NFI BULLETIN
Bulletin of the Nutrition Foundation of India
Volume 28 Number 3 July 2007

Bioavailability of Dietary Iron and Iron Deficiency Anaemia

B.S. Narasinga Rao

Iron deficiency anaemia (IDA) is a major nutritional deficiency disorder in India and other developing countries[1]. The criteria suggested by WHO[2] are generally used to define anaemia among men and women in different age groups (Table 1). These values, though considered arbitrary, presumably correspond to the Mean $\pm$ 2SD levels of haemoglobin distribution in a normal population in the USA, the coefficient of variation being 10 percent of normal haemoglobin distribution. When WHO cut-off point is used, there is considerable overlap between the distribution of haemoglobin among the normal distribution and those who are anaemic. In a study of women on oral iron therapy,[3] using WHO criteria for defining anaemia, 17 percent of women responded to therapy though they were classified as normal, while 35 percent of women classified as anaemic failed to show any change in haemoglobin levels despite treatment. This observation suggests that there is a need for caution in interpreting the observations on anaemia and its treatment among our population.

Large population surveys in rural India indicate that the prevalence of anaemia according to WHO criteria (Table 1) ranges from 42 to 77 percent among different age groups (Table 2). It will be seen that prevalence is higher among children than adults, higher among girls and women than boys and men, respectively. Besides age and sex, prevalence also depends upon the region of the country. Hookworm disease, malaria and other infections, if present, further aggravate anaemia. The most vulnerable groups are preschool children, women and pregnant women among whom prevalence of anaemia may exceed 70 percent.

Dietary Basis of IDA in India

Iron deficiency anaemia in India is essentially due to low dietary iron intake and its poor bioavailability. Dietary iron is contributed mostly by the cereal component of the Indian dietaries. Further, part of the dietary iron derived mainly from grains is contributed by contaminant iron, which is not absorbed. This constitutes about 30 percent of iron intake of various regional groups[4]. The per cent frequency distribution of iron intake (Table 3) of different age groups shows that 67-86 percent of individuals consume less than 50 percent of the Recommended Dietary Allowance (RDA) emphasising the widespread inadequacy of dietary iron intake. The dietary iron intake of different age groups corrected for contaminant iron is given in Table 4.

From National Nutrition Monitoring Bureau (NNMB) data[5], it would appear that the relationship between iron intake (y) and energy intake (x) is related by the following linear relationship: $y = 0.1728 x + 2.0654$ (Figure 1). This is obvious since most of the dietary iron and energy are derived from a common dietary source, i.e., cereals.

Bioavailability of Dietary Iron

Apart from inadequate dietary iron intake, poor bioavailability of dietary iron from foods like cereals, legumes and other vegetable foods which constitute a majority of Indian diets appear to be a major factor leading to iron deficiency among low income Indians[6]. In contrast, iron absorption from diets based on meat and other animal products carrying iron as haem iron have much better bioavailability and can meet the iron requirement[6].

Physiology of Dietary Iron Absorption

Dietary iron absorption from habitual Indian diets in different physiological groups worked out at the National Institute of Nutrition (NIN) is given in Table 5. Vegetable iron to be absorbed from ileum of the small intestine, has to be in an ionisable and a soluble form at the pH 7.0 of the duodenum. Normally, inorganic ferric iron gets precipitated at pH 7.0 or when it is bound to binding agents[7]. On the other hand, ferrous iron, which is soluble at pH 7.0, gets better absorbed. Ascorbic acid or other reducing agents like some reducing sugars, which keep iron in ferrous, and a soluble form at pH 7.0 promote its

CONTENTS

Table 1: WHO criteria for haemoglobin levels indicative of anaemia

Group	Haemoglobin level (g/dl)
Children 6 months-5 years	< 11
Children 6-14 years	< 12
Adult men	< 13
Adult women	
Non-pregnant	< 12
Pregnant	< 11

