

RECOMMENDED DIETARY ALLOWANCES AND ESTIMATED AVERAGE REQUIREMENTS NUTRIENT REQUIREMENTS FOR INDIANS - 2020

**A Report of the Expert Group
Indian Council of Medical Research
National Institute of Nutrition**



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MESSAGE

Developing the Nutrient Requirements for Indians has been one of the stellar contributions of the ICMR-National Institute of Nutrition (ICMR-NIN). This herculean feat has been achieved by NIN with great aplomb and scientific acumen, not once but time and again, but this latest book is an exception in ways more than one.

The changing dietary patterns prompted by a host of factors including agricultural, economic, lifestyle, health and nutrition transitions warrant that these recommendations be revisited from time to time. And newer scientific evidence and population based data become the bedrock of such revisions. It is therefore obvious that the Recommended Dietary Allowances (RDAs) become the guiding lights for evolving newer and relevant strategies for tackling the scourge of undernutrition, overweight/obesity and associated non-communicable diseases (NCDs) as well as the micronutrient deficiencies across the country.

From the earliest attempts to define the nutrient requirements and desirable dietary intakes of nutrients for Indians in the 1940s to the most recent version published in 2011, the RDAs have been revisited, revised and reformulated several times, almost once every decade. For every revision, newer research, analysis methods and data have been the underpinnings. I have no hesitation in saying that the latest version is quite a landmark that will be etched in the history of NIN and ICMR. I don't say this lightly, as this version being brought out in 2020 for the first time includes the Estimated Average Requirements (EAR) and also the Tolerable Upper Limits (TUL) of nutrients along with RDAs. While RDAs are daily dietary nutrient intake levels which would be sufficient to meet the nutrient requirements of nearly all healthy individuals in a particular life stage and gender group, they are not always robust measures for dietary assessment of groups because they refer to an intake level that far exceeds the requirement of a large proportion of individuals within the group.

On the other hand, EARs are the average daily nutrient intake levels estimated to meet the requirements of half of the healthy individuals in a particular life stage and gender group. These are very useful in evaluating the nutritional status of populations or groups. This book therefore aptly alludes to both RDAs and EARs in its title. The TULs, I am sure would be of great help in defining the limits for fortification of foods with nutrients.

Hearty congratulations to each and every scientist of ICMR-NIN, academics and researchers from different parts of the country who contributed to this effort. My sincere commendations to the Expert Committee led by late Prof. M. K. Bhan and Prof. Anura. V. Kurpad for their guidance and contributions. My appreciations to Dr. Hemalatha R, the Director of NIN for leading this effort from the forefront and completing it successfully.

I am sure this will turn out to be a guiding light for making India nutritionally *Atmanirbhar* (self-reliant).



(Balram Bhargava)



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MESSAGE

The present revision of Recommended Dietary Allowances (RDA) is based on the available recent data and technologies. It is well timed keeping in view the economic and epidemiologic transition that India is going through, which necessitates new norms of nutrient requirements for shaping the health of not only present but also future generations of India. In the last decade, there have been advancements in research concomitant with the nutrition transition prompting a paradigm shift in addressing the nutrition health issues confronting us. Considering the available evidences, based on a careful review of the literature, keeping reduction of disease risk as the key criterion along with maintenance of optimal health, the RDA was formulated to meet/satisfy the nutritional needs of all healthy people centred around the estimated average requirements (EAR). The EAR for each nutrient was derived based on a specific criterion of adequacy selected to meet the requirements. Substantial changes have been made for energy requirement for adults considering the fact that the Indians have different body composition and considerably lower basal metabolism compared to western population.

This revision of RDA along with EAR would be useful for all stakeholders including policy makers, regulators, academic, dieticians and industry

My sincere thanks to all the expert committee members for their valuable guidance, suggestions and contributions and the resource persons whose relentless work helped us to bring out this revision of RDA along with EAR at this right juncture of changing nutrition scenario in India. Also, our gratitude to the Director-General, ICMR and the Ministry of Health and Family Welfare, Government of India for their unstinted support.



(Hemalatha R)

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FOREWORD

The formulation of the recommended dietary allowances (RDAs) for Indians had a long and distinguished history. The RDA of energy for Indians was first proposed by the Nutrition Advisory Committee (NAC) of the Indian Research Fund Association (IRFA) [now Indian Council of Medical Research (ICMR)] in 1944. These recommendations were based on the proposals of the Health Committee of the League of Nations made in 1935 but adapted to the lower body weights of Indian adults. The 1944 recommendations for energy for Indians were revised subsequently by the NAC of the ICMR in 1958 in the wake of reports by the Food and Agriculture Organization (FAO) on energy and protein. Subsequently, in 1968 and 1978 the ICMR committee revised the requirements for all nutrients except energy. Considerable additional information and newer statistical approaches for deriving energy requirements were generated and the FAO/WHO/UNU revised approaches in 1985 and again in 2004.

In 1989, the ICMR Expert Group adopted the procedure of 1985 FAO/WHO/UNU Expert Consultations and revised the Indian RDA. The ICMR 2010 committee RDA recommendations further revised and upgraded the RDAs for Indians based on the international data provided by FAO/WHO/UNU expert committee and based on weight and height collected across 16 states by NIN-NNMB rural data.

The present committee took the latest statistical approaches to derive the requirements through definitions of their distributions, such that the estimated average requirement (EAR) could be defined for population requirements, and the upper 95th percentile of the distribution could be used to define the individuals' requirements to place them at low risk of a deficient intake. In addition, since several foods are now being fortified with nutrients, the tolerable upper limits of intake of nutrients were also defined. The need for these new nutrient requirements has been because dietary patterns have changed with economic and nutrition transition occurring during the last decade. The double burden of malnutrition is evident in India. Nutrient requirements have to be reviewed with respect to recent data on energy expenditure, protein metabolism and in case of minerals and vitamins- data on losses and absorption need to be explored to derive EAR and RDA.

The process for generating this report was similar to the earlier recommendations. This draft Report was circulated to the Expert Group members and external Resource Persons well in advance of the meeting for them to go through and make specific comments on the requirements. Each nutrient was considered one by one at the expert group meeting for discussing the specific comments of the members of the expert group and resource persons. The presence of Dr. Maharaj Kishan Bhan, who passed away earlier this year, as Chair of this expert committee was invaluable. He had been a pillar of support to the ICMR-National Institute of Nutrition, serving for 10 years as the Chair of its Scientific Advisory Committee. The ICMR 2020 Committee wishes to place on record its deep appreciation for his guidance and expertise.

I wish to place on record my gratitude and appreciation for the stellar work of the scientists of National Institute of Nutrition, ably led by its Director, Dr. R. Hemalatha, the members of the Expert Committee, and the external reviewers of specific chapters, for bringing out this report in good time.

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1.1 INTRODUCTION

This book defines the nutrient requirements for Indians based on concepts related to the distribution of requirements in normal individuals of all ages. A fundamental part of defining nutrient requirements is that the requirement is not the same in all people. It can vary considerably in healthy individuals. In order to derive a single value for the requirement, two features of this distribution of requirements are used. First, the median of this distribution is called the estimated average requirement (EAR). The EAR is the nutrient requirement used in public health nutrition, to evaluate the nutrient intakes of a population. Second, the 97.5th percentile of the distribution is called the Recommended Daily Allowance. The RDA is for healthy individuals and may be prescribed to satisfy the nutritional needs of specific nutrients in a specific life stage and gender group and ensures that there is a very small risk of the nutrient intake being inadequate. With the RDA, there is also the risk of excess intake, since each individual may not actually require that much. There is no need to consume higher doses on regular basis or for prolonged period without supervision. Therefore, readers of this book will note that there has been a shift from the previous edition, and two distinct nutrient requirements are now presented: the EAR and RDA. In addition, nutrients are also toxic when ingested at very high doses. This has resulted in the definition of the Tolerable Upper Limit of Intake (TUL). Intake of nutrients more than the TUL invites the risk of toxicity. It cannot be overstated that when assessing the health and nutritional status of the population, the EAR is recommended as the unit of requirement.

In India, the first attempt to define nutrient requirements and desirable dietary intakes of nutrients for Indians to maintain good health was made by the Nutrition Advisory Committee of the Indian Research Fund Association [Now Indian Council of Medical Research (ICMR)] in 1944¹. This followed the recommendations made by the Technical Committee of the Health Committee, League of Nations in 1936², Food and Nutrition Board of the National Research Council, USA, 1944³ and Report of the Committee of Nutrition, British Medical Association 1933⁴. During this period, requirements and allowances of only energy, protein, iron, calcium, vitamin A, thiamine, riboflavin, ascorbic acid and vitamin D for Indians were considered. Considering these recommendations, a typical balanced diet based on habitual Indian dietary habits was formulated to provide all the nutrients for a normal adult reference man of 55 kg and a normal adult reference woman of 45 kg body weight⁵. This was used to demonstrate that the diet then consumed by Indians, particularly by the poor, was deficient in several nutrients and could be improved by inclusion of some protective foods.

THE CURRENT NUTRITION SCENARIO IN INDIA

India is in developmental transition and is facing the dual burden of malnutrition. The pre-transition diseases like undernutrition and infectious diseases as well as post-transition, lifestyle-related degenerative diseases such as obesity, diabetes, hypertension, cardiovascular diseases and cancers are wide-spread in India. The last National Family Health Survey (NFHS) – 4 indicates a slight decline in undernutrition, with a prevalence of stunting as 38.4% and wasting as 21.5%⁶. Simultaneously, the burden of overnutrition has increased to about 20% in adult men and women⁶.

Severe clinical forms of Protein Energy Malnutrition (PEM) - kwashiorkor and marasmus have become very rare. Over 50% women (particularly pregnant women) and children suffer from iron deficiency anaemia (IDA), aggravated by helminthic infections. Though blindness due to vitamin A deficiency has become rare, a survey conducted by National Nutrition Monitoring Bureau (NNMB)

shows that milder grades of deficiency as judged by clinical signs like night blindness and Bitot's spots and low serum vitamin A levels, are common⁷. Deficiencies of other micronutrients like some B-complex vitamins particularly riboflavin, folic acid and perhaps vitamin B₁₂ are also common. Rickets has become rare, but recent studies from North and South India show that vitamin D deficiency as judged by serum levels of 25-hydroxy vitamin D exists in adults. This, besides low intake of calcium, may be responsible for the high prevalence of osteoporosis particularly in women. The problem of severe forms of Iodine Deficiency Disorders (IDD) (an environmental problem) has been considerably reduced after universalization of Iodized salt. However due to improper implementation, milder forms of IDD persist in many districts. For every frank case of nutrition deficiency, there are dozens of others who suffer from sub-clinical malnutrition.

REVISION OF HUMAN NUTRIENT REQUIREMENTS

In the wake of reports by the Food and Agriculture Organization (FAO) on calorie^{8,9} and protein¹⁰ in 1950 and 1957 respectively, an attempt was made by the ICMR in 1958 through its Nutrition Advisory Committee (NAC) to revise protein and calorie requirements of Indians, based on data available at that time¹¹. In 1968, the requirements of all nutrients except energy were reviewed by an Expert Committee constituted by ICMR¹². In 1978, the Recommended Dietary Allowances (RDA) for Indians were again reviewed by another Expert Group of the ICMR and RDAs of several nutrients were revised¹³. In the recommendations made by the ICMR Expert Group in 1968 and 1978, a wide range of balanced diets for different age and sex groups were formulated which, if consumed, could ensure a daily intake of all nutrients at the recommended levels. The recommendations on human protein and energy requirements were again revised by a Joint Expert Group of FAO, WHO and United Nations University (UNU) in 1985¹⁴. In arriving at human energy requirement, this International Expert Group followed an entirely new set of guidelines. Based on the new international guidelines, the ICMR 1989 revised the RDA for Indians¹⁶. However, no substantial changes were made in the RDAs for protein, B-complex vitamin, iron and calcium. Also, no definitive recommendations were made for trace elements. Over the years, with the advancement in the data on various nutrients among Indians, the ICMR 2010 committee made changes to the RDAs¹⁷. The ICMR 2010 committee recommendations were generated based on the international data provided by FAO/WHO/UNU expert committee and data generated in India.

Energy allowances for Indians, which were recommended in 1958, had not been revised till 1988. In 1988, an Expert Group was constituted by the ICMR. The previous Expert committees of ICMR, while following the new guidelines of the Joint FAO/WHO/UNU Consultative Group of 1985¹⁴, also considered the updated data on Indians that had accumulated after 1973¹⁵, to define the energy and protein requirements of Indians. The Expert Group also defined the requirement of other nutrients like fat, vitamin D and vitamin A. No changes were, however, made in the recommendations on the requirement of B-complex vitamins, iron and calcium. Further, recommendations on several additional nutrients such as carbohydrate, dietary fiber, water, phosphorus, vitamin E and vitamin K or dietary factors not considered by the earlier ICMR Expert Committees and made provisional recommendations on their desirable intakes to maintain good health. Dietary fat requirements were examined in greater detail and recommendations regarding the requirement in terms of invisible and visible fat were made¹⁶. The reference body weights of normal healthy adult man, woman and children were also altered based on the body weight data on healthy normal adults and children obtained by National Institute of Nutrition (NIN)^{18,19}.

Subsequently, the FAO/WHO/UNU Expert Consultation considered the revision of human nutrient requirements again after 2000. One Committee revised the requirement of micronutrients in 2001²⁰, energy in 2004²¹ and protein in 2007²². In its revision, the international expert group

considered several other micronutrient requirements of humans. The energy requirement, particularly of children 1-10 years was based on stable oxygen use and energy requirement of adults was guided by widespread prevalence of overweight and obesity in the west. In case of proteins, requirement of indispensable amino acids (IAA) was discussed in greater detail and RDA for IAA were also included. With the advancement in research in nutrition, the ICMR 2010 Expert Group¹⁷ had revised and upgraded the RDAs for Indians.

However, in the last decade, there has been further advancement in the research with paradigm shift in nutritional status of the population. Considering the evidences, the RDAs of nutrients for Indians have been revised by the current ICMR Expert Group. Substantial changes have been made in energy requirement for adults considering the fact that the Indians are becoming more sedentary and the basal metabolism of Indians are far different from western population. The present requirement includes the Estimated Average Requirement (EAR), RDA and Tolerable Upper Limit (TUL) as mentioned in chapter-2 (framework for nutrient requirement).

WHY ICMR-NIN DIETARY RECOMMENDATIONS 2020?

- Over the past decade, dietary patterns have changed with economic and nutrition transition at the community level. These rapid changes are relevant to the dual burden of malnutrition in India. Nutrient requirements have to be reviewed with respect to these changes in food intake, access, and lifestyle.
- Until now, a single value, the Recommended Dietary Allowance (RDA) was used for all nutrients. Over the years the nutrient requirements have evolved so that they can be specifically used for individuals or populations. The present recommendations propose the use of the Estimated Average Requirements (EAR) for evaluating population nutrient intake and the Recommended Dietary Allowances (RDA) for setting the safe nutrient intake for an individual.
- Several foods now being fortified with nutrients, the Tolerable Upper Limit (TUL) of intake for nutrients is essential, and therefore the TUL has also been defined.
- New data are incorporated for bioavailability of nutrients, wherever available.

References

1. Nutrition advisory committee of the Indian Research Fund Association (IRFA). A report of the twelfth meeting. New Delhi, India; 1944.
2. LEAGUE ON. The problem of nutrition. Vol. 2. Report on the physiological bases of nutrition drawn up by the Technical Commission of the Health Committee. The problem of nutrition. Vol. 2. Report on the physiological bases of nutrition drawn up by the Technical Commission of the Health Committee. 1936;2.
3. Food and Nutrition Board of the Nutrition Research Council, USA Recommended Dietary Allowances. NRC Reprint and Circular No. 127. 1944.
4. British Medical Association. Report of the Committee on Nutrition. *Brit Med J (Suppl.)* Nov. 25, 1933.
5. Aykroyd WR. The nutritive value of Indian foods and the planning of satisfactory diets. The nutritive value of Indian foods and the planning of satisfactory diets. 1937(23).
6. IIPS I. National Family Health Survey (NFHS-4), 2015–16. International Institute for Population Sciences (IIPS), Mumbai, India. 2017.
7. National Nutrition Monitoring Bureau. Prevalence of micronutrient deficiencies. NNMB Technical Report No. 22. 2003.
8. FAO. Calorie requirements. Report of the Committee on Calorie Requirements, FAO Nutritional Studies, no. 5, Washington, D.C.: 1950.
9. FAO. Calorie requirements. Report of the Second Committee on Calorie Requirements. FAO Nutritional Studies, no. 15, Rome: 1957.
10. FAO. Protein Requirements. Report of the Committee on Calorie Requirements, FAO Nutritional Studies No.16, 1957.
11. Patwardhan VN. Dietary Allowances for Indians. Calorie Requirements. Special Report. Indian Council of Medical Research. 1960(35):1-20.
12. Gopalan C and Narasinga Rao BS. Dietary Allowances for Indians. Special Report Series No. 60. ICMR, New Delhi, 1968.
13. ICMR, Recommended Dietary Intakes for Indians. Report of an Expert Group, New Delhi, 1978.
14. Joint FAO/WHO/UNU, Energy and Protein Requirements. Report of a Joint FAO/WHO/UNU Expert Consultants, WHO Technical Report Series 724, 1985.
15. FAO/WHO, Energy and Protein Requirements. Report of a Joint FAO/WHO ad-hoc Expert Committee, WHO Technical Report Series No. 522, Geneva, 1973.
16. ICMR, Nutrient requirements and Recommended dietary allowances for Indians. A Report of the Expert Group of the Indian Council of Medical Research, National Institute of Nutrition, Hyderabad, 1990.
17. ICMR, Nutrient requirements and recommended dietary allowances for Indians. A report of the expert group of the Indian Council of Medical Research National Institute of Nutrition Hyderabad, 2010.
18. Raghavan KV, Singh D, Swaminathan MC. Heights and weights of well-nourished Indian school children. Indian Journal of Medical Research. 1971; 59(4):648-54.
19. Rao DH, Satyanarayana K, Sastry JG. Growth pattern of well-to-do Hyderabad pre-school children. Indian Journal of Medical Research. 1976; 64(5):629-38.
20. FAO W. Human vitamin and mineral requirements. Report of a joint FAO/WHO expert consultation, Bangkok, Thailand. Food and Nutrition Division, FAO, Rome. 2001:235-47.
21. FAO F. Nutrition Technical Report Series 1: Human Energy Requirements. Report of a joint FAO/WHO/UNU Expert Consultation 2001. Rome FAO, 2004.
22. WHO J. Protein and amino acid requirements in human nutrition. World health organization technical report series. 2007(935):1.

1.2 GENERAL CONSIDERATIONS

Humans need a wide range of nutrients to lead a healthy and active life. The required nutrients for different physiological groups can only be derived from a well-balanced diet. Components of the diet must be chosen judiciously to provide all the nutrients to meet the human requirements in proper proportions for the different physiological activities. The amount of each nutrient needed for an individual depends upon his or her age, body weight and physiological status. Adults need nutrients for maintenance of constant body weight and for ensuring proper body function. Infants and young children grow rapidly and require nutrients not only for maintenance but also for growth. They require relatively more nutrients (3-5 times) per kg body weight than adults. In physiological conditions like pregnancy and lactation, adult woman needs additional nutrients to meet the demand for fetal growth and maternal tissue expansion in pregnancy and milk secretion during lactation. These extra intakes of nutrients are essential for normal growth of infants *in utero* and during early post-natal life.

There are certain general guidelines in arriving at Nutrient Requirement and Dietary Allowances for various groups. The nutrient requirement of an individual and the dietary allowances for a group or a population are distinctly different. The former depends upon the age, body weight and physiological and metabolic status of the individual. The latter must also take into consideration individual variation within the group, quality of the diet, effect of cooking and processing and bio-availability of the nutrient from the diet.

1.2.1. General Principles for deriving human nutrient requirements

Several methods have been employed over the years to arrive at the requirement of different nutrients for individuals of different physiological groups and some of these methods have improved with time. The general principles underlying these methods are:

Dietary intakes: This approach is used to arrive at the energy requirement of children. Energy intakes of normal growing healthy children are used for this purpose. Currently it is not in use as it is considered to overestimate the requirement and not yield correct figures.

Growth: Daily intake of breast milk and its nutrient content are utilized to define the nutrient requirement during early infancy (0-1y). This approach is also no longer in use as it leads to an overestimation of the requirement during early infancy. However, the mode of satisfying the nutrient requirement in early infancy (upto 6 months) is only through breast milk intake.

Nutrient balance: The minimum intake of a nutrient for equilibrium (intake = output) in adults and nutrient consistent retention with satisfactory growth in infants and children, for satisfactory maternal and foetal growth during pregnancy, satisfactory breast milk output during lactation have been used widely in arriving at the protein requirements.

Obligatory loss of nutrients: The minimal loss of any nutrient or its metabolic product (viz. nitrogenous end products of proteins) through normal routes of elimination viz. urine, faeces and sweat is determined on a diet devoid of, or very low in the nutrient under study (viz. protein-free diet). These values are used to determine the amount of nutrient to be consumed daily through the diet to replace the obligatory loss of the nutrient and it represents the maintenance needs of an individual (viz. adults). In infants and children, growth requirements are added to this maintenance requirement. This approach has been widely used in assessing the protein requirement. Other losses of N through sweat, hair etc., are not considered in this method.

Factorial approach: In this approach, the nutrients required for different functions, are assessed individually and added up to arrive at the total daily requirement. This has been the basis of computing the energy requirements (viz., sleep + rest + occupational activity + non-occupational activity). This approach was being used earlier for assessing the protein requirements also.

Nutrient turnover: Results from studies on turnover of nutrients in healthy persons, using isotopically labeled nutrients are employed in arriving at the requirement of certain nutrients. Requirements of vitamin A¹, vitamin C², iron³ and vitamin B₁₂⁴ have been determined through this approach. Earlier, radioactive isotopes were used and currently stable isotopes, which are safer and being increasingly used to determine the turnover of nutrients in the body⁵. Stable isotopes are particularly useful, as they are safer, in determining the turnover of nutrients in infants, children, in women particularly during pregnancy and lactation where use of radioisotopes are contraindicated. Stable isotope labeled nutrients are however expensive and difficult to obtain.

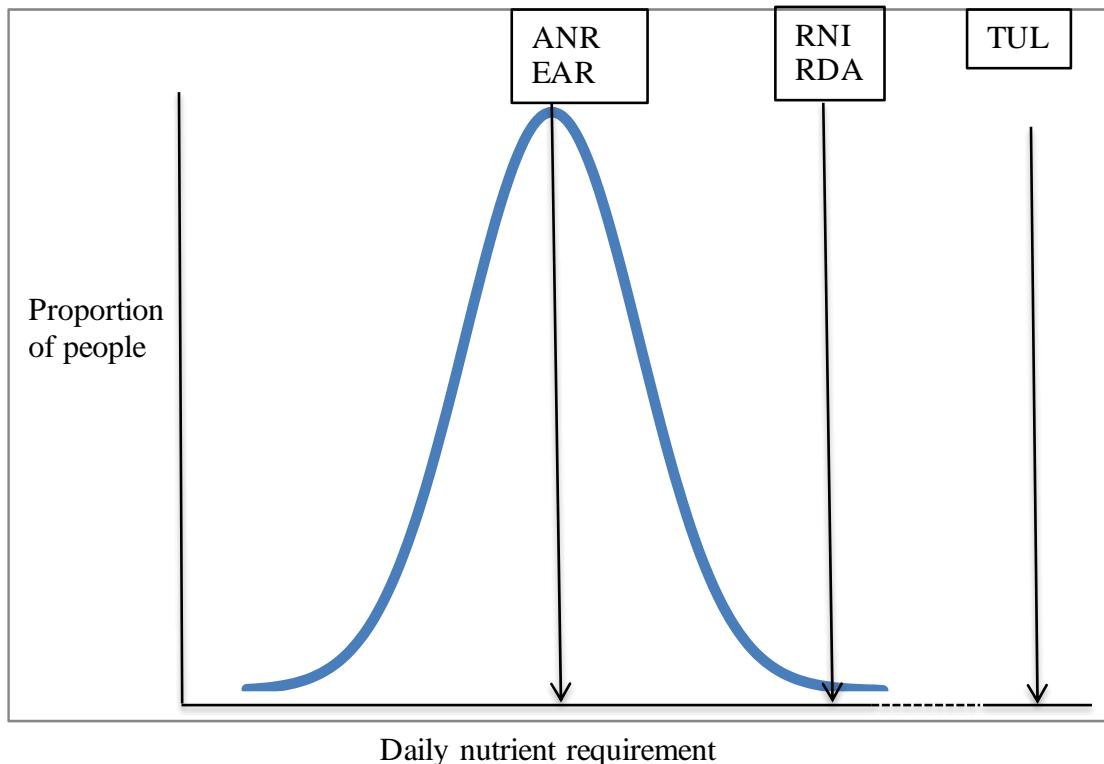
Depletion and repletion studies: This approach is used in arriving at the human requirement of water-soluble vitamins. The level of the vitamin or its coenzyme in serum or cells (erythrocytes, leucocytes) is used as the biochemical marker of the vitamin status. Human requirements of ascorbic acid (vitamin C), thiamine (vitamin B₁) riboflavin (vitamin B₂), and pyridoxine (vitamin B₆) have been determined employing this approach. Healthy volunteers are first fed a diet with very low levels of the vitamin till the biochemical parameter of the vitamin (or its coenzyme) reaches a low level. Response to feeding graded doses of the vitamin with the diet is then determined. The level at which the response increases rapidly corresponds to the level of the requirement of the vitamin.

1.2.2. Frame work for nutrient requirements

Several countries recommend nutrient intakes for their populations, which are used to plan and evaluate the nutrient intakes of healthy people. Nutritional policies, food regulations and nutritional programs are based on these nutrient intake recommendations². The recommended values differ from country to country and could range from a single value for a population group (as in the 2010 ICMR NIN RDA⁶), to four different values that define a ‘lower reference intake’, an ‘average requirement’, a ‘recommended intake’ for individuals from a specific population, and an ‘upper tolerable intake’^{6,7}. These values also differ for a given nutrient depending on the stage in life cycle and gender of the individuals.

In 2007, the United Nations University’s Food and Nutrition Program, in collaboration with the Food and Agriculture Organization (FAO), the World Health Organization (WHO), and the UNICEF, attempted to harmonize the nutrient requirement recommendations used across several countries, and coined the term Nutrient Intake Values (NIV), using primary data from several countries. These were primarily, Dietary Reference Values (DRV, UK), Nutrient Reference Values (NRV, Australia, New Zealand), Reference Values for nutrient supply (Germany, Austria, Switzerland), and Dietary Reference Intakes (DRI, USA, Canada)^{7,8}. The approach to describing the average requirement and the recommended intake are shown in Figure 1.2.1.

Figure 1.2.1: Distribution of the requirements of a theoretical nutrient in a population, showing EAR/ANR and RDA/RNI. The TUL is also depicted as an intake in excess*



* The dashed line on the X-axis depicts a variable distance between the RNI/RDA and the TUL for different nutrients.

Two of the NIV's were recommended for comparability across all countries for specific life stages and genders: average nutrient requirement (ANR) which is equivalent to the Estimated Average Requirement (EAR⁹) and Upper Nutrient Level (UNL) equivalent to the Tolerable Upper Limit (TUL⁹). The other values like the Reference Nutrient Intake (RNI)/ Recommended Dietary Allowance (RDA)/ Recommended Dietary Intake (RDI), which are very similar to each other, and the Lower Reference Nutrient Intake (LRNI), were derived values from the two recommended NIV's. Each of these terms is defined in Table 1.2.1.

An additional term i.e., used is the Acceptable Macronutrient Distribution Ranges (AMDR). The AMDR is a range of macronutrient intakes that is associated with a reduced risk of chronic diseases, but at the same time, provides adequate intakes of essential nutrients. It is usually expressed as a percentage of energy, with a lower and upper limit.

The key measurements within this framework, as defined above, are that of the EAR and its variance, and that of the TUL. The EAR is ideally directly measured experimentally. This has been done for some nutrients, for example, energy, protein and amino acids^{10,11}. However, this also has to be performed at different ages, since growth imposes its own demand on daily nutrient intake, while aging has its unique impact on the requirement. If the measurement is performed easily and non-invasively, then it is ideal for use across ages. Unfortunately, many of these measurements require some degree of invasive procedures, meaning that the measurement is not possible in vulnerable populations; and a careful selection of subjects, and controlled settings, meaning that these are 'laboratory or perfect conditions' measurements, and not 'real world'. The use of stable isotope technology has been key to the development of less invasive measurements, such as the double-

labelled water method for the measurement of energy expenditure¹¹, or the indicator amino acid oxidation method for indispensable amino acid requirements¹². The key measurements used in the framework of nutrient requirements are the *mean* of the requirements or the EAR, and the distribution or variance of the requirements, which yields a *standard deviation* (SD), which is used to calculate the RDA. Note that a normal distribution is required for this method; if the distribution is not normal, then it must be normalized before the SD is used. This is relevant for some nutrient requirements, notably protein and iron.

Table 1.2.1: Definition of terms used in the framework of nutrient requirements

<i>Estimated Average Requirement (EAR)</i>	Refers to the average daily nutrient intake level estimated to meet the requirements of half of the healthy individuals in a particular life stage and gender group. It is used primarily to evaluate populations or groups.
<i>Recommended Dietary Allowance (RDA)</i>	Refers to the daily dietary nutrient intake level that is sufficient to meet the nutrient requirements of nearly all (97–98 percent) healthy individuals in a particular life stage and gender group. This is derived from the EAR as the mean plus 2 standard deviations (SD) of the distribution of requirements. The term is used to primarily evaluate individual diets. <i>The RDA is inappropriate for dietary assessment of groups as it is the intake level that exceeds the requirement of a large proportion of individuals within the group.</i>
<i>Tolerable Upper Level (TUL)</i>	Refers to the highest average daily nutrient intake level that is likely to pose no risk of adverse health effects to almost all individuals in the general population. As intake increases above the TUL, the risk of adverse effects will increase.
<i>Adequate Intake (AI)</i>	These values are used when EAR or RDA cannot be determined. The AI is the recommended average daily intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group of apparently healthy people that are assumed to be adequate.
<i>Lower threshold intake (LTI)</i>	Refers to a value derived from the EAR and is calculated as the EAR minus 2 SD of the distribution of requirements. This value is sufficient to meet the needs of the bottom 2% of individuals. However, countries have used a different cut off such as 5% or 10% to evaluate nutrient insufficiency, although the concern is that these values would set a very low expectation of the individual nutrient intake adequacy level.

If the EAR cannot be experimentally measured, another approach is to deconstruct the total daily requirement into a set of compartments or factors that can be added up. For example, energy requirements are calculated as the sum of the basal requirement, and the requirement of activity and thermogenesis¹¹. This requires assumptions to be made of the mean of each factor and is called the factorial method. The distribution of the requirement is either assumed from observations in the literature or calculated indirectly from the variability of body size or growth, which is a key ‘factor’ in determining the daily requirement.

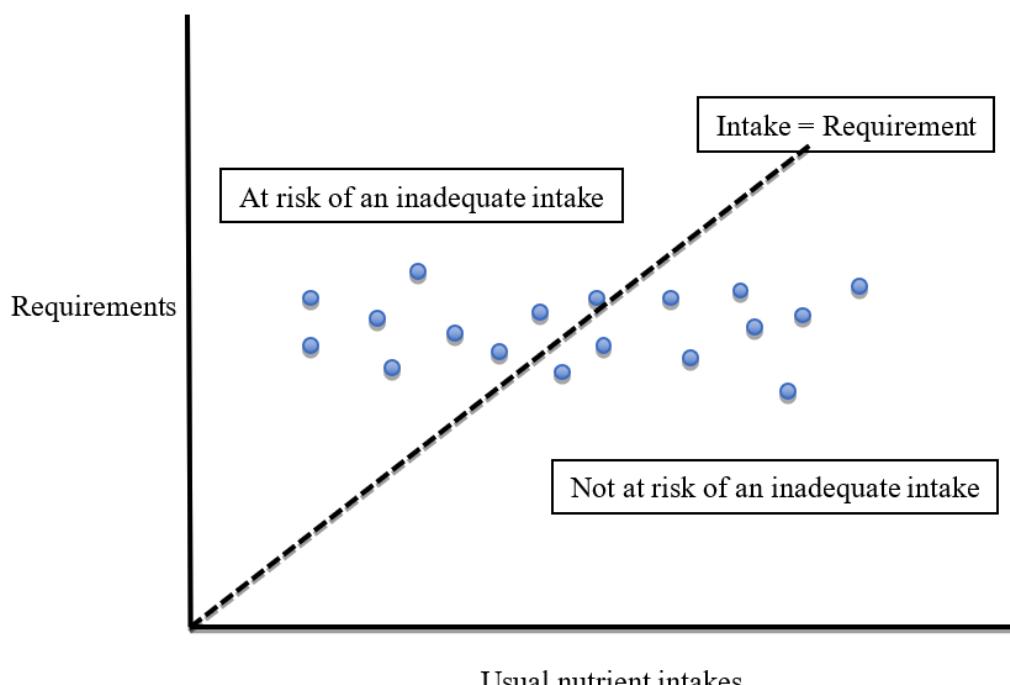
1.2.3. Conceptual basis for calculating the risk of nutrient inadequacy

An individual has inadequate intake when the intake does not meet the requirement of the nutrient. If the distribution of requirements in a population is established (and the individual could be reasonably said to be representative of this population), then the EAR and the RDA will be derived. Since the RDA is the sum of the EAR and 2 SD, recommending this intake for an individual will mean that his/her chances of being at risk of an inadequate intake is <2.5%.

In a population, the risk of inadequacy translates to the proportion of people whose usual intake of a nutrient does not meet their requirement. The risk of an inadequate intake in the population can be easily calculated if population level data on usual intakes and the requirement are available (this is not usually the case). If this were available, one critical requirement of risk calculation is that the intake distribution does not correlate with the requirement distribution (Figure 1.2.2). As can be seen from this theoretical figure, the nutrient intakes of a population are quite variable and have a large range. The tightly regulated nutrient requirements, as expected in biological systems, are represented on the Y-axis. The result is that the scatter does not lie on the line of identity but is flat. Nevertheless, one could now identify the proportion of the population that was eating less than their requirement, as those to the left of the line of identity¹³.

However, obtaining such data (on the intake and requirement of a population) is impractical. Therefore, statistical approximations are used to assess the risk of an inadequate intake. One such method is the probability approach where a continuous risk curve of probability that any intake is inadequate is plotted against the intake value. In this plot, the lower levels of intake will have a probability of inadequacy that is close to 100%, and this declines with increasing intakes, such that higher levels of intake have a 0% probability of an inadequate intake (Figure 1.2.3). By plotting the usual intake distribution against this probability plot, the proportion with the probability of an inadequate intake can be determined.

Figure 1.2.2: Theoretical scatter plot of nutrient intake vs requirement*



*The line of identity refers to a situation where nutrient intake matches the requirement.

The ‘EAR cut point method’ is a simplification of the probability method in which the proportion of the population with an intake lower than the EAR for the nutrient, are considered to be ‘at risk for an inadequate intake’. A word of caution is needed for this method, simple as it seems, since the following assumptions are made for the EAR cut-off point method: a) the nutrient intakes and requirements are independent, b) the requirement distribution is symmetrical around the EAR, c) the variance intakes is larger than the variance of requirements, d) the true prevalence of inadequacy in the population is no smaller than 8-10% or no larger than 90-92%.

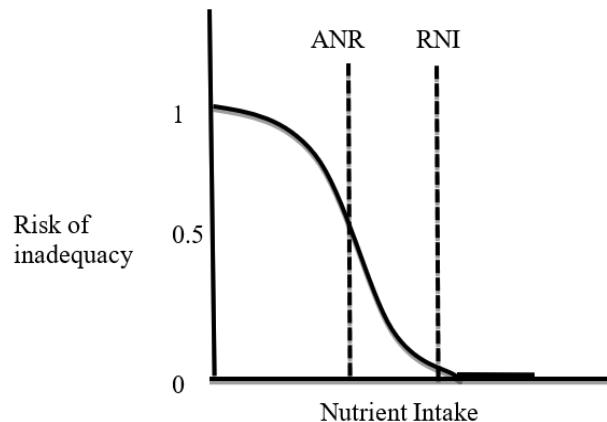
For nutrients whose distributions violate these assumptions, such as iron, adjustments are made¹³. Another example is that of the energy requirement, which is closely related to the intake. This violates the assumption of independence between the intake and requirement consequently for energy, a separate term, the Estimated Energy Requirement (EER) is used; there is *no* RDA for the energy requirement, since the latter would grossly over estimate the requirement.

1.2.4. Tolerable Upper Limit (TUL)

The TUL is the maximum level of habitual intake from all sources of a nutrient or related substance judged to be unlikely to lead to adverse health effects in humans¹⁴. An adverse effect is a change in morphology, physiology, growth, development, reproduction or lifespan of an organism, system, or (sub) population that results in an impairment of functional capacity, an impairment of capacity to compensate for additional stress, or an increase in susceptibility to other influences. The potential risk of adverse effects increases after the intake increases above the Upper Limit (UL). The TUL applies to chronic daily use and it is important to first assess the characteristics of the individual or group, the source of the nutrient, the physiological state of the individual and the duration of sustained high intakes. The bioavailability of a nutrient, which is its accessibility to normal metabolic and physiological processes, also plays a role in the nature and severity of adverse effects at excessive intakes. However, in some cases, the unabsorbed nutrient may also have effects on the lower parts of the intestine. This is particularly relevant for iron, in which the unabsorbed iron may have effects on the intestinal microbiome. The most appropriate approach is to establish the TUL for age/gender/life stage sub-populations, since adverse effects of nutrients are influenced by growth and physiological stages. The increased availability and consumption of fortified foods and food supplements has sparked concerns about excessive intake of nutrients. It is important to assess the safety of fortification by comparing eventual micronutrient intakes with the TUL. High levels of micronutrient additions should be avoided even if a micronutrient has no recommended TUL, particularly if there is no evidence of derived benefit from levels of intake in excess of the RDA.

Risk assessment is a systematic means of evaluating the probability of occurrence of adverse health effects in humans from an excess exposure to an environmental agent¹⁴. Risk assessment has four stages, including hazard identification, hazard characterisation (through dose-response assessment), exposure assessment and risk characterisation. The risk assessment process needs to be rigorous and transparent, particularly with regard to the paucity of the data in human populations. Almost all of the risk assessments have an inherent uncertainty and variability. Identifying and

Figure 1.2.3: Conceptual figure of risk of inadequate intake plotted against nutrient intake*



*At an intake that equals the ANR/EAR, 50% of the population is theoretically at risk of an inadequate intake.

accounting for these variabilities and uncertainties is an essential part of the data analysis for identifying and characterizing the hazard. Various national and international advisory bodies have used the same data, but arrived at different risk assessments due to the different judgments made about identifying adverse effects, the nature of uncertainties in the assessment, and in matching the upper levels with exposure assessments and dietary reference values. Since, the establishment of different upper levels for different nationalities is a source of confusion in public health policy and practice, a collaborative development of the model for establishing upper levels of intake for nutrients and related substances was proposed by a Joint Task Force of the World Health Organization and the Food and Agriculture Organization¹⁴.

There are many terms that are used in this context, such as the no-observed- adverse-effect level (NOAEL), lowest-observed- adverse- effect level (LOAEL) and uncertainty factor (UF) and the acceptable daily intake (ADI). NOAEL is the highest intake of a nutrient at which no adverse effects have been observed in the individuals or groups. LOAEL is the lowest intake at which an adverse event has been identified. UFs are applied to address both the gaps in data and incomplete knowledge. There are many scientific uncertainties associated with extrapolating data to the general population, from the observed values and several judgements need to be made in deriving uncertainty factor for each nutrient. The individual UFs are combined together to arrive at a composite UF for the nutrient. The UFs are lower with data of high quality and when adverse effects are mild and reversible. The TUL of a nutrient is normally derived by dividing the NOAEL (or LOAEL) by the composite UF¹⁵.

The estimation of the LOAEL, NOAEL, and the ADI is based on a well-accepted procedure, in which the LOAEL is first determined, based on animal data available. One level below that is the NOAEL, and a hundredth of that level is the ADI. In case human adverse event data are used, the ADI is usually found to be about one third of the LOAEL. In this framework, the TUL is basically ADI, and refers to the quantity of nutrient coming from all dietary and supplementary sources in a day. From the viewpoint of dietary fortification of nutrients, it is clear that as the nutrient intake progresses beyond the RDA, it could continue to give benefit and this has to be judged based on the evidence available. However, this benefit will occur progressively in a smaller proportion of the population. The key point to consider here is the possibility of harm to the general population. This has to be determined for all proposed fortificants. The role of genetics and the environment in altering the TUL cannot be dismissed. Finally, it is important to consider the harmonization of methods to determine nutrient requirements across countries. This process was considered recently¹⁶, and for some nutrients, such as vitamin A, the requirements in different countries is quite different. As we progress, it is hoped that uniform methods of gathering and evaluating evidence are used.

References

1. Sauberlich HE, Hodges RE, Wallace DL, Kolder H, Canham JE, Hood J, Raica Jr N, Lowry LK. Vitamin A metabolism and requirements in the human studied with the use of labeled retinol. InVitamins & Hormones 1975 Jan 1 (Vol. 32, pp. 251-275). Academic Press.
2. Baker EM, Hodges RE, Hood J, Sauberlich HE, March SC, Canham JE. Metabolism of 14C-and 3H-labeled L-ascorbic acid in human scurvy. The American journal of clinical nutrition. 1971 Apr 1; 24(4):444-54.
3. Green R, Charlton R, Seftel H, Bothwell T, Mayet F, Adams B, Finch C, Layrisse M. Body iron excretion in man: a collaborative study. The American journal of medicine. 1968 Sep 1; 45(3):336-53.
4. Heyssel RM, Bozian RC, Darby WJ, Bell MC. Vitamin B12 turnover in man. The assimilation of vitamin B12 from natural foodstuff by man and estimates of minimal daily dietary requirements. American Journal of Clinical Nutrition. 1966; 18:176-84.
5. Etcheverry P, Hawthrone KM, Liang LK, Abrams SA, Griffin IJ: Effect of beef and soy proteins on the absorption of non-heme iron and inorganic zinc in children. J Am Coll Nutr. 2006; 25: 34-40.
6. ICMR, Nutrient requirements and recommended dietary allowances for Indians. A Report of the expert group of the Indian Council of Medical Research. National Institute of Nutrition, Hyderabad, 2010.
7. King JC, Vorster HH, Tome DG. Nutrient intake values (NIVs): a recommended terminology and framework for the derivation of values. Food and Nutrition Bulletin. 2007 Mar; 28(1_suppl1):S16-26.
8. King JC, Garza C. Harmonization of nutrient intake values. Food and nutrition bulletin. 2007 Mar; 28(1_suppl1):S3-12.
9. Meyers LD, Hellwig JP, Otten JJ, editors. Dietary reference intakes: the essential guide to nutrient requirements. National Academies Press; 2006 Sep 29.
10. WHO J. Protein and amino acid requirements in human nutrition. World health organization technical report series. 2007(935):1.
11. FAO F. Nutrition Technical Report Series 1: Human Energy Requirements. Report of a joint FAO/WHO/UNU Expert Consultation 2001. Rome FAO, 2004.
12. Kurpad AV, Thomas T. Methods to assess amino acid requirements in humans. Current Opinion in Clinical Nutrition & Metabolic Care. 2011 Sep 1; 14(5):434-9.
13. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. DRI dietary reference intakes: applications in dietary assessment. Washington (DC): National Academies Press (US); 2000.
14. World Health Organization/Food and Agriculture Organization. A model for establishing upper levels of intake for nutrients and related substances. Report of a Joint FAO/WHO Technical Workshop on Nutrient Risk Assessment. Geneva: WHO/FAO, 2006.
15. European Food Safety Authority. Tolerable upper intake levels for vitamins and minerals. Scientific Committee on Food. 2006.
16. National Academies of Sciences, Engineering, and Medicine. Global harmonization of methodological approaches to nutrient intake recommendations: Proceedings of a workshop. National Academies Press; 2018 Jul 29.

2. SUMMARY OF RECOMMENDATIONS

Recommended Dietary Allowances & Estimated Average Requirements for Indians - 2020

A SHORT REPORT

REFERENCE BODY WEIGHT

Earlier Expert Committee on RDA used data generated during 1989 on body weights and heights of well-to-do Indian children and adolescents, which was based only on a segment of Indian population and did not have an all India character. The reference weights for man and woman were 60 kg and 50 kg respectively.

The 2010 Committee has considered extensive data on anthropometry collected by NNMB/India nutrition profile from 10 states of India for computing reference body weights. Since the data collected was from rural India, the committee decided to use the 95th centile values of heights and weights for a given age and gender which will be representative of well-nourished population of India. For computing RDA for children (0-3y), WHO growth standards for infants and preschool children were considered.

The present committee has considered the more recent, nationally representative datasets such as the National Family Health Survey - 4 (NFHS-4, 2015-16), National Nutrition Monitoring Bureau (NNMB, 2015-16), the World Health Organization (WHO, 2006-07) and the Indian Academy of Paediatrics (IAP 2015) to derive acceptable reference body weight values through the lifespan. The reference height was taken as 95th centile for adult male and female, and with normal BMI range of 18.5-22.9 kg/m², a reference body weight was calculated.

The definition for reference Indian adult man and woman were modified with regard to age (19-39y instead of 20-39y) and a body weight of 65 kg and 55 kg respectively were fixed for a normal BMI.

ENERGY

The factorial approach used for adults in computation of energy requirement by the earlier committee is retained. Additionally, the current committee has used Doubly Labelled Water (DLW) and heart rate monitoring methods for computation of total energy expenditure for deriving requirements as done in the previous recommendations.

The earlier committee used 5% reduction in BMR from FAO/WHO/UNU equations and higher PAL values for deriving energy requirements for adults. While the present committee reviewed the literature on BMR and PAL based on the evidence, a reduction in the BMR to 10% and 9% for males and females respectively with simultaneous reduction in PAL values is proposed. The current committee uses the lower ranges of PAL reported by FAO/WHO/UNU, 2004 report. The energy requirement for the population >60y of age has been provided as requirements decrease due to a reduction in BMR. Because of change in body weight, a proportionate increase in requirement has been suggested in pregnancy. As data on pregnant Indian women is unavailable the present committee has retained the additional energy requirement proposed by ICMR 2010. In the case of lactation, the average energy utilization for milk production based on actual observation is taken into consideration and an increase has been suggested. No changes from the previous recommendations have been made in the additional requirements of lactating women.

The earlier committee had adopted the FAO/WHO/UNU, 2004 equations for deriving the energy requirement of infants and children since there was an absence of Indian data and also used the body weights reported in the above-mentioned document. However, the present committee has used the WHO child growth standard data for body weight of infants and re-analyzed the energy requirement for infants. With the use of these values 1 kcal/kg body weight/d increment of requirement for infants aged 0-6 months is reported when compared to the previous recommendations. Otherwise the requirement for children above 6 months of age remains the same as suggested by the previous committee. Both the previous and the present committee, have emphasized the importance of physical activity among children. It is recommended that children should be engaged in moderate physical activity. This approach has led to a decrease in energy requirement of children. Among children of 13-17 years, there was an increase in requirements on account of using same quadratic equation generated from FAO/ WHO/ UNU 2004 to which a higher PAL value was used based on a higher physical activity level of Indian children of that age group in ICMR, 2010. The same has been retained by the present committee.

PROTEIN

The present Expert Group of the ICMR adopted the following approaches to define the protein requirements for Indians of different age groups. A median obligatory nitrogen loss of 48 mg/kg (WHO, 2007) has been used to compute mean (0.66 g/kg/day) and safe protein requirements (0.83 g/kg/day) for healthy Indian adults. Considering high quality protein sources as the premise for defining requirements, the present committee has removed the protein digestibility corrections (PDCAAS) applied on safe intakes for all age groups.

A newer protein quality index, digestible indispensable amino acid score (DIAAS), which is based on true ileal digestibility of individual amino acids has been introduced in the current document. Data on true ileal amino acid digestibility values of both high and low quality proteins in Indian adults and children, obtained using dual tracer method has been included in the present document. The low cost Indian vegetarian diets for sedentary, moderate active man and pregnant woman have been modified based on the revised energy requirements. The nutritive values of each food are taken from recently published food composition tables (IFCT, 2017). In addition, the protein contents of each food group have been corrected for true fecal digestibility values (WHO, 2007) to ensure safe protein intakes. The cereal-legume-milk composition of the diet for moderately active man has been improved to 3:1:2.5 as compared to the earlier 11:1:3 (ICMR 2010) within a given low cost window to meet daily protein requirements.

FATS AND OILS

The FAO/WHO recommendations on fat were taken into account for (i) total fat, individual fatty acids and health promoting non-glyceride components (ii) sources of dietary fats in Indians (iii) availability of fat and (iv) energy requirements set on the basis of age, physiological status and physical activity. The recommendations are directed towards meeting the requirements for optimal foetal and infant growth and development, maternal health and combating chronic energy deficiency (children and adults) and Diet Related Non-Communicable Diseases (DR-NCD) in adults. There was a conscious effort to provide physical activity-based recommendations. Consequently, the visible fat intake for sedentary, moderate and heavy activity has been set at 25, 30 and 40 g/d for adult man and 20, 25 and 30 g/d for adult women as against the single level recommended earlier. To achieve intakes of individual fatty acids in Indians that are consistent with FAO/WHO 2008 recommendations the types of visible fats and correct combination of vegetable oils to be used for different food applications has been also emphasized. There is a realization that efforts to increase the dietary levels of total fat and n-3 PUFAs would contribute to lifelong health and well-being. Inclusion of foods which provide LCn-3 PUFAs is also recommended for the prevention of DR-NCD.

DIETARY FIBER

For the first time committee considered recommendations for fiber based on energy intake and the level of about 40 g/2000 kcal has been considered as safe intake.

CARBOHYDRATES

The quantity and quality of CHO are important to maintain good health and have been indicated substantially to impact nutrition related chronic disorders/non-communicable diseases (NCDs). For the first time recommendations have been made for the dietary intakes of carbohydrates. The EAR for CHO has been set at 100 g/day for ages 1 year and above with a RDA of 130 g/day, assuming a coefficient of variance (CV) of 15% based on variation in brain glucose utilization.

MINERALS

The present committee has done extensive deliberations on recommendations for minerals like calcium, phosphorus, zinc, selenium and iodine and have been included as separate chapters in the new document.

Calcium and Phosphorus: Calcium requirement proposed as RDA for adult man and adult woman is 1000 mg/d and is 1.5 times the value proposed by earlier expert group i.e., 600 mg/d for adult man and woman. For pregnant women, the calcium values proposed is similar to the value proposed for adult woman i.e., 1000 mg/d. For lactating woman, an additional amount of 200 mg is added to EAR of 800 mg and a total of 1000 mg has been set as EAR and adding 10% CV, the RDA is set at 1200 mg. For post-menopausal women the recommendation is 1200 mg/d.

The recommended values for phosphorus for all age groups except for infants are 1:1 ratio with calcium. For infants, it is 1.5 times the value recommended for calcium.

Magnesium: EAR was calculated by extrapolating the regression equation from the correlation of intakes and fecal losses and adding the average urinary losses. RDAs were calculated from EARs with 10% coefficient of variation. Requirements of other physiological groups were adjusted to age and growth factors. The EAR was thus estimated to 320 mg per day and RDA at 385 mg per day for adult males.

Sodium and Potassium: Specific recommendations have been made on adequate intakes for sodium and potassium for adult man and woman based on WHO (2012) recommendation. With regard to sodium due to emerging concerns on prevalence of hypertension a safe intake of 2000 mg/day which amounts to 5 g/day of salt is recommended; while an intake of 3510 mg/day is recommended for potassium. The desirable sodium:potassium ratio in mmol from the diet was fixed at 1:1.

Iron: The basis for the recommendations of iron (factorial approach) is similar to what was adopted by the previous committee. Unlike the earlier Committee which used three tier absorption for adjustment of dietary iron 3% for men, 5% for women and 8% for pregnant women, the present Committee recommends the use of only two tiers 5% (men and children) and 8% (all women), which is in conformity with the suggestion made by FAO/WHO, for developing countries and is also based on absorption data generated in India using stable isotopes. Consequently, the average requirement RDA for iron has been reduced significantly among all physiological groups. To achieve this, the committee recommended that the density of ascorbic acid in the daily diet should be at least 20 mg/1000 kcal.

Zinc: Computation of zinc requirements was done considering all the average losses of zinc through bodily fluids and additional requirements due to growth (tissue and blood volume expansion), lactation, pregnancy needs. The absolute requirements were then adjusted for bioavailability to derive

EAR. From the EAR, RDA for adult man and woman is set at 17 and 13 mg/day respectively and specific recommendations for all physiological groups are included in this report.

Copper, Chromium and Manganese: The RDA for Cu, Cr and Mn have been considered separately in view of their importance and a brief account of relevant information on the nutritional significance and suggested adequate dietary intakes for adults are provided in this report.

Selenium: The present Committee recommended 40 µg/day as adequate intake of selenium.

Iodine: Based on intake of Iodine in the diet through food and as fortified salt, the recommendation of 150 µg/day is retained for adults. The recommendations of IOM of 250 µg/day for Iodine during pregnancy, have also been adopted.

VITAMINS

Water Soluble Vitamins

Thiamine and Riboflavin: The daily intake of these vitamins is related to the energy requirements. In the absence of direct studies, the committee recommends the requirements of thiamine (men- 0.6 mg/1000 kcal; women- 0.8 mg/1000 kcal) and riboflavin (men- 0.9 mg/1000 kcal; women- 1.1 mg/1000 kcal) based on ETK-AC (1.15) and EGR-AC cut-off values (1.2), respectively for thiamine and riboflavin.

Niacin: Diet surveys from India show that the average intake of niacin is around 10 mg daily. Based on the EAR of 5.6 mg/1000 Kcals for adults, which was derived by urinary metabolite studies of niacin, 10% CV (20% 2SD) was added to EAR to derive the RDA. Individual requirements were computed based on energy requirements. The EAR (RDA) was set at 12 mg/day (14 mg/day) and 9 mg/day (11 mg/day) for sedentary men and women respectively.

Vitamin B₆: Due to paucity of reference data for different age groups in Indian scenario, expert committee 2020 calculated the vitamin B₆ EAR and RDA based on EAST-AC values for adults. For this the EAST-AC cut-off of 1.8 was considered as suggested by EFSA and the requirements were calculated based on regression analysis in relation to B₆ intakes. Based on this approach the requirement (EAR) of vitamin B₆ for 1000 kcal works out to be 0.616 mg and this was used to extrapolate to other age groups based on the energy requirements. The RDA was set at 2.1 and 1.6 for moderate active men and women respectively.

Folate: The present committee revised the requirements of folate based on some recent Indian data, which includes dietary intakes, and plasma folate and homocysteine levels as functional marker. Based on the available data on serum/plasma folate and the dietary folate intake among healthy Indian adults, the EAR was derived. The requirement to maintain normal plasma folate levels of >10=; nmol/L was considered and the RDA was calculated as 300 µg for adult men and 220 µg for adult women. Additional requirements of 300 µg/day and 100 µg/day were added respectively during pregnancy and lactation for meeting the factorial extra needs.

Vitamin B₁₂: Factorial approach was used for deriving Vitamin B₁₂ requirements and the mean daily excretion used in the previous ICMR 2010 recommendation, of 1 µg/d, was considered. Using mean bioavailability of 50% based on stable isotope kinetic studies done at St. John's Research Institute, an EAR of 2 µg/d for adults is recommended. Distribution of the requirement was calculated based on distribution of bioavailability, and the 97.5th percentile of this distribution was used to define RDA of 2.5 µg/d. For young children, as no specific data is available, an intake of 1 µg/day is suggested keeping in view of low prevalence of vitamin B₁₂ deficiency observed in 1-4y old children in the Comprehensive National Nutrition Survey (CNNS); and for school children and adolescents the adult requirement is suggested. For pregnant women, since studies have shown that the human foetus accumulates 0.1 µg/d and is required for maintaining adequate foetal growth, an additional EAR of

0.2 µg B₁₂/d is suggested adjusting for 50% absorption. With regards to lactating women the B₁₂ requirement was arrived by considering the B₁₂ content of milk and the output in first 6 months, which is around 0.4 µg/d. Adjusting for absorption an additional EAR of 0.8 µg/d is suggested.

Ascorbic acid (Vitamin C): The committee has evaluated all the available evidence on this subject and estimated the EAR and RDA based on replacement levels of body pool saturation of 900 mg, for a metabolic loss of 2.9% per day, compensated for the urinary loss (25% per day), taking absorption efficiency in Indian foods also into consideration. The EAR was set at 65mg per day and RDA at 80 mg per day for adult males. Due importance of ascorbic acid in a meal to improve iron absorption among Indians on a vegetarian diet is also emphasized while making the recommendations.

Fat Soluble Vitamins

Vitamin A: The present Committee revised the carotene conversion ratio to account for tissue conversion, based on recent knowledge, and a general conversion factor of 6:1 is recommended for all carotenoids except β-cryptoxanthine and α-carotene where a CF of 12:1 is recommended. Vitamin A requirements (RDA) for all groups were also revised upwards using factorial computation method.

Considering the recent studies on vitamin A status carried out in India, an upward revision of retinol to 900 µg is recommended during pregnancy. To ensure adequacy at least in vulnerable groups like pregnant and lactating women, the Committee has recommended that a minimum of 50% RE be drawn from animal sources.

Vitamin D: The Committee after considering the available evidence of vitamin D status decided to adopt the IOM recommendation for all age groups. Accordingly an EAR of 400 IU and an RDA of 600 IU is recommended while emphasizing the importance of outdoor physical activity as a means of achieving adequate vitamin D status in a tropical country like India.

Vitamin E & K: The requirement of alpha tocopherol suggested is 0.8 mg/ g of dietary essential fatty acids. This roughly works out to 7.5-10 mg tocopherol per day, similar to FAO/WHO recommendations depending on the edible oil used. The recommendation for vitamin K is 55 µg for adults and is in tune with recommendations of FAO/WHO.

