



ICMR BULLETIN

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CURRENT STATUS OF INHIBIN

Although much has been said about the need for a chemical contraceptive for the male, so far, not a single systemic male contraceptive has been developed and brought to the stage of serious clinical testing. Initially efforts were directed at interruption of spermatogenesis by chemical means. However, studies were hindered by the limited knowledge of male reproductive physiology and during the subsequent 30 years, the development of a male contraceptive did not advance beyond the confines of research laboratories.

Production of an azoospermic state would undoubtedly be the most effective means of male contraception. In several species, it is well documented that FSH regulates germinal cell function, while LH is concerned with testosterone production. Any agent which could selectively inhibit spermatogenesis by suppression of FSH without affecting androgen secretion from the testis would have potential use for male contraception. 'Inhibin' is a peptidic factor of gonadal origin that selectively and specifically lowers the rate of secretion of FSH.

In simple terms, two pituitary gonadotropins act on the testis, FSH stimulates the seminiferous tubules while LH stimulates the Leydig cells; the Leydig cells produce androgens (particularly testosterone) which control LH secretion by negative feedback and the seminiferous tubules produce inhibin which

controls FSH secretion by negative feedback.

Evidence for Existence of Inhibin:

Circulating levels of FSH and LH have been found to be altered in certain pathologic conditions of the human testis. Serum FSH levels are elevated without altering LH and testosterone levels in patients with disturbance of spermatogenesis and normal Leydig cell function^{1,2}. It was found that a substance produced by the seminiferous tubules was involved in the regulation of FSH secretion. The existence of such a substance was first postulated by McCullagh³, who named it 'Inhibin'. Evidence for inhibin activity in human seminal plasma was shown by Franchimont⁴. Since FSH inhibiting activity was found in seminal plasma of normal and oligospermic men, but not in the semen of azoospermic patients, spermatids were postulated to be the most likely source of inhibin⁴. Other investigators could not demonstrate a correlation between specific spermatogenic stages and serum FSH levels, which suggested that the Sertoli cells could be the cellular source of inhibin⁵. In the rat, it has been demonstrated that Sertoli cells in culture secrete a factor - Sertoli cell factor (SCF) - which selectively suppresses FSH production by cultured pituitary cells⁶. However, no direct evidence for secretion of an FSH inhibiting substance by the human Sertoli cell is available.

Interest in inhibin as a potential male contraceptive was stimulated by virtue of its

ability to suppress FSH secretion. However, reports over the last decade have merely established the existence of inhibin in gonadal secretion of several species without making any spectacular studies on its chemistry or physiology.

Interest in its physiological and biochemical properties has resulted in the need for purification of inhibin in sufficient quantity. Various investigators have used different sources for inhibin extraction (Table).

SOURCES OF INHIBIN

Sources	Molecular weight	Investigators
Human seminal plasma	<5,000	Franchimont <i>et al</i> ⁷
	19,000	Thakur <i>et al</i> ⁸
Ovine rete testis fluid	<5,000 to >160,000)	
	)	
Ovine testicular lymph	90,000)	Baker <i>et al</i> ⁹
	)	
	30,000)	
Bovine seminal plasma	15,000 to 19,000	Chari <i>et al</i> ¹⁰ Sairam <i>et al</i> ¹¹
Ram testis	1,500	Sheth <i>et al</i> ¹²

A variety of extraction methods have been used including some which might be expected to lead to a loss of low molecular weight substances, *e.g.* ethanol precipitation, charcoal treatment. The evidence from a number of groups that inhibin like activity is destroyed by heat and by enzymatic treatment with trypsin, pepsin or pronase, has established that the material is peptide or proteinaceous in nature. There is, however, considerable confusion regarding the molecular weight of the active material even when prepared by different investigators from the same starting material. This discrepancy has been attributed to polymer formation, association with a carrier protein or purification artifacts.