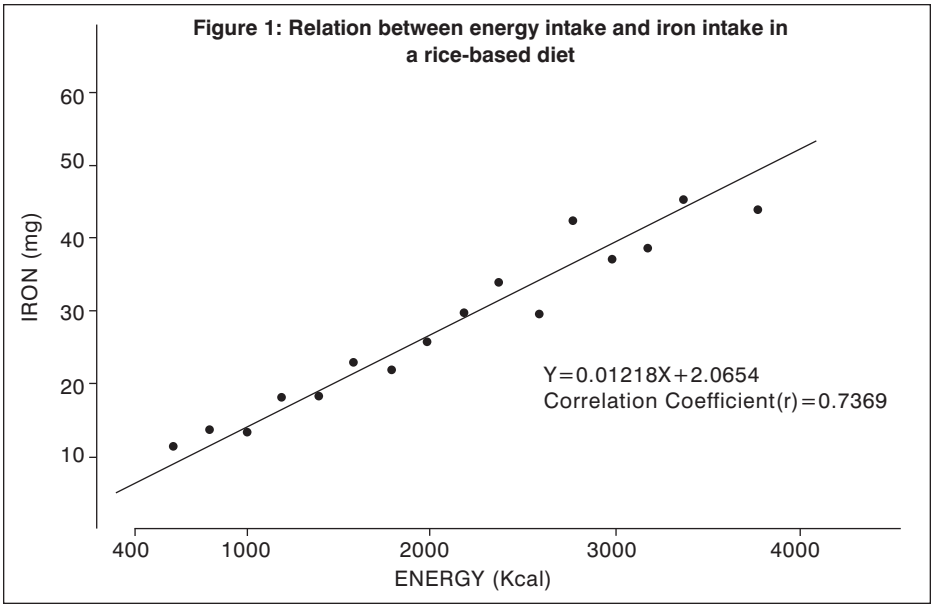

Figure 1: Relation between energy intake and iron intake in a rice-based diet

absorption. On the other hand, in the presence of some favourable chelating agents, even ferric iron can be kept in a soluble form at the pH 7.0 of the duodenum and can be better absorbed. The first step in the intestinal absorption of iron is its uptake by the mucosal brush border receptors. For this, iron should be in a soluble form, which can be ensured by iron binding ligands[7]. The uptake of iron from the intestine by the brush border receptor depends upon the relative affinity of the brush border receptors and iron-binding agents, which keep ferric iron in a soluble form at the intestinal pH of 7.0. Foods also contain orthophosphates, carbonates, phytates, tannic acid, polyphenols all of which form insoluble complexes with iron at pH 7.0 and inhibit its absorption.

Phytates and Tannates in Foods Act as Inhibitors of Bioavailability of Dietary Iron

Phytates are considered to be important binding agents inhibiting iron absorption. But the actual fact is that the phytates by themselves form soluble complexes with iron and can promote its absorption[8], but in the presence of dominating divalent cations like Ca, Mg, Zn, etc., which dominate in foods, phytates form insoluble complexes with iron. On the other hand, polyphenols (tannins) are more powerful inhibitors of bioavailability of dietary iron[9] by forming insoluble complexes with iron. The bioavailability of dietary iron in the foregoing studies was measured by an in vitro method developed by us[10]. This in vitro prediction method highly correlated with iron absorption determined on the same diets by an in vivo extrinsic tag method (correlation coefficient 0.94) and the relationship between percent ionisable iron at pH 7.5 (y) and in vivo absorption (x) on the diets studied was: $y = 0.4827 + 0.4707\ x$ (Table 6). The chemical balance method used yielded two or three times the values obtained from the extrinsic tag method (Table 7).

The first step in the transport of iron across the mucosal cell is the uptake of iron by the brush border receptors[11]. For this uptake, iron must be in a soluble form at pH (7.0) of the deodenum. The amount of iron transferred from the gut lumen to mucosa depends upon the abundance of receptors on the brush border. The receptor population increases in iron deficiency, the increase being more on the distal than the proximal part. The changes are not immediate but occur after one or more days, that is when a cohort of new cells is formed[12].