WATER

The requirement of water was estimated based on a factorial approach, utilising the existing literature of the fluid guidelines provided by the IOM and WHO, with corrections made for body mass and energy requirement to suit the Indian context. The water required from beverages for adult man ranges from 32-58 ml per kg body mass and for woman, it ranges from 27-52 ml per kg body mass, with sedentary working group at lower end and the heavy working group at higher end of the range. For children, the requirement is greater than 60 ml per kg body mass and for adolescent boys it ranges from 47-60 ml per kg body mass, while, for girls it is 39-49 ml per kg body mass. For pregnant woman, based on the working intensity, the water required from beverages ranges from 2.1 to 3.2 litres per day. For old-age, irrespective of gender, the present consensus for water requirement from beverages is 33 ml per kg body mass for sedentary activity and 38 ml per kg body mass for moderate activity.

ANTIOXIDANTS

Realising the importance of dietary antioxidants, the committee deliberated on the information on consumption of antioxidants and recommended a minimum of 400 g/day of fruits and vegetables to obtain sufficient amounts of antioxidant nutrients such as beta-carotene, vitamin C and certain non-nutrients like polyphenols and flavonoids which may protect against chronic diseases. This should be complemented with sufficient amount of vegetable oil so as to obtain vitamin E.

SUMMARY OF EAR FOR INDIANS - 2020

Age Group	Category of work	Body Wt	Energy (**)	Fats/ Oils (visible) (#)	Protein	CHO	Cal cium	Magne sium	Iron	Zinc	Iodine	Thiamine	Ribo flavin	Niacin	Vit B6		Vit B12	Vit C	Vit A	Vit D
		(kg)	(Kcal/d)	(g/d)	(g/d)	(g/d)	(mg/d)	(mg/d)	(mg/d)	(mg/d)	(µg/day)	(mg/d)	(mg/d)	(mg/d)	(mg/d)	(µg/d)	(µg/d)	(mg/d)	(µg/d)	(IU/d)
Men	Sedentary	65	2110	25	42.9		800	320	11	14.0	95	1.2	1.6	12	1.6	250	2	65	460	400
	Moderate		2710	30		100						1.5	2.1	15	2.1					
	Heavy		3470	40								1.9	2.7	19	2.6					
Women	Sedentary	55	1660	20	36.3		800	270	15	11.0	95	1.1	1.6	9	1.6	180	2	55	390	400
	Moderate		2130	25		100						1.4	2.0	12	1.6					
	Heavy		2720	30								1.8	2.6	15	2.1					
Women	Pregnant woman	55 + 10	+ 350	30	+7.6 (2 nd trimester) +17.6 (3 rd trimester)	135	800	320	32	12.0	180	1.6	2.3	+2	1.9	480	+0.2	+10	406	400
Lactation 0-6m 7-12m			+600	30	+13.6	155	1000	270	16	12.0	200	1.7	2.5	+4	+0.22	280	+0.8	+40	720	400
			+520		+10.6	155						1.7	2.4	+4	+0.16	280				
Infants	0-6 m*	5.8	550	-	6.7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	6-12m	8.5	670	25	8.8	-	-	-	2	2.0	130	-	-	-	0.5	71	1	-	170	-
Children	1-3y	11.7	1010	25	9.2	100	400	111	6	2.5	65	0.6	0.8	6	0.8	90	1	22	180	
	4-6y	18.3	1360	25	12.8	100	450	131	8	3.7	80	0.8	1.1	8	1.0	111	1	27	240	400
	7-9 y	25.3	1700	30	19.0	100	500	178	10	4.9	80	1.0	1.3	10	1.3	142	2	36	290	
Boys	10-12y	34.9	2220	35	26.2	100	650	223	12	7.0	100	1.3	1.7	12	1.7	180	2	45	360	400
Girls	10-12y	36.4	2060	45	26.6	100	650	214	16	7.1	100	1.2	1.6	12	1.6	186	2	44	370	400
Boys	13-15y	50.5	2860	50	36.4	100	800	294	15	11.9	100	1.6	2.2	16	2.2	238	2	60	430	400
Girls	13-15y	49.6	2400	35	34.7	100	800	270	17	10.7	100	1.3	1.9	13	1.8	204	2	55	420	400
Boys	16-18y	64.4	3320	40	45.1	100	850	338	18	14.7	100	1.9	2.5	19	2.5	286	2	69	480	400
Girls	16-18y	55.7	2500	35	37.3	100	850	279	18	11.8	100	1.4	1.9	14	1.9	223	2	57	400	400

*: AI; **: There is no RDA for energy. The EAR is equivalent to the Estimated Energy Requirement (EER); #: Visible fat requirement is in proportion to EER;

SUMMARY OF RDA FOR INDIANS – 2020

Age Group	Category of work	Body Wt	Protein	CHO	Cal cium	Magne sium	Iron	Zinc	Iodine	Thiamine	Ribo flavin	Niacin	Vit B6	Folate	Vit B12	Vit C	Vit A	Vit D	
		(kg)	(g/d)	(g/d)	(mg/d)	(mg/d)	(mg/d)	(mg/d)	(µg/day)	(mg/d)	(mg/d)	(mg/d)	(mg/d)	(µg/d)	(µg/d)	(mg/d)	(µg/d)	(IU/d)	
Men	Sedentary	65	54.0	130	1000	385	19	17	150	1.4	2.0	14	1.9	300	2.5	80	1000	600	
	Moderate									1.8	2.5	18	2.4						
	Heavy									2.3	3.2	23	3.1						
Women	Sedentary	55	45.7	130	1000	325	29	13.2	150	1.4	1.9	11	1.9	220	2.5	65	840	600	
	Moderate									1.7	2.4	14	1.9						
	Heavy									2.2	3.1	18	2.4						
Women	Pregnant woman	55 + 10	+9.5 (2 nd trimester) +22.0 (3 rd trimester)	175	1000	385	40	14.5	250	2.0	2.7	+2.5	2.3	570	+0.25	+15	900	600	
	Lactation 0-6m				200	1200	325	23	14	280	2.1	3.0	+5	+0.26	330	+1.0	+50	950	600
	7-12m										2.1	2.9	+5						
Infants	0-6 m*	5.8	8.1	55	300	30	-	-	100	0.2	0.4	2	0.1	25	1.2	20	350	400	
	6-12m	8.5	10.5	95	300	75	3	2.5	130	0.4	0.6	5	0.6	85	1.2	27	350	400	
Children	1-3y	11.7	11.3	130	500	135	8	3.0	90	0.7	0.9	7	0.9	110	1.2	27	390	600	
	4-6y	18.3	15.9	130	550	155	11	4.5	120	0.9	1.3	9	1.2	135	1.2	32	510		
	7-9 y	25.3	23.3	130	650	215	15	5.9	120	1.1	1.6	11	1.5	170	2.5	43	630		
Boys	10-12y	34.9	31.8	130	850	270	16	8.5	150	1.5	2.1	15	2.0	220	2.5	54	770	600	
Girls	10-12y	36.4	32.8	130	850	255	28	8.5	150	1.4	1.9	14	1.9	225	2.5	52	790	600	
Boys	13-15y	50.5	44.9	130	1000	355	22	14.3	150	1.9	2.7	19	2.6	285	2.5	72	930	600	
Girls	13-15y	49.6	43.2	130	1000	325	30	12.8	150	1.6	2.2	16	2.2	245	2.5	66	890	600	
Boys	16-18y	64.4	55.4	130	1050	405	26	17.6	150	2.2	3.1	22	3.0	340	2.5	82	1000	600	
Girls	16-18y	55.7	46.2	130	1050	335	32	14.2	150	1.7	2.3	17	2.3	270	2.5	68	860	600	

* AI

**SUMMARY OF RECOMMENDED INTAKES FOR
OTHER MINERALS AND TRACE ELEMENTS**

SNo.	Minerals/Trace Element	Recommended intake
1	Phosphorous	1000 mg/day
2	Sodium	2000 mg/day
3	Potassium	3500 mg/day
4	Copper	2 mg/day
5	Manganese	4 mg/day
6	Chromium	50 µg/day
7	Selenium	40 µg/day

TOLERABLE UPPER LIMIT (TUL) FOR NUTRIENTS

Age Group	Category of work	Protein	Cal cium	Magne sium	Iron	Zinc	Iodine	Niacin	Vit. B6	Folate	Vit. C	Vit. A	Vit. D
		(PE ratio)	(mg/d)	(mg /d)	(mg /d)	(mg /d)	(µg/day)	(mg/d)	(mg/ d)	(µg/d)	(mg/d)	(µg/d)	(IU/d)
Men	Sedentary	<40%	2500	350	45	40	1100	35	100	1000	2000	3000	4000
	Moderate												
	Heavy												
Women	Sedentary	<40%	2500	350	45	40	1100	-	-	1000	2000	3000	4000
	Moderate												
	Heavy												
Pregnant woman	Pregnant woman	<30%	2500	350	45	40	1100	-	-	1000	2000	3000	4000
	Lactation 0-6m 7-12m	<40%	2500	350	45	40	1100	-	-	1000	2000	3000	4000
Infants	0-6 m	<15%	-	-	40	4	-	-	-	-	-	600\$	1000
	6-12m	<15%	-	-	40	5	-	-	-	-	-	600\$	1500
Children	1-3y	<15%	1500	65	40	7	200	-	-	-	350	600\$	2500
	4-6y	<15%	2500	110	40	12	300	-	-	-	550	900\$	3000
	7-9 y	<15%	2500	110	40	12	400	-	-	-	800	900\$	3000
Boys	10-12y	<15%	3000	350	40	23	600	-	-	600-800 (9-17y)	1050	1700	4000
Girls	10-12y	<15%	3000	350	40	23	600	-	-	-	1300	1700	4000
Boys	13-15y	<15%	3000	350	45	34	900	-	-	-	1550	2800	4000
Girls	13-15y	<15%	3000	350	45	34	900	-	-	-	1800	2800	4000
Boys	16-18y	<15%	3000	350	45	34	1100	-	-	-	1950	2800	4000
Girls	16-18y	<15%	3000	350	45	34	1100	-	-	-	2000	2800	4000

\$: adopted from IOM

3. REFERENCE BODY WEIGHTS

Age, gender, body-structure (height and weight) and body tissue composition largely determine the nutrient requirement of an individual. In growing infants, children, adolescents and adults the body weight, height and tissue composition are indicators of their health and nutritional status. The nutrient requirements are thus adjusted for body weight (a proxy for the built of an individual) in kilograms to account for the wide variation in body sizes observed at a population level. A reference body weight represents what is optimal for age and sex, of an apparently healthy representative population with favourable conditions for growth and development.

The Expert Committee of the ICMR (1989) used anthropometric data of an elite population of India. These data were generated by NIN surveys on children from high socio-economic backgrounds, those studying in public schools or in IIT's in different parts of the country^{1,2}. In 2010, the ICMR expert committee decided to use data on height and weight from the National Nutrition Monitoring Bureau (NNMB) surveys carried out in the year 2000-01, from rural households across 9 states, in order to have a representative data set for the Indian population. The 95th centile values of weights and heights for a given age and sex, from this national survey, were taken to be representative of a well-nourished population and considered as reference values for India. These weights were used to compute the RDA of nutrients (per kg body weight per day), for all age and gender groups, except for children (0-3 years). For children 0-3 years, the WHO standard (2006) for weight and height was used³.

The present committee has considered an alternative approach to define the reference body weight for the Indian population. The currently available, nationally representative datasets such as the National Family Health Survey - 4 (NFHS-4, 2015-16), National Nutrition Monitoring Bureau (NNMB, 2015-16), the World Health Organization (WHO, 2006-07), and the Indian Academy of Pediatrics (IAP 2015) were evaluated to arrive at acceptable reference body weight values through the lifespan. The details are explained in the following sections.

3.1 – Comparison of anthropometric data from national datasets

To define the reference body weights for the population, the ICMR-NIN 2020 expert committee used or compared different datasets with height and weight measurements in different age groups.

3.1.1 Children aged 0-5y

For the age range from 0-5y, the WHO growth standard was selected, since an Indian city (New Delhi) was included in the multicenter assessment. The WHO data was from the multi-center growth reference study (MGRS, 0-60 months)³.

3.1.2 School children and adolescents

For school children and adolescents, the WHO growth standards (2006-07) and the revised IAP growth standards (2015) were compared. The WHO data was from the National Center for Health Statistics (5-18y, NCHS, 1977) dataset⁴. The IAP presented revised growth charts for children and adolescents (5-18 y) in 2015, with data from upper and middle socio-economic status children from 14 different cities⁵. The body weights and heights from these datasets are provided in Table 3.1 and 3.2.

Table 3.1: Body weights (kg) of children and adolescents (5-18y) reported from IAP and WHO growth standards

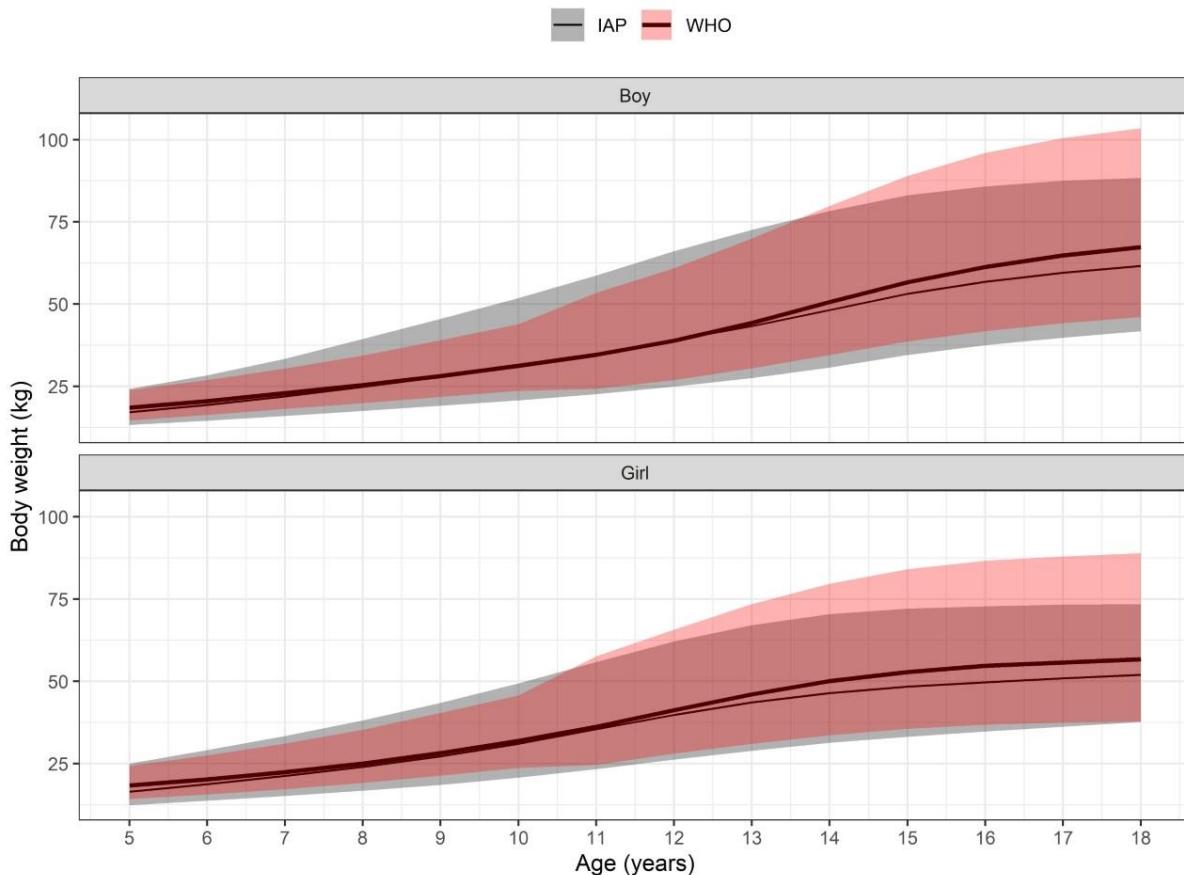
Age (y)	Boys		Girls	
	IAP 2015	WHO 2007	IAP 2015	WHO 2007
5	17.1	18.5	16.4	18.3
6	19.3	20.5	18.7	20.2
7	21.9	22.9	21.2	22.4
8	24.8	25.4	24.0	25.0
9	27.9	28.1	27.2	28.2
10	31.1	31.2	31.0	31.9
11	34.7	34.6	35.4	36.2
12	39.0	38.9	39.8	41.1
13	43.3	44.3	43.6	46.0
14	48.2	50.6	46.4	50.1
15	53.1	56.5	48.4	52.8
16	56.8	61.3	49.7	54.7
17	59.5	64.8	50.9	55.7
18	61.6	67.3	52.0	56.7

Table 3.2: Height (cm) of children and adolescents (5-18y) reported from IAP and WHO datasets

Age (y)	Boys		Girls	
	IAP 2015	WHO 2007	IAP 2015	WHO 2007
5	108.9	110	107.5	110
6	114.8	116	113.5	115
7	120.7	122	119.4	121
8	126.4	127	125.4	127
9	131.8	133	131.4	133
10	137.2	138	137.4	139
11	142.7	143	143.3	145
12	148.4	149	148.4	151
13	154.3	156	152.2	156
14	159.9	163	154.7	160
15	164.5	169	155.5	162
16	168.1	173	156.9	163
17	171.0	175	157.4	163
18	173.6	176	157.8	163

The 3rd, 50th and 97th percentiles values for body weights were plotted against age (Figure 3.1), which showed a considerable overlap between the IAP and WHO data, suggesting that either of these datasets could be used as reference for the Indian population.

Figure 3.1: Comparison of IAP and WHO reference body weights for children and adolescents (5-18y)



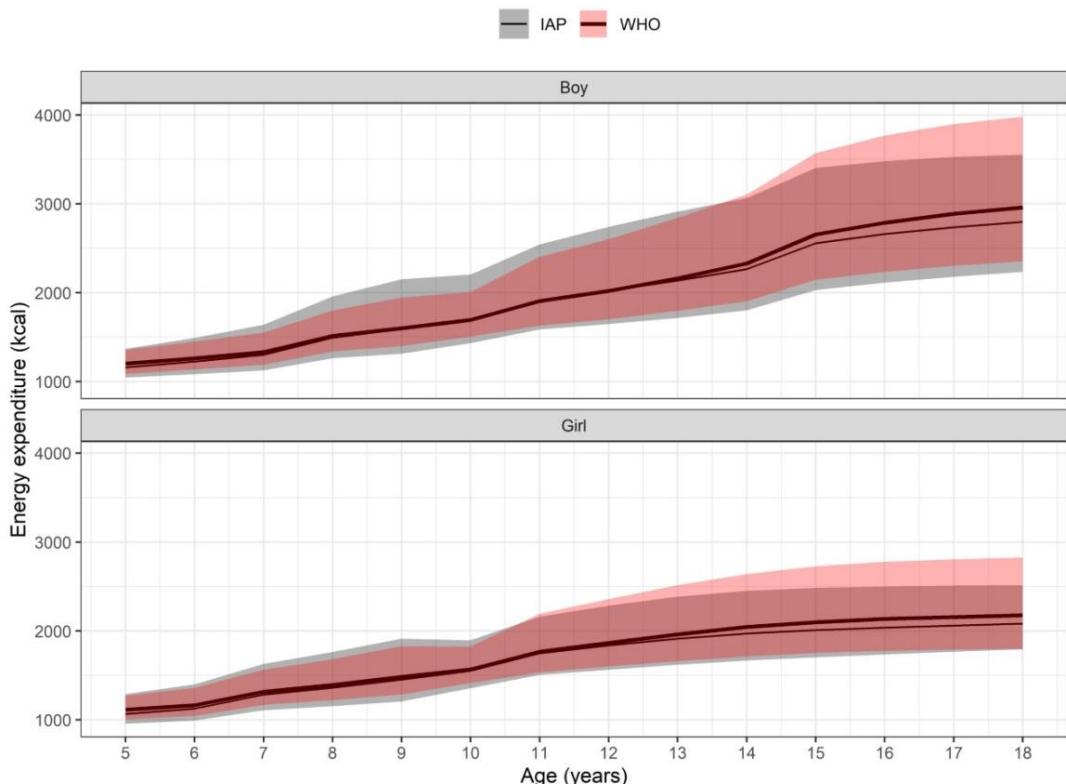
Legend: The line represents mean body weights (kg) and shaded areas represent the range between 3rd and 97th percentiles for each reference

3.1.2.1 Sensitivity analyses with macro- and micronutrient requirement

To test the comparability of references derived from either of these data sets, sensitivity analyses for the energy and iron requirement (to represent macro- and micro-nutrient requirements respectively) were performed, using body weights of 5-18 y from both databases. Figure 3.2 shows the mean (confidence interval, 95%CI) of the energy requirement respectively for ages 5-18 y, when IAP or WHO body weights were used as the reference.

The difference in the energy requirement ranged from 0 to 160 kcal/d across ages, which was less than 5% of average daily requirement of energy for each age. The slightly higher difference at higher ages was due to corresponding increased body weights as per the WHO reference. For the iron requirement, the same analysis showed that the difference in requirement ranged from 0.2 to 1.6 mg/d across ages for boys while for girls it ranged from 0.2 to 1.1 mg/d, which was less than 5% of the daily iron requirement for 5-14y. From 15-18y, this difference was less than 8%. The slightly higher difference at higher ages was because at these ages, the IAP reference weights were lower compared to the WHO reference. To err on the side of caution, since these were small differences, it was decided to use the WHO reference values for children and adolescents.

Figure 3.2: Comparison of energy requirement for children and adolescents based on IAP and WHO body weights



Legend: The line represents the energy requirement (kcal/d) and shaded region represents 95% CI

3.1.3 Adults

Anthropometric data for Indian adults are available from two recent national datasets, the NNMB (2015-16) and the NFHS-4^{6,7}. The NFHS-4 provides information on population's health and nutritional status across Indian states and Union territories. The NNMB survey was conducted on urban population from 16 states - Andhra Pradesh, Gujarat, Karnataka, Kerala, Madhya Pradesh, Maharashtra, Orissa, Tamil Nadu, Uttar Pradesh, West Bengal, Assam, Andaman and Nicobar Islands, Bihar, Rajasthan, Puducherry and Delhi⁸. The main objective of the survey was to assess the diet and nutritional status of urban population and define the prevalence of obesity, hypertension, diabetes and dyslipidaemia among urban men and women. The anthropometric measurements are available for all individuals in 3600 households. From these data, the reference height was taken as 95th centile for adult male and female, and with normal BMI range of 18.5-22.9 kg/m², the reference body weight was calculated (Table 3.3 and 3.4).

Table 3.3: Data on height (cm) of adult Indian population derived from NNMB urban survey 2016

Age (y)	Males				Females			
	n	Mean	Median	95 th	n	Mean	Median	95 th
19-29	13000	167.2	167.5	178.8	15888	153.1	153.1	163.2
30-39	12401	166.2	166.4	177.5	16436	152.4	152.4	162.2
40-49	11758	165.1	165.2	176.0	14584	151.8	151.8	161.4
50-59	8622	164.1	164.2	175.0	405	151.7	151.7	160.5
60 & Above	8457	162.3	162.5	173.1	109	149.4	149.0	160.8

Table 3.4: Reference heights and weights for adult males and females

Gender	Measure	Valid N	Mean	SD	Median	95 th	Proposed reference
Male	Height	170	177.5	0.3	177.5	178.0	177.0
	Weight	170	65.4	4.0	65.6	71.8	65.0
	BMI	170	20.78	1.27	20.83	22.76	20.75
Female	Height	184	162.4	0.3	162.4	163.0	162.0
	Weight	184	54.7	3.5	54.7	60.0	55.0
	BMI	184	20.74	1.33	20.73	22.75	20.95

Note: The reference body weight was calculated based on reference height derived from NNMB urban survey 2016 (177-178 cm for male and 162-163 cm for female) and BMI in the range of 18.5-22.9 kg/m²

Further, to validate the use of the NNMB data, a population subset was selected from the NFHS-4(2015-16) dataset using a strict criterion based on wealth, education, Body Mass Index (BMI), Blood Pressure (BP) and blood parameters like haemoglobin (Hb) and sugar levels to define an apparently healthy population. A regression equation for body weight using the predictors such as wealth index, year of education, Body Mass Index (BMI), anaemia (measured using Hb), blood sugar, blood pressure and age was developed, which explained 85% variation of body weight for males and 78% for females. The reference body weight was estimated from this regression equation for different ages, setting wealth index at the richest class, year of education at graduation level, BMI as 21 kg/m², Hb at 12 g/dl for female and 13 g/dl for male, normal BP levels and blood sugar at 100 mg/dl.

The predicted body weights (confidence interval) were found to be 54.1 (3.3) kg and 64.4 (3.91) kg for females and males respectively. The predicted reference body weight for males and female from NFHS-4 was similar to the proposed body weights using NNMB data, therefore the NNMB data was considered for reference body weights as it is the most recent available dataset.

3.2 Reference body weights

The present expert committee recommended the use of WHO reference body weights for children from 0-18 y, as substantiated by the IAP and WHO comparison and sensitivity analysis for energy and iron requirement, and in confirmation with international standards.

For adult women and men, the weight derived from a normal BMI range (18.5-22.9kg/m²) for the 95% height, from the NNMB data was selected, as the values predicted from the NFHS-4 data were similar to these measured values. The summary of recommended body weights across ages is presented in Table 3.5. Since the requirements are grouped for different ages, a summary of body weights for different age groups is present in the Table 3.6. Reference body weight for normal BMI (21) for corresponding height is given in Table 3.7.

Table 3.5: Recommended body weights across ages for Indian population

Category	Age (y)	Body Weight (kg)	
		Boys	Girls
Infants	0-6 m	6.1	5.6
	6-12 m	8.8	8.2
Children	1	10.9	10.2
	2	13.3	12.7
	3	15.3	15.0
	4	16.5	16.0
	5	18.5	18.3
	6	20.5	20.2
	7	22.9	22.4
	8	25.4	25.0
	9	28.1	28.2
Adolescents	10	31.2	31.9
	11	34.6	36.2
	12	38.9	41.1
	13	44.3	46.0
	14	50.6	50.1
	15	56.5	52.8
	16	61.3	54.7
	17	64.8	55.7
	18	67.3	56.7
Adult Men	>18	65.0	
Adult Women	>18	55.0	

Table 3.6: Reference body weights for Indian population in specified age groups

Age Group	Category	Body weights (kg)
Men	>18 y	65.0
Women	>18 y	55.0
Infants	0-6 m	5.8
	6-12m	8.5
Children	1-3y	11.7
	4-6y	18.3
	7-9 y	25.3
Boys	10-12y	34.9
Girls	10-12y	36.4
Boys	13-15y	50.5
Girls	13-15y	49.6
Boys	16-18y	64.4
Girls	16-18y	55.7

Table 3.7: Recommended weights for corresponding heights at BMI=21

Height (cm)	Weight (Kg)	Height (cm)	Weight (Kg)
130	35.5	161	54.4
131	36.0	162	55.1
132	36.6	163	55.8
133	37.1	164	56.5
134	37.7	165	57.2
135	38.3	166	57.9
136	38.8	167	58.6
137	39.4	168	59.3
138	40.0	169	60.0
139	40.6	170	60.7
140	41.2	171	61.4
141	41.8	172	62.1
142	42.3	173	62.9
143	42.9	174	63.6
144	43.5	175	64.3
145	44.2	176	65.0
146	44.8	177	65.8
147	45.4	178	66.5
148	46.0	179	67.3
149	46.6	180	68.0
150	47.3	181	68.8
151	47.9	182	69.6
152	48.5	183	70.3
153	49.2	184	71.1
154	49.8	185	71.9
155	50.5	186	72.7
156	51.1	187	73.4
157	51.8	188	74.2
158	52.4	189	75.0
159	53.1	190	75.8
160	53.8		

References

1. Raghavan KV, Singh D, Swaminathan MC. Heights and weights of well-nourished Indian school children. Indian Journal of Medical Research. 1971; 59(4):648-54.
2. Rao DH, Satyanarayana K, Sastry JG. Growth pattern of well-to-do Hyderabad pre-school children. Indian Journal of Medical Research. 1976; 64(5):629-38.
3. World Health Organization. WHO child growth standards: length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: methods and development. World Health Organization; 2006.
4. World Health Organization. WHO reference 2007: growth reference data for 5-19 years. World Health Organization; 2007.
5. Khadilkar V, Yadav S, Agrawal KK, Tamboli S, Banerjee M, Cherian A, Goyal JP, Khadilkar A, Kumaravel V, Mohan V, Narayanappa D. Revised IAP growth charts for height, weight and body mass index for 5-to 18-year-old Indian children. Indian pediatrics. 2015 Jan 1; 52(1):47-55.
6. National Nutrition Monitoring Bureau. Diet and nutritional status of rural population. National Nutrition Monitoring Bureau Technical Report No. 21. 2002.
7. IIPS I. National Family Health Survey (NFHS-4), 2015–16. International Institute for Population Sciences (IIPS), Mumbai, India. 2017.
8. National Nutrition Monitoring Bureau. Diet and nutritional status of rural population. National Nutrition Monitoring Bureau Technical Report No. 21. 2002.

4. ENERGY

INTRODUCTION

Body needs energy for maintaining body temperature and metabolic activity and for supporting physical work and growth. Energy is a fuel provided by the macronutrients consumed in the diet: carbohydrate, fat and protein and in some instances, alcohol. The recommended energy allowances are designed to provide sufficient energy that will support satisfactory growth in infants and children and maintain appropriate body size and composition associated with good health in adults. The factors which influence energy needs are age, gender, body size, level of physical activity and, to some extent, climate and altered physiological status such as pregnancy and lactation.

To maintain energy balance, energy intake must be equal to the energy output, which corresponds to a steady state, and results in a stable and healthy body weight. The logical extension of this concept is that if the stable body weight and level of physical activity of an individual are known or defined, then energy balance can be achieved at a single level of intake; the additional needs of the individual (as in the case of children, or during pregnancy and lactation) can be taken care of by specific additional prescribed allowances. This level of intake of an individual, at which he/she remains in steady state or in energy balance, maintaining predetermined levels of body weight and physical activity, is considered to be the individual's energy requirement. It is not essential that a person should be in energy balance on a day-to-day basis. However, over a period of a week or a fortnight, the individual can be in energy balance, that is, his/her daily energy expenditure and daily energy intake averaged over this period should be in a state of balance. Fat, the body's energy store, can take care of any imbalance in daily energy intake and energy expenditure. This definition of individual's energy requirement can then be extended to spell out the energy needs of a group (or a community or a nation), if the composition of the community, age, gender, body weight and habitual pattern of physical activity are known. On the other hand, excess energy is stored in the body leading to positive energy balance and eventually causing overweight or obesity.

The basic concept of estimating energy requirements is fundamentally different from that of protein and other nutrients. Unlike energy, protein is not stored in the body as a reserve and the daily protein intake should match the daily protein metabolism to satisfy a man's daily protein requirements. Further, the recommended dietary intake of protein and other nutrients is given as either the Estimated Average Requirement (EAR) or the safe allowance, which is called the Recommended Daily Intake (RDI), and which covers additional allowances demanded by intra-individual and inter-individual variations. The RDI is an intake at which the risk of inadequacy is less than 2.5%. In the case of energy, however, since energy can be stored as fat when its intake is in excess of the requirement, only the average requirement is used, called the Estimated Energy Requirement (EER). The requirement represents the average daily requirements corresponding to daily average energy expenditure of an individual.

4.1. Energy requirement: Definition and units

The energy requirement of an individual is defined as follows:

The level of energy intake from food that balances energy expenditure when the individual has a body size and composition and level of physical activity, consistent with long-term good health, also allowing for maintenance of economically essential and socially desirable activity. In children and pregnant and lactating women, it includes the energy needs associated with the deposition of tissues during growth, or secretion of milk at rates consistent with good health¹.

The unit of energy, which has been in use in nutrition for a long time, is Kilocalories (kcal). However, recently the International Union of Sciences and International Union of Nutritional Sciences (IUNS) have adopted ‘Joule’ as the unit of energy in the place of kcal. These units are defined as follows.

Joule, a physical unit of energy, is defined as the energy required to move 1kg of mass by 1 meter by a force of 1 Newton acting on it (One Newton is the force needed to accelerate one kg mass by 1 meter per sec²).

Kilo calories (kcal) is defined as the heat required to raise the temperature of one kg of water by 1°C from 14.5°C to 15.5°C. The unit kcal is still popularly used. Both units are used in defining human energy requirement in this report.

The relationship between the two units of energy is as follows:

1 kcal	=	4.184 KJ (Kilo Joule)
1KJ	=	0.239 kcal
1000 kcal	=	4184 KJ = 4.18 MJ (Mega Joule)
1 MJ	=	239 kcal

4.2. Requirements of energy for Indians – A history

The recommended dietary allowances of energy for Indians were first proposed by the Nutrition Advisory Committee (NAC) of the Indian Research Fund Association (IRFA) in 1944². These recommendations were based on the proposals of the Health Committee of the League of Nations made in 1935 but adapted to the lower body weights of Indian adults which were assumed to be 55 kg and 45 kg for the male and female respectively. The 1944 recommendations for energy for Indians² were revised subsequently by the NAC of the ICMR in 1958³. The factorial approach (refer section 4.4) employed in deriving the energy requirements of Indian adults in 1958 was similar to that used by the 1957 FAO/WHO Expert Group on Energy Requirements⁴. For this purpose, the normal man and woman were defined to be of 55 kg and 45 kg body weight respectively and the total daily energy requirements were defined for three categories of activities, namely, sedentary, moderate and heavy. These requirements were derived by employing the factorial approach. These energy requirements of Indian adult man were computed using both Indian and Western data. While the available data on Basal Metabolic Rate (BMR)^{5,6} of Indians were used for computing the energy cost of sleep, the activity component from different daily activities was, however, computed from extensive Western data on energy cost of different activities, since data available then on Indian subjects were limited. Even these limited observations available then on Indians for energy cost of activities, when adjusted for differences in body weights, were comparable to Western observations. Hence, the published values in the West for energy cost of various activities were converted per unit body weight and used in computing the energy cost of different daily activities of Indian Reference Man and Woman in arriving at their daily energy requirements³.

The 1958 RDA for energy for Indians suffered from several shortcomings. Dr. V. N. Patwardhan, the author of the 1958 Report on Energy Requirements of Indians³ recognized this and stated that “it would be difficult to evaluate correctly the total energy requirement of adults in India, for much work had not been done on Indian subjects”. He further stated that attempts to fix total energy requirement would therefore suffer from lack of adequate scientific material, particularly, if attempt is made to recommend ad-hoc allowances for light, moderate and heavy work. Energy allowances for Indians recommended in 1958 suffer from the following limitations, which indicate that the 1958 figures for energy requirements of Indians are possibly an overestimate, particularly, in the case of heavy activity category -

- i. The estimate of energy expenditure during non-occupational activity appeared to be rather high due to the use of higher values for some of the non-occupational activities; no distinction was made in this respect between sedentary category on the one hand and the moderate and heavy activity categories on the other. The latter categories would normally spend less energy during non-occupational activity period than a sedentary person.
- ii. The energy expenditure for occupational activity involving heavy work appeared to be an overestimation. This was because the average energy cost of heavy activity used in the computation of energy expenditure i.e. 5 kcal/kg/h corresponded to the upper limit of the rate of energy expenditure rather than to the practical level of average rate of energy expenditure. From the available evidence based on the observed energy expenditure of miners⁷, it cannot be higher than 4.5 kcal/kg/h. It is also reported that the sustained activity involving heavy work can be carried out only at 35% VO₂ max⁸. Energy allowances recommended in 1958 for heavy activity category are more appropriate for exceptionally heavy activity category like rickshaw pullers, underground miners, dock workers etc., rather than for many of the usual types of heavy work like earth diggers, manual agricultural labourers, stone cutters etc. Further, the energy requirement recommended in 1958 for heavy work was not substantiated by subsequent observation on Indian stone cutters⁹.
- iii. The 1958 computation of daily energy requirement assumed that the level of daily activity was the same throughout the year without taking into consideration the holidays in the case of individual workers and non-agriculture slack season in case of rural workers. During such periods the energy expenditure would more appropriately correspond to sedentary activity. If these seasonal variations in intensity of activity are taken into consideration and the daily energy expenditure is averaged over the entire year, it will roughly correspond to 75% of the energy spent during active working period or season: FAO, 1957 suggested energy requirement in terms of average for a year⁴.

Considerable additional information and newer approaches for deriving energy requirements have emerged after 1980. Revised approaches have been used in 1985¹⁰ and 2004¹¹ by FAO/WHO/UNU. Expert Consultants of ICMR Expert Group revised energy requirement for Indians in 1989¹². In 1989, the ICMR Expert Group adopted the procedure of 1985 FAO/WHO/UNU Expert Consultations¹⁰ that is, using BMR factors for arriving at the energy requirements of Indian Man and Woman from the ICMR recommendations in 2010, the reference weight of an Indian reference man and woman was changed 60 kg and 55 kg respectively based on weight and height collected across 16 states. The details of factors used in estimating energy requirement is described in section 4.4 and the same was used in the ICMR, 2010 recommendations¹³.

4.3. Energy requirements: The current approach

Currently, it is recommended that energy requirement must be assessed in terms of energy expenditure rather than in terms of energy intake. This is because of several reasons. Energy intakes that are derived from dietary recalls are prone to reporting errors as well as many assumptions that relate to converting cooked food portions to energy. The energy intake also is known to vary substantially from day to day; on some days, it may be above the energy expenditure and sometimes, below it. Body energy reserves (fat) help to maintain normal energy expenditure over short periods even when the daily intake is below expenditure. Over a period of time, however, adults tend to maintain energy balance and constant body weight.

It has been emphasized that an analysis of energy intake data is also not helpful since it is possible to have a grossly inadequate intake by individuals for maintaining normal obligatory energy expenditure, lose weight and become substantially underweight. This might happen particularly

among large population groups in underdeveloped and developing countries. On the other hand, energy intake above the energy expenditure is harmful leading to overweight and obesity and is associated with chronic disorders. This may be found among the populations of affluent countries, or affluent sub-populations in developing countries where access to plenty of food is available.

4.4. Determination of energy requirement

The recommended dietary intake for energy is intended for a healthy, well-nourished and active population. The assessment of energy expenditure is a logical approach, where one can specify the energy requirements in terms of energy output for productive work and leisure activity of adults and tissue deposition in infants, children and during pregnancy and milk secretion during lactation. This does imply that there is a need to specify an appropriate body weight of the individual as well as the quantum of physical activity that is considered ‘desirable’ for the same individual. Energy intake far above the actual requirement is harmful, which may lead to obesity related complications. On the other hand, energy intakes far below requirement leads to undernutrition and loss of body weight. Hence, in contrast to many other nutrients, like protein and vitamins, no ‘safe allowances’ are recommended in the case of the energy requirement, but only the ‘average requirement’ is defined.

There are three important terms while defining energy expenditure using physical activity estimations. These are – Physical Activity Ratio (PAR), Physical Activity Level (PAL) and Total Energy Expenditure (TEE). The physical activity ratio (PAR) is expressed as the ratio of the energy cost of an individual activity per minute to the cost of the basal metabolic rate (BMR) per minute.

$$\text{Physical Activity Ratio (PAR)} = \frac{\text{Energy cost of an activity per minute}}{\text{Energy cost of basal metabolism per minute}}$$

The PAR is unit-less, and the distinct advantage of expressing energy expenditure of an activity in terms of PAR values relates to its use for both sexes, at all ages and at all body sizes. This is because these covariates appear in both the numerator and denominator and cancel out. However, this might not be true. In studies with subjects of different body weight, it was found that the PAR increased for the same activity, when measured in heavier individuals¹⁴. In an Indian study, Kuriyan *et al* found that the PAR increased by about 15% in women with a BMI > 25 kg/m² compared to those with a BMI < 18 kg/m², for moderate activities¹⁵. Therefore, while in principle, a constant PAR for specific activities is used for all ranges of body weight, it is important to consider the possibility that in lighter individuals, the PAR may be less. More data are required in this regard.

At present, given the sporadic nature of data (in terms of covering all activities in different genders and age groups) that is specific to a country, PAR values are taken from a detailed table of PAR values for different activities available in the FAO/WHO/UNU 2004 report¹¹. However, for reasons given above, where available, the PAR values for Indians in different activities are used (studies summarized in Table 4.1 and 4.2). These measurements were made by indirect calorimetry for each activity, however, in many studies, the BMR was predicted by the 1985 WHO/FAO/UNU equation¹⁰. Measurements of PAR have also been attempted using heart rate monitoring (HRM) (see section 4.4.1), however, unless a calibration of the individual subject’s heart rate to a range of activity is available, this method should not be used for PAR estimation. This is because the response or slope of the heart rate response to a range of activities will vary by individual, depending on a host of factors such as age, gender and previous fitness. In addition, the heart rate is also known to ‘drift’ upward during activities when they are carried out for intervals over 10 minutes and can considerably alter calculated PAR values¹⁶.

A specific note should be made on the PAR of heavy activities, as they are critical to the prediction of the energy requirement in heavy workers. The energy cost of the activity varies and based on the intensity of the activity performed. The average PAR of heavy activities is summarized in Table 4.3. This is based on the studies which measured energy cost of activity using indirect calorimetry. The studies have been summarized in Table 4.4, 4.5, 4.6, 4.7.

From the table 4.3, it is clear that there is a wide range for PAR values and these are based on the intensity of the activity performed. In order to estimate the energy expenditure, the actual intensity of exercise needs to be considered. Also, special consideration should be given to the individual working for more than 8 hours a day as performing heavy activity for longer duration with negative energy balance is not sustainable. When an individual performs heavy activity (on a treadmill for example) for longer duration without rest, the energy metabolism shifts to anaerobic state and reaches a point at which the individual has to stop the heavy activity. This can be overcome by providing rest periods during the activity. The amount of rest periods depends on the type of activity, and the environment (heat, humidity, position of the body etc.,) in which it is undertaken. However, a simple physiological framework can be obtained from exercise studies. Rodahl *et al* observed that an individual can perform heavy activity without difficulty for 6 h provided they rest for 15 min in every hour of exercise³³

Table 4.1: PAR values for adult Indian males from different studies^{11,13,15, 17-19}

Activities	FAO/WHO / UNU (2004)	ICMR (2010) ^a	Banerjee <i>et. al.</i> , (1972)	Bandyopadhyay <i>et. al.</i> , (1980)	Kanade <i>et. al.</i> , (2001)	Kuriyan <i>et. al.</i> , (2006)
Sleeping	1.00	1.00	-	-	1.00	1.00
Lying resting	1.20	-	-	1.07	-	-
Sitting	1.20	1.50	1.06	1.16	1.25	1.22
Standing	1.40	-	1.43	1.23	1.46	1.29
Personal care (dressing, bathing etc.)	2.40	2.30	-	1.64	-	-
Eating	1.40	1.50	-	1.16	-	-
Household work (general)	2.80 ^b	2.50	-	-	-	-
Light leisure activity	-	1.40	-	-	-	-
Desk-work (sitting and writing)	1.40	1.50	1.14	1.36	-	1.32
Sitting and reading			1.06	1.28		
Ironing	3.50	-	-	-	-	1.64
Sweeping	-	-	-	-	-	3.67
Dusting	-	-	-	-	-	1.56
Cycling	5.60	-	-	-	-	3.33
Walking at 3-2 km/hr	2.80 ^c	2.00	3.07	2.62	-	3.06
Walking at 4-8 km/hr	3.80 ^d	3.20	-	-	-	3.88
Running (7-9 km/hr)	6.34	-	6.34	-	-	-

^a PAR values are same for both males and females

^b Given only for females; ^c Walking slow; ^d Walking quickly

Table 4.2: PAR values for adult Indian females from different studies^{11,13,15,20-22}

Activities	FAO/ WHO/ UNU (2004)	ICMR (2010) ^a	Banerjee <i>et. al.</i> , (1972)	Oberoi (1983)	Sujatha <i>et. al.</i> , (2000)	Kuriyan <i>et. al.</i> , (2006)	Rao <i>et. al.</i> , (2007)
Sleeping	1.00	1.00	-	-	1.00	1.00	-
Lying resting	1.20	-	-	-	1.00	-	0.84
Sitting	1.20	1.50	1.05	-	1.06	1.21	1.01
Standing	1.50	-	1.37	-	1.13	1.30	1.15
Personal care (dressing, bathing etc.)	3.30	2.30	-	-	-	-	-
Eating	1.60	1.50	-	-	-	-	-
Household work (general, cooking)	2.80	2.50	-	-	1.87	2.27	1.74
Washing utensils	1.70	-	-	-	2.44	1.70	2.17
Arranging vessels and folding beds	3.40	-	-	-	3.01	-	-
Light leisure activity	-	1.40	-	-	-	-	-
Desk-work (sitting and writing)	1.40	1.50	1.20	-	-	1.34	-
Sitting and reading	1.50	-	1.08	-	-	-	-
Ironing	1.70	-	-	-	-	1.58	-
Sweeping	-	-	-	-	3.09	2.41	1.58
Dusting	-	-	-	-	-	1.40	-
Cycling	3.60	-	-	-	-	-	-
Walking at 3-2 km/hr	3.00 ^b	2.00	3.23	-	2.67	2.86	2.35
Walking at 4-8 km/hr	-	3.20	-	-	-	3.58	-
Running (7-9 km/hr)	6.55	-	7.00	-	-	-	-
Washing clothes (manual)	2.80	-	-	3.00	3.43	-	2.96
Washing clothes (machine)	-	-	-	1.92	-	-	-

^a PAR values are same for both males and females; ^b Walking slow

As a rough approximation, it means that the 25-30% of the time during the working hours should be provided as a resting period in order to perform heavy activity without physiological difficulty (exhaustion, increase in plasma lactate etc.). These rest periods can be integrated into the duration of the physical activity, by constructing an integrated work-rest index. Thus, to account for rest periods that are taken during exercise, the Integrated Energy Index (IEI) is used. This takes into consideration the duration of rest during the exercise, which, in this case, is taken as 30% of the duration of the specific activity. Using this value for the rest period, the IEI for heavy physical activity works out to be 3.93, 3.84, 5.01 and 3.51 for agriculture, carrying loads, pulling carts and mining activities respectively.

**Table 4.3: Average PAR for various heavy activities
based on the studies**

Activities	Male		Female	
	PAR	Range	PAR	Range
Agriculture activities	5.7	3.1-8.9	4.1	2.4-6.3
Carrying Loads	5.1	3.0-7.9	-	-
Pulling carts	6.7	4.2-9.6	-	-
Mining activities	4.5	2.1-8.4	-	-

Table 4.4: Summary of the PAR values for adult Indian performing agriculture activities from various studies²³⁻²⁵

Activities	Males				Females	
	Ramanamurthy & Belavady 1966		Nag <i>et. al.</i> , 1980		Nag <i>et. al.</i> , 1981	
	PAR	Range ^a	PAR	%CV	PAR	%CV
Ploughing	5.8	4.7-8.9	-	-	-	-
Puddling	6.7	5.7-8.0	-	-	-	-
Working push-hoe	4.9	3.6-7.0	-	-	-	-
Trimming bunds	6.7	5.4-7.9	-	-	-	-
Making channels for irrigation	3.5	3.3-3.7	-	-	-	-
Harvesting	4.1	3.4-4.8	-	-	-	-
Making of bundles	3.7	3.1-4.0	-	-	-	-
Threshing	5.6	4.8-6.4	-	-	-	-
Transplanting	-	-	3.1	16.0	-	-
Germinating seeder	-	-	8.9	17.9	-	-
Bending & holding weights 2 kg	-	-	-	-	2.6	17.0
Spreading grains on floor	-	-	-	-	4.4	14.0
Pounding (helper)	-	-	-	-	2.4	34.7
Winnowing	-	-	-	-	2.5	27.4
Weeding with sickle	-	-	-	-	3.4	15.9
Uprooting (sitting)	-	-	-	-	3.5	40.7
Uprooting (bending)	-	-	-	-	4.5	25.5
Harvesting paddy field	-	-	-	-	3.5	33.6
Transplanting paddy seedling	-	-	-	-	3.6	26.4
Harvesting (bending)	-	-	-	-	3.7	33.9
Carrying load 20-25 kg	-	-	-	-	5.3	40.1
Pounding (single)	-	-	-	-	6.3	13.1
Digging dry soil using spade	-	-	-	-	5.7	26.0

^aThe reported range in the study

Table 4.5: Summary of the PAR values for adult Indian males carrying loads from various studies²⁶⁻²⁸

Activities	Das and Saha 1966		Samanta and Chaterjee 1981		Samanta <i>et. al.</i> , 1987	
	PAR	%CV	PAR	%CV	PAR	%CV
Carrying loads on shoulder (10% gradient)	5.2	13.9	-	-	-	-
Carrying loads on forehead (10% gradient)	5.7	13.3	-	-	-	-
Carrying loads on head (10% gradient)	6.9	19.0	-	-	-	-
Lifting 9 kg load	-	-	3.0	11.9	-	-
Lifting 13.4 kg load	-	-	3.6	13.3	-	-
Lifting 16.3 kg load	-	-	4.8	14.7	-	-
Lifting 20 kg load by porter	-	-	-	-	3.4	13.4
Lifting 30 kg load by porter	-	-	-	-	4.5	10.7
Lifting 40 kg load by porter	-	-	-	-	5.6	10.9
Lifting 50 kg load by porter	-	-	-	-	6.4	9.7
Lifting 60 kg load by porter	-	-	-	-	7.9	7.7

Table 4.6: Summary of the PAR values for adult Indian males pulling carts from various studies^{29,30}

Activities	Datta <i>et. al.</i> , 1978		Datta <i>et. al.</i> , 1983	
	PAR	%CV	PAR	%CV
Rickshaw pulling no load	4.2	16.8	-	-
Rickshaw pulling 1 man	5.5	16.7	-	-
Rickshaw pulling 2 men	6.9	12.4	-	-
Rickshaw pulling 2 men with 50 kg load	8.9	13.5	-	-
Pulling handcart no load	-	-	4.8	25.0
Pulling handcart 185 kg load	-	-	7.0	21.9
Pulling handcart 370 kg load	-	-	9.6	17.1

Table 4.7: Summary of the PAR values for adult Indian males performing mining activities from various studies^{31,32}

Activities	Ramanamurthy and Dakshayani 1962		Pal and Sinha 1994	
	PAR	%CV	PAR	%CV
Stone cutting	8.4	13.4	-	-
Stone splitting (sitting)	3.1	22.0	-	-
Stone splitting (standing)	4.0	21.9	-	-
Drilling with jackhammer - Tunnel face	-	-	3.6	22.5
Drilling with jackhammer - Post and pillar	-	-	3.2	30.9
Drilling with jackhammer - horizontal cut and fill	-	-	4.3	33.8
Drilling with jackhammer - room and pillar	-	-	5.0	29.7
Timbering - preparing face	-	-	4.1	34.6
Grouting	-	-	4.3	33.4
Loading - Cavo loader	-	-	3.8	33.0
Loading - Eimco loader	-	-	4.1	32.3
Loading - LHD operator	-	-	2.1	22.4
Manual shoveling	-	-	4.7	32.6

Using this compendium, and a time distribution of activities in a day, PAR values for activities performed in a day can be aggregated over a 24-hour period to yield the Physical Activity Level (PAL), which is the ratio of the energy expenditure for 24 hours and the BMR over 24 hours.

$$\text{Physical Activity Level (PAL)} = \frac{\text{Total PAR - hours}}{\text{Total time - hours}}$$

Physical Activity Level (PAL) =

$$\frac{\text{Total time - hours}}{\text{Total PAR - hours}}$$

For example, a person spending 8 hours in sleep (with a PAR of 1), 8 hours in domestic and leisure activity (with an average PAR of 2) and 8 hours at work (with an average PAR of 3), would have a total PAR -hour value = $(8 \times 1) + (8 \times 2) + (8 \times 3) = 48$ PAR-hours.

Thus, PAL (for the entire day) = Total PAR-hours / Total time = $48/24 = 2.0$

A large proportion of the daily energy expenditure is accounted for by the Basal Metabolic Rate (BMR). Therefore, Total Energy Expenditure (TEE) is calculated as a multiplication of BMR to PAL, since the PAL and PAR are indexed to the basal metabolic rate:

$$\text{TEE} = \text{PAL} \times \text{BMR}$$

The BMR is either measured directly, or predicted from body weight, gender and age specific equations that were derived from a large international dataset by the FAO/WHO/UNU¹¹. As discussed below in section 4.5, there are potential problems with this approach, given the body composition of Indians. The above principle of using the PAL values for computing daily energy requirement of men and women engaged in light, moderate and heavy activity, has been followed by the FAO/WHO/UNU Expert Consultation in 2004¹¹.

It is important to point out that using PAR and PAL values requires accurate reportage of the distribution of activities during the day and is subject to reporting error. Biological methods that directly measure the TEE, without assumptions of time and PAR (when accurate PAR values may not be available for specific activities) are very useful. The previous and this expert consultation have also used two other principles (a) Individually calibrated heart rate monitoring (HRM) method, and (b) Doubly Labelled Water (DLW) technique. These two techniques, which measure total energy expenditure of free-living persons are described briefly in this section. These methods are used to measure TEE over a 24-hr period including the metabolic response to food and the energy cost of tissue synthesis for adults, which is equivalent to the daily energy requirement. Additional energy for deposition in growing tissues is needed to determine energy requirements in infancy, childhood, adolescence and during pregnancy and for production and secretion of milk during lactation. It can be estimated from calculations of growth (or weight gain) velocity and the composition of weight gain or may be estimated by direct measurements of the TEE, which will include these factors. For lactation, a factorial approach can be used, from the average volume and composition of breast milk.

4.4.1. Heart rate monitor (HRM) method

Several investigations on TEE of healthy well-nourished individuals have been done in a broader spectrum of countries using minute-by-minute heart rate monitor (HRM) and individual calibration of the relationship between heart rate and oxygen consumption. The latter component is critical as explained above, as considerable inter-individual variation can exist. This has become possible with an electronic device that can accurately record minute-to-minute heart rate under free-living conditions, for a whole day or more. The mean TEE measured with this technique is comparable to mean value obtained using DLW or whole-body calorimeter.

4.4.2. Doubly labelled water (DLW) method

The use of the doubly labelled water (DLW) i.e. $^2\text{H}_2^{18}\text{O}$ technique to calculate total production of carbon dioxide (CO_2) over several days and, from this, measurement of the mean respiratory quotient (or food quotient under steady state conditions) and total energy expenditure, was originally developed for use in small animals³⁴. Its application was later validated in humans³⁵. Although questions have been raised about the appropriateness of the assumptions used in the calculation of TEE, the DLW method is considered the most accurate technique for measuring TEE in free-living individuals. TEE measured by this method includes the basal metabolism, metabolic response to food, thermoregulatory needs, physical activity costs, and energy cost of synthesis of growing tissues. Consequently, energy requirements are calculated as the sum of TEE plus the energy deposited as protein and fat in growing tissues and organs. This technique has been applied in studies on infants, children, adolescents and adults in a number of countries. Availability of this method has enabled the measurement of TEE in infants and children directly, which need not be based on energy intake. The technique has been used mostly in Europe and USA and also in some South American countries to a limited extent and has now been used in India in adults and children (Table 4.8)³⁶⁻³⁸.

Table 4.8: DLW studies performed in India

Source	Group studied	n	Mean BMI	Mean TEE (MJ/d)	Mean PAL
Borgonha <i>et al</i> , 2000 ³⁶	Young male active students Bangalore	6	22.7	11.2	1.8
	Young slum -dweller men Bangalore	6	16.9	7.1	1.5
	Young rural active men Bangalore	6	18.1	12.2	1.9
Krishnaveni <i>et al</i> , 2009 ³⁷	8-9 y boys Mysore	30	14.4	6.1	1.4
	8-9 y girls Mysore	28	14.9	5.6	1.4
Sinha <i>et al</i> , 2018 ³⁸	Young male medical students – normal weight	10	20.2	8.8	1.4
	Young male medical students – overweight	9	27.5	9.6	1.3

The limitation of this method is the cost and availability of stable isotope labelled water $^2\text{H}_2\text{O}_{18}$. The validation of DLW method against Whole Body Calorimeter (WBC) has been studied widely in western population, but only one validated study has been done on free-living Indian adults³⁶. This study has been conducted in three groups of subjects, well nourished, upper socio-economic students, undernourished slum dwellers and young rural farmers (Table 4.9). The mean difference between the two methods was 8% in well-nourished students and 0% in the undernourished slum subjects.

It appears that there is no significant difference in TEE as measured by DLW and the classical, currently used factorial method in children. Since there are larger global data sets available on energy requirements of infants, children and adolescents, Western data can be used after correcting for the lower body weights of Indian infants and children.

Table 4.9: TEE measured by the DLW and WBC method along with BMR and BMI³⁶

Description	Well-nourished control subjects	Undernourished slum subjects	Rural subjects
Age (y)	20.2 ± 1.9	21.2 ± 2.1	23.0 ± 2.1
Weight (kg)	64.7 ± 5.7	44.0 ± 2.9	55.3 ± 2.6
Height (M)	1.69 ± 0.10	1.61 ± 0.03	1.74 ± 0.0
BMI (kg/M^2)	22.7 ± 1.8	16.9 ± 0.9	18.1 ± 0.6
Measured BMR (MJ/day)	6.2 ± 0.9	4.9 ± 0.2	6.3 ± 0.3
TEE by calorimeter (MJ/day)	10.3 ± 1.6	7.3 ± 0.2	-
TEE by DLW (MJ/day)	11.2 ± 2.6	7.1 ± 0.9	12.2 ± 1.5
PAL(TEE/BMR)	1.79 ± 0.28	1.54 ± 0.18	1.90 ± 0.19

Values are Mean \pm SD

BMR – Basal Metabolic Rate

TEE – Total energy expenditure

DLW – Doubly labelled water method

PAL – Physical activity level

The present ICMR Expert group has made a few revisions to earlier proposed requirements¹³. These revisions are summarized briefly in this section.

Adult requirements

In case of energy requirement for adults, the comparison of BMR derived from FAO/WHO/UNU equations¹¹ and BMR observed among Indians suggest that the equation can overestimate the BMR by 10-12%⁴⁰⁻⁴⁴. This is perhaps because the FAO/WHO/UNU data set included many young and muscular subjects⁴⁴. The adiposity in Indians is now increasing, and a body composition that includes relatively more fat will result in a lower BMR⁴⁵. The TEE is the product of BMR and PAL.

Studies conducted on groups from the Indian urban population reported significantly different PAR values for adult male and females (lower for sedentary activities) as compared to a Western population^{15,17-32}. This could translate into significant differences in integrated index of PAL. Also, several Indian studies reported PAL level for adult male and female sedentary population in the range of 1.17-1.20^{45,46}. This value is much lower than the value used to derive energy requirements by ICMR, 2010 committee.