Measurement of Inhibin:

The evidence for the existence of inhibin which has been disputed since several investigators have been able to reverse completely the changes in gonadotropin levels which follow castration by the administration of steroids combinations, rests on various assay methods. Given the definition, the obvious response metameter for any biological

assay is the reduction in FSH secretion, which can be measured indirectly by the assessment of a biological effect of FSH or directly by FSH assay. The major problem with inhibin assays is that of specificity; all the bioassays are based on suppression of FSH secretion and/or synthesis, which can be affected by a variety of cell toxins, or by other biologically active substances.

Using a homogeneous inhibin preparation of human origin (human seminal plasma) a sensitive and specific radioimmunoassay for inhibin¹³ has been developed at the Institute for Research in Reproduction (IRR), Bombay. None of the purified hormones tested, such as LH, FSH and prolactin from different species showed any cross-reaction in this radioimmunoassay. Steroid hormones such as testosterone, dihydrotestosterone, estradiol 17 β and progesterone did not interfere with the assay. Using this radioimmunoassay, inhibin has been measured in various biological fluids and tissues. Normospermic semen is found to contain significantly higher levels of inhibin than the oligospermic semen¹⁴. Similar results have been reported with bioassay (Berger *et al*. Personal communication).

Physiological Characterization of Inhibin:

The half-life of inhibin is much shorter in adult rats than in immature rats (2.4 min for adult rats and 90 min for immature rats). The metabolic clearance rate of inhibin is greater in adult rats as compared to the immature rats (0.12 ml/min in adult rats and 0.019 ml/min in immature rats). The slower clearance of inhibin in immature rats leads to longer half-life.

The levels of inhibin are much higher in immature rats as compared to adult rats¹⁵. Inhibition of FSH binding was more pronounced in immature rats as compared to the mature rat testicular receptor preparation at the same dose of inhibin¹⁶. The role of FSH in spermatogenesis indicates that the initiation of spermatogenesis at puberty and also the reinitiation of spermatogenic cycle in regressed testis requires the presence of FSH. It appears that inhibin may control puberty in men.

Half-life studies were carried out in castrated and testosterone treated groups with labelled inhibin. The half-life is prolonged in these two groups as compared to the intact rats. It is tempting to speculate that there are some testicular factors which shorten the half-life or affect the metabolism of inhibin. It is also postulated that inhibin acts in synergism with androgens in the male and estrogens in the female¹⁷. If so, then inhibin alone or in combination with steroids, can be used to bring about 80-90 per cent suppression of FSH.

Site of Action:

The *in vitro* evidence, from a number of laboratories, indicates that inhibin preparations exert suppressive effects on FSH secretion by cultured pituitary cells of rats, while limited experiments in sheep also indicate that a major effect of inhibin contributing to its *in vivo* effect of lowering FSH levels, is a direct one on the pituitary gland. The effect is seen on basal release and cell content as well as on LHRH stimulated release.

Action at the testicular level has been postulated by Franchimont and co-workers⁷. IRR has provided the first experimental evidence of this by demonstrating that inhibin inhibits the binding of FSH to its testicular receptor^{16,18,19}.

Simultaneous incubation of inhibin and ovine FSH ($6 \times 10^{-8}M$) with testicular slices resulted in reduction of cAMP accumulation as compared to controls. When testicular slices were incubated with inhibin the phosphodiesterase (PDE) activity was increased in a dose related manner. This suggests that the observed inhibition of cAMP accumulation is due to reduction in adenyl cyclase and/or increase in PDE activity. When inhibin was incubated in the presence of labelled hCG with testicular receptor, inhibin was unable to inhibit the binding of hCG to its receptor. This suggests that the observed inhibition with inhibin is specific to FSH. From the contraceptive point of view, this is advantageous because inhibin does not affect the LH action at the testicular level. Recently, Daume *et al.*,²⁰ have also reported the presence of a low molecular weight factor from human follicular fluid that inhibits *in vitro* binding of FSH to ovarian granulosa cells.