The acceptor sites on the brush border surface compete for iron with ligand in the gut lumen. Some ligands mainly derived from food promote iron absorption by keeping it in solution at pH 7.0 and others inhibit absorption by precipitating iron. The net result depends upon the balance between the two opposing forces.

Iron once taken up by mucosal cells is bound to specific carriers and is rapidly transferred to the serosal side. The absorbed iron is bound to plasma transferrin. In the mucosal cells, the excess iron is taken up by appoferritin and stored as ferritin.

Despite years of research, the precise molecular mechanisms involved in the uptake of iron into mucosal cells and its transfer to the plasma still remains uncertain. The factor regulating iron absorption remained obscure but there is overwhelming evidence that the rate of iron absorption is closely regulated by the size of the iron stores[12]. In health, this is the only important controlling factor yet identified and there is close inverse controlling point.

Absorbed iron carried in plasma by transferrin is taken up by the transferrin receptor expressed on the surface

Table 2: Prevalence of anaemia according to severity among Indians in rural India

Age (years)	Sex	Percent prevalence of anaemia		
		As per WHO criteria	Moderate Hb < 10 g/dl	Severe Hb < 7 g/dl
1-6	Boys	75.5	56.3	10.9
	Girls	77.1	57.5	7.6
6-15	Boys	56.8	15.8	1.6
	Girls	68.8	18.3	2.4
≥15	Men	42.0	5.1	0.5
	Women	64.0	18.7	1.5
Average	All	58.1	20.7	2.6

Table 3: Percent frequency distribution of intake in individuals of different ages

Percent Recommended Daily Allowance	Preschool Children 1-6 years	Girls 13-15 years	Girls 16-18 years	NPNL women	Pregnant women
< 10	38.5	35.6	36.0	36.1	53.7
30 – 40	17.6	18.9	23.0	20.2	22.0
40 – 50	11.6	14.3	10.8	14.4	9.8
< 50	67.7	68.8	69.8	70.7	85.8
50 – 60	8.7	9.7	8.6	9,7	7.3
60 – 70	6.3	5.5	7.8	7.0	0.0
70 – 80	4.8	4.8	4.7	3.6	1.2
80 – 90	3.8	3.2	3.3	2.7	1.2
90 – 100	93.4	99.2	99.2	95.2	97.6
≥ 100	6.8	6.2	4.7	4.8	2.

Revised values, National Nutrition Monitoring Bureau Special Report No.20, National Institute of Nutrition, Hyderabad, 2000.

NPNL: Non-pregnant and non-lactating.

Table 4: Dietary iron intake by Indians and its adequacy

Age (years)	Gender	Recommended Daily Allowances for Iron (mg/day)	Average dietary iron intake (mg/day)	Adequacy %
1 – 3	All	12	7.7	64
4 – 7	All	18	11.0	61
Adult	Men	28	22.5	89
Women		30	19.7	63
Pregnant women		38	17.1	45
Lactating women		30	23.7	79

Corrected for contaminant iron 30%.

Table 5: Dietary iron absorption % from habitual Indian diets in different physiological groups

Physiological Groups	Mean dietary iron absorption % from Rice-based diet	Mixed cereal diet	Wheat/Millet diet
Adult males	5.0	3.0	2.0
Adult females	8.0	5.0	3.3
Children			
Adolescent males	5.0	3.0	2.0
Adolescent females	8.3	5.0	3.3
10-18 years			
Post-menopause females	5.0	3.0	2.0
Pregnant women	13.3	8.0	5.3
Anaemic males	10.0	6.0	4.0
Anaemic females	16.7	10.0	6.7

of the cells in proportion to their iron requirements. Once bound to the cell, the transferrin iron complex is internalised by the cell and the iron is released from the transferrin. Released iron is either utilised or stored in the form of ferritin.

Body Loss of Iron

In contrast to the dynamics of transfer of iron between body compartments, exchange with external environment is minimal. No adjustable excretory mechanism exists, but a small obligatory loss occurs with physiological turnover of skin epithelium and the cells of gastrointestinal and urinary tracts. Low concentration of iron is also present in sweat, bile and urine. A small quantity of blood is present in faeces; menstrual loss accounts for a significant proportion of iron lost by women of childbearing age.

