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# A REAPPRAISAL OF THE IRON STATUS INDICATORS

Iron deficiency is today the most common nutritional problem. Around 30 per cent of the world's population is estimated to be suffering from iron deficiency anaemia with the highest prevalence rates being seen in developing countries. Two-thirds of children and women of child bearing age in most developing countries are estimated to suffer from iron deficiency. Poor absorption of iron from the predominantly cereal-vegetarian diet is a primary cause of iron deficiency. Chronic blood loss as a result of hookworm infestation and malaria also contribute to the deficiency. Iron deficiency, in its early stages is usually unnoticed due to subtle clinical symptoms. However, a variety of functional disabilities such as reduced work capacity, defective immune response and impairment in learning ability occur even in mild-moderate states of iron deficiency while severe deficiency, manifesting as gross anaemia, may lead to foetal complications.

Data on the prevalence of iron deficiency are mainly based on haemoglobin (Hb) estimations and there are established norms to define anaemia in a population (Table I). While a Hb level less than the expected for a given age and sex is identified as anaemia, increase in Hb levels even from the so called normal to higher levels following iron supplementation may be accepted as an indicator of iron deficiency in an individual. However, it is not possible to obtain such information in all situations.

The precise definition of iron status that is reflected in Hb levels needs to be identified. A battery of haematological (Hb, haematocrit, RBC counts, *etc.*) and iron transport and storage parameters [erythrocyte protoporphyrin (EPP), plasma iron, total iron binding capacity (TIBC), percent transferrin saturation and plasma ferritin] are generally used to detect iron deficiency anaemia at a point of time[2]. The validity of a recently identified parameter *viz.*, circulating transferrin receptor is yet to be established.

**Table I. Criteria for diagnosis of anaemia**

| GROUP | Hb levels (g/l) below which anaemia is considered to exist |
|---|---|
| Children | |
| 6 months - 6 years | 110 |
| 6 - 14 years | 120 |
| Adult males | 130 |
| Adult females | |
| Non-pregnant | 120 |
| Pregnant | 110 |

Source: Reference. 1

## Iron Metabolism

Understanding some aspects of iron metabolism is essential to appreciate the interrelationship between different indicators of iron status. Iron absorption occurs mainly in the duodenum and upper part of the jejunum. The most important factor that regulates iron absorption is the iron status of the body. Thus, when body iron reserves are increased, iron absorption is decreased and vice versa.

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In circulation iron binds to transferrin, its transport protein. Transferrin bound iron through its cell surface receptor is utilised by erythron for Hb synthesis. In erythroblasts, iron is incorporated into protoporphyrin-IX for haemoglobin synthesis and the rest of the body iron is stored in ferritin.

### Body Iron Compartments

The human body contains approximately 30-40 mg iron/kg body wt. However, it varies as a function of age and sex and physiological status. Of this, the storage iron concentration ranges from 0 to 15 mg/kg body wt depending on gender and iron status. The distribution of the stored iron is not uniform and liver contains approximately 60 per cent as ferritin. This storage iron has no known functions other than acting as a reserve to replenish the lost iron from the functional compartment. Iron in the functional compartment is involved in cellular metabolism through incorporation into haemoglobin, myoglobin, iron dependent enzymes and other proteins involved in electron transport. About 85-90 per cent of the functional (non-storage) iron pool is in the erythroid mass. The storage and functional compartments are linked by a small transport compartment, through carrier molecule transferrin.

Three stages of progression in iron deficiency have been identified, using different iron status parameters and the compartments affected[2]. The first stage is represented by a depletion of the storage compartment as evidenced either by a lowered marrow iron or levels of circulating ferritin and is not reflected in any of the haematological parameters or those of iron metabolism (latent phase). The second stage represents alterations in the transport system in addition to depletion in stores and may be reflected in decreased per cent transferrin saturation and increased EPP (depleted erythropoietic pool). During the last stage, Hb levels decrease and all parameters of iron status are affected (iron deficiency anaemia).