Other mechanisms of action may also be operating. Inhibin preparations have been found to decrease the endogenous LHRH content of isolated rat hypothalamus after short term incubation⁷. Similarly, studies at the IRR have shown suppression of hypothalamic LHRH content on administration of low molecular weight testicular inhibin preparation to castrated adult male rats.

It may be difficult to correlate the effects on hypothalamic LHRH with the observed specificity of inhibin action on FSH release and the question must again be raised that more than one gonadal peptide is involved in gonadotropin regulation or that the hypothalamic effects represent a non-specific toxic effect. However, in a number of experimental situations, it has been shown that effects on LH release are earlier and of shorter duration than effects on FSH. This would be consistent with acute effects on LHRH with more prolonged effects on FSH synthesis.

Site of Synthesis:

The probable cell type responsible for inhibin production still remains controversial. The general view is that the Sertoli cells are the probable site of its synthesis in the testis. It has also been demonstrated in *in vitro* culture of pituitary and Sertoli cells^{21,22}. However, Krenger *et al.*²³, have suggested that Sertoli cells in association with germ cells are responsible for inhibin production. Experimental evidence shows high amounts of bioactive and immunoreactive in-

hibin in enriched spermatid fraction obtained from rat testis extract (Sheth *et al.*, unpublished data). Recently, *in vitro* localization of inhibin in spermatid region has been shown by immunofluorescent technique (Arbatti *et al.*, unpublished observations).

Role of Inhibin in Female:

Using identical extraction procedures, inhibin has been purified from sheep testis and ovaries¹⁹.

Daily treatment of adult female or immature male rats for periods longer than 5 days did not result in prolonged suppression of circulating FSH concentrations, LH levels were significantly increased. Female rats showed cyclic vaginal smear changes and normal ovulation; in the male rat, testis weight and number of spermatogenic cells were reduced. It was, therefore, concluded that testicular and ovarian inhibin-like activities are similar. Injections of follicular fluid into male and female rats caused similar effects on FSH and LH. The effect of follicular fluid in unilateral ovariectomized animals suggests that inhibin could play a role in the short term regulation of the number of developing follicles in the ovary. Injection of follicular fluid into male rats caused a probably transient impairment of spermatogenesis through an initial suppression of FSH.

Erickson and Hsueh²⁴ have demonstrated that granulosa cell cultures secrete inhibin-like material which acts directly upon cultured pituitary cells. In view of the preclusive relationship that FSH has on granulosa cells, it would be physiologically meaningful for the specific target cell to provide a selective signal in the modulation of its trophic (FSH) secretion. Thus, an exclusive feedback control system of granulosa cell - FSH axis may operate through follicular inhibin. An inverse correlation between FSH inhibiting activity and follicular size has been observed²⁵. Depalo and associates²⁶, employing an *in vitro* bioassay, used ovarian venous plasma to estimate inhibin activity. Similarities of inhibin of testicular and ovarian origin have been recently established¹⁹. It is not surprising that antigenic similarities were observed between 'female inhibin' and human seminal plasma 'male inhibin' as reflected by the parallel dose responses obtained in radioimmunoassay developed for human seminal plasma inhibin. Inhibin levels showed cyclic variations during menstrual cycles. In all the six cycles

studied, an inhibin peak was observed during late follicular phase, 2 to 3 days earlier than the LH peak²⁷.

The role of FSH in the female is more pronounced as compared to the male. Preliminary studies from IRR, Bombay, on inhibin revealed that it has an anti-implantation effect in mice. It would thus appear that inhibin may be potentially useful for female contraception.

From the reported studies at IRR, Bombay, and other laboratories, it appears that inhibin perhaps exist in multiple forms. Since inhibin is a native hormone, toxicity will be minimal. From various studies, it has been shown that inhibin is capable of suppressing only 70-80 per cent of the FSH secretion. It is possible that inhibin and a steroid (estrogen or androgen) may be involved in the physiological regulation of FSH. To suppress completely circulating FSH levels, administration of either a combination of inhibin and a steroid or a powerful synthetic analogue of inhibin may be necessary. Studies at IRR, Bombay, have demonstrated the identical characteristics of male and female inhibin. Preliminary studies indicate the anti-implantational activity of inhibin.