In normal men, loss of iron from the body has been reported to be 1 mg/day[13]. But in a tropical country like India, dermal loss of iron may be an important source of iron loss. Hussain et al[10] and later Apte and Venkatachalam[14] and Apte[15] carried out several studies to assess the dermal loss of iron in Indians. They reported dermal loss of iron in human volunteers varying from 0.58 to 3.22 mg/day. On the basis of these results, it was suggested that non-haemoglobin loss of iron in Indians is probably in the range of 0.8-2.6 mg/day with an average of 1.7 mg/day, a much higher figure than that reported from West[13].

Assuming an intra-individual variance of 20 percent, an allowance of 2.0 mg of absorbed iron was suggested by the Nutrition Expert Group on RDA[16] in 1968, which in terms of dietary iron will be 20.0 mg/day. To this figure, the menstrual loss of iron in Indian women, which was reported to be another 1.0 mg – the upper limit of loss per day should be added[17]. Thus, total body loss was assessed in a menstruating Indian woman as 3.0 mg of absorbed iron or a total of 30 mg dietary iron/day.

These recommended values were re-examined by the Indian Council of Medical Research (ICMR) Expert Group on RDA[18] in the light of other reports based on turnover studies using radio iron and considered that the Indian figures on sweat loss of iron based on chemical methods as

Table 6: Correlation between per cent ionisable iron and per cent in vivo absorption

Diet	Total iron mg	Ionizable at pH 7.5 Mean (X)	% iron absorption (Y)	Computed from equation
Rice diet (lunch)	8.2	5.9	3.6 (6)	3.3
Wheat diet (lunch)	10.5	4.3	2.2 (5)	2.5
Rice diet	6.0	6.7	3.3 (6)	3.6
Wheat diet (breakfast)	7.3	4.4	2.1 (17)	2.5
Ragi diet	9.2	2.1	1.6 (9)	1.5
Sorghum diet	11.0	2.1	1.7 (4)	1.5
Correlation with in *vivo* % absorption			0.94	

The relationship between % ionisable at pH 7.5 (X) and in vivo percent absorption (Y), Y = 0.4827 + 0.4707 X.

Figures in parentheses indicate the number of subjects studied.

over estimates. These Indian figures on sweat loss of iron were determined by collecting sweat through thermal stimulation, for a short period of time, estimating iron by the chemical method and making a projection for whole body iron loss for 24 hours. On the other hand, other reported studies based on turnover methods using radio iron indicated that the iron losses in subjects in America are not different from either Indian workers in hot and humid climates in South Africa or labourers in Venzulea[12]. Sweat, urine and gastrointestinal tract loss of iron for computing iron requirement was suggested as 14 mg/kg body weight. The same figure was adopted by the 1990 ICMR Expert Group on RDA[19] also. Menstrual losses, however, were retained as 1 mg/day in women during the reproductive age.

However, in view of high prevalence of anaemia in India, Foy and Kondi[20] suspected that body loss of iron may be one source contributing to the high rate of anaemia. Therefore, there is a need to reinvestigate the body loss of iron with turnover studies using isotopic iron among Indians.

Unfavourable Body Stores of Iron among Indians and its Consequences

Good storage iron in a normal adult man, which is about 1000 mg, can meet any sudden increase in iron requirement during pregnancy or sudden blood loss. The body can mobilise 40 mg/day iron to meet these emergency requirements. But an anaemic individual with iron deficiency lacks these iron stores. Even non-anaemic individuals may have normal haemoglobin but may lack iron stores. Such individuals have to depend upon absorbed iron which can provide only 2-4 mg a day and red cell production can increase only by a small margin. Thus, loss of iron from major functional compartments is rapidly corrected when iron stores are adequate, and slowly replenished by absorption, even if bioavailability of iron is satisfactory. The situation is much worse if bioavailability of iron is poor as in the case of Indian diets, which are largely based on plant foods.