### Limitations of Indicators

In all surveys to assess prevalence of iron deficiency, Hb is used as the parameter which obviously indicates only severe deficiency. Thus by clinical symptoms or routine diagnostic parameters, the mild-moderate degrees of iron deficiency are missed. Identification and treatment of these forms of iron deficiency is important to improve the quality of life of a large population. All the haematological and iron status parameters could singly identify severe forms of deficiency. However, more than one measurement was suggested to identify mild-moderate forms of iron deficiency, because the response of the parameters to depletion was variable. Efforts to project circulating ferritin as an indicator of subclinical iron depletion were not universally acceptable.

In a population, individuals are expected to be in a state of change from either poor iron status to good status or vice versa. Studies involving sequential monitoring of various indicators including storage iron at different stages of depletion and repletion along with bone marrow staining for iron as a gold standard, are essential to understand the relative merits and scope of each parameter. However, such studies are not practical in human beings. The monkey has been demonstrated to be a good model of ferrokinetics and is phylogenetically closest to human beings. A series of studies were conducted in monkeys at the National Institute of Nutrition (NIN), Hyderabad, to understand the sequential changes in all iron status parameters during different stages of depletion and repletion. In this article, the results of these studies are highlighted and attempts made to identify sensitive indicators of changes in iron status.

### Monkey Liver Ferritin

Monkey liver ferritin has been purified and characterised for the first time and this was found to have the close resemblance to human ferritin with regard to its physicochemical properties. After raising suitable polyclonal antibodies, an ELISA was developed for quantifying the serum ferritin as a measure of the iron status in monkeys[3].

**Table II. Different modes of depletion and repletion of iron in rhesus monkeys**

| Group | Mode | Duration (months) |
|---|---|---|
| Depletion | | |
| Chronic | Diet | 12 |
| Short-term | Diet | 3 |
| Extended | Diet | 15 |
| Acute (phlebotomy) | Diet+blood loss | 3 |
| Repletion | | |
| Acute | Iron-dextran injection | 3 |
| Short-term (dietary adequate) | Diet | 3 |
| Short-term (dietary 50%) | Diet | 3 |
| Control | Diet | 12 |

**Indicators of Mild Iron Depletion**

Mild iron depletion was produced in rhesus monkeys *(Macaca mulatta)* fed a semi synthetic iron depletion diet over a period of one year (Table II). Various haematological and iron status parameters were monitored every month in all the animals. Liver iron was estimated from the biopsy samples at the end of one year. Bone marrow was aspirated at the same time points and was subjected to iron staining.

Of the iron status indicators tested in milder forms of iron deficiency (12 months), bone marrow iron staining, haemoglobin concentration and haematocrit showed the earliest change (Table III). Serum ferritin and liver iron concentration did not respond to mild iron deficiency. Bone marrow staining was very sensitive to depletion and indicated the highest correlation with changes in haemoglobin. A decrease in haematological parameters without effecting any change in iron storage compartment during mild depletion, indicates the existence of a "labile pool of functional iron", probably represented by erythroid iron. This is more sensitive to changes in dietary iron and is in equilibrium with bone marrow iron. Thus, the degree of change in bone marrow grading is also reflected in haemoglobin, haematocrit and RBC counts. It is concluded from the study on rhesus monkeys fed on semi synthetic iron depletion diet that early changes in haematological parameters are adequate to identify mild forms of dietary iron deficiency and parameters reflecting iron stores, including serum ferritin, are not sensitive to such a change[4].