Overall, a change in either BMR or PAL could lead to a change in the TEE value⁴⁴. With lower levels of BMR and physical activity observed among the Indian adult population, overestimation of BMR and PAL is possible using the earlier recommendations, thus significantly overestimating the daily total energy expenditure. The use of the value of energy requirement recommended previously for adult men and women is likely to result in a positive energy balance. Therefore, the present committee has lowered the energy requirement for adults who are sedentary. In case of moderate or heavy activity, there is no recent evidence suggesting the energy expenditure on the activities is lower than recommendation made by previous expert group. Therefore, the present committee has retained the PAL values for calculating energy requirement. However, the BMR is lowered even for these categories.

Infant requirements

The present committee changed the body weight for infants (0-1y) as per the WHO child growth standards released in 2006³⁹ for deriving energy requirements.

4.5. Energy requirements of adults

There are various approaches to estimate energy requirement for adults – factorial, DLW or HRM methods. A large number of studies have been conducted using DLW for estimation of energy expenditure. The data mostly collected for European and American regions and few studies have been done in India³⁶⁻³⁸. Due to scarcity of data for Indians, the energy requirement for adults has been calculated using factorial approach. The aggregated PAL value has been calculated based on the energy cost of each activity and multiplied to the predicted BMR values. The factorial approach for calculating TEE has also been adopted by FAO/WHO/UNU, 2004¹¹.

The daily energy expenditure of adults depends on their occupational activity (sedentary/moderate/heavy activity), sleep and non-occupational activity, each typically for eight hours in a day. FAO/WHO/UNU have adopted factorial method to estimate the energy requirements of adults. The BMR is largely influenced by the body weight which can be used to predict basal metabolism of the individuals. To this the aggregated energy spent on the activity expressed as PAL is multiplied to arrive at TEE. Therefore, TEE = Predicted BMR x PAL.

A set of equations relating to body weight of adults and their BMR was given by the 1985 and 2004 recommendations of the FAO/WHO/UNU^{10,11}. A study on BMR of Indian subjects by Shetty and co-workers⁴¹ indicated that BMR of Indians was about 5% lower as compared to the reported BMR in developed countries. Based on this observation, a set of equations for computing basal metabolism of Indian adults was proposed which was used by ICMR Expert Group in 1989¹² and in 2010¹³. Subsequent studies indicated that the equation overestimated the BMR by ~10%. Studies from India⁴⁰⁻⁴³ have shown that the BMR predictive equations given by the FAO/WHO/UNU, 2004¹¹ generally overestimate the BMR of adult Indians by 5 to 12%. Soares and Shetty in 1988⁴⁰ examined the validity of Schofield equation for predicting BMR among Indians belonging to different socio-economic status and reported that the predictive equation overestimated the BMR of Indian males by 9.3% with higher difference (12.6%) among rural subjects. In the case of females, this difference was ~9%. On contrary, in another study by Ferro-Luzzi *et al*⁴⁴, it has been shown that there is no difference between measured and predicted BMR. However, the population on which BMR was measured were urban dwellers performing heavy activity and belonged to low socio-economic status. Also, their body fat percent ranged from 9.8 to 24.8%. BMR is highly dependent on body composition and population with low body fat will have higher BMR compared to high body fat population. Considering this evidence, the present committee proposes to lower the BMR by 10% and 9% for adult males and females respectively from values obtained through the Schofield equations¹¹.

Equations for predicting BMR proposed by FAO/ WHO/ UNU, 2004 are given in Table 4.10.

Using the set of equations provided in the Table 4.10, the BMR for adults with defined age, gender and body weight is calculated. And the calculated value will be reduced to 10% and 9% for males and females respectively. For example, for a 60 kg adult (20 y) male, the BMR equation used is (15.1xB.W.(kg)+692.2) and the resultant value is multiplied by 0.90, to obtain the BMR of 1438 kcal/d.

Physical inactivity among Indians has been increasing in the last decade⁴⁶. In the Indian Council of Medical Research-India Diabetes (ICMR-INDIAB) study, conducted in Tamil Nadu, Maharashtra, Jharkhand and Chandigarh, 54.4% were found to be inactive with inactivity being higher in the urban areas at 65% (58.7% among men and 71.2% in women)⁴⁶. The reported PAL values for sedentary population ranged between 1.17-1.20^{45,47}. The studies clearly show that there is a shift in physical activity from active to inactive. The population with heavy activity is not performing intense activity continuously for 8 hrs. It should be mentioned however, that proportion of population engaging in heavy work is quite small. Occupational activity pattern of Indian rural population as per the NNMB survey done in 1996-97⁴⁸ indicates that a majority of rural adults were engaged either in sedentary or moderate activity and a very small population were engaged in heavy activities.

Table 4.10: Equations for prediction of BMR (kcal/24h): FAO/WHO/UNU, 2004¹¹

Sex	Age (years)	Prediction equation proposed by FAO/WHO/UNU Consultation (2004)
Males	18-30	15.1xB.W.(kg)+692.2
	30-60	11.5xB.W.(kg)+873
	>60	11.7xB.W.(kg)+587.7
Females	18-30	14.8xB.W.(kg)+486.6
	30-60	8.1xB.W.(kg)+845.6
	>60	9.1xB.W.(kg)+658.5

BMR of Indians is 10% and 9% lower for males and females respectively than the international values (FAO/WHO/UNU, 2004).

B.W. = Body weight

In the earlier report, the PAR values (energy cost of each activity) were taken from FAO/WHO/UNU, 2004. However, values of energy cost of a population need to be specific to the population being studied. The PAR values used by FAO/WHO/UNU were obtained from the studies conducted in Western populations. Studies carried out in India^{15,17-22} indicate that values differ (lower for sedentary activity) for both in Indian adult males as well as in females (Table 4.1 and 4.2). This could be because PAR is not constant across the range of body weight and extrapolation from a heavy weight individual can overestimate PAR for lower weight individual¹⁴. These could translate into significant differences in an integrated index of activity such as PAL.

Therefore, a recalculation was performed using the PAR values reported for Indian population performing sedentary activity (Table 4.12) and this showed that the PAL was lower from 1.53 that was used in the previous recommendation¹³ to 1.40. This has led to the present recommendation that PAL values should be lower for sedentary activity groups (Table 4.11). Given the limited data available, this is also a prudent value as it is the lower limit of the range suggested by FAO/WHO/UNU¹¹ for sedentary activity. Equally, it should be emphatically stated that it is important to increase physical activity daily to maintain healthy lifestyle. The physical activity should not always be aimed to reduce body weight as it is also necessary to maintain a healthy heart and cognitive function.

For moderate and heavy activities, there is no evidence suggesting that the PAL in Indians is lower than the recommendations made by previous committee. Therefore, the present expert group has retained the PAL values for the moderate and heavy activities. However, the energy requirement of a group should be modified based on the activity performed in a day to maintain energy balance. In case of heavy activity workers, there can be variation in physical activity based on duration and intensity of work performed, which can change their energy expenditure.

A comparison of PAL values for different categories of work for Indian reference adult man and woman as per the previous and current recommendations are given in Table 4.11 along with values recommended by FAO/WHO/UNU Consultation of 2004.

Table 4.11: PAL Values proposed by ICMR Expert Group of 2020, 2010, 1989 compared to the figures proposed by FAO/WHO/UNU Consultation, 2004

Level of activity	ICMR 1989¹²	ICMR 2010¹³	ICMR 2020	FAO/WHO/UNU 2004¹¹
Sedentary Work	1.60	1.53	1.40	1.40-1.69
Moderate Work	1.90	1.80	1.80	1.70 – 1.99
Heavy Work	2.50	2.30	2.30	2.00 – 2.40*

*PAL Value > 2.40 is difficult to maintain over a prolonged period.

A computation of energy expenditure for an adult Indian population using factorial computation is given in Table 4.12. As discussed above, body weights of normal healthy Indian adult individual man and woman are taken as 60 kg and 55 kg respectively. This can be considered as the standard body weight for Indian adult. The PAL values can be calculated as the aggregated PAR over 24 hrs. Then the PAL value is multiplied with calculated BMR to derive at energy expenditure for a day.

Energy expenditure at the three levels of activity at different age groups and at different body weights are given in Table 4.13. Special consideration should be given for the population >60 y as

BMR in the population decreases by nearly 200 kcal, assuming the same reference body weight of 60 kg and 55 kg in both men and women respectively is maintained.

4.6. Energy requirement during pregnancy

4.6.1. Determinants of energy cost during pregnancy

The FAO/WHO/UNU 2004 Consultation on energy requirements¹¹, reviewed the data available on maternal weight gain and foetal body weight and decided to use the gestational weight gain of 10 to 14 kg, with an average of 12 kg, and full-term birth weight of 3.1 to 3.6 kg with a desirable birth weight of 3.3 kg. Data from developed countries indicate that the optimal pregnancy outcome in terms of birth weight, infant growth and survival is seen when pregnancy weight gain is about 12-14 kg.

Energy requirement during pregnancy comprises the normal requirement for an adult woman and an additional requirement needed for gestational weight gain associated with protein and fat accretion in maternal, foetal and placental tissues and increase in BMR. Most of the accretion occurs during the second and the third trimesters. Protein is predominantly deposited in fetus (42%) but also in uterus (17%), blood (14%) placenta (10%) and breast (8%)¹¹. Fat is predominantly deposited in fetus and maternal tissues and contributes substantially to overall energy cost of pregnancy.

Protein and fat gain associated with gestational weight gain of 12 kg, would be 597g and 3.7 kg respectively. Besides weight gain and its associated costs, the BMR also increases during pregnancy. Cumulative increase in BMR during pregnancy is significantly correlated with gestational weight gain. For a 12 kg weight gain, basal metabolism would increase by about 35,000 kcal. The increase in BMR relative to pre-pregnancy values can be considered to be 5.3, 11.4 and 25.3% during the first, second and the third trimesters, respectively¹¹.

Energy requirement during pregnancy also depends upon any deviation from normal physical activity. Several studies in different regions have shown that there is no evidence of any reduced activities during pregnancy, although global literature suggests that women do less arduous tasks towards the end of pregnancy. Some studies in India have also shown that there is a shift towards more sedentary activity even among families whose usual occupation involves manual labour⁴⁹.

Longitudinal measurements of DLW in free living well-nourished women in Sweden, UK and USA have shown a mean increase of 16.5% in TEE by the third trimester value compared to non-pregnant values. This is mostly due to body weight gain, since no difference between pregnant and non-pregnant values was found in TEE, when expressed as per kg body weight. Therefore, for an average 12 kg weight gain, the increment in TEE would be 20, 85 and 310 kcal/day during the first, second and third trimesters respectively¹¹.

4.6.2. Additional energy requirement during pregnancy

The additional energy required during pregnancy (Table 4.14) can be estimated by the factorial method in two ways: using either the cumulative increment in the BMR or in the TEE during pregnancy, plus energy deposited as protein and fat. Most of the additional energy is required in second and third trimester. This increase depends upon the pre-pregnancy body weight and pre-pregnancy BMR. The increment in BMR is due to tissue deposited during pregnancy as protein and fat in the fetus and the mother and depends upon the total Gestational Weight Gain (GWG) during the 9 months of pregnancy which may range from 10 to 14 kg with an average of 12 kg. In the Indian reference women with pre-pregnancy weight of 55 kg, the body weight gain may be 10 kg. Data from NNMB surveys indicate that the mean height of Indian women is about 151 cm and the mean body weight is 47 kg; the average pregnancy weight gain in this population is only 7-8 kg.

Table 4.12: Computation of energy expenditure of adult Indian population using factorial approach

Main daily activities	Duration (h)	Major lifestyles, energy expenditure (PAR values)^a		
		Sedentary	Moderate active	Heavy or vigorously active
Sleep	8	1.0	1.0	1.0
Occupational activity	8	1.3 ^b	2.5 ^c	4.1 ^d
Non-occupational activity	8	1.9	1.9	1.9
Mean		1.41	1.80	2.33
Non-occupational activity details				
Personal care	1	1.6	1.6	1.6
Eating	1	1.2	1.2	1.2
Commuting to work by bus or by vehicle or by walk	1	2.0	2.0	2.0
General household or other activities	2	2.0	2.0	2.0
Walking at various speeds without load	1	3.4	3.4	3.4
Light leisure activity	2	1.4	1.4	1.4
Mean non-occupational activity	8	1.9	1.9	1.9
Men: body wt. (65kg), BMR (1506 kcal)	--	2123	2711	3509
Women: body wt. (55kg), BMR (1184 kcal)	--	1670	2131	2759

^aPAR values from Table 4.1, 4.2, 4.4, 4.5, 4.6 & 4.7

^bPAR is calculated for sedentary activities like desk work, sitting and reading and some time for standing;

^c PAR is calculated for moderate activity like standing, walking at various speed, cycling and carrying light weights;

^dPAR is calculated as IEI assuming 30% of rest period in the activity. Here average of IEI for heavy activities is included - agricultural activities (3.93), carrying loads (3.84), pulling cart (5.05) and mining (3.51) activities (see section 4.4). This should be changed depending upon the activity performed.

Table 4.13: Current estimated energy Requirement of Indian Men and Women at Different Ages and Body Weights

Sex	Body weight (kg)	19-30 y				30-60 y				>60 y			
		BMR (kcal /d) ^a	Sedentary Work TEE (kcal/d) ^b	Moderate Work TEE (kcal/d) ^b	Heavy Work TEE (kcal/d) ^b	BMR (kcal /d) ^a	Sedentary Work TEE (kcal/d) ^b	Moderate Work TEE (kcal/d) ^b	Heavy Work TEE (kcal/d) ^b	BMR (kcal /d) ^a	Sedentary Work TEE (kcal/d) ^b	Moderate Work TEE (kcal/d) ^b	Heavy Work TEE (kcal/d) ^b
Males	45	1235	1728	2222	2839	1251	1752	2253	2878	1003	1404	1805	2306
	50	1302	1823	2344	2996	1303	1824	2346	2997	1055	1478	1900	2427
	55	1370	1919	2467	3152	1355	1897	2439	3116	1108	1551	1995	2549
	60	1438	2014	2589	3308	1407	1969	2532	3235	1161	1625	2089	2670
	65	1506	2109	2711	3465	1458	2042	2625	3354	1213	1699	2184	2791
	70	1574	2204	2834	3621	1510	2114	2718	3473	1266	1772	2279	2912
	75	1642	2299	2956	3777	1562	2187	2812	3592	1319	1846	2374	3033
Females	40	982	1374	1767	2258	1064	1490	1916	2448	930	1303	1675	2140
	45	1049	1468	1888	2412	1101	1542	1982	2533	972	1361	1749	2235
	50	1116	1563	2009	2567	1138	1593	2048	2618	1013	1419	1824	2331
	55	1184	1657	2130	2722	1175	1645	2115	2702	1055	1477	1898	2426
	60	1251	1751	2252	2877	1212	1696	2181	2787	1096	1535	1973	2521
	65	1318	1846	2373	3032	1249	1748	2247	2872	1138	1593	2048	2616
	70	1386	1940	2494	3187	1285	1800	2314	2957	1179	1650	2122	2711

^aBMR calculated using FAO/WHO/UNU, 2004 equation and adjusted to 10% and 9% lower value for males and females, respectively

^bPAL Values: Sedentary – 1.40, Moderate – 1.80; Heavy 2.30

Table 4.14: Current additional energy cost of pregnancy with 12 kg and 10 kg GWG*

	1 st trimester (g/d)	2 nd trimester (g/d)	3 rd trimester (g/d)	Total Deposited (g)
A. Rate of tissue deposition				
Weight gain (g/d)	17	60	54	12000
Protein deposition (g/d)	0	1.3	5.1	597
Fat deposition (g/d)	5.2	18.9	16.9	3741
B. Energy cost of pregnancy (kcal) from energy deposited and increase in BMR				
Protein deposited (kcal/d) ^a	0 (0)	7.2 (6.0)	29.0 (24)	3370 (2808)
Fat deposited (kcal/d) ^a	48.3 (40.2)	175 (145)	156 (130)	34600 (28832)
Efficiency of energy utilization (kcal/d) ^b	4.8 (4.0)	18.2 (15.0)	18.4 (15.3)	3800 (3167)
Basal metabolic rate increase (kcal/d)	48 (40)	95 (79)	237 (198)	35130 (29274)
Total energy cost of pregnancy (kcal/d)	101 (84)	295 (246)	440 (367)	77100 (64247)
C. Energy cost of pregnancy (kcal) from energy deposited and increase in TEE				
Protein deposited kcal/d)	0 (0)	7.2 (6.0)	29.0 (24)	3370 (2808)
Fat deposited (kcal/d)	48.3 (40.2)	175 (145)	156 (130)	34600 (28832)
TEE increase ^c (kcal/d)	20 (17)	84 (70)	311 (259)	38560 (32132)
Total energy cost of pregnancy (kcal/d)	69 (57)	266 (222)	496 (413)	76530 (63775)
Average energy cost of pregnancy based on B and C ^d (kcal/d)	85 (70)	280 (230)	470 (390)	77000 (64170)
Average of 2 nd and 3 rd trimesters (kcal/d)	--		375 (310)	--

*Figures within parentheses correspond to energy costs for 10 kg GWG.

^aProtein and fat deposition with an energy value of 5.65 kcal/g protein deposited and 9.25 kcal/g fat deposited

^bEfficiency of food energy utilization for protein and fat deposition is taken as 0.90

^cIncludes costs of efficiency of energy utilization

^dRounded off to nearest 5 kcal

Additional energy requirement of Indian women during pregnancy have been computed on the basis of the reference Indian women and pregnancy weight gain of 12 kg and 10 kg. The previous committee suggested the average energy requirement of 375 kcal and 310 kcal for 12 kg and 10 kg GWG respectively with an average additional energy requirement of 350 kcal for second and third trimester (Table 4.14). With no recent data on pregnant Indian women being available, the present committee retains the energy requirement proposed by the ICMR, 2010¹³.

Based on the Table 4.14, the following recommendation (Table 4.15) has been made for the additional energy requirements of an Indian woman with pre-pregnancy weight of 55 kg. It should be noted that it is the average value for the 2nd and 3rd trimesters is taken for a single recommendation value.

Hence, an average recommendation of 350 kcal/day through the second and third trimesters, as additional requirement during pregnancy for an Indian woman of 55 kg body weight and pregnancy weight gain between 10 -12 kg can continue as per the previous recommendation.

Appropriate adjustments can also be made to compute the requirements of average women with pre-pregnant weight of 47 kg, gestational weight gain (GWG) of 7-8 kg, and birth weight of 2.8 kg especially in programmes for food supplementation intended to bridge the gap between the requirement and intake in pregnant women.

4.7. Energy requirement during lactation

4.7.1. Determinant of energy cost during lactation

Energy cost of lactation is determined by the breast milk output and its energy content. Milk output is determined by test feeding and weighing, and a correction of 5% is made in arriving at the milk output for the insensible water loss of the baby. The energy content of milk is based on the energy value of protein, fat and lactose of the milk, which are arrived at by analysis and energy estimate by bomb calorimetry. Energy content is 5.65 kcal per g of protein and free amino acids 9.25 kcal per g of fat and 3.96 kcal per g of lactose. The metabolizable energy in human milk is assumed to be 5.3% lower than its gross energy content based on proximate analysis. A study on energy expenditure of Indian lactating woman indicates a negative energy balance, which could be corrected by the fat deposited during pregnancy.

4.7.2. Energy requirement for lactation

There is no evidence for additional demand for lactation besides the energy content of milk secreted for breast feeding. There is no change in TEE during lactation period over that in the non-pregnant period. Frequent sitting for breast feeding itself could be an adaptation for energy conservation. Milk output data collected from developing countries was used by previous committee to compute energy cost of lactation of Indian women (Table 4.16). The present committee retains the energy requirement proposed by ICMR 2010¹³. The energy cost of milk output is corrected for efficiency of utilization of milk energy i.e. 80%.

Daily additional energy requirement of a woman who exclusively breast feed during the first 6 months would be 600 kcal and for partial breast feeding during 7-12 months, it would be 517 kcal or approximately 520 kcal. A study of energy requirement of lactating women based on milk output and

Table 4.15: Additional energy requirement for pregnant women (kcal/d)

Trimester	10 kg GWG	12 kg GWG
	Kcal/d	
1 st trimester	70	85
2 nd trimester	230	280
3 rd trimester	390	470
Average of 2 nd and 3 rd trimester	310	375

energy output computed from actual measurement showed that average energy utilization for average milk production of 624 ml was 549 kcal. This would work out to 594 kcal for 722 ml of milk output and correlating closely with the figures given in Table 4.17.

4.7.3. Fat accretion during pregnancy as a source of energy during lactation

When food scarcity and undernutrition were common in earlier decades in India, lactating women mobilised body fat to provide energy needs for breast milk secretion during the first six months of lactation⁵¹. To prevent aggravation of undernutrition, pregnant and lactating women at risk of undernutrition and those with low pregnancy weight gain would require additional 300 and 500 kcal to meet the energy needs of pregnancy and lactation.

Table 4.16: Energy expenditure during lactation in Indian women⁵⁰

Month postpartum	Mean milk output^a (g/d)	Corrected output^a (g/d)	Gross Energy content^b (kcal/d)	Daily gross energy secreted (kcal/d)	Energy cost of milk production^c (kcal/d)
1	562	590	395	494	468
2	634	666	446	558	520
3	582	611	409	511	484
4	768	806	540	675	639
5	778	817	547	684	648
6	804	844	566	708	671
Mean	688	722	483	605	573
Partial breastfeeding					
7	688	722	484	605	598
8	635	667	452	565	558
9	516	542	367	479	453
10	--	--	--	--	--
11	565	593	402	563	497
12	511	537	364	455	449
Mean	583	612	414	517	511

^aInsensible water losses assumed to be equal to 5% of milk intake.

^b2.8 KJ/g or 0.67 kcal/g of milk output measured by macronutrient analysis

^cBased on energy efficiency of 80%

In the current dual nutrition burden era, there has been reduction in undernutrition rates and increase in overnutrition rates; even in women in their twenties, from low middle income families, a third of women are overweight^{52,53}. A recent study from New Delhi on low middle income lactating women showed that over nutrition is emerging as a major problem both in lactating and non-lactating women, particularly in women over 30 years of age⁵⁴.

Currently the average weight gain during pregnancy is 10-12 kg; of this the fat gain accounts for 2-4 kg. It is important to consider whether utilisation of fat accreted during pregnancy should be taken into account while computing energy requirements during lactation in normally nourished and over-nourished women. The fat store accumulated during pregnancy could yield 18000 to 36000 kcal and provide a maximum of 100 to 200 kcal for milk secretion during the first six months. If this is taken into account, the extra energy allowance during lactation could be reduced by 100 to 200 kcal/day in well-nourished women with an adequate gestational weight gain. This would prevent an increase in overweight in lactating women in India⁵⁴. However, the reduction in the energy

allowance should only be considered if the quality and diversity of the food intake is high enough to meet the requirements of other nutrients.

Table 4.17: Milk production and energy utilization by Indian lactating women on whom total energy balance was conducted⁵⁰

Subject No.	Energy intake	Total energy expenditure	Milk output (ml)	Energy utilization for milk production
Values give in kcal/24h (mean of 6 months)				
1	2255	2555	579.8	481.2
2	2046	2577	861.5	315.1
3	2330	2179	477.3	396.2
4	2313	2156	700.5	581.4
5	2680	2213	860.4	714.1
6	2037	2189	520.8	432.3
7 ^a	1704	1825	-- ^b	--
8	1802	2137	-- ^b	--
Mean	2148	2229	666.7	553.4

^aSubject unwell at times

^bValues were available for infants aged up to 2 months, hence not considered

4.8. Energy requirements of infants and children

4.8.1. Energy requirements of infants

The FAO/WHO/UNU, 2004 derived energy requirement for infants on the basis of the DLW method. A total of 13 studies on healthy, well-nourished and non-stunted infants aged from 0-12 months were included. The data were collected on infants from different countries – UK, USA, Netherlands, Chile and China. There have been no data on infants from India¹¹.

The previous recommendations by the expert group adopted the FAO/WHO/UNU, 2004 recommendations on energy requirement and with no new evidence available the same recommendations will be continued, with modifications in body weights for infants. The 2004 Consultations of FAO/WHO/UNU derived the energy requirement of infants on the basis of DLW method and to this, the energy acquisition due to growth was added to arrive at the total daily energy requirement. However, the present Consultation group has used the body weights of infants 0-12 months of age from WHO child growth standards, 2006 unlike the previous one where body weights were from western data³⁹.

Since there is no data on energy requirement among Indian infants using DLW method or HRM method, the same equation as provided by FAO/WHO/UNU, 2004 was used for Indian infants. The equation to derive energy requirement of infants as reported by FAO/WHO/UNU, 2004 is as follows¹¹:

$$\text{TEE (MJ/d)} = -0.416 + 0.371 * \text{BW (kg)}$$

$$n=320, r=0.85 \text{ and}$$

$$\text{Standard error of estimate} = 0.456 \text{ MJ/d}$$

$$\text{TEE (kcal/d)} = -99.4 + 88.6 * \text{BW (kg)}$$

$$\text{Standard error of estimate} = 109 \text{ kcal/d}$$

Energy deposition during growth [weight gain (g/d) x energy deposited/g] has to be added to TEE to obtain energy requirement. Therefore, the total energy requirement during infancy (kcal/d)=[-99.4 + 88.6*BW (kg)] + [wt gain (g/d) x energy deposited (kcal/g)].

The body weights of infants at different ages and energy requirement levels, which can be adopted for Indian infants, are derived from Tables 4.18 and 4.19. The body weight of infants used to derive the energy deposition during growth are

from WHO child growth standards³⁸. Using the new child growth standards, the energy requirement for infants are 98 kcal/kg/d (1% increase over the previous recommendation) and 80 kcal/kg/d for 0-6 months and 6-12 months respectively. The comparison of energy requirement between the present and previous recommendations is presented in Table 4.23.

4.8.2. Energy requirement of children

The energy requirement for children is arrived at, through the estimation of TEE by doubly labelled water or heart rate monitoring, and the energy needed for growth is added to this estimate.

Although this is a more direct approach to determine the energy requirement of this age group, there is hardly any data on energy expenditure of Indian children using this method. It must be realized that Indian children of this age group particularly in rural areas are more active than children in the Western countries. These Western children are mostly urbanites, less active and hence more prone to obesity. Data from developed countries may therefore not be fully applicable to children from the developing countries, although the computed growth requirement may be applicable to both categories of children. However, rural children from developing countries like India weigh less than the reference children.

FAO/WHO/UNU, 2004 reports¹¹ the energy requirement of children and adolescents based on the estimation of DLW and HRM on children from industrialized (75%) and developing countries and the following quadratic polynomial was derived from body weight of boys and girls:

Boys:

$$\begin{aligned} \text{TEE kcal/day: } & 310.2 + 63.3 * \text{BW(kg)} - 0.263 * \text{BW(kg}^2\text{)} \\ \text{TEE MJ/day: } & 1.298 + 0.265 * \text{BW (kg)} - 0.0011 * \text{BW(kg}^2\text{)} \\ n: & 801, r=0.982, r^2 = 0.964 \end{aligned}$$

Girls:

$$\begin{aligned} \text{TEE kcal/day: } & 263.4 + 65.3 * \text{BW(kg)} - 0.454 * \text{BW(kg}^2\text{)} \\ \text{TEE MJ/day: } & 1.102 + 0.273 * \text{BW(kg)} - 0.0019 * \text{BW(kg}^2\text{)} \\ n: & 808, r = 0.955, r^2 = 0.913 \end{aligned}$$

Table 4.18: Protein, fat and energy deposition during growth in the first year of life¹¹

Age (months)	Protein gain (g/d)	Fat mass gain (g/d)	Weight gain (g/d)	Energy accrued in normal growth*	
				(KJ/g)	(kcal/g)
Boys					
0-3	2.6	19.6	32.7	25.1	6.0
3-6	2.3	3.9	17.7	11.6	2.8
6-9	2.3	0.5	11.8	6.2	1.5
9-12	1.6	1.7	9.1	11.4	2.7
Girls					
0-3	2.2	19.7	31.1	26.2	6.3
3-6	1.9	5.8	17.3	15.6	3.7
6-9	2.0	0.8	10.6	7.4	1.8
9-12	1.8	1.1	8.7	9.8	2.3

*Energy equivalents of 1 g protein = 23.6 KJ (5.65 kcal);
1 g fat = 38.7 KJ(9.25 kcal)

Table 4.19: Current energy requirement of infants during first year of life*

Sex	Age (months)	Weight (kg) ^a	Weight gain (g/d)	Total Energy Expenditure ^b		Energy deposition ^c		Daily energy requirement ^d			
				kcal/d	MJ/d	kca l/d	MJ/d	kcal/d	MJ/d	kcal/kg/d ^f	KJ/kg/d ^f
Boys	0 - 1	3.90	40.0	246	1.030	240	1.005	490	2.035	125	520
	1 – 2	5.05	38.3	348	1.457	230	0.963	580	2.420	115	480
	2 – 3	6.00	31.7	432	1.809	190	0.795	620	2.604	105	430
	3 – 4	6.70	23.3	494	2.069	65	0.273	560	2.342	85	350
	4 – 5	7.25	18.3	543	2.273	51	0.215	590	2.488	80	340
	5 – 6	7.70	15.0	583	2.440	42	0.176	620	2.615	80	340
	6 – 7	8.10	13.3	618	2.588	20	0.084	640	2.672	80	330
	7 – 8	8.45	11.7	649	2.718	18	0.073	670	2.791	80	330
	8 – 9	8.75	10.0	676	2.829	15	0.063	690	2.892	80	330
	9 – 10	9.05	10.0	702	2.940	27	0.113	730	3.053	80	340
	10 – 11	9.30	8.3	725	3.033	23	0.094	750	3.127	80	340
	11 – 12	9.50	6.7	742	3.107	18	0.075	760	3.183	80	330
Girls	0 - 1	3.70	33.3	228	0.956	210	0.879	440	1.835	120	500
	1 – 2	4.65	31.7	313	1.308	200	0.835	510	2.144	110	460
	2 – 3	5.45	26.7	383	1.605	168	0.703	550	2.308	100	420
	3 – 4	6.10	21.7	441	1.846	80	0.336	520	2.182	85	360
	4 – 5	6.65	18.3	490	2.050	68	0.284	560	2.334	85	350
	5 – 6	7.10	15.0	530	2.217	55	0.232	590	2.449	80	340
	6 – 7	7.45	11.7	561	2.347	21	0.088	580	2.435	80	330
	7 – 8	7.75	10.0	587	2.458	18	0.075	610	2.534	80	330
	8 – 9	8.05	10.0	614	2.569	18	0.075	630	2.645	80	330
	9 – 10	8.35	10.0	640	2.681	23	0.096	670	2.777	80	330
	10 – 11	8.60	8.3	663	2.773	19	0.080	680	2.854	80	330
	11 – 12	8.80	6.7	680	2.848	15	0.064	700	2.912	80	330

*Daily energy requirement (kcal/d) is calculated from linear regression analysis of total energy expenditure on weight, plus allowance for energy deposition in tissues during growth

^aBody weights for infants from WHO child growth standards, 2006³⁹

^bTEE (kcal/d) = -99.4 + 88.6kg; TEE (MJ/d) = -0.416 + 0.371kg¹¹

^cWeight gain x energy accrued in normal growth (Table 4.18)

^dRequirement = total energy expenditure + energy deposition

^eRounded off to the nearest 10 kcal;

^fRounded off to the nearest 5 kcal and 10 KJ

Table 4.20: Year-wise body weight, average weight gain and energy cost of weight gain

Age y	Boys			Girls		
	Body Wt kg*	Weight gain kg/y	Energy cost of wt gain kcal/day	Body Wt. kg*	Weight gain kg/y	Energy cost of wt gain cal/day
1	9.9	1.7	9.32	8.9	1.6	8.77
2	12.2	2.3	12.60	11.5	2.6	14.25
3	14.3	2.1	11.51	13.9	2.4	13.15
4	16.3	2.0	10.96	16.1	2.2	12.05
5	18.5	2.2	12.05	18.3	2.2	12.05
6	20.5	2.0	10.96	20.2	1.9	10.41
7	22.9	2.4	13.15	22.4	2.2	12.05
8	25.4	2.5	13.70	25.0	2.6	14.25
9	28.1	2.7	14.79	28.2	3.2	17.53
10	31.2	3.1	16.99	31.9	3.7	20.27
11	34.6	3.4	18.68	36.2	4.3	23.34
12	38.9	4.3	23.51	41.2	5.0	27.34
13	44.3	5.4	29.53	46.0	4.8	26.52
14	50.6	6.3	34.63	50.1	4.1	22.25
15	56.6	5.9	32.55	52.8	2.8	15.18
16	61.3	4.7	25.92	54.7	1.8	10.08
17	64.8	3.5	19.12	55.7	1.1	5.86
18	67.3	2.5	13.81	56.7	0.9	5.10

*WHO-MGRS body weights were used for children 12-59 months and WHO reference values (2007) were used for children and adolescents (5-18y). Refer chapter 3 for details.

Energy cost of weight gain: 2 kcal/g weight gain¹¹

Table 4.21a: Energy requirement and PAL of Indian children and adolescents (boys)

Age (y)	Boys							
	Weight ^a (kg)	TEE ^b (kcal/d)	PAL ^c	Energy cost of growth ^d	Total energy requirement ^e		Total energy requirement ^f	
				kcal/d	kcal/d	MJ/d	kcal/kg/d	KJ/kg/d
1-2	9.9	847.3 ^g	1.63	9.32	857	3.585	85	356
2-3	12.2	1043.3	1.50	12.60	1056	4.418	87	362
3-4	14.3	1161.6	1.40	11.51	1173	4.908	82	343
4-5	16.3	1272.1	1.45	10.96	1283	5.368	79	329
5-6	18.5	1391.2	1.51	12.05	1403	5.871	76	317
6-7	20.5	1497.3	1.54	10.96	1508	6.311	74	308
7-8	22.9	1621.9	1.58	13.15	1635	6.841	71	299
8-9	25.4	1748.3	1.62	13.70	1762	7.372	69	290
9-10	28.1	1881.3	1.65	14.79	1896	7.933	67	282
10-11	31.2	2029.1	1.68	16.99	2046	8.561	66	274
11-12	34.6	2186.0	1.72	18.68	2205	9.224	64	267
12-13	38.9	2374.6	1.76	23.51	2398	10.034	62	258
13-14	44.3	2597.9	1.80	29.53	2627	10.993	59	248
14-15	50.6	2840.2	1.83	34.63	2875	12.028	57	238
15-16	56.6	3048.8	1.84	32.55	3081	12.892	54	228
16-17	61.3	3201.6	1.84	25.92	3228	13.504	53	220
17-18	64.8	3306.8	1.83	19.12	3326	13.916	51	215
18-19	67.3	3378.8	1.83	13.81	3393	14.195	50	211

Note: This Table reflects the energy requirement of boys who are healthy and have a moderate activity level.

^a Weight from Table 4.20

^b Energy (TEE) was predicted from above cited gender specific quadratic equation. This reflects the TEE of moderately active children.

^c PAL was calculated as TEE/BMR. BMR was estimated from age and gender appropriate prediction equations¹¹

^d From Table 4.20

^e Requirement = Sum of TEE and energy cost of growth. Rounded off to the nearest 10 kcal.

^f Rounded off to the nearest 5 kcal.

^g TEE of infants (aged 1-2 years) was reduced by 7% to fit with energy requirement of infants¹¹

Table 4.21b: Energy requirement and PAL of Indian children and adolescents (girls)

Age (y)	Girls							
	Weight ^a (kg)	TEE ^b (kcal/d)	PAL ^c	Energy cost of growth ^d	Total energy requirement ^e		Total energy requirement ^f	
				kcal/d	kcal/d	MJ/d	kcal/kg/d	KJ/kg/d
1-2	8.9	751.9 ^g	1.66	8.77	761	3.184	85	356
2-3	11.5	954.3	1.49	14.25	969	4.052	84	352
3-4	13.9	1083.4	1.41	13.15	1097	4.588	79	330
4-5	16.1	1197.0	1.47	12.05	1209	5.059	75	314
5-6	18.3	1306.3	1.52	12.05	1318	5.516	72	301
6-7	20.2	1397.2	1.56	10.41	1408	5.889	70	292
7-8	22.4	1498.3	1.59	12.05	1510	6.319	67	282
8-9	25.0	1612.2	1.62	14.25	1626	6.805	65	272
9-10	28.2	1743.8	1.65	17.53	1761	7.370	62	261
10-11	31.9	1884.5	1.68	20.27	1905	7.969	60	250
11-12	36.2	2031.0	1.73	23.34	2054	8.595	57	238
12-13	41.2	2181.7	1.75	27.34	2209	9.243	54	225
13-14	46.0	2306.3	1.76	26.52	2333	9.761	51	212
14-15	50.1	2394.4	1.76	22.25	2417	10.111	48	202
15-16	52.8	2445.9	1.75	15.18	2461	10.297	47	195
16-17	54.7	2476.3	1.74	10.08	2486	10.403	45	190
17-18	55.7	2492.5	1.73	5.86	2498	10.453	45	188
18-19	56.7	2505.8	1.73	5.10	2511	10.506	44	185

Note: This Table reflects the energy requirement of girls who are healthy and have a moderate activity level.

^a Weight from Table 4.20

^b Energy (TEE) was predicted from above cited gender specific quadratic equation. This reflects the TEE of moderately active children.

^c PAL was calculated as TEE/BMR. BMR was estimated from age and gender appropriate prediction equations¹¹

^d From Table 4.20

^e Requirement = Sum of TEE and energy cost of growth. Rounded off to the nearest 10 kcal.

^f Rounded off to the nearest 5 kcal.

^g TEE of infants (aged 1-2 years) was reduced by 7% to fit with energy requirement of infants¹¹

Table 4.21c: Daily activity pattern required in a moderately active lifestyle of a 12-15 year child with a PAL of 1.8 compared with a child having a PAL of 1.5*

		PAL = 1.5		PAL = 1.8	
Activity	PAR	Duration (h)	PAR x h	Duration (h)	PAR x h
Sleep	1.0	9	9	9	9
School-related activities					
Walk to School	3.0	0	0	1	3
In class	1.5	6	9	6	9
Recess play time	3.0	1	3	1	3
Home-related activities					
Homework	1.5	2	3	2	3
TV/Sedentary activity	1.5	4	6	2	3
Discretionary Play					
Intermittent fun-play	3.0	2	6	2	6
Focused Games (football, basketball)	8.0	0	0	1	8
Total		24	36	24	44
PAL			1.5		1.8

*Lifestyle has been considered to reflect that of a 12-16 year old school going boy.

PAR values were taken from reference 11

PAL = Total PAR-HR / Total duration

Recess playtime, and ‘intermittent fun-play’ were assumed to be unfocussed play, with rest periods. TV sedentary activity refers to time at home, spent watching TV, or at a computer/ play station, or in light household chores.

The differences between the two lifestyle patterns are a) the extra intense game playtime, b) 50% reduction in sedentary/ TV time and c) active self-transport to school, i.e., not using motorized transport.

Table 4.15 shows energy acquired during growth: energy equivalent for 1g protein = 5.65 kcal, 1 g fat = 9.25 kcal.

The weight gain during growth and overall energy requirement taking growth into consideration for boys and girls, are shown in Tables 4.21a and 4.21b respectively. In these Tables, physical activity levels (computed as the ratio of the predicted TEE and the predicted BMR for each age group) are also presented. Since there is no recent evidence to show that the BMR of Indian children is lower than that of their Western counterparts, the present committee retains the BMR derived from the FAO/WHO/UNU 2004¹¹. BMR prediction equations were used to predict BMR, while TEE was predicted from the quadratic prediction equation given earlier in this section.

It is clear from the Tables (4.21a and 4.21b) that the activity level, or PAL, used in the children’s TEE calculation, was high. This is because of the quadratic equation used to predict TEE; and it is worth considering the type of activities and the time spent in them that could lead to a PAL of 1.8. Table 4.21c depicts a typical lifestyle that would be required for an adolescent aged between 12-16 years, to have a PAL of 1.8.

To illustrate this point, actual measurements of TEE have been performed by the DLW method in Indian children aged 8-9 years³⁷. These values (in middle class South Indian children) were 1458 kcal/day and 1338 kcal/day in boys and girls respectively (Table 4.8). The physical activity level (PAL) in these children was 1.4, which is low and indicative of sedentary habits. It should be

emphasized again, that the prediction equation for TEE (quadratic equation proposed by FAO/WHO/UNU, 2004, assumes a moderately active child as the reference¹¹. This means that for a child aged 8-9, PAL would be about 1.6 (Table 4.21c), which is higher than what was observed in sedentary Indian children (Table 4.8)³⁷. This is a cause for concern, if the observed PAL is indicative of all Indian children. It should be emphasized that children need to be physically active such that they meet the PAL values indicated in Table 4.21a and 4.21b, and that the activity should increase in the teens to a PAL of 1.8. However, as further evidence on Indian children is not yet available these same recommendations as the previous Committee's recommendation is continued.

Table 4.21d below, gives some play activities and their PAR. These can be used as in Table 4.16c, to calculate PAL values in any given lifestyle pattern.

To calculate the requirement of sedentary children, PAL would be 15% less than that in moderately active children, and for highly active children, PAL would be 15% higher. Therefore, for a 9-10-year-old boy, sedentary PAL would be about 1.40 and for highly active in the same age, it would be 1.90. The TEE would change in a similar manner. Tables 4.21e and 4.21f give the requirements (including energy required for growth) for a child who is sedentary, as well as for children who are very active.

It is therefore emphasized that while computing the energy needs of children, the median weight of the group should be taken into account. Recommendations for the energy intake of children, based on Tables 4.21a and 4.21b, should include recommendations for appropriate physical activity as well, without which they cannot remain healthy. While no data are available on the optimum level of physical activity, it is recommended that children should engage in moderately intense physical activity at least for one hour per day. This need not be carried out at a stretch and can be accumulated in bouts of 10-20 minutes. The moderately intense physical activity includes activities that have body displacement and physical effort and can be achieved through individual activities such as walking, running or cycling, or team sports and games.

4.9. Other considerations in computing energy requirements and intake

Source of energy in Indian diets

The main sources of energy in Indian diets, which are predominantly plant food based, are carbohydrate, fat and protein. The recent scientific update considers the unhealthy role of simple sugars and recommends less than 10% of total energy while recommending a wide range of carbohydrate (55-75%) intake from whole grains and legumes, vegetables and fruits⁵⁵.

Table 4.21e: Energy requirements and PAL of Indian boys at different activity levels*

		Sedentary ^a			Vigorous Activity ^b		
Age (y)	Weight (kg)	Total energy requirement ^c (kcal/d)	Total Energy requirement ^d (kcal/kg/d)	PAL	Total Energy requirement ^c (kcal/d)	Total Energy requirement ^d (kcal/kg/d)	PAL
5-6	18.5	1210	65	1.3	1680	90	1.8
6-7	20.5	1270	60	1.3	1760	90	1.8
7-8	22.9	1350	60	1.3	1860	80	1.8
8-9	25.4	1530	60	1.4	2070	80	1.9
9-10	28.1	1610	60	1.4	2190	80	1.9
10-11	31.2	1710	55	1.4	2320	75	1.9
11-12	34.6	1920	55	1.5	2560	75	2.0
12-13	38.9	2040	55	1.5	2720	70	2.0
13-14	44.3	2190	50	1.5	3060	70	2.1
14-15	50.6	2370	50	1.5	3300	65	2.1
15-16	56.6	2690	50	1.6	3520	60	2.1
16-17	61.3	2810	45	1.6	3680	60	2.1
17-18	64.8	2910	45	1.6	3810	60	2.1
18-19	67.3	2970	45	1.6	3890	60	2.1

* To be studied in conjunction with Table 4.21a, which represents energy needs for moderate activity. Energy needs calculated for sedentary and vigorous activity, as given in text above, and include energy required for growth (as in Table 4.16a). Age and weights taken from Table 4.21a; the total energy requirement for each age band is for a child who has attained the 95th percentile for weight. For other weight requirements, use the kcal/kg/day column and multiply by the target weight of the population.

^a Sedentary activity would be typical of a child who did not engage in organized sport/games and went to school by motorized means. See Table 4.21c for example.

^b Vigorous activity would mean walking or cycling long distances every day, engaging in high intensity/energy demanding chores or games for several hours, or intensively practicing sports for several hours a day and several days in a week.

^c Rounded off to the nearest 10 kcal/day

^d Rounded off to the nearest 5 kcal/kg/day

Table 4.21f: Energy requirements and PAL of Indian girls at different activity levels*

		Sedentary Activity ^a			Vigorous Activity ^b		
Age (y)	Weight (kg)	Total energy requirement ^c (kcal/d)	Total Energy requirement ^d (kcal/kg/d)	PAL	Total Energy requirement ^c (kcal/d)	Total energy requirement ^d (kcal/kg/d)	PAL
5-6	18.3	1130	60	1.3	1560	85	1.8
6-7	20.2	1180	60	1.3	1620	80	1.8
7-8	22.4	1330	60	1.4	1710	75	1.8
8-9	25.0	1410	55	1.4	1900	75	1.9
9-10	28.2	1500	50	1.4	2030	70	1.9
10-11	31.9	1590	50	1.4	2150	70	1.9
11-12	36.2	1790	50	1.5	2380	65	2.0
12-13	41.2	1890	50	1.5	2510	60	2.0
13-14	46.0	1990	45	1.5	2640	60	2.0
14-15	50.1	2070	40	1.5	2750	55	2.0
15-16	52.8	2110	40	1.5	2810	55	2.0
16-17	54.7	2150	40	1.5	2860	50	2.0
17-18	55.7	2160	40	1.5	3030	55	2.1
18-19	56.7	2180	40	1.5	3050	55	2.1

* To be studied in conjunction with Table 4.21b, which represents energy needs for moderate activity. Energy needs calculated for sedentary and vigorous activity, as given in text above, and include energy required for growth (as in Table 4.21b). Age and weights taken from Table 4.21b; the total energy requirement for each age band is for a child who has attained the 95th percentile for weight. For other weight requirements, use the kcal/kg/day column and multiply by the target weight of the population.

^a Sedentary activity would be typical of a child who does not engage in organized sport/games and goes to school by motorized means. See Table 4.21c for example.

^b Vigorous activity would mean walking or cycling long distances every day, engaging in high intensity/energy demanding chores or games for several hours, or intensively practicing sports for several hours a day and several days in a week.

^c Rounded off to the nearest 10 kcal/day

^d Rounded off to the nearest 5 kcal/kg/day

Dietary fiber forms an indigestible and important component of plant foods and was until now never considered as sources of energy in the food composition tables. The present Indian food composition has included this in estimation of metabolizable energy by calculating carbohydrate by difference using the difference between 100 minus the sum of proximate components including dietary fibre⁵⁶. Dietary fiber, some of which are soluble and some insoluble undergo fermentation in the colon and yield short chain fatty acids, such as butyric, propionic and acetic acids which are utilized as sources of energy by the colonic cells and by the liver. Hence, they are known to yield energy, 2.6 kcal/g from fermentable foods and no energy from non-fermentable fiber. In conventional foods, 70% of fiber is fermentable: In general, in foods, energy conversion factor for fiber is taken as 2.0 kcal/g. Currently recommended metabolizable energy (ME) factor for different components of food is as follows:

	kcal /g
Protein	4
Fat	9
Carbohydrate	4
Dietary fiber	2

Unlike in earlier years, the present Indian food composition table has recalculated the energy yield of various foods on the basis of their revised content of carbohydrates, proteins, fat and dietary fiber.

4.10. A consideration regarding recommendation for adults

Table 4.23 below gives the current recommendation as a single daily energy requirement value for adults. These values of the daily energy requirement are based on the reference body weights for Indian men and women, which were estimated for a theoretical individual with a BMI of 21 kg/m² and a nominal height that was the 95th percentile of the height distribution from the NNMB, 2016 survey (for more details refer chapter – 3). It is evident that the 95th percentile value for height is an aspirational value. However, if the nominal height were taken to be the 50th percentile value of the NNMB height distribution, along with a BMI of 21 kg/m², the reference body weight would be lower, and consequently, the single value for the daily energy requirement will be lower. The difference in body weight, between using the 50th and 95th percentile value for height in this calculation is about 5 kg.

The present expert committee deliberated on which reference body weight to use for the single value for the daily energy requirement and decided that for the purpose of this specific recommendation, the 95th percentile height value and a BMI of 21 kg/m² would be used, as an aspirational height was desirable. However, it also recommended that as far as possible, energy intake recommendations for a normal healthy population should be based on the actual weight and physical activity level of the target population. When this is unknown, the single value recommendation can be used.

There is always an inherent risk of excess consumption of energy with these approaches, that recommends single daily energy intake in some population. This is mitigated by two approaches: first, to try and ensure that energy requirements for groups are based on their average weight and physical activity levels. If the target group has a higher than ideal body weight, based on their BMI, appropriate considerations should be made for an energy intake appropriate for their ideal weight. Second, that adequate physical activity should be carried out at any BMI and energy intake level. It is critical to maintain a healthy body weight and prevent overweight or obesity. Details on the type and duration of physical activity to be performed are described in the section below.

4.11. Recommendations on physical activity for a sedentary lifestyle

Physical activity is a key determinant of energy expenditure and therefore is fundamental to maintain energy balance and body weight. Additionally, physical activity has protective role to human body as it reduces the risk for coronary heart disease, stroke, diabetes, hypertension and some cancers. For adults, the physical activity includes leisure time physical activity, commute to work (by walking or cycling), occupational, household chores (cleaning, cooking, washing etc.,) or exercises/sports (aerobic exercises).

Based on the WHO 2010, adults (18-64 y) are recommended to perform 150 min of Moderate to Vigorous Physical Activity (MVPA) per week, or 75 min of Vigorous Physical Activity (VPA) per week.⁵⁷ The type of physical activity should be aerobic, meaning an activity which involve large muscles of the body to move in rhythmic manner for a sustained period of time. For example, brisk walking, running, swimming, bicycling etc. In order to meet the recommendation of 150 min/week MVPA, daily 20 min of physical activity can be performed. For individuals having sedentary occupation, the 20 min/d physical activity can be achieved by walking or cycling to work. For elderly, the recommendation on physical activity is same as adults, however, the elderly who cannot perform the recommended physical activity due to certain health problems are advised to be as physically active as per their abilities and conditions allow.

An example of dividing a day's time to perform physical activity for an individual with sedentary occupation is presented in Table 4.22. Note that as the duration of the activity is less in this type of activity pattern, it is essential to include the inclusion of MVPA, which can increase the PAL to 1.43. In order to further increase PAL to 1.5, the duration of MVPA should be increased to 30 min/d. Another way to increase PAL to 1.5 is by avoiding sitting for long durations and increase the time spent in standing and walking/moving around to at least 1-2 h/d. These should be carried out over and above the recommended minimum duration of MVPA (20 min/d).

4.12. Summary of recommended energy requirement for Indians

The final recommended energy levels at different ages are given in Table 4.23. The comparison of energy requirement proposed by present committee and ICMR 2010 is given in Table 4.24.

Table 4.22: Example of type of physical activity performed in a day

Activity	Duration (min)
Sleep	480
Occupation (work)	480
Personal care/eating/watching TV	180
Household chores (cleaning, cooking or washing utensils)	220
Leisure time physical activity (walking in slow pace, gardening, dancing etc.)	60
Aerobic exercise (brisk walking, running, swimming, cycling etc.)*	20

*Recommendation of 150 min/week of MVPA can be met by performing 20 min of MVPA daily. These aerobic exercises should be performed in bouts of at least 10 min duration.

Table 4.23: Energy requirements of Indians at different ages

Age Group	Category	Body weights	Requirement	
			(kcal/d) ^a	(kcal/kg/day)
Men	Sedentary work	65.0	2110	32
	Moderate work	65.0	2710	42
	Heavy work	65.0	3470	53
Women	Sedentary work	55.0	1660	30
	Moderate work	55.0	2130	39
	Heavy work	55.0	2720	49
	Pregnant	55.0 + GWG ^b	+ 350	
	Lactating	55.0+ ^c	+600 +520	
Infants	0-6 m	5.8	550	95
	6-12m	8.5	670	80
Children ^d	1-3y	11.7	1010	86
	4-6y	18.3	1360	74
	7-9 y	25.3	1700	67
Boys	10-12y	34.9	2220	64
Girls	10-12y	36.4	2060	57
Boys	13-15y	50.5	2860	57
Girls	13-15y	49.6	2400	49
Boys	16-18y	64.4	3320	52
Girls	16-18y	55.7	2500	45

^aRounded off to the nearest 10 kcal/d

^bGWG – Gestational Weight Gain. Energy need in pregnancy should be adjusted for actual bodyweight, observed weight gain and activity pattern for the population.

^cGestational Weight Gain remaining after delivery

^dEnergy needs of children and adolescents have been computed for reference children and adolescents; these reference children were assumed to have a *moderate daily physical activity level*. The actual requirement in specific population groups should be adjusted for the actual weight and physical activity of that population (see Table 4.21e and 4.21f), especially when computing the gap between energy requirement and actual intake that needs to be filled by supplementation programmes.

Table 4.24: Energy requirement (kcal/d) as proposed by ICMR 2020

Age group	Category	ICMR 2020	ICMR 2010 ¹³	Difference
		Kcal/d		
Adult Men	Sedentary work ^a	2110	2320	-210
	Moderate work ^a	2710	2730	-20
	Heavy work ^a	3470	3490	-20
Adult Women	Sedentary work ^a	1660	1900	-240
	Moderate work ^a	2130	2230	-100
	Heavy work ^a	2720	2850	-130
	Pregnant	+ 350	+ 350	--
	Lactating (0-6m)	+600	+600	--
	Lactating (7-12m)	+520	+520	--
Infants	0-6 months	550	520	+30
	6-12 months	670	670	--
Children	1-3 y	1010	1060	-50
	4-6 y	1360	1350	+10
	7-9 y	1700	1690	+10
Boys	10-12 y	2220	2190	+30
Girls	10-12 y	2060	2010	+50
Boys	13-15 y	2860	2750	+110
Girls	13-15 y	2400	2330	+70
Boys	16-18 y	3320	3020	+300
Girls	16-18 y	2500	2440	+60

^a Energy requirement calculated as “BMR x PAL”. Refer section 4.5, Table 4.11 and Table 4.13 for details in BMR and PAL values.

References

1. Garry RC, Passmore R, Warnock GM, Durnin JV. Studies on expenditure of energy and consumption of food by miners and clerks, Fife, Scotland, 1952. *Studies on Expenditure of Energy and Consumption of Food by Miners and Clerks, Fife, Scotland, 1952.* 1955(289).
2. Nutrition Advisory Committee of the Indian Research Fund Association (IRFA). Report of the Twelfth Meeting, IRFA, New Delhi, 1944.
3. Patwardhan VN. Dietary Allowances for Indians. Calorie Requirements. Special Report. Indian Council of Medical Research. 1960(35):1-20.
4. FAO. Calorie requirements. Report of the Second Committee on Calorie Requirements. FAO Nutritional Studies, no. 15, Rome: 1957.
5. Kumar S, Kumar N, Sachar RS. Basal metabolic rate in normal Indian adult males. *Indian Journal of Medical Research.* 1961; 49:702-9.
6. Banerjee S. Studies in Energy Metabolism. *Studies in Energy Metabolism.* 1962(43).
7. Rao MN, Sen G, Saha PN, Devi AS. Physiological norms in Indians. Pulmonary capacities in health. *Physiological Norms in Indians. Pulmonary Capacities in Health.* 1961(38).
8. Åstrand I. Degree of strain during building work as related to individual aerobic work capacity. *Ergonomics.* 1967 May 1; 10(3):293-303.
9. Ramanamurthy PS, Dakshayani R. Energy intake and expenditure in stone cutters. *Indian journal of medical research.* 1962; 50:804-9.
10. Joint FAO/WHO/UNU, Energy and Protein Requirements. Report of a Joint FAO/WHO/UNU Expert Consultants, WHO Technical Report Series 724, 1985.
11. FAO F. Nutrition Technical Report Series 1: Human Energy Requirements. Report of a joint FAO/WHO/UNU Expert Consultation 2001. Rome FAO, 2004.
12. Indian Council of Medical Research. Expert Group. Nutrient Requirements and Recommended Dietary Allowances for Indians: A Report of the Expert Group of the Indian Council of Medical Research. Indian Council of Medical Research; 1990.
13. NIN N. requirements and recommended dietary allowances for Indians. A Report of the Expert Group of Indian Council of Medical Research, National Institute of Nutrition, ICMR, Hyderabad. 2010.
14. Haggarty P, Valencia ME, McNeill G, Gonzales NL, Moya SY, Pinelli A, Quihui L, Saucedo MS, Esparza J, Ashton J, Milne E. Energy expenditure during heavy work and its interaction with body weight. *British Journal of Nutrition.* 1997 Mar; 77(3):359-73.
15. Kuriyan R, Easwaran PP, Kurpad AV. Physical activity ratio of selected activities in Indian male and female subjects and its relationship with body mass index. *British journal of nutrition.* 2006 Jan; 96(1):71-9.
16. Jeukendrup A, Diemen AV. Heart rate monitoring during training and competition in cyclists. *Journal of sports sciences.* 1998 Jan 1; 16(sup1):91-9.
17. Bandyopadhyay B, Chattopadhyay H. Energy metabolism in male college students. *The Indian Journal of Medical Research.* 1980 Jun; 71:961-9.
18. Banerjee B. Resting metabolic rate and energy cost of some common daily activities of trained and untrained tropical people. *Energy metabolism in training in tropics.*
19. Kanade AN, Gokhale MK, Rao S. Energy costs of standard activities among Indian adults. *European journal of clinical nutrition.* 2001 Aug; 55(8):708-13.
20. OBEROI K, DHILLON MK, Miglani SS. A study of energy expenditure during manual and machine washing of clothes in India. *Ergonomics.* 1983 Apr 1; 26(4):375-8.
21. Rao S, Gokhale M, Kanade A. Energy costs of daily activities for women in rural India. *Public health nutrition.* 2008 Feb; 11(2):142-50.
22. Sujatha T, Shatrujan V, Venkataramana Y, Begum N. Energy expenditure on household, childcare and occupational activities of women from urban poor households. *British Journal of Nutrition.* 2000 May; 83(5):497-503.