Studies clearly indicate that inhibin has a very short half-life in the adult animal, which may be due to the involvement of other testicular factors. Apart from its possible contraceptive application in the male or the female, inhibin can help to understand the basic role of FSH in reproductive disorders. The availability of synthetic inhibin would certainly solve some of the existing controversies in this field.

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contributed by Dr. A.R.Sheth and colleagues of the Institute for Research in Reproduction, Bombay.

ABSTRACTS

Some Research Projects completed recently

Role of hypersensitivity in intestinal/hepatic amoebiasis

With a view to assess the role of specific amoebic and/or non-specific prior sensitization on the outcome of intestinal/hepatic amoebiasis this study was undertaken.

All the animals studied, became hypersensitive with monoaxenic amoebic antigen and developed fulminating caecal ulcers while approximately 13 per cent animals developed hepatic abscesses. Thirty eight per cent of the animals developed granulomatous lesions as well. Animals sensitized with axenic amoebic antigen showed protection in 30 per cent of animals; caecal lesions were seen in 70 per cent and hepatic lesions in 7 per cent animals. It seemed that axenic amoebic antigen could protect as well as make some of the animals hypersensitive depending upon the concentration of functional antigenic determinants. Animals sensitized with live amoebae developed lesions only in 20-30 per cent animals indicating that live amoebae act as better immunogen. Circulating antibodies seemed to have no role in protection as the animals with lesions had as high antibody titres as the infected animals without lesions. Cell mediated immune response was altered in animals with amoebic liver abscess.

In human studies analysis of the relationship between serum antibodies and the degree of macrophage inhibition did not show much correlation between the two parameters,

indicating thereby that inhibition did not reflect the humoral antibody response.

V.K. Vinayak
Department of Experimental
Medicine
Postgraduate Institute of
Medical Education and
Research
Chandigarh.

Publications:

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Effect of lipid composition on the virulence of Mycobacterium tuberculosis

The alterations in the lipid composition in response to low temperature and fatty acid supplementation in saprophytic avirulent and virulent strains of *M. tuberculosis* were studied. It was observed that total lipids

(total phospholipids as well as the glycerides) increase at lower growth temperature in *M. smegmatis* 607 and *M. phlei* whereas in the pathogenic strain H₃₇R_V, total phospholipids were found to decrease. Among the individual phospholipid components, a general trend of decrease in phosphatidylinositolmannosides and increase in phosphatidylethanolamine was observed. In addition, cardiolipin was found to increase significantly in *M. tuberculosis* H₃₇R_V. Unsaturated fatty acids showed a marked increase at low temperature. The supplementation of exogenous fatty acids produced complex changes with different fatty acids in different strains. No significant changes were observed in lipids of BCG strain (avirulent) under these conditions.

Comparison of human strain of H₃₇R_V (isolated from human sputum) with the stock culture (NCTC London), revealed the presence of high contents of total lipids as well as total phospholipids in virulent human strains as compared to NCTC strain. It was also interesting to note that the total lipids of

the human H₃₇R_V strains increased significantly when the cells were grown in media supplemented with exogenous fatty acids.

Furthermore, virulence was observed to be related with the lipid composition of cells to some extent. *M. tuberculosis* H₃₇R_V cells grown in the presence of palmitoleic acid were found to be more virulent than the control cells.

G.K. Khuller
Department of Biochemistry
Postgraduate Institute of
Medical Education and
Research
Chandigarh.

Publication:

Taneja, R., Malik, U. and Khuller, G.K. Effect of growth temperature on the lipid composition of *Mycobacterium smegmatis* AICC 607. *J Gen Microbiol* 113: 413, 1979.

