The Bhopal Disaster Aftermath OH1 :1

An epidemiological and socio-medical survey

THE BHOPAL DISASTER: ITS AFTERMATH (contd)

Introduction

The disaster that took place on the dark, wintry night of 2/3 December 1984 in Bhopal is the worst man made environmental accident in recorded history. The shocking, official estimates of 2500 human deaths, an equal number of dead cattle and the physical and mental disablement of over two lakhs people, by a mixture of toxic gases including Isocyanate (MIC), do not adequately express the tragedy that has occurred.

The relief efforts, initiated immediately, were handicapped and hampered by the lack of authentic information on the nature of the gases released, by the unwillingness of the Union Carbide to release information and by the lack of relevant information among the State and Central authorities.

The doctors at the Hamidia Hospital, Bhopal, where hundreds of the victims rushed, were faced with an acute emergency which they never anticipated, of whose exact nature they had no inkling, and for the treatment of which they had no ready sources of information.

Since the nature of the toxic gases released into the atmosphere had not been made public either by the Union Carbide or by the Centre (which sent high level technical experts to Bhopal), this had to be a conjecture based on reason and visible evidence.

Soon, two theories emerged to account for the varied symptomatology and stunning mortality of the victims. The

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development and testing of these theories, had they been done properly, would undoubtedly have added immensely to scientific knowledge. What is more important is that it would have relieved the sufferings of thousands of people. The local realities have, however, revealed the power struggles in the medical community and how it ignores in the process, the victims; the lack of human concern leading to withholding of probable proper treatment; the indifference of our medical and scientific community to communicate with our largely illiterate but not unintelligent masses.

The Two Theories

The protagonists of the first theory, the 'pulmonary' theory believe that isocyanates of which MIC is one, damages only those tissues with which they come into direct contact and cannot be carried by blood to internal tissues and organs. Thus MIC can damage only the lungs, eyes and skin and this explains the predominant involvement of the eyes and lungs in the Bhopal victims. They also believe that symptoms, if any, related to other systems must be due to hypoxia caused by lung damage. This theory is strongly supported by a dominant faction in the Gandhi Medical College, Bhopal. They believe that early deaths were due to carbon monoxide poisoning--one of the constituents of the released gases. They adamently refuse to examine any alternative theory.

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This theory cannot fully explain the varied symptoms of the victims; nor the fact of multi-systemic involvement without lung involvement seen in many patients. While another isocyanate, TDI has been shown to cause brain damage, the protagonists of the present theory are silent as to why MIC cannot do so, too. Public Health specialists in the U.S. say that even this can lead to permanent lung involvement and blindness. This is in contrast to the Union Carbide which maintains that MIC can have no lasting damaging effects.

The main protagonist of the second theory, the 'Enlarged Cyanogen Pool' theory, is the Indian Council of Medical Research (ICMR). In fairness to this body, it must be stated at the very outset that it does not reject the first theory but believes that both have important roles to play in explaining the varied symptomatology.

This theory stemmed from the observation that the tissues and blood of the dead victims were bright red in colour. This occurs both in cyanide and carbon monoxide poisoning. Haematological (blood) studies by ICMR ruled out the possibilities of carbon-monoxide poisoning. No doubt that carbon monoxide is also one of the breakdown products of MIC at high temperature. However, being lighter than air it travels upwards and hence cannot kill in an open atmosphere.

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Cyanide on the other hand might have been inhaled directly as hydrogen cyanide or might have been released in the body after the breakdown of the MIC molecule.

There is normally a small cyanogen pool in the body formed by the generation of small amounts of cyanide during normal metabolic processes. These cyanide radicals are converted into relatively harmless thiocyanates by a liver enzyme called rhodanase. Cyanide easily prevents the utilization of oxygen by cells of all the tissues, resulting in rapid death.

The protagonists of cyanogen pool theory believe that MIC in the body gets attached to the haem-globin and slowly releases cyanide to the enlarged cyanogen pool of the body. In these circumstances, its conversion to thiocyanate by rhodanase, can be accelerated by administration of sodium thiosulphate (NaTS). This is the rationale behind using NaTS as an antidote for cyanide poisoning. The resultant thiocyanates are excreted in urine, and this can be used to test the proposed theory itself.

The ICMR conducted a double blind clinical trial in January. Majority of patients who received NaTS showed significant improvement and 10 out of the 19 patients showed an eight fold increase in urinary thiocyanate levels. Unfortunately, and due to reasons best known to itself, the ICMR has not made the details of the findings of this crucial trial, public. The opponents of the theory too claim to have conducted a trial - not double blind - which they say does not confirm the hypothesis. They too have withheld their findings from public

~~scrutiny.~~

The Study by mfc

The mfc had decided at its annual meet held at the end of January 1985, to respond to a series of appeals from various non-governmental organisations (NGOs) and citizen's forums to undertake an epidemiological investigation, so as to support the victims and the NGOs in their struggle for proper relief and a more meaningful rehabilitation process. Some members of mfc visited Bhopal in mid-February to assess the situation and the actual epidemiological survey was conducted between 18-25 March 1985 by 11 members of mfc and 3 friends from the Baroda Medical College.

It must be admitted that the mfc had neither the human power nor the material resources to launch a full scale investigation. Our initial, fact finding survey revealed (i) official secrecy regarding all information on the disaster; (ii) absence of open scientific debates; (iii) lack of encouragement to NGOs. The mfc therefore decided to (i) make an epidemiological assessment of the current health status and health problems of the people; (ii) to examine the findings in the light of the two controversial theories; (iii) to evolve a critique of the medical research and relief programme; (iv) to make recommendations for a more meaningful relief and rehabilitation policy.

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The ICMR summaries of research undertaken and press releases available to us were inadequate and sketchy. We decided that we would go primarily by the broad range of symptomatology with which the patients in the community were presenting. We supplemented this by a thorough physical examination and undertook haemoglobin estimations and lung function tests. A criticism against this approach of reliance mainly on symptoms could be that it lacks objectivity. However, we believe that a thorough study of symptoms is a perfectly valid method of study as has been accepted in a whole range of medical conditions like chronic bronchitis, ischaemic heart disease, arthritis etc.

The study population

Two slums were selected for the study: (i) J P Nagar ~~was~~ situated in ~~the~~ close vicinity of the Union Carbide factory and the worst affected by the gas leak. (ii) Anna Nagar, 10 km away with the least exposure, which served as the control. There was no area which was similar to JP Nagar in socio-economic and environmental characteristics and yet escaped exposure and, therefore, Anna Nagar with the least exposure was the best control that could be chosen.

Rapport was established with the people by explaining to them our objectives and making it very explicity that we were

not there to offer any financial compensation, medical treatment etc. The slum dwellers were given a hand out in Hindi explaining the role of mfc and a commitment was made that the salient findings of our study and our recommendations would be made available to them.

Sample Selection

The families for the study were selected by random sampling. Only subjects above 10 years of age were selected. Those less than ten years were excluded in view of their probable inability to report symptoms correctly. All details were entered in a pre-designed proforma. In addition, lung function tests were done by standard procedures using a portable spirometer by a doctor fully familiar with measuring these under field conditions.

Observations

The two slum populations were similar in age and sex composition, in the number of smokers and of people with long standing respiratory problems like asthma, Tuberculosis etc. The JP Nagar residents who were the more affected, were slightly better off economically but this is of no significance in so far as morbidity rates in JP Nagar are concerned. (For details of actual figures, see our Report).

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The subjects described a broad range of symptoms. Each symptom was described in such graphic detail that it was obviously based on the patient's own experience and could not be malingering or wild imaginations as some are apt to allege. Since these symptoms could arise due to different causes and since the residents of Anna Nagar, the controls, were also exposed to the gas, albeit to a small extent, the latter also reported those symptoms. However, JP Nagar residents had statistically highly significant incidence of these symptoms.

The commonest symptom was breathlessness on accustomed exertion. In addition, they complained of cough, chest pain, blurred vision, head ache, fatigue, loss of memory for recent events, abdominal pain, nausea, watering of eyes and impotence. It is important to note that this survey was conducted more than three months after the disaster, and still the victims suffered with so many effects. Moreover every individual in the JP Nagar sample reported atleast one serious symptom but many in the Anna Nagar sample did not report any such. Probably the most crucial finding of significance was that 35% of the patients had gastrointestinal, central nervous system and eye symptoms but no lung findings, which favours very greatly the possibility of a system poisoning rather than secondary effects of lung damage.

Women in the reproductive age group reported menstrual irregularities such as shortened menstrual cycles, altered pattern

of discharge, pain during menstruation and excessive white discharge. These symptoms were compared not only between the two populations, but also with respect to the pattern in the same group before the gas disaster. It was found to be significantly different

Nearly half of the nursing mothers in JP Nagar reported lactation failure (decreased quantity or stoppage of milk flow)

Salient Findings of the Study

Comparison of symptoms reported in JP Nagar / Anna Nagar

(expressed in percentage)

Number of cases are shown in bracket

Sl No	Symptom	J P Nagar	Anna Nagar	P Value
1	Breathless on usual exertion	87.16 (129)	35.50 (49)	$\ll$ 0.001
2	Chest Pain/tightness	50.0 (74)	26.08 (36)	$\ll$ 0.001
3	Weakness in extremities	65.54 (97)	36.95 (51)	$\ll$ 0.001
4	Fatigue	81.08 (120)	39.85 (55)	$\ll$ 0.001
5	Anorexia	66.21 (98)	28.26 (39)	$\ll$ 0.001
6	Nausea	58.10 (86)	16.66 (23)	$\ll$ 0.001
7	Abdominal pain	53.37 (79)	25.39 (35)	$\ll$ 0.001
8	Flatulence	68.91 (102)	25.36 (35)	$\ll$ 0.001
9	Blurred vision/ photophobic	77.02 (114)	38.40 (53)	$\ll$ 0.001
10	Abnormal distant vision	42.0	21.88	$<$ 0.001

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Salient findings contd....

Sl No	Symptoms	JP Nagar	Anna Nagar	P Value
11	Loss of recent memory	45.27 (67)	11.59 (16)	0.001
12	Ingling & numbness	54.72 (81)	20.28 (28)	0.001
13	Headache	66.89 (99)	42.02 (58)	0.001
14	Muscle ache	72.92 (108)	36.23 (50)	0.001
15	Anxiety/depression	43.92 (65)	10.14 (14)	0.001
16	Impotence	8.10 (12)	0.72 (01)	0.05
17	Hemoglobin (M) (mean gm%)	14.68 (1.79)*	12.70 (1.35)*	0.01
18	Hemoglobin (F) (mean gm%)	12.7 (1.46)*	10.79 (1.34)*	0.001

PFT's —

* Standard deviations

8% of the men reported impotence.

The number of pregnant women in the sample is too small and ~~we intend to~~ study pregnancy outcome ~~separately~~. To come to any conclusion about the effect of the exposure on the outcome of pregnancy we are conducting a detailed study in September 1985.

Many residents had symptoms of anxiety, and some had frank depression. Many had loss of memory for recent events.

Mean pulse rates and respiratory rates were not significantly different in both sexes in JP Nagar and Anna Nagar. Mean hemoglobin concentrations in both males and females were significantly higher in JP Nagar than in Anna Nagar, ~~pointing to~~ suggesting that compensatory mechanisms in the body had begun to respond to the hypoxia (decrease in circulating oxygen).

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The mean values of lung function tests were lower in JP Nagar as compared to Anna Nagar particularly in the age group 15-44 and 45-60. The pattern was primarily restrictive.

An important findings of grave significance is that 65% of the working persons in JP Nagar experienced a drop in income from 20% to 100% as opposed to 9% in Anna Nagar. This reflects the way in which physical/mental disability of the people has affected working capacities.

The causative factor

The presence of such varied symptoms suggests the involvement of more organs and body systems than the lungs alone. These cannot be explained by the pulmonary theory alone even though pulmonary lesions can cause peripheral hypoxia and hence muscular fatigue and so on. On the other hand, the cyanide theory can better explain the varied and apparently unconnected symptomatology. However, the ICMR has not tested the hypothesis vigorously. It has studied only the seriously ill, hospitalised patients and concentrated mainly on the lung symptoms. They do not say whether the non-pulmonary symptoms (symptoms not related to lungs) were also relieved by NaTS and curiously has not made its findings public. One, therefore, may also question whether the cyanide theory is fully valid.

It must be stressed here that the mic is not rejecting the cyanide theory. It is only to point out that the country's main medical research body has failed to be rigorously scientific in testing its own hypothesis.

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Sodium thiosulphate therapy

We have already explained how sodium thiosulphate (NaTS) will help remove cyanoparic radicals from the body. If the ~~cyanide~~ enlarged cyanogen pool theory has been established, even as one of two causative factors the victims should receive NaTS treatment. Some of the local doctors and homoeopaths availed themselves of this therapy after the cyanide theory was proposed, ~~though~~ yet the affected people in the basties were not given the drug.

The ICMR at a meeting held on 4 Feb 85, issued guidelines for NaTS treatment. The medical group of Bhopal, which was opposing the treatment, was also present at the meetings, according to the minutes. Yet they opposed the treatment with the argument that they are not convinced its efficacy. The question is not of a doctor's convictions. A doctor's choice of treatment cannot also be arbitrary. The question is whether there is scientific evidence in favour of NaTS therapy and whether there is equally strong, if not stronger, evidence against the use of NaTS in this situation.

NaTS with its specific action is a better therapeutic agent than the non-specific remedies that are being used for the lung symptoms. A dominant section of the doctors of Bhopal are thus guilty of delaying treatment and by not revealing the findings of its clinical trial, the ICMR too has to accept part of the blame for the continuing suffering of the victims.

After a few weeks of controversy the NaTS therapy has now been accepted but mass detoxification is still being strongly opposed.

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The trial with NaTS is not the only study launched by the ICMR. It has sponsored many other studies on the Bhopal victims, but they lack an integrated approach. Thus lungs, eyes etc., are being examined independent of each other, by different investigators and the ICMR is unwittingly lending support to the first theory, namely, that MIC gas damages only tissues with which it comes into direct contact.

What exactly happened to the gas victims?

So many months after the disastrous gas leak, one still does not know what exactly happened to those who inhaled the gases and are still surviving. This is not because all attempts to unravel the myster, have failed but because an integrated approach had not been taken to do so. Months after the disaster, tens of thousands of the survivers are still suffering from debilitating symptoms which prevent them from going back to work.

The medical community and the officildom have been adhoc in their efforts to render adequate succour to these hapless victims. A powerful medical lobby in Bhopal ~~with unscientific bigotry~~ have opposed NaTS, a treatment, with good potential to the patients. They have no convincing argument for their stand. The IMA, the organisation which has authority over the medical profession, has remained totally mute. The doctors as well as the ICMR have concentrated entirely on those who were hospitalised and have not evolved a holistic, community approach to understanding the problem. The ICMR sponsored local studies with the exception

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of the NaIS trials have lacked the rigour and the epidemiological orientation that are necessary in arriving at a meaningful understanding of the problem.

A point of utmost significance is that the victims of the Bhopal gas disaster mostly belong to the lowest strata of the society and not in a position to fight for their rights, be it medical aid or monetary compensation. It is, therefore, not very surprising that the government and its organisations have shown marginal interest in the after effects. It also reveals a lack of interest among our scientific community in investigating an environmental disaster of an unprecedented nature. On the other hand, one can observe the striking contrast with which all attempts were made to retrieve the Black Box of Kanishka, whose mid-air explosion resulted in the death of only 326 persons but needless to remind of the upper socio-economic class.

Recommendations

Research

1. The research and follow up studies should shift focus from hospital/dispensary based studies of seriously ill patients to family/community based ambulatory patients.

2. Well designed clinical trials should be further initiated using sodium thiosulphate as a therapeutic and epiedemiological tool to further establish the significant role it could play in mass therapy.

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Care, Surveillance and Rehabilitation

3. Psycho social assessment and consequent counselling and rehabilitation are urgently required.

4. Mass treatment with sodium thiosulphate should be initiated maintaining good medical records.

5. A surveillance programme should be undertaken to assess risks to pregnant mothers, unborn babies and new born babies. There should also be close monitoring of the gynaecological problems of women.

6. It is necessary to have a long term surveillance of lung function in view of the postulated damage to lungs and resultant lung fibrosis. Similarly eyes should be examined regularly.

7. A comprehensive listing of all gas disaster victims is a long overdue task necessary for mass treatment, compensation and rehabilitation. This must be done immediately.

Communications

8. There is urgent need for evolving a continuing education strategy for allhealth personnel including doctors working in both governmental and non-governmental centres. These could be through newsletters, handouts and informal group meetings. The areas identified are: (i) sodium thiosulphate therapy; (ii) identification and management of psycho-social stress; (iii) risks to mothers and unborn foetus and need

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for surveillance; (iv) family planning advice till completion of detoxification; (v) role of respiratory physiotherapy; (vi) management of lactation failure; (vii) caution against overdrugging; (viii) need for open minded surveillance of high risk groups; (ix) importance of medical records.

9. There is also urgent need for a dynamic creative non-formal health education of the affected community through group meetings, posters and pamphlets with information and messages built around their life style, culture and existing socio-economic situation.

   The areas identified are: (i) sodium thiosulphate therapy; (ii) ongoing research programmes and informed consent; (iii) risk to unborn and new born babies; (iv) Family Planning advice; (v) respiratory physiotherapy; (vi) management of lactation failure including low cost weaning foods; (viii) importance of records and regular check ups.

10. Occupational Rehabilitation and compensation.

    In the ultimate analysis care of illness, health education, psychosocial counselling would be inadequate measures if they were not backed by adequate monetary compensation and urgent occupational rehabilitation of the disaster victims. This would have to be imaginatively done keeping their previous occupations and the residual disabilities in mind.

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Coordination

11. The government machinery alone cannot handle such a massive task. The government must adopt a policy of enlising the help of all non-governmental agencies and groups wishing to work in Bhopal. This enlistment must be active and supportive.

and finally

12. It is imperative that the victims as well as the entire country must be provided with all the details of how the accident occurred, of the nature of the chemicals released and of the reasons why the detoxification by sodium thiosulphate has been so badly mismanaged.

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NARMADA PROJECT AND THE TRIBALS*

--don't let them drown in despair

PREAMBLE

We are three voluntary groups working since 1980 amongst the tribals who have been ousted from their traditional homelands by the Sardar Sarovar Project on the Narmada river undertaken by the Government of Gujarat. Our experience regarding the problems of these tribal rehabilitees has raised many questions and issues in our minds which we would like to share with you.

When ambitious developmental projects like the Narmada Project are undertaken, a large number of families are asked to leave their lands, houses, familiar surroundings and settle elsewhere. This is justified on the basis of 'public purpose' which is essentially that much larger benefit accrue to much larger populations who would otherwise live at a low standard of living. This, of course, must imply that those who give up everything so that the project can become a reality, must also not suffer a drop in their standard of living. In fact it is necessary that they too improve their lot commensurate with similar improvements in the standard of living of the beneficiaries of the project.

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*An appeal from Chatra Yuva Sangharsh Samiti, Rajpipla
Rajpipla Social Service Society,
Action Research in Community Health, Mangrol,
February 1984.

Our concern is focussed on the social and economic suffering that these oustees are going to face if the governments rehabilitation policy on paper does not match with the actual programme in the field. We have reason to believe that certain ambiguities in policy and trends in implementation seen in that area are going to seriously affect the health, life and future of these people.

BACKGROUND

About 220 villages in the three States of Gujarat, Maharashtra and Madhya Pradesh are going to be affected by the inundation of large tracts in this area. Among them we have worked with the illiterate tribals of 19 villages in Gujarat and most of the facts and issues discussed derive out of this experience. The main occupation of tribals in this area is agriculture. The income from agriculture is supplemented through collection of forest produce, hunting and other forms of labour.

Due to cultural reasons and administrative hurdles at local levels, the overwhelming majority of tribals have joint holdings for generations together. Thus a seemingly large land holding (more than 5 acres) in the name of one person in reality may mean that two or more families are actually earning their livelihood from it.

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There are a few landless labourers. Even these earn their livelihood by cultivating government owned fallow forest land or other waste land. It is not possible to estimate the amount of land brought under such cultivation since this is treated as unauthorised cultivation. Whatever the technicality of such a situation, it is an established fact that they have been living and cultivating such lands for many years and they are entitled to compensation for the loss of this only source of living.

Water Dispute Tribunal

A tribunal was set up to study the problems arising out of the project implementation. Its Award took into account the factors of total dependence on land of these tribals as well as the responsibility of the rehabilitation programme in removing the poverty of the 'oustees'. It, therefore, provided land for land compensation (ie., if more than 25 percent of land owned by any family was acquired for the project they were to be given an equal amount of irrigable land). It also made a specific provision of a minimum of 5 acres of irrigable land to all those whose land holding was less than 5 acres of land. It also stated that first preference would be given for rehabilitation within the command area of the project. However, if the 'oustees' did not want to settle there, they were to be settled in their respective states on a similar basis. In addition, it was decided that the agricultural lands were not be provided free of cost,but the occupancy price should be recovered from them in 20 yearly interest free

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instalments; the first instalment being half the compensation received by the 'oustees'. The tribunal has also made the proviso that if an individual is losing atleast 75 percent of his land he should have the option of surrendering the rest of his land as well. This award on the face of it seemed a relatively fair policy and it was hoped that with proper implementation, it would tackle all the grievances of the tribals.

Some ambiguities

1. The Narmada Tribunal being an inter-state water dispute tribunal took into account the rehabilitation of oustees from Madhya Pradesh and Maharashtra. No reference was made to those from Gujarat. Clearly the that which applies to Maharashtra and Madhya Pradesh should apply to oustees of Gujarat as well; but the government of Gujarat has not earmarked any land in the command area of the project for this rehabilitation. Even though these tribals will be the first to be affected they are expected to buy their own land and only after they do so, other basic amenities like wells, schools and roads are to be provided.

2. There is a government resolution (G.R.) of June 1979 of the Irrigation Department of the Government of Gujarat which states that a minimum of 5 acres of land should be provided for each oustee family for their rehabilitation. There is of course no provision to produce irrigable land in the command area of the project in this resolution.

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3. In the cost estimates in the project report prepared in 1980, the Government has made no provision for the cost of acquiring the total of 40000 acres of land that it would have to acquire to rehabilitate all the oustees. It has instead provided for only 1310 acres of land which is clearly meant for roads and house sites only, whereas in fact the agricultural land to be acquired should be clearly more than the actual amount submerged, especially because a significant proportion of oustees who own less than 5 acres of land are to be provided atleast 5 acres as per the directive of the tribunal. The argument that the land is not to be given free of cost, and hence its cost of acquisition need not be mentioned is not tenable, since clearly some initial investment will have to be made by the government. No mention seems to have been made in the proposals on this matter.

4. The usual analysis of land records do not show the reality of land holdings in a tribal region where joint holdings are significant. We reanalysed the land holdings not by the units as they are officially recorded but by the number of families who are actually dependent on the land. We found that there were 624 land holdings in the 18 villages surveyed whereas the number of families actually dependent on them are 2109, since most are joint holdings. Also if

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their whole social and cultural life disrupted. Naturally they rejected such outrageous propositions.

ii. When the oustees refused the land as was the experience of 5 villages which are affected by Rock filled dykes the government asked them to purchase land from private land owners from their compensation money. The consequences of this were: (a) the oustees with less than 5 acres of land could not buy 5 acres of land which they were entitled to; (b) some could not even buy the land equal to what they had been originally cultivating; (c) the amenities that they were entitled to could not be had asper the law because of the wild scattering.

iii. It has not been possible for the illiterate tribals to deal on an equal footing with the high caste land owners in these land deals thrust upon them by t e governments inaction. They have been/are bound to be cheated and exploited. In the beginning the Additional Collector gave active help and the deals were made relatively easily. However, even now these deals are not yet xompletely registered and the ownership of lands still rests with the original land owners. The whole matter depends on the goodwill of the concerned government official and in our experience there has been a general aloofness and reluctance on their part to get too involved.

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iv) The Government of Gujarat has been pleading from the very beginning that it is virtually impossible to give agricultural land to the oustees as no surplus land is available. Forest lands cannot be given as only a few forests are left and other waste lands are scarce. However the government could instead purchase or acquire private lands in large tracts so that proper rehabilitation can be carried out and rehabilitation of the oustees itself could be proclaimed to be a 'public purpose' in this respect.

v) In some villages the situation has arisen where 80% of the village land has been acquired. The land acquisition authority is refusing to acquire the remaining 20% of the land. This land is owned by 20-30 families;of these families, 4-5 of them happen to retain practically all their lands. If these lands are not to be acquired the isolation of these families will be near total. They will be cut off physically, socially and culturally. They will be deprived of other amenities like school, schops and health services. The land acquisition authority is taking a very rigid view of the land acquisition procedure which states that only land under submergence can be acquired.

vi) All the tribals in the affected villages of Gujarat have so far voluntarily surrendered their lands and other villages are ready to follow suit. There is, however, still widespread uncertainty about the amount of compensation they would be paid. Since all the villages are

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losing their land under the same project, it is only fair that they should get a uniform and generous compensation. The State Government has not yet made up its mind and is vascillating on the issue.The traditional way of computing the amount of compensation by taking current market values for the land is clearly not applicable to tribal land since for many years no sale deeds have been concluded in this area. In its cost estimates the government has put an estimate of average cost of land acquisition at a rate of Rs.2000-00 per acre. This is totally unsatisfactory. This computed average compensation is so low that most of the villagers will be deprived of their land in exchange for a paltry sum insufficient to buy adequate alternative land to survive. The consequence of such myopic action of the government can only be that the 'oustees' will become poorer.

To summarise the situation as it exists today, the government has abandoned its primary responsibility of providing land for land as directed by the tribunal; it has offered a totally inadequate sum of compensation; it has shown no aptitude or willingness to keep down the prices of the surrounding private lands which may have to be acquired by the oustees; it has used 'holding' as the unit and not 'family' in its computation thus hiding a concrete reality of the tribal regions; and its grave lack of responsibility has

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resulted in the ousted tribals becoming a prey to high caste land owners and their brokers. In brief with a policy implementation that is insensitive to the human problem of these tribals it has made the tribunals recommendation of the objective of abolition of poverty of the oustees a myth.

Among others the main claim for this inadequate implementation of the tribunals recommendation has been the cost factor. The government has claimed that land acquisition of the type recommended will push up the cost of the project. The following facts about the governments lop-sided sense of proportions is cost provisions for certain other aspects of the project is not only disturbing but show how hollow the claim is in the first place.

The project report shows that the cost estimate of the whole rehabilitation programme including compensation for lands, houses etc., in all the 220 villages in the three States is Rs.19.83 crores, while the cost estimates of the construction of the staff buildings at Kevadia Colony alone will be Rs.23.45 crores. Added to this cost the miscellaneous and establishment costs of the same magnitude and we begin to sense how distorted is the government's sense of proportion. The scales which the government holds in its hand give more than twice the weigh age to the temporary needs of providing houses only to a few hundred families of the project in one colony alone. In comparison to this, the entire rehabilitation cost of 10 to 12 thousand families scattered over 220 villages of the three States, which can make or mar their entire lives, gets less than half the weightage. What is more surprising that

the staff quarters, the water lines, t e electricity lines, guest homes, the roads in the Kevadia Colony have already been built while the staff are yet to arrive. In contrast the notices to acquire lands from the people to be ousted has already been served but the vital issue of compensation for imminent land loss is still unresolved.

> In acountry like India, with a high po ulation density and high level of poverty, virtually every ecological niche is occupied by some occupational or cultural human group for its sustenance. Each time an ec logical niche is degraded or its resources appropriated by the more powerful in society, the deprived, weaker sections become further impoverished. For instance, the steady destruction of our natural forests, pasture lands and inland and coastal water bodies has not only meant increased economic poverty for millions of tribals, nomads and traditional fisherfolk, but also a slow cultural and social death: a dismal change from rugged self-sufficient human beings to abjectly dependent landless labourers and squalor-stricken urban migrants. Current development can in fact be described as the process by which the rich and more powerful reallocate the nation's natural resources in their favour and modern technology is the tool that subserves this process.
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> ---The State of India's Environment, 1982

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Suggested Changes in Policy

Having worked with the tribals in the 19 villages of Gujarat since 1980, we as a group have just begun to understand the problems from the perspectives of the tribals. We suggest the following changes in the policy of rehabilitation to make it sensitive and relevant to the socio-economic, cultural, psychological and ecological needs of these oustees who are as much the 'public' as any other.

i. The ambiguities in the rehabilitation proposals must be clarified immediately
   -- the rehabilitation of displaced people in all three States is of equal importance;
   -- the land for land compensation with the minimum provision of 5 acres should be implemented efficiently;
   -- the land offered being irrigable, agricultural and as far as possible within the command area of the project and preferably in their respective States.

ii. The government should either buy or acquire private lands in large tracts as it does not have any surplus within its own possession. Under no circumstances should the oustees be asked to purchase their own lands.

iii. The government should take a family as a unit (and not 'holding') and provide minimum of 5 acres of irrigable land to each oustee family irrespective of the fact whether they legally own their land or not or the government can conduct a detailed survey in the villagesto find out the amount of forest land and/or other waste lands

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they are actually cultivating and provide them with alternate land in appropriate amounts.

iv. Not only must individuals whose land is acquired more than 75% be given the option of surrendering the rest but this rule must also be extended to the villages as a whole as well if they are going to loose atleast 75% of their total agricultural land.

v. The compensation for land acquired must be adequate and sufficient to buy new lands of atleast equivalent size keeping the basic objective of rehabilitation ie., abolition of poverty of the oustees in mind.

The forest issue

Following our recent contacts in the interior villages of Gujarat and Maharashtra, we have come to understand another important dimension of our main demand of land for land, although this factor was not totally lost sight of by us earlier. The tribals, especially Dungari Bhils who, through centuries have lived in these forests, cannot simply imagine that they can survive outside the forests. Forest is so much a part of their life that outsiders like us who are quite sympathetic also cannot fully appreciate or understand their attachment to the forests, let alone the aloof and faceless bureaucracy of the Narmada Project.

For these tribals, forests are not on y the fountain of their material needs, but also an integral part of their social,

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cultural and religious life. You have to be with them to understand what forest means to them. No wonder that the inhabitants of these villages are refusing to move outside the forest area. Even those oustees who agree to resettle on non-forest lands, would opt for forest land, if the option is offered to them.

Why is then the Government so adamantly refusing to release forest land for resettlement? The re-evaluation of the government's blanket policy with regard to forest land is urgently called for. The Government's argument is based l on the laudable objective of conservation of forests. The plausibility of this argument is only superficial. It is clear now that the Government itself is destroying large tracks of forest in the name of 'development'. It also sanctions felling of the forest in the name of 'satisfaction of market needs'. Only when it comes to giving forest lands to tribals, the Government raises the bogey of 'Conse vation of forests'. Hidden in this argument is the unfounded charge against the tribals that they are the culprits behind the massive deforestation. The facts if examined closely would reveal exactly the opposite story. The tribals have in fact protected the forests and their needs of forest wood is so small in comparison to the unsatiable hunger of the urban centres for timber that it is sheer perversion to say that they are destroying the forests.

We therefore make a demand that these tribals must be resettled in forest land only so that t ey can preserve their forest bound culture and way of life. In raising this demand we are not at all pitting ourselves against the 'conservation of forest'objective.

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If forests are in danger, the tribals living in forests are not basically responsible for it. The objecgive of 'conservation of forests' and that of resettling the tribals in forest are not incompatable. Indeed they could with imagination become complementary objectives. We, however, challenge these so-called developmental schemes which are both destroying forests and the defenceless tribals.

C O N C L U S I O N

Through this note we have tried to bring to your notice the plight of these oustees of the Narmada project andthe ambiguities and insensitivity of the government rehabilitation policy. Our attempts to bring about appropriate changes in the policy have made very little progress. We need your help to tackle this problem. We must act and act decisively if we do not want to let the tribals down in despair.

> With their forest-based life styles destroyed,
> the tribals are becoming human cannon fodder
> for the country's modernization: poor, unskilled
> and viciously exploited.
>
> --The State of India's Environment, 1982

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OH1 : 3 537 words

"THE TEMPLES OF MODERN INDIA"

-- A summary of the environmental and health hazards of dams

BENEFITS

Electricity for industry (half our total electricity produced today)

Water for irrigation (26.6 million hectares (1979-80) of surface irrigation potential)

INVESTMENT

From 30 dams in 1947 to 1554 in 1979.

From storage capacity of 13650 million c. metres to 173000 million c. metres (1979).

Rs.10,566 crores invested until 1979 ie., 14% of total planned expenditure.

HAZARDS

Evacuees

Large numbers of people/villages are moved - mostly tribals, adivasis and forest dwellers. Thirteen dams produced 5.77 lakhs and 430 villages of 'oustees'. Rehabilitation usually insensitive to socio-economic, cultural and psychological complexities of process.

Silting

Reduces life span of dam, ~~and~~ corrodes banks and affects aquatic flora and fauna downstream. Water loses fertilizing value. Siltation rates have varied from Bhakra (50% increase) to Nizam Sagar (AP) - (1600% increase.)

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Earthquakes

Correlation well established. Have been reported with respect to Mangalam (1963), Parambikkulam (1963), Koyna (1967) Kinnerjani (1969), Mula (1972), Idukki (1977 & 1978) and also Ghirni, Sharavati, Sholayar and Ukai dam.

Water logging

Estimated that 6 million hectares of cultivated land is severely affected by water logging due to ~~increase~~ rise in ground water table.

Soil salinization

Estimated that 7 million hectares of fertile fields are affected by soil salinity affecting crops and plant growth.

Health

Increase in mosquito/fly populations. Spread of malaria filaria and Japanese encephalitis (well documented for Thungabhadra and Sathanur dams). Changes in levels of fluoride, calcium and trace metals due to rise in ground water table causes crippling diseases like genu valgum (knock knees) and fluorosis. Reported from Nagarjunsagar and Parambikkulam Aliyar dams.*

Forests

Loss or sharp reduction of forest cover affecting flora, fauna and ecological balances. Between 1951-1972 estimated loss of 4 lakh hectares of forest lands.

* In Egypt ~~Sudan~~ the Aswan High Dam and associated canals have lead to the spread of Bilharzia transmitted through snails (schistosomiasis) and in Africa the Volta Dam is believed to have led to the spread of river blindness (onchocerciasis)

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Ecology

Apart from deforestation, river and reservoir silting and health hazards already mentioned, ecological changes include - increased water evaporation affecting micro-climate; proliferation of harmful acquatic weeds (Eupatorium, Lantana, Milkania) which smother acquatic growth, are unpalatable to herbivores, increase fish mortality and choke feeder systems, irrigation outlets and block hydro-electric installations.

Floods

Destruction of vegetation which retains water during the rainy season, adds to flood problem. Excessive discharges to avoid damage to dams, causes flash floods downstream (Hirakund, 1980).

Financial drain

The cost escalation over initial cost estimates for important projects in India have varied between 150-1300 percent. ~~cost escalation~~. Apart from the additional drain on the exchequer, cost reduction exercises are affected by inadequate provisions for rehabilitation of oustees and inadequate accounting of costs due to loss of forests, farm lands, fishing and ecological changes.

ALTERNATIVES TO LARGE DAMS

Mini and micro hydro electric plants.
Bio-mass plants utilizing forest produce
Wind and solar energy conversions
Use of small dams, barrages and bunds
Revival of traditional tank irrigation systems.

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PEOPLES PROTESTS

Tehri Bundh Virodhi Sangharsh Samiti

Silent Valley Project protest spearheaded by KSSP.

Bedti Project opposition.

Save Munnar Agitation (Munnar High Dam).

Jungle Bachao, Manav Bachao Andolan (Inchampalli Dam).

Mitti Bachao Abhiyan (Save the Soil campaign).

TO DAM OR NOT TO DAM IS THE QUESTION

--Summarised from 'Temples of Modern India' by 'Madras group' in Patriotic and People Oriented Science and Technology Bulletin (PPST Bulletin) Vol.4, No.1, June 1984.
(~~PPST, Madras C/o~~ R Vijayalakshmi, 17 South Mada Street, Triplicane, Madras 600005).

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MEDICAL RESEARCH IN BHOPAL

-- Are we forgetting the people?

> Concern for man himself and his safety must always form the chief interest of all technical endeavours. Never forget this in the midst of your diagrams and equations.
>
> --Albert Einstein

Preamble

In a tribute to the medical relief workers involved in service to the Bhopal disaster victims the ICMR has noted (1) that 'a disaster of such magnitude, of such suddenness and caused by the release of a highly toxic chemical methyl isocyanate (MIC) into a densely populated habitat is unparallelled in human history. The doctors, medical students, civil servants, governmental, public sector and voluntary bodies and the people themselves rose to the occasion in a human gesture equally unparallelled'...

In the absence of authoritative information on the released gas; the unwillingness of the company to part with authentic information; the unpreparedness of the local bodies and the government health authorities to understand the consequences of the disaster; and the absence of technical or toxicological expertise on MIC among our scientific community, it was imperative that a national body like the Indian Council of Medical Research through its own initiative would have to harness

the scientific medical expertise in the country including the local medical college community to meet this challenge. Considering that the affected population was over 2 lakhs and that the dead were over 6000 (though official estimates are 2000!) this research initiative had to be equally unparallelled in meeting the phenomenal challenges of the world's worst recorded ecological disaster. Do the records of events in the past four months since the disaster bear this out?

The Plan

A report on the first nine days of the Bhopal disaster identified [1] three objectives for the ICMR's research programme:

1. To establish a clinical and patho-physiological profile of the hazard which would also provide clues for improved patient management and clinical outcome
2. To study the long term sequetae of toxin exposure to lung, tissues, foetus, genes and cancer induction
3. To obtain a basic understanding of the biological alterations associated with MIC exposure.

Strangely enough there is no mention in this report of a strategy by which conclusive research data as and when available would be transmitted to the relief and rehabilitation effort in Bhopal, ie., to the treating doctors and through a health education effort to the affected public.

A report of projectization of ICMR supported research effort [2] lists out 17 study projects which covers acute and long term

health effects, lung functions, follow up of children aged 5-15 years, occular changes, pulmonary and neurological changes, growth and development of new borns, clinical and forensic toxicological studies, pathological and microbiological investigations, radiological studies, biochemical and immunological studies, carcinogenicity, mutagenicity, teratogenicity and chromosomal changes, data managment information system, hospital based cancer register, cytofluorometric studies and blood gas analysis. The studies ranging from a time span of 6 months to 5 years would incur a total financial outlay of 1.07 crore rupees.

Some surprising omissions in the list were the assessment of psychological stress and its manifestations in the affected families, studies on health of women (not obstetrical outcome but gynaecological effects) and the assessment of medico social effects like reduced earning capacity and functional disability which would affect rehabilitation efforts. Though there were references to an epidemiological and community based outlook the research endeavour atleast as on paper did not seem to be a coordinated holistic effort on understanding the total problem but basically a series of vertical research programmes initiated and funded according to the interests of the professors involved in the exercise.

Results

It is four months since the tragedy and about three months since many of the research programmes got underway.

As far as a communication strategy goes three press releases

and two lectures by the director generals and a minutes of the meeting on the thiosulphate controversy are the only freely available literature on the research (3-8) efforts. From these all that any member of the scientific community or the general public can gather are:-

i. that there is no evidence of irreversible eye damage or blindness

ii. that the autopsy findings are indicative of severe respiratory damage caused by pulmonary odeme and asphyxia

iii. that studies of exposed persons with lung symptoms/ signs have shown obstructive and or restrictive abnormalities

iv. that a double blind clinical study undertaken using sodium thiosulphate and a placebo has established that sodium thiosulphate administration results in symptomatic improvement and in increased excretion of thiocyanates in the urine. On the basis of clear cut results, the State government has been advised to administer sodium thiosulphate to the exposed population and detailed guidelines have been drawn up and circulated.

v. that two visiting psychiatrists have found that 10-12% of the affected individuals attending the medical clinics in Bhopal are presenting with psychiatric manifestations-- symptoms of anxiety and depression are foremost.

Why this secrecy? or is it administrative over caution?

A more updated report prepared in mid March collating all data as of that date has again become a casualty in the commitment to secrecy (caution!) and no press release has followed.

Issues of concern

An mfc fact finding team which visited Bhopal in mid February at the request of various non governmental agencies and action groups published a report on the realities of medical research and relief which has been widely circulated and is now well known [9]. In mid March an mfc team of 16 members camped in Bhopal and undertook an epidemiological survey which included detailed history taking, physical examination, lung function tests, haemoglobin estimation of a 10 person sample of a severely affected area and a control area [10].

The team also met decision makers, relief and service providers, medical teams of voluntary agencies and others, apart from undertaking a survey of the people's perceptions of relief services and an overview of the services itself. The findings of the team are being analysed and will be reported shortly (a press release is published in this issue) but the experience of the third week of March in Bhopal strengthened the findings of the earlier fact finding team and identified a whole series of issues of concern in the ways in which research efforts were becoming exploitation of peoples' suffering rather than expressions of support to programmes of human welfare.

Lack of dissemination of technical guidelines

1. The medical relief services continue to be starved of authentic and authoritative scientific medical information to support clinical judgment and patient management. In the absence of clear cut guidelines from the seniors in the profession treatment continues to be adhoc, symptomatic and unstandardised. Findings of autopsies, lab investigations and x-rays and other tests are not available to the treating doctors. Doctors have not been alerted to the fact that a wide range of symptomatology like fatigability, weakness, memory problems are all part of the MIC syndrome. In the absence of such information peoples' sufferings have often been passed off as malingering or compensation neurosis.

2. Pill distribution

   The treatment basically consists of a whole series of pills which are efficiently and actively prescribed to the people in a sort of conditioned reflex. In the absence of proper record linkages each patient is collecting large amounts of pills and not feeling the better for it apart from the dangers of over drugging. Other form of care and non-drug therapies have not been thought of and counselling.

3. The thiosulphate controversy: Even after the ICMR studies establishing the validity of thiosulphate administration and the preparation of clear cut guidelines for its administration (6, 8) ~~the use of~~ this specific antidote is not being used as effectively as it should be. It has become a casualty in a medical controversy

between ≠ cyanogen and ≠ carbon monoxide ~~lobby~~ lobbies and the victims rather than being informed and helped are being confused and neglected.

4. Women's health: The mfc fact finding team had highlighted the problems of women who have suffered abortions, still births, diminished foetal movements, suppression of lactation, abnormal vaginal discharges and menstrual disturbances. The studies undertaken by two [redacted] doctors of mfc reported in this bulletin(11) establish the magnitude and severity of the problem. It, however, continues to be neglected by the concerned authorities.

5. Absence of Health Education efforts

Whatever the validity of the research efforts, in the ultimate analysis it should get translated into a strategy of health education and awareness building of the affected people. As of date there are no clinical guidelines or efforts in this direction. The range of areas is phenomenal--advice to mothers of the risk to the foetus and preparation for consequences including options for MTP, advice to couples on contraception till detoxification is over, breathing exercises and antismoking advice to those with fibrosis of lungs, avoidance of overdrugging of pregnant mothers, advice to mothers regarding feeding of infants/children due to lactation suppression, availability of [redacted] thiosulphate and other medical relief measures. None of this has even been recognised as being necessary.

6. Poor epidemiological and medico social orientation of problem assessment

The general impression is that research and relief efforts are [redacted] suffering from an acute clinical and institution based orientation

rather than a community based epidemiological orientation.

Only if all data is field based and is related to known available morbidity patterns (or comparison with controls) can early problems and special trends be identified and urgently acted upon. The danger of getting into the pursuit of a very neat and fool proof epidemiological planning exercise can be equally counter productive.

7. Lack of informed consent: The people are not being informed about the tests being done. Nor is consent being taken for being included in the studies or for procedures to which many of them are being subjected to. This is a minimum medical ethic.

8. Lack of coordination: This is a universal problem and the ultimate casualty are the disaster **victims** themselves. This incordination ~~is~~ is occurring between government services and research efforts in the medical college, between the different research workers themselves between government and non-government relief efforts, between voluntary agencies involved in action, relief, rehabilitation and of all these groups with the disaster victims themselves.

While a more detailed report is awaited we in the mfc appeal to government decision makers in Bhopal, medical college professors, ICMR scientists, IMA, voluntary agencies, action groups that there ~~is an~~ are urgent needs to be actively met :

* Need to evolve a bold, imaginative and open communication strategy to all the doctors and health workers treating the disaster victims who are presently starved of authentic technical/medical information hampering clinical judgment

References

1. ICMR, The Bhopal disaster - current status (the first nine days) and programme of research.
2. ICMR, Projectization of ICMR supported Research
3. ICMR, Medical Research problems in Bhopal (V Ramalingaswami)
4. ICMR, Pathology and clinical toxicology of the Bhopal disaster (S Sriramachari)
5. ICMR, Medical research on Bhopal Gas Tragedy--press release 12 Feb 1985
6. ICMR, ibid, press release, 12 Feb 1985
7. ICMR, ibid, press release 27 Feb 1985.
8. ICMR, Thiosulphate therapy in MIC exposed - minutes of meeting at ICMR, 14 Feb 1985
9. mfc, Medical Relief and Research in Bhopal--the realities and recommendations.
10. mfc, Approach document of a project to evolve a strategy of medical relief and rehabilitation which best meets the peoples' medico social needs and expectations.
11. Rani Bang, Effects of the Bhopal disaster on the women's health--an epidemic of gynaecological disease: reports I & II.
12. Lancet editorial, Calamity at Bhopal, 15 Dec 1984.

is consistent with the fact that the IgM mediated primary humoral response subsides with continuing exposure to an antigen. Our finding that both shipyard groups had significantly higher levels of IgG than the Red Cross controls (table 4) is, however, consistent with the idea that asbestos behaves in the body as a persisting antigen. Immunoglobulin A was also found to be higher in shipyard workers than in the Red Cross controls. The immunoglobulin levels in shipyard experimental subjects were similar to those in shipyard control subjects. Of interest here is the observation of Huuskonen *et al* that patients with asbestosis have higher IgA values than controls.[14] They concluded that the increase in humoral immune response could be used for the evaluation of patients with pulmonary fibrosis.

When we looked at Th and Ts cells, both types were present in significantly greater numbers in the two shipyard groups compared with the Red Cross blood donors, the Ts excess being considerably greater than the Th excess (table 3). The greater excess of Ts cells results in Th/Ts mean ratios that are smaller than those found in the Red Cross controls, a condition also found in immunodeficient people. Our findings are similar to those of Lew *et al* who found raised Ts but not raised Th cells in workers exposed to asbestos.[22]

Although statistically significant differences were found when comparisons were made in several of the laboratory tests, their biological importance remain to be determined. It is also recognised that many comparisons were made and that differences can occur as random events and not the result of real effects. Study hypotheses were selected before inspection of the data, and thus results are not selectively reported.

In conclusion, our observations of the humoral immune response as measured by changes in IgG and IgA, the cellular immune response as measured by mitogen stimulation and the disproportionate increase in Ts cells and decrease in Th Ts cell ratios may, one day, serve as clues to the early diagnosis of susceptibility for asbestos caused malignancy. The test to these clues will depend on a prospective study.

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References

1 Copes R, Thomas D, Becklake MR. Temporal patterns of exposure and nonmalignant pulmonary abnormality in Quebec chrysotile workers. *Arch Environ Health* 1985;40:80–7.
2 Finkelstein MM. A study of dose-response relations for asbestos-associated disease. *Br J Ind Med* 1985;42:319–25.
3 Acheson ED, Gardner MJ, Winter PD, Bennett C. Cancer in a factory using amosite asbestos. *Int J Epidemiol* 1984;13:3–10.
4 Liddell FDK, Thomas DC, Gibbs GW, McDonald JC. Factors, exposure and mortality from pneumoconiosis, respiratory and abdominal malignancies in chrysotile production in Quebec, 1926–75. *Annals of the Academy of Medicine* 1984;13:340–4.
5 Rossiter CE, Harnes PG. UK naval dockyards asbestos study: survey of the sample population aged 50–59 years. *Br J Ind Med* 1979;36:281–91.
6 Hilt B, Langard S, Andersen A, Rosenberg J. Asbestos exposure, smoking habits, and cancer incidence among production and maintenance workers in an electrochemical plant. *Am J Ind Med* 1985;8:565–77.
7 Oliver LC, Eisen EA, Greene RE, Sprince NL. Asbestos-related disease in railroad workers. *Am Rev Respir Dis* 1985;131:499–504.
8 Kubota M, Kagamimori S, Yokoyama K, Okada A. Reduced killer cell activity in lymphocytes from patients with asbestosis. *Br J Ind Med* 1985;42:276–80.
9 Lemaire I, Dubois C, Grondin C, Gingras D. Immunoregulatory role of lung fibroblast growth alteration in asbestos-induced pulmonary fibrosis. *Clin Exp Immunol* 1986;66:201–8.
10 Kagan E, Jacobson RJ, Yeung K-Y, Haidak DJ, Suchnani GR. Asbestos-associated neoplasms of B cell lineage. *Am J Med* 1979;67:325–30.
11 Gellein AR, Macy MG, Uthayakumar S, Newland AC, Rudd RM. Lymphocyte subpopulations in bronchoalveolar lavage fluid of asbestos workers. *Am Rev Respir Dis* 1985;132:824–8.
12 Doll NJ, Bozelka BE, Goldbah S, Anorve-Lopez E, Salvaggio JE. Asbestos-induced alteration of human peripheral blood monocyte activity. *Int Arch Allergy. Appl Immunol* 1982;68:302–5.
13 Ginns LC, Ryu JH, Rogol PR, Sprince NL, Oliver LC, Larson CJ. Natural killer cell activity in cigarette smokers and asbestos workers. *Am Rev Respir Dis* 1985;131:831–4.
14 Huuskonen MS, Rasanen JA, Harkonen H, Asp S. Asbestosis as a cause of immunological stimulation. *Scandinavian Journal of Respiratory Diseases* 1978;59:326–32.
15 Hartmann DP. Immunological consequences of asbestos exposure. *Survey of Immunological Research* 1985;4:65–8.
16 Warheit DB, George G, Hill LH, Snyderman R, Brody AR. Inhaled asbestos activates a complement-dependent chemoattractant for macrophages. *Lab Invest* 1985;52:505–14.
17 Fahey JL, Prince H, Weaver M, *et al*. Quantitative changes in T helper or T suppressor-cytotoxic lymphocyte subsets that distinguish acquired immune deficiency syndrome from other immune subset disorders. *Am J Med* 1984;76:95–100.
18 Schwab R, Stalano-Coico L, Welsker ME. Immunological studies of aging. IX. Quantitative differences in T lymphocyte subsets in young and old individuals. *Diagn Immunol* 1983;1:195–8.
19 Wagner MF, Brown DC, Campbell MJ, Coles RM, Edwards RE, Scott MP. Immunological monitoring of an asbestos-exposed population. *Arch Immunol Ther Exp (Warsz)* 1983;30:201–5.
20 Ueki A, Oka T, Mochizuki Y. Proliferation stimulating effects of chrysotile and crocidolite asbestos fibres on B lymphocyte cell lines. *Clin Exp Immunol* 1984;56:425–30.
21 Rola-Pleszczynski M, Gouin S, Begin R. Pulmonary and systemic immunoregulatory changes during the development of experimental asbestosis. *Clin Exp Immunol* 1984;58:325–34.
22 Lew F, Tsang P, Holland JF, Warner N, Selikoff IJ, Bekesi JG. High frequency of immune dysfunctions in asbestos workers and in patients with malignant mesothelioma. *J Clin Immunol* 1986;6:225–33.
23 Kagan E, Jacobson RJ. Asbestos exposure, immune stimulation and lymphoid malignancy. *Advances in Pathology* 1982;2:549–53.

British Journal of Industrial Medicine 1988;45:469–475

Exposure and response to methyl isocyanate: results of a community based survey in Bhopal

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ABSTRACT In the two weeks immediately after the Bhopal disaster a community based survey was carried out in a series of eight exposed and two non-exposed clusters of households. The primary concern was the effect of the gas (subsequently identified as methyl isocyanate) on the eyes of the victims but data were also sought on respiratory status and the first symptoms of the exposure. No case of blindness was encountered that could be attributed to the gas. The most frequent symptoms reported were burning of the eyes, coughing, watering of the eyes, and vomiting. Among these, the frequency of cough most closely followed the rate of death in the different clusters. Although much greater overall, the frequency of reported diarrhoea appeared to bear a stronger relation to death rates. Reports of photophobia and the clinical finding of superficial interpalpebral erosion of the cornea were more frequent where the death rates were lower. This clinical and epidemiological picture is consistent with different effects of the gas at different doses (as estimated from distance from the factory).

The release of a gas cloud from the Union Carbide factory at Bhopal, India, on the night of 2/3 December 1984 resulted in numerous deaths and injuries among the surrounding population. It is now believed that the immediate cause of the disaster was that water entered a tank where 41 tonnes of methyl isocyanate (MIC) were stored, causing a runaway chemical reaction. The heat of the reaction, possibly augmented by reactions with other materials present in the tank as contaminants, produced vapourisation of such momentum that it could not be contained by the safety systems, which may in any event have been defective or of inadequate capacity (S Varadarajah, report to the Council of Scientific and Industrial Research, India, 1985). There is still considerable confusion about what effects among the survivors can be attributed to exposure to the gas. In the two weeks immediately after the disaster a team of Indian and British doctors carried out a formal community based epidemiological study of exposed and unexposed populations before the mass exodus from the city.

Methods

Several of the worst cases in two hospitals were examined in the days immediately after the disaster to obtain an initial indication of the nature and severity of effects among survivors. Results of slit lamp examination by two ophthalmologists were reported earlier.[1,2] A preliminary assessment of the area of exposure and the effects suffered was made by talking to people who lived at varying distances and directions from the plant. From this it was possible to map out preliminary exposure zones according to reports of human and animal deaths, symptoms, and perceptions of the presence of the gas. Cases seen in the community before the start of formal survey were not included in the study, although they provided valuable information regarding the limits and nature of the exposure.

Eight housing clusters were selected with the exposure zone, covering a range of different severities of exposure as judged by the preliminary inquiries. A household survey was carried out in the second week after the release of the gas. Two non-exposed clusters were selected, having apparently similar socioeconomic status to the exposed groups but located 14 and 17 km away from the plant, where no one reported symptoms of gas exposure within several kilometers. Starting with one arbitrary selection, houses were contacted consecutively in each cluster, moving from door to door with a questionnaire drawn up for the purpose. All inhabitants present in each dwelling at the time of the visit were examined following a standard format. The British doctors used an interpreter. The initial survey lasted from 5 to 12 December, after which time the entire exposure area was

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deserted as people left in case the plant was restarted to "neutralise" the remaining stock of MIC.

The first survey day was spent by the participating doctors practising the administration of the questionnaire and the examination in an attempt to ensure consistency. The questionnaire inquired about name, age, duration of exposure based on experience of acute symptoms and presence in the exposed areas, deaths in the household, the first four symptoms perceived after the exposure, eye symptoms, and treatment received to date. The first four symptoms were requested without prompting by the interviewer, whereas for the eye symptoms a checklist was read out after the first four symptoms had been recorded. A rapid clinical assessment of respiratory signs was recorded (obvious tachypnoea, audible wheeze, use of accessory muscles) so far as possible without alerting the interviewee to this. The initial focus of the inquiry was the eye consequences and to lay a baseline for future possible deterioration of sight.

Data were coded and sorted using the standard SPSS statistical package. Analysis was done using the Mantel Haenszel procedure for combining data from different strata[3] and the Mantel extension of this test.[4] Statistical results are reported as the chi-squared statistic,[2] odds ratio (OR), and confidence interval (CI) for a stated level of probability.

Results

Examination of severely affected hospital inpatients indicated that the main symptoms which were considered life threatening were those involving the respiratory system. The worst eye cases brought forward for slit lamp examination at Ghandi Medical College by two ophthalmologists showed no case of deep corneal damage.[2]

Travel by foot, tricab, and taxi allowed the outer limits of the exposure zones to be mapped out, as shown in the figure. People were affected (mostly burning eyes and coughing) over 1 km north of the factory, despite the light north wind. No deaths were reported to the north of the factory. People from this area tended to flee northward, rapidly getting away from the gas cloud. For analytical purposes, data were available from questionnaires of 379 people in the eight exposed clusters and 119 people in the two unexposed clusters (an average 2·32% of the estimated population in the area surrounding the study clusters). All study clusters were south of the factory. Table 1 shows the age distributions in these clusters. The age distributions of exposed and unexposed groups show some differences, which are most pronounced in the extremes of age. A reporting bias was noted in exposure cluster 7, a factory compound, where men conspicuously understated their ages relative to the estimate of the interviewers (presumably to protect their employment). Sex ratio in the two populations was similar (exposed group 41·7% women, unexposed group 42·9% women). The sex of about 15% of respondents in each group was not recorded.

The duration of exposure across all the exposed groups (n = 379) was as follows: 1% left the areas in which symptoms of gas exposure were reported within one hour, 15% were exposed for one hour, 16% between one and two hours, 16% for two hours, 8% for two to three hours, and 2% for three hours. A further 29% said that they had been present in the area throughout the exposure period (estimated to be about four hours). No details regarding individual exposure were obtained on 13%, three quarters of whom were children under the age of 10 years. Between clusters where deaths occurred (1 to 6) and the remaining two exposure clusters there was no apparent difference in the average duration of exposure nor in the proportion who fled from the exposed area ($\chi^2 = 0·25$).

Each interviewee was asked how many people had died in their household. The results of this are shown in table 2. Deaths were recorded in clusters 1, 2, 3, 5 and 6. Numerous deaths were known to have occurred in cluster 4, in households where there were no survivors, but the limitations of the study method meant that these numbers could not be recorded on a questionnaire. A total of 51 deaths was reported in the houses where the 379 survivors were examined. Based on the estimated population in and around each cluster (800 in 6: 1500 in 1: 2000 each in 2, 3, and 7: 3000 each in 4 and 5: and 4000 in 8), it is possible to calculate that about 1850 deaths occurred in and around the eight clusters used in this study. This covered about 70% of the exposed area.

The most frequently mentioned first symptom among the survivors in all the study clusters was burning eyes. In answer to the open question regarding sequence of symptoms, 56% mentioned burning eyes first, 15% mentioned it second, 7% third, and 3% as the fourth symptom (total 81%). The second most commonly reported symptom was coughing. The next two most frequently mentioned symptoms were watering of the eyes and vomiting (see table 3).

Table 3 shows the relation between initial symptoms and death. It would appear that burning eyes do not have a particularly high reported prevalence in those clusters where death was common. The frequency of reporting cough as one of the first four symptoms, however, follows closely the distribution of deaths. This trend was significant at the 5% level ($\chi^2 = 4·3$, using the Mantel extension as a test of trend with 1 DF). Using 25 mortality as a cut off point, clusters 1, 2, and 6 were strongly associated with reported cough ($\chi^2 = 8·39$; OR = 2·17; 99·5% CI = 1·02–4·59). Diarrhoea and shortness of breath among the

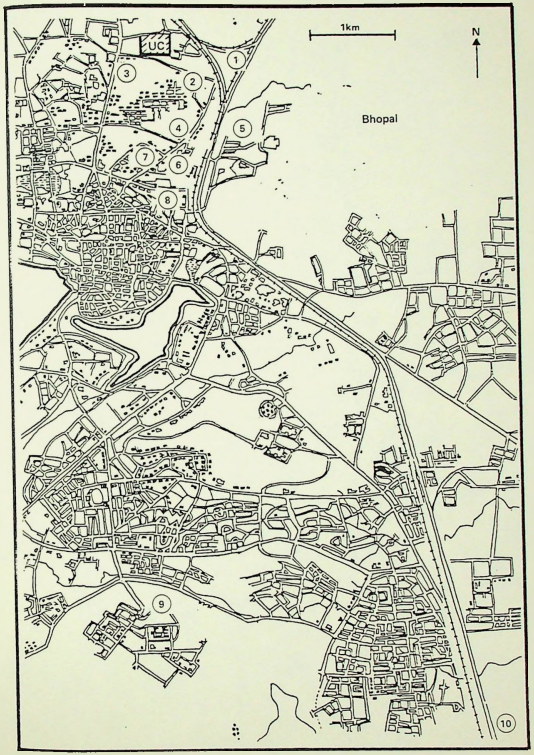

Figure Map of Bhopal showing location of Union Carbide plant (UCIL) and of exposed (1–8) and unexposed (9, 10) clusters. Scale is indicated by 1 km bar; area shown is about 6 km × 9 km.

Table 1 *Age distribution in the study clusters*

		Age (years)									
		0–9	10–19	20–29	30–39	40–49	50–59	60–69	70–79	≥80	NR*
						Exposed clusters					
1	No	12	10	30	5	5	3	2	1		2
	%	17·1	14·3	42·9	7·1	7·1	4·3	2·9	1·4		2·9
2	No	2	3	1	4	4	3				
	%	11·8	17·7	5·9	23·5	23·5	17·7				
3	No	20	13	12	11	1	5	4	2	†	
	%	29·0	18·8	17·4	15·9	1·5	7·3	5·8	2·9	1·5	
4	No	16	4	9	10	3	4	3	1		
	%	32·0	8·0	18·0	20·0	6·0	8·0	6·0	2·0		
5	No	5	5	6	7	3	13	6			7
	%	9·6	9·6	11·5	13·5	5·8	25·0	11·5			13·5
6	No	2	7	2	1	3	1				2
	%	11·1	38·9	11·1	5·6	16·7	5·6				11·1
7	No		7	3	19	17	8	1			
	%		12·7	5·5	34·6	30·9	14·6	1·8			
8	No	1	12	16	4	8	5		1		1
	%	2·1	25·0	33·3	8·3	16·7	10·4		2·1		2·1
						Non-exposed clusters					
9	No	18	11	14	7	1	4				3
	%	27·7	16·9	21·5	10·8	10·8	6·2				4·6
10	No	6	12	16	7	3	4	2			4
	%	11·1	22·2	29·6	13·0	5·6	7·4	3·7			7·4
						Total exposed					
	No	58	61	79	61	44	42	16	5	1	12
	%	15·3	16·1	20·8	16·1	11·6	11·1	4·2	1·3	0·3	3·1
						Total non-exposed					
	No	24	23	30	14	10	8	3			7
	%	20·2	19·3	25·2	11·8	8·4	6·7	2·5			5·9

*NR, no reply.

survivors had a notable relation to the occurrence of death. Diarrhoea was reported with increasing frequency across clusters with increasing mortality (χ^2 = 10·3 using the Mantel extension as a test of trend with 1 DF). Shortness of breath, on the other hand, showed a strongly inverse relation with clustering of deaths (χ^2 = 12·3 by the same test).

Examination of the eyes showed different patterns in the various clusters (table 4). Red eyes were more common among the exposed compared with unexposed people overall, a finding that was stastistically significant at the 90% confidence level (χ^2 = 3·1; OR = 1·71; 90% CI = 1·02–2·08). The characteristic superficial interpalpebral erosion of the cornea and conjunctiva (SIPE) associated with exposure to MIC was observed in seven of the eight exposure groups with differing frequency. This sign was most frequent in clusters 4 and 6 (36% and 50% respectively). These were the two clusters where the lowest proportion of people actually reported burning of the eyes as a symptom immediately after exposure. The possibility of the coincidence of SIPE with low reporting of burning eyes having occurred by chance may be excluded with greater than 99·9% confidence (χ^2 = 59·66 with 1 DF). The apparently inverse relation between burning and SIPE was not found between SIPE and reported watering of the eyes but it was apparent with photophobia and the sensation that the eyes could not be opened (see table 5). Survivors in th clusters where deaths had occurred (1–6) were neari 12 times more at risk of SIPE than other exposed people: in clusters 1–6 there were 52 cases out of 22 survivors compared with two among 101 survivors i clusters 7 and 8 (χ^2 = 17·49; OR = 11·7; 99·9% CI = 1·69–81·4).

There was no evidence of eye problems other tha SIPE and redness in the exposure clusters compare with the non-exposure clusters (9 and 10). There wa no difference in the occurrence of corneal opacity (= 0·05). Bitot spot (χ^2 = 0·14), active infection (χ^2 = 0·83), and evidence of past trachoma (χ^2 = 0·13 Pterygium (χ^2 = 17·6; OR = 3·3) and cataract (χ^2 =

Table 2 *Deaths by cluster*

Cluster	People examined	Households with deaths	No. of deaths
1	70	6	23
2	17	3	10
3	69	1	2
4	50	—†	—†
5	52	2	9
6	18	1	7
7	55*	0	0
8	48	0	0

*Works compound: only men of working age examined.
†In cluster 4 at least three households were encountered where death had occurred. These were not included in the survey.

Table 3 *Death rates in relation to early symptoms*

Cluster No	2 17	6 18	1 70	5 52	3 69	4 50	7 55	8 48
Death rate* (%)	37	28	25	15	3	†	0	0
First four symptoms (%):								
Burning eyes	76	60	86	83	74	60	95	94
Coughing	88	78	77	58	65	64	65	65
Watering eyes	41	33	66	25	22	42	47	58
Vomiting	47	39	37	29	59	15	33	42
Other symptoms (%):								
Photophobia	41	28	43	31	30	24	64	35‡
"Hard to open eyes"	24	6	9	17	9	4	20	10
Diarrhoea	12	6	9	8	4	2	0	0
Short of breath	6	6	9	23	12	12	35	25
Chest pain	6	11	14	17	10	10	16	25
Nausea	0	39	1	12	4	22	15	2
Unconsciousness	0	0	4	0	3	2	4	0
Dizziness	6	0	1	2	4	2	0	0
Choking	0	0	0	4	1	4	11	6
Headache	0	6	0	2	3	0	0	0
Twitching	0	0	0	0	4	0	2	0
Convulsions	0	0	0	2	1	0	0	0

*Number reported dead in each household visited divided by total examined plus number reported dead.
†Number of deaths not known.
‡Includes both volunteered information and direct questioning.

2·3; OR = 3·1) were more common in the non-exposed population. These differences were calculated over all age groups and disappeared with age stratification. There was an increase in fundal changes (dilated retinal vessels or haemorrhages) in the clusters where deaths had occurred (clusters 1–6: 80/272) by comparison with those where no deaths had occurred (clusters 7 and 8: 18/103). Data on four subjects were missing. These findings indicate a positive association, significant at the 2·5% level, between clusters where deaths occurred and fundal changes on ophthalmoscopy (χ^2 = 5·5; OR = 1·97; 97·5% CI = 1·03–3·76).

Unfortunately, the respiratory examination was omitted or no record was made for 55% of exposed survivors. Among the 45% who were examined, 25 (15%) were judged to be in "respiratory distress" on the grounds of a conspicuously increased respiratory rate, an audible wheeze at rest, or use of the respiratory accessory muscles. Overall, there was no hint of an association between fundal changes and obvious respiratory distress (χ^2 = 0·04), which probably indicates lack of representativeness of the available 45% of the respiratory examination data. Considering only those people on whom a respiratory examination was recorded, 16 were considered to be in obvious respiratory distress out of the 131 who were normal on fundoscopy and nine were deemed to be in respiratory distress out of the 40 with abnormal fundi. Fundal changes were thus twice as common among people deemed to be in respiratory distress. This finding, however, was significant only at the 12% level (χ^2 = 2·6; OR = 2·09; for 1 DF). Respiratory distress was observed nearly six times more often in clusters 1–6 where deaths had occurred (22/105) than in the other

Table 4 *Signs on physical examination*

Cluster No	1 70	2 17	3 69	4 50	5 52	6 18	7 55	8 48	9 65	10 54
Normal	30	6	48	19	37	2	21	33	44	30
Red eye	23	4	6	6	5	6	18	—	6	9
SIPE*	9	1	9	18	6	9	2	—	—	—
Bitot spot	5	—	3	2	1	3	1	1	4	2
Pterygium	4	1	4	3	4	—	14	—	9	17
Corneal opacity	6	5	5	3	2	1	7	3	5	4
Cataract	1	—	—	—	1	—	—	1	—	3
Discharge	1	—	—	3	—	1	—	—	3	—
Trachoma	—	2	—	2	1	1	—	6	—	3
Respiratory distress	*8*	*1*	*4*	*3*	*6*	*0*	*1*	*2*		
Examined	18	16	10	10	18	11	43	23		

*SIPE, superficial interpalpebral erosion.
Note: the number of signs in each cluster may exceed the total number of people in the cluster. Some individuals exhibited more than one sign. People, not eyes, were counted—for example, four red eyes equal four people with one or two red eyes each.

Table 5 *Relation between early eye symptoms and clinical eye damage (Cluster number, in descending order)*

	Rank							
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*
Frequency of:								
Burning	7	8	1	5	2	3	6	4
Photophobia	7	1	2	8	5	3	6	4
Difficulty in opening eyes	2	7	5	8	1	3	6	4
SIPE	6	4	3	1	5	2	7	8

SIPE, superficial interpalpebral erosion.

two exposure clusters (3/66), a finding that was significant at the 0·5% level (χ^2 = 9·3; OR = 5·8; 99·5% CI = 1·15–29·6). No abnormal afferent pupillary reflex was detected in any of the clusters.

Discussion

The urgency of implementing this study so soon after the disaster, in substantially less than ideal working conditions, imposed several obvious defects in the data set. It is difficult to extrapolate from the number of deaths reported in this community based survey to the likely number of overall deaths that resulted from the disaster. Although the household survey in each cluster was started from an arbitrarily selected house, after which it proceeded from door to door, there may have been a tendency for some of the teams to move towards the worst affected areas in each community. Overall, however, the patterns of death were consistent with the reported wind directions: first northerly then moving westerly. The highest mortality was observed close to the plant and on the south east side, presumably reflecting the change in wind direction. Allowing for the varying levels of exposure across the reported 100 000 people in communities with quite different density of housing, the official estimate of 2000 to 2500 deaths seems consistent with these data.

Perhaps the most striking impression from the affected communities in the days immediately after the episode was the large number of people with conspicuous difficulty in breathing. Some of these had been too disabled even to attend the local hospitals for treatment. During the two month follow up it was noted, again informally, that many of the children seen during the first visit still had conspicuous breathing difficulties. Such necropsy reports as are available indicate that some deaths were related to acute pulmonary oedema. Failure to record some basic data on respiratory status for the whole study population lost the unique opportunity offered by this survey, carried out so soon after the event. The afferent pupillary reflex indicates damage to the optic nerve and impairment of this reflex could be taken as evidence of neurotoxicity. The fact that this reflex was normal in all groups cannot be taken as evidence that neurotoxicity did not occur. The test may provide a useful baseline, however, for study over a long period.

Our analysis of symptoms related only to clusters where the deaths occurred, not to individuals, so their interpretation in this context limited. During the longer term follow up of survivo now in progress the predictive value of these sym toms in relation to prognosis could possibly be teste The reported mortality in this study involved relative small numbers in each cluster. None the less, t difference between clusters may provide a basis f further inquiries into the distribution and frequency immediate and delayed deaths and the prognosis symptoms among survivors.

The association of high death rates with report cough and diarrhoea among the first four symptoms difficult to reconcile with the apparently inverse rel tion between clusters where death was common a those where shortness of breath was a freque symptom among survivors. A similarly curious findi was the inverse relation between reported burning the eyes as an early symptom of exposure and it clinical finding of SIPE. Clusters 4 and 6, where SIP was most commonly observed and burning of the ey least frequently reported, were investigated by tw different teams. This implies that the apparent inverse relation is not the result of an observer induce bias.

In some clusters the death rates may have been hig enough for most people who had respiratory sy toms to have died, leaving alive only those who we relatively free of symptoms. If this was so th apparently direct relation with another respirator symptom (cough) would be difficult to explain. could be argued that the excess SIPE observed survivors from the clusters where deaths had occurre could result from their running away from th exposure, as was done by many thousands of peopl This might increase the exposure of their eyes an theoretically, their chances of survival. In reality, mo people who ran away went southward from the plan They thus moved with the cloud, not away from which would probably reduce rather than increas their chances of survival. There was also no evident of a difference between clusters where death rates we high and other exposed clusters in respect of pattern exodus immediately after the release of gas.

The second and more plausible explanation is th the relation between dose and overall response is mo complex than the usual log-linear relation. Ther appear to be at least two relatively distinct symptom pathology complexes which result from the exposur (see tables 3 and 4). This could be because differe pathological reactions occur at different doses.

It is theoretically possible that the gas cloud ma have had different constituent components afte certain time or in certain regions. We have shown tha methyl isocyanate can react with methylamine, the product of its initial reaction with water to produce dimethyl urea.[2] Various other oligomeric or polymeric adducts have been identified in the residues in the storage tank (S Varadarajan, 1985). Any of these could have had effects that are distinct from those of MIC. There could also be reactions between endogenous substances and MIC or its breakdown products. Thus people living in different areas could have symptom/pathology patterns relating to exposure to different chemical species. A counterindication to this suggestion is our observation that although liquid phase reactions between MIC and water are rapid, the vapour reactions are (as would be expected) much slower and consistent with the persistence of a concentrated cloud of MIC in its original form for some hours.[2]

On the other hand, laboratory toxicological experiments have shown distinct symptom/pathology groups at different dose levels for animals exposed to pure and continuously generated atmospheres of MIC.[3] Broadly speaking, these may be classified into neuropharmacological (anaesthesia and increased sensitivity to subsequent anaesthesia), eye irritation and damage (including a form of SIPE similar to that seen in Bhopal survivors), and lung damage (acute bronchoconstriction and tachypnoea, pulmonary oedema, and in the longer term, fibrotic and inflammatory reactions). All these effect groups occurred at characteristic and different exposure levels: in particular, sensory and neurological responses were noticed at relatively low doses, the eye damage was apparent at intermediate exposures, and the more obvious and life threatening manifestations of pulmonary damage appeared at higher dose levels. If these differences are considered capable of extrapolation to the responses seen in the Bhopal survivors the differing distribution of symptoms between exposed groups (and by implication, severity of exposure) may be interpreted as the complex response to varying degrees of exposure in a gas cloud composed mainly or entirely of MIC.

The surface of the eye is moist under ordinary circumstances, so damage to this by a substance that is reactive with water[2] will be associated with damage to other moist surfaces, including those of the lungs. The survivors in clusters where deaths had occurred were indeed at 12 times the risk of SIPE and six times the risk of respiratory distress compared with other exposed people. This leads us to the tentative conclusion that the deaths resulting from the exposure, reportedly pulmonary oedema, are associated geographically with cough and diarrhoea as early symptoms, and with SIPE and respiratory distress as clinical findings among the survivors.

The picture emerging from symptom patterns may be confounded by the fact that only the first four symptoms were requested. Later symptoms, however important their relation to pathology, were not recorded. It should also be reiterated that these initial symptoms were not directly associated with death of the individual with the symptom but only with the areas where death rates were higher.

Despite these caveats, there appear to be several geographical contrasts in the survivor population of Bhopal. The most obvious contrast is between the exposure and non-exposure clusters. Secondly, there appears to be a contrast in findings between clusters where deaths occurred (1–6) and the remaining two clusters. Thirdly, table 5 indicates that in certain clusters (notably 4 and 6) relatively few survivors reported burning eyes and shortness of breath, whereas cough, diarrhoea, respiratory distress, and SIPE were more common. In clusters 3, 5, 7, and 8 deaths were more uncommon as were clinical findings of SIPE but a different pattern of symptoms was evident. Burning of the eyes, shortness of breath, photophobia, choking, twitching, and convulsions were all more frequent.

The long term follow up of survivors should allow us to test the predictive value of these signs and symptoms associated geographically with high death rates, information which may be of substantial value in preparing for and coping with the longer term health consequences of the disaster. The groups of symptoms with different patterns of death/survival in the various clusters implies that the gas has different effects on people at different concentrations (or at different distances from the factory). This appears to be a feature of the concentration dependent interaction of the gas and its reaction products with human tissue.

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References

1 Andersson N, Kerr Muir M, Mehra V. Bhopal eye. *Lancet* 1984;ii:1481.
2 Andersson N, Kerr Muir M, Salmon AG, *et al*. Bhopal disaster: eye follow-up and analytical chemistry. *Lancet* 1985;i:761–2.
3 Maniel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of disease. *J Natl Cancer Inst* 1959;22:719–48.
4 Maniel N. Chi-square tests with one degree of freedom: extensions of the Maniel-Haenszel procedure. *Journal of the American Statistical Association* 1963;58:690–700.
5 Salmon AG, Kerr Muir M, Andersson N. Acute toxicity of methyl isocyanate. A preliminary study of the dose response for eye and other effects. *Brit J Ind Med* 1985;42:795–8.

epidemiological information concerning the effects of job difference on the incidence of bladder cancer in those manufacturing benzidine derived dyes, and the first report of a cohort study about cancer experience among the users of these dyes. The results suggest that the risk of bladder cancer exists mainly in the presynthesis process of dye manufacture and the benzidine derived dyes do not appear to be as strongly carcinogenic as benzidine. This supports a study that compared cases of bladder cancer in those exposed to benzidine with those working in the dye industry.[24] It is too early to make firm conclusions based on our results; however, it is necessary to conduct cohort studies in various industries where these dyes are used, and more experiments on their metabolism; such studies are now in progress.

1 Department of Epidemiology, Shanghai Cancer Institute, *et al.* Analysis of cancer incidence, mortality, and survival rates in Shanghai urban area during the period of 1972–1979. *Tumor* 1982;2:385–91. (In Chinese.)

2 Waterhouse J, Muir C, Correa P, Powell J. *Cancer incidence in five continents.* Vol III. Lyon: International Agency for Research on Cancer, 1976. (IARC sci publ No 15.)

3 Haley TJ. Benzidine revisited: a review of the literature and problem associated with the use of benzidine and its congeners. *Clin Toxicol* 1975;8:13–42.

4 International Agency for Research on Cancer. Monographs, suppl 1. *Chemicals and industrial process associated with cancer in humans.* Vols 1–12. Lyon: IARC, 1979.

5 Sakari T, Marita T, Maria-Liite K, Erkki J. Cancer of the urinary in Finland association. *Int Arch Occup Environ Health* 1960;46:43–51.

6 Wynder E, Onderdonk J, Mantel N. An epidemiological investigation of cancer of the bladder. *Cancer* 1963;16:1388–407.

7 Anthony HM, Thomas KM. Tumors of the urinary bladder: an analysis of 1030 patients in Leeds, England. *J Natl Cancer Inst* 1970;45:879–95.

8 Yoshida O, Harada TM. Bladder cancer in workers of dyeing industry. *Igaku no Ayumi* 1971;79:421–2. (In Japanese.)

9 Scott TS. The incidence of bladder tumours in a dyestuff factory. *Br J Ind Med* 1952;9:127–32.

10 Ishizu SK. *Administration of occupational bladder cancer with urinary cytological diagnosis.* Tokyo: Chemical Industrial Co, 1975:5–13.

11 Ishikawa T, the Japanese research group of bladder carcinoma. Clinical application of 2,5-di-acetyl-β-glucaro(1–4)(6–3) dilactone (SIA) to the bladder tumor, with special reference to its effect on frequency of tumor recurrence. *J Urol* 19[illegible] 108:571–6.

12 Kinsley MA. Potential cancer risk in the graphic arts and textile industries. In: Nieburgs HE, ed. *Prevention and detection of cancer. Part I. Prevention,* Vol I. *Etiology.* New York and Basel: Marcel Dekker Inc, 1977:857–99.

13 Boeniger M. *Carcinogenicity and metabolism of azo dyes especially those derived from benzidine.* Cincinnati: National Institute for Occupational Safety and Health, 1980:10–46.

14 Arthur RG. The carcinogenic potential of benzidine-based dyes. *J Environ Pathol Toxicol Oncol* 1984;5:243–59.

15 DHEW. *National occupational hazard survey—survey analysis and supplemental tables.* Cincinnati: US Dept of Health, Education and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, 1977. (DHEW (NIOSH) publ No 78–114.)

16 DHEW. *13-week subchronic toxicity studies of direct blue 6, direct black 38, and direct brown 95 dyes.* Cincinnati: US Dept of Health, Education and Welfare, Public Health Service, National Cancer Institute, Division of Cancer Cause and Prevention, Carcinogenesis Testing Program. 1978:1–[illegible] (DHEW publ No (NIH) 78–1398.)

17 Genin VA. Formation of blastomogenic diphenylamine derivatives as a result of the metabolic effect also dyes. *Voprosy Onkologii* 1977;23:50–2. (In Russian.)

18 Sugimura T, Nagao T, Kawachi M, *et al.* Mutagen-carcinogens in food, with special reference to highly mutagenic pyrolysis products in boiled foods. In: Hiatt HH, ed. *Origins of human cancer. Book C.* New York: Cold Spring Harbor Laboratory, 1977:1561–77.

19 Prival MJ, Valerie PM. Analysis of a method for testing azo dyes for mutagenic activity in salmonella typhimurium in the presence of flavin mononucleotide and hamster liver S. *Mutat Res* 1982;97:103–16.

20 You Xue-yun, Zhou Yong-gui, Ye Xin-liang. Study on the method of the mutagenicity test for benzidine and its derivatives. *Chinese Journal of Industrial Hygiene and Occupational Disease* 1987;5:146–59. (In Chinese.)

21 Thomas MR, Kenneth CM, Ching YW, Charlie KJ. Conversion of Congo red and 2-aminofluorene to mutagens following in vitro reduction by whole cecal bacteria. *Mutat Res* 1983;117:105–12.

22 Dieckhues B. Experiments on the reductive splitting of azo dyes by bacteria. *Zentralblatt fur Bakteriologie, Parasitenkunde, Infektionskrankheiten und Hygiene* 1961;180:244–54. (In German.)

23 Yoshida O, Miyakawa M, Okada H. Reducing direct black EX, a benzidine-derived dye, by colibacillus and bacteria from human soil. *Igaku to Seibutsugaku* 1973;86:361–4. (In Japanese.)

24 Miyakawa M, Harada T, Yoshida O. Reducing of direct black EX, a benzidine-derived dye, in the intestine of rats and man. *Igaku to Seibutsugaku* 1973;86:355–60. (In Japanese.)

25 Ishizu S. Bladder cancer in the dyeing workers. *Japanese Journal of Industrial Health* 1983;25:650–6. (In Japanese.)

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Delayed eye and other consequences from exposure to methyl isocyanate: 93% follow up of exposed and unexposed cohorts in Bhopal

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Abstract

A follow up study three years after exposure to methyl isocyanate in 93% of exposed survivors and "control" residents in 10 Bhopali communities showed an excess of eye irritation, lid infection, cataract, and a decrease in visual acuity among the exposed people. Breathlessness was twice as common in the heavily exposed clusters as those with lower exposure, a trend that could not be explained by different age or smoking patterns (OR 2·05, 95% CI 1·36–3·08). Case referent analysis of outpatient attendances at Bhopal Eye Hospital, considering patients with severe refractive errors and astigmatism as "controls," showed 46% increased risk of trachoma, 36% increased risk of other lid infections, and 45% increased risk of irritant symptoms among previously exposed people. "Bhopal eye syndrome" may thus include full resolution of the initial interpalpebral superficial erosion, a subsequent increased risk of eye infections, corresponsive phenomena (irritation, watering, and phlyctens), and possibly cataracts. It remains to be confirmed whether this reflects a more generalised disease as a consequence of previous exposure to methyl isocyanate or whether it is only the eye that is affected.

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The release of methyl isocyanate (MIC) on the night of 3 December 1984 resulted in the death of at least 1700 people, most of them, it is thought, from acute pulmonary oedema resulting from the burns occurring during the exothermic reaction of MIC with the water content of lung secretions and tissues.[1–3] During this rapid chemical reaction, faster in the liquid than vapour phase, MIC breaks down to methylamine and the toxic dimethyl urea[4]; on its own, MIC is probably capable of alkviating with a variety of tissues containing amino (NH_2) and hydroxyl (OH) groups. The consequences of this on the human eye were studied in eight clusters of exposed households and two clusters of broadly comparable but unexposed households in Bhopal, where all occupants were first examined by a team of Indian and British ophthalmologists in the two weeks immediately after exposure before the mass exodus from the city that occurred when the plant was restarted a fortnight after the incident.[5]

Despite initial fears, no case of blindness attributable to exposure to MIC was found in the clinical examinations immediately after the disaster. The early eye lesions found in Bhopal survivors, typically a superficial interpalpebral erosion (SIPE) of the cornea and conjunctiva,[6] appeared to heal without notable scarring within three months. Follow up at one year confirmed this resolution of the initial superficial erosion, but the complaint of excessive watering and irritation was noted during follow up.[4] Andersson and colleagues studied further the complaint of excessive watering by comparing 989 cases and 532 referents from outpatient records of the Bhopal Eye Hospital. This confirmed that increased risk of irritant eye symptoms was related to duration of exposure to the gas and could not be accounted for by age or social class. The conjunctival epithelium of cases complaining of excessive watering appeared normal on slit lamp examination and no abnormality of tear drainage could be detected using crude duct patency tests.[7]

Parallel to the epidemiological follow up of the survivors in Bhopal, a toxicological model of the eye damage and other effects was developed by Salmon at the London School of Hygiene and Tropical

Medicine using Lister hooded rats, in which species the initial eye lesions closely resembled the human lesions observed in Bhopal survivors.[8,9] Changes 14 months later included an eosinophilic and lymphocytic infiltrate in the conjunctival mucosa and in the perilimbal regions, more prominent in animals that were more heavily exposed.[10] A subsequent larger study in rats confirmed chronic inflammation of the glands of the eyelids which included cystic changes and degeneration of these organs, alterations that were more pronounced in more heavily exposed animals and which increased with time.[11]

We describe here the three year follow up of eight exposure and two non-exposure clusters in Bhopal and the results of a case-control monitoring scheme established at the Bhopal Eye Hospital immediately after the disaster, which now permit evaluation of accumulating clinical evidence that these eye changes noted in exposed animals may also occur among man.

Methods

Eight exposed and two unexposed clusters of households, where all surviving residents were examined in the two weeks after the disaster, were followed up over three years. Selection of the clusters and details of the initial examination have been described elsewhere.[4–6] People examined after the initial two weeks who were not part of the original population of the community eye programme have been excluded from this analysis. Children born to mothers in the original exposure or non-exposure cohorts were included in the analysis, as were people who died in the course of follow up if a "verbal necropsy" from two neighbours or relatives confirmed the likely cause of death.

All examinations followed the same format and were carried out by the same observers. A brief questionnaire was administered in Hindi, inquiring about age, exposure history, main health problems, eye problems, medical treatment, and smoking habits. The eye examination was detailed, including slit lamp and fundoscopy of all residents of the clusters in the Bhopal Eye Hospital community eye programme. One ophthalmologist (MKM) examined nearly 70% of participants at least twice over the three years. Vision was tested with and without the use of a pinhole; colour vision and afferent pupillary reflexes were measured to test for damage to the optic nerve. Standard definitions were used to describe opthalmic conditions encountered. Data were analysed using standard statistical procedures.[12–14]

Results of this cluster cohort analysis generate hypotheses for testing on the much larger base derived from the Bhopal Eye Hospital which, over 18 months, received some 50 000 new outpatients. Soon after starting the hospital, which provides relief for gas exposed victims with eye problems, a record system was inaugurated allowing rapid manual extraction of large series of cases of the eye condition in question to consider their exposure history, and sex, and presenting complaints. Referents (controls) were people from the same community attending hospital with eye problems who were found to have a refractive error greater than 2 diopters or astigmatism (and no other defect). Age and sex were taken into account in the analysis by stratification. Social class is a complicated issue in India and was only taken into account in as much as both cases and referents presented voluntarily at the hospital from the same catchment area complaining of eye problems.

Results

Table 1 portrays the age and sex distribution of people followed up in the 10 cluster cohorts at

Table 1 Age and sex distribution of 10 cluster cohorts: follow up December 1984–April 1988 (Male/fem

	Exposed clusters								Control
	1	2	3	4	5	6	7	8	9
Age group (y)									
0–9	7/7	2/1	11/10	7/9	4/2	0/2	0/0	0/0	7/6
10–19	6/3	1/2	4/9	2/2	3/2	5/2	4/1	0/5	7/4
20–29	11/17	0/1	3/11	2/7	2/3	1/2	2/0	9/6	3/6
30–39	7/0	1/3	4/5	5/5	3/4	0/1	12/3	1/3	3/2
40–49	1/2	2/2	0/2	2/1	1/3	2/2	16/2	4/4	1/5
50–59	1/1	1/2	4/0	4/0	5/9	0/1	3/1	3/1	1/3
⩾60	1/2	0/0	1/2	1/3	5/3	0/0	5/0	0/1	0/0
	34/32	7/11	27/39	23/27	23/26	8/10	42/7	26/20	24/25
Re-examined	66	18	66	50	49	18	49	46	19
Confirmed dead	1	—	1	2	1	—	—	1	2
Not contacted	4	—	2	—	2	—	6	1	3
1984 cohort	70	17	69	50	52	18	55	48	48
Total (excluding births) follow up or confirmed death				364					96
Total seen at initial study 1984				379					11
% Completion of follow up				96%					83

Small discrepancies in totals in clusters 1, 2, 4, 5, and 9 are due to births in these communities.

...ble 2 Eye symptoms in non-, low, and high exposure ...sters: follow up December 1984–April 1988 (percentage)

...ster type	High exposure clusters 1, 2, 4–6	Low exposure clusters 3, 7, 8	No exposure clusters 9, 10
...s followed up for ...ears	201	161	97
...mon burning ...about discharge	137 (68%)	85 (53%)	56 (58%)
...charge	4 (2%)	3 (2%)	2 (2%)
...charge and ...mon burning	8 (4%)	4 (2·5%)	3 (3%)
...s of vision ...s of night vision	90 (45%)	53 (33%)	29 (30%)
	4 (2%)	8 (5%)	3 (3%)

...nse to question "Do you suffer any eye problems?".

...ails of the data missing by the end of three years of ...ow up. Data could not be obtained on only a small ...portion of the original cluster cohorts and some ...s were seen at least once during the three years or ...d died during the follow up period. Only one ...on refused follow up examinations, in cluster 5, ...ong the 379 original residents in the eight ...osed clusters, all but 15 (4%) were examined ...n or had died during the three years. Among the ...riginally unexposed people, all but 20 (17%) ...re re-examined during three years of follow up, ...ss all of them between April and October of the ...d year.

...ye symptoms reported by people followed up ...r three years in response to the question "Do you ...e any eye problems?" (table 2) indicate an excess ...rtant eye symptoms (OR 1·91, χmh 2·98, ...CI 1·25–2·92) and of the complaint of loss of ...on (OR 1·65, χmh 2·29, 95% CI 1·075–2·53) ...se the highly exposed compared with the lower ...osed clusters. The complaint of loss of vision was

...ble 3 Visual acuity loss by age group in the cluster ...sters: follow up December 1984–April 1988. (percentage ...ss among each age group)*

...ster type	High exposure clusters 1, 2, 4–6 (n = 201)	Low exposure clusters 3, 7, 8 (n = 161)	No exposure clusters 9, 10 (n = 97)
...ss of visual ...	48 (24%)	22 (14%)	11 (11%)
...	1 (2%)	3 (14%)	0
...	5 (18%)	4 (13%)	3 (13%)
...	14 (30%)	6 (19%)	0
...	16 (55%)	5 (18%)	2 (18%)
...	7 (30%)	2 (7%)	5 (83%)
...	5 (21%)	2 (10%)	1 ((11%)

...ss in visual acuity between two examinations with ...ng participants unaware of results of first examination ...Age stratified comparison between higher and lower ...osures (OR 2·199, χmh 1df 2·9).

...ble for age specific frequencies are based on numbers ...ected in table 1.

corroborated by formal testing of visual acuity, under conditions where neither subjects nor observers knew the outcome of the baseline 1984 tests (table 3). The differential loss in visual acuity between exposed and unexposed people could not be explained by different age structure of the populations. People in the higher exposure clusters were more than twice as likely to suffer deterioration in visual acuity than people in the lower exposed clusters (OR 2·2, χmh 2·9, 95% CI 1·29–3·75, stratified for age).

Among the other symptoms volunteered in response to the open question "Do you have any other health problems?" chest pain, breathlessness, and vomiting or nausea were more frequent among exposed than non-exposed people and more common among the heavily exposed than those who lived in clusters that received lower exposure (table 4). The complaint of breathlessness was more pronounced among the more heavily exposed and could not be accounted for by different age structures or smoking habits in the different exposure categories (table 5). Stratifying for age and smoking simultaneously, people in higher exposure clusters were twice as likely to report breathlessness as people in the lower exposure clusters (OR 2·05, χmh 3·453, 95% CI 1·36–3·08) and 2·7 times more likely than those in the no-exposure clusters (OR 2·73, χmh 3·69, 95% CI 1·6–4·66).

More acute inflammatory trachomatous changes, trachomatous injection (TI) and follicular trachoma (TF), were found among the higher exposure than lower exposure clusters (OR 3·165, χmh 2·11, 95% CI 1·085–9·23). In the three years of follow up 5% of the unexposed people developed cataracts, whereas 8% of those with lower exposure and 10% of those with higher exposure developed cataract. The risk of developing cataract in the higher exposure clusters was nearly twice that in the other groups (OR 1·99, χmh 2·001, 95% CI 1·014–3·9). There was no new case of altered afferent pupillary reflex or loss of colour vision.

Table 4 Other symptoms in high, low-, and non-exposure clusters: follow up December 1984–April 1988. (percentage affected)*

Cluster type	High exposure clusters 1, 2, 4–6 (n = 201)	Low exposure clusters 3, 7, 8 (n = 161)	No exposure clusters 9, 10 (n = 97)
Breathlessness	109 (54%)	57 (35%)	28 (29%)†
Chest pain	73 (37%)	19 (12%)	20 (21%)
Coughing	67 (33%)	32 (20%)	26 (27%)
Vomiting nausea	48 (24%)	7 (4%)	4 (4%)
Diarrhoea	5 (2·5%)	0	0
Headache	35 (16%)	10 (6%)	14 (14%)
Abdominal pain	12 (6%)	3 (2%)	5 (5%)
Weakness lethargy	12 (6%)	14 (9%)	11 (11%)

*Open response to the question: "Do you have any other health problems?"
†Trend with increasing exposure, χ^2 22·03 2df p = 0·000.

Table 5 Breathlessness by age group in the cluster cohorts: follow up December 1984–April 1988. (percentage affected among each age group)

Cluster type	High exposure clusters 1, 2, 4–6 (n = 201)		Low exposure clusters 3, 7, 8 (n = 161)		No exposure clusters 9, 10 (n = 97)	
No breathless	109 (54%)		57 (35%)		28 (29%)	
	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers
Age (y)	b+ b−	b+ b−	b+ b−	b+ b−	b+ b−	b+ b−
0–9	0 0	11 30	0 0	12 9	0 0	5 16
10–19	1 0	12 15	0 1	10 21	0 1	4 18
20–29	3 2	24 17	1 3	12 15	0 4	8 14
30–39	6 1	15 7	3 3	7 15	3 4	4 0
40–49	4 0	7 7	0 5	7 16	0 1	1 4
50–59	7 2	9 6	3 4	1 4	1 1	2 5
60–69	3 1	7 5	0 2	1 6	0 1	0 0

b+ = Breathless; b− = not breathless.

Stratifying simultaneously for age and smoking:
RR of people with higher exposure compared with lower exposure OR 2·05, χmh 3·45, 95% CI 1·36–3·08.
RR of people with higher exposure compared with no exposure OR 2·73, χmh 3·69, 95% CI 1·6–4·65.
RR of people with higher exposure compared with no exposure OR 1·53, χmh 1·47, 95% CI 0·87–2·69.

The suggestion from the cluster cohort analysis of an association between exposure to MIC and eye infections, irritation, and cataracts was confirmed in the case-referent (case-control) analysis of first attendance outpatients at the Bhopal Eye Hospital. Over the period January 1987 to June 1988, acute infection of the eyelid (blepharitis, chalazion, and sty) was proportionally more common among people exposed to MIC than among those non-exposed. To derive an estimation of relative risk, given that many exposed people attended the hospital for complaints potentially related to MIC, cycatricial trachoma (TC) and tarsal plate involvement (TT) was chosen as a reference entity for acute eyelid inflammation. If these conditions are delayed consequences of exposure then they are unlikely to have developed in the short period since exposure to MIC. This comparison produced a 47% excess risk of acute lid inflammation among the exposed (χmh 2·72, 95% CI 1·114–1·94). Comparison of cases of new trachoma (inflammatory and follicular) and advanced trachoma (TT, TC) illustrated a preponderance of new trachoma among previously exposed people (OR 1·25, χmh 1·99, 95% CI 1·18–1·45). This excess of recent trachoma infection was sustained when severe refractive errors and astigmatism were used as a reference entity, stratifying simultaneously for age and sex (OR 1·42, χmh 3·83, 95% CI 1·21–1·67, table 7).

Table 6 Signs found by examination of the eye: follow up December 1984–April 1988. (percentage affected)

Cluster type	High exposure clusters 1, 2, 4–6 (n = 201)	Low exposure clusters 3, 7, 8 (n = 161)	No exposure clusters 9, 10 (n = 97)
Trachoma (TT, TF)	15 (7%)	4 (2·5%)	2 (2%)
Trachoma (TT, TC, CO)	10 (5%)	2 (1·2%)	3 (3%)
Conjunctivitis	62 (31%)	19 (12%)	51 (53%)
New pterygium	8 (4%)	3 (2%)	7 (7%)
New corneal opacity	22 (11%)	6 (4%)	10 (10%)
New cataract	22 (11%)	10 (6%)	5 (5%)
Loss of visual acuity	48 (24%)	22 (14%)	11 (11%)
Abnormal colour vision	2 (1%)	0	1 (1%)
Abnormal afferent pupillary reflex	0	0	1 (1%)

Numbers represent people affected (one or two eyes).
A single subject may present more than one sign.
"New" indicates occurrence during three years of follow up, derived by subtracting signs detected in 1984.

Over the same 18 month period, the risk of phlyctenular keratoconjunctivitis was higher among people exposed to MIC than among those not exposed. Using pterygium, a non-inflammatory conjunctival lesion, as a comparison entity, the relative risk of phlyctens among exposed people was 2·6 times that in non-exposed people (OR 2·64, χmh 2·76, 95% CI 1·22–5·26) when the data were stratified for age and sex. There was also evidence that people exposed to MIC were more likely than unexposed people to complain of watering of eyes, irritation as a presenting symptom, using the complaint of watering alone as the reference entity (OR 1·3, χmh 6·16, 95% CI 1·18–1·45) or severe refractive errors and astigmatism (OR 1·5, χmh 95% CI 1·33–1·58) as a reference group. When stratified for age and sex. Although there was apparent excess relative risk of cataract among people of all exposure categories together, there was no statistically significant difference between those of higher exposure and those with lower exposure, as portrayed in table 8 (OR 1·3, [illegible] 1·23–1·5), using refractive errors as [illegible]

Discussion

The cohort analysis of clusters in the community

Table 7 Case-referent monitoring with hospital outpatient first attendances January 1987–June 1988

	Not exposed	Exposed to MIC	Relative risk using astigmatism and refractive errors: Crude RR	Age and sex stratified: RR	95% CI
Presenting only as a presenting symptom	1164	1954	1·1	1·11	(1·02–1·21)
Watering and burning or itching as the presenting symptom	999	2704	1·77	1·45	(1·33–1·58)
Old trachoma (TT, TC, CO)	191	392	1·34	1·42	(1·19–1·7)
Phlyctenular keratoconjunctivitis	546	824	0·99	1·06	(0·93–1·2)
Pterygium	46	90	1·28	1·24	(0·85–1·8)
Conjunctivitis	168	142	0·55	0·62	(0·49–0·78)
Chalazion, and blepharitis	1000	1515	0·99	1·02	(0·93–1·12)
Corneal opacity	204	427	1·37	1·36	(1·14–1·62)
Corneal ulcer	309	355	0·75	0·82	(0·69–0·97)
Anaemia	48	50	0·68	0·7	(0·47–1·04)
Renal disease	163	148	0·48	0·69	(0·54–0·88)
Diabetes	54	52	0·63	0·73	(0·5–1·03)
Astigmatism and refractive errors > 5D, no other lesion.	1475	2251	—	—	

Excluded RR estimated using the Mantel-Haenszel procedure.

...ch achieved, suggests a threefold excess of eyelid inflammation, twofold increase of new cataracts, and loss of visual acuity among the more severely exposed sisters. Heavily exposed people were twice as likely to complain of breathing problems as people with little exposure, and three times more likely to do so than unexposed people.

Although it was clear who had been exposed and who had not, by virtue of their residence (non-exposed clusters were 11 km and 14 km away from the Union Carbide plant), neither examiners nor participants knew how the exposed clusters had been classified as lower and higher exposure. This classification was based on early deaths associated with exposure in these areas,[8] not on distance from the Carbide plant or on subsquent long term clinical findings. Clusters 1, 2, and 4–6 were classified as higher exposure whereas clusters 3, 7, and 8 were regarded as having a lower exposure. Thus whereas it is impossible to exclude observer or participant bias between exposed and non-exposed clusters, it is highly unlikely that the differences between higher and lower exposure clusters can be explained by this possible observer bias.

The case-referent analysis confirms an excess of recent infections and irritant symptoms in people exposed to MIC. If this phenomenon is limited to the eye it could possibly be explained as iatrogenic. Indiscriminate and long term use of eye drops, including those containing steroids, has been a notable feature since the disaster. It is virtually impossible, under field conditions, to obtain accurate and detailed medical histories on previous use of medication, which makes this plausible explanation difficult to exclude. Almost everyone attending the Bhopal Eye Hospital reports using some previous medication for their eye problems. Yet the findings of continued irritation and recurrent infections in people previously exposed to MIC are in keeping with histopathology from the toxicology experiments,[9–11] where evidence of dose related and progressive chronic inflammation (eosinophil and lymphoid infiltrates in the conjunctival submucosa and, in the eyelid glands, epithelial dysplasia and chronic inflammatory infiltrates) was found in the absence of any medical treatment. Effects of MIC on the immune system could provide a coherent explanation for the different findings.[15]

In its response to MIC the eye could be considered

...8 First attenders at Bhopal Eye Hospital with cataract: Case-referent monitoring, January 1987–June 1988. MIC exposure history, less than or greater than one hour

	MIC Exposure < 1 hour		MIC Exposure > 1 hour	
...(y)	Cataract	Referent	Cataract	Referent
...–4	1	22	2	13
...–9	3	142	6	171
...–14	7	174	11	211
...–19	5	156	12	177
...–24	27	188	54	254
...–29	73	156	97	192
...–34	72	59	125	109
...–39	56	36	79	59
...–44	16	2	34	11
...–49	0	31	0	11
...–54	5	196	2	162
...–59	2	227	5	241
...–64	13	255	20	307
...–69	3	220	76	220
...–74	61	155	125	162
...–79	81	59	170	104
...–84	104	17	73	10
...–89	20	1	24	4

...ed for age and sex simultaneously: estimated odds ratio ... Mantel-Haenszel statistic = 3·517; 95% confidence ... 1·23–1·5.

a "sentinel organ" for more general phenomena in the rest of the body. So far, there is no evidence of damage to central or peripheral nerve tissue, as tested in the eye by colour vision and afferent pupillary reflexes.[16] Fundoscopy, which permits direct appraisal of arteries and veins, has not yet shown any abnormality associated with exposure to MIC. This corresponds with the absence of cardiovascular findings in Salmon's animal toxicology model.[8-11]

The acute superficial interpalpebral erosion is probably similar in chemical nature to the "burns" caused to the lung by the exothermic reaction of MIC with water in mucous secretions. In the eye this lesion heals rapidly, apparently without scarring (tables 6 and 7). Toxicological study of the lungs showed severe and progressive changes including neoplastic and cystic change of the bronchial epithelium, dramatic increases in peribronchial lymphoid tissue, and abnormal syncitialising activity of the epithelium inside pulmonary tissue.[11] Similar changes in man, if these can be confirmed in postmortem studies, may be related to the complaint of breathlessness in the cluster cohorts, more frequent with increasing severity of exposure and which is not accounted for by smoking or age.

If the apparent excess of cataracts found in this study is confirmed by larger investigations now under way a possible explanation may be found in the dramatic kidney and liver damage found in the toxicological model developed by Salmon.[11] These effects were more advanced in animals that experienced higher doses of MIC and progressed with time. The apparent increased occurrence of infections, and allergic/inflammatory complaints in the human eye, in the light of increasing evidence of multisystem damage in toxicological models, raises the possibility of generalised systemic long term consequences resulting from exposure to MIC. To exclude this, it is necessary to study formally the occurrence of infections, allergies, and deaths from these conditions. Much light will be shed on the issue when systematic evaluation of human postmortem material from lungs, spleen, liver, and kidneys becomes available.

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1 Kamat SR, Mahashur AA, Tiwari AKB, *et al* Early observations on pulmonary changes and clinical morbidity due to the isocyanate gas leak at Bhopal. *J Postgrad Med* 1985;31:63–72.
2 Dave JM. The Bhopal methyl isocyanate incident: an overview. In: Schiefer HB, ed. *Highly toxic chemicals: detection and protection methods: Proceedings of an international symposium September 1985 Saskatoon Canada*. Canada: University of Saskatchewan, Toxicology Research Centre, 1985:1–37.
3 Karol MH. Respiratory effects of inhaled isocyanates. *CRC Crit Rev. Toxicol* 1986;16:349–79.
4 Andersson N, Kerr Muir M, Salmon A, *et al*. Bhopal disaster: eye follow up and analytical chemistry. *Lancet* 1985;i:761–2.
5 Andersson N, Kerr Muir M, Mehra V. Bhopal eye. *Lancet* 1988;i:1481.
6 Andersson N, Kerr Muir M, Mehra V, Salmon AG. Exposure and response to methyl isocyanate: results of a community-based survey in Bhopal. *Br J Ind Med* 1988;45:466–75.
7 Andersson N, Kerr Muir M, Ajwani MK, Mahashabde S, Salmon A, Vaidyanathan K. Persistent eye watering among Bhopal survivors. *Lancet* 1986;ii:1152.
8 Salmon AG, Kerr Muir M, Andersson N. Comparison of toxicology and epidemiology for methyl isocyanate at Bhopal. *Hum Toxicol* 1987;6:410–411. (Abstract.)
9 Salmon AG, Kerr Muir M, Andersson N. [illegible] toxicity of methyl isocyanate: a preliminary study of [illegible] respiratory, eye and other effects. *Br J Ind Med* 1985;42:[illegible]–8.
10 Gassert T, Mackenzie C, Kerr Muir M, Anderson N, Salmon AG. Long term pathology of lung, eye and other tissues following acute exposure of rats to methyl isocyanate. *Lancet* 1986;ii:1403.
11 Mackenzie CD, Andersson N, Cameron K, Kerr Muir M, Salmon A, Gassert T. Longer term consequences of methyl isocyanate exposure in Lister hood rats: possible implications for Bhopal disaster victims. *Am J Expl Pathol* (in press).
12 Mantel N. Chi-square tests with one degree of freedom: extensions of the Mantel-Haenszel procedure. *Journal of the American Statistical Association* 1963;58:690–700.
13 Mantel N, Haenszel W. Statistical aspects of the analysis of data from retrospective studies of diseases. *J Natl Cancer Inst* 1959;22:719–748.
14 Olenick A, Mantel N. Family studies [illegible]. *J Chronic Dis* 1970;22:617–625.
15 Karol MH, Kamat SR. The antibody [illegible] to isocyanate: experimental and clinical findings. *Bull Physiopathol Respir* 1988;23:591–7.
16 Mergler D, Blain L, Lagacé JP. Solvent related colour vision loss: an indicator of neural damage? *Int Arch Occup Environ Health* 1987;59:313–21.

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Assessment of urinary protein 1 and transferrin as early markers of cadmium nephrotoxicity

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Abstract

Transferrin and protein 1, a sex linked α_1-microprotein, were assayed in urine from 58 workers exposed to cadmium (Cd) in a non-ferrous smelter and from 58 age matched referents. These two new markers of nephrotoxicity were compared with urinary β_2-microglobulin (β_2-m), retinol binding protein (RBP), albumin, and β-N-acetylglucosaminidase (NAG). The response of protein 1 to Cd tubulotoxicity was similar to that of β_2-m, RBP, and NAG. In Cd workers, protein 1 had a correlation with urinary Cd ($r = 0{\cdot}56$) similar to β_2-m ($r = 0{\cdot}48$), RBP ($r = 0{\cdot}58$), and NAG ($r = 0{\cdot}49$). Values of these three low molecular weight proteins and of NAG were increased only in workers with urinary Cd higher than 10 μg/g creatinine. Urinary transferrin and albumin were similarly affected by exposure to Cd. Their response, however, was clearly more sensitive than that of low molecular weight proteins. Prevalences of positive values of these two high molecular weight proteins were not only higher but also tended to rise at lower concentrations of Cd in urine or blood. This finding suggests that in some subjects subtle defects in glomerular barrier function may precede the onset of proximal tubular impairment after chronic exposure to Cd. It remains to be assessed whether these subjects are more at risk of developing renal insufficiency.

The early nephrotoxic action of cadmium (Cd) in man can be detected on the basis of several urinary or plasma markers.[1-3] Tests that are currently recommended for the health surveillance of populations exposed to Cd rely on the measurement of urinary proteins which reflect the functional integrity of the tubule or the glomerulus according to their size. Low molecular weight proteins such as β_2-microglobulin (β_2-m),[4] retinol binding protein (RBP),[5,6] or α_1-microglobulin[7] are currently used for screening for proximal tubular injury whereas the assay in urine of a high molecular weight protein such as albumin permits the assessment of glomerular filter selectivity, at least before tubular function is compromised.[8,9] According to recent studies the urinary activity of β-N-acetylglucosaminidase (NAG) is also a sensitive indicator of excessive absorption of Cd.[10,11]

In this study we have assessed two new potential markers of incipient Cd nephropathy, urinary protein 1 and transferrin. Protein 1 is an α_1-microprotein (molecular weight 20 000) which may be the equivalent in man of the androgen dependent rat α_{2u}-globulin.[12] It is a sex linked protein that is excreted in greater amounts in the urine from men after puberty. The origin of urinary protein 1 is unknown but the data we have so far collected suggest that it might constitute a new marker of impaired tubular reabsorption.[12]

Urinary transferrin was determined because recent studies suggest that it may be a more sensitive index of glomerular barrier defect than the microalbuminuria test.[13,14] These two markers have been assayed in the urine from workers exposed to Cd in a non-ferrous smelter and compared with urinary β_2-m, RBP, albumin, and NAG. The relation between these biochemical markers and indicators of exposure to Cd was also examined.

Materials and methods

STUDY POPULATIONS

The study was conducted on 58 male workers exposed to Cd in a non-ferrous smelter. The control group consisted of 58 age matched men recruited from a factory with no risk of exposure to Cd or other heavy metals. None of the exposed workers or referent subjects was occupationally exposed to lead or mercury as shown by the concentrations of these metals in blood (< 35 μg/100 ml) and urine (< 5 μg/g creatinine). The characteristics of the two populations are summarised in table 1.

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Acute Toxicity of Methyl Isocyanate and Ineffectiveness of Sodium Thiosulphate in Preventing Its Toxicity

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LD_{50} (mg.kg^{-1} body wt) of pure methyl isocyanate (MIC) when given subcutaneously was 328.6 and 261.3 in male and female rats, and 81.9 and 85.3 in male and female mice respectively. The oral LD_{50} in male rats was 51.5 mg.kg^{-1}. The LC_{50} for 30 min exposure in a static chamber was 1046.5 mg.m^{-3} in rats (female) and 267.6 mg.m^{-3} in mice (male). Sodium thiosulphate, given before or after MIC treatment had no beneficial effects. Estimation of cyanide in plasma, liver and brain did not show any significant increase in MIC treated rats when compared to controls, while 1/40 LD_{50} of potassium cyanide given subcutaneously, produced significant elevation of cyanide level.

Methyl isocyanate (MIC) an intermediate in the production of carbamate insecticides appears highly le[thal]. Surprisingly very little information is available on the toxicity of MIC[1-5]. Therefore, in this study acute toxicity of pure MIC in experimental animals is investigated by different routes of administration. Since sodium thiosulphate (STS) has been reported to be an effective antidote when administered to MIC victims its role if any, in pure MIC poisoning in rats has also been investigated.

Materials and Methods

Animals—Wistar rats (120-140 g) and Swiss albino mice (18-22 g) bred in the laboratory were used. The animals were housed in polypropylene cages (two rats per cage or four mice per cage of same sex) with dust free rice husk as the bedding material. Pellet chow (Hind Lever feed) and tap water were given *ad libitum*. Before the experiment the animals were fasted overnight while water was given *ad libitum*.

Drugs and chemicals—All chemicals used were of analytical grade. MIC synthesised[6] was of above 98% purity (gas chromatographic analysis). STS (10% solution) was obtained from Dr Franz Kohler Chemie GmbH.

Administration of MIC—MIC was administered by subcutaneous (sc), oral (po) and inhalation routes. For sc route, MIC was dissolved in olive oil, in which it was found to be stable for at least one day (by chromatographic analysis) and injected dorsally. For po route, MIC was fed through a 20 gauge stomach feeding needle.

In order to have a beter safety and also to lower the MIC consumption a static chamber was used for inhalation toxicity, consisting of a 20 l flat bottomed flask with a flat flange joint at the top (Fig. 1). The total capacity was 21.5 l including all the tube connections. The inner diameter of the mouth was 100 mm so that a hand could pass through easily for keeping the animal (rat or mouse) or taking it out. The flask was fitted with two B-14 joints at the sides, one was used as an inlet and the other was used as a port for monitoring inside temperature, humidity, etc. The outlet was at the top of the flask. A closed circuit pump with a displacement capacity of 30 l.min^{-1} connected to the flask ensured mixing of the air.

MIC was loaded using a microlitre syringe in the side test tube on a piece of Whatman grade I filter paper kept in it. When the pump is switched on MIC vapourises instantaneously and diffuses into the inhalation chamber. The pump was switched on every five min for 15 sec, for thorough mixing of the air in-

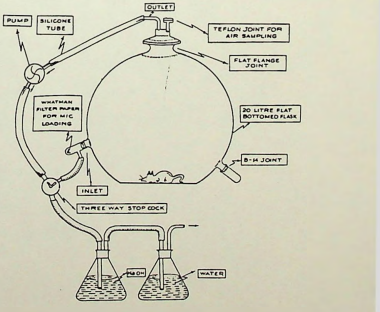

Fig. 1—Assembly for inhalation exposure static type

side the chamber. A teflon joint with a silicone diaphragm fixed at the top was used for sampling the chamber air. As MIC is highly reactive, only silicone tubes were used for the connections and silicone grease for the joints. At a time, only one rat or three mice were exposed (loading complement < 1%) and the duration of exposure was only 30 min. Once the exposure was over MIC was neutralised by turning the three way cock, and removing the test tube connection with the pump on. The chamber air was passed through a gas wash bottle containing sodium hydroxide solution.

Analysis of chamber atmosphere—Three different concentrations (100, 200 and 400 ppm) of MIC were developed inside the chamber without animal to find the effeciency of the inhalation assembly. A 20 ml sample of the chamber atmosphere was withdrawn at intervals of 2,5,10,20 and 30 min. The samples were taken in chilled toluene and injected in gas chromatograph using OV-17 column. More than 90 % recovery was obtained in all the concentrations and intervals. But for all calculations, only theoretical concentration was used i.e. weight of agent injected to volume of the chamber.

LD_{50} calculation—MIC was administered as a single dose or single exposure and the animals were observed for mortality up to 48 hr. For each dose/concentration between 5 to 8 animals were used. LD_{50} and LC_{50} were calculated by probit analysis[7]. STS was given (ip) either immediately after or 10 min prior to MIC administration and protection index calculated.

$$\text{Protection index (PI)} = \frac{LD_{50} \text{ with STS}}{LD_{50} \text{ without STS}}$$

Estimation of cyanide—Cyanide was estimated in plasma, liver and brain in female Wistar rats. The animals were lightly anaesthetised with ether and maximum blood was collected in heparinised vials from the retroorbital plexus. After cervical dislocation, liver and brain were removed and homogenised in 0.1 *N* NaOH (5% homogenate). Cyanide was estimated spectrophotometrically[8]. The analysis was carried out immediately after collecting the blood and tissue samples.

Results and Discussion

LD_{50} studies—Immediately after sc injection the animals looked sedated. Hind limb paralysis and decreased respiratory rate were noted. Higher doses produced gasping (mouth breathing). Mostly death occured after 6 hr. In doses higher than LD_{50} the animals showed severe convulsions and gasping and died within 1 or 2 hr. By po route the symptoms were similar to that of sc route, except that the gasping was more prominent. When the animals were subjected to MIC vapour inhalation, irritation of the eye was the first symptom observed in both rats and mice. Within 5 min after exposure, gasping started and was more prominent in rats. By inhalation exposure also, death occured only after 6 hr. Piloerection was observed in rats. Rectal temperature was markedly reduced and was very low in animals given MIC sc and po than by inhalation. Post mortem (im-

Table 2—LC_{50} Values with MIC in Female Rat and Male Mouse

Log Conc.	Conc. ($mg.m^{-3}$)	No. of animals/ experiment	No. died	Mortality %
		Female Rat		
2.85	707.9	5	0	0
3.00	1000.0	8	4	50.0
3.10	1258.9	6	4	66.7
3.20	1584.9	5	5	100.0
		Male Mouse		
2.30	199.5	6	1	16.7
2.35	223.9	6	2	33.3
2.50	316.2	6	4	66.7
2.75	562.3	6	6	100.0

Table 1—LD_{50} of MIC by Different Routes

Route of administration	Species	Sex	LD_{50} ($mg.kg^{-1}$)	95% confidence limit
sc	rat	M	328.6	319.0-338.6
sc	rat	F	261.3	229.9-296.8
sc	mouse	M	81.9	80.3-83.5
sc	mouse	F	85.3	80.7-90.2
po	rat	M	51.5	49.5-53.6
inh	rat	F	1046.5*	903.6-1211.8*
inh	mouse	M	267.6*	215.5-331.6*

*LC_{50} (t = 30 min) in a static chamber. Values $mg.m^{-3}$
M = male; F = female

mediately after death) showed blood clots and edema in the lungs in all the three routes, both in rat and mouse. Kimmerle and Eben[1] also reported acute lung edema as the cause of death in MIC inhaled rats. A very prominent oozing wound with necrosis of the adjoining skin at the injection site was found 10 days after sc injection in all survived animals.

Table 1 shows LD_{50} and LC_{50} of MIC by different routes. The various concentrations used for the lethality study by inhalation and the percentage of death in each is given in Table 2. MIC is more toxic to mouse than rat. This is contrary to the findings of Dodd *et al.*[9], though the concentration time product of LC_{50} of mouse is more or less same in both studies (present study; 8010 mg.min.m^{-3} and Dodd *et al.* 10102 mg.min.m^{-3}). As MIC is a highly reactive compound, it reacts very quickly with $-OH$, $-SH$ and $-NH_2$ groups[10]. That is why MIC injected sc is less toxic. As it is also a highly corrosive agent particularly to the mucus membranes[1], it may cause extensive damage leading to death. This may be the reason that MIC is more toxic by po route and inhalation.

Effect of STS—STS was given (ip) immediately after administering MIC to rats and mice. Only in mice it gave considerable protection (PI = 2.2).

Table 3—Shift of LD_{50} with Sodium Thiosulphate Treatment

Route of administration of MIC	Species	Sex	Dose of STS (mg.kg^{-1})	Route of administration of STS	LD_{50} (mg.kg^{-1})	P I
sc	rat	F	250	ip*	337.3 (315.6-360.4)	1.3
sc	mouse	F	50	ip*	186.0 (170.8-202.6)	2.2
inh	rat	F	250	ip**	940.8*** (797.8-1107.0)	0.9
inh	mouse	M	10	iv**	243.1*** (208.8-283.3)	0.9

F = female; M = male
*STS given immediately after MIC.
**STS given 10 min prior to inhalation.
***LC_{50} (t = 30 min) in a static chamber. Values are mg.m^{-3}.
Figures in parantheses are 95% confidence limit.

Table 4—Cyanide Levels in Plasma and Tissues After Potassium Cyanide and MIC Treatment

Dose (mg.kg^{-1})	Sacrificing time after poison (hr)	No.	Cyanide level		
			Plasma μg.dl^{-1}	Liver μg.g^{-1}	Brain μg.g^{-1}
Control	—	6	0.70 ± 0.12	3.10 ± 0.25	1.37 ± 0.09
KCN					
0.225 (1/40 LD_{50})	1	5	1.89** ± 0.24	3.73* ± 0.12	1.90 ± 0.30
2.25 (1/4 LD_{50})	1	7	5.59*** ± 0.51	4.13** ± 0.21	1.66 ± 0.25
9.0* (1 LD_{50})	30 min[b]	6	45.03*** ± 2.52	4.65* ± 0.60	2.85*** ± 0.21
MIC					
1 LC_{50}	1	5	1.04 ± 0.21	3.40 ± 0.24	1.50 ± 0.21
1 LC_{50}	4	5	0.95 ± 0.15	3.17 ± 0.17	1.75 ± 0.30
1 LC_{50}	24	5	0.81 ± 0.20	3.49 ± 0.21	1.33 ± 0.10

d = deci litre; Values mean ± SE
*LD_{50} = 9.0 mg.kg^{-1} sc(Sax[11])
[b]Animals were in critical state
LC_{50} = 1046.5 mg.m^{-3} for 30 min exposure.
*P < 0.05; ** < 0.01; *** < 0.001

Where MIC was administered by inhalation STS failed to protect (Table 3) and even the LD_{50} was not shifted significantly (PI = 0.9). Further raising the dose of STS was not beneficial in protecting the animals when MIC was given by inhalation.

To further confirm the results, female rats were given LC_{50} dose of MIC by inhalation and were sacrificed at different time intervals. The amount of cyanide in plasma, liver and brain were estimated. For comparison KCN was given sc at three different doses of LD_{50} (1/40, 1/4 and 1). The results (Table 4) show that the level of cyanide in MIC treated animals was similar to that of the control. But KCN given at a very low dose of 1/40 LD_{50} produced significant raise in the amount of cyanide both in plasma and in liver. Nemery *et al.*[2] also showed that STS could not ptotect against MIC poisoning. Thiocyanate in urine was shown to be decreased rather than increased by Nemery *et al.*[2] and Salmon *et al.*[4]. The present results show that there is no evidence of cyanide produced in the body following administration of MIC (1 LC_{50}) which explains the finding that STS has no beneficial effect in MIC toxicity.

Acknowledgement

The authors are grateful to Dr P K Ramachandran for his keen interest and encouragement and to Dr R K Srivastava for his valuable suggestions. The authors are thankful to Dr B R Gandhe for GC analysis, to Messrs M Srinivasan and Hari Afley for fabricating inhalation exposure chamber and secretarial assistance respectively.

References

1 Kimmerle G & Eben A, *Arch Toxikol*, **20** (1964) 233.
2 Nemery B, Sparrow S & Dinsdale D, *Lancet*, **ii** (1985) 1245.
3 Nemery B, Dinsdale D, Sparrow S & Ray D E, *Br J Ind Med*, **42** (1985) 799.
4 Salmon A G, Kerr Muir M & Andersson N, *Br J Ind Med*, **42** (1985) 795.
5 Ferguson J S, Schaper M, Stock M F, Weyel D A & Alarie Y, *Toxicol Appl Pharmacol*, **82** (1986) 329.
6 Ragueeran C D & Kaushik M P, *J Chromatogr*, **346** (1985) 446.
7 Finney D J, *Probit analysis* (Cambridge University Press, London) 1977.
8 Feldstein M & Klendshoj N C, *J Lab Clin Med*, **44** (1954) 166.
9 Dodd D E, Fowler E H, Snellings W M, Pritts I M & Baron R L, *Fundam Appl Toxicol*, **6** (1986) 747.
10 Cohen S & Oppenheimer E, in *The chemistry of cyanates and their thio derivatives*, edited by S Patai (John Wiley and Sons, New York) 1977, 923.
11 Sax N I, *Dangerous properties of industrial materials* (Van Nostrand Reinhold Co, New York) 1984, 2273.

the Cancer and Steroid Hormone (CASH) group's data proves correct, as seems probable, one would agree with him that "the results of all major studies thus appear to be consistent with some increase in risk in women aged up to about 35".1

The Committee on Safety of Medicines (CSM) makes a "bottom line" statement (*Current Problems* no 26) that there is no need for a change in prescribing practice. I agree, but only if this is interpreted that we continue to present the risks and benefits as fairly as we can so young women but leave to them the decision whether to use the method. This should now mean raising the cancer issue routinely in counselling, not omitting the protective effects against at least two malignancies (ovary and endometrium). The contraceptive and non-contraceptive benefits of combined oral contraceptives may seem so great to many as to compensate for almost any likely lifetime risk of breast cancer: especially as the UK NCCS group has already shown this to be lower for modern low oestrogen pills. One of the authors of UK study (M. C. Pike, personal communication) hypothesises that the risk could be lower still or absent after use of the currently most popular brands with the new progestogens desogestrel and gestodene since they raise sex-hormone binding globulin.

The main findings suggest that the incidence under age 36 changes from one in 500 to one in 300–350 for users of combined oral contraceptives for four or more years. Dr Elizabeth Wilson of the National Association of Family Planning Doctors proposes to explain this as follows: imagine a meeting-hall filled with 1000 average young women. 2 will have to receive treatment for breast cancer before their 36th birthday. Now fill the hall with 1000 women who have all taken the pill for four or more years before this age and there will be 3 under treatment—2 who would have developed breast cancer by then anyway, and 1 who, if she had used a different contraceptive, would either have avoided the disease or (possibly) would have had it later. This helps to put things in perspective: though as Dr Malcolm Potts has said "the jury is still out on the whole matter of the pill and breast cancer".

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1. Potts J. Oral contraceptives and breast cancer: is the CASH study really negative? *Lancet* 1989; i: 552.

LONG-TERM EFFECTS OF METHYL ISOCYANATE

SIR,—The focus on hydrogen cyanide (HCN) in your Round the World correspondent's report (April 29, p 952) regarding the Bhopal disaster is potentially misleading. The release of methyl isocyanate (MIC) resulted in at least 1700 deaths—most of them, it is thought by clinicians who were in Bhopal soon after the event, from acute pulmonary oedema resulting from the burns during the highly exothermic reaction of MIC with water in lung secretions and tissues, and not HCN. During this very rapid reaction MIC breaks down to methylamine and the toxic dimethylurea: it is also probable that, on its own, MIC can alkylate a variety of tissues that contain amino and hydroxyl groups. In vitro MIC itself can also produce "cherry red blood", due to the formation of methylamine.[2] Experiments with laboratory animals produced evidence incompatible with cyanide involvement in the fate of Bhopal survivors.[3,4] Cyanide is not necessary to explain any of the acute effects. Acutely MIC is several times more toxic than HCN, but the real danger of a mistaken emphasis on cyanide is the underestimation of long-term effects. Cyanide is unlikely to produce long-term effects while MIC exposure is associated with considerable long-term effects.

Among survivors from Bhopal chronic effects on the lungs have been documented up to two years after the event.[5] A large case-control study confirmed that increased risk of persistent adverse eye symptoms was related to duration of exposure to the gas.[6] Similar long-term ocular effects were found by other physicians.[7] Several studies of Bhopal survivors suggest long-term immunological effects, including the potential of MIC to produce hypersensitivity reactions.[8] Developmental and reproductive effects seem probable[9,10] and neuromuscular complications may also be a problem area.[7]

Few toxicological studies have been done on the long-term consequence of exposure to MIC, but the evidence is not encouraging. ECG abnormalities were found in rats four months after a single exposure. Chronic respiratory damage and chronic eye inflammation appear to be important features. Formal immunological studies indicate persistent bone marrow suppression, suppression of the lymphoproliferative responses of splenic leucocytes to B and T cell mitogens, and decreased thymus/body weight ratios. MIC is also mutagenic, and increased fetal deaths and reduced placental weights and fetal body weights were observed in mice exposed to MIC during gestation. Carcinogenicity studies are underway.

The evidence from human and toxicological studies is not easily explained by the presence or absence of HCN. It now seems certain, however, that there are long-term multisystem consequences of the exposure to MIC. Further research is best directed at MIC and its degradation products, with special attention to immunological consequences of exposure. To exclude such consequences among survivors formal study of infections and allergies is required. Much light will be shed on the issue when systematic evaluation of human post-mortem material becomes available.

For further references see *Environ Health Perspect* 1987; 72: pp 35, 95, 133, 139, 143, 149, 159, 169, 183, 189. A full list of 32 papers on the toxicology of MIC is available.

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1. Andersson N, Kerr Muir M, Mehra V, Salmon AG. Exposure and response to methyl isocyanate: results of a community based survey in Bhopal. *Br J Ind Med* 1988; 45: 469–75.
2. Salmon AG. Bright red blood of Bhopal victims: cyanide or MIC? *Br J Ind Med* 1986; 43: 502.
3. Nemery B, Dinsdale D, Sparrow S, Ray D. Effects of methyl isocyanate on the respiratory tract of rat. *Br J Ind Med* 1985; 42: 799–805.
4. Bucher JR, Gupta BN, Adkins B, et al. Toxicity of inhaled methyl isocyanate in F344/N rats and B6C3F1 mice. Acute exposure and recovery studies. *Environ Health Perspect* 1987; 72: 53–61.
5. Kamat SR, Patel MH, Pradhan PV, et al. Sequential respiratory psychologic and immunologic studies in relation to MIC exposed subjects at Bhopal: interim report of analysis after two years follow-up. Bombay, India: Department of Respiratory Medicine, Seth GS Medical College.
6. Andersson N, Kerr Muir M, Aiwani MK, Mahashabde S, Salmon A, Vaidyanathan K. Persistent eye watering among Bhopal survivors. *Lancet* 1986; ii: 1152.
7. Misra NP, Pathak R, Gaurr KJBS, et al. Clinical profile of gas leak victims in acute phase after Bhopal episode. *Indian J Med Res* 1987; 86 (suppl): 11–19.
8. Kamil MH, Kamat SR. The antibody response to methyl isocyanate: experimental and clinical findings. *Bull Ist Pneyopathol Resp* 1988; 23: 591–97.
9. ICMR. Health effects of the Bhopal gas tragedy. New Delhi, India: Indian Council of Medical Research, 1986.
10. Kashere S, Darbari BS, Shrivastava AK. Morphological study of placentae of expectant mothers exposed to gas leak at Bhopal. *Indian J Med Res* 1987; 86 (suppl): 77–82.

DIFFICULTY IN DETECTION OF HERNIA AND HYDROCELE IN CRYPTORCHIDISM

SIR,—To avoid treatment of retractile testicles, boys suspected of undescended testes should be examined by experienced investigators.[1] But how accurate is the expert in identifying concurrent inguinal abnormalities such as hernia and hydrocele, which represent contraindications to hormonal treatment?

We examined 121 consecutive boys (135 testes) who were found to be truly cryptorchid by a consultant in paediatric surgery. Median age was 6 years (range 0–15). The preoperative position was compared with the intraoperative finding, and classified as not palpable, ectopic, intracanalicular, in the superficial ring, pubic, at the penile base, and just above the scrotal base. In 7 cases, a hernia was found preoperatively and a hydrocele was found in 1 patient.

The intraoperatively found positions correlated significantly with preoperative findings (Spearman's rho = 0·72, $p < 0·001$), but a further 24 inguinal hernias were found and 6 other boys had unexpected hydrocele (table).

The frequency of hernia is similar to other series.[2] The diagnostic probabilities indicate that palpation alone is not sufficient as a test

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Health effects of the Bhopal gas leak: a review

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Introduction

The world's worst industrial disaster occurred in India on the night of December 2-3, 1984. The accident took place at the Union Carbide plant situated in Bhopal, (pop. 900,000) the capital city of Madhya Pradesh, one of the largest states in India.