23. Ramana Murthy PS, Belavady B. Energy expenditure and requirement in agricultural labourers. Indian Journal of Medical Research. 1966; 54(10):977-.
24. Nag PK, Dutt P. Circulo-respiratory efficiency in some agricultural work. Applied ergonomics. 1980 Jun 1; 11(2):81-4.
25. Nag PK, Chatterjee SK. Physiological reactions of female workers in Indian agricultural work. Human factors 1981; 23(5):607-14.
26. Das SK, Saha H. Climbing efficiency with different modes of load carriage. The Indian journal of medical research. 1966 Sep; 54(9):866.
27. Samanta A, Chatterjee BB. A physiological study of manual lifting of loads in Indians. Ergonomics 1981; 24(7):557-64.
28. Samanta A, Datta SR, Roy BN, Chatterjee A, Mukherjee PK. Estimation of maximum permissible loads to be carried by Indians of different ages. Ergonomics 1987; 30(5):825-31.
29. Datta SR, Chatterjee BB, Roy BN. The energy cost of rickshaw pulling. Ergonomics 1978; 21(11):879-86.
30. Datta SR, Chatterjee BB, Roy BN. The energy cost of pulling handcarts ('thela'). Ergonomics 1983; 26(5):461-4.
31. Ramanamurthy PS, Dakshayani R. Energy intake and expenditure in stone cutters. Indian journal of medical research. 1962; 50:804-9.
32. Pal AK, Sinha DK. The energy cost of metalliferous mining operations in relation to the aerobic capacity of Indian miners. Ergonomics 1994; 37(6):1047-54.
33. Rodahl K. Physiology of Work. CRC Press; 1989 Nov 13.
34. Lifson N, Gordon GB, McClintock R. Measurement of total carbon dioxide production by means of D₂O₁₈. Journal of Applied Physiology. 1955 May 1; 7(6):704-10.
35. Klein PD, James WP, Wong WW, Irving CS, Murgatroyd PR, Cabrera M, Dallosso HM, Klein ER, Nichols BL. Calorimetric validation of the doubly-labelled water method for determination of energy expenditure in man. Human nutrition. Clinical nutrition. 1984 Mar; 38(2):95.
36. Borgonha S, Shetty PS, Kurpad AV. Total energy expenditure and physical activity in chronically undernourished Indian males measured by the doubly labeled water method. Indian Journal of Medical Research. 2000; 111:24-32.
37. Krishnaveni GV, Veena SR, Kuriyan R, Kishore RP, Wills AK, Nalinakshi M, Kehoe S, Fall CH, Kurpad AV. Relationship between physical activity measured using accelerometers and energy expenditure measured using doubly labelled water in Indian children. European journal of clinical nutrition. 2009 Nov; 63(11):1313-9.
38. Sinha S, Devi S, Kurpad AV, Kuriyan R. Declining energy expenditure among millennials: Revisiting the daily energy requirement. Proceedings of Annual conference of Nutrition Society of India – Poster presented at 50thNSI conference (15-17 Nov 2018).
39. WHO Multicentre Growth Reference Study Group. WHO Child Growth Standards based on length/height, weight and age. Acta paediatrica (Oslo, Norway: 1992). Supplement. 2006 Apr; 450:76.
40. Soares MJ, Shetty PS. Validity of Schofield's predictive equations for basal metabolic rates of Indians. The Indian journal of medical research. 1988 Sep; 88:253.
41. Piers LS, Shetty PS. Basal metabolic rates of Indian women1. Energy metabolism during the menstrual cycle, pregnancy and lactation in well-nourished Indian women. 1993 Aug 1; 47:19.
42. Shetty PS, Soares MJ, Sheela ML. Basal metabolic rates of South Indian males. Bangalore, India: FAO. 1986.
43. Henry CJ, Rees DG. New predictive equations for the estimation of basal metabolic rate in tropical peoples. European journal of clinical nutrition. 1991 Apr 1; 45 (4):177-85.
44. Ferro-Luzzi A, Petracchi C, Kuriyan R, Kurpad AV. Basal metabolism of weight-stable chronically undernourished men and women: lack of metabolic adaptation and ethnic differences. The American journal of clinical nutrition. 1997 Nov 1; 66(5):1086-93.

45. Swaminathan S, Sinha S, Minocha S, Makkar S, Kurpad AV. Are we eating too much? A critical reappraisal of the energy requirement in Indians. Proceedings of the Indian National Science Academy. 2018 Nov 13; 84(4):809-19.
46. Anjana RM, Pradeepa R, Das AK, Deepa M, Bhansali A, Joshi SR, Joshi PP, Dhandhania VK, Rao PV, Sudha V, Subashini R. Physical activity and inactivity patterns in India—results from the ICMR-INDIAB study (Phase-1)[ICMR-INDIAB-5]. International Journal of Behavioral Nutrition and Physical Activity. 2014 Dec 1; 11(1):26.
47. Mayer J, Roy PU, Mitra KP. Relation between caloric intake, body weight, and physical work: studies in an industrial male population in West Bengal. The American journal of clinical nutrition. 1956 Mar 1; 4 (2):169-75.
48. National Nutrition Monitoring Bureau. Report of Second Repeat Survey—Rural (1996–1997). Technical report series No. 18. 1996.
49. Kashyap S. Maternal work and nutrition profile of the pregnant and nursing mothers and their offspring among the quarry workers in Delhi—a prospective study. Unpublished Doctorate Dissertation. 1993.
50. Madhvapeddi R, Rao BS. Energy balance in lactating undernourished Indian women. European journal of clinical nutrition. 1992 May 1; 46(5):349-54.
51. Prema K, Madhvapeddi R, Ramalakshmi BA. Changes in anthropometric indices of nutritional status in lactating women [India]. Nutrition reports international. 1981.
52. Shannawaz M, Arokiasamy P. Overweight/Obesity: An Emerging Epidemic in India. Journal of Clinical & Diagnostic Research. 2018 Nov 1; 12: LC01-LC05.
53. Goel A, Devi T, Kalaivani K, Ramachandran P. Dual nutrition burden in urban women from low income families. Indian Journal of Nutrition & Dietetics. 2020; 57:10-24.
54. Goel A, Devi T, Kalaivani K, Ramachandran P. Effect of lactation on nutritional status in urban women from low middle income families. Indian Journal of Nutrition & Dietetics. 2020; 57:222-239.
55. Mann J, Cummings JH, Englyst HN, Key T, Liu S, Riccardi G, Summerbell C, Uauy R, Van Dam RM, Venn B, Vorster HH. FAO/WHO scientific update on carbohydrates in human nutrition: conclusions. European journal of clinical nutrition. 2007 Dec; 61(1):S132-7.
56. Longvah T, Anantan I, Bhaskarachary K, Venkaiah K, Longvah T. Indian food composition tables. Hyderabad: National Institute of Nutrition, Indian Council of Medical Research; 2017.
57. World Health Organization. WHO Global recommendations on physical activity for health. Geneva: World Health Organization; 2011.

5. PROTEIN AND AMINO ACIDS

Dietary proteins provide amino acids for the synthesis of body proteins, which are structural and biologically active proteins (i.e. enzymes, hormones), and for other biologically important nitrogenous compounds. Adequate dietary protein is essential during growth when new tissue proteins are being synthesized. In an adult, dietary protein is essential for synthesis of new proteins to replace those that are being broken down. During pregnancy, additional protein is necessary for synthesis of foetal, placental and maternal body proteins that increase with increase in maternal body weight. In lactating women, additional protein is necessary for synthesis of breast milk proteins.

Dietary proteins should supply the nine nutritionally essential amino acids (EAA), of the total twenty, in proper proportions and in adequate quantities to synthesize tissue proteins in the body. The other eleven amino acids, though required for protein synthesis, are not considered nutritionally essential since the body can synthesize them from other carbon and N sources. Since most of the body N requirements are met by protein nitrogen, protein and N requirements are used interchangeably.

Human protein requirements are determined by methods such as (a) N balance and (b) Obligatory N losses adjusted for efficiency of protein utilization¹. Recently, the Indicator Amino Acid Oxidation (IAAO) method has been used to define protein requirements for adult men and children, however, the method reports significantly higher values than the current recommendation² and are not used in the present report. At present, greater reliance is placed on the N balance method, which reflects the physiological condition of the subject and provides a true estimate of the protein requirement of the body at different ages. The 1985 and 2007 recommendations of FAO/WHO/UNU Consultations^{3,4} are based largely on N balance method.

Globally a considerable experimental data on N balance in adults are available and these are useful in establishing the protein requirements. There have been some systematic studies on N balance in children and protein accretion studies in infants, children, pregnant and lactating mothers consuming their habitual diets, which are useful in arriving at the protein requirement for these physiological groups⁴.

5.1. Protein Requirements

5.1.1. Protein requirements of adults

Taking into consideration the available data on protein requirements of Indians of different age groups in relation to ICMR recommendations of 1978, 1989, 2010 and the FAO/WHO/UNU/Consultations of 1985 and 2007, the present Expert Group of the ICMR adopted the following guidelines in fixing the protein requirement of Indians from different physiological age groups.

1. The human protein requirement should satisfy the currently established indispensable amino acid requirements as recommended by FAO/WHO/UNU Consultation of 2007⁴.
2. N balance data (both long term and short term) obtained in normal, healthy well-nourished subjects should form the basis of fixing adequateN (and protein) requirements.
3. Daily protein requirement of different age groups should be expressed as gram per kg (g/kg). The safe level of requirement should be expressed as Mean + 1.96 SD while Mean – 1.96 SD should indicate the minimal requirement. These two extremes include 95% of the entire population within their limits, which extend to the 97.5 and 2.5 percentile of the populations respectively.
4. The daily requirement of protein for individuals of different age groups should be derived by a two-step process: First, the level of requirement per kg according to age has to be chosen and then

the actual daily requirement has to be computed for a given age by multiplying the requirement per kg by the standard age specific body weights (as per the standard weights provided in Chapter 3 Table 3.6). For adolescents (10-18 years) and adults, the requirement needs to be categorized by gender.

5. In the 1985 recommendations of FAO/WHO/UNU, the available long-term N balance studies were considered, and the minimal protein requirement of 0.60 g/kg/day for adults was recommended³. However, the FAO/WHO/UNU 2007 Consultation considered all the available N balance studies (~ 19) with a total of 235 healthy adult subjects. A zero N balance was considered to be the criterion for estimating the protein requirement. A median N requirement of 105 mg N/kg/day (equivalent to 0.66 g protein/kg/day) was derived from the log normal distribution (corresponding to a median value of 4.65 on the log scale), with 95% confidence intervals as 101 and 110 mg N/kg/day, for the entire sample of 235 subjects weighted equally. In addition, considering an intra- and inter-study variability, and intra- and inter individual variability, the consultation arrived at an estimate of 0.12 as the between-individual SD of the log requirement. On basis of this, the safe intake was calculated as the log median intake plus 1.96×0.12 . This value was further exponentiated to arrive at a safe requirement of 133 mg N/kg/day⁴.
6. Based on the above estimates of median and safe N requirements i.e. 105 mg N/kg/day and 133 mg N/kg/day, the protein requirements would be 0.66 g/kg/day and 0.83 g/kg/day respectively after multiplying by a factor of 6.25. This value is similar to that reported by FAO/WHO/UNU, 2007⁴.

The earlier committee had applied a protein digestibility amino acid score (PDCAAS) correction to the safe protein intake for all the age groups. This was based on the low digestibility of a standard Indian diet of about 85%. However, the protein requirement is estimated for a high-quality protein. Specific dietary proteins to meet this requirement should be corrected for their specific PDCAAS values. The present committee has therefore removed the PDCAAS correction for safe protein intake for all the age groups.

Factorial method for calculating protein requirements for maintenance in adults

The factorial method of assessing protein requirement of an individual involves estimation of obligatory loss of N through faeces, urine and skin (primarily sweat), and miscellaneous means (e.g., hair, tooth brushing, and exhaled ammonia). The obligatory loss of urinary and faecal N has been assessed by maintaining an individual on a protein-free diet and estimating the faecal and urinary N excretion. In the FAO/WHO/UNU 2007 report, the protein requirement is defined as the minimum intake of N that enables N equilibrium (zero N balance). A meta-analysis was conducted on 19 published N balance studies to estimate adequate N intake or maintenance requirements in healthy adults⁶. The studies were classified into three major types a) estimation studies: presented N balance data for individual subjects at ≥ 3 intakes of N, b) obligatory studies: measured endogenous or obligatory N losses with very low amounts of dietary protein, c) test studies: estimated requirements with individuals consuming 1 or 2 specific N intake involving longer experimental periods.

Considering the estimates of individual requirements with a minimum of three N intakes and adjusting for the confounding factors like climate of the study sites, age, sex and dietary protein sources of the subjects, the median basal N requirement was 48 mg N/kg/day⁶. This was derived from the intercept of the linear regression of N balance on intake, which gives the N loss at zero intake and was similar to the median value of 47 mg N/kg/day obtained from 14 studies that had been designed to actually measure the obligatory requirement⁶. There are several factors that can influence this loss, principally the energy intake. In terms of protein, this obligatory N loss corresponds to a value of 0.3 g protein/kg/day. However, the efficiency of utilization of protein being relatively low at about 47%,

the requirement of protein would then be the amount required to replace the obligatory loss divided by the efficiency of utilization. This would be 0.64 g protein/kg/day, which is similar to the median requirement of 0.66 g protein/kg/day, found in the meta-analysis of N balance responses from 19 separate studies on 235 individuals⁶. In addition, assuming a CV of 12.5%, the safe intake would be 0.83 g protein/kg/d.

5.1.2. Protein requirement during pregnancy

Protein requirement during pregnancy has been previously assessed by the factorial method as the additional requirement for foetal growth and expansion of maternal tissue. The FAO/WHO/UNU 1985 Consultation³ and 1989 ICMR Expert Group⁷ on Recommended Dietary Allowances used this approach to derive protein requirements during pregnancy. The 1985 Consultation assessed protein needs on a calculated increment of 925 g protein, i.e. the average gain, plus 30% (2SD of birth weight), and used an efficiency of 70% for the conversion of dietary protein to foetal, placental, and maternal tissues. This gave safe levels of additional protein of 1.2, 6.1 and 10.7 g/day in the first, second and third trimesters respectively. On average, it was also decided that 6 g protein/day was recommended as the extra allowance throughout pregnancy, based on the assumption that more protein was deposited early in pregnancy, and that the rate of deposition was lower in later pregnancy.

The 2007 FAO/WHO/UNU Consultation⁴, while using the factorial and the N balance method, also considered newer data on protein deposition during pregnancy, using the total body potassium (TBK) method, in which the TBK accretion was measured in pregnant women, by whole body potassium counting. Using a conversion ratio of 2.15 mEq potassium/g N, potassium accretion was converted to nitrogen accretion, and a total of 686 g protein deposition during pregnancy was reported. The deposition however was not at a uniform rate across pregnancy. Detailed measurements of FFM, based on TBK and total body nitrogen (TBN) (measured by prompt-gamma neutron activation) in women during and after pregnancy, also showed that there was no net accretion or loss of protein during pregnancy, suggesting that during pregnancy protein was deposited only in the foeto-placental tissue⁴.

Similar data are available in Indian pregnant women⁸. Using a whole-body potassium counter which was constructed at St. John's Research Institute⁹, accurate protein deposition measurements were made in Indian pregnant women. As with data used in the 2007 FAO/WHO/UNU Consultation, the naturally present radioactive isotope of potassium (⁴⁰K) was used to measure potassium accretion during pregnancy. Urban, well-nourished pregnant women belonging to middle socioeconomic status were recruited in the first trimester of pregnancy and followed up till the birth of the baby. Potassium accretion was measured at the 1st trimester (\leq 13 weeks), 2nd trimester (14-26 weeks), 3rd trimester (27-40 weeks) and at birth (\leq 7 days). The mean gestational weight gain (GWG) of these 38 pregnant women was 10.7 kg⁸. Their mean potassium accretion was 0.04 g/d and 0.08 g/d during the second and third trimester respectively, showing that the potassium accretion pattern was not uniform across the trimesters, similar to the pattern observed in the 2007 FAO/WHO/UNU Consultation report. A conversion ratio of 2.15 mEq potassium / g N (as mentioned above) was used to estimate the nitrogen accretion. Assuming that total body nitrogen (TBN) was incorporated into the protein, total body protein (g) can be estimated as $6.25 \times TBN^{10}$. Mean protein accretion in Indian pregnant women gaining 10.7 kg was thus calculated to be 2.7 g/d and 5.7 g/d in the second and third trimester respectively. There was no net accretion or loss of protein during pregnancy, reaffirming that during pregnancy, protein gets deposited only into the foeto-placental tissue. To arrive at the dietary requirement, the protein accretion rates must be adjusted for an efficiency of utilisation of dietary protein. Details on the efficiency of utilization of dietary protein in pregnancy are mentioned below.

Nitrogen balance and efficiency of utilization in pregnancy

In all, 273 metabolic balances were available, the majority of which were on women at or beyond 20 weeks of gestation. The average N retention was 1.8 g/d from 20 weeks onwards and 1.3 g/d before 20 weeks. Miscellaneous N losses unaccounted for in these studies, were estimated to be 0.5 g/d. The average theoretical retention (mean during whole pregnancy) was 0.53 g/d, but the observed N retention after correcting for miscellaneous N losses of 0.5 g/day was 1.1 g/day. The efficiency of N utilization calculated from these studies was very low, at about 26%. However, after the data were cleaned by removing 2 subjects with improbable values, the efficiency of utilization was found to be 42% (which is the value used to calculate the dietary protein requirement for deposition in Table 5.1). This figure was also reasonably close to 47% that was derived in non-pregnant adults⁴.

Factorial approach to defining requirements during pregnancy

Using new data obtained from Indian pregnant women⁸, revised additional protein intakes needed during pregnancy were derived for India (Table 5.1). The protein deposition values, (2.7 g/d and 5.7 g/day for the second and third trimester respectively) were adjusted for the efficiency of dietary protein utilization, which was considered to be 42%. To this, were added the additional maintenance costs, which were based on the mid-trimester body weight of pregnant woman and the adult maintenance protein requirement value of 0.66 g/kg/d. The additional estimated average requirement (EAR) thus obtained for the gestational weight gain (GWG) of 10.7 kg, were 8.2 g/d and 18.9 g/d in the second and third trimesters respectively. Assuming a coefficient of variation of 12.5%, the safe levels for additional protein intakes by trimesters were derived from the EAR. For a GWG of 10.7 kg, the safe levels were calculated to be 10.2 g/d and 23.6 g/d for the second and third trimesters respectively⁸.

Since GWG has been observed to be 10-12 kg in Indian pregnant women from low to middle socioeconomic status, protein deposition and requirements for GWG of 10 kg and 12 kg were also computed. For a GWG of 10 kg, the protein deposition would be 2.5 g/d and 5.3 g/day for the second and third trimesters respectively (Table 5.1). For a GWG of 12 kg, if protein deposition was assumed to be proportional to GWG, a deposition rate of 3.0 g/d and 6.4 g/d was calculated for the second and third trimesters respectively (Table 5.2). Previous surveys¹¹ (NNMB 1979) have also reported GWG of 8 kg. Using the new data⁷, protein deposition rates of 2.0 g/d and 4.3 g/d for the second and third trimesters respectively were calculated for a GWG of 8 kg. The safe levels for additional protein intakes were thus calculated to be 7.6 g/d and 17.6 g/d respectively. Comparison of the additional protein requirements for GWG of 8, 10 and 12 kg are presented in Table 5.3.

Table 5.1: Revised recommendations for additional protein intake during pregnancy, for a 10.0 kg gestational weight gain

Tri-mester	Mid-trimester weight gain ^a (kg)	Additional protein for maintenance ^b (g/d)	Protein deposited (g/d)	Dietary protein requirement for ^c deposition (g/d)	Mean extra protein requirement (g/d) ^d	Safe intake (g/d) ^e
2	2.5	1.6	2.5	6.0	7.6	9.5
3	7.5	4.9	5.3	12.7	17.6	22.0

^a Women gaining 10.0 kg during gestation

^b Mid-term increase in weight x estimated average requirement for maintenance for adult 0.66 g/kg/d

^c Protein deposited, adjusted for a 42% efficiency of utilization

^d Estimated average requirement (EAR); Sum of extra maintenance plus protein deposited

^e Safe Intake = Mean extra protein requirement (EAR) + 1.96 x SD extra protein requirement (corresponding to a CV of 12.5%). This requirement refers to high quality protein that meets criteria for digestibility and amino acid score.

Table 5.2: Revised recommendations for additional protein intake during pregnancy, for a 12.0 kg gestational weight gain

Trimester	Mid-trimester weight gain (kg) ^a	Additional protein for maintenance (g/d) ^b	Protein deposited (g/d)	Dietary protein requirement for deposition (g/d) ^c	Mean extra protein requirement (g/d) ^d	Safe intake (g/d) ^e
2	3.0	2.0	3.0	7.2	9.1	11.4
3	9.0	5.9	6.4	15.2	21.2	26.3

^a Women gaining 12.0 kg during gestation

^b Mid-term increase in weight x estimated average requirement for maintenance for adult 0.66 g/kg/d

^c Protein deposited, adjusted for a 42% efficiency of utilization

^d Estimated average requirement (EAR); Sum of extra maintenance plus protein deposited

^e Safe Intake = Mean extra protein requirement (EAR) + 1.96 x SD extra protein requirement (corresponding to a CV of 12.5%). This requirement refers to high quality protein that meets criteria for digestibility and amino acid score.

Table 5.3: Comparison of the additional protein requirements in pregnancy with the earlier 2010 ICMR recommendation

Safe Intake (g/d)			
GWG 12 kg	Trimester 1	Trimester 2	Trimester 3
ICMR 2010	0.6	8.3	27.2
ICMR 2020 ^a	0.6	11.4	26.3
GWG 10 kg			
ICMR 2010	0.5	6.9	22.7
ICMR 2020 ^a	0.5	9.5	22.0
GWG 8 kg			
ICMR 2010	0.4	5.5	18.2
ICMR 2020 ^a	0.4	7.6	17.6

^aFor Trimester 1, values are retained from ICMR 2010⁵

The protein requirements would increase further, if it is met only by protein in the cereal-pulse-milk based low cost Indian diet, which has a PDCAAS of 82.3%. Since, low cost Indian diet for sedentary non-pregnant and non-lactating women provides about 10.6% calories from protein, during the second and third trimester of pregnancy additional quality adjusted protein requirement demands consumption of more energy. Therefore, consumption of high-quality protein foods (such as milk or eggs) is recommended in pregnancy.

These protein allowances, particularly during the 3rd trimester may appear high, and may suggest that protein supplements are required. However, this is not the case when one considers the diet in its totality. For example, if one considers a sedentary pregnant woman, whose pre-pregnant weight was 55 kg, GWG of 10-12 kg, an energy allowance of 350 kcal /day, and an extra allowance of about 22 – 26 g protein/day, the calculated PE ratio of the required diet would be about 11-13%. Therefore, the required P:E ratio of the diet does not increase dramatically despite the higher protein requirement during pregnancy. Indeed, the use of supplements that are very high in protein (greater than 34% P:E ratio) may have some adverse events.

It is therefore important that the higher intake of protein recommended during pregnancy should preferably be obtained from high-quality protein food sources, and not from commercial, high-protein supplements. It is also important to take into account the fact that these computations have been made for the reference woman gaining 10-12 kg GWG; when adjusted to the average Indian woman with pre-pregnancy weight of 47 kg and GWG of 7-8 kg during pregnancy, the EAR and the safe intake (as mentioned above) will be lower and could be met from cereal, pulse and milk-based vegetarian diets. Examples of low cost Indian vegetarian high protein diets for an adult woman during second and third trimester of pregnancy are given in Annexure 5.2. These vegetarian diets will provide an additional energy and protein requirement at each trimester for a GWG of 10 and 12 kg.

5.1.3. Protein requirements during lactation

A factorial approach has also been adopted to derive the protein requirements during lactation. The requirements have been computed based on secretion of 6.4 g protein per day in the milk for 0-6 months and 5.0 g protein per day for 6-12 months. Earlier the FAO/WHO/UNU 1985 Consultation and ICMR 1989 Expert Group assessed the protein content of milk as N x 6.25. The WHO/FAO/UNU Expert Consultation then in 2007 used the mean concentrations of protein and non-protein N (NPN) in human milk to calculate the mean equivalent milk protein output. The human breast milk contains a relatively high concentration of non-protein N equivalent to about 20-27% of total milk N, much of it being urea. Therefore, for calculating the requirements, protein requirement was assumed to be only for the protein component of the total N in the breastmilk. A factor of 6.25 was used to convert the protein N in milk to protein equivalents. The efficiency of conversion of dietary N (EAR) to milk protein equivalents can be assumed to be 47% on Indian diets, as in case of non-pregnant adults. Taking 12.5% as the coefficient of variation and a 1.96 SD on the EAR, the safe intakes were calculated.

The additional mean (EAR) and safe protein intakes at different months of lactation are given in Table 5.4. Round off figures would be 17 g/d as the safe allowance for a lactating woman during the first 6 months and 13 g/d after 6 months.

Table 5.4: Additional protein requirements during lactation

Months post-partum	Milk output (g/d)	Milk output corrected for IWL ^a (g/d)	Protein concentration ^b (g/L)	NPN protein equivalent ^b (g/L)	True protein secreted (g/d)	NPN protein equivalent (g/d)	Estimated Average requirement ^c (g/d)	Safe intake ^d (g/d)
1-6	688	722	8.9	2.5	6.4	1.8	13.6	16.9
6-12	583	612	8.1	2.1	5.0	1.3	10.6	13.2

^a Milk output corrected for insensible water loss (IWL) during test weighing measurement (5%) (Refer Table 4.16)

^b Protein concentration of breastmilk has been taken from FAO/WHO/UNU 2007 report⁴; N converted to protein by using the factor 6.25;

^c Efficiency of milk protein synthesis taken as 47%

^d Mean + 1.96 SD assuming a coefficient of variation of 12.5%

Nitrogen (N) balance during lactation

A N balance study in Indian lactating women¹² indicated a linear relationship between N balance and protein intake over a range of 60-100 g/d. Minimum protein required to maintain N balance in the subjects studied was found to be 1.5 g/kg/d, which after allowing for a high faecal N excretion, was found to be 1.2 g/kg/d. These figures are close to the derived estimates of protein

requirements during lactation, where, women with a weight of about 60 kg after delivery would meet the additional 19 g/d required for lactation in the first 6 months, by increasing their protein intake to about 1.1 g/kg/d.

Overall, the higher level of protein required during pregnancy and lactation computed from N balance, when compared to normal requirements computed from the factorial method or amino acid index method, suggests that the efficiency of conversion of dietary N into foetal and other tissues during pregnancy or milk protein during lactation must be quite low and not 70% as assumed.

5.1.4. Protein requirements in infants, children and adolescents

Obligatory N loss and maintenance requirement

Protein requirements of infants and children are usually computed by the factorial method. The maintenance requirement is first computed separately and to it the growth requirements are added. In the FAO/WHO/UNU 2007 Report⁴, for the maintenance requirement, 10 studies were available, that examined the relationship between protein intake and N balance both above and below maintenance. Data from these multiple N balance studies among children were analysed following a linear regression approach as described for adults. Individual data were fitted to the linear model.

$$N\text{ Balance} = A + (B \times N \text{ intake})$$

Where A (intercept) is the extrapolated N loss at zero N intake and B (slope) the corresponding efficiency of utilization. The following values were derived by regression analysis of data from N balance studies on children (0.5 to 12 years of age) from these studies.

Maintenance requirement reported by FAO/WHO/UNU 2007 (mg N/kg/d):

Individual studies (n=7)	:57 + (0.56 x N intake)
All studies (n=10)	:57 + (0.58 x N intake)
Only milk or egg based studies (n=4)	:62 + (0.66 x N intake)

While the above studies were based on a regression of N balance on N intake, three studies were available, where 57 subjects were measured for N losses at zero or very low protein intake to directly estimate their obligatory N loss. In these three studies, a mean±SD value of 63 ± 12 mg N/kg/d was obtained as obligatory N loss. It may be noticed that the values for obligatory N loss, of 57 mg N/kg/day (from the N balance studies at different levels of N intake) and 63 mg N/kg/day (from the direct obligatory N loss studies) are similar. These values are also higher than obligatory N loss value of 47 mg N/kg/day in adult.

In addition, the reported efficiency of utilization in these studies on infants and children was on an average 56-58%, and 66% in the case of studies with milk/egg. The maintenance requirement is computed from the obligatory N loss, divided by the efficiency of utilization. For all the N balance studies quoted above, this value would be $57.4/0.58 = 98$ mg N/kg/day, and for the animal protein diets, this would be $61.6/0.66 = 93$ g N/kg/day. The mean value for maintenance requirement reported in FAO/WHO/UNU 2007 Report was 108 and 110 mg N/kg/day for individual and overall estimates respectively, but lower at 93 mg N/kg/day in the animal protein (milk/egg) intake studies, which could be attributed to the better digestibility and efficiency of utilization of animal proteins^{4,6}.

Choosing an appropriate maintenance value in a range of ages with such a limited data set was difficult as pointed out in the FAO/WHO/UNU 2007 report. The value chosen for infants up to 6 months of age was based on the maintenance value for milk/egg protein diets, which was 93 mg N/kg/day⁴. This works out to a protein intake of 0.58 g protein/kg/day. For children above 6 months, the value chosen was 110 mg N/kg/day corresponding to 0.68 g protein/kg/day, which was the mean

maintenance requirement from all the N balance studies quoted above. Since this requirement was close to the adult maintenance value, for children above 6 months of age the maintenance requirement was set at 0.66 g protein/kg/day, or similar to that in adults. In addition, there was no *a priori* reason to think that maintenance value in children would differ from that of adults, although the efficiency of utilization may change as growth occurs. Therefore, the critical change in this model occurs at 6 months of age, which also matches the time at which complementary feeding is initiated in addition to breastfeeding. These new values for the maintenance requirement are lower than 120 mg N/kg/d (or 0.75 g protein/kg/day) reported by 1985 FAO/WHO/UNU consultation.

Protein deposition and growth requirement pattern

Protein deposited for growth from 0.5 to 18 years, together with an expression of amino acid composition of whole-body protein allows a substantial improvement in the factorial estimates for overall protein and nutritionally essential amino acid requirement during growth. The average daily rates of protein deposited have been derived from the measurement of whole body potassium^{13,14}.

Children from age 0-2 y

In a longitudinal study, Butte *et al*¹³ followed 76 individual infants from birth to 2 years with measurements at birth 0.5, 3, 6, 9, 12, 18 and 24 months. Data on total protein of each of the 71 individuals who had at least 5 data points including one at 18 or 24 months were fitted into individual quadratic equations:

$$\text{Total protein deposition} = A + (B \times \text{age}) + (C \times \text{age}^2)$$

The derivatives of equations below describe the protein deposition per day for each child:

$$\text{Protein deposited} = B + (2C \times \text{age})$$

Next, individual weight data were fitted to power curves

$$\ln(\text{weight}) = A + (B \times \ln(\text{age}))$$

Then, for each individual 0-2 y children the ratio of these two equations estimated protein deposited per day per kg of body weight (Table 5.5).

Children from age 4-18 y

Protein deposition needs of children (4-18 y) were reported by Ellis *et al*¹⁴ in a cross-sectional study. A single model was fitted to the entire data set for each sex: Data on protein (yearly cohort averages) were fitted into a single cubic curve for each sex,

Males: Protein (kg) = 5.46 - (1.285 x age) + (0.166 x age²) - (0.00433 x age³), r²=0.992

Females: Protein (kg) = 3.91 - (0.925 x age) + (0.139 x age²) - (0.00428 x age³), r²=0.993

These curves were differentiated to give protein deposition rate estimates.

Males: Protein deposited (kg/year) = -1.285 + (0.332 x age) - (0.0130 x age²)

Females: Protein deposited (kg/year) = -0.925 + (0.279 x age) - (0.0128 x age²)

The weight data were fitted to a cubic curve for each sex.

Males: Weight (kg) = 5.42 - (15.0 x age) + (1.89 x age²) - (0.0557 x age³), r²=0.991

Females: Weight (kg) = 2.53 - (4.47 x age) + (0.73 x age²) - (0.0198 x age³), r²=0.972

The ratio of these two functions (adjusted to give daily values), estimate protein deposition per kg.

Since 2 different datasets for protein deposition at different ages were available, a quadratic equation was used to interpolate data between the two data sets for the missing ages between 2 and 4 years⁴. The final growth and protein deposition values for each year are given in Table 5.5. These values for growth, particularly during the first year of life are slightly lower than previous estimates up to 3 months of age, and slightly higher thereafter.

Variability of protein deposition

The data on the variability of protein deposition is also an important consideration, as it is a part of the total variability of the requirement in a factorial model. In the younger age group of children (0-2 years), directly observed variability (CV) of the rate of protein deposition was available. It was a mean of 24% for the entire age range and was higher as the rate of growth slowed down.¹³ This variability was used in the factorial model for infants up to the age of 6 months. In the older children, directly observed data were not available for the variability of deposition. These were derived from longitudinal data that allowed for estimating the variability of velocity of growth, along with assumptions of fraction of weight as protein, as growth progressed.

Table 5.5: Protein deposition in infants, toddlers and children^a

Age (Y)	Protein deposition g/kg/d				
	Females	Males	Both sexes	SD ^b	Group
0.5	0.266	0.266	0.266	0.035	Infants
1.0	0.168	0.168	0.168	0.031	
1.5	0.108	0.108	0.108	0.029	
2.0	0.076	0.073	0.075	0.026	
3.0	0.044	0.034	0.039	0.022	
4.0	0.026	0.013	0.020	0.019	Pre-school children
5.0	0.022	0.009	0.016	0.017	
6.0	0.038	0.032	0.035	0.016	
7.0	0.048	0.048	0.048	0.016	
8.0	0.051	0.055	0.053	0.016	
9.0	0.050	0.056	0.053	0.017	School children
10.0	0.047	0.054	0.051	0.017	
11.0	0.043	0.050	0.047	0.018	
12.0	0.037	0.045	0.041	0.018	
13.0	0.031	0.041	0.036	0.018	
14.0	0.025	0.036	0.031	0.017	Adolescents
15.0	0.018	0.032	0.025	0.015	
16.0	0.012	0.027	0.020	0.012	
17.0	0.005	0.023	0.014	0.008	
18.0	0.000	0.018	0.009	0.005	

^a Derived from Butte *et al* 2000¹³ and Ellis *et al* 2000¹⁴

^b On average, the CV of protein deposition was 24% (till 2 y) from Butte *et al* 2000¹³ and from longitudinal data on velocity of growth for older children (2-18 y)¹⁴.

Factorial estimates of protein requirements of 0-18y

The estimated average requirement for years 0-18, is calculated, as the sum of maintenance requirement plus protein deposited.

$$\text{EAR (mg/kg/day)} = \text{Maintenance} + (\text{Deposition} / \text{Efficiency of utilization})$$

Maintenance is calculated assuming that maintenance in young children is 0.58 g/kg/d which increase at 6 months to the adult value of 0.66 g/kg/d. In the case of infants below the age of 6 months, efficiency of utilization of protein for growth was assumed to be 66%, while beyond that age, it was 58%. Further safe levels were calculated as the mean requirement plus 1.96 times the SD (with 1SD calculated as the root mean square of the SDs for maintenance and growth). The safe level of protein intake for infants up to the age of 6 months is given in Table 5.6. These values are lower than those provided by the 1985 FAO/WHO Consultation. Values for boys and girls are similar up to the age of 10 years and are given in Table 5.7.

Boys and girls have different growth patterns and their protein deposition rates will be different. Therefore, protein requirement for adolescents is given separately for boys and girls (Table 5.8) although the principles of calculation remain exactly the same as for children up to the age of 10 years.

Table 5.6: Safe level of protein intake (g/kg/d) for infants aged less than 6 months

Age (m)	Maintenance requirement ^a	Growth requirement ^b	Average requirement ^c	Safe level ^d (+1.96 SD)	1985 Report ^e
1	0.58	0.83	1.41	1.77	2.25
2	0.58	0.65	1.23	1.50	1.82
3	0.58	0.55	1.13	1.36	1.47
4	0.58	0.49	1.07	1.24	1.34
6	0.58	0.40	0.98	1.14	1.30

^a calculated from the maintenance requirement (from N balance studies with milk/egg).

^b Protein deposition rates taken from Butte *et al* 2000¹³, adjusted for 66% efficiency of utilization (from N balance studies with milk/egg).

^c Sum of maintenance and protein deposition rate.

^d Mean + (1.96 x SD) values for protein deposition during growth, adjusted for efficiency of utilization, and the maintenance).

^e Values from the 1985 FAO/WHO/UNU consultation³.

These protein requirements of children and adolescents have been based on systematic studies on protein deposition based on total body potassium (TBK) and maintenance requirements, extrapolated from infant to adult¹³. The safe requirements are more systematically derived values than the earlier values, which were based on body weight increases and their protein component. Hence, these values also can be adopted for Indian infants, children and adolescents. Daily total protein intake can be derived from the proposed safe intake per kg per day and the normal body weights of healthy, well-nourished Indian infants, children and adolescents (refer chapter 3, Table 3.3). Safe intakes of protein by Indian children was calculated based on good quality protein as proposed by FAO/WHO/UNU 2007. The protein intake for infants up to 0.5 y are those proposed by FAO/WHO/UNU as they are based on breast milk.

Table 5.7: Safe level of protein intake for children above 6 months up to 10 years (sexes combined)*

Age (y)	g protein / kg body weight /d				
	Maintenance ^a	Growth ^b	Total	Safe level ^c (+1.96SD)	1985 Report ³
0.5	0.66	0.46	1.12	1.31	1.75
1	0.66	0.29	0.95	1.14	1.57
1.5	0.66	0.19	0.85	1.03	1.26
2	0.66	0.13	0.79	0.97	1.17
3	0.66	0.07	0.73	0.90	1.13
4	0.66	0.03	0.69	0.86	1.09
5	0.66	0.06	0.69	0.85	1.06
6	0.66	0.04	0.72	0.89	1.02
7	0.66	0.08	0.74	0.91	1.01
8	0.66	0.09	0.75	0.92	1.01
9	0.66	0.09	0.75	0.92	1.01
10	0.66	0.09	0.75	0.91	0.99

* For total daily protein requirement in each age band, values need to be multiplied by the normative attained weight in that age band. For example, the age band of 10 years represents the class interval from 9.1-10.0 years. The weight of a boy in this age band is 28.0 kg (Chapter 3 in Table 3.3). Then, the total protein requirement will be = 0.91 x 28 = 25.5 g/day.

^a From N balance studies on children

^b Growth requirement is calculated based on protein deposition divided by 0.58 (efficiency of utilization)

^c SD for maintenance based on CV of 12%. SD for growth calculated from SD of protein deposition divided by 0.58 (efficiency of utilisation).

5.2. Amino Acid Requirements

5.2.1. Amino acid requirements for adults, non-pregnant and non-lactating women

The nutritionally essential or indispensable amino acid (IAA) requirements of adult man recommended in the 1985 WHO/FAO/UNU Consultation³, as shown in Table 5.9, were quite low. Many food proteins that were limiting in one or more IAA could supply the recommended IAA, if adequate quantity of the food protein or a combination of food proteins was present in the diet. These earlier recommendations were based on N balance measurements, where several confounding issues, like a high energy intake (that could spare the requirement), were present.

More recent investigations of human amino acid requirements are much more accurate, and are based on measurements of carbon balance, rather than N balance. This is because stable isotopic methods were used, which measured the irreversible oxidative loss of ¹³C carbon, and therefore of the amino acid from the body. These have shown that human IAA requirements are 2-3 times higher than what was recommended earlier⁴. These revised figures are shown in Table 5.9, along with earlier WHO/FAO/UNU 1985 figures for comparison. The WHO/FAO/UNU 2007 Consultation acknowledged that “the (experimental) methods involve a number of assumptions in their interpretation and there is, as yet, no complete consensus as to their relative merits, but that the most reliable approach involved measurements over an entire 24-hour period representative of a normal

day with ^{13}C tracers which can be reliably interpreted in terms of calculation of oxidation rates after some adaptation to the intakes.”

Table 5.8: Safe level of protein intake for adolescent boys and girls (11-18 y)

Age (y)	g protein / kg body weight /d				
	Maintenance ^a	Growth ^b	Total	Safe level ^c (+1.96 SD)	1985 report ³
Boys					
11	0.66	0.09	0.75	0.91	0.99
12	0.66	0.08	0.74	0.90	0.98
13	0.66	0.07	0.73	0.90	1.00
14	0.66	0.06	0.72	0.89	0.97
15	0.66	0.06	0.72	0.88	0.96
16	0.66	0.05	0.71	0.87	0.92
17	0.66	0.04	0.70	0.86	0.90
18	0.66	0.03	0.69	0.85	0.86
Girls					
11	0.66	0.07	0.73	0.90	1.00
12	0.66	0.06	0.72	0.89	0.98
13	0.66	0.05	0.71	0.88	0.98
14	0.66	0.04	0.70	0.87	0.94
15	0.66	0.03	0.69	0.85	0.90
16	0.66	0.02	0.68	0.84	0.87
17	0.66	0.01	0.67	0.83	0.83
18	0.66	0.00	0.66	0.82	0.80

For total daily protein requirement based on attained body weight in each age band, see Table 5.5

^a From N balance studies

^b From Table 5.7 adjusted for efficiency of utilization of 58% from N balance studies on children

^c SD for maintenance based on CV of 12%. SD for growth calculated from SD of protein deposition divided by 0.58 (efficiency of utilization).

The new values for the daily amino acid requirement of adults have therefore, primarily been estimated by using the 24-hour oxidation of ^{13}C -labelled amino acids after some period of adaptation. This was the so-called direct amino acid balance (DAAB) method, requiring measurements with different ^{13}C labelled IAA. However, there were technical difficulties in identifying the enrichment of the specific intracellular precursor pool from which labelled IAA oxidation was taking place. After many experiments that accurately identified the precursor pool for leucine (alpha-ketoisocaproic acid), it was concluded by consensus that the 24-hour ^{13}C -labelled direct amino acid oxidation/balance method could be used definitively only in the case of leucine¹⁵⁻¹⁶. When the same method was applied for lysine¹⁶ and threonine¹⁷, with assumptions, there were problems in arriving at a consensus over the merits of this direct carbon balance method.

Another method of measuring amino acid requirement, which addressed several of the above methodological questions, including the precursor pool enrichment, was the indicator amino acid oxidation and balance (IAAO and IAAB) method^{17,18}. This method also relied on stable isotopes to measure amino acid oxidation, but it differed from the carbon balance approach in that the oxidation of an amino acid *other than* the test amino acid was measured. This amino acid was called the

‘indicator amino acid’. In this case, it was measured by determining leucine balance, since the intracellular pool of leucine from which oxidation occurs (the precursor pool) was alpha-ketoisocaproic acid, which readily equilibrated with the plasma pool, and was hence relatively easily measured.

The leucine balance method was also validated earlier against the N balance method. The theory behind the method is that if one of the amino acids in the diet is below requirement (i.e. limiting), then all other indispensable amino acids cannot be fully utilized for protein synthesis and the excess is therefore oxidized. Experimentally, as the amount of the limiting (test) amino acid is increased, the other essential amino acids will be progressively better utilized and their oxidation rates will progressively fall to a lower limit till the point where the “requirement level” of the test amino acid is reached. The indicator amino acid balance would improve in the same manner. Intakes of the limiting (test) amino acid above this latter intake point, should no longer influence the oxidation of the indicator amino acid, which should remain low and constant, and the balance would remain at zero.

It is critical in these experiments to maintain the intake of the indicator amino acid constant at all levels of intake of the test amino acid, so that the only influence on the oxidation of the indicator amino acid is the intake of the test amino acid. In these difficult experiments, a carbon balance of the indicator amino acid can be obtained, but only after an adequate adaptation to the test diet, as well as with a 24-hour measurement, as is the case for the directly measured carbon balances detailed above. The aim is to detect a “breakpoint” in the curve for oxidation or balance of the “indicator” amino acid against the intake of the test amino acid. Since this method utilizes the carbon balance of a well-characterized amino acid (such as leucine), it is, in effect, a replacement of the N balance method, and when used with the appropriate caveats of adequate adaptation and the measurement of 24-hr balances, is now considered to be the gold standard for measurement for daily amino acid requirements.

The accurate 24h IAAB method was first set up at St John’s Medical College, Bangalore, and was subsequently used to measure the requirements of IAA in Indians, who were both normal and undernourished, over short (1 week) and long (3 week) duration, with and without infections and parasites. These evidence (referred to in Table 5.9 below) provide the primary evidence for the WHO/FAO/UNU 2007 revisions of the indispensable amino acid requirements of man¹⁶⁻²⁸.

Owing to the complexity of the design, shorter indicator methods² have been tested and, in general provide similar estimates of essential amino acid requirement. These shorter methods do away with adaptation to the test diet, as well as shorten the actual period of measurement of isotope kinetics to a few hours in the fed state. They are also simpler to perform, since they rely on the measurement of the breath enrichment of ¹³CO₂ as a surrogate for actual amino acid oxidation. Therefore, the shorter methods are not actual balance measurements, and can be considered surrogate measurements. In some cases, they have provided estimates of amino acid requirement that are higher, but in others, give requirement figures that match the long-term indicator 24-hour balance method. On the other hand, the brevity and non-invasiveness of this method provides a way to measure the amino acid requirements of children².

For over 100 years, there has been a concern that the requirements of nutrients be set at the most desirable level. As Atwater²⁹ wrote: “A man may live and maintain body equilibrium on either a higher or lower nitrogen level. One essential question is: what level is the most advantageous? The answer to this question must be sought in broader questions regarding bodily and mental efficiency, general health, strength and welfare”. The challenge of the future is to identify these functional indices of optimal health, particularly with different environmental challenges, which will guide future recommendations for IAA requirements in humans.

5.2.2. Amino acid requirements for infants, children and adolescents

N balance studies have provided the only empirical data available for determination of indispensable amino acid requirements in children. However, due to problems in interpreting the data, they were not utilized; instead the factorial approach was used to calculate the indispensable amino acid requirement from 6 months through to 18 years⁴. The factorial approach based on the maintenance and growth components of the protein requirement was used to estimate the indispensable amino acid requirements⁴.

Table 5.9: Indispensable Amino Acid Requirements: Adults

Amino acid	FAO/WHO/UNU 2007		FAO/WHO/UNU 1985	
	mg/kg/d	mg/g protein	mg/kg/d	mg/g protein
Histidine	10	15	8-12	15
Isoleucine ¹⁹	20	30	10	15
Leucine	39	59	14	21
Lysine	30	45	12	18
Methionine	10	16	-	-
Cysteine	4	6	-	-
Methionine + Cysteine	15	22	13	20
Threonine	15	23	7	11
Phenylalanine + Tyrosine	25	38	14	21
Tryptophan	4	6	3.5	5.0
Valine	26	39	10	15
Total EAA	184	277	93.5	141
Total Protein*	0.66 g/kg/d		0.60 g/kg/d	
Safe level of protein (Mean+1.96 x SD)*	0.83 g/kg/d		0.75 g/kg/d	

*High quality proteins like egg, milk, meat etc. Numeral superscripts are references for Indian studies.

Maintenance component: The amino acid requirements for maintenance was assumed to be similar to adults based on the observation that the average maintenance N requirement of children (110 mg/kg/d) across a wide age range from 6 months to 18 years was similar to the value of 105 mg/kg/d found for adult⁴. Thus, the adult maintenance protein requirement of 0.66 g/kg/d times the adult maintenance amino acid pattern (amino acid requirement x maintenance protein requirement) was used to calculate the maintenance portion of the amino acid requirements⁴.

The scoring pattern based on amino acid requirement is different depending on the age group. In the case of the infant up to the age of 6 months, the amino acid content of breast milk is recognized as the best estimate of amino acid requirements for this age group. The average essential amino acid composition of mixed human milk proteins is given in Table 5.10⁴. It must be recognized however that this pattern of amino acids may provide an intake that is in excess of the infant's needs.

Table 5.10: Amino acid composition of human milk proteins

Amino acid	mg amino acid/g total milk protein
Lysine	69
Threonine	44
Methionine	16
Leucine	96
Isoleucine	55
Valine	55
Phenylalanine	42
Tryptophan	17
Histidine	21

In the case of older children, no satisfactory experimental data were available to determine the amino acid requirement (except for lysine in India; see below), as in the case of adults. Therefore, a factorial approach was used. Since the maintenance and growth requirements for protein were known (as given above), the amino acid composition of the requirement pattern for maintenance (Table 5.11) was multiplied by the maintenance requirement for protein, for each essential amino acid. For protein deposition with growth, the amino acid composition of mixed tissue protein was multiplied by the protein deposited with growth (adjusted for efficiency of utilization of dietary protein), for each amino acid. Finally, sum of the amino acid requirement for maintenance and growth deposition was taken as the estimated average requirement of each amino acid. This factorial approach is shown in Table 5.11. Whole body, total amino acid requirements have also been determined in healthy school age children in India, by the IAAO method³⁰. The measured lysine requirements in Indian school children was 33.5 mg/kg/day, which was similar to the WHO/FAO/UNU⁴ values and is a partial validation of the factorial method.

Table 5.11: Factorial calculation for daily amino acid requirement in toddlers, children, adolescents and adults⁴

			mg/g protein									
			His	Ile	Leu	Lys	SAA	AAA	Thr	Trp	Val	
Tissue pattern ^a			27	35	75	73	35	73	42	12	49	
Maintenance pattern ^b			15	30	59	45	22	38	23	6	39	
Age (y)	Protein requirement (g/kg/d)		Amino acid requirement ^d (mg/kg/d)									
	Maintenance	Growth ^c	His	Ile	Leu	Lys	SAA	AAA	Thr	Trp	Val	
0.5	0.66	0.46	22	36	73	64	31	59	34	9.5	49	
1-2	0.66	0.20	15	27	54	45	22	40	23	6.4	36	
3-10	0.66	0.07	12	23	44	35	17	30	18	4.8	29	
11-14	0.66	0.07	12	22	44	35	17	30	18	4.8	29	
15-18	0.66	0.04	11	21	42	33	16	28	17	4.5	28	
>18	0.66	0.00	10	20	39	30	15	25	15	4	26	

His: Histidine; Ile: Isoleucine; Leu: Leucine; Lys: Lysine; SAA: Sulphur Amino Acids; AAA: Aromatic Amino Acids; Thr: Threonine; Trp: Tryptophan; Val: Valine

^a Amino acid composition (mg/g protein) of mixed tissue protein

^b Adult maintenance scoring pattern

^c Average values of protein deposition for growth from Table 5.5, adjusted for efficiency of utilization of 58%

^d See text above. Sum of amino acids contained in maintenance pattern and in growth protein deposition

5.3. Protein quality

An important factor in establishing human protein requirement on habitual diets is the quality of the habitual dietary proteins in terms of their nutritionally essential (indispensable) amino acid content. Efficiency of utilization of dietary protein depends upon its digestion and absorption of the released amino acids. Plant proteins, mainly in cereals, legumes and vegetables are of poorer quality than animal proteins, not only because of their lower digestibility but also because they are often limiting in one or more of the nutritionally essential amino acids of human requirement. Cereal proteins are generally deficient in lysine and pulses or legume proteins contain low amounts of methionine. However, when both cereals and pulses (legumes) are present in the diet in proper proportions, proteins from these two sources supplement each other and make good each other's deficiencies in lysine or methionine to a significant extent.

Two factors thus important are: the chemical composition of the protein in terms of the requirement pattern (amino acid score), and the digestibility. The product of these two factors: the AAS and the digestibility, provides an index for assessment of protein quality. These indices are Protein Digestibility Corrected Amino Acid Score (PDCAAS) and the Digestible Indispensable Amino Acid score (DIAAS)³¹. Each of these terms is discussed in detail below.

5.3.1. Amino acid score (AAS)

The chemical composition of the protein, in terms of its amino acids, is evaluated with reference to the requirement pattern for the individual. In earlier quality estimates of the biological value of protein, egg protein amino acid composition was to be used for comparison. However, presently a comparison with the amino acid composition of requirement pattern is suggested. A comparison of the amino acid content of the test protein with the reference value will provide the AAS, as depicted in the equation. The amino acid with the lowest score is called the limiting amino acid in that protein. The AAS is calculated as below and expressed either as a ratio to unity (recommended), or on a percentage scale⁴.

$$AAS = \frac{\text{mg of amino acid in 1 g of test protein}}{\text{mg of amino acid in 1 g of requirement pattern}}$$

AAS of some food groups are presented in Table 5.12. In this, the new amino acid requirements (per kg/day) and the new median protein requirement (0.66 g/kg/day) are used to arrive at the score. The implication of the new essential amino acid requirements is: since lysine is the limiting amino acid in many cereals, the AAS, and hence the PDCAAS and DIAAS (see below for a complete description of this index) will be less than the optimal score of 1 or 100, or less than the requirement for a high-quality protein.

Table 5.12: AAS based on FAO/WHO/UNU 2007 and 1985 Consultation

Protein Source	Lysine Content mg/g protein ^a	Lysine score (%) (45 mg/g protein) ^b	Lysine score (%) (16 mg/g protein) ^c
Wheat	33	73	>100
Rice	37	82	>100
Sorghum	23	53	>100
Millet*	32	71	>100
Nuts* / Seeds	34	77	>100
Vegetables*	47	>100	>100
Legumes*	62	>100	>100
Animal Protein*	86	>100	>100

^aIFCT 2017³², ^bFAO/WHO/UNU 2007⁴, ^cFAO/WHO/UNU 1985³

*The following foods were used to represent these food groups; An average value for Bajra, Ragi and Jowar for millets, groundnuts for nuts, spinach for vegetables, red gram dal for legumes, milk for animal protein.

The amino acid scoring pattern (mg/g of protein requirement) for all the age groups is given in Table 5.13. This is in contrast to the 1985 FAO/WHO/UNU consultation³, where the pattern for pre-school children was used generally for all ages. In general, the new FAO/WHO/UNU 2007⁴ requirements are lower than the earlier values that were used for the pre-school child. These values were recommended by ICMR 2010⁵ expert group and have been retained by the present committee.

The foregoing discussion has spelt out safe intake of proteins per kg body weight per day as a sum of maintenance and growth requirement of infants, children and adolescents. Requirement values

for maintenance and growth have been derived more systematically by FAO/WHO/UNU Consultation in its 2007 Report, and those values differ from the figures given by the 1985 Committee. However, since the diets generally consumed in India are predominantly based on plant proteins with a small contribution from milk or animal food, their biological value will be lower than that of the protein presumed by FAO/WHO/UNU 2007 Committee; there is a need to correct for the lower quality of protein from Indian habitual diet. The nutritive value of proteins from Indian diets had been derived on the basis of AAS and digestibility (based on reported average absorption of dietary proteins from several Indian studies) of 80%⁴, and the AAS has been computed from the newly recommended amino acid requirements for different ages and IAA content of proteins of a cereal-pulse-milk-vegetables based diet (Annexure 1). These computations are given in Table 5.14.

Table 5.13: Amino acid scoring pattern for toddlers, children, adolescents and adults (mg/g protein requirement)

Age (y)	His	Ile	Leu	Lys	SAA	AAA	Thr	Trp	Val
0.5	20	32	66	57	28	52	31	8.5	43
1-2	18	31	63	52	26	46	27	7.4	42
3-10	16	31	61	48	24	41	25	6.6	40
11-14	16	30	60	48	23	41	25	6.5	40
15-18	16	30	60	47	23	40	24	6.3	40
>18	15	30	59	45	22	38	23	6.0	39

Values based on the amino acid requirement and the protein requirement for each age group.

His: Histidine; Ile: Isoleucine; Leu: Leucine; Lys: Lysine; SAA: Sulphur Amino Acids; AAA: Aromatic Amino Acids; Thr: Threonine; Trp: Tryptophan; Val: Valine

Table 5.14: AAS for requirement computation (mg/g protein requirement)

Age (y)	His	Ile	Leu	Lys	SAA	AAA	Thr	Trp	Val
Requirement pattern (mg/g protein)									
3-10	16	31	61	48	24	41	25	7	40
11-14	16	30	60	48	23	41	25	7	40
15-18	16	30	60	47	23	40	24	6	40
>18	15	30	59	45	22	38	23	6	39
Amino acid composition of mixed diet (mg/g protein)*									
	27	40	79	46**	32	92	35	12	53
AAS									
3-10 y	100+	100+	100+	96	100+	100+	100+	100+	100+
11-14 y	100+	100+	100+	96	100+	100+	100+	100+	100+
15-18 y	100+	100+	100+	98	100+	100+	100+	100+	100+
>18 y	100+	100+	100+	100+	100+	100+	100+	100+	100+

*Amino acid contents are calculated from balanced diet for moderately active men (Annexure 5.1). For the purpose of calculation, cereals and millets were presumed to be in the ratio 40:40:20 for rice: wheat: millet (Ragi/Bajra/Jowar). Pulse was presumed to be entirely red gram dhal. Green leafy vegetables were presumed to be represented by spinach alone, while ‘other vegetables’ were presumed to be represented by french beans. Potato was considered to be representative of the roots and tubers food group. Banana was selected as the representative of fruit. Source of nutritive value of foods is IFCT, 2017.

** Presuming wheat to be whole wheat. With refined wheat flour, the lysine value (mg/g protein) reduces by about 6.5%.

Note: Amino acid composition of mixed diet (mg/g protein)is slightly different from the previous report as the new food composition table values³² has been used.

Based on the data from Table 5.14, it is evident that on a cereal-legume-milk based diet, lysine is marginally limiting in all age groups except adults.

5.3.2. Digestibility of protein

The digestibility is the proportion of a food protein that is absorbed after digestion, determined by measuring the digestive losses, and is expressed as the proportion of ingested N or amino acids that is absorbed in the intestine following protein consumption.

$$\text{Digestibility (\%)} = (\text{Ingested} - \text{digestive losses}) / \text{Ingested \%}$$

The measurement of the digestive losses can be made in the faeces or at the level of the terminal ileum; the former method is traditionally and widely used to estimate protein digestibility. In fecal digestibility, the proportion of food protein which is absorbed, is computed from measurement of the N content of the food ingested and the N excreted in faeces, taking into account the extent to which faecal N is endogenous, which is measured as faecal N lost on a protein-free diet. The measurement is called *true* fecal digestibility when it is adjusted for the contribution of endogenous N or amino acids sources (such as enzymes, immunoglobulins, intestinal epithelial debris etc.), otherwise, it is termed *apparent* digestibility.