**Table III. Changes in biochemical indicators of iron status in response to various modes of iron depletion and repletion in rhesus monkeys.**

| Group | Ht | Hb | Bone marrow iron | EPP | Serum iron | TIBC | % TS | Serum ferritin | Liver iron |
|---|---|---|---|---|---|---|---|---|---|
| **Depletion** | | | | | | | | | |
| Chronic | – 10m | – 11m | – 12m | + 9m | = | + 12m | – 12m. | = | = |
| Short-term | = | = | – 90d | = | = | = | = | = | = |
| Extended | – 15m | – 14m | – 15m | = | – 14m | = | – 14m | = | = |
| Acute (phlebotomy) | – 30d | – 30d | – 90d | – 30d | – 30d | + 60d | – 30d | = | = |
| **Repletion** | | | | | | | | | |
| Acute | + 60d | + 30d | + 90d | – 60d | = | – 30d | + 30d | = | + 90d |
| Short-term (dietary adequate) | + 30d | + 30d | + 90d | – 30d | = | – 60d | = | = | = |
| Short-term (dietary 50%) | + 30d | + 30d | + 90d | – 30d | = | = | = | = | = |

Ht: Haematocrit; Hb: Haemoglobin; EPP: Erythrocyte protoporphyrin; TIBC: Total iron binding capacity;
% TS: Per cent transferrin saturation
m : Months; d : Days when first changes were seen.
– Decrease; + Increase; = No change

This is contrary to the classical theory that the first and second stages of deficiency are not reflected in Hb status.

**Different Modes of Depletion and Repletion**

Mildly anaemic monkeys were subsequently continued on the iron depletion diet or were repleted with different supplements of iron for 3 months. Control animals were placed on an iron deficient diet or further subjected to periodic phlebotomy (Table II).

**Effect of Iron Depletion**

In the early stages of depletion, only bone marrow iron was found to be decreased even before a decrease in haemoglobin (TableIII). This finding supports the classical definition of latent phase of deficiency to the extent that a decrease in marrow iron, is the first change during depletion. Chronic and extended dietary depletion resulted in decreased haemoglobin along with bone marrow iron (Table III). Decrease in serum iron and percent saturation were also noted in such dietary depletion. On the other hand, all haematological as well as iron status parameters responded to acute depletion involving phlebotomy. None of these manipulations for iron depletion disturbed either serum ferritin concentrations or liver iron concentration, representing iron stores.

**Effect of Iron Repletion**

During various modes of repletion, increase in haemoglobin was the earliest sign along with that of marrow iron followed by other changes like decrease in EPP and increase in transferrin saturation. Liver iron increased only in acute repletion by intramuscular injection of iron and moderate supplements through diet did not result in any change in liver iron. Serum ferritin and liver iron did not respond to supplements with dietary iron. Only repletion through iron-dextran injection had a positive effect on all the haematological and iron transport indicators. Under this condition liver iron also responded but not serum ferritin. Thus, different parameters responded variably under different modes of depletion/repletion, as their response is perhaps related to the degree and duration of change in iron nutriture that had been taking place before and during the intervention[5].

**Significance of Iron Status Parameters**

The turnover of iron in a given compartment may depend on the pre-existing iron status of the animal and subsequent response of the indicators may depend on the immediate demands, pool size of the compartments and the regulatory mechanisms operating at that time. Haemoglobin stands out as the first and best haematological parameter to reflect the changes in iron status as corroborated by bone marrow iron. This is obvious when two-thirds of body iron is present in haemoglobin and its turnover is much greater than that of stored iron.

While the three stages of iron deficiency suggested earlier provide a convenient conceptual framework of dynamics of iron depletion, evaluation of prevalence data for anemia control programmes in humans did not conform strictly to this pattern in individuals with mild iron deficiency. A decrease in serum ferritin along with bone marrow iron even before a decrease occurred in haemoglobin has been shown as evidence for existence of a "latent" stage of iron deficiency. Only in phlebotomy models of iron depletion was such sensitivity of ferritin demonstrated by other investigators. In a representative sub-sample comprising 200 subjects in a field study carried out in about 6000 rural Hyderabad population, a direct relationship was seen only between cases of hypoferritenaemia (<12 ng/ml) and severe forms of anaemia (Hb <10g/dl; NIN, unpublished data). But no direct proof was available relating serum ferritin to liver or bone marrow iron in conditions where dietary depletion was present. Most of the sequential studies in rats also did not support such a latent phase[6]. The insensitivity of serum ferritin to phlebotomy in the present studies could be explained on the basis of unaltered liver iron stores observed in short-term depletion and repletion.

