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NUTRITION NEWS

● Dietary Guidelines For Indians

A publication on *Dietary Guidelines for Indians* prepared by the Indian Council of Medical Research (ICMR) and the National Institute of Nutrition, will be released shortly. The preparation of this publication was preceded by several meetings of a working group of senior scientists of the National Institute of Nutrition, and a panel of experts under the Chairmanship of Dr Kamala Krishnaswamy, Director, National Institute of Nutrition.

This book meets a long-felt need. In drawing up these guidelines, the experts have kept in mind the socio-cultural context in which these guidelines will be used.

The book is addressed to the common man, nutrition scientists, dieticians, health programmers and policy makers. An abridged version of the book, with emphasis on messages on nutrition, has also been prepared for wide circulation.

The Indian Council of Medical Research and the National Institute of Nutrition deserve to be congratulated on this important effort.

● **The Fourth Annual National Conference Of The Indian Society For Parenteral And Enteral Nutrition** was held at the National Institute of Nutrition, Hyderabad, on March 13 and 14, 1998. Dr Sarath Gopalan Consultant in Paediatric Gastroenterology and Clinical Nutrition at the Pushpavati Singhania Research Institute for Digestive, Liver and Renal diseases, was the Organising Secretary.

Less Recognised Micronutrient Deficiencies In India

Mahtab S. Bamji and A.V. Lakshmi

Optimum health demands an adequate intake of all macro and micronutrients. Since utilisation of one nutrient is often dependent on the adequate supply of some other nutrient, deficiency of any one of them affects not only the biochemical functions dependent on that nutrient, but also the entire metabolic machinery. For instance, iron absorption is facilitated by vitamin C and vitamin B2. Conversion of pyridoxine (vitamin B6) to its active form, pyridoxal phosphate requires riboflavin (vitamin B2). Conversion of vitamin A (retinol) to vitamin A aldehyde (retinal), the form in which it participates in vision, requires vitamin B (niacin). To keep folic acid in its active reduced form, tetrahydrofolate vitamins B12 and B2 are required. Riboflavin is also required for the metabolism of niacin and vitamin K. These are just a few examples.

MICRONUTRIENTS IN INDIAN DIETS

Countrywide surveys conducted by the National Nutrition Monitoring Bureau (NNMB) show that Indian diets are qualitatively adequate in proteins but deficient in some micronutrients. Thus, if caloric needs are met, protein requirements too are fulfilled, but the requirement of several micronutrients (Table 1) remain unmet. While dietary deficiencies of vitamin A and riboflavin are seen in all age, sex and physiological groups, those of other micronutrients, such as iron, calcium, thiamine (vitamin B1), niacin and vitamin C are seen in children and pregnant and lactating women (Table 1)[1]. NNMB data do not provide information on the intake of folic acid and pyridoxine. However, studies conducted at the National Institute of Nutrition many years ago, suggest that intake of these nutrients is also low, particularly in women and children (Table 2)[2,3]. Since Indian diets have not changed substantially over the years, these observations apply even today. Although the problem of vitamin B12 deficiency has not been adequately examined, it may well be prevalent since Indian diets are predominantly vegetarian.

In addition to the micronutrients mentioned above, there are many trace elements such as zinc and selenium, whose deficiency may also exist. Unfortunately, very little information is available on the trace elements,

TABLE 1
Average Nutrient Intake of Children and Adults
(As per cent of RDI)

	Protein	Energy	Calcium	Iron	Vitamin A	Thiamine	Riboflavin	Niacin	Vitamin C
Children									
1-3 yrs	94.5	62.8	61.2	71.7	35.2	66.7	51.4	60.0	48.3
7-9 yrs	90.7	71.8	92.7	67.7	34.8	82.0	46.7	70.0	44.8
Adolescents (13-15 yrs)									
Boys	72.0	81.3	71.8	58.3	48.0	87.5	47.3	78.1	94.5
Girls	74.9	91.8	65.8	78.6	37.8	101.0	58.3	87.1	75.2
Adults									
Males	108.0	87.5	144.2	109.6	48.5	100.0	73.7	90.5	101.0
Females:									
NPNL	108.0	90.6	113.7	87.3	40.5	103.6	56.9	93.6	81.2
Pregnant	65.0	75.6	48.3	35.5	63.0	89.1	53.0	80.0	91.0
Lactating	75.2	89.1	51.5	87.0	32.9	103.0	59.3	90.0	47.5

RDI – Recommended Dietary Intake
NPNL – Non-Pregnant and Non-Lactating

TABLE 2
Folate and Vitamin B6 Intake of Children and Women (As per cent of RDI)

	Folate[1]	Vitamin B6[2]
Children		
1-3 years	–	46
1-12 years	55	–
Pregnant women	38	45.5
Lactating women	33	43.3

Source : *1. Babu, S. (1976)*
2. Bapurao and Tulpule (1980)

whose importance is beginning to be realised. Thus, if the problem of micronutrient deficiency in India is to be addressed, a broader view, encompassing multiple micronutrients, must be taken. Selectively augmenting the intake of one or two will not suffice.