SUMMARY OF THE BASANTI DEVI AMIR CHAND PRIZE (1981) ORATION

Dr. N.C. Nayak, Professor of Pathology, All India Institute of Medical Sciences, New Delhi, delivered the Basanti Devi Amir Chand Prize (1981) Oration on 'Immunological Aspects of Viral Hepatitis with Particular Reference to Immune Response against Liver Specific Protein' at the Indian Council of Medical Research, New Delhi, on September 10, 1981.

Infections with hepatitis viruses are responsible for the majority of acute and chronic hepatitis - health problems of considerable magnitude in India and the South-East Asia region. These viruses do not appear to be cytopathic and the mechanism of hepatocellular injury induced by them remains unknown. Immune reactions against the virus or against altered or normal cellular constituents subsequent to infection have been considered to have important bearing on the pathogenesis of hepatitis.

An attempt was made at the AIIMS, New Delhi, to explore the latter facet by investigating the role of immune responses against the specific liver cell membrane protein (LSP) in human patients and in mice. Antibodies to LSP circulating in blood as well as fixed on hepatocytes can be demonstrated not only in patients with acute and

chronic hepatitis but also in those with some other liver diseases. Development of these antibodies appears to result from rather than causing liver injury.

High lymphocyte mediated hepatocytotoxicity is present in the first 3 weeks of jaundice due to acute hepatitis. The values touch control levels at around 6 weeks or later. Some patients including a few going on to chronicity show resurgence of cytotoxicity.

Mice immunised with rat LSP (1 or 2 doses) manifest both humoral and cellular immune response against this protein as measured by immunofluorescence and cytotoxicity (⁵¹Cr release and morphologic cell loss) respectively. These reactions seem to have two humps, a small one at 3 to 4 weeks and a larger at 14 to 18 weeks. Histologically, there is lymphocytic infiltration in the liver parallel in intensity to the immune reactions, but no significant hepatocytic damage is seen.

It is concluded that even though LSP is actively immunogenic there is, as yet, no evidence to indicate a significant *in vivo* cytopathogenic role of anti LSP immune reactions in man or mouse.



ICMR NEWS

Advisory Group/Study Group meetings:

The following ICMR Advisory Group/Study Group meetings were held at the Council during the month:

Advisory Group on Maternal and Child Health	September 22-24, 1981
Study Group on Diarrhoeal Diseases and Acute Respiratory Diseases	September 28-29, 1981

Dr. S.C. Pal, Director, National Institute of Cholera and Enteric Diseases, Calcutta, participated in the Steering Committee meeting of the WHO Scientific Working Group on Bacterial Enteric Infections held at Geneva between September 2-4, 1981. He also participated in the International Symposium on Recent Advances in Enteric Infections held at Brugge, Belgium, from September 8-11, 1981.

Dr. B.C. Deb, Deputy Director, National Institute of Cholera and Enteric Diseases, Calcutta, participated in a training course on Health Services Research Evaluation with emphasis on Diarrhoeal Diseases Control, held at Dacca, Bangladesh, between September 7-18, 1981.

Dr. V. Ramalingaswami, Director-General, Indian Council of Medical Research, New Delhi, inaugurated the XII Biennial Conference of the Indian Association of Leprologists at Agra on September 9, 1981.

Dr. S.P. Tripathy, Director, Tuberculosis Research Centre, Madras, participated in the meeting of the WHO/IUAT Joint Study Group on Tuberculosis Control at Geneva between September 14-18, 1981.

Dr. V. Ramalingaswami participated in the meeting of the Scientific and Technical Advisory Committee of the WHO Special Programme for Research and Training in Tropical Diseases at Geneva between September 15-19, 1981. He also participated in the meeting of the WHO ICMR Sub-Committee on Cancer, between September 28-30, 1981.

Dr. Usha K. Luthra, Senior Deputy Director-General, ICMR, visited various centres of Cytology/Cytopathology in the USSR between September 15-22, 1981.