**Dynamics of Iron Balance and Compartments**

Iron deficiency may develop suddenly or gradually. Acute depletion, as with phlebotomy, leads to a rapid exhaustion of stored iron (through increased rates of haemoglobin synthesis) and this may lead to redistribution of the remaining body iron. Chronic depletion is a gradual process and normally occurs due to inadequate amounts of available iron in the diet and may induce iron deficiency with simultaneous depletion of all iron stores.

A revised model incorporating a labile pool to explain the observations noted here is proposed in the figure. The upper panel provides a schematic representation of movement of iron among the different compartments during

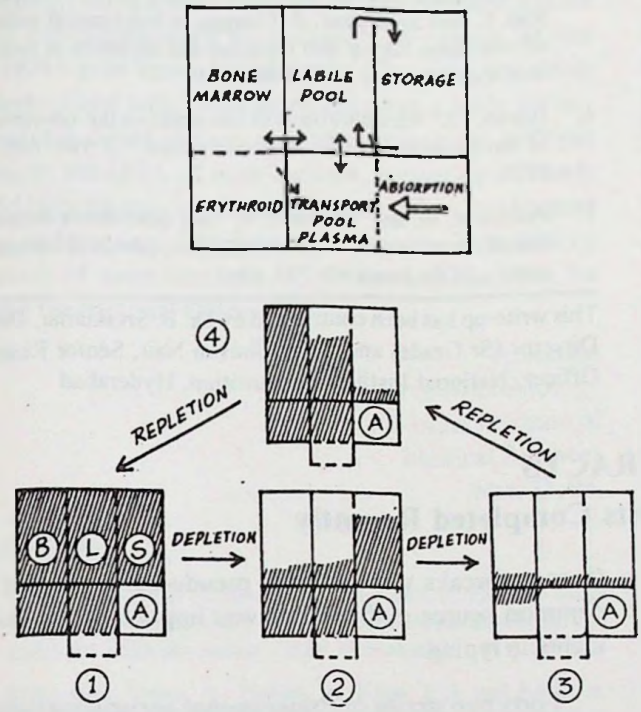



**Fig. Schematic representation of iron compartments (upper panel) and stages operating during depletion or repletion (lower panel).**

The scheme indicates change of L followed by B before any change in S during depletion or repletion.

(B. Bone marrow, L. Labile pool, S. Storage, A. Absorption)

1. Normal with good stores
2. Depletion of labile pool before any change in stores
3. Depletion of storage pool
4. Repletion of labile pool without any change in stores

depletion and repletion. The central concept is that the "labile pool" of iron is the first pool to get depleted or repleted before the stores are changed. For this to occur, movement of iron during repletion is not just opposite to that in depletion and the new scheme (lower panel) accomodates the observed sequential changes in different iron pools. Stage 1 represents the normal iron status wherein all the iron compartments are in equilibrium and are operating at the optimum level. In the course of depletion, stage 2 indicates exhaustion of the labile pool (L) which is considered to be in immediate equilibrium with haematopoietic bone marrow pool (B), before any change occurs in storage pool (S). Exhaustion of the labile pool triggers mobilization of stored iron from the liver and spleen as depicted in stage 3. The cut-off in the supply of labile pool restricts channeling of iron into bone marrow-dependent functional activity *ie.*, decrease in haemoglobin synthesis and RBC formation and explains the delay in accumulation of EPP. On the other hand, repletion fills the labile pool first and then promotes haemoglobin synthesis simultaneous with lowering of EPP as in stage 4. Ultimately the stores are also repleted on continued supplementation reaching a normal state (stage 1). The present studies provide support for the model suggested by Pollycove and Mortimer[7] that ferrokinetics follow a two compartmental model during depletion and three compartmental model during repletion

It is conceivable that if a person is found to have 10 g/dl haemoglobin at a given time, one of the following three situations could arise with regard to the background of the subject : (i) The subject has been stabilised with mild iron deficiency anaemia over a period of time; (ii) the subject had a good iron status earlier but due to the change in the intake, is on a course of depletion at the time of observation; or (iii) the subject, to start with, had iron deficiency anaemia but, is in the process of recovery at the given moment.