The accident was apparently initiated by the introduction of water into the methyl isocyanate (MIC) storage tank resulting in an uncontrollable reaction with liberation of heat and escape of MIC in the form of a gas. Safety systems like the flare tower (to burn excess gas), caustic soda scrubber (for neutralization) and the refrigeration unit were either not functioning or inadequate to deal with the large volume of escaping chemical [1].

This paper reviews health effects of gas exposures from published human studies and discusses some of the clinical and epidemiological issues being debated. Because of the relative paucity of information, the author has also reviewed unpublished data from studies conducted by local physician groups. Some of these studies have helped to highlight specific health problems from the disaster and initiate more organized research to address these problems. The reader may also wish to consult articles by John Bucher [2] and Mehta *et al.* [3] for reviews on human and animal toxicology.

Manufacturing Process

Methyl isocyanate (CH_3-N=C=O) is an intermediate product in the manufacture of carbaryl (Sevin), a carbamate pesticide. The process begins with a mixture of carbon monoxide and chlorine to form phosgene. Phosgene is then combined with monomethylamine to form methyl isocyanate. Methyl isocyanate is further mixed with naphthol to produce the end-product, carbaryl.

Chemical and Physical Properties

MIC is a clear, colorless liquid with a pungent odor (b.p.: 39°C, f.p.: -80°C, s.g.: 0.96, m.w.: 57.1, vapor pressure of 348 mm Hg at 20°C). It is moderately soluble in water and hydrolyses on contact to form carbon dioxide and methylamine. When MIC is pyrolyzed in the temperature range of 427-548°C, decomposition products like hydrogen cyanide and carbon dioxide are formed [4].

Toxicological Properties

MIC is highly irritant to the skin, eyes and mucus membranes of the respiratory tract. This irritant property is based on its reactivity with water which enables it to penetrate tissues and interact with protein. Absorption through the skin is known to occur [5]. Because the ACGIH TLV of 0.02 ppm is less than the mucus membrane irritation threshold, (> 0.4 ppm), and the odor threshold (> 2 ppm), the compound is considered to have poor warning properties [6].

Exposure Conditions

It is estimated that about 27 tons of methyl isocyanate escaped from the two tanks in the plant during a period of one to two hours [1]. The release occurred around midnight with adverse prevailing atmospheric conditions (inversion and a low wind speed) which prevented dispersion of the gas [7]. Eyewitness accounts report that a cloud of gas enveloped the area and moved slowly along.

Because of the unexpectedness, time and brief period of release of the gas, air monitoring was not possible nor was it subsequently attempted.

Based on quantity of the chemical released and area of spread, (40 sq. km) the Central Water & Air Pollution Control Board estimated MIC concentration to be about 27 ppm [7], a figure which is about 1400 times that of the OSHA workplace standard of 0.02 ppm for 8 hrs. This calculation, however, assumes equal concentration over the whole area of contamination and does not account for variability of concentration with distance. Using an analytic dispersion model, Singh

and Ghosh have provided simulations of exposure concentrations at various distances downwind of the plant [8]. Twenty seven sites were identified with ground level concentrations ranging from 85.6 ppm to 0.12 ppm with a median of 1.8 ppm.

Factors contributing to variability in human exposure include distance of residence from the plant, duration of exposure and activity during exposure. No systematic attempts have been made to reconstruct individual exposure based on these criteria. In the actual incident, activity during exposure to the gas was certainly a major dose-regulating factor. The acute irritant effects of MIC created panic, great anxiety and disorientation, resulting in people running out of their homes. The running resulted in increased ventilatory rates in these people, thereby increasing the dose of the chemical delivered to the respiratory system. The Medico Friend Circle study reports exposure history and safety measures taken by people at the time of the gas leak [9]. In a study sample of 158 persons, 124 ran out of their homes. Only 12 persons used a wet towel or blanket as a safety measure.

Major organs of exposure to the gas were the eyes and respiratory tract. Significant amounts of MIC were also, most probably, dissolved in saliva and swallowed into the gastro-intestinal tract. Skin exposure certainly took place but was less clinically important than the respiratory tract.

A cohort of 80,021 has been registered from the population residing around the Union Carbide plant and classified as mild, moderate and severely exposed based on mortality rates in each area [10]. An unexposed population in Bhopal was used to select the control group of 15,931 individuals.

Characteristics of the exposed population

In the exposed areas, about 53% of the population comprise of Hindus, 45.6% are Muslims and the rest are Christians and Sikhs. In 1985, the monthly income of 80% of the population was below Rs 145/ month ($6) and only 1.25% earned over Rs 465/month ($18). By 1988, 65% of the population was earning below Rs 145/ month, indicating some improvement in economic level. However, the above figures indicate that most of the exposed people are close to the government-defined poverty level of Rs 300/month ($12). Ten percent of the population are smokers, 1.4% alcoholics and 5.5% chew tobacco. Only 34% of the population live in a 'pacca' (permanent) house [10].

Mortality

Of the more than 200,000 persons exposed to the gas, the initial death toll within a week following the accident was over 2,500. In Nov. 1989, the Dept. of Relief & Rehabilitation, Govt. of Madhya Pradesh, placed the toll at 3598 [11]. Dr. SR Kamat, a pulmonary physician who has conducted studies on a large number of gas victims, states that most of the later deaths appear to be occurring from respiratory complications (personal communication, 1990). Mortality rates are declined but were still slightly higher in the severely exposed area (8.75/1000) in comparison with the control area (7.5/1000) during the period May 1989-Mar 90 [10].

Overall Morbidity

Symptom prevalence surveys conducted by the Indian Council of Medical Research (ICMR) indicate that morbidity was higher in the exposed areas (26%) as compared with the control area (18%) during the period Nov 1988-Mar 90. About 11% of people experienced 2 or more spells of illness in a 1-year period. Respiratory, ocular and gastro-intestinal symptoms account for most of this morbidity. This trend appears to be persistent in the survey conducted in the latter part of 1990 [12].

Ocular Problems

The intensely irritating effect of MIC on the cornea resulted in severe ocular burning, watering, pain and photophobia [13]. Examination of the eye showed involvement of the corneal and conjunctival epithelium with redness of the eye, corneal ulceration and lid swelling. Fortunately, the corneal ulceration in most cases turned out to be limited to the superficial layers of the cornea. Slit lamp examination showed discrete lesions in a band across the interpalpebral area, punctate keratopathy, conjunctival chemosis and some pigmentary deposition on the cornea [14]. Relatively few cases of iritis were seen [15]. Treatment at the initial stage consisted of saline eyewashes, pupillary dilatation and topical antibiotics.

Andersson and others performed a follow-up study on the eyes of survivors 9 months after the accident and reported that no case of blindness could be found that could be attributed to gas exposure among the nearly 20,000 persons attending the Bhopal Eye Hospital [16]. However, they did find persistent eye watering and other chronic irritant symptoms like burning, itching and redness. Raizada and Dwiwedi [17] studied eye pathology among 1140 exposed persons and found that the main chronic lesions were chronic conjunctivitis, deficiency of tear secretion and persistent corneal opacities.

Animal experiments conducted by Salmon [18] on male Lister hooded rats indicate that the most severe effects on the eye occur at exposure levels of 65 ppm.

Andersson *et al.* [19] performed a follow-up of 93% of exposed and unexposed Bhopal residents 3 years after exposure. Their findings indicated an increased risk of eye infections, hyperresponsive phenomena, (watering, irritation, phlyctens), excess cataracts and resolution of the corneal erosions in exposed persons. These phenomena have been characterized as the 'Bhopal eye syndrome'. The authors state "in its response to MIC, the eye should be considered a 'sentinel organ' for more general phenomena in the body".

Though there is no evidence that severe damage to the eye's external and internal structures has occurred, the single acute exposure seems to have resulted in a chronic inflammatory process. The problems

of persistent eye watering in some cases and tear secretion deficiency in others coupled with chronic conjunctivitis indicate that there is some damage to the eye epithelium. It is conceivable that housing conditions in the Bhopal slum areas like overcrowding, poor ventilation and exposure to dust and smoke may exacerbate the ocular effects causing irritation and infections.

Respiratory Toxicity

Acute symptoms of the respiratory tract were mainly due to the irritant action of MIC on tissues. Because MIC is moderately soluble in water, lesions were seen in both the upper and lower respiratory tract. Predominant symptoms were cough accompanied by frothy expectoration, a feeling of suffocation, chest pain and breathlessness [20]. Other symptoms included dryness and irritation of the throat and rhinorrhea.

Autopsies on 300 victims revealed severe necrotizing lesions in the lining of the upper respiratory tract as well as in the bronchioles, alveoli and lung capillaries. Enlarged and edematous lungs, consolidation, hemorrhage, bronchopneumonia and acute bronchiolitis were seen [21].

Follow-up studies were conducted 2.5-3 months after the accident by a team from the Industrial Toxicology Research Centre (ITRC) at Lucknow, India [22]. Exposed persons were contacted and requested to come forward voluntarily to be examined. A total of 1279 men, women and children were examined. In these studies, radiological examination of the lungs was carried out on 903 subjects to assess damage to the respiratory tract. Out of 164 abnormal X-rays, 65 were determined to have specific radiological changes which were considered to have occurred or aggravated as a result of gas exposure. These were classified into two groups:

Group A (48 subjects) were those with radiological abnormalities thought to have occurred as a result of gas exposure. These changes were haziness in different zones of the lung, hilar prominence, fine mottling and reticulation. 16 of the 48 subjects had respiratory symptoms (cough, chest pain/tightness, breathlessness) clinical signs (adventitious sounds) and abnormal lung function. Twenty subjects had respiratory symptoms but clinical examination and lung function were normal.

Group B (17 subjects) showed abnormalities suggestive of old disease which was aggravated (symptoms appearing after exposure) by exposure to the gas. These abnormalities were tuberculosis, chronic bronchitis and pneumonitis.

Spirometry was carried out on 783 subjects from the above sample to determine respiratory impairment [23]. Vital capacity (VC), forced vital capacity (FVC) and forced expiratory volume in 1 sec ($FEV_{1.0}$, FEV_1) were the main parameters recorded. Impairment was classified as restrictive (VC or FVC $<$ 80% of predicted), obstructive ($FEV_1/FVC < 70\%$) and a combined pattern (FVC $<$ 80% of predicted and FEV_1/FVC ratio $< 70\%$).

The results showed that 39% of the sample was found to have some form of respiratory impairment. The combined pattern of impairment (obstructive and restrictive disease) had the highest prevalence in the sample (22%). Smoking had no effect on the prevalence of this impairment. Females suffered more mild and moderate impairment. Severe impairment was equally distributed (2.4%) among the two sexes.

Broncho-alveolar lavage fluid was analyzed in 36 mild, moderate and severely exposed persons and 12 unexposed normal controls 1-2.5 years after exposure [24]. The results indicated that severely exposed smokers and non-smokers showed a significant increase in alveolar macrophages. Based on these results, the authors concluded that an inflammatory alveolitis may be present in severely exposed subjects and that long-term follow-up must be done to determine if further impairment of lung function occurs.

Sharma *et al.* [26] report the interesting case of a resident of the gas-exposed area who, in his professional capacity, continued to be exposed for a few days to trapped gases in victims. This 60-yr old nonsmoker had no respiratory symptoms (other than bouts of cough) till the beginning of 1989 when he started complaining of severe cough and breathiessness on exertion. Pulmonary function showed a mild obstructive ventilatory defect and CT scan revealed subpleural thickening, punctate lesions and bilateral septal scars, suggesting that extensive pulmonary fibrosis had occurred.

Though isocyanates are known to be allergenic in the lung [26] the respiratory toxicity of MIC appears to be primarily due to its irritant nature. Follow-up studies with lung biopsies done six months after exposure showed evidence of interstitial fibrosis and bronchiolitis obliterans. These findings were similar to those found in several animal studies [27,28] thus revealing the close association between animal data and clinical findings in Bhopal victims.

Reproductive Toxicity

Concerns that the gas leak had effects on reproductive health were raised early in 1985 when reports indicated that menstrual cycle disruption, leucorrhea and dysmenorrhea had occurred in gas-exposed women [29]. Risk to the fetus was considered because of exposure to the gas and factors like stress, anoxia and ingestion of various prescribed drugs like antibiotics, bronchodilators, and analgesics.

An epidemiological survey by Varma [30] showed pregnancy loss and infant mortality to be high in gas-exposed women. In a sample of 865 women who lived within 1 km of the plant and who were pregnant at the time of the gas leak, 43% of the pregnancies did not result in live births. Of the 486 live births, 14% of babies died in the first 30 days as compared to a death rate of 2.6% to 3% for previous deliveries in the 2 years preceding the accident in the same group of women.

A pregnancy outcome survey of gas-exposed women [31] using historic controls, demonstrated a four-fold increase in

spontaneous abortion rate. Alteration in menstrual cycle lenght was also observed.

Animal experiments conducted by Schwetz [32] and Varma [33] exposing pregnant mice to MIC by inhalation showed that this exposure does indeed have a fetotoxic effect. Varma observed a concentration-dependant increase in embryo loss, decrease in fetal and placental weights and a 20% reduction in length of mandible and bones of the extremities.

Varma *et al.* [34] studied the contribution of maternal hormonal changes and pulmonary damage to fetal toxicity of MIC in rats and mice. Their findings showed that fetal toxicity of MIC was partly independent of maternal pulmonary damage and that MIC could be directly fetotoxic. In the Bhopal situation, results from the animal studies, when considered with the findings from human epidemiology, suggest that exposure to MIC is fetotoxic and that this is probably the result of a direct effect on the fetus.

Genotoxicity and Carcinogenicity

Chromosomal studies were done two and a half months after the gas leak to evaluate genetic damage among the sample of gas-exposed survivors studied by the ITRC [35]. Blood was collected from 31 exposed adults and a similar number of age and sex-matched unexposed controls to assess the occurrence of chromosomal aberrations (breaks and gaps) in lymphocytes. The results show a significantly increased ($p < 0.001$) number of breaks and gaps in the exposed subjects. No follow-up studies were done to see if this effect was persistent.

Cytogenetic studies were done 3 years after exposure on a sample of 40 male and 43 female exposed persons [36]. Results from this study showed statistically higher frequencies of chromosomal aberrations in the exposed group as compared to 46 age and sex-matched unexposed controls. The aberrations were in the form of breaks, gaps, dicentrics, rings, tri and quadri-radial configurations and were more pronunced in female subjects. Sister chromatid exchanges were not significantly different. The authors concluded that though the results may indicate a residual effect on T-cell precursors, further studies are required to demonstrate an exposure-effect relationship.

Short-term tests using Chinese hamster ovary cells showed induction of SCEs and chromosomal aberrations both without and with activation by S-9 mix from Aroclor-induced rat liver [37]. Sex-linked recessive tests in Drosophila and the Ames test were negative. The Ames test was also found to be negative by Shelby [38]. Meshram and Rao [39] using a different pre-incubation procedure for the Ames test (10°C for 60 min instead of the standard 37°C) found MIC to be weakly mutagenic.

Shelby *et al.* [38] performed genetic toxicity testing on B6C3F1 mice exposed to MIC by inhalation. Analyses on lymphocytes, bone marrow and lung cells were done using single and multiple exposures. Multiple exposure experiments in the mice showed increased frequencies of SCEs and chromosomal aberrations which were not seen in 2 hr exposures. Delay of cell-cycle time was also reported [40,41].

While animal and in vitro studies demonstrate MIC's potential for genotoxicity, it is not clear that such toxicity has actually occurred in exposed humans.

To assess carcinogenic potential, rats and mice were exposed to MIC for 2 hrs by inhalation. Marginal increases of pheochromocytomas of the adrenal medulla and adenomas of the pancreas were seen but these tumors were not considered clearly related to the exposure [42].

A population-based cancer registry has been established in Bhopal in 1986 to study possible carcinogenic effects of the gas leak. All cases of cancer are being registered and categorised by exposure area [10]. Though it has been predicted that MIC has a significant potential for cancer induction [43], it is not expected that the onset of gas leak-related cancers, if any, will occur before the 30-40 yr lag period.

Immunotoxicity

Following exposure to the gas in Bhopal, there was concern amongst the health authorities that the population might experience an increased rate of infections. Possible reasons for this increased susceptibility included depressed immune function from chemical effects, psychological and physical stress, disruption of normal life (particularly during the 2 mass migrations out of Bhopal), and pulmonary injury.

Immune function was studied [35] in exposed subjects from the ITRC sample two and a half months after exposure to ascertain whether any change had occurred in the immune status. Humoral immunity was assessed by quantitation of immunoglobulins (IgG, IgM, IgA) in over 300 exposed and 10 non-exposed persons. Cell-mediated immunity (CMI) was assessed by phagocytic activity of lymphocytes and quantitation of T-cell rosettes in 19 exposed and 8 non-exposed persons. Results from this study showed that no difference in mean immunoglobulin levels was found when compared to controls. The T-cell population (28%) was found to be less than half that found normally in the Indian population (65%). Significant depression of phagocytic activity of lymphocytes was found as compared to controls.

Concurrent with the human studies, immunotoxicological evaluation of rats exposed to MIC showed a number of positive results (44). Alveolar and peritoneal macrophage function was depressed and exposed rats were susceptible to E. coli endotoxin. Delayed type hypersensitivity was assessed by injecting sheep RBC's into the foot pads and was found to be impaired. Based on these results, the researchers concluded that the gas had a suppressive effect on cell-mediated immunity.

Karol and Kamat [26] found MIC-specific antibodies in guinea-pigs injected with MIC as well as in 12 of 144 human survivors. This showed that MIC was capable of eliciting an immunogenic response. The antibody titers in the human studies were low and transient suggesting a weak response.

Limitations of the human studies include the relatively small sample sizes,

choice of control groups and unclear exposure ascertainment. The above limitations make it difficult to arrive at definitive conclusions regarding immunotoxicity from MIC exposure for the gas victims.

Psychological and Neuro-behavioral Toxicity

Srinivasamurthy and Isaac [45] noted that psychological problems of Bhopal survivors fell into four major categories.

1. Post-traumatic stress disorders which occurred as a result of the tremendous emotional stress of the disaster. Symptoms were anxiety, emotional recall of the event, restlessness, sleep disturbances and generalised weakness and fatiguability.

2. Pathological grief reactions characterised by intense grief, depression, suicidal ideation and guilt feelings arising out a sense of failure to protect their family.

3. Emotional reactions to physical problems: victims with ocular, lung and other problems developed feelings of depression, hostility, insecurity and helplessness.

4. Exacerbation of pre-existing psychiatric problems.

In a psychiatric out-patient service program set up specifically for gas victims, Sethi *et al.* [46] detected 208 persons suffering from mental problems. Of these, 45% were suffering from neuroses, 35% from anxiety states and 9% from adjustment reactions.

Neuro-behavioral tests were conducted on 350 exposed subjects two and a half months after the accident [22]. Auditory and visual memory, attention response speed, and vigilance were found to be significantly impaired in this group as compared to controls. No effect was seen on manual dexterity.

Neuromuscular Toxicity

Neuromuscular symptoms in Bhopal survivors have persisted since the gas leak [10]. These symptoms are mainly tingling, numbness, a sensation of pins and needles in the extremities and muscle aches.

To assess whether MIC was toxic to muscle, Anderson *et al.* [47] evaluated the effects of MIC on rat muscle cells in culture. At lower doses, the formation of muscle fibers was prevented. At higher doses, death of fibroblasts and myoblasts was seen. The findings suggested either an effect on muscle differentiation or selective toxicity to myoblasts.

Clinical Problems

Paucity of information on the toxicology of MIC had created a great deal of confusion and debate about the management of the gas victims. The medical system in Bhopal was severely tested by the twin factors of large numbers of injured people streaming into hospitals and the absence of a definite protocol for treatment of the poisoning. Patients were treated on a symptomatic basis. For ocular problems, atropinization of the eye, local antibiotics and padding were used [14]. Respiratory problems were treated with bronchodilators, steroids, diuretics, antibiotics and oxygen administration.

Questions of clinical importance, some of which still persist to this day, are:

- Were there toxins other than MIC released in the accident?
- What were the specific antidotes?
- Did cyanide poisoning occur in the victims and, if so, how was it manifest and what was the source of the cyanide?
- Does MIC enter the blood-stream and cause multi-systemic disease?
- What were the risks to exposed pregnant women and the unborn child?
- There is no doubt that MIC was clearly the major toxin released in the accident. However, the circumstances leading to the release (hydrolytic and exothermic reaction of water with MIC) raised the possibility of impurities (phosgene) or decomposition products (hydrogen cyanide, nitrogen oxides, carbon monoxide) being present in the gas cloud.

Suspicion that some of the early deaths were due to cyanide poisoning arose from the reporting of rapid fatalities and acute symptoms of syncope, and extreme weakness. Autopsy studies done by Dr. Heeresh Chandra at the local medical college showed two features considered to be characteristic of cyanide poisoning. These were cherry-red discoloration of the blood and other organs, and the unpleasant odor of "bitter almonds" when the lungs were opened [48].

A double-blind clinical trial was performed by the ICMR 2 months after exposure to determine the efficacy of sodium thiosulfate as an antidote to the poisoning [12]. The trial was done on 30 gas-exposed symptomatic patients who were administered sodium thiosulfate, a cyanide antidote, intravenously. The results showed alleviation of symptoms with an 8-10 fold increase in thiocyanate excretion in the urine for 10 out of 15 patients and served to fuel suspicion that cyanide poisoning was involved. (Thiocyanate is the water-soluble detoxification product of cyanide).

On the basis of this study, the ICMR in February 1985 recommended the use of sodium thiosulfate on a mass scale for detoxification of symptomatic gas-exposed persons. Union Carbide denied the possibility of cyanide poisoning and toxicological experts around the world were either unsure or skeptical about cyanide [1].

In this atmosphere of confusion, the local health authorities in Bhopal failed to carry out the recommendations of the ICMR on a widespread and systematic basis.

The scientific hyphotheses seeking to explain the source of the cyanide poisoning and the apparent effectiveness of sodium thiosulfate fell into three categories:

1. *Cyanide from an external source*: That the entry and mixing of water with MIC in the storage tank resulted in a violent exothermic reaction at high temperatures (> 420°C)) which led to the decomposition of MIC to hydrogen cyanide, carbon monoxide and nitrogen oxides. This reaction was first discovered by Blake and Maghsoodi in 1982 and was documented in the NIOSH Occupational health guidelines for MIC [49].

2. *Cyanide from an internal source*: That the MIC was being converted to some form of cyanide after being absorbed into the body resulting in enlargement of the body's cyanogen pool.

3. *Thiosulfate effective against MIC*: That sodium thiosulfate was alleviating the symptoms caused by MIC toxicity. (Sodium thiosulfate was hitherto unknown as an MIC antidote).

Investigators from India [50] investigated the first of the three hypotheses by pyrolysing MIC at 350°C and found that cyanide was produced even at this lower temperature as a decomposition product. They further injected the decomposition products into rats and found that brain cytochrome oxidase activity (the biochemical basis for cyanide toxicity) was significantly depressed.

There has been no evidence to date to support the second hypothesis that MIC is converted to a form of cyanide in the body. Animals exposed to MIC by inhalation have not shown any evidence of cyanide in the blood [51].

Animal studies testing the third speculation have not shown that sodium thiosulfate has any protective effect against direct MIC toxicity [52,53].

If one accepts the possible efficacy of thiosulfate in alleviating symptoms of gas-exposed persons, it must be in the light of the first hypothesis that the cyanide poisoning came from an external source.

Studies to determine chemical composition of the MIC tank residues are currently being undertaken by the ICMR and may shed some light on the toxins released in the accident [10]. Whether cyanide was actually released may never be known with absolute certainty as it involves recreation of the actual conditions leading to the accident.

In the Bhopal situation, administration of an essentially harmless drug like sodium thiosulfate on a mass scale, with adequate follow-up, would have enabled gathering data which would confirm or reject the usefulness of the drug.

Andersson [54], however, points out that acute MIC exposure is more toxic than cyanide and an undue emphasis on cyanide may result in an underestimation of the long-term effects of MIC.

Persistent multi-systemic symptoms have been reported by Bhopal survivors. That exposure to MIC can produce reproductive health problems has been shown in human and animal studies [55]. Ferguson and Alarie [56] have demonstrated that there may be a physiopathological basis for the persistence of multi-systemic symptoms in Bhopal survivors. Their studies on experimental animals have shown that radio-labeled MIC is capable of being absorbed and distributed throughtout the body. These findings have been confirmed by Bhattacharya *et al.* [57] who have shown that MIC binds covalently to tissue proteins in its active form and not as its breakdown product, methylamine.

There is no known antidote for MIC toxicity. If further studies confirm that MIC is indeed distributed in the body, there is a need to develop a method of treatment for MIC poisoning.

Epidemiologic Considerations

In the early period following the accident, clinical treatment of the injured took priority over the planning and conduct of epidemiological studies. This was particularly true given the limited health care resources available for a large number of affected people and the general lack of experience in dealing with a disaster of this nature.

A few cross-sectional studies (Table 1) were done during the early recovery period (6 months) for various systemic health end-points. Virtually all of the epidemiology for the late recovery period is being conducted by the Bhopal Gas Disaster Research Centre, a branch of the Indian Council of Medical Research. About 10 different epidemiological studies were initiated to monitor long-term trends in morbidity and mortality. Results from these studies are currently being awaited. As Bertazzi [58] notes, the selection of a cohort rather than using a population registry for epidemiological studies avoids two major biases: dilution of exposure prevalence and selective migration of people out of the disaster area.

The early cross-sectional studies suffer from a number of defects in study design, resulting in bias and consequent difficulty in clearly establishing causal relationships. In one follow-up study performed 3 months after the accident, exposed persons were contacted and requested to come forward voluntarily for examination [22]. This method of subject selection may have resulted in severely exposed/affected subjects being examined and a consequent over-estimation of health effects. If, however, the severely affected victims were unable to present themselves for examination due to illness, an under-estimation of health effects may have occurred.

Relatively crude methods like mortality rates or distance of residence from the factory have been used in defining community exposures. Koplan *et al.* [59] stated that epidemiologic studies following disasters should accurately estimate exposure to enable correct dose-response relationship modeling. These data are useful for a) identifying exposed and ill persons, b) determine long-term effects and c) link exposure and effects for litigation and compensation.

For the Bhopal gas victims, there is a need to do epidemiological studies to determine morbidity prevalence in the population stratified by estimated pulmonary dose. Such an approach will allow scientifically valid and detailed studies of different health end-points to be performed on relatively small sample sizes [60]. Exposure-based stratified random sampling will also reduce bias due to self-selection and exposure misclassification, as well as permit dose-response and interaction relationships to be understood.

Conclusions

Clinical and toxicological studies have shown that MIC is a potent toxin. Chronic inflammation of the eye and respiratory tract account for a major portion of the morbidity. Certainly the potential exists for these damaged organs to be more susceptible to other environmental insults like infections, irritants, and allergens. For e.g., a person with airway damage

may be more prone to infections or respond adversely to smoke and dust. Pulmonary function limitation may preclude survivors from working on jobs which require moderate or strenuous activity. Progressive pulmonary fibrosis and restrictive lung disease appear to be a major cause for concern among the gas-exposed. Given the completely unexpected and devastating nature of the disaster and the resultant stress, it is expected that a number of survivors will suffer from post-traumatic stress disorders for many years. Establishment of a specialised medical center for dealing with health problems from the gas leak will permit a co-ordinated method of investigation and treatment for the injured.

It is a striking fact that much of the mortality and morbidity could have been averted by the simple expedient of covering the face with a wet cloth. MIC would have been decomposed on contact with the water. Unfortunately, the community was never informed of the existence of such a potent chemical in the factory and contingency measures to be taken in the event of a leak.

For a disaster of this magnitude, there is a relative paucity of knowledge based on epidemiological and clinical investigation. Some of the medical studies done on the survivors suffer from unscientific design and it is difficult to utilise information from these studies with confidence. Stratified sampling techniques using isopleths of exposure from dispersion models may be one way of conducting an epidemiological study without including the total exposed population.

It is imperative that long-term monitoring of the affected community be done for at least the next fifty years. Formal studies of ocular, respiratory, reproductive, immunological, genetic and psychological health must be continued to understand the extent and severity of long-term effects of the disaster.

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Table 1 - Human health effects studies on the Bhopal Gas Leak
Tabella 1 - Studi degli effetti sulla salute umana dell'incidente di Bhopal

Author	*Design*	*n (cases/controls)*	*Study Period (time after gas leak)*	*Major findings*
Ocular Studies				
Andersson et al., 1984, (4)	Case-series	8000 exposed	8 days	chemosis, corneal ulcers, watering photophobia, no blindness
Andersson et al., 1988, (13)	Cross-sectional	261/ 99	2 weeks	photophobia corneal erosions
Dwivedi et al., 1985, (15)	case-series	232	a few weeks	chemosis, redness watering, corneal ulcers
Andersson et al., 1985, (69)	case-series	490	2 months	corneal scars no blindness
Maskati, 1986, (61)	Cross-sectional	261/106	104 days	conjunctivitis corneal opacities
Andersson et al., 1986, (10)	Case-series	989	9-12 months	persistent watering
Raizada, & Dwivedi 1987, (17)	Case-series	1140	2 years	corneal opacities, chronic conjunctivitis
Respiratory Studies				
Mishra et al., 1987, (70)	Case-series	978	2 days	resp. distress, pulm. edema, pneumonitis
Sharma & Gaur, 1987, (62)	Case-series	500 X-rays	> 72 hrs	pulm. edema, emphysema, pneumothorax
Misra & Nag 1988, (20)	Case-series	33	1 week	Dyspnea, upper and lower respiratory tract irritation, pulm. edema, pneumonia
Bhargava et al., 1987, (63)	Case-series	224	1-4 months	Obstructive & restrictive lung disease
Gupta et al., 1988, (22)	Cross-sectional	1109	2.5 months	65 subjects with +ve X-ray changes
Kamat et al., 1985, (64)	Case-series	113	3 & 6 months	Emphysema, pulm. hypertension pleural scars, interstitial depositis
Medico Friend Circle 1986, (9)	Cross-sectional	136/137	4 months	Obstructive & restrictive lung disease
Reproductive Studies				
Varma, 1987, (30)	Survey	865	9 months	Increased pregnancy loss,
		486 live/births/ historic controls	9 months	increased infant mortality
Medico Friend Circle 1986, (31)	Survey	301/historic controls	9 months	increased spont. abortion rate, alteration of menstrual cycle

Table 1 - Human health effects studies on the Bhopal Gas Leak
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Author	Design	n (cases/controls)	Study Period (time after gas leak)	Major findings
Kanhere et al., 1987, (66)	Cross-sectional	134/24	9 months	Decreased placental & fetal weight
Daniel et al., 1987 - (67)	Cross-sectional	18/10	6 months	No effect on spermatogenic function
Genetic Studies				
Deo et al., 1987 (68)	Cross-sectional	22/13	11 days	Cell cycle delay
Saxena et al., 1988, (35)	Cross-sectional	31/31	2.5 months	Increased chromosomal aberrations
Ghosh et al., 1990, (36)	Cross-sectional	83/46	3 years	Increased chromosomal aberrations
Immune Function				
Deo et al., 1987, (68)	Cross-sectional	67/15	4-8 weeks	Decreased response to T & B cell mitogens
Saxena et al. 1988, (35)	Cross-sectional	44 19/8	2.5 months 2-5 months	Decreased T-cell population, decreased phagocytic activity
Karol et al., 1987, (26)	Case-series	144	1-12 months	Transient MIC antibodies in 12 subjects
Psychological Studies				
Sethi et al., 1987, (46)	Case-series	208	2-6 months	Neuroses, anxiety states & adjustment reactions
Neurobehavioral Studies				
Gupta et al., 1988, (22)	Cross-sectional	350/100	2.5 months	Impaired auditory & visual memory, vigilance & attention response speed

Gerard Notarelli, Rosaline Dhara, Drs. Steven Markowitz, Rashid Sheikh, David Kriebel and David Wegman.

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Riassunto

Il peggior disastro industriale della storia si verificò in India la notte tra il 2 e il 3 dicembre 1984 a Bhopal, città di 900000 abitanti e capitale dello stato di Madhya Pradesh. L'incidente fu probabilmente iniziato dall'introduzione di acqua nelle cisterne deposito di Metilisocianato (MIC) e dalla conseguente reazione incontrollata con liberazione di calore e di MIC sotto forma di gas. I sistemi di sicurezza o non funzionarono o furono inadeguati per il grande volume di sostanza chimica coinvolta nella reazione.

Questo lavoro passa in rassegna gli effetti dell'esposizione al MIC così come sono conosciuti da studi umani e discute alcuni degli aspetti clinici ed epidemiologici dibattuti.

Per quanto riguarda l'esposizione della popolazione si stima che circa 27 tonnellate di MIC fuoriuscirono dalle cisterne in un arco di 1-2 ore [1]. L'incidente avvenne intorno a mezzanotte in condizioni metereologiche avverse che prevennirono la dispersione del gas e fecero ristagnare sulla zona una nube tossica [7].

Stime basate sulla quantità di sostanza fuoriuscita e sull'area interessata (40 miglia quadrate) pongono la concentrazione di MIC nell'area a 27ppm, circa 1400 volte lo standard OSHA per i luoghi di lavoro di 0.02 ppm durante le 8 ore [7]. Simulazioni che tenessero conto della variabilità di concentrazione di MIC identificano 27 aree con concentrazioni varianti da 85.6 ppm a 0.12 ppm con una mediana di 1.8 ppm [8]. La dose assunta dipese anche dai comportamenti individuali al momento dell'incidente. In uno studio su un campione di 158 persone, ad esempio, 124 fuggirono dalle case (aumentando quindi con la ventilazione polmonare la quantità di sostanza assunta) e solo 12 utilizzarono stoffa umida davanti alla bocca come forma di protezione [9].

Una coorte di 80021 soggetti fu identificata nella popolazione residente intorno allo stabilimento della Union Carbide e classificata come esposta in modo lieve, moderato e severo sulla base dei dati di mortalità delle diverse aree. Un gruppo di popolazione di Bhopal non esposta (15931 soggetti) venne identificato come controllo. Nelle aree esposte rispettivamente il 53%, 46% e 1% erano di religione indù, musulmana, cristiana o sikh. L'80% della popolazione aveva un reddito inferiore a 6 dollari al mese e solo l'1% superiore a 18 dollari. La soglia di povertà è stabilita dal governo in 12 dollari al mese. Solo il 34% della popolazione viveva in una casa stabile ("pacca" house) [10].

Delle 200000 persone esposte 2500 morirono nella settimana successiva all'incidente. Al novembre 1989 risultava un totale di 3598 vittime decedute prevalentemente per complicazioni respiratorie. Nel periodo maggio 1989 - marzo 1990 la mortalità generale risultava essere più elevata nelle aree esposte (8.75/1000) relativamente alle aree di controllo (7.5/1000) [10].

Surveys sulla prevalenza di sintomi indicano una morbidità più elevata nelle zone esposte (26%) rispetto alle aree di controllo (18%).

L'articolo e la tabella 1 passano in rassegna i lavori condotti sulla patologia oculare, respiratoria, riproduttiva, sulla genotossicità, carcinogenicità e immunotossicità del MIC sulle popolazioni esposte ed i problemi psicologici e di tossicità neuroambientale e neuromuscola.e. Vengono infine valutati i problemi chimici collegati ad una possibile tossicità di altri prodotti presenti nella nube tossica e potenzialmente formatisi durante la reazione di idrolitica ed esotermica.

Considerazioni generali riguardanti gli studi epidemiologici condotti nell'area di Bhopal sono:

1. Nel periodo immediatamente seguente l'incidente la totalità degli sforzi medici si concentrarono sulla cura del numero elevalissimo di intossicati e solo in un secondo periodo vennero pianificati studi al fine di valutare gli effetti cronici ed acuti dell'esposizione alla nube tossica.
2. Gli studi trasversali, i primi ad essere disegnati, soffrirono di numerosi problemi nel disegno dello studio. Gli studi prospettivi vennero disegnati con maggiore attenzione agli aspetti dello distorsioni di selezione.
3. È importante ricordare che larga parte della mortalità e morbidità avrebbe potuto essere evitata con misure di protezione individuale molto semplici, quale il coprirsi il viso con un panno bagnato. Purtroppo la comunità non era mai stata informata né dalla presenza di tossici così potenti nello stabilimento, né di forme di protezione individuale da applicare in caso di incidente.

Summary

The methyl isocyanate (MIC) gas leak from the Union Carbide plant at Bhopal, India in 1984 was the worst industrial disaster in history. Exposure estima-

tes of gas concentrations in the area range from 85 to 0.12 ppm. Of the approximately 200.000 persons exposed, 3598 deaths have resulted as of November 1989. Chronic inflammatory damage to the eyes and lungs appears to be the main cause of morbidity. Reproductive health problems in the form of increased spontaneous abortions and psychological problems have been reported. Questions about the nature of MIC toxicity have been raised by the persistence of multi-systemic symptoms in survivors. Animal studies using radio-labeled MIC given by the inhalation route have shown that the radio-label is capable of crossing the lung membranes and being distributed to many organs of the body. This paper reviews health effects of gas exposure from published studies and discusses some of the clinical and epidemiological issues being debated.

References

1. MOREHOUSE W, SUBRAMANIAM MA. *The Bhopal Tragedy: What Really Happened and What It Means for American Workers and Communities at risk*. Council on International & Public Affairs, New York 1986.
2. BUCHER JR. Methyl Isocyanate: A review of health effects research since Bhopal. *Fund Appl Toxicol* 1987; 9: 367-379.
3. MEHTA PS, MEHTA AS, MEHTA SJ, MAKHIJANI AB. Bhopal Tragedy's Health Effects: A review of Methyl Isocyanate Toxicity. *JAMA* 1990; 264: 2781-2787.
4. BLAKE PG, IJADI-MAGHSOODI S. Kinetics and Mechanism of the Thermal Decomposition of Methyl Isocyanate. *Int J Chem Kinetics* 1982; 14: 945-952.
5. SAX N. IRVING. *Dangerous Properties of Industrial Materials*. 5th ed., New York, Van Nostrand Reinhold Co. 1979; pp. 24.
6. KIMMERLE G, EBEN A. Zur toxicitat von methylisocyanat und dessen quantitiver bestimmung in der luft. *Arch Toxicol* 1964; 20: 235-241.
7. Central Water & Air Pollution Control Board Report: Gas Leak Episode at Bhopal, New Delhi 1985.
8. SINGH MP, GHOSH S. Bhopal Gas Tragedy: Model Simulation of the Dispersion Scenario. *Journal of Hazardous Materials* 1987; 17: 1-22.
9. Medico Friend Circle. *The Bhopal Disaster Aftermatch: An Epidemiological and Socio-Medical Study*. Bangulore, India, 1985.
10. Indian Council of Medical Research. Bhopal Gas Disaster Research Centre, Annual Report. Bhopal, India, 1990.
11. Dept. of Relief & Rehabilitation: *Bhopal Gas Tragedy*. Govt. of Madhya Pradesh, Bhopal, India, 1989.
12. Indian Council of Medical Research. Annual report: Bhopal Gas Disaster Research Centre. *Annual Report*, Bhopal, India, 1991.
13. ANDERSSON N, KERR MUIR M, MEHRA V, SALMON AG. Exposure and response to methyl isocyanate: results of community-based survey in Bhopal. *Br J Ind Med* 1988; 45: 469-475.
14. ANDERSSON N, MUIR MK, MEHRA V. Bhopal Eye. *Lancet* 1984; 2: 1481.
15. DWIVEDI PC, RAIZADA JK, SAINI VK, MITTAL PC. Ocular Lesions following methyl isocyanate contamination: the Bhopal experience. *Arch Ophthalmol* 1985; 103: 1627.
16. ANDERSSON N, KERR MUIR M, AJWANI MK, MAHASHABDE S, SALMON A, VAIDYANATHAN K. Persistent eye watering among Bhopal survivors. *Lancet* 1986; 2: 1152.
17. RAIZADA JK, DWIVEDI PC. Chronic ocular lesions in Bhopal gas tragedy. *Ind J Ophthalmol* 1987; 35: 453-455.
18. SALMON AG, KERR MUIR M, ANDERSSON N. Acute Toxicity of methyl isocyanate: a preliminary study of the dose response for eye and other effects. *Br J Ind Med* 1985; 12: 795-798.
19. ANDERSSON N, AJWANI MK, MAHASHABDE S, TIWARI MK, KERR MUIR M, MEHRA V, ASHIRU K, MACKENZIE CD. Delayed eye and other consequences from exposure to methyl isocyanate 93% follow up of exposed and unexposed cohorts in Bhopal. *Br J Ind Med* 1990; 47: 553-558.
20. MISHRA UK, NAG D. A Clinical Study of Toxic Gas Poisoning in Bhopal, India. *Ind J Exp Biol* 1988; 26: 201-204.
21. Indian Council of Medical Research Health Effects of Exposure to Toxic Gas at Bhopal: an update on ICMR sponsored researches. New Delhi 1985.
22. GUPTA BN, RASTOGI SK, CHANDRA H, MATHUR AK, MAHENDRA PN, PANGTEY BS, KUMAR P, SETH RK, DWIVEDI RS, RAY PK. Effect of exposure to toxic gas on the population of Bhopal: Part I-Epidemiological, clinical, radiological & behavioral studies. *Ind J Exp Biol* 1988; 26: 149-160.
23. RASTOGI SK, GUPTA BN, HUSAIN T, KUMAR A, CHANDRA S, RAY PK. Effect of exposure to toxic gas on the population of Bhopal: Part II-Respiratory Impairment. *Ind J Exp Biol* 1988; 26: 161-164.
24. VIJAYAN VK, PANDEY VP, SANKARAN K, MEHROTRA Y, DARBARI BS, MISRA NP. Bronchoalveolar lavage study in victims of toxic gas leak in Bhopal. *Ind J Med Res* 1989; 90: 407-414.
25. SHARMA S, NARAYANAN PS, SRIRAMACHARI S, VIJAYAN VK, KAMAT SR, CHANDRA H. Objective thoracic CT scan findings in a Bhopal gas disaster victim. *Respir Med* 1991; 85: 539-541.
26. KAROL MH, TASKAR S, GANGAL S, RUBANOFF BF, KAMAT SR. The antibody response to methyl isocyanate: Experimental and clinical findings. *Environ Health Perspect* 1987; 72: 167-173.
27. BOORMAN GA, URAIH LC, GUPTA BN, BUCHER JR. Two hour methyl isocyanate inhalation and 90 day recovery study in B6C3F1 mice. *Environ Health Perspect* 1987; 72: 5-11.
28. FOWLER EH, DODD DE. Respiratory tract changes in guinea pigs, rats and mice following a single six-hour exposure to methyl isocyanate. *Environ Health Perspect* 1987; 72: 107-114.
29. BANG R & SADGOPAL M. Effect of Bhopal disaster on womens' health-an epidemic of gynecological diseases (Part I), 1985, unpublished data.
30. VARMA DR. Epidemiological and experimental studies on the effects of methyl isocyanate on the course of pregnancy. *Environ Hlth Perspect* 1987; 72: 151-155.
31. Medico Friend Circle: Effect of Bhopal Gas Leak on Women's Reproductive Health. Published by Padma Prakash on behalf of Medico Friend Circle at IBCS, Bombay, India, 1986.
32. SCHWETZ BA, ADKINS B Jr, HARRIS M, MOORMAN M, SLOAN R. Methyl Isocyanate: Reproductive and developmental toxicology studies in Swiss mice. *Environ Hlth Perspect* 1987; 72: 147-150.
33. VARMA DR, FERGUSON J, ALARIE Y. Reproductive toxicity of methyl isocyanate in mice. *J Toxicol Env Hlth* 1987; 21: 265-275.
34. VARMA DR, GUEST I, SMITH S, MULAYS. Dissociation between maternal and fetal toxicity of methyl isocyanate in mice and rats. *J Toxicol Env Hlth* 1990; 30: 1-14.
35. SAXENA AK, SINGH KP, NAGLE SL, GUPTA BN, RAY PK, SRIVASTAV RK, TEWARI SP, SINGH R. Effect of exposure to toxic gas on the population of Bhopal: Part IV - Immunological and Chromosomal Studies. *Ind J Exp Biol* 1988; 26: 173-176.
36. GHOSH BB, SENGUPTA S, ROY A, MAITY S, GHOSH S, TALUKDER G, SHARMA A. Cytogenetic Studies in Human Population Exposed to Gas Leak at Bhopal, India. *Environ Hlth Perspect* 1990; 86: 323-326.
37. MASON JM, ZEIGER E, HAWORTH S, IVETT J, VALENCIA R. Genotoxicity studies of methylisocyanate in Salmonella, Drosophila and cultured Chinese hamster ovary cells. *Environ Mutagen* 1986; 9: 19-28.
38. SHELBY MD, ALLEN JW, CASPARY WJ, HAWORTH S, IVETT J, KLIGERMAN A, LUKE CA, MASON JM, MYHR B, TICE RR, VALENCIA R, ZEIGER E. Results of in vitro and in vivo genetic toxicity tests on methyl isocyanate. *Environ Hlth Perspect* 1987; 72: 181-185.
39. MESHRAM GP, RAO KM. Mutagenic and toxic effects of methylisocyanate (MIC) in Salmonella typhimurium. *Ind J Exp Biol Hlth* 1987; 25: 548-550.
40. TICE RR, LUKE CA, SHELBY MD. Methyl Isocyanate: an evaluation of in vivo cytogenetic activity. *Environ Mutagen* 1986; 9: 37-58.
41. CONNER MK, RUBINSON HF, FERGUSON JS, STOCK MF & ALARIE Y. Evaluation of sister chromatid exchange and cytogenicity in murine tissues in vivo and lymphocytes in vitro following methyl isocyanate exposure. *Environ Hlth Perspect* 1987; 72: 115-121.
42. BUCHER JR, URAIH LC. Carcinogenicity and Pulmonary Pathology Associated With a Single 2-Hour Inhalation Exposure of Laboratory Rodents to Methyl Isocyanate. *J Nat Canc Inst* 1989; 81: 586-587.
43. ENNEVER FK, ROSENKRANZ HS. Evaluating the potential for genotoxic carcinogenicity of methyl isocyanate. *Toxicol Appl Pharmacol* 1987; 91: 502-505.
44. DWIVEDI PD, MISHRA A, GUPTA GSD, DUTTA KK, DAS SN, RAY PK. Inhalation toxicity studies of methyl isocyanate (MIC) in rats: Part IV-Immunologic response of rats one week after exposure: effect on body and organ weights, phagocytic and DTH Response. *Ind J Exp Biol* 1988; 26: 191-194.
45. SRINIVASAMURTHY R, ISAAC MK. Mental health needs of Bhopal disaster victims and training of medical officers in mental health aspects. *Ind J Med Res* 1987; 86 (Suppl): 51-58.
46. SETHI BB, SHARMA M, TRIVEDI HK, SINGH H. Psychiatric morbidity in patients attending clinics in gas affected areas in Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 45-50.
47. ANDERSON D, GOYLE S, PHILIPS BJ, TEE A, BEECH L, BUTLER WH. Effect of methyl isocyanate on rat muscle cells in culture. *Br J Ind Med* 1988; 45(4): 269-274.
48. FERA I. The Day After. *Illus Week of India* Dec 30, 1984. In *Bhopal: Industrial Genocide?* Hong Kong, 1985 *Arena Press*.
49. National Institute of Occupational Safety & Health. *Occupational health Guidelines for Chemical Hazards: Methyl Isocyanate*, Us. Dept. of Health Human Services, Washington, D.C., 1978.
50. BHATTACHARYA BK, MALHOTRA RC, CHATTOPADHYAY DP. Inhibition of rat cytochrome oxidase activity by pyrrolysed products of methyl isocyanate. *Toxicol Lett* 1987; 37: 131-134.
51. BUCHER JR, GUPTA BN, ADKINS B Jr, THOMPSON M, JAMIESON CW, THIGPEN JE, SCHWETZ BA. Toxicity of Inhaled Methyl Isocyanate in F344/N Rats and B6C3F1 Mice. I. Acute Exposure and Recovery Studies. *Environ Health Perspect* 1987; 72: 53-61.
52. NEMERY B, SPARROW S, DINSDALE D. Methyl

Isocyanate: Thiosulphate does not protect. *Lancet* 1985; 2: 1245-1246.
53. VIJAYARAGHAVAN R, KAUSHIK MP. Acute toxicity of methyl isocyanate and ineffectiveness of sodium thiosulphate in preventing its toxicity. *Ind J Exp Biol* Aug 1987; 25: 531-534.
54. JANDERSSON N. Long term effects of methyl isocyanate. *Lancet* 1989; 1: 1259.
55. DHARA VR. On the bioavailability of methyl isocyanate in the Bhopal gas leak. Editorial. *Arch Env Health* 1992 ; 47: 385-386.
56. FERGUSON JS, KENNEDY AL, STOCK MF, BROWN WE, ALARIE Y. Uptake and distribution of ^{14}C during and following exposure to [^{14}C] methyl isocyanate. *Toxicol Appl Pharmacol* 1988; 94: 104-117.
57. BHATTACHARYA BK, SHARMA SK, JAISWAL DK. In vivo binding of [^{14}C] methylisocyanate to various tissue proteins. *Biochem Pharmacol* 1988; 37: 2489-2493.
58. BERTAZZI PA. Industrial disasters and epidemiology: a review of recent experiences. *Scand J Work Environ Health* 1989; 15: 85-100.
59. KOPLAN JP, FALK H, GREEN G. Public Health Lessons from the Bhopal Chemical Disaster. *JAMA* 1990; 264: 2795-2796.
60. DHARA VR, KRIEBEL D. The long-term health consequences of the Bhopal gas disaster: dose-response studies. Apha 120th Annual Meeting Exhibition Abstracts. Washington, D.C., 1992; 130.
61. MASKATI QB. Ophthalmic survey of Bhopal victims 104 days after the tragedy. *J Postgrad Med* 1986; 32: 199-202.
62. SHARMA PN, GAUR KJBS. Radiological spectrum of lung changes in gas exposed victims. *Ind J Med Res* 1987; 86 (Suppl): 39-44.
63. BHARGAVA DK, VARMA A, BATNI G, MISRA NP, TIWARI UC, VIJAYAN VK, JAIN SK. Early observations on lung function studies in symptomatic 'gas'-exposed populations of Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 1-10.
64. KAMAT SR, MAHASHUR AA, TIWARI AK, POTDAR PV. Early observations on pulmonary changes and clinical morbidity due to the isocyanate gas leak at Bhopal. *J Postgrad Med* 1985; 31: 61-72.
65. KAMAT SR, PATEL MH, KOLHATKAR VP, DAVE AA, MAHASHUR AA. Sequential respiratory changes in those exposed to the gas leak at Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 20-38.
66. KANHERE S, DARBARI BS, SHRIVASTAVA AK. Morphological study of expectant mothers exposed to gas leak at Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 77-82.
67. DANIEL CS, SINGH AK, SIDDIQUI P, MATHUR BBL, DAS SK, AGARWAL SS. Preliminary report on spermatogenic function of male subjects exposed to gas at Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 83-86.
68. DEO MG, GANGAL S, BHISEY AN, SOMASUNDARAM R, BALSARA B, GULWANI B, DARBARI BS, BHIDE S, MARU GB. Immunological, mutagenic and genotoxic investigations in gas exposed population of Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 63-76.
69. ANDERSSON N, KERR MUIR M, SALMON AG, WELLS CJ, BROWN RB, et al. Bhopal Disaster: Eye follow-up and analytical chemistry. *Lancet* 1985; 1: 761-762.
70. MISRA NP, PATHAK R, GAUR KJBS, JAIN SC, YESIKAR SS, MANORIA PC, et al. Clinical profile of gas leak victims in acute phase after Bhopal episode. *Ind J Med Res* 1987; 86 (Suppl): 11-19.

La Cooperativa Epidemiologia e Prevenzione annuncia il

TERZO CORSO DI PIANIFICAZIONE, GESTIONE E VALUTAZIONE DEGLI SCREENING IN ONCOLOGIA

Istituto per lo Studio e la Cura dei Tumori "Fondazione Pascale"
Via M. Semmola, Napoli

Napoli, lunedì 6 – venerdì 10 settembre 1993

Programma provvisorio

1 – Le basi logiche dello screening e della sua valutazione
2 – Prevenzione primaria verso diagnosi precoce
Le conoscenze eziologiche:
- sul carcinoma della cervice (problema del Papilloma Virus)
- sul carcinoma della mammella (problema del rischio da radiazioni)
- sul carcinoma dell'intestino (problema della famiglia ad alto rischio)
- sugli altri tumori per cui sono stati proposti programmi di screening (endometrio, ovaio, tumori cutanei, prostata, neuroblastoma)

Il problema del riconoscimento dei soggetti ad alto rischio

Rischio attribuibile e rischio prevenibile
3 – Relazione fra screening in campo oncologico e cardiovascolare
4 – Metodologia epidemiologica generale e degli studi valutativi:
- Confronti geografici e temporali
- Studi di coorte, studi casi-controlli
- Studi sperimentali

5 – Analisi dei principali studi valutativi (con esercizi sui tumori della cervice, della mammella e dell'intestino)
6 – Analisi dello osporionzo o doi programmi italiani
7 – Analisi dei costi
8 – Simulazione dell'impatto della diagnosi precoce sulla mortalità, sulla qualità della vita e sulla spesa sanitaria
9 – Indicatori di efficienza dei programmi
10 – Organizzazione del sistema informativo per monitorare l'efficienza e l'efficacia in differenti situazione operative

Docenti: dott. Franco Berrino (Ist. Tumori, Milano)
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dott. Salvatore Panico (Ist. Med. Int. e Malattie Dismetaboliche, Un. Federico II, Napoli)
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Commentary

Public Health Lessons From the Bhopal Chemical Disaster

The 1984 CHEMICAL disaster in Bhopal, India, was first and foremost a terrible human tragedy. For those who were there and even for those at considerable distances who read about it, the reality of 2000 or more persons dead and many more of thousands poisoned by a toxic cloud is horrifying. However, in its particulars and complexities, Bhopal's chemical disaster can also serve as a case example for almost any discipline taught in a school of public health. The disaster has elements of acute and chronic epidemiology, industrial hygiene, toxicology, environmental pollution and planning, disaster preparedness and management, health economics, medical ethics, and environmental protection law, to name a few. Mehta et al[1] review the literature on the Bhopal disaster and its aftermath and focus on the long-term health effects. They recognize the incomplete nature of much of the data and the serious methodologic limitations for study that the circumstances of the event engendered. What conclusions can be drawn from this event? What can we learn to better prepare ourselves for similar events that might occur in the future?

See also p 2781.

There is an inherent catch-22 in doing a health evaluation of a disaster. The time period just following the event—when there remains considerable chaos, confusion, and inaccessibility of means of transport, communication, and data collection—is the very best time to try to establish information that will become invaluable in determining the health effects. This occurs is common to natural disasters such as the Mount Saint Helens volcano eruption or man-mediated ones such as the Bhopal or the nuclear contamination at Chernobyl. This immediate postdisaster time period is also when it is most difficult to organize systematic and valid epidemiologic studies.[2]

The information that is usually of most interest relates to mortality and morbidity (ie, the public health impact), exposure, and environmental damage. Depending on the setting, epidemiologic studies following environmental disasters may focus on some or all of the following: (1) accurate estimates of exposure; (2) correlation of environmental and human exposure data; (3) relationship of exposure (or dose) to observed health effects; (4) the potential interaction of other risk factors with exposure in producing health effects; (5) the natural history of the disaster-related illness (especially if, as with methyl isocyanate [MIC], such illness has not been well documented in the past); (6) impact of therapy or progression of disease (particularly in situations where the therapeutic approach is uncertain—in Bhopal this arose because of questions concerning possible toxic effects of cyanide); (7) effectiveness of screening and diagnostic tests (in determining who was affected and to what extent); (8) identification of markers of prognosis (in Bhopal a critical issue was who among the many individuals with acute respiratory problems would ultimately develop chronic pulmonary toxic effects); (9) evaluation of the effectiveness of disaster plans (including the implementation of warning systems, evacuation procedures, and the provision of emergency medical services); and (10) the psychosocial impact of the disaster on the affected population.[3]

These data are needed for several purposes: (1) to identify exposed and clinically ill persons to provide long-term care and monitoring for their own well-being; (2) to improve contingency planning for future disasters; (3) to determine the short- and long-term health effects; and (4) to link exposure and health consequences for litigation and reimbursement. The acquisition of scientific knowledge is not an intellectual exercise. Rather, it provides information that can help prevent or better control a similar disaster in the future.[4]

A rough estimate of deaths can sometimes be obtained in a disaster setting in a developing country by conducting a survey at the site soon after the event.[5] In Bhopal, this meant that in the first 5 days after the event, sample surveys might have been conducted in selected areas of the city. While there was some disruption of families, much of the local population was still present up to 10 days after the event. Available interviewers with appropriate supervision could have been given brief training and a data collection form and sent out to obtain rough rates of death and illness, along with other variables. In disaster situations there are often many practical impediments to collecting what would otherwise seem to be readily available information. For instance, in Bhopal on the night of the disaster, so many patients were seen at the major hospitals that even rudimentary medical records were not available for most patients; this would complicate unbiased sample selection for clinical epidemiologic studies of hospitalized patients. Such practical constraints ranged from the unavailability of death certificates or medical records to the lack of prior census data, very limited numbers of on-the-scene epidemiologists or investigators, and insufficient envi-

From the Centers for Disease Control, Atlanta, Ga (Drs Koplan and Falk); the All India Institute of Medical Sciences, New Delhi (Dr Ramalingaswami); and the Harvard School of Public Health, Boston, Mass (Dr Green).
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ronmental data (or even the equipment to collect such data). The constraints to the conduct of health studies were especially severe in Bhopal because of the enormous scope of this environmental disaster.

Determining health effects, particularly long-term ones, requires a knowledge of the amount of exposure. Without such information, diseases and conditions that develop over time in a population tend to be more readily noticed, added together, and ascribed to the presumed exposure. Comparing such a newly measured incidence of an adverse health event with a neighboring nonexposed community or with the same community at a time prior to the disaster is problematic. Even neighboring communities often may have characteristics that are different from the case community, such as differing customs, different environmental exposures (the case community may have other toxic hazards in addition to those associated with the disaster), and different food or water sources. A comparison with an earlier time period is often not possible due to the lack of carefully collected data on the health condition in question prior to the disaster and the problem of recall bias. Measuring exposure is made considerably easier when a biomarker of exposure, such as a blood chemistry level or determination of the level of radioactivity, can be obtained.

For the exposure to MIC at Bhopal, such a marker had not been identified. An alternative approach is to establish a dose-response relationship between a measure of environmental exposure and a health event. In the aftermath of the disaster at Bhopal, this was not possible, because wide-scale environmental measurements of MIC (or breakdown products) could not be obtained in time. Approximations were based on less precise measures of exposure such as how close an individual lived to the plant. Actual exposures would be affected by many other factors, such as the height of the patient from the ground, the degree of ventilation of the house, and the shielding of the patient from the MIC vapors. It was also noted that degrees of damage to local vegetation could serve as an approximate, albeit imprecise, indicator of environmental exposure. Indeed, several investigators of the Bhopal disaster used a simplified dose-response approach in looking at late health events. They chose a community within 2 km of the plant and a farther one (approximately 8 km from the plant) and found higher rates of decreased pulmonary function,[6] increased acute ocular symptoms,[7] and increased pulmonary symptoms in children[8] for the individuals living closer to the plant. However, these studies were conducted 3 to 4 months after the disaster, and we have no longer-term follow-up data.

What might have been done epidemiologically, especially in ideal circumstances, is therefore a moot point for Bhopal. The immediate medical response to the disaster appeared to be swift, appropriate, and effective. The epidemiologic response could not be a priority at the time.

Thus the information we have today, 6 years after the event, is not profoundly different from that available in the first weeks after the gas leak; there were considerable acute pulmonary toxic effects with bronchospasm and pulmonary edema, severe irritation of exposed mucous membranes and the cornea, and little evidence of residual ocular effects. However, careful study would be needed to determine the long-term pulmonary damage and damage to other organs.

The article by Mehta et al confirms these early observations, suggests that long-term pulmonary damage has occurred, and raises the issue of teratogenic and fetal damage from MIC, although not proving it. Their statement "none of the data on morbidity and mortality are firm" is disappointing for their desire to present (and the readers to receive) a definitive scientific review. But more important, this lack of firm data extends the tragedy by denying some damaged individuals proper restitution, confounding follow-up and care for exposed individuals, and minimizing the new information available to better prevent and control future MIC exposures. Health authorities in industrial and chemical disasters must first focus on provision of care for the ill and injured but also must see as an urgent priority the establishment of a surveillance and epidemiologic study system that will address the aftermath of the acute exposure.

We readily perceive disaster planning and preparedness to encompass chemical plant operating conditions and safety systems (the cause of the disaster), and procedures to warn, evacuate, and protect nearby populations. The provision of emergency medical services is obvious. But the ability to conduct effective and valid postdisaster epidemiologic and health evaluations also depends on prior planning and available infrastructure. In Bhopal, support came promptly from a variety of local and national resources, including the Indian Council for Medical Research. The article by Mehta et al highlights the importance of the public health response, particularly epidemiology and health surveillance, in disaster planning.

Finally, it is impossible to close a discussion of the Bhopal disaster without reemphasizing the importance of prevention. While there are many lessons to be learned from a careful and thorough investigation of the health effects resulting from the Bhopal disaster, what a terrible price was paid for these lessons. Many difficult issues remain to be addressed: to assure that similar disasters do not occur. Among these are how to prevent such potentially dangerous plants from being located in heavily populated areas, how to ensure the safe operation and maintenance of technologically complex facilities, and how to develop effective disaster plans to better protect workers and nearby residents.*

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1. Mehta PS, Mehta AS, Mehta SJ, Makhijani AB. Bhopal tragedy's health effects: a review of methyl isocyanate toxicity. *JAMA*. 1990;264:2781-2787.
2. Parrish RG, Falk H, Melius JM. Industrial disasters: classification, investigation, and prevention. In: Harrington JM, ed. *Recent Advances in Occupational Health, III*. New York, NY: Churchill Livingstone Inc; 1985:155-168.
3. Joint Report of the Scientific Group on Methodologies for the Safety Evaluation of Chemicals. Health assessment and medical response. In: Bourdeau P, Green G, eds: *Methods for Assessing and Reducing Injury From Chemical Accidents*. New York, NY: John Wiley & Sons Inc; 1989:45-65.
4. Gregg MB, ed. *The Public Health Consequences of Disasters*. Atlanta Ga: Centers for Disease Control; 1989.
5. World Health Organization. *Emergency Preparedness and Response: Rapid Health Assessment in Chemical Emergencies*. Geneva, Switzerland: World Health Organization; January 1990. Publication ERO/EPR/90.1.8. Emergency Relief Operations.
6. Naik SR, Acharya VN, Bhalerao RA, et al. Medical survey of methyl isocyanate gas-affected population of Bhopal. II: pulmonary effects in those victims as seen 15 weeks after MIC exposure. *J Postgrad Med*. 1986;32:185-189.
7. Maskati QB. Ophthalmic survey of Bhopal victims 104 days after the tragedy. *J Postgrad Med*. 1986;32:199-202.
8. Irani SF, Mahashur AA. A survey of Bhopal children affected by methyl isocyanate gas. *J Postgrad Med*. 1986;32:195-198.
9. Bhopal Working Group. The public health implications of the Bhopal disaster. *Am J Public Health*. 1987;77:230-236.

84 SALMON (A·G)

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Correspondence

Bright red blood of Bhopal victims: cyanide or MIC?

Sir,—Several anecdotal observations have contributed to the general perception of the nature of the toxic effects suffered by the victims of the Bhopal disaster. There have now been various reports of scientific investigations of the toxic effects of methyl isocyanate, the substance believed to have been released in the accident, and also various analyses of what conditions might have led to the explosive release of the substance. It has generally been found that symptoms observed in exposed people may be explained by what is now known of the toxicity of methyl isocyanate.[1 2] On the other hand, some have claimed that other toxic compounds were involved, especially cyanides (organic or inorganic). So far as I am aware, no analytical data indicating the presence of such substances as a result of the accident have been presented. The observation of raised thiocyanate concentrations in the urine of both survivors and subsequent visitors to the region has been claimed as evidence of cyanide exposure. While this observation may well be accurate, it appears unlikely to be related to methyl isocyanate exposure on the basis of laboratory investigations of rats exposed to this substance.[1 2]

One particular observation that has been quite widely quoted was made during postmortem studies immediately after the disaster. It was said that the blood (and possibly myoglobin) of victims of acute poisoning was bright red such as is usually associated with oxygenated (arterial) blood. This might be characteristic of cyanide poisoning where tissue uptake of oxygen is inhibited. On the other hand, it seems unlikely that human victims of methyl isocyanate poisoning would have had oxygenated blood; Nemery *et al* specifically measured oxygen levels in the blood of exposed rats and found them to be low, due to lung damage and bronchoconstriction impairing the exchange of blood gases.[2] One might therefore conclude that some other substance was present either as a degradation product or impurity in the gas to which the Bhopal victims were exposed.

To evaluate this possibility we looked at the effect of methyl isocyanate on the ultraviolet/visible absorption spectrum of haemoglobin.[3] A sample of blood obtained by heart puncture from a rat was diluted into 10 mM sodium citrate buffer, pH 6·5: this was designed to produce a haemoglobin solution (by haemolysis of the erythrocytes) of suitable concentration for measurement of the absorption spectrum in a Perkin Elmer 124 UV/vis spectrometer. Formation of methaemoglobin was inhibited by adding a quantity of sodium dithionite. On addition of [illegible] amount of methyl isocyanate (20–50 μl to 2 ml tion) a vigorous reaction was observed in which evolved: this was the expected result of the [illegible] reaction, the first stage of which generates methylamine and carbon dioxide.[4] It was also observed although the main Soret band absorption haemoglobin (ca 430 nm) was unaffected, the peak in the visible region (ca 550 nm) disappeared pale red colour remained in the solution represented the long tail of the ultraviolet peak. The same was observed when a small amount of methyl was added to a solution of haemoglobin. Such tions also caused a shift in pH of the solution the alkaline range (methylamine being quite basic), but when sodium hydroxide was added to duce a similar shift no change in the spectrum seen. Such spectral changes in haem proteins means unexpected when molecules capable of as iron ligands are added: indeed, this is mechanism responsible for the colour diff between haemoglobin and oxyhaemoglobin. spectroscopic change induced by methylamine identical with that produced by oxygenation. does it correspond to methaemoglobin formation which would be prevented by dithionite: in fact, presence of this compound had no effect on modified spectrum.) It does seem possible, however that the methylamine induced spectral change be mistaken for this well known phenomenon by observer using the naked eye, which is a crude such tasks.

If this suggestion is accepted then the evidence involvement of cyanide or other substances MIC, slim as it is, is further reduced.

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References

[1] Salmon AG, Kerr-Muir M, Andersson N. Acute toxicity of methyl isocyanate: a preliminary study of the dose response for [illegible] other effects. *Br J Ind Med* 1985;42:795–8.

[2] Nemery B, Dinsdale D, Sparrow S, Ray DE. Effects of methyl isocyanate on the respiratory tract of rats. *Br J Ind Med* 1985;42:799–805.

[3] Haurowitz F, Hardin R. Respiratory proteins. In: *The proteins*. 2, part A. New York: Academic Press, 1954:296.

[4] Andersson N, Kerr Muir M, Salmon AG, *et al*. Bhopal disaster: follow-up and analytical chemistry. *Lancet* 1985;ii:761–2.

minimal course of treatment with antidepressant drugs, yet the outcome of our unrecognised patients was worse.

Failure to recognise depression seemed to be related to general practitioners' basic knowledge of diagnosing depression and not only to their skills in interviewing or their attitudes towards patients with emotional disorder.

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References

1 Clare AW. Psychiatry in general practice. *J R Coll Gen Pract* 1983;33:195-8.
2 Royal College of General Practitioners, Birmingham Research Unit. *Trends in national morbidity.* London: Royal College of General Practitioners, 1976. (Occasional Paper No 3.)
3 Fahy TJ. Pathways of specialist referral of depressed patients from general practice. *Br J Psychiatry* 1974;124:231-9.
4 Johnson DAW. Treatment of depression in general practice. *Br Med J* 1973;ii:18-20.
5 Johnson DAW. A study of the use of anti-depressant medication in general practice. *Br J Psychiatry* 1974;125:186-92.
6 Marks JN, Goldberg DP, Hillier VF. Determinants of the ability of general practitioners to detect psychiatric illness. *Psychol Med* 1979;9:337-53.
7 Shaw D, Williams P. Screening for psychiatric disorder in general practice. *Psychol Med* 1984;14:365-77.
8 Goldberg D, Huxley P. *Mental illness in the community: the pathway to psychiatric care.* London: Tavistock Publications, 1980:57-107.
9 Islam M. *The doctor, his patient, and the illness.* London: Pitman Medical, 1957.
10 Pendleton D, Schofield T, Tate P, Havelock P. *The consultation: an approach to learning and teaching.* Oxford: Oxford University Press, 1984.
11 Watson JM, Barber JH. Depressive illness in general practice. *Health Bull (Edinb)* 1981;39:312-6.
12 Spitzer RL, Endicott J, Robins E. Research diagnostic criteria: rationale and reliability. *Arch Gen Psychiatry* 1978;35:773-82.
13 Sireling LI, Freeling P, Paykel ES, Rao BM. Depression in general practice: clinical features and comparison with out-patients. *Br J Psychiatry* (in press).
14 Sireling LI, Paykel ES, Freeling P, Rao BM, Patel SP. Depression in general practice: case thresholds and diagnosis. *Br J Psychiatry* (in press).
15 Goldberg DP. *The detection of psychiatric illness by questionnaire.* London: Oxford University Press, 1972. (Maudsley monograph No 21.)
16 Endicott J, Spitzer RL. A diagnostic interview: the schedule for affective disorders and schizophrenia. *Arch Gen Psychiatry* 1978;35:837-44.
17 Wing JK, Cooper JE, Sartorius N. *The measurement and classification of psychiatric symptoms.* Cambridge: Cambridge University Press, 1974.
18 Feighner JP, Robins E, Guze SB, Woodruff RA, Winokur G, Munoz R. Diagnostic criteria for use in psychiatric research. *Arch Gen Psychiatry* 1972;26:57-63.
19 Hamilton M. Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology* 1967;6:278-96.
20 Paykel ES, Klerman GL, Prusoff B. Treatment setting and clinical depression. *Arch Gen Psychiatry* 1970;22:11-21.
21 Paykel ES. The clinical interview for depression. In: Sartorius N, Ban TA, eds. *Assessment of depression* (in press).
22 Carney MWP, Roth M, Garside RF. The diagnosis of depressive syndromes and the prediction of ECT responses. *Br J Psychiatry* 1965;111:659-74.
23 Raskin A, Schulterbrandt JG, Reatig N, McKeon JJ. Differential response to chlorpromazine, imipramine, and placebo. A study of sub-groups of hospitalised depressed patients. *Arch Gen Psychiatry* 1970;23:164-73.
24 Nie NM, Hull CH, Jenkins JG, Steinbrenner K, Bart DH. *Statistical package for the social sciences.* 2nd ed. New York: McGraw Hill, 1975.
25 Schwede L, Spitzoll E. Plots of p-values to evaluate test simultaneously. *Biometrika* 1982;69:493-502.
26 Goldberg D, Huxley P. *Mental illness in the community: the pathway to psychiatric care.* London: Tavistock Publications, 1980:64-5.
27 Burton RH, Freeling P. How general practitioners manage depressive illness: developing a method of audit. *J R Coll Gen Pract* 1982;32:558-61.

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My Student Elective

An eyewitness in Bhopal

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New to Bhopal from Delhi on Sunday 2 December 1984 to begin my elective, which I had arranged to spend in the paediatric department at Hamidia Hospital. In the next two weeks 1337 children were admitted to the department. Of these 119 died and Bhopal became a household name. As the only outsider I was in a unique position to observe the reaction of the city and its medical resources to the world's worst industrial disaster. This report haunts my impressions of how the hospital and staff coped with the disaster and describes some of the clinical features of the toxic gas poisoning.

I spent the first night of my stay with Dr Bhandari, the professor of paediatrics. At about 1 30 am I was woken by the repeated ringing of the doorbell followed by the entry of several people who were coughing violently. At the same time I noticed a sweetish smell, my eyes were mysteriously stinging and watering, and my throat felt dry. I heard the distant sound of a siren, but this being my first night in Bhopal I thought nothing of the incident and went back to sleep. When I woke the next morning I heard that there had been a major leak from the Union Carbide insecticide plant about a mile from the hospital, but the nature of the gas was not yet known. At first the local news reports knew little more than we could deduce from seeing the numbers of affected people who had flooded into the hospital grounds. The earliest reports suggested that about 30 people had died. With each subsequent news report we listened with disbelief as further details about the horrifying story began to emerge. Even after the first full day we were unable to believe the estimate from the BBC World Service of 2000 dead—later even this proved to be conservative.

The dead and dying arrived by the truckload, others came by rickshaw or were carried by relatives. For some the effort of the journey itself proved too much, and they died soon after arrival. Many families were split up during the initial panic, everywhere there were people looking for missing relatives. There were long queues of people trying to identify relatives in the mortuary. From an early stage when the mortuary was full, other unidentified bodies were laid out on a nearby lawn and under hastily erected shelters.

Facilities overwhelmed

The facilities in the hospital and the manpower became increasingly overstretched as the enormity of the disaster became apparent. The doctors were quite overwhelmed. I felt even more helpless; having arrived only the day before I had been unable to see inside the hospital or be introduced to the staff, and was unable to speak the language. For the first few days I was frustrated that I was not able to do more to help. Without a doctor to interpret for me there

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was little that I could do on my own. I joined up with a group of other medical students to help administer eye drops, give injections, set up drips, and distribute food. Doctors worked around the clock giving repeated injections; there was no time to resterilise needles between patients. The doctors were unable to spend much time to help any one child, dividing their time between the ill and the dying. Later on I was able to examine the children as they arrived and suggest treatment. I also helped to ventilate dying children and attempt cardiac massage.

Many hundreds of victims died within the first few hours of the gas leak, death being caused by respiratory failure resulting from the severe pulmonary oedema. The main complaints of those who survived were of a sensation of intense heat, burning eyes, blindness, vomiting, choking, coughing, breathlessness, and a feeling of suffocation. At first the precise toxic effects of the gas were not known so treatment was symptomatic—with antibiotic eye-drops, oxygen inhalation, and intravenous frusemide, steroids, and aminophylline to relieve respiratory distress.

The most severely affected children were attended to in the intensive treatment room, which was about 20 feet square and supplied with only one sink and an electric socket. Rows of infants and older children on drips filled the couches, while others lay on the floor or were held by their parents while they received treatment. Two or three children would be receiving oxygen through nasal cannulae delivered from the same gas cylinder. Most of these children admitted to the intensive treatment room died of respiratory failure within the next few days.

The wide corridors of the hospital were packed with victims, several members of each family requiring urgent treatment. The luckier ones had a bed—for the whole family. By the Wednesday these families who had been camping in the ward corridors were being moved out to tents erected nearby by the army. Relief agencies provided food for the victims—milk, bananas, and biscuits. By the Thursday the initial rush of people coming in had slowed though the death toll was still rising: the official figure now exceeded 2000.

Of the 119 deaths in the paediatric department from 3-14 December, 106 occurred in the first four days after exposure. The cause of death in most cases was cardiac arrest associated with respiratory failure. On examination these children with toxic lung damage were breathless and hyperventilating but not cyanosed. Many had audible wheezes and grunts, and widespread rhonchi and crepitations were heard with the stethoscope. The children were treated with intravenous frusemide and steroids to help reduce the severe pulmonary oedema, and aminophylline to relieve the wheezing. After about a week about 15 children a day were still being admitted to the wards, mainly with respiratory complaints—cough, breathlessness, and respiratory infections. The eye problems, which had caused widespread distress in the first few days, had been due to the direct chemical irritation, causing painful watering eyes, local oedema, and photophobia. The irritant gas had caused superficial ulceration, which led to temporary corneal opacity in some cases. Prophylactic neomycin eye drops helped to keep the rate of infection low, and atropine drops were also given to prevent the formation of posterior synechiae, though it caused further problems of blurred vision and occasionally induced acute glaucoma. Fortunately, the corneal ulceration was superficial and generally healed without scarring. The mobile preventative ophthalmology units played an important role in the follow up of the eye complications in the community. Some children also showed signs of convulsions, hemiparesis, and coma.

On 8 December a German toxicologist, Dr Daunderer, visited Bhopal and reinforced the views held by the head of the forensic medicine department, Dr Heereshchandra, that the effects of the methyl isocyanate gas were mediated via its breakdown into cyanide and other toxic amines. If this were the case there was a specific antidote, sodium thiosulphate. The drug was thought to be harmless, but its efficacy was not proved so its widespread use in the treatment of the victims was vetoed. Sodium thiosulphate was administered to a few patients, some of whom showed marked improvement, but by the time a controlled trial had been started it was too late to expect any effect from an immediate antidote.

Tragedy attracted world attention

For the next few weeks the world's attention focused on Bhopal. Rajiv Gandhi and Mother Teresa came to visit the city, meeting the gas victims in hospital and also in the affected slums around the plant. Newspaper and television reporters arrived from all over the world within a day or two of the tragedy. Everyone was anxious to send back pictures of the carnage in the slum areas, record the death throes of babies in the paediatric wards, and recount the tragic stories of so many left widowed or orphaned by the gas. Some television teams would film patients in the wards, regardless of whether they had been affected by the gas or not.

The next few weeks were spent trying to cope with the immediate toxic effects of the methyl isocyanate gas and later to predict the medium and long term complications likely to arise. The most likely problems expected are thought to be respiratory, especially pulmonary fibrosis, though doctors are also anxious to follow up any neurological, ophthalmologieal, and intellectual sequelae that may emerge. Another fear is the effect of the gas on early pregnancy. Methyl isocyanate exposure was blamed for a marked increase in the rate of premature labour and abortions on the night of the gas leak and in the subsequent few weeks.

I had expected the experiences of my elective period to include a range of the usual childhood diseases—polio, tuberculosis, and diphtheria. Instead it was as if I had arrived during the filming of a disaster film—except it was real.

I am grateful to the following for allowing me access to their departments: Dr N R Bhandari, professor of paediatrics; Dr L P Mishra, professor of medicine; and Dr Mittal, department of ophthalmology.

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What is the current state of knowledge of ecological illness or total allergy syndrome? Is there an immune basis to this condition?

Chemicals as well as pollens can cause sensitivity. This has been discussed by the World Health Organisation and its conclusion was that this is a considerable health problem and that we know little about it.[1] Many patients feel "dis-eased," complaining of lethargy, myalgia, aching joints, abdominal symptoms, depression, fluctuating weight, etc. They are often referred to a psychiatrist because no tests show abnormal results. These are obviously a heterogeneous group which we now know contains patients with food allergy, the postviral syndrome, those with chemical sensitivity, and some with primary or secondary psychological problems, or indeed a mixture of all these. The pioneering work of Randolph has delineated a group of these patients who are sensitive to chemicals and gas fumes and when these are removed from their environment improvement can be remarkable. The term "total allergy syndrome" is valueless. These are patients with multiple sensitivities, so called "universal reactors," as described above, who are difficult to treat, and in whom immunological abnormalities have been found.[2] Their symptoms are not all in the mind, and in environmental units as seen in the United States many of these patients return to normal health. We know little of what causes this syndrome but environmental factors are clearly important. Much work needs to be done before we have a clear understanding as to what is happening in possibly a considerable proportion of the population.—J BROSTOFF, reader in clinical immunology, London.

1 World Health Organisation. Workshop on allergy, responses and hypersensitivities induced by chemicals. *Immunology and Allergy Practice* 1983;5:62.
2 Mike N, Asquith P. *Total allergy syndrome: what evidence can be established.* 2nd Food Allergy Workshop. Oxford: Medicine Publishing Foundation, 1983.

to know what proportion of doctors do believe in an honest approach to their patients, and what proportion of those use the proper terms rather than euphemisms such as growth or tumour, which many patients do not realise are synonyms when used for cancer.

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MISCARRIAGE OR ABORTION

SIR,—We support Professor Beard and his colleagues' suggestion (Nov 16, p 1122) that doctors—like their patients—should use the word "miscarriage" for a spontaneous abortion, thus reducing the distress caused to couples who have a miscarriage. Statistical confusion in Parliament and public debate would also be reduced. In February this year Mr Nicholas Winterton, MP, an opponent of abortion, asked how many deaths were due to abortion. The Minister of Health gave the unintentionally misleading reply that in 1983 in England and Wales there were eight abortion deaths but did not go on to say that all eight were due to miscarriage and none to induced abortion.

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CAROLINE WOODROFFE,
General Secretary

DOGS AND PAGET'S DISEASE

SIR,—Dr O'Driscoll and Dr Anderson report that ownership of dogs in the past was more common among 50 patients with Paget's disease than among 50 age and sex matched diabetic controls (Oct 26, p 919). This case-control comparison was done in Manchester. They suggest that "a canine virus (possibly canine distemper) might be the primary infective agent" in the disease.

We have done two case-control studies—one in Lancaster, UK, the town with the highest recorded prevalence of Paget's disease in any country, and the other in Siena, Italy, which has a low prevalence.[1] Questions about household pets during childhood were included in the interviews. In Lancaster 37 cases were compared with 74 controls matched for sex and age to within five years. Cases and controls were identified from a sequential sample of stored radiographs in a general hospital. In Siena 27 cases from a hospital register were matched with two sets of controls, one comprising patients attending a rheumatology outpatient clinic and the other inpatients on medical wards. Other than for the inpatient controls in Siena all the interviews were at the subjects' homes.

The table shows the numbers of cases and controls who reported having had a dog as a household pet during childhood. Only case-control sets in which the case and at least one control was traced and could reply to the question are included. Matched analysis by the Mantel and Haenszel method was used. 55% of subjects (cases and controls) in Lancaster and 45% in Siena had had a dog as a household pet during childhood. The differences between the cases and controls were small and not significant. Nor were there differences in exposure to other household pets, including cats and birds. These findings do not support those from Manchester.

Laboratory evidence in support of the canine distemper hypothesis has been sought in the past but not found.[2] The prevalence of Paget's disease is higher in Britain than in any other Western European country.[1] Only in France do the rates approach those in Britain. However, the dog population in Britain is estimated as 10·4 per 100 people, while values for eleven other European countries range from 17·2 in France to 5·3 in Switzerland.[3] The value of 9·6 per 100 in Sweden is of particular interest in view of the extreme rarity of Paget's disease there.

NUMBERS OF CASES OF PAGET'S DISEASE AND CONTROLS IN LANCASTER AND SIENA WHO HAD DOGS AS PETS DURING CHILDHOOD

Town	Cases		Controls		Relative risk (and 95% confidence limits)
	Dogs	No dogs	Dogs	No dogs	
Lancaster	19	17	40	32	0·8 (0·4–1·8)
Siena	13	13	19	28	1·4 (0·6–3·6)

We suggest that no firm conclusions be drawn from the Manchester study until there is additional evidence, including that from a large, adequately controlled case-control study in another area.

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2. MRC Environmental Epidemiology Unit. Conference report no 6. The aetiology of Paget's disease of bone. Southampton, 1984.
3. Anderson RS. Trends in pet populations. In: Thrushfield MV, ed. Proceedings of Society for Veterinary Epidemiology and Preventive Medicine, 1983.

SIR,—Although Dr O'Driscoll and Dr Anderson's results show that dog ownership was commoner in patients with Paget's disease than in diabetic patients, it is premature to suggest from these findings that the primary infective agent might be a canine virus. O'Driscoll and Anderson note that the survey provides only circumstantial evidence to implicate dogs in the cause of Paget's disease and stress the need for further studies. The lay press, however, tends to be less discerning; for example, *The Times* of Nov 8, 1985, under the heading of "Distemper jabs to help the family", reported that dogs were probably carriers of Paget's disease. Such reporting only serves to provide ammunition (irrespective of whether it turns out to be live or blank) for anti-dog groups and tends to alienate members of the veterinary profession who are faced with distraught dog owners.

It would be of interest to survey the incidence of Paget's disease in the older veterinarians. Such veterinary surgeons will have been exposed to sick dogs far more than any other group and at times when immunisation of dogs against distemper was either not available or less widely used. It would be expected, therefore, that veterinary surgeons should be over-represented amongst patients with Paget's disease. I know of no colleague with Paget's disease.

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METHYL ISOCYANATE: THIOSULPHATE DOES NOT PROTECT

SIR,—Various sources[1-3] suggest that, after last December's methyl isocyanate (MIC) disaster at Bhopal, in India,[4] there is controversy about cyanide poisoning in some of the victims. It has been claimed that sodium thiosulphate ($Na_2S_2O_3$) was therapeutically effective. However, no hydrogen cyanide seems to have been released with MIC and it is unlikely that MIC is metabolised to or leads to the release of cyanide in the body. On the other hand, $Na_2S_2O_3$ could have therapeutic effects apart from its action as a cyanide antagonist. No published work throws light on this important issue.

While studying rats exposed by inhalation to MIC[5] we evaluated the effects of $Na_2S_2O_3$ on the clinical evolution and daily urinary thiocyanate excretion during one week after exposure to MIC. Eighteen male LAC:P rats of 130–150 g initial body weight were used. Twelve rats (two groups of six) were statically exposed for 1 h in a 50 litre glass tank to MIC at an initial concentration of 0·25 mg/l (about 100 ppm). This corresponds to a time-weighted average level of between 20 and 30 ppm, causing extensive damage (mainly to the airway epithelium) and leading to severe respiratory distress, but no acute deaths.[5] Four of these rats received an intraperitoneal injection of 0·25 ml $Na_2S_2O_3$ in saline (673 μmol per rat) 15 min before exposure to MIC and then daily until the sixth day after exposure; the other eight rats similarly received injections of 0·25 ml saline. Six rats were left unexposed to MIC, three receiving $Na_2S_2O_3$ and three saline injections. After the exposure, the rats were housed individually in metabolism cages for 24 h collection of urine (on 50 μl gentamicin 10 mg/ml) and they were weighed daily. Survivors were killed 7 days after exposure, blood was taken, and

EFFECTS OF SODIUM THIOSULPHATE ON BODY WEIGHT (AS % OF INITIAL BODY WEIGHT) AND URINARY THIOCYANATE LEVELS (µg/l) IN 24 h URINE) AFTER EXPOSURE OF RATS TO METHYL ISOCYANATE

Group	Measurement	Day 1	Day 4	Day 7
Not exposed to MIC				
Saline (n = 3)	Weight	105±3	114±7	122±12
	Urine SCN^-	58±11	64±19	63±19
$Na_2S_2O_3$ (n = 3)	Weight	101±6	99±27	115±13
	Urine SCN^-	40±12	33±20	40±16
Exposed to MIC				
Saline (n = 8)	Weight	87±3 (n = 5)	90±4 (n = 4)	94±8 (n = 4)
	Urine SCN^-	23±4	56±11	61±10
$Na_2S_2O_3$ (n = 4)	Weight	89±4 (n = 3)	90,61 (n = 2)	99 (n = 1)
	Urine SCN^-	20±3	36,12	35

Data (not shown for days 2, 3, 5, and 6) as mean±SD or individual values.

the lungs instilled via the trachea with Bouin's fixative. Urine and plasma thiocyanate levels were measured in Louvain, Belgium (Prof R. Lauwerys) by a modified *p*-phenylenediamine method.

No clinical differences between $Na_2S_2O_3$ treated and saline-treated rats were observed. Contrary to expectation, some rats died in the first 48 h after exposure to MIC (four deaths of the eight saline treated animals, and two of the four $Na_2S_2O_3$ treated animals). A third $Na_2S_2O_3$ treated rat was moribund on the fifth day and was killed. The deaths could have resulted from an exposure level higher than intended (the dose-response curve being rather steep) or from the isolation of the animals after exposure. Body weights, a good index of an animal's wellbeing, were no better in the $Na_2S_2O_3$ animals than in the controls (table). One control $Na_2S_2O_3$-treated rat lost weight during the first 4 days for no obvious reason. Total urinary thiocyanate excretion was lower in the rats receiving $Na_2S_2O_3$ than in those receiving saline, but it was also lower in MIC exposed rats than in non-exposed rats (table). The progress of urinary thiocyanate excretion was directly related to that of body weight. Plasma thiocyanate concentrations on day 7 did not differ between the four groups. The histological appearance of the lungs and airways 7 days after MIC did not appear to be influenced by the $Na_2S_2O_3$ treatment. Bronchioles from both treated and untreated animals showed a hyperplastic repair epithelium with fibrosis and peribronchiolar inflammatory infiltrate.

This study suggests that $Na_2S_2O_3$ does not protect rats from the acute and subacute effects of MIC and that cyanide intoxication is not involved in the clinical syndrome seen after exposure to pure MIC.

We thank Mr C. J. Vesey (St Bartholomew's Hospital, London) and Dr W. N. Aldridge for advice and Mr J. Casters for thiocyanate measurements.

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1. Hamlyn M. Bhopal: Was cyanide the culprit? *Times* 1985; Jan 17.
2. Feru I. The day after (*Illus Weekly India* Dec 30, 1984). In: Bhopal: Industrial genocide? Hong Kong: Arena Press, 1985: 46–55.
3. Anon. New Bhopal dispute: Cyanide poisoning of victims claimed. *Chem Eng News* 1985; 63: 6.
4. Editorial. Calamity of Bhopal. *Lancet* 1984; ii: 1378–79.
5. Nemery B, Dinsdale D, Sparrow S, Ray DE. Effects of methyl isocyanate on the respiratory tract of rats. *Br J Ind Med* (in press).

SODIUM DIETHYLDITHIOCARBAMATE INDUCING LONG-LASTING REMISSION IN CASE OF JUVENILE SYSTEMIC LUPUS ERYTHEMATOSUS

SIR,—Remissions of systemic lupus erythematosus (SLE) are sometimes obtained with steroid therapy, but with a high risk of steroid dependency and serious side-effects, such as severe infections or growth failure. We have been evaluating a T-cell restorative drug,[1,2] sodium diethyldithiocarbamate (Imuthiol; Institut Merieux, Lyon), in a 14-year-old girl whose first symptoms were limited to non-erosive arthritis of the knee joints. A secondary discoid rash with erythematous patches and adherent keratotic scaling and follicular plugging developed. Fixed erythema over the malar eminences tended to spare the labial folds.

When first seen (April, 1984) this girl was neutropenic and thrombocytopenic (less than 80 000/µl). Laboratory tests confirmed SLE: homogeneous antinuclear antibodies at the 1/1000 serum dilution and positive anti-Sm antibody by immunofluorescence assays, in the absence of treatment with drugs associated with drug-induced lupus syndrome. Total haemolytic complement was very low (CH_{10} <7·7 units), as were C_1 (56·6 mg/dl, normal 70–145) and C_4 (4·92 mg/dl, normal 13–40). Severe leucocyturia was found, not associated with proteinuria. Her HLA phenotype was A3/Bw35, Aw32/B⁻. B cells were normal in number, with a mild decrease in T-cells. Labial biopsy was characteristic of SLE. There was no family history, except for a grandmother with an ill-defined connective tissue disease.

Treatment with prednisone 2 mg/kg daily was started in April, 1984. A serious cushingoid syndrome promptly developed, and in September, 1984, we decided to try imuthiol (5 mg/kg, orally, once a week) to see if we could lower the dosage of prednisone. Within 8 weeks we could reduce the steroid dose and after the patient had been on imuthiol for 3 months a complete remission was achieved and prednisone was stopped in April, 1985. Today, a year after the start of treatment with imuthiol and more than 6 months after prednisone had been stopped, the girl is well and laboratory tests are normal (table). No side-effects have been noted in a year of continuous administration of imuthiol.

LABORATORY FINDINGS (APRIL, 1984 TO SEPTEMBER, 1985)

Test*	April, 1984 (P)	Sept (P)	Now (I + P)	March, 1985 (I + P)	Sept (I only)
Platelets	90 000	140 000	230 000	220 000	
Hb	10,7	12,5	14	14,1	
CH_{50}	<7·7	65	61	55	
C_1	39	36	40	56	
C_4	20	23	21	23	
ANF	>1/1000†	1/1000†	1/10	1/100	1/100
ESR	146/150	112/129	88/135	20/47	13/35
Anti-DNA	+	+	+	–	
Anti-Sm	+	–	–	–	
Leucocyturia	>50 000		44 000	4000	

P = prednisone; I + P = imuthiol + reduced doses of prednisone.
*Platelets (/µl); Hb (g/dl); C_1, (mg/dl) (normal 80–140); C_4 (mg/dl) (20–50); leucocyturia (cells/min).
†Homogeneous.

In children with SLE, especially when there is clinical evidence of systemic illness (such as renal, neurological, or haematological involvement) prednisone 2 mg/kg daily is usually recommended. Severe side-effects, that can be lethal, have prompted a search for other treatments. Immunosuppressive agents have not provided conclusive results in SLE.[3] The use of a thymotropic immunotherapeutic agent such as imuthiol might be a new approach to the treatment of connective tissue disease, either in association with reduced dosages of steroids or even as single therapy.

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1. Renoux G. Immunopharmacologie et pharmacologie du diethyldithiocarbamate (DTC). *J Pharmacol (Paris)* 1982; 13: 95–134.
2. Renoux G. The mode of action of imuthiol (sodium diethyldithiocarbamate) and of its metabolites. In: Fenichel RL, Chirigos MA, eds. Immune modulation agents and their mechanisms. New York: Marcel Dekker, 1984: 607–24.
3. Jacobs CJ. Systemic lupus erythematosus. In: Jacobs CJ, ed. Pediatric rheumatology for the practitioner. Berlin: Springer-Verlag, 1982: 313–77.

NO 8520

Rapid hydrolysis of urea has been reported to be a useful test for *C pylori dis*, so urea broth was inoculated with tissue homogenate. Where the bacteria are present in large numbers in the biopsy specimen the urea test is convenient, since it can be read in two hours and is not time-consuming. However, in the 10 culture-positive cases the urea test was positive within 2 hours in only 4 and within 8, 24, and more than 24 hours in a further 1, 2, and 1, respectively. Furthermore, urea testing to 24 hours or more resulted in 3 false-positive reactions (out of 7 culture negatives).

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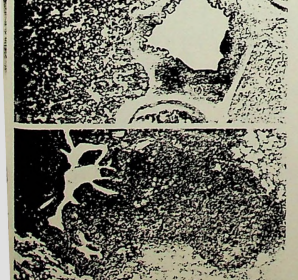
LONG TERM PATHOLOGY OF LUNG, EYE, AND OTHER ORGANS FOLLOWING ACUTE EXPOSURE OF RATS TO METHYL ISOCYANATE

SIR,—A year after the leakage of gas, mainly methyl isocyanate (MIC), at Bhopal, India, in 1984, as many as 320 000 people were still affected,[1] and an increased mortality among those exposed persisted. Long-term effects of MIC exposure include irritation and watering of the eyes[2] and respiratory disease. Severe acute changes in the eyes[3,4] and respiratory tissues[1]—especially conjunctival/corneal alterations and restrictive pulmonary defects—have suggested the possibility of longer term sequelae.

In a pilot study[5] rats exposed to 31, 21, 11, or 0 parts per million (ppm) of MIC vapour for 2 h have now been observed over 14 months. Two MIC exposed animals (one 31 ppm and one 11 ppm) died 6 and 8 months, respectively, after MIC exposure following sudden onset of respiratory distress. Macroscopic and microscopic examination of the six animals killed at 14 months revealed a history of mild respiratory infection in all animals. MIC-exposed animals had at least four times the amount of lymphoid aggregates found in control animals: lymphoid tissue averaged 0·219 mm² per mm² total lung tissue in the animal exposed to 31 ppm, 0·025 mm² in the two 21 ppm rats, 0·039 mm² in the 11 ppm animal, and 0·005 mm² in the two controls. This lymphoid hyperplasia (see figure) was adjacent to bronchiolar airways and consisted largely of lymphocytes, plasma cells, and macrophages. A mild infiltrate of eosinophils was present in the bronchial mucosa of both 21 ppm animals and the 31 ppm animal. There were no eosinophils in the lung tissues of either control. Mild interstitial fibrosis in the peribronchiolar regions was seen in all treated animals.



Rat lungs.

Upper: control.
Lower: exposure to MIC 31 ppm for 2 h, showing lymphoid hyperplasia. Haematoxylin and eosin; × 20.

The major finding in the eyes was an eosinophil and lymphoid infiltrate in the mucosa of the conjunctivae of the eyelids and perilimbal regions. This was most prominent in the 21 ppm animals.

Liver, kidney, and spleen were normal.

These findings suggest that long-term changes in the eyes and lungs may result from a single 2 h exposure to acute sublethal doses of MIC vapours, and indicate that the immune system is most probably directly involved. Immunological effects in man have been observed,[3] which might be causally related to pathological changes or increased susceptibility to infections. Enlarged tonsils and lymphoid hyperplasia were seen in patients 4–6 weeks after the Bhopal incident, and antibodies to proteins modified by reaction with MIC was noted. Kamat et al[1] also suggested the possibility of interstitial fibrosis in many of the 82 people examined. Lung biopsy in 5 cases showed fibrosis (no controls). Hyperreactivity pneumonitis after exposure to various isocyanates has been reported,[7] and low concentrations of isocyanates stimulate lymphocyte proliferation.[8] Presence of eosinophilia has been demonstrated[9] in animals exposed to aliphatic isocyanates. MIC, when linked to albumin, can elicit an antibody response in guineapigs.[10] The long-term finding of lymphoid hyperplasia seen in our animal study may be due to either to persisting exposure-related antigens or to an increased susceptibility to other immunostimulating agents following MIC exposure.

We conclude that the possibilities of persistent antigen/antibody activity and hypersensitisation of the airways of individuals exposed to MIC are worthy of further investigation, and may help uncover the causes of continuing respiratory and eye problems and reports of delayed sudden deaths experienced by Bhopal gas victims.

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1. Lepkowski W. Bhopal: Indian city begins to heal but conflicts remain. *Chem Eng News* 1985; 63 (48): 18–32.
2. Andersson N, Kerr Muir M, Anwari MK, Mahashabde S, Salmon AG, Vaidyanathan K. Persistent eye watering among Bhopal survivors. *Lancet* 1986; ii: 1152.
3. Andersson N, Kerr Muir M, Mehra V. Bhopal eye. *Lancet* 1984; ii: 1481.
4. Andersson N, Kerr Muir M, Salmon AG, et al. Bhopal disaster: Eye follow-up and analytical chemistry. *Lancet* 1985; i: 761–62.
5. Kamat SR, Mahashur AA, Tiwari AKB, et al. Early observations on pulmonary changes and clinical morbidity due to the isocyanate gas leak at Bhopal. *J Postgrad Med* 1985; 31: 63–72.
6. Salmon AG, Kerr Muir M, Andersson N. Acute toxicity of methyl isocyanate: a preliminary study of the dose response for eye and other effects. *Br J Ind Med* 1985; 42: 795–98.
7. Charles J, Bernstein A, Jones B, et al. Hypersensitivity pneumonitis after exposure to isocyanates. *Thorax* 1976; 31: 127–36.
8. Thierman GB, Simms BG, Goldstein AL, Kilian DJ. The effect of organic compounds used in the manufacture of plastics on the responsivity of murine and human lymphocytes. *Toxicol Appl Pharmacol* 1976; 44: 673–81.
9. White WG, Morris MJ, Stagden E, Zapata E. Isocyanate induced asthma in a car factory. *Lancet* 1980; i: 756–60.
10. Karol MH, Rubinoff B, Gangal S, et al. Experimental and clinical immunological response to methyl isocyanate. *Toxicologist* 1986; 6: 78 (abstr 310).

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Inhalation Toxicity Studies of Methyl Isocyanate (MIC) in Rats: Part I—Pulmonary Pathology and Genotoxicity Evaluation

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A single exposure of MIC at 3.20 mg/l chamber concentration for 8 min, through whole body inhalation chamber to male albino rats, caused severe irritation of eyes, lacrimation, corneal opacity and conjuctival hyperaemia, irritation of respiratory tract and gastrointestinal tract, perinasal, perioral wetting, frothiness, gasping and dyspnea. Necropsy revealed consolidation of two small lobes of lungs and ballooning of the gastro-intestinal tract. Microscopically, peripheral emphysema was well marked after 48 hr of exposure, and persisted up to 14 days though exhibiting a trend of reduced severity after 3 days. Congestion of the blood vessels, edaematous changes, cellular reaction and hyperplasia of the lymphoid follicles became marked and persisted up to 14 days. The postexposure (7-14 days) thickening of the alveolar septa and around the peribronchials was progressively noticeable and quite pronounced on 14th day. Food and water consumption and body weights of the animals initially reduced followed by a gradual recovery. No significant micronucleus formation was observed in rat bone marrow preparations 7 and 14 days after exposure. Exposure to higher concentration of MIC, using 10.97, 21.95 and 43.90 mg/l resulted in extensive reaction, pronounced clinical symptoms and death of all exposed rats within 24 hr. It appears from these observations that histopathological injury to lungs and extensive mortality in rats is a consequence of necrotizing/corrosive action of the direct inhalation exposure to MIC.

Methyl isocyanate (MIC, mol. wt. 52.05, sp. gravity 0.9230) is a colourless, highly volatile, inflammable and strong odorous liquid used in the manufacture of carbamate insecticides, such as carbaryl, carbofuran, baygon, kaldicarb, and warfare agents. It reacts violently in presence of moisture, acids, alkalies, etc. It is a hazardous chemical by all routes of contact, irritating skin, eyes and mucous membranes. The possibilities of inhalation and subsequent hazards are greatly enhanced because of its high vapour pressure[1]. The leakage of toxic gas, including MIC, from an industrial reservoir at Bhopal on 2/3 December, 1984, led to widespread morbidity and mortality in humans and several species of animals in and around the area[2]. An accident also occurred in U.K., but of less severity affecting only 35 firemen[3]. The horrifying experience of the illnesses and deaths in humans exposed at Bhopal and U.K., prompted extensive toxicological studies in animals world over. The reports of experimental toxicological studies on MIC, using laboratory animals, have revealed the acute and sub-acute pathological injury in the form of extensive necrosis, erosion of respiratory and olfactory epithelia, haemorrhage, edema, presence of cellular debris, inflammatory cells and fibrin in the airways, chronic alveolitis and atelactasis[4-10]. We initiated toxicological studies on rats exposed to MIC right after the Bhopal episode with a view to comparing the results with observations made by us on toxic gas affected patients in Bhopal (see our data on human patients). Detailed studies were undertaken in order to understand[1] the acute and sub-acute toxicity potential of MIC[2], the spectrum of clinical and pathological manifestations[3], metabolic biochemical mechanisms of biological reactivity of MIC and subsequent injury/lethality. Inhalation route was considered to be the preferred mode of exposure akin to the environmental situations and rats were selected as the experimental animals for these studies.

This report presents our observations on toxicological manifestations, pathological abnormality, genetic toxicity, and relative mortality in male albino rats exposed to MIC.

Materials and Methods

A sample of MIC, with a 99.9% purity (confirmed by Mass spectroscopy) was procured from Regional Research Laboratory (CSIR), Hyderabad. All other chemicals and reagents used in this study were of analytical grade obtained from BDH or E. Merck, India.

Experimental protocols—The all glass whole body inhalation chamber of 21 litres capacity having an internal diam. of 30 cm was used in the present study. The chamber had two parts, (a) upper and (b) lower. The upper part was detachable with 3.130 litres capacity, having arrangements for placing thermometer for recording the chamber temperature, and to fit nebulizer, etc. The lower portion was of

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17.870 litres capacity, with 4 ports (two on each side). One port was connected with a compressed air tube and through this tube the required amount of MIC was injected into the chamber with a Hamilton micro syringe; the second was for collecting air samples of the chamber; the third was of vacuum line and the fourth was provided for attachment to the other monitoring devices or for being connected with other gases in case the same chamber is to be used with more than one gas. The middle of the lower chamber has a perforated teflon plate where animals can be placed. The bottom of the lower chamber is designed for independent collection of stool and urine (Fig. 1).

This type of chamber is appropriate for short-term toxicity studies in static condition with micro amount of MIC in test chemicals[10]. The entire system is being kept inside a fuming hood fitted with a positive pressure exhaust system so that the work environment is safe. The whole set up was checked for being leak proof before each exposure.

The animals were acclimatized in laboratory condition for 7 days prior to MIC exposure. Each time 6 albino rats, adult, male (6-7 weeks) of 150-180 g body weights from ITRC (Ghcru) animal colony were exposed to various concentrations of MIC and analysed by aspiring appropriate samples at various intervals from a side port and estimating through a GLC, model No. Shimatzu GC-7AG. An average chamber concentration of MIC in this set was 3.30 mg/l maintained for a period of 8 min. This was used for all further toxicology studies reported in Parts II-V. Since it gave only a partial mortality (10-20%) and exposed animals showed distinct signs and symptoms of toxicity over a period of time.

Similar arrangements were made to expose animals with compressed air only and these animals served as control. Clinical symptoms, food and water consumption, body weight and mortality rate were observed during and after the exposure for 14 days.

Histopathology—Animals were sacrificed 48 hr, 72 hr, 7 days and 14 days after exposure. Lung was fixed by inflating with the fixative 10% buffered formaline, dehydrated with ethanol, embedded in paraffin and 4-5 μm thick cut sections were stained with haematoxylin-eosin[12] for routine histopathology and Gomori's silver nitrate staining was done for reticulin[13] examination.

Genetic toxicology—Genotoxicity potential of MIC was evaluated in bone marrow preparations from both femurs of exposed rats according to the procedure of Schmid[14].

Results and Discussion

Gross observations—Gross observations during and immediately following MIC exposure were hyperexcitability, lacrimation, perinasal and perioral wetness, respiratory difficulty, gasping, decreased activity and ataxia. 2-3 animals in each batch had nasal, oral and eye bleeding during the exposure. Except in 2 rats which became completely blind the postexposure manifestation included continued frothiness and severe dyspnea even up to 12 hr, conjuctival hyperemia in nearly 50% of animals on 3rd day, development of corneal opacity in both eyes on

Fig. 1—The whole body inhalation chamber placed inside the exhaust system

day, followed by clearing of eyes on 10th day. Food and water consumption of the exposed animals was negligible up to 48 hr and improved in subsequent days. Body weight of the animals initially reduced, and was followed by a gradual improvement with improved water and food intake. In most cases, the severity of above manifestations was directly related to the amount of exposure to MIC. Similar observations have been reported by other workers using MIC[4-10], phosgene and nitrogen dioxide[15] and TDI[16,17].

Lethality—In our experimental set up, exposure to MIC resulted in 100% mortality of rats at a dose of 43.9 mg/l within 30 min, at 21.95 mg/l within 12 hr, and 10.97 mg/l within 24 hr. Exposure to 4.39 mg/l of MIC resulted in 30-40% mortality within 72 hr. It is highly probable that the observed lethality was a consequence of pulmonary insufficiency caused by edema, haemorrhage and narrowing of airways[4,7,9].

Autopsy—Dead animals or those which were in moribund condition, when autopsied, had typical ballooning of the gastro-intestinal tract which was full of a brownish fluid, had enlarged lungs though other organs did not show any noticeable change. Two small lobes of lung were consistently consolidated and the other lobes were enlarged and reddish in colour. Consolidation of the lobes was more pronounced in the moribund animals.

Histological observations—Within the first 48 hr after exposure, severe emphysema was observed in the peripheral area of the lobes (Fig. 2). Congestion was prevalent in the alveolar capillaries with diapedesis, pneumocytes were hypertrophied, and large mononuclear cells were lying loose in the alveoli. Moderate lymphoid hyperplasia and presence of inflammatory infiltrates were observed in the peribronchial areas. Desquamated bronchiolar epithelial cells along with inflammatory mononuclear cells were found inside the bronchiolar lumens (Fig. 3).

After 72 hr peripheral emphysema reduced, alveolar congestion persisted, hypertrophied pneumocytes increased in number, alveolar septa slightly thickened, edema and the changes of bronchioles more pronounced. These changes were more intense in consolidated lobes (Fig. 4).

After 7 days of exposure emphysema was reduced considerably with persistent congestion and presence of numerous inflammatory cells. Some of the alveolar septa were thickened. Bronchiolar epithelial cells were completely denudated exposing the deeper layer and had inflammatory exudate. Lymphoid aggregation in the peribronchial areas was prominent. Polymorphonuclear cells were larger in number than mononuclear cells in the interstitial area. Increased laying down of reticulin was also seen in the peribronchial and perivascular areas (Figs 5 & 6).

After 14 days of exposure, emphysema was significantly reduced as compared to those observed earlier. Many alveoli were apparently unaffected, whereas some isolated foci of cellular reactions comprised primarily the polymorphonuclear leucocytes and occasionally mononuclear cells were still evident in association with haemorrhage and edema (Fig. 7). Alveolar septa were thickened and there was significant reticulation in the foci of cellular reaction (Fig. 8). Peribronchial edema, inflammatory cellular infiltration and fibrosis were also evident along with the exfoliation of bronchiolar epithelium affecting the deeper layers of bronchioles (Fig. 9).

Severe bioreactivity due to exposure to MIC and progressive regeneration during postexposure recovery appears to be responsible for above observations. Our histopathological studies indicate that the primary target of MIC induced injury in respiratory tract was in proximal airways, and then, to some extent, also in alveoli. These findings are consistent with the reports of acute toxicity from MIC[4-10] and toluene di-isocyanate[16,17].

Since MIC is a highly irritant, reactive and necrotizing agent, the initial injury could be because of its direct corrosive action on pulmonary tissue. Lung, in its neutral physiology, has a very humid environment where MIC can react violenty, so that the consequent generation of heat can substantially contribute to its necrotizing action. The extent of pulmonary injury observed in our studies at a chamber concentration of 3.20 mg/l for 8 min varies from that in other studies utilizing 0.02-10 mg/l for 1 hr[4,8] or 0.32-1.04 mg/l for 30 min[10], possibly due to a greater amount of MIC entering the lung and reacting with all available moisture of the upper respiratory tract to escape and reach alveolar areas of lung.

Genetic toxicity potential, tested after 7 and 14 days of exposure, did not exhibit significant induction of micronucleus formation in normochromatic erythrocytes ($0.166 \pm 0.02\%$) when compared to that among controls ($0.133 \pm 0.02\%$). Thereafter, genotoxicity of MIC in this short term study is not substantiated through our observations and this is consistent with a recent report of McConnell *et al*[9], although we feel that no definitive conclusions should be made at this time in the absence of data from long-term studies. Investigations are underway to study the long term effects of MIC exposure in rats.

From the above it appears that the exposure to MIC primarily induces pulmonary injury due to its necrotizing/corrosive action. The pathological ab-

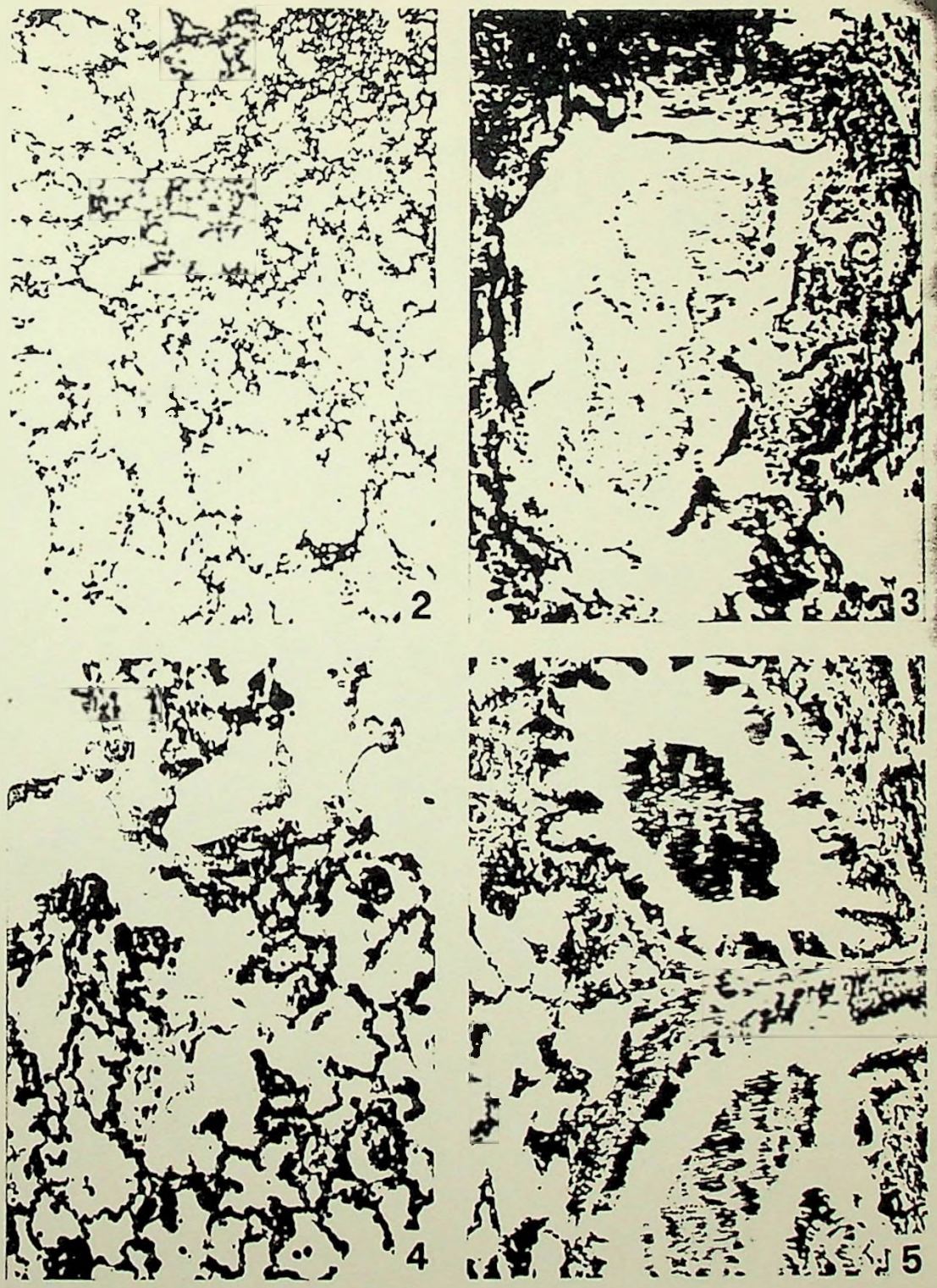
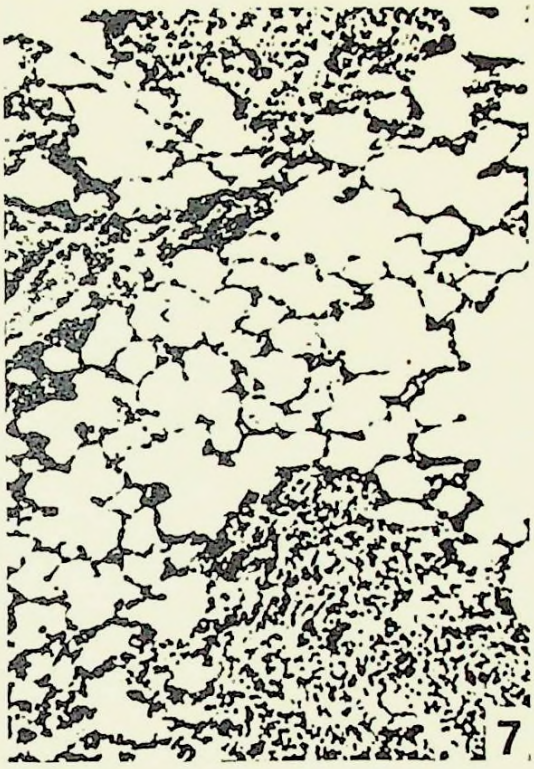


Fig. 2—Section of lungs, showing peripheral emphysema. Two day post-exposure of MIC. H & E × 80.

Fig. 3—Section of lungs showing desquamation of epithelial cells into the lumen of bronchioles, cellular infiltration into the peribronchiolar area. Two day post-exposure of MIC. H & E × 80.

Fig. 4—Section of lungs showing alveolar congestion and presence of edaematous fluid. 3 day post-exposure of MIC. H&E × 130.

Fig. 5—Section of lungs showing desquamation of lining epithelial cells into the bronchioles and presence of inflammatory exudate. 7 day post-exposure of MIC. H & E × 110.



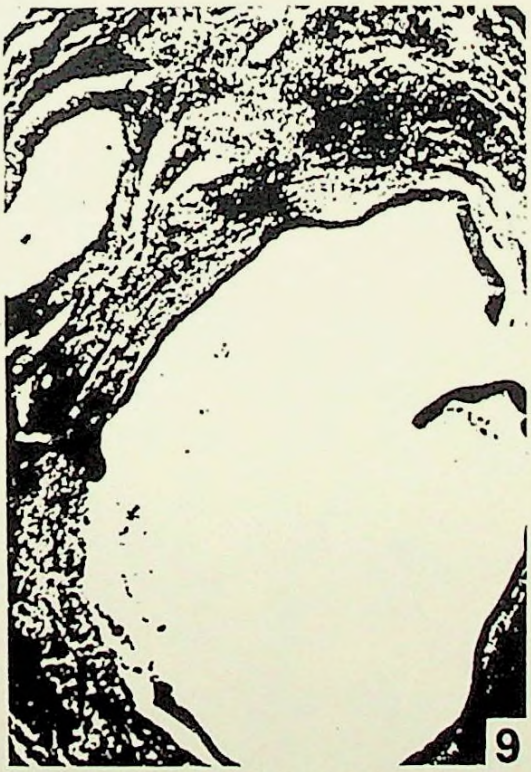


Fig. 6—Section of lungs of the same area of Fig. 7. Showing submucosal reticulination and thickening of the interalveolar septa. 7 day post-exposure of MIC. Gomori's silver nitrate × 110.

Fig. 7—Section of rat lungs showing emphysema, focal cellular reaction and thickening of the interalveolar septa. 14 day post-exposure of MIC. H & E × 110.

Fig. 8—Photomicrograph of the same of the area of Fig. 8. Showing focal area of increased laying down of reticulin. 14 day post-exposure of MIC. Gomori's silver nitrate × 110.

Fig. 9—Section of rat lungs showing desquamation of bronchiolar epithelial cells exposing deeper layer with marked presence of inflammatory cells in the bronchiolar area. 14 day post-exposure of MIC. H & E × 80.

normalities and lethality are dependent upon the dose of MIC exposure. The epithelial erosion is reparable during the postexposure recovery period even though the process of regeneration brings about fibrinogenic response and may be responsible for chronic pulmonary dysfunction. It is expected that these findings will help in understanding and management of toxicosis induced on exposure to MIC in future.

The observations made in this may provide suggestive explanations regarding the abnormal respiratory functions noticed in our human study where we have observed prevalence of combined obstructive-cum-restrictive ventilatory impairment, bronchial obstruction, and restrictive pulmonary defect[18].

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References

1 Kimmerle G & Eben A, *Arch Toxicol*, **20** (1964) 235.
2 Mamath S R, Mahashur A A, Tiwari A K B, Potdar [illegible] Gaur M, Kolhatpar V P, Vaidya P, Parmar D, Rupwa[illegible] Chatterjee T S, Jain K, Kelkar M D & Kinare S G, *pal J Postgrad Med*, **32** (1985) 63.
3 Axford A T, McKerrow C B, Parry J A & Le Quesne P, *Br J Ind Med*, **33** (1976) 65.
4 Nemery B, Dinsdale D, Sparrow S & Ray D E, *Br J Ind* **42** (1985) 799.
5 Dodd D E, Fowler E H, Snellings W M, Pritts I M & [illegible] R L, *Fundam Appl Toxicol*, **6** (1986) 747.
6 Fowler E H & Dodd D E, *Fundam Appl Toxicol*, **6** (1 756.
7 Noorman G A, Brown R, Gupta B N, Uraih L C & Bucher R, *Toxicol Appl Pharmacol*, **87** (1987) 446.
8 Dinsdale D, Nemery B & Sparrow S, *Arch Toxicol* (1987) 385.
9 McConnell E E, Bucher J R, Schwetz B A, Gupta B N, Sh by M D, Luster M I, Brody A R, Boorman G A, Rich C, Stevens M A & Adkins B (Jr), *Environ Sci Techn* **21** (1987) 188.
10 Pant S C, Srivastava R K & Vijararaghavan R, *Bull Envi Contam Toxicol*, **38** (1987) 876.
11 Farland H N, *Fund Appl Toxicol*, **3** (1983) 603.
12 McManus J F A & Mowry R W, in *Staining methods, Histologic and histochemical* (Happer and Row Publishers, East 33rd Street, New York), 1960.
13 Goman G, *Am J Path*, **13** (1937) 993.
14 Schmid W, *Mut Res*, **31** (1975) 9.
15 Alarie Y, *CRC Crt Rev Toxicol*, **2** (1973) 299.
16 Ducan B, Scheel L D, Fairchild E J, Killens R & Graham S, *Am Industr Hyg Assoc J*, **23** (1962) 447.
17 Kaufman J & Burkons D, *Arch Environ Hlth*, **23** (1971) 29.
18 Rastogi S K, Gupta B N, Hussain T, Kumar A, Chandra S & Ray P K, *Indian J Exp Biol*, **26** (1988) 161.

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Special Communication

Bhopal Tragedy's Health Effects

A Review of Methyl Isocyanate Toxicity

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SIX YEARS AGO, on December 3, 1984, a toxic gas leak at a Union Carbide pesticide plant in Bhopal, India, released methyl isocyanate (MIC) and its reaction products. The number of persons "exposed" and "injured" remains uncertain.[1] Official estimates from the Indian government place the dead at around 1800.[2] Others estimate mortality to have been between 2500 and 5000 and the number of injured to have been up to 200 000.[3,4]

See also p 2795.

Until the Bhopal incident, neither deaths nor cases of toxic effects from MIC exposure had been recorded in the *Index Medicus*.[5] We have extensively surveyed the medical literature concerning effects of MIC exposure on the victims of the disaster and laboratory studies in animals. A great deal has been learned, but many questions still remain unanswered.

THE BHOPAL PLANT

In 1969, the Union Carbide Corporation built a formulation plant in Bhopal, India, to mix and package pesticides imported from the United States. The plant was expanded in 1980 to manufacture carbamate pesticides from chemical intermediaries produced on site.[6,7] Bhopal, the capital of the State of Madhya Pradesh, lies 744 km south of New Delhi and has a population of about 900 000 people. The railway station was 3 km from the plant, and a sizable population that worked in the textile mill was located in the area around the station.[7]

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In 1984, squatters' rights were granted to those in the burgeoning settlements surrounding the plant.[2,6] The Indian Council of Medical Research estimated that 100 000 people lived within a 1-km radius of the plant at the time of the tragedy.

Methyl isocyanate, produced at the plant, was stored in liquid form in two steel tanks, each holding 57 120 L , and was used as needed to produce carbaril pesticides.[8,9] For safety reasons, MIC storage tanks at plants in Belgium, the Federal Republic of Germany, Japan, Korea, and the United States (West Virginia) have smalier capacities, holding an estimated 17 500 L with allowable filling of 50%.[4,6]

MIC CHEMISTRY/TOXICOLOGY

Methyl isocyanate is a colorless liquid with a molecular weight of 57.1, boiling point of 39°C, specific gravity of 0.96, and vapor density of 2.[10,11] Contact with water causes an exothermic reaction, resulting in the formation of carbon dioxide, methylamine gases, and *N*, *N*'-dimethyl urea.[8,9] The reaction is enhanced in the presence of acids, alkalis, and amines. Contact with metals and certain catalysts causes violent polymerization, and fire involving MIC results in hazardous decomposition products such as hydrogen cyanide (HCN), oxides of nitrogen, and carbon monoxide.[9,10] The National Institute for Occupational Safety and Health's 1978 publication[12] sets forth the following safety and warning guidelines for MIC: (1) The permissible exposure limit of MIC is 0.02 ppm averaged over an 8-hour work shift, which is also expressed as 0.05 mg of MIC per cubic meter of air. (2) The documentation of threshold limit values states that at 2 ppm no odor is detected but subjects experience eye, nose, and throat irritation and lacrimation. At 4 ppm the symptoms of irritation are more marked. Exposure is unbearable at 21 ppm. (3) MIC is regarded as a material with poor warning properties.

In 1964, Kimmerle and Eben[13] first reported on acute toxic effects of inhaled MIC in animals, establishing the lung as the primary site of injury. Up until 1984, data provided by industries using MIC in pesticide and herbicide manufacturing processes mainly dealt with chemical properties rather than toxicology.[11] Since the Bhopal incident, however, priority was assigned to MIC toxicology assessment.[13,14]

THE BHOPAL INCIDENT

The widely accepted explanation for the cause of the release of MIC and its reaction products from the Bhopal plant was the entry of water into tank 610, which contained approximately 40 900 kg of MIC. This resulted in an exothermic reaction causing approximately 24 545 kg of MIC and 12 800 kg of reaction products to escape within a few hours.[6,8] Estimated time of release of MIC and its reaction products was 12:30 AM on December 3, 1984. By 2 AM, most of the contents of tank 610 had escaped, spreading as a cloud over a large, densely populated area of approximately 40 km^2. The first deaths were reported to the police by 3 AM and by morning, more than 1000 people were reported dead, some as far as 8 km from the plant.[6,15]

Meteorological conditions at the time of the MIC leak aggravated the effects of its toxicity. The temperature that night was 10°C and the air was dry. The winds were from north to northwest at 1 to 2 m/s (3 to 4 mph) in the direction of settlements located in the vicinity of the plant. Temperature inversion also reduced the dilution of the gas cloud. Thus, the area with the largest number of dead and severely injured was approximately 6.65 km^2 south of the plant.[16]

Ninety thousand patients were seen within the first 24-hour period at nearby

hospitals, clinics, and small area health facilities.[17] Bhopal has four major hospitals and several clinics that provide primary health care. Three hundred physicians staff these 1800 beds.[18] Little was known about possible treatment for toxic effects due to MIC exposure, and several days after the accident it was still uncertain whether the effects being observed from the gas leak were due to MIC, phosgene, HCN, or other MIC reaction products.[2,19] Treatment of the exposed population was thus limited to symptom management because no scientific agreement could be reached on what might constitute an appropriate antidote.

ACUTE TOXIC EFFECTS

About 200 000 people were acutely affected by the MIC leak. In all, 2500 are estimated to have died during the first week. Immediate health effects observed were respiratory problems and eye irritation.[2,19] Six thousand people suffered from acute respiratory distress, plus symptoms involving the circulatory, gastrointestinal, and central nervous systems. Total exposure time, concentration levels of MIC, age, sex, and physical condition of victims played a major role in the development of acute and chronic disease.[16,20,21] There is no consensus on MIC concentration levels in Bhopal's atmosphere immediately following the gas leak. None of the data on morbidity and mortality are firm.

Hydrogen cyanide has been implicated in the acute injury caused by the gas leak.[22] Blake and Ijadi-Maghsoodi[23] and Bhattacharya et al[24] have shown that pyrolysis of MIC at high temperatures (350°C to 540°C) resulted in the formation of HCN and other degradation products. The cherry-red color of the blood and viscera of the victims, increased urinary thiocyanate levels in some survivors, and reported symptomatic relief by administration of sodium thiosulfate are cited as evidence supporting the causative role of HCN.[22,23,25] Others dispute this evidence.[22-24] In vitro, MIC itself can produce "cherry-red blood" due to formation of methylamine.[27,28] Experiments with laboratory animals produce evidence incompatible with cyanide toxic effects in Bhopal victims.[29] Elevated urinary thiocyanate levels in Bhopal victims, often measured months after the exposure, cannot be equivocally attributed to the original toxic gas exposure because of the relatively rapid clearance of thiocyanate.[30,31] High thiocyanate levels also may be explained by other factors such as water contamination, smoking, and dietary habits of the affected population.[31] As a result, the role of HCN as a causative factor remains ill-defined at this time.

No attempt has been made to recreate the incident experimentally to examine effects on animals. However, animal inhalation exposure studies mimicked the gradation of human responses that occurred at different distances from the chemical plant. Overall objectives of these studies were to determine the consequences of injury following acute exposure to high MIC vapor concentration and to elucidate the pathogenesis of early mortality. In one study, rats and mice were subjected to lethal and sublethal concentrations of MIC by inhalation.[32] Exposure to a 20- to 30- ppm concentration of MIC for 2 hours caused severe dyspnea and weakness followed by death within 15 to 18 hours. A second wave of deaths occurred 8 to 10 days later, preceded by respiratory distress. Exposure to a 3-ppm concentration caused no respiratory abnormalities, while at 10 ppm, obstructive airway lesions were evident even 13 weeks after exposure. These phases corresponded to those seen in Bhopal victims. Lethal exposure studies done on rats, mice, and guinea pigs revealed extensive degenerative changes in epithelial lining extending from the nasal cavity to the level of the bronchioles with mucus, fibrin, and sloughed-off epithelium plugging the airways.[32-35]

RESPIRATORY EFFECTS

Involvement of the respiratory system was the most common and serious health problem of the victims. Misra et al[36] retrospectively reviewed charts of 978 patients admitted to Hamidia Hospital during the first 3 days after exposure. In 544 victims, symptoms included breathlessness (99%), cough (95%), throat irritation or choking (46%), chest pain (25%), and hemoptysis (12%). Cyanosis was conspicuous by its absence. A more detailed report from the same institution[37] on the range of radiologic pulmonary changes in 500 victims during the first 3 days of exposure described interstitial edema (41%), alveolar and interstitial edema (41%), and destructive lesions with cavitation, pneumomediastinum, or emphysema (8%).

Kamat et al[38] studied 82 individuals with persistent symptoms between the seventh and 55th days after exposure. Seventy-eight percent showed a restrictive pulmonary functional defect which in one fourth improved with bronchodilator therapy, suggesting a secondary obstructive element. Inability to maintain normal maximal-minimal ventilation and oxygen uptake at rest was seen in 45 individuals (55%). Twenty-four subjects had impaired ability to increase oxygen uptake on exercise, and in 78 there were extensive roentgenographic changes suggesting interstitial depositions. Flow volume studies of 35 individuals showed changes in small airways in 12 and incoordination in central airways in 15. Blood carboxyhemoglobin levels measured in 69 individuals were raised in 66, while methemoglobin levels measured in 80 were raised in 63. In a separate study,[39] the authors reassessed victims over a 6-month period. Of 78 subjects studied at 3 months, results of pulmonary function tests were unchanged in 46%, improved in 38%, and worsened in 12%. Only 50% of 52 subjects studied at the end of 6 months showed improvement. Pate et al[40] did sequential measurements of maximal expiratory flow volume on the above subjects for 18 months and found chronic respiratory disability with flow volume reductions due to restrictive lung disease with alveolitis.