THE NEGLECTED PROBLEM

Clinical deficiency is the tip of the proverbial iceberg. For every case of clinical deficiency, there are many others who suffer from sub-clinical malnutrition. Sub-clinical vitamin deficiencies can be identified through appropriate biochemical measurements. Numerous studies done at the National Institute of Nutrition (NIN) show a very high incidence of riboflavin deficiency in children[4,5] and adults (as yet unpublished) as judged by the erythrocyte glutathione reductase activation test (Table 3). Several studies conducted at NIN show that more than 60 per cent of young women suffer from folate deficiency, judged by red blood cell folate levels and that over 25 per cent of women suffer from vitamin B6 deficiency as judged by erythrocyte aspartate aminotransferase activation coefficient. Deficiencies of these nutrients are greater in magnitude during pregnancy. Subjects with sub-clinical vitamin deficiencies may appear normal but suffer from subtle functional deficits, such as impaired psychomotor function and reproduction, increased susceptibility to infections, reduced capability to handle offending xenobiotics, increased susceptibility to oxidant stress and degenerative diseases, reduced synthesis of important macro-molecules, such as DNA, collagen and others. Public health workers and even scientists tend to ignore such malfunctions because they are not obvious killers or cripplers. Yet, in terms of the overall performance of the community, they may also contribute substantially to the loss of 'disability-associated life years' (DALY loss).

Deficiencies of B-complex vitamins have drawn lesser attention from public health nutritionists as compared to nutrients such as iron, vitamin A and iodine, as the former may not lead to such obviously crippling morbidities as the latter would. Yet, they are metabolically important. Riboflavin – one of the most deficient nutrients in Indian diets – along with thiamine and niacin, is a key player in the conversion of dietary energy into the energy currency of the body – ATP, by facilitating oxidation-reduction reactions. It is also required for drug metabolism and for the generation of reduced glutathione – a powerful anti-oxidant. Pyridoxine is needed for protein metabolism and for the generation of important neurotransmitters such as serotonin, dopamine and others. Folic acid, along with vitamin B12 is involved in single carbon transfer reactions and, hence, is crucial for the synthesis of the genetic material, DNA, and for cell maturation, including red blood cell formation.

Ignoring these deficiencies would imply that either nutritionists do not consider them metabolically important, or feel that present estimates of their requirements are wrong. Either assumption will be unacceptable. Some examples of the functional consequences of vitamin B deficiencies are discussed to emphasise the consequences of their deficiencies which may go beyond the obvious.

RIBOFLAVIN DEFICIENCY

The characteristic features of advanced riboflavin deficiency are orolingual (angular stomatitis, glossitis, and cheilosis), dermal (seborrhoeic dermatitis), corneal (vascularisation) and haematological manifestations; in the earlier stages, fatigue, itching and burning of the eyes, and some personality changes may also occur. The incidence of the orolingual and dermal lesions in India is high – about 5 to 10 per cent – particularly in pregnant women and in school-going children. These lesions, however, are not specific to riboflavin deficiency. Their treatment, sometimes, requires other B-complex vitamins, particularly pyridoxine, suggesting the presence of multiple deficiencies. Angular stomatitis may also occur due to fungal infection.

Respiratory infections in children lead to excessive elimination of riboflavin in urine, and this, in addition to the low dietary intake may contribute significantly to the problems. Studies in mice show that Klebsiella pneumoniac infection alters

TABLE 3
Prevalence of Biochemical Riboflavin Deficiency, as Judged by EGR-AC[1] Among Low-income Group Women and Children (Percentage Distribution)

	Number	Adequate	Low risk	Medium risk	High risk	Source
Rural children 1-5 yrs	105	21	33	21	25	Unpublished
Rural school boys 5-11 yrs	114	0	5.2	15.5	79.3	Bamji *et al*[4] 1982
Urban school children 7-11 yrs	103	2	4	9	85	Prasad *et al*[5] 1987
Rural women 15-45 yrs	105	8	21	21	50	Unpublished
Urban women 18-35 yrs	415	8	10	14	68	Bamji, Prema and Jacob (WHO) Task Force Unpublished