Monitoring the iron status (at least at two points) could normally be expected to provide such information. However, actual data did not provide any such clues nor was any analysis really attempted.

As indicated from these experiments, if it is a fact that various iron status parameters do respond differently during repletion and depletion, it is possible theoretically to use more than one parameter along with haemoglobin to obtain information on the previous iron status of the subject in relation to the current condition. Viewed in this context, EPP seems to be sensitive to repletion rather than depletion. Similarly transferrin saturation seems to respond to depletion better than repletion. These results may be reflecting the typical sensitivity of the indicators and the changes taking place in the iron compartments. The present studies highlight the potential of obtaining wholesome information on iron status using selected parameters. With more vigorous testing it is possible to identify the ideal combination of tests which will yield a comprehensive picture of the iron status of an individual with a single sample at one time point.

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7. Pollycove, M. and Mortimer, R. The quantitative determination of iron kinetics and haemoglobin synthesis in human subjects. *J Clin Invest 40*: 753, 1961.

This write-up has been contributed by Dr. B. Sivakumar, Deputy Director (Sr.Grade) and K. Madhavan Nair, Senior Research Officer, National Institute of Nutrition, Hyderabad

# ABSTRACTS

## Some Research Projects Completed Recently

### Molecular fingerprinting of bacteria causing nosocomial infections.

Plasmid fingerprinting was applied for tracing the source of nosocomial infection with plasmid containing bacteria. In addition, bacterial chromosomal fingerprinting after restriction endonuclease digestion was done to determine the relationship between isolates of bacteria causing nosocomial infection but not containing plasmids.

Forty seven strains of *Klebsiella pneumoniae* were subjected to klebocin typing by scrape and streak method using 6 indicator strains. All the strains were found to be positive to klebocin thereby offering no discrimination. By klebocin extraction method these 47 strains could be grouped into 11 distinct klebocin types. However, on repeated testing only 35 strains gave similar pattern indicating a reproducibility of 74.5 per cent. Three major categories of klebocin types predominated even in epidemiologically unrelated strains making discrimination difficult. The two outbreaks (August 1994, June 1995) investigated were caused by a klebocin type designated 444, a group indicating resistance to klebocin produced by all six producer strains and essentially untypable by klebocin typing. Whole cell protein analysis (WCPA) by SDS-PAGE could subdivide the 7 strains from the first outbreak into 5 groups while the 3 strains from the second outbreak were found to be homogenous. Ribotyping of the same isolates indicated 5 groups in the first outbreak and 2 groups in the second outbreak were found to be homogenous. Ribotyping of the same isolates indicated 5 groups in the first outbreak and 2 groups in the second. Ribotyping had better discrimination and indicated that these outbreaks were actually pseudo-outbreaks and not common source outbreaks as was implied by the use of klebocin typing.

Forty two strains of *Pseudomonas aeruginosa* isolated from bronchoalveolar lavage from intubated patients between December 1993 to June 1994 were also analyzed. Pyocin typing of these strains by scrape and streak method using 22 indicator strains revealed excellent discrimination in 40 strains. But on repeated testing the group designation changed indicating that the system had low reproducibility. SDS-PAGE of whole cell protein profile indicated presence of 45 protein bands of different molecular weights, individual isolates had 37 to 42 protein bands ranging from 340 to 143 kDa. On the basis of Dice Index of similarity the strains could be grouped into 20 types. Since all strains could be typed the system had adequate typability. Similar results were obtained on repeated testing indicating good reproducibility. Ribotying of these isolates could group them into 18 groups; the major group comprised 8 isolates.