Tissue Depletion of Iron and its Consequence

Anaemia, when present, is indicated by a reduction in haemoglobin. However, tissue iron content, which is stored as ferritin, is also concurrently reduced. The physiological consequence of this is related to impaired oxygen delivery and other abnormalities. The functional deficiencies are mucosal and epithelial abnormalities like angular stomatitis, glossitis, impaired immune function and susceptibilities to infection. Skeletal muscle dysfunction resulting in reduced work capacity, behavioural and neurological abnormalities have also been reported in anaemic persons. These abnormalities are related to severity of tissue iron deficiency and to severity of anaemia. Anaemic persons have certainly tissue iron deficiency but those with normal haemoglobin may also have various degrees of tissue iron deficiencies and suffer from associated consequences.

Micronutrient Deficiency with Anaemia and their Consequences

The main micronutrient whose deficiency leads to anaemia in India and other developing countries is iron. Anaemia is also reported to be associated with deficiencies of other micronutrients such as folate and vitamin B_{12}. In India, prevalence of anaemia is high in low-income groups in rural areas and urban slums. Dietary intake of these anaemic populations is deficient in several other micronutrients besides iron and energy[21] (Table 8). Those belonging to vulnerable groups (pre-school children and women), besides iron deficiency, suffer from other nutritional deficiencies (Table 8). Pre-school children suffer from chronic energy deficiency, vitamin A and B-complex vitamins deficiency, particularly riboflavin, thiamine and perhaps pyridoxine, ascorbic acid and zinc besides iron. Non-pregnant non-lactating women suffer from energy and micronutrient deficiencies like vitamin A, riboflavin, besides iron.

The main micronutrients whose deficiencies are known to lead to anaemia are iron, folate and vitamin B_{12}. All the three are directly related to the development of anaemia in the synthesis of erythrocytes and their

Table 7: Iron absorption from Indian diets determined by extrinsic tag and chemical balance method – a comparison

Diet	No. of subjects	Iron intake mg/d	Iron absorption	
			Extrinsic tag technique	Chemical balance method
Rice-based diet with milk	4	30.0	3.6 $\pm$ 0.14	9.2 $\pm$ 1.94
Mixed cereal based diet with milk	4	42.0	3.5 $\pm$ 0.27	8.7 $\pm$ 2.82
Rice-wheat-meat diet	4	35.5	2.6 $\pm$ 0.39	6.0 $\pm$ 0.12
Rice-wheat-fish diet	4	28.8	2.7 $\pm$ 0.13	5.8 $\pm$ 0.88

Absorption figure indicate Mean $\pm$ SE.

Table 8: Dietary intake of nutrients and their adequacy in preschool children (1-3 years) and non-pregnant and non-lactating (NPNL) women of low income groups

Nutrient	Preschool children (1-3 years)		NPNL women	
	Mean intake	As % of RDA	Mean intake	As % of RDA
Energy (Kcal)	807	65	2106	73
Protein (g)	20.9	91	53.8	108
Fat (g)	12.9	37	29.7	100
Calcium (mg)	239	48	509	127
Iron (mg)	8.7	72.5	25.1	84
Retinol (μg)	133	33	295	49
Thiamine (mg)	0.3	50	1.2	114
Riboflavin (mg)	0.4	57	0.9	72
Niacin (mg)	5	62.5	13	94
Ascorbic acid (mg)	15	38	40	100
Folic acid (μg)	57.5			
Zinc (mg)	4.3	54	15.4	100

maturation. Lack of folate and vitamin B_{12} leads to megaloblastic anaemia. Deficiencies of other micronutrients like vitamin A, riboflavin, pyridoxine and zinc, and cobalt are also reported to be related to development of anaemia. Vitamin A deficiency has also been shown to result in a fall in haemoglobin levels in adult men volunteers[22]. This was reversible with supplementation of vitamin A and iron but not with vitamin A alone. While the precise mechanism remains to be elucidated, some of the suggested mechanisms include impaired mobilisation of iron stores, possibly due to an effect of vitamin A deficiency on the transferrin receptors. Supplementation of vitamin A deficient individuals with vitamin A alone increases the haemoglobin concentration by about 1 g/dl[23]. In several studies, the addition of vitamin A alone has been shown to improve response to iron supplements[24]. Night blindness among pregnant and lactating women of South Asian countries was attributed to severe anaemia as well as vitamin A deficiency[25].