The key difference between fecal and ileal digestibility is related to the colonic microbial N contribution, and therefore, confounding of the estimation of true digestibility. The end products of protein digestion (amino acid, di- and tri-peptides) that are unabsorbed in the small intestine are mainly metabolized by colonic bacteria with the production of ammonia, other bacterial metabolites and amino acids. Ammonia and many of the bacterial metabolites can be absorbed by the colon whereas amino acid absorption in the colon remains questionable. The protein digestibility values obtained by the fecal analysis method are thus overestimated when compared to the ileal analysis method. In addition, differences between fecal and ileal digestibility are particularly important for protein sources which are poorly digested in the upper intestine, increasing the quantity to be fermented in the colon. Furthermore, in the Protein Digestibility Corrected Amino Acid Score (PDCAAS) approach, the same value of faecal protein digestibility is applied to each amino acid. It is known that different amino acids from a same dietary protein source have different digestion and absorption rates, and that each amino acid should be treated as an individual nutrient. A recent FAO expert consultation³¹ proposed that protein quality should be assessed by using ileal digestibility of each IAA in relation to its content in the protein under consideration and has led to a new digestibility index called the Digestible Indispensable Amino Acid Score (DIAAS). This has now been considered as a more accurate methodology for determining digestibility and for its application in protein quality assessment. In the present report both faecal and ileal digestibility values are provided for assessing protein quality, using PDCAAS and DIAAS.

Methods to determine protein digestibility

The classic procedure for determining fecal digestibility in-vivo has been the faecal index method, in which the N excreted in the faeces is subtracted from the amount ingested and the value expressed as a percentage of intake. To determine true digestibility, it is necessary to correct for the amount of faecal N excreted when the subject is consuming either a protein free diet, or a diet with just enough of a highly digestible protein to prevent excessive loss of body protein. Since true digestibility measurements consider the metabolic faecal N which is not of dietary origin, it is always higher than the apparent digestibility. Apparent protein digestibility values increase with increasing protein intakes, whereas true digestibility values are independent of protein intake⁴. The true fecal digestibility values estimated in humans are provided in Table 5.15.

A recently convened FAO expert working group report described approaches to different methods of estimating true ileal protein or IAA digestibility³³. Briefly, the ileal digestibility measure in humans requires the direct collection of ileal digesta, which is performed either by using naso-ileal intubation methods or collection through ileostomy. These methods are invasive and not possible to deploy as routine methods. Stable isotope based methods have been proposed as non-invasive or minimally invasive alternatives, which include the IAAO and the dual isotope tracer approach^{2,34}. The best method currently available to comprehensively measure IAA ileal digestibility is the dual tracer method, which has been developed and used to determine true IAA digestibility for specific foods in Indian adults and children³⁵⁻³⁸. Since this method measures and compares the appearance of labelled amino acids from an intrinsically labelled test and standard protein, it is not confounded by endogenous protein secretion, and is hence a measure of true ileal digestibility. The true ileal IAA digestibility values obtained by this method in South Indian adults and children are provided in Table 5.16 and 5.17 below.

Table 5.15: True fecal digestibility values for various protein sources in humans*

Protein sources	True digestibility (%)	Protein sources	True digestibility (%)
American mixed diet	96	Oatmeal	86
Beans	78	Oats, cereal	72
Brazilian mixed diet	78	Peanut butter	95
Chinese mixed diet	96	Peanuts	94
Corn, cereal	70	Peas, mature	88
Corn, whole	87	Rice, cereal	75
Cottonseed	90	Rice, polished	88
Egg	97	Soy flour	86
Farina	99	Soy protein isolate	95
Filipino mixed diet	88	Sunflower seed flour	90
Indian rice + beans diet	78	Triticale	90
Indian rice diet	77	Wheat flour, white	96
Indian rice diet +milk	87	Wheat gluten	99
Maize	85	Wheat, cereal	77
Maize + beans	78	Wheat, refined	96
Maize + beans +milk	84	Wheat, whole	86
Meat, fish	94		
Milk, cheese	95		
Millet	79		

*Source WHO/FAO/UNU, 2007⁴

Differences in protein digestibility from different foods may arise from inherent differences in the nature of food protein (protein configuration, amino acid bonding), from the presence of non-protein constituents, anti-nutritional factors (dietary fibre, tannins and phytate) and processing conditions that alter the release of amino acids from proteins by enzymatic processes³⁹.

5.3.3. Protein quality indexes

Qualitative differences in food protein sources can be determined by many methods. In this section, some of the traditional methods have been described in brief, followed by discussion on the more recent methods of estimation using the fecal protein and ileal IAA digestibility to compute the PDCAAS and DIAAS.

Table 5.16: True ileal mean indispensable amino acid digestibility of select legume and animal protein sources in South Indian adults

IAA	Chick Pea ^a (%)	Yellow pea (%)	Whole mung bean ^a (%)	De-hulled mung ^a (%)	Egg White ^b (%)	Whole Egg (%) ^b	Chicken Meat ^b (%)	Spirulina ^c (%)
Methionine	71.8	56.1	52.2	64.3	79.8	85.9	92.7	85.9
Phenylalanine	80.9	80.6	73.4	75.1	93.0	95.6	94.4	94.0
Threonine	72.5	66.8	42.5	54.5	88.7	96.2	93.7	87.7
Lysine	60.0	62.1	63.0	63.4	88.8	88.9	95.5	81.1
Leucine	79.5	79.0	67.5	76.3	87.9	87.6	89.1	87.5
I-Leucine	81.4	79.5	75.8	82.9	83.5	85.4	88.8	83.5
Valine	75.6	77.4	63.2	80.0	82.1	86.6	89.6	84.8
Mean IAA	74.6	71.6	63.2	70.9	86.3	89.4	92.0	86.4

^aKashyap *et al*, 2019³⁸; ^bKashyap *et al*, 2018³⁶; ^cDevi *et al*, 2018³⁵

Table 5.17: True ileal mean indispensable amino acid digestibility of select plant and animal protein source in South Indian children (1.5 – 2 years)*

IAA	Rice (%)	Finger millet (%)	Mung bean (%)	Egg (%)
Methionine	79.7	60.1	54.0	85.9
Phenylalanine	83.9	69.4	77.2	89.6
Threonine	73.4	67.2	61.6	90.1
Lysine	78.3	74.5	64.8	93.6
Leucine	78.7	67.2	68.0	86.3
Iso-leucine	80.5	75.4	63.0	85.4
Valine	75.2	65.3	68.0	81.2
Mean IAA	78.5	68.4	65.2	87.4

*Shivakumar *et al*, 2018³⁷

Traditional methods of protein quality assessment

5.3.3.1 Protein efficiency ratio (PER)

PER is one of the earliest and widely used approaches to assess the nutritional quality of proteins for humans by using a rat growth assay. This index determines the ability of a dietary protein to support growth in rapidly growing young rats and is dependent on the amount of protein incorporated in the test diet. The PER is expressed as the body weight gain in gram per gram of test protein consumed. In spite of its use, there have been major criticisms to the use of the method. The most common being its inability to account for the maintenance protein requirement and the errors associated with changes in the body composition of the growing rats. It is now known that PER tends to be overestimated for animal proteins and under estimated for vegetable proteins due to the higher growth rate of rats as compared to humans. Additionally, PER is a measure of total protein quality and does not indicate individual amino acid utilisation³.

5.3.3.2 Biological Value (BV)

Biological value has long been considered as the method of choice for protein quality assessment. It is defined as the “percentage of absorbed N retained in the body” with the test protein as the only source of N in the diet. These values are obtained by measuring the fecal and urinary N loss after test protein ingestion and correcting for the amounts excreted when a N-free diet is fed. It determines quality of a single protein and does not provide information on interaction of protein with other foods in the diet, thus limits its application in human protein requirement studies. In addition, the strict experimental protocol and protein-free feeding protocol is unsuitable for routine application in humans³.

5.3.3.3 Net protein Utilisation (NPU)

Similar to BV, NPU estimates N (test protein) retention, but in this case by determining the “percentage of the dietary protein (N) retained in the body”. It includes both the digestibility (defined as the percentage of the food N absorbed from the gut) and the BV of the test protein. It is directly related to dietary intake of protein unlike BV which is an estimate of absorbed N. Retention of N is measured by balance studies or direct carcass analysis. Although it is the most reliable method among the other traditional methods, the tedious experimental protocol like BV restricts its routine application in humans³.

5.3.3.4 Protein Digestibility Corrected Amino Acid Score

In 1989, the joint FAO/WHO Expert Consultation on Protein Quality Evaluation (FAO/WHO 1990)⁴⁰ suggested that protein quality could be assessed adequately by expressing the content of the first limiting essential amino acid of the test protein compared to the content of the same amino acid in a reference pattern of essential amino acids. This reference pattern was based on the essential amino acid requirements of the preschool-age child as published in 1985 (FAO/WHO/UNU 1985)³. Subsequently, this percentage is corrected for the apparent and true fecal digestibility of the test protein to derive PDCAAS.

$$\text{PDCAAS} = \text{Apparent or true fecal crude protein digestibility} \times \text{Limiting AAS}$$

To calculate the PDCAAS, first the AAS for the limiting IAA of a food protein or mixed meal must be estimated (Table 5.18). For the mixed meal, a weighted average digestibility is used. The AAS of the IAA is then corrected for the apparent or true fecal crude protein digestibility value (Table 5.15), estimated in humans or animal models, as per the data available for a particular food or a mixed diet. Where data is available, it is preferable to use the true ileal protein digestibility estimated in

humans. The PDCAAS index is then calculated as the protein digestibility corrected AAS based on the limiting AA requirement for the specified age group.

Proteins with PDCAAS values exceeding 1 (i.e. 100%) are not considered to contribute additional benefit in humans and are truncated to 1. A PDCAAS score below 1 indicates that at least one amino acid is limiting and a score of 1 indicates that there is no limiting amino acid, in the food or mixed diet to be assessed. Thus, PDCAAS determines the effectiveness with which the absorbed dietary N or amino acids meets the IAA requirement at safe levels of protein intake⁴¹. However, the PDCAAS has been subject to constant criticism, of using a single crude protein or N digestibility value instead of a specific digestibility value of each IAA and using faecal instead of true ileal digestibility.

Table 5.18: PDCAAS of a mixed diet (lysine had the lowest score) for different age groups

Age Group	AA with lowest score	AAS ^a	PDCAAS = AA Score ^b X true fecal digestibility ^b
3-10 y	Lysine	96	96 x 84/100 = 81
11-14 y	Lysine	96	96 x 84/100 = 81
15-18 y	Lysine	98	98 x 84/100 = 82
> 18 y (adult)	Lysine	102	102 x 84/100 = 86

^aTable 5.14, ^b WHO 2007⁴

^b Average true fecal digestibility of a mixed Indian diet (based on rice, legume and milk), Table 5.15

5.3.3.5 Digestible Indispensable Amino Acid Score

To overcome the shortcomings of PDCAAS, FAO recommended replacing the PDCAAS with the new scoring system termed the digestible IAA score (DIAAS)³¹. In DIAAS, the constituent indispensable amino acids (IAA) of a protein source are corrected for its true ileal digestibility and the lowest scoring AA is termed as limiting and this defines it's the DIAAS score. Therefore, there is a need to determine true ileal IAA digestibility of habitually consumed foods and mixed diets, preferably in humans; however, a porcine model has also been recommended as a possible alternative as its digestive physiology is very similar to humans⁴².

$$\text{DIAAS} = \text{True ileal digestibility of limiting amino acid} \times \text{Limiting AAS}$$

In DIAAS calculation, the relative digestible content of the IAAs in the protein is compared to the requirement pattern of same IAAs of a particular age group. Hence, a reference ratio of the digestible amino acid content of each IAA in the test protein to the content of the corresponding amino acid in 1 g of the reference protein is calculated, and the lowest value is considered to be the DIAAS (expressed as percentage). A DIAAS score of 100 indicates that the protein source is of high quality and can satisfy the body protein requirement of a particular age. A DIAAS score below 100 indicates that the protein source is limiting in at least one amino acid. A key difference between the DIAAS and the PDCAAS is that DIAAS requires the use of true ileal digestibility of each amino acid determined preferably in humans or growing pigs or growing rats in that order⁴².

The DIAAS calculation of any food protein involves the following steps– 1) IAA content is expressed as mg/g of protein in the food, 2) individual IAA content (mg/g of protein) are multiplied by their respective true ileal digestibility values to obtain the true ileal digestible IAA content in the food, 3) A ratio of true ileal digestible IAA content (mg/g of protein) and reference IAA pattern (mg/g of protein) is calculated to obtain a digestible IAA reference ratio, 4) The ratio is expressed as a percentage, and the lowest value is considered to be the DIAAS of that food.

The DIAAS of a mixed diet is calculated using the true ileal digestibility of IAA from each food. An example of estimating the DIAAS of a mixed meal is provided in Table 5.19. In this, the digestibility of lysine is taken as the key IAA digestibility (as lysine is the limiting AA in a typical Indian mixed meal), but the same method can be applied for all amino acids. The following steps are involved - 1) protein content (g/100g of mixed diet) of each food is calculated, 2) The IAA content of each food is expressed in mg/g of protein, 3) the individual IAA content (mg/g of protein) of each food is then multiplied by the respective true ileal IAA digestibility values to obtain the true ileal digestible IAA content in mixture (mg/g of protein) 4) A ratio of total true ileal digestible IAA content (mg/g of digestible protein) and reference pattern of limiting amino acid (mg/g of protein) is calculated, to derive a digestible IAA reference ratio, 5) The ratio is expressed as a percentage and lowest value is considered to be the DIAAS of that food.

5.3.3.6 Complementing IAA with mixed diets

In assessing the PDCAAS and DIAAS of the dietary protein intake, it is necessary to consider a diet simulating the habitual diet of any country, or a well-balanced diet to provide all nutrients at the recommended level. A typical Indian habitual diet, well-balanced, but predominantly based on cereals, deriving its proteins from other sources like pulses, vegetables and milk and milk products, is given in Annexure 5.1. This is a low cost well-balanced diet, designed by improving a diet for the poor by improving the content of pulses (legumes), vegetables, green leafy vegetables and milk as well as the fat content. Cereals may be derived from more than one source, viz., rice, wheat and millets. The protein digestibility on mixed vegetarian diets is usually about 84.2% calculated as an average of the true fecal digestibility of Indian rice diet + milk and Indian rice diet + beans⁴. The true fecal digestibility values for different foods and mixed diets are provided in Table 5.15, and the computation of PDCAAS for each age group is provided in Table 5.18.

Proteins from different sources may be present in a diet and they may complement each other and a limiting amino acid in a mixed diet may be different from individual protein sources present in the diet. For example, cereal proteins may be limiting in lysine, but if a mixture of cereals and legumes or legumes and milk proteins are present, lysine, deficiency may be reduced considerably. The mixed proteins from cereals and legumes may complement each other and PDCAAS of proteins from a cereal-pulse based diet will be more than that of a cereal protein.

5.4. Protein energy ratio

Protein energy interrelationship

Protein utilization and deposition are dependent on intake of adequate energy. Adequate non-protein energy from carbohydrate and fat is essential for dietary amino acid to be utilized for protein synthesis and for amino acid related functions in the body. If adequate dietary energy is not available, dietary protein is inefficiently utilized. Similarly, an increase in the energy and protein intake (N intake) has been shown to be separately effective in improving the N balance. The slope of N balance with increase in N intake is steeper at a higher intake of energy than at a lower intake. This has been demonstrated both in children⁴⁴ and adults in India⁴⁵. On the basis of international data, the relation of N balance to N intake and energy intake is given by the following formula⁴:

$$\text{N Balance} = 0.17 \times \text{N Intake} + 1.006 \times \text{Energy Intake} - 69.13$$

The slope of this equation indicates that N balance improves by 1 mg/kg/day per extra 1 kcal/kg/day. A study conducted in Indian preschool children provided a mixed diet (cereal, pulse and milk based) found that decreasing energy intake by 20% increased the protein requirement by 20%⁴⁴. This study reported that at an energy intake of 80 kcal/kg, protein requirement was 1.64 g/kg, while at 100 kcal/kg energy intake, the protein requirement decreased to 1.33 g/kg⁴⁴. Similar relationships

between energy intake and protein intake for N equilibrium were observed in adults engaged in heavy manual labour⁴⁵ in India. The study reported a negative N balance in these adults with a reduction in energy intake by 20% from a habitual energy and protein intake⁴⁵. Therefore, it is essential to consider the protein and energy requirements together on habitual Indian diets. Protein requirement of different age groups can be expressed as ratio of protein energy to total dietary energy requirement (PE ratio). This PE ratio will differ for different ages and also between individuals engaged in different activities (lifestyles). In Table 5.20, safe recommended intake of protein is expressed as the ratio of recommended energy intake. If the PE ratio of any diet is compared with PE ratio of the recommended intake, it will indicate whether the diet will satisfy the protein requirement, when adequate energy is consumed. It will also indicate the level of energy intake below which protein also becomes deficient⁴⁶.

The important issue to consider is the way the PE ratio changes with the energy intake. Since protein requirement is constant at different levels of activity, when the energy requirement changes, the PE ratio also changes, becoming higher with reduced energy requirement, as in sedentary people. In addition, the requirement of protein in the food will also depend on the activity levels.

In children, the PE ratio is usually low, owing to the high energy needs. In the elderly, there has been some suggestion that the protein requirement should be increased. While there is no evidence that if there is an increased protein requirement, it is likely that in the elderly, a sedentary way of life may lead to a drop in their energy requirement. With a constant protein requirement, this drop in the energy requirement will lead to an increased PE ratio of the required diet. Therefore, while the use of PE ratios can be of great value, the issues involved are complicated; hence care is required in both calculating and using such ratios.

Protein quality of a diet is important and will reduce the proportion of protein required in the diet to meet essential amino acid requirements. It is not recommended that high protein diets (with a PE ratio of greater than 15%) to be routinely advocated (see section 5.5). The use of commercial high protein supplements for the elderly, or for pregnant and lactating women, is not encouraged. There is also no evidence that protein intake alone can increase muscle protein deposition in the absence of exercise. With regard to the latter, it is also important to know that if an individual were to exercise, energy requirements would increase and actually decrease the requirement PE ratio. Finally, utilization of protein is dependent on adequate intake of energy and micronutrients. The recommended intake of protein has to be accompanied by a balanced diet that meets all the micronutrient requirements.

5.5 Tolerable Upper Limit (TUL)

The consumption of a wide range of dietary protein intakes in humans are known⁴⁷, with intakes 2 to 3 times more than the RDA and PE ratio of the diet nearing 34%⁴. With an increase in intakes there is a related increase in concentrations of nitrogenous substances, especially urea, both in the blood and urine, which form a part of the body's regulatory process⁴⁷. Nonetheless, a number of undesirable effects have been reported, especially at the very high intakes, but also at more modest levels in children.

Groups of people with high protein intake, for example Eskimos, who consume only meat, maintain a protein intake below 50 percent (estimated as 44%) of energy by eating fat⁴⁸. Additional evidence for a maximum limit for dietary protein can be derived from historical records and anthropological studies, and these provide evidence that very high protein intakes might be toxic⁴⁹. Analysis from old literature noted that consumption of more than about 45% of the dietary energy as protein led to nausea and diarrhea within 3 days and to death in a few weeks, a condition known as "rabbit starvation".

Table 5.19: DIAAS calculation (using lysine only) for different age groups

Food	Composition ^a			True ileal IAA digestibility ^b	Protein content in mixture	True ileal digestibility IAA content in mixture ^c	
	Weight	Protein	Lys				
	(g)	(g/100g food)	mg/g protein				
	A	B	C	D	E = (Ax B)/100	Ex CxD	
Whole wheat	170	10.6	33.1	0.84	18.0	500.6	
Rice	170	7.9	37.0	0.94	13.5	469.5	
Bajra	29	11.0	31.9	0.82	3.1	81.7	
Ragi	29	7.2	28.3	0.82	2.0	47.4	
Jowar	29	10.0	23.1	0.80	2.8	52.5	
Red gram dal	60	21.7	61.6	0.57	13.0	457.2	
Milk	200	3.3	85.9	0.96	6.5	537.7	
Total	686				59.1	2146.4	
Amino acids: mg/g protein (total for each amino acid/ total protein content in the mixture)							
Age group	Reference pattern: mg/g protein					Digestible IAA reference ratio ^d	DIAAS for mixture ^e (%)
3 – 10 years	48					0.76	76
11-14 years	48					0.76	76
15 – 18 years	47					0.77	77
>18 years	45					0.81	81

^aIFCT 2017³², ^bCVB Feed tables 2016⁴³, ^cFor example, calculation is shown only for lysine, where possible all IAA should be included in the calculation.

^dDigestible IAA reference ratio= (Digestible IAA in 1 g of mixed diet/mg of the same IAA in 1 g of the requirement pattern), ^eDIAAS for mixed diet (lowest value of the “digestible IAA reference ratio” expressed as % for each reference pattern³¹;

Note: In this case as this is a mixed diet if the calculated DIAAS exceeded 100%, it would be truncated to 100%

Table 5.20: Protein energy ratio (%) for different age groups

Group		Protein requirement g/kg/d ^a	Energy requirement kcal/kg/d	PE Ratio of requirement
Pre-school children ^b	1-5 years	0.95	79	4.8
School children ^b	6-10 years	0.91	68	5.4
Adolescents ^b	11-17 y (Boys)	0.88	56	6.3
	11-17 y (Girls)	0.86	51	6.7
Adults				
Men (Sedentary)		0.83	32	10.4
Women (Sedentary)		0.83	30	11.1
Men (Moderate active)		0.83	39	8.5
Women (Moderate active)		0.83	37	9.0

PE Ratio = Protein Energy ratio; these values refer to the requirement

^a Safe requirement of high-quality protein

^b Assuming moderately active children and adolescents

Rabbit meat has a very low-fat content, so consumption of enough rabbit meat to satisfy energy requirements resulted in very high protein intake⁴⁹. However, there have only been case reports, in one of the two Arctic explorers, reporting adverse effects (weakness, nausea, and diarrhea) on high levels of intake (45 % energy as protein) that resolved on reduction of protein intake^{50,51}.

The evidence on high protein intakes on various organ functions and clinical conditions are equivocal. There have been reports of high protein intakes being implicated in chronic diseases such as osteopenia and osteoporosis, renal stones, renal insufficiency, cancer, coronary artery disease, and obesity⁵¹⁻⁵⁵. On the contrary, there are other studies reporting benefits of high protein on diabetes and bone health^{56,57}.

In early life, there is evidence relating a higher protein intake ($\geq 15\%$ energy as protein) during infancy to elevated risk of obesity in adulthood and an early adiposity rebound, in normal growing children⁵². Animal protein sources, particularly dairy products were mostly responsible for this association with adiposity⁵⁵. An Indian study, with nutritional intervention to improve growth, using a relatively lower protein intake (11-13 % of energy) has demonstrated an increased propensity for fat mass gain. In this study, it was observed that the weight gain component of severely undernourished children (6-60 months), comprised predominantly of fat mass (40%)⁵⁸.

High protein supplements during pregnancy (>34% of energy) has shown untoward effects on pregnancy outcome⁵⁹. A high protein supplement (470 kcal and 40 g protein/day) administration to a group of pregnant women in New York was associated with a small, non-significantly higher weight gain, and a higher, non-significant increase in neonatal death with no difference in foetal growth⁶⁰. The TUL for pregnancy has been tentatively set to 30%. From the available literature, with contradicting results on the benefits or ill-effects of a high protein diet, it is difficult to make a recommendation on the TUL. Nevertheless, knowing that a wide range of protein intakes are consumed, it must be assumed that any untoward effects are subtle, long-term and unreported. It would however be prudent to limit the protein intake to less than 40 %, 15 % and 34% of energy, for adults, children and pregnant women respectively.

5.6. Summary of recommended protein requirements for Indians

The final protein requirements for different ages is given in Table 5.21.

Table 5.21: Summary of recommended protein requirements for Indians

Groups		Body weight ^a (kg)	ICMR 2020				
			EAR ^b (g/kg/d)	RDA ^b (g/kg/d)	EAR ^b (g/d)	RDA ^b (g/d)	TUL ^{b,c} (PE ratio)
Adult Men*	Sedentary	65	0.66	0.83	42.9	54.0	<40%
	Moderate						<40%
	Heavy Work						<40%
Adult Women#	Sedentary	55	0.66	0.83	36.3	45.7	<40%
	Moderate						<40%
	Heavy Work						<40%
Pregnant Women ^d	2nd Trimester				+7.6	+9.5	<30%
	3rd Trimester				+17.6	+22.0	<30%
Lactating Women ^e	0-6 months				+13.6	+16.9	<40%
	6-12 months				+10.6	+13.2	<40%
Infants ^f	0-6 months	5.8	1.16	1.40	6.7	8.1	<15%
	6-12 months	8.5	1.04	1.23	8.8	10.5	<15%
Children ^g	1-3y	11.7	0.79	0.97	9.2	11.3	<15%
	4-6y	18.3	0.70	0.87	12.8	15.9	<15%
	7-9y	25.3	0.75	0.92	19.0	23.3	<15%
Boys ^h	10-12y	34.9	0.75	0.91	26.2	31.8	<15%
Girls ^h	10-12y	36.4	0.73	0.90	26.6	32.8	<15%
Boys ⁱ	13-15y	50.5	0.72	0.89	36.4	44.9	<15%
Girls ⁱ	13-15y	49.6	0.70	0.87	34.7	43.2	<15%
Boys ^j	16-18y	64.4	0.70	0.86	45.1	55.4	<15%
Girls ^j	16-18y	55.7	0.67	0.83	37.3	46.2	<15%

^a Body weights are taken from Chapter 3; WHO child growth standard (for 0-5 years)⁶¹, WHO growth reference (for 6-17 years)⁶² and NNMB 2016 urban data (for adults)⁶³

^b EAR, Estimated Average Requirement; RDA, Recommended Dietary Allowance; TUL, Tolerable Upper Limit; PE Ratio, Protein: Energy Ratio

^c TUL for lactating women is not available but set at the value for non-pregnant non-lactating women.

^d Additional protein requirement for pregnant women with GWG of 10 Kg, at 2nd and 3rd trimester(Table 5.1). For GWG of 12 Kg, see Table 5.2.

^e Additional protein requirement for lactating women during 0-6 and 6-12 months (Table 5.4)

^f Average protein requirements of 1-6 months and 0.5-1y (Table 5.6 and 5.7) were used to calculate EAR and RDA of 0-6 and 6-12 month infants respectively

^g Average protein requirements of 1.5-3y, 4-6y and 7-9y (Table 5.7) were used to calculate EAR and RDA of 1-3y, 4-6y and 7-9y children respectively

^h Average protein requirements of 10-12y (Table 5.7 & 5.8) were used to calculate EAR and RDA

ⁱ Average protein requirements of 13-15y (Table 5.8) were used to calculate EAR and RDA

^j Average protein requirements of 16-17y (Table 5.8) were used to calculate EAR and RDA

* For people consuming cereal based diet with low quality protein, the protein requirements are 1 g/kg per day. For men it is 65g/day

For women it is 55g/day

References

1. FAO. Protein Requirements. Report of the Committee on Calorie Requirements, FAO Nutritional Studies No.16, 1957.
2. Elango R, Ball RO, Pencharz PB. Recent advances in determining protein and amino acid requirements in humans. *British Journal of Nutrition*. 2012 Aug; 108(S2):S22-30.
3. Joint FAO/WHO/UNU, Energy and Protein Requirements. Report of a Joint FAO/WHO/UNU Expert Consultants, WHO Technical Report Series 724, 1985.
4. FAO/WHO/UNU, Expert Consultation on Protein and Amino Acid Requirements in Human Nutrition. WHO Technical Report Series No.935, 2007.
5. 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, ICMR, New Delhi, 2010.
6. Rand WM, Pellett PL, Young VR. Meta-analysis of nitrogen balance studies for estimating protein requirements in healthy adults. *The American journal of clinical nutrition*. 2003 Jan 1; 77(1):109-27.
7. 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, ICMR, New Delhi, 1989.
8. Kuriyan R, Naqvi S, Bhat KG, Thomas T, Thomas A, George S, Nagarajarao SC, Sachdev HS, Preston T, Kurpad AV. Estimation of protein requirements in Indian pregnant women using a whole-body potassium counter. *The American journal of clinical nutrition*. 2019 Apr 1; 109(4):1064-70.
9. Naqvi S, Bhat KG, Preston T, Devi S, Joseph J, Sachdev HS, Kurpad AV, Kuriyan R. The development of a whole-body potassium counter for the measurement of body cell mass in adult humans. *Asia Pacific journal of clinical nutrition*. 2018 Nov; 27(6):1190.
10. Butte NF, Ellis KJ, Wong WW, Hopkinson JM, Smith EB. Composition of gestational weight gain impacts maternal fat retention and infant birth weight. *American journal of obstetrics and gynecology*. 2003 Nov 1; 189(5):1423-32.
11. National Institute of Nutrition. Diet Surveys. National Nutrition Monitoring Bureau (NNMB), National Institute of Nutrition, Hyderabad, 1979.
12. Rao BN, Pasricha S, Gopalan C. Nitrogen balance studies in poor Indian women during lactation. *Indian Journal of Medical Research*. 1958; 46:325-31.
13. Butte NF, Hopkinson JM, Wong WW, Smith EO, Ellis KJ. Body composition during the first 2 years of life: an updated reference. *Pediatric research*. 2000 May; 47(5):578-85.
14. Ellis KJ, Shypailo RJ, Abrams SA, Wong WW. The Reference Child and Adolescent Models of Body Composition: A Contemporary Comparison a. *Annals of the New York Academy of Sciences*. 2000 May; 904(1):374-82.
15. Kurpad AV, Young VR. Human indispensable amino acid requirements: new paradigms of measurement and the implication for protein quality. *Indian journal of physiology and pharmacology*. 1999 Jan 1; 43(1):5-24.
16. Kurpad AV, El-Khoury AE, Beaumier L, Srivatsa A, Kuriyan R, Raj T, Borgonha S, Ajami AM, Young VR. An initial assessment, using 24-h [13C] leucine kinetics, of the lysine requirement of healthy adult Indian subjects. *The American journal of clinical nutrition*. 1998 Jan 1; 67(1):58-66.
17. Kurpad AV, Raj T, Regan MM, Vasudevan J, Caszo B, Nazareth D, Gnanou J, Young VR. Threonine requirements of healthy Indian men, measured by a 24-h indicator amino acid oxidation and balance technique. *The American journal of clinical nutrition*. 2002 Oct 1; 76(4):789-97.

18. Kurpad AV, Raj T, El-Khoury A, Beaumier L, Kuriyan R, Srivatsa A, Borgonha S, Selvaraj A, Regan MM, Young VR. Lysine requirements of healthy adult Indian subjects, measured by an indicator amino acid balance technique. *The American journal of clinical nutrition*. 2001 May 1; 73(5):900-7.
19. Kurpad AV, Regan MM, Varalakshmi S, Gnanou J, Lingappa A, Young VR. Effect of cystine on the methionine requirement of healthy Indian men determined by using the 24-h indicator amino acid balance approach. *The American journal of clinical nutrition*. 2004 Dec 1; 80(6):1526-35.
20. Kurpad AV, Regan MM, Raj T, Gnanou JV. Branched-chain amino acid requirements in healthy adult human subjects. *The Journal of nutrition*. 2006 Jan 1; 136(1):256S-63S.
21. Kurpad AV, Raj T, El-Khoury A, Kuriyan R, Maruthy K, Borgonha S, Chandukudlu D, Regan MM, Young VR. Daily requirement for and splanchnic uptake of leucine in healthy adult Indians. *The American journal of clinical nutrition*. 2001 Dec 1; 74(6):747-55.
22. Kurpad AV, Regan MM, Raj T, Varalakshmi S, Gnanou J, Thankachan P, Young VR. Leucine requirement and splanchnic uptake of leucine in chronically undernourished adult Indian subjects. *The American journal of clinical nutrition*. 2003 Apr 1; 77(4):861-7.
23. Raj T, Dileep U, Vaz M, Fuller MF, Kurpad AV. Intestinal microbial contribution to metabolic leucine input in adult men. *The Journal of nutrition*. 2008 Nov 1; 138(11):2217-21.
24. Kurpad AV, Regan MM, Raj T, El-Khoury A, Kuriyan R, Vaz M, Chandukudlu D, Venkataswamy VG, Borgonha S, Young VR. Lysine requirements of healthy adult Indian subjects receiving long-term feeding, measured with a 24-h indicator amino acid oxidation and balance technique. *The American journal of clinical nutrition*. 2002 Aug 1; 76(2):404-12.
25. Kurpad AV, Regan MM, Raj T, Vasudevan J, Kuriyan R, Gnanou J, Young VR. Lysine requirements of chronically undernourished adult Indian men, measured by a 24-h indicator amino acid oxidation and balance technique. *The American journal of clinical nutrition*. 2003 Jan 1; 77(1):101-8.
26. Kurpad AV, Regan MM, Nazareth D, Nagaraj S, Gnanou J, Young VR. Intestinal parasites increase the dietary lysine requirement in chronically undernourished Indian men. *The American journal of clinical nutrition*. 2003 Dec 1; 78(6):1145-51.
27. Kurpad AV, Regan MM, Raj TD, Rao VN, Gnanou J, Young VR. The daily phenylalanine requirement of healthy Indian adults. *The American journal of clinical nutrition*. 2006 Jun 1; 83(6):1331-6.
28. Kurpad AV, Regan MM, Raj TD, Gnanou JV, Rao VN, Young VR. The daily valine requirement of healthy adult Indians determined by the 24-h indicator amino acid balance approach-. *The American journal of clinical nutrition*. 2005 Aug 1; 82(2):373-9.
29. Reeds PJ, Hutchens TW. Protein requirements: from nitrogen balance to functional impact. *The Journal of nutrition*. 1994 Sep 1; 124(suppl_9):1754S-64S.
30. Pillai RR, Elango R, Muthayya S, Ball RO, Kurpad AV, Pencharz PB. Lysine requirement of healthy, school-aged Indian children determined by the indicator amino acid oxidation technique. *The Journal of nutrition*. 2009; 140(1):54-9.
31. FAO. Dietary protein quality evaluation in human nutrition. Report of an FAO Expert Consultation. Food and nutrition paper no 92. Rome: FAO, 2013.
32. Longvah T, Anantan I, Bhaskarachary K, Venkaiah K, Longvah T. Indian food composition tables. Hyderabad: National Institute of Nutrition, Indian Council of Medical Research; 2017.
33. FAO. Research Approaches and Methods for Evaluating the Protein Quality of Human Foods Proposed by an FAO Expert Working Group. Rome: FAO, 2014.
34. Engelen MP, Com G, Anderson PJ, Deutz NE. New stable isotope method to measure protein digestibility and response to pancreatic enzyme intake in cystic fibrosis. *Clinical nutrition*. 2014 Dec 1; 33(6):1024-32.

35. Devi S, Varkey A, Sheshshayee MS, Preston T, Kurpad AV. Measurement of protein digestibility in humans by a dual-tracer method. *The American journal of clinical nutrition*. 2018 Jun 1; 107(6):984-91.
36. Kashyap S, Shivakumar N, Varkey A, Duraisamy R, Thomas T, Preston T, Devi S, Kurpad AV. Ileal digestibility of intrinsically labeled hen's egg and meat protein determined with the dual stable isotope tracer method in Indian adults. *The American journal of clinical nutrition*. 2018 Nov 1; 108(5):980-7.
37. Shivakumar N, Kashyap S, Kishore S, Thomas T, Varkey A, Devi S, Preston T, Jahoor F, Sheshshayee MS, Kurpad AV. Protein-quality evaluation of complementary foods in Indian children. *The American journal of clinical nutrition*. 2019 May 1; 109(5):1319-27.
38. Kashyap S, Varkey A, Shivakumar N, Devi S, Reddy B H R, Thomas T, Preston T, Sreeman S, Kurpad AV. True ileal digestibility of legumes determined by dual-isotope tracer method in Indian adults. *The American journal of clinical nutrition*. 2019 Oct 1; 110(4):873-82.
39. Jansman AJ, Longstaff M. Nutritional effects of tannins and vicine/convicine in legume seeds. 1993. InProceedings of the 2nd International Workshop on Antinutritional Factors (ANFs) in Legume Seeds. Recent Advances of Research in Antinutritional Factors in Legume Seeds (eds. AFB Van der Poel, J. Huisman, HS Saini). EAAP Publication, Wageningen, The Netherlands (pp. 301-316).
40. Joint FA, Consultation WE. Protein Quality Evaluation FAO Food and Nutrition Paper 1990;51.
41. Schaafsma G. The protein digestibility–corrected amino acid score. *The Journal of nutrition*. 2000; 130(7):1865S-7S.
42. Guilloteau P, Zabielski R, Hammon HM, Metges CC. Nutritional programming of gastrointestinal tract development. Is the pig a good model for man? *Nutrition research reviews*. 2010 Jun; 23(1):4-22.
43. CVB Feed Tables. Chemical compositions and nutritional values of feed ingredients. Product Board Animal Feed, CVB, The Hague, 2016.
44. Iyengar AK, Rao BN, Reddy V. Effect of varying protein and energy intakes on nitrogen balance in Indian preschool children. *British Journal of Nutrition*. 1979 Nov; 42(3):417-23.
45. Iyengar A, Rao BN. Effect of varying energy and protein intake on nitrogen balance in adults engaged in heavy manual labour. *British Journal of Nutrition*. 1979 Jan; 41(1):19-25.
46. Millward DJ. Optimal intakes of protein in the human diet. *Proceedings of the Nutrition Society*. 1999 May; 58(2):403-13.
47. Wu G. Dietary protein intake and human health. *Food & function*. 2016; 7(3):1251-65.
48. Krogh A, Krogh M. A study of the diet and metabolism of Eskimos undertaken in 1908 on an expedition to Greenland. 1915.
49. Speth JD, Spielmann KA. Energy source, protein metabolism, and hunter gatherer subsistence strategies. *Journal of Anthropology and Archaeology*, 1983, 2:1–31.
50. McClellan WS, Du Bois EF. Clinical calorimetry XLV. Prolonged meat diets with a study of kidney function and ketosis. *Journal of Biological Chemistry*, 1931, 87:651–668.
51. McClellan WS, Rupp VR, Toscani V. Clinical calorimetry XLVI. Prolonged meat diets with a study of the metabolism of nitrogen, calcium and phosphorus. *Journal of Biological Chemistry*, 1930, 87:669–680.
52. Koletzko B, Demmelmair H, Grote V, Prell C and Weber M. High protein intake in young children and increased weight gain and obesity risk. *The American journal of clinical nutrition*. 2016;103(2):303-304.

53. Pimpin L, Jebb S, Johnson L, Wardle J. and Ambrosini GL. Dietary protein intake is associated with body mass index and weight up to 5 y of age in a prospective cohort of twins. *The American journal of clinical nutrition*. 2015;103(2): 389-397.
54. Rolland-Cachera MF, Akrout M. and Péneau S. Nutrient intakes in early life and risk of obesity. *International journal of environmental research and public health*. 2016; 13(6):564.
55. Günther AL, Remer T, Kroke A. and Buyken AE. Early protein intake and later obesity risk: which protein sources at which time points throughout infancy and childhood are important for body mass index and body fat percentage at 7 y of age? *The American journal of clinical nutrition*. 2007; 86(6):1765-1772.
56. Rizzoli R, Biver E, Bonjour JP, Coxam V, Goltzman D, Kanis JA, Lappe J, Rejnmark L, Sahni S, Weaver C, Weiler H. Benefits and safety of dietary protein for bone health-an expert consensus paper endorsed by the European Society for Clinical and Economical Aspects of Osteoporosis, Osteoarthritis, and Musculoskeletal Diseases and by the International Osteoporosis Foundation. *Osteoporosis international*. 2018 May 8:1-6.
57. Hamdy O, Horton ES. Protein content in diabetes nutrition plan. *Current diabetes reports*. 2011 Apr 1; 11(2):111-9.
58. Radhakrishna KV, Kulkarni B, Balakrishna N, Rajkumar H, Omkar C, Shatrujan V. Composition of weight gain during nutrition rehabilitation of severely under nourished children in a hospital based study from India. *Asia Pacific journal of clinical nutrition*. 2010 Mar; 19(1):8.
59. Merialdi M, Carroli G, Villar J, Abalos E, Gulmezoglu AM, Kulier R, de Onis M. Nutritional interventions during pregnancy for the prevention or treatment of impaired fetal growth: an overview of randomized controlled trials. *The Journal of nutrition*. 2003 May 1; 133(5):1626S-31S.
60. Rush D, Stein Z, Susser M. A randomized controlled trial of prenatal nutritional supplementation in New York City. *Pediatrics*. 1980 Apr 1; 65(4):683-97.
61. WHO Multicentre Growth Reference Study Group. WHO Child Growth Standards based on length/height, weight and age. *Acta paediatrica* (Oslo, Norway: 1992). Supplement. 2006 Apr; 450:76.
62. WHO. Development of a WHO growth reference for school-aged children and adolescents. *Bulletin of World Health Organization*. 2007; 85:660-7.
63. National Nutrition Monitoring Bureau (NNMB). Diet and nutritional status of urban population in India and prevalence of obesity, hypertension, diabetes and hyperlipidemia in urban men and women. National Institute of Nutrition, Indian Council of Medical Research, NNMB Technical Report No. 27. Hyderabad, India, 2016.

Annexure 5.1

Low cost Indian vegetarian diet for an adult man doing moderate activity

Food group composition^a	Amount (g/d)	Digestible Protein content^b (g/d)	Nutrients (Based on present amount)	
Cereals & Millets	425	32	Protein ^b (g)	57
Pulses (legumes)	60	10	Total fat ^c	48
Green leafy vegetables (GLVs)	150	9	Energy (Kcal)	2452
Other vegetables	100		Calcium (mg)	674
Roots & tubers	100		Iron (mg)	21
Fruits	150		Vitamin A (µg) ^d	965
Milk and milk products	200		Riboflavin (mg)	1
Fats & oils	30		Thiamine (mg)	2
Sugar & Jaggery	28		Vitamin C (mg)	86
PE ratio (%)	9		Niacin (mg)	13

^aCereals and millets are presumed to be in the ratio 40:40:20 for rice: wheat: millets (Ragi/Bajra/Jowar). For calculation purpose, nutritive values of three millets are averaged. Spinach, french beans, potato and banana represents green leafy vegetables, other vegetables, roots and tubers and fruits respectively. Source of nutritive values of foods: IFCT, 2017.

^b Represents digestible protein content of the diet. Protein contents of food groups (except GLVs, other roots and tubers, fruits) are corrected for true faecal digestibility values (Table 5.16).

^c Represents total dietary fat

^d Vitamin A calculation: (β carotene content/8) + (Sum of other carotenoids/16)

Additional note:

- This vegetarian diet with a PE ratio of 9% would meet protein and energy requirements of a moderately active man. PDCAAS value of this diet is 85.6%. Milk intake could be substituted by intake of milk products or curd. This diet will cost around Rs. 20 per day and compares well with the 2019 per capita daily food expenditure.

Annexure 5.2

**Low cost Indian vegetarian high protein diet for an adult woman
during 2nd and 3rd trimester of pregnancy (10 kg GWG)**

Food group composition ^a	2nd trimester Amount (g/d)	Digestible protein content ^b (g/d)	3rd trimester Amount (g/d)	Protein content ^b
Cereals & Millets	325	24	260	20
Pulses (legumes)	65	11	80	14
Green leafy vegetables (GLVs)	250	9	250	9
Other vegetables	100		100	
Roots & tubers	50		30	
Fruits	50		50	
Milk and milk products	300	9	750	23
Fats & oils	30		22	
Sugar & Jaggery	20		5	
Energy (kcal)	2019		2031	
Protein (g) ^b	54		65	
PE Ratio (%)	11		13	

^a Cereals and millets are presumed to be in the ratio 40:40:20 for rice: wheat: millets (Ragi/Bajra/Jowar). For calculation purpose, nutritive values for all three millets are averaged. Spinach, french beans, potato and guava represents green leafy vegetables, other vegetables, roots and tubers and fruits respectively. Source of nutritive values of foods: IFCT, 2017.

^b Represents digestible protein content of the diet. Protein contents of each food group (except for GLVs, roots and tubers, fruits) are corrected for true faecal digestibility values (Table 5.16).

Additional Notes:

- The PDCAAS value of the above mixed diets are ~1.0, due to the additional provision of legume and milk. These vegetarian diets with a P:E ratio of 11% and 13%, for GWG of 10 kg and 11% and 14% for GWG of 12 kg, will provide 350 kcal additional energy as compared to a standard diet for a sedentary non-pregnant and non-lactating woman.
- As shown in the above table, for a 10 kg GWG, milk and legume amounts are increased as compared to the low-cost diet for moderately active man to meet additional protein requirement of 9 g and 22 g in 2nd and 3rd trimester respectively.
- For a 12 kg GWG, additional 60 ml and 125 ml of milk to be provided to suffice extra protein requirement of 2 g and 4.3 g for 2nd and 3rd trimester respectively, when compared to 10 kg GWG.
- Milk intake could be substituted by intake of milk products or curd. Each 100 ml milk intake can be substituted by 15 g legume intake.
- Change in the pulse: cereal ratio from 1:7 in the standard diet (annexure-5.1), to about 1:5 and 1:3 in the 2nd and 3rd trimester respectively. In addition, milk intake increased 1.5 to 4-fold as compared to the standard diet (Annexure-5.1) to meet additional protein requirement.

In a sedentary lifestyle with a solely vegetarian diet, it is difficult to reach a PE ratio of 13%, unless milk intake (particularly toned milk) is substantial (about ~ 700 ml/day), pulse: cereal intake ratio is about 1:3, root, tubers and visible fat are reduced. Inclusion of non-vegetarian foods would help to meet the requirement for high quality protein.

6. FATS AND OILS

6.1 Dietary fat: Chemistry and functions

Dietary fat (lipids) provides energy and essential fatty acids, serves as a vehicle for fat-soluble vitamins and facilitates their absorption. Since fat provides high energy value (9 kcal or 37.7 kJ/g) as compared to carbohydrates or proteins (4 kcal or 16.7 kJ/g), fat content of a diet contributes significantly to its caloric density. Fat enhances texture, taste and flavour of food, reduces its gastric emptying time and thereby affects satiety. Dietary fats have also been at the receiving end of tremendously bad press since the 1980s, mainly due to their putative deleterious role in cardiovascular diseases. However, research in the last decade have thrown up serious doubts about the strength of the ‘diet-heart hypothesis’ leading to the modification of dietary fat recommendations in the Western world.

Dietary fat consists of heterogeneous mixtures of triacylglycerols (triglycerides) and small proportions of phospholipids, glycolipids, monoacylglycerols, diacylglycerols and unsaponifiable fraction composed of fat-soluble chemicals collectively designated as non-glyceride components. Fatty acids, the building blocks of various lipids, are classified into 3 groups: saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs). Most of the SFAs consisting of straight, even-numbered chains of 4-24 carbon atoms are classified as short ($C<10:0$), medium ($C12:0$ and $C14:0$) or long ($C16:0-C24:0$) chain fatty acids. The double bonds in MUFAs and PUFAs can be either *cis* or *trans* relative to the plane of acyl chain while the nutritionally significant MUFAs and PUFAs have double bonds in *cis* configuration. Unsaturated fatty acids (MUFAs and PUFAs) containing one or more double bonds in *trans* configuration are called *trans* fatty acids (TFAs). PUFAs are grouped into two series (n-6 or n-3) depending on whether the double bond closest to the methyl end is located at C6 or C3 position. Humans can synthesize SFAs and MUFAs besides obtaining them from the diet, while they cannot synthesize the parent PUFAs, namely, linoleic acid (LA, $C18:2n-6$) and alpha-linolenic acid (ALA, $C18:3n-3$). LA and ALA are dietary essential fatty acids, and are metabolized by consecutive chain elongase and desaturase enzymes to long chain (LC) n-6 PUFAs {arachidonic acid (AA, $C20:4n-6$) is the predominant LC n-6 PUFA} and LC n-3 PUFAs {eicosapentaenoic acid (EPA, $C20:5n-3$) docosapentaenoic acid (DPA, $C22:5n-3$) and docosahexaenoic acid (DHA, $C22:6n-3$)} respectively¹. These are incorporated into the membrane lipids¹. The current human diets generally furnish high LA levels and low ALA levels. Also, the conversion of ALA to LCn-3 PUFAs is slow and variable due to competitive interactions among LA, ALA and the various intermediates formed during their metabolism to LCPUFAs¹. In addition, several other nutritional and hormonal factors can influence the metabolism of LA and ALA to their respective LC PUFAs.

Functions of fatty acids

In the body, fatty acids used for generation of cellular energy and biosynthesis of membrane lipids and lipid mediators^{1,2}, are essential for development of central nervous system³, modulate lipoprotein metabolism and risk for diet-related non-communicable diseases (DR-NCD), namely, coronary heart disease (CHD), diabetes and cancers⁴⁻⁸.

a) Modulation of membrane structure and functions

As the integral components of cell membranes, fatty acids affect membrane fluidity and lipid protein interactions which alter activity of membrane-related transport systems, ion channels, membrane bound enzymes, cellular receptors for hormones and neurotransmitters. AA and EPA of membrane phospholipids also give rise to an array of potent bioactive eicosanoids (thromboxanes,

prostacyclins and leukotrienes). Eicosanoids derived from AA have strong pro-inflammatory, pro-aggregatory and vaso-constricting effects as compared to the opposing or weak effects of eicosanoids derived from EPA^{1,2}. Recent studies have identified that PUFAs of n-6 and n-3 series and their metabolic products regulate production of lipid cellular mediators namely, lipoxins (from AA), E series resolvins (from EPA), and D-series resolvins and neuro-protectins D1 (from DHA). Lipoxins and resolvins (from both EPA and DHA) have potent anti-inflammatory effects and neuro-protectin D1 has potent anti-inflammatory and neuro-protective effects⁹. Since LA, ALA, and their respective LC PUFAs have distinct biological effects, their absolute levels and their ratio (n-6: n-3) modulate physiological functions. Although a dietary ratio in the range of 5:1 to 10:1 has been recommended¹⁰, emphasis should be on increasing the absolute levels of ALA and LCn-3 PUFAs¹¹.

b) Role of AA and DHA in foetal and infant early growth and development

During the foetal and early infant development, there is a rapid accretion of AA and DHA in infant brain, DHA in retina and AA in the whole body for meeting the demands of rapidly growing tissues/organs. Small amounts of DHA are also present in cell membranes throughout the body. AA and DHA have different and specific roles in neural and behavioural functions. DHA is crucial for the function of rhodopsin for vision and postsynaptic receptors for neurotransmission¹⁻³. The foetus depends completely on the maternal source of LA, ALA, AA and DHA (maternal tissues/stores and dietary intake) and infant obtains these PUFAs through breast milk^{12,13}. Human breast milk is unique in that it provides AA and DHA in addition to other fatty acids. DHA (as provided by human milk) is considered conditionally essential during the early human development. This is because of the high variability in the formation of DHA from ALA and because of its crucial role in normal retinal and brain development in the human. Studies have shown a close association between higher levels of maternal fish consumption during pregnancy and beneficial effects on maternal health as well as foetal and infant development.

c) Role of dietary fatty acids in preventing CHD and other diet-related non-communicable diseases (DR-NCD)

A strong and consistent association is documented between dietary fats and DR-NCD (particularly CHD), from metabolic studies, clinical trials and epidemiological studies^{4-8,14-16}. Elevated serum levels of total cholesterol, low density lipoprotein (LDL) cholesterol and total triglycerides; low serum levels of high density lipoprotein (HDL) cholesterol and increased ratios of total cholesterol: HDL cholesterol are associated with increase in CHD risk and CHD events. Dyslipidemia associated with metabolic syndrome (risk factor for type 2 diabetes) elicits high serum levels of triglycerides (very low density lipoproteins, VLDL and small dense lipoprotein, sdLDL) in addition to the above cited lipid abnormalities.

Dietary fatty acids modify the concentrations of plasma triglycerides and lipoprotein cholesterol fractions which affect CHD risk significantly. Lauric (C12:0), myristic (C14:0) and palmitic (C16:0) acids increase serum LDL and total cholesterol as well as the risk of CHD and CHD events. TFAs are similar to SFAs in increasing LDL cholesterol but, in addition, they lower the protective effects of HDL cholesterol and increase lipoprotein (a) which further increases the CHD risk. Recent studies have shown that replacement of SFAs with PUFAs (LA, ALA, LC n-3 PUFAs) lower the risk of CHD and CHD events. However, the beneficial effects of individual PUFAs on various risk factors of CHD and CHD events are not similar. Compared to higher fat intakes, diets low in fat with high carbohydrate result in a metabolic pattern that increases the risk of type 2 diabetes and CHD. These changes include a reduction in serum HDL cholesterol and increase in the triglyceride concentrations; they show higher responses in postprandial glucose and

insulin concentrations. Studies on experimental animals¹⁷ and limited data in humans suggest that high intakes of either SFAs and or TFAs may contribute to insulin resistance whereas PUFAs may prevent insulin resistance^{5-7,16}. The LC n-3 PUFA provided from fish and other sea foods lower serum triglycerides, postprandial lipemia and have beneficial effects on endothelial function, inflammation, vascular reactivity and ventricular arrhythmias. A strong inverse relationship is documented between fish or LC n-3 PUFA intake and CHD and some types of cancers^{5,8,14}.

Studies on the LDL cholesterol raising effects of dietary cholesterol have shown variable results; some studies have shown that the cholesterolemic effect of dietary cholesterol is reduced when diets provide high levels of PUFAs. Since reactive oxygen species increase the risk of DR-NCD, an adequate intake of natural antioxidants from varied sources is consistent with good health and well being^{4,5,10}.

Non-glyceride components and their nutritional and health promoting effects

Non-glyceride components of fats from animal foods contain cholesterol and fat soluble vitamins (A, E, D) whereas plant foods and vegetable oils have, in addition to fat soluble vitamins (E, D, K and carotenoids), plant sterols and a wide range of other chemical compounds. Plant sterols and some of the unique non-glyceride components (oryzanol and sesame lignans) lower serum LDL cholesterol^{18,19}. Vitamin E, carotenoids, sesame lignans, oryzanol and polyphenols have antioxidant effects. Hypocholesterolemic and antioxidant effects of a combination of nonglyceride components are greater than their individual effects. Increasing plant sterols and other non-glyceride components from natural plant foods and vegetable oils could therefore provide an additional dietary means for prevention and correction of dyslipidemia and increasing antioxidant potential of human diets¹⁹.

6.2 Recommendations of FAO and WHO on dietary fats

The World Health Organization (WHO) and the Food and Agricultural Organization (FAO) have been updating international recommendations on fats in human health.

Recommendations of the first Expert Consultation²⁰ took into account the then available information on functions of fats in food (source of energy and essential fatty acids, vehicle for fat soluble vitamins, cell structure, membrane functions and control of serum total cholesterol) and the safety aspects. In view of the adverse effects of erucic acid, it was recommended that levels of erucic acid in Brassica oils (rapeseed/mustard) should be reduced or these oils should be blended or mixed with other oils.

The second Expert Consultation¹⁰ set the following recommendations after reviewing the many crucial and varied roles played by different types and levels of dietary fats and oils in human nutrition and health:

- Requirements of fats and oils for adults were set between 15-30% E (35% E for active individuals who are in energy balance), at least 20% E for women in reproductive age and 30-40% E for children up to 2 years.
- Infants should be breast fed. Formula milk given to infants should mimic the fatty acid composition of human milk with respect to all fatty acids (including AA and DHA).
- Recommendations for fatty acids considered elevated serum LDL cholesterol as a major risk factor for CHD. An upper limit of 10% E SFAs and <300 mg/d of dietary cholesterol, desirable intakes of LA between 4-10% E and ratio of LA: ALA between 5:1 to 10:1 were recommended.

Intake of leafy vegetables, legumes, fish and sea foods was to be encouraged to achieve a ratio of LA: ALA between 5:1 to 10:1.

- The report acknowledged the need to ensure adequate intakes of essential fatty acids in pregnant and lactating women to meet the requirements for foetal and infant development.
- The recommendations endorsed that consumers should substitute partially hydrogenated vegetable oils (PHVO) which is the major source of TFAs with liquid oils and soft fats. Food manufacturers should reduce TFAs in processed foods and foods high in TFAs should not be considered as low in SFAs.
- Nutritional significance of antioxidants, namely carotenoids, tocopherols and other non-glyceride components was highlighted. Foods high in PUFAs should contain at least 0.6 mg tocopherol (vitamin E) equivalents per gram to stabilize unsaturated fatty acids.
- Use of red palm oil should be encouraged in countries where vitamin A deficiency is a public health problem.

Using the criteria to define the strength of evidence between exposure and disease (convincing, probable, possible and insufficient), WHO/FAO Expert Group on Diet, Nutrition and Prevention of Chronic Diseases endorsed that qualitative composition of fats in the diet has a significant role to play in modifying risk factors of CVD and set the following ranges for population nutrient goals (%E): total fat, 15-30 (at least 20% E is consistent with good health), SFAs, <10%; PUFAs, 6-10; n-6, 5-8; n-3, 1-2; TFAs,<1; MUFAs, by difference; cholesterol <300mg/day⁴.

- The FAO/WHO Expert Consultation on fats and fatty acids in Human Nutrition held in November 2008 in Geneva, Switzerland, reviewed the scientific evidence on nutrient intake values for total fat and fatty acids for different life stages. It also assessed the risks to adequate growth, development and maintenance of health and provided recommendations for infants, children, adults and for women during pregnancy and lactation^{3,5-8,12,21}. Following the same criteria employed in WHO/ FAO to define the strength of evidence⁴, the 2008 Consultation used evidence of sufficient strength to be ‘convincing’ or ‘probable’ to formulate a dietary recommendation²². Some of their conclusions and recommendations are as follows:

There is convincing evidence on the following:

- Energy balance is critical to maintain healthy body weight and ensure optimal nutrient intakes, regardless of macronutrient distribution of energy as % total fat and % total carbohydrates.

SFAs

- Replacing SFAs (C12:0 – C16:0) with PUFAs decreases LDL cholesterol concentration and the total/HDL cholesterol ratio. A similar but lesser effect is achieved by replacing these SFAs with MUFAs.
- Replacing SFAs (C12:0 – C16:0) with carbohydrates decreases both LDL and HDL cholesterol concentration but does not change the total/HDL cholesterol ratio.
- Replacing SFAs (C12:0 – C16:0) with TFAs decreases HDL cholesterol and increases the total /HDL cholesterol ratio.

- Considering the data from epidemiological studies on morbidity and mortality due to coronary heart disease (CHD) and controlled clinical trials (using CHD events and death), it was also agreed that replacing SFAs with PUFAs decrease the risk of CHD.

MUFAs

- Replacing carbohydrates with MUFAs increases HDL cholesterol concentrations.
- Replacing SFA (C12:0 – C16:0) with MUFA reduces LDL cholesterol concentration and total/HDL cholesterol ratio.