It is thus clear, that pyocin typing had excellent discrimination but poor to no reproducibility while klebocin typing had adequate reproducibility but not sufficient discrimination. Therefore, these methods cannot be of use for reliable fingerprinting of isolates leading to tracing of the source of infection in an outbreak. Whole cell protein analysis by SDS-PAGE offered a reliable, reproducible and fairly discriminating method for identification of clones of bacteria.

Plasmid analysis was not found to be a very discriminatory or sensitive indicator for fingerprinting. But

ribotyping, using a DIG labelled 18 mer oligonucleotide probe complementary to a conserved region of the 16 S rRNA gene showed good results. The probe was stable and hybridized with DNA extracted from a wide variety of bacteria. Since the rrn operon is present in multiple copies in the DNA of each bacteria, a number of bands would light up making determination of the clonal nature of the isolates easy. Discrimination could be improved by selection of more than one restriction endonuclease for cutting the bacterial DNA.

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**Publications:**

1. Khan, I.A., Rattan, A., Khan, F.G. and Kalia, A. Fingerprinting of *Salmonella typhimurium* – comparison between traditional and molecular methods. *Indian J Med Microbiol 13:* 141, 1995.
2. Khan, I.A., Rattan, A., Fatima, T., Khan, F.G. and Kalia, A. Application of whole cell protein analysis by SDS-PAGE to establish the source of *Salmonella typhimurium*. *J Infect 33:* 169, 1996.
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**Immunological studies in patients with cervical lesions using recombinant human papillomavirus fusion proteins.**

The study was carried out to determine the humoral and cellular immune response against human papillomavirus (HPV) 16 in patients with benign and malignant lesions of the uterine cervix and to correlate the response with the clinical status of the patients.

Serum samples from 13 histopathologically confirmed patients with carcinoma cervix and 9 healthy asymptomatic women were analysed for detection of antibody response to HPV16E7 and L1 fusion proteins in a Western blot assay. Antibodies to HPV16E7 fusion protein were detected in 9 (69.2%) patients and 1 (11.1%) healthy woman, whereas all the 13 patients and 9 healthy women revealed the presence of antibodies to HPV16L1 fusion protein.

ELISA was carried out with the synthetic peptides of two B cell epitopes of HPV16E7 gene product for detecting the presence of antibodies in 30 patients cytopathologically confirmed to have cervical intraepithelial neoplasia (CIN)-I, 21 patients of CIN-II, 13 patients of CIN-III, 30 patients of histopathologically confirmed carcinoma cervix and 30 healthy asymptomatic women. Two synthetic peptides of HPV16E7 comprising the amino acid sequences QAEPDRAHYNIVTF (a,a. 44-57) and QLNDSSEEEDEI (a.a. 27-38) were designated as peptides PI and PII respectively. Thirteen (43.3%), 4(19%), 7(53.8%) and 8(26.6%) patients with CIN-I, CIN-II, CIN-III and carcinoma cervix respectively had antibodies to PI peptide whereas 9(30%), 16(76%), 6 (46.1%) and 18(60%) patients with CIN-I, CIN-II, CIN-III and carcinoma cervix respectively had antibodies to PII peptides. None of the healthy women had antibodies to PI and PII peptides of HPV16E7. Lymphoproliferative assays (LPA) were carried out with synthetic peptide (EPDRAHYNIVTFCC; a.a. 46-59) of HPV16E7 T cell epitope for detection of T cell responses. Two (6.6%) of the 30 controls and 19 (63.3%), 9( 42.8%), 4(30.7%) and 11(36.6%) patients with CIN-I, CIN-II, CIN-III and carcinoma cervix respectively showed positive response to T cell epitope. No correlation could be established between the severity of the disease and T cell responses.

The study thus suggests that peptides PI and PII are the two major immunodominant B cell epitopes of the HPV16E7 and PII is more immunodominant compared to PI. However, more studies need to be carried out to see whether antibody detection to these two peptides can be used to monitor the prognosis and biological progression of patients with CIN.