Riboflavin deficiency may exacerbate iron deficiency by increasing intestinal loss of iron, reducing iron absorption, impairing mobilisation of intracellular iron and increasing crypt proliferation. Riboflavin deficiency may impair the synthesis of globin and reduce the activity of NADH-FMN oxidoreductase so that iron becomes trapped in ferritin. Supplementation of riboflavin-deficient individuals with riboflavin caused an increase in haemoglobin level and an increase in haematological response to iron supplements in iron deficient Gambian men and lactating women[26,27]. Similarly, supplementation of both iron and riboflavin (a supplement containing both) increased serum ferritin levels of anaemic lactating women more than iron supplements alone[28] although the haemoglobin level did not improve significantly. A study in Europe reported an improvement in haemoglobin level when riboflavin was added to iron supplements and given to anaemic pregnant women[29] as did a study of children in Thailand[30]. Based on rat studies[31], it is reported that zinc reduces iron absorption by competing for the carrier involved. It is not clear to what extent this operates in human subjects. In rats, zinc supplementation reduced iron status (serum ferritin and haematocrit values), supplementation of both zinc and iron restored it. This needs to be tested in humans in view of the current interest to supplement children with zinc in view of reported zinc deficiency among them and these children will be iron deficient also.

The interaction between iron deficiency and other micronutrient deficiencies can work in a different way. The functional consequences of iron deficiency anaemia such as impaired learning ability and immune competence are also seen in deficiencies of iodine, vitamin A and protein energy malnutrition (PEM). Therefore, when these deficiencies coexist with anaemia, these functional parameters will respond better to correction of all deficiencies than to correction of iron alone.

The interaction between iron and micronutrients can also exist at tissue level. Structural changes occur in the skin, mucosal membrane and gastrointestinal tract as a result of iron deficiency as well as of other micronutrients. For example, angular stomatitis, glossitis, gastrointestinal changes and koilonychias are manifestation of B-deficiency (pyridoxine) as well as of zinc. These changes respond to iron therapy as well as to vitamin therapy. Pyridoxine or zinc deficiency may be the contributing factors in the development of pathological changes in severe iron deficiency[32,33]; cobalt is known to have a non-specific effect in raising the haemoglobin level of those on iron therapy. Protein-energy malnutrition is reported to regulate erythropoiesis[34].

In light of the interaction between micronutrient deficiencies like vitamin A, riboflavin, pyridoxine and energy deficiency with anaemia, there is a need to investigate whether the prevalence of these micronutrient deficiencies along with iron deficiency exacerbates anaemia among low income group Indians.

●●●

Diets of poor are deficient in a wide range of nutrients, both macro and micro. The problem cannot be solved by the administration of a cocktail of few synthetic nutrients, which are now made available by the pharmaceutical firms. There are several scores of phytonutrients, which are present in plant foods (especially green leafy vegetables) and which are now known to play an important part in health promotion and disease prevention. The rational approach in the management of anaemias, apart from iron and folate administration, would be to promote balanced diets containing not only wheat and rice but also pulses and legumes, fruits and vegetables and milk.

The author is Former Director, National Institute of Nutrition, Hyderabad.

References

1. WHO. Technical Report Series 580. Control of Nutritional Anaemia with Special Reference to Iron Deficiency. Report of an IAEA/USAID/WHO Joint Meeting. 1975.

2. *Nutritional Anaemia*. Report of a WHO Scientific Group. World Health Organ. Tech. Rep. Ser. 405, 5-37. 1968.

3. Lynch SR, Anaemia, Iron deficiency Anaemia. In: Sadler MJ (ed). *Encyclopaedia of human nutrition.* New York. Academy Press. 81-84.2000.