New Appointments:

Dr. A.J. Baxi, Assistant Director, Blood Group Reference Centre, Bombay, has been appointed as Deputy Director at the Centre.

ICMR AIDED SYMPOSIA/SEMINARS/WORKSHOPS/CONFERENCES

Symposium/Seminar/Workshop/Conference	Date	Institution/Department
Workshop on Scientific Methods in Morphosciences.	August/September, 1981	Department of Anatomy, Maulana Azad Medical College, New Delhi (Delhi Association of Morphological Sciences).
UNESCO-IBRO Workshop on New Techniques in Neurosciences with special reference to the Neurophysiology of Higher Nervous Activity.	August 30 - September 12, 1981	National Institute of Mental Health and Neurosciences, Bangalore.
Symposium on Science, Technology and Society.	September 7-12, 1981	Bombay (Indian Women Scientists Association).

Symposium/Seminar/Workshop/ Conference	Date	Institution/Department
Symposium on Leprosy.	September 9-12, 1981	Central Jalma Institute for Leprosy, Agra (Indian Association of Leprologists).
Workshop on Gastrointestinal Endoscopy.	September 19, 1981	Department of Gastroenterology, Medical College, Trivandrum (Society of Gastrointestinal Endoscopy of India).
Seminar on Involvement of Medical Colleges/Institutions in the Delivery of Comprehensive Health Care to the Rural Community.	September 25-27, 1981	Medical College, Nagpur.
Third National Conference on Drug Abuse.	September 28 - October 1, 1981	Department of Psychiatry, All India Institute of Medical Sciences, New Delhi.
Symposium on Fertility Control and Teratology.	October 3, 1981	JN Medical College, Aligarh Muslim University, Aligarh.
Symposium on Regulation of Fertility.	October 3-5, 1981	School of Life Sciences, University of Hyderabad, Hyderabad (Endocrine Society of India).
National Symposium on Cataract and Corneal Disorders.	October 12-17, 1981	Dr. Rajendra Prasad Centre for Ophthalmic Sciences, New Delhi.
4th All India Congress of Cytology and Genetics, 1981.	October 17-21, 1981	University of Bhagalpur, Bhagalpur.
First National Conference on Environmental Biopollution.	October 19-21, 1981	Marathwada University, Aurangabad (Indian Aerobiological Society).
Seminar-cum-Workshop in Surgery for Postgraduates and Surgeons.	October 31 - November 1, 1981	All India Institute of Medical Sciences, New Delhi.
Second Workshop on <i>In vitro</i> Cultivation of Erythrocytic Forms of <i>P. falciparum</i> and Preparation of Antigens for Serology of Malaria.	November 2-15, 1981	Department of Microbiology, St. John's Medical College, Bangalore.
Conference on Industrial and Environmental Toxicology.	November 6-7, 1981	Industrial Toxicology Research Centre, Lucknow (Society of Toxicology, India).