DNA samples from biopsy material from 30 carcinoma cervix patients were tested for the presence of HPV DNA in dot blot hybridization experiments. Twenty three (76.6%) DNA samples were positive for HPV DNA under low stringency conditions and 10(43.4%) of these 23 samples showed the presence of HPV 16 DNA. ELISA showed the presence of antibodies to HPV16E7 in 7 (70%) of the 10 patients with HPV16DNA, 6 (46.1%) of the 13 patients with HPV DNA and 6 (85.7) of the 7 patients with no evidence of HPV DNA by dot blot hybridization. LPA reactivity to HPV 16E7 T cell epitope was observed in 3 of 10 (30%) HPV16 positive, 7 of 13 (53.8%) HPV16 negative but other HPV positive and 1 of 7 (14.3%) HPV negative cervical cancer patients. These findings suggest that LPA reactivity to HPV16E7 T cell epitope was depressed in most HPV16 positive cervical cancer patients.

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# ICMR NEWS

The following meetings of various technical committees/groups of the Council were held:

**Meetings of the Task Force (TF)/Project Review Committee (PRC) held at New Delhi:**

| | |
|---|---|
| TF on Case Control Study to Identify Risk Factors for Acute Myocardial Infarction in Indians | September 30-October 1, 1997 |
| PRC on Oncology and Pathology | October 7, 1997 |

**Participation of ICMR Scientists in Scientific Events:**

Dr. V. Kumaraswami, Dy. Director, Tuberculosis Research Centre (TRC), Chennai, participated in the II meeting of the Task Force on Community Directed Treatment of Lymphatic Filariasis and Onchocerciasis at Ouagadougou, Burkino Faso (September 16-19, 1997).

Dr. P. Bhaskaram and Dr. B. Sivakumar, Dy. Directors (Sr.Grade) and Dr. K.V.R. Sarma, Asstt. Director, National Institute of Nutrition, (NIN), Hyderabad, participated in the XVIII International Vitamin A Consultative Group meeting at Cairo (September 22-26, 1997).

Dr. J.M. Deshpande, Director, Enterovirus Research Centre, Mumbai, participated in the meeting of Global Polio Laboratory Network at Geneva (September 25-26, 1997).

Dr. Manjula Datta, Dy. Director, TRC, Chennai, participated in the meeting of the Working Group for the Development of New TB Vaccine of the WHO Global Programme for Vaccines and Immunization/Vaccine Research and Development at Geneva (October 4, 1997).

Dr. V.K. Vijayan, Dy. Director (Sr. Grade), TRC, Chennai and Dr. A.K. Mukherjee, Sr. Research Officer, Regional Occupational Health Centre, Calcutta, participated in the IX International Conference on Occupational Respiratory Diseases at Kyoto (October 13-16, 1997).

**Honours/Awards:**

Dr. Kamala Krishnaswamy, Director, NIN, Hyderabad, has been elected as Fellow of the Indian National Science Academy, New Delhi.