Two months after exposure, the results of demographic and clinical studies[41,42] of 1109 subjects supported the results of the studies noted above. Thirty-nine percent of 783 patients showed ventilatory impairment by lung spirometry tests, with females exhibiting a higher prevalence (44%) of abnormalities in comparison with males (32%). Naik et al[43] reported pulmonary function studies on 569 persons. Group 1 comprised 446 persons residing within 0.5 to 2 km of the plant, and group 2 comprised 123 persons who lived farther than 8 km from the plant. At least 109 days after the exposure, 72% of group 1 had respiratory symptoms that showed persistent and, in some cases, worsening pulmonary function test results. In group 2, 17% suffered from respiratory ailments, a much higher incidence than would be expected in a control population. Six to 8 months after the exposure, lung biopsies done on three of the victims revealed alveolar wall thickening, interstitial fibrosis, and exudative reaction in terminal bronchioles with bronchiolitis obliterans. Pulmonary hypertension related to obstructive functional defect was also detected in some victims.[4,36]

Experimental studies done by Pai et al[44] on rabbits and rats after exposure to MIC at concentrations of 3 and 10 mg/L, respectively, revealed inflammatory reaction, destruction of alveolar architecture, and pulmonary edema. In the majority of animals, epithelial necrosis of the bronchioles was extensively damaged. These findings suggested that MIC predominantly damaged lung tissue due to its corrosive action. Agarwal and Dutta et al[45] showed that a

level exposure to MIC immediate-
led the cell lining of the respiratory
in both mice and rats. Animals that
ived the exposure showed excess
ous tissue occluding the airways and
pering oxygen exchange. Alarie et
exposed Swiss-Webster mice to
at 0.5 to 7.6 ppm for 90 minutes and
ed MIC to be a more potent senso-
tant of the upper respiratory tract
formaldehyde, chlorine, or ammo-
It was a strong pulmonary irritant
almost the same concentrations,
ing MIC a chemical with poor warn-
characteristics. Nemery et al[46] con-
ed the highly irritating properties
IC. In longer-term studies done by
rman et al,[49,50] mice were exposed for
urs to MIC concentration levels of 0
trol), 3, 10, and 30 ppm, and fol-
d up for 91 days. At 30-ppm concen-
ons, extensive necrosis of the respi-
ry tract, including trachea and main
chi, was observed. Though regen-
ion of [redacted]helium occurred rapidly,
luminal and mural fibrosis of the
bronchi persisted in many of the
at the conclusion of the 91-day
y. Ursell et al[51] assessed the ultra-
ctural changes of nasal and respira-
epithelium and confirmed the
helial necrosis and gradual regener-
n with persistent fibrosis in experi-
tal animals. Tepper et al[52] studied
iopulmonary effects in rats
o 6 months after MIC exposure
0 ppm for 2 hours and found evi-
ce of pulmonary hypertension. This
documented by electrocardiogram
ges and supported by postmortem
ysis that showed right ventricular
ertrophy. Srivastava et al[53] exposed
to sublethal concentrations (137
for 30 minutes) of inhaled MIC so as
void the induction of pulmonary ede-
and then studied lung morphologic
acteristics and mechanisms. Maxi-
dynamic surface tension de-
eed while total lung capacity, lung
ning, and compliance increased,
ngs that correlated with histologic
ges of emphysema. In a study on
structure of respiratory tract in
exposed to 0.25 mg/L of MIC for 1
. Dinsdale et al[54] showed a "raft" of
ls and fibrin lining most of the air-
during the first week after expo-
but with repair well advanced
2 to 3 days. The majority of the
ts were lined with normal epithe-
within 3 weeks of exposure, but
ed foci of hyperplasia and occluded
s persisted.
e clinical studies described above
methodological problems. They
retrospective; lack suitable con-
and have biases in history taking,
keeping, and patient responses.

Radiologic and pulmonary function tests are limited and poorly organized. Nevertheless, there appears to be adequate evidence of fibrosing bronchiolitis obliterans as an effect of long-term injury, but there is no credible evidence regarding its frequency in the exposed population. Well-designed animal experiments corroborate the development of acute lung injury and chronic restrictive lung disease along with persistent airway obstruction.

OPHTHALMIC EFFECTS

On the first day of the disaster, 6000 people were seen as outpatients and another 2000 were seen as inpatients by the Department of Ophthalmology, Hamidia Hospital.[42] Presenting manifestations included severe watering of the eyes, photophobia, profuse lid edema, and corneal ulcerations. Survivors with the most severe eye effects were studied by a team of Indian and British physicians under the auspices of the Royal Commonwealth Society for the Blind.[44] A week following exposure, several victims were still experiencing photophobia, while a minority had eyelid swelling. In all cases, slit-lamp examination revealed a discrete superficial interpalpebral erosion of the cornea and conjunctiva with the typical whorling pattern of new epithelialization. Blindness or irreversible eye damage was not seen. In the 2 weeks immediately after the disaster, the same investigators[45] carried out a community-based epidemiological study of eight exposed (379 subjects) and two unexposed (119 subjects) clusters of households. The sample represented 2% of the estimated population in the area surrounding the study clusters. There was no evidence of eye problems other than superficial interpalpebral erosion and redness in the exposure clusters compared with the nonexposure clusters.

Dwivedi et al[26] reported on ocular disease in 232 children admitted to Hamidia Hospital. Symptoms included severe ocular burning (100%), eye swelling (95%), redness (94%), watering of the eyes (89%), ocular pain (76%), and photophobia (44%). Ninety-three percent had only mild involvement, with conjunctival vascular engorgement and chemosis; 6% had corneal involvement ranging from punctate keratopathy to epithelial denudation; and only 1% (three patients) had more severe involvement, with stromal haze and iritis.

Approximately 15 weeks after exposure, Maskati[27] studied two groups from similar socioeconomic backgrounds. Group 1 comprised 261 subjects residing within 2 km of the plant, and group 2 comprised 106 people residing 8 km from the plant. Findings included burning eyes (38%), diminished vision (19%), corneal opacities (6.5%), cataracts (4%), and night blindness (1.5%) in group 1 subjects, as compared with 10%, 7.5%, 2%, 2.8%, and 0%, respectively, in group 2 subjects. Eleven people in group 1, compared with three in group 2, were suffering from cataracts (six of the 11 were between 21 and 50 years of age).

Raizada and Dwivedi[28] evaluated chronic ocular lesions in 1140 patients who had been hospitalized earlier with acute oculorespiratory symptoms. Main lesions noted were chronic conjunctivitis (14%), refractive changes (35%), deficiency of tear secretions (5.6%), and persistent corneal opacities (0.5%). No changes were seen in the iris, lens, vitreous, or retina that could be attributed to MIC exposure. Khurrum and Ahmad[29] also studied 2280 patients 2 to 3 years after their acute eye injury. Observed abnormalities included chronic conjunctivitis (15%), refractive vision changes (3.5%), deficiency of tear secretions (6.7%), and persistent corneal opacities (9%).

Prior to Bhopal, Pozzani and Kinkead[40] showed that MIC application to rodent eyes caused severe necrosis. In experimental studies, low molecular weight ethyl and methyl isocyanates have been proven to be skin and eye irritants.[41,42] After the Bhopal incident, Harding and Rixon[43] induced opacities in young rat lenses incubated in 50-mmol/L MIC, thereby speculating that MIC could induce cataracts. Gupta et al[54] exposed rats to 10- to 30-ppm concentrations of MIC for 2 hours, doses sufficient to cause pulmonary injury or death. Eye irritation developed, but there was no irreversible eye damage.

All of the clinical trials were observational studies of the case series design with descriptive accounts of the eye injuries that occurred in a sample of patients exposed to MIC. The studies were susceptible to selection and measurement bias. The need for Bhopal area residents to have kept their eyes open while fleeing from the disaster may have been responsible for the higher eye injury rate as compared to the rates seen in rats. From these studies, we can conclude that MIC causes acute eye injury and probably does not cause permanent serious eye damage.

MATERNAL-FETAL, GYNECOLOGICAL, AND GENETIC EFFECTS

Varma[46] conducted an epidemiological survey of 3270 families 9 months after exposure to MIC. Retrospective medical history of the same families for

2 years prior to the incident served as the control, thereby minimizing variables such as cultural differences, language barriers, and socioeconomic factors. Forty-three percent of 865 pregnancies in the exposed group terminated in fetal loss, representing a threefold to fourfold greater incidence of fetal loss as compared with the normal incidence of 6% to 10% in Bhopal as estimated by the Indian Council of Medical Research. The spontaneous abortion rate appeared to be higher in those exposed to MIC while in the first trimester of pregnancy. Of 486 live births, 14% of infants died within 30 days as compared with a 3% rate recorded for 1983 and 1984 in the same group. Shilotri et al[66] surveyed 100 women of childbearing age residing within a 10-km distance from the plant. Of 38 women who were pregnant at the time of the disaster, 29 had spontaneous abortions and two had premature deliveries. Both surviving infants had multiple anomalies: spina bifida, meningomyelocele, limb deformities, and heart disease. Autopsies revealed lung disease, similar to MIC-induced lung changes described in adults.

Deo et al[67] studied immunologic, mutagenic, and genotoxic effects on the exposed population and concluded that MIC, being highly reactive, interacted with enzymes involved in DNA replication and repair and, thus, may be the cause of serious repercussions in the development of the embryo. Kanhere et al[68] studied the morphologic characteristics of 134 human placentas in pregnant women exposed to MIC, following full-term and premature births, and after medical termination of pregnancy. An increase in hydropic degeneration of placental tissue was noted after medical termination of pregnancy. Some increase in calcification was noted in placentas of infants born prematurely. Even though these changes were not statistically significant, they were indicative of poor perfusion and anoxia.

Fifteen weeks post-MIC exposure, Naik et al[69] measured blood and urine thiocyanate levels in 310 subjects residing within 8 km of the plant. As compared with healthy, nonsmoker controls, thiocyanate levels in the exposed population were significantly higher. In the 1940s, hypothyroidism and thyroid goiter were discovered as complications of thiocyanate treatment for hypertension.[70] Maternal hypothyroidism can cause serious sequelae in the infant.[70] This is an aspect of fetal toxic effects that has not been studied, and epidemiological studies on infants born several months following the disaster will be needed to uncover such effects due to MIC exposure.

Fifteen weeks after MIC exposure, Shilotri et al[66] surveyed 198 women who resided within 10 km of the plant. One hundred women complained of gynecologic symptoms and had an increased incidence of leukorrhea, abnormal uterine bleeding, and abnormal Papanicolaou smears. In another study, Deo et al[67] studied semen samples of 19 subjects 100 to 120 days after MIC exposure and reported normal sperm counts, morphologic features, and motility. The authors acknowledged that the survey may have been too late to detect damage to mature sperms because the duration of spermatogenesis is 74 days in man.

Using lymphocyte culture methods, Goswami[71] studied chromosomes for chromosomal aberrations and sister chromatid exchange frequencies, parameters regarded as good indicators of genetic damage. As late as 12 months after the exposure, 71% of the exposed population showed evidence of chromosomal damage as compared with 21% incidence in a control population residing 20 to 50 km from the plant.

Animal experiments were done to study MIC's effects on pregnant mice and rats. Varma et al[72] reported 80% resorption of implants when pregnant mice were exposed for 90 to 180 minutes to 9 to 12 ppm of MIC on the eighth day of gestation. Schwetz et al[73] reported increased dead fetuses and decreased neonatal survival when pregnant Swiss mice were exposed to 1 to 3 ppm of MIC for 6 hours a day during the 14th to 17th days of gestation. These studies[6,72] also revealed a higher incidence of fetal malformations, such as reduction in extremity bone length, diaphragmatic hernia, myocardial thinning, cleft palate, and hydrocephalus, as compared with controls. To determine if maternal pulmonary irritation was essential for the development of fetal toxic effects, MIC was injected intraperitoneally in pregnant mice.[6] Increased resorption of fetal implants, even without pulmonary irritation, indicated the involvement of nonpulmonary factors. Bhattacharya et al[74] reported covalent binding of MIC tagged with carbon 14 (^{14}C) to brain, liver, kidney, and lung proteins when administered intraperitoneally or by inhalation to female Wister rats. Ferguson et al[75] studied uptake and distribution of ^{14}C during and following exposure to [^{14}C]MIC in pregnant mice and found ^{14}C in all tissues examined, including uterus, placenta, and fetus.

Genotoxic and mutagenic studies were carried out using a variety of in vitro and in vivo assays. Mason et al[76] were unable to demonstrate MIC-induced mutation by *Salmonella* reversion assay or *Drosophila* sex-linked recessive lethal assay. There was increased sister chromatid exchange frequencies and chromosomal aberrations in mammalian cell culture of Chinese hamster ovary cells. Studies by Kligerman et al,[77] Tice et al,[78] and Shelby et al[79] showed that MIC induced chromosomal damage in actively dividing bone marrow cells, thereby indicating MIC's potential to induce systemic genotoxic and/or cytotoxic activity.

All of the clinical studies on maternal, fetal, gynecologic, and genetic effects were retrospective cohort studies. However, well-controlled animal experimental studies support the clinical observations. Radiolabeled ^{14}C studies show carbamylation and MIC's ability to cross the blood-tissue barrier. The forms of ^{14}C distributed in blood and tissues have not been identified and their toxicity remains to be established. Preliminary evidence from genetic and mutagenic experiments emphasizes the need for case-controlled, cohort studies of the victims.

PEDIATRIC EFFECTS

In the first 2 weeks after the disaster, the Hamidia Hospital Pediatric Department had admitted 1337 children for treatment of MIC toxic effects.[7] One hundred nineteen deaths were reported in the first 12 days, with 106 deaths occurring in the first 4 days of exposure. The majority of children presented with symptoms of breathlessness, cough, painful watering eyes, photophobia, diarrhea, and vomiting. Some had convulsions, hemiparesis, and coma. Bharucha and Bharucha[81] reported on neurologic manifestations. Twenty-four of 47 children had lost consciousness for varying periods up to 24 hours. Three children had convulsions. An interesting observation by physicians who had examined the children early in the illness was the generalized hypotonia and weakness.

Irani and Mahashur[82] studied 211 children who were exposed to MIC and surveyed them 100 days following the gas leak. Group 1 comprised 164 children residing within 2 km of the plant. Group 2 comprised 47 children residing more than 8 km from the plant. In group 1, cough (83.5%), eye symptoms (79.8%), and breathlessness (34.5%) were the most common symptoms. These symptoms were found in only 8.5% of the children in group 2. In group 1, 40% to 45% of the children had persistent pulmonary symptoms and signs. Eye symptoms of the children in group 1 decreased from 79.8% to 34.5% at the end of the 100-day study. Some of the children exhibited psychological problems and psychiatric problems relating

eir medical status and social dis-
rtions.

All the pediatric clinical studies were
servational in nature and have seri-
s methodological shortcomings in
udy design. Lack of attention to this
egulation is unfortunate, since it is the
ildren who may have long-term toxic
fects.

MMUNOLOGIC EFFECTS

Two and one-half months after expo-
re, Saxena et al[36] studied humoral im-
unity (measurement of serum immu-
oglobulin levels) and cell-mediated
mmunity (measurement of T-cell ro-
ettes and phagocytosis) of exposed vic-
ms and found suppression of cell-medi-
ed immunity only. Karol et al[34,35] found
ransient, low-titer, MIC-specific anti-
odies in 12 of 144 exposed persons as
ompared with no such antibodies in
ontrols. Although the titers were low
nd tra[illegible]nt, the results suggest that a
ingle exposure to MIC was capable of
nducing antibodies that were main-
ained for several months.

Dwivedi et al[37] studied immunologi-
al response in rats 7 days after MIC
xposure and found impaired alveolar
nd peritoneal macrophage function and
mpaired delayed hypersensitivity re-
ponses. Karol et al[35] injected guinea
igs with MIC in its reactive isocyanate
orm and showed antibodies specific to
TC hapten 3 weeks later. Tucker et al[37]
xposed female mice subacutely to MIC
o 3 ppm of MIC for 6 hours a day for 4
onsecutive days) and failed to show pri-
ary immune system toxic effects with-
n 5 days of exposure when antibody
esponse to sheep erythrocytes, natural
iller cell activity, and lymphoprolifera-
ive responses to mitogen were studied.
esistance to infectious agents, *Lis-*
eria monocytogenes, mouse malaria
arasite, and influenza viruses, or to
ansplantable tumor cells was not com-
romised. Using the same model, Hong
t al[37] examined bone marrow parame-
ers in two groups of mice, receiving 1
nd 3 ppm of MIC, respectively, on days
, 3, and 21, after a 4-day inhalation
xposure (6 h/d). Myelotoxicity was in-
uced as evidenced by hypocellularity
nd suppression of pluripotent stem
ells, granulocyte-macrophage progeni-
ors, and erythroid precursors. Hema-
opoietic parameters returned to nor-
al by 21 days in the 1-ppm dose group,
owever, in the 3-ppm dose group, indicat-
ng that bone marrow toxic effects per-
isted for a relatively long period at
oses of 3 ppm even when there was no
ther clinical abnormality.

Few immunologic toxicity studies of
IC have been reported. Further studi-
s are needed to clarify whether injury
to the human immune system is caused primarily by MIC or is secondary to lung disease, and to establish its biological significance.

HEMATOLOGICAL AND BIOCHEMICAL EFFECTS

Two and one-half months after the incident, hematological and biochemical studies on 695 MIC-exposed victims showed hemoglobin levels greater than 7.4 mmol/L and a higher total RBC count in 78%.[39] Though WBC counts were normal in most victims, polymorphonuclear leukocytosis (11.9%), lymphocytosis (40%), and increased erythrocyte sedimentation rate (36.4%) were noted. Blood glutathione level was significantly lowered in 40% of the population examined. Serum ceruloplasmin level was generally about twofold higher. Results of routine liver function tests were normal. Fifteen weeks following exposure, blood carboxyhemoglobin, methemoglobin, and thiocyanate levels were significantly higher in MIC-exposed victims as compared with controls. This persistence was attributed to consumption of food and water contaminated by breakdown products of MIC. Fifteen weeks following the incident, Naik et al[40] found thiocyanate levels from Bhopal lake and tap waters to be twice as high as in water supplied for domestic consumption in Bombay (720 km from Bhopal). At the end of 9 months, thiocyanate levels had returned to baseline levels.

In rats and guinea pigs, neither intravenous injection of MIC nor in vivo exposure to 1000 ppm of MIC by inhalation resulted in any inhibition of erythrocyte choline esterase activity or alteration in hemoglobin electrophoretic mobility. This suggests that neither erythrocyte choline esterase inhibition nor structural alteration of hemoglobin was a major contributing factor to death resulting from MIC exposure.[29] Bucher et al[41] exposed rats and mice to 0, 1, 3, and 6 ppm of MIC for 6 hours on 4 consecutive days. No significant changes were observed in red blood cell indexes, platelet count, total leukocyte count, or serum creatinine, blood urea, or methemoglobin levels. Counts of segmented neutrophils increased while lymphocytes decreased. These researchers attributed the slight changes in the hematological picture to pulmonary injury caused by MIC at these exposure levels.

Hematological and biochemical toxicity studies were minimal. If MIC were to gain entry into the bloodstream, systemic toxic effects with abnormal changes reflected in hematological and biochemical values might be expected. However, no significant abnormalities were noted in Bhopal area residents.

NEUROMUSCULAR EFFECTS

Bharucha and Bharucha[42] reported neurological manifestations in 129 adults and 47 children affected by MIC exposure. Of 129 adults screened, 50% were reported to have lost consciousness in the initial hours following exposure. Other symptoms reported included muscle weakness, tremors, vertigo, ataxia, and fatigue. In another study,[42] 33 victims who were 0.1 to 8 km from the plant were clinically evaluated. Eighteen persons fainted after inhaling the toxic gas and remained unconscious for a variable duration. Three of them had prolonged unconsciousness and hyperreflexia; one had features suggestive of encephalopathy.

Experimental studies were carried out on cell cultures isolated from muscle of 2-day-old rats after exposing them to MIC concentrations of 0.025 to 0.5 μL per 5-mL culture. Light and electron microscopic studies of nuclei of both cell types (fibroblasts and myoblasts) were carried out. At higher concentrations, both cell types were killed. At lower concentrations, myoblasts appeared to be more susceptible to MIC's toxic effects than fibroblasts. This study concluded that MIC directly affects muscle tissue and prevents differentiation in in vitro situations.[43] Sethi et al[44] studied the effect of acute exposure on rats to 3.52 and 35.32 ppm of MIC. Along with known acute ophthalmological and pulmonary signs, animals were observed to have progressively increasing ataxia, uncoordinated movements, and immobility.

Clinical studies on neurological effects of MIC toxicity were simple observations susceptible to many possible biases related to subject selection and characteristics observed. No valid conclusions can be drawn. Animal experimental data on neuromuscular effects of MIC are also minimal.

PSYCHOLOGICAL EFFECTS

Sethi et al[45] collected data on psychiatric problems from patients attending an outpatient clinic located in a severely affected area for a 4-month period (February 1985 to May 1985). Of 208 patients with psychiatric symptoms, 45% were diagnosed as having neurotic depression, 35% suffered from anxiety states, and 9.7% were found to have adjustment reaction. Psychoses were uncommon. Cumulative data were also obtained from 10 satellite government clinics set up in moderately and severely affected areas. Of 855 patients reporting with medical problems, 193 adults (22%) also had psychiatric symptoms.

Diagnostic categories included neurotic depression (37%), anxiety states (25%), adjustment reaction—prolonged depressive type (20%), and adjustment reaction with predominant disturbance of emotions (15%). Again, psychoses were uncommon.

It has been recognized that disasters can have substantial effects on the mental health of victims. The most commonly recognized disturbance is posttraumatic stress disorder (PTSD). In the general population, this is rare, occurring in less than 1%.[96] Studies have suggested that 15% to 35% of Vietnam veterans developed PTSD, about 30% to 59% of people may do so after natural disasters, and 80% may develop PTSD after man-made disasters.[97,98] During a follow-up period of 2.5 years, the prevalence of PTSD in a group of 459 fire fighters exposed to the 1983 Ash Wednesday brush fires in Southern Australia varied little from 30%, thereby emphasizing the long-term psychiatric morbidity after a natural disaster.[99]

The number of factors producing stress, as well as previous studies of PTSD and stress due to economic factors, suggest that psychiatric morbidity in Bhopal is expected to be high as compared with populations not exposed to group trauma.[96,100] Regrettably, the clinical studies of psychological effects among the Bhopal survivors were uncontrolled observations on a small sample of the population, and therefore valid conclusions cannot be drawn.

CONCLUSION

The Bhopal tragedy has fallen from the public spotlight, leaving pressing questions still unanswered. We do not have reliable figures on the number of persons who died or were injured; nor do we know why the gas leaked or whether MIC was the only culprit. Most of the clinical studies on the victims were observational in nature and uncontrolled. Bias was pervasive and there was insufficient information to allow careful operational definition of crucial matters, such as criteria for inclusion and exclusion of subjects and controls, and effects of independent and dependent variables on study outcome. Data quality therefore could not always be ensured. Initially, systematic study was prevented by the chaos and trauma that followed the disaster; later, inadequate resources compromised study design. On a strictly scientific level, these observational studies do not conclude causality. However, many of the conclusions are supported by experimental studies, and by analyzing both clinical and experimental studies much can be learned about MIC's effects on the health of the people of Bhopal.

The Bhopal tragedy has raised a number of public health issues, in terms of policy in regard to accident prevention, contingency planning in case of disasters, and actions to be taken before and after disasters that would minimize death and suffering.[4,15] Comprehensive right-to-know laws, providing communities and governments with public information about the character and extent of releases of toxic materials from industrial processes, need to be enacted. Multinational corporations need to operate under a minimum set of standards, even if local laws are weak. It is important that governments act both within their own countries and through the United Nations to create the minimal regulations that will be needed to prevent future disasters, create contingency plans, and produce sound risk assessments.

International funds need to be established for research in this area. Primary objectives should include the provision of the facilities needed to obtain immediate epidemiologic data at the onset of a major toxic disaster, monitor the health of the surviving victims, provide what treatment is possible, study the causes of toxic effects, develop a cure, and plan for future prevention. We need a clearinghouse for information about industrial activities so as to make communities, workers, and the health professionals aware of health and safety precautions.

As we engage in an assessment of chemical technology, the lesson learned from Bhopal is that the tragedy of man-made disasters can be reduced by encouraging and planning for competent medical and scientific analyses. Such research should be done promptly with appropriate methodological designs and with full public disclosure of the results.[101] Data and knowledge from such analyses can be used to treat and adequately assess the condition of victims and, it is hoped, prevent tragedies of this magnitude from occurring in the future.

References

1. Weill H. Disaster at Bhopal. *Bull Eur Physiopathol Respir* 1988;23:387-390.
2. Bogard W. *The Bhopal Tragedy.* Boulder, Colo: Westview Press Inc; 1989.
3. Lepkowski W. Bhopal: Indian city begins to heal, but conflicts remain. *Chem Eng News.* December 2, 1985:63:18-32.
4. Jasanoff S. The Bhopal disaster and the right to know. *Soc Sci Med.* 1988;27:1113-1123.
5. Calamity at Bhopal. *Lancet.* 1984;2:1378-1379. Editorial.
6. Morehouse W, Subramaniam AM, eds. *The Bhopal Tragedy.* New York, NY: Council on International and Public Affairs; 1986.
7. Everest L, ed. *Behind the Poison Cloud.* Chicago, Ill: Banner Press; 1985.
8. Union Carbide Corporation. *Bhopal Methyl isocyanates Incident: Investigation Team.* Report. Danbury, Conn: Union Carbide Corp; March 1985.
9. Smith AB, Zenz C. Methyl isocyanate. In: Zenz C, ed. *Occupational Medicine Principles and Practical Applications.* Chicago, Ill: Year Book Medical Publishers Inc; 1988:745-746.
10. *Occupational Health Guidelines for Methyl Isocyanate.* Washington, DC: US Dept of Health and Human Services; September 1978:1-4. NIOSH publication 81-123.
11. Union Carbide Corporation. *Methyl Isocyanate Technical Report F-41443A.* New York, NY: Union Carbide Corp; 1976.
12. Kimmerle G, Eben A. Toxicity of methyl isocyanate and how to determine its quantity in air. *Arch Toxicol.* 1964;20:235-241.
13. Bucher JR, Schwetz BA, Shelby MD, Mellon EE. Introduction: the toxicity of methyl isocyanate. *Environ Health Perspect.* 1987;72:72.
14. Bucher JR. Methyl isocyanate: a review of health effects research since Bhopal. *Fundam Appl Toxicol.* 1987;9:367-379.
15. Bhopal Working Group. The public health implication of the Bhopal disaster: APHA technical report. *Am J Public Health.* 1987;77:230-236.
16. Lorin HG, Kulling PE. The Bhopal tragedy: what has Swedish disaster medicine planners learned from it? *J Emerg Med.* 1986;4:311-316.
17. Tarbakhsh SA. The Bhopal disaster. *J R Soc Health.* 1987;107:1-2.
18. Shrivastava P, ed. *Bhopal: Anatomy of a Crisis.* Cambridge, Mass: Ballinger Publishing Co; 1987:37.
19. Bucher JR. The toxicity of methyl isocyanate: where do we stand? *Environ Health Perspect.* 1987;72:197-198.
20. Marwick C. Bhopal tragedy's repercussions may reach American physicians. *JAMA.* 1985;253:2001-2003.
21. Zaidi SH. Bhopal and after. *Am J Ind Med.* 1986;9:215-216.
22. Correspondents, India: long-term effects of MIC. *Lancet.* April 29, 1989;1:952.
23. Blake PG, Ijadi-Maghsoodi S, Kinetics and mechanism of thermal decomposition of MIC. *Int J Chem Kinet.* 1982;14:945-952.
24. Bhattacharya BK, Malhotra RC. Chatterjee AK, Vijayvargiya DP. Inhibition of rat brain cytochrome oxidase activity by pyrolysed products of methyl isocyanate. *Toxicol Lett.* 1985;27:131-134.
25. Acharya VN, Naik SR, Pottis AV, et al. Sequential study of thiocyanate levels in Bhopal victims following methyl isocyanate gas leakage. *J Postgrad Med.* 1986;32:192-194.
26. Sainani GS, Joshi VR, Mehta PJ, Abraham P. Bhopal tragedy: a year later. *J Assoc Physicians India.* 1988;33:755-756.
27. Salmon AG. Bright red blood of Bhopal victims: cyanide or MIC? *Br J Ind Med.* 1988;43:502-504.
28. Anderson N. Long-term effects of methyl isocyanate. *Lancet.* 1989;1:1259.
29. Nemery B, Sparrow S, Dinsdale D. Methyl isocyanate: thiosulphate does not protect. *Lancet.* 1985;2:1245-1246.
30. Hayes WJ Jr, ed. *Pesticides Studied in Man.* Baltimore, Md: Williams & Wilkins; 1982:123-125.
31. Hamilton A, Hardy HL. Cyanides. In: *Industrial Toxicology.* Acton, Mass: Publishing Sciences Group Inc; 1974:221-228.
32. Bucher JR, Gupta BN, Adkins B Jr, et al. Toxicity of inhaled methyl isocyanate in Fischer 344 rats and B6C3F1 mice. I. *Environ Health Perspect.* 1987;72:53-61.
33. Fowler EH, Dodd DE. Toxicity of inhaled methyl isocyanate in rats and guinea pigs. II. Comparative effects of short-term, high-concentration exposure to methyl isocyanate. V: morphologic changes in rat and guinea pig lungs. *Environ Health Perspect.* 1987;72:39-44.
34. Dodd DE, Frank FR, Fowler EH, Trochimowicz HJ, Milton RM. Biological effects of short-term, high-concentration exposure to methyl isocyanate. I. Range-finding study objectives and inhalation exposure design. *Environ Health Perspect.* 1987;72:13-19.
35. Fowler EH, Dodd DE. Respiratory tract

...ngers in guinea pigs, rats, and mice following a ...gle six-hour exposure to methyl isocyanate va... Environ Health Perspect. 1987;72:109-116.
...r, Misra NP, Pathak R, Gaur KJBS, et al. Clinical ...dies of gas leak victims in acute phase after Bho... episode. Indian J Med Res. 1987;86(suppl):11-...
...r, Sharma PN, Gaur KJBS. Radiological spec... of lung changes in gas exposed victims. Indi... J Med Res. 1987;86(suppl):39-44.
...h, Kamat SR, Mahasur AA, Tiwari AKB. Early ...servation on pulmonary changes and clinical mor...lity due to the isocyanate gas leak at Bhopal. J Postgrad Med. 1988;34:63-72.
...h, Kamat SR, Patel MH, Kolhatkar VP, Dave ..., Mahashur AA. Sequential respiratory ...anges in those exposed to the gas leak at Bhopal. ...dian J Med Res. 1987;86(suppl):29-38.
...h, Patel MH, Kolhatkar VP, Potdar VP, Shekha... et Kl., Shah HN, Kamat SR. Methyl isocyanate ...rvivors of Bhopal: sequential flow volume loop ...anges observed in eighteen months' follow-up ...dy. Indian J Med Res. 1987;72:59-65.
...h, Gupta BN, Rastogi SK, Chandra H, et al. Ef... of exposure to toxic gas on the population of ...hopal, I: epidemiological, clinical, radiological and ...ulmonary studies. Indian J Exp Biol. 1988;26:149-...
...t, Rastogi SK, Gupta BN, Husain T, Kumar A, ...handra S, Ray PK. Effects of exposure to toxic ...as on the population of Bhopal, II: respiratory ...acrimetry. Indian J Exp Biol. 1988;26:161-164.
...t, Nair J, Acharya VN, Bhalerao RA, et al. ...edical survey of methyl isocyanate gas affected ...opulation of Bhopal: part II. J Postgrad Med. ...86;32:155-191.
...t, Pant SC, Srivastava RK, Vijayaraghavan R. ...ucomorphologic changes induced by methyl iso...yanate in lungs of rats and rabbits. Bull Environ ...ontam Toxicol. 1987;38:876-883.
...h, Amber L. Methyl isocyanate tests: new evi...ence of lasting lung damage. Chem Eng News. ...arch 2, 1988:6-4. News of the Week. Editorials.
...h, Dutta KK, Gupta GSD, Mishra A, Joshi A, ...asan GS, Ray PK. Inhalation toxicity studies of ...ethyl isocyanate (MIC) in rats: part I—pulmo...ary pathology and genotoxicity evaluation. Indi... J Exp Biol. 1988;26:177-182.
...h, Alarie Y, Ferguson JS, Stock MF, Weyel DA, ...ecker M. Sensory and pulmonary irritation of ...ethyl isocyanate in mice and pulmonary irritation ...d possible cyanide-like effects of methyl isocya... te in guinea pigs. Environ Health Perspect. ...87;72:159-167.
...h, Nemery B, Dinsdale D, Sparrow S. Toxicity of ...ethyl isocyanate in experimental animals. ...ull Eur Physiopathol Resp. July-August ...86;22:315-322.
...h, Boorman GA, Uraih LC, Gupta BN, Bucher ...L. Two-hour methyl isocyanate inhalation and 90-...y recovery study in B6C3F1 mice. Environ ...ealth Perspect. 1987;72:63-69.
...h, Boorman GA, Brown R, Gupta BN. Pathologic ...anges following acute methyl isocyanate inhala... and recovery in B6C3F1 mice. Toxicol Appl ...harmacol. 1987;87:445-456.
...h, Uraih LC, Talley FA, Mitsumori K, Gupta BN, ...ucher JE, Boorman GA. Ultrastructural changes ...the nasal mucosa of Fisher 344 rats and B6C3F1 ...ice following an acute exposure to methyl ...socyanate. Environ Health Perspect. 1987;72:77-88.
...h, Bucher JE, Wiester MJ, Costa DL, Watkinson ...H, Weber MF. Cardiopulmonary effects in awake ...e four and six months after exposure to methyl ...socyanate. Environ Health Perspect. 1987;72:...
...h, Srivastava RK, Vijayaraghavan R, Kumar P, ...ta S. Effects of inhaled sublethal concentration of ...ethyl isocyanate on lung mechanics in rats. Indi... J Exp Biol. 1987;25:535-538.
...h, Nemery B, Dinsdale D, Sparrow S. Ultrastruc...al changes in the respiratory tract of rats follow... methyl isocyanate inhalation. Arch Toxicol. ...86;59:260.
...h, Amerbauer N, Kerr Muir M, Mehra V, Salmon AG. Exposure and response to methyl isocyanate: results of community-based survey in Bhopal. Br J Ind Med. 1988;45:469-475.
56. Dwivedi PC, Raizada JK, Saini VK, et al. Ocular lesions following methyl isocyanate contamination: the Bhopal experience. Arch Ophthalmol. 1985;103:1627.
57. Maskati QB. Ophthalmic survey of Bhopal victims 104 days after the tragedy. J Postgrad Med. 1986;32:199-202.
58. Raizada JK, Dwivedi PC. Chronic ocular lesions in Bhopal gas tragedy. Indian J Ophthalmol. 1987;35:453-545.
59. Khurrum MA, Ahmad SH. Long-term follow-up of ocular lesion of methyl isocyanate gas disaster in Bhopal. Indian J Ophthalmol. 1987;35:136-137.
60. Pozzani UC, Kinkead ER. Animal and human response to methyl isocyanate. Presented at the Annual Meeting of the American Industrial Hygiene Association; May 16-20, 1966; Pittsburgh, Pa.
61. Gassert T, Mackenzie C, Muir MK, Anderson N, Salomon AG. Long-term pathology of lung, eye, and other organs following acute exposure of rats to methyl isocyanate. Lancet. 1986;2:1403.
62. Rye WA. Human response to isocyanate exposure. J Occup Med. 1973;15:306-307.
63. Harding JJ, Rixon RC. Lens opacities induced in rat lenses by methyl isocyanate. Lancet. 1985;1:762.
64. Gupta BN, Stefanski SA, Bucher JR, Hall LB. Effect of methyl isocyanate (MIC) gas on the eyes of Fischer 344 rats. Environ Health Perspect. 1987;72:105-108.
65. Varma DR. Epidemiological and experimental studies on the effects of methyl isocyanate on the course of pregnancy. Environ Health Perspect. 1987;72:153-157.
66. Shilotri NP, Raval MV, Hinduja IN. Gynecological and obstetrical survey of Bhopal women following exposure to methyl isocyanate. J Postgrad Med. 1986;32:203-205.
67. Deo MG, Gangal S, Bhisey AN. Immunological, mutagenic, and cytogenetic investigations in gas-exposed population of Bhopal. Indian J Med Res. 1987;86(suppl):63-76.
68. Kanhere S, Darbari BS, Shrivastava AK. Morphological study of placentae of expectant mothers exposed to gas leak at Bhopal. Indian J Med Res. 1987;86(suppl):77-82.
69. Naik SR, Acharya VN, Bhalerao RA, et al. Medical survey of methyl isocyanate gas affected population of Bhopal. J Postgrad Med. 1986;32:175-184.
70. Taylor ES. On maternal-fetal transfer of thyroxine in congenital hypothyroidism due to a total organification defect or thyroid agenesis. Obstet Gynecol Surv. 1990;45:115-116.
71. Goswami HK. Cytogenetic effects of methyl isocyanate exposure in Bhopal. Hum Genet. 1986;74:81-84.
72. Varma DR, Ferguson JS, Alarie Y. Reproductive toxicity of methyl isocyanate in mice. J Toxicol Environ Health. 1987;21:265-275.
73. Schwetz BA, Adkin B Jr, Harris M, Moorman M, Sloane R. Methyl isocyanate: reproductive and developmental toxicology studies in Swiss mice. Environ Health Perspect. 1987;72:149-152.
74. Bhattacharya BK, Sharma SK, Jaiswal DK. In vivo binding of [1-^{14}C] methyl isocyanate to various tissue proteins. Biochem Pharmacol. 1988;37:2489-2493.
75. Ferguson JS, Kennedy AL, Stock MF, Brown WE, Alarie Y. Uptake and distribution of ^{14}C during and following exposure to (^{14}C) methyl isocyanate. Toxicol Appl Pharmacol. 1988;94:104-117.
76. Mason JM, Zeiger E, Haworth S, Ivett J, Valencia R. Genotoxicity studies of methyl isocyanate in *Salmonella*, *Drosophila*, and cultured Chinese hamster ovary cells. Environ Mutagen. 1987;9:19-28.
77. Kligerman AD, Campbell JA, Erexson GL, Allen JW, Shelby MD. Sister chromatid exchange analysis in lung and peripheral blood lymphocytes of mice exposed to methyl isocyanate by inhalation. Environ Mutagen. 1987;9:29-36.
78. Tice RR, Luke CA, Shelby MD. Methyl isocyanate: an evaluation of in vivo cytogenetic activity. Environ Mutagen. 1987;9:37-58.
79. Shelby MD, Allen JW, Caspary WJ, et al. Results of in vitro and in vivo genetic toxicity tests on methyl isocyanate. Environ Health Perspect. 1987;72:183-187.
80. Sutcliffe M. An eyewitness in Bhopal. BMJ. 1985;290:1883-1884.
81. Bharucha EP, Bharucha NE. Neurological manifestations among those exposed to toxic gas at Bhopal. Indian J Med Res. 1987;86 (suppl):59-62.
82. Irani SF, Mahashur AA. A survey of Bhopal children affected by methyl isocyanate gas. J Postgrad Med. 1986;32:195-198.
83. Saxena AK, Singh KP, Nagle SL, et al. Effect of exposure to toxic gas on the population of Bhopal, IV: immunological and chromosomal studies. Indian J Exp Biol. 1988;26:173-176.
84. Karol MH, Taskar S, Gangal S, Rubanoff BF, Kamat SR. The antibody response to methyl isocyanate. Environ Health Perspect. 1987;72:169-175.
85. Karol MH, Kamat SR. The antibody response to methyl isocyanate: experimental and clinical findings. Bull Eur Physiopathol. 1988;23:591-597.
86. Dwivedi PD, Mishra A, Gupta GSD, Dutta KK, Das SN, Ray PK. Inhalation toxicity studies of methyl isocyanate (MIC) in rats, IV: immunologic response of rats one week after exposure. Indian J Exp Biol. 1988;26:191-194.
87. Bucher AN, Bucher JR, Germoler DR, Silver MT, Vore SJ, Luster MI. Immunological studies on mice exposed subacutely to methyl isocyanate. Environ Health Perspect. 1987;72:139-141.
88. Hong HL, Bucher JR, Ganjee J, Boorman GA. Myelotoxicity induced in female B6C3F1 mice by inhalation of methyl isocyanate. Environ Health Perspect. 1987;72:143-148.
89. Srivastava RC, Gupta BN, Athar M, et al. Effects of exposure to toxic gas on the population of Bhopal, III: assessment to toxic manifestations in humans. Indian J Exp Biol. 1988;26:165-172.
90. Troup CM, Dodd DE, Fowler EH, Frank FR. Biological effects of short-term, high-concentration exposure to methyl isocyanate, II: blood chemistry and hematologic evaluation. Environ Health Perspect. 1987;72:21-28.
91. Bucher JR, Gupta BN, Thompson M, Adkins B Jr, Schwetz BA. The toxicity of inhaled methyl isocyanate in F344/N rats and B6C3F1 mice, II: repeated exposure and recovery studies. Environ Health Perspect. 1987;72:133-138.
92. Misra UK, Nag D, Nath P, Khan WA, Gupta BN, Ray PK. A clinical study of toxic gas poisoning in Bhopal, India. Indian J Exp Biol. 1988;26:201-204.
93. Anderson D, Goyle S, Phillips BJ, Tee A, Beech L, Butler WH. Effects of methyl isocyanate on rat muscle cells in culture. Br J Ind Med. 1988;45:269-274.
94. Sethi H, Dayal R, Singh RK. Acute and subacute toxicity of inhaled methyl isocyanate in Charles Foster rats. Ecotoxicol Environ Safety. 1988;16:68-74.
95. Sethi BB, Sharma M, Trivedi JK, Singh H. Psychiatric morbidity in patients attending clinics in gas-affected areas in Bhopal. Indian J Med Res. 1987;86(suppl):45-50.
96. Kinzie JD. Post-traumatic stress disorders. In: Kaplan HI, Sadock BJ, eds. *Comprehensive Textbook of Psychiatry/I*. 5th ed. Baltimore, Md: Williams & Wilkins; 1989.
97. Raphael B. After the horror. BMJ. 1988;296:1142-1144.
98. Titchener JL, Kapp FT. Family and character change at Buffalo Creek. Am J Psychiatry. 1976;133:295-299.
99. McFarlane AC. Long-term psychiatric morbidity after a natural disaster. Med J Aust. 1986;145:561-563.
100. Brenner MH. *Mental Illness and the Economy*. Cambridge, Mass: Harvard University Press; 1973.
101. Cassel CK, Leaning J. Chernobyl: learning from experience. N Engl J Med. 1988;321:254-255.

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Effect of Exposure to Toxic Gas on the Population of Bhopal: Part II—Respiratory Impairment

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The respiratory status of 783 cases, of either sex exposed to the toxic gas was investigated. Lung spirometry revealed 39% prevalence of ventilatory impairment in the affected population. The female population exhibited a higher prevalence (44.1%) of pulmonary abnormalities in contrast with the male population (33.9%). The toxic gas inhalation produced three patterns of respiratory disorders as indicated by simple spirometric tests. Bronchial obstruction was recorded in 3.7% male and 3.6% female population respectively, while restrictive pulmonary defect was noticed in 13.4% cases. The combined obstructive-cum-restrictive ventilatory disorder was observed in 22.2% population. The possible mechanisms involved in the pulmonary dysfunction induced by toxic gas are discussed.

Exposure to the toxic gas caused various pulmonary abnormalities resulting in a number of respiratory symptoms, viz. cough, dyspnoea, chest pain, etc. in the population of Bhopal. No epidemiological study is so far reported in the literature involving exposure to the human population to the toxic gas. Some studies had been conducted with isocyanates[1-5]. Cumulative effect on lung function of the persons exposed to toluene diisocyanate (TDI) has been reported[6-11]. Significant decrease occurred in FVC, FEV_1, PEFR, $FEF_{50\%}$ of VC on account of TDI exposure[12-15]. A recent study[16] has suggested that infiltrated lung diseases with pulmonary function abnormalities may occur in workers exposed to TDI.

Materials and Methods

Spirometry was performed in 783 cases (371 males and 412 females) examined in the respiratory screening programme during February/March 1985 at Bhopal. The subjects who were unable to perform the spirometric manoeuvres were excluded from the study. Among the 371 male cases, there were 179 (48.7%), non-smokers and 172 (51.7%) smokers while in the female population there were no smokers.

The spirometric lung functions were recorded using calibrated Vitalograph Spirometer in standing position with nose clip on. The spirometer was regularly checked for calibration of volume and speed statically and dynamically by air displacement method at ATPS (normal barometric pressure of 760 mm Hg, air saturated with water vapour). The following parameters were recorded:

1, Vital capacity (VC); 2, Forced vital capacity (FVC); 3, Forced expiratory volume in 0.75 sec ($FEV_{0.75}$); 4, Forced expiratory volume in 1 sec (FEV_1); 5, FEV_1/FVC ratio; 6, Indirect maximum breathing capacity (IMBC); 7, Air velocity index (AVI).

The population exposed to toxic gas was asked to perform in the manner demonstrated to them, at least three VC and FVC manoeuvres. The values from the largest of the three reproducible curves were taken into consideration and expressed at body temperature and ambient pressure saturated with water vapour (BTPS). IMBC was calculated from $FEV_{0.75}$ by multiplying it by 40. AVI was calculated from the predicted VC and IMBC values as follows:

$$AVI = \frac{\% \text{ of normal IMBC}}{\% \text{ of normal VC}}$$

Normal value of AVI was considered to be 1.0. Higher values indicated restrictive pattern and lower values obstructive pattern of respiratory impairment. The FEV_1/FVC percentage between 70 and 80 was considered normal and values less than 70% indicated central airway obstruction.

Peak expiratory flow rate (PEFR) was recorded with Peak Flow Meter (Wright Standard Model, UK) and the highest of the three values attained was recorded.

The predicted normal values for the various lung function tests were derived from the regression equations laid down for healthy north Indian population[17]. For the female population, the predicted values were calculated from the predictiion equations laid down for healthy women[18]. The pulmonary impairment was classified according to Rastogi *et al.*[17] as follows:

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1 Restrictive	VC or FVC less than 80% of the predicted values
2 Obstructive	FEV_1/FVC ratio less than 70%
3 Combined or mixed	FVC less than 80% of the predicted value and FEV_1/FVC ratio less than 70%

Further classification of pulmonary impairment was done as follows:

1 Mild respiratory impairment, between 61 and 80%.

2 Moderate respiratory impairment, between 40 and 60%.

3 Severe respiratory impairment, less than 40%.

Statistical procedure—The lung function test results were analysed by applying student's paired/unpaired t test in the study population. Chi square test was used for the determination of the significance of the prevalence of various categories of respiratory impairment in the male and female population exposed to the toxic gas.

Results

The physical characteristics of the male and female population studied for lung function tests are detailed in Table 1. Sexwise no significant differences were observed in their mean ages. In the male population, the age and height differences between smokers and non-smokers were noticed ($P<.001$ and $P<.05$ respectively). The mean values of anthropometric measurements of the population suffering from respiratory impairment are shown in Table 2. In the male population, the mean age of the cases with restrictive lung disorder was significantly ($P<.05$) less than the mean values recorded in obstructive and mixed lung abnormality cases. While in the female population, the mixed respiratory impairment cases were older than the other cases.

Data in Table 3 show no significant difference in the prevalence of pulmonary abnormality between the smokers 37.5%) and non-smokers (31.1%).

Prevalence of obstructive respiratory impairment in the study population is detailed in Table 4. Sexwise no significant difference was observed in the prevalence of airway obstruction in the lungs. Smoking exhibited a significant influence on the prevalence of bronchial obstruction ($P<0.05$). Mild airway obstruction was noted in 3% of the population examined. Mild bronchial obstruction was equally prevalent in the male and female population. Smokers exhibited a significantly higher prevalence (5.7%) in comparison with non-smokers ($P<0.05$). Sexwise, it was noted that both male and female populations were equally affected by the severe broncho-spasm.

Prevalence of restrictive respiratory impairment is presented in Table 5. 13.4% population suffered from lung restriction. However, sex and smoking habits had no effect on it. Mild restrictive lung impairment was prevalent in 12.3% cases. The results showed a significantly higher prevalence of restriction in the male population ($P<0.05$). Moderate

Table 1 Physical Characteristics of Population Exposed to the Toxic Gas

Sex and smoking habits	N	Age (yrs) X ± SE (X)	Height (cms) X ± SE (X)	Weight (kgs) X ± SE(X)	Distance from UCIL X ± SE(X)
Male Population	371	33.8 ± 0.60	162.5 ± 0.35	54.5 ± 0.5	2.5 ± 0.06
Non-smokers	179	30.8 ± 0.82	163.5 ± 0.50	55.2 ± 0.7	2.6 ± 0.09
Smokers	192	36.7 ± 0.81	161.7 ± 0.49	53.9 ± 0.6	2.4 ± 0.07
Female population	412	32.9 ± 0.55	149.3 ± 0.29	48.3 ± 0.5	2.7 ± 0.05

Table 2 Physical Characteristics of the Population showing Respiratory Impairment

Physical characteristics	Males (N = 126)			Female (N = 182)		
	Obstructive (N=14) X ± SE(X)	Restrictive (N=55) X ± SE(X)	Mixed (N=57) X ± SE(X)	Obstructive (N=15) X ± SE(X)	Restrictive (N=50) X ± SE (X)	Mixed (N=117) X ± SE(X)
Age (years)	40.4 ± 3.3	34.6 ± 1.7	39.2 ± 1.7	31.0 ± 2.6	36.4 ± 1.6	39.9 ± 1.0
Height (Cms)	161.2 ± 1.7	163.7 ± 0.9	161.9 ± 0.8	149.7 ± 1.3	150.1 ± 0.8	147.6 ± 0.6
Weight (kgs)	52.1 ± 2.5	54.1 ± 1.5	54.2 ± 1.5	47.4 ± 2.1	50.6 ± 1.5	48.3 ± 0.9
Distance from UCIL Factory (Kms)	1.9 ± 0.5	2.4 ± 0.14	2.1 ± 0.15	2.8 ± 0.3	2.7 ± 0.1	2.6 ± 0.1

and severe lung restriction was recorded in 0.3% and 0.6% population respectively.

Mixed respiratory impairment was recorded in 22.2% of the population studied (Table 6). Its prevalence was significantly higher in the female population ($P<0.05$). Smoking did not make any difference in the prevalence of mixed respiratory impairment. The mild category of mixed respiratory impairment was observed in 14.9% cases. Sexwise, it was the female population which suffered more ($P<0.01$). 4.8% and 2.4% of the study population suffered from moderate and severe types of mixed respiratory impairment (Table 6). Sexwise moderate category was more prevalent in the female cases ($P<0.01$), while severe mixed impairment was equally present in both sexes.

Discussion

Various types of pulmonary impairment were recorded in the population exposed to the toxic gas. It was observed that the study population suffered from three types of impairment, viz. obstructive, restrictive and combined. The majority of the population suffered from the mixed type of respiratory

Table 3 Prevalence of Respiratory Impairment in Population Classified According to Sex and Smoking habits.

Sex and smoking habits	Total	Persons with normal spirometry		Respiratory Impairment						Total impairment	
				Persons with obstructive impairment*		Persons with restrictive impairment**		Persons with mixed type of impairment***			
		N	%	N	%	N	%	N	%	N	%
Male population	371	245	66.0	14	3.7	55	14.8	57	15.3	126	33.9
Non smokers	179	125	69.8	3	1.6	26	14.5	25	13.9	54	30.1
Smokers	192	120	62.5	11	5.7	29	15.1	32	16.6	72	37.5
Female population	412	230	55.8	15	3.6	50	12.1	117	28.3	182	44.1
Total	783	475	60.6	29	3.7	105	13.4	174	22.2	30.8	39.3

* FEV_1/FVC % less than 70%
FVC less than 80% of the predicted values
FVC less than 80% of the predicted value and FEV_1/FVC ratio less than 70%

Table 4 Prevalence of Obstructive Pulmonary Impairment in the Population Exposed to the Toxic Gas

Sex and smoking habits	Total	Persons with normal spirometry		Obstructive pulmonary impairment						Total Impairment	
				Mild		Moderate		Severe			
		N	%	N	%	N	%	N	%	N	%
Male population	371	245	66.0	13	3.5	-	-	1	0.2	14	3.7
Non smokers	179	125	69.8	2	1.1	-	-	1	0.5	14	1.6
Smokers	192	120	62.5	11	5.7	-	-	-	-	11	5.7
Female population	412	230	55.8	11	2.6	3	0.7	1	0.2	15	3.6
Total	783	475	60.6	24	3.0	3	0.7	2	0.2	29	3.7

Table 5 Prevalence of Restrictive Respiratory Impairment in Population Exposed to the toxic Gas

Sex and smoking habits	Total	Persons with normal spirometry		Restrictive Respiratory Impairment						Total Impairment	
				Mild		Moderate		Severe			
		N	%	N	%	N	%	N	%	N	%
Male population	371	245	66.0	55	14.8	-	-	-	-	55	14.8
Non smokers	179	125	69.8	26	14.5	-	-	-	-	26	14.5
Smokers	192	120	62.5	29	15.1		-	-	-	29	15.1
Female population	412	230	55.8	42	10.1	3	0.7	5	1.2	50	12.1
Total	783	475	60.6	97	12.3	3	0.3	5	0.6	105	13.4

Table 6 Prevalence of Mixed Respiratory Impairment in Population Exposed to the Toxic Gas

Sex and smoking habits	Total	Persons with normal spirometry		Mixed Respiratory Impairment						Total Impairment	
				Mild		Moderate		Severe			
		N	%	N	%	N	%	N	%	N	%
Male population	371	245	66.0	42	11.3	6	1.6	9	2.4	57	15.4
Non smokers	179	125	69.8	16	8.0	4	2.2	5	2.7	25	13
Smokers	192	120	62.5	26	13.5	2	1.0	4	2.0	32	
Female population	412	230	55.8	75	18.2	32	7.7	10	2.4	117	28.4
Total	783	375	60.6	117	14.9	38	4.8	19	2.4	174	22

impairment followed by the restrictive type. The results indicated that toxic gas inhalation caused a mild form of pulmonary impairment.

Spirometry studies revealed that majority of the pulmonary functions were subnormal. The spirometric parameters, i.e. VC, FVC, $FEV_{0.75}$, FEV_1, FEV_1/FVC ratio and IMBC revealed decreased mean observed values in contrast with their respective reference values.

Thus the present study provides ample evidence of pulmonary hazard in the population exposed to the toxic gas as indicated by the decreased respiratory function values and high prevalence of respiratory impairment. The physiological mechanisms responsible for causing lung impairment induced by toxic gas inhalation are not yet clear. Some investigators[19] believe that isocyanates have an acute irritating effect on the respiratory tract resulting in the bronchospastic reactions, while others[20], suggest that isocyanate induced respiratory disease may be immunlogically mediated. A third hypothess[21] is that the isocyanates are pharmacologically active and on inhalation may react with components of the lung tissue leading to changes or to the inhibition of biological functions. Two types of pulmonary responses to isocyanate exposure have been suggested[22]. One is the direct irritant response due to triggering of normal protective mechanism of the upper respiratory tract and the second is host generated and truly allergic.

References

1 Brugsch H G & Elkins H B, *N Eng J Med*, 268 (1963)
2 Zapp J A, *Arch Industr Health*, 15 (1957) 324.
3 Stokinger H E & Scheel L D, *J Occup Med*, 15 (197
4 Dodson V M, *J Occup Med*, 13 (1971) 238.
5 Munn A, *Ann Occup Hyg*, 8 (1965) 163.
6 Gandevia B, *Br J Industr. Med*, 20 (1963) 204.
7 Peters J M, *Proc Royal Soc Med*, 63 (1970) 372.
8 Paggiaro P L, Loi A M O, Rossi B, Ferrante F P, Ro & Baschieri L, *Clin Alergy*, 14 (1984) 463.
9 Wegmann D H, Peters J M, Pagnotto L & Fine L J, *dustr Med*, 34 (1977) 196.
10 Adams W G F, *Br J Industr Med*, 32 (1975) 72.
11 Diem J, Jones R N & Hendrick D J, *Am Rev Respir Di* (1982) 420.
12 Maxon F G, *Arch Environ Health*, 8 (1964) 755.
13 Peters J M, Murphy R L H & Pagnotto L D & Van G F, *Arch Environ Health*, 16 (1968) 642.
14 Wegmen D H, *J Occup Med*, 16 (1974) 258.
15 Mekerrow C B, Davies H J & Parry Jones A, *Proc Roy Med*, 63 (1970) 376.
16 Charles J, Bernstein A, Jones B, Jones D J, Edwa Seal R R M E & Seaton A, *Thorax*, 31 (1976) 12
17 Rastogi S K, Mathur N & Clerk S H, *Indian J Chest Di lied Sci*, 25 (1983) 186.
18 Bhattacharya A K, Banerjee S, *Indian J Med Res*, 54 62.
19 Baur X, Romme H H & Fruhmann G, *Respiration*, 38 289.
20 Karol M H, Loset H H & Alaire Y C, *Am Ind Hyg Assoc* (1978) 454.
21 Butcher B T, Karr R M & O'Neil C L, *J Allergy Clin Im* 64 (1979) 146.
22 Rye W A, *J Occup Med*, 15 (1973) 306.

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Effect of Exposure to Toxic Gas on the Population of Bhopal: Part III—Assessment of Toxic Manifestations in Humans—Haematological and Biochemical Studies

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Various haematological parameters like total RBC count, total and differential leucocyte count, haemoglobin content, packed cell volume, erythrocyte sedimentation rate and various biochemical parameters like blood urea, glutathione (GSH), glutamic oxaloacetic transaminase (GOT) glutamic pyruvic transaminase (GPT), ceruloplasmin, total bilirubin, glutamic transpeptidase (γ-GTP) in blood, creatinine and GPT in urine were studied in the population exposed to the toxic gas in Bhopal 2½ months after the gas disaster. In a majority of subjects studied (67.1%) haemoglobin values were higher than 12 g/100 ml and the total RBC count was on the higher side in 77.8% subjects. Total leucocyte count did not reveal any abnormality. Increased polymorphonuclear leucocytosis was found in 11.9% subjects. A lymphocyte count of more than 40% was found in 31.6% cases. An eosinophil count of more than 6% was observed in 25.7% subjects. The ESR was found elevated in 36.4% subjects of the overall population. Blood glutathione was found significantly lowered in about 40% of the population examined. Serum ceruloplasmin rose to about 2 times of the highest reported value in the controls in a significant number of exposed subjects. Creatinine content in urine was also raised in these subjects. Other biochemical parameters were found to be within the normal range.

To study the effect of the toxic gas on the haemopoietic system of the exposed population various haematological parameters like total RBC count, total and differential leucocyte count, haemoglobin percentage, packed cell volume, erythrocyte sedimentation rate and blood urea were studied during the phase I (11 Feb to 3 March 1985) and phase II (15 March to 10 April 1985) studies at Bhopal. Despite a few papers describing clinical and experimental findings there is still no agreement on the biological mechanism responsible for the gross effects on the humans. Various biochemical parameters in blood serum and urine in male and female populations exposed to the toxic gas were studied to find the changes that occurred in these parameters and whether the biochemical changes can be interpreted to postulate a theory of the possible mechanism of action of the toxic gas in the human system.

Materials and Methods

Out of 1109 persons exposed to the toxic gas 695 who volunteered themselves for haematological and biochemical studies were examined during phase I and II studies. These subjects comprised persons of different age groups including children, of either sex, belonging to different religions and having had varying degrees of exposure to toxic gas on account of their living at different distances from the Union Carbide Factory at the time of gas leakage.

Blood from medial cubital vein was obtained with the help of sterilized syringes and various haematological parameters were studied, using standard methods in haematology. Haemoglobin value was measured by Alkaline Haematin Method[1], RBC count and total and differential leucocyte counts were done according to the methods described by Dacie and Lewis[1] and packed cell volume and erythrocyte sedimentation rate were studied by Wintrobe method. Blood urea was estimated by Nesslers method as described by King and Wootton[2].

The following biochemical parameters in blood and urine were estimated.

Blood: Reduced glutathione (GSH)

Serum: Glutamic oxaloacetic transaminase (GOT EC 2.6.1.1), glutamic pyruvic transaminase (GPT EC 2.6.1.2), ceruloplasmin, total bilirubin, and γ-glutamyl transpeptidase (γ-GTP EC 2.3.2.2).

Urine: Creatinine, glutamic pyruvic transaminase.

The biochemical assay of blood or urine samples were performed using standard methods.

GSH—Ellman[3] modified by Jollow[4]; CP—Curzon and Vallet[5]; GOT—Reitman and Frankel[6]; GPT—Reitman and Frankel[6]; γ-GTP—Orlowski and Szewezuk[7]; Bilirubin—Wootton[8]; Creatinine—Wootton[8].

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Results and Discussion

Estimation of haemoglobin—A majority of the population (67.1%) had haemoglobin values of higher than 12 mg/100 ml, while one-third of the population (32.5%) had values between 8 and 12 mg/100 ml. The percentage of population having haemoglobin level of less than 8 g/100 ml was 0.4.

When haemoglobin values were correlated with different age groups, no significant pattern was observed. The percentage of population having more than 12 g/100 ml haemoglobin ranged from 61.9% in the 26-35 years age group to 75.3 in the 16-25 years age group (Table 1).

There was a direct relationship between haemoglobin level in the individuals residing at different distances from the Union Carbide Factory. The percentage of cases having haemoglobin level of more than 12 g/100 ml was 65.5, 67.3 and 82.2 in persons residing within 2, 2-4 and 4-6 km respectively.

Out of the total population residing within 2 km from the factory 65.5% revealed a haemoglobin level of over 12 mg/100 ml and a similar trend was observed in 80% of the population living beyond 4 km (Table 2). A haemoglobin level of more than 12 g/100 ml was recorded in a higher percentage of male population than in the female in different age groups. However, the difference was statistically insignificant.

Red blood cell count—RBC count was more than 4 million/mm^3 in a majority of the population (77.8%). Agewise distribution pattern of pers having total RBC count of more than 4 million/m was 87.1, 82.9, 77.1, 74.3, 70.1 and 72.0% respe ively in subjects in age groups of less than 15, 16-2 26-35, 36-45 and more than 56 years.

Total RBC count of less than 3 million/mm^3 observed in a very small percentage of populatio The percentage of population in different a groups which had an RBC count between 3 and million/mm^3 was between 12.9 and 28.0. A ver small percentage of population had a total RBC count of less than 3 million/mm^3. No significant effect of the distance from UCIL factory was noted on the red blood cell count (Table 3).

The increased haemoglobin and high RBC count suggest the effect of the toxic gas on the bone marrow leading to polycythenin type of disorder in haemopoetic system. Such a disorder is also caused by various chemicals/drugs which are known to reduce the oxygen capacity of blood. Inspite of the high RBC count the normal value of PCV probably suggested the shrinkage of RBC.

Total leucocyte count—TLC count was high (more than 10,000/mm^3) in 11.6% of overall population. It was almost similar in males and females being 10.3 and 12.6% respectively. The percentage of population having TLC of less than 5,000 was 6.4. Low leucocyte count (less than 5,000) was more in female (8.2%) than males (4.6%). The TLC (more than 10,000) has similar distribution in each population group residing between 2, 2-4, 4-6 km and more than 6 km from the factory (Table 4). Leucocytosis

Table 1—Haemoglobin Values in Relation to Age and Sex

Age group (years)	Haemoglobin (gm %)									Total		
	<8			8-12			12+					
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
≤ 15	-	-	-	3	6	9	8	14	22	11	20	31
16-25	-	-	-	(27.3)	(30.0)	29.0)	(72.7)	(70.0)	(71.)			
16 - 25	-	-	-	17	25	42	54	74	128	71	99	170
				(29.9)	(25.2)	(24.7)	(76.1)	(74.8)	(75.3)			
26 - 35	-	1	1	20	46	66	55	54	109	75	101	176
		(0.9)	(0.6)	(26.7)	(45.6)	(37.5)	(73.3)	(53.5)	(61.9)			
36 - 45	-	-	-	24	29	53	50	37	87	74	66	140
				(13.5)	(43.9)	(37.8)	(67.5)	(56.1)	(62.2)			
46 - 55	-	1	1	8	9	17	25	14	39	33	24	57
		(4.2)	(1.7)	(24.2)	(37.5)	(29.8)	(75.8)	(58.3)	(68.5)			
≥ 56	-	-	-	5	3	8	13	4	17	18	7	25
				(27.8)	(42.8)	(32.0)	(72.2)	(57.2)	(68.0)			
Total	-	2	2	77	118	195	205	197	402	282	317	599
		(0.6)	(0.4)	(27.3)	(37.2)	(32.5)	(72.7)	(62.2)	(67.1)			

Figures in parenthesis indicate percentage of population studied.

Table 2—Haemoglobin Values in Relation to Distance from the Factory (sex wise)

Distance from the factory (km)	Haemoglobin (gms %)											
	<8			8–12			>12			Total		
	Male	Female	Total	Male	Female	Total	Male	Female	total	Male	Female	Total
<2	-	1	1	50	74	124	121	117	238	171	192	257
		(0.5)	(0.3)	(29.2)	(38.5)	(34.2)	(70.8)	(61.0)	(65.5)			
2–4	-	1	1	26	37	63	71	61	132	97	99	196
		(1.0)	(0.5)	(26.8)	(37.3)	(32.2)	(73.2)	(61.7)	(67.3)			
4–6	-	-	-	1	4	5	9	14	23	10	18	28
				(10.0)	(22.2)	(17.8)	(90.0)	(77.8)	(82.2)			
>6	-	-	-	-	3	3	4	5	9	4	8	12
					(37.5)	(25.)	(100.0)	(62.2)	(75.0)			
Total	-	2	2	77	118	195	205	197	402	282	317	599
		(0.6)	(0.4)	(27.3)	(37.2)	(34.5)	(72.7)	(62.2)	(67.1)			

Figures in parenthesis indicate percentage of population studied.

Table 3—RBC count According to Distance from the Factory (sex wise)

RBC count millions/ mm³	Distance from the Factory (km)												Total		
	<2			2–4			4–6			>6					
	M	F	Total	M	F	Total	M	F	Total	M.	F	Total	M.	F	Total
<3	1	10	11	2	2	4	-	-	-	-	-	-	3	12	15
	(0.6)	(5.2)	(3.)	(2.1)	(2.)	(2.)							(1.1)	(3.8)	(2.5)
3–4	17	53	70	11	29	40	4	4	4	1	3	4	29	89	118
	(10.0)	(27.6)	(19.3)	(11.3)	(29.3)	(20.4)		(22.2)		(25.0)	(42.8)	(36.4)	(10.3)	(28.2)	(19.2)
>4	153	129	282	84	68	152	10	14	24	3	4	7	250	215	465
	(89.4)	(67.2)	(77.7)	(86.6)	(68.7)	(77.6)	(100.0)	(77.7)	(85.7)	(75.0)	(57.2)	(63.6)	(88.6)	(88.0)	(77.8)
Total	171	192	363	97	99	196	10	18	28	4	7	11	282	316	598

Figures in parenthesis are the percentages from the column totals.

Table 4—Total Leucocyte Count in Relation to Distance from the Factory

Leucocyte	Distance from the Factory (km)												Total		
	<2			2–4			4–6			>6					
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	female	total
<5000	4	14	18	8	7	15	1	4	5	0	1		13	26	39
	(2.3)	(7.3)	(4.9)	(8.2)	(7.1)	(7.7)	(10.0)	(22.2)	(17.8)			(9.1)	(4.6)	(8.2)	(6.5)
5000–10000	148	151	299	81	80	161	7	13	20	4	6	10	240	250	499
	(86.5)	(78.6)	(82.4)	(83.5)	(80.8)	(82.1)	(70.0)	(72.2)	(71.4)			(90.9)	(85.1)	(79.1)	(81.7)
>100000	19	27	46	8	12	20	2	1	3	-	-	0	29	40	69
	(11.1)	(14.1)	(12.6)	(8.2)	(12.1)	(10.2)	(20.0)	(56)	(10.8)			(0.0)	(10.3)	(12.6)	(11.6)
Total	171	192	363	97	99	196	10	18	28	4	7	11	282	316	598

Figures in parenthesis are the percentage of the row totals

was maximum (28.0%) in age group over 56 years. The respective percentage in the age groups less than 15, 16-25, 26-35, 36-45, 46-55 were 6.4, 8.2, 16.6, 8.6 and 8.8 respectively (Table 5).

Differential leucocytes count—All the cases studied were divided into two groups, i.e. those having less than 70% polymorphs and those with more than 70%. Increased polymorphonuclear leucocytosis was found in 71 subjects (11.9%) of the overall population. Similarly the cases were divided in two groups having less than or more than 40% lymphocytes. The number of subjects showing lymphocytosis more than 40% was found to be 188 (31.6%). Eosinophils were found to be more than 6% in 171 subjects (28.7%) (Table 6).

The percentage of cases showing increased polymorphonuclear leucocytosis was almost similar in different age groups, i.e. 16.1, 11.1, 11.4, 14.4 and

Table 5—Age and sexwise Distribution of total Leucocyte count

Age group	Total Leucocyte Count											
	< 5000			5000–10000			>10000			Total		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
≤15	-	-	-	11	18	29	-	2	2	11	20	31
				(100.0)	(90.0)	(93.5)		(10.0)	(6.4)			
16 – 25	1	9	10	61	85	146	9	5	14			
	(1.4)	(10.1)	(5.9)	(85.9)	(5.8)	(85.9)	(12.7)	(5.0)	(8.2)	71	99	170
26 – 35	5	8	13	60	73	133	10	19	29	75	100	175
	(6.7)	(8.0)	(7.4)	(80.0)	(73.0)	(76.0)	(13.3)	(19.0)	(16.6)			
36 – 45	4	6	10	67	51	118	3	9	12	74	66	140
	(5.4)	(9.1)	(7.1)	(90.5)	(77.2)	(84.3)	(4.0)	(13.7)	(8.6)			
46 – 55	3	3	6	27	19	46	3	2	5	33	24	57
	(9.1)	(12.5)	(10.5)	(81.8)	(79.2)	(80.7)	(9.1)	(8.3)	(8.8)			
≥ 56	-	-	-	14	*4	18	4	*3	7	18	7	25
				(77.8)	(72.0)	(72.0)	(22.2)		(28.0)			
Total	13	26	39	240	250	490	29	40	69	282	316	598
	(4.6)	(8.2)	(6.5)	(85.1)	(79.1)	(81.9)	(10.3)	(12.6)	(11.6)			

Figures in parenthesis are the percentages of row totals * percentages were not worked out as denomination was less than 10.

Table 6—Age and Sexwise Distribution of Differential Leucocyte count Values

Age group		Polymorphs		Lymphocytes		Eosinophils	
		< 70	> 70	< 40	> 40	< 6	> 6
≤15	Male	8	3	10	1	7	4
	Female	18	2	14	6	12	8
	Total	26	5	24	7	19	12
16 – 25	Male	64	7	48	23	49	22
	Female	87	12	60	39	70	29
	Total	151	19	108	62	119	51
26 – 35	Male	68	7	51	24	53	22
	Female	87	13	70	30	74	26
	Total	155	20	121	54	127	48
36 – 45	Male	64	10	54	20	59	15
	Female	53	10	47	16	43	20
	Total	20	101	36	102	35	
46 – 55	Male	31	2	24	9	26	7
	Female	22	2	11	13	14	10
	Total	53	4	35	22	40	17
≥ 56	Male	17	1	12	6	11	7
	Female	5	2	6	1	6	1
	Total	22	3	18	7	17	8
	Total	524	71 (11.9%)	407	188(31.8%)	424	171(28.7%)

12.0% in age groups upto 15, 16-25, 26-35, 36-45 and more than 56 years, respectively, except in the age group 46-55 years where only 7.1% subjects showed polymorphonuclear leucocytosis (more than 70%).

Lymphocyte count of more than 40% was found in 22.6% and 26.3% of subjects up to 15 and 36-45 years age groups. High lymphocyte count in age groups 16-25, 26-35, 46-55 and more than 56 years was found to be 36.4, 30.8, 38.6 and 28.0% respectively.

The maximum number of cases showing eosinophil count more than 6% was found in age group up to 15 years (33.8%) as compared 30.0, 26.5, 20.2 and 32.0% in age groups 16-25, 26-35, 36-45 and more than 56 years respectively.

The differential leucocyte count was not found to be significantly affected in groups of population residing at different distances from the factory.

Erythrocyte sedimentation rate—ESR was found elevated (more than 20 mm in females and more than 10 mm in males) in 36.4% of the overall population. It was raised in 39.2 and 33.8% males and females respectively. .The distance from the factory had no significant effect on ESR values in the study population. ESR values were raised in 36.9, 38.2,

Table 7 — ESR values in Relation to Distance (Sex wise)

Distance	Erythrocyte Sedimentation Rate (ESR)					
	Male			Female		
	< 10	> 10	Total	< 20	> 20	Total
< 2	100	71	171	129	63	192
	(58.4)	(41.6)		(67.2)	(32.8)	
2 - 4	60	37	97	61	38	99
	(61.8)	(38.2)		(61.7)	(38.3)	
4 - 6	8	2	10	15	3	18
	(80.0)	(20.0)		(83.3)	(16.7)	
> 6	2	0	2*	4*	3*	7*
Total	170	110	280	209	107	316
	(60.7)	(39.3)		(66.1)	(33.8)	

Figures in parenthesis are percentages of row totals

* Percentages are not worked out where denominator is less than 10

Table 8—Age and Sex wise Distribution of ESR Values

Age group (Years)	ESR mn/1st Hr (Wintrobe)					
	Male			Female		
	< 10	> 10	Total	< 20	> 20	Total
≤ 15	7	4	11	13	7	20
	(63.6)	(36.4)		(65.0)	(36.0)	
16 - 25	45	24	69	74	25	99
	(65.2)	(34.8)		(74.7)	(25.3)	
26 - 35	46	29	75	63	37	100
	(61.3)	(38.7)		(63.0)	(37.0)	
36-45	41	37	74	40	26	66
	(55.4)	(44.6)		60.6	(39.4)	
46 - 55	23	10	33	14	10	
	(69.7)	(30.3)		(58.3)	(41.7)	
> 56	8	10	18	5*	2*	7
	(44.4)	(55.6)				
Total	170	110	280	209	107	316
	(60.7)	(39.3)		(66.1)	(33.9)	

Figures in parenthesis are percentage of row total

* Percentage are not workout where denominator is less than 10

17.8 and 33.3% population residing less than 2, 2-4, 4-6 and more than 6 km from the factory respectively. The age group of more than 56 years had maximum number of persons (48.0%) having elevated ESR. This was followed by 42.1, 37.7, 35.1 and 29.1% prevalence of elevated ESR values in age groups 36-45, 26-35 less than 15, 46-55 and 16-25 years respectively (Table 7 and 8).

Blood urea—The majority of cases with raised blood urea values (more than 40 mg/100 ml) were found in persons in age group 36-45 years followed by age group 16-25 (25.0%) and 26-35 (21.2%). The overall prevalence of raised blood urea was found to be 20.5% in males and 12.7% in f with an average of 16.3% subjects showing el values (Table 9).

The distance from the factory did not have significant effect on the pattern of abnormal b urea values (Table 10).

Reduced glutathione—GSH level was signific reduced in 30% of the exposed population. A pressed level of GSH in RBC may be due to the hibitory effect of the toxic gas on the biosynth sis of various SH dependent and other related e zymes of glutathione synthetic pathway. Prolonge decrease in the GSH content even after 2½ month

Table 9—Age and Sex wise Blood Urea Levels

Age group (years)	Blood urea (mg %) < 40 Male	Female	Total	> 40 Male	Female	Total	Total Male	Female	Total
≤ 15	9	20	29	0	1	1	9	21	30
	(100.0)	(95.2)	(6.7)	(0.0)	(4.8)	(3.3)			
16 25	50	78	128	11	9	20	61	87	148
	(81.9)	(89.9)	(86.6)	(18.1)	(10.4)	(13.5)			
26 -35	38	78	116	8	9	17	46	87	133
	(82.6)	(89.7)	(87.2)	(17.3)	(10.3)	(12.8)			
36 - 45	52	39	91	16	11	27	68	50	118
	(76.5)	(78.0)	(77.1)	(23.5)	(22.0)	(22.4)			
46 - 55	20	14	34	6	3	9	26	17	43
	(76.9)	(82.3)	(79.1)	(23.1)	(17.7)	(20.9)			
≥ 56	8	5	14	5	1	6	14	6	20
	(64.3)	(83.3)	(70.0)	(35.7)	)16.7)	(30.0)			
Total	178	234	412	46	34	80	224	268	492
	(79.5)	(87.3)	(83.7)	(20.5)	(12.7)	(16.3)			

Figures in parenthesis are the percentages of row totals

Table 10—Blood urea levels of the population according to distance from the factory (sex wise)

Distance from factory (km)	Blood Urea (mg %) < 40 Male	Female	Total	> 40 Male	Female	Total	Total Male	Female	Total
< 2	111	125	236	33	29	62	144	154	298
	(77.1)	(81.2)	(79.2)	(22.9)	(18.8)	(20.8)			
2 - 4	58	94	152	13	3	16	71	97	168
	(81.6)	(96.9)	(90.5)	(18.4)	(3.1)	(9.5)			
4 - 6	5*	10	15	-	2	2	5*	12*	17
			(88.2)			(11.8)			
> 6	4*	5*	9*	-	-	-	4*	5*	9*
Total	178	234	412	46	34	80	224	268	492
	(79.5)	(87.3)	(83.7)	(20.5)	(12.7)	(16.3)			

Figures in parenthesis are the percentage of row totals

* Percentages are not worked out as the denominator is less than 10.

exposure to the toxic gas may lead to the impairment in redox potential of the biological system as well in the detoxification mechanism (Table 11).

Glutamic oxaloacetic transaminase (GOT)—The value of serum GOT was found to be almost normal in these populations and there was no significant difference between males and females or persons of different age groups (Table 12).

Table 11— Examination of whole Blood of the Exposed Population

Age groups (in years & sex)	Subjects	Glutathione u mole/100 ml	Haemoglobin gm %
Control value		M = 100-140 F = 100 - 140	M = 13 - 15 F = 11 - 13
10 - 20			
Male	22	100 (73-140)	14.7 (13.5-17.0)
Female	38	91 (60-135) 15%	14 (12-18) 58 %
21 - 40			
Male	82	90 (54 - 130) 35%	14.9 (13.0 - 18.0) 57 %
Female	103	94 (57 - 129) 25 %	14.3 (9.5 - 18.0) 63 %
41 - 65			
Male	42	91 (54 - 125) 30 %	15.3 (12.5 - 18.0) 57 %
Female	40 (65 - 124)	89.0 (65 - 124.)	14.3 (12.0 - 17.5)
Average			
Male	146	94 (54 - 140)	15.0 (12.5 - 18.0)
Female	181	91 (57 - 135)	14.2 (9.5 - 18.0)

Values in parenthesis denote the range
Values in % represent the percentage of the population surveyed exhibiting higher values than the maximum limit of the control.

Table 12— Biochemical Examination of Serum of the Exposed Population

Age group and sex (in years)	Subjects	G P T LU/ml	G O T LU/ml	Y- G T P LU/ml
Control 10 -20		5-35	5-40	upto 500
Male	10	9.38 ± 0.44 (7.0 - 15.4)	9.54 ± 0.54 (4.9 - 15.4)	145.65 ± 19.23 (26.9 - 403.5)
Female	12	10.44 ± 0.23 (8.6 - 13.0)	10.68 ± 0.45 (5.4 - 15.4)	169.57 ± 20.71 (87.0 - 260.9)
21 - 40				
Male	40	11.2 ± 0.5 (6.0 - 32.3)	10.0 ± 0.27 (4.02 - 18.4)	148.87 ± 14.44 (43.5 - 521.7)
Female	58	10.48 ± 0.17 (6.0 - 17.4)	10.0 ± 0.20 (5.4 - 17.4)	162.74 ± 9.19 (60.9 - 347.8)
41 - 65				
Male	23	10.23 ± 0.29 (7.0 - 15.4)	9.90 ± 0.35 (6.0 - 18.4)	160.68 ± 22.38 (43.5 - 434.8)
Female	14	12.26 ± 0.38 (7.0 - 19.0)	12.06 ± 0.53 (5.4 - 17.4)	144.85 ± 17.02 (43.5 - 217.4)
Average				
Male	73	10.30 ± 0.41 (6.0 - 32.6)	9.82 ± 0.39 (4.0 - 18.4)	151.73 ± 18.68 (26.9 - 521.7)
Female	84	11.03 ± 0.26 (6.0 - 19.0)	10.92 ± 0.46 5.4 - 17.4)	159.05 ± 15.64 (43.5 - 347.8)

Values in parenthesis denote the range

Glutamic pyruvic transaminase (GPT)—The value of GPT was also found to be within normal range of 5-35 I.U./ml and no significant difference was observed in the values in respect of age and sex of the exposed population (Table 12).

Ceruloplasmin—The ceruloplasmin content was found significantly raised in more than 45% of the population examined. In about one third of the population examined (35%) it was even more than one and a half to two times higher than the highest normal value of 50 mg/100 ml reported in literature. In 10% population the value was raised by 5-14% only. The elevated ceruloplasmin level was found in both the sexes but it was more prominent in the female population (Table 13).

Ceruloplasmin, an acute phase reactant protein, is found raised in various diseases including inflammatory disorders. It is also responsible for the incorporation of iron into transferrin which is ultimately taken up by the bone marrow for the synthesis of haemoglobin. Significant increase in the ceruloplasmin content observed among 40% of the exposed population surveyed may be an indication of inflammation in the body tissues and a cause of high levels of haemoglobin in these subjects. The enzyme is also responsible for the transport of copper into var-

Table 13— Biochemical Examination of Serum of the Exposed Population

Age group and sex (in years)	Subjects	Bilirubin mg %	Ceruloplasmin mg %
Control		0 - 1.5	25 - 50
10 - 20			
Male	10	0.52 ± 0.12 (0.2 - 0.8)	57.61 ± 10.33 (23.7 - 125.6) 55.6 %
Female	12	0.40 ± 0.04 (0.2 - 0.8)	43.13 ± 5.20 (19.0 - 75.8) 27.3 %
21 - 40	40	0.65 ± 0.07	44.45 ± 3.40
Male		(0.2 - 1.5)	(19.0 - 90.1) 36.6 %
Female	58	0.49 ± 0.05 (0.2 - 1.3)	48.21 ± 2.82 (19.0-113.8) 68.5 %
41 - 65			
Male	23	0.74 ± 0.11 (0.2 - 1.4)	45.14 ± 4.28 (21.3 - 106.7) 30.4 %
Female	14	0.52 ± 0.17 (0.1 - 1.1)	49.61 ± 5.54 (26.1 - 92.4) 21.4 %
Average			
Male	73	0.64 ± 0.10 (0.2 - 1.5)	49.1 ± 6.00 (19.0 - 125.6) 37.0%
Female	84	0.47 ± 0.09 (0.1 - 1.3)	47.02 ± 4.52 (19.0 - 113.8) 54.4 %

Values in parenthesis denote the range
Values in % represent the percentage of the population (surveyed) higher value than the maximum limit of the control

Table 14—Biochemical Examination of Urine in the Exposed Population

Biochemical parameters	Control Male/Female	Exposed population Male	Exposed population Female
1. Glutamate pyruvate transaminase (GPT)	18.83 ± 0.74 (14.63 – 22.61) (20)	23.45 ± 0.80 (14.83 – 25.27) (20)	20.85 ± 0.75 (18.62 – 26.60) (27)
2. Creatinine (g/24 hrs/1.5 litre urine)	1.71 ± 0.156 (1.1–2.39) (20)	3.30 ± 0.23 (2.25–5.04) (20)	2.95 ± 0.24 (1.99–3.89) (27)

Values in parenthesis indicate the range
Numbers in brackets underneath indicate subjects.

ious organs including brain as well as for the regulation of copper into various biogenic amines. A persistent increase in its level may cause higher transport of copper to various organs including brain and may disturb the level of biogenic amines. It may also lead to various neurological disorders.

Total bilirubin—Total bilirubin content was found within the normal range in 314 subjects exposed to the toxic gas. The values ranged between 0.1 and 1.4 mg/100 ml (Table 13).

γ-Glutamic transpeptidase (γ-GTP)—The values of γ-GTP were found to be within normal range. The values in 314 subjects, both male and female, ranged from 26.9-521.7 I.U./ml (Table 12).

The normal value of GPT, GOT, γ-GTP and bilirubin in serum of these subjects suggests the normal functioning of their liver.

Urinary creatinine—The value of creatinine in urine of these subjects was found to be significantly higher than in the control subjects. It ranged between 2.20 and 5.04 g/24 hr in males and 1.99 and 3.89 mg/24 hr in females with an average value of 3.30 and 2.95 g/24 hr respectively (Table 14). No change was observed in the urinary enzyme [illegible] GOT and GPT in these subjects after 24 [illegible]

The release of creatinine in urine of these [illegible] indicates an increased breakdown of [illegible] phosphate (a high energy phosphate compo[illegible] a result of muscular distress produced after [illegible] sure to the gas. The data are in accordance [illegible] general observation of the subjects at Bhopal [illegible] they reported whole body pain. Creatinine [illegible] is also markedly intensified in disease states [illegible] diabetes, exophthalmic goitre, poliomyelitis, [illegible] thenia gravis and progressive muscular atrophy [illegible] the absence of a significant change in serum [illegible] bin, GOT, GPT and γ-GTP activity along with [illegible] significant change in urine enzymes level, it may be suggested that liver and kidney possibly may be target sites for gas toxicity.

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References

1 Dacie J V & Lewis S M, *Practical haematology*, 5th edition (ELBS & Churchill Livingstone, Edin., 1975.
2 King E J & Wootton I D P, *Microanalysis in medical biochemistry*, 4th edition (J & A Churchill Ltd, London), 1964.
3 Ellman G L, *Arch Biochem Biophys*, 82 (1959) 70.
4 Jollow D J, Mitchell J R, Zampaglione N & Gillette J [illegible] *Pharmacology*, **11** (1974) 151.
5 Curzon G & Vallet L, *Biochem J*, 74 (1960) 279.
6 Reitman S & Frankel S A, *Am J Clin Pathol*, 28 (1957) 56.
7 Orlowski M & Szewczuk A, *Clin Chem Acta*, 7 (1962) 755.
8 Wootton I D P, *Microanalysis in medical biochemistry* (J & A Churchill Ltd, London), 1964, 35, 79.
9 Salter J M, in *Protein metabolism: Diseases of metabolism*, edited by G G Duncan, 5th edition, 1965, 98.

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A Clinical Study of Toxic Gas Poisoning in Bhopal, India

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The results of a clinical study of the victims of toxic gas released from Union Carbide Plant at Bhopal, India are presented. Thirty three patients, 22 males and 11 females were studied: their mean age was 38.3 years (range 8-60). All the patients lived close to the Union Carbide Plant, where the accident took place (0.1-3 km). Eye and respiratory tract irritation were the most important clinical features and resulted in keratitis in 18% and pneumonia in 79% patients. 55% patients fainted after inhaling the toxic gas and remained unconscious for a variable duration; three of them who had prolonged unconsciousness, had hyperreflexia and extensor planter response and one patient had features suggestive of encephalopathy. Polymorphonuclear leucocytosis (77%) and raised blood urea (67%) were commonly observed. The patients who lived close to the site of accident had significantly higher frequency of fainting ($P < 0.05$) and respiratory involvement ($P < 0.1$). Long-term follow-up studies are recommended to assess the sequelae of toxic gas poisoning.

On second and third December, 1984 in one of the biggest and most unfortunate industrial accidents of the century at Bhopal, India, a highly toxic gas stored in Union Carbide Plant leaked, affecting large number of population and claiming a number of lives. In this communication the results of a clinical study of the affected patients in the first week of the accident and a short term follow up of some of the patients for 3 months are presented.

Materials and Methods

The study was conducted within a week of the accident on 33 patients (22 males and 11 females) who were admitted in the medical wards of Hamida Hospital, Bhopal. Their mean age was 38.3 years (range 8-60 years) and they belonged to low or middle socio-economic status. At the time of the accident these patients were present within 0.1 to 3 km from the Union Carbide Plant. These patients were subjected to detailed medical history and physical examination beside hematological investigations which included hemoglobin estimation, blood counts, bleeding and coagulation time, blood sugar, blood urea, serum creatinine, serum bilirubin, SGOT, SGPT and alkaline phosphatase. A follow-up study was conducted after 3 months of the initial examination when the patients were subjected to a symptom checklist and clinical examination.

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Results

Around midnight, on the day of accident, most of the patients woke up because of severe burning in the eyes, irritating cough, dyspnoea, suffocation which soon became intolerable. Some patients in anxiety and panic ran for a safer place and medical help (14 patients), while other stayed in their houses and waited for the crisis to pass over (19 patients). Ophthalmological and respiratory involvement dominated the clinical picture and in the severely affected patients neurological involvement was also observed.

Opthalmological manifestations—All the patients experienced severe burning the eyes associated with watering and redness. 73% patients complained of diminution of vision and photophobia, and 24% had pain in the eyes. At the time of examination all the patients had marked congestion of the eyes. Keratitis was present in 6 cases and was bilateral in 2. The corneal opacity was of macula grade in 4 patients and leucoma grade in 2. Characteristically only the exposed portion of the cornea was affected being directly exposed to the toxic gas. On fundus examination, superficial flame shaped haemorrhages were observed in 2 cases, in both of them they were in the superior temporal region. In majority of the cases symptoms improved by 3rd day (range 2-7), except in 2 in whom they persisted till 2nd week due to severe keratitis.

Respiratory manifestations—All the patients had severe cough; irritation and dryness of throat, rhinorrhoea, suffocation and breathlessness. 81% patients

complained of chest pain because of tracheitis and 73% had excessive frothy expectoration. 91% patients were markedly dyspnoeic. Pharyngitis and laryngitis were present in 12% patients and were associated with difficulty in swallowing and hoarsness respectively. Crepitations and rhonchi were present bilaterally in 94% patients. In 64% patients dyspnoea improved by 4th day (range 2-7) but inspite of the treatment 46% patients were dyspnoeic at rest in 2nd week.

Neurological manifestations—Severe cough and dyspnoea were followed by fainting in 55% patients. The duration of unconsciousness ranged from 30 min to 3 days. One patient had prolonged unconsciousness, he also had myoclonic jerks localised to the right upper extremity and generalised hyperreflexia. His clinical picture was suggestive of encephalopathy. Three patients who had prolonged unconsciousness (more than 12 hr) had brisk deep tendon jerks and extensor planter response. Weakness of the legs was present in 2 patients, it was more marked in the proximal muscles; one patient had difficulty in getting up from the squatting position and the other had difficulty in walking. In both these cases the deep tendon jerks were absent and sensations were normal. Mild to moderate headache (55%), giddiness (46%), burning sensation in hands and feet (9%) and hypoaesthesia (3%) were also noted.

Miscellaneous manifestations—Shortly after exposure to toxic gas 64% patients had 1-3 vomitings, 15% had 1-2 loose motions and 6% complained of pain in abdomen. The patient with encephalopathy developed gastric haemorrhage on 4th day. Three cm liver enlargement was present in a patient but he did not have jaundice. 91% patients had sinus tachycardia which persisted in the first week. Generalised bodyache and fever were also present in 12% patients during the first week. A summary of the physical signs in these patients are presented in Table 1.

By the end of 2nd week, 3 patients were free of symptoms and were discharged from the hospital. 29 patients had varying degree of illness and were still in the hospital.

To study the effect of extent of exposure to the toxic gas on the clinical picture, the physical activity undertaken by the patient at the time of the accident and the distance from Union Carbide Plant were associated to the severity of the clinical picture. Dyspnoea at rest on 7th day and history of loss of consciousness were taken as the indicators of the severity of the clinical picture. The statistical analysis was done by the chi square test. The proximity of the patient to the accident site was found to be significantly associated to the severity of the clinical picture, i.e. dyspnoea at rest ($P < 0.1$) and loss of consciousness ($P < 0.05$).

The results of the laboratory investigations are presented in Table 2. The chest x-ray films of 19 patients were examined; bilateral patchy consolidation suggestive of pneumonitis was present in 15 patients, bilateral airspace consolidation suggestive of pulmonary oedema and prominent bronchovascular markings were present in 2 each. Left sided pneumothorax in addition to pneumonia was present in one patient.

Follow-up studies—Three months after the initial examination; one fourth of these patients could be followed up. By this time all the patients were discharged from the hospital but most of them still required medical supervision and treatment (88%). Rawness in

Table 1—Physical Signs in Patients (n = 33) Exposed to Toxic Gas

Physical signs	At the time of admission %	On 10th day of exposure %
Respiratory		
Dyspnoea at rest	90.9	45.5
Laryngitis	12.1	12.1
Crepts and rhonchi	93.7	81.8
Ocular		
Congestion	97.0	42.4
Keratitis	18.2	18.2
Retinal haemorrhages	—	6.1
Neurological		
Altered sensorium	54.5	3.0
Pyramidal signs	15.1	12.1
Myoclonic jerk	3.1	3.1
Muscle weakness	6.1	6.1
Hyporeflexia	6.1	6.1

Table 2—Laboratory Investigations in Patients Exposed to Toxic Gas

Investigations	N	Mean	Range	No. of abnormal values (%)
Haemoglobin (gm/100 ml)	22	12.8	8-14.6	18.2
Total leucocytes (count/mm^3)	22	13645.5	9200-24000	77.3
Polymorphs (%)	22	75.8	52-93	72.7
Platelets (count/mm^3)	6	221667	150.00-400.00	0
Bleeding time (min)	6	1.8	1-2.5	0
Coagulation time (min	6	3.95	2.5-4.5	0
Blood urea (mg/100 ml)	18	51.6	22-93	66.7
Serum creatinine (mg/100 ml)	18	0.6	0.4-1.2	0
Serum bilirubin (mg/100 ml)	19	0.5	0.4-0.8	0
SGOT (U/L)	19	29.7	6-110	26.3
SGPT (U/L)	19	17.4	17.41	5.3
Alkaline phosphatase (KA)	9	21.4	8-92	33.3

throat and dyspnoea, cough, reduction in visual acuity, headache, burning in hand and feet, depression and irritability were the commonly reported symptoms. In one patient diabetes mallitus was discovered during the follow-up period.

Discussion

The subjects included in this study resided close to Union Carbide Plant (0.1-3 km) and were heavily exposed to the toxic gas. Eye and respiratory system involvement dominated the clinical picture. The ophthalmological involvement was characterized by conjunctivitis and keratitis. Corneal opacity in 6 patients in this study suggests serious injury to cornea, however in the absence of slit lamp examination the frequency of lesser degree of corneal involvement and punctate keratitis can not be commented. Irritant toxics like TDI (toluene diisocyanate) and MIC (methyl isocyanate) have been reported to cause corneal oedema by producing alteration in the metabolism of corneal epithelium in human[1] and permanent eye damage in animals[2] respectively. Superficial retinal haemorrhages which were present in 2 cases in the present study may be attributed to hypoxia following exposure to toxic gas. The mechanism of haemorrhage in the funds is usually by diapedesis of red blood cells through the capillary endothelium whose permeability has been altered by hypoxia[3].

The respiratory involvement in the patients of this study included irritation of the upper respiratory tract, bronchitis, and pulmonary oedema. The radiological picture was also suggestive of bilateral patchy pneumonia (79%) and pulmonary oedema (10.5%). In animal studies inhaled isocyanate vapours have been reported to cause respiratory irritation at low concentration (2 ppm) and coagulation necrosis at high concentration (5 ppm). The histopathological picture was suggestive of peribronchial pneumonia[4]. The respiratory effects observed in the present study can be compared to that produced by several irritant gases like nitrogen dioxide, sulphur dioxide, chlorine, phosgene, carbon monoxide and hydrocarbons which produce actute pulmonary oedema. However, pulmonary oedema is transient and no more than a part of lungs reacts to the insult. The more serious and long lasting effects are fibrinous bronchiolitis, peribronchiolitis fibrosa obliterans and severe airway obstruction[5].

More than 50% patients in the present study fainted and remained unconscious for 30 min to 3 days. Those who had prolonged unconsciousness had hyperreflexia and extensor planter response. One patient had clinical picture suggestive of encephalopathy. With other irritant gases like TDI poisoning hyper reflexia, extensor planter response and coma have been reported[6]. In another study on TDI poisoning in which 35 firemen were exposed while combating a fire in a polyurethane plant a majority of the patients had irritation of respiratory tract, half of them had gastrointestinal and neurological symptoms, the latter included euphoria, ataxia, headache and loss of consciousness. During next 3 weeks difficulty in concentration, poor memory and confusion were reported by 61% patients[7]. Similar studies have also been carried out by ICMR[8,9].

In view of the suggestion of MIC as a possible component of the toxic gas an experimental study gives an idea about the dose response relationship. 0-4 ppm MIC for 1-5 min produces no effect, only the odour can be perceived; 2 ppm produces lacrymation, irritation of nose and throat; 4 ppm produces more marked irritation and at 21 ppm the exposure is intolerable[10]. The patients of the present study who were close to Union Carbide Plant probably inhaled a large quantity of toxic gas and developed more severe illness as evidenced by respiratory involvement ($P < 0.1$) and loss of consciousness ($P < 0.05$). The results of a detailed follow up study conducted after 2-3 months subsequent to the exposure of the toxic gas have already been reported[11-14]. The results of a further follow up study conducted after 15 months of the exposure will be presented later which would highlight a comparative account of the clinical profile in the acute phase and that during the follow up period.

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References

1 Luckenbach M & Kielar R, *Am J Ophthalm*, 90 (1980) 682.
2 Zang C, *Isocyanates quoted in Developments in occupational medicine*, (Year Book Medical Publisher, Chicago) 1980: 365.
3 Michelson I C *Textbook of the fundus of the cty*, Illed (Churchill Livengstone, London) 1980, 127.
4 Bernstein L, *All Clin Immunol*, 70 (1982) 24.
5 Fraser R G & Prae J A P, *Diagnosis of the diseases of the chest*, *Iled* (W B Saunder's Co, Philadelphia) 1978, 1259.
6 Brugsch H G & Elkins H B, *New Engl J Med*, 268 (1963) 353.
7 Quesne P, Axford A T, Kckerrow C B & Jones A P, *Br J Ind Med*, 33 (1976) 72.
8 Misra N P, Pathak R, Gaur K J B S, Jain S C, Yesikar S S, Manoria P C, Sharma K N, Tripathi B M, Asthana B S, Trivedi H H, Sharma V K, Malhotra Y, Verma A, Bhargava D K & Batni G, *Indian J Med Res*, 86 (Suppl) (1987) 11.

9 Bharucha E P & Bharucha N E, *Indian J Med Res.* **86** (Suppl) (1987) 59.

10 B.R.M.A *Isocyanates in industry, operating and medical code of practice*, (British Rubber Manufacture Association Ltd, Birmingham) 1977.

11 Gupta B N, Rastogi S K, Chandra H, Mathur A K, Mathur N, Mahendra P N, Pangtey B S, Kumar S, Kumar P, Seth R K, Dwivedi R S & Ray P K, *Indian J Exp Biol*, **26** (1988) 149.

12 Rastogi S K, Gupta B N, Tanveer Hussain, Kumar A, S & Ray P K, *Indian J Exp Biol*, **26** (1988) 161.

13 Srivastava R C, Gupta B N, Athar M, Behari J R, Hasan S K, Bharti R S, Singh A, Misra M & Ray P, *an J Exp Biol*, **26** (1988) 165.

14 Saxena A K, Singh K P, Nagle S L, Gupta B N & Ray P, *an J Exp Biol*, **26** (1988) 173.

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Effect of Exposure to Toxic Gas on the Population of Bhopal: Part IV—Immunological and Chromosomal Studies

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The present studies were conducted in Bhopal 21 months after the gas tragedy to find out whether the toxic gas had altered the immune response and caused chromosomal aberrations in the exposed population. Immunological studies showed a significant depression in phagocytosis and T-cell rosettes whereas no deviations in immunoglobulin levels were found. This indicated that the toxic gas had suppressed the cell mediated immune status and did not alter the humoral immunity of the exposed population. Chromosomal studies were performed on 31 adult subjects of either sex and random age groups. The buffy coat lymphocytes of peripheral venous blood were cultured. 100 metaphase plates of each culture were observed for frequency of chromatid breaks and gaps. Appropriate control studies were also done. Statistically significant differences were observed in respect of chromosomal aberrations in the two groups.

Recently several reports have shown adverse pharmacologic, toxicologic and physiologic effects of different toxic gases. In spite of available literature on MIC and its effects on eye, lungs, behavior and on immune system in rodents[1-4], there is a paucity of data on the effects of MIC on immune system of human beings. Preliminary reports indicated sensitization of animals by inhalation of *p*-tolyl isocyanate or toluene diisocyanate and the detection of specific antibodies within 2 week of exposure[5], respiratory distress, pulmonary oedema, abnormalities of the corneal epithelium and the immuno modulatory effects after single or repeated exposures of MIC[1-4,6]. In the present study an attempt has been made to assess the effect of toxic gas on immune system of human beings.

The present studies were conducted during February to April, 1985 on the population exposed to the toxic gas to ascertain whether the toxic gas had altered the immune response of the subjects. The humoral immunity was assessed by quantification of immunoglobulins (IgG, IgA and IgM) and cell mediated immunity (CMI) by T-cell rosettes and phagocytosis.

Chromosomal aberrations in the peripheral blood lymphocytes were recorded in the exposed population for the assessment of genetic and carcinogenic potential of the gas.

Materials and Methods

Peripheral blood was obtained from the exposed subjects by using plastic disposable sterilized syringes and needles. Each blood sample was divided into two portions, one was kept for serum separation and the other taken into heparinized tubes for lymphocyte separation.

Lymphocyte separation—Lymphocytes were separated from peripheral heparinized blood using Ficoll-Hypaque (Pharmacia, Sweden) density gradient centrifugation. Mononuclear cells at the buffy interface were gently removed and washed three times with RPMI 1640.

Phagocytosis—One ml lymphocytes (1×10^6 cells/ml) were taken into petri dish containing cover slip and incubated at 37°C for 2 hr. The cover slips were washed twice with warm PBS to remove non-adherent cells. One ml opsonized sheep erythrocytes (SRBC) (1×10^8 cells/ml) were added and further incubated at 37°C for 1 hr. After incubation, the cover slips were washed thrice with warm PBS and then fixed in methanol for 10 min. Dried cover slips were stained with Leishman's stain and mounted on microscope slides. 200 cells were counted, using light microscope, to determine the percentage of phagocytosis.

T-Cell rosettes—It was determined by spontaneous rosetting of lymphocytes with SRBC in 44 subjects. Equal volume of 0.5% SRBC in media with 5% fetal calf serum (FCS) was added to lymphocyte suspension (2×10^6 cells/ml) and incubated at 37°C for 15 min. The tubes were centrifuged at 1000 rpm for 5 min and left at 4°C overnight. Cells (200) were counted under light microscope to determine the percentage of T-rosettes.

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Immunoglobulins (IgG, IgA and IgM)—Serum immunoglobulins were estimated by the single radial immunodiffusion method. The wells of tripartigen plates were filled with 5 µl of serum and left for incubation at 37°C. For IgG and IgA the minimum diffusion time given was 50 hr whereas for IgM it was 80 hr. The standard control sera and tripartigen plates supplied by M/s Hoechst India.

Chromosomal studies—Cytogenetic study was performed on 31 adult subjects of either sex and random age group. Peripheral venous blood (5 ml) was drawn in a heparinized disposable syringe. The buffy coat lymphocytes were obtained by incubating the blood sample held in the syringe at 37°C at an inclination of 70° for 45 min. These lymphocytes were suspended in media TC 199 at 37°C for 68 hr in presence of phytohaemagglutinin-P (PHA-P, Difco) and homologous serum. 8 Drops of colchicine (0.01%) was added three hours before harvesting. The cultured lymphocytes were treated with 0.667% hypotonic solution for 20 min and fixed in 3:1 methanol glacial acetic acid at 37°C for 15 min. The air dried slides were prepared and stained with 4% buffered Giemsa for 30 min and trypsin-G banding was done according to methods of Kato and Yoshida[7]. 100 well-spread metaphases from each subject were studied for chromosomal aberrations. The results were compared with 31 control cultures from residents of Bhopal who were not exposed to the toxic gas.

Statistical analysis—The results are expressed as mean ± SE. Comparisons were made with appropriate controls using student's t test. Differences were considered significant at $P < 0.05$.

Results and Discussion

Experiments on phagocytosis were conducted to study whether the toxic gas had altered the ability of peripheral blood mononuclear cells to engulf and process the micro-organisms. It is the first line of defence in a host against pathogens. Increased phagocytosis by phagocytic cells may contribute to the elimination of microbial infection. In subjects suffering from toxic damage of bone marrow by chemicals, the normal mechanism of bacterial killing and digestion by phagocytic cells does not take place.

This study was conducted on 19 exposed and 8 control subjects. 57.9% of the subjects showed suppression in phagocytic ability of peripheral blood mononuclear cells when compared to controls. The phagocytosis was found to be 17.2 ± 3.0% in the exposed group whereas in controls it was 32.5 ± 0.9% (Table 1). Whether these reductions in phagocytic response are due to the decrease in actual cell numbers for phagocytes in the exposed population or due to a reduction in their functional ability is clear. More studies will be required for finding suitable answer for the same, particularly because used same amount of whole blood cells for doing phagocytosis. Any reduction in number of phagocytes will show a relative increase in other cell populations particularly in PMNs, lymphocytes etc. The cell mediated immune system is under the influence of thymus and the cells are referred to as thymus-dependent or T-cells. They are also responsible for the elimination of spontaneously developing neoplastic cells which might be a potential threat to the individual. Cell mediated immunity is responsible for the protection of the host against various infective agents. The T-cell rosettes were estimated to study whether there was any effect on the cell mediated immune response of the gas exposed victims. The T-cell population in exposed subjects was found to be 28.6 ± 0.5%. It is reported to be 65.1% in the normal Indian population[8]. The suppression of T-cells in the exposed population was found to be statistically significant.

It has generally been observed that the immunoglobulin levels in a normal healthy individual are fairly stable. The deviation in immunoglobulin levels may indicate the presence of infection or close relationship to a causative agent. Quantification of immunoglobulins (IgG, IgA and IgM) was carried out to ascertain whether the toxic gas had altered the humoral immune response of the exposed population (Table 2). Immunoglobulin G was estimated in the sera of 389 subjects exposed to gas. The mean

Table 1—Phagocytosis of SRBC by Peripheral Blood Mononuclear Cells

	Exposed group	Control group
Percent phagocytosis (Mean ± SE)	17.2 ± 3.0	32.5 ± 0.9
Range	1.0-39.2	29.1-38.0
Number of cases	19	8
Suppression in percent of cases	57.9	—
P value	< 0.001	

Table 2—Immunoglobulin Profile of IgG, IgA and IgM

Exposed group	IgG	IgA	IgM
Mean ± SE (mg/100 ml)	1242 ± 12.3	213.9 ± 4.4	177.0 ± 5.0
Range	300-3474	20-364	39.0-436.7
No. of cases	389	312	309
Control group			
Mean ± SE mg/100 ml)	1184 ± 88.9	216.9 ± 22.8	111.5 ± 12.9
Range	600-1726	90.9 ± 338.4	40.0-192.0
No. of cases	10	10	10

value was found to be 1242 mg/100 ml with a range of 300 to 3474 mg/100 ml. Four per cent of the exposed cases showed raised IgG levels, i.e. 2747.5 mg/100 ml (range 2334 to 3474 mg/100 ml), whereas 7% of the cases showed low IgG values, i.e. 407.4 mg/100 ml with a range of 300 to 620 mg/100 ml. In the case of control group, it was found to be 1184 mg/100 ml (range 600 to 1726 mg/100 ml).

Immunoglobulin A was estimated in the sera of 312 exposed cases and the mean value was found to be 213.9 mg/100 ml with a range of 20 to 364 mg/100 ml. Lower values, i.e. 70.8 mg/100 ml (range 20 to 118.9 mg/100 ml) were observed in 7% of the cases. In control group, IgA was found to be 216.9 mg/[illegible]ml (range 90.9 to 338.4 mg/100 ml).

Immunoglobulin M was estimated in 309 exposed cases and the mean value was found to be 177.0 mg/100 ml with a range of 39.0 to 436.8 mg/100 ml. The higher values, i.e. 390.0 mg/100 ml, were found in 8% of the exposed cases (range 294.7 to 436.8 mg/100 ml) whereas in 2% it was low, i.e. 39.2 mg/100 ml (range 39.0 to 39.5 mg/100 ml). In the control group, the mean value was found to be 111.5 mg/100 ml with a range of 40.0 to 192.0 mg/100 ml. Our studies indicated that the mean values of immunoglobulin (IgG, IgA and IgM) in the exposed population were not significantly different from that of controls (Table 2).

In chromosomal studies metaphase observations were recorded and analysed for the presence of breaks and gaps. The number of breaks and gaps in 100 metaphases of each subject were noted. The data are presented in Tables 4 and 5.

The present study shows that exposure to the toxic gas caused a significant degree of chromosomal aberrations. Our studies support the findings of Andersen *et al.*[9] who, in a standard Ames test, found that [illegible]luene diisocyanate and 4,4-methylenediphenylisocyanate are mutagenic for bacteria (*Salmonella typhimurium*) in the presence of a conventionally prepared microsomal fraction (S_9).

Table 3—High/Low Immunoglobulin Values in Population Exposed to Toxic Gas

	IgG	IgA	IgM
High			
Mean ± SE (mg/100 ml)	2847.5 ± 0.9	—	390.9 ± 10.4
Range	2334-3474	—	294.7-436.8
Percent of cases	4		8
Low			
Mean ± SE (mg/100 ml)	407.4 ± 22.8	70.8 ± 5.8	39.2 ± 0.1
Range	300-620	20-118.9	39.0-39.48
Percent of cases	7	7	2

The present studies were conducted 2½ months after the gas tragedy at Bhopal. The CMI status of the exposed subjects as studied by the estimation of T-cells and phagocytosis indicated that the toxic gas nad suppressed the phagocytic ability of the peripheral blood mononuclear cells. The T-cells were also found affected by the toxic gas showing that the toxic gas had significantly lowered the CMI status of the exposed population. The marginal deviations in immunoglobulin profile may be due to non-specific

Table 4—Breaks and Gaps in 100 Metaphases in Each Exposed and Control Subjects

Subject	Exposed		Control	
	Breaks	Gaps	Breaks	Gaps
1	12	15	5	6
2	12	26	5	7
3	13	27	6	6
4	11	13	6	5
5	12	13	5	6
6	13	12	6	6
7	13	12	6	6
8	13	13	6	7
9	14	15	7	6
10	14	16	4	6
11	13	15	4	5
12	16	17	4	7
13	14	15	7	6
14	18	18	7	5
15	14	18	6	5
16	17	17	7	5
17	17	20	6	7
18	16	18	5	6
19	19	20	6	5
20	17	17	7	5
21	15	16	6	5
22	16	17	5	5
23	17	18	5	6
24	14	17	6	5
25	17	15	7	6
26	19	20	5	7
27	17	18	4	5
28	19	18	4	4
29	15	18	5	4
30	17	16	6	5
31	17	19	6	7

Table 5—Analysis of Chromosomal Aberrations (Breaks)

	Control	Exposed	't'
Number	31	31	20.82
Mean ± SD	5.61 ± 0.99	15.19 ± 2.30	($P < 0.001$)

Analysis of Chromosomal Aberration (Gaps)

	Control	Exposed	't'
Number	31	31	23.44
Mean ± SD	5.68 ± 0.87	16.23 ± 2.29	($P < 0.001$)

infections. It can be concluded from the present studies that the toxic gas had suppressive effect on CMI while it had no effect on humoral aspect of the immune system.

The chromosomal study was conducted at a time when the clinical signs and symptoms were well marked. A follow-up study should be performed after such time as the clinical signs and symptoms have subsided to ascertain the residual effects of the exposure to the toxic gas on chromosomes.

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References

1 Nemery B, Dinsdale D, Sparrow S & Ray D E, *Br J* **42** (1985) 799.
2 Salmon A G, Kerrmuir M & Andersson A, *Br J Ind* (1985) 795.
3 Ferguson J S, Schaper M, Stock M F, Weyel D A & *Toxic Appl Pharm.* 82 (1986) 329.
4 Luster M I, Tucker A N, Germolec D R, Silver M T, T Y, Vore S J & Bucher J R, *Toxic Appl Pharm.* 140.
5 Karol M H, Dixon C, Brady M & Alarie Y, *Pharm.* 53 (1980) 260.
6 Verma D R, in *Hazard assessment of chemicals*, Vol by Jitendra Saxena (Hemisphere Publishing Washington), 1987.
7 Kato & Yoshida, *Chromosoma*, 36 (1973) 270.
8 Singh S & Nath I, *Indian J Med Res.* 72 (1980) 385.
9 Andersen M, Binderup M L, Kiel P, Larsen H & *Scand J Work Envir Hlth.* 6 (1980) 221.

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EDITORIAL

BHOPAL TRAGEDY — A YEAR LATER

GS Sainani, VR Joshi, PJ Mehta & P Abraham

In the night of 3rd and 4th December 1984, world's worst industrial disaster occurred. A reservoir released a cloud of forty tonnes of ...ous methyl isocyanide (MIC), leaving behind ...ands dead and affecting the health of over ...undred thousand people in the vicinity. At ...me very little was known about MIC and its ...gical affects. This increased the magnitude of ...tragedy. Are we wiser now?

From various medical and non medical ...rts, it is clear that MIC not only produces an ...e illness but due to its irritant nature, causes ...ise inflammation of the skin, the eyes, the ... the throat and the lungs. Many died as a ...ult of pulmonary oedema. The affected individ...s complained of dry cough, throat irritation, ...noea on exertion and chest pain. There are ...istent rales in the chest with evidence of ...ratory obstruction. In almost one third cases, ...symptoms could be graded as severe. Chest X-... showed evidence of interstitial, alveolar and ...ural pathology. Lung functions have shown a ...ed pattern of restrictive and obstructive dys...ction. The obstruction may be at the small and ... large air-ways. There is reduced arterial ...genation. The acute effects go beyond local ...ation. There is central nervous system dysfun...tion. Immediate loss of consciousness, loss of ...ncter control, confusion, memory defect, ...ia, euphoria and paraesthesiae have been ...ed. Gastrointestinal manifestations consisted ...vampy abdominal pain, nausea, vomiting and ...rrhoea[2]. Abortions and premature deliveries ...curred. Kidneys and liver have been variably ...ected.

There had been some confusion over the ...chanism of toxicity of MIC. This really com...nded the tragedy, hampering effective thera...utic relief. The big question was whether MIC ...cause cyanide poisoning? The evidence avai...le so far suggests, such a possibility. MIC can ...ease hazardous decomposition products i.e. ...ic gases and vapours such as hydrogen ...anide, oxides of nitrogen and carbon monoxide. ...hen involved in fire)[3]. Union Carbide Corpora-tion, U.S.A., in its guidelines for treatment of MIC toxicity, states amongst other measures: "If cyanide poisoning is suspected use, amyl nitrite; if there is no effect, use sodium nitrite and sodium thiosulphate IV." Examination of the urine with picric acid gave some indication of excretion of thiocyanates produced by the host's detoxifying mechanism[4]. Patients who received thiosulphate showed remarkable improvement. Prof. Heeresh Chandra of Bhopal has, based on autopsy studies of over 300 patients, postulated that death is due to poisoning by irrespirable gases resulting in cytotoxic or histotoxic death. Myocardial cells show not only pigmentary changes, but flocculent changes of the mitochondria, which are believed to be characteristic of cytotoxic death[4].

At autopsy the lungs are grossly increased in size and weight. There is tracheitis with denudation of mucosa. Necrotising bronchiolitis is present at places. Alveolar septa are congested or destroyed. The most important finding however is pulmonary oedema with little evidence of inflammation.

Brain shows oedema, blood vessel congestion, sub-arachnoid and intraventricular haemorrhage and petechial haemorrhages in the white matter and the cortex. Kidneys have shown acute tubular necrosis. Liver has shown necrosis in some cases.

Venous blood of victims has been uniformly described to be red or cherry red in colour. This colour can be due to carbon monoxide, cyanide, aliphatic and aromatic nitrites and organic thiocyanates. MIC can increase cyanogen pool leading to chronic cyanide poisoning. Another possible mechanism is carbamylation of terminal amino groups of the chains of haemoglobin resulting in shift to the left of the Bohr effect. This is an important mechanism as the effect can be reversed by more potent sulfur donors.

The question of chronic toxicity or after effects is yet to be answered. Affected people have continued to complain of burning and watering of the eyes. Many have diminished vision. There is macular oedema and central serous retino-

PTO

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Inhalation Toxicity Studies of Methyl Isocyanate (MIC) in Rats: Part IV—Immunologic Response of Rats One Week after Exposure:Effect on Body and Organ Weights, Phagocytic and DTH Response

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Immunotoxicological evaluation of rats exposed to MIC was done on animals after 7 days of exposure to it. Initial and final body weights, percent organ weights and alveolar and peritoneal macrophage functions were evaluated. No increase in the body weight was found in treated animals after 1 week of exposure whereas percent thymus and liver weights were significantly enhanced. Both alveolar and peritoneal macrophage functions were significantly impaired in MIC exposed animals as compared to controls. Delayed type hypersensitivity, enumerated against sheep red blood cells was found significantly impaired in MIC exposed animals as compared to the controls.

Currently investigation on the toxicological effects of chemicals on the immune system is gaining importance, since the complex immune system[1] with several types of cells is working independently or together to carryout homeostasis to provide resistance against infections caused by bacteria, fungi and virus, and also against toxic chemicals, carcinogens[2-4] etc. The rationale behind monitoring of immunotoxicological effects of a chemical is (a) that the basic process of host defence mechanism is well understood, (b) that it represents a system in which different cells from the system can be removed easily and their functions examined *in vitro*, and (c) that even a small dose of a toxicant can have a significant effect on the immune system of the body much earlier than any other system[6].

An extensive study of the toxicological effect of methyl isocyanate (MIC) has been undertaken in our laboratories after the disaster in Bhopal, where a toxic gas leakage took a toll of thousands of lives. An identical but smaller accident occurred in Kem (1967), where 35 firemen were exposed to isocyanate fumes. They were studied for neurological and lung function abnormalities[7,8]. However, there is no significant data on the immunotoxic effect of MIC. Previous studies on human indicated that in low concentration isocyanate stimulates lymphocyte proliferation, while it inhibits T-cell blastogenic response with mitogens in the spleen and lymph nodes of mice[9]. The limited data have shown increased airway sensitivity leading to asthma in isocyanate workers[10,11], and hyperactive pneumonitis[12] which have been related to the immunotoxic effects of isocyanate. In our study of Bhopal population exposed to toxic gas we have found significant increase in absolute lymphocyte counts, T cell counts, DTH response and phagocytic response[13].

The present study has, therefore, been carried out to investigate the early effect of MIC (7 day post exposure) on the immune response of the experimental animals with reference to phagocytic response, delayed type hypersensitivity (DTH) and percent organ weights of all immunologically important organs.

Materials and Methods

This study was done on random bred Wister strain male rats of between 130 and 155 g body weight. Three sets of animals were evenly arranged in terms of their total group body weights and were exposed to an acute dose of MIC inside the inhalation chamber. The animals were marked for identification, maintained at 21°-24°C and weighed daily for the change in their body weight, and a complete record of food and water intake maintained. The animals were checked twice a day for behavioral changes and morbidity. Control groups were made from age, sex and weight matched normal rats maintained on similar diet and placed in inhalation chamber with air only for the same period. The exposed animals were weighed and sacrificed after 1 week. One set of exposed and control group was used for body weight and organ weight evaluation, another for alveolar and peritoneal macrophage function test and the third group of exposed and normal animals, for DTH determination. Immediately after sacrifice, a complete necropsy was performed and thymus, spleen, liver, peripheral and mesentric lymph nodes, adrenal gland and kidney were removed, trimmed and weighed.

For phagocytosis mononuclear cells were obtained from peritoneal cavity and lungs by flushing

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Table 2—Percent Organ Weight in MIC Treated Rats as Compared to Control

[Values are mean ± SE. Figures in parentheses represent number of animals]

	Normal controls	Treated	*P*-Value
Thymus	0.123 ± 0.019 (9)	0.182 ± 0.004 (14)	< 0.001
Spleen	0.364 ± 0.021 (8)	0.407 ± 0.031 (15)	NS
Liver	3.550 ± 0.200 (9)	4.74 ± 0.240 (9)	< .01
L.N. (P)	0.071 ± 0.012 (5)	0.107 ± 0.016 (9)	NS
L.N. (M)	0.299 ± 0.064 (5)	0.430 ± 0.057 (9)	NS
Adrenal	0.025 ± 0.0023 (8)	0.029 ± 0.0056 (9)	NS
Kidney	0.737 ± 0.037 (5)	0.784 ± 0.026 (9)	NS

L.N. (P) = Lymph node (parietal); L.N. (M) = Lymph node (Mesentric)

Table 3—Macrophage Function in MIC Exposed Animal as Compared to Control

[Figures in parentheses are number of animals]

Groups	Phagocytosis (%)			
	Peritoneal		Alveolar	
	Range	Mean ± SE	Range	Mean ± SE
Control	14.0-42.22 (9)	26.16 ± 2.65	17.0-39.0 (7)	27.34 ± 3.10
Treated	11.11-21.50 (7)	15.60 ± 1.55	8.23 ± 24.07 (7)	15.22 ± 1.80
P Value		< 0.01		< 0.01

water intake, it was found to be impaired and this may have caused some weight reduction in treated animals. However, there was no significant change in percent organ weight of animals exposed to MIC in comparison to control except in case of liver and thymus. The increase in liver and thymus weights could have resulted from hypertrophy of these organs due to toxic effects of MIC.

Lymphoreticular system plays an important role in host defence against foreign invaders and toxicants. Macrophage actively clears the foreign body from the system on one hand, and on the other produces the B cell differentiation factor[15]. Phagocytosis is one of the most important functions of macrophage. Macrophage undoubtedly plays an important role in host resistance to many microorganisms. Macrophages also make a diverse group of products[16] as a result of external stimuli due to lipopolysaccharide, phagocytic particles, immune complexes and lymphokines etc. Macrophages also participate in formation of a variety of complement proteins, interferon and interleukin-1 (IL 1). Though by studying phagocytosis alone one cannot pinpoint the defect in immune system, grossly reduced/increased phagocytic function may indicate the ability of macrophages to perform abnormally or in an activated form.

In the present study the functional capacity of both the alveolar and peritoneal macrophages was found to be significantly impaired in rats exposed to MIC as compared to that in the normal untreated controls, indicating a gross impairment in the macrophage related immune response of the exposed animals. The observation is consistent with those in the previous reports of Tse[11] and White *et al.*[17] who observed eosinophilia, lymphopenia and neutropenia in animals after exposure to aliphatic isocyanate. An impaired macrophage function in MIC, therefore, has an immunosuppressive effect and animals may become more susceptible to various infections, as reported by Howard[18] about immunosuppressed animals and also shown by endotoxin sensitivity test by Saxena *et al*[19].

Cellular immune parameters have been widely used in toxicological evaluation of various chemicals because immune system represents an integrated system of host defence and also because changes in the immune response appear long before the involvement of any other system of the body[6]. DTH has proved to be one of the sensitive parameters of cellular immune competence. The rationale behind the DTH response assay is that the *in vivo* testing reflects systemic immunity of the host more closely than the *in vitro* test. Evaluating the cell mediated immunity (CMI) using foot pad assay requires both afferent arm (antigen recognition and processing) and the efferent arm (lymphokine production and increased permeability) for adequate function of the immune system[17]. Any defect, therefore, in efferent or afferent arm leads to impairment in DTH re-

them with 5 ml of cold minimum essential medium, containing 10 units of heparin/ml. The peritoneal and alveolar fluids were collected in silconized glass tubes. Viability of peritoneal and alveolar exudate cells was studied using trypan blue dye exclusion test.

Phagocytosis—The phagocytic activity of peritoneal cells was carried out using sheep red blood cells (SRBCs) according to the method of Koller *et al.*[14] with minor modification. Briefly, the cells from peritoneal and alveolar exudate (1×10^6) were carefully layered over glass microslides (22 mm^2) kept in plastic petridishes of 4.0 cm diam. The cells were allowed to adhere to the surface of the microslide by incubating for 1 hr at 37°C in humidified atmosphere of 5% CO_2. The microslides kept in petridishes were flooded with 5 ml PBS (*p*H 7.2) and the nonadherent cells were removed by sucking out the fluid with pasteur pipette. The process was repeated thrice.

SRBCs were coated with anti-sheep red blood cells anti-bodies (anti-Sheep Hemolysin 1:500 dilution) by incubating it at 37°C for 1 hr and washed by PBS at the end of the opsonization process (2000 rpm, 15 min), and finally a SRBC suspension (1%) was made by phagocytic assay.

The adherent cells on microslides kept in petridishes were covered with 1 ml of processed SRBC suspension and further incubated for 1 hr at 37°C. The microslides were washed with PBS, stained with Giemsa and examined under oil immersion in the light microscope.

The assay of macrophage function was carried out by its capacity to phagocytose SRBCs. A macrophage was considered positive for phagocytosis if three or more SRBCs were seen engulfed. The number of such macrophages was counted along with non-phagocytic macrophages microscopically, and their percentage determined.

DTH response—DTH response to SRBCs was done by the following protocol. Sensitization of rats was done on 1st day on the abdominal side with 1×10^8 SRBCs in 0.1 ml of Hank's balanced salt solution (HBSS). On 5th day, rats were challenged in the left hind foot pad with 1×10^7 SRBC in a volume of 0.05 ml. Diameter of foot pad was measured with caliper before challenge injection and then 24 and 48 hr after injection to observe induration. An equal volume (0.05 ml) of normal saline was injected in the right hind foot pad which served as control and the diameter was read after 24 and 48 hr in the same way with the caliper to determine non-specific swelling. The non-specific swelling was substracted from the left foot pad induration to determine the actual swelling caused by hyper-sensitivity response. The above control protocol was followed for controls as well as animal exposed to MIC and induration in rats exposed to MIC was compared with that in the controls.

Results

Control and experimental values of the body weight of the animals have been shown in Table 1. In control group there was a significant gain in body weight after 7 days of air exposure, whereas in experimental group there was a reduction in weight range although the mean weight did not show any significant change. The organ weights have been expressed in gm organ weight/100 g body wt in Table 2. Only thymus and liver showed statistically significant increase in weight. Results of phagocytosis against SRBCs presented in Table 3, showed significant impairment in rats exposed to MIC when compared to control rats. Phagocytosis was significantly lowered in both alveolar and peritoneal macrophages. Both range and mean values of phagocytosis were significantly low in rats exposed to MIC as compared to normal controls.

Results of DTH in MIC exposed as well as control rats showed that there was a significant impairment of DTH response ($P < 0.001$) in MIC exposed animals as compared to normal controls. The DTH response (cm) in control (n = 8) and experimental animals (n = 10) being 0.2009 ± 0.007 and 0.0037 ± 0.007 respectively.

Discussion

Immunotoxic effect of MIC was studied after 1 week of exposure. There was a significant increase in the body weight of the normal control animals, whereas in the group exposed to MIC the weight range after treatment decreased, although no significant change in the mean body weight was observed. In fact, during the regular monitoring of food and

Table 1—Change in Body Weight in MIC Treated Rats as Compared to Control

Groups	No. of animals	Initial body wt(g)		Final body wt(g)		
		Range	Mean ± SE	Range	Mean ± SE	
Control	6	145-150	146.6 ± 0.40	150-175	162.5 ± 3.56	< 0.001
Treated	8	130-155	138.0 ± 3.87	115-147	139.37 ± 3.61	NS

sponse. DTH is primarily an *in vivo* manifestation of the activation of specialised T cell subsets. These T cells are called T_{DH}[20]. T_{DH} subset can respond to a diverse array of antigens. Any change in T cell function which is one of the most important parameters of CMI can, therefore, be measured by DTH assay. In the present study a significant impairment of DTH response was observed in rats exposed to MIC as compared to that in normal rats, which clearly shows a defect in CMI and T cell function. It must be mentioned here that CMI and humoral immunity are inter-related and that when CMI gets affected humoral immunity too must invariably be affected. Similarly in our studies on the human subjects[13] exposed to toxic gas at Bhopal a phytohemogglutinin and purified protein derivative mitogenic response was found decreased in exposed subjects when compared to that in normal controls. A decrease in phagocytic response of human subjects exposed to toxic gas was also observed by us. These data are consistent with the observations made in rats exposed to MIC.

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References

1 Ray P K, Ed, *Immunobiology of transplantation, cancer and pregnancy* by (Pergamon Press, New York, 1983, 1-24
2 Ray P K, Dohadwala M & Bandyopadhyay S, *Cancer mo Pharmacol*, 4 (1985) 59.
3 Dohadwala M & Ray P K, *Cancer Chemo Pharmacol* (1985) 135.
4 Srivastava S P, Singh K P, Saxena A K, Seth P K & Ray *Biochem Pharmacol*, 36 (1987) 4055
5 Singh K P, Saxena A K, Zaidi S I A, Dwivedi P D, Srivas S P, Seth P K & Ray P K, *J Appl Toxicol*, (1987) Co nicated.
6 Ray P K & Singh K P, in *Research in industry*, edited by M ky M J, Srivastava H C, Varsya Bhartendu (Oxford IBH Publishing Co Pvt Ltd), 1987, 168-178.
7 Axford A T, Mc Kerrow C B, Parry Jones A & Le Quene M, *Br J Indust Med*, 33 (1976) 65.
8 Le Quene P M, Axford A T, Mc Kerrow C B & Perry Jo A, *Br J Indus Med*, 33 (1978) 72.
9 Thruman G B, Sunims B G, Goldstein A L & Killian D J *Toxicol Appl Pharmacol*, 44 (1976) 617.
10 Sagha G K & Alarie Y, *Toxicol Appl Pharmacol*, 50 (1979) 533.
11 Tse C S & Pesce A J, *Toxicol Appl Pharmacol*, 51 (1979) 39.
12 Charles J, Bernstein A, Jones B, Jones D J, Edwards J H, Seal RME & Seaton A, *Thorax*, 31 (1976) 127.
13 Saxena A K, Singh K P, Nagale S L, Gupta B N, Ray P K, Srivastava R K, Tewari S P & Singh Randhir, *Indian J Exp Biol*, 26 (1988) 173.
14 Koller L D, Roan J G & Brouner J A, *J Environ Pathol Toxicol*, 3 (1980) 407.
15 Bobak D & Whisler R, *J Immunol*, 125 (1980) 2764.
16 Hocking W G & Golde D W, *N Engl J Med*, 301 (1979) 580.
17 White W G, Morris M J, Sugden E & Zapata E, *Lancet* (1980) 756.
18 Howard R J, *Curr Prob Surg*, 17 (1980) 275.
19 Saxena A K, Singh K P, Dutta K K, Gupta G S D, Nagale S L, Misra A, Zaidi S I A & Ray P K, *Indian J Exp Biol*, 26 (1988) 195.
20 Crowle A J, *Ad Immunol*, 20 (1975) 197.

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METHYL ISOCYANATE SURVIVORS OF BHOPAL — SEQUENTIAL FLOW VOLUME LOOP CHANGES OBSERVED IN EIGHTEEN MONTHS FOLLOW-UP*

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ABSTRACT

One hundred and thirteen symptomatic victims of methyl isocyanate (MIC) toxicity were investigated by peforming maximal expiratory flow volume measurement sequentially for 18 months. Expiratory flows remained low and inspiratory flows declined. Abnormalities of the flow volume loop included saw tooth (12.3%), doming (16.5%), hesitation (25.8%) in inspiration and doming (30%) and concavity (53.6%) during expiration. The changes persisted despite improved spirometric values.

Introduction

Deadly methyl isocyanate gas spewed forth on Second December 1984, from the Union Carbide factory at Bhopal. Pulmonary function assessment was carried out on 113 subjects who were victims of Bhopal gas tragedy and these have been reported[2,3]. Many of these victims were assessed with careful flow volume loop measurements at intervals of three, six, 12 and 18 months, in order to elucidate more fully the ventilatory and small airway abnormalities. Toluene diisocyanate has been documented to induce occupational asthma[1]. Oxygen exchange, blood gases and immunoglobulin changes were also studied in the methyl isocyanate victims. A group of 160 normals were assessed for flow volume to establish our laboratory normal standards. Flow rates were evaluated for evidence of airway obstruction[4]. Flow rates and lung volumes have been correlated[5]. Extensive data are available for flow volume abnormalities in chronic obstructive pulmonary disease[6]. Expiratory flow volume curves have been worked out for South Indian men[7].