1– Erythrocyte glutathione reductase activation coefficient

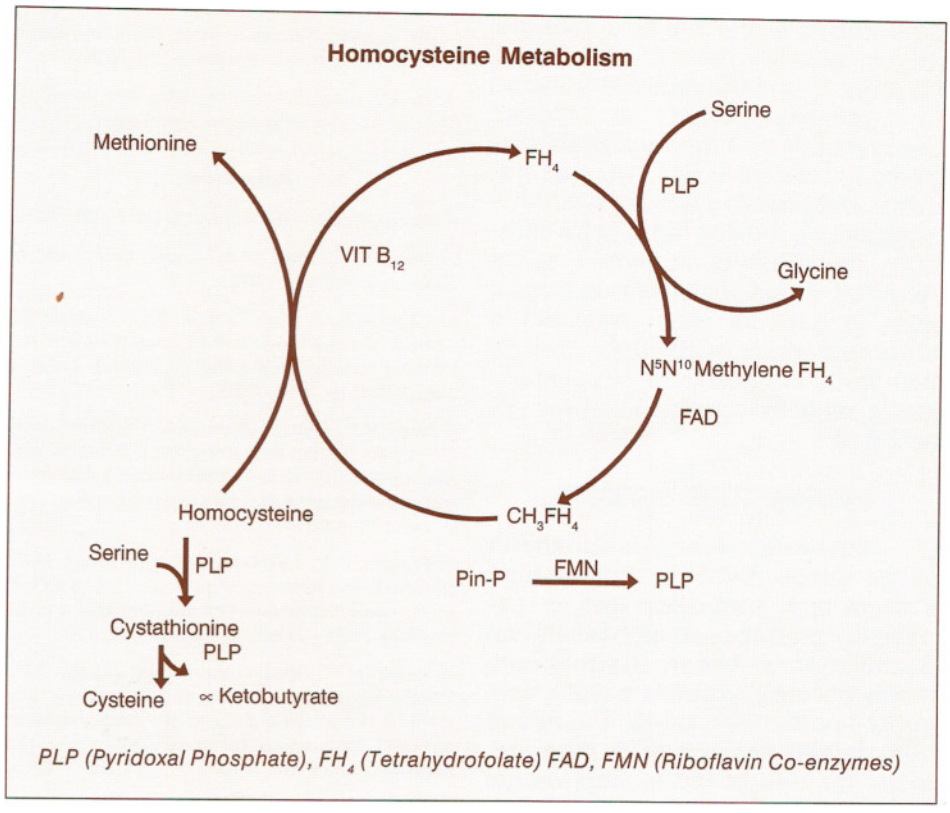
riboflavin metabolism and mobilises riboflavin from the liver, and perhaps other tissues, into the blood[7]. This may be due to the requirement of this vitamin for mounting the acute phase of defence reactions associated with phagocytosis. Separate studies in rats suggest that phagocytic activity of the white blood cells is affected in riboflavin deficiency.

Psychomotor performance tests, such as hand-steadiness, have been found to be significantly impaired in riboflavin-deficient rural and urban school children[4,8]. In experimentally-induced riboflavin deficiency American adult men lost some of the hand grip strength[9]. Impairment of such neuromotor functions may be due to the role of riboflavin in energy transduction reactions in the mitochondria. Individuals suffering from riboflavin deficiency may perform suboptimally, and to judge the impact of riboflavin deficiency, simply on the basis of the well-established clinical signs and symptoms which are not life threatening or crippling, may not be justified. The incidence of riboflavin deficiency has been found to be higher in men suffering from cataract compared to matched controls[10]. Riboflavin deficiency has been implicated in the aetiology of cataract[10]. Experimental studies in humans show that physical work increases riboflavin requirement beyond the present recommendations of 0.6 mg per 1,000 kilo calories.

Riboflavin is crucial for embryogenesis. Limb-reduction in infants, whose mothers had used thalidomide during pregnancy, was attributed to its anti-riboflavin action. Riboflavin is transported to the foetus or to the avian egg by the oestrogen-induced protein or the riboflavin-cancer protein (RCP). Genetic absence of this protein in the avian egg results in its failure to hatch. Neutralisation of this protein through active or passive immunisation has an abortifacient effect in rats and monkeys[11], suggesting the importance of this vitamin during pregnancy.

MOLECULAR BASIS OF SKIN LESIONS

Deficiencies of both these vitamins give rise to similar oral and skin lesions. The work done by the authors suggests that these lesions may be due to impaired maturity (reduced cross-linking) of the connective tissue protein, collagen[12]. This biochemical lesion would weaken the dermis and make the overlying epithelial tissue susceptible to mechanical stress and infection. The formation of epithelium of the skin also requires a mature cross-linked collagen.



PLP (Pyridoxal Phosphate), FH_4 (Tetrahydrofolate) FAD, FMN (Riboflavin Co-enzymes)

Collagen synthesis is vital to the process of wound healing. In riboflavin, as well as pyridoxine-deficient rats, wound healing has been found to be impaired. The implications of this functional defect in humans needs to be examined.

Further studies on the biochemical basis of reduced collagen cross-linking in the deficiencies of these vitamins show that the levels of the amino acid, homocysteine is elevated in the skin of rats suffering from either of these deficiencies. Homocysteine is known to impair collagen cross-linking and this may well explain the collagen defect in these deficiencies[12].