## ICMR AIDED SYMPOSIA/SEMINARS/WORKSHOPS/COURSES/CONFERENCES

| Symposium/Seminar/Workshop/ Course/Conference | Date & Place | Contact Address |
|---|---|---|
| IV National Conference of the Society of Biomaterials and Artificial Organs on New Horizons in Biomaterials Research | October 3-4, 1997; (at Hyderabad) | Dr. P.R. Chatterjee, Convenor of the Conference, Indian Institute of Chemical Technology, Hyderabad-500007. |
| IX National Congress of Veterinary Parasitology | October 6-8, 1997; (at Ludhiana) | Dr. H.S. Bali, Organising Secretary of the Congress, Department of Veterinary Parasitology, College of Veterinary Science, Punjab Agricultural University, Ludhiana-141004. |
| Guha Research Conference-1997 | October 13-17, 1997; (at Palampur) | Dr. Amit Ghosh, Co-convenor-GRC'97, Institute of Microbial Technology, Chandigarh-160014. |
| Seminar on Current Status of Thalassaemia in India and National Workshop on Carrier Detection and Antenatal Diagnosis of Thalassaemia | November 13 and 14-15, 1997; (at Lucknow) | Dr. Sarita Agarwal, Organising Secretary, Department of Medical Genetics, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow-226014. |
| International Conference on Advances in Reproductive Medicine | November 15-18, 1997; (at Calcutta) | Dr. Sudersan Ghosh Dastidar, Organising Secretary-ICARM'97, Infertility Clinic and I.V.F. Centre, 208, Rash Behari Avenue, Calcutta-700029. |
| Asia-Pacific Congress on Head and Neck Cancer | December 11-14, 1997; (at Mumbai) | Dr. Ashok R. Mehta, Chairman of the Congress, Department of Surgery, Tata Memorial Hospital, Mumbai-400012. |

## COUNCIL'S TRAINING PROGRAMMES FOR 1997-98

**Reproductive Biology**

*At the Institute for Research in Reproduction, Mumbai:*

- Workshop on Basic Molecular Biology for Clinicians (December 2-11, 1997).

**Nutrition**

*At the National Institute of Nutrition, Hyderabad:*

- Postgraduate Certificate Course in Nutrition (December 1, 1997-February 28, 1998).

**Occupational Health**

*At the National Institute of Occupational Health, Ahmedabad:*

- Orientation Course on Occupational Health for Industrial Medical Officers (September 30-October 11, 1997).
- Training Course on Environmental Hygiene Practice (November 10-14, 1997).

**Laboratory Animal Technology**

*At the National Centre for Laboratory Animal Science, National Institute of Nutrition, Hyderabad:*

- Training Course for Laboratory Animal Supervisors (September 1-November 29, 1997).

**Haematology**

*At the Institute of Immunohaematology, Mumbai:*

- Training Course in Transfusion Medicine for Blood Bank Medical Officers (August 27-October 24, 1997).
- Training Course in Advanced Haematology and Immunohaematology (October 6-24, 1997).

## ICMR PUBLICATIONS

| | *Price (Rs.)* |
|---|---|
| Nutritive Value of Indian Foods (1985), by C. Gopalan, B.V. Ramasastri and S.C. Balasubramaniam, Revised and Updated (1989), by B.S. Narasinga Rao, K.C. Pant and Y.G. Deosthale (Reprinted 1995) | 23.00 |
| Growth & Physical Development of Indian Infants and Children (1972, Reprinted 1989) | 10.00 |
| Studies on Weaning & Supplementary Foods (1974, Reprinted 1996) | 15.00 |
| Studies on Pre-School Children (1974, Reprinted 1984) | 6.00 |
| A Manual of Nutrition (Second Edition 1974, Reprinted 1995) | 6.00 |
| Low Cost Nutritious Supplements (Second Edition 1975, Reprinted 1994) | 4.00 |
| Menus for Low Cost Balanced Diets and School Lunch Programmes Suitable for North India (Second Edition 1977, Reprinted 1994) | 4.50 |
| Menus for Low Cost Balanced Diets and School Lunch programmes Suitable for South India (Fourth Edition 1996) | 6.00 |
| Some Common Indian Recipes and their Nutritive Value (Fourth Edition 1977, Reprinted 1995) by Swaran Pasricha & L.M. Rebello | 10.00 |
| Nutrition for Mother & Child (Third Edition 1978, Reprinted 1994) by P.S. Venkatachalam & L.M. Rebello | 9.00 |
| Some Therapeutic Diets (Fifth Edition 1996) by Swaran Pasricha | 7.00 |
| Nutrient Requirements & Recommended Dietary Allowances for Indians (1990, Reprinted 1995) | 16.00 |
| Fruits (Second Edition 1996) by Indira Gopalan & M. Mohan Ram | 20.00 |

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|---|---|
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