Material and Methods

The victims who presented to our hospital in Bombay from the first week onward from Bhopal formed the material for the study.

All persistently symptomatic subjects underwent a detailed questionnaire (British Medical Research Council Respiratory Questionnaire, 1976 edition) and had detailed physical examination, chest radiography, spirometry (Stead-Wells, W.E. Collins), bronchodilator assessment with nebulised salbutamol, minute ventilation, arterial blood gases at rest and on exercise, CoHb and MetHb (co-oximeter, Instrumentation Laboratories Inc.)

In relevant patients, electrocardiography (number 52), mechanics of breathing (48), bronchoalveolar lavage (12), rheumatoid and antinuclear factor (32), lung biopsy (8) and immunological antibody studies (93) in association with Dr. M.H. Karol, University of Pittsburgh were also carried out.

Flow volume norms were derived with Fleisch No. 3 Pneumotachograph and Hewlett Packard X-Y Recorder in 160 normals. The latter recorded a maximal flow volume loop after a calibration with H.P. calibration set up (No. 09911) and oscilloscope. Typical two loops were measured after 10 records were faithfully reproduced. The volumes were derived by integration. All normals were healthy nonsmokers belonging to ages 17-60 years and had normal spirometric values by Indian standards reported earlier[8].

For comparing our standards, 25 chronic obstructive pulmonary disease and 12 interstitial fibrosing alveolitis patients were also studied similarly. Manual calculations from the loop were carried out for expiratory and inspiratory flow rates at Peak V^{25}, V^{50}, V^{75} and related ratios. Multiple regression analyses for age, sex, height and weight were undertaken to derive prediction equation in normals. For comparison, the data on MIC exposed subjects has been also presented

* Based on Preliminary report presented at the Fifth National Congress on Respiratory Diseases at Jaipur, December 1985.

TABLE—1

RESPIRATORY FLOW RATES IN NORMAL INDIANS

(a) Expiratory flow rates:

Age group/yrs.	No.	PEFR	V_{E25}	V_{E50}	V_{E75}
Male:					
17–20	20	8.02±2.29	7.38±2.05	4.67±1.42	2.37±0.92
21–30	40	8.03±1.22	7.42±1.27	4.69±0.92	2.18±0.56
31–40	20	7.93±1.36	7.37±1.30	4.68±0.86	1.99±0.34
>40	10	6.79±0.70	6.42±0.66	4.04±0.46	1.72±0.28
Total	90	7.33±1.54	6.84±1.48	4.32±0.99	1.96±0.53
Female:					
17–20	17	5.13±0.61	4.68±0.65	3.55±0.43	2.04±0.47
21–30	26	6.01±1.05	5.66±1.04	3.97±0.90	2.19±0.57
31–40	15	5.49±0.85	5.13±0.76	3.46±0.85	1.83±0.46
>40	12	5.12±0.98	4.73±0.89	2.96±0.59	1.56±0.41
Total	70	5.55±0.92	5.16±0.97	3.61±0.84	1.99±0.54

(b) Inspiratory flow rates:

Age group/yrs	No.	PIFR	V_{I25}	V_{I50}	V_{I75}
Male:					
17–20	20	5.29±1.51	4.34±1.32	5.17±1.40	4.62±1.38
21–30	40	5.19±1.05	4.28±0.92	5.05±1.05	4.48±0.96
31–40	20	4.99±0.98	4.07±0.80	4.86±0.88	4.29±0.79
>40	10	4.26±0.51	3.57±0.54	4.17±0.59	3.68±0.52
Total	90	4.70±1.13	3.88±0.97	4.59±1.09	4.03±1.01
Female:					
17–20	17	3.48±0.51	3.04±0.50	3.47±0.40	2.91±0.54
21–30	26	3.90±0.80	3.90±0.80	3.86±0.78	3.25±0.77
31–40	15	3.72±0.97	3.32±0.89	3.56±0.91	2.93±0.84
>40	12	3.13±0.65	2.74±0.53	3.05±0.58	2.33±0.52
Total	70	3.58±0.80	3.16±0.80	3.55±0.79	2.95±0.79

as real and standardised (for age 30 and Height 1 cm in males and 160 cm in females) values.

Results

The flow volume loops for males (Fig. 1) a females (Fig. 2) are similar to those of North Americ subjects, but lower in absolute values.

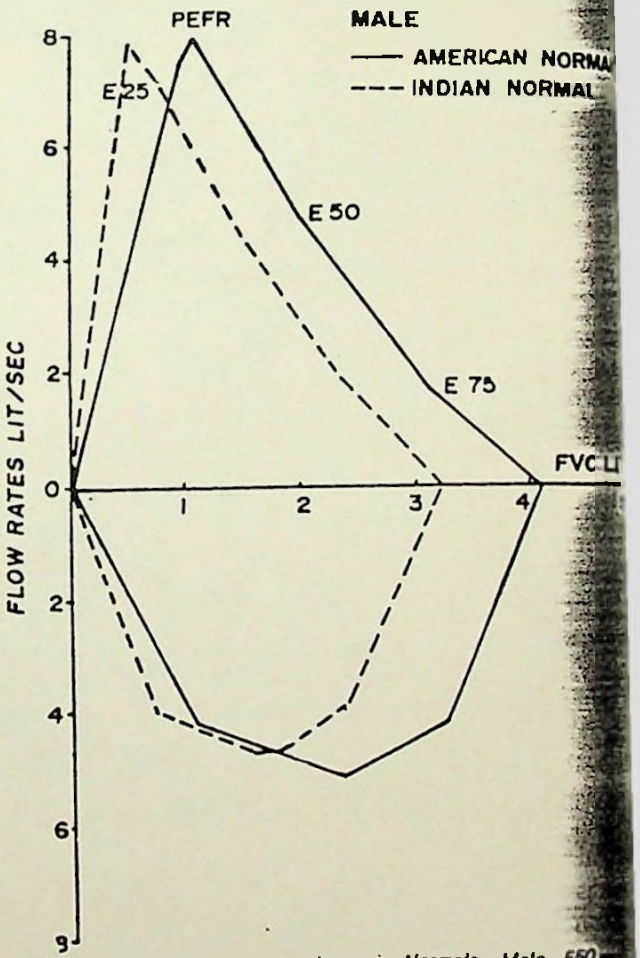

Fig. 1 Mean Flow Volume loops in Normals—Male. E50— Flow rates at 50% on the expiratory slope.

TABLE—2

PREDICTION FORMULAE FOR NORMALS

	Male	*Female*
PEF	2.024—0.025 Age + 0.043 Ht. —0.010 Wt.	5.964—0.028 Age + 0.010 Ht. +0.038 Wt.
V_{E25}	3.612—0.021 Age + 0.027 Ht.	4.525—0.024 Age + 0.036 Wt.
V_{E50}	3.085—0.013 Age + 0.010 Ht.	2.204—0.031 Age + 0.020 Wt.
V_{E75}	—0.332—0.016 Age + 0.017 Ht.×	0.975—0.019 Age + 0.011 Ht.×
PIF	0.014—0.021 Age + 0.034 Ht.	1.341—0.020 Age + 0.027 Wt.
V_{I25}	—1.91 —0.01 Age + 0.041 Ht.	1.720—0.016 Age + 0.024 Wt.
V_{I50}	0.061—0.019 Age + 0.033 Ht.	2.128—0.023 Age + 0.024 Wt.
V_{I75}	—0.733—0.020 Age + 0.032 Ht.	2.55 —0.029 Age + 0.021 Wt.

× Unreliable prediction formula. Prediction by Weight (kg) has large errors in males for all parameters; Prediction by height (for all parameters in females except PEF, has large errors.

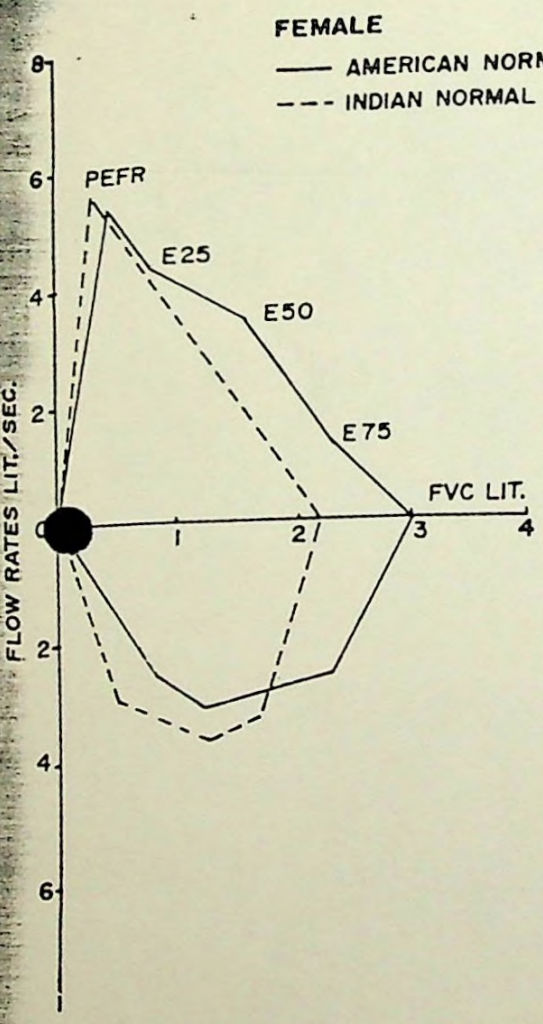

Fig. 2 Mean Flow volume loops in normals-female.

Clinical observations on MIC exposed subjects

Of 113 subjects 23% were females, 33% smokers and 10 below 20 years and 41 between 21-30 years. The distance from the Union Carbide factory at the time of exposure was upto one kilometre in 16 subjects and beyond two kilometres in nine subjects. Severity of immediate clinical illness was derived by number and severity of respiratory, eye, abdominal and neurologic symptoms, duration of hospitalisation, history and duraton of complications (e.g.unconsciousness). Thus 30 had initially mild, 57 moderate and 26 severe illness. This showed a significant relationship to distance from the factory ($P < 0.05$). By the time we studied these subjects, 69% had improved, 4% had worsened and 27% had unchanged disability.

As shown in Table. 3, the initial (first assessment) symptomatology was: eye congestion (43%), blurred vision (29%), abdominal pain (30%), cough (98%), sputum (42%), dyspnoea (97%), chest pain (69%), muscle weakness (32%), poor memory (29%) and poor concentration. (11%). Later dyspnoea (mainly on exertion), cough, chest pain, muscle weakness, poor concentration, and body pains persisted as prominent symptoms. The mean respiratory symptom score in 113 subjects initially studied was 2.92 ± 1.55, it was 2.08 ± 1.25 at six months and 2.8 ± 1.2 at 18 months.

Radiologically at initial stage, 36% showed 1-2 zone, 24% 5-6 zone involvement. Mainly linear (82%), punctate (37%) and reticulonodular (27%) interstitial deposits were seen. In 15 to 19% changes consistent with those due to emphysema, and cardiac enlargement were seen while pleural scars (21%) and patchy consolidations (4%) were other findings. After three months 38% showed improvement and 16% showed worsening. At 18 months, 97% still showed linear, 23% punctate and 22% reticulonodular deposits. Initially carboxyhaemoglobin was raised (mean, 5.97%) in 94.3% of 70 subjects tested and methaemoglobin was (in 111 subjects), mean 1.76% high (over 1%) in 83% subjects. After three months, (but not when studied in few days) both the CoHb and MetHb values came down ($P < 0.05$ and $P < 0.01$ for respective two parameters).

Oxygen uptake values at rest were low initially but continued to improve significantly later. FVC and PEF showed significant improvement but not $MEFR_{0.25-0.75}$. The radiographic extent of zone involvement initially was related to methaemoglobin reading ($P < 0.05$). Also initially clinical respiratory score showed a relation to FVC ($P < 0.05$) and FEV^1 ($P < 0.01$).

Flow rates in MIC exposed subjects

Generally, the values are lower than for normals, particularly for inspiratory flow rates (Table 4). Over next three to six months most flow rates improved significantly (except V_{E75} which showed only insignificant changes).

The improvement in flow rates continues over the follow up for PEF and V_{I25} (18 months). After an initial improvement for PIF, V_{I50} and V_{I25} (after 12 months), V_{E50} (after three months), V_{I75} (after six months) all the flow rates decreased at 18 months. In contrast, forced vital capacity has improved steadily after six months significantly ($P < 0.05$ for all differences).

The trends for males (nonsmokers and smokers) and females are shown graphically in Fig. 3-5. For

TABLE—3

SEQUENTIAL SUMMARY OF CLINICAL AND FUNCTIONAL BACKGROUND OF MIC EXPOSED SUBJECTS (n:113).

	Initial (%)	At 12 months (%)
(a) Clinical:		
Cough	98	69
Sputum	42	58
Dyspnoea	97	93
Chest pain	69	48
Muscle weakness	32	53
Poor memory	29	20
Poor concentration	11	50
Eye congestion	43	3
Blurred vission	29	9
Abdominal pain	30	15
Abnormal lung signs	51	—

	Initial (%)	At 12 mon (%)
(b) Chest radiograph:		
Normal	2	0
Cardiac enlargement	19	1
Emphysema, Pulm. hypertension	15	0
Pleural scar	21	2
Consolidation	4	0
Interstitial Deposits:		
Linear	82	65
Punctate	37	40
Micronodular/Nodular	27	12
Reticular	27	8

	Initial (70) Mean ±SD	After 3 months (73)
(c) Carboxyhaemoglobin %	5.97 < ± 11.1	2.26 ± 1.42 P<0.05
Methaemoglobin (111) %	1.76 < ± 0.74	0.89 ± 0.73 P<0.01

(d) Oxygen uptake resting:

	Mean±SD
Initial	197.5±58.5
3 months	222.5±51.0
12 months	246.6±91.4

(e) Blood gases and PH	Initial	12 months
pO_2	100.8±12.1	94.2±11.9
pCO_2	33.3±3.7	33.5±3.1
pH	7.49±0.04	7.43±0.03

(f) Spirometric indices:

Period (month) (No.)	0(113)	3(80)	6(76)	12(94)	18(74)
Standardised to age 30 and ht. (167cm.M; × = P<0.05. 160 cm .F).					
FVC × Lit.	2.31 ± 0.59	2.38 ± 0.53	2.40 ± 0.58	3.10 × ± 0.77	3.17 × ± 0.56
FEV_1/FVC %	96.7 ± 3.9	95.9 ± 4.1	95.5 ± 8.9	86.2 ± 9.0	81.4 ± 8.5
PEF × Lit/min.	389±123	448±107	448±103	487±105	610±240
MEFR (0.25-0.75)	200.8 ± 81.5	213.8 ± 94.5	212.9 ± 93.6	185.4 ± 117.3	173.6 ± 56.3

TABLE—4

SEQUENTIAL CHANGES IN RESPIRATORY FLOWRATES IN MIC EXPOSED SUBJECTS

Standardised° Values Period (months). (number of subject)	0(52)	3(79)	6(70)	12(73)	18(64)
PEF Lit/Sec.	5.71 + ±1.79	6.69 + ±1.86	6.75 + ±1.71	6.88 ±2.20	6.95 + ±1.94
PIF Lit./Sec.	3.40° ±1.08	3.60 ±1.20	3.67 ±1.32	3.92° ±1.62	3.29 ±1.11
V_{E25} L/Sec.	5.47° ±1.64	6.12° ±1.83	6.08 ±1.80	6.13 ±2.24	6.06 ±1.95
V_{E50} L/Sec.	3.48° ±1.13	3.86 + + ±1.42	3.72 ±1.22	3.62 ±1.49	3.46° ±1.32
V_{E75} L/Sec.	1.90 ±0.67	2.10 ±0.93	1.99 ±0.74	1.90 ±0.88	1.78 ±0.79
V_{I25} L/Sec.	2.47° ±0.94	2.83 ±1.01	2.98 ±1.10	3.47° ±1.45	3.02° ±1.00
V_{I50} L/Sec.	3.23° ±1.04	3.48 ±1.18	3.45 ±1.25	3.77° ±1.56	3.17° ±1.01
V_{I75} L/Sec.	2.85 ±0.93	3.05 ±1.01	3.25 ±1.20	3.14 ±1.39	2.77 ±1.0
FVC Lit.	2.30 × ±0.70	2.19 ±0.56	2.20 ±0.55	2.48 ±0.75	2.68 × ±0.97

° to height 167 cm (M) and 160 cm(F) and age 30 years.
X, °, +: P<0.05 + + Significant increase upto 3 months and then decline upto 18 months: P<0.05
† NS P<0.05 for females only. †: P<0.05 ": NS
§ Significant increase upto 6 months and then decline at 18 months: P ∠ 0.05

male smokers (in six, 15, 14, 11 and nine subjects on the respective occasions Fig. 3) the initial pattern

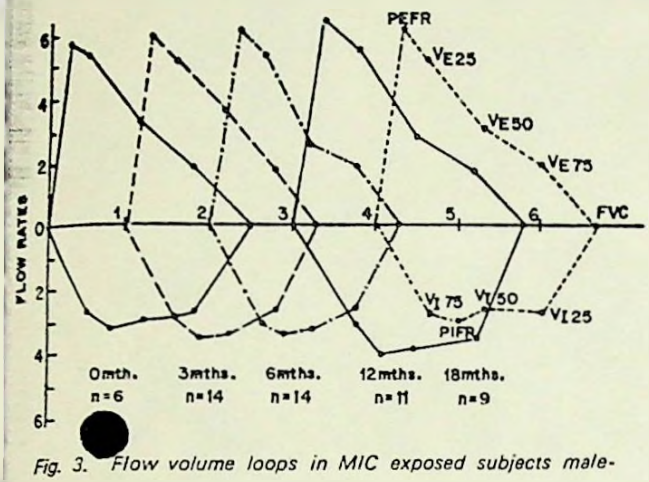

Fig. 3. Flow volume loops in MIC exposed subjects male-smoker (see text)

shows low flow rates particularly on inspiration. There is inspiratory hesitation developing at 18 months and obstructive small airway pattern on expiration at six to 12 months. Some improvement seen at three to six months declined later.

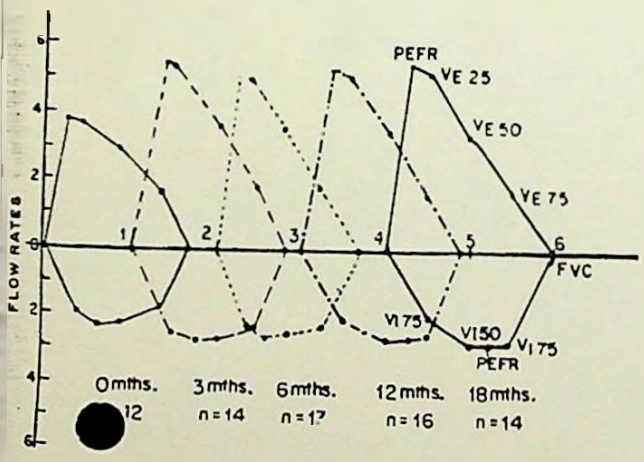

Fig. 4. MIC exposed subjects male non-smoker (see text)

Fig. 4 shows the results in male non-smokers (in 34, 50, 43, 46 and 41 subjects respectively). This group has higher flow rates. There is no evidence of inspiratory hesitation but concavity on descending expiratory limb is seen at 12 and 18 month stage.

Fig. 5 shows the results in females all non-smokers (in 12, 14,13,16, and 14 subjects respectively). This group has lower readings generally while expiratory flows show some improvement, inspiratory flows do not improve to that degree. However there is no evidence of concavity on the expiratory curve.

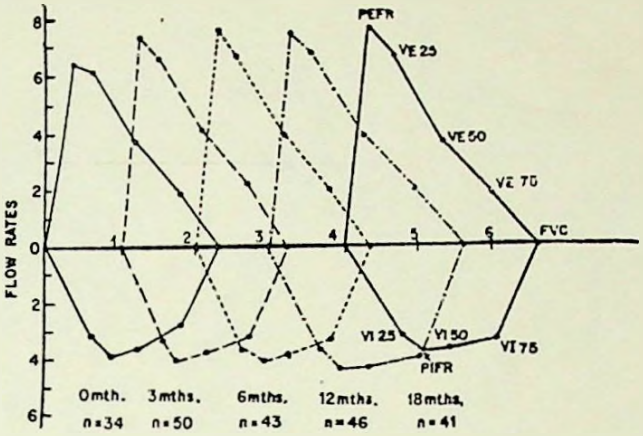

Fig. 5. Flow volume loops in MIC exposed subjects-Non-smokers, Female (see text)

TABLE—5

SEQUENTIAL PATTERN OF ABNORMALITIES FOR FLOW VOLUME LOOP IN MIC EXPOSED SUBJECTS

Period (months)	0	3	6	12	18
No.	52	79	70	73	64
(a) Inspiration:					
Normal	*45.4*	*44.3*	*42.8*	*30.1*	*59.7*
Sawtooth	*12.3**	*18.7*	*30.0*	*15.0*	*0**
Doming	*16.5**	*12.5*	*35.7**	*21.8*	*8.3**
Hesitation	*25.8*	*31.3*	*42.8*	*32.8*	*34.7*
(b) Expiration:					
Normal	*19.6*	*15.6*	*50.0*	*10.9*	*12.5*
Doming	*30.0*	*31.3*	*35.7*	*63.4*	*48.6*
Concavity	*53.6*	*59.8*	*14.3*	*43.8*	*41.6*

* $P < 0.05$

Table. 5 shows the nature of abnormalities in flow volume loop in MIC exposed subjects. These abnormalities were categorised as hesitation (poor starting of inspiration with fluctuations due to incoordination), sawtooth (indicating incoordination of inspiratory movement due to variable intra or extrathoracic central airway obstruction)[8], doming (inability to sustain peak flow indicating fixed central airway obstruction)[9] and expiratory concavity (indicating small airway disease)[6]. We did not find any correlation between clinical symptoms, spirometric values and flow rate restriction in MIC exposed subjects.

At the initial stage 45.4% inspiratory and 19.6% expiratory loops were normal (Table 5). While this proportion did not improve for expiration, there

is insignificant increase of normal pattern (59.7%) for inspiratory loop at 18 months. Similarly frequency of sawtooth and doming abnormalities significantly reduced at 18 months. However proportions showing hesitation on inspiration (suggestive of mild airway incoordination) did not change. The expiratory abnormalities continued unchanged but there was transistory improvement at six month stage.

It is true that lower FVC values may account for lower flow rates, in our population (in comparison to the U.S. subjects[6]) we do not think this is the whole explanation. The inspiratory and expiratory flow patterns could be considered related to mechanical properties of lung (mainly elastic recoil); but these are also effort dependent.[5]

Discussion

We have found larger S.E. (by two to three times) for prediction formulae than those by Bass, possibly indicating that in comparison to spirometry, the utility of F-V loop for flow rates is lower in our population. The limitations in using such prediction norms for flow rates viz. population selection, technical limitation, cooperation, and standardisation of methods have been stressed by Knudson et al[13] earlier. Perhaps the pattern of flow volume loop may indicate airway abnormalities more definitively. While expiratory flow rates would reduce earlier in obstructive airway disease, inspiratory flow rates would decrease as the disease progresses. Jordanoglou and Pride[10] and Kryger[9] have analysed causes of abnormal pattern particularly on inspiration. Variable intra and extrathoracic airway changes, airway distortion, laryngeal or upper airway obstruction and small airway disease have been implicated for various abnormalities observed. However the actual proof for any of the causes is still not available.

Significant restrictive functional changes (oxygen exchange studies), airway inflammation and distortion (bronchoscopy) increased cellular exudation (broncho-alveolar lavage) as well as bronchiolitis and alveolitis (biopsy) have been documented[2]. While we have observed some improvement in spirometry, oxygen exchange and blood gases, the decreases in flow rates seem to be progressive. This has been marked in 11 of 99 subjects studied showing positive immunologic (RAST, IgE and ELISA, IgM, IgA) antibodies specific to MIC (being reported seperately; Karol M. H., et al 1986). The changes in our flow volume studies have been similar to those observed by Alarie et al[14] in guinea pigs after exposure to MIC. This group also found that abnormal flow patterns persisting beyond six weeks upto eight months. We have not found overt evidence of pure obstructive lung disease and large reversibility in contrast to reported occupational asthma in exposure to toluene diisocyanate[3].

Conclusion

The pattern of chronic respiratory disability showing flow volume reductions alongwith restrictive lung damage with alveolitis on acute exposure to MIC observed by our group with improvement in some aspects suggest the need for more extensive and long term studies amongst the survivors.

Acknowledgement

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REFERENCES

1. Kamat, S.R., Mahashur, A.A., Tiwari, A.K.B. et al. (1986). Early observations on pulmonary changes and clinical morbidity due to the isocyanate gas leak at Bhopal. J. Postgrad. Med. *31*, 63-72.
2. Kamat, S.R., Patel, M.H., Kolhatkar, V.P., Dave, A.A. and Mahashur, A.A. (1986). Sequential respiratory changes in those exposed to the gas leak at Bhopal. Ind. J. Med. Res. *83*, (to be published).
3. White, W.G., Sugden, E., Morris, M.J. and Eapata, E. (1980). Isocyanate induced asthma in a car factory. Lancet. 1, 756-760.
4. Morris, J.F., Koski, A. and Breese, J.D., Normal values and evaluation of forced expiratory flow. (1975). Ame. Rev. Respir. Dis. *111*, 755-762.
5. Hyatt, R.E., Schilder, D.P. and Fry, D.L. (1958). Relationship between maximum expiratory flow and degrees of lung inflation. J. Appl. Physiol. *13*, 331-336.
6. Bass, R. (1973). The flow volume loop: normal standards and abnormalities in chronic obstructive pulmonary disease. Chest. *63*, 171-176.
7. Singh, H.D. (1984). Mean Expiratoy flow volume curve in South Indian men. Lung India *2*, 293-296.
8. Kamat S.R., Sarma, B.S., Raju, V.R.K. et al. Indian norms for Pulmonary Functions (1977). J. Assoc. Phys. India. *25*, 531-540.
9. Kryger, N., Bode, P., Antia R., and Anthonisen, N. (1974). Diagnosis of the upper and central airways obstruction. Am. J. Med. *61*, 85-93.

10. Jordanoglou, J. and Pride, N.B. (1968). A comparison of maximum inspiratory and expiratory flow in health and in lung disease. Thorax. *23*, 38-45.

11. Kamat, S.R., Tyagi, N.K., Rashid, S.S A. (1982). Lung function in Indian adult subjects. Lung India. *1*, 11-21.

12. Jain, S., Pande, R.S., Ahuja, S.K. (1984). Pulmonary studies in Myasthenia Gravis. Indian J. Med. Rsearch. *79*, 806-812.

13. Knudson, R J., Lebowitz, R.D., Helberg, C.J. and Burrows, B. (1983). Changes in the normal maximal expiratory flow volume curve with growth and aging. Ame. Rev. Respir. Dis *127*, 725-734.

14. Alarie, Y., Ferguson, J.S., Stock, H.P., Woyel, D.A. and Schaper, M. (1986). Sensory and pulmonary irritation of methyl isocyanate in mice and pulmonary irritation and possible cyanide like effects of methyl isocyanate in guinea pigs. Environ. Hlth. Persp. (In press).

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Effects of Inhaled Sublethal Concentration of Methyl Isocyanate on Lung Mechanics in Rats

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Rats exposed for 30 min to a concentration of 137 ppm (1/3 LC_{50}) of methyl isocyanate (MIC) showed respiratory distress. After 24 hr of exposure the animals were free from distress. There was no external haemorrhage and the lung body weight index did not show presence of pulmonary edema in these rats. Characteristics of pressure-volume curves drawn from the excised air filled lungs, phospholipids content of the lung lavage, minimum dynamic surface tension and histomorphological studies showed that the lung surfactant was not affected. However, the lungs of these rats developed emphysema as was evident by decreased maximum dynamic surface tension, increased total lung capacity, greater quantity of air trapped in the lungs at the end of deflation process and significant increase in the compliance and specific compliance. Lung emphysema was further confirmed by histophatological studies. Besides, there was very marked inflammatory reaction in the alveolar septa and the walls of capillaries were damaged. Red blood cells were spilled in the interstitial and alveolar air spaces. The results show that sublethal concentration of inhaled MIC damage lung parenchyma and produce emphysema. These seem to be lingering effects of MIC independent of its pulomonary edema inducing capability.

Methyl isocyanate (MIC) a highly reactive, volatile, flammable and toxic liquid causes corrosive damage to tissues exposed by inhalation and topical application[1]. Intoxication by inhalation produces pulmonary edema, mottled red lungs, dyspnea and respiratory failure associated with inflammation and squamous metaplasia of respiratory epithelium[2]. Exposure to sublethal concentrations of MIC may produce increased secretion, cough, pain in breathing and obstruction in airspaces causing temporary respiratory distress. However, no attempt has been made to study the effect of inhaled sublethal concentrations of MIC on the morphology and lung mechanics in experimental animals. Being an industrial chemical, most of the studies have been confined to generate data on perceptible and irritant concentrations for human subjects[3] and the lethal concentrations by systemic and inhalation routes of administration in experimental animals[4,5].

The present study has been designed to find the effect of inhaled MIC on the lung mechanics of rats independent of pulmonary edema and haemorrhage.

Materials and Methods

Male Wistar rats weighing 85-100 g raised in the Animal House of the Establishment and fed on standard Hind Lever Feed were used in the present study. Ten animals were used in each treated and control group, except for histomorphological studies where lungs of three rats were processed.

Methyl isocyanate (MIC) was synthesized in the Chemical Laboratories of this Establishment by reaction of acetylchloride with sodium azide in the presence of phase transfer catalyst.

With a view to minimising the risk of exposure of the investigators, the rats were exposed to MIC in a specially designed static exposure chamber[6]. Two rats at a time with loading complement of less than 1.0% were exposed for 30 min to vapours of MIC. Seven µl of MIC was spontaneously evaporated in a small glass test tube attached to the inhalation chamber and the monitored concentration of MIC was 0.32 mg/l (137 ppm). This is slightly less than 1/3 LC_{50}. Calculated LC_{50} (using the static chamber for 30 min exposure) was 1.08 mg/l (95% of confidence limit 0.89-1.3 mg/l which is equal to 465 ppm. The control rats were also confined for the same duration to the environment of the exposure chamber but without MIC.

After 24 hr of exposure, the rats were bled to death through a cut in the abdominal aorta under pentobarbital sodium anaesthesia.

Lung body-weight index (LBI)—The lungs were removed, freed from adhering tissues, blood and weighed. The LBI was calculated by the formula:

$$\text{LBI} = \frac{\text{Lung weight}}{\text{Body weight}} \times 100$$

Dynamic surface tension—Dynamic surface ten-

sion, maximum (γ max) and minimum (γ min) of lung homogenates was determined[7]. Lungs freed from adhering tissues and blood were homogenised in cold normal saline. The homogenate was filtered through cotton, diluted with normal saline to 100 ml/g lung tissue, poured in the trough of the Wilhelmy Assembly and allowed to age for 40-50 min at 27°C. Thereafter the surface film was compressed cyclically from a maximum of 55 cm^2 to a minimum of 19 cm^2 at the rate of 4 cycles/min for about 30 min. The surface tension area loops were recorded on a X-Y recorder.

Pressure-volume curves (P-V curves)—Excised rat lungs were cannulated through the trachea and degassed under vacuum. Subsequently, they were inflated with air at the rate of 1 ml/min to a maximum pressure of 40 cm H_2O and then deflated (1 ml/min) to 0 cm H_2O intrapulmonary pressure. The lung volumes at each measured pressure point were used to construct P-V curves. The stability index (SI), expansion index (EI), total lung capacity (TLC or V_{max}) and the volume of air at 0 cm H_2O pressure (V_0) and 5 cm H_2O pressure (V_5) were compiled from the P-V curves[8,9]. The slope of the deflation curve between 55 to 80% of TLC was taken as the specific compliance and from that compliance was calculated (compliance = specific compliance × TLC/100)[10].

Lung phospholipids—The lungs *in situ* were perfused with cold normal saline through the pulmonary circulation to remove the blood from the pulmonary capillary bed. Thereafter, the trachea was cannulated and the lungs were lavaged with 5 ml cold normal saline. The process was repeated 5 times. The lipids, in the pooled lavage, were extracted and fractionated for phosphatidylcholine and phosphatidylethanolamine by Thin Layer Chromatography[11,12]. Total phospholipids (TPL), phosphatidylcholine (PC) and phosphatidylethanolamine (PE) were estimated spectrophotometrically[13].

Histomorphological studies—Excised lungs of rats were inflated with air to an intrapulmonary pressure of 10 cm H_2O. The trachea was ligated and the whole lung was fixed in the buffered formaline. Representative samples of tissue from various lobes of the lungs were cut into small pieces and processed for routine histological examination.

Mean values for each group of animals were tested for significance by Student's t test. A level of significance of $P < 0.05$ (two tailed) was chosen.

Results

Gross effects—All the MIC exposed rats developed temporary respiratory distress (mouth breathing). After 24 hr, the animals were apparently normal with no symptom of respiratory distress.

Lung body weight index—LBI of experimental and control rats (10 animals in each group) was 0.59 ± 0.02 and 0.69 ± 0.03 respectively. The difference was marginally significant ($P < 0.05$).

Dynamic surface tension—Significant decrease in γ max ($P < 0.01$), γ min and stability ratio ($P < 0.05$) was observed in MIC exposed rats (Table 1).

Pressure volume curves—The mean of the P-V curves is shown in Fig. 1. Summary of results obtained from the analysis of P-V curves is presented in Table 2.

In MIC exposed rats, the total lung capacity and the volume of air trapped in the lungs at the end of deflation process were markedly increased ($P < 0.001$).

The stability index, expansion, index, the volume of air retained during deflation (% TLC) compliance and specific compliance were significantly increased from the control values.

Table 1—Pulmonary Dynamic Surface Tension and Stability Ratio of Control and MIC Exposed Rats

[Values are mean ± SE of 10 animals in each group]

Group	Surface tension (dynes/cm)		Stability ratio (SR)
	Minimum (γ min)	Maximum (γ max)	
Control	23.0 ± 1.00	41.0 ± 1.90	0.56 ± 0.03
MIC exposed	20.7 ± 0.36[a]	33.4 ± 0.72[b]	0.46 ± 0.03[a]

P value: [a] < 0.05; [b] < 0.01. SR = 2 (γ max − γ min)/γ max + γ min

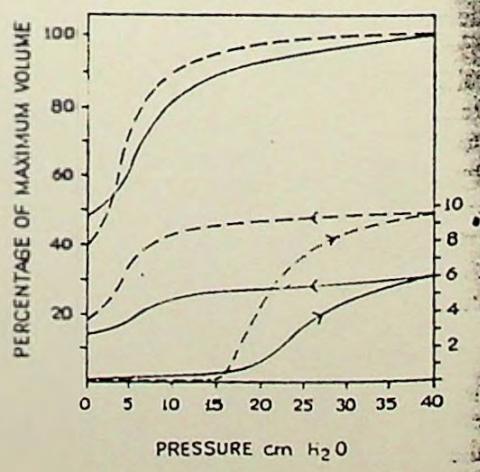


Fig. 1—Pressure-volume curves of control and exposed rats (Lower panel: inflation limb (–) and the deflation limb (–) of control (—) and exposed (—) rats. The upper panel: deflation limbs of control (—) and exposed (—) rats taking TLC as 100%)

Table 2—Results Computed from P-V Curves of Lungs of Rats

[Values are mean ± SE of 10 animals in each group]

Group	Total lung capacity (CC/100 g body wt; TLC)	Volume of air at 0 cm H_2O (CC/100 g body wt; V_0)	Stability index (SI)	Expansion index (EI)	% Air evacuated as of TLC (V_5-V_0)	Compliance	Specific compliance
Control	6.00 ± 0.25	2.86 ± 0.14	1.00 ± 0.02	0.57 ± 0.01	13.3 ± 1.2	0.348 ± 0.031	3.90 ± 0.19
MIC exposed	9.59 ± 0.24[b]	3.74 ± 0.15[a]	1.14 ± 0.02[b]	0.68 ± 0.01[b]	31.3 ± 1.4[b]	0.762 ± 0.045[b]	7.68 ± 0.41[b]

P values: [a] < 0.01; [b] < 0.001; $SI^7 = 2 \times V_5 + V_0/2 \times TLC$

$EI^8 = V_5 - V_d/TLC - V_d$; Vd = 10% of TLC.

Lung phospholipids—Extractable TPL, PC and PE did not show statistically significant deviation from the control values (Table 3). However, TPL and PC tended to be low and percent of PC in TPL was marginally increased.

Histomorphological studies—In Fig. 2 are shown the histomorphological features of a lung from a control rat observed under light microscope. The airways, air sacs and the alveolar septa are normal. The lining of the respiratory bronchioles is intact. On exposure to MIC epithelial cell lining of respiratory bronchioles and the walls of the finer capillaries in the alveolar septa were degenerated (Fig. 3). Alveolar ateleçtasis was seen only in the peripheral alveoli. Larger blood vessels were clogged with blood. However, the most significant finding was that red blood corpuscles were seen scattered in the alveolar septa and alveolar spaces (Fig. 4).

Table 3—Phospholipids Contents (mg/g) of Lung Lavage of Control and MIC Exposed Rats

[Values are mean ± SE of 10 animals in each group]

Group	Total phospholipids (TPL)	Phosphatidylcholine (PC)	Phosphatidylethanolamine (PE)	% of PC in TPL
Control	1.79 ± 0.13	1.35 ± 0.13	0.21 ± 0.03	73.7 ± 4.05
MIC exposed	1.55 ± 0.13[NS]	1.26 ± 0.26[NS]	± 0.03[NS]	77.8 ± 2.52[NS]

NS: not significant

Discussion

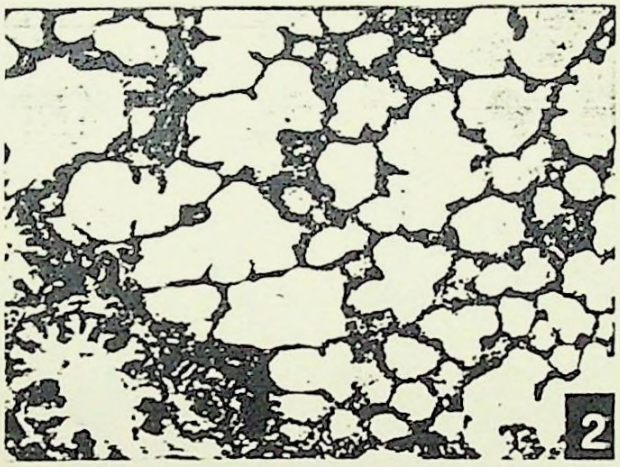
Pulmonary edema and haemorrhage are commonly occurring gross effects induced by agents including MIC, which have profound irritant effect on the respiratory tract[1,14,16]. The presence of edematous fluid and blood in the air spaces of the lung may mask the underlying mechanisms involved in causing changes in the lung mechanics by such agents.

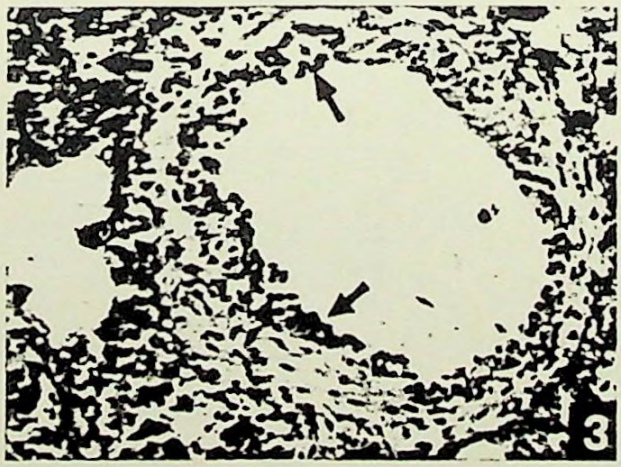
Unlike lethal exposure to MIC where LBI increases due to pulmonary edema and haemorrhage, the lungs of rats in the present study (1/3 LC%) were free from these effects as is evident by no increase in LBI. The reduced LBI observed here may be due to lesser intake of water (including food) as these animals were under acute respiratory distress for a few hours after exposure to MIC. Hence, it is reasonable to assume that the findings on P-V curves, dynamic surface tension and TPL were primarily due to the effects of MIC on the lungs independent of the influence of accumulated edematous fluid and blood. The marginal decrease in LBI of MIC exposed rats was possibly due to the trauma as these rats did not take food and water during the 24 hr post exposure period.

It is evident that MIC, in the concentration and duration of exposure used, had no destabilizing effect on the alveoli, mediated through the lung surfactant system since the TPL, PC were not decreased significantly. Furthermore, γ min was not increased. The histomorphological studies also did not reveal alveolar atelectasis.

Significant increase in TLC, V_0, sp. compl. and SI computed from P-V curves of lungs of rats exposed to MIC indicated presence to emphysema. This contention is further substantiated by the finding that dynamic maximum surface tension (γ max) was appreciably lowered in the homogenates prepared from these lungs. Emphysematic lungs have been reported to retain large volumed of air (increased V_0) and had relatively lower maximum dynamic surface tension[15]. Emphysema was also seen in the histomorphological studies carried out in the MIC exposed lungs.

The most striking finding of the histomorphological studies under light microscopy was the presence





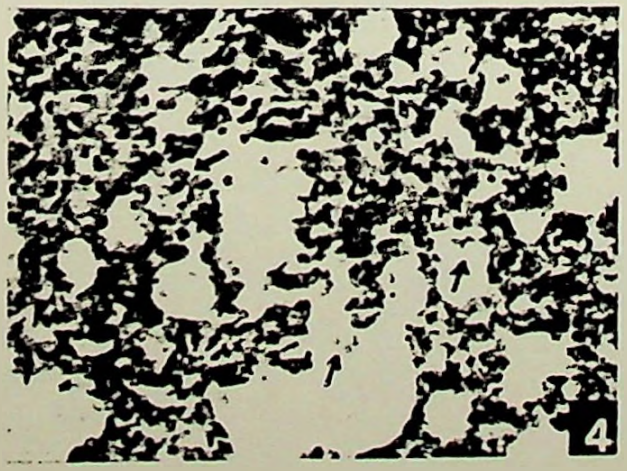


Fig. 2—Normal lung from a control rat showing ho... alveolar pattern with normal alveolar septa, ducts and bro... oles with intact epithelial lining H & E × 280. Fig. 3—Nor... ed epithelial lining of bronchiole has undergone exc... mage. H & E × 300. Infiltration of inflammatory cells in the ... spaces (arrow) seen. Fig. 4—Infiltration of large number ... blood cells in the alveolar spaces is seen. H & E × 380. (Alve... septa are inflammed. There is no evidence of oedematous ... The emphysema is also very marked)

of randomly distributed red blood cells in the alveolar airspaces. Other histomorphological findings were inflammatory response with severe interstitial pneumonia and disintegrated walls of finer pulmonary capillaries in the lungs of rats which had recovered from initial respiratory distress. Furthermore, there was no externally visible haemorrhage in these lungs. Pulmonary injury induced by sublethal concentration of MIC appears to have adverse effects on the lung mechanics independent of its pulmonary edema inducing effect.

Acknowledgement

The authors are indebted to Dr P.K. Ramachandran, Director, for keen interest and guidance. Thanks are due to Mr Hari Afley for secretarial assistance.

References

1 Kimmerle G & Eben A, *Arch Toxicol*, **20** (1964) 23.
2 Fowler E H & Dodd D E, *Fundam Appl Toxicol*, **6** (1986) 756.
3 Rye W A, *J Occup Med*, **15** (1973) 306.
4 Smyth H F Jr, Carpenter C P, Weil C S, Pozzani U C, Striegel J A & Nycum J S, *Am Ind Hyg Ass J*, **30** (1969) 470.
5 Vernot E H, Macewen J D, haun C C & Kinkead E R, *Toxicol Appl Pharmacol*, **42** (1977) 417.
6 Vijayaraghavan R & Kaushik M P, *Indian J Exp Biol*, **25** (1987) 531.
7 Mendenhall R M, *Rev Sci Instrum*, **42** (1971) 878.
8 Gruenwald P, *J Appl Physiol*, **18** (1963) 665.
9 Srivastava R K, Sachan A S & Sharma S K, *Indian J Exp Biol*, **14** (1976) 428.
10 Szapiel S V, Fulmer J D, Hunninghake G W, Elson N A, Kawanami O, Aerrans V J & Crystal R C, *Am Rev Respir Dis*, **123** (1981) 680.
11 Folch J, Lees M & Sloane-Stanley G H, *J Biol Chem*, **226** (1957) 497.
12 Abramson D & Blecher M, *J Lipid Res*, **5** (1964) 628.
13 Bartlett G R, *J Biol Chem*, **234** (1959) 466.
14 Nemery B, Dinsdale D, Sparrow S & Ray D E, *Br J Ind Med*, **42** (1985) 799.
15 Clements J A, Hustead R F, Johnson R P & Gribetz I, *Appl Physiol*, **16** (1961) 444.
16 Ferguson J S, Schaper M, Stock M F, Weyel D A & Abra... *Toxicol Appl Pharmacol*, **82** (1986) 329.

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ffects of methyl isocyanate on rat muscle cells in ulture

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STRACT Since the Bhopal disaster, in which the causal agent was methyl isocyanate (MIC), xposed ple have complained of various disorders including neuromuscular dysfunction. In an ttempt to gain some information about the response of muscle tissue to MIC its effects were vestigated in cells in culture isolated from muscle of 2 day old rats. After treatment with a range of MIC concentrations (0·025–0·5 μl/5 ml culture) the total number of nuclei of the two main cell types fibroblasts and myoblasts) and the number of nuclei in muscle fibres (myotubes) were recorded. At wer doses which had little effect on the total number of nuclei, the formation of muscle fibres—that fusion of muscle cells—was prevented as the proportion of nuclei in myotubes was decreased. At gher oses both cell types were killed. This would suggest either an effect on muscle differentiation a selective toxicity towards myoblasts. The observations were supported by light and electron icroscopy.

the industrial disaster in Bhopal, India, in 1984 the akage of 40 tons of methyl isocyanate (MIC) gas om a pesticide plant claimed over 2500 lives and left estim ted 15 000 people suffering from a variety of lment Three months later 50 000 of the people ontinued to have disorders of the lungs and eyes and ceration of the gastrointestinal tract and many omplained of kidney, liver, and neuromuscular dysnction. Since then, various researchers have been athering toxicological information.

Kimmel nd Eben showed that MIC was highly ritant to skin and mucosae and produced pulmonary dema.[2] Nemery *et al* suggested that MIC at high oncentrations probably caused peracute death roug reflex inhibition of breathing and rats survivg exp sure exhibited narrowing of the airways and aemorrhagic pulmonary oedema.[3] The lesions were paired rapidly but renewed inflammation and perironchial fibrosis were apparent. Nemery *et al* also owed that cyanide intoxication is not concerned in he clinical syndrome seen after exposure to MIC nor oes sodium thiosulphate protect rats from the acute nd subacute effects of MIC.[4] Cellular immunity has been shown to be slightly compromised by exposure to MIC.[5]

In investigations of the genotoxic potential of MIC it has been reported to be non-mutagenic in the standard Ames[6] and preincubation assay,[7] and Anderson *et al* showed that negative results were also obtained in the Ames test when urine from animals treated with MIC was tested.[8] Shelby *et al* have also reported a negative sex linked recessive lethal test in Drosophila.[9] Positive results have been shown, however, for point mutation in the mouse lymphoma assay and in Chinese hamster ovary cells in culture for chromosome aberrations and sister chromatid exchange in the absence of rat liver S9.[7] In vivo there was marginal evidence of an effect for chromosome aberrations and sister chromatid exchanges in the B6C3F$_1$ hybrid mouse but results were not reproducible.[7] The results from the bioassay using the same strain are eagerly awaited since they would give an indication of the potential carcinogenicity of MIC in man.

In man there have been complaints of neuromuscular dysfunction[1] but little or nothing is known about the toxicology of such effects. In an attempt to gain some information on muscular function we examined the effect of MIC administered to rat muscle cells in culture.

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Materials and methods

ISOLATION OF MUSCLE CELLS CULTURE TECHNIQUES

Cells were isolated from muscle of 2 day old rats by treatment with dispase solution. Cells were cultured in Dulbecco's medium with 10% fetal calf serum in 25 cm^2 flasks and placed in a CO_2 gassing incubator at 37°C until treatment.

TREATMENT REGIMEN

MIC was added directly to the flasks and left for two hours and then replaced by fresh medium. Flasks were then incubated until fixation.

EXPERIMENTAL APPROACHES

Various experimental protocols were used. The first study with treatments on days 1 and 2 was to determine a suitable range of doses for the final study in which the compound was administered at days 2, 4, and 6 in culture in order to treat the myoblasts at different stages of development.

CELL FUSION COUNTS ON STANDARD CULTURES

Counts were made on 10 fields for each culture. The total number of nuclei of all cell types and the number of nuclei in myotubes was recorded.

Results

STUDY 1

In the first study (doses ml:0·25, 0·5, 1, and 2 μl treated at day 1 and fixed at day 9) only cultures with 0·25 μl MIC had any cells (data not shown).

In those cultures treated on day 2 with lower dose treatments and fixed at day 9 there was little effect on total nuclei but myotube formation was much reduced at 0·25 and 0·05 μl (table 1).

Table 1 *Study 1: the effect of MIC treatment on day two of culture on the growth and fusion of rat muscle cells (fixed on day nine)*

MIC (μl 5 ml culture)	Total nuclei per culture (10^6)	Nuclei in myotubes (10^6)	% Fusion
0	3·20	0·51	15·0
0·009	3·43	0·57	14·9
0·028	3·70	0·40	10·8
0·083	2·85	0·00	0·0
0·25	2·88	0·03	1·0

[a]Calculated from counts made on 10 representative microscope fields per culture using duplicate cultures.

[b]% Fusion = $\frac{\text{Nuclei in myotubes } (10^6)}{\text{Total nuclei per culture}} \times 100$

To achieve these low concentrations, MIC was dissolved in culture medium and then diluted.

Table 2 *Study 2: the effect of MIC treatment at various times after culture establishment (day 0) on the growth and fusion of rat muscle cells*

Day of treatment	MIC (μl 5 ml culture)	Total nuclei per culture (10^6)	Nuclei in myotubes (10^6)	% Fusion
1[c]	0	2·76	0·36	13·0
	0·025	3·11	0·24	7·7
	0·05	2·79	0·28	10·0
	0·125	2·43	0·12	4·9*
	0·25	2·01*	0·04	2·0*
	0·5	0·0*	0·0	0·0*
4[d]	0	2·89	0·51	17·6
	0·025	2·46	0·36	14·6
	0·05	3·15	0·64	20·3
	0·125	2·15	0·02	0·9*
	0·25	2·65	0·02	0·8*
	0·5	0·0*	0·0	0·0*
6[d]	0	2·89	0·51	17·6
	0·025	3·37	0·70	20·8
	0·05	0·59*	0·02	3·4*
	0·125	0·0*	0·0	0·0*
	0·25	0·0*	0·0	0·0*
	0·5	0·0*	0·0	0·0*

[c] Fixed on day 5
[d] Fixed on day 8
Footnotes as for study 1
* $p = < 0·05$ (values compared with control means).

STUDY 2

The results for control cultures were similar to those in the first study with mean values of 2·76 × 10^6 nuclei (13·0% fused) on day 5 and 2·89 × 10^6 (17·6% fused) on day 8 (table 2). Because of the larger number of control cultures in this study it was possible to observe the variation obtained with this counting method. The total nuclei were relatively consistent (a range of 2·2 to 3·48 × 10^6) but fusion was more variable (8·1 to 25·6%).

With treatment on day 1, nuclear counts were significantly reduced at 0·25 μl (more than two standard deviations below the control mean) and fusion was significantly lower at both 0·25 and 0·125 μl in cultures fixed on day 5. No cells were present after treatment with 0·5 μl.

After treatment on day 4 and fixation on day 8 total counts were similar to controls at doses up to 0·25 μl but fusion was inhibited at 0·125 and 0·25 μl. There were no cells present after treatment with 0·5 μl.

Treatment on day 6 resulted in a decrease in nuclear counts and fusion at a dose of 0·05 μl and cells were destroyed at higher concentrations.

MICROSCOPY

On days 4 and 6 at the light microscope level the control cultures consisted of a mixed population of long strap-like multinucleate muscle cells and mononucleated spindle cells resembling fibroblasts (fig 1). The fibroblasts showed many fatty cytoplasmic vacuoles while both fibroblasts and muscle cells had dense cytoplasmic inclusions. At the electron microscope level two cell types could be readily identified by

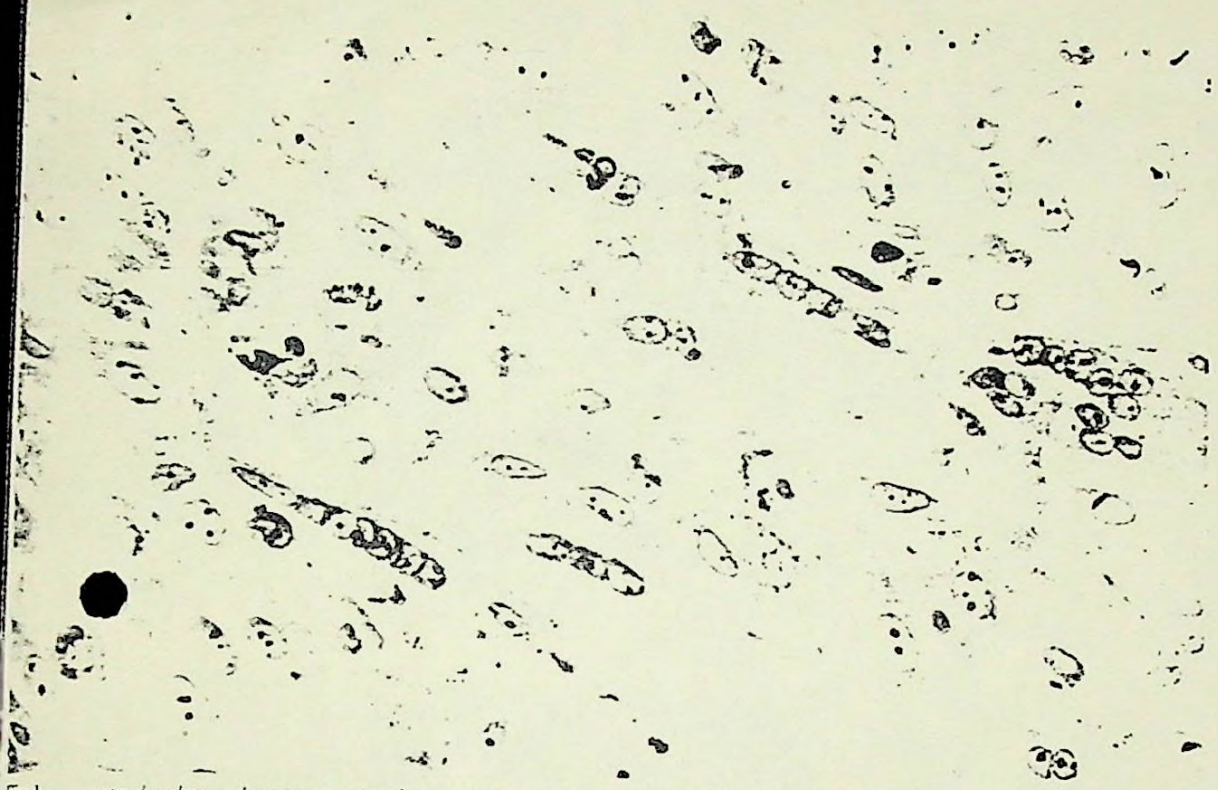
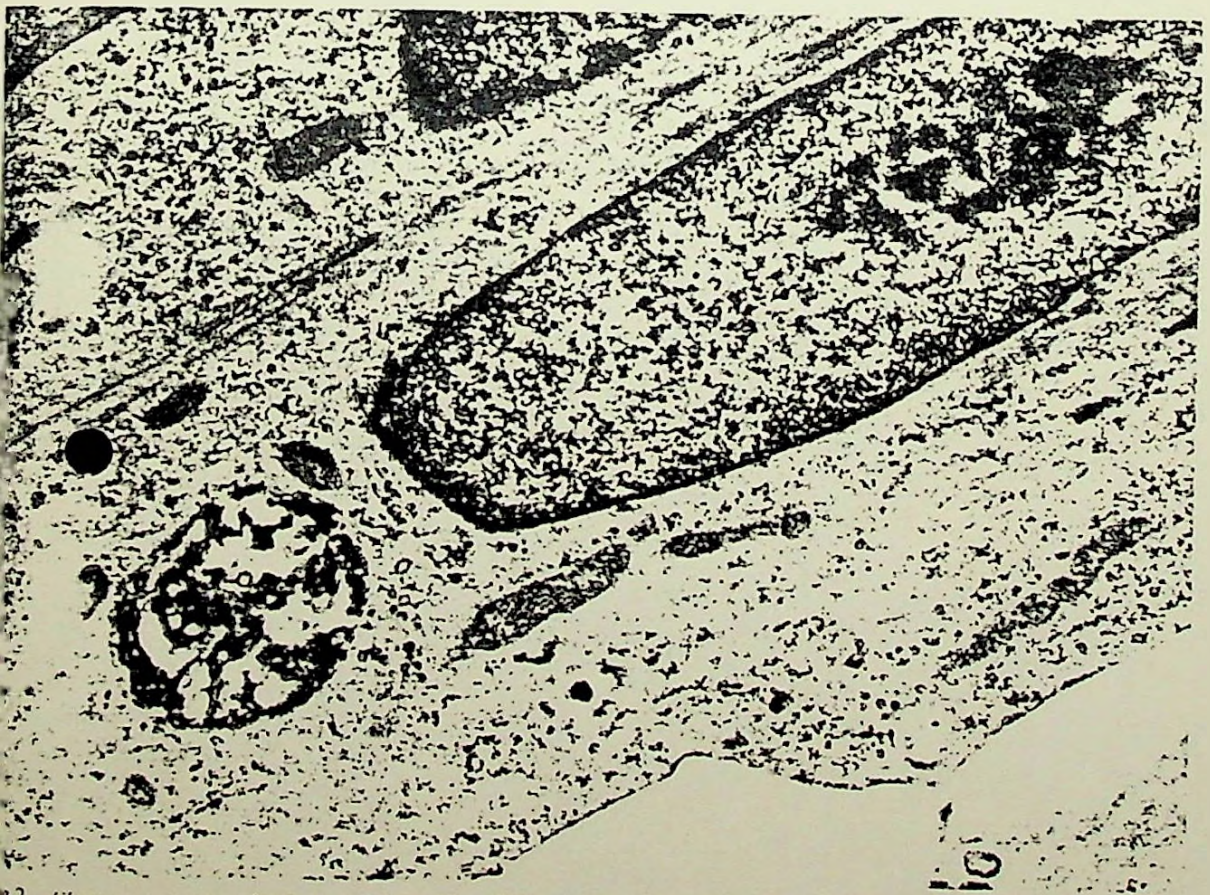


Fig 1 ...ntrol culture showing a mixed population of syncytial strap-like muscle cells and spindle fibroblasts. (Tolu... blue × 300).



g 2 Electron micrograph of a control muscle cell showing well organised fibrils and an autophagic vacuole. ...).

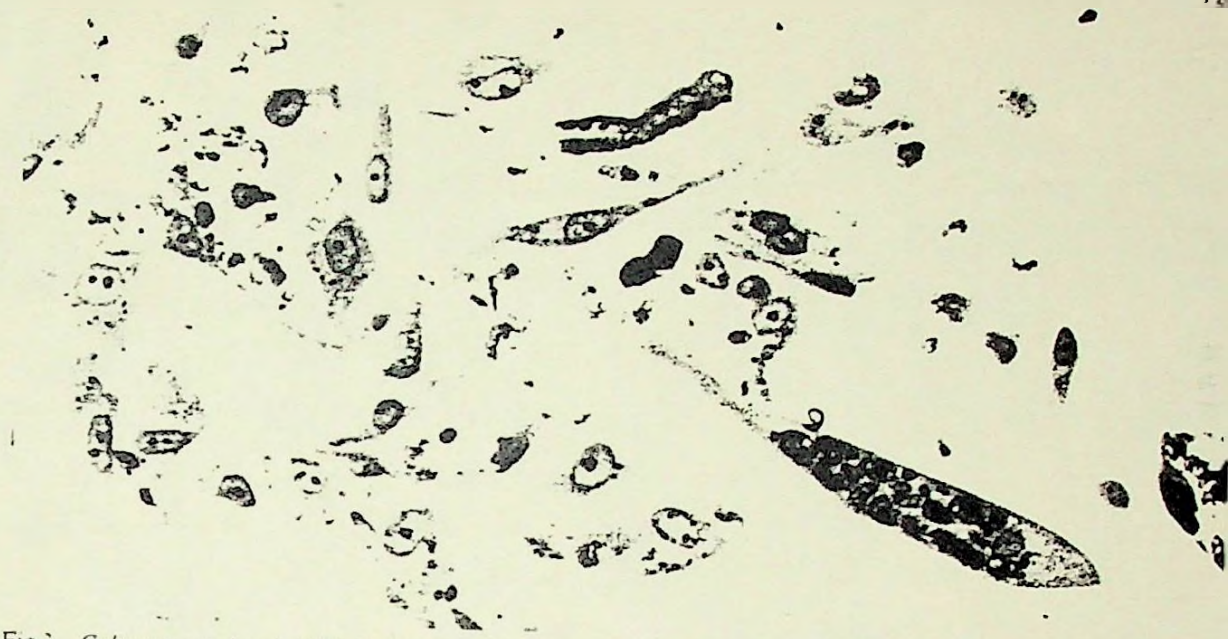


Fig 3 *Culture treated with MIC (0·125 µl 5 ml culture) showing a sparse population of muscle cells, spindle cells, and necrotic debris. Toluidine blue × 300).*

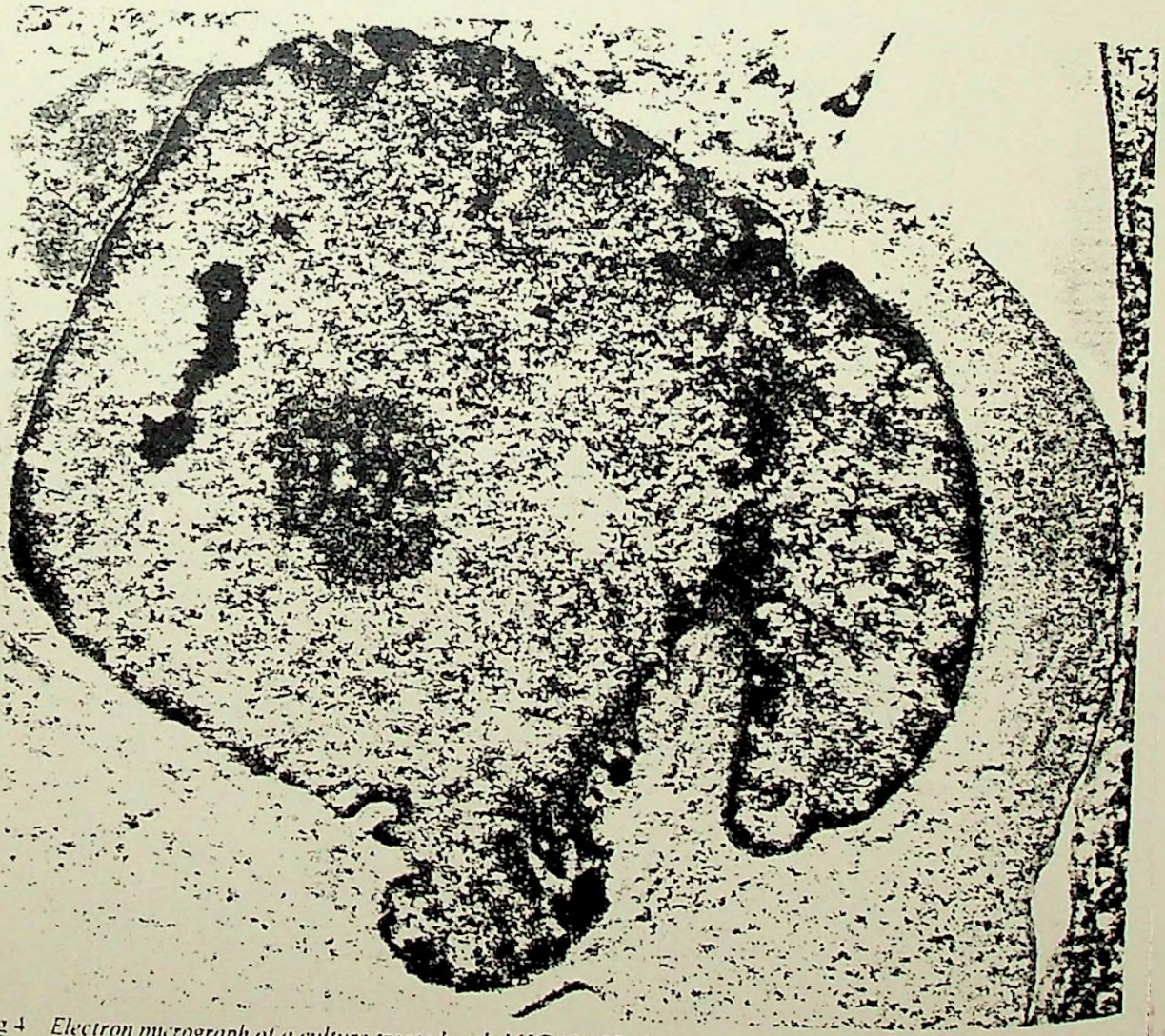


Fig 4 *Electron micrograph of a culture treated with MIC (0·125 µl/5 ml culture) showing a muscle cell with a simplified profile and part of a normal fibroblast. × 20 000).*

Fig 5 *Culture treated with MIC (0·25 µl 5 ml culture) showing necrotic cells and no intact muscle cells. Toluidine blue × 300).*

6 *Electron micrograph of a culture treated with MIC (0·25 µl/15 ml culture) showing residual necrotic cell. 15 000).*

the characteristic fibres of the muscle cells and the form of the endoplasmic reticulum of the fibroblasts (fig 2).

Cultures receiving 0·05 *µl* MIC on day 4 were essentially the same as the controls at both the light and electron microscope level. At a treatment level of 0·125 *µl* MIC changes were observed. The syncytial muscle cells were much less abundant and necrotic cells were scattered throughout the culture (fig 3). At the electron microscope level the mononuclear cells could be identified as both myoblasts and fibroblasts. The myoblasts were less abundant than the fibroblasts and often had a simplified profile (fig 4). The fibroblasts were similar to the controls.

At the treatment level of 0·25 *µl* MIC all the cells of the culture were necrotic and there was no evidence of the syncytial muscle cells (figs 5 & 6).

Discussion

The results suggest that MIC is toxic for a mixed culture of fibroblasts and muscle cells but that the myoblasts appear to be more susceptible to the acute toxic action of MIC. Even at relatively low concentrations it was toxic. Lower doses tended to prevent myotube formation since the proportion of nuclei in myotubes was decreased at concentrations which had little effect on total number of nuclei as shown at doses of 0·125 *µl* and 0·25 *µl* after treatment on day 1 and fixation on day 5 and at a dose of 0·125 *µl* and 0·25 *µl* after treatment on day 4 and fixation on day 8. At higher doses the total number of nuclei were reduced. These findings would suggest either an effect on muscle differentiation or selective toxicity to myoblasts. Aft treatment on day 6 effects were more severe because this stage cells were detaching as monolayer sheets the higher doses and both cell types appeared to killed.

The importance of the findings for muscle tissue the intact animal is not clear where muscle cells a under the control of the nervous system. It may speculated that MIC would directly affect musc tissue and prevent differentiation as shown in the vitro situation. If MIC were also to directly attack t nervous tissue this might superimpose a lack coordination in addition to a lack of differentiation

References

1 Zaidi M. Bhopal and after. *Am J Ind Med* 1986;9:215-6.

2 Kimmerle R, Eben A. Zur Toxicitat von Methylisocyanat un dessen quantitativer Bestimmung in der Luft. *Arch Toxic* 1964;20:235-41.

3 Nemery B, Dinsdale D, Sparrow S, Ray DE. Effects of meth isocyanate on the respiratory tract of rats. *Br J Ind Me* 1985;42:799-805.

4 Nemery B, Sparrow S, Dinsdale D. Methyl isocyanate: thiosu phate does not protect. *Lancet* 1985;ii:1245.

5 Silver M, Germalec D, Vore S, Tucker A, Luster M. Immunotox cology of methyl isocyanate in mice. *The Toxicologist* 1986;6:3 (Abstract No 311.)

6 National Toxicology Programme. Research Triangle Park, Nort Carolina: Department of Health & Human Services, 1983:5-6 (Tech bulletin No 9.)

7 Shelby MD, Zeiger E, Caspary W, Mason J, Tice R. Genetic toxicity of methyl isocyanate (MIC). *The Toxicologist* 1986;6: 28. (Abstract No 312.)

8 Anderson D, Blowers SD, Nemery B. Investigation of the Ame test of urine samples from rats exposed to methyl isocyanate. *Br J Ind Med* 1986;43:566-7.

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Effect of Exposure to Toxic Gas on the Population of Bhopal: Part I—Epidemiological, Clinical, Radiological & Behavioral Studies

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Systematic follow-up studies were conducted to obtain information on after effects of exposure to the toxic gas on the population of Bhopal. A total of 1109 subjects were studied on location in Bhopal. This paper deals with the general demographic data and the findings of clinical, radiological and behavioral studies. The majority of the subjects had symptoms pertaining to respiratory, cardiovascular, gastrointestinal and musculoskeletal systems. The prevalence of symptoms pertaining to respiratory and gastrointestinal system was significantly higher in population residing within 4 km than among those residing beyond that distance from the factory. A number of clinical signs in regard to various systems were observed. 739 Subjects (81.8%) out of a total of 903 subjects studied radiologically showed no abnormality in their chest X-rays, whereas 91 subjects (10.1%) showed radiological changes suggestive of definite pathology like pulmonary tuberculosis, pneumonitis, emphysema, etc. Specific radiological changes were observed in 73 subjects (8.1%) and out of these, in 48 subjects the abnormal findings on X-ray was because of exposure to the toxic gas. In 17 subjects radiological changes suggestive of old disease were observed. Since the symptoms appeared only after exposure to the toxic gas it was presumed that in those cases the old pathology was aggravated because of the exposure to the gas. Pneumonitis was commonly seen in the right zone. There was no correlation between the radiological picture and the clinical presentation. The cases with clear-cut pneumonitis had no evidence of toxaemia. The results of behavioral studies revealed that memory mainly visual perceptual and attention/response speed alongwith attention/vigilance were severely affected in the exposed population.

Background Information

Bhopal (Lat. 23.16N, Long. 77.24°E), the capital of Madhya Pradesh, having a population of approximately 9 lakhs and located 568 km south of Delhi, met with the world's worst chemical disaster on the night of Sunday, 2 December 1984, and early hours of Monday, 3 December 1984. The storage tanks in Union Carbide Plant which were storing methyl isocyanate (MIC) leaked leading to a variety of health problems in the population of Bhopal. Henceforth, we will mention in all our subsequent papers this exposed population as the population exposed to toxic gas released from the Union Carbide Plant. This is a local pesticide plant situated at the north east and about 2 km from the Bhopal railway station. The escaping gas quickly spread as a fogged cloud over a large and thickly populated areas of about 7-8 km mostly to the south and east of the plant (Map 1).

The effect on the people, living in the shanty settlements just over the plant fence, was immediate. Many died in their beds, never knowing what killed them. Others staggered out of their homes, blinded and choking to die on the street within minutes. Many more succumbed later on, some after reaching hospitals and emergency aid centres. The worst affected areas of the city are situated 1-3 km from the factory (Map 2).

The early acute effects beside the heavy death toll were frightening particularly of long term prognosis. As the cloud of gas swept over parts of the city, stricken residents experienced a burning sensation in the eyes, nose and throat. Many ran out of their homes, coughing and gasping for a breath. Some had begun uncontrolable vomiting. Others collapsed right away whereas ulcers formed on the cornea of some victims leading to blindness.

Most deaths have been attributed to various forms of respiratory distress. In some people, the toxic gas caused such massive internal secretions that their lungs became clogged with fluids, while in others, spasmodic constrictions of bronchial tubes led to suffocation.

Many of those who survived the first day were found to have impaired lung function. Their breathing was shallow, making it difficult for them to get enough oxygen. The tragic occurrence gave rise to certain important questions like what are the long term effects with regard to the physiological functioning of various systems and the severity of the damage caused to the population exposed to the gas.

Introduction

The systematic studies were carried out to obtain

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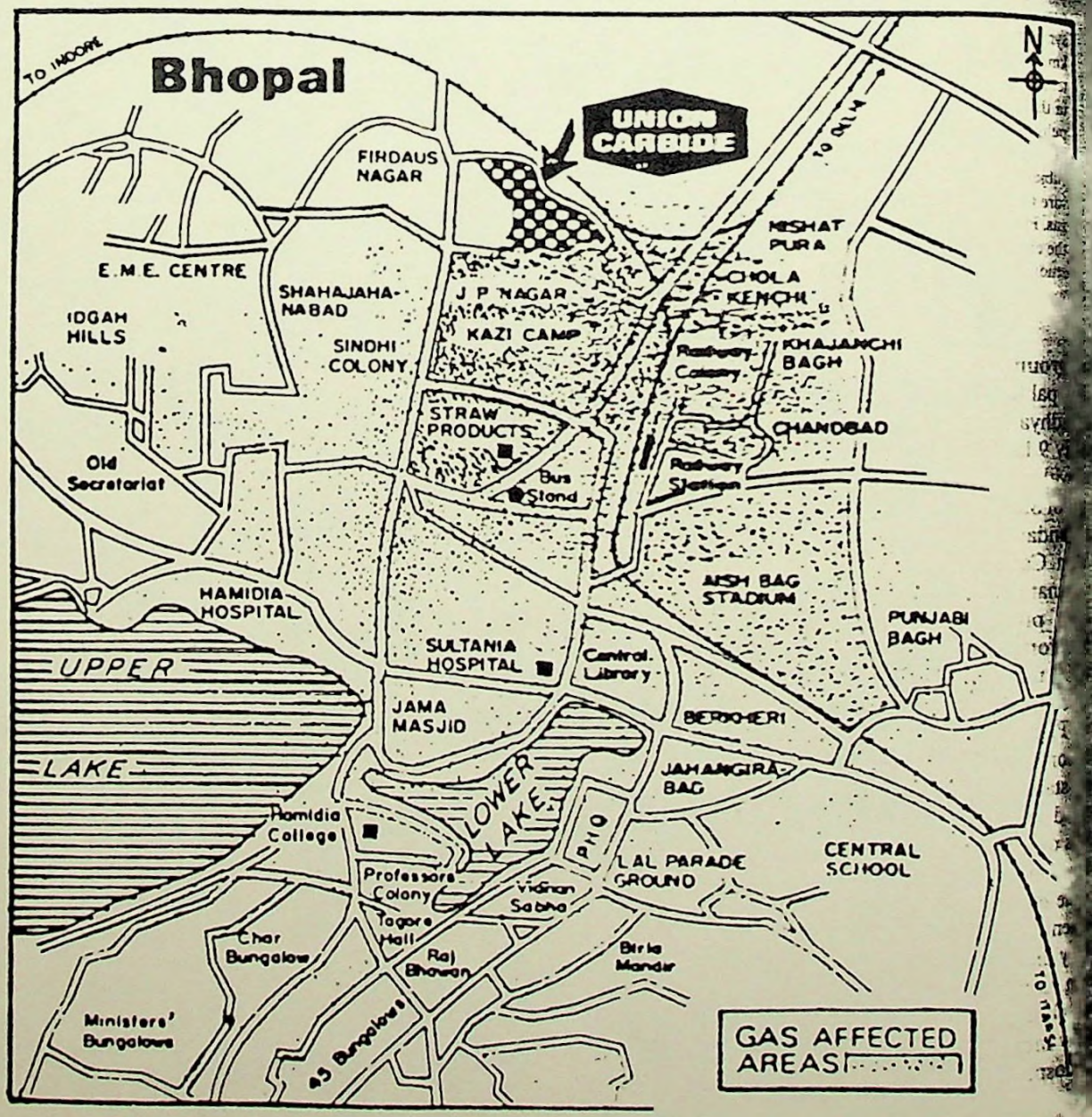
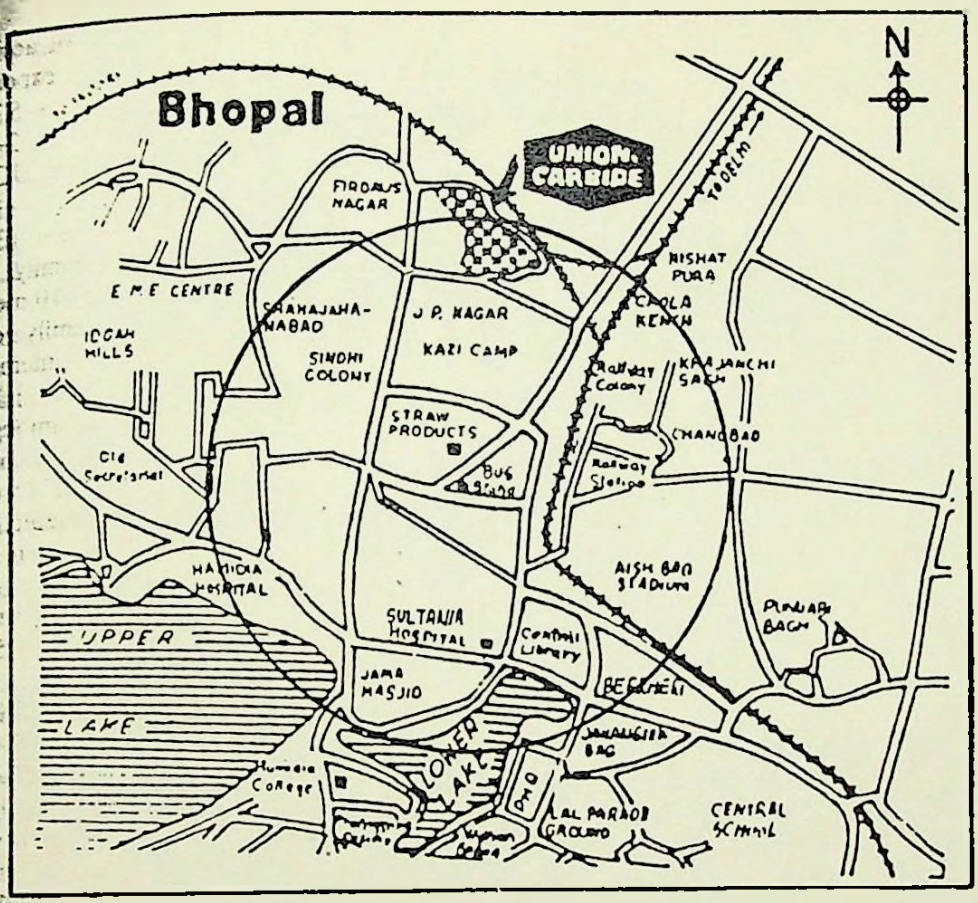
information on after effects of exposure to the toxic gas on the population of Bhopal. The scientific information on toxicity of the gas and related compounds is available to some extent[1-7]. Some studies have been reported on the toxic effects of toluene di-isocyanate and diphenyl methane di-isocyanate in the literature[8-11]. The present study was undertaken to note clinical, radiological and behavioural changes in the population exposed to the toxic gas.

Materials and Methods

A team of scientists/technicians from ITRC reached Bhopal on 11 February, 1985 to conduct follow-up studies of the population exposed to the toxic gas. This team comprised specialists in medicine, pulmonary physiology, radiology, toxicology, behavioural toxicology, biochemistry, industrial hygiene, immunology, cytogenetics and statistics. The follow-up study centre was started at Noor Manzil Road, Bhopal. The persons exposed to the toxic gas were contacted and requested to come forward voluntarily for getting themselves examined. During the first phase (11 Feb. to 3 March 1985) 685 affected persons of either sex (including children) of various age groups and from different affected areas like Chola Road, Kazi Camp, Railway Station, Chanchbagh, etc. were examined. During the second phase (18 March to 10



Map 1—Map of Bhopal city showing gas affected areas



Map 2—Location of UCIL factory in Bhopal. Maximum number of study subjects were drawn from the areas in the circle

592 persons were covered. The following parameters were studied in each case—History taking, clinical examination, chest X-ray and behavioural studies.

History taking—A complete history including bio-social and occupational background of each subject was recorded on the basis of a questionnaire prepared according to WHO epidemiological-study procedure.

Clinical examination—A complete clinical examination that covered general physical examination, examination of respiratory, cardiovascular, gastro-intestinal, musculoskeletal and central nervous system of the subjects was done.

Radiological examination—A full sized (30×42.5 cm) plate was used for chest X-ray postero-anterior view in 903 exposed persons using a portable X-ray machine International General Electric Company IGE). The X-ray films were read by a panel of experts including a radiologist, a chest disease specialist and an industrial medicine specialist.

Behavioural studies—Psychological tests were conducted in 350 random subjects of either sex representing different socio-economic groups and drawn from different age groups. Children were excluded from this study. The following tests were conducted.

Digit Span Test: This is a subtask of Wechsler Adult Intelligence Scale (WAIS) and Wechsler Memory Scale (WMS) used for testing immediate auditory memory.

Benton Visual Retention Test: This is a visual memory test and also measures attention and visual ability.

Digit Symbol Test (DST): This is also a substask of Adult Intelligence test and is used to measure perceptual motor speed. This is considered as the most valid detector of cerebral dysfunction.

Bourdon Wiersma Vigilance Test: This also measures perceptual motor speed. The score represents perceptual accuracy as percentage.

Simple Reaction Time (SRT): It is the length of time between the onset of a stimulus and execution of motor response. This is a sensitive indicator of toxic effects.

Santa Ana Test: This is used for measuring perceptual motor coordination in terms of manual dexterity.

Personality tests like Rorschach Inkblot and Ey-

senck Personality Inventory (EPI): These are used to detect non intellectual personality disturbances, changes in mood, readiness for affective reactions, neuroticism and the dimension of extroversion/introversion, etc. The above tests were also conducted in control subjects which were equally distributed according to age, sex and socio-economic status, comparable to the exposed population, the only difference being that the subjects of this group were not exposed to any toxic or irritant gas.

Statistical analysis—The following statistical tools were used for the analysis of data pertaining to memory, perceptual motor speed, attention/response speed and manual dexterity.

Analysis of variance: In order to test the significance of the variations among three different groups, viz. control, exposed male and exposed female, the analysis of variance as suggested by R.A. Fischer, was worked out.

Student's t test: This test is applied to find out the significance between two groups. The expression standard error (SE) of the difference between two groups is represented by:

$$SE\,(Diff) = EMS\left(\frac{1}{n_1}+\frac{1}{n_2}\right)$$

where EMS = mean square within groups
n_1 and n_2 denote the sizes of the two groups.

Observations

General profile of the study population—Out of 1109 persons studied 202 (18.2%) were Hindus, 894 (80.6%) were Muslims and 13 (1.2%) belonged to other religions. This high proportion of Muslims in the population under study is because of the fact that most of the residents in the areas worst affected by the gas exposure were Muslims.

Among the population studied, 32.6% were unmarried and 67.4% married. In the age group < 15 years 100%, in 16-25 years 54%, in 26-35 years 2%, in 46-55 years 3% and in the age group above 56 years 3% were unmarried. The rest were married.

As regards food habits 134 subjects (12.1%) were vegetarian while 975 subjects (87.9%) were non-vegetarian.

Out of 1109 subjects, 259 (23.4%) belonged to the nuclear type of family while 850 (76.6%) belonged to the joint type. 29% in age group of < 15 years, 24% in 16-25 years, 25% in 26-35 years, 19% in 36-45 years, 17% in 46-55 years and 20% in more than 56 years age group respectively belonged to the nuclear type. The remaining persons belonged to joint families.

Out of the total population studied (1109 subjects, 671 subjects (60.5%) lived within a distance of 2 km from UCIL factory at the time of accident. This population was worst affected by exposure to the gas. 345 subjects (31.1%), 63 subjects (5.7%) and 30 subjects (2.7%) lived at a distance of 2-4, 4-6 and more than 6 km respectively from UCIL factory (Table 1).

Out of 1109 subjects studied, 386 (34.8%) had fewer than 5 members in the family, while 573 (51.7%) and 150 (13.5%) had 6 to 10 members and more than 11 members in the family respectively. Six to ten members or more than 11 members reflect crowding in the family.

As regards the level of literacy among the study population 32.4% were illiterate, 21.6% had received only primary education, 10.3% were educated up to Junior High School, or Middle, 8.7% up to High School, 11.4% up to Intermediate and 15.6% were degree holders.

In the study population of 1109 subejcts 438 (39.5%) slept in the open while 671 (60.5%) lived in closed (covered) houses.

Out of 1109 cases studied, 993 (89.5%) had a per capita income of Rs 300 or less, 78 (7.0%) had a per capita income of Rs 301-500, 24 (2.2%) had a per capita income of Rs 501-800 and 14 (1.3%) had a per capita income of Rs 801 or more per month (Table 2).

Symptomatology—Of the overall complaints, the majority, i.e. 22.1 and 20.2% respectively were re-

Table 1—Agewise Distribution of Population Living at Various Distances from Union Carbide Factory

Age group years	Distance (km)			
	2	2-4	4-6	6
< 15	112	53	10	1
16-25	170	86	20	14
26-35	152	95	18	6
36-45	102	68	9	8
46-55	70	22	3	1
> 56	65	21	3	—
Total	671 (60.5%)	345 (31.1%)	63 (5.7%)	30 (2.7%)

Table 2—Agewise Distribution of Population on the Basis of Per Capita Income

Age groups (years)	Per capita income (Rs per month)			
	300	301-500	501-800	801
< 15	162	9	2	3
16-25	260	17	10	3
26-35	247	18	4	2
36-45	161	21	4	1
46-55	84	5	3	4
> 56	79	8	1	1
Total	993 (89.5%)	78 (7.0%)	24 (2.2%)	14 (1.3%)

lated to respiratory and ocular system, followed by those related to gastrointestinal system (16.4%) and the musculoskeletal system (12.3%). Psychogenic complaints constituted 11% of the overall symptoms.

Complaints related to cardiovascular system, skin, urinary system, genital system and CNS were only 3.1, 1.2, 0.8, 0.2 and 0.1% respectively. Headache, ear or dental problems and thirst, included in miscellaneous group, formed 10.4% of the total complaints.

Complaints and systems involved were also analysed according to distance from the factory (Tables 3 & 4). The average number of complaints per person showed a decline with the increase of distance from the factory. The prevalence of symptoms pertaining to respiratory and gastrointestinal system was significantly higher in the population residing than 4 km than in population living beyond 4 from UCIL factory.

Sleep was normal in 69.8% subjects while 31.2% complained of insomnia.

Clinical findings—The majority of subjects (66.0%) was of average built whereas 17.5% population had a good physique, 12.5% had a poor general appearance and 4.0% were obese.

Examination of the tongue showed a normal clean tongue in 735 subjects (66.3%) while 309 subjects (27.9%) had a coated tongue. Furring, ulceration, dryness and cyanosis was observed in 0.8%, 0.5%, 4.2% and 0.3% cases respectively.

The conjunctivae were normal in 574 subjects (51.9%) while jaundice noted in 15 subjects (1.4%) and congestion in 338 subjects (16.4%).

During the examination of oral cavity, the teeth and gums were found normal in 790 subjects (71.2%) and 1039 subjects (93.6%) respectively. The incidence of caries was found in 195 subjects (17.6%) and the teeth were missing in 124 subjects (11.2%). Bleeding, hypertrophy and infection of gums were noted in 42 subjects (3.8%), 26 subjects (2.3%) and 2 subjects (0.2%) respectively.

Lymphadenopathy was seen in 95 subjects (8.5%).

Tachycardia (pulse rate of more than 80 beats/min) was found in 67.8% subjects while the remaining 32.2% had a pulse rate of less than 80/min.

163 (14.7%) out of 1109 subjects examined had systolic blood pressure of more than 140 mm Hg and 141 subjects (12.7%) had a high diastolic pressure of more than 90 mm Hg.

Abnormal breath sounds on auscultation were heard in 131 subjects (11.8%). Out of this 5.8% had rhonchi, 3.4% crepts and 2.6% diminished breath sounds. In the rest (88.2%) of the population normal bronchovascular breath sounds were heard in all areas of chest.

Clinical examination of heart and blood vessels did not reveal any significant abnormality in the majority of subjects examined.

Out of the total persons examined, 895 (80.5%) had no abnormality in the abdomen on palpation, while 211 (19.9%) subjects showed mild, tender and

Table 3 : Agewise Number of Complaints and Systems Involved

System Involved*	Age groups ≤ 15	16–25	26–35	36–45	46–55	56	Total %
A	36 (2.5)	68 (2.9)	54 (2.3)	80 (2.5)	21 (2.5)	15 (2.4)	274 (3.1)
B	281(20.1)	549 (23.8)	483(20.7)	317 (22.0)	191(23.5)	142(22.9)	1964(22.1)
C	280(20.1)	316(13.7)	407(17.4)	233(16.2)	123(15.0)	101(16.3)	1460(16.4)
D	171(12.2)	353(1513)	330(14.1)	209(14.5)	209(14.5)	64(15.1)	1295(14.5)
E	10(0.7)	14(0.6)	14(0.6)	19(1.3)	13(1.5)	1 (0.6)	71 (0.8)
F	0	5(0.2)	3(0.1)	6(0.4)	5(0.6)	1(0.2)	20 (0.2)
G	322(23.0)	444(19.3)	460(19.7)	270(18.8)	160(19.6)	144(23.2)	1900(20.2)
H	132 (9.4)	238(10.0)	312(13.3)	150(10.4)	90(11.0)	52(8.4)	1800(20.2)
I	18(1.2)	36(1.5)	20(0.8)	14(0.9)	5(0.6)	10(1.6)	103(1.2)
J	-	6(0.3)	01(0.4)	-	2(0.2)	-	9)0.1)
K	149(10.6)	268(11.6)	249(10.6)	137(9.54)	66(8.0)	59(9.5)	928(10.1)
Total complaints	1399	2298	2333	1435	815	619	6899
Total population studied	182	284 282	174	83	82	1097	
No. of complaints/	7.6	8.1	8.3	8.2	8.8	7.6	-

Figures in parenthesis are the percentage of total complaints

A = Carmovascular; B= Respiratory; C= Gastrointestinal; D= Musculoskeletal; E= Urinary; F = Genital; G= Ocular; H = Psychological; I= Dermatological ; J= Central Nervous system; K = Miscellaneous.

palpable liver. Spleen was found palpable in 5 (0.4%) subjects.

Radiological findings—Radiological findings of 903 X-rays of chest are given below:

Normal	739
Abnormal	164
Group A	48
Group B	17
Group C	8
Pneumonitis	7
Pulmonary tuberculosis	55
Primary complex	7
Chronic bronchitis	4
Emphysema	2
Bronchiectasis	2
Cardiac abnormalities	11
Collapse	2
Effusion	1
Tension bullae	1
Total	903

739 Subjects (81.8%) out of a total of 903 studied radiologically showed no abnormality in their X-rays. X-ray picture of 91 subjects (10.1%) showed radiological changes suggestive of definite disease conditions like pulmonary tuberculosis, pneumonitis, chronic bronchitis, emphysema, etc. Specific radiological changes were observed in 73 subjects (8.1%). These subjects have been classified as follows:

Group A: X-ray showing radiological abnormalities because of exposure to the toxic gas — 48 subjects

Group B: Radiological changes suggestive of old pulmonary disease, but symptoms appearing only after exposure to the toxic gas — 17 subjects (In this category old pulmonary disease is aggravated by exposure to the toxic gas)

Group C: Radiological findings not definitely related to exposure to the toxic gas — 8

Total — 73

The subjects have been divided according to positive findings in respect of symptomatology, X-ray findings, PFT values, and findings on the clinical examination (Table 5).

To screen out malingerers the findings of symptomatology, clinical examination, X-ray and pulmonary function tests were correlated. Those subjects whose chest X-ray did not show any abnormality and whose lung function values were normal where no abnormality was detected on clinical examinatioin were very strongly suspected to be malingers giving a false statement of suffering from respiratory symptoms like cough, breathlessness and/or tightness in chest, etc. for the lure of some compensation or otherwise, their symptoms

Table 5—Distribution of 48 Radiologically Positive Cases with Respect to Symptomatology, Clinical and Pulmonary Function Examination

Sl No.	Symptomatology	Clinical	LFT	Total cases
1	—	—	N	4
2	—	+	N	2
3	+	—	N	20
4	+	+	N	6
5	+	—	Impaired	13
6	+	+	Impaired	3

Table 4 : Distancewise Complaints and System Involved

System Involved	Distance from Factory		
	<2 Kms	2-4 Kms	>4 Kms
A-Cardiovascular	207 (3.5)	60 (2.4)	7(1.3)
B-Respiratory	1311 (22.2)	551(22.3)	102(19.4)
C-Gastrointestinal	957 (16.2)	446 (18.1)	57 (10.8)
D-Musculoskeletal	872 (64.8)	329(13.3)	94(12.9)
E-Urinary	49 (0.8)	20 (0.8)	2(0.4)
F-Genital	11 (0.2)	05(0.2)	4 (0.8)
G-Ocular	1175(19.9)	516(20.9)	108(20.8)
H-Psychological	609 (10.3)	286 (20.9)	80 (15.2)
I-Dermatological	65 (1.1)	24 (1.0)	14 (2.7)
J-Central Nervous System	03 (0.1)	06(0.2)	-
K-Miscellaneous	646 (10.9)	226(9.1)	56 (10.7)
Total	5905	2469	525
Total population survey studied	655	351	91
Average No. complaints/person	9.0	7.0	5.8

Figures in parenthesis are the percentage from total complaints

No.of subjects in over 6 km distance was small hence it was pooled with 4-6 kms category.

...ic in nature consequent on the severe tra... ...essed by them. Such cases need to be stud... ...great detail during follow-up studies.

... (6.8%) subjects showed definite radiological ... of tubercular infection. Of these 55 subjects ... showed findings of pulmonary tuberculosis ... (0.77%) of primary complex. The incidence of ... infection (6.8%) is much more than Na... Tuberculosis Survey[7] findings of 1.8%. ... most of X-rays where findings were sug... of pulmonary tuberculosis showed that they ... cases. There is a strong possibility that ... tuberculosis cases could not survive the ... effects of the gas and those with healed tuber... could.

... other pulmonary and cardiac abnormalities ... in the population exposed to the toxic gas ... similar to such abnormalities in any group of ... population. Seven cases showed presence of ...nitis in their X-rays. Out of these cases in... ...ent of the right lower zone was seen in 5 sub... ...of left lower zone in 1 subject, and of both low... ... in 1 subject, while left-sided pleural effu... ...was observed in 1 subject only. Tension bullae ... the right lower zone in one subject and lung col... ...lapse in two cases—one on the right lower lobe and ... on the middle lobe-were observed. 73 subjects ... showed radiological abnormalities which ... suspected to be related to the exposure to the ... gas. Out of these in 48 subjects the radiological ... observed appeared to have been caused by ... to the toxic gas. The radiological findings ... cases are given in Table 6. The radiological ... observed in these X-rays are definitely ab... but they do not confirm any definite diagno... These findings were haziness seen in different ... of the lungs, hilar prominence, fine mottling, ...lation, etc. The changes are more marked in ... right than in the left lung. Haziness was more ...monly seen in the lower zone (40 X-rays out of ... showing haziness in the right lower zone whereas ... showing haziness in the left lower zone). One case ... haziness in the right upper zone whereas, no ... showed haziness in the left upper zone. Com... the middle zone on right and left sides, it was ...served that 3 cases showed haziness in the right ... zone where 6 cases showed haziness in the ... middle zone. Preponderance of abnormalities, ... haziness, hilar prominence, etc. on the right side ... probably because of the anatomical configuration ... the right and left bronchus. The right bronchus ... wider probably permitted more of the gas to ... the right lung and hence more abnormalities ... observed on the right side. Although haziness ... served in almost all X-rays in this group it was surprising to note that hardly a few subjects presented with fever also.

Hilar shadows were prominent in 47 out of 48 cases in this group. Prominence of the right hilum was more common (in 47 out of 48 cases) whereas left hilum was prominent in 41 out of 47 cases. Costophrenic angle, heart size and trachea did not reveal any significant abnormality. There was no correlation between radiological picture and clinical presentation. The cases with clearcut pneumonitis had no evidence of toxaemia.

Out of these 48 cases, 37 subjects were within 2 km distance from UCIL factory at the time of exposure and 10 were at distance of 2-4 km, while only one person was at a distance of more than 4 km. As 37 out of 48 subjects were within 2 km from the factory they are likely to have been exposed to a significant amount of gas, and the resultant radiological changes in their X-rays are due to high exposure.

Group B: Cases showing radiological changes suggestive of old disease which was aggravated by exposure to toxic gas.

Tuberculosis	7
Chronic bronchitis	6
Pneumonitis	4
Total	17

The radiological findings of 17 subjects revealed the old diseases as tuberculosis, chronic bronchitis and pneumonitis. These old diseases seem to be aggravated by exposure to the toxic gas as the symptoms in these subjects appeared only after the gas exposure.

Pneumonitis was more commonly seen in the right lower zone similar to the observation in group A subjects.

Behavioral findings

(1) Memory

(a) Auditory memory (immediate)—The exposed group particularly the females were significantly inferior to control group. This showed that the effect of exposure in reducing the auditory memory was more in case of females than in males. Exposure to toxic gas significantly reduced the auditory memory of the individuals.

(b) Visual memory—The exposed male group showed statistically significant ($P < 0.001$) differences with the control group (Table 7a, b). Further the immediate memory was below in males as compared to females. Group results showed that immediate visual memory of the subjects was affected because of exposure to toxic gas.

There was significant ($P < 0.01$) difference in delayed visual memory among the three groups. It was

Table 6: Radiological Changes suspected to be Due to the Gas Exposure

No.	Right Lung					Left Lung					Heart Size	Trachea	Remarks
	Upper	Middle	Lower	Hilar	Costoph.	Upper	Middle	Lower	Hilar	Costoph.			
2	N	N	N	P	N	N	N	H	P	Clear	N	Central	
26	N	H	H	P	N	Linear markings	N	N	P	Clear	N	Central	
44	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	
54	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Chronic bronchitis & bronchiectasis
87	N	N	H	P	Clear	N	H	N	P	Clear	N	Central	Few calcified shadows
156	N	N	N	P	Clear	N	H	N	P	Clear	N	Central	Fine mottling
123	N	N	H	P	Clear	N	N	N	N	Clear	N	Central	Increased bronchial markings
133	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	
201	N	N	H	P	Clear	N	N	H	P	Clear	N	Central	
207	N	N	H	P	Clear	N	N	N	N	Clear	N	Central	Increased bronchovascular markings
219	N	N	H	N	Clear	N	N	H	N	Clear	N	Central	Increased vascular markings
315	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	
316	N	N	H	P	Clear	N	N	H	P	Clear	N	Central	Calcified shadows & fibrosis
357	N	N	H	P	Clear	N	N	H	P	Clear	N	Central	
365	N	N	H	P	Clear	N	N	H	P	Clear	N	Right	Scattered calcified shadows
430	N	H	H	P	Clear	N	N	N	P	Clear	N	Central	Congenital deformity of 3rd rib right side
446	N	N	H	P	Clear	N	N	N	N	Clear	N	Central	
484	N	N	H	P	Clear	N	N	N	N	Clear	N	Central	Cystic shadows in right base
463	N	N	H	P	Clear	N	N	N	N	Clear	N	Central	Scattered calcified shadows & surrounding Pneumonitis along Rt hilum
476	N	N	H	P	Clear	N	N	N	N	Clear	N	Central	Left border of heart straightened
495	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Right dome of diaphragm is raised due to collapse right side
509	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Scattered calcified shadows
519	N	N	N	P	Clear	N	N	N	P	Clear	N	Central	Fine mottling both lung fields
548	N	N	N	P	Clear	N	N	N	P	Clear	N	Central	Rt hilars prominent & surrounding haziness
561	N	H	N	P	Clear	N	N	H	P	Clear	N	Central	Fine mottling
608	N	N	H	P	Clear	N	N	H	P	Clear	N	Central	
623	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	
627	N	N	N	P	Clear	N	N	N	P	Clear	N	Central	Scattered calcified shadows & Rt dome of diaphragm raised
651	N	N	H	P	Not well defined	N	H	N	P	Clear	N	Central	
701	H	N	H	P	Clear	N	N	H	P	Clear	N	Central	
868	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Fine mottling in both lungs. Old fractures 8th and 9th rib post part left side
871	N	N	H	P	Clear	N	N	N	P	Clear	enlarged	Central	Calcification left infraclavicular region
882	N	N	H	P	Clear	N	N	H	P	Clear	N	Central	Scattered calcified shadows
897	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Fine mottling & scattered calcification.

Table 6 contd.....

1	2	3	4	5	6	7	8	9	10	11	12	13	14
905	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Scattered calcified shadows
936	N	N	N	P	Clear	N	N	N	P	Clear	N	Central	Scattered calcified shadows C fine mottling
971	N	N	N	P	Clear	N	H	N	P	Clear	N	Central	Scattered calcified shadows C mottling
1034	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	
1011	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	
1019	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Surrounding consolidation around Rt hilar region
1025	N	N	H	P	Clear	N	H	N	P	Clear	N	Central	
1070	N	N	H	P	Clear	N	N	H	P	Clear	N	Central	Scattered calcification
1105	N	N	H	P	Clear	N	N	H	P	Clear	N	Central	Fine mottling C old fracture of 4th, 5th and 6th rib left side
1128	N	N	N	P	Clear	N	N	N	P	Clear	N	Central	Increased bronchovascular marking C fine mottling and scattered calcification
[illegible]	N	N	H	P	Clear	N	H	N	P	Clear	N	Central	Scattered calcification
[illegible]	N	N	N	P	Clear	N	N	N	P	Clear	N	Central	Fine mottling calcification
1204	N	N	H	P	Clear	N	N	N	P	Clear	N	Central	Left dome of diaphragm raised, fine mottling

N = Normal n = Hazy P = Prominent

Table 7: Analysis of Variance

Source of Variation	Immediate Memory (Auditory)			Immediate Memory (Visual)			Delayed Memory for 10 sec. (visual)		
	DF	MS	F	DF	MS	F	DF	MS	F
Between group	2	15.045	4.61	2	176.80	44.22***	2	117.785	25.02 ***
Within group	453	3.265		368	3.98		362	4.71	

* Significant at p = 0.05 ** Significant at p =.0.01

*** Significant at p = 0.001

Table 7 (b) Mean Memory Scores and Test of Significance of Different Comparison

Characters	Groups	Sample size (n)	Mean	SD	Comparisons	Diff. of means	S.E.D.	Level of Significant	
IAM	Control	100	8.96	1.75	Control vs Exposed	0.44	0.2044	2.163	P = 0.05
	Exposed	356	8.52	1.83	Control vs Male	0.25	0.2232	1.120	NS
	Male	190	8.71	1.78	Control vs Female	0.66	0.2287	2.886	p = 0.01
	Female	166	8.30	1.87	Male vs Female exposed	0.41	0.1920	2.135	p = 0.05
IVM	Control	100	6.92	1.35	Control vs exposed	2.13	0.2335	9.122	p = 0.001
	Exposed	271	4.79	2.20	Control vs Male	2.37	0.2546	9.309	P = 0.001
	Male	159	4.55	2.20	Control vs Female	1.79	0.2745	6.521	P = 0.001
	Female	112	5.13	2.16	Male vs Female	0.58	0.2461	2.357	P = 0.05
DVM	Control	100	5.35	1.93	Control vs Exposed	1.80	0.2547	7.067	P = 0.001
	Exposed	265	3.55	2.25	Control vs Male	1.84	0.2780	6.619	P = 0.001
	Male	156	3.51	2.29	Control vs Female	1.74	0.3005	5.790	P = 0.001
	Female	109	3.61	2.20	Male vs Female	0.10	0.2709	0.369	NS

IAM Immediate auditory memory
IVM Immediate visual memory
DVM Delayed visual memory for 10 seconds

Very highly significant P = 0.001***
Highly significant P = 0.01**
Significant P = 0.05*

found that the exposed group as such was significantly inferior ($P < 0.01$) to control group whereas there was no difference between exposed male and female indicating that both were equally affected by exposure to toxic gas. It was thus concluded that visual memory was more affected than auditory memory by the exposure.

Perceptual motor speed: Both Digit Symbol and Bourdon-Wiersma test measure the perceptual motor speed the second test includes attention vigilance as an additional parameter (Table 8). The changes were significant ($P < 0.001$) while in the Digit Symbol Test such a level of significance was not observed. The analysis of questionaire and result

Table 8: ANOVA Table of Perceptual Motor Speed Tests

Source of variation	D.W. Vigilance Test			Digit Symbol Test		
	D.F.	M.S.	F	D.F.	M.S.	F
Between group	2	1394.045	10.58•••	2	55.74	0.325 (NS)
Within group	387	131.790		371	171.58	

Table 8 (b) Mean and Test of Significance of Different Comparisons

Character	Group	Size	Mean	SD	Comparisons	Diff. of means	SE (D)	Level of significance
Vigilance	Control	100	51.00	8.06	Control vs exposed	6.12	1.3313	4.597 •••
	Exposed	290	44.88	12.42	Control vs Male	6.26	1.4725	4.251 •••
	Male	155	44.74	12.10	Control vs Female	5.95	1.5146	3.928 •••
	Female	135	45.05	12.82	Male vs Female	0.31	1.3515	0.229(NS)
Digital Symbol Test	Control	100	44.52	12.85	Control vs Exposed	NS		NS
	Exposed	274	43.37	13.17	Control vs Male			
	Male	157	43.57	13.37	Control vs Female			
	Female	117	43.11	12.94	Male vs Female			

• Significant at p = 0.05
•• Significant at P = 0.01
••• Significant at P = 0.001
NS Non Significant

Table 9 (a): ANOVA of Attention Response Speed

Sources of variation	Simple Reaction Time (Light)			Simple Reaction Time (Sound)		
	D.F.	M.S.	F	D.F.	M.S.	F
Between group	2	0.0400	4.166•	2	0.0397	7.065•••
Within group	424	0.0096		424	0.0056	

Table 9: (b): Mean and Test of Significance of Different Comparisons

Character	Group	size (n)	Mean	SD	Comparisons	Diff. of mean	SE (D)	t	Level of significance
Simple reaction time (SRT) (light)	Control	70	0.27	0.05	Control vs Exposed	0.035	0.0128	2.734	••
	Exposed	357	0.31	0.11	Control vs Male	0.030	0.0137	2.190	•
	Male	190	0.30	0.10	Control vs Female	0.040	0.0139	2.878	••
	Female	167	0.31	0.11	Male vs Female	0.010	0.0104	0.962	(NS)
Simple reactions Time (SRT) (sound)	Control	70	0.18	0.04	Control vs Exposed	0.035	0.0098	3.571	•••
	Exposed	357	0.22	0.08	Control vs Male	0.030	0.0105	2.857	••
	Male	190	0.21	0.08	Control vs Female	0.040	0.0107	3.738	•••
	Female	167	0.22	0.08	Male vs Female	0.010	0.0079	1.266	(NS)

• Significant at p = 0.05
•• Significant at p = 0.01
••• Significant at p = 0.001
NS Non Significant

Distribution of Cases with Poor Scores

	Exposed n = 289	Male n = 154	Female n = 135	χ^2
	230	117	113	
	(79.58)	(75.97)	(83.70)	2.645
	256	133	123	
	(88.58)	(86.36)	(91.11)	1.603
	165	93	72	
	(57.09)	(60.9)	(53.33)	1.462
	13	4	9	
	(4.50)	(2.60)	(6.67)	1.907*

χ^2 = 1.907

χ^2 5% = 3.841

Interviews with the exposed subjects revealed that these subjects were having a lack of concentration and had poor attention. The study also revealed that attention/vigilance was significantly impaired in the population affected by the toxic gas.

Attention response speed: This was studied by using the Reaction Time Test. It was tested by both light and sound stimuli. It was observed that the exposed group had a significantly delayed response as compared to that of the control group. The exposure to the toxic gas equally affected the reaction time (attention-response speed) for both the stimuli in the exposed population (Table 9).

Manual dexterity—To see the effect of exposure on manual dexterity in comparison groups namely, control, exposed male and exposed female groups, the analysis of variance of the data was made. The F value was found to be less than one. It was, therefore, concluded that exposure to the toxic gas did not affect the manual dexterity of the exposed population.

The questionnaire is a method for detecting non-intellectual personality disturbances. The analysis of the questionnaire revealed that 79.6% cases had poor scores on general lability items, whereas 88.6% with poor scores had a tendency to general fatigue with somatic complaints. Lack of extroversive activity was observed in 57.1% cases. Only 4.5% cases had neurotic tendencies.

It was concluded that the highest percentage of cases with poor score was in general fatigue with somatic complaints (88.6%), followed by general lability symptoms (72.5%) and neuroticism found only in 4.5% of the population.

When the data of the exposed population for males and females were separately compared the results indicated that formally the females were more affected than the males. The distribution of cases with poor scores is given in Table 10. Statistically significant differences were observed between the control and exposed groups of all parameters used.

It was observed (Table 10) that the majority of cases with poor scores were in the second category, i.e. general fatigue with somatic complaints (88.6%) followed by 79.6% cases in the first category, i.e. general lability items.

The effects of toxic gas on the population was mainly on ocular, gastrointestinal and musculoskeletal systems. Radiologically 48 cases showed specific changes because of exposure to the toxic gas, out of these 37 were staying within 2 km of the factory. The results of behavioral tests revealed that memory, mainly visual perceptual and attention/response speed alongwith attention/vigilance were severely affected in the exposed population.

A majority of exposed subjects staying within a distance of 6 km were affected by one or the other health problems. Besides clinical and radiological morbidity changes in behaviour were objectively measured in a large number of subjects. Thus the gas manifested its effect in various systems of body depending on exposure, dose and individual susceptibility.

References

1 IPCS-IRPTC Information sheet on Methyl Isocyanate, International Programme on Chemical Safety—International Registry of Potentially Toxic Chemicals, Geneva, 1985, 7 Jan.

2 Kumar C D & Mukherji S K, *Sci Today*, **19** (1985) 11.

3 Editorial, *Calamity at Bhopal*, *Lancet*, **11** (1984) 1378.

4 Rye W A, *J Occup Med*, **15** (1973) 306.

5 Munn A, *Ann Occup Hyg*, **8** (1965) 163.

6 B R M A, British Rubber Manufacture Association Limited, Birmingham, 1977.

7 Berstein L, *Aller Clin Immunol*, **70** (1982) 24.

8 Fuchs S & Valade P, *TDI Arch Mal Prog*, **12** (1951) 191.

9 Reinl W, *Sickness caused by working with plastics and lacquers on polyurethanes*, *For be and Lack*, **60** (1954) 69.

10 Walworth H T & Virchow W E, *Am Ind Hyg Ass J*, **20** (1959) 205.

11 Williamson K S, *Trans Ass Ind Med Off*, **15** (1965) 29.

12 Hanninen H & Lindstrom K, *Behavioral test battery for toxicopsychological studies* (Instt. Occup. Health, Helsinki), 1979.

13 Benton A L, *The visual retention test—Clinical & experimental* (Psychological Corp., New York), 1972, 92.

14 Eysenck H & Eysenck S, *Manual of eysenck personality inventory*, *Repr Hodder and Stonghton*, *Dunton Green Sevenoaks*, *Kent*, 1976, 24.

15 Devos G A, *J Proj Tech*, February 28, 1951.

16 Hanninen H, *Br J Industr Med*, **28** (1971) 374.

17 Hanninen H, in *Behavioral toxicology*, edited by C. Xintaras, B.L. Johnson and I. De Groot (US Department of Health, Education and Welfare, Washington, DC), 1974, 73.

18 Hanninen H, Eskelinen L, Huszan K & Jurminen M, *Scand J Work Environ Health*, **2** (1976) 240.

19 Klopper B & Davidson H H, *The Rorschach technique: An introductory manual* (Harcourt, Brace and World Inc., New York) 1962, 245.

20 Lindstrom K, *Work Environ Health*, **10** (1973) 151.

21 Lindstrom K, Harkonen H & Hernberg S D, *Scand J Work Environ Health*, **2** (1976) 129.

22 Mira Y & Lopez E, *Hans Huber Berne*, 1965, 164

23 Rorschach H, Psychodiagnostik, *Hans Huber, Ber* 277.

24 Takala M I, *Ann Sci Fenn Ser B*, **81** (1953) 132.

25 Wechsler D *The measurement and appraisal of adul ence* (The Williams and Wilkins Co.) 1958, 297.

26 Wechsler D, *J Psychol*, **19** (1945) 87.

27 *Tuberculosis in India: A sample survey*, 1955-58 Council of Medical Research, New Delhi) 1959.

Rhoplat file (Technical Papers)

Editorial

On the Bioavailability of Methyl Isocyanate in the Bhopal Gas Leak

Methyl isocyanate (MIC), the toxin that escaped from the Union Carbide plant at Bhopal, India, in December, 1984, is now well known for its highly irritant properties.[1] This toxin manifested its primary acute effects on the lungs and eyes of the victims who came in contact with the gas. More than 3 000 lives were claimed, and the toxin is responsible for chronic health effects in an additional 50 000 survivors. Chronic lung effects, i.e., pulmonary fibrosis, currently appear to be the main cause of morbidity and mortality among the surviving population.[2] Clearly, the varying exposure of victims to MIC was a major factor in determining toxicity. Singh and Ghosh computed exposure estimates from an analytic dispersion model, which incorporated meteorological and topographical factors around the Union Carbide plant.[3] These estimates ranged from 0.12 ppm to 85 ppm and are higher than the workplace threshold limit value (TLV) of 0.02 ppm. Proximity of residence to the Union Carbide plant and activity (e.g., running versus remaining at home) are variables that determine the total dose of MIC experienced by the victims.

Was methyl isocyanate bioavailable in Bhopal? On contact with water, MIC decomposes to methylamine and dimethylurea.[4] For this reason, systemic absorption into the bloodstream through the respiratory tract was not initially seriously considered because MIC was expected to decompose on contact with fluids in the respiratory tissues.

The question of whether MIC became a systemic poison after environmental exposure was debated subsequent to early studies, which reported that multiple systems were involved in the Bhopal victims.[5] Clinical manifestations of toxicity were observed in the gastrointestinal, reproductive, and central and peripheral nervous systems. Gastrointestinal symptoms included anorexia, nausea, vomiting, and abdominal pain. Endoscopic examination of patients revealed congestion and hemorrhages in the lower esophagus and stomach. Reproductive manifestations included an increased incidence of spontaneous abortions and stillbirths. Neurological problems included coma, tremors, vertigo, hearing loss, muscle weakness, and peripheral neuropathy.

It can be argued that some of these manifestations could be explained by the effects of hypoxia and acidosis, which were secondary to suffocation and respiratory damage.[6] This view was supported by results from a study done by Fedde et al.,[7] which demonstrated that a drop in blood PaO_2 and pH and a rise in pCO_2 occurs following exposure of mice to MIC. The most likely mechanism that would produce altered ventilation-perfusion relationships and impaired gas exchange in this study was thought to be obstruction of small airways from sloughing of epithelium into the bronchioles. Undoubtedly, hypoxia secondary to respiratory damage must have occurred in many gas-exposed victims. However, clinical studies conducted by the Industrial Toxicology Research Centre at Lucknow, India, revealed that some multi-systemic complaints were persistent and occurred even in those patients who did not have significant respiratory damage.[8] In a sample from a gas-exposed population studied 3 mo after the accident, biochemical indicators of stress response were observed: blood ceruloplasmin levels were increased 200% over control values in more than 45% of those tested.[9] In these studies, urinary creatinine was significantly higher in study subjects (2.95–3.3 g/24 h · 1.5 l of urine) than in controls (1.71 g/24 h · 1.5 l of urine). Blood glutathione was significantly depressed in approximately 40% of the population examined. In some animal studies, systemic toxicity has been found in target organs other than the lung.[10-13]

Bucher,[14] in his review of the health effects research done on MIC, states that if MIC is shown to bind to normal hemoglobin (carbamoylation), this would provide evidence that the chemical crosses the alveolar barrier, and would, therefore, support MIC's potential for systemic exposure. Cyanates can be absorbed from the gastrointestinal tract, and they exert antisickling properties when administered orally. In patients with sickle-cell disease, the cyanate anion (NCO^-) produces irreversible inhibition of red blood cell sickling via carbamoylation of the amino-terminal valine residue of sickle-cell hemoglobin.[15] The life span of the erythrocyte is increased, and improvement of hemolytic anemia is seen.[16] Though once considered as potential therapeutic agents in the long-term management of sickle-cell anemia, cyanates have not been used for this purpose because of their toxicity.

At the University of Pittsburgh, Ferguson et al.[17] studied the uptake and distribution of radiolabeled (^{14}C) MIC in guinea pigs. The animals were exposed to concentrations of 0.5–15 ppm for periods of 1–6 h. Their findings showed that the radiolabel was rapidly absorbed and distributed in all tissues of the body, including the uterus, placenta, and fetus. Clearance of the radiolabel was gradual for 3 d. A significant amount of this absorption occurred in the nasal mucosa and upper-respiratory tract. Less absorption occurred in the tracheal route than in the upper-respiratory passages. The authors concluded that "these findings may help to explain the toxicity of MIC or MIC reaction products on organs other than the respiratory tract."[17]

Bhattacharya et al.,[18] who are at the Defence Research and Development Establishment, Gwalior, India, studied distribution and binding of MIC to tissue proteins throughout the body. They used radiolabeled MIC (label on isocyanate fraction, $CH_3—N=\overset{14}{C}=O$) and dosed female Wistar rats by inhalation and intraperitoneal (i.p.) routes. Their results indicated that MIC carbamoylates globin, total blood proteins, and liver proteins. The radiolabel was also distributed in its ac-

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Pregnancy outcome in women exposed to toxic gas at Bhopal

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A study was undertaken to compare the effects of exposure to the toxic gas in pregnant women in Bhopal with pregnant women in a similar, unexposed area. A high incidence of spontaneous abortions (24.2%) in the pregnant women exposed to the toxic gas was observed as compared to those in the control area (5.6%). Other indices of adverse reproductive outcome, such as the rate of still birth and congenital malformation were not found to be different. The perinatal and neonatal mortalities were significantly higher in the affected area (6.9 and 6.1% respectively), as compared to the control area (5.0 and 4.5% respectively).

Bhopal gas disaster known as one of the worst industrial accidents in history, has left several unanswered questions regarding long term sequelae including potential genotoxic/teratogenic effects of the toxic gas that leaked out on the fateful night of December 2, 1984.

In utero exposure of embryo/foetus to a chemical can produce a variety of toxic effects. In severe circumstances, the embryo/foetus may die leading to spontaneous abortion or still birth depending upon the period of gestation at which the exposure has occurred. Interaction with DNA can produce genetic diseases and congenital malformation. In less severe cases, a toxic agent may interfere with processes of intrauterine growth leading to intrauterine growth retardation. In utero exposure may also lead to effects on growth and development in later life causing physical and/or mental retardation.

A study was carried out to monitor the adverse effects in pregnant women who were exposed to toxic gas at Bhopal, in comparison to the data collected on pregnant women in a similar, but unexposed area. The outcome of pregnancy and neonatal status are reported here.

Material & Methods

The study was carried out in 10 severely affected areas of Bhopal city, around the Union Carbide Factory identified on the basis of records of casualities. The following localities from gas exposed areas were included: J.P. Nagar, Kazi Camp, Tila Jamalpura, Shahjanabad, Straw Product, Bus Stand- Ibrahimganj, Kenchi Chhola, Railway Colony, Station Bazaria and Chandbarh. A door-to-door population survey was carried out during April-May, 1985 to identify women who were pregnant at the time of exposure (as assessed by recording those with the last menstural period before 18th November, 1984).

As this made the study partly retrospective and partly prospective, the identified study population were classified into the following three groups:-

(*i*) Those who aborted between December 3, 1984 and the date of the commencement of survey (381).

(*ii*) Those who delivered within this period (934).

(*iii*) Those who were found to be pregnant at the time of survey (1251).

Information in abortions and on women who had delivered before the commencement of survey was obtained on the basis of history given by the woman and confirmed by interview of neighbours and a review of the records of the nursing home and hospitals wherever possible (125 cases). Spontaneous abortions were reconfirmed through a quick survey (as suggested by the concerned Project Advisory Committee held in December, 1985). The women who were pregnant at the time of survey were prospectively followed till delivery. All the babies born since December 3, 1984 and who were alive were examined to detect congenital malformations (both major and minor malformations), if any. In cases where the child had died or there was a still birth, a set of photographs of common congenital malformations were shown to either parents or others who had seen the child, to verify the presence or absence of congenital malformations.

A control area of similar socio-economic status was selected including the following localities of Bhopal: Anna Nagar, Vishwakarma Nagar, Habibganj, Janta Colony (near Arera Colony), Panchsheel Nagar, Harshvardhan Nagar, Ambedkar Nagar, Banganga and Roshanpura. Study of women in the control area could not be done simultaneously as it was more important and urgent to the pregnant women in the affected area. Thus, study in the control area was started a year later, taking 3rd Dec., 1985 as the cut-off point; all women pregnant on this date were registered to obtain a cohort of similar type in respect of period of gestation.

The study on the control area was thus totally prospective. A door to door survey was carried out to identify the pregnant women, who were carefully followed for outcome of pregnancy. Statistical analysis was done using the Student's 't' test and proportion test for testing the difference between averages and rates respectively.

Results

The total number of households surveyed was 18,978 covering a population of approximately 86,000 in affected area and 13,539 households covering a population of approximately 60,000 in control area. Of these, 2566 women from the affected area and 1218 women from the control area were identified as pregnant on December, 3rd 1984 and 1985 respectively, and the outcome of pregnancy was studied. There were 2153 deliveries in the affected area, of which 1071 (49.7%) deliveries were conducted in hospitals and 39 (1.8%) were conducted in MCH Centres and Nursing Homes, the rest, 1043 (48.4%) having been conducted at home. There were 1180 deliveries in control area, 627 (53.1%) women having delivered in Hospitals, one (0.08%) delivery being conducted in a nursing home and 552 (46.8%) deliveries having been conducted in home.

Socio-economic profile : Most of the women were from the low socio-economic group. The mean per capita income was Rs.122 ± 69 in control area and Rs.96 ± 87 in affected area. Though the per capita income was slightly higher, the literacy rate was found to be lower in the control area (32.5%) than in the affected area (39.4%).

Religion : The proportion of Muslims was considerably higher in the affected area (40.3%), as compared to the control area (14.4%). This was mainly because no other area of similar socio-economic status with such high proportion of Muslims could be found within the unexposed areas of Bhopal.

Consanguinity : Consanguinity was observed to be 15.6 per cent in the affected area as compared to 6.7 per cent in control area. This difference in consanguinity rate was probably due to higher proportion of Muslims in the affected area.

Age : The mean age of the women in affected and control areas was 24.9 and 24.4 yr respectively. In the affected area, 21.6 per cent (341) women were aged 30 yr or above, as compared to 15.7 per cent (74) in the control area, the difference being statistically significant ($P < 0.01$).

Previous obstetric history : Previous obstetric history was taken for all the women included in the study. Average parity of the overall sample in affected area was 2.8 of which 2.5 were live births, 0.04 were still births, 0.23 were spontaneous abortions and 0.02 were induced abortions. In the control area the average parity was 2.1, of which 1.9 were live births, 0.03 were still births, 0.17 were

abortions. The average number of living male and female children was 1.2 each in the affected area and 0.7 and 0.9 in the control area respectively.

Gestation period (as on December 3) : The period of gestation at which women enter the cohort was considered important, as the outcome of pregnancy is associated with this factor. There was considerable variation in the distribution of women according to gestation on 3rd December in the affected and control area. There was 57.3 per cent pregnancies with period of gestation up to 20 wk on 3rd December, 1984 in affected area. The corresponding figure for control area was 39.9 per cent. Percentage of women with gestation 21-27 wk was 15.7 and 22.2 in affected and control area respectively. The remaining 27.0 per cent pregnancies in affected area and 37.9 per cent pregnancies in control area were of gestation 28 wk or more. This difference may be due to the fact that those women (from the affected area) who were in the third trimester of pregnancy perhaps went elsewhere for their delivery recognizing that pregnancy constituted increased risk particularly in the wake of the general atmosphere of shock, fear and controversies prevalent at the time of the disaster. These women obviously could not be included in the study as they were not present at the time of survey (Table I).

Outcome of pregnancy :

Abortion - Abortion having been defined as termination of pregnancy before 20 wk of gestation, only those women who were up to 20 wk of gestation on 3rd December 1984/1985 were considered to be at the risk of abortion. These including 1468 women in the affected area as against 485 women in the control area. Among these, 355 (24.2%) pregnancies from the affected area and 27 (5.6%) pregnancies from the control area ended in spontaneous abortions. A diagnosis of abortion was recorded only when the women gave history of amenorrhoea of at least 8 wk and/or had actually observed expulsion of products of conception. All spontaneous abortions were confirmed through a re-survey done by doctors, using a detailed information check list; in 125 cases of spontaneous abortions, records were available from hospital or doctors. The history of D & C was confirmed from hospital records (43 cases). The abortion rate was significantly higher in affected area ($P < 0.001$; Table II).

In the affected area, the abortion rate was 32.5 per cent in women (341) of age 30 yr or above compared to 22.1 per cent in women below 30 yr. This difference was statistically significant. Whereas no case of spontaneous abortion was reported from control area in women (74) 30 yr or above.

Religion and consanguinity were not found to have any association with the abortion rate. In the affected area, abortion rate was 26.3 per cent among Hindus and 21.9 per cent among Muslims.

In the control area, the abortion rate which slightly higher among the Muslims (9.1%), was not significant as compared to 5.0 per cent in the Hindus (Table III). Among consanguinous and non-consanguinous marriages abortion rates were 26.6 and 23.9 per cent respectively in affected area and 6.6 and 5.6 per cent respectively in control area (Table IV). Abortion rates were significantly higher in affected area irrespective of religion or consanguinity.

Intermediate foetal deaths – In the affected area, 32 (1.2%) intermediate foetal deaths (pregnancy termination between 21-27 wk of pregnancy) were recorded, as compared to 8 (0.7%) cases of intermediate foetal deaths in the control area (Table II).

Still births – Taking into consideration the pregnancies which terminated at 28 wk of gestation and above, there were 2153 deliveries in affected area, of which 56 were stillbirths and 2117 live births as 20 deliveries had resulted in twin births. This gave a stillbirth rate of 2.57 per cent. In the control area, the total number of deliveries at or after 28 wk of gestation was 1180. Of these, 27 were stillbirths and 1160 live births giving a still birth rate of 2.27 per cent. No difference was observed in the stillbirth rates from affected and control area. In control area 7 deliveries (0.59%) resulted in twin births as compared to 20 (0.93%) in the affected area, the difference being not significant (Table V).

Congenital malformations : There were 31 congenital malformations in the affected area out of 2173 births. In the control area 15 of a total of 1187 births showed congenital malformations, giving a rate of 14.2 per 1000 births and 12.6 per 1000 births in the affected and control areas respectively (Table V). Details of the nature of congenital malformations observed in the two areas are depicted in Table VI. As in case of abortions, religion did not show any relationship with the congenital malformations. The latter were found to be slightly higher in consanguinous as compared to non-consanguinous marriages in both the affected and the control areas. It was 1.9 and 2.5 per 1000 births for consanguinous marriages and 1.4 and 1.2 per 1000 births in non-consanguinous marriages in affected and control areas respectively. These differences were statistically significant.

Perinatal and neonatal mortality : Of the 2153 deliveries in the affected area, 2117 were live births (including 20 twin deliveries). Of these, 95 babies died within 7 days of birth and 34 between 8-28 days of birth, giving a perinatal mortality rate of 69.48 per 1000 births and a neonatal mortality rate of 60.9 per 1000 live births. In the control area, of the 1160 live births (including 7 twin deliveries) out of 1180 deliveries, 33 babies died within 7 days of birth while another 19 died between 8-28 days, giving a perinatal mortality rate of 50.54 per 1000 births and neonatal mortality of 44.8 per 1000 live births. Both perinatal and neonatal mortality were significantly lower in the control area than in the affected area (Table V).

Babies who survived beyond 28 days *i.e.*, (1988

Table I. Period of gestation of studied women as on 3rd December, 1984/1985

Period of gestation (wk)	Affected area No.	Control area No.
<12	934 (36.4)	250 (20.6)
13-20	534 (20.9)	235 (19.3)
21-27	401 (15.7)	270 (22.2)
28-36	450 (17.6)	307 (25.2)
>37	242 (9.4)	154 (12.7)
Total	2561	1216
Gestation period not known	5	2

Figures in parentheses are the percentages

Table II. Outcome of pregnancy

	Affected area	Control area
Total number of pregnancies	2566	1218
Number of women at risk of abortion	1468	485
Number of spontaneous abortions	355	27
Abortion rate (%)	24.2*	5.6*
Number of induced abortions	26	3
Intermediate foetal death (21-27 wk of gestation)	32	8
Number of deliveries	2153	1180

* $P < .001$

Table III. Spontaneous abortions in relation to religion

Religion	Affected area			Control Area		
	Number of			Number of		
	At risk of abortion	Spont. abor.	Rate /100	At risk of abortions	Spont. abor.	Rate /100
Hindu	832	219	26.3	402	20	5.0
Muslim	607	133	21.9	77	7	9.1
Others	29	3	10.3	6	—	—
Total	1468	355	24.2	485	27	5.6

Table IV. Spontaneous abortions in relation to consanguinity

Consanguinity	Affected area			Control Area		
	Number			Number		
	At risk of abortion	Spont. abort.	Rate /100	At risk of abortion	Spont. abort.	Rate /100
Yes	248	66	26.6	33	2	6.6
No	1208	289	23.9	448	25	5.6
Not known	12	—	—	4	—	—
Total	1468	355	24.2	485	27	5.6

Table V. Neonatal outcome

	Affected area	Control area
No. of deliveries	2153	1180
No. of twin births	20	7
No. of live births	2117	1160
No. of still births	56	27
Still birth rate/1000 deliveries	26.0	22.9
No. of early neonatal deaths (0-7 days)	95	33
Total perinatal loss	151	60
PMR/1000 births	69.48*	50.54*
No. neonatal deaths (0-28 days)	129	52
NMR/1000 live births	60.9**	14.8**
No. of congenital malformations	31	15
Rate/1000 births	14.2	12.6

P values, * $< .001$; ** $< .001$

Table VI. Congenital malformations

Type of malformation	Exposed area	Control area
Congenital talipes equinovarus	13	02
Congenital heart disease	07	03
Meningo myelocoel	02	—
Exomphalus	01	—
Micro opthalmaia with lenticular opacity	01	—
Imperforate anus	01*	02
Congenital aplasia of both eyes	01	—
Haemangioma scalp	01	—
Hirschsprung disease	01	—
Hypospadius	01+	01
Cleft lip with cleft palate	01	—
Rocker bottom foot	01	—
Multiple congenital anamolies	—	02
Anencephaly	—	02
Microcephaly	—	01
Polydactyly	—	01
Foetal ascitis	—	01
Total	31	15

* With recto-vaginal fistula.
+ With absence of external ear

babies from the affected area and 1108 babies from the control area) are being followed up for morbidity and mortality during infancy.