4. Prabhavati T, Narasinga Rao BS. Contaminant iron in foods and its bioavailability predicted by the in vitro method. *Ind. J. Med. Res*. 74, 37-41.1981.

5. Narasinga Rao BS(1983). Bioavailability of dietary iron. *Proc. Nutr. Soc.* Ind. 28: 1-6.

6. Narasinga Rao BS and Subba Rao K. Studies on the iron binding ligands and the intestinal brush border receptors in iron absorption. *Ind. J. Biochem. & Biophys*. 29: 214-218.1992.

7. Subba Rao K and Narasinga Rao BS. Studies on iron chelation by phytate and the influence of other mineral ions on it. *Nut. Rep. Int.* 28 : 771-781.1983.

8. Bagepalli S Narasinga Rao and Tatineni Prabhavati. Tannin content of foods commonly consumed in India and its influence on ionisable iron. *J. Sci. Food Agric*. 33 : 89-96.1982.

9. Narasinga Rao BS and T. Prabhavathi. An in vitro method for predicting the bioavailability of iron from foods. *Amer. J. Clin. Nutr.* 31 : 169-175.1978.

10. Greenberger NJ, Balcerzak SP and Ackerman GA. Iron uptake by isolated brush borders changes induced by alterations in iron stores. *J. Lab. Clin. Med.* 73 : 711-721.1969.

11. Green R, Charlton RW, Seftel H, Bothwell T, Mayers F, Adams B, Funich C and Layrisse M. Body excretion in man. A collaborative study. *Amer. J. Med.* 45 : 336-353.1968.

12. Hussain R, Patwardhan VN, Sriramachari SS. Dermal loss of iron in healthy men. *Ind. J. Med. Res.* 48 : 235-242.1961.

13. Apte SV and Venkatachalam PS. Factors influencing dermal loss of iron in human volunteers. *Ind. J. Med. Res*. 50: 817-822.1962.

14. Apte SV. Dermal loss of iron in Indian adults. *Ind. J. Med. Res*. 51: 1101-1104.1963.

15. Gopalan C and Narasinga Rao BS. *Dietary Allowances for Indians.* Special Report Series No.60. Indian Council of Medical Research.1968.

16. Apte SV and Venkatachalam PS (1963). Iron losses in Indian women. *Ind. J. Med. Res*. 51 : 958.1963.

17. Indian Council of Medical Research: Recommended Dietary Intakes for Indians. Report of an Expert Group, 1980.

18. Indian Council of Medical Research. Nutrient Requirements and Recommended Dietary Allowances for Indians: A Report of the Expert Group of the Indian Council of Medical Research, 1990.

19. Foy H and Kondi A. Anaemias: Anaemias of the tropics. Relation to intake, absorption and losses during growth, pregnancy and lactation. *J. Trop. Med. & Hyg*. 60: 105-118.1957.

20. National Institute of Nutrition: Report of the second repeat survey 1996-1997, Hyderabad. National Nutrition Monitoring Bureau, India, 1999.

21. Hodges RE, Sauberlich HE, Canhaun JE, Wallace DL, Rucker RB, Mejia LA et al. Haematopoietic studies in vitamin A deficiency. *Amer. J. Clin. Nutr.* 31 : 876-885.1973.

22. Sommer A, West KP Jr. *Vitamin A deficiency: Health Survival and Vision*. Oxford, Oxford University Press, 1996.

23. Mejia L, Chew F. Haematological effect of supplementing anaemic children with vitamin A alone and in combination with iron. *Amer. J. Clin. Nutr.* 48 : 595-600.1988.

24. Suharno A, West CE, Muhilal, Karyodi D, Haut Vast JGAJ. Supplementation with vitamin A for iron nutritional anaemia in pregnant women in West Java, Indonesia. *Lancet*, 342: 1325-8.1993.