PUFAs

Linoleic acid (LA) and alpha-linolenic acid (ALA) are indispensable since they cannot be synthesized by humans. Minimum intake levels for essential fatty acids to prevent deficiency symptoms are estimated to be 2.5% E LA plus 0.5% E ALA.

TFAs

TFAs from commercial Partially Hydrogenated Vegetable Oils (PHVO) increase CHD risk factors and CHD events to a greater extent than what was thought earlier (6.16).

- a) Based on epidemiologic studies and randomized controlled trials of CHD events, 6%E has been fixed as the minimum recommended consumption level of total PUFAs for lowering LDL and total cholesterol concentrations, increasing HDL cholesterol concentrations and decreasing the risk of CHD events.
- b) Whilst ALA may have individual properties in their own right, there is evidence that the n-3 LCPUFAs may contribute to the prevention of CHD and possibly other degenerative diseases of aging.
- c) Based on both the scientific evidence and conceptual limitations, there is no compelling scientific rationale for recommending a specific ratio of n-6 to n-3 fatty acids or LA to ALA, especially if intakes of n-6 and n-3 fats lie within the recommendations established in this report.

In promoting the removal of TFAs, which are predominantly a by-product of industrial processing (partial hydrogenation) usually in the form of PHVO, particular attention must be given to what would be their replacement; this is a challenge for the food industry.

d) Recommendations for total fat and fatty acids:

- Minimum total fat intakes for adults^a
15% E to ensure adequate consumption of total energy, essential fatty acids and fat-soluble vitamins for most individuals.
20% E for women of reproductive age and adults with BMI <18.5, especially in developing countries where dietary fat may be important to achieve adequate energy intake in malnourished populations.
- 30-35% E as maximum total fat intakes for most adults^a
- Recommended intakes of individual fatty acids as summarized in Table 6.1

^aTo optimize health, special attention should be given to both the overall dietary pattern, in terms of types of food consumed, and total energy intakes, in relation also to anthropometric (age group, BMI) and lifestyles characteristics.

6.3 Recommended dietary allowances for Indians in 1990

Earlier recommendations on fat requirement for Indians²³ considered the FAO/WHO 1977 recommendations²⁰ for i) total fat calories between 15-30% E and LA requirements for different groups (adult and children 3% E, pregnant women 4.5% E, lactating women 5.7% E) ii) took into account the LA content from invisible fat, and arrived at a minimum level of 5% E (12 g/p/d) visible fat derived from oils having at least 20% LA. Although a minimal intake of 12 g visible fat can meet LA requirement, a higher level of intake of 20 g/day (10% E) was recommended to provide energy density and palatability to the diet. In pregnant and lactating women, a higher visible fat (30 and 45 g/p/d respectively) was recommended to provide higher level of LA and the necessary calorie density. In young children, ~15%E (25g/child) visible fat was recommended to provide adequate calorie density in their bulky cereal-based diets. The upper limit of visible fat was estimated to be ~50 g/p/day (<20% E) considering the amount of fat from all foods in the diets of urban high income group (10-15% E).

6.4 Sources of fat in Indian diets

A small amount of fat is present in almost every food item (invisible fat). The fat in processed and ready to eat foods (hidden fat) and visible fat (vegetable oil, ghee, butter and *vanaspati*) used for cooking together contribute to total fat intake of an individual.

Fats present as integral components of foods (invisible fat)

Edible plant foods have a low content of fat and SFAs (except for nuts and oilseeds) and are fairly good sources of MUFA and PUFAs. In most cereals, millets, legumes and pulses, fat content ranges between 1.5-3% (higher contents in maize, bajra, bengalgram and soyabean). In cereals, millets and most oilseeds, LA is the major fatty acid whereas pulses / legumes, green leafy vegetables, some oilseeds (soyabean, rapeseed/mustard, perilla seed and flaxseed) and fenugreek are good sources of both LA and ALA²³. Animal foods (fatty dairy products like butter, ghee, whole milk, cream, fatty cheese and fatty meats) provide cholesterol, high amounts of SFAs and are a natural source of TFAs (<5 % of total fatty acids). Structural fats (lean meats) have a fairly high content of LC PUFAs²⁴. The meats of ruminants grazed on grass and in the wild contain less fat, SFAs and higher LCn-3 PUFAs (ratio of LCn-6PUFAs/LCn-3PUFAs is less than 2) as compared to meats of those in captivity fed on grain based rations. Poultry meat contains less fat and cholesterol but appreciable amounts of PUFAs including LC PUFAs. Egg has high cholesterol but is a good source of LA, ALA and DHA^{24,25} and Table 6.7. Fish has less fat, SFAs and cholesterol and is a good source of LCn-3 PUFAs. Fat content and relative contents of EPA and DHA vary in fish and other sea foods²⁴⁻²⁶ and Table 6.7. If farm-raised fish are not fed abundant EPA and DHA, they will have far less of these nutrients than wild caught fish. The total quantity of invisible fat and its fatty acid composition depend on the kind of diet consumed^{17,24,28}.

Visible fats

Vegetable oil used in cooking is the major type of visible fat consumed; *vanaspati* (PHVO) and ghee are the other sources. India has a wide range of edible vegetable oils (groundnut, rapeseed/mustard, soybean, sunflower, sesame, safflower, ricebran, cottonseed and linseed). The type of vegetable oil consumed varies from one part of the country to the other. *Vanasapti* promoted as *desi ghee* is used largely in north India (Haryana, Punjab, Himachal Pradesh, Uttar Pradesh) as cooking medium. In most parts of the country, *vanasapti* is used as a substitute for ghee in Indian sweets and savoury foods. It is also used in preparing commercially fried, processed, ready-to-eat, packaged, frozen, premixed foods and street foods. In recent years, health claims have affected the choice of cooking oil(s) in the urban population. The relative proportions of fatty acids are known to vary in different visible fats (Table 6.2). Depending on the percentage of various fatty acids, fats

and oils can be grouped as oils containing: i) high SFAs ii) high MUFAAs iii) low (<20%), medium (20-40%) or high (>40-70%) LA and iv) both LA and ALA. The traditional rape-mustard seed oils contain ~50% erucic acid (C22:1). Concerns about possible deleterious effects of erucic acid (lipidosis and fibrosis in experimental animals) in humans led to the development of low / zero erucic acid rapeseed variety and the oil is sold as canola oil²⁰. Butter, ghee, coconut oil and palm kernel oils are rich sources of short and medium chain SFAs. Partial hydrogenation of vegetable oils results in the formation of several C 18:1 and C18:2 trans isomers; the chemical composition of these isomers is different from those of ruminant fats. During refining of vegetable oils, deodorization step contributes to formation of C18:2 trans isomers, the contents should be <1 % of total fatty acids. PHVO (vanasapti, bakery fats and margarines) is the main modifiable source of TFAs in Indian diets.

Besides fatty acids, vegetable oils also contain minor components in the non-glyceride fraction which have specific health significance. The composition of non-glyceride components in dietary fats and oils is given in Table 6.3. Plant sterols and other unique components (oryzanol and sesamol) present in the non-glyceride component of vegetable oils contribute to lowering of total and low density lipoprotein cholesterol.

6.5 Fat intake in Indians: An update

Total fat intake is largely variable among rural and urban Indian population. Earlier diet surveys (2008) by the National Nutrition Monitoring Bureau³⁰ show that daily intake of visible fats in rural India (range 6-22 g, median 13 g/consumption unit) is about the same as reported about 25 years back (range 3-20 g, median 10 g/consumption unit)³⁰. The intake of total fat and PUFAs calculated by putting together the total fat (~14g/consumption unit, 6.5% E), and contents of LA and ALA from cereals, millets, pulses/legumes and milk and any one vegetable oil (median 13 g/consumption unit) shows that diets of the rural population (including children, pregnant and lactating women) provide <14 % total fat calories (AMDR-Acceptable Macronutrient Distribution Ranges: 20-35% E). Depending on the type of vegetable oils consumed, levels of LA range between 3 to 7% E (AMDR: 2.5-9% E) except when coconut oil / vanasapti are used. The levels of ALA are generally low (~ 0.2% E) except when mustard /rapeseed oil, linseed oil or soyabean oils are used (AMDR 0.5-2% E).

Subsequent diet surveys by National Nutrition Monitoring Bureau show that the total fat intake in rural (2012)³³ and urban (2016)³⁴ India increased (rural - 20% E; urban - 23% E) compared to the previous survey conducted in 2008 (< 14% E). The visible fat intake in rural and urban population is 5% E (14gm/consumption unit) and 13% E (28gm/ consumption unit) respectively. Although the total fat intake in lactating women in urban and rural population is within the recommended range (urban-22.3% E, rural - 20.3% E), the fat intake in rural pregnant women (15.5 % E) is less compared to the urban pregnant women (23% E). The fat intake in urban children is within the recommended range (26.2% E and 25.0% E for the age group of 3-6 years and 7-9 years respectively) whereas in rural children the fat intake is less (19.8% E and 17.1% E for the age group of 3-6 years and 7-9 years respectively). In urban and rural girls and boys of all the age groups, the fat intake is less (Urban <24% E and Rural < 21% E) compared to the recommended intake (AMDR for girls and boys 25% E). Overall the fat intake in rural population particularly pregnant women, children, girls and boys are less. Efforts to increase the dietary levels of total fat in the rural population would contribute to lifelong health and wellbeing.

Table 6.1: FAO/WHO 2008 recommendations for dietary fatty acids (% E)

1	2	3	4	5	7	8	9	10
Physiological groups/age/ gender	SFAs	MUFAs	TFAs ^c	Total PUFAs ^d (n-6 +n-3)	Total n-3 PUFAs ^e	LA (n-6)	ALA (n-3)	LCn-3 PUFAs ⁱ
Adult Man	U-AMDR 10	By difference ^b	<1	AI 2.5-3.5 ^f AMDR 6 ^g -11 ^h	AMDR 0.5 -2	AI 2-3 ^f AMDR 2.5-9	L- AMDR >0.5	AMDR 250 -2000 ^k mg/d
Adult Woman (NPNL)								300 ^j mg
Pregnant woman								300 ^j mg
Lactating woman								
Infants	0-6 m	HM	HM	-	HM	HM	LA - AI HM composition as % E of total fat AA - AI 0.2-0.3 ^m	AI 0.2-0.3 ^m
	6-24 m	<10	By difference ^b	<1	U-AMDR 15	-	AI 3 - 4.5 U-AMDR 10	AI 0.4-0.6 U-AMDR 3
Children	3-6 y	U-AMDR 8 ^a	By difference ^b	<1	U-AMDR 11	-	n	n
	7-9 y							
Boys	10-12 y	U-AMDR 8 ^a	By difference ^b	<1	U-AMDR 11	-	n	n
	13-15 y							
	16-17 y							
Girls	10-12 y	U-AMDR 8 ^a	By difference ^b	<1	U-AMDR 11	-	n	n
	13-15 y							
	16-17 y							

Reference: 21

% E : percentage total energy; AMDR: accepted macronutrient dietary range; L-AMDR lower limit of AMDR; U-MDR upper limit of AMDR; AI : adequate intake; HM : human milk; AA : arachidonic acid; DHA : docosahexaenoic acid; SFAs: saturated fatty acids; MUFAs: monounsaturated fatty acids; PUFAs: polyunsaturated fatty acids; TFAs : trans fatty acids; LA: linoleic acid; ALA: alpha-linolenic acid

^a children from families with evidence of familial dyslipidemia (high LDL cholesterol) should receive lower SFA but not reduced total fat intake;

^b Total MUFAs: Total fat (%E) - SFAs (% E) - PUFAs (% E), can make upto 15-20%E according to total fat intake;

^c Total TFAs : from ruminants and partially hydrogenated vegetable oils;

^d Total PUFAs : LA +AA+ ALA+EPA+DPA+ DHA;

^e Total n-3 PUFAs : LA+EPA+DPA+DHA; ^fminimum intake levels to prevent deficiency symptoms;

^g minimum recommended level for lowering LDL and total cholesterol, increasing HDL cholesterol concentrations and decreasing the risk of CHD events;

^h to prevent risk of lipid peroxidation particularly when tocopherol intake is low;

ⁱ LCn-3 PUFAs : EPA+DPA+DHA from 1-2 fish meals including oily fish / week; ^jincluding 200mg DHA ;

^k including supplements (fish oil/ algal oil)for secondary prevention of CHD (to prevent increased risk of lipid peroxidation and reduced cytokine production,

^l conditionally essential due to limited synthesis from ALA, critical role in retinal and brain development;

^m % fatty acids AA 0.4-0.6, ALA 0.4-0.6 DHA 0.2-0.36;

ⁿ have not yet been adequately established, recommendations set to be the same as in adults;

Cholesterol :<300mg/day; Natural antioxidants from wide variety of foods (including visible fats).

Table 6.2: Approximate fatty acid composition of dietary fats and oils consumed in India (% of total fatty acids)

Fats/ oils	SFAs*	MUFAs**	LA	ALA
High medium chain SFAs				
Coconut	92 ^{a, d}	6	2	-
Palm kernel	83 ^{b,d}	15	2	-
Butter/Ghee	68 ^{c,e,f}	29	2	1
High SFAs & MUFAs				
Palmolein	39	46	11	<0.5
High MUFAs & Moderate LA				
Groundnut ⁱ	19	41	32	<0.5
Rice bran ^h	17	43	38	1
Sesame ^h	16	41	42	<0.5
High LA				
Cottonseed ^h	24	29	48	1
Corn ^h	12	35	50	1
Safflower ^h	9	13	75	-
Sunflower ^h	12	22	62	-
LA & ALA				
Soybean ^h	14	24	53	7
Canola ^h	6	60 ^j	22	10
Mustard/rapeseed ^h	4	65 ^k	15	14
Flaxseed	10	21	16	53
High TFAs				
Vanasapti ^h	61	34 ^g	5	-

Reference 23,

* SFAs include < C10:0, C12:0 (lauric), C14:0 (myristic), C16:0 (palmitic), C18:0 (stearic) < C 10:0- ^a15, ^b9 ^c15; C12:0 and C14:0- ^d65, ^e14

** Mainly cis C18:1 (oleic) other MUFAs when present indicated against superscripts
TFAs ^f5; ^g5 (Data from Indian Food Composition Tables - 2017);
SFAs and MUFAs
C20-24 ^h1 to 4, ⁱ~8; ^jC22:1 (erucic) ~2, ^kC22:1 (erucic) ~50 and C 20:1(gadoleic) ~ 5

Table 6.3: Non-glyceride components in dietary fats and oils

Non-glyceride components	Oil	Biological and health function
Plant sterols	All vegetable oils	Hypocholesterolemic
Tocopherols	All vegetable oils	Vitamin, Antioxidant
Tocotrienols	Palm , Rice bran	Vitamin, Antioxidant
Carotenes	Red Palm	Provitamin, Antioxidant
Oryzanols	Rice bran	Hypocholesterolemic Antioxidant
Sesamin	Sesame	Hypocholesterolemic Anti-inflammatory
Sesamolin, Sesamol	Sesame	Antioxidant
Hydroxytyrosol, Phenolic acids	Olive	Antioxidant

Reference: 19

In the urban middle and upper income groups, the daily intake of visible fat ranges between 22-45g/p/d³⁵⁻³⁶ and total fat in their diets furnish 20-33% E³⁶⁻³⁷. Studies on plasma lipid fatty acid compositions in urban upper middle income groups have shown that a large proportion of Indian subjects have inadequate n-3 PUFA nutritional status^{25, 17,37}. It is necessary to increase n-3 PUFAs in the diets of the urban segments for providing fat quality consistent with good health^{25,17,29}.

6.6 Recommended intake of dietary fats for Indians

Recommendations for dietary fats in Indians have been revised taking into account recent FAO and WHO recommendations^{4,9,16,20,22,23} for: i) total fat, individual fatty acids and health promoting non-glyceride components ii) sources of dietary fats in Indians and iii) availability of fat. The recommendations are directed towards meeting the requirements for optimal foetal and infant growth and development, maternal health and for combating chronic energy deficiency (children and adults) and DR-NCD in adults.

Quantity of visible fat

a) Minimum levels

Adults: Taking into account: i) ~10 % E fat from all foods except visible fats; average of ~7% E in rural India and 12 -14% E in urban segments^{25,17,29}, ii) unfavourable effects of low fat-high carbohydrate diets⁵⁻⁸ and iii) depending on energy requirements set on the basis of age, physiological status and physical activity (Refer Chapter on Energy), the minimal intakes of visible fat in Indian adults should range between 20-40 g/person/day (Table 6.4).

Pregnant and lactating women: The minimum level of total fat should be 20% E and AMDR is the same as for general population. Pregnant and lactating females, should consume at least 200 mg/d DHA for optimal adult health and foetal and infant development^{12,13,22}. To furnish 20% E total fat, diets of pregnant and lactating women should contain at least 30 g visible fat (Table 6.4).

Infants (0- 6 m): The recommended method of feeding healthy infants is breast milk. Fat content of human milk is relatively constant at 3-4% by weight and delivers 50-60% E. Human milk substitutes / infant formulas should have fat and individual fatty acid contents (including AA and DHA) similar to the levels in human milk. Addition of AA and DHA will enable the formula-fed infant to achieve the same blood LC PUFA nutritional status as that of the breast-fed infants^{12,13,21}. Pre-term infants have a higher requirement for AA and DHA to allow rapid brain and body growth.

Infants (6-24 m): Fat intake should be reduced to 35% E gradually, depending on the physical activity of the child from age 6 months to 2 years^{21,22}. The mix of fat from breast milk and complementary foods should provide infants with at least 3–4.5% E from LA (U-AMDR 10 %E) and 0.4-0.6 % E from ALA (U-AMDR 3%E)²¹. In situations where breast milk intake is low, the level of fat, LA, ALA, AA and DHA in complementary foods should be increased so as to achieve the recommended intakes. Young preschool children's diet should carry enough fat to provide optimal energy density and adequate calories⁴¹. Twenty five gram visible fat is recommended in diets of young children assuming 10% E fat from breast milk and infant formula plus fat from complementary foods (except visible fat). This level of visible fat would provide adequate energy density (reduce volume / bulkiness) in the child's diet and contribute to prevention of chronic energy deficiency (Table 6.4). It would also ensure adequate supply and absorption of fat soluble vitamins⁴⁰. The provision of dietary sources of DHA to infants should come from human milk / infant formula and complementary foods (Table 6.1).

Table 6.4: Recommendations for dietary fat intake in Indians

Age/Gender/ physiological groups	Physical activity	Minimum level of Total fat (% E) ^a	Fat from foods other than visible fats ^d % E	Visible fat ^g	
				%E	g/p/d
Adult Men	Sedentary	20	10	10	25
	Moderate				30
	Heavy				40
Adult Women	Sedentary	20	10	10	20
	Moderate				25
	Heavy				30
	Pregnant women	20	10	10	30
	Lactating women				30
Infants	0 – 6 m	40-60	Human milk ⁱ		
	6 - 24 m	35 ^b	10 ^c	25	25
Children	3-6 y	25	10	15	25
	7-9 y				30
Boys	10 – 12 y				35
	13 – 15 y				45
	16 – 18 y				50
Girls	10 – 12 y				35
	13 – 15 y				40
	16 – 18 y				35

^aReference^{22,23}

^bgradually reduce depending on physical activity

^c Human milk /infant formula+ complementary foods

^d if higher than 10% E, visible fat requirement proportionately reduces

^g cooking oils, butter, ghee and margarine

ⁱ infant formulae/ milk substitutes should mimic contents of fat and fatty acids in human milk including arachidonic and docosahexaenoic acid.

Children and adolescents (2-17y): Total fat intake below 25% E is considered to affect growth in children and adolescents²¹. To provide 25% total fat calories, the minimum level of visible fat in diets of children and adolescents should range between 25-30 and 35-50 g/day respectively (Table 6.4). Since the requirement of fatty acids for adolescents has not been adequately established, the recommendations for this age group are set to be the same as in adults^{21,22}.

b) Maximum levels

In Table 6.4, minimum level of total fat (and visible fat) in the diet is suggested. However, the level of total fat (U-AMDR) that can be included in the diet should not exceed 30% E (about 60g visible fat/day). Fat intake exceeding 35% E may increase the risk of DR-NCD and should be avoided. However, fat intake in the daily diet can be between 20-30% E.

Quality of fat

a) Type of visible fat

The quantity and fatty acid composition of both visible fat and fat from all other foods (invisible fats) contribute to the intake of various fatty acids in the total diet. The data on fatty acid intake in Indian adults determined by taking into account the contribution of various fatty acids from all foods (invisible fat) and either 20g or 50g visible fats (in diets of either rural or urban

population respectively) shows that a complete dependence on just one vegetable oil does not ensure the recommended intake of fatty acids for optimal health and prevention of DR-NCD^{25,29}. To achieve intakes of individual fatty acids in Indians that are consistent with FAO/WHO 2008 recommendations (Table 6.1); the types of visible fats and correct combination of vegetable oils to be used for different food applications are summarized in Table 6.5^{25,29}. A long term ‘in home’ study with oil combinations (which increase ALA) showed improvement of LC n-3 PUFA nutritional status in adults³⁷.

Table 6.5: Recommendations on type of visible fat[#]

<p>1. Use correct combination / blend of 2 or more vegetable oils (1:1)##</p> <p><i>Oil containing LA + oil containing both LA and ALA (Table 6.1, Columns 8 & 9)*</i></p> <p>Groundnut / Sesame^a/ Rice bran^b/ Cottonseed + Mustard/ Rapeseed** Groundnut /Sesame^a/ Ricebran^b/ Cottonseed + Canola Groundnut / Sesame^a/ Rice bran^b / Cottonseed + Soyabean Palmolein^c+ Soyabean Safflower / Sunflower + Palm oil/Palmolein^c + Mustard/ Rapeseed**</p> <p><i>Oil containing high LA + oil containing moderate or low LA*** (Table 6.1 Column 8)</i></p> <p>Sunflower / Safflower + Palmolein^c / Palm oil^c / Olive Safflower / Sunflower + Groundnut / Sesame^a/ Ricebran^b/ cottonseed</p>
2. Limit use of butter/ghee ^d (Table 6.1, Column 2)
3. Avoid use of PHVO as medium for cooking / frying (Table 6.1 columns 2 & 4)
4. Replacements for PHVO (Table 6.1, column 4)

Frying : oils which have higher thermal stability -- palm oil^c/ palmolein^c, sesame^a, ricebran^b, cottonseed -- single / blends (home /commercial)

Bakery fat, shortening, Mithai / Indian sweets etc -- Food applications which require solid fats : coconut oil/ palm kernel oil/ palm oil / palmolein/ palm stearin and / their solid fractions and / their blends

Reference: 17, 19, 23, 29, 42, 43.

#Wide sources as part of a well balanced diet

All vegetable oils contain tocopherols and plant sterols

^aSesame lignans, ^boryzanol + tocotrienols, ^ctocotrienols, ^dvitamin A & D

##Furnish greater variety of nonglyceride components

* Approximately 30-40% PUFAs with >3 %ALA

**Combinations with rapeseed/ mustard reduce erucic acid levels.

***Approximately 40-50 % LA and <0 .5 % ALA, recommended only when intake of ALA from other foods / unconventional foods is increased and or adequate amount of fish is consumed (Table 6.7)

b) Quality of total fat from dietary components other than visible fats

The recommendations to ensure that the individual fatty acids from fats present as integral component of foods and ‘hidden’ fats from processed foods (foods other than visible fat) contribute towards (along with visible fats consumed) ensuring optimal intakes of various fatty acids (Table 6.1) are summarized in table 6.6. Inclusion of foods which provide LCn-3 PUFAs is recommended for the prevention of DR-NCD and life-long health and well being (Table 6.7). Individuals / populations who do not consume fish should achieve higher intake of ALA (Table 6.7). Foods enriched with DHA and/or EPA from marine microalgae is a vegetarian source for LCn-3 PUFA.

In brief, fat *per se* does not affect body weight but because it increases the palatability of the diet, it may increase energy intake and risk of DR-NCD. Instead of recommending “prefer ‘low fat’ diets and avoid ‘fatty foods’ as a way to lose body weight and prevent DR-NCD”, a better advise would be on type of fat, namely, “consume / increase proportion of ‘good fats’ (polyunsaturated including n-3 and monounsaturated)”; limit /decrease proportion of ‘bad fats’ (saturated) and avoid/eliminate industrially produced trans fats (food additives) with focus on energy balance and physical activity.

Table 6.6: Recommendations for optimizing quality of fat from foods other than visible fats*

To increase n-3 PUFAs : (Table 6.1 Columns 7,9,10)

Consume foods which have high contents of ALA and / LCn-3 PUFAs (Table 6.7). Individuals / populations who do not consume fish should achieve higher intake of ALA. Oils and foods enriched with DHA and or EPA from marine microalgae are vegetarian source for LCn-3 PUFA

To minimize TFAs : (Table 6.1 Column 4)

Avoid foods prepared in PHVO (processed, premix, ready-to- eat and fast foods. Consume low fat milk and dairy foods.

To limit SFAs (Table 6.1, Column 2)

Consume low fat milk and dairy foods
Moderate consumption of beef, mutton

To increase MUFAs & PUFAs, antioxidant vitamins and minerals

Consume whole nuts but total energy and fat calories should be within the recommended limits.

References: 17, 23, 25, 29, 42, 43.

Table 6.7: Approximate quantity of foods required to furnish 0.1 g n-3 PUFAs

Plant Foods (ALA)	g	Vegetable oils (ALA)	G
<i>Cereal /Millet</i>		Mustard / Rapeseed	0.7
Wheat, Bajra	70	Soybean	1.5
Oats (germ)	70	Canola	0.5
Wheat (germ)	1.4	Flaxseed	0.2
<i>Pulses</i>		LC n-3PUFAs from animal foods	
Black gram, Rajmah & Cow pea	20	<i>Fish^b</i>	
Soybean	7	Low / medium fat fish ^c	20-50
Other pulses	60	Oily fish ^d (>5 % fat)	10
<i>Vegetables</i>			
Green leafy	60		
Purslane ^a	25		
Radish seed (sprouted)	14		
Spirulina (dried)	12		
<i>Spices</i>		<i>Poultry</i>	
Fenugreek Seed	5	Egg	
Mustard Seed	2	Standard ^e	
		DHA enriched (flaxseed) ^{f,g,h}	2-3 eggs
		DHAenriched (meal from marine sources) ^{f,g,h}	1 egg
		Chicken ^h	1/3 rd egg
			100
<i>Nuts</i>		<i>Lean meats</i>	
Walnuts	2	Lamb, sheep, goat, beef, pork ⁱ	150
Almonds	25		
		<i>Fish oils</i>	
		Cod liver ^j	0.5
		Muscle oil	0.3
<i>Unconventional oilseeds</i>			
Flaxseed (linseed)	0.5	Vegetarian source of LC n-3 PUFAs	To see contents on label
Perilla seed	0.5	Algal oil-based DHA products	

References: 17, 22-28.

^a Richest source of ALA of any green leafy vegetable examined, source of EPA.

^b Good source of LCn-3 PUFAs, oil and LCn-3 PUFAs. Contents vary markedly with species, season, diet, packaging and cooking methods.

^c Bam, beley, bhekhti, jew fish, lobster, pomphret, prawn, rohu, surmai, bombay duck, shark, thread fin, shrimp, cod, haddock, tuna, katla, mullet, sardines, halibut, albacore, mullet, mussels, crab, red tilapia, tilapia, cat fish, haddock.

^d Seer (white and black), mackerel, sardines, salmon, eel, cat fish (*Mystusnemurus*), red pomphrethilsa, purava.

^e Poultry feeds not including flaxseeds or fish meal, eggs also contain ~0.03g ALA.

^f Contain ~0.3g ALA.

^g Poultry feeds containing either flaxseed or fish meal.

^h Varies depending on ALA / fish meal in poultry feed.

ⁱ Varies depending on nutrient composition of the diet, animals grazed on pastures have higher n-3 content than grain fed.

^j 600, 5 and 1 µg /g oil respectively for vitamins A, D and E.

References

1. Ratnayake WN, Galli C. Fat and fatty acid terminology, methods of analysis and fat digestion and metabolism. *Annals of nutrition and metabolism.* 2009 Jan 1; 55(1/3):8-43.
2. Galli C, Calder PC. Effects of fat and fatty acid intake on inflammatory and immune responses. *Annals of nutrition & metabolism.* 2009 Jan 1; 55(1/3):123-39.
3. Crawford MA, Bazinet RP, Sinclair AJ. Fat Intake and CNS Functioning. *Annals of nutrition & metabolism.* 2009 Jan 1; 55(1/3):202-28.
4. WHO/FAO. Diet, nutrition and the prevention of chronic diseases: Report of a Joint WHO/FAO Expert Consultation. TRS 916, Geneva, 2003.
5. Elmadafa I, Kornsteiner M. Fats and fatty acid requirements for adults. *Annals of nutrition & metabolism.* 2009 Jan 1; 55(1/3):56-75.
6. Melanson EL, Astrup A, Donahoo WT. The relationship between dietary fat and fatty acid intake and body weight, diabetes, and the metabolic syndrome. *Annals of nutrition & metabolism.* 2009 Jan 1; 55(1/3):229-43.
7. Sanders TA. Fat and fatty acid intake and metabolic effects in the human body. *Annals of nutrition & metabolism.* 2009 Jan 1; 55(1/3):162-72.
8. Skeaff CM, Miller J. Dietary Fat and Coronary Heart Disease. *Annals of nutrition & metabolism.* 2009 Jan 1; 55(1/3):173-201.
9. Levy BD. Resolvins and protectins: natural pharmacophores for resolution biology. *Prostaglandins, Leukotrienes and Essential Fatty Acids (PLEFA).* 2010 Apr 1; 82(4-6):327-32.
10. FAO. Fats and oils in human nutrition: Report of a Joint FAO/WHO Expert Consultation FAO Technical Papers 57. Rome, FAO, 1994.
11. Harris WS. The omega-6/omega-3 ratio and cardiovascular disease risk: uses and abuses. *Current atherosclerosis reports.* 2006 Nov 1; 8(6):453-9.
12. Brenna JT, Lapillonne A. Background paper on fat and fatty acid requirements during pregnancy and lactation. *Annals of nutrition & metabolism.* 2009 Jan 1; 55(1/3):97-122.
13. Koletzko B, Cetin I, Brenna JT, Perinatal Lipid Intake Working Group. Dietary fat intakes for pregnant and lactating women. *British Journal of Nutrition.* 2007 Nov; 98(5):873-7.
14. Willett WC. The role of dietary n-6 fatty acids in the prevention of cardiovascular disease. *Journal of Cardiovascular Medicine.* 2007 Sep 1; 8:S42-5.
15. Rastogi T, Reddy KS, Vaz M, Spiegelman D, Prabhakaran D, Willett WC, Stampfer MJ, Ascherio A. Diet and risk of ischemic heart disease in India. *The American journal of clinical nutrition.* 2004 Apr 1; 79(4):582-92.
16. Uauy R, Aro A, Clarke R, L'abbé MR, Mozaffarian D, Skeaff CM, Stender S, Tavella M. WHO Scientific Update on trans fatty acids: summary and conclusions. *European Journal of Clinical Nutrition.* 2009 May; 63(2):S68-75.
17. Ghafoorunissa SA. Dietary Fat and Diet-Related Chronic Diseases: Indian Perspective. Touch. (Heinz Nutrition Foundation India). 2005; 7:2-6.
18. Ellegård LH, Andersson SW, Normén AL, Andersson HA. Dietary plant sterols and cholesterol metabolism. *Nutrition reviews.* 2007 Jan 1; 65(1):39-45.
19. Ghafoorunissa. Impact of quality of dietary fat on serum cholesterol and coronary heart disease: focus on plant sterols and other non-glyceride components. *National Medical Journal of India.* 2009 May 1; 22(3):126-32.
20. FAO report of a Joint FAO/WHO Expert Consultation on Dietary Fats and Oils in Human Nutrition FAO Technical Papers 3. Rome, FAO, 1977.

21. Uauy R, Dangour AD. Fat and fatty acid requirements and recommendations for infants of 0–2 years and children of 2–18 years. *Annals of nutrition & metabolism*. 2009 Jan 1;55(1/3):76-96.
22. World Health Organization. Interim summary of conclusions and dietary recommendations on total fat & fatty acids. From the joint FAO/WHO expert consultation on fats and fatty acids in human nutrition. 2008 Nov 10:10-4.
23. Misra A, Sharma R, Gulati S, Joshi SR, Sharma V, Ghafoorunissa IA, Joshi S, Laxmaiah A, Kurpad A, Raj RK, Mohan V. National Dietary Guidelines Consensus Group. Consensus dietary guidelines for healthy living and prevention of obesity, the metabolic syndrome, diabetes, and related disorders in Asian Indians. *Diabetes Technol Ther*. 2011; 13(6):683-94.
24. ICMR, Nutrient requirements and recommended dietary allowances for Indians. Report of the Expert Group of the Indian Council of Medical Research, National Institute of Nutrition, Hyderabad, 1990.
25. Ghafoorunissa. Fats in Indian diets and their nutritional and health implications. *Lipids*. 1996 Mar 1; 31:S287-91.
26. McCance and Widdowson's The composition of foods : Amino acids mg/100g food, Fatty acids g/100g food By A.A.Paul, D.A.T Southgate and J.Russell Seventh edit Ministry of agriculture, Fisheries and food (MAFF), 1998 Elsevier/North -Holland Biomedical press.
27. Whelan J, Rust C. Innovative dietary sources of n-3 fatty acids. *Annual Reviews of Nutrition*. 2006 Aug 21; 26:75-103.
28. Ghafoorunissa. Fish and fish oil for nutritional security sustainable Indian fisheries, Pandian TJ (ed) 272-288 National Academy of Agricultural Sciences, New Delhi, 2001.
29. Ghafoorunissa. Requirements of dietary fats to meet nutritional needs & prevent the risk of atherosclerosis-An Indian perspective. *Indian Journal of Medical Research*. 1998 Nov 1; 108:191-202.
30. Codex alimentarius commission, Report Of The Fifteenth session of The Codex Committee on fats and oils, London, United Kingdom, 4–8 November 1996.
31. NNMB. Diet and nutritional status of population and prevalence of hypertension among adults in rural areas. Technical Report Series No 24, National Institute of Nutrition (ICMR), Hyderabad, 2008.
32. NNMB. Report for the year 1979. Technical Report Series 6, National Nutrition Monitoring Bureau, National Institute of Nutrition (ICMR), Hyderabad, 1980.
33. NNMB. Report for the year 2012. Technical Report Series, National Nutrition Monitoring Bureau, National Institute of Nutrition (ICMR), Hyderabad, 2012.
34. NNMB. Report for the year 2016. Technical Report Series, National Nutrition Monitoring Bureau, National Institute of Nutrition (ICMR), Hyderabad, 2016.
35. NNMB. Report on Urban Population, National Nutrition Monitoring Bureau, National Institute of Nutrition (ICMR), Hyderabad, 1982.
36. Goyal U, Sadana B, Verma S. Contribution of various foods to fat and fatty acids intake among urban and semi-urban women of Punjab. *Journal of Human Ecology*. 2005 Nov 1; 18(3):217-20.
37. Ghafoorunissa, Vani A, Laxmi, R, and Sesikaran B. Effects of dietary alpha-linolenic acid from blended oils on PUFA nutritional status and biochemical indices of coronary heart disease in Indians. *Lipids*. 2002; 37(11): 1077-86.
38. Radhika G, Van Dam RM, Sudha V, Ganesan A, Mohan V. Refined grain consumption and the metabolic syndrome in urban Asian Indians (Chennai Urban Rural Epidemiology Study 57). *Metabolism*. 2009 May 1; 58(5):675-81.
39. Misra A, Khurana L, Isharwal S, Bhardwaj S. South Asian diets and insulin resistance. *British Journal of Nutrition*. 2008 Oct; 101(4):465-73.
40. Jayarajan P, Reddy V, Mohanram M. Effect of dietary fat on absorption of β carotene from green leafy vegetables in children. *Indian Journal of Medical Research*. 2013 May 1; 137(5).

41. Narasinga Rao, B.S. Low dietary fat intake as the main cause of widespread energy deficiency especially among preschool children in the developing countries. *Malaysian Oil Science and Technology* 1995; 4: 139–46.
42. Skeaff CM. Feasibility of recommending certain replacement or alternative fats. *European journal of clinical nutrition*. 2009 May; 63(2):S34-49.
43. L'Abbé MR, Stender S, Skeaff CM, Tavella M. Approaches to removing trans fats from the food supply in industrialized and developing countries. *European Journal of Clinical Nutrition*. 2009 May; 63(2):S50-67.

7.1. DIETARY FIBER

INTRODUCTION

Dietary fiber (DF) is broadly defined as the endogenous edible components present in plant materials or analogous carbohydrates which are resistant to digestion by the enzymes secreted in small intestine of mammalian digestive system. They are digested and absorbed in the mammalian with complete or partial fermentation in the large intestine. Dietary fiber includes polysaccharides, oligosaccharides, lignin and associated plant substances. Countries like Denmark, Finland, Norway, Sweden and Japan have defined the Dietary fiber as edible material that cannot be degraded by human endogenous enzymes, as measured by AOAC method 985.29. However, these countries had different approaches for the inclusion or exclusion of inulin and fructo-oligosaccharides in the dietary fiber. Dietary fiber promotes one or more of the beneficial physiological effects such as laxation, reduction in blood cholesterol, and/or blood glucose attenuation. In the past three decades, dietary fiber has received large attention as one of the important macro nutrients.

Structure

Dietary fiber structure plays significant role in their physiological action with in human body. The dietary fiber structure provides the information about the solubility of dietary fiber in water and consequently their classification as soluble or insoluble dietary fiber. Plant food contains various types of dietary fiber with different name and structure. Cellulose and hemicelluloses are the predominant dietary fiber which contain β -(1 \rightarrow 4) linked glucose units and arabinoxylan (AX) (1 \rightarrow 4)- β -D-xylan backbone with single α -L-arabinofuranose units as side branches, respectively. On the other hand, β -glucan is linear polysaccharide and joined by β -(1 \rightarrow 3) and β -(1 \rightarrow 4)-D-glucopyranose units, therefore classified as soluble dietary fiber¹. Heteroxylans consists (1 \rightarrow 4)- β -D-xylan backbone highly substituted by single arabinose units, single glucuronic acid unit, and more complex short side chain containing arabinose, xylose and galactose while some heteroxylans of fruits and vegetable cell walls contain glucuronoxylans and glucurono-arabinoxylans (1 \rightarrow 4)- β -D-xylan backbone with glucuronic acid units and/or α -L-arabinofuranose units as substituents. Structurally the mannans (linear) abundantly found in date, green coffee bean and *Aloe vera* consists of β -(1 \rightarrow 4)-linked β -D-mannopyranosyl residues whereas galactomannans prominently found in grain legumes which contains, mannans substituted with side chains of α -1, 6-linked galactose residues. However, glucomannans which is dominantly found in fungal cell wall consists β -(1 \rightarrow 4)-linked linear mannan chain with interspersed glucose residues in the main chain and are often acetylated².

Type and sources

Dietary fiber is classified according to their chemical structure, physico-chemical properties including water solubility, viscosity and fermentability. Dietary fiber is further subdivided into polysaccharides including non-starch polysaccharides (NSP) and resistant starch (RS) with the monomeric unit (MU) number of 10, and resistant oligosaccharides (RO) with MU number between 3 to 9³. Depending upon solubility in a buffer at specific pH and their *in vitro* fermentability using an aqueous enzyme solution representative of human alimentary enzymes, dietary fiber is classified as water soluble (well fermented fiber including gums, pectin and mucilage) and water insoluble dietary fiber (less fermented which includes hemicelluloses, cellulose, lignin). Most of the RS are insoluble in water while solubility of NSP is related to the monomeric unit number, composition and the linkage associated with the polymer. However, most of the resistant oligosaccharides are water soluble. Interestingly, certain non-starchy polysaccharides (soluble fibers) including β -glucan,

high molecular weight guar gum, psyllium and pectin have gel forming nature in intestinal tract thus may influence the glucose and lipid metabolism⁴. Fiber consumption through food significantly depends on the fiber content and portion of edible food since the outer layers of a food material has high concentration of dietary fiber. Some type of dietary fibers including RS, β -glucan, mucilage, gums, are found only in particular food such as β -glucan specific to barley, oat, mushrooms etc.; RS is predominantly found in green banana, pectin in fruits and vegetables⁵.

Nutritional and health significance

Dietary fiber is considered to be a key component in healthy diet and known to provide many health benefits. Both insoluble and soluble dietary fibers play important physiological role in human health⁶. Insoluble fiber includes lignins, cellulose and some hemicelluloses which are the components of cell walls in grains and vegetables. Insoluble fiber works to promote laxation, speeding up intestinal transit time and adding bulk to the stool and therefore can prevent certain conditions such as constipation, hemorrhoids and diverticulitis. Soluble fiber includes pectins, gums, mucilages and some hemicelluloses. This type of fiber takes up fluid in the gastrointestinal tract, forming a thick, gummy substance. Soluble fiber delays gastric emptying and intestinal transit time. Because of the large water-holding capacity of soluble dietary fiber, it binds to bile acids and help to reduce blood cholesterol levels. Soluble fiber gets fermented by bacteria in the large intestine, and the gastrointestinal tract catabolism leads to the generation of various bioactive materials, such as short-chain fatty acids (SCFAs), that can markedly enhance the gastrointestinal tract biomass and change the composition of the gastrointestinal (GI) tract flora⁷.

It has been demonstrated by extensive research in the past three decades that sufficient dietary fiber intake has benefits for health maintenance and disease prevention including cardiovascular disease, diabetes, colon cancer and weight regulation through reducing the digestion and absorption of macronutrients and also decrease the contact time of carcinogens within the intestinal lumen⁸. By modulating food ingestion, digestion, absorption and metabolism, dietary fiber reduces the risk of hyperlipidemia, hypercholesterolemia and hyperglycemia. Emerging research has also begun to investigate the role of dietary fiber in immunomodulation⁷. Therefore, research on dietary fiber has drawn much attention recently, particularly in the growing nutraceutical industry⁹.

Dietary fiber has been shown to inhibit absorption of micronutrients like minerals and some vitamins. However, the inhibitory effect was found to be confined to insoluble fiber component¹⁰⁻¹². In fact, soluble fiber such as inulin and their oligosaccharides were shown to promote absorption of Mg, Ca, Fe, and Zn by increased permeability. Insoluble fiber binds the divalent elements minerals non-specifically and reduces their absorption. More recently, β -glucan, a soluble fiber component from bran of grains like barley, oats, and wheat, has gained utmost nutraceutical significance^{10, 13}.

Requirements

Traditionally, fiber was considered as a non-nutrient component of food in the Indian context. The Indian Food Composition database has been updated methodologically to include the total, insoluble and soluble fractions of dietary fiber and is reported in IFCT 2017 (www.ifct2017.com)¹⁴. The amount of total dietary fiber and its components in commonly consumed cereals, pulses, vegetables and fruits is presented in Table 7.1. While computing total carbohydrates value, the total dietary fiber content should be deducted. The soluble fiber undergoing fermentation in the colon would provide 2 Kcal per g of fiber. Hence, the kcal of soluble dietary fiber needs to be considered while computing the total energy of the food.

With economic transition, health transition too has taken place in India, resulting in abdominal obesity, insulin resistance, hypertension and cardiovascular risk aggravating the burden of diseases among its people. With more and more proportion of population shifting to processed, refined and

convenient foods, the importance of dietary fiber and its digestibility is acquiring greater significance particularly in the context of health transition. Very few studies have dealt with this aspect. Joshi and Agte¹⁵ estimated the digestibility of neutral detergent fiber (NDF) of diets with wheat and rice as staple foods and found NDF intake of 37.7 g with the digestibility of 34.1%. The overall fiber (intake 38g /d) digestibility was about 35%, in which lignin digestion was only 8%, hemicellulose was 53% and cellulose 30%. This 35% digestibility of fiber adds to the calorie content (about 13 g of digested fiber adds approximately 50 Kcal/d to the daily calorie intake). Similar database on fiber digestibility needs to be generated for all the major Indian foods to correctly assess the energific value of the diets and true energy intake. It is clear that this digestible proportion of fiber will depend on the nature of the food, its proportion in the diet and the eating habits and lifestyle of the persons.

Data from diets of the Western part of the country show that the dietary fiber content is about 30-40 g/d¹⁶. Intake was increasing with increasing level of energy intake, 39-47 g/d in young men¹⁷. The fiber intake is lower in women (15-30 g/d) and is much less in tribal population (15-19 g/d)¹⁷ (Table 7.2). Another report from North India shows that the average total fiber intake per day is about 52 g¹⁸. More data need to be generated in the Indian context to understand the phenomenon of health transition.

Assessment of requirements of fiber is based mainly on normal large bowel function reflected to provide enough bulk and substrates for fermentation. Thus, stool weight and gut transit time were taken as indicators of large bowel function. Further, higher risks of chronic disease associated with lower dietary fiber intake, has helped to identify the requirements from the general public health point of view. There have been no studies on evaluating the dietary fiber requirements in Indians. However, the recommendations of US, SACN and other agencies that a minimum intake of 25-30 g of fiber is conducive for long-term good health^{10,12} can be considered as a positive guideline. WHO has recommended that the consumption of >25 g of total dietary fiber through the wholegrain cereals, fruits and vegetable for the prevention of chronic diseases¹⁹. Intakes in excess of 60 g of fiber over a day can reduce the absorption of nutrients and may cause irritation in the bowel apart from leading to diarrhea. Based on energy intake, a level of about 40 g/2000 kcal in a diet is considered reasonably safe. However, with a steep increase in the consumption of processed and refined foods, consumption of fiber, at least in the urban high-income groups, may become critical. While there is a need to generate a strong evidence base systematically, it may be suggested to gradually diversify the diets to include increasing amounts of whole grains, pulses, legumes, vegetable and fruits to achieve the recommended fiber intake.

Fiber is absent in the circulatory system as it is not absorbed. Therefore, measuring the nutritional status of DF may not be possible. Consumption of DF for various potential health benefits could be taken into consideration to calculate the DRI. However, due to insufficient information to calculate EAR followed by RDA, the dietary intake has been calculated based on the fiber intake of urban Indian population and given in Table 7.3. Fiber intake was not recorded for the healthy infants up to the age of 12 months who are fed human milk where, the DF is absent. Though there is no documentation on the consumption of DF as energy intake, the recommended intakes of DF will be added advantage on various other health benefits including constipation, reducing blood glucose and lipid levels and contribute for low energy- nutrient-rich food.

Table 7.1: Different dietary fiber fractions in selected Indian foods

Food group	Food Name	Dietary Fiber (g/100g)		
		Total	Insoluble	Soluble
Cereals and millets	Bajra (<i>Pennisetum typhoideum</i>)	11.49	9.14	2.34
	Jowar (<i>Sorghum vulgare</i>)	10.22	8.49	1.73
	Ragi (<i>Eleusine coracana</i>)	11.18	9.51	1.67
	Rice, raw, milled (<i>Oryza sativa</i>)	2.81	1.99	0.82
	Wheat, whole (<i>Triticum aestivum</i>)	11.23	9.63	1.60
Grain legumes	Bengal gram, whole (<i>Cicer arietinum</i>)	25.22	22.70	2.52
	Green gram, whole (<i>Vigna radiata</i>)	17.04	14.59	2.44
	Rajmah, red (<i>Phaseolus vulgaris</i>)	16.57	13.86	2.70
	Soya bean, brown (<i>Glycine max</i>)	21.55	16.56	5.00
Green leafy vegetables	Amaranth leaves, green (<i>Amaranthus gangeticus</i>)	4.41	3.21	1.20
	Colocasia leaves, green (<i>Colocasia anti-quorum</i>)	5.60	4.32	1.29
	Drumstick leaves (<i>Moringa oleifera</i>)	8.21	6.12	2.10
	Tamarind leaves, tender (<i>Tamarindus indica</i>)	10.70	9.34	1.36
Other vegetables	Brinjal (<i>Solanum melongena</i>)	3.98	2.84	1.14
	Drumstick (<i>Moringa oleifera</i>)	6.83	5.60	1.23
	Ladies finger (<i>Abelmoschus esculentus</i>)	4.08	2.80	1.28
Fruits	Apple, big (<i>Malus sylvestris</i>)	2.59	1.43	1.16
	Pine apple (<i>Ananas comosus</i>)	3.46	2.88	0.59
	Sapota (<i>Achras sapota</i>)	9.60	8.46	1.14
Roots and tubers	Carrot, orange (<i>Ducus carota</i>)	4.18	2.81	1.37
	Potato, brown skin (<i>Solanum tuberosum</i>)	1.71	1.13	0.58
	Tapioca (<i>Manihot esculenta</i>)	4.61	3.85	0.76
Condiments and spices	Chillies, red (<i>Capsicum annuum</i>)	31.15	26.55	4.60
	Fenugreek seeds (<i>Trigonella foenumgraecum</i>)	47.55	27.63	19.92
	Pepper, black (<i>Piper nigrum</i>)	33.16	30.61	2.54
Nuts and oil seeds	Almond (<i>Prunus amygdalus</i>)	13.06	10.55	2.52
	Gingelly seeds, black (<i>Sesamum indicum</i>)	17.16	13.57	3.59
	Mustard seeds (<i>Brassica nigra</i>)	14.10	10.63	3.47
Mushrooms	Button mushroom, fresh (<i>Agaricus</i> sp.)	3.11	2.76	0.35
	Shiitake mushroom, fresh (<i>Lentinula</i> sp.)	3.02	2.03	0.99
	Oyster mushroom, dried (<i>Pleurotus</i> sp.)	39.12	35.64	3.48

Source: Indian Food Composition Tables (www.ifct2017.com)

Table 7.2: Estimates of dietary fiber intakes in different income segments* reported from Western India

Different segments	Intake of fiber (g/d)	
	Male	Female
Rural	39	30
Tribal	19	15
Industrial	41	31
HIG	31	21
MIG	43	22
LIG	24	20

Source: Reference (Agte *et al.*, 1994)²⁰

LIG, MIG, HIG: Low, middle and high socioeconomic groups

Table 7.3: Average intake of dietary fiber with reference to intakes of Urban India

Age (Yrs) / status	Males		Females		Total	
	(g/day)	(g/1000kcal)	(g/day)	(g/1000kcal)	(g/day)	(g/1000kcal)
1-3	10.8 (6.8-15.4)	12.50 (8.92-16.50)	10.3 (6.2-14.9)	11.94 (8.70-15.90)	10.6 (6.5-15.2)	12.28 (8.79-16.15)
4-6	17.0 (12.2-23.0)	14.80 (11.14-18.66)	16.3 (12.0-21.7)	14.89 (11.39-18.81)	16.6 (12.1-22.5)	14.87 (11.28-18.71)
7-9	20.3 (15.0-27.5)	15.64 (12.01-19.89)	19.5 (14.4-25.9)	15.74 (12.47-19.91)	19.9 (14.7-26.9)	15.68 (12.27-19.89)
10-12	25.1 (18.6-32.4)	16.78 (13.08-21.14)	22.5 (16.5-30.0)	16.40 (12.87-20.73)	23.9 (17.6-31.4)	16.56 (12.99-20.90)
13-15	28.0 (20.7-38.0)	17.41 (13.26-21.88)	25.3 (18.9-32.8)	16.99 (13.24-21.68)	26.6 (19.8-35.6)	17.20 (13.25-21.82)
16-17	31.9 (23.2-41.6)	18.23 (13.92-22.85)	25.6 (19.3-33.7)	17.12 (13.34-21.47)	28.6 (20.8-38.0)	17.67 (13.69-22.13)
18-60	35.6 (26.0-46.6)	18.32 (14.02-22.55)	30.0 (22.4-38.9)	17.41 (13.60-21.45)	32.4 (23.9-42.8)	17.84 (13.79-22.01)
60 & Above	34.5 (25.3-44.8)	18.76 (14.76-22.69)	26.8 (19.9-35.9)	17.56 (13.80-21.81)	30.2 (21.9-40.4)	18.08 (14.25-22.25)
Activity Status						
Sedentary	35.7 (26.2-46.6)	18.65 (14.38-22.70)	29.7 (22.1-38.6)	17.52 (13.72-21.58)	31.9 (23.5-42.1)	17.97 (13.98-22.07)
Moderate	34.8 (25.2-45.9)	17.64 (13.45-22.22)	28.5 (20.9-37.7)	15.90 (12.49-20.20)	33.4 (24.2-44.3)	17.32 (13.24-21.91)
Heavy	32.9 (22.7-45.0)	14.84 (12.61-18.59)	19.0 (19.0-19.0)	20.08 (20.08-20.08)	32.4 (21.0-42.2)	14.96 (12.74-18.66)
Physiological Status						
NPNL			29.8 (22.2-38.7)	17.26 (13.49-21.36)		
Pregnant			30.8 (23.0-40.6)	17.83 (14.07-21.44)		
Lactating			32.5 (23.9-39.7)	17.29 (12.99-21.21)		

Values are Median (P25, P75); Source: NNMB Urban Survey 2016

NPNL-Non Pregnant and Non Lactation

References

1. Stephen AM, Champ MM, Cloran SJ, Fleith M, van Lieshout L, Mejborn H, Burley VJ. Dietary fibre in Europe: current state of knowledge on definitions, sources, recommendations, intakes and relationships to health. *Nutrition research reviews*. 2017 Dec; 30(2):149-90.
2. Kumar V, Sinha AK, Makkar HP, de Boeck G, Becker K. Dietary roles of non-starch polysaccharides in human nutrition: a review. *Critical reviews in food science and nutrition*. 2012 Oct 1; 52(10):899-935.
3. McCleary BV, Prosky L, editors. Advanced dietary fibre technology. Development of dietary fiber methodology pp. 77–86 Oxford: Blackwell Science; 2001.
4. Cummings JH (2001). The effect of dietary fiber on fecal weight and composition. In CRC Handbook of Dietary Fiber in Human Nutrition, pp. 183–252 [GA Spiller, editor]. Boca Raton, FL: CRC Press.
5. Choo CL, Aziz NA. Effects of banana flour and β -glucan on the nutritional and sensory evaluation of noodles. *Food Chemistry*. 2010 Mar 1; 119(1):34-40.
6. EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA). Scientific Opinion on the substantiation of health claims related to xanthan gum and changes in bowel function (ID 837) pursuant to Article 13 (1) of Regulation (EC) No 1924/2006. *EFSA Journal*. 2011 Jun; 9(6):2272.
7. Kaczmarczyk MM, Miller MJ, Freund GG. The health benefits of dietary fiber: beyond the usual suspects of type 2 diabetes mellitus, cardiovascular disease and colon cancer. *Metabolism*. 2012 Aug 1; 61(8):1058-66.
8. Anderson JW, Baird P, Davis RH, Ferreri S, Knudtson M, Koraym A, Waters V, Williams CL. Health benefits of dietary fiber. *Nutrition reviews*. 2009 Apr 1; 67(4):188-205.
9. Cheung PC. Mini-review on edible mushrooms as source of dietary fiber: Preparation and health benefits. *Food Science and Human Wellness*. 2013 Sep 1; 2(3-4):162-6.
10. Lunn J, Buttriss JL. Carbohydrates and dietary fibre. *Nutrition Bulletin*. 2007 Mar; 32(1):21-64.
11. Coudray C, Demigne C, Rayssiguier Y. Effects of dietary fibers on magnesium absorption in animals and humans. *The Journal of nutrition*. 2003 Jan 1; 133(1):1-4.
12. Timm DA, Slavin JL. Dietary fiber and the relationship to chronic diseases. *American Journal of Lifestyle Medicine*. 2008 May; 2(3):233-40.
13. Sheard NF, Clark NG, Brand-Miller JC, Franz MJ, Pi-Sunyer FX, Mayer-Davis E, Kulkarni K, Geil P. Dietary carbohydrate (amount and type) in the prevention and management of diabetes: a statement by the American Diabetes Association. *Diabetes care*. 2004 Sep 1; 27(9):2266-71.
14. Longvah T, Anantan I, Bhaskarachary K, Venkaiah K, Longvah T. Indian food composition tables. Hyderabad: National Institute of Nutrition, Indian Council of Medical Research; 2017.
15. Joshi S, Agte V. Digestibility of dietary fiber components in vegetarian men. *Plant foods for human nutrition*. 1995 Jul 1; 48(1):39-44.
16. Joshi S, Mane S, Agte V. Analysis of insoluble fiber components in common Indian foods and habitual diets. *Indian Journal of Clinical Biochemistry*. 1991 Jul 1; 6(2):97-103.
17. Agte VV, Chiplonkar SA, Tarwadi KV. Factors influencing zinc status of apparently healthy Indians. *Journal of the American College of Nutrition*. 2005 Oct 1; 24(5):334-41.
18. Singh N, Makharia GK, Joshi YK. Dietary survey and total dietary fiber intake in patients with irritable bowel syndrome attending a tertiary referral hospital. *Indian J Gastroenterol*. 2008 Mar; 27(2):66-70.
19. WHO. Report of a Joint WHO/FAO Expert Consultation on Diet, Nutrition and prevention of chronic diseases, Technical Report Series No 916.WHO, Geneva, 2003.
20. Agte V, Chiplonkar S, Joshi N, Paknikar K. Apparent absorption of copper and zinc from composite vegetarian diets in young Indian men. *Annals of nutrition and metabolism*. 1994; 38(1):13-9.

7.2. CARBOHYDRATES

INTRODUCTION

Carbohydrates (CHO) are a major and vital source of energy in the diet. The quantity and quality of CHO are important to maintain appropriate health and have been indicated to substantially impact nutrition related chronic disorders/non-communicable diseases (NCDs). For this purpose, the chapter on requirements of CHO including added sugars based on current evidence is included in this publication, to communicate to consumers to eat right amount and type of CHO.

Classification

The main classification of CHO is based on chemistry while the aspects of physical effects (food matrix), functional/ physiological effects and health consequences are also important. They are chemically classified, based on degree of polymerization (DP) into a) DP 1-2: sugars (monosaccharides and disaccharides) and polyols; b) DP 3-9: oligosaccharides (malto-oligosaccharides and non-digestible oligosaccharides) and c) DP ≥ 10 : polysaccharides (starch and non-starch polysaccharides). However, for ease of translation into nutritional effects, CHO are classified based on their digestion and absorption in the human small intestine into i) available/digestible/glycemic CHO that are absorbed and digested in the small intestine and available for further metabolism ii) unavailable/non-digestible/non-glycemic CHO that are resistant to digestion in the small intestine and reach the large intestine where they are either partially fermented by colonic bacteria to short chain fatty acids that are absorbed and metabolized to provide energy or are excreted¹⁻³.