Discussion

In this study, those women who were pregnant at the time of gas exposure from moderate to severely affected areas were studied for adverse pregnancy outcome in order to determine possible embryotoxic effects of MIC. Control area of similar socio-economic status from unexposed area of Bhopal was selected and pregnant women were registered. Women were followed till outcome of pregnancy to obtain comparative data. Although both the affected and the control areas fall in the low socio-economic group, there was a slight increase in per capita income in control area. The difference in the income in the control area could be due to the fact that the study in the control area started one year after the exposure. The proportion of Muslims was also higher in affected area (40.3%) as compared to control area (14.4%), as there was no other area in Bhopal of similar socio-economic status with such a high proportion of Muslims. These factors were taken into account while comparing indicators of pregnancy outcome.

The variation in the distribution of period of gestation seen in affected area as compared to control area may perhaps be due to the fact that women from affected area nearing term went elsewhere for their delivery and could not be registered for the study.

The abortion rate was more than four times in the affected area as compared to control area. Religion and consanguinity did not show association with abortion rate. Abortion rate in affected area was significantly higher in women 30 yr of age or above (32.5%) as compared to women below 30 yr of age (22.1%). There was no case of abortion in women 30 vr or above in control area. Abortion rate in the affected area after standardizing for age according to the control area was found to be 23.6 per cent as compared to 5.6 per cent in control area, which was still very high.

Stillbirth rate was found to be similar in the affected and control areas, but both perinatal and neonatal mortality rates were observed to be higher in the exposed area. Though per capita income was slightly higher in the control area, it cannot account for higher mortality rates in the affected area, as none of the mortality rates were associated with income. Further, both the control and affected areas belonged to the low income groups. The difference in religion could not also account for this as perinatal and neonatal mortality were not different among Hindus and Muslims. As the study was partly retrospective, information on birth weight was not available for the total study sample and there were differences in methodology for recording birth weights. Hence, frequency distribution of birth weight could not be related to perinatal and neonatal mortality in affected and control area. In relation to age perinatal mortality was higher in women of 30 yr or above and there were 20.4 per cent women in that age group in affected area as compared to 16.4 per cent in control area. Standardized perinatal mortality in exposed area considering control area as standard was seen to be still higher. In the control area, the perinatal mortality was not higher in the older age group. After standardization the neonatal mortality in the exposed area of 60.6 from 60.9 per 1000 live births was still significantly higher than 44.8 in control area. The only factor that could not be studied in relation to these rates was the time factor, as the study in the control area was started a year later.

When compared to the national averages, the perinatal mortality is higher in affected area than the national average of 53.6 per 1000 deliveries but neonatal mortality although higher than in control area is below the national average of 67.2 per 1000 live births[1].

Any exposure to a genotoxic gas can produce damage to DNA in somatic and/or in germ cells. Direct consequences of mutations include both alteration of chromosomes and if severe it may lead to obvious adverse reproductive outcome such as increased rate of abortion, still births, congenital malformations and perinatal mortality in exposed population. The congenital malformation rate did not show any significant difference in both areas (14.2 per 1000 births in affected area and 12.6 per 1000 births in control area). These rates are also not significantly different from several hospital based studies reported in Indian literature[2].

Our study showed a marked increase in the spontaneous abortion rate but how much of it is due to genetic damage caused by MIC could not be assessed from this study as no cytogenetic studies were carried out on abortuses. Considerable proporation of the abortions may be due to the stress and trauma associated with the disaster itself. Although the possibility of possible genetic damage cannot be ruled out, the highlights of this study was an increase in abortion rate, perinatal mortality and neonatal mortality in the areas exposed to the gas leak as compared to the unexposed areas.

In women exposed to toxic gas, the lungs were one of the target organs and the respiratory tract showed series of pathological changes. The deffusion of gases was affected and many pregnant women, who were exposed had respiratory problem, which could affect the foetus. Although the present study does not show any alarming incidence of acute toxicity, the long term genetic effects revealing subsequently cannot be ruled out.

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References

1. *Family Welfare Programme in India-Year Book 1985-86* (Figures quoted are for the year 1983) (Ministry of Health and Family Welfare, Govt. of India, New Delhi). 1987 p 100.
2. Verma, I.C. and Mathews, A.R. Congenital malformation in India.In: *Peoples of India - Some genetical aspects*, G.V. Satyavati, Ed. *Proc. XV. Int.Cong. of Genetics, New Delhi, India* (Indian Council of Medical Research, New Delhi) 1983 p 70.

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Health effects of the Bhopal gas leak: a review

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Introduction

The world's worst industrial disaster occurred in India on the night of December 2-3, 1984. The accident took place at the Union Carbide plant situated in Bhopal, (pop. 900,000) the capital city of Madhya Pradesh, one of the largest states in India.

The accident was apparently initiated by the introduction of water into the methyl isocyanate (MIC) storage tank resulting in an uncontrollable reaction with liberation of heat and escape of MIC in the form of a gas. Safety systems like the flare tower (to burn excess gas), caustic soda scrubber (for neutralization) and the refrigeration unit were either not functioning or inadequate to deal with the large volume of escaping chemical [1].

This paper reviews health effects of gas exposures from published human studies and discusses some of the clinical and epidemiological issues being debated. Because of the relative paucity of information, the author has also reviewed unpublished data from studies conducted by local physician groups. Some of these studies have helped to highlight specific health problems from the disaster and initiate more organized research to address these problems. The reader may also wish to consult articles by John Bucher [2] and Mehta *et al.* [3] for reviews on human and animal toxicology.

Manufacturing Process

Methyl isocyanate (CH_3-N=C=O) is an intermediate product in the manufacture of carbaryl (Sevin), a carbamate pesticide. The process begins with a mixture of carbon monoxide and chlorine to form phosgene. Phosgene is then combined with monomethylamine to form methyl isocyanate. Methyl isocyanate is further mixed with naphthol to produce the end-product, carbaryl.

Chemical and Physical Properties

MIC is a clear, colorless liquid with a pungent odor (b.p.: 39°C, f.p.: -80°C, s.g.: 0.96, m.w.: 57.1, vapor pressure of 348 mm Hg at 20°C). It is moderately soluble in water and hydrolyses on contact to form carbon dioxide and methylamine. When MIC is pyrolyzed in the temperature range of 427-548°C, decomposition products like hydrogen cyanide and carbon dioxide are formed [4].

Toxicological Properties

MIC is highly irritant to the skin, eyes and mucus membranes of the respiratory tract. This irritant property is based on its reactivity with water which enables it to penetrate tissues and interact with protein. Absorption through the skin is known to occur [5]. Because the ACGIH TLV of 0.02 ppm is less than the mucus membrane irritation threshold, (> 0.4 ppm), and the odor threshold (> 2 ppm), the compound is considered to have poor warning properties [6].

Exposure Conditions

It is estimated that about 27 tons of methyl isocyanate escaped from the two tanks in the plant during a period of one to two hours [1]. The release occurred around midnight with adverse prevailing atmospheric conditions (inversion and a low wind speed) which prevented dispersion of the gas [7]. Eyewitness accounts report that a cloud of gas enveloped the area and moved slowly along.

Because of the unexpectedness, time and brief period of release of the gas, air monitoring was not possible nor was it subsequently attempt ed.

Based on quantity of the chemical released and area of spread, (40 sq. km) the Central Water & Air Pollution Control Board estimated MIC concentration to be about 27 ppm [7], a figure which is about 1400 times that of the OSHA workplace standard of 0.02 ppm for 8 hrs. This calculation, however, assumes equal concentration over the whole area of contamination and does not account for variability of concentration with distance. Using an analytic dispersion model, Singh

and Ghosh have provided simulations of exposure concentrations at various distances downwind of the plant [8]. Twenty seven sites were identified with ground level concentrations ranging from 85.6 ppm to 0.12 ppm with a median of 1.8 ppm.

Factors contributing to variability in human exposure include distance of residence from the plant, duration of exposure and activity during exposure. No systematic attempts have been made to reconstruct individual exposure based on these criteria. In the actual incident, activity during exposure to the gas was certainly a major dose-regulating factor. The acute irritant effects of MIC created panic, great anxiety and disorientation, resulting in people running out of their homes. The running resulted in increased ventilatory rates in these people, thereby increasing the dose of the chemical delivered to the respiratory system. The Medico Friend Circle study reports exposure history and safety measures taken by people at the time of the gas leak [9]. In a study sample of 158 persons, 124 ran out of their homes. Only 12 persons used a wet towel or blanket as a safety measure.

Major organs of exposure to the gas were the eyes and respiratory tract. Significant amounts of MIC were also, most probably, dissolved in saliva and swallowed into the gastro-intestinal tract. Skin exposure certainly took place but was less clinically important than the respiratory tract.

A cohort of 80,021 has been registered from the population residing around the Union Carbide plant and classified as mild, moderate and severely exposed based on mortality rates in each area [10]. An unexposed population in Bhopal was used to select the control group of 15,931 individuals.

Characteristics of the exposed population

In the exposed areas, about 53% of the population comprise of Hindus, 45.6% are Muslims and the rest are Christians and Sikhs. In 1985, the monthly income of 80% of the population was below Rs 145/ month ($6) and only 1.25% earned over Rs 465/month (S18). By 1988, 65% of the population was earning below Rs 145/ month, indicating some improvement in economic level. However, the above figures indicate that most of the exposed people are close to the government-defined poverty level of Rs 300/month (S12). Ten percent of the population are smokers, 1.4% alcoholics and 5.5% chew tobacco. Only 34% of the population live in a 'pacca' (permanent) house [10].

Mortality

Of the more than 200,000 persons exposed to the gas, the initial death toll within a week following the accident was over 2,500. In Nov. 1989, the Dept. of Relief & Rehabilitation, Govt. of Madhya Pradesh, placed the toll at 3598 [11]. Dr. SR Kamat, a pulmonary physician who has conducted studies on a large number of gas victims, states that most of the later deaths appear to be occurring from respiratory complications (personal communication, 1990). Mortality rates are declined but were still slightly higher in the severely exposed area (8.75/1000) in comparison with the control area (7.5/1000) during the period May 1989-Mar 90 [10].

Overall Morbidity

Symptom prevalence surveys conducted by the Indian Council of Medical Research (ICMR) indicate that morbidity was higher in the exposed areas (26%) as compared with the control area (18%) during the period Nov 1988-Mar 90. About 11% of people experienced 2 or more spells of illness in a 1-year period. Respiratory, ocular and gastro-intestinal symptoms account for most of this morbidity. This trend appears to be persistent in the survey conducted in the latter part of 1990 [12].

Ocular Problems

The intensely irritating effect of MIC on the cornea resulted in severe ocular burning, watering, pain and photophobia [13]. Examination of the eye showed involvement of the corneal and conjunctival epithelium with redness of the eye, corneal ulceration and lid swelling. Fortunately, the corneal ulceration in most cases turned out to be limited to the superficial layers of the cornea. Slit lamp examination showed discrete lesions in a band across the interpalpebral area, punctate keratopathy, conjunctival chemosis and some pigmentary deposition on the cornea [14]. Relatively few cases of iritis were seen [15]. Treatment at the initial stage consisted of saline eyewashes, pupillary dilatation and topical antibiotics.

Andersson and others performed a follow-up study on the eyes of survivors 9 months after the accident and reported that no case of blindness could be found that could be attributed to gas exposure among the nearly 20,000 persons attending the Bhopal Eye Hospital [16]. However, they did find persistent eye watering and other chronic irritant symptoms like burning, itching and redness. Raizada and Dwiwedi [17] studied eye pathology among 1140 exposed persons and found that the main chronic lesions were chronic conjunctivitis, deficiency of tear secretion and persistent corneal opacities.

Animal experiments conducted by Salmon [18] on male Lister hooded rats indicate that the most severe effects on the eye occur at exposure levels of 65 ppm.

Andersson *et al.* [19] performed a follow-up of 93% of exposed and unexposed Bhopal residents 3 years after exposure. Their findings indicated an increased risk of eye infections, hyperresponsive phenomena, (watering, irritation, phlyctens), excess cataracts and resolution of the corneal erosions in exposed persons. These phenomena have been characterized as the 'Bhopal eye syndrome'. The authors state "in its response to MIC, the eye should be considered a 'sentinel organ' for more general phenomena in the body".

Though there is no evidence that severe damage to the eye's external and internal structures has occurred, the single acute exposure seems to have resulted in a chronic inflammatory process. The problems

of persistent eye watering in some cases and tear secretion deficiency in others coupled with chronic conjunctivitis indicate that there is some damage to the eye epithelium. It is conceivable that housing conditions in the Bhopal slum areas like overcrowding, poor ventilation and exposure to dust and smoke may exacerbate the ocular effects causing irritation and infections.

Respiratory Toxicity

Acute symptoms of the respiratory tract were mainly due to the irritant action of MIC on tissues. Because MIC is moderately soluble in water, lesions were seen in both the upper and lower respiratory tract. Predominant symptoms were cough accompanied by frothy expectoration, a feeling of suffocation, chest pain and breathlessness [20]. Other symptoms included dryness and irritation of the throat and rhinorrhea.

Autopsies on 300 victims revealed severe necrotizing lesions in the lining of the upper respiratory tract as well as in the bronchioles, alveoli and lung capillaries. Enlarged and edematous lungs, consolidation, hemorrhage, bronchopneumonia and acute bronchiolitis were seen [21].

Follow-up studies were conducted 2.5-3 months after the accident by a team from the Industrial Toxicology Research Centre (ITRC) at Lucknow, India [22]. Exposed persons were contacted and requested to come forward voluntarily to be examined. A total of 1279 men, women and children were examined. In these studies, radiological examination of the lungs was carried out on 903 subjects to assess damage to the respiratory tract. Out of 164 abnormal X-rays, 65 were determined to have specific radiological changes which were considered to have occurred or aggravated as a result of gas exposure. These were classified into two groups:

Group A (48 subjects) were those with radiological abnormalities thought to have occurred as a result of gas exposure. These changes were haziness in different zones of the lung, hilar prominence, fine mottling and reticulation. 16 of the 48 subjects had respiratory symptoms (cough, chest pain/tightness, breathlessness) clinical signs (adventitious sounds) and abnormal lung function. Twenty subjects had respiratory symptoms but clinical examination and lung function were normal.

Group B (17 subjects) showed abnormalities suggestive of old disease which was aggravated (symptoms appearing after exposure) by exposure to the gas. These abnormalities were tuberculosis, chronic bronchitis and pneumonitis.

Spirometry was carried out on 783 subjects from the above sample to determine respiratory impairment [23]. Vital capacity (VC), forced vital capacity (FVC) and forced expiratory volume in 1 sec ($FEV_{0.75}$, FEV_1) were the main parameters recorded. Impairment was classified as restrictive (VC or FVC < 80% of predicted), obstructive (FEV_1/FVC < 70%) and a combined pattern (FVC < 80% of predicted and FEV_1/FVC ratio < 70%).

The results showed that 39% of the sample was found to have some form of respiratory impairment. The combined pattern of impairment (obstructive and restrictive disease) had the highest prevalence in the sample (22%). Smoking had no effect on the prevalence of this impairment. Females suffered more mild and moderate impairment. Severe impairment was equally distributed (2.4%) among the two sexes.

Broncho-alveolar lavage fluid was analyzed in 36 mild, moderate and severely exposed persons and 12 unexposed normal controls 1-2.5 years after exposure [24]. The results indicated that severely exposed smokers and non-smokers showed a significant increase in alveolar macrophages. Based on these results, the authors concluded that an inflammatory alveolitis may be present in severely exposed subjects and that long-term follow-up must be done to determine if further impairment of lung function occurs.

Sharma *et al.* [26] report the interesting case of a resident of the gas-exposed area who, in his professional capacity, continued to be exposed for a few days to trapped gases in victims. This 60-yr old nonsmoker had no respiratory symptoms (other than bouts of cough) till the beginning of 1989 when he started complaining of severe cough and breathlessness on exertion. Pulmonary function showed a mild obstructive ventilatory defect and CT scan revealed subpleural thickening, punctate lesions and bilateral septal scars, suggesting that extensive pulmonary fibrosis had occurred.

Though isocyanates are known to be allergenic in the lung [26] the respiratory toxicity of MIC appears to be primarily due to its irritant nature. Follow-up studies with lung biopsies done six months after exposure showed evidence of interstitial fibrosis and bronchiolitis obliterans. These findings were similar to those found in several animal studies [27,28] thus revealing the close association between animal data and clinical findings in Bhopal victims.

Reproductive Toxicity

Concerns that the gas leak had effects on reproductive health were raised early in 1985 when reports indicated that menstrual cycle disruption, leucorrhea and dysmenorrhea had occurred in gas-exposed women [29]. Risk to the fetus was considered because of exposure to the gas and factors like stress, anoxia and ingestion of various prescribed drugs like antibiotics, bronchodilators, and analgesics.

An epidemiological survey by Varma [30] showed pregnancy loss and infant mortality to be high in gas-exposed women. In a sample of 865 women who lived within 1 km of the plant and who were pregnant at the time of the gas leak, 43% of the pregnancies did not result in live births. Of the 486 live births, 14% of babies died in the first 30 days as compared to a death rate of 2.6% to 3% for previous deliveries in the 2 years preceding the accident in the same group of women.

A pregnancy outcome survey of gas-exposed women [31] using historic controls, demonstrated a four-fold increase in

spontaneous abortion rate. Alteration in menstrual cycle lenght was also observed.

Animal experiments conducted by Schwetz [32] and Varma [33] exposing pregnant mice to MIC by inhalation showed that this exposure does indeed have a fetotoxic effect. Varma observed a concentration-dependant increase in embryo loss, decrease in fetal and placental weights and a 20% reduction in length of mandible and bones of the extremities.

Varma *et al.* [34] studied the contribution of maternal hormonal changes and pulmonary damage to fetal toxicity of MIC in rats and mice. Their findings showed that fetal toxicity of MIC was partly independent of maternal pulmonary damage and that MIC could be directly fetotoxic. In the Bhopal situation, results from the animal studies, when considered with the findings from human epidemiology, suggest that exposure to MIC is fetotoxic and that this is probably the result of a direct effect on the fetus.

Genotoxicity and Carcinogenicity

Chromosomal studies were done two and a half months after the gas leak to evaluate genetic damage among the sample of gas-exposed survivors studied by the ITRC [35]. Blood was collected from 31 exposed adults and a similar number of age and sex-matched unexposed controls to assess the occurrence of chromosomal aberrations (breaks and gaps) in lymphocytes. The results show a significantly increased ($p < 0.001$) number of breaks and gaps in the exposed subjects. No follow-up studies were done to see if this effect was persistent.

Cytogenetic studies were done 3 years after exposure on a sample of 40 male and 43 female exposed persons [36]. Results from this study showed statistically higher frequencies of chromosomal aberrations in the exposed group as compared to 46 age and sex-matched unexposed controls. The aberrations were in the form of breaks, gaps, dicentrics, rings, tri and quadri-radial configurations and were more pronunced in female subjects. Sister chromatid exchanges were not significantly different. The authors concluded that though the results may indicate a residual effect on T-cell precursors, further studies are required to demonstrate an exposure-effect relationship.

Short-term tests using Chinese hamster ovary cells showed induction of SCEs and chromosomal aberrations both without and with activation by S-9 mix from Aroclor-induced rat liver [37]. Sex-linked recessive tests in Drosophila and the Ames test were negative. The Ames test was also found to be negative by Shelby [38]. Meshram and Rao [39] using a different pre-incubation procedure for the Ames test (10°C for 60 min instead of the standard 37°C) found MIC to be weakly mutagenic.

Shelby *et al.* [38] performed genetic toxicity testing on B6C3F1 mice exposed to MIC by inhalation. Analyses on lymphocytes, bone marrow and lung cells were done using single and multiple exposures. Multiple exposure experiments in the mice showed increased frequencies of SCEs and chromosomal aberrations which were not seen in 2 hr exposures. Delay of cell-cycle time was also reported [40,41].

While animal and in vitro studies demonstrate MIC's potential for genotoxicity, it is not clear that such toxicity has actually occurred in exposed humans.

To assess carcinogenic potential, rats and mice were exposed to MIC for 2 hrs by inhalation. Marginal increases of pheochromocytomas of the adrenal medulla and adenomas of the pancreas were seen but these tumors were not considered clearly related to the exposure [42].

A population-based cancer registry has been established in Bhopal in 1986 to study possible carcinogenic effects of the gas leak. All cases of cancer are being registered and categorised by exposure area [10]. Though it has been predicted that MIC has a significant potential for cancer induction [43], it is not expected that the onset of gas leak-related cancers, if any, will occur before the 30-40 yr lag period.

Immunotoxicity

Following exposure to the gas in Bhopal, there was concern amongst the health authorities that the population might experience an increased rate of infections. Possible reasons for this increased susceptibility included depressed immune function from chemical effects, psychological and physical stress, disruption of normal life (particularly during the 2 mass migrations out of Bhopal), and pulmonary injury.

Immune function was studied [35] in exposed subjects from the ITRC sample two and a half months after exposure to ascertain whether any change had occurred in the immune status. Humoral immunity was assessed by quantitation of immunoglobulins (IgG, IgM, IgA) in over 300 exposed and 10 non-exposed persons. Cell-mediated immunity (CMI) was assessed by phagocytic activity of lymphocytes and quantitation of T-cell rosettes in 19 exposed and 8 non-exposed persons. Results from this study showed that no difference in mean immunoglobulin levels was found when compared to controls. The T-cell population (28%) was found to be less than half that found normally in the Indian population (65%). Significant depression of phagocytic activity of lymphocytes was found as compared to controls.

Concurrent with the human studies, immunotoxicological evaluation of rats exposed to MIC showed a number of positive results (44). Alveolar and peritoneal macrophage function was depressed and exposed rats were susceptible to E. coli endotoxin. Delayed type hypersensitivity was assessed by injecting sheep RBC's into the foot pads and was found to be impaired. Based on these results, the researchers concluded that the gas had a suppressive effect on cell-mediated immunity.

Karol and Kamat [26] found MIC-specific antibodies in guinea-pigs injected with MIC as well as in 12 of 144 human survivors. This showed that MIC was capable of eliciting an immunogenic response. The antibody titers in the human studies were low and transient suggesting a weak response.

Limitations of the human studies include the relatively small sample sizes,

choice of control groups and unclear exposure ascertainment. The above limitations make it difficult to arrive at definitive conclusions regarding immunotoxicity from MIC exposure for the gas victims.

Psychological and Neuro-behavioral Toxicity

Srinivasamurthy and Isaac [45] noted that psychological problems of Bhopal survivors fell into four major categories.

1. Post-traumatic stress disorders which occurred as a result of the tremendous emotional stress of the disaster. Symptoms were anxiety, emotional recall of the event, restlessness, sleep disturbances and generalised weakness and fatiguability.

2. Pathological grief reactions characterised by intense grief, depression, suicidal ideation and guilt feelings arising out a sense of failure to protect their family.

3. Emotional reactions to physical problems: victims with ocular, lung and other problems developed feelings of depression, hostility, insecurity and helplessness.

4. Exacerbation of pre-existing psychiatric problems.

In a psychiatric out-patient service program set up specifically for gas victims, Sethi *et al.* [46] detected 208 persons suffering from mental problems. Of these, 45% were suffering from neuroses, 35% from anxiety states and 9% from adjustment reactions.

Neuro-behavioral tests were conducted on 350 exposed subjects two and a half months after the accident [22]. Auditory and visual memory, attention response speed, and vigilance were found to be significantly impaired in this group as compared to controls. No effect was seen on manual dexterity.

Neuromuscular Toxicity

Neuromuscular symptoms in Bhopal survivors have persisted since the gas leak [10]. These symptoms are mainly tingling, numbness, a sensation of pins and needles in the extremities and muscle aches.

To assess whether MIC was toxic to muscle, Anderson *et al.* [47] evaluated the effects of MIC on rat muscle cells in culture. At lower doses, the formation of muscle fibers was prevented. At higher doses, death of fibroblasts and myoblasts was seen. The findings suggested either an effect on muscle differentiation or selective toxicity to myoblasts.

Clinical Problems

Paucity of information on the toxicology of MIC had created a great deal of confusion and debate about the management of the gas victims. The medical system in Bhopal was severely tested by the twin factors of large numbers of injured people streaming into hospitals and the absence of a definite protocol for treatment of the poisoning. Patients were treated on a symptomatic basis. For ocular problems, atropinization of the eye, local antibiotics and padding were used [14]. Respiratory problems were treated with bronchodilators, steroids, diuretics, antibiotics and oxygen administration.

Questions of clinical importance, some of which still persist to this day, are:

- Were there toxins other than MIC released in the accident?
- What were the specific antidotes?
- Did cyanide poisoning occur in the victims and, if so, how was it manifest and what was the source of the cyanide?
- Does MIC enter the blood-stream and cause multi-systemic disease?
- What were the risks to exposed pregnant women and the unborn child?
- There is no doubt that MIC was clearly the major toxin released in the accident. However, the circumstances leading to the release (hydrolytic and exothermic reaction of water with MIC) raised the possibility of impurities (phosgene) or decomposition products (hydrogen cyanide, nitrogen oxides, carbon monoxide) being present in the gas cloud.

Suspicion that some of the early deaths were due to cyanide poisoning arose from the reporting of rapid fatalities and acute symptoms of syncope, and extreme weakness. Autopsy studies done by Dr. Heeresh Chandra at the local medical college showed two features considered to be characteristic of cyanide poisoning. These were cherry-red discoloration of the blood and other organs, and the unpleasant odor of "bitter almonds" when the lungs were opened [48].

A double-blind clinical trial was performed by the ICMR 2 months after exposure to determine the efficacy of sodium thiosulfate as an antidote to the poisoning [12]. The trial was done on 30 gas-exposed symptomatic patients who were administered sodium thiosulfate, a cyanide antidote, intravenously. The results showed alleviation of symptoms with an 8-10 fold increase in thiocyanate excretion in the urine for 10 out of 15 patients and served to fuel suspicion that cyanide poisoning was involved. (Thiocyanate is the water-soluble detoxification product of cyanide).

On the basis of this study, the ICMR in February 1985 recommended the use of sodium thiosulfate on a mass scale for detoxification of symptomatic gas-exposed persons. Union Carbide denied the possibility of cyanide poisoning and toxicological experts around the world were either unsure or skeptical about cyanide [1].

In this atmosphere of confusion, the local health authorities in Bhopal failed to carry out the recommendations of the ICMR on a widespread and systematic basis.

The scientific hypotheses seeking to explain the source of the cyanide poisoning and the apparent effectiveness of sodium thiosulfate fell into three categories:

1. *Cyanide from an external source*: That the entry and mixing of water with MIC in the storage tank resulted in a violent exothermic reaction at high temperatures (> 420°C)) which led to the decomposition of MIC to hydrogen cyanide, carbon monoxide and nitrogen oxides. This reaction was first discovered by Blake and Maghsoodi in 1982 and was documented in the NIOSH Occupational health guidelines for MIC [49].

2. *Cyanide from an internal source*: That the MIC was being converted to some form of cyanide after being absorbed into the body resulting in enlargement of the body's cyanogen pool.

3. *Thiosulfate effective against MIC*: That sodium thiosulfate was alleviating the symptoms caused by MIC toxicity. (Sodium thiosulfate was hitherto unknown as an MIC antidote).

Investigators from India [50] investigated the first of the three hypotheses by pyrolysing MIC at 350°C and found that cyanide was produced even at this lower temperature as a decomposition product. They further injected the decomposition products into rats and found that brain cytochrome oxidase activity (the biochemical basis for cyanide toxicity) was significantly depressed.

There has been no evidence to date to support the second hypothesis that MIC is converted to a form of cyanide in the body. Animals exposed to MIC by inhalation have not shown any evidence of cyanide in the blood [51].

Animal studies testing the third speculation have not shown that sodium thiosulfate has any protective effect against direct MIC toxicity [52,53].

If one accepts the possible efficacy of thiosulfate in alleviating symptoms of gas-exposed persons, it must be in the light of the first hypothesis that the cyanide poisoning came from an external source.

Studies to determine chemical composition of the MIC tank residues are currently being undertaken by the ICMR and may shed some light on the toxins released in the accident [10]. Whether cyanide was actually released may never be known with absolute certainty as it involves recreation of the actual conditions leading to the accident.

In the Bhopal situation, administration of an essentially harmless drug like sodium thiosulfate on a mass scale, with adequate follow-up, would have enabled gathering data which would confirm or reject the usefulness of the drug.

Andersson [54], however, points out that acute MIC exposure is more toxic than cyanide and an undue emphasis on cyanide may result in an underestimation of the long-term effects of MIC.

Persistent multi-systemic symptoms have been reported by Bhopal survivors. That exposure to MIC can produce reproductive health problems has been shown in human and animal studies [55]. Ferguson and Alarie [56] have demonstrated that there may be a physiopathological basis for the persistence of multi-systemic symptoms in Bhopal survivors. Their studies on experimental animals have shown that radio-labeled MIC is capable of being absorbed and distributed throughtout the body. These findings have been confirmed by Bhattacharya *et al.* [57] who have shown that MIC binds covalently to tissue proteins in its active form and not as its breakdown product, methylamine.

There is no known antidote for MIC toxicity. If further studies confirm that MIC is indeed distributed in the body, there is a need to develop a method of treatment for MIC poisoning.

Epidemiologic Considerations

In the early period following the accident, clinical treatment of the injured took priority over the planning and conduct of epidemiological studies. This was particularly true given the limited health care resources available for a large number of affected people and the general lack of experience in dealing with a disaster of this nature.

A few cross-sectional studies (Table I) were done during the early recovery period (6 months) for various systemic health end-points. Virtually all of the epidemiology for the late recovery period is being conducted by the Bhopal Gas Disaster Research Centre, a branch of the Indian Council of Medical Research. About 10 different epidemiological studies were initiated to monitor long-term trends in morbidity and mortality. Results from these studies are currently being awaited. As Bertazzi [58] notes, the selection of a cohort rather than using a population registry for epidemiological studies avoids two major biases: dilution of exposure prevalence and selective migration of people out of the disaster area.

The early cross-sectional studies suffer from a number of defects in study design, resulting in bias and consequent difficulty in clearly establishing causal relationships. In one follow-up study performed 3 months after the accident, exposed persons were contacted and requested to come forward voluntarily for examination [22]. This method of subject selection may have resulted in severely exposed/affected subjects being examined and a consequent over-estimation of health effects. If, however, the severely affected victims were unable to present themselves for examination due to illness, an under-estimation of health effects may have occurred.

Relatively crude methods like mortality rates or distance of residence from the factory have been used in defining community exposures. Koplan *et al.* [59] stated that epidemiologic studies following disasters should accurately estimate exposure to enable correct dose-response relationship modeling. These data are useful for a) identifying exposed and ill persons, b) determine long-term effects and c) link exposure and effects for litigation and compensation.

For the Bhopal gas victims, there is a need to do epidemiological studies to determine morbidity prevalence in the population stratified by estimated pulmonary dose. Such an approach will allow scientifically valid and detailed studies of different health end-points to be performed on relatively small sample sizes [60]. Exposure-based stratified random sampling will also reduce bias due to self-selection and exposure misclassification, as well as permit dose-response and interaction relationships to be understood.

Conclusions

Clinical and toxicological studies have shown that MIC is a potent toxin. Chronic inflammation of the eye and respiratory tract account for a major portion of the morbidity. Certainly the potential exists for these damaged organs to be more susceptible to other environmental insults like infections, irritants, and allergens. For e.g., a person with airway damage

may be more prone to infections or respond adversely to smoke and dust. Pulmonary function limitation may preclude survivors from working on jobs which require moderate or strenuous activity. Progressive pulmonary fibrosis and restrictive lung disease appear to be a major cause for concern among the gas-exposed. Given the completely unexpected and devastating nature of the disaster and the resultant stress, it is expected that a number of survivors will suffer from post-traumatic stress disorders for many years. Establishment of a specialised medical center for dealing with health problems from the gas leak will permit a co-ordinated method of investigation and treatment for the injured.

It is a striking fact that much of the mortality and morbidity could have been averted by the simple expedient of covering the face with a wet cloth. MIC would have been decomposed on contact with the water. Unfortunately, the community was never informed of the existence of such a potent chemical in the factory and contingency measures to be taken in the event of a leak.

For a disaster of this magnitude, there is a relative paucity of knowledge based on epidemiological and clinical investigation. Some of the medical studies done on the survivors suffer from unscientific design and it is difficult to utilise information from these studies with confidence. Stratified sampling techniques using isopleths of exposure from dispersion models may be one way of conducting an epidemiological study without including the total exposed population.

It is imperative that long-term monitoring of the affected community be done for at least the next fifty years. Formal studies of ocular, respiratory, reproductive, immunological, genetic and psychological health must be continued to understand the extent and severity of long-term effects of the disaster.

Acknowledgements

The author wishes to thank the following persons for their helpful comments and criticisms: Carol and

Table 1 - Human health effects studies on the Bhopal Gas Leak
Tabella 1 - Studi degli effetti sulla salute umana dell'incidente di Bhopal

Author	*Design*	*n (cases/controls)*	*Study Period (time after gas leak)*	*Major findings*
Ocular Studies				
Andersson et al., 1984, (4)	Case-series	8000 exposed	8 days	chemosis, corneal ulcers, watering photophobia. no blindness
Andersson et al., 1988, (13)	Cross-sectional	261/ 99	2 weeks	photophobia corneal erosions
Dwivedi et al., 1985, (15)	case-series	232	a few weeks	chemosis, redness watering, corneal ulcers
Andersson et al., 1985, (69)	case-series	490	2 months	corneal scars no blindness
Maskati, 1986, (61)	Cross-sectional	261/106	104 days	conjunctivitis corneal opacities
Andersson et al., 1986, (10)	Case-series	989	9-12 months	persistent watering
Raizada, & Dwiwedi 1987, (17)	Case-series	1140	2 years	corneal opacities, chronic conjunctivitis
Respiratory Studies				
Mishra et al., 1987, (70)	Case-series	978	2 days	resp. distress, pulm. edema, pneumonitis
Sharma & Gaur, 1987, (62)	Case-series	500 X-rays	> 72 hrs	pulm. edema, emphysema, pneumothorax
Misra & Nag 1988, (20)	Case-series	33	1 week	Dyspnea, upper and lower respiratory tract irritation. pulm. edema, pneumonia
Bhargava et al., 1987, (63)	Case-series	224	1-4 months	Obstructive & restrictive lung disease
Gupta et al., 1988, (22)	Cross-sectional	1109	2.5 months	65 subjects with +ve X-ray changes
Kamat et al., 1985, (64)	Case-series	113	3 & 6 months	Emphysema, pulm. hypertension pleural scars, interstitial depositis
Medico Friend Circle 1986, (9)	Cross-sectional	136/137	4 months	Obstructive & restrictive lung disease
Reproductive Studies				
Varma, 1987, (30)	Survey	865	9 months	Increased pregnancy loss,
		486 live/births/ historic controls	9 months	increased infant mortality
Medico Friend Circle 1986, (31)	Survey	381/historic controls	9 months	Increased spont. abortion rate, alteration of menstrual cycle

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Author	Design	n (cases/controls)	Study Period (time after gas leak)	Major findings
Kanhere et al., 1987, (66)	Cross-sectional	134/24	9 months	Decreased placental & fetal weight
Daniel et al., 1987 - (67)	Cross-sectional	18/10	6 months	No effect on spermatogenic function
Genetic Studies				
Deo et al., 1987 (68)	Cross-sectional	22/13	11 days	Cell cycle delay
Saxena et al., 1988, (35)	Cross-sectional	31/31	2.5 months	Increased chromosomal aberrations
Ghosh et al., 1990, (36)	Cross-sectional	83/46	3 years	Increased chromosomal aberrations
Immune Function				
Deo et al., 1987, (68)	Cross-sectional	67/15	4-8 weeks	Decreased response to T & B cell mitogens
Saxena et al. 1988, (35)	Cross-sectional	44 19/8	2.5 months 2-5 months	Decreased T-cell popuiation, decreased phagocytic activity
Karol et al., 1987, (26)	Case-series	144	1-12 months	Transient MIC antibodies in 12 subjects
Psychological Studies				
Sethi et al., 1987, (46)	Case-series	208	2-6 months	Neuroses, anxiety states & adjustment reactions
Neurobehavioral Studies				
Gupta et al., 1988, (22)	Cross-sectional	350/100	2.5 months	Impaired auditory & visual memory, vigilance & attention response speed

Gerard Natarelli, Rosaline Dhara, Drs. Steven Markowitz, Rashid Sheikh, David Kriebel and David Wegman.

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Riassunto

Il peggior disastro industriale della storia si verificò in India la notte tra il 2 e il 3 dicembre 1984 a Bhopal, città di 900000 abitanti e capitale dello stato di Madhya Pradesh. L'incidente fu probabilmente iniziato dall'introduzione di acqua nelle cisterne deposito di Metilisocianato (MIC) e dalla conseguente reazione incontrollata con liberazione di calore e di MIC sotto forma di gas. I sistemi di sicurezza o non funzionarono o furono inadeguati per il grande volume di sostanza chimica coinvolta nella reazione.

Questo lavoro passa in rassegna gli effetti dell'esposizione al MIC così come sono conosciuti da studi umani e discute alcuni degli aspetti clinici ed epidemiologici dibattuti.

Per quanto riguarda l'esposizione della popolazione si stima che circa 27 tonnellate di MIC fuoriuscirono dalle cisterne in un arco di 1-2 ore [1]. L'incidente avvenne intorno a mezzanotte in condizioni metereologiche avverse che prevennirono la dispersione del gas e fecero ristagnare sulla zona una nube tossica [7].

Sitme basate sulla quantità di sostanza fuoriuscita e sull'area interessata (40 miglia quadrate) pongono la concentrazione di MIC nell'area a 27ppm, circa 1400 volte lo standard OSHA per i luoghi di lavoro di 0.02 ppm durante le 8 ore [7]. Simulazioni che tenessero conto della variabilità di concentrazione di MIC identificano 27 aree con concentrazioni varianti da 85,6 ppm a 0,12 ppm con una mediana di 1·8 ppm [8]. La dose assunta dipese anche dai comportamenti individuali al momento dell'incidente. In uno studio su un campione di 158 persone, ad esempio, 124 fuggirono dalle case (aumentando quindi con la ventilazione polmonare la quantità di sostanza assunta) e solo 12 utilizzarono stoffa umida davanti alla bocca come forma di protezione [9].

Una coorte di 80021 soggetti fu identificata nella popolazione residente intorno allo stabilimento della Union Carbide e classificata come esposta in modo lieve, moderato e severo sulla base dei dati di mortalità delle diverse aree. Un gruppo di popolazione di Bhopal non esposta (1993 soggetti) venne identificato come controllo. Nelle aree esposte rispettivamente il 53%, 46% e 1% erano di religione indù, musulmana, cristiana o sikh. L'80% della popolazione aveva un reddito inferiore a 6 dollari al mese e solo l'1% superiore a 18 dollari. La soglia di povertà è stabilita dal governo in 12 dollari al mese. Solo il 34% della popoiazione viveva in una casa stabile ("pacca" house) [10].

Delle 200000 persone esposte 2500 morirono nella settimana successiva all'incidente. Al novembre 1989 risultava un totale di 3598 vittime decedute prevalentemente per complicazioni respiratorie. Nel periodo maggio 1989 - marzo 1990 la mortalità generale risultava essere più elevata nelle aree esposte (8.75/1000) relativamente alle aree di controllo (7.5/1000) [10].

Surveys sulla prevalenza di sintomi indicano una morbidità più elevata nelle zone esposte (26%) rispetto alle aree di controllo (18%).

L'articolo e la tabella 1 passano in rassegna i lavori condotti sulla patologia oculare, respiratoria, riproduttiva, sulla genotossicità, cancinogenicità e immunotossicità del MIC sulle popolazioni esposte ed i problemi psicologici e di tossicità neuroambientale e neuromuscolare. Vengono infine valutati i problemi chimici collegati ad una possibile tossicità di altri prodotti presenti nella nube tossica e potenzialmente formatisi durante la reazione di idrolitica ed esotermica.

Considerazioni generali riguardanti gli studi epidemiologici condotti nell'area di Bhopal sono:

1. Nel periodo immediatamente seguente l'incidente la totalità degli sforzi medici si concentrarono sulla cura del numero elevatissimo di intossicati e solo in un secondo periodo vennero pianificati studi al fine di valutare gli effetti cronici ed acuti dell'esposizione alla nube tossica.
2. Gli studi trasversali, i primi ad essere disegnati, soffrirono di numerosi problemi nel disegno dello studio. Gli studi prospettici vennero disegnati con maggiore attenzione agli aspetti delle distorsioni di selezione.
3. È importante ricordare che larga parte della mortalità e morbidità avrebbe potuto essere evitata con misure di protiezione individuale molto semplici, quale il coprirsi il viso con un panno bagnato. Purtroppo la comunità non era mai stata informata nè dalla presenza di tossici così potenti nello stabilimento, nè di forme di protezione individuale da applicare in caso di incidente.

Summary

The methyl isocyanate (MIC) gas leak from the Union Carbide plant at Bhopal, India in 1984 was the worst industrial disaster in history. Exposure estima-

tes of gas concentrations in the area range from 85 to 0.12 ppm. Of the approximately 200,000 persons exposed, 3598 deaths have resulted as of November 1989. Chronic inflammatory damage to the eyes and lungs appears to be the main cause of morbidity. Reproductive health problems in the form of increased spontaneous abortions and psychological problems have been reported. Questions about the nature of MIC toxicity have been raised by the persistence of multi-systemic symptoms in survivors. Animal studies using radio-labeled MIC given by the inhalation route have shown that the radio-label is capable of crossing the lung membranes and being distributed to many organs of the body. This paper reviews health effects of gas exposure from published studies and discusses some of the clinical and epidemiological issues being debated.

References

1. MOREHOUSE W, SUBRAMANIAM MA. *The Bhopal Tragedy: What Really Happened and What it Means for American Workers and Communities at risk*. Council on International & Public Affairs, New York 1986.

2. BUCHER JR. Methyl Isocyanate: A review of health effects research since Bhopal. *Fund Appl Toxicol* 1987; 9: 367-379.

3. MEHTA PS, MEHTA AS, MEHTA SJ, MAKHIJANI AB. Bhopal Tragedy's Health Effects: A review of Methyl Isocyanate Toxicity. *JAMA* 1990; 264: 2781-2787.

4. BLAKE PG, IJADI-MAGHSOODI S. Kinetics and Mechanism of the Thermal Decomposition of Methyl Isocyanate. *Int J Chem Kinetics* 1982; 14: 945-952.

5. SAX N. IRVING. *Dangerous Properties of Industrial Materials*. 5th ed., New York, Van Nostrand Reinhold Co. 1979; pp. 24.

6. KIMMERLE G, EBEN A. Zur toxicitat von methylisocyanat und dessen quantitiver bestimmung in der luft. *Arch Toxicol* 1964; 20: 235-241.

7. Central Water & Air Pollution Control Board Report: Gas Leak Epidemic at Bhopal, New Delhi 1985.

8. SINGH MP, GHOSH S. Bhopal Gas Tragedy: Model Simulation of the Dispersion Scenario. *Journal of Hazardous Materials* 1987; 17: 1-22.

9. Medico Friend Circle. *The Bhopal Disaster Aftermath: An Epidemiological and Socio-Medical Study*. Bangalore, India, 1985.

10. Indian Council of Medical Research. Bhopal Gas Disaster Research Centre. Annual Report, Bhopal, India, 1990.

11. Dept. of Relief & Rehabilitation: *Bhopal Gas Tragedy*. Govt. of Madhya Pradesh, Bhopal, India, 1989.

12. Indian Council of Medical Research. Annual report: Bhopal Gas Disaster Research Centre. *Annual Report*, Bhopal, India, 1991.

13. ANDERSSON N, KERR MUIR M, MEHRA V, SALMON AG. Exposure and response to methyl isocyanate: results of community-based survey in Bhopal. *Br J Ind Med* 1988; 45: 469-475.

14. ANDERSSON N, MUIR MK, MEHRA V, Bhopal Eye. *Lancet* 1984; 2: 1481.

15. DWIVEDI PC, RAIZADA JK, SAINI VK, MITTAL PC. Ocular Lesions following methyl isocyanate contamination: the Bhopal experience. *Arch Ophthalmol* 1985; 103: 1627.

16. ANDERSSON N, KERR MUIR M, AJWANI MK, MAHASHABDE S, SALMON A, VAIDYANATHAN K. Persistent eye watering among Bhopal survivors. *Lancet* 1986; 2: 1152.

17. RAIZADA JK, DWIVEDI PC. Chronic ocular lesions in Bhopal gas tragedy. *Ind J Opthalmol* 1987; 35: 453-455.

18. SALMON AG, KERR MUIR M, ANDERSSON N. Acute Toxicity of methyl isocyanate: a preliminary study of the dose response for eye and other effects. *Br J Ind Med* 1985; 42: 795-798.

19. ANDERSSON N, AJWANI MK, MAHASHABDE S, TIWARI MK, KERR MUIR M, MEHRA V, ASHIRU K, MACKENZIE CD. Delayed eye and other consequeces from exposure to methyl isocyanate 93% follow up of exposed and unexposed cohorts in Bhopal. *Br J Ind Med* 1990; 47: 553-558.

20. MISHRA UK, NAG D. A Clinical Study of Toxic Gas Poisoning in Bhopal, India. *Ind J Exp Biol* 1988; 26: 201-204.

21. Indian Council of Medical Research. Health Effects of Exposure to Toxic Gas at Bhopal: an update on ICMR sponsored researches, New Delhi 1985

22. GUPTA BN, RASTOGI SK, CHANDRA H, MATHUR AK, MAHENDRA PN, PANGTEY BS, KUMAR P, SETH RK, DWIVEDI RS, RAY PK. Effect of exposure to toxic gas on the population of Bhopal: Part I-Epidemiological, clinical, radiological & behavioral studies. *Ind J Exp Biol* 1988; 26: 149-160.

23. RASTOGI SK, GUPTA BN, HUSAIN T, KUMAR A, CHANDRA S, RAY PK. Effect of exposure to toxic gas on the population of Bhopal: Part II-Respiratory Impairment. *Ind J Exp Biol* 1988; 26: 161-164.

24. VIJAYAN VK, PANDEY VP, SANKARAN K, MEHROTRA Y, DARKARI BS, MISRA NP. Bronchoalveolar lavage study in victims of toxic gas leak in Bhopal. *Ind J Med Res* 1989; 90: 407-414.

25. SHARMA S, NARAYANAN PS, SRIRAMACHARI S, VIJAYAN VK, KAMAT SR, CHANDRA H. Objective thoracic CT scan findings in a Bhopal gas disaster victim. *Respir Med* 1991; 85: 539-541.

26. KAROL MH, TASKAR S, GANGAL S, RUBANOFF BF, KAMAT SR. The antibody response to methyl isocyanate: Experimental and clinical findings. *Environ Health Perspect* 1987; 72: 167-173.

27. BOORMAN GA, URAIH LC, GUPTA BN, BUCHER JR. Two hour methyl isocyanate inhalation and 90 day recovery study in B6C3F1 mice. *Environ Health Perspect* 1987; 72: 5-11.

28. FOWLER EH, DODD DE. Respiratory tract changes in guinea pigs, rats and mice following a single six-hour exposure to methyl isocyanate. *Environ Health Prospect* 1987; 72: 107-114.

29. BANG R & SADGOPAL M. Effect of Bhopal disaster on womens' health-an epidemic of gynecological diseases (Part I), 1985, unpublished data.

30. VARMA DR. Epidemiological and experimental studies on the effects of methyl isocyanate on the course of pregnancy. *Environ Hlth Perspect* 1987; 72: 151-155.

31. Medico Friend Circle: Effect of Bhopal Gas Leak on Women's Reproductive Health. Published by Padma Prakash on behalf of Medico Friend Circle at IBCS, Bombay, India, 1986.

32. SCHWETZ BA, ADKINS B Jr, HARRIS M, MOORMAN M, SLOAN R. Methyl Isocyanate: Reproductive and developmental toxicology studies in Swiss mice. *Environ Hlth Perspect* 1987; 72: 147-150.

33. VARMA DR, FERGUSON J, ALARIE Y. Reproductive toxicity of methyl isocyanate in mice. *J Toxicol Env Hlth* 1987; 21: 265-275.

34. VARMA DR, GUEST I, SMITH S, MULAY S. Dissociation between maternal and fetal toxicity of methyl isocyanate in mice and rats. *J Toxicol Env Hlth* 1990; 30: 1-14.

35. SAXENA AK, SINGH KP, NAGLE SL, GUPTA BN, RAY PK, SRIVASTAV RK, TEWARI SP, SINGH R. Effect of exposure to toxic gas on the population of Bhopal: Part IV - Immunological and Chromosomal Studies. *Ind J Exp Biol* 1988; 26: 173-176.

36. GHOSH BB, SENGUPTA S, ROY A, MAITY S, GHOSH S, TALUKDER G, SHARMA A. Cytogenetic Studies in Human Population Exposed to Gas Leak at Bhopal, India. *Environ Hlth Perspect* 1990; 86: 323-326.

37. MASON JM, ZEIGER E, HAWORTH S, IVETT J, VALENCIA R. Genotoxicity studies of methylisocyanate in Salmonella, Drosophila and cultured Chinese hamster ovary cells. *Environ Mutagen* 1986; 8: 19-28.

38. SHELBY MD, ALLEN JW, CASPARY WJ, HAWORTH S, IVETT J, KLIGERMAN A, LUKE CA, MASON JM, MYHR B, TICE RR, VALENCIA R, ZEIGER E. Results of in vitro and in vivo genetic toxicity tests on methyl isocyanate. *Environ Hlth Perspect* 1987; 72: 181-185.

39. MESHRAM GP, RAO KM. Mutagenic and toxic effects of methylisocyanate (MIC) in Salmonella typhimurium. *Ind J Exp Biol* 1987; 25: 548-550.

40. TICE RR, LUKE CA, SHELBY MD. Methyl Isocyanate: an evaluation of in vivo cytogenetic activity. *Environ Mutagen* 1986; 9: 37-58.

41. CONNER MK, RUBINSON HF, FERGLSON JS, STOCK MF & ALARIE Y. Evaluation of sister chromatid exchange and cytogenicity in murine tissues in vivo and lymphocytes in vitro following methyl isocyanate exposure. *Environ Hlth Perspect* 1987; 72: 115-121.

42. BUCHER JR, URAIH LC. Carcinogenicity and Pulmonary Pathology Associated With a Single 2-Hour Inhalation Exposure of Laboratory Rodents to Methyl Isocyanate. *J Nat Canc Inst* 1989; 81: 586-587.

43. ENNEVER FK, ROSENKRANZ HS. Evaluating the potential for genotoxic carcinogenicity of methyl isocyanate. *Toxicol Appl Pharmacol* 1987; 91: 502-505.

44. DWIVEDI PD, MISHRA A, GUPTA GSD, DUTTA KK, DAS SN, RAY PK. Inhalation toxicity studies of methyl isocyanate (MIC) in rats: Part IV-Immunologic response of rats one week after exposure: effect on body and organ weights, phagocytic and DTH Response. *Ind J Exp Biol* 1988; 26: 191-194.

45. SRINIVASAMURTHY R, ISAAC MK. Mental health needs of Bhopal disaster victims and training of medical officers in mental health aspects. *Ina J Med Res* 1987; 86 (Suppl): 51-58.

46. SETHI BB, SHARMA M, TRIVEDI HK, SINGH H. Psychiatric morbidity in patients attending clinics in gas affected areas in Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 45-50.

47. ANDERSON D, GOYLE S, PHILIPS BJ, TEE A, BEECH L, BUTLER WH. Effect of methyl isocyanate on rat muscle cells in culture. *Br J Ind Med* 1988; 45(4): 269-274.

48. FERA I. The Day After. *Illus Week of India* Dec 30, 1984. In *Bhopal: Industrial Genocide?* Hong Kong, 1985. *Arena Press*.

49. National Institute of Occupational Safety & Health. *Occupational health Guidelines for Chemical Hazards: Methyl Isocyanate*, Us. Dept. of Health Human Services, Washington, D.C., 1978.

50. BHATTACHARYA BK, MALHOTRA RC, CHATTOPADHYAY DP. Inhibition of rat cytochrome oxidase activity by pyrolysed products of methyl isocyanate. *Toxicol Lett* 1987; 37: 131-134.

51. BUCHER JR, GUPTA BN, ADKINS B Jr, THOMPSON M, JAMIESON CW, THIGPEN JE, SCHWETZ BA. Toxicity of Inhaled Methyl Isocyanate in F344/N Rats and B6C3F1 Mice. I. Acute Exposure and Recovery Studies. *Environ Health Perspect* 1987; 72: 53-61.

52. NEMERY B, SPARROW S, DINSDALE D. Methyl

Isocyanate: Thiosulphate does not protect. *Lancet* 1985; 2: 1245-1246.

53. VIJAYARAGHAVAN R, KAUSHIK MP. Acute toxicity of methyl isocyanate and ineffectiveness of sodium thiosulphate in preventing its toxicity. *Ind J Exp Biol* Aug 1987; 25: 531-534.

54. ANDERSSON N. Long-term effects of methyl isocyanate. *Lancet* 1989; 1: 1259.

55. DHARA VR. On the bioavailability of methyl isocyanate in the Bhopal gas leak. Editorial. *Arch Env Health* 1992 ; 47: 385-386.

56. FERGUSON JS, KENNEDY AL, STOCK MF, BROWN WE, ALARIE Y. Uptake and distribution of ^{14}C during and following exposure to [^{14}C] methyl isocyanate. *Toxicol Appl Pharmacol* 1988; 94: 104-117.

57. BHATTACHARYA BK, SHARMA SK, JAISWAL DK. In vivo binding of [^{14}C] methylisocyanate to various tissue proteins. *Biochem Pharmacol* 1988; 37: 2489-2493.

58. BERTAZZI PA. Industrial disasters and epidemiology: a review of recent experiences. *Scand J Work Environ Health* 1989; 15: 85-100.

59. KOPLAN JP, FALK H, GREEN G. Public Health Lessons from the Bhopal Chemical Disaster. *JAMA* 1990; 264: 2795-2796.

60. DHARA VR. KRIEBEL D. The long-term health consequences of the Bhopal gas disaster: dose-response studies. Apha 120th Annual Meeting Exhibition Abstracts. Washington, D.C., 1992: 130.

61. MASKATI QB. Ophthalmic survey of Bhopal victims 104 days after the tragedy. *J Postgrad Med* 1986; 32: 199-202.

62. SHARMA PN, GAUR KJBS. Radiological spectrum of lung changes in gas exposed victims. *Ind J Med Res* 1987; 86 (Suppl): 39-44.

63. BHARGAVA DK, VARMA A, BATNI G, MISRA NP, TIWARI UC, VIJAYAN VK, JAIN SK. Early observations on lung function studies in symptomatic 'gas'-exposed populations of Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 1-10.

64. KAMAT SR, MAHASHUR AA, TIWARI AK, POTDAR PV. Early observations on pulmonary changes and clinical morbidity due to the isocyanate gas leak at Bhopal. *J Postgrad Med* 1985; 31: 63-72.

65. KAMAT SR, PATEL MH, KOLHATKAR VP, DAVE AA, MAHASHUR AA. Sequential respiratory changes in those exposed to the gas leak at Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 20-38.

66. KANHERE S, DARBARI BS, SHRIVASTAVA AK. Morphological study of expectant mothers exposed to gas leak at Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 77-82.

67. DANIEL CS, SINGH AK, SIDDIQUI P, MATHUR BBL, DAS SK, AGARWAL SS. Preliminary report on spermatogenic function of male subjects exposed to gas at Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 83-86.

68. DEO MG, GANGAL S, BHISEY AN, SOMASUNDARAM R, BALSARA B, GULWANI B, DARBARI BS, BHIDE S, MARU GB. Immunological, mutagenic and genotoxic investigations in gas exposed population of Bhopal. *Ind J Med Res* 1987; 86 (Suppl): 63-76.

69. ANDERSSON N, KERR MUIR M, SALMON AG, WELLS CJ, BROWN RB, et al. Bhopal Disaster: Eye follow-up and analytical chemistry. *Lancet* 1985; 1: 761-762.

70. MISRA NP, PATHAK R, GAUR KJBS, JAIN SC, YESIKAR SS, MANORIA PC, et al. Clinical profile of gas leak victims in acute phase after Bhopal episode. *Ind J Med Res* 1987; 86 (Suppl): 11-19.

La Cooperativa Epidemiologia e Prevenzione annuncia il

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Istituto per lo Studio e la Cura dei Tumori "Fondazione Pascale"
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Napoli, lunedì 6 – venerdì 10 settembre 1993

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- sul carcinoma della cervice (problema del Papilloma Virus)
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- sul carcinoma dell'intestino (problema della famiglia ad alto rischio)
- sugli altri tumori per cui sono stati proposti programmi di screening (endometrio, ovaio, tumori cutanei, prostata, neuroblastoma)

Il problema del riconoscimento dei soggetti ad alto rischio

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3 – Relazione fra screening in campo oncologico e cardiovascolare
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- Confronti geografici e temporali
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10 – Organizzazione del sistema informativo per monitorare l'efficienza e l'efficacia in differenti situazione operative

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OHI : 28.

People's right to information — Interview with Dr. Thelma Narayan

I did my schooling in Bhopal and we used to stay in the Polo Bungalow (which is now a thirty-bed Community Hospital). The Union Carbide Factory came up in the seventies just opposite where we stayed.

I belong to a group called Medico Friend Circle, a group of doctors and other people who are involved in Health issues. It is an All India group. Dr. Ravi Narayan, my hunsband, is the convenor of this group and the organisational office of this group is here in Bangalore. Other voluntary groups, who knew about MFC needed an objective assessment of the situation, and requested our group to send a team to Bhopal.

About eleven MFC members along with three friends from Baroda Medical College had gone to make a study. What we learned was that on day one, on the 3rd day of December itself the Professor of Forensic Medicine, Dr. Heeresh Chandra who conducted the autopsies on the bodies felt that the picture he saw was something like Cyanide poisoning. The blood was cherry red in appearance whereas normally it would be bluish. He suggested that the antidote to Cyanide poisoning, namely Sodium Thiosulfate be given. The local medical community scoffed at him and said that he was a doctor of the dead and that he shouldn't be talking about the living.

At the same time there was a telex message from the medical director of Union Carbide from America, Dr. Bipin Avashia, saying that the treatment should be symptomatic. He also happened to mention that if Cyanide poisoning was suspected to give Sodium Thiosulfate. But a few days later when he did come to Bhopal he withdrew the statement.

A German toxicologist was invited to come to Bhopal by the Government of India. He arrived about four or five days after the disaster, Dr. Mar Daunderer. He brought with him various tools of investigation. Based on that he also felt that it was some Cyanide-like poisoning and he suggested that Sodium Thiosulfate be given to the patient. Anyway there was a lot of opposition to this and he was soon packed off to Germany.

ICMR carried out a very detailed study on the efficacy of Sodium Thiosulfate, the study, called a double blind study is one of the scientific method of studies. On the analysis of the data ICMR found that when given Sodium Thiosulfate the patients had very significant symptomatic relief. On the basis of this study they made certain recommendations to the Government of India that the affected victims should be given Sodium Thiosulfate. They gave certain criteria to select the patients. This was at the end of January and a press release was given in the first week of February to the same effect. The Central Government obviously accepted this. But there was a hitch at the State level because ultimately health is a State subject and the State Health authorities have to take action on any recommendations given by either ICMR or the Central Government. No action was taken on this recommendation.

The local voluntary groups who had a scientific background kept raising this issue of Thiosulfate. Despite all their protests Thiosulfate was not given. The State health authorities had another meeting with ICMR. They took action in May and made Thiosulfate available through their clinics and dispensaries. The doctors in these clinics were totally unconvinced about its efficacy. We made a short survey and found that the drug was being given so slowly that it would take about seven years to cover the affected population. This was a totally ridiculous situation.

In June four groups of activists formed Jan Swasthya Samiti involving the basti people and volunteers from different sections. The West Bengal Drug Action Forum sent doctors on a voluntary basis to run clinics. Volunteers from MFC, and from Delhi and Bombay also helped. This group of doctors and other health workers devised a questionaire by which patients were monitored before and after the injections to see whether there would be a significant improvement.

In Bhopal one of the most active groups was Zahreeli Gas Kand Sangharsh Morcha with Dr. Anil Sadgopal and a band of volunteers.

OH1 : 29

"RATIONALE FOR THE USE OF SODIUM THIOSULPHATE AS AN ANTIDOTE IN THE TREATMENT OF THE VICTIMS OF THE BHOPAL GAS DISASTER - A REVIEW"

June 7, 1985

b y

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and

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Summary

Multisystemic involvement, persistence of symptoms, continued alteration of blood gas percentages indicative of poor oxygen carrying capacity of blood with resultant tissue anoxia, suggest that some toxin still exists in the body (the cyanogenic pool) and needs to be eliminated.

Significant symptomatic relief, increased excretion of urinary thiocyanates and improvement in the blood gas picture after administration of sodium thiosulphate indicates that it is an effective antidote in the treatment of the victims of the Bhopal gas disaster.

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C O N T E N T S

Part - I

Part - II

Review of literature on cyanide poisoning

Part - I

1. Introduction

Since the gas disaster in December, 1984, at Bhopal which killed thousands of people and left approximately 70,000 seriously injured and 45,000 with mild to moderate disability, controversy has surrounded the issue of an effective form of therapy for the affected population. This has led to the withholding of sodium thiosulphate (NaTS - an antidote, recommended by ICMR in February, 1985) and hence to the prolonged and unnecessary suffering of people.

After months of uncertainty the Madhya Pradesh Government Health Services officially sanctioned the use of sodium thiosulphate in April, 1985, and it reached the peripheral dispensaries and health centres only in May. To cover the affected population, at the rate set by the M.P. Government, i.e. 13 centres giving approximately 300 injections a day, complete detoxification will take years, by which time it may just be too late.

If the majority of victims are to receive this specific therapy in time it is essential to pressurize the government to tackle the problem on a war footing. There is a need to utilize all available resources in as efficient a manner as possible. Dispensaries and health centres run by non-governmental agencies too, need to be involved.

To undertake this task, medical personnel from the government and voluntary sector need to understand the rationale behind the use of sodium thiosulphate as one of the specific therapies for the gas affected victims.

This paper is intended to sort out some of the confusion that has arisen from the controversy regarding the use of sodium thiosulphate and hopefully to clarify some of the questions.

2. Clinical picture in the sub-acute phase

2.1 A common picture has emerged from observations by three independant studies, viz.

a) "Health effects of exposure to toxic gas at Bhopal. An update on ICMR sponsored researches", 10th March, 1985.[1]

b) "Medical survey on Bhopal gas victims" (March 1985), conducted by Nagrik Rahet aur Punarvas Committee, Bhopal, in collaboration with Voluntary Health Association of India, Delhi and Bhopal Relief Trust, Bombay.[2]

c) "The Bhopal disaster aftermath - a socio-medical and epidemiological survey" (March, 1985) - Medico Friend Circle.[3] (unpublished data)

2.2 In the affected areas, most persons had more than one symptom or group of symptoms. These have been grouped according to the body systems they belong to, with some inevitable overlap.[3]

a) Respiratory system - Breathlessness on rest and on accustomed exertion, dry cough, cough with expectoration, pain/tightness in the chest.

Persistent tachypnoea is a common feature.[1] Rales and rhonchi are present in 9.4%.[3] In some patients symptoms were out of proportion to the physical and radiological observations. 40% of those who complained of respiratory symptoms had ventilatory impairment, 12% had restrictive lung disease, 6% obstructive airway disease, 22% obstructive-cum-restrictive defect. Ventilatory defects were not observed in patients with mild exposure to toxic gas.[1]

Blood gas analysis in 35 patients indicates abnormality in the oxygen carrying capacity of haemoglobin. 23 patients with severe exposure had PaO_2 60 mm Hg. Arterial CO_2 tension 35 mm Hg. was observed in 12.[1]

b) Gastro-intestinal system - Loss of appetite, nausea, vomiting, abdominal pain and burning, flatulence, diarrhoea.

Endoscopic examination has revealed superficial gastritis and oesophagitis.[1]

c) Eye - Blurring of vision, irritation and burning of eyes, redness, difficulty in seeing bright light. 80% of people within ½ km had ophthalmic symptoms, as did 60% of those at 2 km and 40% of those at 8 km distance.[2]

d) Reproductive system : Women - Frequent menstrual periods, polymenorrhoea, abnormalities of menstrual flow - scanty or heavy, blackish in colour, excessive vaginal discharge, suppression of lactation.

Men - loss of libido.

e) Neuromuscular - Muscle weakness, sensation of pins and needles, tingling and numbness.

f) Mental - Headache, anxiety, apprehension, confusion, irritability, forgetfulness, grief, guilt or apathy, disturbed sleep, depression.

2.3 The picture which emerges is one of multisystemic involvement.

2.4 Present explanations for the cause of the above picture are:

a) after-effects of lung damage.

b) increase in the cyanogenic pool in the body.

c) psychosomatic manifestations as a result of the severe trauma experienced.

2.5 In each individual there is possibly an overlap and interplay of the three causative factors which result from variations in parameters such as distance from disaster site, type of house, action taken at the time of disaster, degree of bereavement, etc. The percentages of patients with symptoms predominantly due to any one of the above factors is not known.

3. A Flashback

3.1 Autopsy Findings

To begin with, there was a great deal of uncertainty as to the nature and composition of the gas that escaped. Initial autopsy findings showed a cyanide-like poisoning as the cause of death. Instead of the normal postmortem lividity and cyanosis, there was a pinkish discolouration of the organs. The arterial and venous blood and the organs had a characteristic bright "cherry-red" colour. Lung weight was 2.5 to 3 times the normal, heavily water-logged by oedema. There was also a varying degree of oedema of the brain. Autopsy findings even up to the 21st of December, 1984, remained similar.

3.2 Haemoglobin Examination

Spectroscopic and spectrophotometric studies of haemoglobin were carried out from the 14th to 21st of December. Control blood samples, samples of blood preserved in the deep freeze and fresh samples from subsequent autopsies were analysed. The samples did not show any evidence of the presence of carboxyhaemoglobin or cyanomethaemoglobin.[1] Further, samples from the victims showed the presence of the characteristic twin bands of oxyhaemoglobin.[1] This ruled out carbon monoxide poisoning which is also characterised by a cherry red appearance of the blood. Arterialization of venous blood as seen here is a characteristic of

cyanide poisoning also. Later, experimental studies in all animal species carried out so far have shown that MIC also produces a cherry-red appearance of the blood. A recent study also indicates a 26-60% reduction of the free amino groups in the haemoglobin of persons exposed to the toxic gas.

On estimating 2-3 DPG (diphosphoglycerate) in blood, levels were found to be raised in the gas-exposed population -- thereby indicating anoxia.

Haemoglobin percentage was found to be significantly higher among the affected population in Anna Nagar indicating compensatory polycythemia (to anoxia).[3]

3.3 Urinary Thiocyanates - Thiocyanates are normally present in urine and smokers show levels 50% higher than non-smokers.

The presence of increased levels of urinary thiocyanates (the form in which cyanide is excreted) in the affected victims also seemed to indicate a cyanide like poisoning. Taking 20 Delhi-based urine samples as a control, a base-line of 0.5 mg/100 ml of thiocyanate in urine was set as the upper limit of normal. In persons who were exposed to the toxic gas the urinary thiocyanate levels estimated were always greater than 2 mg/100 ml. In a study at Bhopal, it was observed that over 65% of the samples from the gas affected population and 70% of samples from hospital workers (indirect exposure) showed urinary thiocyanate levels greater than 1 mg/100 ml, while only 33% of samples from the non-exposed population had similar levels. After administration of NaTS, the level of thiocyanate in urine increased several folds initially and then showed a dip. On the basis of the experiments it is felt that urine thiocyanate level is a reasonably good indicator that will corroborate the other clinical manifestations of the toxic exposure and in such cases, administration of NaTS will produce significant increase in the thiocyanate excretion in urine.[4]

3.4 The initial Double Blind Study with Thiosulphate

Thirty patients were selected. They were given two injections of either sodium thiosulphate or glucose. Urinary thiocyanate levels were determined at 3 and 5 hour intervals and compared to the baseline level before the injection. In patients given sodium thiosulphate there was an 8-10 fold increase in excretion of thiocyanate in a significantly large number (10 out of 15). Only one of 15 receiving the glucose injection showed such increase.[1]

Subsequently 230 cases were treated with NaTS with over 1,000 injections. Of these complete records are available for 167 (87 men, 69 women and 11 children). The symptomatology at the time of inclusion of therapy was weakness and breathlessness. This was present at rest in 29 cases and in another 132 cases moderate exercise elicited the symptoms. Following NaTS administration, there was no effect in 9 cases. The remaining 158 persons showed varying grades of improvement for variable periods of time. There were no deaths. Adverse reactions were: 5 cases of feverishness, 1 case each of transient loss of memory, exaggerated reflexes, sense of 'heat' over the body, skin rash and transient venospasm.[4]

In another study various clinical symptoms were scored on an arbitrary scale so that in the worst case the score totalled 100. Of the 100 patients given NaTS therapy, 60 showed a decrease in score, 19 showed an increase, while 21 showed no change. The worse the initial symptoms the smaller the improvement after therapy. Of another 10 patients given intravenous glucose 8 showed reduction in score, but nocchange in their lung function. Another 10 were given intravenous aminophylline, all of whom claimed they felt better with their FVC, FEV_1, MEFR all showing improvement. However, the scoring system adopted needs improvement as different symptoms should not be given equal weightage nor should the scores be just added linearly. The question also arises as to why the results of lung function tests on the persons given NaTS were not reported. The findings of this study are, therefore, not conclusive.[4]

3.5 Blood gas analysis

In the initial study, blood gas was analyzed in 20 patients, using ABL gas analyzer and an oxymeter. PaO_2 was lower than normal with a range of 47.3 to 85.6 mm Hg. Those showing higher values had a history of treatment with sodium thiosulphate. 8 out of 14 untreated patients had arterial $PaCO_2$ of 35 mm Hg or less, which is below normal. The venous sample showed a normal or low PvO_2 value but an elevated $PvCO_2$ in 9 of 13 cases. The level of $PvCO_2$ after treatment with sodium thiosulphate was increased markedly in comparison to pre-treatment levels. The increase in $PvCO_2$ was proportional to improvement in clinical symptoms. The inference from the above is that oxygen transport was found to be affected leading to tissue anoxia which eventually produced less carbon dioxide in the peripheral veins. On treatment with NaTS, the increase in $PvCO_2$ occurred with predictable reproducibility. It was thought that owing to the inability of haemoglobin to take up carbon dioxide it was carried in solution in the blood with the partial pressure going up to 70 mm Hg.

On cardiac catheterisation the difference in levels of $PvCO_2$ between central and peripheral venous samples was negligible. Serial data showed that after administration of NaTS, $PvCO_2$ in both central and peripheral veins showed a significant rise indicating better uptake and utilisation of oxygen by the tissues. Studies have shown that pure MIC had no effect on cytochrome oxidase, but its degradation products did thus affecting oxygen uptake.[4]

3.6 Animal Experiments

MIC has an LD 50 dose of 85 mg in mice, but with thiosulphate therapy it is shifted to 195 mg. For rats, the figures are 270 and 344 respectively. Normal rabbit lungs weighed 6 gms, with MIC they weighed 29 gms and had a large number of haemorrhagic patches. When given NaTS immediately after MIC, lungs weighed 24 gms and appearance was near normal. Generally longer the duration after exposure, greater was the lung oedema observed. Salbutamol had no blocking effect on the action of MIC. Haematocrit increased to a high level.[4]

3.7 Cyanogenic Pool

The continued presence of the cyanide radical in the body of the gas affected victims increases the cyanogenic pool in the body. This was responsible for the multisystemic symptoms. An improvement in symptoms could occur only if the cyanogenic pool was depleted. (Refer to Part-II for greater details).

ICMR studies show that NaTS therapy gives significant symptomatic relief to people suffering after-effects of exposure to toxic gas. This relief can be objectively quantified by appropriate investigations. In general NaTS therapy has also been found to be harmless, although allergic reactions may occur in 1 out of 1,000 or 10,000 cases arising from imprities in the NaTS. Hence it has been recommended for use, besides other therapeutic measures, in selected patients.

4 Guidelines for the use of sodium thiosulphate (ICMR)
Criteria for selection of Patients -

1. Patients suffering from acute and/or chronic symptoms relating to the respiratory, gastro-intestinal and neuromuscular systems presumably related to possible exposure to MIC gas.

2. Patients presenting with recurrence of symptoms after having obtained some measure of relief from the acute phase.

3. Recorded cases of acute pulmonary oedema and/or coma occuring immediately following the episode and those who are currently symptomatic.

Contradictions

1. Pregnancy - routine use not advised, decision depends on severity of symptoms.

2. Renal disease.

Procedure

1. Record name, address.

2. Detailed record of symptoms and clinical findings should be made with special reference to a tendency

for early fatigue, exhaustion, respiratory signs and symptoms. This information should be recorded before and after treatment.
(Refer Appendix-I for a sample proforma)

3. Check urine for albumin.

4. Dose :

   a) Adults - 1 gm (10 cc of 10% NaTS or 4 cc of 25% NaTS) given intravenously, daily for a maximum of 5-6 days.

   b) Children - less than 2 years : 3 cc of 10% NaTS.
   2-8 years : 5 cc of 10% NaTS.
   more than 8 years : Adult dose.

5. Disposable syringes and needles are to be used for this purpose.

6. Alkaline mixture diuretic with sodium chloride should be administered to the patients to facilitate increased excretion of thiocyanate for the duration of therapy.

7. The patient should be kept under observation for at least one hour after the injection.

8. Pulse rate, respiratory rate, response to exercise and level of physical activity should be recorded during therapy and afterwards in order to assess the progress/improvement.

Side-effects/Precautions

1. It is a safe drug. Minor side-effects - feverishness, bodyache etc. respond to symptomatic treatment and antihistaminics.

2. Adverse reactions, if any, should be brought to the notice of local senior physicians for prompt management, and also to the health authorities.

3. Routine precautionary measures to manage anaphylactic reactions which are kept as stand-by's when giving any I.V. injections should be available in the dispensary, eg. I.V. fluids, steroids and antihistamine injections.

4. If extravascation of the drug occurs, the patient will have intense burning locally which is transitory and needs reassurance.

Drug Interactions - Nil.

Availability

1. The following are available with Dr. Tandon, Medical Superintendent, JP Hospital, 1250, South TT Nagar, Bhopal (Timings : 9 am - 1 pm and 4 pm - 6 pm) :
   a) Inj. Sodium Thiosulphate.
   b) Disposable syringes and needles.
   c) Alkaline mixture.
   d) Printed forms for recording and monitoring patients' symptoms and signs.
   e) Cotton, spirit, steroids, antihistaminies etc.
2. Presently NaTS stocks have been given to peripheral government dispensaries and polyclinics, Red Cross and a couple of voluntary agencies.

3. There is an urgent need for government and voluntary agencies to work in a coordinated manner and cover the affected population within 3-6 months.

4. OXFAM has also made an offer to help voluntary agencies interested in using thiosulphate for the affected population. Contact Nagpur office - Field Director, OXFAM (India) Trust, P.O. Box 71, Nagpur 440 001.

4. Conclusions

Scientific investigations show the continued presence of an increase in the cyanogenic pool in persons exposed to the toxic gas, even months after the disaster.

This is one of the factors responsible for continued morbidity among the affected population. The other known causative factors are structural lung damage and manifestations following severe psychic trauma.

Many areas, even regarding existing theories, are yet unknown and there is an urgent need for further study.

When on the basis of experiments and investigations therapeutic interventions are suggested it is essential that the logic for such measures is made open for scientific scrutiny. This information should also be made available to the treating medical personnel since it is irrational to expect physicians to prescribe drugs without being aware of the rationale for their use.

This is perhaps one of the many reasons why NaTS is at present being given half-heartedly at the cost of much ill-health, suffering and economic loss for thousands of helpless victims.

After the draft of this paper was ready, we came across a report of the Pollution Control Board giving the chemical analysis of samples of air collected on the 5th and 6th December, 1984. Despite the fact that these samples had been collected in the open air 3 to 4 days after the gas leak, these measurements show that the air near the MIC storage tank of the Union Carbide factory contained cyanide at a concentration of 4,533 mg/m^3 (4.5 ppm). This is nearly half the Maximal Allowed Concentration (MAC) and about 1/50th of the Lethal Concentration (LC) set for hydrogen cyanide. The cyanide level reduced to half this value 50 metres away from the tank and cyanide was not detected in samples collected from three other localities further away from the factory - the clear inference being that cyanide was still leaking from the MIC storage tank even on the 5th and 6th of December. The most probable origin of the cyanide must have been the thermal decomposition of the MIC stored in the ill-fated tank.

When one ties up the above mentioned evidence with the reports that cyanide was detected by the German toxicologist Dr. Max Daunderer in the blood of the gas victims and also by the CBI team in the ambient air using chemical spot tests, a few days after the tragedy, the case of NaTS therapy for the surviving gas-affected population becomes almost irrefutable.

5. References for Part-I

1. "Health effects of exposure to toxic gas at Bhopal", An update on ICMR sponsored researches, 10th March, 1985.

2. "Medical survey on Bhopal gas victims", conducted by Nagrik Rahat aur Punarvas Committee, Bhopal, in collaboration with Voluntary Health Association of India, Delhi and Bhopal Relief Trust, Bombay, March, 1985.

3. "The Bhopal disaster aftermath - a sociomedical and epidemiological survey", Medico Friend Circle, March, 1985.(unpublished)

4. Minutes of the third meeting of the Working Group on Thiosulphate Therapy of the MIC exposed population, 4th April, 1985.

Appendix - I

PROFORMA FOR THE CLINICAL MONITORING OF SODIUM THIOSULPHATE ADMINISTRATION

PEOPLE'S HEALTH PROGRAMME

Sl.No. _____ Date of Registration_____

Name _____ Sex_____ Age_____

Smoking : Yes/No Tobacco chewing : Yes/No

	What work do/did do	Monthly Income
Present Employment -		
Past Employment -		

Exposure to Gas

Heavy Moderate Mild

Where were you when the Gas leaked _____

Where did you run and how _____

Were you admitted to Hospital? If yes, which hospital?

_____

Did any family member die?

Treatment : Government Private

Fees _____

Expenses_____ Drug _____

Others _____

Compensation _____

What have you heard about proper treatment for the gas?

What have you heard about sodium thiosulphate?

SUMMARY TREATMENT AND INVESTIGATION

Previous Complaints	Yes	No	What
Nervous System	----	----	----
Eyes	----	----	----
Nose and Throat	----	----	----
Chest	----	----	----
G.I.T.	----	----	----
Skin	----	----	----
Genito Urinary	----	----	----
Musculo-skeletal	----	----	----

Treatment	Date	Details

Investigation	Date	Details

PRESENT SYMPTOMS/CHECK LIST

I. Nervous System

HEADACHE/HEAVINESS :

DIZZINESS :

ANXIETY :

POOR MEMORY :

LOSS OF SLEEP :

TINGLING AND NUMBNESS :

II. Eyes

BURNING OF EYES :

WATERING OF EYES :

DIMNESS/BLURRING OF VISION:

III. Nose and Throat

RUNNING NOSE/SNEEZING :

SORE THROAT :

IV. Chest and Cardiovascular system

PALPITATION :

BREATHLESSNESS :

CHEST PAIN :

DRY COUGH :

COUGH WITH EXPECTORATION :

HAEMOPTYSIS

V. Gastrointestinal System

ULCERS IN MOUTH :

LOSS OF APPETITE :

NAUSEA :

DISTENSION OF ABDOMEN :

PAIN ABDOMEN :

DIARRHOEA :

CONSTIPATION :

HAEMATEMESIS :

VI. Skin

ITCHING :

VII. Urogenital System

BURNING ON PASSING URINE :

LEUCORRHOEA :

IMPOTENCE :

PAIN ON INTERCOURSE :

VIII. SCANTY/HEAVY BLEEDING :

SHORTENED/LENGTHENED CYCLES :

IX. Musculoskeletal System

TIREDNESS/WEAKNESS :

FEVER :

PAIN IN THE CALF MUSCLES :

BACK PAIN/BODYACHE :

PAIN IN THE EXTREMITES :

X. Any others :

PRESENT SIGNS-CHECK LIST

ANAEMIA :

PULSE RATE :

RESPIRATORY RATE :

CYANOSIS :

CORNEA AND CONJUNCTIVA

ACUITY OF VISION :

SKIN INFECTION :

SKIN RASH :

SHIFT OF MEDIASTINUM :

NATURE AND CHARACTER OF BREATH SOUND :

ADDED SOUNDS :

OHI : 31.

SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

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Madhukar Pai
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Sundaram Medical Foundation
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5th November 1998

Dear Madhukar Pai, Anand Zakharia and Prabir Chaterjee

Re: *Invitation to Coordinate the Fact Finding Team on Medical Research in Bhopal – 15 Years on.*

It is now almost fourteen years since the Union Carbide Bhopal gas disaster. Yet the medical, legal, environmental, social and economic condition of the survivors is no better. In fact in many ways, it is much worse than it was in the immediate aftermath of the disaster in December 1984. Institutional indifference, apathy and corruption has compounded the difficulties faced by the survivors in their everyday struggle for justice and a dignified life. The inability of institutions to effectively respond to the world's worst industrial disaster and the resilient struggles of the survivors since 1984, has made it imperative that creative initiatives are taken to ensure that the people and lessons of the world's worst industrial disaster are not forgotten.

In the last fourteen years a number of groups have actively worked on the issues facing survivors. As you may know, a number of independent actions have also been taken to look at specific issues facing those affected. For instance, the Permanent Peoples' Tribunal on Industrial Hazards and the Environment (1990-94) gave survivors of Bhopal and other industrial disasters an international forum to testify on the continuing impact on their lives; and the International Medical Commission on Bhopal (1994) in which international experts assessed the ongoing health effects and state of medical care in Bhopal. Those working consistently in Bhopal, however, now feel that it is time to take stock of these and other issues and explore new directions for future long term work with and for the people of Bhopal.

Thus, in an attempt to go beyond previous studies, we propose an independent Fact Finding Mission, with specific teams to examine the current status of the issues faced by the survivors of the disaster; as well identify areas where critical interventions can be made. We hope that several teams comprised of experts, researchers and volunteers will come together to conduct studies and collate information over a period of six months. It is envisaged that a final report will be presented by July 1999 as a precursor to organizing working groups on specific project areas.

This is the first opportunity in 14 years that such a comprehensive initiative has been proposed. It is clear that the issues of Bhopal are very complicated and the reports of each team will have implications on other areas. The studies, highlighting the most effective ways forward will give those working on Bhopal fresh direction and support. The overall initiative, and the subsequent commissioning of working groups will be an effective tool in compelling an otherwise indifferent

SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

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government to remain accountable to the continuing suffering of the people of Bhopal. We intend that results from the working groups be presented in time for the fifteenth anniversary of the disaster next year.

We would like to invite you to participate in this independent initiative as a joint coordinator of the Fact Finding team on Medical Research in Bhopal, as well as taking responsibility for the completion of the final report. We have outlined, in consultation with those already working in Bhopal and survivors organizations, the issues which we feel are most pertinent and will need to be examined:

Medical Research

- Consolidation of studies carried out by government, private and other agencies and a reassessment of their major findings
- Analysis of the design and implementation of government studies
- Analysis of various studies conducted by Central Govt institutions for rehabilitation
- Outline of studies required for long term monitoring of health effects
- Assessment of the health status of gas affected persons, and their continuing exposure to hazards at both, the occupational and domestic levels
- What are the existing public health initiatives, and what is required?

We have requested Padma Prakash of the Economic and Political Weekly to assist in the coordination of this team.

We undertake to provide resources in terms of office-cum-accommodation and food in Bhopal. Whilst we expect to provide volunteers locally, we will need your assistance in identifying trained volunteers, specific logistics needed for the work, and whether it will be possible for the institution that you represent to offer any support. Any other suggestions on the parameters outlined would also be appreciated.

You will find enclosed a Backgrounder summarizing the issues facing those in Bhopal, the Parameters of the Fact Finding teams and Terms of Reference and a list of all those we are requesting to be part of this venture.

We will be launching the Mission on 30th Novemvber 1998, in Delhi. We very much hope it will be possible for you to attend. We would be extremely grateful if you could confirm your involvement as soon as possible.We look forward to your participation as a joint coordinator of the Fact Finding Team and thank you in advance for your co-operation and support.

With warm regards

E. Deenadayalan & Tarnjit Birdi

E. Deenadayalan & Tarnjit Birdi
On behalf of the Organizing Committee

SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

Backgrounder

Introduction :

On the midnight of 2nd - 3rd December 1984, the worst industrial disaster of this century was caused by Union Carbide Corporation, USA in the capital city of Bhopal, Madhya Pradesh, a city with about one million people. Over 40 tonnes of Methyl Isocyanate and other lethal gases including Hydrogen Cyanide, leaked from Carbide's pesticide factory in the northern end of the city killing over 8,000 people in its immediate aftermath, causing multisystemic injuries to over 500,000 people. The number of deaths has risen to over 16,000 in the subsequent years and there appears no end to the physical and mental suffering caused by exposure to the poisonous gases. Breathlessness, diminished vision , loss of appetite, pain, menstrual irregularities, recurrent fever, persistent cough, neurological disorders, fatigue, weakness, anxiety and depression are the most common symptoms. Research findings on chromosomal aberrations suggest that the future generations of the survivors will possibly carry ravages of the industrial toxins. Union Carbide continues to withhold toxicological information on the leaked gases thereby impeding medical treatment. The majority of those affected by the gases are people who earned their livelihood through hard physical labour and today their economic condition forces them to continue with their jobs, thus exposing themselves to further health risks. Little has changed in the living environment of the survivors, most of whom live in congested slums without facilities for safe drinking water, sanitation and clean air Judicial systems in both USA and India have failed to ensure adequate compensation and justice for the survivors. The settlement amount, an average of US $ 940 for each survivor, paid by Union Carbide resulted in a nominal loss to its shareholders of merely 50 cents per share. Compensation sums awarded for personal injury have been unjust and inadequate and in over 90% of cases the victims have received only about Rs15,000/- (or about $430). Nearly two hundred thousand persons directly affected by Union Carbides gases remain to be compensated. For a large number of the victims the sums received as compensation have been spent in repaying debts incurred in medical treatment in the last several years. Officials of Union Carbide who have been charged with manslaughter and other criminal offences are absconding from Indian courts where criminal proceedings against them been pending for the last six years. Every week in a public park in the city , hundreds of gas-affected women hold public meetings calling for the trial of the prime-accused Warren Anderson, former Chairman of the corporation, who is known to be on vacation at Vero Beach, Florida, USA.

Medical impact of the disaster:

Most of the information on the medical consequences of the Union Carbide disaster in Bhopal has been generated by the Indian Council of Medical Research (ICMR), an agency of the Indian government that carried out 25 research studies from 1985 to 1994. All ICMR studies in Bhopal were prematurely terminated by December 1994.

The ICMR established that the toxins from Carbide's factory have crossed into the blood stream of those exposed and have caused damage to the lungs, brain, kidneys, muscles as well as gastro- intestinal, reproductive, immunological and other systems.

Six monthly morbidity surveys by ICMR from 1987 to 1991, show that the number of people with exposure-related symptoms actually increased in that period. According to one study, there were three times more persons with respiratory symptoms in 1991 as compared to 1987. The damage to the respiratory system and particularly the lungs comprises the most obvious and very significant part of the overall health damage. Bronchial asthma, Chronic Obstructive Airways Disease, recurrent chest infections, and fibrosis of the lungs are the principal effects of exposure induced lung injury. The prevalence of pulmonary tuberculosis among the exposed population has been found to be more than three times that of the national average.

Damage caused to the eyes of the survivors have led to early-age cataracts found to be three times more prevalent among the exposed population compared to an unexposed population at the far end of the city. Pregnancy outcome studies on women who were pregnant at the time of the disaster have shown that the spontaneous abortion rate was almost three times that of the national average. In the exposed population the stillbirth rate was three times, perinatal mortality was two times and neonatal mortality was one and a half, times more than the comparative national figures. Study on growth and development of the children whose mothers were exposed to the toxic gases during pregnancy revealed that majority of children had delayed gross motor and language sector development. Studies have also presented evidence of chromosomal aberrations of gaps and breaks in the chromosomal material as well as increased sisterchromatid exchange indicating likelihood of congenital abnormalities among future generations of the exposed persons.

Toxic gas exposure was found by ICMR researchers to have had a detrimental effect on the immune system. Immunoglobulin levels were significantly raised , T-cells were reduced in number and there was a tendency towards the reversal of T4/T8 ratio indicative of immuno-suppression.

Various studies conducted by non-government organizations have pointed out that the medical consequences of the Union Carbide disaster have been under-assessed by the ICMR and certain exposure related injuries have been overlooked.

The pregnancy outcome survey referred to above was carried out by Medico Friend Circle in September '85 and showed that the spontaneous abortion rate among gas exposed women was several times higher than that reported by ICMR. A survey of psychiatric morbidity carried out by a group of independent doctors from Bombay found that nearly 40% of those exposed suffered from post-traumatic stress disorder, a condition not studied by ICMR.

An epidemiological survey coupled with clinical investigation carried out by the International Medical Commission on Bhopal, composed of 14 medical specialists from 11 different countries, reported in January 1994 significant multi-organ symptoms persistent among the exposed population. Clinical examinations have shown significant lung impairment, marked reduction in control over limb movements and reduced memory function caused due to exposure. Their findings include evidence of a range of neurotoxic injuries in the exposed population.

State of Medical Care and Monitoring :

The medical care of the survivors has largely remained symptomatic since the time of the disaster and continues to be ineffective in providing sustained relief. Union Carbide continues to withhold information on the composition of the leaked gases and their long term effects on the human body. In the absence of such information, doctors in Bhopal indiscriminately prescribe antibiotics, steroids and psychotropic medicines causing more harm than good. A study undertaken by the International Medical Commission on Bhopal confirmed that therapies prescribed for the ailing survivors are aimed at temporary symptomatic relief rather than long term amelioration of chronic disease processes. The major emphasis of the medical relief programmes of the Madhya Pradesh government has been to build hospitals so much so that Bhopal now has more per capita hospital beds than is recommended by the World Health Organization. Yet as per the reports of the ICMR the number of diseased persons has gone up with the years. For the last several months almost no medicines are available to the gas victims at any of the hospitals and clinics meant for them. People who do not have sufficient means for their basic needs are asked to buy medicines and even syringes and IV sets by doctors at these institutions that are supposed to provide free medical care. There has been hardly any government initiative in providing community based medical care to chronically ill survivors. Also lacking are initiatives for provision of medical care through systems of medicine such as Ayurveda, Unani and Yoga that have demonstrated their superior efficacy in the treatment of exposure related illnesses. The growing inadequacies of government medical care has led to unregulated proliferation of private and expensive medical clinics. With the termination of 25 medical research projects of the ICMR in December 1994, long term monitoring of the health condition of the survivors has been abandoned. The Centre for Rehabilitation Studies funded by the state government since March 1995 is yet to initiate any research work. The official agency for monitoring exposure related deaths has been wound up in December 1992 and there is no official record of deaths that continue to occur. With the premature termination of research and monitoring there is almost no current data on the prevalence of tuberculosis, cancers and infertility among the exposed population all of which are reported to be on the rise by doctors involved with the treatment of the survivors.

A recent issue of major concern is the proposed handing over of the health infrastructure set up the government to the so called Bhopal Hospital Trust set up by Union Carbide. Despite vigorous opposition by the survivors, the state government has begun handing over the eight community clinics to the Bhopal Hospital Trust (BHT) set up by Union Carbide Corporation. Preparations for transfer of the

four Red Cross clinics are also on. Ironically, one of the main factors impeding appropriate medical treatment at these government clinics has been withholding of medical information by Carbide and the closure of the Red Cross clinics followed from withdrawal of financial support by the corporation. The source of funds of the BHT is the value of shares of Union Carbide that had been judicially attached to ensure that the representatives of the corporation face criminal charges related to the disaster. By means of interventions in the Supreme Court through the sole trustee of BHT, Sir Ian Percival (an attorney working for Union Carbide from 1984 to '92) the corporation has been able to get the shares dis-attached and continues to abscond justice. In the last two years Percival has spent Rs. 7 crores on the construction of a 260-bed hospital ,which happens to be 8 kms away from the gas affected area, and Rs. 5 crores on his own fees, travel and office expenses. Quite clearly providing medical care to the survivors is not among the priorities of BHT. Its sole purpose is to build a humane image for the corporation while helping it to abscond criminal justice on the massacre. Percival's plans of health care administration have been severely criticized by national and international professional groups including the IMCB. Among other misgivings, concerns have been expressed regarding transparency of activities at the proposed medical research centre to be set up by the BHT.

Economic and Social Impact :

There has hardly been any systematic effort to document the social and economic impacts of the disaster. Official information on orphaned children and families that lost their breadwinners in the immediate or long aftermath is scanty, if available at all. Over 70% of the exposed population has been in the unorganized sector, with people earning subsistence wages through day labour or petty trade. A large number of men and women who pushed hand carts, carried loads, dug soil, repaired cars and did other jobs can no longer pursue their trades after being exposed to Carbide's gases. Gas exposed factory workers in textile and paper mills are more sensitive to occupational hazards and are absent from work due to illness as much as 15 days in a month. Over 90 % of the survivors have received a compensation of Rs 15,000 which is just enough for the cost of medicines for five years, and many of these people will be in need of medical care till the end of their lives. Given the complete inadequacy of official rehabilitation efforts the loss of regular income has driven tens of thousands of families to chronic starvation conditions. Loss of income also makes people borrow money from local money lenders who charge upto 200% interest so that chances of paying back are low and debts keep growing. Gas exposed women's inability to carry out reproductive functions have led to their desertion by their husbands and gas exposed young women continue to suffer social discrimination in marriage.

Environmental problems :

Thirteen years after the disaster, Union Carbide's toxic legacy continues to harm people in more ways than one. Communities in the vicinity of the Carbide factory continue to be exposed to toxic chemicals that are injurious to the lungs, liver and kidneys and can cause cancer. Water in over 200 wells around t[illegible]rbide factory have been declared unfit for human consumption by the municipal authoriti[illegible]is is a result of routine dumping of hazardous chemicals during the operation of the fac[illegible] thus

contaminating soil and groundwater in and around the factory premises. Analysis carried out by the Citizens Environmental Laboratory (CEL), Boston in 1991 show presence of toxic chemicals in the community wells around the factory. This report presented at the company's annual shareholders meeting drew the attention of the senior officials to the problem. The Corporation gave the job to Arthur D. Little Inc. who sponsored a collaborative investigation with an Indian government agency in 1994 in to the matter of contamination caused by dumping chemicals inside the factory premises. This study done without public knowledge recommends a fuller investigation for better assessment of the environmental contamination. The findings of this study confirm the worst apprehensions of activists and people in the community. Meanwhile, in 1996 the company management has dug up bottom soil from the "Solar Evaporation Ponds" and buried the heavily contaminated sludge under three metres of farm soil in a bid to cover up evidence of environmental damage. Survivors' organizations have been calling for an official assessment of the damage wrought by Union Carbide so that the corporation could be asked to pay the costs of environmental rehabilitation and supply of safe drinking water for the affected communities.

Economic Social and Environmental Rehabilitation :

The government programmes for economic rehabilitation have been badly designed and only few have been implemented. While an estimated population of 50,000 is in need of alternate jobs currently less than 100 gas victims have found employment under the government's scheme. 42 worksheds that had been built between 1985 to 1987 were allotted to so called NGOs between 1994 to 1996. However, apart from two worksheds employing about 50 women, none of them have ever been made available for employment of the survivors. Official promises have been on record that 50 % jobs in the Railway Coach Repair Factory would be reserved for gas affected people. However, only 205 of the 1000 employees of this factory are gas affected persons. In 1987 a special industrial area for training and employment of over 10,000 survivors was inaugurated and 152 worksheds were constructed at a cost of Rs.8 crores. However, till date not a single survivor has found any employment. A programme offering women survivors tailoring jobs ran successfully from 1986 to 1992 employing 2300 women and making an yearly profit of Rs.1 crore. The rehabilitation centres where these jobs were offered were also places where women survivors could gather, share their concerns and organize themselves. However, this programme was terminated without any reason in July 1992.

Till date the government has no record of the social condition of the persons who have been widowed, orphaned, or have been permanently disabled as a result of the gas disaster. The state government has deemed its work of social rehabilitation to be over by constructing 2500 houses and a few schools. There has been no official attention towards the urgent need of life long pension for widows, orphans, chronically ill and disabled survivors. The Supreme Court's final order with regard to provision of insurance coverage to about one lakh children likely to suffer delayed effects of the lethal gases is also being ignored by the Central government.

Despite the expenditure of over Rs. 70 crores in environmental rehabilitation basic necessities such as clean drinking water and sanitary facilities remain unavailable to majority of the gas affected communities.

Legal Aspects :

Subsequent to the disaster the Indian government through the Bhopal Gas Leak Disaster (Processing of Claims) Act in March 1985 arrogated to itself, sole powers to represent the victims in the civil litigation against Union Carbide. On behalf of the victims the Indian government filed a suit for compensation of more than 3 billion US $ in the Federal Court of the Southern District of New York. However, in May 1986 the case was sent to the Indian courts on grounds of forum non-convenience, under the condition that Union Carbide would submit to their jurisdiction. During the proceedings at the Bhopal District Court, Union Carbide was directed to pay an interim relief sum of Rs.350 crores so that the delay in the adjudication of the case does not adversely affect the claimants. However, Union Carbide refused to pay interim relief and its appeal against this decision reached the Supreme Court. On Feb 14,1989 in a sudden departure from the matter of interim relief , the Supreme Court passed an order approving the settlement that had been reached between the government of India and Union Carbide without the knowledge of the claimants in Bhopal. According to the terms of the settlement, in exchange of payment of US $ 470 million the Corporation was to be absolved of all liabilities, criminal cases against the company and its officials were to be extinguished and the Indian government was to defend the Corporation in the event of future suits. The settlement sum, nearly one-seventh of the damages initially claimed by the government, while being far below international standards is also lower than the standards set by the Indian Railways for railway accidents. There were widespread protests by the Bhopal victims against the betrayal by the government and many organizations and individuals including prominent members of the parliament supported the call to oppose the infamous settlement. Several petitions seeking review of the order on settlement were filed and the Supreme Court announced its revised judgement on October 3,1991. This final judgement upheld the settlement amount paid by Carbide but directed the Indian government to make good any shortfall during the distribution of compensation. Also the criminal cases against the Corporation and its officials were reinstated in the final judgement. The Supreme Court also directed Union Carbide to finance a 500-bed hospital for the medical care of the victims.

Compensation:

The amount paid as compensation (Rs. 715 crores) has multiplied as a result of the increase in the value of the dollar and the accruing interest. Out of this amount, about Rs. 850 crores have been paid to nearly 3.2 lakh claimants and a balance of about Rs. 1100 crores remains to be disbursed. The procedures for compensation disbursement have been tortuous and thoroughly unjust. More than 90% of the claimants have been paid a sum less than Rs. 25,000 as compensation for personal injuries out of which nearly Rs. 10,000 have been routinely deducted against interim monetary relief paid by the government from 1990. The remaining money does not half cover the medical expenses borne by the

claimants in the last several years let alone provide for future expenses. Out of the 15,168 death claims adjudicated 65% have been rejected or converted into personal injury cases where compensation sums are lower. Judges at the claim courts are completely ignorant of the medical consequences of the toxic exposure and the administration of compensation is riddled with corruption so that claimants inability to pay bribes often results in denial of compensation. In response to an official announcement for fresh registration of claims in December 1996 over 4 lakhs claims have been filed. Majority of these claims have been filed by persons residing outside the gas affected area including elite neighbourhoods and it is most likely that genuine victims will be the ultimate loser in the disbursement of compensation.

Criminal Case

A First Information Report for causing death by negligence and a number of other serious offences was registered on December 3,1984 at the local police station. On December 1,1987 the government's prosecution agency the Central Bureau of Investigation (CBI) pressed charges in the Bhopal District Court against UCC and its Asian and Indian subsidiaries namely Union Carbide Eastern (UCE), Hong Kong and Union Carbide India Limited (UCIL) respectively as well as nine officials including the then Chairman, Warren Anderson. The twelve accused were charged under sections 304 (Part IF), 326, 324 and 429 of the Indian Penal Code, with culpable homicide, causing grievous hurt, causing death of and poisoning animals and other serious offences punishable by imprisonment upto ten years and fines. The Corporation blamed a fictitious saboteur and later a disgruntled worker for causing the disaster and organized public relations campaign to distance itself from criminal liability. The CBI with the cooperation of the workers in the factory presented a strong case linking key managerial decisions to the disaster. As the proceedings in the Bhopal District Court began, Union Carbide and its officials chose to ignore the Court's summons claiming that Indian courts had no jurisdiction over them. Finally Anderson was served summons through the Interpol and on his repeated refusal to obey them, the Chief Judicial Magistrate (CJM),Bhopal proclaimed him an absconder. After the criminal immunity granted under the settlement was revoked by the October 1991 final judgement of the Supreme Court a non-bailable arrest warrant was issued against Anderson and the shares of Union Carbide in its Indian subsidiary were attached by the CJM, Bhopal. Five years have passed since the issuance of arrest warrants against the accused Corporation and its officials, yet the Indian government has not taken any steps towards seeking the extradition of the foreign accused. Union Carbide deregistered UCE, Hong Kong in 1992 and now operates in Asia through Union Carbide Asia Ltd. and Union Carbide Asia Pacific Inc.(UCAP) both wholly owned subsidiaries of the parent US based Corporation. Ramasami Natarajan the former CEO of UCE is now the President of UCAP, Hong Kong. The CBI has expressed , in Court, its inability to proceed against UCE as it has deregistered itself. On Sep. 13, 1996, in response to an appeal moved by Keshub Mahindra and other accused officials of Union Carbide India Ltd. (UCIL), the Supreme Court passed an order diluting the charges of culpable homicide to death caused by negligence (Sec. 304A of the I P C), thereby reducing the maximum sentence from 10 years to 2 years. Trial of the Indian accused are currently going on before the CJM, Bhopal, and only five of the over two hundred witnesses for the prosecution have testified in the last four

months. Meanwhile, the managers of Eveready Industries India Limited (new name of Union Carbide India Ltd.) have dismantled most of the Bhopal factory that is supposed to be under the custody of the CBI as evidence in the criminal case. In the absence of any preemptive action by the CBI , survivors organizations sought judicial and executive intervention into the erasure of the memory of the disaster as well as destruction of evidence. However, these attempts were unsuccessful.

Memorial

In a meeting of the state cabinet in end June 1988, the government decided to take back the 60 acre stretch of land in Bhopal on which the Union Carbide pesticide factory had been built. In July, the Madhya Pradesh state government committed itself to constructing a memorial at the site of the Union Carbide factory in Bhopal. The shape or content of this memorial remains to be decided.

Unresolved Issues of the Union Carbide Disaster in Bhopal

Medical :

1. Research and Monitoring

i. Lack of research on continuing gynaecological, neurological, endocrinal, chromosomal and mental health impacts of the disaster.
ii. Lack of administrative set up for carrying out long term, and possibly trans generational research activities in Bhopal.
iii. Absence of monitoring of continuing exposure related mortality and morbidity.

2. Information

i. Lack of information on health impacts available with government and private doctors involved with the medical care of the survivors.
ii. No official initiative towards disseminating information on health impacts and preventive and ameliorative measures to the survivors.

3. Health care

i. Absence of perspective and administrative set up to respond to the chronic nature of exposure related diseases.
ii. Absence of protocols for the proper treatment of exposure induced illness
iii. Absence of a system of recording health status and efficacy of medical interventions.
iv. Absence of a community based health care approach and overwhelming emphasis on hospital based treatment.
v. Negligence of Indigenous systems of medicine.
vi. Unavailability of medicines and facilities for investigations at government hospitals and clinics.
vii. Indiscriminate use of Steroids, Antibiotics, Psychotropic and symptomatic drugs.
viii. Absence of drug- free therapies such as Yoga.

4. Health Education and Public Health improvement

i. Absence of official initiatives towards health education among survivors.
ii. Lack of official initiatives towards provision of clean air, water and sanitation facilities to the survivors.

iii. Absence of criminal case against Union Carbide for contamination of ground water and soil.
iv. Absence of criminal case against Eveready Industries India Limited for dismantling and demolition of the factory.

2.Civil

i. Inadequate amount of compensation
ii. Wrongful rejection of claims
iii. Wrongful denial of registration of claims
iv. Lack of fora for review and redress
v. Utilization of over Rs 1000 crore expected to be left over after disbursal of compensation to all claimants
vi. Continuing legal liability of Union Carbide Corporation, USA and Government of India for medical, economic and social rehabilitation of survivors.
vii. Legal liability of Union Carbide Corporation, USA for contamination of groundwater and soil in the vicinity of its factory.
viii. Ensuring democratic control over the funds currently held by Bhopal Hospital Trust

Economic, Social and Environmental Rehabilitation :

1. Information

i. No identification of individuals, families and communities most in need of economic and social rehabilitation .
ii. No information on production and training skills available locally
iii. No information on locations requiring environmental rehabilitation.
iv. No information on technologies for decontaminating soil and groundwater poisoned by Union Carbide

2. Programmes

i. Absence of long term perspective and administrative structure to carry out long term programmes of economic and social rehabilitation.
ii. Absence of integration of economic and social rehabilitation programmes with programmes of medical care and monitoring.
iii. Lack of innovation and imagination in the design of economic and social rehabilitation programmes.
iv. No scope for participation of survivors in design and implementation of rehabilitation programmes.
v. Absence of an ecological perspective in planning and implementation of environmental rehabilitation programmes.

3. Administration

i. Absence of a well defined coordinating agency
ii. Absence of community based organization
iii. Absence of a mechanism for feed back and review
iv. Gross mis- utilization and mis- appropriation of funds Legal :

Legal Issues

1. Criminal

i. Revision of charges against accused Union Carbide India Limited and its officials including Keshub Mahindra and others pending since September 1996
ii. Slow pace of trial of Indian accused

Terms of Reference

Towards developing a comprehensive overview of the current issues faced by those affected by the Bhopal gas disaster, it is envisaged that the fact finding mission will:

1. Analyze the continuing impact of the disaster on different areas of the lives of survivors of the disaster.
2. Identify specific failures, including policy and institutional, in the last fourteen years and their subsequent repercussions on the people of Bhopal.
3. Compile documentation on all the various aspects related to the Bhopal gas disaster.
4. Outline larger policy changes as a result of the disaster.
5. Provide concrete suggestions for effective democratic interventions.

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SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

Parameters of the Fact Finding Mission

In a meeting of the Organizing Committee we identified the following 14 aspects of Surviving Bhopal that we feel that the Fact Finding Mission must address. We have also arrived at broad parameters for each Fact Finding Team which are briefly outlined below. We would appreciate your response to these outlines and any suggestions you have to make, either on expanding the scope of the Fact Finding Teams' study and analysis, or the inclusion of any other aspect that we may have overlooked.

1. Medical Care

- Estimated figures of exposure-related deaths per month, persons chronically ill, persons acutely ill and persons with exposure-related injuries
- The extent and nature of knowledge on treatment and treatment efficacy available with doctors, RMPs etc
- The most commonly prescribed drugs and lines of treatment
- Available facilities for medical care in different systems of medicine: No. of beds, doctors, specialists, equipments, investigation facilities, availability of medicines and attendance per day and utilisation of beds in government, private and other hospital/clinics.
- Systems of registration and medical record keeping
- Issues confronting employees of various hospitals and clinics, especially in gas affected areas
- Possibilities of improvement in health care

2. Medical Research

- Consolidation of studies carried out by government, private and other agencies and a reassessment of their major findings
- Analysis of the design and implementation of government studies
- Analysis of various studies conducted by Central Govt institutions for rehabilitation
- Outline of studies required for long term monitoring of health effects
- Assessment of the health status of gas affected persons, and their continuing exposure to hazards at both, the occupational and domestic levels
- What are the existing public health initiatives, and what is required?

3. Legal Issues

- The current status of legal actions pending before different courts
- The situation with respect to compensation distribution
- An investigation of the systematic institutional corruption forced on the survivors
- The policy and legal fall outs of the disaster
- The possibilities of further legal action
- The status of survivors' access to justice in terms of legal aid and counselling
- Compilation of a list of documents available with the CBI

4. Economic Rehabilitation

- Outline of existing government and non-government rehabilitation programmes
- The estimated figure of persons unemployed due to exposure related ill health
- Profile of the range of occupations in different sections of the gas affected population and the estimated per capita income.
- Outline of the skills available for income generation
- List of the potential markets for goods produced through income generating projects

12. Memorial on the Disaster and its People

- To present the current status of the State government plans to build a memorial
- To highlight future possibilities
- To outline how peoples' participation in constructing and managing the memorial can be ensured

13. Disaster Management

- To assess the disaster management policies and programmes, implemented by the state bodies in the immediate aftermath of the disaster
- To examine whether Union Carbide Corporation and Union Carbide India Limited had any strategies for management of a possible disaster at the Bhopal factory, whether they were implemented and their effectiveness
- Based on the Bhopal experience, what would be the proposed disaster management programme in the event of an industrial tragedy?

14. Media's Response

- To present a critique of national and international media response in the last fifteen years
- Compilation of media articles, reports etc....

SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

Proposed List of Participants

Proposed Members of Fact Finding Mission:

Justice Bhagwati, Dr PM Bhargava, Mahashweta Devi, Prof. Satish Dhawan Sayeda Hamid, Dr L.C Jain, Girish Karnad, Sugatha Kumari, Arundhati Roy

Proposed Coordinators of Fact Finding Teams:

Medical Care:	Dr C.K Jacob , Medical Superintendent, Christian Medical College Hospital, Velore Satinath Sarangi, Sambhavna Trust, Bhopal
Medical Research:	Dr Mohan Rao, School of Community Health and Social Medicine, JNU, New Delhi. Dr Padma Prakash, Economic and Political Weekly, Mumbai.
Law:	S. Muralidhar, Advocate Supreme Court, New Delhi. Sandeep Sharma, Advocate, Bhopal.
Economic Rehabilitation:	David Selvraj, VISTHAR, Bangalore Jabbar Khan, Bhopal Gas Peedit Mahila Udyog Sangathan, Bhopal.
Labour:	Sujata Goteshkar, Workers Solidarity Centre, Mumbai. Ashim Roy, Hind Mazdoor Kisan Panchayat, Ahmedabad.
Social Rehabilitation:	Shiv Vishwanathan, Centre for the Study of Developing Societies, Delhi. Dr Veena Das, Department of Sociology, Delhi University (**)
Environment:	Ravi Aggrewal, Shrishti, New Delhi Ashish Kothari, Kalpavriksh, Pune
Union Carbide:	Ward Morehouse, Council on International and Public Affairs, New York, USA.
Scientific Institutions:	Raghunandan, Delhi Science Forum.
Role of State and Central Governments:	Babu Matthew, National Law School, Bangalore Mohan Mani, Centre for Workers' Management,Bangalore.
NGO's and Peoples' Organisations:	C.R Bijoy, PUCL, Coimbatore Kalpana Kanabiran, Asmita Resource Centre for Women
Memorial:	Sheba Chachi, New Delhi K.T Ravindran, Architect, New Delhi
Disaster Management:	*To be decided*
Media's Response:	Prafu! Bidwai, Columnist, New Delhi.

BHOPAL ?

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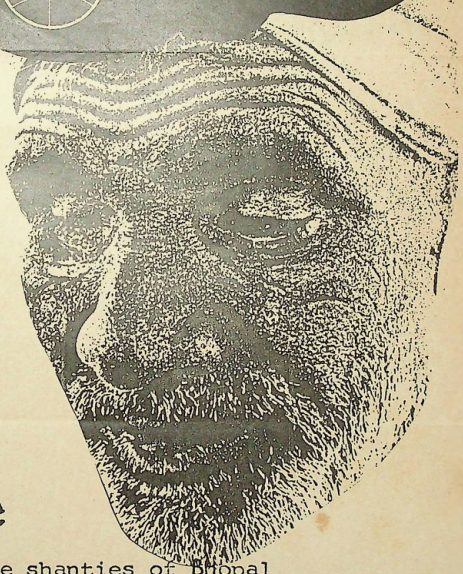


The Deaths Continue

Death still stalks the shanties of Bhopal twenty months after the disaster wreaked by Union Carbide. Poisoned by a killer multinational, neglected by the government, ignored by the media, the gas victims continue to suffer from illnesses that are yet to be properly diagnosed, let alone cured. And they continue to die. Like Moolchand of Patel Nagar, Bhopal.

The Story of Moolchand

Moolchand died at 5.15 p.m. on 14 ~~July~~ June, 1986, on bed No. 17 of the MIC Ward, nine months after admission to the Hamidia Hospital. His tragic tale is the tale of many many more who wait, spent and dispirited, for a slow, troubled death.

Moolchand came to Bhopal in 1950 in the tumultuous aftermath of the partition of the country. A Sindhi migrant from Pakistan, Moolchand had little to survive on when he started life anew at Bhopal. He eked out a living as a scrap merchant, carrying his load of waste around the city on a bicycle. The only one to help him was his son, Vasudev. And they did manage to survive and sustain their small family through the years. Until the night of 2nd December, 1984.

That fateful night, Moolchand awoke with a start to find his room thick with acrid, irritating fumes of gas. He was afraid, and alone, for his family had fled. So he too ran, like thousands of others, to save his life. And he survived. But just. The toxins had ravaged his body so much he was unable to work. His business suffered. He could not breathe properly, his body was wracked by coughing, he lost his appetite, he couldn't see well. And then began his frustrating search for a cure, or even for some relief from suffering.

Each morning he joined the queues at the government clinics with his wife Pramvati, who was similarly affected. He took drugs and drugs and more drugs. But to little avail. There was little respite from his painful condition. In despair he went to private doctors. His savings were all gone. He had to sell off his wife's jewels to pay for the expensive medicines the doctors prescribed. Again, to little effect, because he continued to suffer and his condition deteriorated.

On 2 September 1985, in a last desperate bid to save his life, a pale and emaciated Moolchand went, once again, to the government polyclinic at Bharat Talkies. He shuttled for days between the clinic and Hamidia Hospital. Until, on 9 September 1985, he was admitted to the MIC ward. He still complained of breathlessness, cough with expectoration, muscular weakness in the legs and generalised weakness. Clinical and pathological examinations ruled out tuberculosis but pointed to bilateral crepitation. The diagnosis: "MIC-induced lung diseases"!

His wife, herself suffering, was his constant companion at the hospital. She made the daily four-kilometre trip from their home in Patel Nagar to the hospital with their son. Moolchand was prescribed pain-killers, broncho-dilators, steroids.. ... "He used to take almost half a kilogram of medicines everyday", reminisces another patient in the ward.

Then one day Moolchand slipped and fell in the bathroom. (This is not uncommon in government hospitals where reeking toilets and bathrooms are covered with the slippery slime of dirt and excreta). He fractured his thigh bone. The fracture was improperly set. As a result, one leg was an inch shorter than the other when the cast was removed. Already weak, Moolchand was now further immobilised , confined to his hospital bed. He lay gasping for six months, his family driven to the brink of deprivation. Till he finally lost the will to live. As a final act of resignation and despair, he stopped eating just prior to his death. He died in the evening of 14 ~~July~~ June, 1986.

Many, like Moolchand, continue to die. In the absence of a meaningful and adequate system for monitoring the conditions of victims, many of these deaths go unreported. Says Professor Heeresh Chandra, Director of the Medico-legal Institute, under whose charge the autopsy of Moolchand's body was carried out, "Many of the gas victims are dying, yet very few dead bodies are brought in for autopsy." Despite many suggestions, the government has not made autopsy compulsory. Such an omission attains crucial significance in the context of the opinion expressed by Michael Ciresi, the lawyer retained by the Government of India, that the nexus between exposure to gas and subsequent deaths has to be established beyond reasonable doubt if damages for death are to be claimed from Union Carbide Corporation.

The complete absence of such initiative was blatantly made clear in an interview with the Director of Claims, Mr. Khare. We asked how Moolchand's family could file a claim for compensation. Sitting in his office tucked away in a maze of government buildings, seven kilometres away from Patel Nagar, Mr. Khare confessed, "We do not make any attempts to inform people that they can still file claims." When pressed, he tried to justify this glaring omission by saying - "Somehow some people do come!"

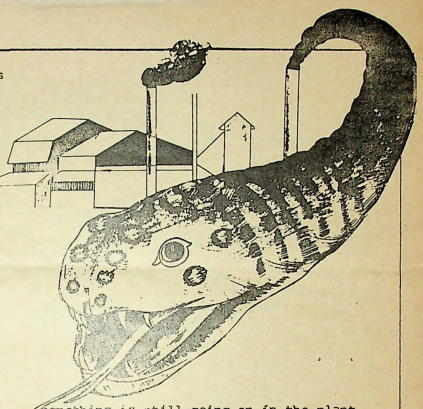
Such wilful neglect condemns the gas victims to prolonged suffering and death, even as the death dealing multinational goes unscathed.

TOXIC EMISSIONS CONTINUE

Shortly before noon on 19th June, 1986, clouds of black smoke were visible at the UC plant in Bhopal. Investigations by BGIA revealed a raging fire next to the administrative building inside the plant. On enquiry, security guards said that plastic cans containing toxic chemicals were being destroyed. The fire continued for almost an hour.

This is not the first time since the December, 1984, gas leak that toxic materials have been disposed off in this manner by Union Carbide without informing the public. In February last year, toxic fumes from the incinerator inside the plant entered a double storied school building near the eastern wall of the plant while the school was in session. About 50 children, all below twelve years, started coughing violently and some started vomiting. A number of children complained of giddiness and nausea. The teacher, not knowing how to respond to such a situation, declared the school closed for the day and the children were asked to go home. The matter was taken up with the authorities but no action was forthcoming. The emissions from the incinerator continued unabated. It was only after a group of young people from Shaktinagar, where the school is located, went up to the gate and hurled stones that the authorities got the message and the emissions stopped.

Incidents such as these aggravate the panic-bound psyche of the gas victims, besides causing injury to their already damaged bodies. Many of the victims continue to talk about the poison clouds of the December leak and visions of that catastrophe recur in their dreams. Mention is often made, sometimes in hushed tones and sometimes in anger, that



Something is still going on in the plant. But who is bothered about the gas victims anyway?

WITHOUT COMMENT

Last month, the UCO bank in Bhopal was robbed in broad daylight by a band of yet unidentified persons. They are reported to have introduced themselves as "Hum log gas peedit hain, bank lootne aye hein" (We are gas victims, we have come to loot the bank). Reading the report, an old gas victim commented, "Gas peediton ke naam se kitnon ne loot liya, yeh ek aur sahi" (So many have looted in the name of gas victims, this is just one more instance).

STREET VS STAGE

Rangmandal, the theatre group of Bharat Bhavan, the elite cultural centre of Bhopal staged a play early this month based upon the Bhopal gas disaster. 'Banjh Ghati' (Barren Valley), staged twenty months after the disaster, was, however, eclipsed by the forceful 'Dastan-e-Gas Khand' (Tale of the gas disaster) enacted at the same time on the streets opposite the Bhavan by 'Shuruat' a group of amateur artistes. This group has been performing the play since early last year in many parts of Bhopal and elsewhere, including the gas affected slums. Among the crowd which preferred to watch the street theatre were many young artistes of Bharat Bhavan itself.

SURAKSHA

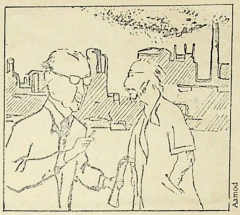
सुरक्षा

A new project, involving the gas affected children in painting, music, story telling, poetry and drama, has started in the Jayaprakash Nagar Basti of Bhopal. It has been organised by David Bergman, a young Britisher who has been in Bhopal for the past three months. (David and a friend undertook a cycle tour from England to Bhopal to raise funds for the gas victims of the Bhopal disaster.) The group which has been set up for the purpose is called SURAKSHA.

At present, five teams of local artists (of 2/3 individuals per team), each conduct a workshop in the basti once a week. A large covered area is used for these activities. The project is now in its third week, and 30-50 children are regularly involved.

Suraksha feels that although there is an obvious need for such opportunities to be given to basti children everywhere, there is a special need for them in Bhopal today. Apart from the physical disabilities the children are still suffering from, many of them have also gone through extreme mental anguish and stress during and after the gas disaster. Emphasis is, therefore, being placed in these workshops on psychological therapy.

For the next few months, Suraksha plans to limit itself to working in this basti with these five teams of artists. Indeed, much needs to be done just to make these activities with the children successful. Relationships of friendship and trust need to be built up between the artists and the permanent workers of Suraksha, on the one hand, and between Suraksha and the children, on the other. The children must be involved in all aspects of the project. Attempts must also be made to get the general acceptance of the project within the basti community. Only then can they become involved in the general organisation of Suraksha and in the supervising of the activities. Finally, much discussion needs to be done with the artists themselves to work out coherent, structured activities which have an overall aim behind them. Such activities can then continue over a number of weeks and months, with new teams of artists joining in at different points.



"The gas leak is okay. What we've got to guard against is the leaks to the press."

We have

the right to know

From September/October onwards, it is hoped that Suraksha will be working in two or three independent "activity huts" structures of timber and bamboo - around JP Nagar and Shakti Nagar. Apart from the core of activities being carried out by local artists and others from the basti, individuals and groups working with children from other parts of the country are also being invited to come to Bhopal to work intensively for two or three weeks. Such groups could exchange ideas with local teams so that their work and methods could be continued in their absence.

Once a good understanding and friendship has been built up with the children, Suraksha is interested in organising a nutritional study of the children. Such a survey is especially important now because of the large amounts of drugs that their bodies are being subjected to.

The success of all this is very much dependant on two to three more permanent workers committing themselves for at least three months to this project. So the group requests interested people to come and work with them. The group also invites ideas and suggestions from interested people. If interested, write to:

SURAKSHA
A-73 SANT KANWAR RAM NAGAR
BHOPAL 462 018.

A writ petition on the issue of the right to know has been filed recently in the Supreme Court of India by the environmental action group, Kalpavriksh. This petition is an intervention in the ongoing case against the Sriram Foods and Fertilizers Company which was responsible for the Oleum gas leak in Delhi, on the first anniversary of Bhopal, which resulted in the death of one person and injuries to many others.

Kalpavriksh had earlier intervened in the case, pleading that the plant be shut down and relocated. As readers might recall, the Supreme Court had then ruled that the plant could reopen subject to its management meeting certain conditions, prominent among which is the one which holds the top management personally responsible for paying compensation in the event of future accidents.

The right to know petition argues that citizens have the right to be informed about the nature of the dangers posed to them by hazardous industries. The petition is due for hearing later this year.

Support the call by KSSP for a **Global boycott** of all products of Union Carbide by Environment and Consumer Groups with immediate effect.

For more information and details on campaign, please write to:

KSSP – Kerala Sastra Sahitya Parishad,
Parishad Bahvan,
Trivandrum 695037
Kerala, South India.

AIRPLAN

In May, 1985, representatives from environment groups from across the world, including India, met at Eerbeek, a village in Holland. The subjects of concern were the growing menace of AIR POLLUTION, particularly Acid Rain and the issues raised by Bhopal.

Out of this meeting grew a new global network linking environmental groups concerned about these issues. AIRPLAN, as this network is called, coordinates international campaigns and is a forum for exchange of information and action plans.

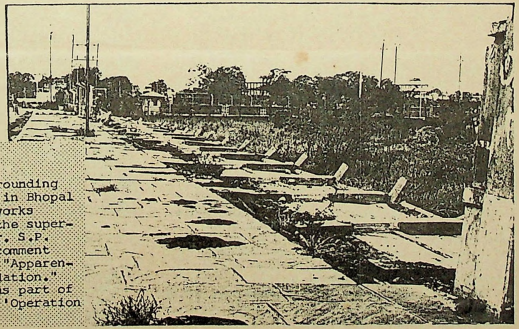
AIRPLAN has been around for a very short while but has already demonstrated the potential of such a network. Recently, an appeal sent out by it resulted in international pressure being applied on the government of an East European country to free two environmentalists who had been jailed for raising ecological issues. In order to maintain a regular channel for communication and information exchange, AIRPLAN publishes a bimonthly newsletter, two issues of which have already come out.

AIRPLAN welcomes interested groups to join and strengthen its network.

Their address:

PO BOX 5627
1007 AP Amsterdam
The Netherlands.

AIR POLLUTION ACTION NETWORK



FAITH WITHOUT FOUNDATION

A portion of the wall surrounding the Union Carbide factory in Bhopal collapsed recently. The works manager, Mr. Mukund, and the supervisor of the MIC plant, Mr. S.P. Choudhary, were heard to comment during their spot visit – "Apparently, the wall has no foundation." Incidentally, this wall was part of safety system No.5 during 'Operation Faith'.

Quacks have a field day

Every gas affected person in Bhopal must have visited a private medical practitioner at some point in time over the past year and a half. Every private medical practitioner must have treated anywhere between 2,000 and 10,000 gas affected patients during this period. Indeed, this group of "businessmen" could not have had things better - the boom in their trade stands in stark contrast to the gloom and depression pervading every other economic activity in the area.

In normal times, the present period of the year is referred to as the "slack season" by these private practitioners. But these are not normal times - the stream of patients does not end, the 'shops' stay open till late in the night. Clearly, the potential of these private doctors to do good or cause harm to their patients is phenomenal. The question is - how safe are the lives of the thousands of gas affected people in the hands of these 200 odd self-professed medicos?

The first thing one notices while visiting a private doctor in the gas affected areas of Bhopal is his sign board. Often oversized, it proclaims the doctor to be a specialist in a variety of areas of medical knowledge. One sign board, for instance, advertises its owner as a specialist in medicine, surgery, gynaecology, paediatrics and sex problems. Some sign boards mention the degrees the "doctor" has acquired. These are usually a curious mixture of unevenly spaced letters amenable to a variety of interpretation - U.V.P., V.V.P., M.I.A.M.S., M.A.M.S. etc.

On a rough estimate, eight out of every ten private doctors have received no formal training in medicine. Their medical qualifications usually comprise an apprenticeship with another private doctor, coupled with lessons received from correspondence courses offered by ubiquitous "institutes" characterised by their anonymity and dubious reputations. Many doctors take such correspondence courses simultaneously in more than one system of medicine. Thus, it is not rare to meet a private doctor who claims to treat his patients by the allopathic, homoeopathic, ayurvedic and Unani systems. As one doctor interviewed put it, "We try different systems and see which one fits - my patient must find relief." The treatment is, by and large, oriented to provide instant relief. "People are workers here", one doctor explained, "they have to be fit for work, so they want quick relief lest they lose their wages."

Talking to some of these private doctors, one picks up very interesting bits of information. One learns, for instance, that Sodium Thiosulphate is a drug from the U.S. and is not of much use in India because the blood circulation, the pulse rate and breathing rates of Indians are different from those of people in the U.S. One also learns that the outermost layer of the eye, contrary to what class nine biology students are taught in schools, is called the Retina. One also encounters a host of very interesting theories which attempt to explain the effect of the toxins in the body. One such theory is that the gas has produced "heat in the body" which cannot be tackled by ordinary medicines, since the gas has interfered with the formation of pancreatic juices and saliva, causing problems of digestion.

Private doctors have several pertinent observations to make about the total health situation in the bastis. They are in a position to do so since many have been in the area for almost twenty years. We list some of these observations:

- While the number of patients is still considerable, there has been a 50 per cent fall in the past year. One reason is that many gas victims have resigned to their suffering.
- Patients who visited their clinics only once a month before the gas disaster now do so once or even twice every week.
- A large number of patients have been suffering without respite ever since the gas leak and despite the treatment received from the private medicos and government clinics.
- More than half the patients who come to their clinics have symptoms directly related to gas exposure.
- Diseases which were prevalent earlier now occur more frequently and with intensity.
- The variety and nature of symptoms is baffling. They include breathlessness, gastric disorders, loss of appetite, fatigue, weakness, body aches, dizziness, abdominal pain, headache, menstrual irregularities, impotency, loss of vision, burning sensation in the eyes, pain during intercourse, anxiety, insomnia, headache and chest pain.

According to another theory, "MIC affected patients need hard drugs, potent drugs, drugs which are costly. Only then can they get better."

These beliefs, coupled with the hard-sell tactics of big drug companies (smooth talking medical representatives and stylishly printed literature which invariably adorns the walls of the clinics) have resulted in utter irrational and dangerous "therapy". Antibiotics are prescribed at the first signs of cough, fever or boils. Rarely are patients prescribed the full course; where they are, patients discontinue use half way through the course, rendering themselves more vulnerable to further infections. Injections, the ultimate panacea in the eyes of many patients, are readily given by the doctors to patients with money to spend on these "miracle" drugs. Another favourite is steroids (which provide a "sense of well-being" though they impair the natural immunity system of the body). Tonics, which, according to one doctor, are prescribed "to give energy to the gas victims", formulations such as "Protinules", "Protinex", "Charkaferrol" and "Ferradol" and useless drugs such as Kaolin-Pectin mixture for children with diarrhoea are also routinely prescribed. Moreover, drugs such as Phenylbutazone and Oxyphenabutazone preparations (e.g. Actigesic, Bestophen, Suganril and Reducin) which have been categorised by the drug controller of India, as restricted for use in the treatment of ankylosing spondylitis and gouty arthritis (and whose potent toxic after effects have been demonstrated by the fact that Ciba Geigy the principal manufacturers of these drugs is reported to have recorded 1674 deaths due to these drugs) are routinely given for body aches.

The private medical practitioners charge between Rs. 5/- and Rs. 25/- per patient per visit. An average household in the gas affected area would have spent at least Rs. 2,000/- in the last eighteen months on medicines and consultation fees of these private doctors. There are many who have borrowed money for this purpose at rates of interest sometimes as high as 120%.* The government run hospitals, on the contrary, charge nothing. The obvious question is - why do the victims prefer these private doctors to the hospitals run by the government. There are several reasons,...

*(These figures give lie to the ill-conceived notions of most government doctors who maintain that the gas victims are pretending to be sick as they want compensation).

The government run hospitals and clinics are open only during the day and in the early evening, times when people are out working, while private clinics are open till late in the night.

The attitudes of the doctors towards their patients is an impressive blend of smart salesmanship and a veneer of professionalism cultivated over several years of experience and practice. In sharp contrast, the government doctors who are too pressed for time do not spend enough time or give personal attention to their patients. The private medicos give a patient hearing to each person and spend considerable time in clinical examinations. Indeed, the private doctors score over even the ICMR researchers and senior government doctors when asked to provide an oral list of symptoms presently displayed by the gas affected people.

The shortage of government doctors means long queues at the clinics. There are several other inadequacies in the governmental medicare system. As one private doctor stated very candidly - "The government doctors prescribe the same medicines as we do, they have much greater facilities, if they happen to improve their services, we will be out of business."

That doctor, like most others operating in the gas affected bastis of Bhopal, does not lose sleep over this possibility, fully aware that such a change in the governmental attitude is extremely improbable.

AN APPEAL

In the first issue of 'BHOPAL' which we mailed to you a month ago, we laid out the objectives of the Bhopal Group for Information and Action. We have received a positive response to that issue and to the idea of BGIA. Our thanks to all those who've responded.

In the last month, we've begun the task of collecting material on Bhopal and setting up the documentation centre. We've also found a place near the Union Carbide factory to house the centre.

As we mentioned last time, we see our small group here as a mere extension of a shared desire of a vast community to sustain work on Bhopal and related issues. Moreover, the task of documentation, bringing out the newsletter and running and information service can be sustained only with your active support.