25. Fairweather-Tait SJ, Powers HJ, Mineski MJ, Whitehead J, Downes R. Riboflavin deficiency and iron absorption in adult Gambian men. *Ann. Nutr. Metab*. 36 : 34-40.1992.

26. Powers HJ, Bates CJ, Prentice CM,, Lamb WH, Jepson M, Bowman H. The relative effectiveness of iron and iron with riboflavin in correcting microcytic anaemia in children in rural Gambia. *Hum. Nutr. Clin. Nutr.* 37, 413-25.1983.

27. Powers HJ, Bates CJ, Lamb WH. Haematological response to supplement of iron and riboflavin in pregnant and lactating women in rural Gambia. *Hum. Nutr. Clin. Nutr.* 39: 117-29.1985.

28. Decker K, Dotis B, Glatzle D, Hinslemann M. Riboflavin status and anaemia in pregnant women. *Nutr. Met.* 21 (Suppl.1), 17-19.1977.

29. Buzina R, Jusic M, Milnovic N, Sapunar J, Brubancher G (1979). The effect of riboflavin administration on iron metabolism parameters in a school going population. *Int. J. Vit. Nutr. Res*. 49 : 136-43.1979.

30. Yadrick MK, Kenney MA, Winterfeldt EA. Iron, copper and zinc status : Response to supplementation with zinc or zinc and iron in adult female. *Amer. J. Clin. Nutr.* 49 : 145-50.1989.

31. Jacobs A, Cavill I. The oral lesions of iron deficiency anaemia: Pyridoxine and riboflavins tatus. *Brit. J. Haematol.* 14: 291-5.1968.

32. Bothwell TH, Charlton RW, Cook JD, Fuich CA. *Iron metabolism in man*. Oxford Black Well Scientific Publciation.1979.

33. Finch CA. Erythropoiesis in protein-calorie malnutrition. In: Olesur RE (Ed.). *Protein Calorie Malnutrition*. (The Nutrition Foundation), New York, Academic Press, 280-9.1976.

NUTRITION NEWS

- The 39th Annual Meeting of Nutrition Society of India will be held at National Institute of Nutrition, Hyderabad on November 15-17, 2007.
- The Xth Asian Congress of Nutrition will be held at the International Convention Centre, Taipei, Taiwan on September 9-13, 2007. The details of the Congress can be accessed from the website: http://www.2007acn.org.tw. The Message from Dr C. Gopalan (President FANS and Founder President Asian Nutrition Congress series) to X ACN is given in the next column.

MESSAGE

Greetings to the organisers and delegates of the 10th Asian Congress of Nutrition, which I am sure, will be a grand success.

The Asian Congress of Nutrition, which was started in Hyderabad, India, has continued to flourish and grow in strength. It is indeed heartening that the small seedling planted in Hyderabad in 1971 has now blossomed into a sturdy tree.

The Asian Congress of Nutrition was set up to provide a Forum for Asian nutrition scientists and for fostering their solidarity; and to provide them an opportunity for exchange of ideas with respect to promotion of nutritional well-being of Asian people. Right from the very start we had kept our doors open for participation of outstanding non-Asian nutrition scientists. This policy continues; the planning and organisation of each congress will, of course, be in Asian hands.

Today nutrition scientists of Asia face a 'double burden' – with poverty-related nutritional problems in children, at one end of the age spectrum, and, nutrition-related chronic degenerative diseases at the other end. These are formidable challenges. The challenges, which Asian nutrition scientists will have to face in future, could be even more formidable.

Current reports on global warming hold grim threats of coastal catastrophies and marine crises; of diminishing food grain production; of glacial meltdown resulting in flooding, subsequently followed by drying of rivers and increasing susceptibility to mosquito borne diseases and a significant loss of biodiversity. These threats are not all 'distant'; some of them could even materialise in the course of the century. Though Asia's contribution to global warming is insignificant as compared to that of North America or Europe, Asian countries are likely to bear the brunt of the deleterious effects of global warming. Nutrition scientists of Asia and the Asian Congress of Nutrition will therefore have to play even a more significant role in future.

May I wish the Congress all success!

C. Gopalan