Symposium/Seminar/Workshop/ Conference	Date	Institution/Department
Symposium on the Heart.	November 6-8, 1981	Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum (Indian Aca- demy of Sciences).
Satellite Congress on Diabetes in the Young.	November 21-22, 1981	Ootacamund(The Coimbatore Branch of the Diabetes Association of India).
Symposium on Tropical Immunology.	November 23, 1981	Cancer Research Insti- tute, Tata Memorial Centre, Bombay (Indian Immunology Society).
International Symposium on Early Diagnosis and Prevention of Disabi- lity.	November 26-28, 1981	Department of Pediatrics, B.J. Medical College, Pune (Indian Society of Human Genetics).
Seminar on Teaching and Research in Medical Sociology in India.	November, 1981	University of Jodhpur, Jodhpur.
International Symposium on Incidence of Infections and Chronic Diseases - Geographical Perspectives.	December 1-4, 1981	Department of Geography, University of Madras, Madras.
National Seminar on Health for All - A National Dialogue on Operational Aspects of Comprehensive Primary Health Care and Rural Development.	December 12-13, 1981	Birla Institute of Medi- cal Research, Gwalior.
International Conference on the Applications of Mossbauer Effect.	December 14-18, 1981	Jaipur (Indian National Science Academy).
Symposia on Nutrition & Environment and Current Concepts in Membrane Biochemistry.	December 21-24, 1981	Department of Biochem- istry, University Coll- ege of Sciences, Calcutta.
30th Annual Conference of the Anato- mical Society of India.	December 28-30, 1981	Gandhi Medical College, Bhopal.
International Workshop on Nutrition and the Development of the Child.	January 8-15, 1982	Department of Biochemis- try, M.S. University, Baroda.
Workshop on Evaluation of Mutagenic Potential of Environmental Agents: Microbial and Mammalian Systems.	February 15-19, 1982	Division of Biochemistry and Food Technology, Bhabha Atomic Research Centre, Bombay (Environ- mental Mutagen Society of India).

**PROGRAMME OF SCIENTIFIC LECTURES AT THE
INDIAN COUNCIL OF MEDICAL RESEARCH, NEW DELHI**

Date	Topic	Speaker
10.9.1981	Immunological Aspects of Viral Hepatitis with particular reference to Immune Response against Liver Specific Protein (Basanti Devi Amir Chand Prize Oration, 1981)	Dr. N.C. Nayak, New Delhi
24.9.1981	Influenza, the Perennial Scourge: A Challenge to Mankind by a Changing Virus (Dr. J.B. Srivastav Oration, 1981)	Dr. B. Lalitha Rao, Pune.
29.10.1981	Evaluation of Methods for Fertility Regulation (Kshanika Oration Award, 1980)	Dr. K. Prema, Hyderabad.

COUNCIL'S TRAINING PROGRAMMES FOR 1981

Leprosy:

At the Central Jalma Institute for Leprosy, Agra:

Training course in Leprosy for medical officers: (February 15 to March 28, 1981, and August 31 to October 14, 1981).

(2) M.Sc. (Applied Nutrition) course: (June 1, 1981 to February 28, 1982).

(3) Annual certificate course in Public Health Nutrition: (October/November, 1981).

(4) Annual certificate course in Nutrition: (December 1, 1981 to February 28, 1982).

Virology:

At the National Institute of Virology, Pune:

(1) Diploma course in Medical Virology of Poona University: (July, 1981).

(2) *Workshop on Japanese Encephalitis in India.

Endocrinology:

At the National Institute of Nutrition, Hyderabad:

Annual certificate course in Endocrinological Techniques and their Application: (August to September, 1981).

Nutrition:

At the National Institute of Nutrition, Hyderabad:

(1) Annual certificate course in Human Nutrition for teachers from Agricultural Universities: (June/July, 1981).

Haematology:

At the Blood Group Reference Centre, Bombay:

(1) A certificate course in Blood Banking Technology and Basic Haematology for medical officers: (February 1 to March 15, 1981).

(2) Training course in Blood Banking Methodology for technicians: (May 12-26, 1981).

Mental Health:

At Bangalore, Chandigarh and Delhi:

Training course on Extension of Mental Health Services in the Community: (July, 18 - August 29, 1981).

Occupational Health:

At the National Institute of Occupational Health, Ahmedabad:

COMMUNITY HEALTH CELL
326, V Main 1 Block
Koramangla
Bangalore-560034
India



NIOH/WHO Training Course on Air Pollution and Health: (December 1-10, 1981).

Oncology:

At the Cancer Research Institute, Tata Memorial Centre, Bombay:

Training course on Carcinogenicity, Mutagenicity and Teratogenicity: (December, 1981).

Clinical Pharmacology:

*At the Department of Pharmacology, Seth G.S. Medical College, Bombay.