At the moment, BGIA requires the following assistance from you and we trust you will try your best to help:

- We need a list of all documents concerning Bhopal which you may possess or have access to. This list is vital for the building up of the documentation centre.

- We need some equipment and financial assistance. Equipment includes a typewriter, storage cabinets, stationery and paper.

- We need contributions to keep the newsletter going. We urge you to
  - contribute a minimum of Rs. 24/- a year in advance (Rs 30/- for institutions and $ 30/- for foreign groups); and
  - undertake to sell a minimum of five copies of the newsletter on a regular basis at Rs. 2/- a copy.

More than anything else, we value your comments, criticisms and advice.

'BHOPAL' is brought out by the Bhopal Group for Information and Action.
D-42 FIRDOS NAGAR
BHOPAL 462 018

This issue was produced by Satinath Sarangi, S. Ravi Rajan and Vinod Raina, with assistance from N R Mohandas.

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BHOPAL

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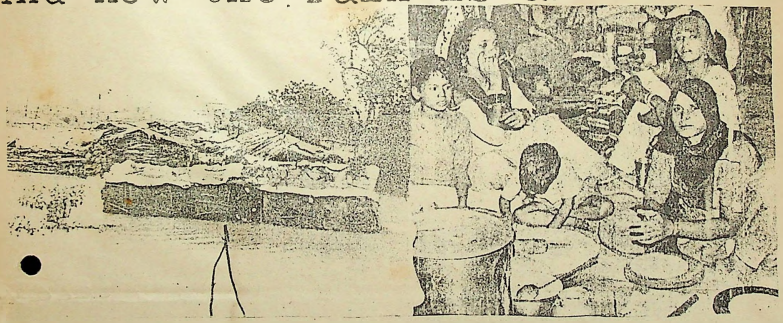
And now the rain is after them



A flooded basti in one of the gas-affected areas (left); People crowd into the only shelter available - the front yard of a vegetable storage depot (Right) courtesy "Nai Dunia" and "Free Press Journal"

The nightmare continues. First the gas, then an indifferent response from a corrupt administration and now the worst rains in this century. Will Bhopal's hibakushas ever live in peace?

THE INSECURE SECURITY STAFF OF CARBIDE

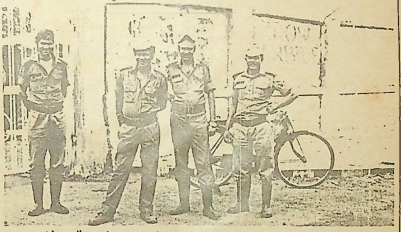
On the night of the disaster (December 2/3 1984), KR Deshmukh and YN Singh, security guards, were on duty inside the UCIL Bhopal plant. This is how they narrate their experience.

"Both of us were on night duty from 10 p.m. to 6 a.m. At 12.30 a.m., during the tea break, we were in the canteen when gas started leaking. Some of the people from the MIC Plant were also at the canteen. Soon the gas started coming out more violently and the workers tried to control it by spraying water but when it got out of control they ran for their lives. The nine security guards on duty, three of whom were quite badly exposed by now, were asked by Mr. Chouhan, the Asstt. Security Officer, to stay on and do their duty. We were never told about the toxic materials inside the plant nor were we given any safety appliances like masks. So, not knowing what to do, we stayed on in a state of helplessness and panic. Around 3.30 a.m., people from Jai Prakash Nagar started coming inside the plant - they were coughing, choking, blinded, stumbling, groping and all crying out for help. We took them to the plant dispensary, sprinkled water on their faces and gave them water to drink. Major Mehra, our Security Officer, told us to mind our posts and said that one person was enough to attend to the people who were streaming in. We refused to go by his orders and continued helping the people who were now coming in large numbers and in more acute conditions. Some were dying inside the plant. By 8 O'clock in the morning more than a thousand people must have come inside the plant. We were all very worried about the welfare of our family members but to our relief we found when we rushed home in the morning that none of our family members was dead. They were suffering quite badly and so were we. We were getting breathless and our eyes were swollen and for the next two days we just could not report for our duties at the plant."

This account of the disaster and its immediate aftermath was given to us by the two secuiity guards in the course of a detailed interview we had with them. Further discussions with them as well as other security guards brought out the insecure state of the security guards of the UCIL Bhopal plant.

Deshmukh (33) and Singh (32) are two of the forty-three security guards employed by the Star Security and Emergency Services (SSES) and posted at the UCIL Bhopal Plant. The SSES, owned by Mr. RN Nagu, an ex-Inspector General of Police, is an agency that is contracted by several industries in Madhya Pradesh and is managed from the office in Mr. Nagu's posh house atop Shyamla Hills beside the lake.

While the plant was in operation, the security guards had to work under hazardous conditions. "Since most of us have had our posts near the plants we have inhaled a lot of those poisons which used to emit from



them", they said, "and pain in the chest was an ailment common to us. Apart from small emissions, massive leaks were also not uncommon SIX MONTHS PRIOR TO THE DISASTER", said Deshmukh, "While I was on night duty, gas had leaked around midnight. There was almost no wind so very little spread outside the plant but the whole plant was engulfed in it. Workers fled from the plant and people from Jai Prakash Nagar came enquiring about the reasons for the irritating 'mirchi ki dhans' (smell of burnt chillies) they were getting. Conditions became better after two hours and workers returned at 4 O'clock." While all this was happening, Deshmukh and his colleagues PL Gopalan and MC Tiwari, not knowing how to respond, hid themselves in the bathroom behind the security office. According to the security guards, even today hazardous practices are followed. Last month, according to the orders of the management, daily wage labourers were employed to burn waste napthal lying in the plant.

This went on from 8.30 a.m. to 5 p.m. for about 10 days till some passengers of a train passing near the plant who were exposed to the nauseating fumes reported to Hanumanganj police station about it. Sulphonic acid tanks are kept open leading to the formation of acrid fumes that affected the people of Jai Prakash Nagar.

While such hazardous practices were being carried out almost routinely in this plant, the security guards were not informed about the barest essentials of safety practices. "Only after the gas disaster, the Carbide management told us that we should have put wet clothes over our faces." They did not have access to any safety equipment like gas masks and goggles and in the event of disaster were left to find their own means of survival.

With such hazardous conditions of work for the security guards, attention was never paid towards their medical care either by the Carbide management or by Mr. Nagu. Even basic human considerations were absent in their attitudes towards the workers. The salaries of the security workers, who could not report for duty because they were affected by the toxic gases on the night of the disaster, were deducted for the number of days they were absent. The three security guards who were severely affected while on duty lost 15 days' salary. Even now Deshmukh remains absent for 8-10 days in a month because of the ailments he suffers due to exposure to the toxic gases and loses his salary.

The security workers are paid low salaries and the rise in their pay has not been commensurate with the rise in prices. "Since 1975 prices of things have gone up 5 to 6 times, our salaries have gone up by 2 times only", says Deshmukh who gets Rs. 600/- (less when there are deductions) and has to pay Rs. 150/- as rent for his one-room family accommodation. "And Nagu Sahab says", adds Singh, "if you can't support your family with the salary we give you, you are free to leave". Since 1975, about 60 of the workers have left their jobs, some who could not support their families left quietly while others, who were bold enough to ask for a raise of salaries, were dismissed. Dismissals have almost always been without notice and on the basis of trumped-up charges. Prior to the gas disaster, SR Sakalya and Shiv Dhyan Singh, who were identified as "leaders" by the Carbide management, were dismissed on concocted charges of "sleeping during duty hours". After the gas disaster, three security guards who were quite vocal, MP Pathak, Padam Singh and Govind Kurosia have been dismissed on charges of theft. Both Deshmukh and Singh were quite sure that these were just the manageeent's way of suppressing dissent.

Since the gas disaster, the condition of the security workers has become much more precarious. They have had to spend large sums of money on the medical care of their family members as well as themselves.

The majority of them have not received the ex-gratia relief of Rs. 1,500/- distributed by the MP Government. Absenteeism due to poor health conditions lead to cuts in the salary and requests for a sympathetic consideration from both the officials of SSFS as well as from the Carbide management met with callousness. While employees of the plant, and contract employees in the plant canteen have received compensation, the security workers have not received any. They are unsure how long they will continue to be employed - as Mr. Nagu has pointed out to them, their future was linked to the future of the UCIL plant, and the UCIL management has 'informed' them that they are not responsible for 'contract workers'. Almost all of them come from places distant from Bhopal like Kerala, Bihar, Uttar Pradesh and some districts of Madhya Pradesh. They have no land to go back to, , so the options before them are limited. "We are never sure - it is possible that tomorrow we get a letter saying our services are not required. Where can we go? We have spent the best years of our life and our bodies have taken in all these poisons - who will give us jobs now?" - asked some of the workers.

In their desperation the security workers have tried several means to have their grievances heard but their experience so far has been rather disappointing. Initially they had joined hands with the Union Carbide Karmachari Sangh (UCKS) (the recognised trade union of the UCIL Bhopal employees) and contributed to their funds, but in the matter of obtaining compensation from the company and getting alternative jobs from the government, the UCKS leadership did not take into account the problems of the security workers. They have met the Chief Minister and submitted their grievances at least ten times, only to be given empty assurances. For the last two months, they have been members of the AITUC (the workers' trade union of CPI) whose leadership has made promises to them of obtaining permanent government jobs. While the promises are yet to be fulfilled some of the workers are considering militant action to press their demands.

With their low number, poor economic conditions and faced with a repressive management, the security workers at UCIL, Bhopal, have a limited possibility of overcoming their present state of continued insecurity.

Carbide's Fraud

Why is Union Carbide spending $2 million on a rehabilitation project for twelve people ?

The Bhopal Technical and Vocational Training Centre stands by the Upper Lake in a remote part of the town, Lal Ghati, amidst a mango orchard, surrounded by rich farmland. With a large and spacious mansion as its nucleus - an old country home now converted to its present use, the Centre comprises four acres of most beautiful aspect with a view of the lake that only the richest homes in Bhopal can command. Workers and staff dart about the place looking efficient, in the process of establishing a yet larger and more efficient Technical and Vocational Training Centre.

The Director of the Centre says she would like the Centre to be like Tagore's Shantiniketan - a laudable sentiment. But the more one finds out about the Bhopal Technical and Vocational Training Centre, the more intriguing its operation and activities - and indeed, its very existence, become. For the financier of this enterprise is none other than the Union Carbide Corporation.

Despite its name, the Centre offers neither technical nor vocational training at present. Everyday, twelve students are brought in, six blind and six deaf, to attend classes in Braille and in sign language respectively. Their instructors have been brought in from the Arizona State University (ASU) which operates the Centre; every two months new instructors from ASU fly in as replacement (and stay at the exclusive Jehan Numa Palace Hotel). Training in welding and sewing will commence at some point for a similar number of people. The 'latest equipment' and expertise are being imported, and everything used at the Centre, down to the wall charts, is from the US. Experts and officials from ASU fly in and out constantly; there can be few groups of twelve students who are pampered quite as much.

"The technology and know-how for training in these areas (i.e. Braille, sign language, welding) is inadequate in India", said an ASU professor, a liaison officer in the project. "This is a giant leap for Bhopal into the 20th century", he proclaimed. When asked to explain the strikingly modest number of participants, he became defensive - "This is only a prototype", he said, "it represents a beginning."

Admitting that Carbide was financing the $ 2 million project, he said that Carbide and ASU both were anxious to avoid publicity. One instructor from ASU candidly told us - "We have been asked to keep a low profile." That Union Carbide, hardly a spendthrift on public welfare, should suddenly be coy and furtive about its philanthropy is strange enough. But more intriguing still is the fact that hardly any of the twelve trainees are gas victims. If Carbide was funding this, surely they would admit only those injured in the gas leak "But how can you tell who is a gas victim?" responded the Director, who narrowly missed Congress(I) nomination to the Rajya Sabha this year - "everybody is claiming to be a gas victim."

The mystery was cleared up when highly placed sources informed us that Union Carbide was reportedly trumpeting its munificence to the Bhopal public in corporate journals in the US. If brought under public scrutiny here, the farcical nature of its relief scheme would become apparent. The story of a $ 2 million charitable project would presumably do much to appease corporate liberals in the US, and would improve Carbide's reputation there - "there", of course, being the only place it matters.

What is disturbing is the high-level cooperation this project has obtained from the Central Government. "All their papers, including their foreign exchange remittance permits, were in order - we couldn't touch them", said one state administrative officer.

It is imperative that Union Carbide is not permitted to make capital of this masquerade.

LEGAL UPDATE

In the din following the judgement of John Keenan returning the case to India, many of the several manoeuvers in the US courts since then have escaped public notice. Following, in chronological order, are some of the more important events.

May 12, 1986: Judge John F. Keenan of the Federal District Court for the Southern District of New York finally ruled on the forum non-conveniens motion made by Union Carbide Corporation in July 1985. Keenan's decision was to return the case to India, with three important conditions:

"1. Union Carbide shall consent to submit to the jurisdiction of the courts of India, and shall continue to waive defenses based upon the statute of limitations;

"2. Union Carbide shall agree to satisfy any judgment rendered by an Indian court, and if applicable, upheld by an appellate court in that country, where such judgment and affirmance comport with the minimal requirements of due process;

"3. Union Carbide shall be subject to discovery under the model of the United States Federal Rules of Civil Procedure after appropriate demand by plaintiffs."

May 20, 1986: Hearing in Chambers. Keenan entertained oral arguments and notions relating to his May 12th forum ruling.

Attempts at reaching on out-of-court settlement did not end with the rejection in March by the Indian government of a $350 million settlement offer. In fact, with the Keenan decision on forum, it became even more important for the individual plaintiff attorneys to attempt such a settlement, as they stand to lose both their clients and their contingency fees if the case moves to India. On May 20th Stanley Chesley and F. Lee Bailey (individual members of the Plaintiffs' Executive Committee) made oral arguments requesting that the Court "conduct a hearing to determine whether the settlement proposed for these plaintiffs is fair and reasonable."

At this hearing, Carbide's attorney, Bud Holman, raised three issues. He requested that the period for Carbide to respond to the conditions in Keenan's May 12th Order be extended to as many as 60 days, only after which the appeal period would begin. Holman also asked that two changes be made in the conditions; that the words "against it" be inserted in the second condition so that it would read "Union Carbide shall agree to satisfy any judgement rendered against it by an Indian court..." to insure that it refers to Union Carbide and not to Union Carbide India Limited.

Holman in addition requested Keenan to amend the third condition which requires Carbide to "be subject to discovery under the model of the United States Federal Rules of Civil Procedure..." Holman argued that Keenan has authority to demand from the Indian Government that it submit to similar rules of discovery--an argument that Keenan had already addressed in his May 12th ruling in these words: "While the Court feels that it would be fair to bind the plaintiffs to American discovery rules, too, it has no authority to do so." (emphasis added).

May 21, 1986: The individual plaintiff attorneys entered a Motion for a Fairness Hearing requesting in written form that the court consider the following questions in determining whether the settlement proposed is "fair and reasonable:"

"A. To what extent have the victims been informed as to what amounts of money they would receive individually if the present offer were accepted?

"B. Do these victims wish to renounce the retainers they have signed with their American counsel, or do they wish American counsel to continue to negotiate on their behalf even if the case is transferred to India?

Yet Another Survey

A state cabinet sub-committee of the MP government decided recently to conduct a medical check-up for each of the five lakh odd people who have filed compensation claims. According to "The Hindustan Times" which reported this on July 26, the chief minister has said that such a medical record (which would include X-rays and blood and other tests) would be necessary for determning the amount of compensation to be paid to each gas victim.

The proposal, however, is shrouded with controversy. The Claims and Compensation Secretariat is reportedly insisting that the previous surveys be studied in order to avoid the mistakes that were committed while conciewing and conducting them. The Chief Medical Officer feels that such an exercise would be a waste of time.

The survey form is believed to be over eleven pages long, of which nine are to be filled inby the examining doctor and the rest by a CCS surveyer. Given the government's track record in implementing projects and the logistical enormity of the present enterprise, the survey might be the major preoccupation of government departments for a long time.

Meanwhile, one wonders what happened to the previous surveys, including the much talked about ICMR epidemiological survey. If they are as bad as to necessitate another major survey, what is the guarantee that the present one, more ambitious than its predecessors, would be successful?

Carbide to shift Bombay unit

BOMBAY, Aug 4 (UNI): Giving in to mounting public pressure after the Bhopal disaster, the Union Carbide company has expressed willingness to shift its chemical plant elsewhere from Chembur in north-east Bombay.

MEETING AT WARDHA

A meeting was held at Sevagram, Wardha, from July 27 to July 29, to consider the need and feasibility of carrying out a comprehensive epidemiological (nature and extent of disease) survey of the gas affected areas of Bhopal.

The participants included members from Drug Action Forum (West Bengal), Lawyers Collective (Bombay), Medico Friend Circle, Bhopal Group for Information and Action and many individuals.

A tentative evaluation of existing surveys suggested that there was need for a more thorough study of the gas-affected population to provide a basis for compensation, medical relief and rehabilitation. It was decided that, after a rigorous critique of all previous surveys and a review of existing literature on MIC and its break-down products, a study design should be prepared in consultation with all known experts in the field. The Government of India would then be urged to carry out such a survey with the participation of non-governmental organisations. To demonstrate the feasibility of the design, a pilot study would be conducted in one of the gas affected areas.

The meeting also arrived at the consensus that a National Convention of activists, environmentalists and scientists should be held to mobilize action on the larger issues thrown up by the Bhopal disaster.

In view of the announced (see page) medical survey by the Government, the proposed design for an epidemiological survey assumes added urgency.

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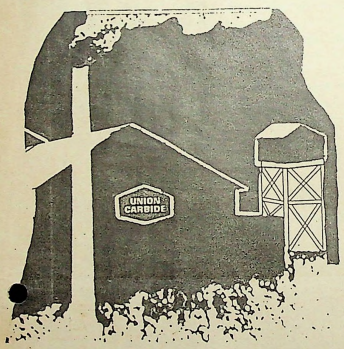

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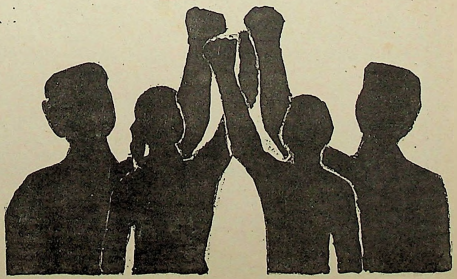

On the SECOND ANNIVERSARY of the world's **worst** industrial disaster, let us pledge to fight multinational interests and governmental apathy to ensure that there shall be

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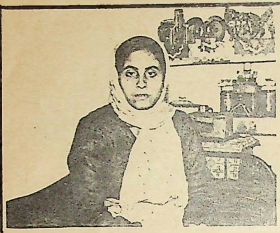


SAZDA BANU (26, Clerk, Widow of Ashraf Mian - UCIL worker killed by Phosegene in 1981; Umrao Dulha Bagh)

On December 3, '84 I arrived at Bhopal by the Lucknow-Bombay Express from Kanpur. The train stopped at the platform at 1.30 a.m. I started coughing, as soon as I got off the train. The platform was deserted. My two children were with me. One was 5 and the other 4. The elder was called Arshad Mohammed Khan and the younger one Shoeb Mohammed Khan. We were all coughing, after which I don't remember anything. Someone carried me to the hospital from where I was later dropped at my brother's house. My children were abandoned at the platform. At 7 a.m. I asked for my children. Then my brother went to the station to look for them and brought them back. At 9 a.m. my elder son Arshad Mohammed Khan expired. After my husband died I got no compensation. My in-laws have filed a court case and they are asking for the money. I have got nothing. I still have not got the 1500 rupees. My in-laws sent me out of the house after my husband died. My husband died on December 25, '81. This also happened because of the manager's mistake. His duty time was over and he was asked to open a valve. The valve had not been opened or even touched for three years, and there was phosegene gas inside. My husband asked him three times if there was gas inside. Three times the manager said, No. After the valve leaked, my husband was kept in the factory dispensary, and later taken to the hospital. I have not got anything yet.

GAURI SHANKAR (23, Unemployed; Indira Nagar)

Ever since I was hit by the gas I have had breathing trouble all the time. I can't ride my cycle. I used to earn 50 rupees daily but now even a cup of tea is a problem. Before the gas I had some tools with me and I was thinking of opening a workshop. But after the gas I was forced to sell some and mortgage the rest to get medicines. I am not very old, I am a youth. I think that if I get a good doctor, I will get better. Carbide should be punished. Carbide is a mass-murderer, which has taken thousands of lives, thousands of women, thousands of cattle and birds. Birds no longer flew in the sky. In my heart I feel like setting Carbide on fire, blowing it up. When I was admitted in the Indore hospital, I used to read poems about Union Carbide and wrote some myself.

RADHA BAI (50, Beedi-making/housework; Jaiprakash Nagar)

My husband is dead. He died in the month of 'Chait' on poornima. He was then under treatment at the DIG Bungalow hospital, he got some medicines from there and took them and then he said, It is Chaiti Punno* so I will take a bath. He pulled up one bucket of water (from the well) and while pulling the second, he fell into the well, he had become giddy. Now I make bidis. The government people said, I could not get relief for his death was not due to the gas.

* Festival on the full moon night of the Chait month (March-April).

RAJU (11, Shaktinagar)

I used to study in class 2 before the gas leak. Now I have unbearable chest pain. I do not go to school because of my poor health. There is pain in my chest and I feel dizzy. I was in the MIC Ward for seven days.

GHANSHYAM (32, Unemployed; Indira Nagar)

I used to drive an autorickshaw, but after the gas leak this became difficult. I cannot look at bright light. I cannot face oncoming headlights. Nowadays I do nothing except go daily to get my eyes treated. I have eruptions in my eyes. There is no use in the treatment. Now I sit at home. I don't go anywhere.

GYANA BAI (40, Housework/Basket weaving; Indira Nagar)

I was pregnant with a son at the time of the gas, now he has trouble with his eyes and cannot see well at night. I can't make baskets properly now. Now I work on one bamboo for two days and make baskets on the third day. Before I would strip the bamboo in a day and make four baskets. When I sit in the sun to strip bamboo, my head reels and darkness comes before my eyes. I go in and lie down, and my eyes burn.

AHMAD ALI (45, Unemployed, Bapna Colony)

I was seriously ill and was in Hamidia Hospital for eight days. I had medical certificates with me. I went with my certificates to the Textile Mill and said, Saab, these are my certificates. I have come back to my job. Then the manager said, Your health is not good and the workload here has increased; give me your resignation. So I gave my resignation. I had worked in the Mill for fifteen years. I was a permanent worker. They said, We need your original certificates (for gratuity etc.). Then they accepted my resignation, but I have not got my certificates back. I have not filled the claim form, nor have I got 1500 rupees. If I had the certificates, I could have filled the claim form.

PREM BAI (40, widowed by gas, Shaktinagar)

I don't know where the lawyers came from. They said that they will get a response for me immediately. The loss of your loved one will be taken care of, they said. My husband died on December 3, '84. I don't know the lawyers' name, address or anything. They took my papers, photo and other things. Other lawyers came and they also took my signature. They said they will make a claim for me.

JAGADISH (35, Worker in Straw Products; Shaktinagar)

The government doctors, they just chased me out of there. When I was admitted to Hamidia hospital, I asked the doctor to keep me there. My condition was very bad. The doctor said, You get out right now. I thought I would wait and try again. In the afternoon the doctor said, Go now, you have been discharged. I don't want to talk to you.

ABID ALI RIZWI (32, Union Official; Chandbad)

I am the Vice-president of the Textile Mill Workers Union. The main problem of the workers after the gas incident is lack of stamina. They have to stand at the machines for eight hours non-stop. Now their capacity to do so is finished. In one or two hours, they are done for. They need rest. If they work for two to four days, they have to go on leave. Absenteeism has greatly increased now, and no division has all its workers present.

NARAINI BAI (35, Housework; Shaktinagar)

We have no money and no job to earn our livelihood. Before the leak, I used to work as a labourer. Now I can't do that. After the gas leak, I have worked only for eight or ten days. I get breathless. I feel dizzy, my head aches. When I rest, I feel slightly better. When I get back to work, I feel sick again.

MAHESH (10, class 3 student; JP Nagar)

I lost many of my friends during the gas leak. My friend Goonja died. Most of my playmates are no more. I get out of breath and cannot run when I play.

RAJESH TIWARI (28, Unemployed; Kainchi Chhola)

Instead of helping the people, the government is putting people on the dole. But people have their rights. After the gas I could see that no political party supports the people. I took part in processions and sit-ins, so that gas victims should get what they are entitled to. But they have not got anything. Carbide should definitely be punished because the dreadful incident that occurred here should not happen again elsewhere.

KANTH BAHADUR (35, Unemployed; Shaktinagar)

I came here from Nepal and immediately after that the gas leak occurred. The gas had a very adverse effect on me. I was in the Hamidia Hospital for eight days and in the TB Hospital for a full month. I lost my job after that.

so I went to the Satguru Complex to meet the Chief Medical Officer Dr. Dheer. I gave him my application and showed him my papers. He said, We will reply to you later. I said that I had heard this response in many places and that he should tell me something final. He asked me whether I was trying to become a 'leader'. He became very angry with me and asked me to get out. All my attempts at showing my medical card were ignored. I fainted under this stress. I was taken by the police to the police station. So much for the want of a job. Dr. Dheer had charged me with goondaism under Sec. 151, IPC. All sorts of lies were written about me in the police report. As they were preparing to hit me, I said, At one level you are keeping me alive and at another you are beating me with sticks. I threw my treatment card at the policemen. I was released several days later, on April 12, '86 and I found out that the police had extracted 250 rupees from my family, in addition to 150 rupees taken from me. After my release, I wrote to the Prime Minister, Health Minister, Collector, Dr. Dheer, and newspapers viz. Dainik Bhaskar, Nai Duniya and MP Chronicle. I sent letters to everyone. Now I am waiting to see who replies.

LAXMI BAI (28, Employed in Carbide Rehabilitation Centre; Shaktinagar)

I feel Carbide is responsible for what happened. Both Carbide and the Government must be punished. Carbide because the gas escaped from its factory and the Government because it gave Carbide permission to be sited where it is. The Government is colluding with Carbide. If it had to punish Carbide, it would have done so by now. I have not voted after the gas leak. Before, I used to vote for Congress. Each and every party must be beaten with shoes. Who bothered to come to us when we lay stricken after the gas leak?

MOHAMMED KHALID (30, Light decorator; Indira Nagar)

My wife, my brother, my sister, my father - my whole family was affected. My wife is in great difficulty. She had a live child in her womb, but the child died. After that she was treated for 8 or 9 months. It cost 90 rupees to pay for just three days medicine.

GOMTI BAI (30, Bidi making/house work; Indira Nagar)

I used to make a thousand bidis a day. Now I don't make even 500 and my head starts to reel. My legs ache when I sit for long. I feel giddy. The tobacco makes me cough a lot, I did not cough before. I feel breathless.

BAL MUKUND (40, Unemployed; JP Nagar)

My legs are useless. I cannot walk. I gave in an application to set up a small shop. But it wasn't granted. Before the gas leak, I used to work at a wood merchant's, but now I am unemployed.

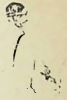
GAFOORAN BEE (60, Housework; Indira Nagar)

I feel as if there is a fire in my head and it seems as though the same gas is leaking. When I cough it feels as though it is the gas again. What do I know about the case? I can neither read nor write. I have got my claim form filled. But I don't know how much it asks for. Who knows when we will get the compensation?

KAMLA BAI (35, Bidimaking/Housework; Indira Nagar)

I don't feel as hungry as I used to before the gas. I can't eat more than two rotis. Many things don't taste good now. My mouth tastes bad.

HARI SHANKAR (40, Street Magician; Vijay Nagar)



Before the gas leak I used to perform magic on the streets. Now I can't do it. I get breathless. I speak for some time and leaves me breathless and my chest pains. I went to find work at the Chandbad Rehabilitation Centre but they told me that I was 'overage'.

SHANKAR LAL (45, Ex-UCIL employee; Kainchi Chhola)

I used to work at the incinerator in Carbide. I used to get about 1200 rupees a month. After being laid off from Carbide, I got a job a year later (January '86) in the Irrigation Department. I now get 430 rupees a month. This does not meet my household expenses. Because of illness I often go late to my new job, and I get shouted at. I have to go to the hospital often. At the most I can work for 10 or 15 days a month. Then I fall sick and I go without pay.

TULSA BAI (55, Housework; Vijaynagar)

After the gas leak, when I went to the village, people said, How dark you have become, and how weak. Even now I remember the gas leak. Sometimes I completely forget about it. And sometimes, it comes back to my heart. I had been on a procession. We went to break the gates of Carbide. To break the gates and voice our demands - to ask, Why have you caused the gas leak and brought us pain? But the police did not let us go. The police beat us up.

RAMESH (24, Cycle Mechanic; JP Nagar)

Five to six months after the leak, government officials came and collected applications for loans. In March '86 they told me that my loan had been sanctioned. Then they asked me to get a photograph of myself and a 'no dues' certificate. I got no dues certificates from 22 banks. Day after day, hungry and thirsty, I went from bank to bank. For three months I did only this.

SANTO BAI (10, Student, Married; Shaktinagar)

I study in the 5th class. After the gas incident I failed. Or else I would have been in the 6th class. My health was poor, so I wrote my exams badly. I cannot concentrate on my studies. Reading makes me giddy, and makes my head spin. In my class 5 or 6 girls failed. This never happened before.

KOMAL BAI (30, Washes dishes; Indira Nagar)

My husband died in 1985. He was very sick after the leak, they said he was suffering from typhoid. We were unable to admit him in a hospital because we had no money. We knew nothing about Carbide. I had never seen it before. This factory should be shifted from here. Some people say Carbide will start functioning here again and sometimes people say it will remain closed. They sometimes say there is another gas leak. Only last month people in Kazi Camp ran away saying the same thing.

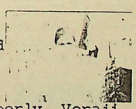
ACCHO KHAN (56, Porter; Indira Nagar)

Before the gas leak I used to carry loads. Now I do nothing. Because of the gas I cannot do anything. I don't even have the strength to talk or run, my lungs are ruined. I have small children. I survive on the 10 to 15 rupees that I earn.

VIJAY (29, Textile worker; Rajendra Nagar)

I work at the loom at the Textile Mill. After the gas leak my stomach aches. I cough a lot. I get tired when I move around. I go to work for just 10 days in a month. There is a difference of 800 rupees in my earnings. This should be compensated by Carbide because the gas leaked from their factory.

ANKUMARI (29, Housework; Vijay Nagar)



The government brought the factory from outside and killed thousands of people. When we fight and put pressure on the government, only then will we get money. Otherwise we will not get anything. They have been distributing 1500 rupees for two years and even that they have not done properly. Voraji says, We will give it to you tomorrow. Rajiv Gandhi says, We will give it to you one day before that. Now they are cleaning the mohallas because they want votes. This happens every year. After the votes they say, We do not know you.

SURESH (8, class 2 student; Shaktinagar)

There is very little to eat. Very little to wear. Papa just doesn't get a job - he has no permanent job. Before the leak, he used to work on a boring machine. Now he cannot work on that machine.

Carbide must be punished. Take them to the police station, then hit them, and then jail them - those Carbide fellows.

I can't play like I used to before the gas leak. Now I am weak. My hands and legs ache when I run. I get breathless soon. If I run I fall down immediately.

JAMEEL KHAN (35, Saw Mill Worker; Rajgarh Colony)

I work as a helper at the Saw Machine. I have been doing since childhood. After the leak I find it difficult to work. Previously I could stand the saw dust, but now it has become unbearable and it makes me cough. I get 400 rupees a month. Before the leak I earned 600 rupees including overtime.

SUMATI BAI (50, Assistant in Aanganwadi*; Shaktinagar)

I participated in a number of protest marches. We had gone on a procession to Kamala Park and there the police beat us all up badly. They hit me on my hand, and till today, a full year later, I feel the pain - I can't even lift my hand. I can't do any work with this hand. I can't lift any loads. I can't fill vessels with water and carry them.

We felt we ought to punish Carbide by ourselves, that is why I participated in the procession - otherwise why should we go? We benefitted from the protest. We got grain.

* Child-care centre.

SAYEEDAN BEE (60, Housework; Indira Nagar)

After the gas everything has been razed to the ground. If we had stayed for two more minutes (during the gas) we would all have been wiped out. I escaped death, but what am I good for now? I have become half-dead. When will we get compensation from Carbide? I know nothing about it.

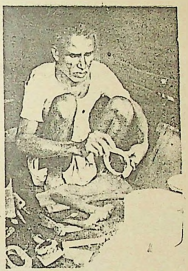
GOPI LAL (40, Cartpuller; Rajnagar Colony)

I push loaded carts. I am breathless most of the time. I go to far off places pushing my cart-loads. My earnings have been reduced by half. There are lots of carts these days. Everyone seems to own a cart bought through bank loan. There weren't so many carts in the days before the leak and business used to be good. Now I can't find any work.

KANTI BAI (30, Housework; Vijay Nagar)

I have five children. All of them suffer from ailments. related to the gas leak. The youngest, Somnath, is suffering the most. He is two years old. He was born two days before the gas leak. He is always out of breath. The medicines just ease his pain for two days after which it comes back again. I admitted him in Hamidia Hospital. There the doctors said he had TB. He was given three injections a day for eight days and then he was discharged. After some time he became just as sick again. Before the gas no one in my family had suffered from TB.

BARJOR SINGH (45, Blacksmith; Kabad Khana)



At the time of the gas I was at home. All at once it hit us. We looked outside. Everything was dark. We stayed home out of fear. If we had stayed outside for two or three minutes, we would all have died. Now I have trouble working. My hands and legs have no life in them. My hands have become scaly. I experience numbness and tingling. My hands and legs have become black. I have no property. What I get is what I beat out of iron.

BHANWAR LAL (46, Worker at Straw Products; JP Nagar)

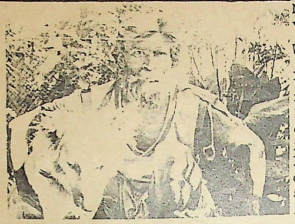
The amount of work I used to do before the leak I can never do again. Now I have reached such a state that I feel like giving it all up and sitting at home. But I am helpless. I have six children, and I am the only earner.

SHAMMU KHAN (50, Cycle shop owner; Indira Nagar)

People are still going around in circles for their 1500 rupees relief money. The assets of Carbide are still intact. Neither is the government taking it over, nor is it using Carbide's assets to help the poor victims. The people are not quiet, it's just that they are being lulled. Like when a child cries, one soothes it by diverting its attention saying a tiger is coming or a goat is coming. Neither does the tiger come nor does the goat. And the child eventually sleeps. The government is working in a similar fashion. We will have to cry out all over again.

RAEESA BEE (35, Housework/knitting machine work; Bapna Colony)

I used to go to the rehabilitation centre to do work. After three months of work in the centre, they said, You people will get loans for machines. You will have to repay in instalments. Because of the gas, my condition is bad, I cannot make 350 rupees a month to pay the instalments. They had taken the responsibility of sending somebody to teach me, but nobody came. In 3 months, I could not learn properly at the centre with 12 women working on one machine.



NARMADA PRASAD (38, Health worker in a voluntary health clinic; Shaktinagar)

When I go in search of labour, people just look at my face and dismiss me - they take only strong men. When I did find a job, for which others are paid 18 rupees, they told me, Dada, we will give you 12 rupees. So I said I will do it for 12 rupees itself, for with that I could buy 2 or 3 kilos of atta. I had only gone a small distance when someone said, Dada, you can't do this - this job is too strenuous for you.

RAM SINGH THAKUR (54, Unemployed; JP Nagar)

Before the leak I was a manager of a wineshop earning around 900 rupees a month. After the leak my eyes have become weak. When I reported for work 1½ months later they fired me, saying if I could not read or write I wasn't fit for them. They chased me away. If I can find work now, I will even work as a chowkidar*.

*watchman.

DEVI BAI (48, Housework; Vijaynagar)

I joined the gas victims' struggle thinking that I could get proper treatment. I would be cured, that may be the poor would be helped. Most of the people joined the struggle in the hope of the same things. Union Carbide should be punished for injuring so many lakhs of people.

MUSHTAQ ALI (25, Railway porter; Model Ground)

At the time of the gas leak I was on duty at the station. I got back to work eight days after that. I feel breathless when I work. I cannot lift the amount I used to. Every month I am sick for fifteen days. Because of this my earnings have suffered badly. Before the gas leak I used to run from one carriage to another. Now if I run from here to the carriage, I am breathless.

SHANTI BAI (35, Housework; JP Nagar)

When I cook my eyes start burning. During the day it is not so bad. But in the evening the burning becomes unbearable. My hands and feet ache, and my chest pains. When I lift water, I can never lift the amount I used to.

SAGEERA BEE (30, Housework; Rajgarh Colony)

I have spent nearly 8000 rupees on private treatment. I had to borrow this money at 25% a month interest. I have sold my goats. Our son has become very weak. Day by day his condition becomes worse. He is 14 months old.

SHANTI BAI (26, employed in rehabilitation centre; Shaktinagar)

I work at a sewing centre. In a month, we get jobs for 20 days, sometimes for 15 days and at other times for 12; never do we get employed for a full month. My monthly wages are 250 rupees sometimes, 200 rupees at others and at times 275 rupees. Never do we get more than this. If I work for a full day, I experience pain in the hands and legs, and a burning sensation in the throat and chest pain.

MAMTA (6, Housework; Shaktinagar)

They distribute bread at the aanganwadi. Then they let us go. I like bread. We don't get any toys to play with at the aanganwadi. We are taught अ and आ .

RAMCHARAN (20, class 11 student; Indira Nagar)

My health was good before the leak. Ever since the leak, I feel giddy, my eyes have become weak and I can't read well. This year 50% of my class has failed. Before the gas leak we had an 80% pass percentage and our school had a good record. My condition worsens day by day. Previously we never realised that this factory would be so lethal and dangerous. After the leak everyone knows. We have seen this factory around for ages but we were never told what it manufactured.

PRADEEP (12, class 6 student; Kainchi Chhola)

It is Carbide's fault that the poison escaped. The big-big officers in Carbide should be punished. They should be hung.

SUNIL KUMAR (14, class 9 student; JP Nagar)

Just as people were killed by the gas, something similar should be done to punish Carbide. This punishment cannot be given by the Bhopal Court. Two years have passed and Carbide and the Government are still arguing, but they are doing nothing. I think if the owner (of Carbide) were here, I would slay him - and it is not just me but the whole colony that thinks so. Carbide has destroyed my family- seven people, my mother, father, three sisters and two brothers died. Many of my friends also died.

TWO YEARS AFTER

Two years ago, the whole world held its breath at the horror that Union Carbide's satanic factory had caused, through UC's merciless and hell-bent quest for profits. For years, people had warned of the madness of pouring poisons into our environment in the name of prosperity. Carbide, through its relentless cost-cutting and its systematically hazardous management of ultra-hazardous materials, has taken a short-cut to the devastation that we dreaded. Two years have passed. Neither the death of thousands nor the misery and suffering of lakhs has made Carbide flinch from its attitude to 'business as usual'. On the contrary, it has done everything to impede justice to the people of Bhopal. But Carbide will not go unpunished.

The dimensions of the tragedy are heightened by the scandals that surround every aspect of it. The government's relief and rehabilitation plans still remain so many castles in the air. Fully two years after the event the government has become, if possible, even more fanatical about secrecy than it ever was. Compounded with Carbide's criminal refusal to divulge any information on the nature of the gases, the victims are again the same. With all the medical studies commissioned into effects of the gas (nearly 20 by the ICMR alone), the treatment remains practically the same as it was in the first week of the disaster. The ICMR studies including the epidemiological survey are still 'Confidential'.

We wanted to review all aspects of the disaster in this issue. The question was - how? With unmitigated hostility and suspicion from government officials towards BGIA, collecting information continues to be hazardous. We wrote letters to all the chief officials in charge of gas relief with a detailed list of questions on government action and planning. Not one responded. We decided therefore, to let the gas victims speak for themselves - this would be the best way of conveying the nature of their suffering, the most revealing and comprehensive means of understanding their condition.

Physical suffering is unabated - the medical treatment, such as it is, has had little effect. Perhaps most devastating is their anguish at their helplessness. Their chief source of pride - their work, their ability to provide for themselves, has been taken away from them. But their voices are as eloquent as ever.

Any hope for proper and long-term relief and compensation seems possible only as a result of such eloquent struggle. But how long can a deprived and debilitated community struggle? Gradually, hope is likely to give way to despair. When that happens, the industry, government and the general public shall write 'finis' to Bhopal and march on relentlessly till another one is let loose. We must not let that happen.

'BHOPAL' is brought out by the Bhopal Group for Information and Action.
D-42 FIRDOS NAGAR
BHOPAL 462 018

This issue was produced by Goutam Banerjee, Vinod Raina, S Ravi Rajan, Arvind Rajagopal, Satinath Sarangi, and MK Srinivasan with assistance from NR Mohandas.

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BHOPAL

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The Deaths Continue

Death still stalks the shanties of Bhopal twenty months after the disaster wreaked by Union Carbide. Poisoned by a killer multinational, neglected by the government, ignored by the media, the gas victims continue to suffer from illnesses that are yet to be properly diagnosed, let alone cured. And they continue to die. Like Moolchand of Patel Nagar, Bhopal.

The Story of Moolchand

Moolchand died at 5.15 p.m. on 14 June, 1986, on bed No. 17 of the MIC Ward, nine months after admission to the Hamidia Hospital. His tragic tale is the tale of many many more who wait, spent and dispirited, for a slow, troubled death.

Moolchand came to Bhopal in 1950 in the tumultuous aftermath of the partition of the country. A Sindhi migrant from Pakistan, Moolchand had little to survive on when he started life anew at Bhopal. He eked out a living as a scrap merchant, carrying his load of waste around the city on a bicycle. The only one to help him was his son, Vasudev. And they did manage to survive and sustain their small family through the years. Until the night of 2nd December, 1984.

That fateful night, Moolchand awoke with a start to find his room thick with acrid, irritating fumes of gas. He was afraid, and alone, for his family had fled. So he too ran, like thousands of others, to save his life. And he survived. But just. The toxins had ravaged his body so much he was unable to work. His business suffered. He could not breathe properly, his body was wracked by coughing, he lost his appetite, he couldn't see well. And then began his frustrating search for a cure, or even for some relief from suffering.

Each morning he joined the queues at the government clinics with his wife Premvati, who was similarly affected. He took drugs and drugs and more drugs. But to little avail. There was little respite from his painful condition. In despair he went to private doctors. His savings were all gone. He had to sell off his wife's jewels to pay for the expensive medicines the doctors prescribed. Again, to little effect, because he continued to suffer and his condition deteriorated.

On 2 September 1985, in a last desperate bid to save his life, a pale and emaciated Moolchand went, once again, to the government polyclinic at Bharat Talkies. He shuttled for days between the clinic and Hamidia Hospital. Until, on 9 September 1985, he was admitted to the MIC ward. He still complained of breathlessness, cough with expectoration, muscular weakness in the legs and generalised weakness. Clinical and pathological examinations ruled out tuberculosis but pointed to bilateral crepitation. The diagnosis: "MIC-induced lung diseases"!

His wife, herself suffering, was his constant companion at the hospital. She made the daily four-kilometre trip from their home in Patel Nagar to the hospital with their son. Moolchand was prescribed pain-killers, broncho-dilators, steroids .. ... "He used to take almost half a kilogram of medicines everyday", reminisces another patient in the ward.

Then one day Moolchand slipped and fell in the bathroom. (This is not uncommon in government hospitals where reeking toilets and bathrooms are covered with the slippery slime of dirt and excreta). He fractured his thigh bone. The fracture was improperly set. As a result, one leg was an inch shorter than the other when the cast was removed. Already weak, Moolchand was now further immobilised , confined to his hospital bed. He lay gasping for six months, his family driven to the brink of deprivation. Till he finally lost the will to live. As a final act of resignation and despair, he stopped eating just prior to his death. He died in the evening of 14 July, 1986.

Many, like Moolchand, continue to die. In the absence of a meaningful and adequate system for monitoring the conditions of victims, many of these deaths go unreported. Says Professor Heeresh Chandra, Director of the Medico-legal Institute, under whose charge the autopsy of Moolchand's body was carried out, "Many of the gas victims are dying, yet very few dead bodies are brought in for autopsy." Despite many suggestions, the government has not made autopsy compulsory. Such an omission attains crucial significance in the context of the opinion expressed by Michael Ciresi, the lawyer retained by the Government of India, that the nexus between exposure to gas and subsequent deaths has to be established beyond reasonable doubt if damages for death are to be claimed from Union Carbide Corporation.

The complete absence of such initiative was blatantly made clear in an interview with the Director of Claims, Mr. Khare. We asked how Moolchand's family could file a claim for compensation. Sitting in his office tucked away in a maze of government buildings, seven kilometres away from Patel Nagar, Mr. Khare confessed, "We do not make any attempts to inform people that they can still file claims." When pressed, he tried to justify this glaring omission by saying - "Somehow some people do come."

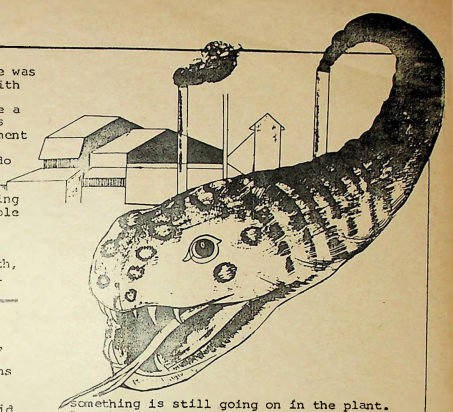
Such wilful neglect condemns the gas victims to prolonged suffering and death, even as the death dealing multinational goes unscathed.

TOXIC EMISSIONS CONTINUE

Shortly before noon on 19th June, 1986, clouds of black smoke were visible at the UC plant in Bhopal. Investigations by BGIA revealed a raging fire next to the administrative building inside the plant. On enquiry, security guards said that plastic cans containing toxic chemicals were being destroyed. The fire continued for almost an hour.

This is not the first time since the December, 1984, gas leak that toxic materials have been disposed off in this manner by Union Carbide without informing the public. In February last year, toxic fumes from the incinerator inside the plant entered a double storied school building near the eastern wall of the plant while the school was in session. About 50 children, all below twelve years, started coughing violently and some started vomiting. A number of children complained of giddiness and nausea. The teacher, not knowing how to respond to such a situation, declared the school closed for the day and the children were asked to go home. The matter was taken up with the authorities but no action was forthcoming. The emissions from the incinerator continued unabated. It was only after a group of young people from Shaktinagar, where the school is located, went up to the gate and hurled stones that the authorities got the message and the emissions stopped.

Incidents such as these aggravate the panic-bound psyche of the gas victims, besides causing injury to their already damaged bodies. Many of the victims continue to talk about the poison clouds of the December leak and visions of that catastrophe recur in their dreams. Mention is often made, sometimes in hushed tones and sometimes in anger, that



Something is still going on in the plant. But who is bothered about the gas victims anyway?

WITHOUT COMMENT

Last month, the UCO bank in Bhopal was robbed in broad daylight by a band of yet unidentified persons. They are reported to have introduced themselves as "Hum log gas peedit hain, bank lootne aye hain" (We are gas victims, we have come to loot the bank). Reading the report, an old gas victim commented, "Gas peediton ke naam se kitnon ne loot liya, yeh ek aur sahi" (So many have looted in the name of gas victims, this is just one more instance).

STREET VS STAGE

Rangmandal, the theatre group of Bharat Bhavan, the elite cultural centre of Bhopal staged a play early this month based upon the Bhopal gas disaster. 'Banjh Ghati' (Barren Valley), staged twenty months after the disaster, was, however, eclipsed by the forceful 'Dastan-e-Gas Khand' (Tale of the gas disaster) enacted at the same time on the streets opposite the Bhavan by 'Shuruat' a group of amateur artistes. This group has been performing the play since early last year in many parts of Bhopal and elsewhere, including the gas affected slums. Among the crowd which preferred to watch the street theatre were many young artistes of Bharat Bhavan itself.

SURAKSHA

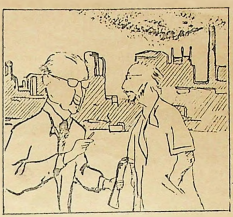
सुरक्षा

A new project, involving the gas affected children in painting, music, story telling, poetry and drama, has started in the Jayaprakash Nagar Basti of Bhopal. It has been organised by David Bergman, a young Britisher who has been in Bhopal for the past three months. (David and a friend undertook a cycle tour from England to Bhopal to raise funds for the gas victims of the Bhopal disaster.) The group which has been set up for the purpose is called SURAKSHA.

At present, five teams of local artists (of 2/3 individuals per team), each conduct a workshop in the basti once a week. A large covered area is used for these activities. The project is now in its third week, and 30-50 children are regularly involved.

Suraksha feels that although there is an obvious need for such opportunities to be given to basti children everywhere, there is a special need for them in Bhopal today. Apart from the physical disabilities the children are still suffering from, many of them have also gone through extreme mental anguish and stress during and after the gas disaster. Emphasis is, therefore, being placed in these workshops on psychological therapy.

For the next few months, Suraksha plans to limit itself to working in this basti with these five teams of artists. Indeed, much needs to be done just to make these activities with the children successful. Relationships of friendship and trust need to be built up between the artists and the permanent workers of Suraksha, on the one hand, and between Suraksha and the children, on the other. The children must be involved in all aspects of the project. Attempts must also be made to get the general acceptance of the project within the basti community. Only then can they become involved in the general organisation of Suraksha and in the supervising of the activities. Finally, much discussion needs to be done with the artists themselves to work out coherent, structured activities which have an overall aim behind them. Such activities can then continue over a number of weeks and months, with new teams of artists joining in at different points.



"The gas leak is okay. What we've got to guard against is the leaks to the press."

We have

the right to know

From September/October onwards, it is hoped that Suraksha will be working in two or three independent "activity huts" structures of timber and bamboo - around JP Nagar and Shakti Nagar. Apart from the core of activities being carried out by local artists and others from the basti, individuals and groups working with children from other parts of the country are also being invited to come to Bhopal to work intensively for two or three weeks. Such groups could exchange ideas with local teams so that their work and methods could be continued in their absence.

Once a good understanding and friendship has been built up with the children, Suraksha is interested in organising a nutritional study of the children. Such a survey is especially important now because of the large amounts of drugs that their bodies are being subjected to.

The success of all this is very much dependant on two to three more permanent workers committing themselves for at least three months to this project. So the group requests interested people to come and work with them. The group also invites ideas and suggestions from interested people. If interested, write to:

SURAKSHA
A-73 SANT KANWAR RAM NAGAR
BHOPAL 462 018.

A writ petition on the issue of the right to know has been filed recently in the Supreme Court of India by the environmental action group, Kalpavriksh. This petition is an intervention in the ongoing case against the Sriram Foods and Fertilizers Company which was responsible for the Oleum gas leak in Delhi, on the first anniversary of Bhopal, which resulted in the death of one person and injuries to many others.

Kalpavriksh had earlier intervened in the case, pleading that the plant be shut down and relocated. As readers might recall, the Supreme Court had then ruled that the plant could reopen subject to its management meeting certain conditions, prominent among which is the one which holds the top management personally responsible for paying compensation in the event of future accidents.

The right to know petition argues that citizens have the right to be informed about the nature of the dangers posed to them by hazardous industries. The petition is due for hearing later this year.

Support the call by KSSP for a Global boycott of all products of Union Carbide by Environment and Consumer Groups with immediate effect.

For more information and details on campaign, please write to:

KSSP – Kerala Sastra Sahitya Parishad,
Parishad Bahvan,
Trivandrum 695037
Kerala, South India.

AIRPLAN

In May, 1985, representatives from environment groups from across the world, including India, met at Eerbeek, a village in Holland. The subjects of concern were the growing menace of AIR POLLUTION, particularly Acid Rain and the issues raised by Bhopal.

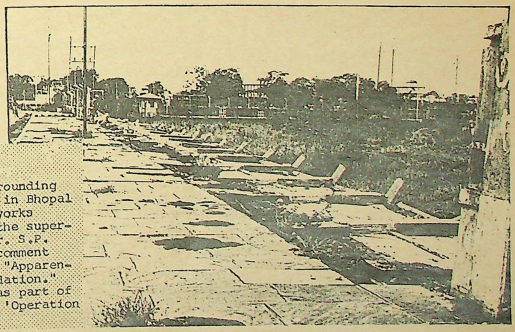
Out of this meeting grew a new global network linking environmental groups concerned about these issues. AIRPLAN, as this network is called, coordinates international campaigns and is a forum for exchange of information and action plans.

AIRPLAN has been around for a very short while but has already demonstrated the potential of such a network. Recently, an appeal sent out by it resulted in international pressure being applied on the government of an East European country to free two environmentalists who had been jailed for raising ecological issues. In order to maintain a regular channel for communication and information exchange, AIRPLAN publishes a bimonthly newsletter, two issues of which have already come out.

AIRPLAN welcomes interested groups to join and strengthen its network.

Their address:

PO BOX 5627
1007 AP Amsterdam
The Netherlands.



FAITH WITHOUT FOUNDATION

A portion of the wall surrounding the Union Carbide factory in Bhopal collapsed recently. The works manager, Mr. Mukund, and the supervisor of the MIC plant, Mr. S.P. Choudhary, were heard to comment during their spot visit – "Apparently, the wall has no foundation." Incidentally, this wall was part of safety system No.5 during 'Operation Faith'.

Quacks have a field day

Every gas affected person in Bhopal must have visited a private medical practitioner at some point in time over the past year and a half. Every private medical practitioner must have treated anywhere between 2,000 and 10,000 gas affected patients during this period. Indeed, this group of "businessmen" could not have had things better - the boom in their trade stands in stark contrast to the gloom and depression pervading every other economic activity in the area.

In normal times, the present period of the year is referred to as the "slack season" by these private practitioners. But these are not normal times - the stream of patients does not end, the shops stay open till late in the night. Clearly, the potential of these private doctors to do good or cause harm to their patients is phenomenal. The question is - how safe are the lives of the thousands of gas affected people in the hands of these 200 odd self-professed medicos?

The first thing one notices while visiting a private doctor in the gas affected areas of Bhopal is his sign board. Often oversized, it proclaims the doctor to be a specialist in a variety of areas of medical knowledge. One sign board, for instance, advertises its owner as a specialist in medicine, surgery, gynaecology, paediatrics and sex problems. Some sign boards mention the degrees the "doctor" has acquired. These are usually a curious mixture of unevenly spaced letters amenable to a variety of interpretation - U.V.P., V.V.P., M.I.A.M.S., M.A.M.S. etc.

On a rough estimate, eight out of every ten private doctors have received no formal training in medicine. Their medical qualifications usually comprise an apprenticeship with another private doctor, coupled with lessons received from correspondence courses offered by ubiquitous "institutes" characterised by their anonymity and dubious reputations. Many doctors take such correspondence courses simultaneously in more than one system of medicine. Thus, it is not rare to meet a private doctor who claims to treat his patients by the allopathic, homoeopathic, ayurvedic and Unani systems. As one doctor interviewed put it, "We try different systems and see which one fits - my patient must find relief." The treatment is, by and large, oriented to provide instant relief. "People are workers here", one doctor explained, "they have to be fit for work, so they want quick relief lest they lose their wages."

Talking to some of these private doctors, one picks up very interesting bits of information. One learns, for instance, that Sodium Thiosulphate is a drug from the U.S. and is not of much use in India because the blood circulation, the pulse rate and breathing rates of Indians are different from those of people in the U.S. One also learns that the outermost layer of the eye, contrary to what class nine biology students are taught in schools, is called the Retina. One also encounters a host of very interesting theories which attempt to explain the effect of the toxins in the body. One such theory is that the gas has produced "heat in the body" which cannot be tackled by ordinary medicines, since the gas has interfered with the formation of pancreatic juices and saliva, causing problems of digestion.

Private doctors have several pertinent observations to make about the total health situation in the bastis. They are in a position to do so since many have been in the area for almost twenty years. We list some of these observations

- While the number of patients is still considerable, there has been a 50 per cent fall in the past year. One reason is that many gas victims have resigned to their suffering
- Patients who visited their clinics only once a month before the gas disaster now do so once or even twice every week
- A large number of patients have been suffering without respite ever since the gas leak and despite the treatment despite the treatment received from the private medicos and government clinics.
- More than half the patients who come to their clinics have symptoms directly related to gas exposure
- Diseases which were prevalent earlier now occur more frequently and with intensity
- The variety and nature of symptoms is baffling. They include breathlessness, gastric disorders, loss of appetite, fatigue, weakness, body aches, dizziness, abdominal pain, headache, menstrual irregularities, impotency, loss of vision, burning sensation in the eyes, pain during intercourse, anxiety, insomnia, headache and chest pain

According to another theory, "MIC affected patients need hard drugs, potent drugs, drugs which are costly. Only then can they get better."

These beliefs, coupled with the hard-sell tactics of big drug companies (smooth talking medical representatives and stylishly printed literature which invariably adorns the walls of the clinics) have resulted in utter irrational and dangerous "therapy". Antibiotics are prescribed at the first signs of cough, fever or boils. Rarely are patients prescribed the full course; where they are, patients discontinue use half way through the course, rendering themselves more vulnerable to further infections. Injections, the ultimate panacea in the eyes of many patients, are readily given by the doctors to patients with money to spend on these "miracle" drugs. Another favourite is steroids (which provide a "sense of well-being" though they impair the natural immunity system of the body). Tonics, which, according to one doctor, are prescribed "to give energy to the gas victims", formulations such as "Protinules", "Protinex", "Sharkaferrol" and "Ferradol" and useless drugs such as Kaolin-Pectin mixture for children with diarrhoea are also routinely prescribed. Moreover, drugs such as Phenylbutazone and Oxyphenabutazone preparations (e.g. Actigesic, Bestophen, Suganril and Reducin) which have been categorised by the drug controller of India, as restricted for use in the treatment of ankylosing spondylitis and gouty arthritis (and whose potent toxic after effects have been demonstrated by the fact that Ciba Geigy the principal manufacturers of these drugs is reported to have recorded 1674 deaths due to these drugs) are routinely given for body aches.

The private medical practitioners charge between Rs. 5/- and Rs. 25/- per patient per visit. An average household in the gas affected area would have spent at least Rs .2,000/- in the last eighteen months on medicines and consultation fees of these private doctors. There are many who have borrowed money for this purpose at rates of interest sometimes as high as 120%.* The government run hospitals, on the contrary, charge nothing. The obvious question is - why do the victims prefer these private doctors to the hospitals run by the government. There are several reasons,...

*(These figures give lie to the ill-conceived notions of most government doctors who maintain that the gas victims are pretending to be sick as they want compensation).

The government run hospitals and clinics are open only during the day and in the early evening, times when people are out working, while private clinics are open till late in the night.

The attitudes of the doctors towards their patients is an impressive blend of smart salesmanship and a veneer of professionalism cultivated over several years of experience and practice. In sharp contrast, the government doctors who are too pressed for time do not spend enough time or give personal attention to their patients. The private medicos give a patient hearing to each person and spend considerable time in clinical examinations. Indeed, the private doctors score over even the ICMR researchers and senior government doctors when asked to provide an oral list of symptoms presently displayed by the gas affected people.

The shortage of government doctors means long queues at the clinics. There are several other inadequacies in the governmental medicare system. As one private doctor stated very candidly - "The government doctors prescribe the same medicines as we do, they have much greater facilities, if they happen to improve their services, we will be out of business."

That doctor, like most others operating in the gas affected bastis of Bhopal, does not lose sleep over this possibility, fully aware that such a change in the governmental attitude is extremely improbable.

AN APPEAL

In the first issue of 'BHOPAL' which we mailed to you a month ago, we laid out the objectives of the Bhopal Group for Information and Action. We have received a positive response to that issue and to the idea of BGIA. Our thanks to all those who've responded.

In the last month, we've begun the task of collecting material on Bhopal and setting up the documentation centre. We've also found a place near the Union Carbide factory to house the centre.

As we mentioned last time, we see our small group here as a mere extension of a shared desire of a vast community to sustain work on Bhopal and related issues. Moreover, the task of documentation, bringing out the newsletter and running and information service can be sustained only with your active support.

At the moment, BGIA requires the following assistance from you and we trust you will try your best to help:

- We need a list of all documents concerning Bhopal which you may possess or have access to. This list is vital for the building up of the documentation centre.

- We need some equipment and financial assistance. Equipment includes a typewriter, storage cabinets, stationery and paper.

- We need contributions to keep the newsletter going. We urge you to
  - — contribute a minimum of Rs. 24/- a year in advance (Rs 30/- for institutions and $ 30/- for foreign groups); and
  - -- undertake to sell a minimum of five copies of the newsletter on a regular basis at Rs. 2/- a copy.

More than anything else, we value your comments, criticisms and advice.

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This issue was produced by Satinath Sarangi, S. Ravi Rajan and Vinod Raina, with assistance from N R Mohandas.

Medline Search on Bhopal – 15 years 14/11/98

OH1 : 30

PC-SPIRS 3.40 MEDLINE (R) 10/97-12/97

NIMHANS Library

TI: A study of cognitive functions in methyl-iso-cyanate victims one year after bhopal accident.
AU: Misra-UK; Kalita-J
AD: Department of Neurology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, India.
SO: Neurotoxicology. 1997; 18(2): 381-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: There is a paucity of information regarding the late effects of methyl-iso-cyanate (MIC) poisoning. In this study, the clinical and cognitive functions of 52 MIC victims were evaluated one year after the Bhopal gas accident. There were 15 severely, 14 moderately, and 23 mildly affected patients. Their mean age was 38.2 (range 15-65) years and 30 patients were males. Their clinical picture was dominated by respiratory symptoms in 36 patients, of which 17 had abnormal chest radiographs. Neurological examination of these patients was normal. Psychometry in 33 patients revealed significant impairment of standard progressive matrices (SPM), associate learning and motor speed and precision tests ($p < 0.01$) in severely exposed MIC patients. In the moderately affected group associate learning and motor speed and precision were significantly impaired. Motor speed and precision tests had significant positive correlation with eye signs ($r = 0.42$) and disability score ($r = 0.68$). Our results suggest significant cognitive impairment in severely exposed MIC victims.

PC-SPIRS 3.40 MEDLINE (R) 1/96-1/97

TI: Long term morbidity in survivors of the 1984 Bhopal gas leak.
AU: Cullinan-P; Acquilla-SD; Dhara-VR
AD: National Heart and Lung Institute, London, UK.
SO: Natl-Med-J-India. 1996 Jan-Feb; 9(1): 5-10
This source is Available in S.J.M.C Library
Call Number: From: 1988+
LA: ENGLISH
AB: BACKGROUND: The extent and nature of long term health sequelae among survivors of the Bhopal gas disaster are not known. In 1994 an International Medical Commission was set up with the aim of assessing respiratory, neurological and other health effects attributable to gas exposure. METHODS: An epidemiological survey of a representative sample of gas-exposed inhabitants of Bhopal was conducted in January 1994; for reference, a group of unexposed persons in the same city were surveyed. Questionnaires regarding health and exposure were administered to 474 persons, and a random sample (n=76) were subjected to respiratory and neurological testing. Responses to the questionnaire and the results of clinical testing were analysed according to a measure of individual gas exposure. RESULTS: A large number of subjects reported general health problems (exposed v. unexposed; 94% v. 52%) and episodes of fever (7.5/year v. 2.5/year); adverse outcome of pregnancy (e.g. still-births, 9% v. 4%) and respiratory symptoms (81% v. 38%), with a strong gradient by exposure category. This was not accounted for by differences in smoking, and was consistent with the results of spirometric testing.
Neurological and psychiatric

in high exposure categories and the results of neurological examination and testing tended to confirm this finding. Ophthalmic symptoms demonstrated a similar pattern. Although a number of other symptoms were reported (with the

 there was no clear evidence of

TI: Respiratory morbidity 10 years after the Union Carbide gas leak at Bhopal: a cross sectional survey. The International Medical Commission on Bhopal [see comments]
AU: Cullinan-P; Acquilla-S; Dhara-VR
AD: Department of Occupational and Environmental Medicine, Imperial College (National Heart and Lung Institute), London.
SO: BMJ. 1997 Feb 1; 314(7077): 338-42
This source is Available in S.J.M.C Library
Call Number: From 1914+
LA: ENGLISH
AB: OBJECTIVE: To examine the role of exposure to the 1984 Bhopal gas leak in the development of persistent obstructive airways disease. DESIGN: Cross sectional survey. SETTING: Bhopal, India. SUBJECTS: Random sample of 454 adults stratified by distance of residence from the Union Carbide plant. MAIN OUTCOME MEASURES: Self reported respiratory symptoms; indices of lung function measured by simple spirometry and adjusted for age, sex, and height according to Indian derived regression equations. RESULTS: Respiratory symptoms were significantly more common and lung function (percentage predicted forced expiratory volume in one second (FEV1), forced vital capacity (FVC), forced expiratory flow between 25% and 75% of vital capacity (FEF25-75), and FEV1/FVC ratio) was reduced among those reporting exposure to the gas leak. The frequency of symptoms fell as exposure decreased (as estimated by distance lived from the plant), and lung function measurements displayed similar trends. These findings were not wholly accounted for by confounding by smoking or literacy, a measure of socioeconomic status. Lung function measurements were consistently lower in those reporting symptoms. CONCLUSION: Our results suggest that persistent small airways obstruction among survivors of the 1984 disaster may be attributed to gas exposure.

TI: Relationship between lung inflammation, changes in lung function and severity of exposure in victims of the Bhopal tragedy [see comments]
AU: Vijayan-VK; Sankaran-K
AD: Cardiopulmonary Medicine Unit, Indian Council of Medical Research, Madras, India.
SO: Eur-Respir-J. 1996 Oct; 9(10): 1977-82
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: The world's worst chemical industrial disaster, which occurred at Bhopal on 2-3 December, 1984, resulted in considerable respiratory morbidity in the exposed population. Therefore, a study was planned to evaluate the relationship between lower respiratory tract inflammation, lung function and severity of exposure. Sixty patients exposed to methyl isocyanate and presenting with respiratory symptoms were studied using bronchoalveolar lavage (BAL) 1-7 yrs after the accident. Pulmonary function tests included forced vital capacity (FVC) and forced expiratory volume in one second (FEV1). An index of severity of exposure was derived retrospectively on the basis of the acute symptoms in the victims themselves or the occurrence of death among their family members. Total lung inflammatory cells ($p < 0.01$) and absolute numbers of macrophages ($p = 0.01$) and lymphocytes ($p < 0.05$) increased as severity of exposure increased.

PC-SPIRS 3.40 MEDLINE (R) 1998/01-1998/10

1 of 1
Marked Record

TI: Improving the U.S. EPA Toxic Release Inventory database for environmental health research.
AU: Neumann-CM
AD: Department of Public Health, Oregon State University, Corvallis 97331-6406, USA. neumannc@ccmail.orst.edu
SO: J-Toxicol-Environ-Health-B-Crit-Rev. 1998 Jul-Sep; 1(3): 259-70
LA: ENGLISH
AB: In 1986, Congress passed the Emergency Planning and Community Right-to-Know Act (EPCRA) in response to the tragic death of thousands of people in Bhopal, India, following the accidental release of the toxic gas methyl isocyanate (MIC) from a Union Carbide facility. As a component of EPCRA, certain manufacturers are required to report annually the total mass (pounds per year, lb/yr) of toxic chemicals released into the environment (air, water, land, or underground injection), treated on-site, or shipped off-site for further waste treatment. This information is compiled by the U.S. Environmental Protection Agency (EPA) into a publicly accessible database known as the Toxic Release Inventory (TRI). The TRI database is designed to encourage pollution prevention and waste reduction by increasing public access to and knowledge of environmental chemical releases. EPCRA has been generally considered by industry, government, and community representatives as one of the most successful environmental laws in U.S. history. Over the past few years, EPA has initiated a three-phased expansion to EPCRA reporting requirements that will enhance the overall usefulness of the TRI database. The focus of this article is to discuss these changes and highlight several current uses of the TRI database in environmental health research.

PC-SPIRS 3.40 MEDLINE (R) 1/97-9/97

1 of 1

TI: The lessons of Bhopal [toxic] MIC gas disaster scope for expanding global biomonitoring and environmental specimen banking.
AU: Sriramachari-S; Chandra-H
AD: Institute of Pathology (ICMR), Safdarjang Hospital Campus, New Delhi, India.
SO: Chemosphere. 1997 May; 34(9-10): 2237-50
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Bhopal Toxic gas tragedy represents one of the worst chemical accidents of the world. Autopsy and toxicological studies, apart from presenting evidence of acute and even chronic cyanide toxicity, provided a unique example of the incriminated chemical being traced to the bodies of the victims. The entry of methyl isocyanate (MIC) into the blood stream was established by the presence of carbamoylated end-terminal amino acids of haemoglobin and other tissue proteins. The presence of MIC trimer and a few other identified as well as unidentified tank residue constituents in the blood and viscera further established a close nexus of the products of pyrolysis of MIC in the aerosol inhaled by the victims. The Bhopal studies exemplify the scope for biological monitoring (BM) and environmental specimen banking (ESB) in chemical accidents as part of the global efforts.

FEV1/FVC % (p = 0.05) was also significantly lower as severity of exposure increased. Moderately exposed subjects had significantly lower FEV1/FVC % (p < 0.05) compared to those mildly exposed. In nonsmokers, BAL neutrophils, both percentage and absolute numbers, showed significant negative correlations with FEV1 % predicted (rs = -0.350, p < 0.05; and rs = -0.374, p < 0.01, respectively). Neutrophil percentage was negatively correlated with FEV1/FVC % (rs = -0.378; p < 0.01). Absolute lymphocytes had significant negative correlations with FVC % pred (rs = -0.318; p < 0.05). Macrophages had significant positive correlations with FVC % pred (rs = 0.322; p < 0.05) and FEV1 % pred (rs = 0.433; p < 0.01). Radiographic abnormalities (International Labour Organization (ILO) classification) were associated with decline in FEV1 % pred (p < 0.05). This study suggests that pulmonary function abnormalities occur in gas-exposed subjects as a consequence of an abnormal accumulation of lung inflammatory cells (lymphocytes and neutrophils), and that the intensity of lung inflammation and reduction in pulmonary function are greater in severely exposed subjects. As it has been observed that decline in pulmonary function is associated with radiographic abnormalities, there is a suggestion that injury following toxic gas exposure can lead to irreversible lung damage.

3 of 5
Marked Record

TI: Late consequences of accidental exposure to inhaled irritants: RADS and the Bhopal disaster [editorial; comment]
AU: Nemery-B
SO: Eur-Respir-J. 1996 Oct; 9(10): 1973-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH

4 of 5
Marked Record

TI: International Medical Commission, Bhopal: a model for the future.
AU: Bertell-R; Tognoni-G
AD: International Institute of Concern for Public Health, Toronto, Ontario, Canada.
SO: Natl-Med-J-India. 1996 Mar-Apr; 9(2): 86-91
This source is Available in S.J.M.C Library
Call Number: From: 1988+
LA: ENGLISH

5 of 5
Marked Record

TI: Major chemical accidents in industrializing countries: the socio-political amplification of risk.
AU: de-Souza-Porto-MF; de-Freitas-CM
AD: Study Center of Workers' Health and Human Ecology, National School of Public Health, Rio de Janeiro, Brazil.
SO: Risk-Anal. 1996 Feb; 16(1): 19-29
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Accidents in the chemical industry, such as those that took place in Seveso (1976) and Bhopal (1984), may kill or injure thousands of people, cause serious health hazards and irreversible environmental damage. The aim of this paper is to examine the ever-increasing risk of similar accidents becoming a frequent ocurrence in the so-called industrializing countries. Using figures from some of the worst chemical accidents in the last decades, data on the Bhopal disaster, and Brazil's social and institutional characteristics, we put forward the hypothesis that present social, political and economic structures in industrializing countries make these countries much more vulnerable to such accidents and create the type of setting where--if and when these accidents occur--they will have even more catastrophic consequences. The authors argue that only the transformation of local structures, and stronger technical

cooperation between international organizations, industrialized and industrializing countries could reduce this vulnerability.

PC-SPIRS 3.40 MEDLINE (R) 1/95-1/96

1 of 5
Marked Record
TI: Persistently toxic. The Union Carbide accident in Bhopal continues to harm [news]
AU: Mukerjee-M
SO: Sci-Am. 1995 Jun; 272(6): 16, 18
This source is Available in S.J.M.C Library
Call Number: From: 1949+
LA: ENGLISH

2 of 5
Marked Record
TI: Comparative toxicity of methyl isocyanate and its hydrolytic derivatives in rats. II. Pulmonary histopathology in the subacute and chronic phases.
AU: Sriramachari-S; Jeevaratnam-K
AD: Institute of Pathology, New Delhi, India.
SO: Arch-Toxicol. 1994; 69(1): 45-51
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: This paper describes the long-term (subacute and chronic) histopathological effects in the lungs of rats subjected to a single exposure to methyl isocyanate (MIC) by both the inhalation and subcutaneous (s.c.) routes as well as the role of methylamine (MA) and N,N'-dimethyl-urea (DMU), the hydrolytic derivatives of MIC in eliciting the observed changes. At the subacute phase, the intraalveolar and interstitial edema were prominent only in the inhalation group as against the more pronounced inflammatory response in the s.c. route. With the progress of time the evolution of lesions appeared to be similar, culminating in the development of significant interstitial pneumonitis and fibrosis. MA, one of the hydrolytic derivatives of MIC, also caused interstitial pneumonitis progressing to fibrosis, albeit to a lesser extent than MIC, indicating its contribution to the long-term pulmonary damage. The diffuse interstitial pulmonary fibrosis observed at 10 weeks after a single exposure to MIC by either route is of greater significance in the context of the occurrence of pulmonary fibrosis in the late autopsies of Bhopal gas victims and also clinical sequelae in some of the survivors.

3 of 5
Marked Record
TI: Comparative toxicity of methyl isocyanate and its hydrolytic derivatives in rats. I. Pulmonary histopathology in the acute phase.
AU: Jeevaratnam-K; Sriramachari-S
AD: Defence Research and Development Establishment, Gwalior, India.
SO: Arch-Toxicol. 1994; 69(1): 39-44
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: The present study describes the acute histopathological changes induced by methyl isocyanate (MIC) in the lungs of rats at 24 h after a single exposure to varied concentrations/doses of MIC by inhalation and subcutaneous (s.c.) routes and also delineates the effects due to the hydrolytic derivatives of MIC, viz., methylamine (MA) and N,N'-dimethyl urea (DMU). MIC, either inhaled or administered s.c., resulted in a wide range and extent of histopathological changes in the lungs, proportional to the exposure concentration/dose. The salient, effects of inhaled MIC are acute necrotizing bronchitis of the entire

respiratory tract accompanied by varying degrees of confluent congestion, hyperemia and interstitial and intra-alveolar edema, while MIC administered s.c. led to prominent vascular endothelial damage, congestion and severe interstitial pneumonitis with apparently normal bronchial epithelium; and intra-alveolar edema only with the high dose. The only noteworthy lesion produced by MA and DMU (to some extent) was interstitial pneumonitis, suggesting their possible involvement in the subsequent inflammatory response of MIC. Except, for the endothelial changes, the overall spectrum of the histopathological lesions is quite comparable to those observed in the lungs of Bhopal victims during the acute phase.

4 of 5
Marked Record

TI: Chronic lung inflammation in victims of toxic gas leak at Bhopal.
AU: Vijayan-VK; Sankaran-K; Sharma-SK; Misra-NP
AD: Cardio-Pulmonary Medicine Unit, Indian Council of Medical Research, Madras.
SO: Respir-Med. 1995 Feb; 89(2): 105-11
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Bronchoalveolar lavage (BAL) studies in 20 patients at Bhopal, 1.3 +/- 0.4 yr and 2.7 +/- 0.6 yr after toxic gas exposure had revealed that the lower respiratory tract inflammation had progressed from initial macrophage alveolitis to macrophage-neutrophilic alveolitis. The interval between the two lavages was 1.4 +/- 0.6 yr. BAL studies in a new group of 24 patients 5.1 +/- 1.0 yr after exposure had confirmed chronic inflammation of the lower respiratory tract as evidenced by macrophage-neutrophilic alveolitis in these subjects as well. Clinical, radiographic and pulmonary function abnormalities were persistent in a proportion of subjects in both groups. Fibronectin (FN) levels were estimated in BAL fluid in 41 patients. Elevated FN levels were seen in 12 (29.3%) subjects and nine of these 12 had radiographic abnormalities. Severely exposed subjects (n = 30) had significantly higher BAL fibronectin levels compared to normal subjects and mild/moderately exposed subjects. Repeat FN estimations in BAL samples from 10 patients had revealed that five had abnormally high FN including three who had high FN on both occasions. The number of patients showing abnormal decline in pulmonary function was higher in patients with elevated FN than in patients with normal FN. Thus, persisting clinical, roentgenographic and ventilatory abnormalities, as well as macrophage-neutrophilic alveolitis along with abnormally elevated FN levels in a proportion of subjects, suggest the possibility that lung fibrosis can occur in subjects exposed to toxic gas at Bhopal.

5 of 5
Marked Record

TI: The ultimate poison center call--Bhopal [editorial]
AU: Wax-PM
SO: J-Toxicol-Clin-Toxicol. 1995; 33(1): 18
This source is Available only few issues in S.J.M.C. Library
LA: ENGLISH

1 of 2
Marked Record

TI: The Bhopal tragedy [letter]
AU: Murlidhar-V
SO: Natl-Med-J-India. 1994 May-Jun; 7(3): 150-1
This source is Available in S.J.M.C Library
Call Number: From: 1988+
LA: ENGLISH

2 of 2
Marked Record

TI: Isolation of an unknown compound, from both blood of Bhopal aerosol disaster victims and residue of tank E-610 of Union Carbide India Limited--chemical characterization of the structure.
AU: Chandra-H; Saraf-AK; Jadhav-RK; Rao-GJ; Sharma-VK; Sriramachari-S; Vairamani-M
AD: Medicolegal Institute, Gandhi Medical College Building, Bhopal, India.
SO: Med-Sci-Law. 1994 Apr; 34(2): 106-10
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: A total of more than 28 chemical entities/reaction products in the form of gases, vapour and particulate matter were reported from the tank E-610 of methyl isocyanate (MIC) storage tank of Union Carbide India Limited on the night of 2/3 December 1984 in Bhopal. In earlier studies, methyl isocyanate and its trimer, with a few other compounds, were reported in the human victims preserved in deep freeze. Randomly selected samples were analysed by gas chromatograph coupled with mass spectrometer (ITD-800, Finnigan MAT, UK). Four of the cases showed the peaks and fragmentation pattern identified with one of the unidentified compound of molecular weight 269 amu in the Tank Residue, which constituted about 0.2 area per cent on GC-ITD. After isolation by column chromatography and being exposed to characterization, it was identified as a Spiro compound. It was possibly formed by the polymerization of five molecules of methyl isocyanate.

1 of 5
Marked Record

TI: The public health physician's role in chemical incidents.
AU: Gunnell-DJ
AD: Somerset Health Authority, Taunton.
SO: J-Public-Health-Med. 1993 Dec; 15(4): 352-7
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Chemical incidents such as the methylisocyanate release at Bhopal and the aluminium sulphate incident in Lowermoor, Cornwall, are uncommon. However, five chemical incidents occurred in Somerset in 12 months between 1990 and 1991, and District Health Authorities are required to have plans to deal with such events. A survey of the plans held by the Consultants in Communicable Disease Control in South Western and Wessex Regional Health Authorities is discussed and the roles of public health physicians and emergency organizations are outlined.

2 of 5
Marked Record

TI: The Bhopal accident and methyl isocyanate toxicity.
AU: Varma-DR; Guest-I
AD: Department of Pharmacology and Therapeutics, McGill University, Montreal, Canada.
SO: J-Toxicol-Environ-Health. 1993 Dec; 40(4): 513-29
This source is Available only few issues in S.J.M.C. Library
LA: ENGLISH
AB: The Bhopal accident, the world's worst industrial disaster, in which nearly 40 metric tons of methyl isocyanate (MIC) was released from the Union Carbide pesticide plant, occurred nearly 10 yr ago during the night of December 2 and 3, 1984. Over 3000 people residing in areas adjacent to the plant died of pulmonary edema within 3 d of the accident. Follow-up studies revealed pulmonary, ophthalmic, reproductive, immunologic, neurological, and hematologic toxicity among the survivors. Despite high reactivity, MIC can traverse cell membranes and reach distant organs, perhaps as a reversible conjugate with glutathione, which may explain some of the systemic effects of MIC. MIC can be degraded as a result of pyrolysis and interaction with water, but none of the breakdown products can duplicate the toxicity observed in Bhopal and in animal models. MIC may be the most toxic of all isocyanates because of its very high vapor pressure relative to other isocyanates and because of its ability to exert toxic effects on numerous organ systems.

3 of 5
Marked Record

TI: The Bhopal gas disaster: it's not too late for sound epidemiology [editorial]
AU: Dhara-VR; Kriebel-D
SO: Arch-Environ-Health. 1993 Nov-Dec; 48(6): 436-7
This source is Available in S.J.M.C Library
Call Number: From: 1960+
LA: ENGLISH

4 of 5
Marked Record

TI: The epidemiology of disasters and adverse reproductive outcomes: lessons learned.
AU: Cordero-JF
AD: Division of Birth Defects and Developmental Disabilities, Centers for Disease Control, Atlanta, GA 30333.
SO: Environ-Health-Perspect. 1993 Jul; 101 Suppl 2: 131-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: A disaster has been defined as a disruption of human ecology that exceeds the capacity of the community to function normally. Little is known about the adverse effects of natural disasters on reproductive outcomes. Important lessons can be derived from several disasters caused by human factors, such as the Minamata Bay disaster. Adverse reproductive outcomes include infertility, early pregnancy loss, stillbirths, congenital malformations, and serious developmental disabilities such as cerebral palsy and mental retardation. Recent disasters like the Chernobyl and Bhopal explosions have provided important lessons on the need for accurate and sound information about the risk of prenatal exposures for adverse reproductive outcomes. To study questions of adverse reproductive outcomes and disasters requires a well-planned approach. It should include early development of surveillance for adverse reproductive outcomes, analytic studies on the risk of disasters from direct and indirect effects, sensitive methods to measure early pregnancy loss, and long-term follow-up programs to assess outcomes such as developmental disabilities.

5 of 5
Marked Record

TI: Environmental release of chemicals and reproductive ecology.
AU: Bajaj-JS; Misra-A; Rajalakshmi-M; Madan-R
AD: Department of Medicine, All India Institute of Medical Sciences, New Delhi.
SO: Environ-Health-Perspect. 1993 Jul; 101 Suppl 2: 125-30
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Reproductive ecology is defined as "the study of causes and mechanisms of the effects of environmental risk factors on reproductive health and the methods of their prevention and management." Major areas of concern, within the purview of this paper, relate to adverse pregnancy outcomes, effects on target tissues in the male and the female, and alterations in the control and regulatory mechanisms of reproductive processes. Teratogenic potential of chemicals, released as a result of accidents and catastrophes, is of critical significance. Congenital Minamata disease is due to transplacental fetal toxicity caused by accidental ingestion of methyl mercury. Generalized disorders of ectodermal tissue following prenatal exposure to polychlorinated biphenyls have been reported in Taiwan and Japan. The Bhopal gas disaster, a catastrophic industrial accident, was due to a leak of toxic gas, methyl isocyanate (MIC), in the pesticide manufacturing process. The outcome of pregnancy was studied in female survivors of MIC exposure. The spontaneous abortion rate was nearly four times more common in the affected areas as compared to the control area (24.2% versus 5.6%; $p < 0.0001$). Furthermore, while stillbirth rate was found to be similar in the affected and control areas, the perinatal and neonatal mortality rates were observed to be higher in the affected area. The rate of congenital malformations in the affected and control areas did not show any significant difference. Chromosomal aberrations and sister chromatid exchange (SCE) frequencies were investigated in human survivors of exposure. The observed SCE frequencies in control and exposed groups indicated that mutagenesis has been induced. Strategies for the management, prediction, and preventability of such disasters are outlined.

1 of 9
Marked Record

TI: Critical appraisal of entomological data of Madhya Pradesh for 1991 and its relevance to the National Malaria Eradication Programme.
AU: Saxena-VK; Narasimham-MV; Kalra-NL
AD: National Malaria Eradication Programme, Bhopal.
SO: J-Commun-Dis. 1992 Jun; 24(2): 97-108
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1978-1992
LA: ENGLISH
AB: Entomological data generated in five entomological zones, of Madhya Pradesh State during 1991 including, Bhopal, Bilaspur, Gwalior, Indore and Raipur were analyzed. The entomological parameters that were studied included per man hour (pmh) density, abdominal physiology and parity status. The inferences were related to i) resting behaviour (exophily/endophily) ii) duration of indoor resting period of mosquitoes iii) man-vector contact iv) efficacy of residual insecticide and v) vulnerability of the area to focal malaria outbreaks. The data chiefly pertains to the putative malaria vector Anopheles culicifacies in all the five zones under study. The studies have brought out that A. culicifacies, traditionally endophilic and endophagic, has demonstrated radical departure in its resting and feeding behaviour. In Gwalior zone the species shows high preference for exophily. In Bhopal and Indore zones there is differential resting behaviour with respect to season. The species shows, for most part of the year exophily but is also endophilic during post monsoon period. In Bilaspur zone the species shows marked exophily and endophagy. Irrigation practices seem to have affected the mosquito population density patterns in these regions as brought out in Indore and Bilaspur zone, where high density pattern is observed between November and February. These findings have obvious implications in selecting the appropriate intervention methods and the period of spray in areas where residual spray is the method of choice for interruption of transmission.

2 of 9
Marked Record

[redacted]: Health effects of the Bhopal gas leak: a review [published erratum appears in Epidemiol Prev 1992 Dec;14(53):48]
AU: Dhara-R
AD: Dept. of Environmental & Community Medicine UMDNJ, Robert Wood Johnson Medical School, Piscataway 08854.
SO: Epidemiol-Prev. 1992 Sep; 14(52): 22-31
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: The methyl isocyanate (MIC) gas leak from the Union Carbide plant at Bhopal, India in 1984 was the worst industrial disaster in history. Exposure estimates of gas concentrations in the area range from 85 to 0.12 ppm. Of the approximately 200,000 persons exposed, 3598 deaths have resulted as of November 1989. Chronic inflammatory damage to the eyes and lungs appears to be the main cause of morbidity. Reproductive health problems in the form of increased spontaneous abortions and psychological problems have been reported. Questions about the nature of MIC toxicity have been raised by the persistence of multi-systemic symptoms in survivors. Animal studies using radio-labeled MIC given by the inhalation route have shown that the radio-label is capable of crossing the lung membranes and being distributed to many organs of the body.

This paper reviews health effects of gas exposure from published studies and discusses some of the clinical and epidemiological issues being debated.

3 of 9
Marked Record

TI: On the bioavailability of methyl isocyanate in the Bhopal gas leak [editorial]
AU: Dhara-VR
SO: Arch-Environ-Health. 1992 Sep-Oct; 47(5): 385-6
This source is Available in S.J.M.C Library
Call Number: From: 1960+
LA: ENGLISH

4 of 9
Marked Record

TI: Global estimates of acute pesticide morbidity and mortality.
AU: Levine-RS; Doull-J
AD: Department of Family and Preventive Medicine, Meharry Medical College, Nashville, TN 37208.
SO: Rev-Environ-Contam-Toxicol. 1992; 129: 29-50
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Mathematical models have projected increasing numbers of pesticide poisoning throughout the world, rising from 500,000 cases/yr in 1972 to 25,000,000 cases/yr in a 1990 estimate. Among 148 outbreaks (excluding Bhopal and three probable epidemics of pesticide-related suicide) reported between 1951-90, the known number of cases was 24,731 with 1065 deaths (4.3% case fatality); these are probably underestimates. Among the known outbreaks, the most commonly identified agents were organophosphates (58), carbamates (23), chlorinated hydrocarbons (23), and organic mercurials (11). Food was the most common vehicle of exposure in these epidemics (83 outbreaks), followed by skin contact (26), multiple types of exposure (22), and respiratory exposure (16). Two countries, the United States and Thailand, accounted for more than half the reports. Both the mathematical models and the outbreak investigations support the need for continuing investigation and improved surveillance throughout the world.

5 of 9
Marked Record

TI: The industrial safety professionals: a comparative analysis from World War I until the 1980s.
AU: Dwyer-T
AD: IFCH/DCS, Universidade Estadual de Campinas, Sao Paulo, Brazil.
SO: Int-J-Health-Serv. 1992; 22(4): 705-27
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: The birth of industrial society produced demand for the services of professionals specialized in matters related to industrial safety. Three professions--safety engineering, industrial medicine, and ergonomics--are examined. These professions are observed to either submit to single sets of demands, to integrate contradictory demands, or to experience scission. Until the late 1960s their growth appears to have been relatively peaceful and uncontroversial. From this period onward, controversy breaks out over questions related to industrial safety, and professions and government administrations grow. Increasingly, the traditional approach of safety professionals is called into question, and they adopt new orientations. These changes are mapped through the examination of data drawn principally from the United States, France, Great Britain, and to a lesser extent Brazil. The traditional standards approach competes with cost-benefit analysis and with systemic safety for influence; in addition, an emergent approach that analyzes accident causes in terms of social relations of work is detected. From Bhopal to Chernobyl, new

technologies subject civilian populations to risks of catastrophic accidents, and the action of safety professionals comes under the spotlight. The analysis constructed permits new understandings of the past and the future of these professions.

6 of 9
Marked Record

TI: Sequential respiratory, psychologic, and immunologic studies in relation to methyl isocyanate exposure over two years with model development.
AU: Kamat-SR; Patel-MH; Pradhan-PV; Taskar-SP; Vaidya-PR; Kolhatkar-VP; Gopalani-JP; Chandarana-JP; Dalal-N; Naik-M
AD: Department of Respiratory Medicine and Psychiatry, Seth G. S. Medical College, Bombay, India.
SO: Environ-Health-Perspect. 1992 Jul; 97: 241-53
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Of 113 methyl isocyanate (MIC)-exposed subjects studied initially at Bhopal, India, 79, 56, 68, and 87 were followed with clinical, lung function, radiographic, and immunologic tests at 3, 6, 12, 18, and 24 months. Though our cohort consisted of subjects at all ages showing a varied severity of initial illness, fewer females and young subjects were seen. Initially all had eye problems, but dominant symptoms were exertional dyspnea, cough, chest pain, sputum, and muscle weakness. A large number showed persistent depression mixed with anxiety, with disturbances of personality parameters. The early radiographic changes were lung edema, overinflation, enlarged heart, pleural scars, and consolidation. The persistent changes seen were interstitial deposits. Lung functions showed mainly restrictive changes with small airway obstruction; there was impairment of oxygen exchange. Oxygen exchange improved at 3-6 months, and spirometry improved at 12 months, only to decline later. The expiratory flow rates pertaining to large and medium airway function improved, but those for small airways remained low. There were changes of alveolitis in bronchoalveolar lavage fluid on fiber optic bronchoscopy, and in 11 cases positive MIC-specific antibodies to IgM, IgG, and IgE were demonstrated. On follow up, only 48% of the subjects were clinically stable, while 50% showed fluctuations. Thirty-two percent of the subjects had lung function fluctuations. Detailed sequential behavior over 2-4 years was predicted for dyspnea, forced vital capacity, maximum expiratory flow rate (0.25-0.75), peak expiratory flow rate, VO2, and depression score. A model for clinical behavior explained a total variance of 52.4% by using the factors of cough, PCO2 and X-ray zones in addition to above five parameters. The behavior of the railway colony group (1640 patients) revealed a similar pattern of illness. When this observed pattern of changes was transferred to the affected Bhopal city sections (with an equitable age-sex distribution), our model results were again validated. Thus the picture of MIC-induced disease seems similar despite the differences for age-sex and initial severity of illness in our cohort and in the population of Bhopal city as predicted by our model.

7 of 9
Marked Record

TI: Toxicity of the methyl isocyanate metabolite S-(N-methylcarbamoyl)GSH on mouse embryos in culture.
AU: Guest-I; Baillie-TA; Varma-DR
AD: Department of Pharmacology and Therapeutics, McGill University, Montreal, Quebec, Canada.
SO: Teratology. 1992 Jul; 46(1): 61-7
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Methyl isocyanate, the chemical involved in the 1984 accident at Bhopal, India, forms a labile conjugate, S-(N-methylcarbamoyl)GSH (SMG), by way of a reversible reaction with GSH. We studied the toxicity of SMG on mouse embryos explanted on day 8 of gestation and cultured in rat serum for 42 hr. SMG caused

concentration-dependent decreases in growth and development over the range 0.1-2 mM, without causing significant mortality. At a concentration of 2 mM, SMG completely arrested embryo development, but heartbeat was absent in only one of nine embryos at 42 hr. At a concentration of 0.25 mM, SMG reduced embryo size to 75% and protein content to 63% of the control; 18% of embryos failed to rotate. At this concentration (0.25 mM), which was selected for all other studies, spinal kinks and somite pair distortion in the region of the forelimb were evident in 38% of embryos; no other abnormalities were noted. DNA content of and thymidine incorporation by embryos and yolk sacs was reduced by SMG, although this was more pronounced in the yolk sac than in embryos. At subtoxic concentrations, the L-cysteine precursor (-)-2-oxo-4-thiazolidine-carboxylic acid did not, but GSH did, inhibit embryotoxicity of SMG. It is concluded that SMG exerts embryotoxic and dysmorphogenic effects and may contribute to systemic toxicity of methyl isocyanate.

8 of 9
Marked Record

TI: Trojan horse. Did a protective peptide exacerbate Bhopal injuries? [news]
AU: Rennie-J
SO: Sci-Am. 1992 Mar; 266(3): 15-6
This source is Available in S.J.M.C Library
Call Number: From: 1949+
LA: ENGLISH

9 of 9
Marked Record

TI: Accident analysis of large-scale technological disasters applied to an anaesthetic complication [see comments]
AU: Eagle-CJ; Davies-JM; Reason-J
AD: Department of Anaesthesia, Foothills Hospital, University of Calgary, Alberta, Canada.
SO: Can-J-Anaesth. 1992 Feb; 39(2): 118-22
This source is Available in S.J.M.C Library
Call Number: From: 1966+
LA: ENGLISH
AB: The occurrence of serious accidents in complex industrial systems such as at Three Mile Island and Bhopal has prompted development of new models of causation and investigation of disasters. These analytical models have potential relevance in anaesthesia. We therefore applied one of the previously described systems to the investigation of an anaesthetic accident. The model chosen describes two kinds of failures, both of which must be sought. The first group, active failures, consists of mistakes made by practitioners in the provision of care. The second group, latent failures, represents flaws in the administrative and productive system. The model emphasizes the search for latent failures and shows that prevention of active failures alone is insufficient to avoid further accidents if latent failures persist unchanged. These key features and the utility of this model are illustrated by application to a case of aspiration of gastric contents. While four active failures were recognized, an equal number of latent failures also became apparent. The identification of both types of failures permitted the formulation of recommendations to avoid further occurrences. Thus this model of accident causation can provide a useful mechanism to investigate and possibly prevent anaesthetic accidents.

1 of 10

TI: Fetal loss and contraceptive acceptance among the Bhopal gas victims.
AU: Kapoor-R
AD: International Institute for Population Sciences, Bombay, India.
SO: Soc-Biol. 1991 Fall-Winter; 38(3-4): 242-8
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: The rates of fetal loss and family planning acceptance among Bhopal gas victims from 1984 to 1989 were compared to those of a control group. In all, 136 eligible women in the affected area and 139 women in the control area were interviewed. Care was taken to ensure that these women had conceived at least once during the previous five years. The The fetal loss rate among the gas-affected women was abnormally high (26.3 per cent) compared to that of women in the control area (7.8 per cent). Family planning acceptance in both areas was similar, with most women using permanent methods. In the case of temporary methods, the percentage of use was higher in the gas-affected area.

2 of 10

TI: GC-MS identification of MIC trimer: a constituent of tank residue in preserved autopsy blood of Bhopal gas victims.
AU: Chandra-H; Rao-GJ; Saraf-AK; Sharma-VK; Jadhav-RK; Sriramachari-S
AD: Medicolegal Institute, Bhopal, India.
SO: Med-Sci-Law. 1991 Oct; 31(4): 294-8
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Based on the external and internal findings of Bhopal gas disaster victims, it was apparent that the gases and particulate matter came out as an aerosol. This was possibly the pyrolysed, reformulated, reconjugated suspension of constituents of the tank E-610 of Union Carbide India Limited, Bhopal, while it was claimed to be methyl isocyanate (MIC) only. It was postulated by the manufacturer of MIC, that the material inhaled by the victims of the Bhopal gas disaster does not cross the lung barrier (UCC press conference on 14th December 1984). It was observed that the more the victims ran, the more aerosol they inhaled and the fatalities were observed in such victims. The tissues, which were preserved in the deep freeze, were randomly selected and analysed by GC coupled with MS (ITD) Finnigan MAT, UK. 14 out of 34 autopsy cases showed MIC trimer peak in extracts of blood. This was one of the constituents of the aerosol and was also located in the tank residue, thereby proving that the trimer had passed the lung barrier.

3 of 10

TI: GC-NPD and GC-MS analysis of preserved tissue of Bhopal gas disaster: evidence of methyl carbamylation in post-mortem blood.
AU: Sriramachari-S; Rao-GJ; Sharma-VK; Jadhav-RK; Saraf-AK; Chandra-H
AD: Institute of Pathology (Indian Council of Medical Research), Safdarjung Hospital, New Delhi.
SO: Med-Sci-Law. 1991 Oct; 31(4): 289-93
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Twenty-five preserved autopsy blood samples of Bhopal toxic gas exposed victims were analysed by gas chromatography (GC) coupled with either Nitrogen-Phosphorous detector (NPD) or mass spectrometer (MS) for the presence of methyl carbamyl valine in terms of valine methyl hydantoin (VMH). 84% of

these samples showed a positive test for VMH on GC-NPD and the identity of the peaks were further confirmed on GC-MS. The concentration of VMH in the gas-affected positive blood samples ranged from 2.56 to 51.28 nanomoles. These results indicate entry of methyl isocyanate (MIC), one of the constituents of the toxic cloud caused by the disaster, into the blood stream of victims who had inhaled gas.

4 of 10

TI: Industrialization and emerging environmental health issues: lessons from the Bhopal disaster.
AU: Murti-CR
AD: Madras Science Foundation, India.
SO: Toxicol-Ind-Health. 1991 Sep-Nov; 7(5-6): 153-64
this source is not Available in S.J.M.C.Library
LA: ENGLISH

5 of 10

TI: Objective thoracic CT scan findings in a Bhopal gas disaster victim.
AU: Sharma-S; Narayanan-PS; Sriramachari-S; Vijayan-VK; Kamat-SR; Chandra-H
AD: Department of Radiology, G.B. Pant Hospital, New Delhi, India.
SO: Respir-Med. 1991 Nov; 85(6): 539-41
this source is not Available in S.J.M.C.Library
LA: ENGLISH

6 of 10

TI: Mass deaths by gas or chemical poisoning. A historical perspective.
AU: Eckert-WG
AD: Milton Helpern International Center of Forensic Sciences, Wichita State University, Kansas.
SO: Am-J-Forensic-Med-Pathol. 1991 Jun; 12(2): 119-25
this source is not Available in S.J.M.C.Library
Call Number: From 1980+
LA: ENGLISH
AB: This review chronicles the characteristics of deliberate and accidental mass poisonings that occurred in World Wars I and II, in Bhopal, and in other historical cases up to and including modern wars. It also considers approaches to the investigation of such cases from the medicolegal as well as general standpoints.

7 of 10

TI: Technological disasters--towards a preventive strategy: a review.
AU: Andersson-N
AD: Centre for Tropical Disease Research (CIET), Faculty of Medicine, Universidad Autonoma de Guerrero, Acapulco, Mexico.
SO: Trop-Doct. 1991; 21 Suppl 1: 70-81
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1971-1992
LA: ENGLISH
AB: Technological or man-made disasters are a growth industry. Widely publicized industrial disasters like those in Bhopal and Chernobyl are only the tip of the iceberg of human and environmental risk from technological development. Other less well publicized disasters, including the contamination of food, water and air, have affected millions of people. The 'slow' technological disasters - like air pollution, pesticides, radiation, lead, asbestos and other industrial hazards - also compromise human intellectual, behavioural and physical development. Although it can be argued that there are hazards attached to virtually every industrial activity and that it is almost impossible to remove completely the risk of technological disasters, it is possible to reduce this risk by decentralizing or deconcentrating knowledge on technological processes. Global recommendations may provide a framework for priority action, but they are obviously not applicable everywhere with the same

intensity. A measurement-based approach is described that is beginning to have an effect in several developing countries.

8 of 10

TI: Cytogenetic studies on MIC gas-exposed persons in Bhopal [letter; comment]
AU: Das-BC
SO: Hum-Genet. 1991 Aug; 87(4): 513-5
this source is not Available in S.J.M.C.Library
LA: ENGLISH

9 of 10

TI: An overview of process hazard evaluation techniques.
AU: Gressel-MG; Gideon-JA
AD: National Institute for Occupational Safety and Health, Division of Physical Sciences and Engineering, Cincinnati, OH 45226.
SO: Am-Ind-Hyg-Assoc-J. 1991 Apr; 52(4): 158-63
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1970-1975
LA: ENGLISH
AB: Since the 1985 release of methyl isocyanate in Bhopal, India, which killed thousands, the chemical industry has begun to use process hazard analysis techniques more widely to protect the public from catastrophic chemical releases. These techniques can provide a systematic method for evaluating a system design to ensure that it operates as intended, help identify process areas that may result in the release of a hazardous chemical, and help suggest modifications to improve process safety. Eight different techniques are discussed, with some simple examples of how they might be applied. These techniques include checklists, "what if" analysis, safety audits and reviews, preliminary hazard analysis (PHA), failure modes and effect analysis (FMEA), fault tree analysis (FTA), event tree analysis (ETA), and hazard and operability studies (HAZOP). The techniques vary in sophistication and scope, and no single one will always be the best. These techniques can also provide the industrial hygienist with the tools needed to protect both workers and the community from both major and small-scale chemical releases. A typical industrial hygiene evaluation of a facility would normally include air sampling. If the air sampling does detect a specific hazardous substance, the source will probably be a routine or continuous emission. However, air sampling will not be able to identify or predict the location of a nonroutine emission reliably. By incorporating these techniques with typical evaluations, however, industrial hygienists can proactively help reduce the hazards to the workers they serve.

10 of 10

TI: Developmental toxicity of methylamines in mice.
AU: Guest-I; Varma-DR
AD: Department of Pharmacology and Therapeutics, McGill University, Montreal, Canada.
SO: J-Toxicol-Environ-Health. 1991 Mar; 32(3): 319-30
This source is Available only few issues in S.J.M.C. Library
LA: ENGLISH
AB: Monomethylamine (MMA), dimethylamine (DMA), and trimethylamine (TMA) are endogenous substances as well as metabolites of methyl isocyanate, the chemical involved in the 1984 accident at Bhopal, India. Although methylamines exert several toxic effects including inhibition of protein turnover and oocyte RNA synthesis, their reproductive toxicity has not been investigated. We therefore studied the possible developmental toxicity of these amines using pregnant CD-1 mice and mouse embryo culture as experimental models. Intraperitoneal injections (daily from d 1 to 17 of gestation) of TMA at 2.5 and 5 mmol/kg/d significantly (p less than .001) decreased fetal body weight but not the placental weight or maternal body weight gain; however, 5 of 11 mice treated with 5 mmol/kg TMA died. Similar treatment with DMA and MMA did not exert any

obvious maternal or fetal effects. All three methylamines, when added to embryos in culture, caused dose-dependent decreases in size, DNA, RNA, and protein content as well as embryo survival; the order of toxicity was TMA greater than DNA greater than MMA. The ability of methylamines to adversely affect fetal development suggests that these amines, especially trimethylamine, may act as endogenous teratogens under certain conditions.

PC-SPIRS 3.40 MEDLINE (R) 1990

1 of 12
Marked Record

TI: Industry's voluntary program: Community Awareness and Emergency Response Program and the Emergency Planning and Community Right-to-Know Act.
AU: Cooper-JS
AD: Environmental Policy Group, Hill and Knowlton Public Affairs Worldwide, Washington, D.C. 20007.
SO: Toxicol-Ind-Health. 1990 Oct; 6(5): 13-21
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: This paper describes the chemical industry's Community Awareness and Emergency Response (CAER) Program, and voluntary and mandatory actions by the chemical industry to comply with the major environmental legislation. The chemical industry started the voluntary CAER Program soon after the Bhopal Disaster in 1984; it is coordinated through the Chemical Manufacturer's Association. This program, which began in March 1985, is a long-term industry commitment to develop a community outreach program and to improve local emergency response planning. The Congress of the United States began, in 1985, to consider proposals for mandatory programs. This led to enactment of the Superfund Amendments and Reauthorization Act of 1986, known as SARA. A portion of this Act, entitled Title III is also known as the Emergency Planning and Community Right-to-Know Act. Although this legislation has many mandatory requirements, it should be emphasized that a significant degree of voluntary industrial participation is needed if the purposes of the statute are to be achieved. Title III has created an intricate and still evolving system that ties together the EPA, industrial plant managers, state emergency response commissions, local emergency planning committees and fire departments with jurisdiction over the facility. Each of these groups has a different role and responsibilities but must work cooperatively with other participants. Because of the intricate network of participants, the magnitude of the information flow, and the continuing evolution of the system, unique public relations problems exist in order to comply with Title III.(ABSTRACT TRUNCATED AT 250 WORDS)

2 of 12
Marked Record

TI: Air toxics in the U.S.: magnitude of the problem and strategy for control.
AU: Berry-DK
AD: Air Quality Management Division, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711.
SO: Toxicol-Ind-Health. 1990 Oct; 6(5): 1-12
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Over the past several years, substantial concern has been expressed by some in Congress, environmental groups, and members of the public concerning the lack of progress in regulating toxic air pollutants by the U.S. Environmental Protection Agency (EPA). As a result, a number of amendments to the Federal Clean Air Act have been introduced to require EPA to regulate in a relatively rapid timeframe, a large number of potentially toxic pollutants that are released to the ambient air. This paper discusses EPA's current understanding of the magnitude and nature of the air toxics problem in the U.S., and the pollutants and source categories that pose the most significant risk to the public. The focus of the discussion is on routine releases, as opposed to

catastrophic, accidental releases such as the one in Bhopal, India. The paper then discusses the strategy that EPA has put in place to deal with the problem and presents the status of a number of regulatory and non-regulatory activities under way to better understand the problem and to mitigate it. The strategy involves important roles for: (1) EPA to regulate national problems using a variety of Federal authorities in addition to the Clean Air Act, and (2) States to develop their own air toxic control programs to deal with unique local problems involving high risk point sources and multipollutant, multisource problems in large urban or industrialized areas.

3 of 12
Marked Record

TI: The current practice of health risk assessment: potential impact on standards for toxic air contaminants.
AU: Paustenbach-DJ; Jernigan-JD; Finley-BL; Ripple-SR; Keenan-RE
AD: ChemRisk, Division of McLaren/Hart Environmental Engineering Corp., Alameda, California.
SO: J-Air-Waste-Manage-Assoc. 1990 Dec; 40(12): 1620-30
___this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Since the Bhopal incident, the public has placed pressure on regulatory agencies to set community exposure limits for the dozens of chemicals that may be released by manufacturing facilities. More or less objective limits can be established for the vast majority of these chemicals through the use of risk assessment. However, each step of the risk assessment process (i.e., hazard identification, dose-response assessment, exposure assessment, and risk characterization) contains a number of pitfalls that scientists need to avoid to ensure that valid limits are established. For example, in the hazard identification step there has been little discrimination among animal carcinogens with respect to mechanism of action or the epidemiology experience. In the dose-response portion, rarely is the range of "plausible" estimated risks presented. Physiologically based pharmacokinetic (PB-PK) models should be used to understand the difference between the tissue doses and the administered dose, as well as the difference in target tissue concentrations of the toxicant between rodents and humans. Biologically-based models like the Moolgavkar-Knudson-Venzon (MKV) should be developed and used, when appropriate. The exposure assessment step can be significantly improved by using more sensitive and specific sampling and analytical methods, more accurate exposure parameters, and computer models that can account for complex environmental factors. Whenever possible, model predictions of exposure and uptake should be validated by biological monitoring of exposed persons (urine, blood, adipose) or by field measurements of plants, soil, fish, air, or water. In each portion of an assessment, the weight of evidence approach should be used to identify the most defensible value. In the risk characterization, the best estimate of the potential risk as well as the highest plausible risk should be presented. Future assessments would be much improved if quantitative uncertainty analyses were conducted. Procedures are currently available for making future assessments. By correcting some of these shortcomings in how health risk assessments have been conducted, scientists and risk managers should be better able to identify scientifically appropriate ambient air standards and emission limits.

4 of 12
Marked Record

TI: Industry's voluntary program: Community Awareness and Emergency Response Program and the Emergency Planning and Community Right-to-Know Act.
AU: Cooper-JS
AD: Environmental Policy Group, Hill and Knowlton Public Affairs Worldwide, Washington, D.C. 20007.
SO: Toxicol-Ind-Health. 1990 Oct; 6(5): 13-21
___this source is not Available in S.J.M.C.Library

LA: ENGLISH
AB: This paper describes the chemical industry's Community Awareness and Emergency Response (CAER) Program, and voluntary and mandatory actions by the chemical industry to comply with the major environmental legislation. The chemical industry started the voluntary CAER Program soon after the Bhopal Disaster in 1984; it is coordinated through the Chemical Manufacturer's Association. This program, which began in March 1985, is a long-term industry commitment to develop a community outreach program and to improve local emergency response planning. The Congress of the United States began, in 1985, to consider proposals for mandatory programs. This led to enactment of the Superfund Amendments and Reauthorization Act of 1986, known as SARA. A portion of this Act, entitled Title III is also known as the Emergency Planning and Community Right-to-Know Act. Although this legislation has many mandatory requirements, it should be emphasized that a significant degree of voluntary industrial participation is needed if the purposes of the statute are to be achieved. Title III has created an intricate and still evolving system that ties together the EPA, industrial plant managers, state emergency response commissions, local emergency planning committees and fire departments with jurisdiction over the facility. Each of these groups has a different role and responsibilities but must work cooperatively with other participants. Because of the intricate network of participants, the magnitude of the information flow, and the continuing evolution of the system, unique public relations problems exist in order to comply with Title III.(ABSTRACT TRUNCATED AT 250 WORDS)

5 of 12
Marked Record
TI: Public health lessons from the Bhopal chemical disaster [published erratum appears in JAMA 1991 Feb 20;265(7):869] [comment]
AU: Koplan-JP; Falk-H; Green-G
AD: Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Atlanta, GA 30333.
SO: JAMA. 1990 Dec 5; 264(21): 2795-6
This source is Available in S.J.M.C Library
Call Number: From: 1918+
LA: ENGLISH

6 of 12
Marked Record
TI: Bhopal tragedy's health effects. A review of methyl isocyanate toxicity [see comments]
AU: Mehta-PS; Mehta-AS; Mehta-SJ; Makhijani-AB
AD: Humana Hospital, Aurora, Colo.
SO: JAMA. 1990 Dec 5; 264(21): 2781-7
This source is Available in S.J.M.C Library
Call Number: From: 1918+
LA: ENGLISH

7 of 12
Marked Record
TI: Effects of methyl isocyanate on rat brain cells in culture.
AU: Anderson-D; Goyle-S; Phillips-BJ; Tee-A; Beech-L; Butler-WH
AD: British Industrial Biological Research Association, Carshalton, Surrey.
SO: Br-J-Ind-Med. 1990 Sep; 47(9): 596-601
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1991
LA: ENGLISH
AB: Since the disaster in Bhopal, India, people exposed to methyl isocyanate (MIC) have complained of various disorders including neuromuscular dysfunction. In an attempt to get information about such dysfunction we have previously shown that MIC can affect muscle cells in culture. The present communication

reports investigations into the effect of MIC on brain cells in culture. MIC was toxic to brain cells and the response was dose related. The observations were supported by light and electron microscopy.

8 of 12
Marked Record

TI: Cytogenetic studies in human populations exposed to gas leak at Bhopal, India.
AU: Ghosh-BB; Sengupta-S; Roy-A; Maity-S; Ghosh-S; Talukder-G; Sharma-A
AD: Human Genetics Unit, Center for Advanced Study in Cell and Chromosome Research, Calcutta, India.
SO: Environ-Health-Perspect. 1990 Jun; 86: 323-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Frequencies of chromosomal abnormalities, sister chromatid exchanges, and replicative index were assessed following peripheral lymphocyte culture in 129 individuals from Bhopal, India. Of these, 83 persons (40 male and 43 female) had been exposed directly to the methyl isocyanate (MIC) gas after the accident at the Union Carbide plant on December 2 and 3, 1984. The remaining 46 samples were taken from age-matched unexposed persons in the same city. Chromosome aberrations were recorded at first cycle metaphase (M1) and sister chromatid exchanges, at second cycle metaphase (M2), following standard schedule. The frequency of chromosomal aberrations was, in general, higher in individuals from the exposed populations, with the females showing a higher incidence. Nondisjunction of chromosomes or laggard was rare. The frequencies of sister chromatid exchanges and depression in mitotic and replicative indices could not be related to exposure or sex. The persistence of chromosomal abnormalities in the form of replicating minutes and exchange configurations, even 1114 days after exposure to the gas, may indicate a residual effect on T-cell precursors.

9 of 12
Marked Record

TI: Delayed eye and other consequences from exposure to methyl isocyanate: 93% follow up of exposed and unexposed cohorts in Bhopal.
AU: Andersson-N; Ajwani-MK; Mahashabde-S; Tiwari-MK; Muir-MK; Mehra-V; Ashiru-K; Mackenzie-CD
AD: TUC Centenary Institute of Tropical Medicine, London School of Hygiene & Tropical Medicine.
SO: Br-J-Ind-Med. 1990 Aug; 47(8): 553-8
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1991
LA: ENGLISH
AB: A follow up study three years after exposure to methyl isocyanate in 93% of exposed survivors and "control" residents in 10 Bhopali communities showed an excess of eye irritation, eyelid infection, cataract, and a decrease in visual acuity among the exposed people. Breathlessness was twice as common in the heavily exposed clusters as those with lower exposure, a trend that could not be explained by different age or smoking patterns (OR 2.05, 95% CI 1.36-3.08). Case referent analysis of outpatient attendances at Bhopal Eye Hospital, considering patients with severe refractive errors and astigmatism as "controls," showed a 40% increased risk of trachoma, 36% increased risk of other lid infections, and 45% increased risk of irritant symptoms among previously exposed people. "Bhopal eye syndrome" may thus include full resolution of the initial interpalpebral superficial erosion, a subsequent increased risk of eye infections, hyperresponsive phenomena (irritation, watering, and phlyctens), and possibly cataracts. It remains to be confirmed whether this reflects a more generalised disease as a consequence of previous exposure to methyl isocyanate or whether it is only the eye that is affected.

10 of 12
Marked Record

7 of 11
Marked Record

TI: Early observations on pulmonary changes and clinical morbidity due to the isocyanate gas leak at Bhopal.
AU: Kamat-SR; Mahashur-AA; Tiwari-AK; Potdar-PV; Gaur-M; Kolhatkar-VP; Vaidya-P; Parmar-D; Rupwate-R; Chatterjee-TS; et-al
SO: J-Postgrad-Med. 1985 Apr; 31(2): 63-72
This source is Available only few issues in S.J.M.C. Library
Call Number: from 1955-1985
LA: ENGLISH

8 of 11
Marked Record

TI: The Bhopal tragedy.
AU: Das-JJ
SO: J-Indian-Med-Assoc. 1985 Feb; 83(2): 72-4
this source is not Available in S.J.M.C.Library
LA: ENGLISH

9 of 11
Marked Record

TI: American personal-injury lawyers at Bhopal. Ethics and public policy in mass disaster.
AU: Curran-WJ
SO: N-Engl-J-Med. 1985 Oct 24; 313(17): 1068-70
This source is Available in S.J.M.C Library
Call Number: From: 1945+
LA: ENGLISH

10 of 11
Marked Record

TI: Bhopal tragedy focuses on changes in chemical industry.
AU: Aydelotte-C
SO: Occup-Health-Saf. 1985 Mar; 54(3): 33-5, 50, 59
this source is not Available in S.J.M.C.Library
LA: ENGLISH

11 of 11
Marked Record

TI: Bhopal tragedy's repercussions may reach American physicians [news]
AU: Marwick-C
SO: JAMA. 1985 Apr 12; 253(14): 2001-3
This source is Available in S.J.M.C Library
Call Number: From: 1918+
LA: ENGLISH

PC-SPIRS 3.40 MEDLINE (R) 1984

1 of 1
Marked Record

TI: Calamity at Bhopal [editorial]
SO: Lancet. 1984 Dec 15; 2(8416): 1378-9
This source is Available in S.J.M.C Library
Call Number: From: 1930+
LA: ENGLISH

1 of 11
Marked Record

TI: Bhopal disaster: eye follow-up and analytical chemistry [letter]
AU: Andersson-N; Kerr-Muir-M; Salmon-AG; Wells-CJ; Brown-RB; Purnell-CJ; Mittal-PC; Mehra-V
SO: Lancet. 1985 Mar 30; 1(8431): 761-2
This source is Available in S.J.M.C Library
Call Number: From: 1930+
LA: ENGLISH

2 of 11
Marked Record

TI: An eyewitness in Bhopal.
AU: Sutcliffe-M
SO: Br-Med-J-Clin-Res-Ed. 1985 Jun 22; 290(6485): 1883-4
this source is not Available in S.J.M.C.Library
LA: ENGLISH

3 of 11
Marked Record

TI: Chronobiologic considerations of the Bhopal methyl isocyanate disaster [letter]
AU: Reinberg-A; Smolensky-MH
SO: Chronobiol-Int. 1985; 2(1): 61-2
this source is not Available in S.J.M.C.Library
LA: ENGLISH

4 of 11
Marked Record

TI: Bhopal tragedy--a year later [editorial]
AU: Sainani-GS; Joshi-VR; Mehta-PJ; Abraham-P
SO: J-Assoc-Physicians-India. 1985 Dec; 33(12): 755-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH

5 of 11
Marked Record

TI: Aftermath of Bhopal tragedy [editorial]
AU: Das-JJ
SO: J-Indian-Med-Assoc. 1985 Oct; 83(10): 361-2
this source is not Available in S.J.M.C.Library
LA: ENGLISH

6 of 11
Marked Record

TI: Ocular lesions following methyl isocyanate contamination: the Bhopal experience [letter]
AU: Dwivedi-PC; Raizada-JK; Saini-VK; Mittal-PC
SO: Arch-Ophthalmol. 1985 Nov; 103(11): 1627
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

This source is Available only few issues in S.J.M.C. Library
Call Number: from 1955-1985
LA: ENGLISH

13 of 17
Marked Record

TI: The Bhopal tragedy--what has Swedish disaster medicine planning learned from it?
AU: Lorin-HG; Kulling-PE
SO: J-Emerg-Med. 1986; 4(4): 311-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: On December 3, 1984, a leak of methylisocyanate (MIC) from a chemical plant in Bhopal, India, affected 150,000 to 200,000 people. More than 10,000 people were severely injured and approximately 2,500 died. In this article a survey of symptoms, treatment, and rescue work is given. On the basis of this, we discuss ways to help reduce the effects of a major release of an irritant gas. People living in the vicinity of potential health hazards need information on how to behave in case of accidents. Rescue workers and medical personnel must be trained to operate under "toxic conditions." There must be planning for treatment of thousands of patients at the same time, a circumstance that will often require temporary "satellite hospitals" to be opened. As symptoms and injuries are of the same kind, even if the magnitude and the effect may differ, treatment can, in many ways, be standardized. Therefore members of the health care team, irrespective of their daily different specialty fields, can work with the most urgent missions.

14 of 17
Marked Record

TI: Bhopal tragedy--a year later [letter]
AU: Misra-NP
SO: J-Assoc-Physicians-India. 1986 Apr; 34(4): 307
this source is not Available in S.J.M.C.Library
LA: ENGLISH

15 of 17
Marked Record

TI: Bright red blood of Bhopal victims: cyanide or MIC? [letter]
AU: Salmon-AG
SO: Br-J-Ind-Med. 1986 Jul; 43(7): 502
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1991
LA: ENGLISH

16 of 17
Marked Record

TI: Bhopal and after [editorial]
AU: Zaidi-SH
SO: Am-J-Ind-Med. 1986; 9(3): 215-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH

17 of 17
Marked Record

TI: From Flixborough to Bhopal: is legislation enough? [editorial]
AU: Baxter-PJ
SO: Br-J-Ind-Med. 1986 Jan; 43(1): 1-3
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1991
LA: ENGLISH

AU: Maskati-QB
SO: J-Postgrad-Med. 1986 Oct; 32(4): 199-202
This source is Available only few issues in S.J.M.C. Library
Call Number: from 1955-1985
LA: ENGLISH

7 of 17
Marked Record

TI: Sequential study of thiocyanate levels in Bhopal water following methyl isocyanate gas leakage.
AU: Acharya-VN; Naik-SR; Potnis-AV; Bhalerao-RA
SO: J-Postgrad-Med. 1986 Oct; 32(4): 192-4
This source is Available only few issues in S.J.M.C. Library
Call Number: from 1955-1985
LA: ENGLISH

8 of 17
Marked Record

TI: Medico-legal aspects of the Bhopal tragedy.
AU: Parikh-CK
SO: Leg-Med. 1986: 28-39
this source is not Available in S.J.M.C.Library
LA: ENGLISH

9 of 17
Marked Record

TI: Gynaecological and obstetrical survey of Bhopal women following exposure to methyl isocyanate.
AU: Shilotri-NP; Raval-MY; Hinduja-IN
SO: J-Postgrad-Med. 1986 Oct; 32(4): 203-5
This source is Available only few issues in S.J.M.C. Library
Call Number: from 1955-1985
LA: ENGLISH

10 of 17
Marked Record

TI: A survey of Bhopal children affected by methyl isocyanate gas.
AU: Irani-SF; Mahashur-AA
SO: J-Postgrad-Med. 1986 Oct; 32(4): 195-8
This source is Available only few issues in S.J.M.C. Library
Call Number: from 1955-1985
LA: ENGLISH

11 of 17
Marked Record

TI: Medical survey of methyl isocyanate gas affected population of Bhopal. Part II. Pulmonary effects in Bhopal victims as seen 15 weeks after M.I.C. exposure.
AU: Naik-SR; Acharya-VN; Bhalerao-RA; Kowli-SS; Nazareth-H; Mahashur-AA; Shah-S; Potnis-AV; Mehta-AC
SO: J-Postgrad-Med. 1986 Oct; 32(4): 185-91
This source is Available only few issues in S.J.M.C. Library
Call Number: from 1955-1985
LA: ENGLISH

12 of 17
Marked Record

TI: Medical survey of methyl isocyanate gas affected population of Bhopal. Part I. General medical observations 15 weeks following exposure.
AU: Naik-SR; Acharya-VN; Bhalerao-RA; Kowli-SS; Nazareth-H; Mahashur-AA; Shah-S; Potnis-AV; Mehta-AC
SO: J-Postgrad-Med. 1986 Oct; 32(4): 175-84

1 of 17
Marked Record

TI: Persistent eye watering among Bhopal survivors [letter]
AU: Andersson-N; Kerr-Muir-M; Ajwani-MK; Mahashabde-S; Salmon-A; Vaidyanathan-K
SO: Lancet. 1986 Nov 15; 2(8516): 1152
This source is Available in S.J.M.C Library
Call Number: From: 1930+
LA: ENGLISH

2 of 17
Marked Record

TI: Ophthalmic survey of Bhopal victims--100 days after the tragedy.
AU: Maskati-QB
SO: Indian-J-Ophthalmol. 1986; 34: 328-31
This source is Available in S.J.M.C Library
Call Number: From: 1973+
LA: ENGLISH

3 of 17
Marked Record

TI: Acute ocular lesions in Bhopal gas tragedy.
AU: Raizada-JK; Dwivedi-PC
SO: Indian-J-Ophthalmol. 1986; 34: 324-7
This source is Available in S.J.M.C Library
Call Number: From: 1973+
LA: ENGLISH

4 of 17
Marked Record

TI: Bhopal: a bibliography.
AU: Mac-Sheoin-T
SO: Int-J-Health-Serv. 1986; 16(3): 441-68
this source is not Available in S.J.M.C.Library
LA: ENGLISH

5 of 17
Marked Record

TI: Toxic chemical disasters and the implications of Bhopal for technology transfer.
AU: Weiss-B; Clarkson-TW
SO: Milbank-Q. 1986; 64(2): 216-40
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: The dramatic disaster in 1984 at Bhopal, India, may be overshadowed in total impact by less immediate health effects characterized by long latency, cumulative damage, and subtle impairments. Transfer of chemical technology must be accompanied by transfer of the corresponding infratechnology, toxicology, only then can the process of technology transfer be managed with fewer risks, fewer costs, and fewer tragic surprises.

6 of 17
Marked Record

TI: Ophthalmic survey of Bhopal victims 104 days after the tragedy.

TI: Penetration of methyl isocyanate through organic vapor and acid gas respirator cartridges.
AU: Moyer-ES; Berardinelli-SP
SO: Am-Ind-Hyg-Assoc-J. 1987 Apr; 48(4): 315-23
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1970-1975
LA: ENGLISH
AB: Methyl isocyanate (MIC) is a volatile, toxic chemical [Threshold Limit Value (TLV) = 0.02 ppm] used to manufacture carbamate pesticides. The principal manufacturer of MIC is Union Carbide, and the site of production is Institute, West Virginia. In light of the December 1984 Bhopal, India disaster and possible safety problems at the Institute facility, NIOSH conducted this research as a basis upon which to recommend protective equipment that might be used in an emergency situation where extremely high MIC concentrations might be encountered. Both protective clothing and respirators were evaluated. In particular, NIOSH studied air-purifying respirators in order to assess their effectiveness against MIC vapor penetration. NIOSH does not recommend any air purifying respirator for MIC because of its high toxicity and lack of warning properties and because no effective end of service life indicator currently is available for MIC. This report addresses only MIC penetration through air-purifying cartridges at challenge concentrations designed to simulate emergency escape conditions. Another report addresses the protective clothing issue. The results presented are for two different manufacturers' organic vapor (OV) and acid gas cartridges. Penetration tests were conducted at three or four MIC challenge concentrations and at three different humidity conditions. In general, breakthrough times (1% of challenge concentration) were very short (less than 20 min). Also, high relative humidity was found to decrease the breakthrough time of MIC.

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Marked Record

TI: Bright red blood of Bhopal victims? [letter]
AU: Nemery-B
SO: Br-J-Ind-Med. 1987 Apr; 44(4): 287
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1991
LA: ENGLISH

stillbirths, infant mortality, and fetal abnormalities, no clinical or experimental studies on the reproductive toxicity of MIC were reported in scientific journals for several months after the accident. We therefore conducted, 9 months after the accident, a preliminary survey of 3270 families in Bhopal and experimental studies on the effects of MIC in pregnant mice. It was found that 43% of pregnancies in women residing near the Union Carbide pesticide plant did not result in the birth of a live child. Likewise, exposure of mice to relatively low concentrations of MIC (9 and 15 ppm) for 3 hr caused complete resorption in more than 75% of animals. A decrease in fetal and placental weights was observed at 2 to 15 ppm MIC. In general, the experimental findings in mice corroborate the epidemiological data from Bhopal. The mechanism of the fetal toxicity of MIC remains to be established.

25 of 29
Marked Record

TI: Biological effects of short-term, high-concentration exposure to methyl isocyanate. I. Study objectives and inhalation exposure design.
AU: Dodd-DE; Frank-FR; Fowler-EH; Troup-CM; Milton-RM
SO: Environ-Health-Perspect. 1987 Jun; 72: 13-9
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Early reports from India indicated that humans were dying within minutes to a few hours from exposure to methyl isocyanate (MIC). Attempts to explain the cause(s) of these rapid mortalities is where Union Carbide Corporation concentrated its post-Bhopal toxicologic investigations. The MIC studies involving rats and guinea pigs focused primarily on the consequences of acute pulmonary damage. All MIC inhalation exposures were acute, of short duration (mainly 15 min), and high in concentration (ranging from 25-3506 ppm). MIC vapors were statically generated in a double chamber exposure design. Precautionary measures taken during exposures are discussed. Guinea pigs were more susceptible than rats to MIC exposure-related early mortality. A greater than one order of magnitude difference was observed between an MIC concentration that caused no early mortality in rats (3506 ppm) and an MIC concentration that caused partial (6%) early mortality in guinea pigs (225 ppm) for exposures of 10 to 15 min duration. For both species, the most noteworthy clinical signs during exposure were lacrimation, blepharospasm, and mouth breathing. Fifteen minute LC50 tests with 14-day postexposure follow-up were conducted, and the LC50 (95% confidence limit) values were 171 (114-256) ppm for rats and 112 (61-204) ppm for guinea pigs. Target exposure concentrations for the toxicologic investigations of MIC-induced early mortality were established. A short summary of pertinent results of Union Carbide Corporation's post-Bhopal toxicologic investigations is presented.

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Marked Record

TI: The Bhopal tragedy.
AU: Srivatsa-LP
SO: J-Toxicol-Clin-Exp. 1987 Jan-Feb; 7(1): 47-9
this source is not Available in S.J.M.C.Library
LA: ENGLISH

27 of 29
Marked Record

TI: Spectre of the Bhopal disaster [letter]
SO: Am-J-Public-Health. 1987 Jul; 77(7): 878-9
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1942-1991
LA: ENGLISH

28 of 29
Marked Record

21 of 29
Marked Record

TI: K.A.P. survey of contraception in Bhopal and surrounding villages.
AU: Ranganekar-G; Sapre-S; Singh-H
SO: Indian-J-Med-Sci. 1987 Jun; 41(6): 119-23
This source is Available in S.J.M.C Library
Call Number: From: 1947+
LA: ENGLISH

22 of 29
Marked Record

TI: Bhopal aftermath re-assessed [news]
AU: Jayaraman-KS
SO: Nature. 1987 Oct 29-Nov 4; 329(6142): 752
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1993
LA: ENGLISH

23 of 29
Marked Record

TI: The antibody response to methyl isocyanate: experimental and clinical findings.
AU: Karol-MH; Taskar-S; Gangal-S; Rubanoff-BF; Kamat-SR
SO: Environ-Health-Perspect. 1987 Jun; 72: 169-75
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: As a result of the industrial accident in Bhopal, India (December 1984) in which thousands of people were exposed to methyl isocyanate (MIC), concern was raised for possible long-term health effects. The well-recognized immunologic consequences of exposure to other industrial isocyanates prompted investigation of an antibody response to MIC. Using procedures which had been developed in this laboratory to evaluate isocyanate immunotoxicity, animal studies were undertaken to develop and test reagents which could be used to detect antibodies to MIC in the exposed population. Guinea pigs were injected with MIC in its reactive isocyanate form. Three weeks later, blood was drawn and serum evaluated using ELISA. To detect antibodies, an antigen was prepared by reaction of MIC with guinea pig serum albumin. Antibodies were detected in each of the four animals injected with MIC. Titers achieved were 1:5120 to 1:10,240. Inhibition assays revealed antibody specificity directed toward the MIC hapten. Analogous antigens prepared by reaction of MIC with human serum albumin were used to evaluate sera from individuals exposed in Bhopal to MIC. Antibodies were detected in 12 of 144 exposed persons. Antibodies were specific for MIC, as evidenced by inhibition assays, and belonged to the IgG, IgM and IgE classes. However, titers were generally low and transient and were found in persons having had the highest MIC exposures. Total IgE values of sera were not significantly different from those of control sera obtained from Bombay residents. The results indicate that exposure to methyl isocyanate resulted in production of specific antibodies. However, the low titers observed and the transient nature of the response suggest little health consequence should result form the antibody response.