Total Carbohydrates are calculated ‘by difference’ or by direct measurement of the individual components that are then added to give a total. The first method is used widely by many organizations and researchers around the world since the early 20th century^{4,5}. It is derived by subtracting the moisture, protein, fat, ash and (alcohol) content of a food (that are directly determined), from the total weight of the food. This method has problems as it will also include the non-CHO components (viz., lignin, tannins, polyphenols, organic acids etc.,) including the analytical errors of each of these^{6,7}. It does not allow identification of the types of CHO and thus their health effects. The direct measurement of total CHO and its components should be more preferred as it helps to relate with potential health benefits⁸.

“Free sugars or Added sugars” refers to “all monosaccharides and disaccharides added to foods and beverages by the manufacturer, cook or consumer, as well as to sugars naturally present in honey, syrups, fruit juices and fruit juice concentrates”. However, free/added sugars do not include sugars naturally present in milk (lactose), whole fruit (fructose, glucose) and vegetables (fructose, sucrose)⁹⁻¹¹.

Functions

CHO are the major source of easily available energy in human diets comprising more than 60-78% of total energy intake, particularly in India¹²⁻¹⁵. In addition CHO rich foods also provide important micronutrients and phytochemicals. CHO is important to maintain glycemic homeostasis and gastrointestinal integrity and function⁷. Free sugars consumption may promote a positive energy balance as they contribute to the overall energy density of diets.

Digestion and Absorption

The monosaccharides i.e., glucose and galactose are actively absorbed in the small intestine via sodium glucose co-transporter 1(SGLT1) while fructose is taken up by facilitative transport¹⁶.

The di-, oligo- and poly-saccharides are hydrolyzed to their constituent monosaccharides and absorbed. Starch that is gelatinized by processing or cooking is digested majorly by pancreatic amylase in the small intestine. Resistant starch is the sum of starch and products of starch digestion that are not absorbed in the small bowel¹⁷. The non-digestible CHO are partially fermented by the commensal bacteria which contain enzymes capable of hydrolyzing them¹⁸.

Metabolism

CHO are principally metabolized to provide energy. The brain, central nervous system and red blood cells are almost totally dependent on glucose as a source of energy. After absorption, the monosaccharides are transported to liver which then enters the systemic circulation. Plasma insulin increases in response and the glucose is taken up by cells (via GLUT transporters) and tissues (adipose and skeletal muscle) and is metabolized by glycolysis. Fructose uptake by tissues are non-insulin dependent. The amount of energy yielded per unit weight of glucose, sucrose and starch respectively is 3.74, 3.92 and 4.18 kcal/g. The available energy from short chain fatty acids absorbed after fermentation in the colon is 1.9, 2.2, 1.9-2.2 and 1.4-2.4 kcal/g from non-starch polysaccharides, resistant starch, oligosaccharides and polyols¹⁹.

The glucose breaks down to pyruvate by the glycolysis pathway. Pyruvate may be converted to lactate anaerobically or to acetyl CoA aerobically. Once it is converted to acetyl CoA, glucose is not retrievable. Glucose is metabolized and is stored as glycogen, which is the main source of energy. Making glucose from protein is called gluconeogenesis. When there is inadequate supply of CHO, ketone bodies are formed from fat fragments and in contrary when CHO is abundant, the body uses the glucose to make fat de novo²⁰.

Dietary Sources of CHO

The major dietary sources of CHO are sugars, cereals & millets, roots & tubers, pulses & legumes and to a limited extent from vegetables, fruits and dairy. Indian dietaries are predominantly high in starch from cereals, millets and root vegetables. Examples are rice, wheat, sorghum, maize and potatoes. Sugars from fruits and vegetables are limited as consumption is very low. Sources of free/added sugars in diets are sugar sweetened carbonated beverages, fruit juices and concentrates, sweets & desserts, cakes, biscuits, chocolates & candies and beverages such as tea and coffee.

CHO serve as the main source of energy for majority of the Indian population. The median intake of CHO by the Indian men and women are 320 g/d each providing 72 En% in the rural areas¹² and 300 and 261 g/day respectively, providing about 60 En% in the urban areas¹³. Analysis of trends in intake from across NNMB surveys done from 1975 to most recent will provide important information. There is a need to generate/analyse existing data on the dietary contributors from different food groups to total CHO & added sugar intakes, its relation to energy intakes, prevalence of obesity and other diet related chronic conditions by systematically reviewing the data particularly from India and South East Asian countries and secondary data analysis.

Health implications

The nature and type of dietary CHO is more critical rather than the quantity of CHO for desired health effects. The evidence on the impact of dietary CHO on cardio metabolic and oral health is equivocal. CHO from whole grains, legumes, vegetables and whole fruits are associated with reduced risk of type-2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) and the effects may mainly depends on specific components of CHO^{21,22}. Association of consumption of white rice with increased risk and consumption of brown rice with reduced risk of T2DM in the Asian population, specifically in Japan and China is indicated, but the evidence is limited³. The contraindications of high intakes of unrefined complex CHO is little or is not well studied. There is

no conclusive evidence that diets higher in total CHO causes weight gain, however increased regular consumption of free sugars is associated with a greater risk of weight gain & higher body mass index in children, adolescents and adults^{3,23}, development of T2DM & NCDs^{24,25} and dental caries^{26,27}. The sugar-sweetened beverages do not induce satiety to the same extent as solid forms of CHO and the discretionary calories from free sugars may replace the calories from the healthier nutrient dense foods, leading to an unhealthy diet¹¹.

Requirements and recommended intake of CHO

Total Carbohydrates

The minimal amount of CHO required, either from endogenous or exogenous sources, is determined by the brain's requirement for glucose. The average minimum amount of glucose utilized by the brain is approximately 100 g/day¹⁰. The diets primarily consist of CHO, proteins and fats as the sources of energy; therefore, non-availability of glucose to the brain does not arise. However, the alternate source of energy, when the CHO consumption is very low and availability of glucose is less (eg. in prolonged starvation) energy needs is partially met by utilizing keto acids obtained from break down of fats or utilizing energy from endogenous protein catabolism²⁸⁻³¹. This means that the brain can adapt to a CHO-free diet that is sufficient in energy. The endogenous glucose production rate, in the post absorptive phase, correlates well with the estimated size of the brain from birth to adult life³². The brain size after the age of 5y increases modestly while the consumption of glucose by the brain is fairly constant after 1y; thereby the requirement of CHO for children above 1y is similar to adults³³. Also the rate of decrease in brain mass³⁴ and that of decrease in glucose oxidation³⁵ with advancing age is likely to be modest; therefore the requirement is same for all adults. The difference in brain glucose utilization between genders is negligible. The estimated average requirement (EAR) for CHO has been reported to be 100 g/day for ages 1y and above with a Recommended Dietary Allowance (RDA) of 130 g/day, assuming a coefficient of variance (CV) of 15% based on variation in brain glucose utilization¹⁰. RDA is equal to EAR + 2 times the CV.

For children below 1y of age, an Adequate Intake (AI) value is suggested. The children below 6 months of age, solely depend on human milk for supply of CHO. The CHO present in human milk is lactose. The human milk consumption during 2nd to the 6th month of lactation by full term infants exclusively fed human milk, is reported to be equivalent to 720-780 ml/day commensurate with requirements for maintenance and growth³⁶⁻³⁸ and the corresponding lactose content is 74 g/L^{39,40}. Considering human milk intake of 700 ml/day for Indian 0-6 month infants (as reported elsewhere in this report, the AI for CHO suggested is 52 g/day, rounded to 55 g/day (rounded upwards to the nearest 5 g). For children 7-12 months of age, the CHO content in human milk as well as that provided by complementary food is considered to derive the AI. Therefore, considering the CHO content of 44 g from human milk (based on an average volume of 600ml/day of human milk consumed by Indian infants) and that obtained from median intake of complementary foods by rural and urban 7-12 month Indian infants, to be 50 g/day^{12,13}, and is similar to the usual intakes (51 g/day) reported in the IOM document^{41,42}, the AI suggested for Indian infants in this age group is 94 g/day, rounded to 95 g/day.

During pregnancy, due to the establishment of the placental-fetal unit, there is an increased requirement for the energy yielding substrates to support maternal storage depots during early pregnancy as well as sustain growth and development of the fetus in the last trimester⁴³. The developing fetus in utero utilizes glucose for energy needs. Assuming, the average infant brain weight to be 380 g³⁴ and glucose utilization rate similar to that of adult brain (8.64 g/100g brain weight/day)¹⁰, the glucose requirement for the fetal brain in utero would be 32.8 g/day rounded to 35 g/day. Therefore, the suggested EAR for CHO for pregnant women is 135 g/day which is

derived as the EAR of the non-pregnant non-lactating women (100 g/day) plus the additional requirement for the fetus during the last trimester (35 g/day). Considering a CV of 15% for variation in brain glucose utilization, the RDA for pregnant women is set at 175 g/day.

During lactation, the requirement for CHO increases to support adequate lactose content in human milk. The lactate is synthesized from glucose and therefore the requirement of CHO increases. The lactose content of human milk is estimated to be 74 g/L with minimal variation during the lactation period. Assuming the average amount of human milk secreted is 700 ml/day, the additional requirement of CHO will be 52 g/day (74×0.7) rounded to 55 g/day. Therefore, the EAR for CHO during lactation would be 155 g/day that is derived as the EAR of the non-pregnant non-lactating women (100 g/day) plus the additional amount required to replace CHO secreted in human milk (55 g/day). Considering a CV of 15% for variation in brain glucose utilization, the RDA for lactating women is set at 200 g/day.

The above DRI values may be adapted (Table 7.2.1) until recommendations can be made as more specific data is available from India, related to infant or adult brain size, lactose content of breast milk of healthy Indian women and the amount of CHO present in complementary foods consumed by Indian infants commensurate with good health and nutritional status.

However, apart from CHO, there are other major essential sources of energy in the diet i.e., proteins and fat. Energy from all these sources is essential for various metabolic and physiological needs and a fine balance between them has to be ensured to establish nutritional adequacy and maintain optimal health. Thus, the Acceptable Macronutrient Distribution Range (AMDR) as a proportion of total energy (En%) intake from CHO, proteins and fats has been suggested for individuals based on evidence from epidemiological studies and intervention trials for their association with reducing the risk of chronic disease in the long run and ensuring sufficient intakes of other essential nutrients and physical activity to maintain energy balance. More recently, a 25 year follow-up cohort study from four US communities and meta-analysis of seven multinational studies indicate that the consumption of low CHO diets (providing less than 40 En%) as well as high CHO diets (more than 70 En%) was associated with higher risk of mortality. Moderate CHO consumption (50-55 En %) was associated with the lowest risk of mortality⁴⁴.

Rapid transition in India and shifts in lifestyle factors (diet and physical activity) contribute to over nutrition (overweight and obesity) linked non-communicable diseases (NCDs) at a rate surpassing under nutrition prevalence. This escalation in NCDs seen globally and particularly in low and middle income countries like India across all socio economic classes adds larger proportion to premature mortality⁴⁵. India ranks 2nd in the world with the largest number of people with diabetes (77 million) and will continue to increase to 101 million by the year 2030⁴⁶⁻⁴⁸. Life style factors such as diets, particularly cereal grains and physical inactivity influence, not only obesity but also cardio-metabolic factors such as insulin resistance, pre diabetes and diabetes. The ICMR-INDIAB study has reported a prevalence of 7.3% diabetes and 10.3% prediabetes⁴⁹. Further they rapidly convert from normoglycemia to dysglycemia^{50,51}. Pradeepa *et. al.*, reported that female gender, diabetes, hypertension and high socioeconomic status are associated with generalized and central obesity⁵¹. Recent Global Health Observatory data had indicated 166 millions of overweight and obese adults in India⁵². Further it is known that dyslipidemia (low HDL, high triglyceride) is associated with total CHO and glycaemic load¹⁴. Dietary CHO provide maximum calories in Indian diets and more than half is contributed by refined grains in urban adults and 75% in rural adults. CHO intake is closer or a little above the upper margin of the range recommended by WHO (55 - 75% calories)⁵³. Bulk of the calories is contributed by cereals and millets^{12,13,54}. The percent energy from CHO is in the range of >60-78% in urban and rural areas¹²⁻¹⁵. Therefore, considering cultural aspects, changed scenario of physical activity levels as well as cost considerations, 55-60En% from CHO may be more advantageous with proteins contributing 10-15 En% and fats contributing 20-30

En%, while meeting the requirements of all other essential nutrients and non-nutrients that have possible health benefits. The refined cereal grain intake should be reduced or replaced by whole grains. More than half the intake of cereal grains must consist of whole grains (intact grains).

A tolerable upper limit (TUL) is not set for CHO probably because 1) there is insufficient data on dietary intakes that indicate risk of adverse effects and/or 2) because of the inter-relationship between macronutrients to supply energy, it is not possible to identify if an observed adverse effect is caused either due to a high or a low intake of a certain macronutrient or both (a high intake of one and a low intake of another). Due to a lack of an established TUL, it is suggested to consume CHO within the suggested ranges of AMDR to maintain a healthy diet pattern and thereby avoid undesirable health effects.

Added Sugars

The World Health Organization in 2003 recommended that not more than 10% of total energy should be from free sugars/added sugars with a goal to reduce or prevent diet-related chronic diseases⁹. The Scientific Advisory Committee on Nutrition³ and the World Health Organization¹¹ in 2015 have provisionally recommended a further reduction of free sugars to less than 5%.

The systematic reviews and meta-analysis presented in the WHO¹¹ and SACN³ documents indicate a positive association between consumption of free sugars and development of dental caries, primarily in children⁵⁵. This risk is lower when the free sugar intake contributes less than 10% of the total energy intake and decreased further when the free sugars contributed less than 5% of total energy intake. Similar observation in adults was not possible due to limited studies, but the risk may exist as was suggested that the aetiology of development of dental caries would be similar in adults too. The systematic review commissioned by WHO showed moderate evidence of an association of a reduction in free sugar intakes and a decreased risk of weight gain in children and adults⁵⁶. An association between higher intakes of sugar sweetened beverages and higher incidence of type 2 diabetes mellitus in adults was reported⁵⁷. Further, the WHO suggests including weight gain and dental caries as the key outcomes in studies related to free sugars intake¹¹, as it is known that the risk of developing T2DM and CVD is often mediated through the effects of overweight and obesity, among other risk factors. Indians are at high risk of developing obesity (more fat in the body from day 1 of birth) and are at high risk of developing metabolic syndrome and diabetes⁵⁸. Hence it is good to restrict dietary free sugars to 5-7 En%. More research is required from India to define the upper limit for free sugars/added sugars that is commensurate with reduced risk of obesity, chronic diseases and dental caries.

The suggestions elucidated above may be adopted until more data specific to India is generated through systematic reviews and meta-analysis of their roles in reducing the risk of chronic diseases while satisfying requirements of other essential nutrients conducive to good health.

Glycaemic index, glycaemic load and health implications

CHO-containing foods influence the post-prandial glycaemic response. The glycemic index (GI) of a food is the blood glucose response after consuming a CHO containing food relative to a CHO containing reference food (usually 50g available carbohydrate), viz., glucose or white bread under standard conditions⁵⁹. The glycemic index (GI) of foods is categorized into three groups: a GI value below 55 is defined as low, 56 to 69 as moderate and 70 and above as high^{60,61}. Glycemic load (GL) considers the GI and the total amount of available CHO present in the food consumed ($GL = GI/100 \times CHO$ content). GL value is categorized as low (≤ 10), moderate (10-19) and high (≥ 20)⁶². A weighted GI/GL value can also be calculated for mixed meals or whole diets^{63,64}. Foster-Powell *et al*⁶⁵ and Atkinson *et al*⁶⁶ have compiled international tables with the average GI and GL values of several common foods derived from multiple studies, including a few Indian foods (Table 7.2.2).

Both the GI and GL of the food are important determinants of the post-prandial plasma glucose response. A food with very high GI but if consumed in lower amounts, will provide only a small amount of CHO and hence will have a small GL and vice versa. Therefore, portion size of the food consumed is also important in eliciting the glycemic response. The amount of CHO, type of CHO component, amount of protein and fat, food matrix, nutrient interactions, type of food processing, other food components, etc., influence the glycemic response^{62,67-72}. Many researchers have associated the consumption of natural and minimally processed cereals, vegetables and fruits with health benefits by virtue of their low GI/GL⁶². Low GI/GL foods that contain sugars (sucrose) and undesirable fats may have a lower glycemic response but are energy dense and are not necessarily healthy⁷³. Therefore, GI/GL and glycemic response alone cannot be used; other attributes mentioned above should also be considered before associating a health benefit. The longitudinal observational study among 53644 Danish subjects over 12 years revealed that substituting saturated fat with CHO with low GI reduced cardiovascular risk while CHO with high GI increased the risk of myocardial infarction⁷⁴. Another 5 year follow up cohort study among 64,633 Japanese men and women indicated an association between dietary glycemic load and higher risk of T2DM among women and a higher dietary glycemic index was associated risk of T2DM among men who consumed a higher dietary fat⁷⁵. Though findings from observation studies^{76,77} (Jenkins, 2002; Greenwood *et al*, 2013) indicate predictable effects of dietary GI and GL on circulating glucose, hemoglobin A1c, insulin, triacylglycerol and high density lipoprotein (HDL), results from meta-analysis of RCTs and prospective cohort studies are inconclusive³. Radhika *et al*¹⁴ have reported that dyslipidemia (low HDL, high triglycerides) is associated with total CHO and GL in Indian adults. As such, it is plausible to expect a low GI diet to reduce risk of T2DM and CVD. However, sufficient evidence needed to recommend substantial dietary changes based on GI/GL are not available.

In India more research into glycemic response of individual foods, mixed meals and whole diets is required to be estimated under standard conditions in a sufficiently large number of subjects. Therefore, a GI and GL data base for Indian foods and common Indian recipes from different geographical regions is the need of the hour. Recommendations on the use of GI or GL can be made as more data becomes available from India.

Recommendations for further research

The following priority areas of research are recommended:

- There is a need to estimate content of CHO and its components in foods by direct measurement rather than adopting ‘the difference’ method and inclusion of the same in the national FCT document.
- To develop a database on glycaemic index and glycaemic load of various CHO rich foods and commonly consumed mixed dishes in different regions of India.
- Systematic reviews & meta-analyses to be commissioned for data generated in the previous 10-20 years from India and nearby countries, relating to role of CHO and its components on health implications such as obesity, T2DM, CVD, Dental Caries etc.,
- Evaluation of different approaches to promote the reduction of free/added sugars intake including the intake of sugar-sweetened beverages particularly in children and adolescents.
- To generate evidence on different dietary exposure levels to understand the mechanism of sugar consumption and its effect on energy intakes and health.
- Studies to investigate the levels at which adverse effects occur with chronic high intakes of CHO, fibre, fat, and protein.

- Research on the contribution of chronic exposure to total CHO, type of CHO and dietary patterns to insulin resistance, T2DM, CVD and other chronic disorders such as oral health.
- Long-term studies on the role of glycaemic index in preventing chronic diseases, such as diabetes and coronary heart disease, in healthy individuals.
- Research on efficacy of low GI and low GL whole food diets on risk factors of chronic disorders and its complications.
- To conduct high quality research with sufficient duration of dietary exposures in RCTs with appropriate data on mean change, variances etc., to better inform interpretation of outcomes.

Table 7.2.1: Dietary Reference Intakes for CHO

Adequate Intake (AI), Estimated Average Requirements (EAR) and Recommended Dietary Allowances (RDA) for dietary CHO by age group and physiological status

Group	Criterion	EAR	RDA
Infants, Boys and Girls			
0-6 mo.	Mean content of CHO in human milk	55 g/d(AI)	
6-12 mo.	Mean intake of CHO from human milk and complementary foods	95 g/d (AI)	
Children			
1 - 3 y, Boys	Extrapolated from Adult Values	100 g/d	130 g/d
1 - 3 y, Girls		100 g/d	130 g/d
4 - 6 y, Boys		100 g/d	130 g/d
4 - 6 y, Girls		100 g/d	130 g/d
7 - 9 y, Boys		100 g/d	130 g/d
7 - 9 y, Girls		100 g/d	130 g/d
Adolescents			
10 - 12 y, Boys	Extrapolated from Adult Values	100 g/d	130 g/d
10 - 12 y, Girls		100 g/d	130 g/d
13 - 15 y, Boys		100 g/d	130 g/d
13 - 15 y, Girls		100 g/d	130 g/d
16 - 18 y, Boys		100 g/d	130 g/d
16 - 18 y, Girls		100 g/d	130 g/d
Adults			
18 y and above, Men	Amount of glucose utilized by brain	100 g/d	130 g/d
18 y and above, Women		100 g/d	130 g/d
Pregnant Women	EAR of Adult women plus amount of glucose utilized by fetal brain	135 g/d	175 g/d
Lactating Mothers, 0-6 mo.	EAR of Adult women plus mean content of CHO in human milk	155 g/d	200 g/d
Lactating Mothers, 7-12 mo.		155 g/d	200 g/d

Table 7.2.2: Glycaemic Index and Glycaemic Load of some commonly consumed Indian foods*

Foods	Glycaemic Index (Glucose=100)	Serving size (g/ml)	Available CHO (g)	Glycaemic Load per serve
Cereals including breakfast cereals, Pulses and Legumes and Cereal Pulse combinations				
Whole wheat bread	74± 2	30	15	11
Wheat Roti	66±9	30	16	10
Wheat Chapathi	52± 4	60	32	21
Poori	70 ±13	150	41	28
Semolina Upma	68± 1	150	42	28
White rice, boiled	73± 4	150	40	29
Brown rice, boiled	68± 4	150	33	16
Bajra Roti	57±5	75	50	29
Barley [#]	28±2	NA	NA	NA
Sweet corn	53±4	150	32	17
Corn flakes	81± 6	30	26	21
Porridge, rolled oats	55± 2	250	22	13
Instant oat porridge	79± 3	250	26	17
Rice porridge	78± 9	150	36	25
Millet porridge	67± 5	150	36	25
Muesli	57± 2	30	19	11
Chickpeas	28± 9	150	30	8
Lentils	32± 5	150	17	5
Soya beans	16± 1	150	6	1
Rajmah, soaked & steamed	70 ±11	150	25	17
Dhokla	33± 2	100	20	6
Cheela, green gram	38 ±1	150	26	10
Cheela, bengal gram	36±1	150	28	10
Dosa	66± 11	150	39	26
Idli	69± 9	250	52	36
Pongal	68 ±23	250	52	35
Vegetables, Fruits and their products				
Potato, boiled	78± 4	150	28	14
Potato, instant mash	87± 3	150	20	17
Potato, french fries	63± 5	150	29	22
Carrot, boiled	39± 4	80	6	3
Sweet potato, boiled	63± 6	150	28	17
Plantain/green banana	55± 6	120	21	8
Green Peas	54 ±14	80	7	4
Vegetable soup [#]	48± 5	NA	NA	NA
Apple	36± 2	120	15	6
Watermelon	76± 4	120	6	4
Orange	43± 3	120	11	5
Banana	51± 3	30	20	12
Pineapple	59± 8	120	13	7
Mango	51± 5	120	17	8
Papaya	60±16	120	29	17
Dates	42± 4	60	40	42
Strawberry Jam	49± 3	30	20	10
Apple juice	41± 2	250	28	11
Orange juice	50± 2	250	23	12
Dairy products				
Milk, full fat	39± 3	250	12	3
Milk, skim [#]	37± 4	NA	NA	NA
Ice cream	51± 3	50	13	8
Yogurt, fruit [#]	41± 2	NA	NA	NA
Snacks				
Chocolate	40± 3	50	10	4
Popcorn	65± 5	20	11	8
Potato chips	56± 3	50	21	11
Soft drink / Soda [#]	59± 3	NA	NA	NA

Sources: Extracted from International tables on GI and GL Values published by *Foster-Powell *et al.*, 2002⁶⁵; [#]Atkinson *et al.*, 2008⁶⁶.

References

1. Cummings JH, Stephen AM. Carbohydrate terminology and classification. European journal of clinical nutrition. 2007 Dec; 61(1):S5-18.
2. EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA). Scientific opinion on dietary reference values for carbohydrates and dietary fibre. EFSA Journal. 2010 Mar; 8(3):1462.
3. England PH. SACN Carbohydrates and Health Report. London: TSO. 2015.
4. Atwater WO, Woods CD. The Chemical Composition of American Food Materials. Experiment Station Bulletin no. 28.
5. United States Department of Agriculture (USDA) (2007). Available from <http://www.ars.usda.gov/nutrientdata>.
6. Southgate DA. Digestion and metabolism of sugars. The American journal of clinical nutrition. 1995 Jul 1; 62(1):203S-10S.
7. UN Food and Agriculture Organization. Carbohydrates in human nutrition: report of a joint FAO–WHO expert consultation.
8. McCance RA, Lawrence RD. The Carbohydrate Content of Foods. The Carbohydrate Content of Foods. 1929(135).
9. World Health Organization. Diet, nutrition, and the prevention of chronic diseases: report of a joint WHO/FAO expert consultation. World Health Organization; 2003 Apr 22.
10. Table M. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. National Academy Press: Washington, DC, USA; 2005.
11. World Health Organization. Guideline: sugars intake for adults and children. World Health Organization; 2015 Mar 31.
12. NNMB. Diet and nutritional status of rural population, prevalence of hypertension and diabetes among adults and infant and young child feeding practices. Report of third repeat survey. National Nutrition Monitoring Bureau, Technical Report No: 26. National Institute of Nutrition (ICMR), Hyderabad, 2012.
13. NNMB. Diet and Nutritional Status of Urban Population in India and prevalence of obesity, hypertension, diabetes and hyperlipidemia in urban men and women- a brief NNMB urban nutrition report. NNMB Technical Report No.27. National Nutrition Monitoring Bureau National Institute of Nutrition (ICMR), Hyderabad, 2017.
14. Radhika G, Ganesan A, Sathya RM, Sudha V, Mohan V. Dietary carbohydrates, glycemic load and serum high-density lipoprotein cholesterol concentrations among South Indian adults. European journal of clinical nutrition. 2009 Mar; 63(3):413-20.
15. Sowmya N, Lakshmipriya N, Arumugam K, Venkatachalam S, Vijayalakshmi P, Ruchi V, Geetha G, Anjana RM, Mohan V, Krishnaswamy K, Sudha V. Comparison of dietary profile of a rural south Indian population with the current dietary recommendations for prevention of non-communicable diseases (CURES 147). The Indian journal of medical research. 2016 Jul; 144(1):112.
16. Thorens B, Mueckler M. Glucose transporters in the 21st Century. American Journal of Physiology- Endocrinology and Metabolism. 2010 Feb; 298(2):E141-5.
17. Champ M, Langkilde AM, Brouns F, Kettlitz B, Le Bail-Collet Y. Advances in dietary fibre characterisation. 2. Consumption, chemistry, physiology and measurement of resistant starch; implications for health and food labelling. Nutrition research reviews. 2003 Dec; 16(2):143-61.
18. Flint HJ, Scott KP, Duncan SH, Louis P, Forano E. Microbial degradation of complex carbohydrates in the gut. Gut microbes. 2012 Jul 14; 3(4):289-306.

19. Elia M, Cummings JH. Physiological aspects of energy metabolism and gastrointestinal effects of carbohydrates. *European Journal of Clinical Nutrition*. 2007 Nov 9; 61(S1):S40.
20. Glimcher LH, Lee AH. From sugar to fat: How the transcription factor XBP1 regulates hepatic lipogenesis. *Annals of the New York Academy of Sciences*. 2009 Sep; 1173(Suppl 1):E2.
21. Forouhi NG, Misra A, Mohan V, Taylor R, Yancy W. Dietary and nutritional approaches for prevention and management of type 2 diabetes. *Bmj*. 2018 Jun 13; 361:k2234.
22. Anand SS, Hawkes C, De Souza RJ, Mente A, Dehghan M, Nugent R, Zulyniak MA, Weis T, Bernstein AM, Krauss RM, Kromhout D. Food consumption and its impact on cardiovascular disease: importance of solutions focused on the globalized food system: a report from the workshop convened by the World Heart Federation. *Journal of the American College of Cardiology*. 2015 Oct 6; 66(14):1590-614.
23. Malik VS, Pan A, Willett WC, Hu FB. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. *The American journal of clinical nutrition*. 2013 Oct 1; 98(4):1084-102.
24. Malik VS, Popkin BM, Bray GA, Després JP, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. *Diabetes care*. 2010 Nov 1; 33(11):2477-83.
25. Hauner H, Bechthold A, Boeing H, Brönstrup A, Buyken A, Leschik-Bonnet E, Linseisen J, Schulze M, Strohm D, Wolfram G. Evidence-based guideline of the German Nutrition Society: carbohydrate intake and prevention of nutrition-related diseases. *Annals of Nutrition and Metabolism*. 2012; 60(Suppl. 1):1-58.
26. Sheiham A, James WP. A new understanding of the relationship between sugars, dental caries and fluoride use: implications for limits on sugars consumption. *Public health nutrition*. 2014 Oct; 17(10):2176-84.
27. Breda J, Jewell J, Keller A. The importance of the World Health Organization sugar guidelines for dental health and obesity prevention. *Caries research*. 2019; 53(2):149-52.
28. Carlson MG, Snead WL, Campbell PJ. Fuel and energy metabolism in fasting humans. *The American journal of clinical nutrition*. 1994 Jul 1; 60(1):29-36.
29. Owen OE, Smalley KJ, D'Alessio DA, Mozzoli MA, Dawson EK. Protein, fat, and carbohydrate requirements during starvation: anaplerosis and cataplerosis. *The American journal of clinical nutrition*. 1998 Jul 1; 68(1):12-34.
30. Streja DA, Steiner G, Marliss EB, Vranic M. Turnover and recycling of glucose in man during prolonged fasting. *Metabolism*. 1977 Oct 1; 26(10):1089-98.
31. Sokoloff LO. Metabolism of ketone bodies by the brain. *Annual review of medicine*. 1973 Feb; 24(1):271-80.
32. Dobbing J, Sands J. Quantitative growth and development of human brain. *Archives of disease in childhood*. 1973 Oct 1; 48(10):757-67.
33. Sokoloff L, GG F, EE K. Cerebral nutrition and energy metabolism.
34. Dekaban AS, Sadowsky D. Changes in brain weights during the span of human life: relation of brain weights to body heights and body weights. *Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society*. 1978 Oct; 4(4):345-56.
35. Robert JJ, Cummins JC, Wolfe RR, Durkot M, Matthews DE, Zhao XH, Bier DM, Young VR. Quantitative aspects of glucose production and metabolism in healthy elderly subjects. *Diabetes*. 1982 Mar 1; 31(3):203-11.
36. Butte NF, Garza C, Smith EB, Nichols BL. Human milk intake and growth in exclusively breast-fed infants. *The Journal of pediatrics*. 1984 Feb 1; 104(2):187-95.
37. Atkinson S, Alston-Mills BR, Lönnnerdal BO, Neville MC. Major minerals and ionic constituents of human and bovine milks. In *Handbook of milk composition* 1995 Jan 1 (pp. 593-622). Academic press.

38. Neville MC, Keller R, Seacat J, Lutes V, Neifert M, Casey C, Allen J, Archer P. Studies in human lactation: milk volumes in lactating women during the onset of lactation and full lactation. *The American Journal of Clinical Nutrition*. 1988 Dec 1; 48(6):1375-86.
39. Dewey KG, Lönnérdaal B. Milk and nutrient intake of breast-fed infants from 1 to 6 months: relation to growth and fatness. *Journal of Pediatric Gastroenterology and Nutrition*. 1983 Jan 1; 2(3):497-506.
40. Nommsen LA, Lovelady CA, Heinig MJ, Lönnérdaal B, Dewey KG. Determinants of energy, protein, lipid, and lactose concentrations in human milk during the first 12 mo of lactation: the DARLING Study. *The American journal of clinical nutrition*. 1991 Feb 1; 53(2):457-65.
41. Heinig MJ, Nommsen LA, Peerson JM, Lonnerdal BA, Dewey KG. Energy and protein intakes of breast-fed and formula-fed infants during the first year of life and their association with growth velocity: the DARLING Study. *The American journal of clinical nutrition*. 1993 Aug 1; 58(2):152-61.
42. Specker BL, Beck A, Kalkwarf H, Ho M. Randomized trial of varying mineral intake on total body bone mineral accretion during the first year of life. *Pediatrics*. 1997 Jun 1; 99(6):e12-.
43. Knopp RH, Saudek CD, Arky RA, O'sullivan JB. Two phases of adipose tissue metabolism in pregnancy: maternal adaptations for fetal growth. *Endocrinology*. 1973 Apr 1; 92(4):984-8.
44. Seidelmann SB, Claggett B, Cheng S, Henglin M, Shah A, Steffen LM, Folsom AR, Rimm EB, Willett WC, Solomon SD. Dietary carbohydrate intake and mortality: a prospective cohort study and meta-analysis. *The Lancet Public Health*. 2018 Sep 1; 3(9):e419-28.
45. GBD 2017 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet* (London, England). 2018 Nov 10; 392(10159):1923.
46. Ogurtsova K, da Rocha Fernandes JD, Huang Y, Linnenkamp U, Guariguata L, Cho NH, Cavan D, Shaw JE, Makaroff LE. IDF Diabetes Atlas: Global estimates for the prevalence of diabetes for 2015 and 2040. *Diabetes research and clinical practice*. 2017 Jun 1; 128:40-50.
47. Cho N, Shaw JE, Karuranga S, Huang Y, da Rocha Fernandes JD, Ohlrogge AW, Malanda B. IDF Diabetes Atlas: Global estimates of diabetes prevalence for 2017 and projections for 2045. *Diabetes research and clinical practice*. 2018 Apr 1; 138:271-81.
48. Huang Y, Karuranga S, Malanda B, Williams DR. Call for data contribution to the IDF Diabetes Atlas 9th Edition 2019. *Diabetes research and clinical practice*. 2018 Jun 1; 140:351-2.
49. Anjana RM, Deepa M, Pradeepa R, Mahanta J, Narain K, Das HK, Adhikari P, Rao PV, Saboo B, Kumar A, Bhansali A. Prevalence of diabetes and prediabetes in 15 states of India: results from the ICMR-INDIAB population-based cross-sectional study. *The lancet Diabetes & endocrinology*. 2017 Aug 1; 5(8):585-96.
50. Anjana RM, Rani CS, Deepa M, Pradeepa R, Sudha V, Nair HD, Lakshmipriya N, Subhashini S, Binu VS, Unnikrishnan R, Mohan V. Incidence of diabetes and prediabetes and predictors of progression among Asian Indians: 10-year follow-up of the Chennai Urban Rural Epidemiology Study (CURES). *Diabetes care*. 2015 Aug 1; 38(8):1441-8.
51. Pradeepa R, Anjana RM, Joshi SR, Bhansali A, Deepa M, Joshi PP, Dhandania VK, Madhu SV, Rao PV, Geetha L, Subashini R. Prevalence of generalized & abdominal obesity in urban & rural India-the ICMR-INDIAB Study (Phase-I)[ICMR-INDIAB-3]. *The Indian journal of medical research*. 2015 Aug; 142(2):139.
52. Ministry of Health and Family Welfare (MoHFW), Government of India, UNICEF and Population Council. 2019. Comprehensive National Nutrition Survey (CNNS) National Report. New Delhi. Available from <https://nhm.gov.in/WriteReadData/1892s/1405796031571201348.pdf>.
53. World Health Organization. Diet, nutrition, and the prevention of chronic diseases: report of a joint WHO/FAO expert consultation. World Health Organization; 2003 Apr 22. Available from: <https://www.who.int/dietphysicalactivity/publications/trs916/en/>.

54. National Sample Survey Office. Nutritional Intake in India, 2011-12. 560, NSS 68th Round. National Statistical Organization, Government of India. New Delhi, India. 2014.
55. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *BMJ*. 2013 Jan 15; 346:e7492.
56. Moynihan PJ, Kelly SA. Effect on caries of restricting sugars intake: systematic review to inform WHO guidelines. *Journal of dental research*. 2014 Jan; 93(1):8-18.
57. Greenwood DC, Threapleton DE, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, Burley VJ. Association between sugar-sweetened and artificially sweetened soft drinks and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. *British Journal of Nutrition*. 2014 Sep; 112(5):725-34.
58. Wells JC, Pomeroy E, Walimbe SR, Popkin BM, Yajnik CS. The elevated susceptibility to diabetes in India: an evolutionary perspective. *Frontiers in public health*. 2016 Jul 7; 4:145.
59. Brouns F, Bjorck I, Frayn KN, Gibbs AL, Lang V, Slama G, Wolever TM. Glycaemic index methodology. *Nutrition research reviews*. 2005 Jun; 18(1):145-71.
60. Brand-Miller J, Foster-Powell K, Holt S. *The New Glucose Revolution Complete Guide to Glycemic Index Values*. Marlowe; 2003.
61. International Standards Organisation. Food products—Determination of the glycaemic index (GI) and recommendation for food classification.
62. Venn BJ, Green TJ. Glycemic index and glycemic load: measurement issues and their effect on diet–disease relationships. *European journal of clinical nutrition*. 2007 Dec; 61(1):S122-31.
63. Wolever TM, Jenkins DJ. The use of the glycemic index in predicting the blood glucose response to mixed meals. *The American journal of clinical nutrition*. 1986 Jan 1; 43(1):167-72.
64. Salmerón J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL, Willett WC. Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes care*. 1997 Apr 1; 20(4):545-50.
65. Foster-Powell K, Holt SH, Brand-Miller JC. International table of glycemic index and glycemic load values: 2002. *The American journal of clinical nutrition*. 2002 Jul 1; 76(1):5-6.
66. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. *Diabetes care*. 2008 Dec 1; 31(12):2281-3.
67. Wolever TM. The glycemic index. *World review of nutrition and dietetics*. 1990; 62:120-85.
68. Suzuki H, Fukushima M, Okamoto S, Takahashi O, Shimbo T, Kurose T, Yamada Y, Inagaki N, Seino Y, Fukui T. Effects of thorough mastication on postprandial plasma glucose concentrations in non-obese Japanese subjects. *Metabolism*. 2005 Dec 1; 54(12):1593-9.
69. Henry CJ, Lightowler HJ, Kendall FL, Storey M. The impact of the addition of toppings/fillings on the glycaemic response to commonly consumed carbohydrate foods. *European journal of clinical nutrition*. 2006 Jun; 60(6):763-9.
70. Nuttall FQ, Mooradian AD, Gannon MC, Billington C, Krezowski P. Effect of protein ingestion on the glucose and insulin response to a standardized oral glucose load. *Diabetes care*. 1984 Sep 1; 7(5):465-70.
71. Collier G, O'Dea K. The effect of coingestion of fat on the glucose, insulin, and gastric inhibitory polypeptide responses to carbohydrate and protein. *The American journal of clinical nutrition*. 1983 Jun 1; 37(6):941-4.
72. Sugiyama M, Tang AC, Wakaki Y, Koyama W. Glycemic index of single and mixed meal foods among common Japanese foods with white rice as a reference food. *European journal of clinical nutrition*. 2003 Jun; 57(6):743-52.
73. Freeman J. The glycemic index debate: does the type of carbohydrate really matter? *Diabetes forecast*. 2005; 58: 11.

74. Jakobsen MU, Dethlefsen C, Joensen AM, Stegger J, Tjønneland A, Schmidt EB, Overvad K. Intake of carbohydrates compared with intake of saturated fatty acids and risk of myocardial infarction: importance of the glycemic index. *The American journal of clinical nutrition*. 2010 Jun 1; 91(6):1764-8.
75. Oba S, Nanri A, Kurotani K, Goto A, Kato M, Mizoue T, Noda M, Inoue M, Tsugane S, Japan Public Health Center-based Prospective Study Group. Dietary glycemic index, glycemic load and incidence of type 2 diabetes in Japanese men and women: the Japan Public Health Center-based Prospective Study. *Nutrition journal*. 2013 Dec 1; 12(1):165.
76. Jenkins DJ, Kendall CW, Augustin LS, Franceschi S, Hamidi M, Marchie A, Jenkins AL, Axelsen M. Glycemic index: overview of implications in health and disease. *The American journal of clinical nutrition*. 2002 Jul 1; 76(1):266S-73S.
77. Greenwood DC, Threapleton DE, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, Burley VJ. Glycemic index, glycemic load, carbohydrates, and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. *Diabetes care*. 2013 Dec 1; 36(12):4166-71

8. MINERALS

8.1. CALCIUM AND PHOSPHORUS

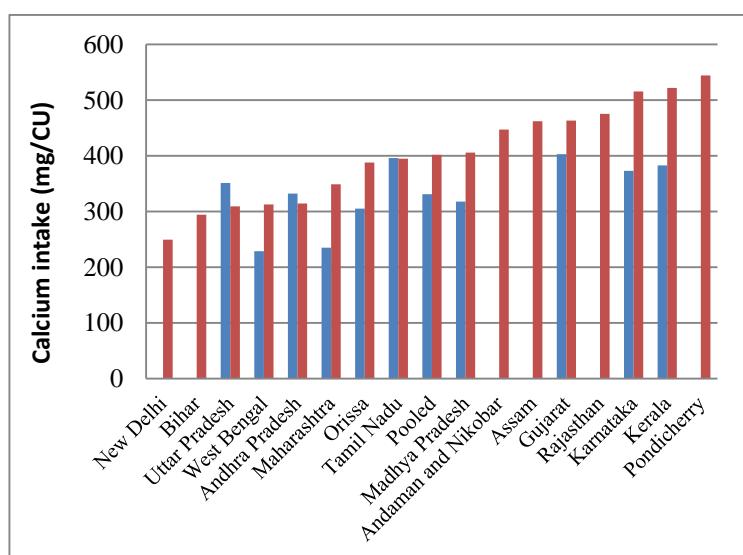
The requirements of calcium (Ca) and phosphorus are considered together as their function and requirements are closely linked. Once the requirements of calcium are assessed, it would be easier to fix the requirements of phosphorus. Calcium is a major element in the body and an adult man of 60 kg has nearly 1 kg of Ca, most of which is present in the bone. An important body function of Ca lies in the formation of the bone. Though small in quantity, non-skeletal Ca has the other important functions like neuromuscular excitation, blood coagulation, membrane permeability and others. Importance of Ca in these functions is reflected in the precision with which plasma Ca level is regulated. Calcium in bone plays a role in maintaining blood level even in the face of dietary Ca inadequacy. Blood calcium level is maintained within narrow limits by the interplay of vitamin D and several hormones, like parathyroid hormone (PTH), thyrocalcitonin, cortisol and gender steroids by controlling absorption, excretion and bone turnover.

Calcium is required by an adult man for replacing Ca lost from the body through stools, urine, bile and sweat. The endogenous fecal losses are estimated to be about 1.4 mg/kg in children, while it is about 2.1 mg/kg in adults. Urinary losses are roughly 40 mg in childhood reaching 80 mg before puberty and tapering down to 40 mg in adults, while median sweat losses have been estimated at 35 mg per m² body surface area¹. About 20-50% of Ca in the diet is absorbed and such absorption is greatly facilitated by vitamin D. Additional Ca is required during growth for skeletal development and during lactation for the calcium secretion in breast milk.

8.1.1. Dietary calcium intake

Calcium intake is fairly high, being in the range of 1 g or more a day in communities that consume plenty of milk as in the West, milk being a rich source of calcium. However, in developing countries where milk intake is low, most dietary calcium comes from cereals. Since these are only a moderate source, the daily intake of Ca in such communities is in a low range of 300-600 mg a day. Based on NNMB (Urban 2016 and Rural 2012) data, the median milk consumption in Urban and Rural India is 122 ml/Consumption Unit (CU) and 85 ml/CU respectively^{2,3}. Calcium intakes from the same data, show intakes of 402 mg/CU (Urban) and 331 mg/CU (Rural) (Figure 8.1.1). Other rich sources of Ca among plant foods are the millet ragi (*Eleusine coracana*), the pseudo cereal, rajkeera (*Amaranthus*) and the green leafy vegetables.

Figure 8.1.1: Calcium intakes (mg) per consumption unit (CU) in Urban (red bars) and Rural (blue bars) India
(Source: Ref 2 & 3)



8.1.2. Calcium requirement

Calcium requirement has been measured by long-term balance studies. Such studies among the Western population, whose habitual diets contain high levels of Ca from generous amounts of milk, have indicated a requirement of Ca of the order of 1 g/day. Population groups in many developing countries subsist on a much lower calcium intake of about 500 mg without any ill effects. Long-term balance studies in such populations indicate that they are in positive Ca balance even on much lower intakes. This is because the body can adapt to different levels of intakes of Ca and maintain a positive Ca balance. These observations were the basis for the RDAs for Ca suggested by the two previous RDA Committees in 2010 and 1990^{4,5}. These recommendations were supported by the review of Nordin, who presented evidence resolving the paradox of low fracture rates associated with low calcium intakes in developing world against the higher fracture rates in the developed world with higher Ca intake⁶. The dramatic differences in the dietary levels of both animal protein and sodium, known to limit calcium loss in urine, were shown to be mainly responsible for the pronounced differences in calcium requirements. It was estimated that a reduction in animal protein from 60g to 20g or sodium from 150 to 50 m mol/d could decrease calcium requirement by about 200 mg/d; a combination of both could be additive, accounting to differences upto 400 mg. Such observations were also endorsed by the 1997 IOM Committee⁷ and WHO Expert Group⁸. In fact, WHO Expert group presented a different set of recommendations of estimated calcium intakes for population from less developed countries subsisting on low levels of animal protein in the diets along with those recommended for population from developed countries (Annexure 8.1).

However, recent epidemiological studies indicate that the net effect of protein on bone turnover is slightly positive, and therefore have a protective effect¹. Several observational and clinical studies have examined the effect of high-protein diets on bone¹. Over a 4-year period in the Framingham Osteoporosis Study, a higher protein intake (84 to 152 g/day), was positively associated with change in femoral neck and spine BMD¹. Other factors that influence calcium absorption include sodium and potassium in the diet. High intakes of sodium increase urinary calcium excretion. In contrast, adding more potassium to a high-sodium diet might help decrease calcium excretion, particularly in postmenopausal women⁷.

Three major approaches are commonly employed in arriving at the requirements of calcium:

1. Calcium balance studies on subjects consuming variable amounts of calcium.
2. A factorial model using calcium accretion based on bone mineral accretion data obtained isotopically or by scanning.
3. Clinical trials investigating the response of change in calcium balance or bone mineral content/density or fracture rate to varying calcium intakes.

All the three approaches finally seek to arrive at requirements as the minimum amount of calcium needed to accrue enough bone mineral content for good bone health (during growth) and bone integrity or compensate the losses or extra demands to maintain the desired level of bone mineral content and bone integrity. With the advent of Dual-energy X-ray absorptiometry (DEXA), estimation of desirable retention of calcium in a dose-dependent, non-invasive manner over a long period at multiple time-points has become possible. Whole body mineral content and density (also multiple bone sites at risk) along with body composition could be examined using DEXA. There have been many studies on populations from developing countries, fully exploiting the advantages of DEXA measurements and transforming it into a powerful tool of bone health. However, such studies are few in Indian population. A recent multi-centric study supported by ICMR has generated large database on bone health of Indian population using DEXA for the first time and are discussed below.

Adults

As indicated above, based on the observations that the body can adapt to different levels of intakes of Ca and maintain a positive Ca balance even at 400 mg dietary calcium intake (RDA committee 1990)⁵ suggested a lower level of calcium intake. On the other hand, there is evidence of widespread Ca depletion as indicated by bone density measurements, particularly in women after repeated episodes of pregnancy and lactation. The strong adaptation to a chronic low-level consumption of calcium seems to be resulting in compromises in stature and restriction in the function of the skeletal system. The fracture rate at the neck of the femur was shown to occur 12-15 years earlier in women from low income group as compared to that in high income group⁹. There have been reports of moderately higher levels of circulating parathyroid hormone in Indians suggesting strains on calcium economy¹⁰.

Attaining peak bone densities is essential to prevent osteoporotic fractures in later life. Also attaining optimal accretion rate of bone mass during puberty is critical for optimum body size and skeletal maturity^{11,12}. There is concern that women from low income group are exposed to a greater risk of developing bone abnormalities due to poor nutrition and their occupational or non-occupational activity¹³ aggravating the situation. Current level of consumption providing less than 400 mg Ca/ d/ CU is not able to protect them from poor bone health and some segments of the population exhibit bone density (spinal) z-scores described as osteoporotic. The previous Committee in 2010, in view of the balance studies and ICMR multi centric study data as indicated above, chose the upper value of calcium requirements for adults (600 mg/d). This was based on reevaluation of all the calcium balance studies carried out on Indian adults published earlier¹⁴⁻¹⁸.

Annexure 8.2 denotes that the Indian adult achieves zero retention at an intake of 334 mg and for retaining 40 mg/d of calcium to adjust for the insensitive losses, the calculated intake will be 480 mg/d. Considering the +2SD (as 25%), the allowance was calculated to 600 mg/d (Annexure 8.1). However, it is important to note that the calcium zero retention set at 334 mg for men, by the previous committee, is much lower than other countries. For example, IOM has set EAR of 800 mg based on Hunt and Johnson study in 2007¹⁹, where the calcium zero retention was 741 mg. There is a further uncertainty as to the true retention rates as there is a wide variability seen from various countries such as china, where zero retention rate has been reported to be 400 mg, Japanese have reported 600 mg and WHO/FAO at 540 mg²⁰.

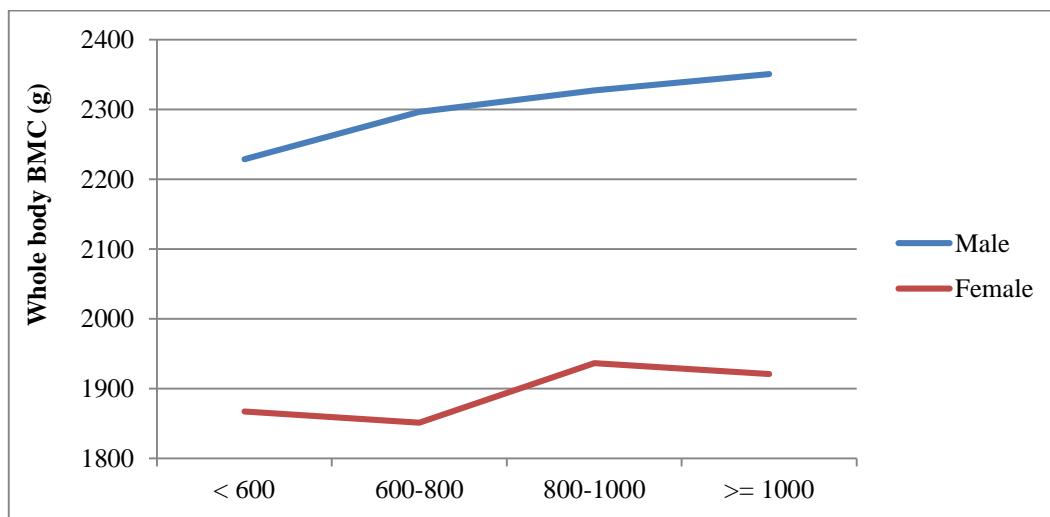
A review of the available ICMR multi-centric studies on bone health in Indian men and women is appropriate here²¹. These studies were carried out in two parts: the first, with the objective of establishing BMD reference values for males and females using DXA. Data on whole body bone mineral content (WBBMC) were obtained in 400 healthy men and women drawn from upper income group with a BMI ranging between 16.5- 25.0 and with an intake of calcium around 1g/d. The results are shown in Table 8.1.1 and Figure 8.1.2.

The results show that WBBMD at all sites was significantly lower than Hologic standards²¹. The z scores of bone density are much lesser than those of Hologic Standards, despite the Ca intakes recording at about 1g. Factors other than calcium intake seem to determine the peak bone content and density. It is interesting to note, while there was an upward trend in WB BMC in healthy men, no such trend was observed in women, where the calcium requirements are more important (Figure 8.1.2). Further, the slope was highest from below 600 to 600-800 ranges in men and 600-800 and 800-1000 ranges in women, suggesting the intakes of 800-1000 mg may be reasonably adequate for this population. Further, a calcium intake was not a determinant of bone density in this population, unlike Vitamin D, which was shown to be positively associated with bone density²¹.

Table 8.1.1: DEXA indicators of bone health in reference groups

		Male	Female	Pooled
Body Mass Index (kg/m²)	N	404	405	809
	Mean	22.5	21.8	22.1
	SD	2.2	2.2	2.2
Age in completed years	N	404	405	809
	Mean	24.8	24.4	24.6
	SD	2.7	2.8	2.8
Average calcium intake (mg/day)	N	400	404	804
	Mean	1150	1012	1081
	SD	553	437	502
Whole body BMD (g/cm²)	N	263	266	529
	Mean	1.13	1.10	1.11
	SD	0.08	0.08	0.09
Hip BMD (g/cm²)	N	404	405	809
	Mean	0.96	0.89	0.93
	SD	0.19	0.15	0.18
Forearm BMD (g/cm²)	N	404	405	809
	Mean	0.60	0.53	0.56
	SD	0.08	0.09	0.09
Spine BMD (g/cm²)	N	404	405	809
	Mean	0.95	0.93	0.94
	SD	0.19	0.17	0.18

Figure 8.1.2: Mean WBBMC among healthy men and women aged 20 to 30 years
 (Source: Ref 21)



The second part of the ICMR multi-centric studies was aimed at estimating the prevalence of osteopenia (rarefaction of bone to less than 1 z score and osteoporosis (to less than 2 z score) in Indian population aged between 30-70+ y (about 750 each of males and females) drawn from the three different socioeconomic backgrounds. The data on WB BMC was not available at all centers and further analysis could not be done.

However, the data from ICMR-NIN was available and are reported. The results indicated that with decreasing income of the groups, there was graded decrease not only in anthropometry and BMI, but also in bone mineral content and calcium intake (mean intakes were 933, 606 and 320 mg/d). Parallel to a decrease in the calcium intake, bone densities were also lower with a decreasing income. Those above 50 years suffered from much worse bone densities than those less than 50 years in the same group.

In order to further extract information in relation to requirements, all the pooled data was subdivided into quartiles of calcium intake and compared for various bone-related indicators (Table 8.1.2).

Table 8.1.2: Whole body bone mineral content (WBBMC) controlled for BMI according to quartiles of calcium intake in adult Indian men and women

Quartiles	Men		Women	
	Diet Ca mg/d	WBBMC * g	Diet Ca mg/d	WBBMC* g
1 st	<344	2088 ± 22 ^a	<326	1611 ± 20 ^a
2 nd	344-581	2137 ± 21 ^{ab}	326-540	1675 ± 19 ^b
3 rd	581-872	2171 ± 21 ^b	540-811	1730 ± 20 ^c
4 th	>872	2250 ± 22 ^c	>811	1848 ± 20 ^d

* The values are expressed as mean±SE. The WBBMC values carrying different superscripts were significantly different in women and men ($p<0.01$).

Here too, there is a graded lowering in bone mineral content in tune with lowering range of intakes of calcium even after adjustment for BMI changes. It could have been more useful if the bone mineral content above a threshold quantity was shown functionally relevant to fix the requirement. In fact, though the pooled data show statistically significant correlation between Ca intake and TBBMC, the R^2 values of 0.15 and 0.125 in males and females, respectively, show poor predictability of BMC content from the intake of calcium. These data on bone health status using DXA in Indian population suggest that an intake of 800 mg calcium/d is associated with better bone health status.

Unlike the past, recently there is a significant increase in the consumption of milk and milk products raising the level of calcium intake, though not enough to meet the requirements given for RDA (WB-BMC) compared to other developed countries. Based on the dietary intake versus bone density projections from ICMR multi-centric data, an adequate calcium intake of 800 to 1000 mg/day for adults appears to be required for optimal bone health among healthy adults. The current committee based on the evidence from balance studies and bone density studies has considered an EAR of 800 mg for the adult population. Adding 25% CV, RDA has been set at 1000 mg for adult males and females.

Post-menopausal women

It is well documented that menopause is accompanied by a sustained rise in obligatory urinary calcium of about 30 mg daily. According to FAO/WHO⁸, calcium absorption certainly does not increase at this time - and probably decreases; this extra urinary calcium leads to a negative calcium balance. There is a consensus that these events are associated with an increase

in bone resorption but controversy continues over whether this is the primary event, or is a response to an increased calcium demand, or both. A published review on calcium nutrition and osteoporosis is available in Indian females²². The authors cite a study carried out in two groups of women aged 20-90 years, with median intakes of calcium of 800 (with lifetime milk consumption) or 480 mg/d (who consumed no milk or less), in whom BMD was measured. The group with higher intake of calcium entered osteoporotic and fracture zones of bone density 10 years later than those with lower intake. In addition, women engaged in hard work and those with lifetime vegetarian habit are stated to be at reduced risk of osteoporosis.

Studies on the 3 socio economic groups at NIN show that even after the age of 50 years, the extent of osteoporosis in the spine is only 16% in the HIG group (with higher calcium intakes of around 1 g) compared to the LIG group with 65% osteoporosis (calcium intakes around 400mg). However, it is logical to recommend sufficient additional calcium (+200mg/d) proportionate to what is recommended by FAO/WHO after menopause to cover at least the extra obligatory loss of 30 mg/d calcium in the urine. Therefore, an additional allowance of 200 mg has been added to the EAR and the RDA has been set at 1200 mg for post-menopausal women.

Pregnant and lactating women

IOM suggests no additional requirements for pregnant women, lactating women, provided the intakes are sufficient for healthy adults¹. However, in a study conducted at NIN (Kulkarni *et al* 2020, unpublished), provision of calcium supplementation of 750 mg to a baseline intakes of 400 to 500 mg improved bone density in this low-income group. Hence, an additional amount of 200 mg has been added to 800 and a total of 1000mg has been set as EAR and adding 10% CV, the RDA is set at 1200mg for lactating women. This is also in line with previous recommendation (ICMR RDA – 2010).

Evidence suggests that there is an increase in absorption of calcium, especially in women consuming less than 1200 mg Ca/d. Hypercalciuria is often present during normal pregnancy as a consequence of the doubling of intestinal calcium absorption that occurs, and pregnancy itself increases the risk of kidney stones¹. Studies on effects of calcium intakes are lacking in pregnant women. However, studies in adolescent girls suggest that that calcium intake of 1,500 to 1,700 mg/day do not interfere with iron or zinc absorption in adolescent girls. However, as calcium intakes among this age group could be higher than those studied, there is little evidence to shed light on the larger issue¹.