Biostatistics:

At the Institute for Research in Medical Statistics, New Delhi:

- (1) Certificate course in Planning and Evaluation of Health Programmes: (April, 1981).
- (2) Diploma course in Biostatistics: (September, 1981).
- (3) Certificate course in Orientation to Biostatistics: (September, 1981).
- (4) Certificate course in Statistical Techniques in Medical Research: (January, 1982).

Laboratory Animal Technology:

At the National Institute of Nutrition, Hyderabad:

- (1) Laboratory Animal Supervisors Training Course: (January 2 to March 31, 1981).

- (2) Laboratory Animal Technicians Training Course: (August 1 to September 30, 1981).

COURSES FOR TALENT SEARCH SCHEME FELLOWS

Cell-Biology and Cyto-Genetics:

At the Indian Institute of Science, Bangalore: (March, 1981).

Use of Radioisotopes in Medicine:

At the National Institute of Nutrition, Hyderabad: (July, 1981).

Immunological Techniques:

At the Central Jalma Institute for Leprosy, Agra: (November, 1981).

Haematological Techniques:

*At the Department of Pathology, Christian Medical College, Vellore.

Clinical Endocrinology:

*At the Institute for Research in Reproduction, Bombay.

*Dates to be announced later.

SOME ICMR PUBLICATIONS

	Price Rs.
Menus for Low-Cost Balanced Diets and School Lunch Programmes suitable for South India (1959), Third Edition 1977, Reprinted 1978	1.50
Menus for Low-Cost Balanced Diets and School Lunch Programmes suitable for North India (1959), Second Edition 1977	1.50
Nutrition for Mother and Child (1962), Third Edition 1978	4.50
Some Common Indian Recipes and Their Nutritive Value (1964), Fourth Edition 1977, Reprinted 1980	6.50
Nutritive Value of Indian Foods (1971), Reprinted 1981	9.00
Dietary Allowances for Indians (1971), Reprinted 1980	5.50
Diet Atlas of India (1971)	8.00
Nutrition Atlas of India (1971)	10.50

	Price Rs.
Atlas of Histopathology of Liver: Hepatitis and Cirrhosis (1974)	10.00
Studies on Pre-School Children (1974), Reprinted 1977	3.50
Studies on Weaning and Supplementary Foods (1974), Reprinted 1977	4.00
Medicinal Plants of India Vol. I (1976)	30.00
Research in Malaria - An Outline (1977)	Gratis
Health Hazards of Mycotoxins in India (1978)	5.00
Japanese Encephalitis in India, Revised Edition 1980	5.00
Recommended Dietary Intakes for Indians (1981)	4.00

These publications are available on prepayment of cost by cheque or postal order (postal charges will be extra) drawn in favour of the Director-General, Indian Council of Medical Research, New Delhi. Money orders are not acceptable. All correspondence in this regard should be addressed to the Chief, Division of Publication and Information, Indian Council of Medical Research, Post Box 4508, Ansari Nagar, New Delhi-110029.

TISSUE CULTURE

A Workshop on "Animal Cell Culture and Differentiation" (November 20 - December 15, 1981) will be held at the Department of Zoology, University of Poona, Pune, in collaboration with the National Institute of Virology, Pune.

Prof. S.P. Modak, Department of Zoology, University of Poona, Pune-411007, may be contacted for details on or before 3rd October, 1981.

RADIOIMMUNOASSAY KITS AVAILABLE

The reagents listed below (procured under the Indo-US Sub-Commission Programme) are available at the Reagent Bank, Institute for Research in Reproduction, Bombay, in limited quantities, free of charge, to qualified investigators for research and diagnostic purposes only:

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Human	Rat	Subhuman
h TSH	r LH	primate pregnancy
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h FSH	r FSH	
h GH		
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B hCG		

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Only one vial, or one kit (one kit = one vial of antigen (50 ug) and one vial of antiserum) of the reagents, animal or human for RIA will be supplied at a time. Request for larger amounts may also be considered.

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