24 of 29
Marked Record

TI: Epidemiological and experimental studies on the effects of methyl isocyanate on the course of pregnancy.
AU: Varma-DR
SO: Environ-Health-Perspect. 1987 Jun; 72: 153-7
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Although press reports indicate that the leakage of methyl isocyanate (MIC) on December 3, 1984, in Bhopal has led to an increase in spontaneous abortions,

This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

15 of 29
Marked Record
TI: Mental health needs of Bhopal disaster victims & training of medical officers in mental health aspects.
AU: Murthy-RS; Isaac-MK
SO: Indian-J-Med-Res. 1987; 86 Suppl: 51-8
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

16 of 29
Marked Record
TI: Psychiatric morbidity in patients attending clinics in gas affected areas in Bhopal.
AU: Sethi-BB; Sharma-M; Trivedi-JK; Singh-H
SO: Indian-J-Med-Res. 1987; 86 Suppl: 45-50
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

17 of 29
Marked Record
TI: Sequential respiratory changes in those exposed to the gas leak at Bhopal.
AU: Kamat-SR; Patel-MH; Kolhatkar-VP; Dave-AA; Mahashur-AA
SO: Indian-J-Med-Res. 1987; 86 Suppl: 20-38
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

18 of 29
Marked Record
TI: Clinical profile of gas leak victims in acute phase after Bhopal episode.
AU: Misra-NP; Pathak-R; Gaur-KJ; Jain-SC; Yesikar-SS; Manoria-PC; Sharma-KN; Tripathi-BM; Asthana-BS; Trivedi-HH; et-al
SO: Indian-J-Med-Res. 1987; 86 Suppl: 11-9
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

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Marked Record
TI: Scientific studies on Bhopal gas victims. Part-A.
SO: Indian-J-Med-Res. 1987; 86 Suppl: 1-86
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

20 of 29
Marked Record
TI: Early observations on lung function studies in symptomatic "gas" exposed population of Bhopal.
AU: Bhargava-DK; Verma-A; Batni-G; Misra-NP; Tiwari-UC; Vijayan-VK; Jain-SK
SO: Indian-J-Med-Res. 1987; 86 Suppl: 1-10
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

survivors. Available evidence, which is limited, suggests that chronic damage, when present, is, or resembles, fibrosing bronchiolitis obliterans, the expected consequence when permanent injury results from acute, high-level irritant gas exposure. Definition of the follow-up population is uncertain, and exposure information is lacking. Dose-response relationships are not likely to emerge from follow-up studies.

9 of 29
Marked Record

TI: The public health implications of the Bhopal disaster. Report to the Program Development Board, American Public Health Association. Bhopal Working Group.
SO: Am-J-Public-Health. 1987 Feb; 77(2): 230-6
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1942-1991
LA: ENGLISH

10 of 29
Marked Record

TI: Bhopal--what is their suffering?
AU: Srivatsa-LP
SO: J-Toxicol-Clin-Exp. 1987 Sep-Oct; 7(5): 323-9
this source is not Available in S.J.M.C.Library
LA: ENGLISH

11 of 29
Marked Record

TI: Preliminary report on the spermatogenic function of male subjects exposed to gas at Bhopal.
AU: Daniel-CS; Singh-AK; Siddiqui-P; Mathur-BB; Das-SK; Agarwal-SS
SO: Indian-J-Med-Res. 1987; 86 Suppl: 83-6
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

12 of 29
Marked Record

TI: Morphological study of placentae of expectant mothers exposed to gas leak at Bhopal.
AU: Kanhere-S; Darbari-BS; Shrivastava-AK
SO: Indian-J-Med-Res. 1987; 86 Suppl: 77-82
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

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Marked Record

TI: Immunological, mutagenic & genotoxic investigations in gas exposed population of Bhopal.
AU: Deo-MG; Gangal-S; Bhisey-AN; Somasundaram-R; Balsara-B; Gulwani-B; Darbari-BS; Bhide-S; Maru-GB
SO: Indian-J-Med-Res. 1987; 86 Suppl: 63-76
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH

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Marked Record

TI: Neurological manifestations among those exposed to toxic gas at Bhopal.
AU: Bharucha-EP; Bharucha-NE
SO: Indian-J-Med-Res. 1987; 86 Suppl: 59-62

with standard control group of same socioeconomic status in a non-gas affected slum area of Bhopal. The main chronic lesions noticed were chronic conjunctivitis, refractive changes, deficiency of tear secretion and persistent corneal opacities of various forms.

6 of 29
Marked Record
TI: Long term follow up of ocular lesion of methyl-isocyanate gas disaster in Bhopal.
AU: Khurrum-MA; Ahmad-SH
SO: Indian-J-Ophthalmol. 1987 May-Jun; 35(3): 136-7
This source is Available in S.J.M.C Library
Call Number: From: 1973+
LA: ENGLISH

7 of 29
Marked Record
TI: The antibody response to methyl isocyanate: experimental and clinical findings.
AU: Karol-MH; Kamat-SR
AD: Department of Industrial Environmental Health Sciences, Graduate School of Public Health, University of Pittsburgh, PA 15261.
SO: Bull-Eur-Physiopathol-Respir. 1987 Nov-Dec; 23(6): 591-7
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Sera from 99 subjects exposed to the industrial gas leak in Bhopal on December 2, 1984 were studied along with sera from guinea pigs exposed to methyl isocyanate (MIC) to determine the production of antibodies specific to (MIC). Each of the four guinea pigs injected with the reactive isocyanate produced MIC-specific antibodies in titres of 1:5120 to 1:10240, when tested with MIC-guinea pig albumin antigen conjugate. Analogous antigens prepared by reaction of MIC with human serum albumin were used to probe production of antibodies in 264 serially obtained human sera from 99 subjects from Bhopal. MIC-specific antibodies belonging to IgG, IgM and IgE classes were detected in eleven subjects. Though titres were low and transient (declining after several months) these findings indicate that the single large exposure to MIC resulted in an immunologic response. This finding was concomitant with chronic respiratory effects following MIC exposure.

8 of 29
Marked Record
TI: Disaster at Bhopal: the accident, early findings and respiratory health outlook in those injured.
AU: Weill-H
AD: Tulane University School of Medicine, Department of Medicine, New Orleans, Louisiana 70112.
SO: Bull-Eur-Physiopathol-Respir. 1987 Nov-Dec; 23(6): 587-90
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: In December, 1984, in Bhopal, India, a massive leak of methyl isocyanate (MIC) resulted from operational and equipment malfunctions in a pesticide plant. Many thousands of residents of the city, most in proximity to the plant, suffered sublethal and lethal respiratory injuries, the expected consequences of high-level exposure to this type of potent irritant chemical vapour. Animal toxicologic information was limited prior to the accident, but has since confirmed that the lung is the major target of these lethal injuries, invariably with pulmonary oedema. Early concerns regarding acute cyanide intoxication were not supported by subsequent scientific inquiry. Superficial corneal erosions did not result in permanent eye injury. The primary medical (and, presumably, legal) issue which is unresolved, and perhaps unresolvable, is the incidence and determinants of long-term respiratory injury in the

1 of 29
Marked Record

TI: The Bhopal disaster.
AU: Tachakra-SS
SO: J-R-Soc-Health. 1987 Feb; 107(1): 1-2
this source is not Available in S.J.M.C.Library
LA: ENGLISH

2 of 29
Marked Record

TI: Methyl isocyanate: a review of health effects research since Bhopal.
AU: Bucher-JR
AD: National Toxicology Program, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina 27709.
SO: Fundam-Appl-Toxicol. 1987 Oct; 9(3): 367-79
this source is not Available in S.J.M.C.Library
LA: ENGLISH

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Marked Record

TI: Toxicity of inhaled methyl isocyanate in experimental animals--a review of studies published less than two years after the Bhopal disaster [published erratum appears in Bull Eur Physiopathol Respir 1987 Nov-Dec;23(6):667]
AU: Nemery-B; Dinsdale-D; Sparrow-S
AD: Toxicologie Industrielle et Medecine du Travail, Universite Catholique de Louvain, Bruxelles, Belgique.
SO: Bull-Eur-Physiopathol-Respir. 1987 Jul-Aug; 23(4): 315-22
this source is not Available in S.J.M.C.Library
LA: ENGLISH; FRENCH

4 of 29
Marked Record

TI: Acute inhalational injury.
AU: Schwartz-DA
SO: Occup-Med. 1987 Apr-Jun; 2(2): 297-318
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: Recent events, such as the Bhopal, India tragedy, dramatically illustrate the potential consequences of acute exposure to toxic inhalants. This article covers problems in clinical management of the toxic gases, mists and fumes that are capable of inducing acute lung injury or systemic toxicity.

5 of 29
Marked Record

TI: Chronic ocular lesions in Bhopal gas tragedy.
AU: Raizada-JK; Dwivedi-PC
SO: Indian-J-Ophthalmol. 1987; 35(5-6): 453-4
This source is Available in S.J.M.C Library
Call Number: From: 1973+
LA: ENGLISH
AB: A comprehensive eye checkup programme was carried out in 1140 cases of affected community to evaluate the exposure response and exposure effect with Methyl Isocynate on human eyes. The final evaluation of these cases was made

response of muscle tissue to MIC its effects were investigated in cells in culture isolated from muscle of 2 day old rats. After treatment with a range of MIC concentrations (0.025-0.5 microliter/5 ml culture) the total number of nuclei of the two main cell types (fibroblasts and myoblasts) and the number of nuclei in muscle fibres (myotubes) were recorded. At lower doses which had little effect on the total number of nuclei, the formation of muscle fibres--that is, fusion of muscle cells--was prevented as the proportion of nuclei in myotubes was decreased. At higher doses both cell types were killed. This would suggest either an effect on muscle differentiation or a selective toxicity towards myoblasts. The observations were supported by light and electron microscopy.

12 of 12
Marked Record

TI: Inhibition of methyl isocyanate toxicity in mice by starvation and dexamethasone but not by sodium thiosulfate, atropine, and ethanol.
AU: Varma-DR; Ferguson-JS; Alarie-Y
AD: Department of Pharmacology and Therapeutics, McGill University, Montreal, Quebec, Canada.
SO: J-Toxicol-Environ-Health. 1988; 24(1): 93-101
This source is Available only few issues in S.J.M.C. Library
LA: ENGLISH
AB: Effects of starvation (24 and 48 h), dexamethasone, sodium thiosulfate, atropine, and ethanol on the toxicity of methyl isocyanate (MIC) vapor, which escaped during the Bhopal accident of December 3, 1984, were studied in male Swiss-Webster mice. Toxicity to MIC appeared to be biphasic; majority of animals died between 1 and 2 d or between 7 and 21 d after exposure to 40 ppm MIC. Starvation (24 or 48 h) or an injection of 2 mg dexamethasone/kg prior to exposure inhibited the toxicity of MIC, especially during the first 6-7 d; administrations of sodium thiosulfate, alcohol, and atropine before or of dexamethasone after the exposure to MIC were ineffective. Starvation increased serum corticosterone levels. The antidotal effects of both starvation and dexamethasone might be due to suppression of the inflammatory response to MIC.

Call Number: From: 1972+
LA: ENGLISH

8 of 12
Marked Record

TI: Effect of exposure to toxic gas on the population of Bhopal: Part I--Epidemiological, clinical, radiological & behavioral studies.
AU: Gupta-BN; Rastogi-SK; Chandra-H; Mathur-AK; Mathur-N; Mahendra-PN; Pangtey-BS; Kumar-S; Kumar-P; Seth-RK; et-al
SO: Indian-J-Exp-Biol. 1988 Mar; 26(3): 149-60
This source is Available in S.J.M.C Library
Call Number: From: 1972+
LA: ENGLISH

9 of 12
Marked Record

TI: Bhopal gas disaster: clinical & experimental studies.
SO: Indian-J-Exp-Biol. 1988 Mar; 26(3): 149-204
This source is Available in S.J.M.C Library
Call Number: From: 1972+
LA: ENGLISH

10 of 12
Marked Record

TI: Exposure and response to methyl isocyanate: results of a community based survey in Bhopal.
AU: Andersson-N; Kerr-Muir-M; Mehra-V; Salmon-AG
AD: Department of Occupational Health, London School of Hygiene and Tropical Medicine, UK.
SO: Br-J-Ind-Med. 1988 Jul; 45(7): 469-75
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1991
LA: ENGLISH
AB: In the two weeks immediately after the Bhopal disaster a community based survey was carried out in a series of eight exposed and two non-exposed clusters of households. The primary concern was the effect of the gas (subsequently identified as methyl isocyanate) on the eyes of the victims but data were also sought on respiratory status and the first symptoms of the exposure. No case of blindness was encountered that could be attributed to the gas. The most frequent symptoms reported were burning of the eyes, coughing, watering of the eyes, and vomiting. Among these, the frequency of cough most closely followed the rate of death in the different clusters. Although much rarer overall, the frequency of reported diarrhoea appeared to bear a stronger relation to death rates. Reports of photophobia and the clinical finding of superficial interpalpebral erosion of the cornea were more frequent where the death rates were lower. This clinical and epidemiological picture is consistent with different effects of the gas at different doses (as estimated from distance from the factory).

11 of 12
Marked Record

TI: Effects of methyl isocyanate on rat muscle cells in culture.
AU: Anderson-D; Goyle-S; Phillips-BJ; Tee-A; Beech-L; Butler-WH
AD: British Industrial Biological Research Association, Carshalton, Surrey, UK.
SO: Br-J-Ind-Med. 1988 Apr; 45(4): 269-74
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1991
LA: ENGLISH
AB: Since the Bhopal disaster, in which the causal agent was methyl isocyanate (MIC), exposed people have complained of various disorders including neuromuscular dysfunction. In an attempt to gain some information about the

determining the liability of the Union Carbide Company (U. S. A.) for the Bhopal disaster; the criteria for determining compensation; and the international remedies available to the Indian government in the event that Bhopal victims fail to get justice within the Indian court system. The article discusses two applicable sets of proposed international standards--the U. N. Draft Code of Conduct on Transnational Corporations, and the U. N. International Law Commission's Draft on International Liability for Injurious Consequences Arising out of Acts Not Prohibited by International Law. The scattered 'hard' and 'soft' jurisprudence of international environmental law establishes liability and accountability for environmental hazards. It makes both state and non-state entities liable to pay compensation to the victims of environmental pollution. This jurisprudence, in addition to domestic law analogies, can influence Indian courts in determining the amount of damages payable to the victims of the Bhopal disaster. The authors conclude that the Bhopal disaster has demonstrated that enforceable international standards are clearly and urgently needed for hazardous industries, especially those operating in developing countries. Such standards would eliminate, or at least narrow, the gap between standards prevailing in the developed countries and those in the Third World. Even without enforcement, international standards could provide norms for measuring the performance of individual companies engaged in hazardous activities such as the manufacture of MIC at Bhopal.

4 of 12
Marked Record

TI: A clinical study of toxic gas poisoning in Bhopal, India.
AU: Misra-UK; Nag-D; Nath-P; Khan-WA; Gupta-BN; Ray-PK
SO: Indian-J-Exp-Biol. 1988 Mar; 26(3): 201-4
This source is Available in S.J.M.C Library
Call Number: From: 1972+
LA: ENGLISH

5 of 12
Marked Record

TI: Effect of exposure to toxic gas on the population of Bhopal: Part IV--Immunological and chromosomal studies.
AU: Saxena-AK; Singh-KP; Nagle-SL; Gupta-BN; Ray-PK; Srivastav-RK; Tewari-SP; Singh-R
SO: Indian-J-Exp-Biol. 1988 Mar; 26(3): 173-6
This source is Available in S.J.M.C Library
Call Number: From: 1972+
LA: ENGLISH

6 of 12
Marked Record

TI: Effect of exposure to toxic gas on the population of Bhopal: Part III--Assessment of toxic manifestations in humans--haematological and biochemical studies.
AU: Srivastava-RC; Gupta-BN; Athar-M; Behari-JR; Dwivedi-RS; Hasan-SK; Bharti-RS; Singh-A; Misra-M; Ray-PK
SO: Indian-J-Exp-Biol. 1988 Mar; 26(3): 165-72
This source is Available in S.J.M.C Library
Call Number: From: 1972+
LA: ENGLISH

7 of 12
Marked Record

TI: Effect of exposure to toxic gas on the population of Bhopal: Part II--Respiratory impairment.
AU: Rastogi-SK; Gupta-BN; Husain-T; Kumar-A; Chandra-S; Ray-PK
SO: Indian-J-Exp-Biol. 1988 Mar; 26(3): 161-4
This source is Available in S.J.M.C Library

1 of 12
Marked Record

TI: Aluminium phosphide: worse than Bhopal [letter]
AU: Kabra-SG; Narayanan-R
SO: Lancet. 1988 Jun 11; 1(8598): 1333
This source is Available in S.J.M.C Library
Call Number: From: 1930+
LA: ENGLISH

2 of 12
Marked Record

TI: The Bhopal disaster and the right to know.
AU: Jasanoff-S
AD: Program on Science, Technology and Society, Cornell University, Ithaca, NY 14853.
SO: Soc-Sci-Med. 1988; 27(10): 1113-23
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: The chemical disaster in Bhopal jolted activist groups around the world into renewing their demands for right-to-know legislation granting them broader access to information about hazardous technologies. This article explores the obstacles to creating effective right-to-know policies when technology is transferred across national boundaries. The events leading to the Bhopal accident are first examined in order to assess how far the tragedy can be attributed to gaps in knowledge or to breakdowns in communication. Using Bhopal as a case study, the article then considers three issues that are central to the design of right-to-know policies: who has a right to receive information about hazards; who has the duty to disclose such information, and, where necessary, to produce missing information; what information should be available for disclosure? This inquiry suggests that the circles of those with a right to know and those with a duty to disclose should both be larger than under existing right-to-know laws. More systematic risk information should also be generated, including probabilistic estimates of risk and environmental impact analyses. Finally, the article asks whether such improvements in knowledge and communication would prevent disasters of the kind that occurred in Bhopal. It concludes that for knowledge to be meaningful it must be correlated with the power to act preventively. This implies, in turn, that those with a right to know have to be given an opportunity to participate in technology transfer decisions before it is too late to choose a technology that is well adapted to the technical and cultural circumstances of the importing country.

3 of 12
Marked Record

TI: Some international law aspects of the Bhopal disaster.
AU: Tyagi-YK; Rosencranz-A
AD: International Law & Economics, School of International Studies, Jawaharlal Nehru University, New Delhi, India.
SO: Soc-Sci-Med. 1988; 27(10): 1105-12
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: This article explores certain international law aspects of the Bhopal disaster, namely the principles and rules of international law establishing international accountability for environmental damage; the criteria for

1 of 2
Marked Record

TI: Hydrogen cyanide and Bhopal [letter; comment]
AU: Varma-DR
SO: Lancet. 1989 Sep 2; 2(8662): 567-8
This source is Available in S.J.M.C Library
Call Number: From: 1930+
LA: ENGLISH

2 of 2
Marked Record

TI: Bronchoalveolar lavage study in victims of toxic gas leak at Bhopal.
AU: Vijayan-VK; Pandey-VP; Sankaran-K; Mehrotra-Y; Darbari-BS; Misra-NP
SO: Indian-J-Med-Res. 1989 Dec; 90: 407-14
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH
AB: Bronchoalveolar lavage using flexible fibreoptic bronchoscope was carried out in 50 patients 1-2 1/2 yr after exposure to the 'toxic gas' at Bhopal. Thirty six patients in the analysis were categorised into 3 groups (viz., mild, moderate and severe), depending upon the severity of exposure. There was an increase in cellularity in the lower respiratory tract (alveolitis) of the severely exposed patients (in both smokers and non-smokers), compared to normals (P less than 0.05). The increase in cellularity in severely exposed non-smokers was due to abnormal accumulation of macrophages (P less than 0.01), and in severely exposed smokers, to macrophages (P less than 0.01) and neutrophils (P less than 0.05). Mild and moderately exposed patients did not show significant change in cellularity in lower respiratory tract, compared to normal individuals (P greater than 0.2). There was a trend towards increasing cellularity, as the severity increased (P less than 0.0001) and higher numbers of total cells were seen in severely exposed smokers, suggesting that smoking is a risk factor. It appears, therefore, that subjects severely exposed to the toxic gas at Bhopal may have a subclinical alveolitis characterised by accumulation and possibly activation of macrophages in the lower respiratory tract. Smokers, who were exposed to the gas had in addition, accumulation of neutrophils.

TI: Bhopal: deal is less than final [news]
AU: Jayaraman-KS
SO: Nature. 1990 Aug 9; 346(6284): 503
This source is Available only few issues in S.J.M.C. Library
Call Number: From: 1956-1993
LA: ENGLISH

11 of 12
Marked Record

TI: Pregnancy outcome in women exposed to toxic gas at Bhopal.
AU: Bhandari-NR; Syal-AK; Kambo-I; Nair-A; Beohar-V; Sexena-NC; Dabke-AT; Agarwal-SS; Saxena-BN
AD: Department of Paediatrics, Gandhi Medical College, Bhopal.
SO: Indian-J-Med-Res. 1990 Feb; 92: 28-33
This source is Available in S.J.M.C Library
Call Number: From: 1943+
LA: ENGLISH
AB: A study was undertaken to compare the effects of exposure to the toxic gas in pregnant women in Bhopal with pregnant women in a similar, unexposed area. A high incidence of spontaneous abortions (24.2%) in the pregnant women exposed to the toxic gas was observed as compared to those in the control area (5.6%). Other indices of adverse reproductive outcome, such as the rate of still birth and congenital malformation were not found to be different. The perinatal and neonatal mortalities were significantly higher in the affected area (6.9 and 6.1% respectively), as compared to the control area (5.0 and 4.5% respectively).

12 of 12
Marked Record

TI: Search for chromosomal variations among gas-exposed persons in Bhopal [see comments]
AU: Goswami-HK; Chandorkar-M; Bhattacharya-K; Vaidyanath-G; Parmar-D; Sengupta-S; Patidar-SL; Sengupta-LK; Goswami-R; Sharma-PN
AD: Department of Genetics, Bhopal University, India.
SO: Hum-Genet. 1990 Jan; 84(2): 172-6
this source is not Available in S.J.M.C.Library
LA: ENGLISH
AB: A chromosomal survey using standard lymphocyte cultures employing different media and G-banding techniques was initiated in 1984. This study became particularly important following the tragic gaseous exposure of the population in Bhopal at midnight on 2 December 1984. We have been able to formulate a chromosomal profile for each person whom we have studied; during 1986-1988, 154 persons were examined twice. Among seemingly normal individuals, as many as 20% might possess some chromosomal abnormality; of these, 50% may develop, at a later date, some kind of pathological complication (such as tumours, recurrent abortion or transmission of defects to their offspring). The people exposed to methyl isocyanate have repeatedly shown Robertsonian translocations, mostly in acrocentric chromosomes 13 and 21. Other types of translocations have been studied among all exposed (53) and normal (101) persons; the involvement of chromosomes 5, 9, 11, 14 and 16 is statistically significant (P = less than 0.001). One of the major clinical symptoms is dyspnoea; we have estimated that almost all seriously dyspnoeic patients have developed at least two categories of chromosomal aberrations, one of which is Robertsonian translocation, in at least 10% metaphases. Our chromosomal survey will be of significance because we are able to identify people with chromosomal aberrations that might be correlated with future pathological consequences of the accident. The "chromosomal load" that can be sustained with an apparently normal phenotype can also be measured.

SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

Terms of Reference

Towards developing a comprehensive overview of the current issues faced by those affected by the Bhopal gas disaster, it is envisaged that the fact finding mission will:

1. Analyze the continuing impact of the disaster on different areas of the lives of survivors of the disaster.

2. Identify specific failures, including policy and institutional, in the last fourteen years and their subsequent repercussions on the people of Bhopal.

3. Compile documentation on all the various aspects related to the Bhopal gas disaster.

4. Outline larger policy changes as a result of the disaster.

5. Provide concrete suggestions for effective democratic interventions.

SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

Parameters of the Fact Finding Mission

In a meeting of the Organizing Committee we identified the following 14 aspects of Surviving Bhopal that we feel that the Fact Finding Mission must address. We have also arrived at broad parameters for each Fact Finding Team which are briefly outlined below. We would appreciate your response to these outlines and any suggestions you have to make, either on expanding the scope of the Fact Finding Teams' study and analysis, or the inclusion of any other aspect that we may have overlooked.

1. *Medical Care*

- Estimated figures of exposure-related deaths per month, persons chronically ill, persons acutely ill and persons with exposure-related injuries
- The extent and nature of knowledge on treatment and treatment efficacy available with doctors, RMPs etc
- The most commonly prescribed drugs and lines of treatment
- Available facilities for medical care in different systems of medicine: No. of beds, doctors, specialists, equipments, investigation facilities, availability of medicines and attendance per day and utilisation of beds in government, private and other hospital/clinics
- Systems of registration and medical record keeping
- Issues confronting employees of various hospitals and clinics, especially in gas affected areas
- Possibilities of improvement in health care

2. *Medical Research*

- Consolidation of studies carried out by government, private and other agencies and a reassessment of their major findings
- Analysis of the design and implementation of government studies
- Analysis of various studies conducted by Central Govt institutions for rehabilitation
- Outline of studies required for long term monitoring of health effects
- Assessment of the health status of gas affected persons, and their continuing exposure to hazards at both, the occupational and domestic levels
- What are the existing public health initiatives, and what is required?

3. *Legal Issues*

- The current status of legal actions pending before different courts
- The situation with respect to compensation distribution
- An investigation of the systematic institutional corruption forced on the survivors
- The policy and legal fall outs of the disaster
- The possibilities of further legal action
- The status of survivors' access to justice in terms of legal aid and counselling
- Compilation of a list of documents available with the CBI

4. *Economic Rehabilitation*

- Outline of existing government and non-government rehabilitation programmes
- The estimated figure of persons unemployed due to exposure related ill health
- Profile of the range of occupations in different sections of the gas affected population and the estimated per capita income.
- Outline of the skills available for income generation
- List of the potential markets for goods produced through income generating projects

5. Labour
- The status of Carbide workers and their families today
- Outline of how labour unions have responded to the disaster
- The impact of exposure on survivors' capacity to work
- Effect on their opportunities of livelihood in the job market e.g special covenants

6. Social Rehabilitation
- Estimated figures of those needing social support as a consequence of the disaster
- The social impact of the disaster (qualitative study of 50 families?)
- Outline of the specific impact of the gas disaster on women in terms of economic, social status etc.
- How the gas disaster has impacted children in terms their of childhood, education, health and livelihood
- Other possibilities of social rehabilitation

7. Environment
- The environmental impact of the factory and toxic contamination
- Assessment of government and non-governmental studies related to environmental impact of the disaster
- The pattern of industrial development in and around Bhopal since the disaster

8. Union Carbide
- An overview of Union Carbide Corporation, Union Carbide Eastern and Union Carbide India Limited and their assets, annual profits, facilities, products, board of directors, number of employees, number of countries, new projects, rating as a corporation, other disasters, health and safety records

9. Scientific Institutions
- Assessment of studies conducted by different scientific institutions and their major findings
- Possibilities of further involvement from scientific institutions

10. Role of State and Central Governments
- Outline of the government bodies and their responsibilities
- To provide details of programmes, policies and their analysis
- Analysis of expenditure and accounts
- Outline of the infrastructure of government departments meant for the welfare of gas survivors, e.g, number of personnel
- Outline the database available with state and central governments

11. Non-Governmental Organizations and Peoples' Organizations
- To provide a list of NGOs and Peoples' organizations
- To outline major demands, objectives and activities
- To identify their sources of funding and other support
- Strengths and weaknesses, achievements and failures
- To list national and international support
- Suggestions for improved functioning

12. Memorial on the Disaster and its People

- To present the current status of the State government plans to build a memorial
- To highlight future possibilities
- To outline how peoples' participation in constructing and managing the memorial can be ensured

13. Disaster Management

- To assess the disaster management policies and programmes, implemented by the state bodies in the immediate aftermath of the disaster
- To examine whether Union Carbide Corporation and Union Carbide India Limited had any strategies for management of a possible disaster at the Bhopal factory, whether they were implemented and their effectiveness
- Based on the Bhopal experience, what would be the proposed disaster management programme in the event of an industrial tragedy?

14. Media's Response

- To present a critique of national and international media response in the last fifteen years
- Compilation of media articles, reports etc...

SURVIVING BHOPAL: 15 YEARS ON

A FACT FINDING MISSION

Backgrounder

Introduction :

On the midnight of 2nd - 3rd December 1984, the worst industrial disaster of this century was caused by Union Carbide Corporation, USA in the capital city of Bhopal, Madhya Pradesh, a city with about one million people. Over 40 tonnes of Methyl Isocyanate and other lethal gases including Hydrogen Cyanide, leaked from Carbide's pesticide factory in the northern end of the city killing over 8,000 people in its immediate aftermath, causing multisystemic injuries to over 500,000 people. The number of deaths has risen to over 16,000 in the subsequent years and there appears no end to the physical and mental suffering caused by exposure to the poisonous gases. Breathlessness, diminished vision , loss of appetite, pain, menstrual irregularities, recurrent fever, persistent cough, neurological disorders, fatigue, weakness, anxiety and depression are the most common symptoms. Research findings on chromosomal aberrations suggest that the future generations of the survivors will possibly carry ravages of the industrial toxins. Union Carbide continues to withhold toxicological information on the leaked gases thereby impeding medical treatment. The majority of those affected by the gases are people who earned their livelihood through hard physical labour and today their economic condition forces them to continue with their jobs, thus exposing themselves to further health risks. Little has changed in the living environment of the survivors, most of whom live in congested slums without facilities for safe drinking water, sanitation and clean air. Judicial systems in both USA and India have failed to ensure adequate compensation and justice for the survivors. The settlement amount, an average of US $ 940 for each survivor, paid by Union Carbide resulted in a nominal loss to its shareholders of merely 50 cents per share. Compensation sums awarded for personal injury have been unjust and inadequate and in over 90% of cases the victims have received only about Rs15,000/- (or about $430). Nearly two hundred thousand persons directly affected by Union Carbides gases remain to be compensated. For a large number of the victims the sums received as compensation have been spent in repaying debts incurred in medical treatment in the last several years. Officials of Union Carbide who have been charged with manslaughter and other criminal offences are absconding from Indian courts where criminal proceedings against them been pending for the last six years. Every week in a public park in the city , hundreds of gas-affected women hold public meetings calling for the trial of the prime-accused Warren Anderson, former Chairman of the corporation, who is known to be on vacation at Vero Beach, Florida, USA.

Medical impact of the disaster:

Most of the information on the medical consequences of the Union Carbide disaster in Bhopal has been generated by the Indian Council of Medical Research (ICMR), an agency of the Indian government that carried out 25 research studies from 1985 to 1994. All ICMR studies in Bhopal were prematurely terminated by December 1994

The ICMR established that the toxins from Carbide's factory have crossed into the blood stream of those exposed and have caused damage to the lungs, brain, kidneys, muscles as well as gastro- intestinal, reproductive, immunological and other systems.

Six monthly morbidity surveys by ICMR from 1987 to 1991, show that the number of people with exposure-related symptoms actually increased in that period. According to one study, there were three times more persons with respiratory symptoms in 1991 as compared to 1987. The damage to the respiratory system and particularly the lungs comprises the most obvious and very significant part of the overall health damage. Bronchial asthma, Chronic Obstructive Airways Disease, recurrent chest infections, and fibrosis of the lungs are the principal effects of exposure induced lung injury. The prevalence of pulmonary tuberculosis among the exposed population has been found to be more than three times that of the national average.

Damage caused to the eyes of the survivors have led to early-age cataracts found to be three times more prevalent among the exposed population compared to an unexposed population at the far end of the city. Pregnancy outcome studies on women who were pregnant at the time of the disaster have shown that the spontaneous abortion rate was almost three times that of the national average. In the exposed population the stillbirth rate was three times, perinatal mortality was two times and neonatal mortality was one and a half times more than the comparative national figures. Study on growth and development of the children whose mothers were exposed to the toxic gases during pregnancy revealed that majority of children had delayed gross motor and language sector development. Studies have also presented evidence of chromosomal aberrations of gaps and breaks in the chromosomal material as well as increased sisterchromatid exchange indicating likelihood of congenital abnormalities among future generations of the exposed persons.

Toxic gas exposure was found by ICMR researchers to have had a detrimental effect on the immune system. Immunoglobulin levels were significantly raised , T-cells were reduced in number and there was a tendency towards the reversal of T4/T8 ratio indicative of immuno-suppression.

Various studies conducted by non-government organizations have pointed out that the medical consequences of the Union Carbide disaster have been under-assessed by the ICMR and certain exposure related injuries have been overlooked.

The pregnancy outcome survey referred to above was carried out by Medico Friend Circle in September '85 and showed that the spontaneous abortion rate among gas exposed women was several times higher than that reported by ICMR. A survey of psychiatric morbidity carried out by a group of independent doctors from Bombay found that nearly 40% of those exposed suffered from post-traumatic stress disorder, a condition not studied by ICMR.

An epidemiological survey coupled with clinical investigation carried out by the International Medical Commission on Bhopal, composed of 14 medical specialists from 11 different countries, reported in January 1994 significant multi-organ symptoms persistent among the exposed population. Clinical examinations have shown significant lung impairment, marked reduction in control over limb movements and reduced memory function caused due to exposure. Their findings include evidence of a range of neurotoxic injuries in the exposed population.

State of Medical Care and Monitoring :

The medical care of the survivors has largely remained symptomatic since the time of the disaster and continues to be ineffective in providing sustained relief. Union Carbide continues to withhold information on the composition of the leaked gases and their long term effects on the human body. In the absence of such information, doctors in Bhopal indiscriminately prescribe antibiotics, steroids and psychotropic medicines causing more harm than good. A study undertaken by the International Medical Commission on Bhopal confirmed that therapies prescribed for the ailing survivors are aimed at temporary symptomatic relief rather than long term amelioration of chronic disease processes. The major emphasis of the medical relief programmes of the Madhya Pradesh government has been to build hospitals so much so that Bhopal now has more per capita hospital beds than is recommended by the World Health Organization. Yet as per the reports of the ICMR the number of diseased persons has gone up with the years. For the last several months almost no medicines are available to the gas victims at any of the hospitals and clinics meant for them. People who do not have sufficient means for their basic needs are asked to buy medicines and even syringes and IV sets by doctors at these institutions that are supposed to provide free medical care. There has been hardly any government initiative in providing community based medical care to chronically ill survivors. Also lacking are initiatives for provision of medical care through systems of medicine such as Ayurveda, Unani and Yoga that have demonstrated their superior efficacy in the treatment of exposure related illnesses. The growing inadequacies of government medical care has led to unregulated proliferation of private and expensive medical clinics. With the termination of 25 medical research projects of the ICMR in December 1994, long term monitoring of the health condition of the survivors has been abandoned. The Centre for Rehabilitation Studies funded by the state government since March 1995 is yet to initiate any research work. The official agency for monitoring exposure related deaths has been wound up in December 1992 and there is no official record of deaths that continue to occur. With the premature termination of research and monitoring there is almost no current data on the prevalence of tuberculosis, cancers and infertility among the exposed population all of which are reported to be on the rise by doctors involved with the treatment of the survivors.

A recent issue of major concern is the proposed handing over of the health infrastructure set up the government to the so called Bhopal Hospital Trust set up by Union Carbide. Despite vigorous opposition by the survivors, the state government has begun handing over the eight community clinics to the Bhopal Hospital Trust (BHT) set up by Union Carbide Corporation. Preparations for transfer of the

four Red Cross clinics are also on. Ironically, one of the main factors impeding appropriate medical treatment at these government clinics has been withholding of medical information by Carbide and the closure of the Red Cross clinics followed from withdrawal of financial support by the corporation. The source of funds of the BHT is the value of shares of Union Carbide that had been judicially attached to ensure that the representatives of the corporation face criminal charges related to the disaster. By means of interventions in the Supreme Court through the sole trustee of BHT, Sir Ian Percival (an attorney working for Union Carbide from 1984 to '92) the corporation has been able to get the shares dis-attached and continues to abscond justice. In the last two years Percival has spent Rs. 7 crores on the construction of a 260-bed hospital ,which happens to be 8 kms away from the gas affected area, and Rs. 5 crores on his own fees, travel and office expenses. Quite clearly providing medical care to the survivors is not among the priorities of BHT. Its sole purpose is to build a humane image for the corporation while helping it to abscond criminal justice on the massacre. Percival's plans of health care administration have been severely criticized by national and international professional groups including the IMCB. Among other misgivings, concerns have been expressed regarding transparency of activities at the proposed medical research centre to be set up by the BHT.

Economic and Social Impact :

There has hardly been any systematic effort to document the social and economic impacts of the disaster. Official information on orphaned children and families that lost their breadwinners in the immediate or long aftermath is scanty, if available at all. Over 70% of the exposed population has been in the unorganized sector, with people earning subsistence wages through day labour or petty trade. A large number of men and women who pushed hand carts, carried loads, dug soil, repaired cars and did other jobs can no longer pursue their trades after being exposed to Carbide's gases. Gas exposed factory workers in textile and paper mills are more sensitive to occupational hazards and are absent from work due to illness as much as 15 days in a month. Over 90 % of the survivors have received a compensation of Rs 15,000 which is just enough for the cost of medicines for five years, and many of these people will be in need of medical care till the end of their lives. Given the complete inadequacy of official rehabilitation efforts the loss of regular income has driven tens of thousands of families to chronic starvation conditions. Loss of income also makes people borrow money from local money lenders who charge upto 200% interest so that chances of paying back are low and debts keep growing. Gas exposed women's inability to carry out reproductive functions have led to their desertion by their husbands and gas exposed young women continue to suffer social discrimination in marriage.

Environmental problems :

Thirteen years after the disaster, Union Carbide's toxic legacy continues to harm people in more ways than one. Communities in the vicinity of the Carbide factory continue to be exposed to toxic chemicals that are injurious to the lungs, liver and kidneys and can cause cancer. Water in over 200 wells around the Carbide factory have been declared unfit for human consumption by the municipal authorities. This is a result of routine dumping of hazardous chemicals during the operation of the factory thus

contaminating soil and groundwater in and around the factory premises. Analysis carried out by the Citizens Environmental Laboratory (CEL), Boston in 1991 show presence of toxic chemicals in the community wells around the factory. This report presented at the company's annual shareholders meeting drew the attention of the senior officials to the problem. The Corporation gave the job to Arthur D. Little Inc. who sponsored a collaborative investigation with an Indian government agency in 1994 in to the matter of contamination caused by dumping chemicals inside the factory premises. This study done without public knowledge recommends a fuller investigation for better assessment of the environmental contamination. The findings of this study confirm the worst apprehensions of activists and people in the community. Meanwhile, in 1996 the company management has dug up bottom soil from the "Solar Evaporation Ponds" and buried the heavily contaminated sludge under three metres of farm soil in a bid to cover up evidence of environmental damage. Survivors' organizations have been calling for an official assessment of the damage wrought by Union Carbide so that the corporation could be asked to pay the costs of environmental rehabilitation and supply of safe drinking water for the affected communities.

Economic Social and Environmental Rehabilitation :

The government programmes for economic rehabilitation have been badly designed and only few have been implemented. While an estimated population of 50,000 is in need of alternate jobs currently less than 100 gas victims have found employment under the government's scheme. 42 worksheds that had been built between 1985 to 1987 were allotted to so called NGOs between 1994 to 1996. However, apart from two worksheds employing about 50 women, none of them have ever been made available for employment of the survivors. Official promises have been on record that 50 % jobs in the Railway Coach Repair Factory would be reserved for gas affected people. However, only 205 of the 1000 employees of this factory are gas affected persons. In 1987 a special industrial area for training and employment of over 10,000 survivors was inaugurated and 152 worksheds were constructed at a cost of Rs.8 crores. However, till date not a single survivor has found any employment. A programme offering women survivors tailoring jobs ran successfully from 1986 to 1992 employing 2300 women and making an yearly profit of Rs.1 crore. The rehabilitation centres where these jobs were offered were also places where women survivors could gather, share their concerns and organize themselves. However, this programme was terminated without any reason in July 1992.

Till date the government has no record of the social condition of the persons who have been widowed, orphaned, or have been permanently disabled as a result of the gas disaster. The state government has deemed its work of social rehabilitation to be over by constructing 2500 houses and a few schools. There has been no official attention towards the urgent need of life long pension for widows, orphans, chronically ill and disabled survivors. The Supreme Court's final order with regard to provision of insurance coverage to about one lakh children likely to suffer delayed effects of the lethal gases is also being ignored by the Central government.

Despite the expenditure of over Rs. 70 crores in environmental rehabilitation basic necessities such as clean drinking water and sanitary facilities remain unavailable to majority of the gas affected communities.

Legal Aspects :

Subsequent to the disaster the Indian government through the Bhopal Gas Leak Disaster (Processing of Claims) Act in March 1985 arrogated to itself, sole powers to represent the victims in the civil litigation against Union Carbide. On behalf of the victims the Indian government filed a suit for compensation of more than 3 billion US $ in the Federal Court of the Southern District of New York. However, in May 1986 the case was sent to the Indian courts on grounds of forum non-convenience, under the condition that Union Carbide would submit to their jurisdiction. During the proceedings at the Bhopal District Court, Union Carbide was directed to pay an interim relief sum of Rs.350 crores so that the delay in the adjudication of the case does not adversely affect the claimants. However, Union Carbide refused to pay interim relief and its appeal against this decision reached the Supreme Court. On Feb 14,1989 in a sudden departure from the matter of interim relief , the Supreme Court passed an order approving the settlement that had been reached between the government of India and Union Carbide without the knowledge of the claimants in Bhopal. According to the terms of the settlement, in exchange of payment of US $ 470 million the Corporation was to be absolved of all liabilities, criminal cases against the company and its officials were to be extinguished and the Indian government was to defend the Corporation in the event of future suits. The settlement sum, nearly one-seventh of the damages initially claimed by the government, while being far below international standards is also lower than the standards set by the Indian Railways for railway accidents. There were widespread protests by the Bhopal victims against the betrayal by the government and many organizations and individuals including prominent members of the parliament supported the call to oppose the infamous settlement. Several petitions seeking review of the order on settlement were filed and the Supreme Court announced its revised judgement on October 3,1991. This final judgement upheld the settlement amount paid by Carbide but directed the Indian government to make good any shortfall during the distribution of compensation. Also the criminal cases against the Corporation and its officials were reinstated in the final judgement. The Supreme Court also directed Union Carbide to finance a 500-bed hospital for the medical care of the victims.

Compensation:

The amount paid as compensation (Rs. 715 crores) has multiplied as a result of the increase in the value of the dollar and the accruing interest. Out of this amount, about Rs. 850 crores have been paid to nearly 3.2 lakh claimants and a balance of about Rs. 1100 crores remains to be disbursed. The procedures for compensation disbursement have been tortuous and thoroughly unjust. More than 90% of the claimants have been paid a sum less than Rs. 25,000 as compensation for personal injuries out of which nearly Rs. 10,000 have been routinely deducted against interim monetary relief paid by the government from 1990. The remaining money does not half cover the medical expenses borne by the

claimants in the last several years let alone provide for future expenses. Out of the 15,168 death claims adjudicated 65% have been rejected or converted into personal injury cases where compensation sums are lower. Judges at the claim courts are completely ignorant of the medical consequences of the toxic exposure and the administration of compensation is riddled with corruption so that claimants inability to pay bribes often results in denial of compensation. In response to an official announcement for fresh registration of claims in December 1996 over 4 lakhs claims have been filed. Majority of these claims have been filed by persons residing outside the gas affected area including elite neighbourhoods and it is most likely that genuine victims will be the ultimate loser in the disbursement of compensation.

Criminal Case

A First Information Report for causing death by negligence and a number of other serious offences was registered on December 3,1984 at the local police station. On December 1,1987 the government's prosecution agency the Central Bureau of Investigation (CBI) pressed charges in the Bhopal District Court against UCC and its Asian and Indian subsidiaries namely Union Carbide Eastern (UCE), Hong Kong and Union Carbide India Limited (UCIL) respectively as well as nine officials including the then Chairman, Warren Anderson. The twelve accused were charged under sections 304 (Part IF), 326, 324 and 429 of the Indian Penal Code, with culpable homicide, causing grievous hurt, causing death of and poisoning animals and other serious offences punishable by imprisonment upto ten years and fines. The Corporation blamed a fictitious saboteur and later a disgruntled worker for causing the disaster and organized public relations campaign to distance itself from criminal liability. The CBI with the cooperation of the workers in the factory presented a strong case linking key managerial decisions to the disaster. As the proceedings in the Bhopal District Court began, Union Carbide and its officials chose to ignore the Court's summons claiming that Indian courts had no jurisdiction over them. Finally Anderson was served summons through the Interpol and on his repeated refusal to obey them, the Chief Judicial Magistrate (CJM),Bhopal proclaimed him an absconder. After the criminal immunity granted under the settlement was revoked by the October 1991 final judgement of the Supreme Court a non-bailable arrest warrant was issued against Anderson and the shares of Union Carbide in its Indian subsidiary were attached by the CJM, Bhopal. Five years have passed since the issuance of arrest warrants against the accused Corporation and its officials, yet the Indian government has not taken any steps towards seeking the extradition of the foreign accused. Union Carbide deregistered UCE, Hong Kong in 1992 and now operates in Asia through Union Carbide Asia Ltd. and Union Carbide Asia Pacific Inc.(UCAP) both wholly owned subsidiaries of the parent US based Corporation. Ramasami Natarajan the former CEO of UCE is now the President of UCAP, Hong Kong. The CBI has expressed , in Court, its inability to proceed against UCE as it has deregistered itself. On Sep. 13, 1996, in response to an appeal moved by Keshub Mahindra and other accused officials of Union Carbide India Ltd. (UCIL), the Supreme Court passed an order diluting the charges of culpable homicide to death caused by negligence (Sec. 304A of the I P C), thereby reducing the maximum sentence from 10 years to 2 years. Trial of the Indian accused are currently going on before the CJM, Bhopal, and only five of the over two hundred witnesses for the prosecution have testified in the last four

months. Meanwhile, the managers of Eveready Industries India Limited (new name of Union Carbide India Ltd.) have dismantled most of the Bhopal factory that is supposed to be under the custody of the CBI as evidence in the criminal case. In the absence of any preemptive action by the CBI , survivors organizations sought judicial and executive intervention into the erasure of the memory of the disaster as well as destruction of evidence. However, these attempts were unsuccessful.

Memorial

In a meeting of the state cabinet in end June 1988, the government decided to take back the 60 acre stretch of land in Bhopal on which the Union Carbide pesticide factory had been built. In July, the Madhya Pradesh state government committed itself to constructing a memorial at the site of the Union Carbide factory in Bhopal. The shape or content of this memorial remains to be decided.

Unresolved Issues of the Union Carbide Disaster in Bhopal

Medical :

1. Research and Monitoring

i. Lack of research on continuing gynaecological, neurological, endocrinal, chromosomal and mental health impacts of the disaster.
ii. Lack of administrative set up for carrying out long term, and possibly trans generational research activities in Bhopal.
iii. Absence of monitoring of continuing exposure related mortality and morbidity.

2. Information

i. Lack of information on health impacts available with government and private doctors involved with the medical care of the survivors.
ii. No official initiative towards disseminating information on health impacts and preventive and ameliorative measures to the survivors.

3. Health care

i. Absence of perspective and administrative set up to respond to the chronic nature of exposure related diseases.
ii. Absence of protocols for the proper treatment of exposure induced illness
iii. Absence of a system of recording health status and efficacy of medical interventions.
iv. Absence of a community based health care approach and overwhelming emphasis on hospital based treatment.
v. Negligence of Indigenous systems of medicine.
vi. Unavailability of medicines and facilities for investigations at government hospitals and clinics.
vii. Indiscriminate use of Steroids, Antibiotics, Psychotropic and symptomatic drugs.
viii. Absence of drug- free therapies such as Yoga.

4. Health Education and Public Health improvement

i. Absence of official initiatives towards health education among survivors.
ii. Lack of official initiatives towards provision of clean air, water and sanitation facilities to the survivors.

Economic, Social and Environmental Rehabilitation :

1. Information

i. No identification of individuals, families and communities most in need of economic and social rehabilitation.
ii. No information on production and training skills available locally.
iii. No information on locations requiring environmental rehabilitation.
iv. No information on technologies for decontaminating soil and groundwater poisoned by Union Carbide

2. Programmes

i. Absence of long term perspective and administrative structure to carry out long term programmes of economic and social rehabilitation.
ii. Absence of integration of economic and social rehabilitation programmes with programmes of medical care and monitoring.
iii. Lack of innovation and imagination in the design of economic and social rehabilitation programmes.
iv. No scope for participation of survivors in design and implementation of rehabilitation programmes.
v. Absence of an ecological perspective in planning and implementation of environmental rehabilitation programmes.

3. Administration

i. Absence of a well defined coordinating agency
ii. Absence of community based organization
iii. Absence of a mechanism for feed back and review
iv. Gross mis- utilization and mis- appropriation of funds Legal :

Legal Issues

1. Criminal

i Revision of charges against accused Union Carbide India Limited and its officials including Keshub Mahindra and others pending since September 1996
ii. Slow pace of trial of Indian accused

iii. Absence of criminal case against Union Carbide for contamination of ground water and soil.
iv. Absence of criminal case against Eveready Industries India Limited for dismantling and demolition of the factory.

2.Civil

i. Inadequate amount of compensation
ii. Wrongful rejection of claims
iii. Wrongful denial of registration of claims
iv. Lack of fora for review and redress
v. Utilization of over Rs 1000 crore expected to be left over after disbursal of compensation to all claimants
vi. Continuing legal liability of Union Carbide Corporation, USA and Government of India for medical, economic and social rehabilitation of survivors.
vii. Legal liability of Union Carbide Corporation, USA for contamination of groundwater and soil in the vicinity of its factory.
viii. Ensuring democratic control over the funds currently held by Bhopal Hospital Trust

Editorial: OH

THE CHALLENGE OF BHOPAL

" The growing multinational culture must be destroyed because it leads to economic chaos, increased social disparities, mass poverty and filthy affluence in coexistence, envronmental degradation, and ultinately civil strife and war.

To get a balanced, rational development and to preserve the environment, a new development process is needed. The biggest intellectual and political challenge of our times is to articulate and demonstrate this new kind of development."

— A statement of shared concern-Citizens report on state of Indias Environment 1982.

Its six months since the worst industrial and environmental disaster in recorded history. Bhopal has not only been a nightmare for those who were there on the night of 2/3 December, 1984. It is also a portent of events to come.

World Environment Day (5th June) has come and gone. There have been the usual meetings, seminars and lectures: the usual ~~xxxxxxxxx~~ lip-service to ecological sensitivity, the usual narrations of the health and social hazards of environmental pollution; the usual pious recommendations of what can and should be done.

How many more Bhopals will we need in this county before we are shaken from our apathy?

...2...

- from our callousness to our disadvantaged and exploited fellow human beings who are alway's the worst hit in such disasters.
- from our insensitivity to nature, our forests, our rivers and our land.
- from our insensate rush for chemicalising and technologising our lifestyles.
- from our race for profits even at the cost of the health of our workers, our people.

The medical community in India will be increasingly called upon to respond to the medical and health problems caused by more and more ecological disasters. What will our response be?

Will we see every disaster as a chance to refine our clinical skills, satisfy our charity and welfare urges, exploit the research potential for career development and use the opportunity to ask for more and more sophisticated gadgetry for our institutions?

Or will we be challenged by these disasters to raise our voice collectively to oppose the unhealthy trends in our society; to use our knowledge and social potential to support the growing awareness for healthier and more egalitarian social system; to use our research skills to strengthen and conscientise our fellow human beings to an increasing health and ecological awareness.

The dilemma of a man who enters a room to find a tap running and a wash basin overflowing, faces us today. Will we choose to be floor moppers or tap turners off?

....3...

Overpowered, compromised and hypnotised by the products and high pressure sales tactics of the multinational pharmaceutical industry, our sensitivities have been so dullened that we are quite content to be merely ' floor moppers'. Can we ever be tap-turners off? The International movement of physicians for prevention of Nuclear war is a thought provoking example showing that if we want to, we can.

Bhopal too is a challenge? So are many other more insidous developments in our country. The growing investment in nuclear - energy now discredited as an energy resources in the West, the gradual take over of the cottage industry in food by big business, the increasing professional vested interest in over investigation - each of this though different from the other has a growing similarity representing either a subservience to the profit motive or an insensitivity to health hazards or both. We feature some of these aspects in this bulletin. We also feature investigation in Bhopal which raise some of these issues for our readership.

Minimata, Seveso, long island were too distant to make any impact. Amlai, Chembur, Handigodu, Harsiar, Zuari, Nagda, Mavoor, Silent valley, Thal-vaishet here not stimulated us either. Will Bhopal do so?

Occupational Medicine = Protection and Promotion of Health

Occupational Health Services

Designing the World of Work

Work Layout
- project schemes
- work procedures
- supply with working equipment
- work organization and procedures
- ergonomic design of the work place
- safety and ergonomic audits of machinery and appliances
- rules on working hours
- work rhythms

Work environment
- climate
- working materials and chemical substances
- noise
- lighting
- radiation
- vibration
- colour design

Work analysis
- workplace inspection
- measurements and monitoring
- occupational risk assessment
- evaluation and registration of exposure levels
- assessment of multifactorial exposures
- substitutes for hazardous substances (Commission on Substitutes for Hazardous Materials)
- stress and strain assessments
- personal protective equipment (choice and try-out)
- workplace related skin protection scheme
- workplace and job register
- health circles
- employees surveys

Assessment of Health and Occupational Health Care Counselling

Examinations
- pre-employment
- pre-placement
- surveillance tests
- periodic health assessments
- youth protection check-ups
- general occupational medical check-ups and return-to-work examinations
- specific occupational medical check-ups
- check-ups for test drivers
- health preparations for international travel
- health check-ups, health coaching
- screening programmes
- laboratory (blood, urine, X-ray, opto- and audiometry, E.C.G., spirometry, stress testing)

Rehabilitation at plant level
- employment adapted to individual capacities
- redesign of workplaces, choice of appropriate technical auxiliaries
- job transfers
- changes in work organization and devices
- gradual reintegration
- in-plant rehabilitation centre
- rehabilitation sports
- initiation of occupational retraining measures
- regular meetings with LVA (Social Insurance Pension Funds for Hourly Employees)
- regular meetings with BKK (Volkswagen Health Insurance Fund)

Primary Care, Accident and Emergency Medicine

Initial treatment
- first-aid stations: to give first aid in cases of health indispositions, diseases and work accidents
- on-site clinics: first medical aid, general medical counselling
- emergency response service: emergency medicine, first-aid training, emergency medical exercises (mega-code)
- physiotherapy, neuraltherapy
- psychological services/offering short term counselling and referral (e.g. substance abuse)
- laboratory (blood chemistry, function tests)
- medical care during test driving

Care for specific groups of employees
- adolescent employees
- elderly employees
- women
- pregnant women
- disabled employees
- mentally disturbed employees
- employee assistance programmes (e.g. counselling in life crises)
- employees, suffering from coronary artery disease
- employees, suffering from skin diseases, atopic individuals
- athletes
- care for travellers abroad
- chronically ill employees (epileptics, diabetics, rheumatics)

On-site hygiene
- vaccinations
- education and counselling in infectious diseases
- investigation of known contacts (e.g. in case of tuberculosis)
- food and water hygiene

Information, Education Behavioural Counselling*

- healthy nutrition
- smoking cessation
- kinetotherapy
- remedial exercise
- training programmes for the prevention of back disorders and for persons with a sedentary work
- training in safe manual materials handling
- training for work at video display terminals
- autogenic training
- muscle relaxation programme
- wellness programmes
- stress management
- self-care education
- substance-abuse prevention
- individual psychologic counselling
- lectures
- training courses and informative events
- brochures
- action weeks
- rotating exhibitions
- specific consultation hours

*in co-operation with the Volkswagen Works Sickness Fund and duly qualified technical personnel

VOLKSWAGEN
Gesundheitswesen

Occupational Medicine = Protection and Promotion of Health

Occupational Health Services

Health Policy/ Planning and Strategy

- co-ordination of Group and Branch health activities
- standards, guidelines
- environmental medicine
- ergonomics
- tropical and travel medicine
- medical care during test driving
- health coaching
- assisting in experts' bodies, commissions and committees
- participation in congresses, conferences and symposia
- participation in research projects
- initiating and conducting inter-company workshops
- presentations in national and international conferences
- scientific publications
- consulting and expert testimony
- research in co-operation with scientific institutions case-control studies/cohort studies
- teaching at technical colleges and universities

Documentation, Evaluation and Reporting on Health

- collecting and registrating data relevant to health and searching for early signs of impaired health (first-aid register, medical records)
- stress analyses for main occupational risks
- registration and evaluation of epidemiologic data
- epidemiologic surveys
- monitoring changes in occupational risks
- management of workers' compensation in case of occupational diseases
- impairment and disability evaluation
- investigating work-related impacts on sick leave and mortality
- evaluating the dose-response relationship of hazardous chemicals at the workplace
- structural analysis of absenteeism
- extended analysis of sick absence and work accidents
- occupational medical stress and strain register
- reporting on health
- stastistical surveys

Postgraduate education

- emergency medical training programme (mega-code)
- attending specific occupational medical meetings, congresses and seminaries
- sit in at hospitals
- general medical training at regional level
- training workshops by the Employers' Liability Insurance Associations
- postgraduate education within the specialized training for an occupational medicine physician
- continuing education of all on-site healthcare professionals

Partners of Co-operation

Within the company

- human resources
- production
- works council
- occupational safety department
- planning
- research and development
- environmental protection
- coaching company
- commissions
  - on occupational safety
  - on planning
  - on substitutes for hazardous substances
  - working group on occupational dermatology
  - on ergonomics
  - on the mediation of jobs adapted to the capacities and disabilities of handicapped employees

Externally

- Volkswagen Works Sickness Fund
- Employers' Liability Insurance Associations
- health insurance funds, Social Insurance Pension Fund for Hourly Employees (LVA), German Federal Social Insurance Institution for Salaried Employees (BfA)
- hospitals, doctors
- rehabilitation centres
- universities, specialitists' associations
- Medical Associations
- German Red Cross and other relief organizations
- municipal rescue services, disaster control
- labour court, social court
- German Federal Institute for Occupational Safety and Occupational Medicine (BAuA), German Federal Ministry of Labour and Social Affairs (BMA), Institute of the Employers' Liability Insurance Associations for Occupational Safety (BIA), World Health Organization (WHO), International Labour Organisation (ILO), German Society of Occupational and Environmental Medicine (DGAUM), Central Organ of the German Statutory Accident Prevention and Insurance Institutions in Industry (HVBG), Professional Association of German Occupational Physians (VDBW)

draft

OH - 1

Dear Mr. Vasudevan Nair.

I am forwarding a letter regarding Bhopal from a group very well known to us. As you know the health impact of the Bhopal disaster remains a continuing tragedy and an issue of gross social injustice. I therefore thought it would be appropriate for Health Action to feature this update. You could directly contact Mr. E. Deenadayalan at "The other Media" (email : admn7@del3.vsnl.net.in) in this connection. They have been consistently involved with this issue over many years. Through the Medico Friend Circle Ravi and I were also involved.

Many thanks and with best wishes.

C.C.Fr. Sebastian Ouseperumbil, Director, CHAI

Dear Deena.

Greetings from Community Health Cell!

a) Thank you for the note regarding Bhopal update. We are certainly in support of the campaign for Justice in Bhopal. Health Action is a journal with a fairly wide all India circulation. I have forwarded your mail to the editor in charge. Mr. Vasudevan Nair recommending publication. I am a member of the editorial board. His email is <chai@pol.net.in>. He will probably contact you. Do directly contact him for any changes, illustrations etc.

b) I wonder if you have received my confirmatory letter regarding the fact finding mission regarding primitive tribes in the Andaman and Nicobar islands.

c) A local media person is visiting CHC tomorrow to prepare a film on Bhopal. Shall give him all the current information.

d) The issue should be raised at the Jan Swasthya Abhiyan which meets in Bhopal on 15th July. A protest could be planned with memoranda.

Ragards,

Thelma

Subject: Appeal for support to the campaign for Justice in Bhopal
Date: Wed, 26 Jun 2002 17:55:13 +0530
From: "The Other Media" <admin@del3.vsnl.net.in>
To: "Mohan Mani" <nbamm@vsnl.net>, "Unnikrishnan (Dr.)" <unnikru@nda.vsnl.net.in>, "Thelma Narayan (Dr.)" <sochara@blr.vsnl.net.in>, "Srinivas Murthy" <murthy@nimhans.kar.nic.in>, "K.P. Sasi (Netwaves)" <sasi36@rediffmail.com>, "SCMI" <scmi@bgl.vsnl.net.in>, "Madhu Bhushan" <awhrci@vsnl.com>, "Visthar" <vistar@bgl.vsnl.net.in>

Bhopal file — laser print on other side

Part 1.1 Type: Plain Text (text/plain) Encoding: quoted-printable

To Mr Vasudevan Nair, Editor-in-charge, Health Action

Dear Mr Vasudevan Nair,

I am forwarding a letter regarding Bhopal from a group very well known to us. As you know the health impact of the Bhopal disaster remains a continuing tragedy and an issue of gross social injustice. I therefore thought it would be appropriate for Health Action to feature this update. You could directly contact Mr E Doonadayalan at "The Other Media" (email & phone) in this connection. They have been consistently involved with this issue over many years. Though the Medico Friend Circle has also been involved.

Rest is well.

Many thanks in anticipation

JN 27/6.

cc. Fr Sebastian Kureppumbhil, Director CHMI

Dear Deena,

Greetings from CHC!

a) Thank you for the note regarding Bhopal update. We are certainly in support of the Campaign for Justice in Bhopal. Health Action is a journal with a fairly wide all-India circulation. ⇒ I have forwarded your mail to the editor-in-charge Mr Vasudevan Nair & recommending publication. I am a member of the editorial board. The email id is ——— He will probably contact you. Do directly contact him for any changes, illustrations etc.

b) I wonder if you have received my confirmatory letter regarding the fact finding regarding pesticide issues in the Andaman & Nicobar islands.

Dr. TN

27/6/02 c) A local media person Rajarshi D'Rozario 27/6 is meeting CHC tomorrow to prepare a piece on Bhopal. I shall pass him all the current information.

d) The above should be raised at the forthcoming Abhiyan which meets in Bhopal on 15th July. A protest could be planned with memoranda.

6/27/02 11:08 AM

David Selvaraj, Visthar
URGENT
An Appeal for support to the Campaign for Justice in Bhopal

Dear friends,

Many of you will remember the December '84 chemical disaster in Bhopal when half a million people were surrounded by deadly poison clouds in their sleep. The disaster killed more than 8000 in its immediate wake. The death toll today is well over 20, 000 and rising with more than 30 survivors dying every month. The poisons entered the blood stream of the exposed people and damaged almost every organ of their bodies. Today well over 120,000 survivors are in desperate need of medical attention for chronic exposure induced diseases. Tens of thousands of children born to exposed parents suffer from growth retardation and worse. An overwhelming majority of the exposed people earned their living through hard labour. Thousands of families are on the brink of starvation because the breadwinners are too sick to work. Over 20,000 people are today forced to drink water contaminated with cancer and other disease causing chemicals that have been recklessly dumped and have seeped in to the ground water.

As you know, the principal author of this continuing disaster in Bhopal- the worst of its kind in the world- is Union Carbide Corporation. There is substantial evidence that this American multinational with complete control over the pesticide factory in Bhopal, was deliberately negligent in its location, design, operation and maintenance. Two years before the disaster, the corporations safety experts warned of a "potential for the release of toxic materials" in a business confidential memo. Warren Anderson, the company's chairman and other senior executives ignored the warning and went ahead with reducing plant personnel, shutting down vital safety systems and keeping people in the neighbourhood in the dark about the deadly chemicals stored, used and produced in the factory.

The Indian government has been in cahoots with the corporation before, during and in the aftermath of the disaster. Government agencies have actively colluded to underplay the number of the dead, grossly under-assess the extent of injuries and suppress medical information potentially disadvantageous to the corporation. In February 1989, the Indian government entered in to a collusive settlement with Union Carbide without the knowledge of, let alone consultation with, the victims of Bhopal. The settlement cost the corporation 43 cents per share and 95% of the victims got a paltry sum of 25 thousand rupees for injuries they are likely to suffer all their lives. From this amount the government took back the money it had paid as relief to the victims - about rupees ten thousand from every individual. In the distribution of compensation people were denied their legal entitlement to the interest on the compensation sum. This has resulted in a balance of well over rupees 1300 crores in the settlement fund after almost all victims have been paid.

The Indian government's complicity in the crimes of Bhopal is most glaring in its willful neglect in prosecuting Union Carbide and its officials. Charged with homicide and other serious offences Union Carbide and its officials are absconding from the proceedings in the Bhopal district court for the past ten years. Different political parties have come to power at the centre in the last ten years. None of these has taken the tiniest step to extradite the corporate culprits. According to a secret internal memo, Indian government's deliberate inaction stems from concerns that it would "jeopardize the investment climate". As you can imagine, in addition to such systemic reasons, inducements in the form of deposits in individual Swiss bank accounts can not be ruled out.

In February 2001, Union Carbide in a "vanishing act" merged into the Dow Chemical Company of Midland, Michigan, USA. Dow, now the largest chemical corporation in the world, has inherited the medical, social, environmental and criminal liabilities arising out of the Bhopal disaster. Defying established legal procedures and judicial principles in USA and India, Dow's pr speak on Bhopal is that it does not hold itself liable for a

factory they never operated in a place they never have been. Continuing with Union Carbide's tradition of liability evasion, Dow refuses to provide for long term health care, income support to the destitute and the disabled and cleaning up the ongoing contamination.

Meanwhile in the longest struggle of its kind the survivors of Bhopal continue to struggle facing the severest of odds. Members of the three organizations of survivors majority of whom are women continue to meet every week to talk, plan and agitate. Ever since the merger of Union Carbide with Dow, hundreds of women survivors have twice stormed the headquarters of Dow, India in Mumbai. In response Dow has sued for 77,000 Rupees against 17 persons and organisations for damages done to its office. Faced with the agitation of Bhopal survivors and their supporters in the USA Dow today promises to offer concrete 'humanitarian" programs for the victims of Bhopal. This could as well be pr speak. Quite clearly the surest way to get Dow to fulfill its responsibilities in Bhopal is to establish its accountability [inherited through the merger] in the ongoing criminal case. This is the job of the prosecution - the Central Bureau of Investigation. And this is what the CBI was told to do by the Chief Judicial Magistrate of the Bhopal district court during the hearing on the case on April 10, 2002.

Two current happenings have overtaken the ongoing pressure building on Dow. In the hearing on the criminal case on the disaster on May 23rd, 2002 when the CBI was supposed to present its results of investigation into the Dow-Carbide merger - it presented an application that would in effect end the entire case against Warren Anderson and by extension against Union Carbide/Dow.

In the application the CBI, without doubt following instructions from the highest authorities in the government, sought to dilute the criminal charges from homicide 304 [ii] IPC , punishable by 10 years imprisonment and fine to negligence 304[a] IPC, punishable by maximum two years imprisonment or fine. Through this second betrayal of the Bhopal people, the Indian government seeks to convert the worst industrial massacre in history in to a crime equivalent to a car accident. Worse still, if the CBI's application is granted there would no longer be any legal basis left for extraditing Anderson or the authorized representatives of the corporation. The Indo-US extradition treaty does not cover car accidents. The CJM, Bhopal is expected to deliver a decision on CBI's application during the next hearing on the criminal case on July 17-18, 2002.

The other current burning issue of Bhopal also demonstrates the utter apathy of the central government towards the victims of Union Carbide. On June 7th the Group of Ministers [GoM] on Bhopal in the central government declared that the residents of the 20 municipal wards of Bhopal hitherto considered unaffected by the disaster would be given compensation from the balance of the settlement fund. Given that there has been no evidence of exposure among these residents [in fact the ICMR drew its sample of unexposed persons from these wards] the only qualification the residents seem to have is that they are mostly Hindus. The GoM took this decision on a demand put forth by Uma Bharati the MP from Bhopal better known for her cheerleader role in the demolition of the Babri mosque by the saffron goons in 1992. The cynical decision of the GoM is a blatant attempt to rob the people of the 36 gas affected wards to appease the Hindu vote bank. Its Gujarat all over again.

The state government in its turn has opposed the demand of the BJP MP. This opposition articulated by the Minister for Gas Relief, however, is not motivated by any respect for the legal rights of the victims. The state government would that the funds are handed over for it to spend on provision of drinking water supply, disposal of residual chemicals and other activities. Funding these vital activities, as outlined by the survivors, is strictly the responsibility of Dow Chemicals. Four years back the state government allowed Union Carbide to abandon the factory site without paying for its rehabilitation. Today it seeks to rob the gas victims to bail out Dow. Additionally, the Comptroller and Auditor General's yearly reports on MP government's mishandling of moneys meant for gas victims make it the last agency to be entrusted with public funds for this purpose. The GoM on Bhopal is likely to finalize its decision in mid-July.

Last year in Britain a senior civil servant put a memo around her department. Saying "today is a good day to

bury bad news" - it was September 11th. In similar vein both the central and state governments hope that the noise of the war will bury the news of the betrayal. This and the fact that the issue of Dow's criminal and other liabilities are reaching a decisive stage seem to have influenced the timing of the government's latest decisions on Bhopal.

The survivors of Bhopal have decided to oppose the two decisions of the Indian government both in the court and through a relay hunger strike in New Delhi starting from June 26th. Their demand is that:

i. The Home Minister direct the CBI to withdraw its application and immediately move on extradition of Anderson and the representative/s of Union Carbide from the USA.

ii. The GoM take back its decision to distribute compensation to the residents of the 20 wards from the balance of the settlement funds and make arrangement for payment of interest to the victims of the 36 wards with immediate effect.

Friends, through this letter we seek your support to this last ditch battle of the survivors. The dilution of charges against Union Carbide and its officials is a license granted to multinational corporations to kill and maim the ordinary people in this country. More than ever before your support is critical to defeat the evil designs of our pseudo-nationalist government.

Please support the current struggle for justice in Bhopal by :

1. Visiting the site at Jantar Mantar (if you are in Delhi) where 200 survivors of Union Carbide gas disaster from Bhopal are sitting in Dharna and 50 of them are on relay hunger strike.

2. Organizing media visible hunger strikes in solidarity of the Bhopal survivors wherever you are.

3. Publicizing the current issues of Bhopal through all means available to you.

4. Organizing signature campaign against the decision of the Home Ministry to dilute criminal charges and that of the Group of Ministers on Bhopal [c/o Ministry of Chemicals and Fertilizers] to rob the victims of their legal entitlement.

5. Joining in the email / fax action at the corpwatchindia and ..... Web sites.

Please contact us at any of the addresses below and send your ideas / suggestions and confirmation of participation.

Bhopal
Rashida Bi, President, Bhopal Gas Peedit Mahila Stationery Karmachari Sangh

House no. 12, Gali no. 2, near Naseer Masjid, Bag Umrao Dulha, Bhopal

Balkrishna Namdeo. President. Gas Peedit Nirashrit Morcha,

A - 542, Housing Board Colony, Aishbag, Bhopal Tel : 0755 - 757619

Abdul Jabbar, Convenor, Bhopal Gas Peedit Mahila Udyog Sangathan

51, Rajendra Nagar, Bhopal, Tel : 0755 - 242727

Satinath Sarangi, Member, Bhopal Group for Information and Action

B-2 / 302, Sheetal Nagar, Berasia Road, Bhopal contact Tel: 0755 - 730914

Contact email : justiceinBhopal@yahoo.co.in

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Health Effects of the Dec. '84 Union Carbide disaster in Bhopal

Death Toll

In Nov '89 the Dept of Relief and Rehabilitation placed the death toll at over 6000. In December 1992 the official agency for monitoring the deaths among the persons exposed to the gas was wound up. Data published by the Department of Relief and Rehabilitation in December 1998 shows that in the year 1997 the death rates were 10 4 in the gas-exposed population compared to 6.07 in the unexposed population. Based on an exposed population of 5,72,242 [ICMR's estimate] this works out to over 2000 deaths attributable to exposure in the year 1997 alone.

Overall Diseased

Symptomatic disease in the population surveys conducted by the ICMR indicate that persons who were ill was higher in the exposed area [26%] as compared with the control area [18%] when assessed during the period November 1988 to March 1990.

Results from the survey carried out by the International Medical Commission on Bhopal show that a large number of people reported general health problems [exposed 94% vs unexposed 52%] and episodes of fever [exposed 7.5 per year vs unexposed 2.5 per year]. Respiratory, neurological [nervous system], psychiatric [mental illness] and ophthalmic [eye related] symptoms also showed a strong gradient by exposure category.

Problems with the eye

The intensely irritating effect of MIC [methyl isocyanate] on the eyeball resulted in severe burning, watering, pain and aversion to light in the eye. Examination showed redness and ulcers in some parts of the eye and lid swelling.
Examination also showed discrete lesions in a band across the inner aspect of the eyelids, whitish spots, swelling and some pigmentary deposition on the eye.

Anderson et. al. performed a survey of exposed and control populations 3 years after the disaster and found aversion to light, burning and watering sensation, signs of red eye, glazing of the eye. Their findings indicated an increased risk of eye infections, hyper responsive phenomena, excess cataracts and resolution of the ulcerations in the eye in exposed persons. These phenomena have been characterised as the "Bhopal eye syndrome".

Though there is no evidence that severe damage to the eye's external and internal structures has occurred, the single acute exposure seems to have resulted in a chronic inflammatory process.

Respiratory toxicity

Acute symptoms of the respiratory tract were mainly due to the irritant action of MIC on tissues. Because MIC is moderately soluble in water, lesions were seen in both the upper and lower respiratory tract. Though isocyanates are known to be allergenic in the lung, the respiratory toxicity of MIC appears to be primarily due to its irritant nature.

Follow up studies with lung biopsies done six months after exposure showed evidence of thickening of tissues and induration of air sacs. These findings were similar to those in several animal studies revealing the close association between animal data and clinical findings in Bhopal victims.

Autopsies on 300 victims revealed severe death of cells resulting in lesions in the lining of the upper respiratory tract as well as in the air sacs and lung capillaries. Enlarged and fluid filled lungs, consolidation, internal bleeding and acute inflammation of the lungs were seen.

Reproductive toxicity

Concerns that the gas leak had effects on reproductive health were raised early in 1985 when reports indicated that monthly periods cycle disruption, whitish discharge and painful periods had occurred in gas-exposed women.

An epidemiological survey by Varma showed spontaneous abortion loss and infant death to be very high in gas-exposed women.

NR Bhandari et al. documented significant increase in spontaneous abortion, Infants and infant death rate in the womb in a pregnancy outcome study carried out in 18, 978 households.

Fetotoxicity [ill effects on the unborn child] of MIC has been confirmed through animal experiments.

Genetictoxicity

Chromosomal studies done two and half months after the gas leak to evaluate genetic damage showed significant increase in number of breaks and gaps in the exposed subjects.

Cytogenetic studies done 3 years after exposure showed higher frequencies of chromosomal alterations in the exposed group.

Immunotoxicity

Immune function was studied in exposed subjects by Saxena et al. two and half months after exposure. No difference in mean immunoglobulin levels [part of bodies defense system] was found compared to controls. The defense cell population was found to be less than half of that found normally in the Indian population. Significant depression of fighter activity of defense cells was found as compared to controls.

Psychological and Neuro-behavioural toxicity

Srinivasamurthy and Isaac noted Post-traumatic stress disorder, pathological grief reaction, emotional reactions to physical problems and exacerbation of pre-existing psychiatric problems among gas victims.

Neurobehavioural tests were conducted on 350 exposed subjects two and a half months after the accident. Auditory and visual memory, attention response speed and vigilance were found to be significantly impaired.

Neuromuscular toxicity

Neuromuscular symptoms in Bhopal survivors have persisted since the gas leak. The symptoms are mainly tingling, numbness, a sensation of pins and needles in the extremities and muscle ache.

Anderson et. al. evaluated the effects of MIC on rat muscle cells in culture. At lower doses, the formation of muscle fibers was prevented. At higher doses, death of cells that help in muscle building.

In summary, the exposure to toxic gases during the Bhopal disaster has resulted in serious, wide ranging and long term adverse effects on the health of about 500,000 people who were living in that area. Following chromosomal damage, it is also effecting the next generation born to those exposed.

[This note has been prepared from reports of studies undertaken in Bhopal]

- Materials reviews → Govt/Public lib/research institutes in this field/ NGOs, Medical colleges & Dental, IMA

→ Refining a problem

Research/awareness/training

IV NIAS Course
Indian Administrative Service Officers
25-29 August 2003

"Disaster Management"

Programme

International and Strategic Studies Unit
National Institute of Advanced Studies
Bangalore

Monday, 25 August

0945 Welcome
Prof. S. Rajagopal, Homi Bhabha Visiting Professor, NIAS

0950 Iroductory Remarks & Inauguration
Prof. R. Narasimha, Director, NIAS

1000 **Health and Humanitarian Challenges in Disaster Response**
Dr. Ravi Narayan, Co-ordinator
Peoples Health Movement Secretariat (Global), Community Health Centre, Bangalore

1115 Tea/Coffee

1130 **Case Study of Health Initiatives: Earthquake, Flood and Cyclone Disasters and Aftermath**
Dr. Sanjiv Lewin, Coordinator
St. John's National Academy for Sciences, St. John's Medical College, Bangalore

1300 Lunch

1430 **Psycho-social Challenges of Disasters**
Dr. Srinivasa Murthy, Professor
Department of Psychiatry, National Institute of Mental Health and Neurosciences
Bangalore

1600 Tea/Coffee

Tuesday, 26 August

1000 Floods and Cyclones
Ms. Rita Missal, Social Development Officer
United Nations Development Programme , Bhubaneswar

1115 Tea/Coffee

1130 **Case Study on "Orissa Floods"**
Ms. Rita Missal

1300 Lunch

1430 Interactive session: Participants, Speaker and
Mr. N. Hari Krishna, OXFAM UK (Hyderabad)

1600 Tea/Coffee

1615 **Drought Management in Rajasthan**
Mr. Madhukar Gupta, Divisional Commissioner Commissioner's Office
Bikaner

Wednesday, 27 August

1000 **Earthquakes: An Overview**
Prof. R N Iyengar and Prof. B. K Raghu Prasad
Department of Civil Engineering, Indian Institute of Science, Bangalore

1115 Tea/Coffee

1130 **Case Study on "Gujarat Earthquake"**
Prof. R. N. Iyengar and Prof. B. K. Raghu Prasad

1300 Lunch

1430 Interactive session: Participants, Speaker and
Ms. Prema Gopalan, Director
Swayam Shikshan Prayog, Mumbai

1600 Tea/Coffee

1615 **Gujarat Earthquake-2001: Initial Impressions & Response-Lessons for the Future**
Mr. Madhukar Gupta

Thursday, 28 August

0900 **Chemical Disasters: An overview**
Mr. N. R Krishnan, Former Secretary, Ministry of Environment and Forests, Government of India

1015 Tea/Coffee

1030 **Case Study on "Bhopal Tragedy"**
Mr. N. R. Krishnan

1130 Interactive session: Participants, Speaker &
Dr. Thelma Narayan, Coordinator
Community Health Cell, Bangalore

1300 Lunch

1400 **Emergency Preparedness Plan of Department of Atomic Energy**
Mr K. Muralidhar, Secretary-Atomic Energy Commission & Head, MSG, Mumbai

1500 Visit to Regional Remote Sensing Service Centre, Department of Space
Banashankari, Bangalore

Friday, 29 August

0915 **Disaster Management: Organisational Structure – India and Abroad, a Comparison**
Maj. Gen. M. K. Paul (Retd), Controller, NIAS

0945 Discussion

1000 **Disaster Management at Railway Accident Spots**
Mr. G. Ram Mohan, Former Commissioner, Railway Safety Southern Circle-Bangalore

1115 Tea/Coffee

1130 **Case Study of "Railway Accidents"**
Mr. G. Ram Mohan

1300 Lunch

1430 Interactive session: Participants, Speaker and
Mr. Max Martin, Media Expert, Bangalore

1600 Tea/Coffee

1615 Participants feedback

1645 Certificate distribution

IV NIAS Course
Indian Administrative Service Officers
25-29 August 2003

Disaster Management

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National Institute of Advanced Studies
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CHEMICAL DISASTER MANAGEMENT

PART II

The Bhopal Gas Tragedy

The Bhopal Gas tragedy occurred on the night intervening 2-3 December, 1984. About 40 tonnes of poisonous methyl isocyanate (MIC) vapour escaped from the pesticide manufacturing facility of Union Carbide (India) through a 33 metre tall stack over a two hour period. Aided by a gentle wind, the vapour, which being heavier than air and would have otherwise settled down, got dispersed over an area of about 40 sq.km. in a south-southeasterly direction from the plant (**Annexe 1**). An officially estimated 2500 people died within hours of exposure to the noxious gas and about ten times that number were physically affected to varying degrees from temporary eye irritation and racking cough to permanent pulmonary disability. Years after, birth defects were also reported in children born after the disaster. Unofficial estimates placed the number of dead at 8000 or even more and the affected population at around two lakhs. Most of the dead and affected were poor slum dwellers. A large number of cattle also perished.

BACKGROUND:

Union Carbide (India) Ltd. (UCIL) was a subsidiary of Union Carbide Corporation (UCC) of US in which the parent company held 50.9% of stock. In 1969, UCIL was given a licence to manufacture carbaryl family of pesticides using imported raw materials. In 1979, the company was licensed to make the intermediates (MIC and alpha naphthol) too and full indigenous production commenced in 1980.

Paper delivered by Mr Krishnan IAS, retd as NIDM course on disasters - Aug 2003

for lib - Bhopal resource file.

In 4/9

Among the carbaryls, the most well known was Sevin having diverse applications in protecting plantation crops, horticultural produce and oil seeds. As an household insecticide, Sevin was effective against cockroaches, mosquitoes , bugs, fleas etc. It was heralded as just the versatile chemical needed on-form and off-form.

In the initial stages, the Bhopal unit was managed by foreign technical personnel but later, they were replaced by Indian engineers who had been given extensive training at the UCC's, West Virginia plant in the US. The US plant which was also manufacturing Sevin was considered to be a model in chemical industrial safety. A general feeling of confidence that nothing could go wrong at the Bhopal plant was prevalent among the engineers and technicians as the plant was modelled after the US one.

UCIL was licensed to manufacture 5250 tonnes of carboryl pesticides per year. Actual production, however, remained far below this level due to poor demand.

Year	Production (Tonnes)
1981	2704
1982	2308
1983	1657

The carbaryl pesticides were facing stiff competition from another group of pesticides known as synthetic pyrethroids. UCIL reportedly suffered a loss of Rs.5 Crores in the first ten months of 1984. The fall in demand and the resultant idle capacity and losses forced the company to consider dismantling the facility and shifting it out of India. This also led to reduction of staff and other cost-cutting measures.

PROPERTIES OF MIC

MIC, a colourless liquid, has a low boiling point of 39 degrees C at normal atmospheric pressure and has a flash point of –7 degrees C. As these physical characteristics show, MIC is highly volatile. It is very reactive, chemically and biologically, because of the presence of the isocyanate group in the molecule and its affinity for water. The affinity for water accounts for MIC's effects of causing irritation in the eyes, cough and draining the lungs of fluids. Since MIC vapour is 2.2 times heavier than air, it expels air from the lungs on inhalation leading to choking and death. From the health hazard point of view, MIC's high hazard potential is shown by its low threshold limit value (TLV) of 0.02 part per million (ppm) in air compared to 50 for carbon monoxide, another well known air pollutant.

MIC STORAGE

The MIC produced in Bhopal was being stored in three underground stainless steel tanks numbered 610, 611 and 619 as shown in **Annexe** 2. These tanks were encased in concrete and refrigerated to ensure temperatures close to 0 degree C. In any case, the temperatures were not to go above 15 degrees C. An inert atmosphere of nitrogen at 2 psi pressure was to be maintained in the tanks. This pressure had to be raised to around 10 psi to force the liquid MIC out of the tanks to the process area for further reaction with alpha napthol.

A technical feature of the plant was that to vent any excess MIC going into the production process, a Process Vent Header (PVH) connected to a Gas Scrubber was provided. The scrubber arrangement provided for neutralisation of MIC by caustic soda solution. Similarly, in

the event of a need to ease the pressure in the MIC tanks, that is, if the pressure exceeded 40 psi, a rupture valve was provided to make the MIC flow through a Relief Valve Vent Header (RVVH) to the gas scrubber where the gas would be neutralized by caustic soda. The PVH and RVVH were connected by a jumper line which normally remained closed. Finally, all escaping gases were to be ignited in a flare tower.

Instrumentation panels were provided in a control room to monitor pressure, temperature etc. in the MIC tanks. A siren system was available to provide alarm to the general public.

EVENTS BEFORE 2, DECEMBER, 1984

The refrigeration unit meant to keep down the temperatures in the MIC tanks close to 0 degree C were decommissioned in June, 1984. Instruments recording pressure, temperature etc. and their control systems were found to be faulty needing repairs. On 22, October, the MIC plant was shut down for maintenance. On 25, November, the flare tower was disconnected and the jumper line between PVH and RVVH was opened. The opening of the jumper line was a modification done at the instance of the engineers of the UCC in the US.

On 26, November, work on restarting the manufacture of pesticides began with the deficiencies noted above remaining as they were. The restarting operation began with pumping MIC out of tank 610 (which had about 40 tonnes of MIC) to the process area but it was noticed that pressure could not be built up. On 2, December, another attempt was made, this time to force MIC out of tank 611 which had 42 tonnes. But this attempt also failed due to non-building up of pressure. Tank 619 had been kept empty.

EVENTS – LATE EVENING 2, DECEMBER, EARLY MORNING 3, DECEMBER.

The failure to get MIC forced out of the tanks led the Plant Supervisor to order washing of the lines with water to remove any blockage. This order was complied with by the Plant Operator without observing the safety procedure of isolating the lines from the MIC tanks by inserting a water arresting device called 'slip blind'. Incidentally, the Supervisor, who had been posted recently, had no experience of handling MIC.

The sequence of events from this point were as follows:

*"09.15 pm Washing of relief valve lines without isolation

09.30 pm Operator notices the lines are blocked

10.00 pm MIC plant supervisor orders washing to continue

10.20 pm Pressure in the tank 610 is 2 Psi. 610 failed to get pressurised

10.45 pm Shift changed

10.45 pm Water entry from RVVH to PVH and the tank

11.00 pm Operator logs pressure in tank 610 as 10 Psi

* B.BOWNDE R :INDUSTRIAL HAZARD MANAGEMENT—AN ANALYSIS OF THE BHOPAL ACCIDENT .. Project Appraisal, Volume 2, number 3, September 1987, p.160.

- 11.30 pm to 11.45 pm First leak of MIC detected. Plant Supervisor notified about the high pressure and MIC leak

11.50 pm MIC operator sees yellow drip from RVVH

12.00 am Supervisor ordered stopping of washing operations

12.15 am Tea break for operator

12.20 am Attempt to start the vent gas scrubber pump

12.25 am Plant superintendent, on being informed about the leak, arrives t the spot

12.30 am Pressure gauge reading over range. Concrete tanks get very hot.

12.40 am MIC operator reports escape of MIC through the vent line at 33 meters

01.00 am Toxic Gas Alarm was alerted, but switched off. Police official on patrol reports to police control room that something had gone wrong at UCIL.

01.15 am Police control room informs the city police chief.

01.30 am UCIL staff when telephoned report that everything is normal

01.45 am Additional District Magistrate informs the Works Manager of UCIL at his residence about the leak.

02.00 am to 02.30 am The safety valve reseated, but 40 to 45 tonnes of MIC escaped before that. Public siren was restarted at full blast."

*((UCC, 1985, Technica 1985; Quoted in B. Bowonder "Industrial Hazard Management: An Analysis of the Bhopal accident", Project Appraisal, September 1987)

3, December, 1984 (early morning).

The gentle cold wind carried the MIC vapours in a south-southeasterly direction from the plant over shanty colonies, Bhopal Railway Station and beyond. People who were sleeping with doors open or in huts with no doors and on the railway platform perished on inhaling the gas. Those who had kept their doors closed survived but those who on hearing the cries of people running on the streets and followed them succumbed.

The Government hospital in Bhopal called Hamidia Hospital was suddenly beseiged by 12,000 patients, a figure which swelled to 55,000 the next day. The hospital had the capacity to accommodate only 750 patients. Immediate medical requirements like oxygen cylinders were in extremely short supply.

The increase in the number of affected people on 4, December, was said to be due to the residual MIC vapour remaining suspended in the cool winter air, recondensing and settling down to ground level thus affecting more people. This explanation does not appear to be quite acceptable in view of the known high reactivity of MIC which would have led to its total neutralisation within a few hours leaving little residues.

Further, MIC being heavier than air would have settled down fast.

EVENTS ON AND AFTER 3, December, 1984.

News of hundreds of deaths on the early morning of 3, December, spread like wild fire throughout the city leading to an exodus of people to safer places. Rumours of possibilities of such accidents in the next few days created a panic situation among the public.

Coping with the flood of patients affected to different degrees proved to be a herculean task for the state health administration. All available government medical and para medical personnel in Bhopal and in nearby districts were mobilised. Medical supplies started coming in but demand far outstripped supplies, particularly the demand for oxygen cylinders.

The state government authorities realised that the plant still contained enough quantities of MIC in the system and until this MIC was not taken care of, panic would prevail among the public. A request was made to the Government of India to depute the Director-General of the Council for Scientific and Industrial Research (CSIR) Dr.S.Varadarajan, an eminent chemist, to Bhopal and guide the efforts to detoxify the Carbide Plant. Under his leadership, a team was formed consisting of experts from the Regional Research Laboratories of the CSIR in Hyderabad (later renamed Central Institute for Chemical Technology) and the National Chemical Laboratory, Pune. The team, after holding detailed discussions with the Plant officials, finally decided that the best way to dispose of the residual MIC in the plant was to convert it to the final

product, Sevin by restarting and running the plant. Extensive precautions including aerial spraying of the plant area with water by helicopters were taken while the plant was restarted and run. The residual MIC was successfully converted to carbaryl pesticide.

IMMEDIATE CAUSE OF THE ACCIDENT:

To understand the immediate cause of the accident it would be necessary to look into the chemical behaviour of MIC. MIC, though normally stable, undergoes an autoreaction called polymerisation in which three MIC molecules combine with each other to form a larger molecule called cyanuric ester. This reaction is exothermic, that is, it releases heat as it progresses. As this polymerisation reaction is inhibited by a chemical called phosgene (COCl2), small quantities of it are added to the stored MIC . On the other hand, the reaction is catalysed, that is, accelerated by hydrochloric acid which is formed when phosgene comes into contact with water. Traces of iron also help catalyse the polymerisation. Hence, utmost care was needed to be taken by the plant designers and operators to prevent the possibility of ingress of water into the MIC tanks. Pipelines carrying MIC had to be made of stainless steel instead of iron to avoid contamination of MIC.

A committee of scientists, appointed by the Government of India, reconstructed the sequence of chemical events that led to the accident. According to the team, the failure of the plant operator's efforts to pump MIC out of the tanks was due to non-building up of pressure inside the tanks. This, in turn, was due to leakage of nitrogen and hence attention should have

been paid to detect the leakage. The plant supervisor wrongly felt that the non-building up of pressure was due to blockages in the MIC pipelines. His orders on 2, December, to wash the pipelines with water compounded by the fact that the operator did not use a slipblind led to water entering the MIC tanks. The water reacted with phosgene to release hydrochloric acid setting off the polymerisation reaction of MIC. Traces of iron impurities from the iron pipes carrying MIC would have catalysed the reaction further.

The polymerisation reaction, being exothermic, released heat raising the temperatures inside the tanks to almost 300 degree C. vapourising and thermally decomposing the MIC. The pressures inside the tanks could have gone upto more than 44 psi, high enough to break the rupture disc. About 40 tonnes of MIC could have escaped into the atmosphere without being neutralised by the caustic soda scrubber. The scrubber, anyway, had capacity to neutralise only 8.8 tonnes of MIC per hour and could not have handled the sudden rush of gas. Part of the MIC which could have found its way to the flare tower could also not be destroyed by the flare as the tower had been disconnected. Further, had the jumper line between the PVH and RVVH remained closed as before 25, November, some of the MIC could have found its way to the process plant area thus reducing the quantity released to the atmosphere. (ANNEXE 2A)

It is known through laboratory experiments that one of the thermal decomposition products of MIC could be another deadly poison, hydrogen cyanide (HCN). It was surmised that some HCN could also have escaped into the atmosphere.

LONG TERM RELIEF TO VICTIMS:

The population affected directly, like the dead and physically injured, and indirectly through loss of breadwinners was nearly 1 lakh spread over 30 wards lying on the eastern half of Bhopal Municipal Corporation area. Besides cash, assistance in kind like supply of free rations was given to the affected population and continues to this day. Medical facilities were augmented considerably through provision of dedicated hospitals for the gas affected. Compensation courts were set up to identify the people affected and determine the compensation payable to them from out of the Fund created for the purpose. Many claim cases, it is reported, still remain to be decided causing extreme hardship and agony to the affected. Vocational training schools to impart skills to the survivors of the affected families have been set up.

FAILURE ANALYSIS:

A question that arises naturally at this juncture is whether the Bhopal gas tragedy could have been averted or at least, its impact could have been minimised. For this, we have to look into the categories of errors normally associated with chemical accidents. An enumeration of these errors, as we saw earlier in Part I, had been made by the APCTT. Following this categorisation, * Bowonder and Miyake have identified the various errors that occurred at Bhopal. According to them the management and operating staff at Bhopal could be faulted on 63 counts (**Annexe 3**).

The proximate causes of the accident clearly fall under 'technoware' and 'humanware' errors. It would

be tempting to state that despite these deficiencies, the accident could have been prevented had some of the 'humanware" errors like washing the MIC lines with water been avoided. Perhaps, had an experienced supervisor familiar with MIC plants, instead of a new one with no such experience, been in place, the non-building up of pressure in the MIC tanks could have been traced to a leak of nitrogen gas and this aspect could have been looked into first. If no leak were to be found on inspection, then the washing of the lines could have been resorted to after taking the precaution of inserting a slipblind.

Admittedly, there were human errors galore in Bhopal but the weight of 'technoware' errors like basic design defect was significant. Use of inappropriate materials, insufficient safety margins, equipment malfunctioning were clearly 'technoware' errors for which the parent company should be held responsible.

Of the 'inforware' deficiencies, the fatal one was the non-availability of information on the toxicity of MIC. Much of the information on MIC manufacture was proprietory in nature and hence public availability of information on its toxicity was limited. The local plant management seemed to be more concerned with the accidental release of phosgene as it was considered more lethal than MIC. Knowledge on the long term impact of MIC on human health is becoming available only now after the disaster. On the possible genetic impact on the new born, the jury is still out.

From the public administration stand point, the most serious error was in allowing the plant to be located close to the city. There were no zoning

regulations either in 1969 when the plant was first set up or in 1979 when the production facilities were expanded. It was true that initially no squatter settlements existed in close neighbourhood of the plant They sprang up later as encroachments on government land. These encroachments were legalised in 1983. Sadly, most of the victims lived in these settlements.

When the Government of India gave the licence to UCIL to manufacture Carbaryl pesticides, little attention was paid by the licensing authority to the technological aspects of manufacture and handling of MIC. Alternative routes were available for manufacturing carbaryls which did not need storage of MIC. Besides this, the design of the Bhopal plant suffered from many inherent defects like insufficient scrubbing systems to neutralise escaping MIC, storage of MIC in large quantities, lack of safety systems to contain leaks and lack of backup systems for controls etc.

The absence of a disaster management plan or an emergency relief plan proved a big handicap in dealing with the aftermath of the gas leak. Had regulations like the Hazardous Chemicals (Storage, Handling and Transportation) Rules, 1989 or the Rules on Emergency Planning, Preparedness and Response for Chemical Accidents, 1996 been in place in 1984 then the probability of occurrence of the disaster and its impact could have been lessened. The Environment (Protection) Act, 1986 was itself a fallout of the Bhopal gas disaster. The Supreme Court directed that such an enactment be considered by the Government of India. Again, had an Environmental Impact Assessment (EIA) been done prior to grant of license, the technological and safety aspects could have been looked into. Bhopal has many lessons to offer.

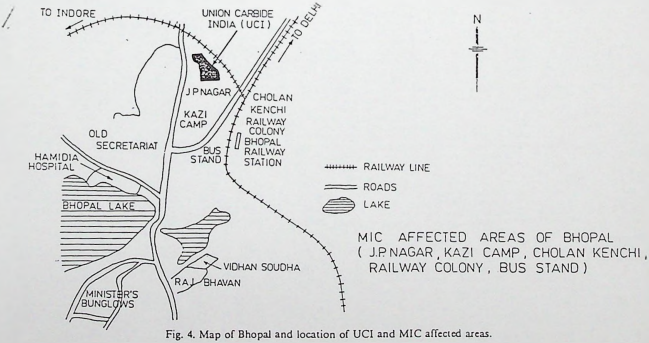
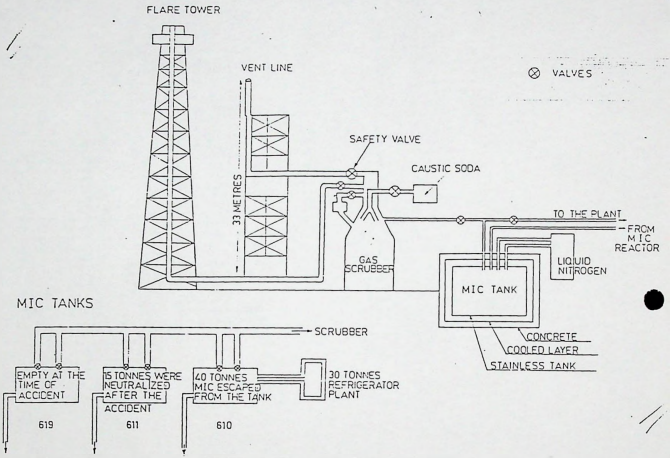
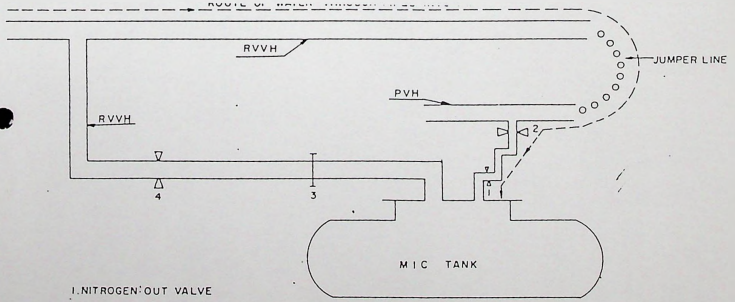


Fig. 4. Map of Bhopal and location of UCI and MIC affected areas.


FLARE TOWER
VENT LINE
VALVES
SAFETY VALVE
CAUSTIC SODA
33 METRES
TO THE PLANT
FROM M.I.C REACTOR
GAS SCRUBBER
LIQUID NITROGEN
MIC TANK
CONCRETE
COOLED LAYER
STAINLESS TANK
MIC TANKS
SCRUBBER
EMPTY AT THE TIME OF ACCIDENT
619
15 TONNES WERE NEUTRALIZED AFTER THE ACCIDENT
611
40 TONNES MIC ESCAPED FROM THE TANK
610
30 TONNES REFRIGERATOR PLANT

PART II ANNEXE 3 2A



1. NITROGEN OUT VALVE
2. PVH ISOLATION VALVE
3. RUPTURE DISC
4. SAFETY VALVE PVH PROCESS VENT HEADER
   RVVH RELIEF VALVE VENT HEADER

(Source: Fera, 1985)

FIG. 4. HOW THE WATER ENTERED.

TABLE 2

Causes of Bhopal accident: An analysis

	Technoware errors	Operator failure/design failure	Poor hazard management/safety management practices	Poor regulatory practices
1.	Capacity of vent gas scrubber insufficient		*	
2.	Refrigeration plant not functioning		*	
3.	No automatic sensors for MIC storage tanks		*	
4.	Pressure indicator not working		*	
5.	Sufficient gas masks not avialable		*	
6.	Flare tower was disconnected		*	
7.	Vent gas scrubber not kept in active mode		*	
8.	Plant modification connecting RVVH and PVH		*	
9.	Use of steel pipelines instead of stainless steel		*	
10.	There was only a manual mechanism for scrubber operation		*	
11.	No online monitor for monitoring contamination		*	
12.	Corroded valves not changed	*		
13.	Water curtain could rech only 10 m	*		
14.	No indicator for monitoring position of valves in the control room	*		
15.	Absence of hot lines		*	
	Humanware errors			
1.	MIC plant operator had no prior experience		*	
2.	Reduction in operating staff		*	
3.	Failure of shift operator to communicate about pressure increase	*	*	
4.	Repressurizing the tank without checking reasons for non-repressurization	*	*	
5.	Issuing orders for washing MIC pipelines	*	*	
6.	Not following safety precautions while washing	*	*	
7.	Not confirming leak when police officials called	*	*	
8.	Not operating the toxic alarm siren (switched off)		*	

PART II ANNEXE 3 (CONTD.)

TABLE 2 (continued)

	Technoware errors	Operator failure/design failure	Poor hazard management/safety management practices	Poor regulatory practices
9.	Failure to recognize the seriousness of the leak	•		
10.	Failure to use the empty tank		•	
11.	Failure to inform works' manager	•	•	
	Inforware errors			
1.	Panic reaction since no emergency plan		•	
2.	No risk analysis before plant modification		•	
3	Information on possiblity of runaway reaction not communicated		•	
4.	Doctors did not know the line of treatment		•	
5.	Information on precautions against MIC exposure not disclosed		•	
6.	Significance of toxic gas alarm not known to public		•	
7.	Information on toxicity of MIC not disseminated properly		•	
8.	Considering phosgene as more toxic		•	
	Orgaware errors			
1.	Absence of emergency rehearsals to check systems and procedures		•	
2.	Poor emergency planning on site		•	
3.	Poor emphasis on systems' safety		•	
4.	Not relocating the facility when applied for licence		•	
5.	Absence of hazard assessment procedures		•	
6.	No improvements in safety even after six accidents		•	
7.	Not developing expertise for handling toxics		•	
8.	Treating hazardous and non-hazardous facilities alike		•	
9.	Safety audit results not communicated to UCIL		•	
10.	Non-review of safety procedures even after newspaper reports		•	
11.	Storing large quantities of MIC		•	

PART II ANNEXE 3 (CONTD)

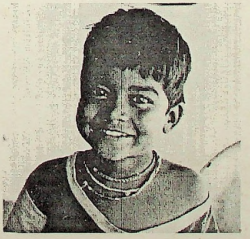
TABLE 2 (continued)

	Technoware errors	Operator failure/design failure	Poor hazard management/safety management practices	Poor regulatory practices
12.	Large manpower turnover		•	
13.	Non-disclosure of critical information		•	
14.	Poor commitment to safety		•	
15.	Absence of an emergency procedures manual		•	
16.	Absence of system for analyzing accidents		•	
17.	Heavy reliance on inexperienced opertors		•	
18.	Neglecting the warning of Factory Inspector		•	
19	Carrying out plant modifications without hazard analysis		•	
20.	Inability of the operating managers to make operators comply		•	
	Climoware errors			
1.	Weak factory inspection procedures			•
2.	Factory inspectors not trained for complex tasks			•
3.	No hazard management system			•
4.	No emergency plan for the city of Bhopal			•
5.	Not disseminating information on wind movement			•
6.	Absence of a zoning policy or industrial location policy			•
7.	Not resolving medical controversy in time		•	•
8.	Absence of toxicological information on MIC		•	•
9.	Public not educated about the true risk		•	•

– The failure of the operators to inform the Works Manager as soon as the leak started [16].

Most of the Humanware errors arise due to lack of training, development of skills and absence of procedures and lack of experience in handling such situations. In hazardous facilities, all possible emergency actions should have been anticipated and interventions prescribed so that no actions, however elementary, have to be improvised by decision of the operator [34].

VOICES FROM BHOPAL



On the night of 2nd-3rd December 1984, 40 tons of toxic gas was released from a Union Carbide pesticides plant in Bhopal, India. In the immediate aftermath, 3000 died and over 400,000 others were exposed and have continued to die and suffer. Misery in Bhopal increases daily as victims begin to suffer the long term effects of toxic exposure and the consequences of damage to their immune systems which makes them prone to debilitating infections. Meanwhile, the struggle for justice continues. Victims continue to speak out about the need for proper rehabilitation programs and for punishment of Union Carbide.

The Union Carbide plant was set up in 1969 as part of the effort to bring Green Revolution prosperity through high yield agriculture that is dependent on heavy inputs of chemical fertilizers and pesticides. The plant was located in an already densely populated area despite city planning codes which require facilities handling hazardous substances to be located away from human settlements. Nonetheless, the plant was not designed to fully accomodate safety precautions, it was poorly maintained and negligently operated. On several occasions prior to the disaster, workers attempted to point out potential hazards but were ignored by the management which was intent on a cost-cutting drive.

The government failed to enforce regulations of safety standards at the Carbide plant because they remained convinced that the benefits of foreign investment and accompanying reliance on chemical agriculture would bring "development" to India. While there is, today, a general concern about the harmful sociological and ecological implications of such "development", the Government response to periodic developmental crises remains essentially symptomatic. Governmental response to the Bhopal disaster has been symptomatic at all levels. Medical care, job generation and housing issues have only been dealt with superficially.

According to a recent study, 70 to 80% of the population in severely affected areas and 40 to 50% in mildly affected areas continue to suffer from breathlessness, fatigue, loss of appetite, loss of acuity of vision, menstrual irregularities, anxiety, depression and a host of other problems. Damages to the respiratory, reproductive, nervous, musculoskeletal and immune systems of the gas victims have been documented in epidemiological studies carried out so far. The 1990 report of studies carried out by the Indian Council of Medical Research states that the death rates among the affected population is more than double that of the unexposed population. Significantly, higher incidence of spontaneous abortions, still births and infant mortality among the gas victims have also been documented inthis report.

Despite these deteriorating health conditions, a proper line on medical treatment is yet to be available. Gas affected people continue to be given symptomatic treatment offering only temporaty relief, if at all. While Union Carbide continues to withhold information on the effect of released gases on the human body and the means to deal with these effects, research projects sponsored by the Government of India have also yielded litte towards a cure. Many observers suggest that there is a governmental will not to know what the released gases were or their long term effects as a way to avoid admission of the magnitude of the tragedy. Hiding the magnitude of the tragedy allows the government to both ignore immediate responsibilities to provide aid to victims and to ignore the need to change economic policies which promote investment into hazardous industries, particularly those operated by multinational corporations.

Another symptomatic response to the disaster has been the government's disproportionate emphasis on interim relief rather than on job generation. While the interim relief available since last June has significantly lessened the economic deprivation of gas affected people, its disbursement has been highly corrupt and inefficient. Further, the interim relief payments of Rs 200 per person per month will only

be given for three years. The government has made no long term plan to create jobs suitable for a permanently disabled population. Prior to the disaster, the majority of the victims earned their living through hard physical labour. Exposure to Union Carbide's gases has led to a substantial reduction in their capacity to work. Hence, there is an urgent need for provision of jobs to the gas victims in accord with their health condition. It has been suggested that a substantial number of gas victims can be employed to provide medical, educational and other services that are essential for the rehabilitation of the community.

A third symptomatic response spearheaded by the governemnt has been a "Bhopal Beautification Plan" which legitimated the demolition of a larger number of houses in the areas adjacent to the Carbide plant. In June 1990, residents were given a few moments' notice before bulldozers rolled over their homes. They were not compensated for loss of property; further, the long process of compensation for gas exposure compensation was disrupted because identification of claimants is based on residential addresses. These unnecessary and illegal demolitions to beautify Bhopal occurred while gas affected people continue without access to clean drinking water, hygienic living conditions or pucca houses. These basic needs of gas affected people have been ignored despite clear documentation of gas induced damage to their immune systems which makes them susceptible to infections.

Meanwhile, the case against Union Carbide is yet to cross the preliminary stages of litigation. Currently, the Supreme Court is hearing the review petitions that challenge the validity of the February 1989 settlement between Union Carbide and Government of India. Unless the settlement is struck down, Union Carbide will be absolved of all civil and criminal liabilities in return for a sum of 470 million dollars. Conservative estimates have placed the cost of medical care and surveillance alone in the range of 600 million dollars. Further, it is clear that a settlement of 470 million dollars will not have any deterrent effect on the hazardous operations of big corporations. Among gas victims, faith in the legal process in not strong; the courts are seen to be part of the establishment that benefited from Carbide's operations. Victims forcefully argue that they would have been ignored completely had they not carried out sustained public protest, insisting that their lives not be sold in exchange for the glamour of Indian participation in global capitalism.

People's struggle in Bhopal has been strong and sustained since the gas tragedy. Victims and those who support their cause believe solidarity to be the only way to justice and to transformation of the institutional structures which caused the Bhopal tragedy. Solidarity is seen as the only way to insist that communities like those in Bhopal will not accept impositions of risk that serve the interests of multinational corporations. Solidarity against Union Carbide is the only path to a world without "Bhopals".

There will be tears of sorrow,

tears of compassion,

there must not be tears of despair.

BANO BI (35)

CHHAWNI, MANGALWARA

The night the gas leaked, I was sewing clothes sitting next to the door. It was around midnight. The children's father had just returned from a poetry concert. He came in and asked me, "what are you burning that makes me choke?" And then it became quite unbearable. The children sleeping inside began to cough. I spread a mat outside and made the children sit on it. Outside we started coughing even more violently and became breathless. Then our landlord and my husband went out to see what was happening. They found out that some gas had leaked. Outside there were people shouting "Run, run, run for your lives."

We left our door open and began to run. We reached the Bharat Talkies crossing where my husband jumped into a truck full of people going to Raisen and I jumped into one going towards Obaidullahganj. It was early morning when we reached Obaidullahganj. The calls for the morning prayers were on. As we got down, there were people asking us to get medicines put on our eyes and to get injections. Some people came and said they had made tea for us and we could have tea and need not pay any money.

Meanwhile, some doctors came there. They said the people who are seriously ill had to be taken to the hospital. Two doctors came to me and said that I had to be taken to the hospital. I told my children to come with me to the hospital and bade them to stay at the hospital gate till I came out of the hospital. I was kept inside for a long time and the children were getting worried. Then Bhairon Singh, a hindu who used to work with my husband, spotted the children. He too had run away with his family and had come to the hospital for treatement. The children told him that I was in the hospital since morning and described to him the kind of clothes I was wearing.

Bhairon Singh went in to the hospital and found me among the piles of the dead. He then put me on a bench and ran around to get me oxygen. The doctors would put the oxygen mask on me for two minutes and then pass it on to someone else who was in as much in agony as I was. The oxygen made me feel a little better. The children were crying for their father so Bhairon told them that he was admitted to a hospital in Raisen. When I was being brought back to Bhopal on a truck,

we heard people saying that the gas tank has burst again. So we came back and went beyond Obaidullahganj to Budhni, where I was in the hospital for three days.

I did not have even a five paisa coin on me. Bhairon Singh spent his money on our food. He even hired a taxi to take me back to Bhopal to my brother's place. My husband also had come back by then. He was in a terrible condition. His body would get stiff and he had difficulty in breathing. At times, we could give up hopes of his survival. My brother took him to a hospital. I said that I would stay at the hospital to look after my husband. I still had a bandage over my eyes. When the doctors at the hospital saw me, they said "why don't you get admitted yourself, you are in such a bad state." I told them that I was alright. I was so absorbed with the sufferings of my children and my husband that I wasn't aware of my own condition. But the doctors got me admitted and since there were no empty beds, I shared the same bed with my husband in the hospital. We were in that hospital for one and a half months.

After coming back from the hospital, my husband was in such a state that he would rarely stay at home for more than two days. He used to be in the Jawahar Lal Nehru Hospital most of the time. Apart from all the medicines that he used to take at the hospital, he got medicines like Deriphylline and Decadron from the store. He remained in that condition after the gas disaster. I used to take him to the hospital and when I went for the Sangathan meetings, the children took him to the hospital. He was later admitted to the MIC ward and he never came back from there. He died in the MIC ward.

My husband used to carry sacks of grain at the warehouse. He used to load and unload railway wagons. After the gas, he could not do any work. Sometimes, his friends used to take him with them and he used to just sit there. His friends gave him 5-10 rupees and we survivied on that.

We were in a helpless situation. I had no job and the children were too young to work. We survived on help from our neighbors and other people in the community. My husband had severe breathing problems and he used to get into bouts of coughing. When he became weak, he had fever all the time. He was always treated for gas related problems. He was never treated for tuberculosis. And yet, in his post-mortem report, they mentioned that he died due to tuberculosis. He was medically examined for compensation but they never told us in which category he was put. And now they tell me that his death was not due to gas exposure, that I can not get the relief of Rs. 10,000 which is given to the relatives of the dead.

I have pain in my chest and I get breathless when I walk. The doctors told me that I need to be operated on for ulcers in my stomach. They told me it would cost Rs. 10,000. I do not have so much money. All the jewellery that I had has been sold. I have not paid the landlord for the last six years and he harasses me. How can I go for the operation? Also, I am afraid that if I die during the operation, there would be no one to look after my children.

I believe that even if we have to starve, we must get the guilty officials of Union Carbide punished. They have killed someone's brother, someone's husband, someone's mother, someone's sister- how many tears can Union Carbide wipe? We will get Union Carbide punished. Till my last breath, I will not leave them.

"How many tears can Union Carbide wipe.?"

ABDUL ZAHOOR (30)

BAUG UMRAODULHA

I get swelling in my stomach. I become extremely uneasy and cry out in pain. Sometimes this happens all through the night. I am tired of getting x-rays done and the doctors say nothing about my disease. I have gone to all kinds of doctors, the big ones too. I have been to Sajad Nursing Home, the J. P. Hospital and to the Hamidia Hospital. I have even gone to the Hakims and Homeopathic doctors. But it has been like this. Pain, pain, all the time. I become weak, had body aches and fever for a long time after the gas, but earlier I didn't have this pain in the stomach. They have done my medical examination but now they tell me I have been put in "B" category. I had shown them my medical papers. Still. I haven't been able to work for the last two years and have stayed in bed all the time. I depend on my brothers for food and my treatment. Something has to be done for this pain in the stomach. I am getting the interim relief of Rs 200 per month, but that isn't enough for my treatment. I have to spend 700 to 800 rupees on treatment every month. And yet there is no relief.

NATTHIBAI (55)

RAJENDRA NAGAR

My husband's name was Dukhishyam. He got a lot of gas in him. On the night the gas leaked, both of us ran towards the forest. He remained sick afterwards. He used to get breathless, cough and his eyes would get very big. He could not see properly after the gas. Twice he was admitted to the hospital. Right after the gas leak he was admitted to the hospital. He was a little better when he came back from the hospital. The second time he was admitted, he never came back. He died in the M.I.C. ward. I gave an application for Rs 10,000 in interim relief, but they haven't done anything about it yet. Last year, he died in Kunwar (autumn). They haven't yet told me whether I will get Rs 10,000 or not. I gave them all the medical presciptions of my husband with my application.

I stay sick. I have come back from the hospital on 13th of this month (November 1990). I was there for one and a half months. I never got breathless before the gas, I used to work as a labourer. Now I get badly breathless and my chest pains. I was in the hospital during the Festival of Lights. This gas has destroyed us completely.

AJEEZA BI (30)

CONGRESS NAGAR, KAZI CAMP

Ever since the gas, my head aches 24 hours a day. I have pain in my stomach and sometimes I feel giddy. My daughter, Nasreen, can not see properly, can not thread a needle and she is only eleven. My other daughter, Sofia, also stays sick and she is eight. I have three children from before the gas disaster and after the gas I have aborted thrice. All three times it happened in the hospital. Once I was six months pregnant, the second time I was seven months pregnant and the third time I carried the baby for eight months. They were all born dead. All with black skin like the colour of coal and all shrunken in size. The doctors never told me why such a things was happening to me.

ASAD (14)

IBRAHIMPURA

I get breathless and often I am down with fever. Also I cough a lot. I go to school but I can not study. I forget things easily and my eyes burn. I study in a government school. After the gas, for three years I could not write my examinations. I can not remember things. Ever since the gas, I am always taking medicines. Those doctors who were examining me, I told them that I have breathing problems. But they have sent this notice that says I've been put in "B" category.

SABRA BI (40)

CONGRESS NAGAR, KAZI CAMP

I have been in and out of several hospitals since the gas disaster. In 1986, I was told that they are registering claims of the gas victims. So I took my children and stood in the queue to get my claim form filled. It was a long queue and there were at least 250 people before me. When my turn came up, the fellow who was filling the claim forms said that he will not fill claims for children. He said only people over eighteen years could file claims. When I insisted, he asked me to put the medical prescriptions of the children along with their father's claim form. But that did not work. Later, when people were receiving notices to get themselves medically examined, there were no notices for my children. So their medical examinations were not done.

I was in the hospital when the people who were carrying on the survey- the Tata Insitute people- came to my place. They took down the names that were listed in the family ration card. But all the names were not there. The ration card was issued fifteen years back and only three of my six children were listed on it. When the claim forms were being filled, my daughter Afroz was twelve years old, Gulnaaz was ten years old, Mehenaaz was nine years, Neelofar was seven years and the youngest, a son, Firdous, was two years old. The government fellows did not put any of these children's claims in their register.

BADRUDDIN, (50)

PULBOGDA

I was asked to report to the Identification Center on 23rd of last month (October 1990). There they told me that the names of my children that were on the notices did not match with the names they had on their records. Names of my two sons and one daughter were wrongly recorded. I went to the Collector's office to get the names corrected. I was made to go from one office to another. It took three days to get the names corrected. And my daughter's name is yet to be corrected. I showed them all her medical papers, even her affidavit, but they are yet to correct the name.

SHAMEEM BANO,(30)

BUDHWARA

Three of my children have yet to file their claims. All three were born before the gas disaster. The oldest, Samad, is 12 years old; then Malka is 8 years old and the third son, Amjad, is 7 years old. When they were filling the claim forms, I told them to file the children's claims. But they said such young children can not file claims. "We will put these children's claims along with their father's papers, " they said. But they did not even write down the names of these children. Earlier, when the survey people had come, I told them the names of all my children. But they took down names from the ration card. Our ration card is 20 years old. It does not have the names of all the children. I told those government fellows that my children have been left out. All they say is "We will see, we will see."

"If someone kills just one person he is put in jail for twenty years. And here, the Carbide officials have not been put behind bars for even twenty minutes. They should be hanged."

SHAKILA BANO (30)

JAI PRAKASH NAGAR

Right after the disaster, I was admitted to the Katju Hospital. Then I was admitted to the Hamidia Hospital for a long time in the M.I.C. ward. The doctors told me that my x-ray pictures showed that my lungs were badly damaged. I had filed my claims and was called for medical examination. The medical examination, they said, was necessary to make my case strong for compensation. They did all kinds of examinations: they did blood tests, sputum tests, urine tests and also took x-ray pictures. I was once agian admitted to the Jawaharlal Nehru Hospital after that. Now they sent me this notice which says I have been put in category "B". It says that I only suffered temporary injury due to the gas and I am alright now. Even now I cough so badly all the time, I throw up blood sometimes. I have pain in my chest. When I get admitted to the hospital, the doctors do not let me go home. "You are still very sick," they say, and ask me to stay on at the hospital.

RAMKISHAN (40)

PHUTA MAKBARA, CHHOLA ROAD

I used to work at the Formulation plant in Union Carbide's factory. I had been working there ever since the sixth month of the year 1973. When I joined, I used to work as a casual worker. For six years, I worked as a casual worker. They made me a permanent worker in the third month of 1980. I was working in the Formulation plant on the night the gas leaked. The tank of MIC which leaked was only 400 feet from the Formulation plant. When the gas started leaking, some people cried out "Run, Run" and we left our work and ran towards the west.

Later, the factory was closed down. There was nothing for me to do. The government offered me jobs but they were all away from Bhopal. I was given jobs in Mhow, Rajgarh and Indore, none in Bhopal. My wife had taken in a lot of gas and she was pregnant at that time. So I could not stay away from Bhopal. Now I work as a daily wage labourer. I get jobs 15 to 20 days in a month and make about 20 to 25 rupees in a day. There are quite a few Carbide workers who could not find employment after the factory was closed down. I personally know about one hundred of such workers. After the factory was closed, I was given six months salary as compensation, nothing else.

I was just a worker there, how could I know what poisons were stored in there? I was never told that there were such dangerous chemicals inside the factory. If I knew, I would not have worked in that factory. The plant used to smell awfully at times but we were just workers, how could we know? When we worked there, our eyes used to hurt and our skin itched but whoever knew that such a disaster could happen?

JAYA MANE (28)

RAJENDRA NAGAR

I get breathless when I walk and now my head aches so badly that I can not do any sewing or reading. The doctors had done my medical examination. But later they sent a notice which said they found me to be only temporarily injured. I do not know whether we can ask them to do another medical examination. I have started getting interim relief but the bank is quite far. I had to spend Rs 20 to go to the bank.

SHER KHAN (45)

CHHOLA ROAD

I work at the railway coach factory. I have been living in this house for the last fifty years. This year, they announced from a jeep that they would demolish the houses on both sides of the road. People in my community and the tenants who had rented shops were very troubled. Quite a few cried their hearts out. Those who protested got arrested. Some people who tried to argue with the government officials were beaten up by the cops. And the bulldozers went on demolishing house after house as we watched in silence and in sorrow. After my house was demolished, I was so sad I could not eat food for four days. It was raining then and my children were crying. I cried too.

I have made a tiny shelter out of whatever was left after the demolition. I still have the registration papers for my house. This house has been there for the last sixty years and they never told us that we had encroached upon government land. This anti-encroachment drive is a lot of bunkum. It was as if the government had declared war on the people. All day they would carry on the demolitions and at night they rested till they blew the bugle the next morning. They cut off the water connections, the electricity connections and turned us homeless. They have not given us any compensation or any land. First, we were attacked by Union Carbide gas and then by the government's bulldozers. Where do we go?

MOHINI (32)

MAHAMAYEE KA BAUG

Our organization, the Bhopal Gas Peedit Mahila Udyog Sangathan started from a sewing centre. After the gas disaster a rehabilitation centre run by an organization was started in September '85 with government help. About 600 women used to be given sewing jobs from this centre. There were 30 of us employed who were

"We know that the struggle against Union Carbide will be a long one and we are determined to carry on with our struggle till justice is done."

employed for cutting cloth at the centre and this cut cloth was given to the women for sewing at their homes. In December 1986 this centre was closed down.All of a sudden the women who were dependent on the sewing job became jobless. The 30 of us decided that something must be done to get the centre reopened. So we, alon with 600 women marched to the Chief Minister's residence. We went on several demonstrations and had to face the police on many occasions. In April '87, 225 of us were arrested and put in jail. It was a long and hard struggle. Most of us were quite sick due to the gas. During one demonstration a women named Hamida Bi fell unconscious with chest pain and died 4 days later. We finally managed to get the centre reopened and now 2300 women are getting sewing jobs.

After we got the centre reopened our organization grew in number and we took up the issues of medical treatment and economic rehabilitation of the gas victims. We also campaigned against Union Carbide and organised rallies demanding punishment to the guilty officials of the company and adequate compensation for all gas victims. We opposed the unholy settlement between Union Carbide and Rajiv Gandhi's government. On five separate occasions more than 3000 women from the Sangathan have gone to Delhi and voiced our opposition to the settlement.

We have also filed a petition in the Supreme Court challenging the validity of the settlement and now it is being heard. Earlier in August 1988, we had filed a petition seeking interim relief from the Government. On 13th March 1990, the Supreme Court ordered the Government to pay Rs.200 per person per month to all the residents of the 36 gas-affected municipal wards of Bhopal for three years. This amount is being disbursed now but there are a lot of problems in the manner in which this is being done. We know that the struggle against Union Carbide will be a long one and we are determined to carry on with our struggle till justice is done.

SAHODRA BAI,

LAKHERAPURA

My husband Shantilal died on 12th May 1990. After the gas, he had difficulty in breathing. He never went to work after the gas. He couldn't earn any money. The children earned something by doing odd jobs. I can not see properly and I get breathless. I can not do any work. Union Carbide is responsible for my husband's death. I should be given the relief money of Rs 10,000 and should get enough compensation so that we have enough to eat and get ourselves treated

SHAHAZADI BAHAR (35)

KOLIPURA, BARKHEDI

I joined the Sangathan in 1988. I was looking for a sewing job. I went to a number of places all around Bhopal. Then one of my friends asked me to become a member of the Sangathan. She asked me to come for the Sangathan meetings and talk about my problems there. So I filled a form and became a member of the Sangathan. Now I am so closely attached with the Sangathan that when I do not go for the meetings, I miss it as people miss their dear ones.

The world is very selfish. I, too, joined the Sangathan with some selfish motive. I thought I could get some sewing job through the Sangathan. But though I have not been benefited, there are others who have. Quite a few people have got monetary relief of Rs 1000, Rs 3000 and Rs.750 per month. And now the provision of interim relief of Rs 200 per month per person is a big victory for the Sangathan. This has brought in a new hope and a new determination. We are certain that we will win this battle.

The Bhopal victims are entitled to compensation. We need hospitals, medicines, jobs, clean air and water. We have to have medical treatment centers in the community itself. The bigger things are, the more they create problems. Hamidia hospital is so big but we can not get treatment there, only those with money are treated properly. We need jobs that do not need hard physical work. I get breathless when I walk and can not see properly. Two of my daughters are being treated for tuberculosis.

They should not have allowed Union Carbide to set up its factory. When these companies want to set up some factory, they mention some product in the agreement (with the government) and they start producing something else. Then the people in the neighborhood do not get to know what is being produced. Workers in the factory are forbidden to speak to people in the community. Such factories should not be allowed in the first place. And even if they are allowed to be set up, the neighboring community must be consulted.

The officials of Union Carbide should be given the severest punishment. If someone kills just one person, he is put in jail for 20 years. And here the Carbide officials have not been put behind bars for even 20 minutes. They should be hanged. I am certain that the Sangathan will win the battle. The struggle for truth will be a success. Truth always wins, it only takes a little longer.

"The doctor in charge of the MIC ward has written on my papers that my lungs are badly damaged. I have to take 8 to 12 tablets in a day to be able to talk, move about or just breathe."

DINKAR RAO (16)

NAGAR NIGAM COLONY, KAZI CAMP

When the gas leaked, we were all sleeping. We started coughing and getting choked. I thought someone was burning red chillies in the neighborhood. But my mother said it was some kind of gas. She knew, she read a lot of books. She asked everybody to stay indoors but my father did not listen. He opened the door and went out to see. Thick clouds of gas filled the room. Our parents covered us up with a quilt from all sides. So we were a little protected. But my parents took in a lot of that gas. That is why they fell so sick.

My father could not do any work after the gas disaster. He used to remain sick and in 1986 he died. My mother used to be sick also. Doctors took x-ray pictures and said her lungs have been badly damaged. Some doctors said she has got tuberculosis but we do not believe that. In 1986, she was admitted to the Jawahar Lal Nehru Hospital. She used to get breathless and used to cough all the time. She could not go to office to work. My mother died in February 1988 in the hospital. Since then, I have become a full time worker in the Sangathan.

We are opposing the settlement between Union Carbide and Government of India done in February 1989. The settlement would have meant that Union Carbide officials would have been let off without any punishment. We can not let this happen. Carbide's officials must be punished. If these officals are let off easily, they will go on killing people and making them sick. What happened in Bhopal should not happen anywhere else.

SULEMAN KHAN (50)

ASHOKA GARDEN

I have been working as a booking agent in the Madhya Pradesh State Road Transport Corporation for the last 24 years. I was on duty right after the disaster and during Operation Faith when the Corporation's buses were used to carry people who were running away from Bhopal. I started having serious health problems about a month after the gas leak. In May '86 I was transfered to Piparia, 150 kms away from Bhopal. There were no facilities for medical treatment of gas victims in the Piparia hospital. So I had to absent myself from my duty and come to Bhopal. They stopped my salary. I wrote many applications to get myself transfered back to Bhopal. More than 2 years later, they transfered me back but I am still not getting my salary.

I was admitted in the MIC ward in June '87 and remained there for more than three years. I was so breathless they had to put me on oxygen. The doctor incharge of the MIC ward has written on

my papers that my lungs are badly damaged. I have to take 8 to 12 tablets in a day to be able to talk, move about or just to breathe. I have written 17 letters to the officials of the Corporation, 7 letters to the Chief Minister and 4 letters to the Prime Minster. I have requested them to pay me my salary, give me some monetary relief and do something for my medical treatment. These medicines don't seem to be working.

In August '87 I was called for medical examination by the Directorate of Claims. I was in the MIC ward at that time. Then after 2 years when I was still in the hospital they sent me a notice which said that I have been put in Catagory 'B'. My wife was also admitted in the MIC ward. She too has been told by the Claims Directorate that she has suffered only temporary injury. Like they said for me. I wrote a letter saying that there was something terribly wrong in putting me in Catagory 'B'. The doctor in charge of the MIC ward put in his recommendation in that letter. It has been almost a year since and they haven't replied yet.

PUNIYA BAI (65)

CHHOLA ROAD

This was an old house . My husband's parents built it a long time back. And now the government has demolished it. They did not give us any notice before breaking it down. It has been six months since the bulldozer demolished my house and they haven't talked about giving any compensation. I do not want money, I should be given a house somewhere. They broker down the house in the middle of the rainy season. The children had no shelter and there was no place to light a fire or to cook food. The house had an identification number which was a proof that we have been affected by the gas. Once we are shifted away from this place we will lose that proof. Then the government fellows will not believe us when we tell them we have been exposed to the gases from Carbide.

CHHOTELAL (50)

BARKHEDI

I used to work as a porter for transport companies. Since the gas, I have not been able to work for a single day. The gas killed my daughter, she died in the morning after the gas leak. I am breathless all the time and I cough badly. My eyes have become weak too. I have been admitted to the MIC ward more than five times since 1987. Last year I was there for 9 months at a stretch. This year I have come home after eight and

"I was in the hospital during the Festival of Lights."

GANGA BAI (63)

RAJENDRA NAGAR

I have started getting interim relief of Rs 200 per month. But there are lots of problems. To get my photograph taken, I would have to go to Indrapuri, which is twelve kilometers away. The bank was fifteen kilometers away in Bairagarh. My son took me on his bicycle to the bus stand and I had to change two buses to reach the bank. And I had to make three trips to the bank before I got the money. Now they have brought the bank a little closer. Even then I had to spend Rs 24 on an auto rickshaw. The banks are very crowded and the queues are long. Once I fell down on the ground while I was waiting in the queue.

SULEMAN (45)

PUTLIGHAR, SHAHAJANABAD

I am in Bhopal for the last 10 years. Before the gas disater, I used to sell vegetables on a push cart. I have become too weak to work now. After I was exposed to the gas, I tried pushing the cart but I became so breathless it was impossible to move. Some time back, I went to sell vegetables but was so ill afterwards that I had to be admitted to the hospital. Five bottles of intraveneous medicines were given to me because I was so sick. Since then, I have not tried to take the vegetable cart around. For a few days, I worked as a watchman but that too was difficult. Now I have given up. I stay in my sister's place, her family arranges for my food. All the treatment that I have taken until now has done no good. Factories like Union Carbide's should not be allowed anywhere in the world. Not even if they build it far from human settlements.