IMPLICATIONS OF HYPER-HOMOCYSTEINAEMIA

Homocysteine is generated in the body through de-methylation of methionine. Its metabolic disposal is shown in the figure above. Genetic, physiological, pathological, dietary factors (deficiency of folic acid, B12 and pyridoxine) and certain drugs are known to elevate homocysteine levels in blood[13]. Though a similar observation in vitamin B2 deficiency in humans has not been made, as mentioned earlier, significant increase in homocysteine was observed in the skin of riboflavin-deficient rats.

Many subjects with hyper-homocysteinaemia (normal range 7-17 μmol/L), and even homocysteineurea (a genetic disorder due to the deficiency of the metabolising enzymes) respond to treatment with pyridoxine, folic acid and vitamin B12. In recent years, many retrospective, as well as prospective studies have shown moderate hyper-homocysteinaemia to be an independent risk factor for occlusive arterial disease[13]. The mechanism involved is complex and not fully understood for want of a suitable animal model. Hydrogen peroxide generated through the oxidation of homocysteine may damage endothelial cells and impair its function[14]. Excess homocysteine may also alter the activity of coagulation factors.

Information on plasma homocysteine levels in Indians is not available. However, recent unpublished observations of the authors in a few subjects with coronary heart disease and matched controls show higher levels of homocysteine in both the groups (range: 7-74.5 μmols/l). Considering the widely prevalent

deficiencies of the vitamins involved in homocysteine metabolism (see figure on page 7) and the fact that Indians, in general, have a higher susceptibility to coronary heart diseases and to syndrome X, hyper-homocysteinaemia and coronary heart disease among Indians need to be carefully investigated. A recent study reported regression in carotid plague area in patients with unexplained atherosclerosis with high plasma homocysteine levels after supplementation with folic acid, vitamins B6 and B12[15].

NEURAL TUBE DEFECTS

Folic acid deficiency is also shown to be associated with neural tube defects and pregnancy loss in genetically predisposed individuals. An association between thermolabile methylenetetrahydrofolate reductase, hyper-homocysteinaemia and neural tube defects has also been reported. India has one of the highest neural tube defects – 5 in every 1,000 births[16]. To prevent this disorder, the folic acid status has to be built prior to pregnancy, since neural tube fusion occurs within two weeks of conception.

The ICMR recommendation of RDA for folic acid is only 100 μg for men and non-pregnant and non-lactating women. This level is much lower than the international recommendation of 170-200 μg. If higher RDA is used, many more Indians will fall under the deficient category. Also, the requirement of pyridoxine for Indians needs to be reinvestigated, in the light of recent data in plant foods which shows that they contain almost 50 per cent of pyridoxine that occurs in glycosylated form and the availability of this form is poor. While the administration of iron folic acid supplements during pregnancy is absolutely essential, the diet alone may not be able to meet the elevated requirement of these nutrients. An all out effort to augment their consumption in adequate amounts and thus derive a balance of nutrients should be the strategy. To achieve this, a coordinated effort on the part of agricultural scientists, nutritionists and planners is required. In the USA, fortification of foods with folic acid has been made mandatory, UK may do the same. But food fortification is not an easy strategy in a country such as India. We must ensure the adequacy of these essential nutrients in the daily diet.

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FOUNDATION NEWS

● Study Circle Lectures

Prof Indira Chakroborty, former Professor of Nutrition, All India Institute of Hygiene and Public Health, Calcutta on 'Street Foods' on January 21, 1998.

Dr Ramesh Bhat, Deputy Director, National Institute of Nutrition (NIN), Hyderabad on 'Fungal Toxins in Food: Recent Studies' on February 24, 1998.

Dr Gill Hardy, Director, Oxford Nutrition UK on 'Advances in Immuno Nutrition' on March 20, 1998.

● Task Force Meeting

A Task Force Meeting on micronutrient research projects, undertaken by NFI, was held on February 27.

● Annual General Meeting

The Annual General Meeting of the Foundation will be held on March 24.

● Two research foundations, with objectives and programmes closely related to NFI, which have been accommodated in the NFI building are:

Flourosis Research and Rural Development Foundation has been headed by Dr A.K. Susheela for several years and is reputed to be an important international centre for flourosis research. Dr Susheela herself is a leading scientist in this field.

The centre has been carrying out work on the problem of flourosis in India, addressing the problem of excess of fluoride in water as a part of an overall effort for ensuring quality of drinking water in the country.

Centre for Research on Nutrition Support Systems (CRNSS): While NFI's programmes have largely been community-oriented, the objective of this centre is to address problems related to clinical nutrition in the hospital setting. Dr Sarath Gopalan is the Honorary Executive Director.

● Fund Raising

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