MOHAMMAD NAFEES (25)

BUDWARA

My children are named Assu and Sharik. One is about 7 years old and the other is 6 years old. When the gas leaked, my elder son was eight months old; my younger son was born four months after the

disaster. Both of them have difficulty in breathing and they cough a lot. The doctor says they have got tuberculosis. They have been taking treatment for tuberculosis for the last four years. Even now they are under treatment. I get tablets and capsules for them from the hospital. Sometimes I have to buy capsules from the store; one capsule costs one and a half rupee. You tell me that one should not take drugs for tuberculosis for so long. But what do we know? We do what the doctor tells us. No one in my family ever had tuberculosis. Both my wife and me also have breathing trouble. Before the gas I used to spend a lot of time working at the bakery. Now I can't sit close to the oven.

SHAHIDA BI (25)

NEAR BHARAT TALKIES

A few months after the gas disaster, I had a son. He was alright. After that I had another child in the hospital. But it was not fully formed. It had no limbs and no eyes and was born dead. Then another child was born but it died soon after. I had another child just one and a half months back. It's skin looked scalded and only half its head was formed. The other half was like a membrane filled with water. It was born dead and was white all over. I had a lot of pain two months before I delivered. My legs hurt so much that I couldn't sit or walk around. I got rashes all over my body. The doctors said that I will be okay after the childbirth but I still have these problems.

NARAYANI BAI (35)

MAHAMAYEE KA BAUG

This is the sixth time I have been admitted to the MIC ward. I have been here sice the last month of 1985. When I feel a little better the doctors send me home but I can't stay there for long. My breathlessness become acute and my husband has to bring me back to the hospital. The doctors say that the gases have damaged my lungs badly. They say nothing can be done about my disease. Before the gas I had never seen the insides of a hospital. And now I have spent most of the last five years on this hospital bed. I used to work as an assistant at a day care centre and now I can not do any work. My husband

"If these officials are let off easily, they will go on killing people and making them sick. What happened in Bhopal shoud not happen anywhere else."

"What do these people in the Directorate of Claims have against me? Why don't they want me to be properly compensated?"

Kaluram also can not go to his job. He used to carry loads. My son works as a tailor, he is the only one earning in the family.

MOHAMMAD AJEEZ (22)

KUMHAR MOHALLA, VIDHAN SABHA

I am 22 years old and the notice for interim relief that came in my name said I was 46 years old. When I went to get my pass book made, they told me to get my age corrected on the notice. I went back to them two days later and they said if you pay us Rs 50, we will get the age corrected. Then they asked me to pay them this bribe after two days. My school examinations mark-sheet says I am 23. I have got certificates that have my date of birth and yet I am being harassed in this manne

ABDUL JABBAR (36)

RAJENDRA NAGAR

I am the convenor of the Bhopal Gas Peedit Mahila Udyog Sangathan. I am a gas victim myself, my father died because of the gas. We in the Sangathan are fighting against a killer multinational and an apathetic government. Union Carbide is trying its best to evade accountability for the genocide it has commited. It is trying to wriggle out of the situation by using its wealth and its political power. The new government at the Centre seems to have taken a strong stand against Union Carbide. But the government has yet to take effective action for medical treatment of the gas victims. We have long been asking the government to set up a Medical Commission on Bhopal. The Medical Commission would concentrate on evolving a proper medical treatment forthe gas affected people. The Commissiom should also look into the medical categorization that the Directorate of Claims has done. The government has to provide opportunities for people to become self-dependent.

People outside Bhopal seem to have forgotten the gas disaster. Earlier a lot of concern was expressed for the Bhopal victims but now that seems to have died down. Even today hundreds of thousands of the gas affected people continue to suffer from gas-related illnessess and people are still dying painful deaths. Yet most people seem to believe that the Bhopal issue is over. This is indeed unfortunate.

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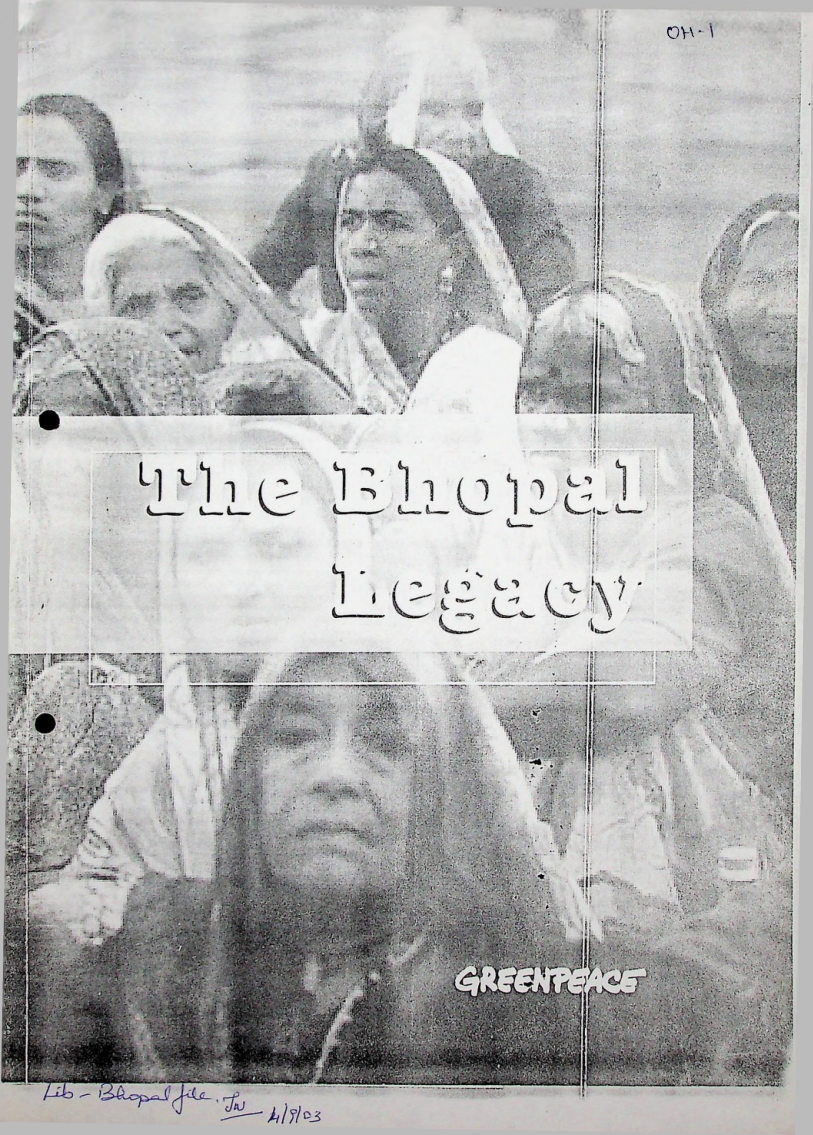

The Bhopal Legacy

GREENPEACE

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Occupational Health Hazards faced by poor rural women in Karnataka.

A Pilot study in Raichur District

Paper prepared by :
Amrutha S.V. and Revathi Narayanan
Mahila Samakhya Karnataka, India.

To be presented at Nepal Conference on
Women Workers - Gender concerns vis-a-vis
Occupational Health. March 23-26, 1999
at Kathmandu, Nepal.

OCCUPATIONAL HEALTH HAZARDS FACED BY POOR RURAL WOMEN IN KARNATAKA

A PILOT STUDY IN RAICHUR DISTRICT

(AMRUTHA.S.V. AND REVATHI NARAYANAN : MAHILA SAMAKHYA, KARNATAKA)

WOMEN'S WORK IN KARNATAKA

The report of the Voluntary Health Association of Karnataka published in 1997 entitled "State of people's health in Karnataka", gives the following picture of women and work in Karnataka.

The 1991 census enumerated 5.1 million female workers (5.0 million main workers and 0.1 million marginal workers). 4.3 million in rural areas and 0.8 million in urban areas. Marginal workers or those who worked for less than 183 days during the year preceding the census were considerably more among females; many women combine household work with outside work for livelihood. Over one half of main workers are agricultural labourers. Marginal workers and agricultural labourers together constitute nearly two third of total workers. A large number of women workers are engaged in the unorganised sector. In rural areas, among workers other than cultivators and agricultural labourers, female workers are mainly engaged in beedi making, plantation work, retail shopkeeping, basket making, tending cattle etc. In urban areas, they are mainly engaged in beedi making, agarbathi making, domestic work, retail shopkeeping, spinning, as house maids, teachers, clerks etc. Because of the nature of work, working environment and poor remuneration, they have to suffer several kinds of discrimination and disabilities which have a telling effect on their health.

The effect of woman's work on women's health has three aspects:

1. Employment places an additional ENERGY BURDEN on women along side domestic work and reproductive demands.

2. Among the wage-earners it denotes a greater AUTONOMY in money related decisions and improved health outcomes for the children and the family.

3. Among the non wage-earners, there is little recognition for the additional work load of the woman's contribution. One estimate based on the simplest calculation valuing women's work at home at Rs.10.00 per day yields an annual CONTRIBUTION TO THE GNP of morethan Rs.40,000 crore (On closing the gender gap: increasing the value of women in India: Rohde JE. Indian J Paediatrics, 1991:58:295-9)

Significantly, the poorer the family, the more it depends on the economic productivity of a woman. With 32.1% of Karnataka (1987-88) below the poverty line (Statical outline of India:

1994-95 Ed. JK Mukhopadhyay, Tata Services Ltd, 21st Ed., Oct 1994.), a large proportion of families look to the woman for support.

For the non-wage and subsistence wage earners, like the Mahila Samakhya sangha women, there is hardly any recognition of their contribution except in the negative sense. If women did not work, there would be even less to eat. Thus, their level of earning is not large enough to ensure greater autonomy and better health outcomes for their children ; however their work does place an increased energy burden on them. Through the collectives, Mahila Samakhya is working to improve their financial status and decision making (through savings and various economic activities in the sanghas); to enhance their health status and access to healthcare through a combination of strategies. These are by demanding greater accountability from government health systems through the sanghas and by encouraging sangha women to become more self reliant, for preventive and curative healthcare, using their knowledge of herbal medicine.

THE MAHILA SAMAKHYA PROGRAMME AND ITS WORK WITH RURAL WOMEN :

Mahila Samakhya is a programme of the Department of Education, Ministry of Human Resource Development, Government of India. It was born out of the emphasis given in the New Education Policy of 1986 to the need for education programmes to play " a positive, interventionist role" in bringing about women's equality. The programme was launched in ten districts in the states of Gujarat, Karnataka and Uttar Pradesh in 1989, currently it is also in Andhra Pradesh, Bihar, Madhya Pradesh, Assam and Kerala. In Karnataka, the Mahila Samakhya programme is working in the seven districts of Bidar, Bijapur, Mysore, Gulbarga, Raichur, Koppal and Bellary.

The programme is called Mahila Samakhya: Mahila means women, and Samakhya is a compound of the Sanskrit words "sama" meaning equal, and "akhya" meaning to be valued or weighed.

Mahila Samakhya believes that despite the many educational and developmental programmes undertaken over the past four decades, women, as a gender, remain an exploited and oppressed lot, and the poorest rural women of India, regardless of caste and community, are the most powerless group of all. They play little or no role in decision - making processes at any level, but must remain the passive "beneficiaries" of welfare and development schemes which they have played no part in designing.

Our main objective is to reverse these processes by empowering the poorest rural women with the freedom and knowledge with which to determine their own destinies. A "time and space" is created for them to gather together and engage in a process of collective reflection, analysis, learning, and action, defining their own needs and priorities.

The basic strategy of the programme is the building of village level collectives or "sanghas" which are not merely activity oriented, but which enable women to raise their self-image and confidence, and recognise their own strengths. The idea is to create an atmosphere in which women feel the possibility of changing their lives.

Mahila Samakhya, Karnataka has been working in the fields of literacy, health, panchayat raj, economic development programmes and various issues that contribute to the overall status of women. Gender is a cross-cutting issue in all aspects of work.

Mahila Samakhya Karnataka has recently started work in the field of occupational health hazards among sangha women. Earlier, the health aspects of the programme were focused more on herbal medicine. The benefits of herbal medicine are manifold- providing basic health care to poor women who have hardly any access to the Government-run primary healthcare system, empowering women by recognising and valuing their knowledge, encouraging the planting of more and more herbal medicinal plants, providing medicine for hitherto ignored ailments that are suffered in silence by women. A large number of training camps have been held for sangha women, several of whom are now preparing and dispensing medicines in their villages. Diabetes, anaemia, white discharge, excess bleeding, uterine prolapse, asthma, toothache, even baldness has been successfully treated!

This area of work focused more on curative aspects rather than on the preventive aspects of health care. However, in the course of our work we have became increasingly conscious of the need to enhance the health status of sangha women. The obstacles in this route are several and severe. Poverty forces sangha women to take up occupations that are hazardous to their health.

Many of the sangha women suffer from general poor health and from symptoms that can clearly be correlated with their occupation. Being poor landless labourers, they bear the brunt of unhealthy and unsafe agricultural practices. Being forced by circumstances to undertake these occupations, they suffer from occupational health hazards as described below. Their poverty, combined with poor access to healthcare makes them vulnerable to several kinds of diseases. This is increased by their low levels of nutrition, frequent child-bearing and a lifestyle that generally leads to poor health conditions.

CASE STUDIES FROM MAHILA SAMAKHYA SANGHAS IN RAICHUR DISTRICT

Discussions were held with sangha women from 3 villages by researchers from Mahila Samakhya (Amrutha, Leela, Dakshayani) and PRIA (Sumedha Sharma, Harsh Jaitli)

35 year old Basamma belongs to the Mahila Samakhya Sangha in Mallapura village of Raichur District in Karnataka. Basamma, who belongs to the SC community, was deserted by her husband when she was 20. Desperate to earn her living, Basamma began working with the Woddar community. Her work consist of carrying heavy headloads of

stones, jelly and similar material. Three years ago Basamma found that her vision was getting blurred. She could not see properly. She went to the Government hospital in Raichur town where, with treatment, her vision begin to improve. But this was short lived and a week later she became completely blind. Her family took her to Hyderabad where a detailed examination was done. The doctor told her that the damage to her eyes was irreversible and had been caused by the heavy loads she used to carry for more than 10 years. This had led to the damage of the optic nerves. Today Basamma is completely dependent on others. Her daughter looks after the household with the help of her mother, while the young son earns what he can, to support the family.

The other case is that of 30-year - old Lallamma of Jegarkal village. She is a landless labourer who works for her living on the fields of others. For the past 2 years, Lallamma has a persistent cough with blood in her sputum. She finds it increasingly difficult to work and has to depend on her son, since her husband is also sickly. Despite several visits to the Government hospital, Lallamma has got no relief from her illness nor is she clear about the exact reasons for it.

This story repeated itself in the life of 30-year-old Gudamma of the same village. Her work also consists of carrying heavy head load and doing agricultural work. She found that her vision was becoming blurred and stopped carrying heavy loads. In addition, Gudamma suffers from elephantiasis because of working long hours in the wet fields without any protection for her feet. Husband and wife are forced to work under difficult conditions to look after their four children and themselves.

Shanthamma (35 years old) from the same sangha is also an agricultural labour. She is suffering from chest pain, headache and when she coughs, the sputum is dark brown or black.

Discussions with the sangha women in nearby Jagirvenkatapura revealed that all of them are poor landless labourers, who suffer from lower back pain, chest pain, headache and recurring bouts of fever. They also suffer from persistent cough and black sputum. In the discussions, they expressed their fear that the symptoms are those of tubercolosis.

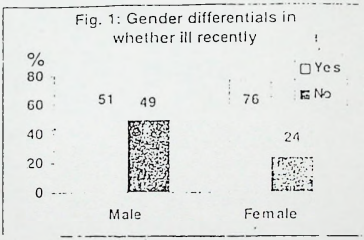
Discussions with sangha women in other areas reveal that they suffer skin and respiratory ailments from constant exposure to pesticides. Continuous bending gives rise to lower back pain and pain in the legs.

Further, more girl children start agricultural work even as early as 10 years. Thus she is vulnerable to all these hazards during a critical period of physical growth. In her late teens, she has the added burden of bearing children, frequent miscarriages and often violence within the household.

To look at these problems in a larger context, we look at a Karnataka level study on the status of rural women and how gender discrimination affects their health.

ACCESS TO HEALTHCARE FOR RURAL WOMEN IN KARNATAKA

A major study on the status of rural women in Karnataka (Srilatha Batliwala, B.K. Anitha, Anita Gurumurthy, Chandana S. Wali : Women's Policy Research and Advocacy (WOPRA) Unit of National Institute of Advanced Studies, Bangalore, Karnataka) focuses attention on the gender discrimination that women, especially poor rural ones, face in their access to healthcare. The study is based on about a thousand case studies covering representative regions of the state. Sangha women from Mahila Samakhya Raichur and Bijapur were part of the study. The following figures illustrate some of the major findings with relation to the health of poor rural women.



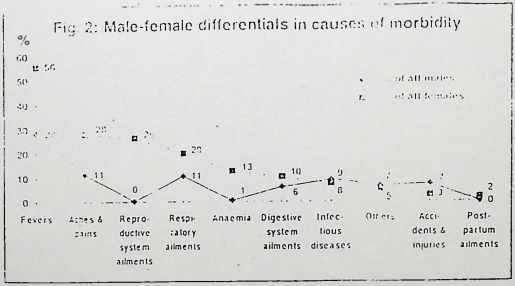
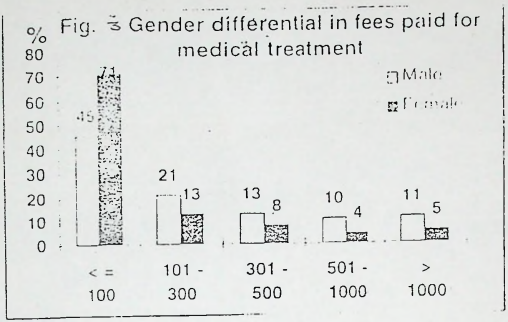


Fig 1 and 2 show that generally, women fall ill oftener and have higher morbidity than men. This can be related to their general poor health status.



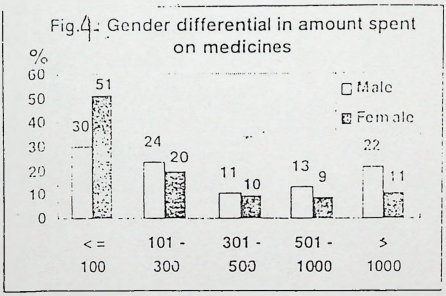


Figure 3 and 4 shows that rural women spend much less on medical treatment than rural men, except if the treatment costs less then Rs.100/-. Most of the diseases arising out of the occupational health hazards described in this paper would warrant expenditures on healthcare greater than Rs.100/-

The study clearly establishes the discrimination and consequent poor health status suffered by rural women. These women are also forced by poverty to eke out a meagre living under exploitative and health-threatening circumstances.

The following work is proposed by Mahila Samakhya, for the coming year.

1. To select a few villages and conduct a detailed study of occupational related health hazards of sangha women.
2. To enhance women's awareness about health hazards and emphasise the use of simple preventive measures.
3. To look for alternative sources of income through the sangha, which will not be hazardous to health.
4. To identify and link with medical help within formal and informal systems.
5. To improve knowledge about existing agricultural practices so that they can improve productivity and resist exploitation.
6. To develop linkages with government agencies working in this field.
7. To identify worker- friendly agricultural practices which can also enhance productivity and income, leading to her better status and consequent better health status and healthcare.

SERICULTURE : MAJOR HEALTH HAZARDS FOR WOMEN

In Karnataka, apart from agriculture, sericulture is also a major occupation for poor women which is a occupational health hazards. In a study by R.K. Datta , Geetha Devi, Ganapathi Rao, CSR&TI, Mysore - 8. From Ms. Swaminathan Report presented at National Seminar on The Technological Empowerment of Women in Agriculture, the hazards faced by women in this industry are described .

Sericulture, an agro-based cottage industry in India, is identified as a potential sector for employment and income generation for rural masses and for women in particular. The support of 60 million people living in 60,000 villages has helped India to come out as a second largest producer of cocoons and silk. In fact silk is processed from reeling of the cocoons, while silkworm eggs are produced by processing live seed cocoons. In both, the role of women becomes vital for maintaining the quality.

The study was done in Ramanagaram, (near Bangalore) the biggest silk producing town in India, shows the following occupational health hazards and its treatements of women working in sericulture.

Sl.No	Activity	Health Hazards	% of women's income spent on medicines
1.	Charkha and the cottage basins	Skin disease on palm and on the body, Asthama, back pain, body ache, shoulder pain and other ailments.	20% of the income of the women is spent on medicines.
2.	Reeling (8 - 10 hours)	Skin disease and asthama	
3.	Seed production a) sorting of cocoons b) moth examination c) picking and handling of eggs for loose eggs preparation.	Allergy due to scales and smell, cough, watering of eyes, nasal irritation, itching of skin dandruff and bronchoitis, bleeding nose, nausea due to the bad smell of melted cocoons, dead moths and pupae, fever, back pain, headache etc.	30% of women's income spent on medicines.

- Women apply lemon juice, tamarind paste, medical ointments and other herbal medicines.
- No formal medical care is provided nor is protective equipment adequate.
- A few government reeling units, however, provide ointment to their workers.

A committee was set up by the Central Silk Board to study health hazards and has made its recommendation.

i) Awareness generation among both women workers and entrepreneurs about hazards, rewards and punitive systems for managements.

ii) Modifying technologies, using protective equipment, increasing preventive and curative healthcare facilities for workers.

BHOPAL'S STORY

(At the onset I must state that I am not one of those affected by the gas leak disaster. I have worked with the survivors as a medical researcher supporting their struggle for justice).

The actors in this drama are the Union Carbide Corporation (American multinational), Union Carbide India Limited (UCC´s Indian subsidiary), the Indian government, the Supreme Court of India, the people who lived in Bhopal in December 1984 (Bhopal is the capital city of Madhya Pradesh in Central India).

The story begins not so long ago, in fact just sixteen years ago...

It was a night in early December. It was a night like any other. Windows and doors were shut right against the chill. The entire city huddled in deep sleep. Well, not quite. For, in a pesticide factory across the road, there was panic as workers working in the night shift, watched with growing horror a huge underground steel tank burst through the concrete floor and vomit its deadly content into the night air. Since all the safety systems had been dismantled or were in a state of disruption, the 40 tons of deadly gases moved, unhampered, downwind only to invert in a malicious perversity to hang over the city for the next seven hours.

As the vapours entered the homes of the poor people through the cracks in the doors, holes in the walls or the gaps in the roof where a missing tile had not been replaced, a pungent smell of burning chillies and peppers filled the air. Couple woke up coughing, spluttering, eyes burning, throats choking. Stumbling from their beds, they threw open their doors for a breath of fresh air only to be assaulted by the poisonous fumes waiting outside. Soon the air was rent with the screaming and wailing of hundreds of people as a nameless dread took hold of them. Those who could, got on to a bicycle or a rickshaw. Those who could not, ran a race for their lives, downwind , where they thought security lay. Men who ran faster, breathed harder and collapsed earlier. Whole families sleeping on the pavements of roads, railway platforms were wiped out in one fell blow.

Did I say it was a night like any other night? It was no longer a night like any other night. Death reaped a grim harvest that night. As the sun rose to shine dimly through the poisonous cloud on the morning of 3rd December, the still living staggered back to look for their own. Mothers who had abandoned their children knew that they will carry this guilt coiled tightly in the pit of their stomachs forever. Moving through the dead and the dying, the carcasses of cattle *littered* the streets, bewildered and benumbed, little did the people of Bhopal know that their *misery* was only just beginning. Soon the hospitals began to be flooded as the *patients* looked in vain for a treatment that did not appear to *exist*. The next few years saw the damage wreaked on the human body. Every system - the respiratory system, gastro-intestinal system, muscular-skeletal, reproductive system, the eyes, the immune system began to manifest progress MULTIMEDIAive irreversible damage. People began to age prematurely. Psychiatric morbidities began to rise. Death continued to stalk the slums of Bhopal with a determined deliberateness.

A health system unable to cope with the enormous numbers of constantly ill population and with the complex nature of injuries began to blame the survivors. They were called malingerers and a new term ´ compensation neurosis ´ was coined to dismiss their distress and suffering. With no more tears to shed, with a grief too deep to mourn, people continued with the necessary task of living. While no one wanted to own up responsibility, the Indian government was only too willing to appropriate the rights of the survivors to litigate. Not just that. The Indian government through its agencies of research, the Indian Council of Medical Research, appropriated the right to medical information about the bodies

of the gas affected people. Information gathered through medical research was under the official Secrets Act.

The only recourse left for the gas victims was to organise and demand their right for medical relief, compensation and justice. However, every attempt to raise these issues was met with ruthless opposition. Peaceful demonstrations were disrupted by the police, demonstrators beaten up and their leaders labelled as terrorists and jailed with grave charge such as "attempt to murder".

Siding with the American multinational UCC, the Indian government on behalf of the victims signed a settlement for a pittance. What was in effect, on out-of-court settlement was blessed by the Indian Supreme Court as a full and final settlement and against all future claims. The rights of children yet to be born was thus quashed. In a twist of irony, if any new claims come up in future, it will be the Indian government that will have to defend the right of UCC. It was a complete sell out.
UCC did not have to pay a single cent more than its insurance coverage. Compensation to individuals ranged from US $ 5000 for deaths to US $ 500 for injuries. Life in India was indeed cheap.

More that 10,000 have been killed in the disaster and more that 200,000 have been injured permanently. Yet, all criminal cases against the culprits have been dismissed. In effect, the Indian Supreme Court's judgement appears to favour the hand of God and not the act of a greedy, immoral, American multinational as the cause of the disaster.

The people of Bhopal, however, have not given up. They know that truth is on their side. Yet, they know they will have to fight and struggle for every inch of their way. They know their lives have been destroyed forever. They know their children's lives have been destroyed forever. And who knows, perhaps the lives of the children's children as well. They know that in a world which is owned by the multinationals, ruled by money, dialogue does not work. For them Warren Anderson, the new UCC chief is an absconder and a fugitive from justice. They demand that he be extradited and tried for the murder of 10,000 people and suffering of several generations. There is indeed no room for dialogue.

The people of Bhopal have become seasoned fighters with the women in the forefront of the struggle. They have nothing to lose and very little to gain for themselves. But they continue to struggle with their supporters, within and outside the country for a better world so that their suffering may not have been in vain.

Their struggle is our struggle, the struggle of all people all over the world whose lives are being poisoned slowly and not so slowly by transnational capital.

I would like to change the title of the story form "continuing tragedy of Bhopal" to "Continuing struggle of Bhopal".

This is not the story of one. This is the story of hundreds of thousands of people. I can not end the story here because the struggle continues...

Sathyamala
Medical friend circle
India

(Testimony presented at the People's Health Assembly, GK-Savar, Bangladesh, 4 - 8th December, 2001)

Health Effects of the Dec. '84 Union Carbide disaster in Bhopal

Mortality

In Nov '89 the Dept of Relief and Rehabilitation placed the death toll at over 6000. In December 1992 the official agency for monitoring exposure related deaths was wound up. Data published by the Department of Relief and Rehabilitation in December 1998 shows that in the year 1997 the mortality rates were 10.4 in the gas-exposed population compared to 6.07 in the unexposed population. Based on an exposed population of 5,72,242 [ICMR's estimate] this works out to over 2000 deaths attributable to exposure in the year 1997 alone.

Overall Morbidity

Symptom prevalence surveys conducted by the ICMR indicate that morbidity was higher in the exposed area [26%] as compared with the control area [18%] when assessed during the period November 1988 to March 1990.

Results from the survey carried out by the International Medical Commission on Bhopal show that a large number of subjects reported general health problems [exposed 94% vs unexposed 52%] and episodes of fever [exposed 7.5 per year vs unexposed 2.5 per year]. Respiratory, neurological, psychiatric and ophthalmic symptoms also showed a strong gradient by exposure category.

Ocular problems

The intensely irritating effect of MIC on the cornea resulted in severe ocular burning, watering, pain and photophobia. Examination showed involvement of the corneal and conjunctival epithelium with redness of the eye, cornea ulceration and lid swelling [1]. Slit lamp examination showed discrete lesions in a band across the interpalpebral area, punctuate keratopathy, conjunctival chemosis and some pigmentary deposition on the cornea [2].

Anderson et. al. performed a survey of exposed and control populations 3 years after the disaster and found photophobia, burning and watering sensation, signs of red eye, superficial interpalpebral erosion, corneal opacity, discharge and fundal changes. Their findings indicated an increased risk of eye infections, hyperresponsive phenomena, excess cataracts and resolution of the corneal erosions in exposed persons. These phenomena have been characterised as the "Bhopal eye syndrome"[3].

Though there is no evidence that severe damage to the eye's external and internal structures has occurred, the single acute exposure seems to have resulted in a chronic inflammatory process.

Respiratory toxicity

Acute symptoms of the respiratory tract were mainly due to the irritant action of MIC on tissues. Because MIC is moderately soluble in water, lesions were seen in both the upper and lower respiratory tract. Though isocyanates are known to be allergenic in the lung, the respiratory toxicity of MIC appears to be primarily due to its irritant nature [4]

Follow up studies with lung biopsies done six months after exposure showed evidence of interstitial fibrosis and bronchiolitis obliterans. These findings were similar to those in several animal studies revealing the close association between animal data and clinical findings in Bhopal victims [5]

Autopsies on 300 victims revealed severe necrotising lesions in the lining of the upper respiratory tract as well as in the bronchioles, alevoli and lung capillaries. Enlarged and oedamatous lungs, consolidation, haemorrhage, bronchopneumonia and acute bronchiolitis were seen [6]

Reproductive toxicity

Concerns that the gas leak had effects on reproductive health were raised early in 1985 when reports indicated that menstrual cycle disruption, leucorrhoea and dysmenorrhoea had occurred in gas-exposed women [7].

An epidemiological survey by Varma showed pregnancy loss and infant mortality to be very high in gas-exposed women [8]

NR Bhandari et. al. documented significant increase in spontaneous abortion, perinatal and neonatal mortality in a pregnancy outcome study carried out in 18, 978 households [9].

Fetotoxicity of MIC has been confirmed through animal experiments [10,11,12].

Genotoxicity

Chromosomal studies done two and half months after the gas leak to evaluate genetic damage showed significant increase in number of breaks and gaps in the exposed subjects. [13]

Cytogenetic studies done 3 years after exposure showed higher frequencies of chromosomal aberrations in the exposed group [14].

Immunotoxicity

Immune function was studied in exposed subjects by Saxena et. al. two and half months after exposure [15]. No difference in mean immunoglobulin levels was found when compared to controls. The T-cell population was found to be less than half of that found normally in the Indian population. Significant depression of phagocytic activity of lymphocytes was found as compared to controls.

Psychological and Neuro-behavioural toxicity

Srinivasamurthy and Isaac noted Post-traumatic stress disorder, pathological grief reaction, emotional reactions to physical problems and exacerbation of pre-existing psychiatric problems among gas victims [16]

Neurobehavioural tests were conducted on 350 exposed subjects two and a half months after the accident. Auditory and visual memory, attention response speed and vigilance were found to be significantly impaired [17].

Neuromuscular toxicity

Neuromuscular symptoms in Bhopal survivors have persisted since the gas leak [18]. The symptoms are mainly tingling, numbness, a sensation of pins and needles in the extremities and muscle ache.

Anderson et. al. evaluated the effects of MIC on rat muscle cells in culture [20]. At lower doses, the formation of muscle fibers was prevented. At higher doses, death of fibroblasts and myoblasts was seen.

References

1. Anderson N, Kerr Muir M, Mehra V, Salmon AG. Exposure and response to methyl isocyanate:results of community - based survey in Bhopal. Br. J Ind Med. 1988;45:469-475.
2. Anderson N, Muir MK, Mehra V. Bhopal Eye. Lancet. 1984;2:1481
3. Anderson N, Ajwani MK, Mahashabde S, Tiwari MK, Kerr Muir M, Mehra V, Ashiru K, Mackenzie CD. Delayed eye and other consequences from exposure to methyl isocyanate: 93% follow up of exposed and unexposed cohorts in Bhopal. Br J Ind Med. 1990;47:553-558.
4. Karol MH, Taskar S, Gangal S, Rubanoff BF, Kamat SR. The antibody response to methyl isocyanate: experimental and clinical findings. Environ Health Perspect. 1987;72:167-173.
5. Boorman GA, Uraih LC, Gupta BN, Bucher JR. Two hour methyl isocyanate inhalation and 90 day recovery study in B6C3F1 mice. Environ Health Perspect. 1987;72:107-114.
6. Indian Council of Medical research. Health effects of exposure to toxci gas at Bhopal: an update on ICMR sponsored researches. Dec 1985.
7. Bang R, Sadgopal M. Effect of Bhopal disaster on women's health - an epidemic of gyanecological diseases [PartI]. 1985. Unpublished data.
8. Varma DR. Epidemiological and experimental studies on the effects of methyl isocyanate on the course of pregnancy. Environ Hlth Perspect. 1987;72:151-55.
9. Bhandari NR, Syal AK, Kambo I et. al.. Pregnancy outcome survey in women exposed to toxic gas at Bhopal. Ind J Med Res 92:28-33 (1990)

10. Schwetz BA, Adkins B, Jr., Harris M, Moorman M, Sloan R. Methyl Isocyanate: Reproductive and developemental toxicology studies in Swiss mice. Environ Hlth Perspect. 1987;72:147-150.
11. Varma DR, Ferguson J, Alarie Y. Reproductive toxicity of methyl isocyanate in mice. J Toxicol Env Hlth 1987;21:265-275.
12. Varma DR, Guest I, Smith S, Mulay S. Dissociation between maternal and fetal toxicity of methyl isocyanate in mice and rats. J Toxicol Env Hlth. 1990;30:1-14.
13. Saxena AK, Singh KP, Nagle SL, Gupta BN, Ray PK, Srivastav RK, Tewari SP, Singh R. Effect of exposure to toxic gas on the population of Bhopal: Part IV-Immunological and Chromosomal Studies. Ind J Exp Biol. 1988;26:173-176.
14. Ghosh BB, Sengupta S, Roy A, Maity S, Ghosh S, Talukdar G, Sharma A. Cytogenetic Studies in Human Populations Exposed to Gas Leak at Bhopal, India. Environ Hlth Perspect. 1990;86:323-326.
15. Saxena AK, Singh KP, Nagle SL, Gupta BN, Ray PK, Srivastav RK, Tewari SP, Singh R. Effect of exposure to toxic gas on the population of Bhopal: Part IV-Immunological and Chromosomal Studies. Ind J Exp Biol. 1988;26:173-176.
16. Srinivasamurthy R, Isaac MK. Mental health needs of Bhopal disaster victims and training of medical officers in mental health aspects. Ind J Med Res 1987;86(Suppl):51-58.
17. Gupta BN, Rastogi SK, Chandra H, Mathur AK, Mahendra PN, Pangtey BS, Kumar P, seth RK, Dwivedi RS, Ray PK. Effect of exposure to toxic gas on the population of Bhopal: Part I-Epidemiological, clinical, radiological & behavioural studies. Ind J Exp Biol 1988;26:149-160.
18. Indian Council of Medical Research. Bhopal Gas Disaster Research Centre. Annual report. Bhopal, India. 1991.
19. Anderson D, Goyle S, Philips BJ, Tee A, Beech L, Butler WH. Effect of methyls isocyanate on rat muscle cells in culture. Br J Ind Med 1988;45(4):269-74.

IN MEMORY OF BHOPAL

Auther: Binu Mathew

Tears are still flowing down from the eyes of the Bhopal gas victims. Their tears have not dried even after 17 years since one of the worst ever industrial disasters occurred. For someone like Selma, daughter of Razia Bee of Mangalwara, tears run down in blood red colour as though symbolising the plight of the whole victims.

It was on the night of December 2, 1984 that 40 tons of Methyl Isocyanate gas leaked from the Union Carbide Factory (UCC) situated in one of the most densely populated neighbourhood of Bhopal, Madhya Pradesh, India. The first time the management of the Carbide plant came to know about the leak was at 11:00 pm. The factory alarm meant for workers was started by a desperate worker at 12:50 pm. The management not only turned it off within minutes but also delayed the sounding of the public siren until as late as 2:00pm by which time all the gas that could leak had leaked.

8000 people died in the immediate aftermath of the disaster. After 17 years, the death toll has risen to over 20,000 and 10-15 people are dying every month from exposure related diseases. Over 120,000 children, men and women continue to suffer acutely from a host of exposure related illnesses and their complications.

Damage to the respiratory system has led to the prevalence of pulmonary tuberculosis which has been found to be more than three times the national average. In the years following the disaster, the stillbirth rate was three times, prenatal mortality was two times and neonatal mortality was one and a half times more than the comparative national figures. According to a study by Dr. Daya Varma, Mcgill University, Canada, 40% of the women pregnant at the time of the disaster aborted. Another study reported nearly five times increase in the rate of spontaneous abortion as a result of the Union Carbide disaster.

The survivors complain of breathlessness, coughing, chest pains, fatigue, body aches, abdominal pain, numbness and tingling in the limbs, weak sight and runny eyes, anxiety attacks, bad memory, concentration difficulties, irritability, headache and mental illness.

An unusually large number of women have menstrual irregularities and excessive vaginal secretions. Mothers complain of retarded physical and mental growth in children exposed in infancy or born after the disaster. According to a study conducted by Ingrid Eckerman, there are reports on

intellectual impairment and epilepsy. Failure to grow, delay in gross motor and language sector development was found in children born a considerable time after their mother's exposure to the gas.

The worst part of the disaster is probably yet to come. Researchers have found chromosomal aberrations in the exposed population indicating a strong likelihood of congenital malformations in the generations to come.

A detailed study of of psychological disorders caused by the disaster has not yet been taken up. But post-traumatic stress disorders, pathological grief reactions, emotional reactions to physical problems and exacerbation of pre-existing psychiatric problems have been noted.

To top it all is the rupture in family and social relations in the affected areas due to death, illness and poverty.

All this misery would have been much less had UCC revealed the exact nature of the compositon of the gases released from the plant. To this day they have not done that.

On the December 3 morning of 1984 when victims started to pour into the hospitals, the bewildered Bhopal doctors contacted the plant doctor and the reply they got from him was that " It is only like tear gas. Just wash with water". Without information on the nature of the gases, docotors are still giving symptomatic treatment, which gives only partial relief.

Within the first week of the disaster 4 'medical experts' came to Bhopal on a visit sponsored by UCC. In their interviews to the media, they stated that the leaked gases would not have any long term health effects on the exposed population. This was in sharp contrast to the subsequent research findings. One of these experts was Brian Ballyentine, who was also a toxicologist for the Pentagon. Another expert, Dr Hans Weil, Prof. and Chairman of Pulmonary Medicine at the Tulane University Medical School, New Orleans, has a history of fudging medical data to minimize liabilities of Corporations (a prime example being that of Johns Manville Inc. in the Asbestosis case), and had been reprimanded in the past by a US court for his unethical conduct. He examined victims in Bhopal and said "they have an encouraging prognosis and most would recover fully."

After the disaster Dr. Max Daunderer, a toxicologist from Munich, demonstrated the efficacy of intravenous sodium thiosulphate injections in detoxifying the exposed persons and providing substantial relief in symptoms. This was further confirmed by studies carried out by the Indian Council for Medical Research. Through helpful government officials, UCC succeeded in undermining official attempts for large scale administration of sodium thiosulphate. The company was quick to realise

that the administration of this drug would establish that its toxins had indeed reached the blood stream and caused much more damage than the company would like people to believe.

Greatest sell off of all was the out of court damage settlement reached between Government of India and UCC. On December 14, 1989 it was announced in the Supreme Court of India that Union Carbide will pay $ 470 million as damages to the disaster.

The first suit filed by Melvin Belli, an American lawyer, claimed damages upto $15 billion. Later the Indian Government arrogating itself the sole power to represent all the victims, filed a suit for $3.3 billion. 4 years after filing the suit and without informing the victims, the government settled for nearly one-seventh of the original claim. Of the $470 million settlement $200 million was covered by UCC's insurance and another $200 million had already been put aside. Out of an annual revenue of $8 billion a year, the corporation had to find just $70 million to close the books on the worst industrial disaster in history.

After news of the $470 million settlement, Carbide's stock actually increased $2 a share. The then chairman, Robert Kennedy who owned 35,000 shares in the company, personally benefitted $70,000.

The settlement clearly shows a double standard in treating victims of industrial disasters in India and elsewhere. Union Carbide and eight other companies paid US $ 4.2 billion as potential damages for Silicone Breast Implants to 650,000 claimants. This amount was 9 times more than what the Bhopal victims were given and less than a 10th of the $5 billion court award against Exon Valdez for polluting the Alaskan coast. Approximately US $ 40,000 was spent on the rehabilitation of every sea otter affected by the Alaska oil spill. Each sea otter was given rations of lobsters costing US $ 500 per day. Thus the life of an Indian citizen in Bhopal was clearly much cheaper than that of a sea otter in America. If the award amount of $470 million where distributed equally among all the victims of Bhopal disaster each would get around only $200.

Many of the people did not get even that much relief. More than 250,000 claims were never documented or classified, making it hard for these victims to obtain compensation. The largest amount paid for death was around $ 2,000.

Many of the victims in the gas trgedy were poor, illiterate people. They had no idea of compensation or the importance of keeping records. When the government agencies demanded documents, they had nothing to provide. And some who had documents lost it in the 1992 Hindu – Muslim riot.

There is no provision for providing compensation for severly affected children who are born after the disaster.
According to the settlement the liability to provide adequate compensation and facilities for the handicapped victims requiring long-term follow up and treatment rest with the Indian Government and not with Union Carbide Corporation.
Union Carbide was also exonerated of the responsibility to clean up the affected area. On the occasion of the 15th anniversary of the disaster Greenpeace named the area in the vicinty of the Union Carbide factory in Bhopal a "Global Toxic hotspot". Their report based on samples collected from in and around the factory premises – indicates severe contamination of the ground water and soil with heavy metals and carcinogenic chemicals. In 1990 the Bhopal Group for Information and Action(BGIA) reported the presence of atleast seven toxic chemicals based on a report by the Citizens Environmental Laboratory, Boston. Toxic chemicals are reported even in the breast milk of mothers around factory vicinity. Indian government has not taken this matter seriously, only in one community is there a provision made for supplying drinking water in tankers. All the other neighbourhoods are using toxic drinking water.

The Indian Governments attempts in rehabilitating the disaster victims is a picture of total neglect, apathy, inefficiency and corruption. As per offici records of the Gas Relief and Rehabilitation Department, Government of Madhya Pradesh, a total of $ 80 million have been spent on relief and relief and rehabilitation of the survivors of Bhopal since the disaster. The compensation money has multiplied in terms of Indian rupees as a result of the increase in the value of the dollar and the accruing interest. However the interest has not been paid to the claimants as it should have and a balance of about $200 million is likely to remain after all compensaton are to be settled at the present rate.

For legal redressal of this disaster and the wrong done to the victims, seven individuals and five organizations filed a class action suit on November 15, 1999 in the Federal District Court of New York, against Union Carbide Corporation and its former chairman Warren Anderson. The suit charged the Corporation and the official with grave violations of international law and the human rights arising from their "reckless and depraved indifference to human life" in perpetrating the disaster. The support of the Indian Government is crucial to the success of this legal action. But the request of the victims to the Indian Government to present an "amicus curiae" brief has so far fallen on deaf ears.

As the victims are moving from one hell to hell, the perpetrators of the crime are romping free around the world. On Dec. 7th 1984, Warren Anderson, and other Indian officials were arrested on charges of culpable homicide, criminal conspiracy and other serious offences. The arrested officials were lodged in the posh guest house of Union Carbide and Warren Anderson with an annual salary of Rs.10 million, was released on the same day on a bail of Rs.20,000. Summons from the Bhopal court drew no response from him and in January 1992 proclamations were published in Washington Post directing Anderson to face trial in the Bhopal court. In March 1992 the Chief Judicial Magistrate issued a non-bailable arrest warrant against Warren Anderson. He continues to abscond criminal justice.

The hope of the victims ever to receive justice received a setback with the merger of Union Carbide Corporation with Dow Chemicals, in February this year. With the merger, UCC has vanished as an entity and Dow became the second largest chemical corporation in the world. In its submission to the Securities and Exchange Commission, USA, Union Carbide has deliberately omitted the the mention of pending criminal liabalites of the corporation. This fact was brought by victims organisation to the notice of the SEC, but met with indifference and deliberate silence.

Manu's law, the ancient Hindu law practised in India, held a person who had injured another to pay damages, not according to the status of the victim but according to the status of the wrongdoer. This law, has never been repealed. This rule of the ancient common law of India, enforced by many rulers in the last thousand years and more, is still the unacknowledged law of India. And it goes on, as we now see in the case of the Bhopal victims too.

Health Effects of the Dec. '84 Union Carbide disaster in Bhopal

Death Toll

In Nov '89 the Dept of Relief and Rehabilitation placed the death toll at over 6000. In December 1992 the official agency for monitoring the deaths among the persons exposed to the gas was wound up. Data published by the Department of Relief and Rehabilitation in December 1998 shows that in the year 1997 the death rates were 10.4 in the gas-exposed population compared to 6.07 in the unexposed population. Based on an exposed population of 5,72,242 [ICMR's estimate] this works out to over 2000 deaths attributable to exposure in the year 1997 alone.

Overall Diseased

Symptomatic disease in the population surveys conducted by the ICMR indicate that persons who were ill was higher in the exposed area [26%] as compared with the control area [18%] when assessed during the period November 1988 to March 1990

Results from the survey carried out by the International Medical Commission on Bhopal show that a large number of people reported general health problems [exposed 94% vs unexposed 52%] and episodes of fever [exposed 7.5 per year vs unexposed 2.5 per year]. Respiratory, neurological [nervous system], psychiatric [mental illness] and ophthalmic [eye related] symptoms also showed a strong gradient by exposure category.

Problems with the eye

The intensely irritating effect of MIC [methyl isocyanate] on the eyeball resulted in severe burning, watering, pain and aversion to light in the eye. Examination showed redness and ulcers in some parts of the eye and lid swelling.
Examination also showed discrete lesions in a band across the inner aspect of the eyelids, whitish spots, swelling and some pigmentary deposition on the eye.

Anderson et. al. performed a survey of exposed and control populations 3 years after the disaster and found aversion to light, burning and watering sensation, signs of red eye, glazing of the eye. Their findings indicated an increased risk of eye infections, hyper responsive phenomena, excess cataracts and resolution of the ulcerations in the eye in exposed persons. These phenomena have been characterised as the "Bhopal eye syndrome".

Though there is no evidence that severe damage to the eye's external and internal structures has occurred, the single acute exposure seems to have resulted in a chronic inflammatory process.

Respiratory toxicity

Acute symptoms of the respiratory tract were mainly due to the irritant action of MIC on tissues. Because MIC is moderately solubie in water, lesions were seen in both the upper and lower respiratory tract. Though isocyanates are known to be allergenic in the lung, the respiratory toxicity of MIC appears to be primarily due to its irritant nature.

Follow up studies with lung biopsies done six months after exposure showed evidence of thickening of tissues and induration of air sacs. These findings were similar to those in several animal studies revealing the close association between animal data and clinical findings in Bhopal victims.

Autopsies on 300 victims revealed severe death of cells resulting in lesions in the lining of the upper respiratory tract as well as in the air sacs and lung capillaries. Enlarged and fluid filled lungs, consolidation, internal bleeding and acute inflammation of the lungs were seen.

Reproductive toxicity

Concerns that the gas leak had effects on reproductive health were raised early in 1985 when reports indicated that monthly periods cycle disruption, whitish discharge and painful periods had occurred in gas-exposed women.

An epidemiological survey by Varma showed spontaneous abortion loss and infant death to be very high in gas-exposed women.

NR Bhandari et. al. documented significant increase in spontaneous abortion, Infants and infant death rate in the womb in a pregnancy outcome study carried out in 18, 978 households.

Fetotoxicity [ill effects on the unborn child] of MIC has been confirmed through animal experiments.

Genetictoxicity

Chromosomal studies done two and half months after the gas leak to evaluate genetic damage showed significant increase in number of breaks and gaps in the exposed subjects.

Cytogenetic studies done 3 years after exposure showed higher frequencies of chromosomal alterations in the exposed group.

Immunotoxicity

Immune function was studied in exposed subjects by Saxena et. al. two and half months after exposure. No difference in mean immunoglobulin levels [part of bodies defense system] was found compared to controls. The defense cell population was found to be less than half of that found normally in the Indian population. Significant depression of fighter activity of defense cells was found as compared to controls.

Psychological and Neuro-behavioural toxicity

Srinivasamurthy and Isaac noted Post-traumatic stress disorder, pathological grief reaction, emotional reactions to physical problems and exacerbation of pre-existing psychiatric problems among gas victims.

Neurobehavioural tests were conducted on 350 exposed subjects two and a half months after the accident. Auditory and visual memory, attention response speed and vigilance were found to be significantly impaired.

Neuromuscular toxicity

Neuromuscular symptoms in Bhopal survivors have persisted since the gas leak. The symptoms are mainly tingling, numbness, a sensation of pins and needles in the extremities and muscle ache.

Anderson et. al. evaluated the effects of MIC on rat muscle cells in culture. At lower doses, the formation of muscle fibers was prevented. At higher doses, death of cells that help in muscle building.

In summary, the exposure to toxic gases during the Bhopal disaster has resulted in serious, wide ranging and long term adverse effects on the health of about 500,000 people who were living in that area. Following chromosomal damage, it is also effecting the next generation born to those exposed.

[This note has been prepared from reports of studies undertaken in Bhopal]

OH-1

INDIAN ASSOCIATION OF OCCUPATIONAL HEALTH

51st NATIONAL CONFERENCE

"Challenges for Working life and Environment in New Millennium"

SCIENTIFIC PROGRAMME

February 1st to February 4th, 2001

Organised by IAOH Delhi Chapter

India Habitat Centre
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51st National Conference of IAOH
India Habitat Centre Lodhi Road, New Delhi
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CHALLENGES FOR WORKING LIFE AND ENVIRONMENT IN NEW MILLENNIUM

Scientific Programme

1st February 2001

Time	Topic	Speaker
9.00-10.00 a.m.	Registration & Assembly	
	PLENARY SESSION - I	
Chair Persons	**Dr. T.K. Joshi & Dr. S.M. Shanbagh**	
10.00-11.00 a.m.	The ILO Overall Approach Towards Occupational Health	**Dr. I. Fedotov**
11.00-11.15 a.m.	**TEA**	
11.15-12.00 noon	**3rd Central Council Meeting**	
	TECHNICAL SESSION - I	
Chair Persons	**Dr. A.K. Mandal & Dr. Abhoy Shanker**	
11.15-12.15 p.m.	Paper Presentations	
	PLENARY SESSION - II	
Chair Persons	**Dr. Bharat Singh & Dr. Deepak Tyagi**	
12.15-1.00 p.m.	Key Note Address on Hospital Waste Management	**Dr. T.K. Joshi**
1.00-2.00 p.m.	**LUNCH**	
	SYMPOSIUM - A	
Chairman	**Dr. Naresh Trehan & Dr. R.R. Kasliwal**	
2.00-2.45 p.m.	CAD-An Indian Perspective	**Dr. Kasliwal**
2.45-3.00 p.m.	A.E.D.-A New Methodology For Sudden Cardiac Death	**Dr. N. Chandra**
3.00-3.15 p.m.	Heart Failure Clinic - A New Concept	**Dr. Sanjay Mittal**
3.15-3.45 p.m.	Pannel Discussion Question & Answers	**Dr. Kasliwal** **Dr. N. Chandra** **Dr. S. Srivastava** **Dr. Sanjay Mittal**
	PLENARY SESSION - III	
Chair Persons	**Dr. R.R. Kasliwal & Dr. Sameer Srivastava**	
3.45-4.30 p.m.	Minimal Invasive Surgery at The dawn of New Millennium	**Dr. Naresh Trehan**
4.30-6.00 p.m.	**INAUGURATION**	
6.00 p.m.	**TEA**	
7.00-8.00 p.m.	**Cultural program (IMA House)**	
8.00 p.m. onward	**Cocktail Dinner (IMA House)**	

2-00 PM →

2nd February 2001

Time	Topic	Speaker
8.30-9.00 a.m.	REGISTRATION	
	PLENARY SESSION - IV	
Chair Persons	**Dr. Barry Castleman & Dr. A.K. Mandal**	
9.00-10.00 a.m.	Occupational Health Problems of Chainsaw use in Forest Workers	**Dr. M. Tanaka**
	Finger Function of Forest Workers	**Dr. K. Tanaka**
10.00-10.15 a.m.	**TEA**	
	PLENARY SESSION - V	
Chair Persons	**Dr. Sudha Ramchandran**	
10.15-11.15 a.m.	ADM ORATION	**Dr. S.M. Shanbagh**
	PLENARY SESSION - VI	
Chair Persons	**Dr. N.K. Ganguly & Dr. G.K. Kulkarni**	
11.15-12.00 noon	Occupational Health In Developing Countries	**Dr. Barry Levy**
	PLENARY SESSION - VII	
Chair Persons	**Dr. Qamar Rahman & Dr. Rajiv Garg**	
12.00-1.00 p.m.	Controverses in Asbestos and Man made Fibres	**Dr. Arthur Frank**
1.00-2.00 p.m.	**LUNCH**	
	PLENARY SESSION - VIII	
Chairman	**Dr. T.K. Joshi & Dr. M. Tanaka**	
2.00-3.00 p.m.	Role of Emerging Technology In Mitigation of Occupational & Environmental Health Hazards	**Dr. R.A. Mashelkar**
	SYMPOSIUM - B	
Chair Persons	**Dr. S.P. Aggarwal & Dr. Bharat Singh**	
3.00-4.30 p.m.	Symposium on **Hospital Waste Management**	**Dr. S.P. Aggarwal** **Dr. Bharat Singh** **Dr. C.P. Singh** **Dr. T.K. Joshi** **Dr. B. Sengupta** **Dr. Dwarkanath**
4.30-4.45 p.m.	**TEA**	
	SYMPOSIUM - C	
Chair Persons	**Dr. Arthur Frank & Ms. Rani Advani**	
4.45-6.00 p.m.	Symposium on **Banning Asbestos**	**Dr. B. Castleman** **Dr. Arthur Frank** **Dr. A.K. Mandal** **Dr. S.K. Dave** **Dr. Rani Advani** **Dr. Qamar Rahman**

3rd February 2001

Time	Topic	Speaker
	TECHNICAL SESSION - II	
Chair Persons	**Dr. S.K. Sahoo & Dr. N.K. Jain**	
9.00-10.00 a.m.	Paper Presentation	
	TECHNICAL SESSION - III	
Chair Person	**Dr. S.K. Jain & Dr. Rajkumar**	
10.00-11.00 a.m.	Paper Presentation	
11.00-11.15 a.m.	**TEA**	
	PLENARY SESSION - IX	
Chair Persons	**Dr. N. Jayanandan & Dr. Rita Bakshi**	
11.15-12.00 noon	**CKR Oration**	**Dr. M.R. Jape**
	PLENARY SESSION - X	
Chair Persons	**Dr. S.K. Dave & Dr. S.K. Puri**	
12.00-1.00 p.m.	Global Asbestos Challenges In India	**Dr. B. Castleman**
1.00-2.00 p.m.	**LUNCH**	
	TECHNICAL SESSION - IV	
Chair Persons	**Dr. Shivramkrishan & Dr. O.P. Agarwal**	
2.00-2.40 p.m.	Miners Health in India	**Dr. S.K. Dave**
2.40-3.00 p.m.	Air Pollution	**Dr. Rajiv Garg**
3.00-3.20 p.m.	Bhopal Gas Disaster and Its aftermath	**Dr. V.K. Vijyan**
3.20-3.35 p.m.	**TEA**	
	TECHNICAL SESSION - V	
Chair Persons	**Dr. N. Chakraborty & Dr. Rakesh Garg**	
3.35-5.00 p.m.	Paper Presentation	
5.00-6.00 p.m.	GBM	
6.00-7.00 p.m.	First Central Council Meeting	
7.30 p.m. onward	**Banquet**	

4th February 2001

Time	Topic	Speaker
	TECHNICAL SESSION - VI	
Chair Persons	**Dr. P.K. Sishodiya & Dr. P.K. Duggal**	
9.00-10.00 a.m.	Paper Presentation	
	TECHNICAL SESSION - VII	
Chair Person	**Dr. Vivek Chandra Rao & Dr. Amita Sharma**	
10.00-11.00 a.m.	Paper Presentation	
11.00-11.15 a.m.	**TEA**	
Presentations		
	PLENARY SESSION - XI	
Chair Person	**Dr. S.V. Datar**	
11.15-12.00 p.m.	L & T Oration	**Dr. Chandrasekharan**
12.00-1.00 p.m.	**Valedictory Function**	
1.00-2.00 p.m.	**LUNCH**	

3rd Council Meeting – 11.15 a.m. on 1-02-2001

General Body Meeting – 5-6 p.m. on 3-02-2001

1st Council Meeting – 6 p.m. on 3-02-2001

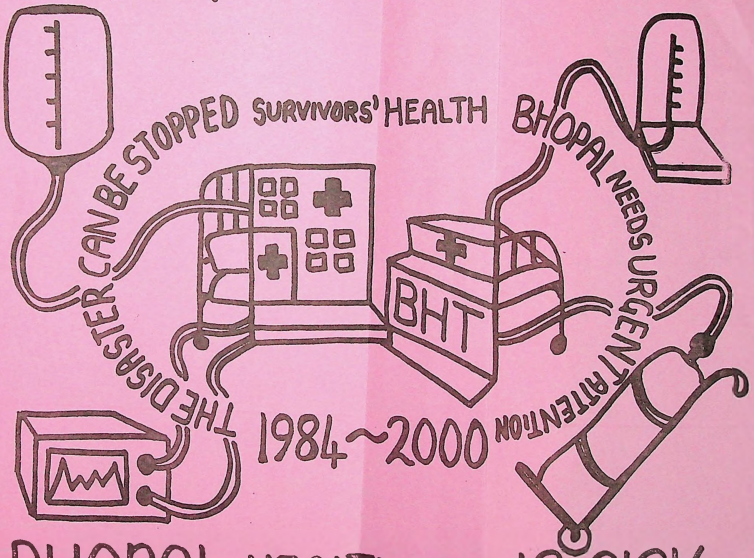
Organising Secretary

DR. RAJIV GARG

Sr. Specialist, I.G. ESI Hospital
Jhilmil, Delhi-110095
Tel.: 91-11-2152197, 2152264
Res.: 91-4610103, 91-4622062
E-mail : drrajivgarg@yahoo.com

OH-1

STOP the ONGOING MEDICAL DISASTER



BHOPAL HEALTHCARE IS SICK

Basic Information	Department of Bhopal Gas Tragedy Relief and Rehabilitation	Sambhavna Trust/ Bhopal Peoples' Health and Documentation Clinic	Bhopal Hospital Trust/ Bhopal Memorial Hospital Trust
stablished	1985	1995/1996	1992/1998
esources	Grants from Union government	Individual donations and voluntary work contribution from India and other countries	Sale of Union Carbide's shares that were confiscated in the criminal case
ctivities	Medical relief and research through the Centre for Rehabilitation Studies	Medical care Monitoring Research Community organisation Health education	Medical treatment
dministration	Bureaucrats headed by Principal Secretary	Board of Trustees includes three individuals who have been involved in medical care of survivors from the time of the disaster	Board of Trustees, none of whom have had any involvement in medical care of survivors. At least two trustee posts remain vacant. Remains without representation of social workers.
Total expenditure	Approx Rs. 250/ crores	Approx Rs. 49.36/ lacs	Well over Rs.100/ crores
Staff	140 doctors and about four times as many para-medical and other staff	20 including 4 doctors	About 100 doctors and about four times as many para-medical and other staff.
Population served	About 5,00,000	Over 20,000	NA
Facilities	7 hospitals, 2 poly clinics, 5 civil dispensaries	1 clinic	1 hospital, 5 clinics
Beds	260	None	424
No. of Beneficiaries per day	About 4750 (Gas victims+Non gas victims)	About 100	About 1000
No. of persons registered for care	NA	9742	About 9,000

Problem	Solution	Bhopal Gas Tragedy Relief & Rehabilitation Dept. M.P. Government	Sambhavna Trust	Bhopal Memorial Hospital Trust
• Lack of information on the health status of the gas victims.	• Epidemiological and clinical research. • Monitoring of health conditions of the gas victims undergoing treatment.	• Information gathered by the Centre for Rehabilitation Studies (CRS), that was established with a corpus of Rs. 5 crores, is ridiculous and laughable. • Absence of monitoring of health status of survivors undergoing treatment.	• Community based survey of health status and health care in five severely affected communities with population of over 10,000. Three of these reports have been published. • Information on the health status of persons undergoing treatment are recorded and computerised on a regular basis.	• Absence of epidemiological or clinical research. No published information. • Absence of system to monitor the health status of the gas victims undergoing treatment.
• Lack of information on continuing exposure related mortality.	• Community based methods for monitoring ongoing exposure related mortality. • Development of criteria to establish the nexus or its absence between exposure and subsequent death.	• Based on the 1998 report of the CRS, a total of 2165 deaths in 1997 are attributable to gas exposure. There is no information on mortalities in subsequent years. • Scrutiny committee consisting of the Head of the Departments of Gandhi Medical College and District officials for monitoring exposure related deaths was dissolved in December 1992.	• Successful implementation of the technique of Verbal Autopsy since 1995 for monitoring exposure related mortality in the gas affected communities. • 86% of the deaths in gas affected communities found to be related to the gas disaster.	• Absence of system to monitor exposure related mortality.
• Lack of information on treatment protocol of the gas induced illnesses.	• Developing appropriate treatment protocols for exposure related symptom complexes.	• Treatment protocol published by Indian Council of Medical Research (ICMR) is 11 years old. Scant attention has been paid in this publication to problems other than respiratory. More than 90% of the Government doctors are unaware of any such publication let alone are familiar with its contents.	• Constant efforts being made towards developing treatment protocols for specific symptom complexes.	• No efforts in this direction.
• Indiscriminate use of harmful and unnecessary drugs in the treatment of gas victims.	• Ban on stocking and prescribing harmful and unnecessary drugs.	• According to a joint study conducted by Socially Active Medicos (Indore) and the Bhopal Group for Information and Action, Bhopal, 27% of the drugs used in government hospitals are either harmful or unnecessary and 13% of the drugs are banned in other countries. • A study conducted by International Medical Commission on Bhopal, highlighted indiscriminate use of steroids, antibiotics, psychotropic and symptomatic drugs in government hospitals.	• 2.7% of drugs used in the clinic were harmful/unnecessary. The mistake has since been corrected. • Vetting of clinic's drug list by specialist in pharmacology.	• Delhi based Dr. Atanu Sarkar analysed the drugs prescribed in the BMHT run clinics, and found that: 26% of drugs prescribed are harmful 49% are unnecessary 8% are both harmful and unnecessary.

Harmful health effects of drugs used in the treatment of gas victims.	Including the use of drug-free therapies and therapies based on medicinal herbs in the medical treatment of the survivors.	Absence of drug-free therapies such as yoga, massage etc. Budgetary allocation for Ayurved and Unani system of treatment is only 1% of the total medical budget.	Successful treatment of gas-induced disorders, through Yoga and massage therapy. Treatment through Ayurvedic medicines and Panchakarma procedures.	No arrangement of treatment through drug-free therapies and medicinal herbs.
Alarming rise in number of TB patients in the gas affected communities.	Community based program for identification, prevention and treatment of TB.	Investigation and treatment of TB is hospital based. No initiatives in health education, follow-up and prevention of TB through community involvement.	Successful development of a system to monitor, prevent and treat TB through community based health education involving voluntary participation from individuals in the gas affected communities. Decrease in number of non-complying patients through intensive follow up in the communities.	Hospital based diagnosis and treatment of TB is in a worse condition than government hospitals. Absence of community based programmes for identification prevention and treatment of and health education on TB.
Absence of any scientific research on treatment of exposure-induced illness.	Continuous scientific research on gas induced illnesses and their treatment.	No scientific research on treatment of exposure induced illnesses.	Scientific research on effects of Pranayama and Yogasana on gas related respiratory disorders. Scientific papers presented in national and international conferences.	Absence of scientific information of treatment of exposureinduced illnesses.
Failure of treatment to provid sustained relief.	Continuous monitoring of efficacy of treatment.	No information on efficacy of therapeutic intervention.	More than 75% of persons have reported more than 25% relief in their illness, on their fifth visit to the clinic.	Absence of information on efficacy of treatment.
Lack of proper attention and information on gas induced gynaecological disorders.	Special attention to gathering information on gynaecological health problems, their investigation and treatment.	No reseach on gynaecological consequences of the disaster in spite of a special hospital for women care. Lack of essential facilities, equipment & specialists to deal with gynaecological problems.	Ongoing scientific research on gas induced gynaecological health problems. Facilities for regular Pap's smear examinations and other equipment available only at Sambhavna.	Lack of information on gas induced gynaecological health problems. Lack of necessary equipment and specialists.
Lack of proper attention and information on gas induced mental health problems.	Special attention to gathering information on mental health problems and their treatment.	In spite of the ICMR reporting a high prevalence of mental illness in the survivors, no current reliable information on mental health problems. Just one psychiatrist in the entire system of health care.	Regular visit by mental health specialist to treat problems such as panic disorders, depression, irritability, insomnia and others. Shirodhara technique of Panchakarma has been successfully employed to cure mental illness.	Lack of current information on gas induced mental illness and non availability of mental health care professionals.
Patients dropping out of treatment.	Counseling and monitoring of patients who drop out of treatment.	Absence of facilities to monitor and counsel drop out patients.	House visits made by community health workers to monitor and counsel drop out patients.	Absence of facilities to monitor and counsel drop out patients.

Stop Press

ON DECEMBER 1, 2000, THE CHIEF MINISTER MR. DIGVIJAY SINGH ANNOUNCED AT A"MEET THE PRESS" MEETING THAT THE DEPARTMENT HAS OUTLIVED ITS UTILITY.,SINCE THE "CITY'S RSIDENTS ARE NO LONGER SUFFERING FROM THE AFTER EFFECTS OF THE BHOPAL GAS DISASTER.

If you think otherwise, please let him know :

Mr. Digvijay Singh
Chief Minister
State of Madhya Pradesh Mantralaya
Ballabh Bhavan, Bhopal
Tel: 755-551433 / 551396 / 551581
Email : cm@mpchiefminister.com

• Information on health impacts of exposure, their prevention and treatment not available to gas victims.	• Community health education among survivors.	• Leaving aside the health education of gas victims, the investigation reports of blood, sputum, urine and smear samples collected by the ICMR in the course of their study involving more than 80,000 gas victims have not being given to concerned persons.	• With the help and cooperation of voluntary workers from gas affected communities, health workers have been successfully conducting health education programs.	• No initiatives towards health education among survivors.
• Corruption and misuse of resources.	• Public scrutiny of income and expenditure of the institutions.	• The Comptroller and Auditor General's reports have charged the M.P. Government with mal-administration of funds in the relief and rehabilitation of gas victims.	• Yearly accounts of income and expenditure are displayed publicly every year and are publicly available.	• The matter of unauthorised withdrawal and expenditure of funds by the former trustee of the Bhopal Hospital Trust, Mr Ian Percival still pending in the Supreme Court. Rs. 5 Crores spent by Percival on office refurbishment, travel and other accounts. Evidence of corruption in the purchase of equipment for the hospital.
• Lack of scientific consultation on gas induced illnesses and their treatment.	• Ensuring participation of specialist doctors and scientists in the treatment of gas victims.	• Absence of specialist consultants in the treatment of gas victims.	• National and international advisory groups give specialist advice. Three of the trustees have been and continue to be involved in the treatment of the gas victims since the disaster.	• Absence of specialist consultant. None of the trustees have any experience in the medical care of gas victims.
• Lack of medical information on gas induced illnesses and their treatment.	• Generation of scientific information on health impacts of the gas disaster.	• No efforts in this direction.	• Scientific and medical information collected by our documentation centre is publicly available.	• No efforts in this direction.
• Lack of information on health and health care in the gas affected communities.	• Dissemination of information on health impacts and preventive and ameliorative measures to survivors.	• No system of issuing health books to gas victims.	• A health book issued to every person registered for care at the clinic.	• Initially the health books were issued to persons taking treatment but these were taken back when severe irregularities regarding treatment were brought to light through analysis of information in these health books. Presently no system for providing health books to patients.
• Lack of equipment for diagnosis of exposure induced illnesses.	• Ensuring availability and utilisation of medical equipment.	• Equipment available but not being utilised.	• Several types of diagnostic equipment are not available.	• Equipment available but limited utilisation.

Disclaimer

Formal request [acknowledgement received] for information for this publication

To,
The Director
Bhopal Memorial Hospital & Research Centre
Raisen Bypass Road [Karond]
Bhopal 462038

Through Ms. Sanghamitra Mohanty
Public Relations Oficer

November 27, 2000

Sub : Request for information on the Bhopal Memorial Hospital & Research Centre

Dear Sir,

Our trust, Sambhavna is a registered public charitable Trust with the primary objective of improving the condition of health and health care of the survivors of the December '84 Union Carbide disaster in Bhopal, through medical care, research, health education and information dissemination.

On the occasion of the 16th anniversary of the disaster [December 2-3, 1984] we wish to publish a fact sheet on the health and health care situation of the gas affected people. In this fact sheet we wish to include information on different institutions providing health care to the survivors of the disaster.

I will much appreciate your sending the following information .

1. When was Bhopal Memorial Hospital Trust (BMHT) established, what is its relation to the Bhopal Hospital Trust, who are the current members of the Board of the BMHT and what is the status of the BHT ?
2. What are the current facilities available to the patients at the Bhopal Memorial Hospital & Research Centre, how many beds and staff does it currently have, how many patients have been registered in this centre and what is the current number of patients per day at this centre?
3. How many community clinics does the BMHT run, how many beds and staff do they have, how many patients have been registered in these clinics and what is the current number of patients per day at these clinics?

I will much appreciate your prompt response.

Thanking you,

Yours sincerely,

Satinath Sarangi
Managing Trustee

INFORMATION PROMISED BUT NEVER GIVEN.
SS.

The Bhopal Memorial Hospital Trust

In October 1991 the Supreme Court of India directed Union Carbide Corporation, USA, principal accused of the world's worst industrial disaster, to finance a 500-bed hospital for the longterm medical care of survivors. In response, Union Carbide set up the Bhopal Hospital Trust (BHT) in February 1992 in England with its former attorney as the sole trustee and with just US $1000 as its contribution. The rest of the BHT funds came from the sale of Union Carbide's Indian shares that were confiscated by the Bhopal District Court in response to the company absconding the criminal case.

On 11th August 1998, the BHT was Indianised to form the Bhopal Memorial Hospital Trust {BMHT}. The Supreme Court judge who overrode the order of the Bhopal court and allowed the sale of confiscated shares in the face of legal opposition of the survivors, now retired, is the current Chairperson of the BMHT. Of the three Bhopal-based trustees of the BMHT, two have left.

The BHT and now the BMHT's plans for health care of the survivors of the disaster include running a 260-bed hospital (situated five kms away from the severely affected communities without any public transport) and 10 community clinics, in different parts of the gas affected area. While five community clinics are currently running, the inauguration of the hospital awaits the visit of a Head of State.

The BHT/BMHT's plans for health care have been severely criticised by national and international groups including the International Medical Commission on Bhopal. Inadequacies pointed out by health care professionals include absence of a community health care perspective; undue attention and expenditure on facilities not related to gas exposure such as cardio-thorasic surgery, and alarming use of harmful and unnecessary drugs in the community clinics.

Given its principal role in the medical disaster in Bhopal that followed in the wake of the gas leak it is indeed ironic that Union Carbide Corporation, USA today projects a caring image through its hospital project in Bhopal, in its publicity campaigns and more prominently on its website, www.bhopal.com.

Meanwhile official complaints against unauthorised withdrawal and expenditure of Rs. 5 crores by the sole trustee of the BHT lie pending in the Supreme Court. A case of violation of the Child Labour Act by the BHT lies pending with the Deputy Labour Commissioner for employing children as young as 10 years old in the construction of the hospital building.

Department of Bhopal Gas Tragedy Relief and Rehabilitation

The Department of Bhopal Gas Tragedy Relief and Rehabilitation was set up by the Madhya Pradesh Government on August 29, 1985 in view of the longterm medical, social, economic and environmental relief and rehabilitation needs of the survivors of the disaster. In the last 16 years the Department has spent well over Rs. 400 crores. Of this about Rs. 200 crores have been spent on medical relief, Rs. 60 crores on economic rehabilitation, Rs. 70 crores on social rehabilitation and Rs. 70 crores on environmental rehabilitation.

Responsibility for the funds for the rehabilitation of survivors is meant to be shared between this Department and the Department of Chemicals and Petrochemicals, Government of India, in a ratio of 1:3. Under the First Action Plan (1990 to 1995) the Department received a sum of Rs. 193.50 crores from the Union Government. While work under the First Action Plan remains to be completed, an application for a grant of Rs. 316.54 crores for a Second Action Plan has been pending before the Union Government since 1996. The Union Government refused to grant this and, alleging misuse of funds, in June 2000 announced the institution of a high level investigation into expenditures made by this Department. Uncertainty regarding availability of funds has remained a constant problem. In 1998-99, serious official consideration was given to winding up the Department all together.

The Department has frequently been in the news with condemnatory reports of corruption and inefficiency. The March 1999 report of the Comptroller and Auditor General is a damning indictment of the Department's administration of funds. This official audit highlights over payments, cost inflation and unauthorised expenditure as some of the instances of maladministration involving over Rs. 20 crores. Currently, due to lack of funds, provision of health care is almost the only activity of this department. For administrative purposes the whole of the gas affected area has been made into a separate medical district under the control of a Chief Medical Officer.

A 60-member State Advisory Committee was formed in 1987 to "prevent over-governmentalisation (sic) of the Bhopal Relief and Rehabilitation Programme by involving non-offiicals in the decision making process". In practice, the Committee (which has the Chief Minister as its Chairman) has two meeting's per year (in a good year) and very few, if any, of its decisions are implemented. A writ petition against the Department alleging violation of the right to life of the survivors filed by the Bhopal Gas Peedit Mahila Udyog Sangathan and the Bhopal Group for Information and Action is currently being heard by the Supreme Court of India.

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The Sambhavna Trust

The Sambhavna Trust was set up as a registered charitable trust in June 1995 with the sole objective of improving the health condition of the survivors of the Bhopal disaster. Through the Bhopal Peoples' Health and Documentation Clinic situated close to the affected communities, the Trust provides medical care, health education, and research and monitoring facilities to the survivors free of charge. The clinic opened on September 1, 1996 and has registered nearly 10,000 survivors to date, mostly from the severely affected communities.

Objectives of the Trust

- To provide appropriate medical care through modern medicine as well as Ayurveda, Yoga and other alternative therapies.
- To generate information on the health consequences of gas exposure and to educate survivors on preventive and curative measures.
- To carry out medical research that addresses current and projected medical needs.
- To function as a repository of medical and other information on the Bhopal gas disaster and make such information accessible to all.
- To relate the medical and other activities of the government and other agencies with reference to survivors.
- To support and conduct seminars, conferences, research, training programmes and other educational and academic events towards the welfare of survivors.

Our Trustees

The seven member Board of Trustees that administers Sambhavna consists of doctors, scientists, writers and social workers who have been involved with various aspects of the disaster ever since its occurrence. Two of the Board members are from Bhopal. Several members of the Board have received national and international recognition for their scientific and social contributions. Except the Managing Trustee who is paid a monthly honorarium of Rs. 4500/-, all trustees work on a voluntary basis and meet three to four times a year. Decisions are made through a consensus among the members of the Board.

International support

We have a five member International Advisory Group made up of professionals from Italy, the UK and the USA who have provided longterm support to the cause of the Bhopal survivors. The volunteer-based Bhopal Medical Appeal-UK generates awareness and support for the clinic through publicity campaigns. A 14-member panel of medical advisors from Canada, South Africa, Sweden, the UK and the USA provides valuable technical support as needed.

Sambhavna Clinic staff

Among the 20 staff members at Sambhavna: nine are survivors of the disaster, 17 are employed fulltime, eight are women, nine are under 30 and four are over 40. Four are qualified in medicine, two of whom are specialists. Five are post graduates, four are graduates and seven are educated to school level. We are paid favourably in comparison to other local non-government institutions and a ratio of 1:4 is adhered to in determining minimum and maximum salaries. Most if not all, decisions regarding the day-to-day and longterm work of the clinic are taken by consensus at the weekly meeting of the fulltime staff members. The job of coordinator is chosen (by drawing lots) every two months from among the staff members.

Funds and fundraising

The Sambhavna Trust collects its funds mainly through small donations from a large number of individuals.

Appeals for funds are publicised through newspapers in India and abroad. The Pesticide Trust UK (Now renamed Pesticide Action Network, PAN-UK) collects donations on the Sambhavna Trust's behalf and transfers them to the account of the Sambhavna Trust after obtaining the required permission from the Ministry of Home Affairs. The trust has so far received Rs. 3.6 lacs approx. through individual donations in India and a total of Rs. 40.66 Lacs approx. have been transferred from PAN-UK. In the last four years well over 6000 individuals have sent donations towards the running of the Bhopal Peoples' Health and Documentation Clinic.

Expenditures

The clinic runs on a very tight budget requiring judicious attention to all expenditures. The Trust has incurred a total expenditure of Rs. 49.30 lacs approx an annual average of Rs.12.36 lacs or Rs. 1.03 lacs in an average month. The bulk of expenditure has been on payment of salaries, purchase of medicines, equipment and the clinic building. Audited accounts of income and expenditures of the Trust are publicly available and displayed every year.

Activities of Sambhavna

What we do

We believe that medical care should be based on the principle of "First do no harm" and that therapy should not compound the injuries sustained as a result of exposure. Development of integrated systems of therapy based on the specific symptom complexes presented by survivors (as opposed to attention to individual symptoms) is ongoing. We have also developed our own system of registration of persons under care to confirm and record history of exposure as well as monitoring the health status of individuals. We are active in five main areas:

* Medical care
* Community health work
* Research and monitoring
* Documentation
* Seminars and training

Medical care

At Sambhavna we offer both Allopathic and Ayurvedic systems of medicine. We also give Yoga instruction. We have a fully equipped pathology laboratory and we prepare and distribute many of our own medicines.

Allopathic care

Allopathic care at the clinic distinguishes itself in its attention to development of treatment protocols for specific symptom complexes, weeding out unnecessary and harmful drugs and including Ayurveda and Yoga in the overall treatment regime. We have general medicine, gynaecological care and mental health care units. Facilities such as Pap's smear tests are routinely available in Bhopal only at Sambhavna's gynaecological clinic.

Ayurvedic care

Ayurvedic health care at our clinic includes the use of medicines described in the standard Ayurvedic texts rather than factory-manufactured drugs; predominant use of herbal drugs over mineral preparations and use of modern investigative facilities and tools for objective assessment. We have two units of Ayurvedic care: general medicine and Panchakarma Chikitsa.

Yoga

We use Yoga at Sambhavna to treat several chronic diseases suffered by the gas exposed including those of the respiratory, musculo-skeletal, neurological and endocrine systems. More than half the people doing Yoga for exposure-induced respiratory disorders at the clinic have been able to discontinue medication they have been dependent on for over 10 years.

Pathology laboratory

Routine, microscopic and bio-chemical tests for blood, urine, sputum, and vaginal and cervical smears are carried out in house. In addition, samples are collected and sent to the medical college or private laboratories for more specific tests.

Preparation and distribution of medicines

We make 64 Ayurvedic medicines using locally collected or purchased herbs. Single herb and herbal mixture powders, decoctions, oils, tablets and poultices are all prepared on the premises. In dispensing medicines the utmost care is taken to ensure that people are well informed about their prescriptions.

• Community health work

Our approach to community health consists of empowering the community and its individuals to take control of their own health. Our four community health workers are the only ones of their kind in all Bhopal.

Health and health care surveys

We have carried out door-to-door surveys of five communities with a total population of about 10,000 in order to generate a database on the demography, health and health care status as well as the social, economic and environmental condition of the residents. While outlining priorities these surveys have also been a means of making contacts in the community and building local health committees. Our health workers also identify people in need of special medical attention and ensure that this is made available either at Sambhavna or elsewhere.

TB care and health education

Despite official knowledge regarding the unusually high prevalence of TB in the survivor population (over three times the national average according to an ICMR study) there are no official initiatives in this direction. Our work in TB care consists mainly of education, identifying those with symptoms of TB, having their tests done and, where required, ensuring medicine availability. We also follow up to ensure compliance with dosage. Much of this work is shared by "patient leaders", former TB patients who provide much effective inspiration in the community.

Monitoring and house visits

We evaluate the quality of our care and patient satisfaction in order to find out the efficacy of treatment and to ensure compliance of treatment regimes by visiting a random sample who have dropped out of care at Sambhavna. Our health workers find out the reasons for dropping out, whether the person found relief of their symptoms and whether he/she is receiving treatment elsewhere. People thought to be in need of continued care are encouraged to revisit the clinic.

• Research and monitoring

The government stopped monitoring exposure related mortality in 1992. We have picked up this activity by employing a technique called Verbal Autopsy - a scientific method of proven validity used for establishing the cause of death of individuals in a community. Other research projects include an assessment of drug distribution in gas affected Bhopal and on the use of Yoga therapy in the treatment of chronic respiratory disorders related to the disaster.

• Documentation

Much of the information on the disaster and its aftermath is lost, unavailable or classified. Of the information that is actually available, a large part remains within a tight-knit circle of bureaucrats, scientists, medical researchers and academics. Government efforts to collect and distribute this information are absent and non-governmental initiatives towards documentation of the continuing disaster are rare. We collect, collate, and distribute medical and other information related to the disaster. Quite possibly this is the only public repository of its kind.

• Seminars and training

We have organised several seminars involving local, national and international medical professionals, scientists, environmentalists and survivors' organisations. We have also participated in various local and national seminars. Our research paper, "Effects of yoga practices for respiratory disorders related to the Union Carbide gas disaster in 1984" was presented at the XVI World congress of Asthma at Buenos Aires, October 17-20, 1999.

Our Vision for the Future

The medical situation in Bhopal is likely to remain an issue of serious concern for at least the next 30 to 50 years. An estimated 35,000 children exposed in utero or conceived by exposed parents in the three years following the disaster are likely to require special medical attention all their life. Late manifesting cancers also call for longterm health surveillance and treatment facilities. Chromosomal aberrations, observed in an unusually large number of those exposed, warrant that health surveillance of the exposed population is carried out at least for two generations.

Over four years of work have produced encouraging results and given us some degree of confidence regarding our role and relevance. They have also instilled the courage to dream and plan for the future of the clinic. For the next five years these consist mainly of upgrading and expanding facilities (medical care, diagnostic equipment, the pathology laboratory and medicine manufacture), creating new services and becoming further involved with the affected communities. The increasing patient-load on the doctors calls for the appointment of additional physicians/ specialists and corresponding increase in space and para-medical personnel.

The results of our community health work have been most encouraging, particularly in terms of involving more and more people. There is a strong possibility of building on this to create a community-based network for monitoring activities. We have collected information from different parts of the world on the development of health surveillance techniques that are implemented by lay members of the community. It is possible to train survivors in Bhopal in such techniques as 'lay epidemiology' and 'verbal autopsy' and in using simple equipment for water/air quality monitoring so that a community can be self sufficient in the longterm monitoring of its health and that of its future generations.

We believe

- That the disaster in Bhopal is not an isolated event. Workers and communities are routinely poisoned all over the world.
- The reduction and eventual elimination of hazardous chemicals from the planet and our daily lives is the only solution to the growing number of slow and silent Bhopals in our midst.
- Until that happens, the safety of our health and lives depends on watchful monitoring, strict enforcement of regulations and exemplary punishment to offending agencies. In this respect, ensuring justice in Bhopal can be seen as a public health initiative with potential for significant and widespread change.
- The limits of modern medicine in taking care of modern industrial diseases are becoming increasingly apparent. Evolution of an appropriate system of health care, monitoring and research for survivors of Bhopal is of consequence to all people worldwide.
- It is possible to combine traditional and western systems of health care to provide sustained relief to the chronically ill survivors of Bhopal.
- It is possible for an individual to be an active participant in the process of healing and the community to be involved in all aspects of public health.
- It is possible to evolve systems of health surveillance and environmental monitoring through active participation of the community of survivors. Our work in documenting longterm consequences of exposure is part of the survivors' ongoing struggle of memory against forgetting.
- It is possible to depend upon the compassion of ordinary individuals and generate enough funds to run our clinic without the help of corporate charities, big business, large grants from foundations or government assistance.
- It is possible to generate opportunities for hope through creative and collective intervention in a situation of despair.

Ten Ways to Help Sambhavna

1 **You can support the day-to-day running of the clinic.**

Currently it costs us a little more than Rs.1,12,500/- per month to run the clinic. This includes the cost of medicines, salaries, medical investigations and publications.

2 **You can help us purchase land and build a bigger and better clinic.**

We plan to:

- plant a medicinal herb garden
- establish a demonstration and production unit of herbal medicines
- build rooms for different kinds of therapy as well as for training and education
- install a solar power system for our energy requirements

3 **You could contribute to our core fund.**

At the current rate of expenditure we would need about Rs. 3 crores to generate enough interest to run the Sambhavna Clinic indefinitely.

4 **You can help by giving new/used equipment.**

At this point, we are badly in need of:

- a spirometer (for testing lung function) Rs. 18 lakhs
- an ECG and an EEG machine Rs. 90 lakhs+
- a network of three computers for registration, for clinical data entry by doctors and other staff members and recording of prescriptions

If you have steel cupboards, weighing machines or furniture that you don't use, we could.

5 **You can help with the cost of medicines .**

Medicines used by the clinic cost Rs. 13,500/- per month. Ayurvedic medicines cost substantially less than allopathic ones.

6 **You can contribute to staff salaries.**

Collectively we pay ourselves Rs. 67,500/- per month. We have 17 full time staff including physicians, therapists, registration staff and a further three who work part time. We also deserve a raise!

7 **You may be interested in donating to specific activities.**

Panchakarma therapy (traditional therapy based on detoxification of the body) is one of our specific projects in need of support. This fivefold therapy provides sustained relief of many of the chronic conditions suffered by the gas affected. On the basis of this success we are keen to expand the present facilities and employ one full time Panchakarma doctor as well as purchasing additional equipment.

8. **Help us to further educate ourselves.**

Our health workers, physicians, yoga therapists and others need training and re-training in specific skills both in the field and in clinical settings. For a two week training course the average cost per person is Rs. 4500/-. Participation in a seminar or workshop costs about Rs. 3600/- per person.

9. **You can help us to host our seminars, workshops and training camps.**

Sambhavna has also held a number of seminars, workshops and training camps since it began you can provide support through volunteer work, donations and ofcourse, active participation.

10. **Help us to fund our volunteers.**

In addition to local volunteers from among the survivors and other supporters we have had a few volunteers coming from different parts of India, the UK and the USA.

If you would like to help us in any of the above ways, then pelse send a cheque or postal order to the clinic address mentioning your preference for a particular area of work.

Publish by the Sambhavna Trust

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Mumbai, July 8, 2003.

BRIEF REPORT OF BHOPAL MEETING..July 4,5, 2003.

FOLLOW UP OF DESH BACHAO DESH BANAO ABHIYAN
ACTIONS PLANNED THROUGH OUT INDIA FOR COMING DAYS

The meeting at Gandhi Bhavan, Shyamala Hills, Bhopal was attended by about 25 representatives from 12 states and was chaired by National Coordinator Medha Patkar.

Brief report about the discussions and decisions taken in the meeting is as follows.

1. Condolences were offered at the beginning of the meeting by observing silence for the sad and untimely sudden demise of Shobha Wagh (Narmada B. Andolan), Om Srivastava (Asha, Udaipur) and Com. Balkrishna Gupta (CPI, Bhopal).
2. Members reported about the follow up that has taken place in last three months in various states after culmination of Desh Bachao Desh Banao Abhiyan .It was decided that it should be continued in the following way by National Convenor from that state and the State Coordinator of the state in the coming months.
   a. Get affiliation forms filled from all the concerned organizations.
   b. List of allies- individuals and organizations- is prepared by Prayas, Pune office, based on the forms filled during the DBDB abhiyan. Each convener will receive those soon, which may be completed, added to and used.
   c. Get in touch with all those individuals and organisations who filled in forms during DBDB abhiyan and motivate them to get involved in day-to-day activities of NAPM.
   d. Start contacting all above for proper implementation of follow up action programmes mentioned below. Organise meetings, camps regularly.
   e. Those who haven't, may plan workshops for young activists, convention of allied organizations and individuals and strategy meetings/ conferences on sectoral issues highlighted/ supported during DBDB abhiyan.
3. **NATIONAL STRUGGLES**
   a. **NARMADA struggle** is on. About 12,000 families are expected to face unjust submergence this monsoon due to increase of Sardar Sarovar dam height to 100+3 mtrs. without proper rehabilitation of all those who have been affected at even 90 mtrs. itself! While the Maharashtra cabinet has approved the main demand and initiated the joint planninmg, everything is not in order. On the other hand M.P. govt. is too callaus and denying land based rehabilitation. Several activists, political leaders and other eminent persons are visiting valley in teams between July and September. WARN & WATCH CENTRES have been started in the valley, warning the Maharashtra and Madhya Pradesh state governments that if all issues related to rehabilitation are not resolved and a concrete commitment not to raise dam height further till rehabilitation of affected families is completed; is not given then, SATYAGRAHA WILL BEGIN ANY TIME IN AUGUST.
   b. **PLACHIMADA ANTI COCA COLA STRUGGLE** is in full swing. Just after the culmination of DBDB, people in Plachimada have observed first anniversary of their struggle. Support came from left parties and many organizations of dalits, adivasis and environmentalists. The local gram panchayat has cancelled the license of the plant. Coca Cola Company went in to Trivendrum High Court. Court asked the state to intervene. The

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state govt. cancelled the decision of gram panchayat! People of Plachimada supported by NAPM have decided to organize YATRA from Plachimada and other parts of Kerala to culminate at Trivendrum, on August 9 in the form of MASSIVE DEMONSTRATIONS in front of the Secratariate.

c. **STRUGGLE AGAINST SELLING OF SHIVANATH RIVER** in Chhattisgarh has made Chief Minister to decide in favour of canceling the sale of Sivanath to privatize the river water. As he informed Medha Patkar when they met during journey, the cabinet has resolved the same, however the Finance and Legal Departments of State Govt. are examining the implications. There is no formal announcement on the cancellation of the contract.Local people are prepared to intensify the struggle in case State Govt. betrays! Binayak Sen, on behalf of NAPM will be in touch with local movements/ groups.

d. **STRUGGLE AGAINST PRIVATISING JAMBUDWEEP ISLAND** and OFFERING it to SAHARA, India for TOURISM, has yielded some positive results.100Ha. Of the land has been assured to the fisher people for their traditional fishing activities. NFF is determined to carry out a SHOW DOWN from Oct. 1st, in case the assurance is not fulfilled. It was decided that NAPM would not only participate at Jambudweep but also mobilize support actions all over country.

e. **GANGA PEOPLES'** process has been well initiated in Bihar by organising 3-day workshop in Patana in the last week of May. 4 state level meetings will be held in coming days in W.B, N.E., U.P. and Uttaranchal. Impact assessment study will be conducted regarding various completed projects in Ganga Bassin including Farrakka, Kosi and Suvarna. Chetana yatra BRAHMAPUTRA TO YAMUNA will be organized against INTER LINKING of RIVERS project and for LINKING RIVERINE PEOPLES.

f. **ISSUE OF LAND RIGHTS TO DALITS, ADIVASIS AND LANDLESS** was decided to be taken up as an additional National Struggle issue. Anti Globalisation Committee in Maharashtra has organized a daylong dharana in Mumbai on July 17. A.P.V.V.U. in Andhra Pradesh is planning to organize 21 days TRUCK YATRA in Oct. this year starting from 3 corners of the state and culminating in Hyderabad. It was decided to raise the demand of PUBLISH WHITE PAPER ABOUT AVAILABILITY OF LAND with each State Govt. We expect and request all our allies to raise the question through appropriate programs ' Tell us where is land. Tell us why dalits, adivasis and landless cant be given land.' Each state unit of NAPM must organize at least a sample survey about availability of land. APVVU along with other organizations will also organize SATYAGRAHA at various places en route the yatra.

4. After thorough discussion following Action Programmes have been finalized. Many of these are organized by NAPM or the constituent organizations whereas some are by others and supported by NAPM.

ACTION PROGRAMMES NAPM APPEALS ALL TO PARTICIPATE

July 9/10 : Inauguration of WARN & WATCH Centers in Narmada Valley.
11-13 : NAPM Maharashtra State camp for activists at Nashik.
12-13 : Workers Camp about Destruction of Traditional Industries, Madurai.
15 : PROTEST ALL OVER INDIA AGAINST SENDIG TROOPS TO IRAQ
16 : Right to Food Conference in Mumbai by Right to Food campaign.
17 : DHARANA IN FRONT OF ASSEMBLY FOR LAND RIGHTS TO DALITS AND ADIVASIS, in Mumbai and allover the state by Anti Globalisation. Committee.
19, 20 : Public Hearing at Delhi Against Forcible Eviction

20 : Anti Imperialism Convention at Kolkata, W.B.

25-27 : Work Shop on " Land, forest and Water" at Indore organized by Jana Sahayog Trust and Jan Sangharsha Morcha, M.P.

August 6 : "Anti Nuclear Power Station" Demonstrations at Kudan Kulam, T.N.

7 : Workers' Demonstrations against Job loss at Madurai, T.N.

8-9 : Seminar to oppose WTO Cancun Ministerial Meeting, at Delhi, by Anti WTO Peoples` Campaign

8 : Conference On Dalit right to Water atRaigad,Maharashtra.

9 : (A) Yatra From Plachimada and elsewhere to culminate at Trivendrum in the form of massive demonstrations against Coca Cola Plant.

(B) Workers Convention at Asansol, W.B.

9 to 15 : **Demonstrations, Rallies, blackening of MNC boards and other programmes ALL OVER COUNTRY against Globaloisation, Communalism.**

9 onwards : SATYAGRAHA BEGINS IN NARMADA VALLEY.

26 : ANTI WTO RALLY in Delhi by Anti WTO Campaign (involving left to the center parties and peolpes' movements).

27 : Demonstrations in front of Commerce Ministry in Delhi for the demand of INDIA QUIT WTO by NAPM (Others in Anti WTO campaign are to be invited to join)

30 : Public Meeting against Globalisation in Martandam, T.N.

Sept. 1 : Rally on Right to Work at Akabarpur, Dt. Ambedkarnagar,U.P.

SEPT. 4 : NATIONWIDE SATYAGRAHA FOR "INDIA QUIT WTO"

Oct.1 : NFF Showdown in JAMBUDWEEP, W.B. & Support action all over India

Oct. : TWENTY ONE days TRUCK YATRA & SATYAGRAHA starting from 3 corners of A.P. and culminating at Hyderabad on the issue of LAND & UNTOUCHABILITY

NOV. 4 : "KRISHI BACHAO, ROZGAR DILAO" Kisan Naujawan Rally in Patana by Samajwadi Jana Parishad

Jan. 2004 : Fisher people of the world reaching Mumbai during WSF to press for their 42 demands.

5. A resolution on sending troops to Iraq was passed unanimously and sent to Prime Minister. Signatures to be collected all over India and sent to P.M. on July 15.

6. POLITICAL INTERVENTION

A. Some members expressed that NAPM should take stand of "Non Congress Non BJP led NDA" for the forthcoming elections instead of appealing to defeat BJP, while others felt that Congress and other parties in the secular force should not be considered on par with BJP and its allies.

B. NAPM will extend support to its constituents Samajwadi Jana Parishad and Kranti Dal candidates in M.P. assembly elections.NAPM expects them to bring out election manifestos at the earliest.

C. NAPM welcomes the initiative of Samajwadi Jana Parishad to start the process for evolving ALTERNATIVE POLITICAL STREAM to face worsening socio- economic and political situation in the country. NAPM will participate in this process after SJP's letter is received. NAPM will continue exploring avenues for electoral and non electoral political intervention.

D. NAPM will plan 'lokmanch' in Delhi inviting leaders of political parties for a public dialogue and debate with the senior activists in the country. Yogendra Yadaw, Vijay Pratap, Rajendra Ravi and Surendra Mohanji are requested to plan and make it a success. NAPM

will try and use the SC judgment on declaration of assets by the candidates

7. **WORLD SOCIAL FORUM**

Medha Patkar and Vijay Pratap reported about International Council meeting of WSF that took place recently in Miami,U.S.A. Detailed discussion took place about the internal code and mode oif operation of WSF, future of WSF and the WSF- India. They reported on the various working groups ('commissions' in Spanish) on various aspects of WSF process and principles and that Medha Patkar and Chico Whitacker are to coordinate group on 'Strategy'. Vijay Pratap is also in the coordinating team of the same. The decision taken was " NAPM maintains its Lucknow decision as far as joining the IOC. Though we participate at the WSF 2004 in Mumbai, we maintain a space for CRITICAL COLLABORATION. The constituents of NAPM should make use of this space at ALL LEVELS."

8. **PUBLICATIONS AND BULLETINES**

A. Hindi booklets of AYODHYA DECLARATION (A.D.) are available with NAPM conveners.

B. A.D. in English with few modifications suggested in this meeting will be available by July end with NAPM conveners.

C. Translation of A.D. in regional languages is on and booklets will be available in the respective states by end July.

D. Publication of NAPM Bulletins in Marathi and Bengali is regular. Publication of English bulletin from Bangalore is not yet regular. It was decided that the following members be requested to take up the responsibility till the alternate arrangement is made. Sanjay Sangvai (Convenor), Arundhati(U.P.) & Gabriele D.(T.N.) to work as Executive Editors. INITIATIVE GROUP, Mumbai (an independent group of youngsters related with move ments) is requested to help the printing and dispatch from Mumbai.

9. **FUND RAISING AND FINANCE**

A. Interim accounts of DBDB Abhiyan was presented by Sanjay M.G. Utilisation Certificate of the same will be obtained and published in NAPM related publications.

B. Each National Convener will contribute at least Rs. 500/- per month towards the expendi ture of the National Office. Thomas Kocherry has already contributed at the time of the last National Convention at Palghar.

C. It was a unanimous opinion of all that NAPM should be very carefully following all legal requirements in relation to accounting. Registration of a charitable trust is also thought necessary. The need of organizational legal requirement of Auditing NAPM accounts as also the registration requirement will be enquired and Sanjay M.G. will present a note on the same in the next meeting.

D. It was reported that Arundhati Roy has included NAPM in the list of organosations to receive a share of her prize money and the same is received by NAPM.

10. **ABOUT JAIPUR INITIATIVE:** A meeting bringing together various like-minded groups and individuals, including NAPM was held in the run-up to the Gujarat elections in July` 2002. A number of NAPM conveners and constituents attended that meeting. Follow-up meetings were held in Wardha, Delhi and Bhopal, which were attended by some NAPM conveners and perma nent invitees. It was decided in those meetings that the process leading to a wider alliance among national networks, which is long term, should be taken forward.

The matter was deliberated upon and it was felt that a new front and a new campaign involving a majority of the same people involved in the recently

concluded "Desh Bachao, Desh Banao" campaign does not appear feasible at this point. It was decided to communicate this to the organisers of the Ranchi meeting through Kishen Patanayakji and Amarnathbhai who were to be present at Ranchi.

11. **VARIOUS CELLS** to facilitate an in-depth grasp and response to various emerging challenges were set up in the last meeting held at Lucknow following DBDB abhiyan. Key concerns areas of our national movement were listed down. The conveners also decided to form cells/committees that would guide NAPM formulate its strategy and programmes related to those areas. Hence, the following cells with the convener of each mentioned, are being formed:

1) **Agriculture**- P. Chennaiah, A.P.; 2) **Labour**- Datta Iswalkar, Mumbai.; 3) **Land, Water, Forests**- Ananad/ Michael, Gujrat; 4) **Energy**- Sanjay M.G., Mumbai.; 5) **Food Security**- Ulka Mahajan, Maharashtra.; 6) **Right to Work**- Sukhendu Bhattacharjee, W.B.; 7) **Inter-linking of Rivers**- Sudhir Vombatkere; 8) **Media & Communications** - Sanjay Sangvai, Maharashtra.; 9) **Political intervention**- Yogendra Yadav, Delhi.; 10) **Cultural Cell**- Rashtra Sewa Dal, Mahrashtra.
Those who want to join these cells are most welcome to send their names.

12. **LOKTANTRIK SAMAJWADI PARTY** has expressed its desire to get affiliated to NAPM. It was decided to hold discussions with its leadership to know their views on various issues and aspects in DBDB abhiyan and Ayodhya Declaration, before taking any decision.

13. To counter distribution of **TRISHUL** by VHP IN U.P., distribution of **GEETA** booklets was undertaken by some organizations under the banner of NAPM.Some of our allies have expressed dissatisfaction over the same. The meeting discussed the matter at length and suggested:
    - **A.** Some other secular booklets of Mahatma Gandhi or others could have been distributed but surely not Geeta, which justifies several evils includingWAR!
    - **B.** All the State, Regional and NATIONAL conveners should make it a regular practice to make formal or at least informal consultations with the other constituents before announcing any new programme in the name of NAPM.

14. **WORK SHOP ON URBAN POOR:** It was decided that Delhi NAPM would organize a work shop in the third week of September, on the issue of Urban Poor, involving other organizations working on same issue in Delhi. Representatives from Mumbai, Nashik, Bangalore, Hyderabad, Chennai, Madurai, Kolkata etc. will be invited. Responsibility is given to Rajendra Ravi.

15. **BILL ON THE UNORGANISED SECTOR WORKERS** is likely to get introduced in the coming session of parliament. The proposed bill is not up to the mark. NAPM with other likeminded forces and forums will initiate the process to pressurize Government on this issue. Responsibility is with P. Chennaiah.

16. **WTO VIRODHI BHARTIYA JAN ABHIYAN (INDIAN PEOPLES' CAMPAIGN AGAINST WTO)** is to come out with 5 booklets on different important issues regarding WTO on the eve of Cancun Ministerial Meeting; scheduled on Sept.10 to 15, 2003.The abhiyan has planned a 2 day conference in Delhi on August 8 & 9 and public rally in Delhi on Aug. 26.It was decided that the National Coordinator of NAPM will write to the committee requesting them to arrange the rally in more effective, attractive and participative way.

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In the rally on Aug. 26 NAPM- Delhi will participate in a good number. Representatives from all states including far off southern states will also participate.
NAPM will organize additional action on Aug. 27 in Delhi in front of Commerce Ministry demanding INDIA QUIT WTO! Others in Anti WTO abhiyan will be invited to join.

17. **SOUTH ASIAN ALLIANCE FOR POVERTY ERADICATION (SAAPE)** has been launched recently in its first general assembly in Colombo, Srilanka held on June 12 to 16. A conference on POVERTY, POLITICS AND PEOPLES was also organized. Sanjay M.G. at tended the same on behalf of NAPM. It is an alliance at South Asia level similar to NAPM. The purpose of forming SAAPE is to understand the similarity of WTO/IMF/WB strategies in the South Asian countries and explore the possibility to counter it through better solidarity amongst the South Asian Peoples' Movements and with the help of European Union wherever possible.

18. A **conference of DAM AFFECTED** people is going to be organized in Thailand in December this year. The activists concerned and interested in attending may please write to the National Office of NAPM.NAPM coveners should send names of experienced, articulate dam affected people, activists, close supporters to enable a final selection of ten.

THE MEETING ENDED WITH OFFERING SINCERE THANKS TO Mr. Bhargavaji of Gandhi Bhavan for providing excellent facilities to all. National Covenor from Bhopal Mr. A.K.BHANAGE took lot of efforts to arrange the logistics during the meeting.

PLEASE ACKNOWLEDGE THE RECEIPT OF THIS REPORT.

MUMBAI P. Chennaiah Sanjay Mangala Gopal Medha Patkar
July 15, 2003. NATIONAL CO COORDINATORS NATIONAL COORDINATOR

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http://webdrive.service.emory.edu/users/vdhara/www.BhopalPublications/

Dear Friends,

I have put a few scientific publications relevant to Bhopal on the Emory University webdrive. You can see these by clicking on the link above. If you wish to acquire a copy, you can open the article by double-clicking on it and then save it on your own computer. I will add more articles as I acquire electronic copies of them. If you have articles which you would like me to add to this site, please send them to me in electronic format. Because of space restrictions, I would like to put in those which are peer-reviewed and published in the literature.

HRM - Please save the articles & make a list of articles - we can later decide which ones we need
JS
8/5/03

best wishes,

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TH
8/5/03

Main identity

From: "Ruchita Khurana" <ruchita@toxicslink.org>
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Sent: Monday, December 15, 2003 4:51 PM
Subject: IMAGING BHOPAL TODAY : Photographs by Raghu Rai, on December 19, 2003

Toxics Link
Environment and Health Public Lecture Series

On 3rd December, 1984, a lethal gas leaked from the Union Carbide/DOW factory in Bhopal (India), killing 8000 and injuring half a million. Since then, several thousands have died a slow death and lakhs are chronically ill. Today, many from the second generation and an increasing number of victims from the third generation, are living a life which has become an unending disaster. The gas inhaled that day, 19 years later, still continues to corrode their vital organs, making it impossible for them to lead a normal life. Union Carbide/ DOW' criminality and government apathy to the issue, compounds this tragedy – which is the largest of its kind.

The responsibility of chemicals and their health and environmental impact, lies with the industries from which they emerge. This norm is not alien to corporate responsibility, according to which, the mishandling of toxic chemicals is labelled criminal neglect. Although global standards and global corporate responsibility is emerging on the agenda for the civil society in India, the most notorious culprits for the Bhopal disaster are yet to be convicted for culpable homicide.

This brutal and impudent violation of our country's ecological space was captured on camera by the world - renowned photographer, Raghu Rai, who revisited the site between December 2001 to May 2002.

We invite you to the screening of these photographs on the Bhopal Gas tragedy, a unique collection that narrates a shocking story of corporate irresponsibility:

IMAGING BHOPAL TODAY : Photographs by Raghu Rai

Followed by a panel discussion on Corporate responsibility.
Panelists:
Mr. Raghu Rai, eminent photo journalist and Padamshree award winner
Dr. Usha Ramanathan, Programme Director, International Environmental Law Research Centre, law researcher and teacher
Prof. Surupa Mukherjee, International Campaign for justice in Bhopal (ICJB)

Moderated by Mr. Ravi Agarwal, Director, Toxics Link.

Venue: Conference Room 1, India International Centre, Lodhi Road, New Delhi
Date: Friday, 19 December 2003, Time: 6:30 p.m.
(In collaboration with India International Center)

12/16/03

A historic ruling

A U.S. Court of Appeals sustains the plea of the survivors of the Bhopal gas disaster and orders Union Carbide Corporation to undertake the removal of contamination in and around the abandoned pesticide plant.

V. VENKATESAN

AFTER nearly 20 years of struggle for justice and due compensation, the survivors of the 1984 Bhopal gas tragedy, the world's worst environmental and industrial disaster, won a major legal victory against Union Carbide Corporation (UCC), the perpetrator of the disaster and the then owner of the pesticide plant in Bhopal, in the U.S. Court of Appeals for the Second Circuit, New York.

Setting a significant precedent in the history of environmental litigation, on March 17, the court approved "injunctive environmental remediation" against UCC to clean up the pollution it caused in Bhopal. The judgment was delivered by Circuit Judges Wilfred Feinberg, Amalya L. Kearse and Reena Raggi.

The term "injunctive environmental remediation" encompasses any work that has to be done to remove contamination or pollution from a given site in order to restore it to certain applicable environmental standards. In this case, for example, remediation might entail a complete decontamination of the soil, the filtration and removal of contaminants in the groundwater to safe drinking levels, the removal of all the waste matter on the site such as asbestos wall cladding through the "treatment" or processing of such waste and/or transporting it to a location outside India.

Haseena Bi, one of the survivors of the tragedy, and several organisations in Bhopal representing survivors were plaintiffs in a class action suit against UCC filed before the U.S. District Court for the Southern District of New York seeking damages and injunctive relief for the severe pollution of their land and drinking water. A class action suit enables individuals and organisations to make a complaint both individually and on behalf of all other classes of persons similarly situated. The plaintiffs alleged that thousands of residents in and around the abandoned pesticide plant

A.M. FARUQUI

In Bhopal, members of the 'Bhopal Gas Peedit Mahila Udhyog Sangathan' protest on January 10 against the lack of medical facilities for the victims, the distribution of polluted drinking water in the affected areas, and the non-rehabilitation of the widows of gas victims.

AFP

Demanding an end to the dumping of genetically engineered products in Asia, Greenpeace activists in the Philippines block a shipment of genetically engineered soya from the United States.

quirements needed for safe handling, storage, transport and use under existing international instruments, as well as domestic regulations and any agreement made between the exporter and the importer. On the compliance issue, MOP1 had a long debate on how to deal with countries that do not comply with their obligations under the Protocol. The European countries were especially keen to get a strong compliance regime so that member-countries would take their obligations seriously.

India took an ambiguous stand on compliance, not supportive of a strong compliance mechanism. This was probably because of the perception that it would one day be an exporter of genetically modified foods and products and therefore would not want to confront a rigorous compliance regime. India's weak stand on compliance is not a wise one. First, it would encourage bad practices at home. A not-always-literate farming community, traders who are not in tune with international practices, and the overall poor level of awareness about the implications of genetically modified crops for the environment and human health could create dangerous situations. Strong compliance is needed at home, to protect us from ourselves and from others.

Despite the progress made at MOP1, it remained a matter of concern that the conclusions failed to take on board a central concern of developing countries: that of the social and economic impacts of genetic modification technology. This is of crucial significance to India and other developing countries where the impact on small farmers and their livelihoods could be considerable. Although it is very important to monitor genetically modified crops for their impact on biodiversity and the environment as well as the health of humans and animals, it is equally important to watch out for the social and economical implications of this technology for farmers and consumers in developing countries. The social and economic costs of this technology could be highly significant in the agricultural situation of the South and this should be monitored as critically as health and the environment.

It is a pity that the discussions at the Biosafety Protocol did not take up the social and economic aspects even though some country delegations, especially the African Union, flagged the issue repeatedly. The African countries in fact were trying to keep the focus on this aspect alive at all levels of the discussions accompanying the meetings. Although India did mention socio-economic concerns in the official interventions, there was little follow-up or lobbying to create a strong pressure group that would put the issue on the main agenda. As a result, it was not included in the main conclusions of MOP1.

It is short-sighted to overlook the fact that genetic modification technology could turn out to be counter-productive in the agricultural economies of developing countries. If it were to displace small farmers, the impact would be detrimental. In fact, in genetic modification research there is considerable emphasis on producing through genetic engineering products that are at the moment produced only in developing countries. One may recall what happened to vanilla. Madagascar, once the largest producer of vanilla, earned sizable revenue for its farmers through its export. Determined to break this monopoly, U.S. laboratories succeeded in synthesising vanilla and the markets, flooded with the cheaper version, turned away from the natural vanilla produced by Madagascar. This resulted in big economic losses and hardship for farmers in Madagascar. Similarly, sugar-producing countries in Asia have suffered at the hands of another laboratory-based substitution. Cornstarch is used to make high fructose corn syrup, which has displaced sugar in large amounts from sectors like confectionery. In fact, most of the genetically modified corn that is being grown by the U.S. and Canada is used either in the production of high fructose corn syrup or as animal feed.

A strong line of genetic modification research in the West currently is attempting to produce the characteristics of coconut and palm oil in the more common canola (a form of mustard). Canola grows in countries in the temperate zone, whereas coconut and oil palm grow in the tropics. Many farmers in Asia earn a livelihood from the export of coconut and palm oil, both of which are sought-after in the U.S. and Europe for their special properties like high lauric acid content. When genetic modification technology creates canola plants that produce oils with high lauric acid content, it would mean the loss of markets for farmers growing coconuts and oil palm in countries like India and Malaysia.

Given the potential of this technology to damage the agricultural prospects of developing countries, its social and economic impact must be taken on board in the international agreements on biosafety, through the Biosafety Protocol.

In effect this would mean that countries should have the right to refuse the import of a genetically modified crop that could displace the produce of their own farmers without attracting penalties and sanctions of the kind that happened in the E.U.-U.S. case. The E.U., which has had a *de facto* moratorium on genetically modified crops, refused the exports of corn and soybean from the U.S. and was hauled by that country to the World Trade Organisation Dispute Settlement Court and threatened with huge fines for refusing U.S. imports, an act, the U.S. claimed, was trade distorting. Such a situation could easily arise for India and other developing countries if they were to refuse imports of genetically modified foods on socio-economic grounds, unless this was regulated through the internationally binding nature of the Biosafety Protocol. It is in India's interest to ensure that socio-economic concerns as provided for in Article 26 of the Protocol are brought on to the main agenda. ■

in Bhopal were exposed to toxins because of the contamination of soil and water. They accused UCC of causing pollution by utilising improper technology in the design of the Union Carbide India Limited (UCIL) facility in Bhopal and then recklessly dumping large quantities of toxic materials at the plant site. They claimed that pollutants from the plant continued to seep into the local environment causing serious health problems for nearby residents. "If nothing is done to resolve this problem in terms of the relief sought, UCC will have bequeathed another large-scale environmental catastrophe to Bhopal," the plaintiffs warned.

In March 2003, the District Court rejected the suit on the grounds that Haseena Bi's claims were time-barred, that organisations could not be representatives of individual plaintiffs, and that it would be impossible for a U.S. court to implement a decision that required a U.S. corporation to clean up contaminated land. The plaintiffs then filed an appeal before the Second Circuit Court of Appeal on the basis of internal documents of UCC and points of law.

RAJ PATIDAR/REUTERS

A torchlight procession in Bhopal in December 2002 on the anniversary of the gas disaster.

It is important to understand the Appeals Court's decision in terms of the overall nature of the claims made in the class action suit. Plaintiffs had claimed the following as relief: (1) Damages for personal injury caused by exposure to contaminants in drinking water and soil through the underground aquifer from the UCIL factory; (2) monetary damages for loss of value of property and private hand pumps; (3) claims for medical monitoring of an estimated 20,000 people living in the 10 municipal wards around the former Carbide plant where contamination has been found; (4) environmental clean-up and remediation of off-site contamination on private properties/residences/hand pumps of plaintiffs; and (5) environmental clean-up and remediation of the former UCIL factory itself.

The Appeals Court has reinstated virtually all the claims. The court maintained that the plaintiffs' personal injury claims must be allowed to go forward but stated that the statute of limitations for such claims must be limited to three years before the filing of the complaint in November 1999. Of course, the three-year limitation period eliminates the claims of Haseena Bi who had stated that the injuries and symptoms resulting from contamination approximately dated back to 1990. But the case is "class action" litigation and Haseena Bi's personal injury claims are not the only ones to be included. Other plaintiffs can advance their personal injury claims, subject to the three-year time period. The District Court had not addressed the issue.

The Appeals Court reversed the District Court's conclusion that monetary relief for property damage and loss of value of property and private hand pumps must be dismissed on the basis of the three-year limitation. The court affirmed the argument of the plaintiffs that because such claims are "continuous" and "ongoing" in nature the defence of a three-year limitation is not applicable. The Appeals Court also held that notice of personal injury damage did not amount to a constructive notice of property damage. This means that Haseena Bi and the approximately 20,000 residents of the 10 municipal wards in Bhopal that have been affected are free to prosecute such claims against UCC.

The Appeals Court declined to address the District Court's dismissal of the medical monitoring claims on technical grounds. Essentially, this means that the medical monitoring claims on behalf of the 20,000 or so plaintiffs continue to remain viable for individuals and the class.

The Appeals Court reversed the District Court's dismissal of claims for "injunctive relief" regarding property, that is, the clean-up of individual properties and hand pumps off-site.

The Appeals Court did, however, affirm the District Court's dismissal of the plaintiffs' claims that UCC should be made to pay for and undertake proper environmental clean-up and remediation of the former UCIL plant site. The court did so with an important caveat: it rejected the District Court's conclusion that such clean-up or equitable relief would be either "impossible" or would automatically "interfere" with India's interest in handling its own environmental problem. The term "equitable relief" is used to suggest that the court orders the defendant to do something, in terms of an activity, as opposed to merely paying damages for the harm it caused.

According to the plaintiffs' counsel, H. Rajan Sharma (see interview), the decision seems to suggest that such equitable relief for clean-up and remediation of the source of pollution, that is, the plant where thousands of tonnes of waste were improperly stored and disposed of, would be feasible and appropriate if either the Indian government or the Madhya Pradesh government were to make a submission indicating

'Some reason to be cautiously optimistic'

Interview with H. Rajan Sharma, lawyer of the survivors.

H. Rajan Sharma, an international lawyer and author currently based in New York, represents the survivors of the Bhopal gas disaster in courts in the United States. He obtained his Juris Doctor degree from the American University, Washington D.C. He has written extensively on international law and politics. His most recent article, 'Dispute Resolution Mechanisms in International Environmental & Investment Disputes' was published by the Permanent Court of Arbitration at The Hague and included in a collection of *The Peace Palace Papers* by the International Bureau of the Permanent Court of Arbitration. Sharma has been profiled by *The American Lawyer* magazine and has been nominated for inclusion in 'The Best Lawyers in America'. In an e-mail interview to **V. Venkatesan,** he answers questions on the Appeals Court's decision.

BY SPECIAL ARRANGEMENT

► *What makes the Appeals Court decision significant?*

Never before in judicial history has a court sitting in one country ordered a multinational corporation to go some 8,000 miles [12,800 km] to clean up and remediate an environmental mess in another country literally halfway around the world. It is unprecedented. And that the precedent should be set in a case about Union Carbide's conduct in Bhopal certainly seems more than appropriate.

► *What is the next stage in the progress of this case? Are you optimistic about the outcome?*

The case will go back before the District Court. It is, however, virtually impossible to predict the outcome or to speculate how the District Court will approach these issues. On the other hand, we believe that the legal effect of the Appeals Court rulings is very much in favour of the Bhopal victims and survivors. To that extent, I believe there is some reason to be cautiously optimistic.

► *Why is it important that Union Carbide Corporation (UCC) assumes responsibility for the clean-up of the site itself? What are the specific implications of this?*

It is extremely important that Union Carbide be made to pay for and undertake the clean-up of the UCIL [Union Carbide India Limited] plant site. To understand this, one has to appreciate the hundreds of thousands of metric tons [tonnes] of extremely toxic waste and hazardous chemicals that have been buried in over 11 waste pits on the site, the landfill for the three solar evaporation ponds which contain several thousand metric tons of waste buried under the surface with just a thin plastic liner, and the other asbestos wall cladding, tons of crude Sevin, alpha naphthol and Sevin tarry residue, etc., on the site. These materials are gradually leaching into the groundwater aquifer beneath the surface of the plant and spreading through the drinking water supply of at least 10 neighbourhoods surrounding the factory. Some of these toxic and carcinogenic chemicals, scientifically determined in sample tests of the water supply, have been found to be at extremely high levels in the drinking water of these areas.

"receptivity" to an order from a U.S. court directing UCC to pay for and undertake such a clean-up. Furthermore, the Appeals Court expressly instructed the District Court to wait for and hold open the possibility of granting injunctive relief "until the entry of final judgment" in the case.

INDEED, there is no parallel to the December 1984 Bhopal gas tragedy in terms of the magnitude of destruction and the number of deaths. But the process of rendering justice to the victims has proved to be a deplorable legal tragedy. While the criminal case against those responsible for the disaster has been proceeding in the Bhopal District Court at a snail's pace, the civil case seeking due compensation appears to have been closed after the Indian Government and UCC arrived at a settlement before the Supreme Court in 1989. Under the settlement, UCC and its Indian subsidiary, UCIL, agreed to pay, and paid, $470 million to the Government of India on behalf of all the victims. Since then, the survivors of the tragedy have been questioning the unjust nature of the settlement and improper consideration of the compensation claims of individual victims and survivors. Besides, they have been deploring the extent of indifference within India and outside to the magnitude of the tragedy, and its continuing consequences for the health of the survivors and their families.

It was in this context that the organisations of the survivors and the relatives of those killed took their legal battle to the U.S. in November 1999. It coincided with the publication of the report by the Greenpeace Research Laboratories, Department of Biological Sciences, University of Exeter, based on its independent testing of the soil and water in Bhopal. The report found substantial to severe contamination of land and drinking water supplies with heavy metals and persistent organic contami-

Tests conducted by the University of Exeter laboratories in the United Kingdom found, for example, that one carcinogenic chemical was present in the drinking water at nearly 1,705 times the maximum level permitted by the World Health Organisation. Other studies have found these chemicals in the breast milk of women living in the affected areas. Here, you have the possibility of another "slow motion Bhopal", where thousands of people over several generations may be injured or even killed by the underground contamination spreading through the water supplies of the area. In fact, although more studies are needed to determine the precise extent of the groundwater contamination, it is at least conceivable that eventually such high levels of contamination might spread to the drinking water supply of Bhopal as a whole.

The Indian government and the M.P. government now have an opportunity to redeem themselves by preventing this "slow motion Bhopal". On a more practical note, the M.P. government and the Indian government have been aware of this problem for some time but have been unable to address it properly. The M.P. government has asked the company that purchased UCIL to clean up the plant site and remove the source of the contamination. That company, Eveready Industries India Limited, has expressly and publicly refused to do so, claiming that the plant site was surrendered to the M.P. authorities in 1998 and it has no further responsibilities regarding the plant.

The Indian government has been trying to figure out how to handle the large-scale and massive nature of the task of properly remediating the plant site but is daunted by the complexity, magnitude and expense of the task. At one point, I believe they asked the Indian Department of Defence to take a look at the problem. But the problem is simply too huge and complex to be properly handled by government agencies and too expensive for them to contract it out to foreign companies. The Indian Supreme Court too has looked at the matter and asked that the international principle of "polluter pays" should be applied to the issue. Moreover, there is absolutely no reason why Indian taxpayers and the Indian public should be made to pay hundreds and hundreds of crores to clean up the plant site and the off-site contamination caused by Union Carbide, a company that has already done such egregious harm to the country and its people.

The importance of making Carbide responsible for clean-up is, in other words, a most basic proposition of justice: that this notorious corporate criminal should be held responsible for cleaning up the environmental mess that it has made in Bhopal, instead of being allowed to "socialise" this cost to the Indian public and taxpayer while it manages to "privatise" the profits from its unlawful conduct in the form of the sale of UCIL and other plant assets. From 1989 onwards, Union Carbide was actively involved with the Bhopal plant site in terms of what it called its "Bhopal Site Rehabilitation & Asset Recovery Project." Clearly, the emphasis was on "asset recovery" because, by its own admission in our case, Union Carbide has publicly conceded that it basically abandoned the plant site and any proposed remediation efforts in 1994 when the Indian Supreme Court allowed it to sell its shares in UCIL.

► *How will the submissions made by the Government of India and the Madhya Pradesh government before the District Court help the plaintiffs?*

We do intend to approach them. All the insurmountable expenses and difficulties faced by the Indian government or the M.P. government regarding both on-site and off-site remediation can be avoided by the simple expedient of making just one submission to the U.S. court stating that they would be receptive to an order from a U.S. court requiring Carbide to undertake injunctive relief. There is simply no reason why the Indian or M.P. government should hesitate to do so. They are not required to become parties to the case or do anything that would adversely affect or even inconvenience them. It would be astonishing if the Government of India or the M.P. government would fail to avail itself of this opportunity, especially since they would benefit from it almost as much as the Bhopal survivors.

► *How did the Appeals Court address the District Court's argument that India's interests will get impugned by any grant of equitable relief from U.S. courts?*

The District Court ruled that any grant of equitable relief will automatically interfere with India's interests in the context of this case (or, indeed, any other case). Simply put, the District Court held that any grant of such equitable relief by the U.S. courts for remediation affecting property located outside the U.S. would automatically and inevitably be inappropriate because it would interfere with or impugn a foreign sovereign's interests. This was the settled proposition of U.S. law that we were arguing against with no actual precedents in our favour because it has never been done before. Yet, our arguments prevailed over the settled, antiquated rule.

The Appeals Court said: "There may be circumstances in which it is appropriate for a court to grant injunctive relief with respect to the remediation of an environmental problem in a foreign country." It is not very dramatic-sounding, but the legal significance of the ruling is, I believe, nothing less than historic. ■

nants both within and around the former UCIL plant.

In their class action suit, the survivors sought monetary and equitable relief under various common law theories for environmental harm allegedly attributable to the UCIL plant, but not related to the gas leak. The District Court had dismissed these claims, along with others. The Appeals Court returned the case to the District Court in November 2001 in order to permit the latter to consider the claims afresh, as in its view the judge had erred in dismissing them (*Frontline*, January 4, 2002). The dismissal of the claims by the District Court again in March 2003 on other grounds forced the plaintiffs to approach the Appeals Court again.

IF the plaintiffs' legal battle is to succeed finally, they need the cooperation of both the Union and Madhya Pradesh governments. The decision itself presents the invitation to submit a communication in express, specific language: "Madhya Pradesh has neither been made a party to this lawsuit nor sought to intervene, and the record contains no communication from Madhya Pradesh or the Indian government indicating its receptivity to an order of a United States court compelling work on the property... we believe the District Court should be free to revisit its dismissal of the claim for plant-site remediation in the event that the Indian government or the State of Madhya Pradesh seeks to intervene in the action or otherwise urges the court to order such relief."

In other words, if the District Court is to order UCC and its inheritors, Dow Chemicals, to undo the contamination in Bhopal, the two governments must first show their willingness to facilitate the execution of the order. It is up to the two governments now to seize the opportunity and help the plaintiffs-survivors obtain justice, even if it is belated. ■

An institute reborn

The reincarnation of State Observatory, Nainital, as an autonomous institute, ARIES, augurs well for the overall development of astronomy in India.

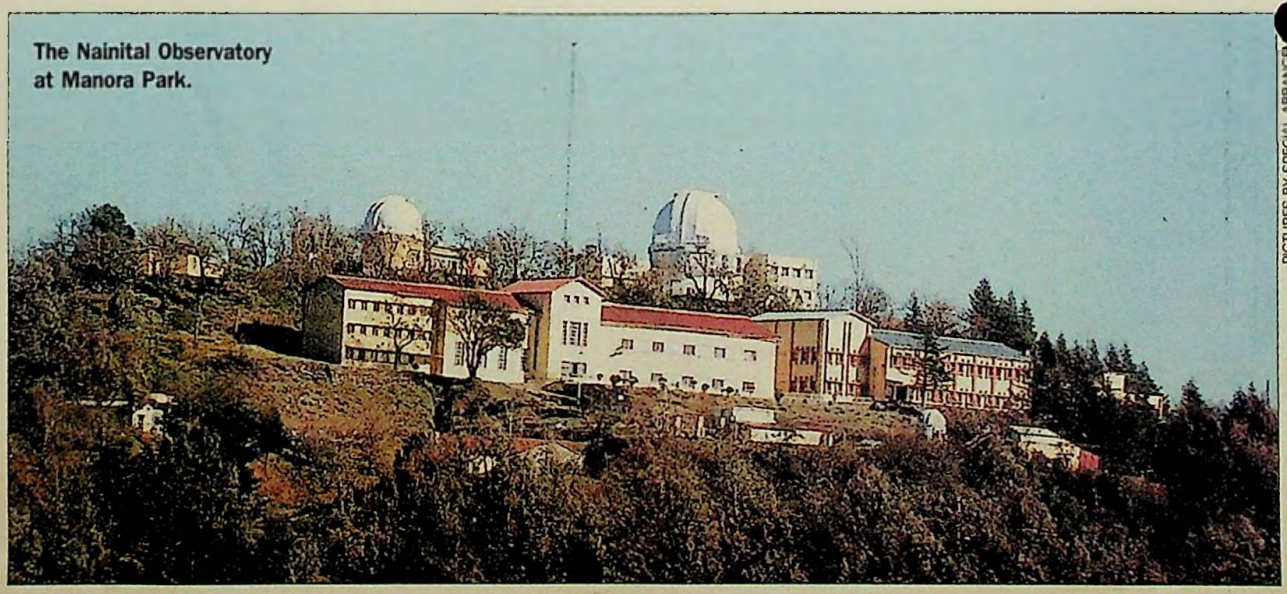
R. RAMACHANDRAN

A NEW national institute of astronomy is born under the zodiacal sign of Aries. Well, not quite. The 50-year-old, historical Uttar Pradesh State Observatory (UPSO) at Nainital, an institute specialising in optical astronomy, underwent an important metamorphosis on March 22. And, for the observatory staff and authorities, the timing of its rebirth is of some significance. No astrological mumbo-jumbo here. Fifty years ago – before it moved to Nainital from the dust and haze of the plains in 1961 – UPSO was founded at Varanasi on April 20, 1954, at the initiative of Dr. Sampurnanand, the then Education Minister and later the Chief Minister of the State, and Prof. A.N. Singh, a Professor of Mathematics at Lucknow University. To signify the sun sign of the month of April, Aries, the observatory in its new avatar will be called ARIES, short for Aryabhata Research Institute of Observational Sciences. The acronym is no doubt highly contrived but equally unusual is the happy coincidence of an institution's golden jubilee also marking the beginning of a new phase that promises a good deal better and brighter times ahead.

On January 7, the Union Cabinet took the decision to convert this State observatory into an autonomous national research institute under the Department of Science and Technology (DST) at the Centre. The new institute was registered as a society on March 12 and, according to the government order, the institute and all its assets (movable and immovable) were formally transferred from the Government of Uttaranchal (under whose administrative control it has been functioning as State Observatory, Nainital (SON) since the creation of the new State in November 2000) to the Centre with effect from March 22.

The observatory is equipped in the main with four telescopes – a 32-year-old 104-cm aperture reflector (called Sampurnanand Telescope), a 36-year-old 56-cm reflector, a 43-year-old 52-cm reflector, a 43-year-old 38-cm reflector – and associated instrumentation. It also has a 15-cm reflecting telescope, acquired in 1960, and a 25-cm refracting telescope, with which the observatory began functioning at Sarnath. These are at present used for public outreach activities. Besides, the observatory also has two 15-cm refractors acquired during 1988-92, which enable solar observations. The total assets transferred, which include the 32.38 hectares of land at Manora Peak (altitude 1,951 metres) in Nainital and 4.48 ha of land at the newly identified observation site at Devasthal, about 50 km from Nainital (altitude 2,500 m), have been valued at Rs.10-15 crores, depending on whether the land is regarded as agricultural or not. Of the total 111 employees at the time of its transfer, 40 (mainly non-academic personnel) have opted to stay with the State government. They will be absorbed in other State institutions in due course. Effective April 1, Prof. Ram Sagar, the Director of the erstwhile State Observatory, took charge as the Director of the new institution.

ARIES now has a 12-member governing council headed by Dr. K. Kasturirangan, the former Chairman of the Indian Space Research Organisation who is currently a member of the Rajya Sabha. The other scientist-members of the council include Prof. V.S. Ramamurthy, Secretary, Department of Science and Technology; Prof. J.V. Narlikar Emeritus Professor, Inter-University Centre for Astronomy and Astrophysics (IUCAA), Pune; Prof. G. Srinivasan, Raman Research Institute (RRI), Bangalore; Prof. P.C. Agrawal, Tata Institute of Fundamental Research (TIFR), Mumbai; Prof. Ramanath Cowsik, former Director and now Distinguished Professor, Indian Institute of Astrophysics (IIA), Bangalore; Dr. S.D. Sinvhal former Di-



The Nainital Observatory at Manora Park.

PICTURES BY SPECIAL ARRANGEM