Infants and young children

For infants, AI has been set at 300 mg based on breast milk intake and calcium content, which is lower than the previous recommendations of 500 mg set as RDA. The IOM data was used, as children for the first 2 years are expected to grow at same level, given the right environment as observed from WHO multi-centric study¹. IOM review of studies have shown mean intake of 780 ml milk for the first six months, and an average of 264 mg of calcium content in 100 ml of breast milk have been reported. The total intake of calcium comes to 210 mg of calcium for 0-6 months, which has been the AI recommended by IOM in this age group. Similarly, for infants 6-12 months, with a mean intake of 600 ml breast milk and taking the calcium content as 210 mg in 100 ml would give 130 mg of calcium from breast milk in this age group. Adding 140 mg of calcium from solid foods based on studies on infants fed formula feeds; the calcium needed in this age group would be 270 mg of calcium. With mean intakes of breast milk at 700 ml for first six months and 600 ml for the next six months in Indian population, the recommended AI of 300 mg for the current one will be enough to meet the requirements of both age groups of Infants.

Children and adolescents

Cross sectional data given on WB-BMC in healthy boys and girls aged 5 to 17 years²³ were used to calculate retention rates per year and calcium accretion per day was calculated based on the assumption that 32.3% of bone is calcium¹. Information available from clinical trials in which additional calcium was given and balance studies in children were used to confirm the requirements in children and are explained below.

Calcium balance studies (short-term) reported were on children, mainly of 3-11 years of the pre-pubertal age²⁴⁻²⁶. Two unpublished balance studies on children aged 3-7 years (n= 3 or 4) were used to derive the retention rates by the previous committee. The levels of calcium studied were 76, 174, 450 and 610mg/d with energy intake varying between 945-1300 kcal/d. For comparison, the mean values of the earlier balance studies and the individual values of the most recent studies were considered. While balance studies after long-term consumption at each given level are best suited for fixing requirements, validity of short-term balance data can be enhanced by covering wide range of intakes even with less number of observations. The strength of the estimate will be determined by the best fit of the data independently collected over time to a set model. Almost linear relationships between the intake, excretion and retention provide a strong basis for the current recommendations. These data are presented in Annexure 8.3.

The data were used to generate two types of information as is evident from the figures in Annexure 8.3. All the pooled data of individuals as well as that for means appeared to be homogenous with high correlation coefficients and all the data on means also showed the same characteristics (values of r, slopes and intercepts). The means were used to obtain intra-class coefficient. The resulting regression equations were used to derive the level of calcium intake at which, 0 retention is obtained or the line can be extrapolated to obtain the excretion at 0 intake, which is a reflection of obligatory loss. Both these estimates were utilized to obtain calcium requirements at pre-pubertal age.

$$\text{Where intake (X) and retention (Y) were related: } Y = -78.5 + 0.44 X$$

At an intake of 200 mg, there was 0 mg retention as total losses are equal to the intake. Based on the data available on healthy children, the previous committee used IOM⁷ value on bone accretion rates for children to be 125-130 mg at the minimum level. Considering a retention value of 125 mg for full longitudinal bone growth, using the above equation, the intakes required are 462 mg of calcium and considering about 30mg/d for sweat losses at 40% absorption, the intakes needed are 540 mg per day (EAR) and taking mean+2SD level ($2 \times 12.5\% = 25\%$), the total calcium needed for RDA to meet requirement is 700 mg. This level of intake would ensure the requirements of calcium for most of the pre-pubertal population²⁷.

Unfortunately, there are no calcium balance data on pubertal boys or girls from India. However, there has been at least one comprehensive, randomized, placebo controlled clinical trial on middle-income, semi-urban Indian schoolchildren wherein bone mineral content and densities were measured in the whole body and at four sites using DEXA over 14 months of supplementation²⁸. The children habitually received 745 mg calcium in the diets; the placebo controls received 920 mg (through milk vehicle) and the supplemented group 1145 mg calcium every day for 14 months (Table 8.1.3). There was a significant improvement in the calcium status and total bone mineral content in adolescents. The bone mineral accretion data are presented in Tables 8.1.3 and 8.1.4. Increments in bone mineral content are available at two levels of calcium intake in addition to that at the habitual intake at the start. An attempt was therefore made to compare their intakes and bone accretion in two sub-age groups. The design and composition of the groups did not permit segregation of data for boys and girls. A plot of the bone mineral content

with age shows that it increases with age in two phases: one slower phase below 11 years and the other steeper phase between 12-15 years (Annexure 8.4). An attempt was also made, therefore, to calculate mean increase in bone mineral content per day based on the estimated calcium intake from habitual basal diet + two supplements (placebo and supplement) into two broad age subgroups 7-11 and 12-16 years and compare the same with literature values (Tables 8.1.3 & 8.1.4).

Table 8.1.3: The bone changes in calcium treated vs. placebo groups in randomized, controlled trials in adolescents

Source (Ref)	No.	Age (y)	Gender	Length of Study (m)	Calcium Intake Controls (mg/day)	Calcium Intake Treatment (mg/day)	Group Mean Differences	
							Total BMC (g)	Change in BMD (%)
29	94	11.9 ± 0.5	F	18	960	1,314	13.32	Total and Lumbar 10
27	48	9–13	F	12	728	1,200	35.52	Total and Lumbar 7-10
28	122	7-16 / 11.5	F/ M	14	745	1145	22.00	Femur neck 11
30	82	12.5±0.3	F	18	600	1125	37.0	Total body 11
31	149	7.9±0.1	F	12	<880	+850	-	Many sites density

It is striking that the results obtained are in conformity with the literature values on velocity of peak bone formation and the requirements of calcium worked out earlier in pre-pubertal children. They are substantially lower than those during pubertal peak velocity (Tables 8.1.3 and 8.1.4). An intake of 921 mg/d in the total group works out to about 980 mg calcium per day in the pubertal group (>10 y group) and is sufficient to accrue a mean 390 mg mineral in the pubertal group (Table 8.1.3). Similarly, the diet was providing about 850 mg calcium/d in the pre-pubertal sub-group, which was sufficient to accumulate 240 mg of bone mineral content per day. The figures of calcium retention obtained are compared with the requirements suggested for optimum growth of US children (Tables 8.1.3 –8.1.5).

It is well known that the calcium balance and retention both for maintenance and growth are mostly dependent on: race; geographical location; intake level of animal protein; body status of sodium and vitamin D; and of intake of calcium. In general, populations from developing countries are at calcium equilibrium at much lower intakes of calcium and are less prone to calcium deficiency⁸ than those in developed countries. Even in the case of adolescents, the cross-sectional increase in bone calcium and the longitudinal increment were much lower than those noted in children from developed Western countries (Tables 8.1.3 and 8.1.4). The Expert Committee of WHO warned that calcium requirements determined for populations of developed countries cannot be extrapolated to those in developing countries. At the levels of calcium intake far above

habitual and requirement levels of 650 and 800 mg/d, the mineral laid was no better or only marginally better in both subgroups of children justifying the RDA suggested (Table 8.1.). It may also be noted that with increasing intake, the calculated net absorption also decreased in children (Table 8.1.6).

Table 8.1.4: Calcium accretion rates in Indian children (pre-adolescent and adolescent) at different levels of calcium intake

Age group/ Sub-group	Treatment	Dietary Ca mg/d	Increment in bone mineral content	Mean incremental mineral mass (calcium) mg/d	Ca requirement= bone Ca + integ 30-40 mg/d +Obl Urine 40-50 mg/d. (calculated absorption, %)
Sub-group Mean (7-11 y)	Habitual	660	85	233 (77)	147 (22.2)
	Placebo	850	101	240 (80)	150 (17.6)
	Supplement	1060	125	298 (99)	170 (16.0)
Sub-group Mean (11-15y)	Habitual	830	161	440 (140)	230 (27.7)
	Placebo	980	164	390 (130)	220 (22.4)
	Supplement	1230	184	438 (146)	236 (19.2)
Group Mean (10 y)	Habitual	745	123	337 (112)	192 (25.8)
	Placebo	920	133	315 (105)	185 (20.1)
	Supplement	1145	155	368 (123)	203 (17.7)

Supplement = Supplemented with calcium and other micronutrients. Whole body bone mineral content was determined at the start and after 14 months supplementation and average increments were used²⁸. Plotting of bone mineral content of children at the beginning of the study on habitual intakes of calcium with the age and the slope provided estimate of annual increment in the corresponding group. Assuming 25% dietary calcium absorption, an intake of 800 mg is adequate to provide these retentions.

Taking in view of all the above evidence, the current committee has used bone accretion rates for healthy population (Table 8.1.6) and have used factorial approach to derive EAR and RDA for all age groups with RDA ranging from 550 mg in children 4 to 6 years and 1050 mg for children aged 16-18 years based on healthy reference Indian children data²³. A similar approach was used for children aged 1-3 years with a calcium accretion rates of 75 grams derived from longitudinal data from normal birth weight children (NIN unpublished study, Kulkarni B *et al*).

Table 8.1.5: Calcium accretion rates in adolescents

Source (Ref)	Type of study	Age (Years)	Ca intake (mg/d)	Ca retention (mg/d)	Absorp- tion (%)
32	Gain in body and skeletal mass and Ca kinetics	10-18 Male Female	500-700 500-550	292-421 305-330	60
33	Stable isotope	5-12	907	130	28
34	BMC, Suppl.	6-14	908 Placebo 1612 Suppl.	130	
35	Balance studies	12-15 Boys Girls	823- 2164	282 212	
36	Stable isotope	11-14 Girls 19-31 Women	1330	494 283	38 22
37,38	Balance	11-14 Girls 19-30 Women 12-15 Boys	791 1019 700-2001	326 171 171	
39	DEXA Velocity	8-14 Boys Girls	1140 1113	359 284	36.5 29.6

Table 8.1.6: Whole Body Bone mineral content (WB-BMC) in healthy boys and girls aged 5 to 17 years. (Source: Ref 23)

Age	Male		Female		Male	Female
	BMC (g)	Increment per year (g)	BMC (g)	Increment per year (g)	Calcium accretion/day (mg)	Calcium accretion per day (mg)
5+	584		566			
6+	678	94	664	98	83	87
7+	794	116	766	102	103	90
8+	913	119	881	115	105	102
9+	945	32	947	66	28	58
10+	1061	116	1122	175	103	155
11+	1170	109	1314	192	96	170
12+	1380	210	1496	182	186	161
13+	1641	261	1660	164	231	145
14+	1690	49	1780	120	43	106
15+	1832	142	1839	59	126	52
16+	2174	342	1862	23	303	20
17+	2325	151	1968	106	134	94

Fecal endogenous losses were taken at 1.4 mg/kg in children and the total fecal losses were estimated using the average body weights of WHO growth standards for boys and girls. Urinary excretion was taken at 40 mg in all age groups as done by previous committee. Sweat losses was taken at 30 mg in all age groups as done by previous committee. Absorption was taken at 40% for all age groups as done by previous committee (ICMR RDA 2010). CV was set at 12.5% and for 2 SD at 25% while the previous committee set 2 SD at various ranges (25-30%). The details of deriving the same are provided in Annexure 5.

Table 8.1.7: EAR and RDA of calcium for various physiological groups

Age Group	Category	ICMR 2020 (mg/day)	
		EAR	RDA
Men	>18 y	800	1000
Women (NPNL)	>18 y	800	1000
Pregnant women		800	1000
Lactating women		1000	1200
Post-menopausal women		1000	1200
Infants	0-6 m 6-12m	-	300 (AI)
Children	1-3y	400	500
Children	4-6y	450	550
Children	7-9 y	500	650
Boys	10-12y	650	850
Girls	10-12y	650	850
Boys	13-15y	800	1000
Girls	13-15y	800	1000
Boys	16-18y	850	1050
Girls	16-18y	850	1050

Tolerable Upper limit (TUL) for calcium

The ICMR committee report on TUL has been used to fix the TUL by the present committee⁴⁰. The TUL committee looked into various studies in India, and observed that the values recommended by IOM are similar, except for in one study, where an intake of 2100 mg/day resulted in hypercalciuria in 9% of participants and hypercalcemia in 11% of participants. Nevertheless, the committee recommended a TUL of 2500 mg as the upper limit for adults. The TUL committee considered that only few studies on calcium supplementation have reported adverse events and no studies with long term follow up. The TUL committee suggested that high oxalate foods in plant-based diets might also increase the risk of nephrolithiasis. Further, high calcium intakes might also interfere with absorption of iron and zinc and may exacerbate their deficiency. For children aged 1-3 years, 1500 mg has been set as the TUL, beyond which, there is a risk of increased adverse effects in taking calcium. For children aged 4 to 9 years, a value of 2500 mg has been set and TUL value for adolescents aged 9-17 years has been set at 3000 mg. For Adult men and women, a TUL of 2500 mg has been set. Similarly, for pregnant women and lactating women, TUL of 2500 mg has been set.

Table 8.1.8: Tolerable upper limit (TUL) of calcium for various physiological groups

	1-3 years	4-9 years	9-17 years	Adults	Pregnant and lactating women
Calcium (mg/day)	1500	2500	3000	2500	2500

8.1.3. Phosphorus Requirements

The Committee also considered the desirable intake of phosphorus (P). Since P deficiency is unlikely to occur on the types of diets consumed in India, ensuring an adequate P intake may not present a problem. The previous Committee (ICMR RDA 2010) suggested that an elemental Ca:P ratio of 1:1 may be maintained in most age groups, except in infancy, where the ratio suggested is 1:1.5. The present Committee too adopts the same recommendations and the phosphorus values are modified in tune with the calcium recommendations (to maintain the same ratios) (Table 8.1.9).

Table 8.1.9: EAR and RDA of phosphorus for various physiological groups

Age Group	Category	ICMR 2020 (mg/day)	
		EAR	RDA
Men	>18 y	800	1000
Women (NPNL)	>18 y	800	1000
Pregnant women		800	1000
Lactating women		1000	1200
Post-menopausal women		1000	1200
Infants	0-6 m 6-12m	-	450 (AI)
Children	1-3y	400	500
Children	4-6y	450	550
Children	7-9 y	500	650
Boys	10-12y	650	850
Girls	10-12y	650	850
Boys	13-15y	800	1000
Girls	13-15y	800	1000
Boys	16-18y	850	1050
Girls	16-18y	850	1050

References

1. Del Valle HB, Yaktine AL, Taylor CL, Ross AC, editors. Dietary reference intakes for calcium and vitamin D. National Academies Press; 2011 Apr 30.
2. NNMB, Report of Third Repeat Surveys (Rural). National Nutrition Monitoring Bureau, National Institute of Nutrition (ICMR). Hyderabad, 1996-97.
3. NNMB, Report of Urban Surveys. National Nutrition Monitoring Bureau, National Institute of Nutrition (ICMR). Hyderabad 2016.
4. ICMR, Nutrient requirements and Recommended dietary allowances for Indians, A Report of the Expert Group of the National Institute of Nutrition (ICMR), 2010.
5. ICMR, Nutrient requirements and Recommended dietary allowances for Indians, A Report of the Expert Group of the National Institute of Nutrition (ICMR), 1990.
6. Nordin BC. Calcium requirement is a sliding scale. *The American journal of clinical nutrition*. 2000 Jun 1; 71(6):1381-3.
7. Institute of Medicine. Dietary Reference Intakes for Ca, Mg, Vitamin D and F. Food and Nutrition Board, USA, Washington DC, NA Press, 1997.
8. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.
9. Shatrujan V. Osteoporosis in the Asian region: newer questions. *Diet, Nutrition and Chronic Diseases*. 1998;81-3.
10. Sivakumar B, Krishnamachari KA. Circulating levels of immunoreactive parathyroid hormone in endemic genu valgum. *Hormone and Metabolic Research*. 1976; 8(04):317-9.
11. Matkovic V, Jelic T, Wardlaw GM, Illich JZ, Goel PK, Wright JK, Andon MB, Smith KT, Heaney RP. Timing of peak bone mass in Caucasian females and its implication for the prevention of osteoporosis. Inference from a cross-sectional model. *The Journal of clinical investigation*. 1994 Feb 1; 93(2):799-808.
12. Nieves JW, Golden AL, Siris E, Kelsey JL, Lindsay R. Teenage and current calcium intake are related to bone mineral density of the hip and forearm in women aged 30–39 years. *American journal of epidemiology*. 1995 Feb 15; 141(4):342-51.
13. Shatrujan V, Kulkarni B, Kumar PA, Rani KU, Balakrishna N. Bone status of Indian women from a low-income group and its relationship to the nutritional status. *Osteoporosis International*. 2005 Dec 1; 16(12):1827-35.
14. Tandon GS, Teotia SP, Yadav SC, Garg SK. A study of calcium balance in normal individuals and postmenopausal state. *Journal of the Indian Medical Association*. 1973 Sep 1; 61(5):214.
15. Nageswara RC, Narasinga RB. Absorption of calcium from calcium lactate and calcium sulphate by human subjects. *The Indian journal of medical research*. 1974 Mar; 62(3):426.
16. Srikantia SG, Siddiqui AH. Metabolic studies in skeletal fluorosis. *Clinical science*. 1965; 28:477-85.
17. Rao BN, Krishnamachari KA, Sarathy CV. 47 Ca turnover in endemic fluorosis and endemic genu valgum. *British Journal of Nutrition*. 1979 Jan; 41(1):7-14.
18. Ahuja MM, Mohanan P. Calcium phosphorus and nitrogen balance studies in renal calculus disease. *The Indian journal of medical research*. 1970 Apr; 58(4):444.
19. Hunt CD, Johnson LK. Calcium requirements: new estimations for men and women by cross-sectional statistical analyses of calcium balance data from metabolic studies. *American Journal of Clinical Nutrition*, 2007; 1054:63.
20. Fang A, Li K, Shi H, He J, Li H. Calcium requirements for Chinese adults by cross-sectional statistical analyses of calcium balance studies: an individual participant data and aggregate data meta-regression.
21. ICMR Taskforce study. Population based reference standards of peak bone mineral density of Indian males and females- An ICMR multi-centre task force study. Indian Council of Medical Research, New Delhi 2010.

22. Teotia SP, Teotia M. Nutritional bone disease: The continuing challenge to neonatal bone health. Postgrad Med. 2009; 23:30-8.
23. Khadilkar AV, Sanwalka NJ, Chiplonkar SA, Khadilkar VV, Mughal MZ. Normative data and percentile curves for dual energy X-ray absorptiometry in healthy Indian girls and boys aged 5–17 years. Bone. 2011 Apr 1; 48(4):810-9.
24. Sivakumar B. Iron and Calcium balance studies from a special nutritional beverage in children. National Institute of Nutrition, Annual Report, 20-21, 1999-2000.
25. Murthy HBN, Reddy SK, Swaminathan M and Subramanyan V. The metabolism of Nitrogen, calcium and phosphorus in undernourished children. Brit J Nutr. 1955;9:203-215.
26. Perera WD, Reddy V. Effect of vitamin D supplements on calcium absorption in children. Indian Journal of Medical Research. 1971; 59(6):961-4.
27. Chan GM, Hoffman K, McMurry M. Effects of dairy products on bone and body composition in pubertal girls. The Journal of pediatrics. 1995 Apr 1; 126(4):551-6.
28. Shatrugna V, Balakrishna N, Krishnaswamy K. Effect of micronutrient supplement on health and nutritional status of schoolchildren: bone health and body composition. Nutrition. 2006 Jan 1; 22(1):S33-9.
29. Lloyd T, Andon MB, Rollings N, Martel JK, Landis JR, Demers LM, Eggle DF, Kieselhorst K, Kulin HE. Calcium supplementation and bone mineral density in adolescent girls. Jama. 1993 Aug 18; 270(7):841-4.
30. Cadogan J, Eastell R, Jones N, Barker ME. Milk intake and bone mineral acquisition in adolescent girls: randomised, controlled intervention trial. Bmj. 1997 Nov 15; 315(7118):1255-60.
31. Bonjour JP, Carrie AL, Ferrari S, Clavien H, Slosman D, Theintz G, Rizzoli R. Calcium-enriched foods and bone mass growth in prepubertal girls: a randomized, double-blind, placebo-controlled trial. The Journal of clinical investigation. 1997 Mar 15; 99 (6):1287-94.
32. Rao BJ. Nutrient requirement of adolescents. In Proc. Nutr. Soc. India 1985 (Vol. 31, pp. 41-62).
33. Abrams SA, Copeland KC, Gunn SK, Gundberg CM, Klein KO, Ellis KJ. Calcium absorption, bone mass accumulation, and kinetics increase during early pubertal development in girls. The Journal of Clinical Endocrinology & Metabolism. 2000 May 1; 85(5):1805-9.
34. Johnston Jr CC, Miller JZ, Slemenda CW, Reister TK, Hui S, Christian JC, Peacock M. Calcium supplementation and increases in bone mineral density in children. New England journal of medicine. 1992 Jul 9; 327(2):82-7.
35. Jackman LA, Millane SS, Martin BR, Wood OB, McCabe GP, Peacock M, Weaver CM. Calcium retention in relation to calcium intake and postmenarcheal age in adolescent females. The American journal of clinical nutrition. 1997 Aug 1; 66(2):327-33.
36. Wastney ME, Ng J, Smith DB, Martin BR, Peacock M, Weaver CM. Differences in calcium kinetics between adolescent girls and young women. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology. 1996 Jul 1; 271(1):R208-16.
37. Weaver CM, Martin BR, Plawecki KL, Peacock M, Wood OB, Smith DL, Wastney ME. Differences in calcium metabolism between adolescent and adult females. The American journal of clinical nutrition. 1995 Mar 1; 61(3):577-81.
38. Braun M, Martin BR, Kern M, McCabe GP, Peacock M, Jiang Z, Weaver CM. Calcium retention in adolescent boys on a range of controlled calcium intakes-. The American journal of clinical nutrition. 2006 Aug 1; 84(2):414-8.
39. Bailey DA, Martin AD, McKay HA, Whiting S, Mirwald R. Calcium accretion in girls and boys during puberty: a longitudinal analysis. Journal of bone and mineral research. 2000 Nov; 15(11):2245-50.
40. Indian Council of Medical Research: To examine a) allowance of vitamins/minerals more than one RDA in health/dietary supplements and nutraceuticals and b) Safe Upper limits. A Report of the Expert Committee of the Indian Council of Medical Research, 2018.

**Calcium requirements recommended by FAO/WHO (2004),
ICMR 1989 and ICMR 2010**

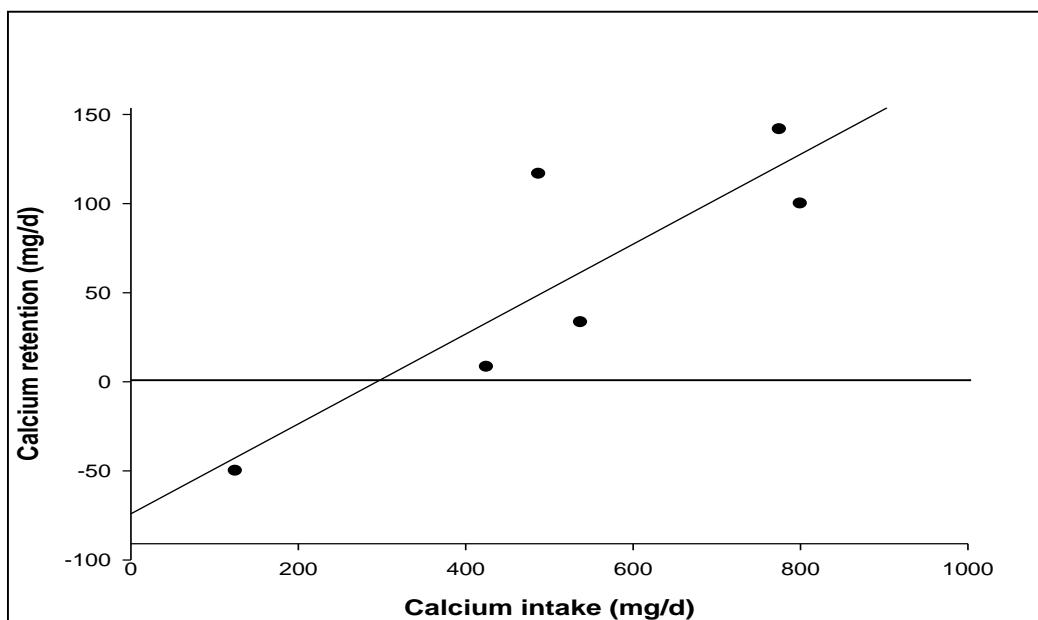
Category	FAO/WHO 2004		ICMR, RDA 1989 (mg/d)	ICMR, RDA 2010 (mg/d)
	High protein intake, developed country (mg/d)	Low protein intake, less developed country (mg/d)		
Infants	400	300*	500	500
Children 1-3 y	500	500	400	600
4-6 y	600	550	400	600
7-9 y	700	700	400	600
Adolescents 10-18 y	1300	1000**	600	800
Adult females	1000	750	400	600
Pregnant women	1200	800	1000	1200
Lactating women	1000	750	1000	1200
Post menopausal women	1300	800	NA	800
Adult men 19-65 y	1000	750	400	600
Adult men 65+ y	1300	800	NA	NA

* Human milk

** Particularly during growth spurt

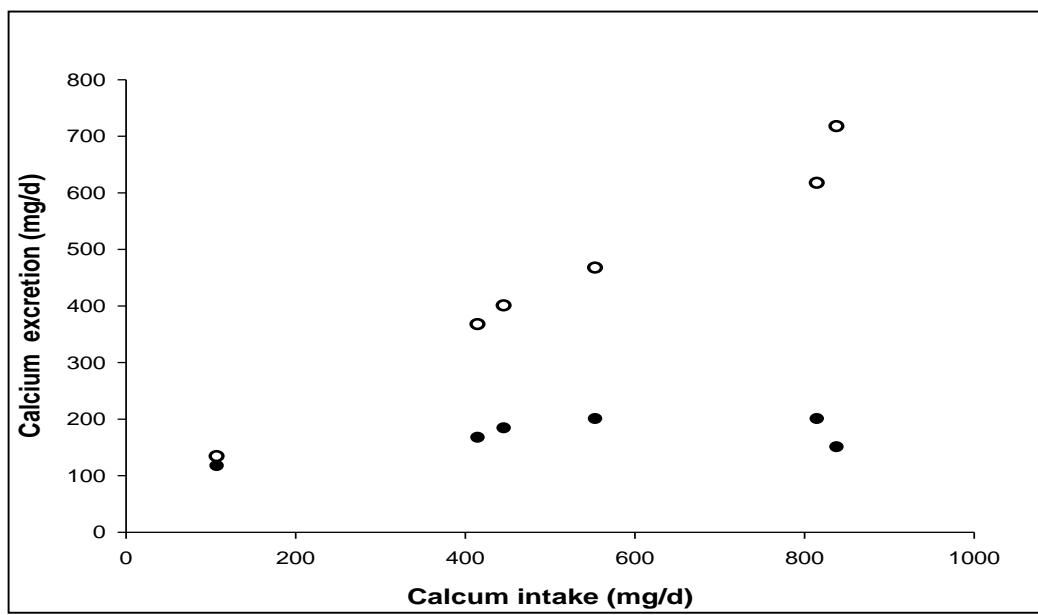
Annexure 8.2

Plot of mean values of calcium retention and intake in Indian adults



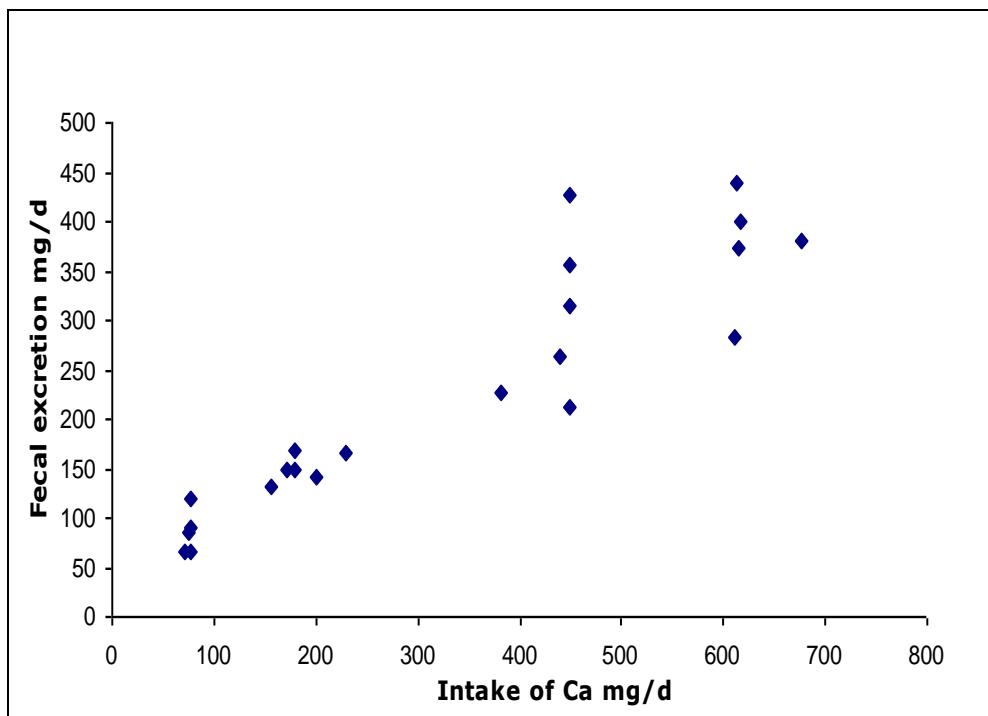
n=6, r= 0.9204; Y = -92.39 + 0.2765 X.

Plot of total urinary and fecal calcium excretion and intake in Indian adults

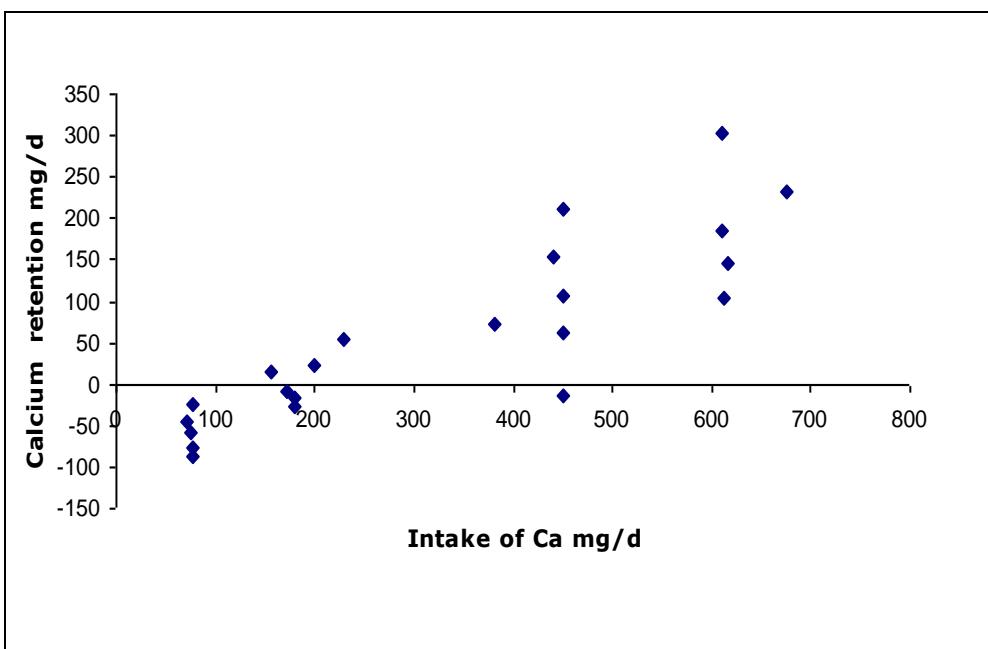


The obligatory loss obtained was 80 mg/d ($r= 0.9945$; $Y = 79 + 0.765 X$, compared to WHO equation: $Y= 142+0.779X$).

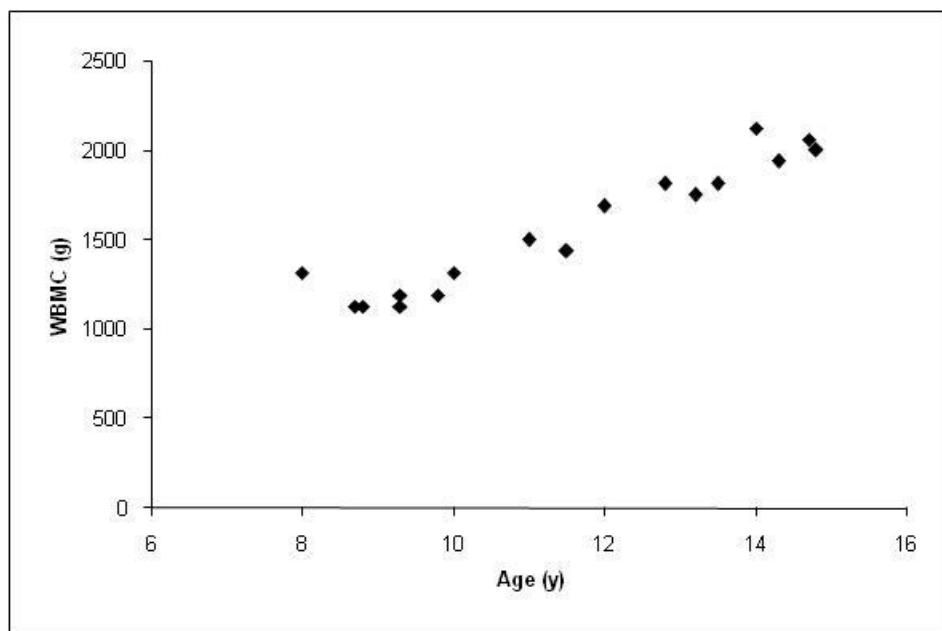
Plot of intake and excretion of Ca in adolescents



Plot of intake and retention of Ca in adolescents



Plot of age related changes in whole body mineral content of school children



Whole body mineral content of middle-income, semi-urban school children is plotted against their mean age. The mineral content increased at a slower rate below the age of 11.5 y (with a slope of about 116 g/y) as compared to the pubertal spurt (with a slope of 170 g/y) during 11-16y²⁸.

Annexure 8.5

Factorial method for estimating calcium requirements in children and adolescents

S No	Age group (years)	1-3	4-6	7-9	10-12	13-15	16-17
1	Weight (Kg)	12	18.4	25.2	35.4	50.3	58
2	Retention of calcium per day (mg)	75	85	95	145	180	180
3	Fecal endogenous loss (mg)	17	26	35	50	70	81
4	Urinary excretion (mg)	40	40	40	40	40	40
5	Sweat loss (mg)	30	30	30	30	30	30
6	Total calcium requirements (Adding steps 2 to 5)	162	181	200	265	320	331
7	Absorption (%)	40	40	40	40	40	40
8	Total intake needed (EAR)	405	452	501	661	801	828
9	RDA (EAR + 2SD)	506	565	626	827	1001	1035

8.2. MAGNESIUM

Since the report of McCollum *et al*¹ inducing magnesium deficiency in rats and dogs in 1933, magnesium deficiency in humans has been always found to be associated with other disease states mostly as inborn errors of absorption and not as an isolated magnesium deficiency. Like calcium, magnesium is closely associated with skeletal system and is homeostatically regulated by calcitropic hormones. About 20-25 g magnesium is present in adult human body and about 60-70% of it occurs in the bone, 25-30% in the muscle, 6-8% in soft tissues and 1% in the extracellular fluid. In adults, magnesium is mostly an integral part of the bone crystal structure along with calcium and phosphorus, but in growing children, a substantial portion of it is in the surface of the bone and its matrix. Magnesium as well as calcium form complexes with phospholipids of cell membranes and nucleic acids. Magnesium is also important for maintaining electrical potential in nerves and muscle membranes. Magnesium deficiency leads to neuromuscular dysfunction.

8.2.1 Diet

Magnesium is widely distributed in foods. As it is the metal ion in chlorophyll, plants form major source of magnesium. Animal products, along with legumes and cereals help to ensure adequate consumption of Magnesium by man. Magnesium is absorbed both by passive diffusion and by a carrier-mediated transport in the intestinal tract. In metabolic studies the absorption of magnesium ranged from 50-70% and in self-selected diet over long periods, the absorption was found to be around 25-30%. Thus, there is no chance of dietary magnesium deficiency occurring under normal dietary situation unless accompanied by a malabsorption syndrome or abnormal disease condition. Magnesium intake in different regions of India was found to range from 540 mg to 1002 mg and average absorption of magnesium ranges from 13% to 50%. A point to note is that there are a few research articles which have recently suggested of evidence that Magnesium intakes have been declining substantially since the beginning of the century in the western world². NNMB urban survey (2016) has noted that the median adult magnesium intake among Indian adults was 323 mg/d.

8.2.2. Deficiency

Symptoms of abnormal neuromuscular function occur in magnesium depletion associated with malabsorption syndromes like inflammatory bowel disease or sprue, primary idiopathic hypomagnesemia and severe protein energy malnutrition. In severe deficiency, the subjects suffer often from tetany and convulsions. Hypomagnesemia, hypocalcemia and hypokalemia are always associated with magnesium deficiency and are reversed by magnesium repletion. The symptoms are largely as a result of associated hypomagnesemia, hypocalcemia and hypokalemia². A syndrome of magnesium-dependent, vitamin D-resistant rickets was described in Indian child case reports³.

8.2.3. Biochemical function

Magnesium is the main intracellular earth metal cation and the second most abundant intracellular cation, with a free cytosol concentration of around 0.5 mmol/L². Magnesium is an important cofactor of many regulatory enzymes, particularly the kinases, and is fundamental in the energy transfer reactions involving high energy compounds like ATP and creatine phosphate and thus muscle contraction. This also explains the key role of magnesium in heart health and skeletal system. Blood magnesium is maintained in a narrow range like that of calcium. In view of this close association of occurrence and functions of Ca and Mg, there have been mutually reinforcing as well as contraindicative roles of these two divalent anions, particularly in bone

health and hypertension. A large number of interventional and clinical studies reveal variable effects of magnesium supplementation on essential hypertension⁴ and pregnancy-induced hypertension and pre-eclampsia^{5,6,7}, other than the associated changes that take place due to hypokalemia and its correction. Several studies have linked its role in vascular complications of Diabetes and stroke, while some studies have implicated its neuro-protective nature in several experimental models of ischemic and excitotoxic brain injury².

In a report by Singh RB *et al.*⁸ lower levels of plasma magnesium and zinc and reduced zinc / copper ratio were associated with coronary artery disease in Indians. The dietary intake of magnesium reported by them was 430 mg and 370 mg/d in the rural and urban population, respectively, confirming the earlier data of Rao and Rao collected in 1980⁹. These are further confirmed by reports from North India by Kapil and others^{10,11}.

8.2.4. Requirements

Based on the balance studies and tracer turnover studies, the requirement of magnesium has been worked out for adults. The earlier Committee did not suggest any RDA as there was no possibility of any Mg deficiency, in our population. The intakes were estimated to vary between 540 mg -1000 mg/d in different regional diets in India. The absorption also varied between 20-50%. At any intake tested, the retention was positive and was above 20-30 mg/d.³

These intakes suggest that there is no scope for Mg deficiency as the recommendations of FAO/WHO were 260 mg/d for adult males⁵ and 420 mg/d according to US National Academy of Sciences.

Recent stable isotopic studies with ²⁵Mg and ²⁶Mg indicated that 50% and 90% labelled Mg from maternal milk can be absorbed by an infant¹². Some recent studies in 26 adolescent females⁷ showed that 52.6.7% Mg was absorbed in a day, resulting in 174 mg retention of Mg daily. On the basis of 50% absorption, FAO/WHO⁶ has recommended desirable intake of Mg as 4 mg/kg/day for international use.

The data of Rao and Rao⁹ were reanalyzed for determining the safe intakes. The data given as mean values with each diet were treated as a cluster and subjected to correlations (intra-class) for possibly arriving at the intakes for equilibrium or zero balance. As to be expected, there was no significant linear correlation between Mg intake on one hand and any of the parameters like urinary excretion or balance, both as absolute values or as % of intake on the other. This obviously makes the task difficult to work out the requirements based on retention data directly. However, there was a significant positive correlation between intake and fecal loss (Figure 8.2.1A). The regression equation applicable was $Y = -184 + (0.916) X$, where X is intake and Y is excretion of Mg (mg/d) in stool. With increasing intake in the diet, there is an increase in the excretion of Mg. By extrapolation of the regression line, the fecal loss was 184 mg at 0 intake and this, in principle corresponds to obligatory fecal loss. Also there was a curvilinear relationship between intake and urinary excretion (Fig 8.2.1B). From this, it appears that the urinary loss is more or less constant around 135 mg in the range of intakes of 540-796 mg and then steadily increases to a mean of 191 mg in ranging intakes of 863-1002 mg. Therefore, one can expect an average urinary loss of 130 mg at habitual intake range and the total amount of Mg loss appeared to be in equilibrium (in all situations the balance was adequately positive). Thus, a total intake of $184 + 135 = 319$ or 320 mg of dietary Mg could be considered as the safe average requirement. This exercise serves as the basis for estimating the EAR for adult males to be 320 mg/d. The variance in requirements cannot be determined from the available data. Thus, a coefficient of variation (CV) of 10 percent is assumed¹³, thus RDA for magnesium is computed as 385 mg/d.

The EARs of adult women, pregnant and lactating women were adjusted to weight. EARs for children have been estimated based on relative body weights, as a function of reference weights and growth, using the formula:

$$\text{EAR}_{\text{child}} = \text{EAR}_{\text{adult}} \times F, \text{ where}$$

$$F = (\text{Weight of child}/\text{weight of adult})^{0.75} \times (1+GF)$$

$$GF = 0.57 \text{ for ages up to 1 years,}$$

$$0.25 \text{ for ages 1 to 3,}$$

$$0.06 \text{ for ages 4 to 6,}$$

$$0.13 \text{ for ages 7 to 9,}$$

$$\text{In ages 10 to 15} - 0.11 \text{ for boys and } 0.08 \text{ for girls,}$$

$$\text{In ages 16 to 18} - 0.08 \text{ for boys and } 0.03 \text{ for girls.}$$

RDAs were calculated from EARs with a coefficient of Variation of 10%.

Table 8.2.1. Recommended Daily Allowance of Magnesium for Indians

Physiological Group	Body weight (Kg)	RDA 2020		RDA 2010 (mg/day)	TUL* (mg/day)	H-AR	IOM - 2000 RDA (mg/day)
		EAR (mg/day)	RDA (mg/day)				
Adult Men	65	320	385	340	350	350	420
Adult Women	55	270	325	310	350	265	320
Pregnant	55+GWG	320	385	310	350	390	350
Lactating	55+	270	325	310	350	255	310
Infants (0-6 mo)	5.8	-	30(AI)	30	-	-	30
Infants (6-12 mo)	8.5	-	75(AI)	45	-	-	75
Children (1-3y)	11.7	111	135	50	65	65	80
Children (4-6y)	18.3	131	155	70	110	110	130
Children (7-9y)	25.3	178	215	100	110	110	130
Boys (10-12y)	34.9	223	270	120	350	200	240
Girls (10-12y)	36.4	214	255	160	350	200	240
Boys (13-15y)	50.5	294	355	165	350	200	410
Girls (13-15y)	49.6	270	325	210	350	200	360
Boys (16-18y)	63.1	338	405	195	350	340	410
Girls (16-18y)	55.2	279	335	235	350	300	360

*TULs are only for non-dietary pharmacological doses of magnesium, whereas dietary intake can go well above the TUL value.

There is a need for some renewed balance studies on Mg requirements for fixing RDA for Indians of all physiological groups.

8.2.5. Tolerable upper limits

Magnesium, when ingested as a naturally occurring substance in foods, has not been demonstrated to exert any adverse effects. However, adverse effects of excess magnesium intake have been observed with intakes from nonfood sources such as various magnesium salts used for pharmacologic purposes. Thus, a Tolerable Upper Intake Level (UL) cannot be based on magnesium obtained from foods. Therefore, magnesium intake that could result in adverse effects was from that obtained from its pharmacological use.

Magnesium is generally used in drugs for its cathartic effect, and causes osmotic diarrhea on excessive consumption. This diarrhea is not demonstrated with normal or even large dietary intakes of magnesium. Other effects of hyper magnesium include metabolic alkalosis, dehydration, paralytic ileus and rarely cardiac arrest.

Figure 8.2.1: Correlation between magnesium intake and fecal excretion

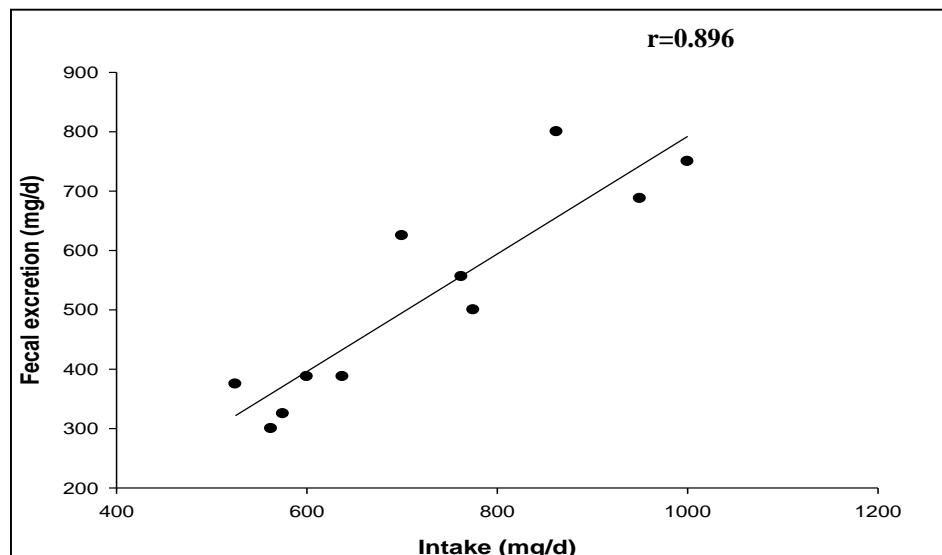
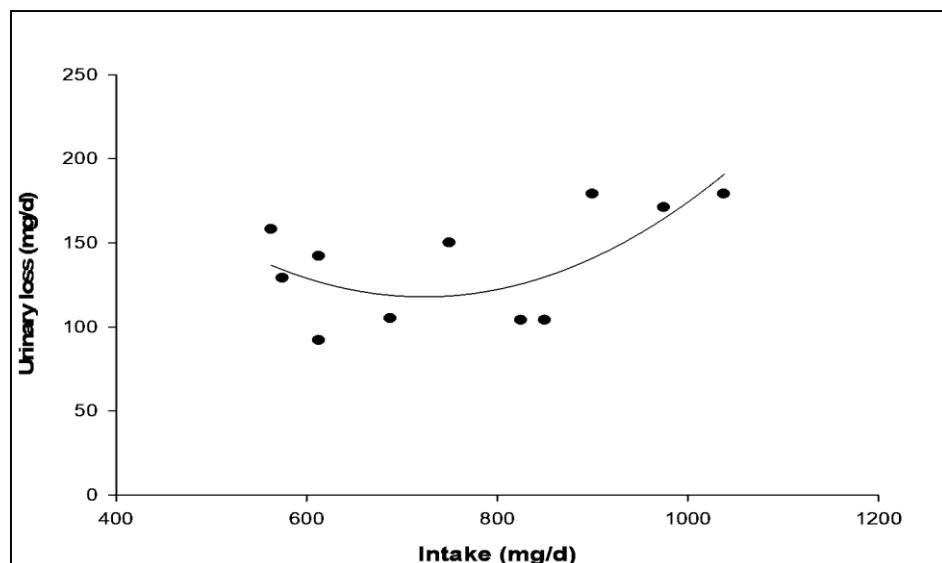


Figure 8.2.2: Relationship between magnesium intake and urinary loss



The LOAEL identified for magnesium-induced diarrhea in adults is 360 mg (15 mmol)/day of magnesium from nonfood sources based on the results of Bashir *et al.*¹⁴ With an uncertainty factor of 1.0, TUL was calculated at 350 mg per day of non-dietary consumption for adolescents and adults including the pregnant and lactating. TUL for ages 1 to 9 are calculated on body-weight basis and reference weights¹³.

References

- 1 Kruse HD, Orent ER. Studies on magnesium deficiency in animals. 1. Symptomatology resulting from magnesium deprivation. *Journal of Biological Chemistry*. 1932; 96:519-39.
- 2 Saris NE, Mervaala E, Karppanen H, Khawaja JA, Lewenstam A. Magnesium: an update on physiological, clinical and analytical aspects. *Clinica chimica acta*. 2000 Apr 1; 294(1-2):1-26.
- 3 Reddy V, Sivakumar B. Magnesium-dependent vitamin-D-resistant rickets. *The Lancet*. 1974 May 18; 303(7864):963-5.
- 4 Tillman DM, Semple PF. Calcium and magnesium in essential hypertension. *Clinical Science*. 1988 Oct; 75(4):395-402.
- 5 Roberts JM, Balk JL, Bodnar LM, Belizán JM, Bergel E, Martinez A. Nutrient involvement in preeclampsia. *The Journal of nutrition*. 2003 May 1; 133(5):1684S-92S.
- 6 Makrides M, Crowther CA. Magnesium supplementation in pregnancy. [Update in Cochrane Database Syst Rev. 2001; (4): CD000937; PMID: 11687087]. *Cochrane Database Syst Rev*. 2000.
- 7 Bucher HC, Guyatt GH, Cook RJ, Hatala R, Cook DJ, Lang JD, Hunt D. Effect of calcium supplementation on pregnancy-induced hypertension and preeclampsia: a meta-analysis of randomized controlled trials. *Jama*. 1996 Apr 10; 275(14):1113-7.
- 8 Singh RB, Gupta UC, Mittal N, Niaz MA, Ghosh S, Rastogi V. Epidemiologic study of trace elements and magnesium on risk of coronary artery disease in rural and urban Indian populations. *Journal of the American College of Nutrition*. 1997 Feb 1; 16(1):62-7.
- 9 Rao N, Rao N. Absorption and retention of magnesium and some trace elements by man from typical Indian diets. *Annals of Nutrition and Metabolism*. 1980; 24(4):244-54.
- 10 Pathak P, Kapil U, Kapoor SK, Saxena R, Kumar A, Gupta N, Dwivedi SN, Singh R, Singh P. Prevalence of multiple micronutrient deficiencies amongst pregnant women in a rural area of Haryana. *The Indian Journal of Pediatrics*. 2004 Nov 1; 71(11):1007-14.
- 11 Kapil U, Verma D, Goel M, Saxena N, Gnanasekaran N, Goindi G, Nayar D. Dietary intake of trace elements and minerals among adults in underprivileged communities of rural Rajasthan, India. *Asia Pacific journal of clinical nutrition*. 1998 Mar; 7:29-32.
- 12 Liu YM, Neal P, Ernst J, Weaver C, Rickard K, Smith DL, Lemons J. Absorption of calcium and magnesium from fortified human milk by very low birth weight infants. *Pediatric research*. 1989 May; 25(5):496-502.
- 13 Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. *Dietary reference intakes*. In: *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride* 1997. National Academies Press (US).
- 14 Bashir Y, Sneddon JF, Staunton HA, Haywood GA, Simpson IA, McKenna WJ, Camm AJ. Effects of long-term oral magnesium chloride replacement in congestive heart failure secondary to coronary artery disease. *The American journal of cardiology*. 1993 Nov 15; 72(15):1156-62.

8.3 SODIUM AND POTASSIUM

SODIUM (Na^+):

INTRODUCTION

Sodium is the 6th most abundant element on earth and the principal cation in extracellular fluid in the body, and is an essential nutrient necessary for maintenance of plasma volume, acid–base balance, transmission of nerve impulses and normal cell function. The sodium-potassium gradient across the cellular membrane is maintained by a sodium-potassium pump or Na^+/K^+ ATPase, which uses considerable energy in the body at rest. Sodium is a macro element (required in milligram quantities), and functions as the “osmotic skeleton” of the extracellular fluid. The body content of sodium and its concentration in body fluids is under tight homoeostatic control. Kidney is the primary organ responsible for maintaining sodium balance through aldosterone action on renal tubular function. When the dietary intake is zero, level of aldosterone increases and urinary sodium rapidly decreases. Converse is true when sodium intake is high. Man and other mammals have evolved on a no-added salt diet. Records of primitive people suggest that they did not use much salt and were free from hypertension¹. On land, which may be considered to be a sodium-restricted environment, powerful inbuilt mechanisms such as renal-renin-angiotensin aldosterone and the kinin-prostaglandin systems have evolved a mechanism to maintain blood pressure and renal blood flow on low or minimal sodium intake.

Dietary Sources

The main source of sodium is common salt (sodium chloride), by weight table salt is approximately 40 percent sodium and 60 percent chloride. The salt content of natural diets, predominantly plant-based foods in India, does not exceed 300-400 mg of Na (1g of NaCl). Sodium comes from domestic cooking as well as from processed foods such as bakery products, pickles, dry fish, nuts etc. Sodium is also found naturally in a variety of foods, such as milk, meat and shellfish. Diet provides 90% sodium from sodium chloride (salt) and only 10-15% originates from natural foods. High amounts of sodium are found in many condiments (e.g. soy and fish sauces). Thus, a diet high in processed foods and low in fresh fruits and vegetables is often high in sodium. The total body content of Na^+ in a 60 kg man is approximately 92 g.

Absorption & Excretion

Sodium is rapidly absorbed from the gastrointestinal tract and the usual routes of excretion are urine, feces and skin. In healthy individuals, nearly 100% of ingested sodium is absorbed during digestion, and urinary excretion is the primary mechanism for maintaining sodium balance. Even in hot, humid climates, there are only minimal loses through feces and sweat.

Adequate Intakes for Sodium

Sodium content of breast milk is higher immediately after delivery (65-75 mmol/L) and falls precipitously by day three. Mature human milk contains 7 mmol/L of sodium². Although the minimum intake level necessary for proper bodily function is not well defined, it is estimated to be as little as 200–500 mg/day. Based on the National Academy of Sciences, USA “Dietary Reference Intakes for Sodium and Potassium (2019)” the following adequate intakes [AI] have been proposed for sodium for the different age groups. The AI for infants 0-6 months and 7-12 months is 110 mg/day and 370 mg/day respectively. Children [1-3y] the AI is 800 mg/day while [4-8y] is 1000 mg/day; 9-13y it is 1200 mg/day and 14-18y it is fixed at 1500 mg/day irrespective

of gender; and >19 years is fixed at 1500 mg/day. The AI for adults of 19 years of age and older is based on the lowest levels of sodium intakes evaluated in randomized controlled trials for which there was no evidence of deficiency, evidence from the best-designed balance study, and insufficient evidence of harmful effects from observational studies. Sodium AIs for children and adolescents were extrapolated based on sedentary Estimated Energy Requirements. For infants, the AI's were derived from estimates of sodium intakes in breastfed infants.

The function of sodium and potassium in the body are closely linked. As sodium consumption increases, increased consumption of potassium may be even more beneficial because, in addition to other benefits, it can mitigate the negative effects of elevated sodium consumption on blood pressure. Potassium increases urinary sodium excretion which diminishes body sodium. In addition, potassium is thought to induce vascular smooth muscle relaxation and thus decrease peripheral resistance. The blood pressure lowering effects of potassium intake are greater in individuals ingesting higher sodium, and the Na:K ratio had a stronger effect on the risk of cardiovascular disease than sodium or potassium alone. Studies have reported that maintaining a 1:1 ratio of the two nutrients is an important factor in cardiovascular disease and mortality^{3,4}.

Deficiency and Excess

Normal sodium levels are usually in the range of 136-145 mmol/L. Low sodium levels referred to as 'Hyponatremia' occurs when the concentration of sodium in the blood is abnormally low. Hyponatremia occurs due to one or more factors, ranging from an underlying medical condition, kidney and liver problems, adrenal gland insufficiency, drinking too much water and chronic diarrhea. When this happens, the body's water levels rise, and cells begin to swell. Symptoms of hyponatremia include nausea, vomiting, confusion, restlessness, muscle weakness, seizures and coma. On the other hand high blood levels of sodium also known as 'Hypernatremia', is caused due to low fluid intake, diarrhea and kidney dysfunction. Typically the initial symptom of hypernatremia is thirst. If the volume of water lost is greater than sodium loss and if water replacement is inadequate due to failure of thirst mechanism, then hypernatremia could occur. However, hypernatremia can cause serious symptoms such as muscle twitching, seizures and coma.

Hypertension and salt intake studies from India

The average Indian diet has undergone a drastic nutrition transition over the last three decades. The intake of unprocessed cereals, fruits and vegetables has decreased while consumption of meat products and processed foods has increased⁵. High BP is the leading cause of non-communicable disease (NCD) in India and the prevalence rates of hypertension are expected to almost double from 118 million in 2000 to 213 million in 2025⁶. The Global burden of disease study (2010) reported excess salt intake to be the seventh leading cause of global mortality from cardiovascular disease⁷. Studies done in adolescent and adult Indian population indicate a clear link between high salt and occurrence of hypertension⁸⁻¹⁰. The daily intake in Indians varies between 2.3 g - 9.2 g depending on the dietary habits. About 90 percent of the sodium we eat is in the form of sodium chloride. A single teaspoon of salt contains 2.3 grams of sodium, which is the maximum recommended amount per day. A person with high blood pressure should get no more than 1,500 milligrams per day, and the limit drops even lower if you have kidney or liver problems. A single dash of table salt contains about 150 mg of sodium. Indians have been taking much more salt as against the 5 g/day or 2 g sodium/day limit recommended by WHO¹¹, said a new study conducted by the Public Health Foundation of India (PHFI). The study found that salt intake in Delhi and Haryana was 9.5 g/day and 10.4 g/day in Andhra Pradesh¹². All member states of WHO including India, have set a target of 30% reduction in mean population salt intake by the year 2025, as part of the '25 by 25' initiative for the control of NCDs¹³.

Studies conducted in different regions of India show that salt consumption is way beyond the WHO-recommended maximum of 5 g/day/person. A population survey of 1395 persons from both urban and rural [North & South] India indicated a salt intake of 9-10 g/day¹⁴. Two other studies conducted in 8000 and 1900 individuals from Southern India suggested higher dietary intake of salt which was an independent predictor of systolic BP^{15, 16}. Yet another study from South India in subjects with diabetes, hypertension and renal dysfunction indicated intake of high dietary salt¹⁷. A study from western India showed higher salt intake and high sodium content in ready-to-eat foods¹⁸. The ICMR-INDIAB study (2015) reported high salt intake (>6.5 g/day) with a significantly higher risk for hypertension in three states and one union territory¹⁹. A cross-sectional study carried out in infants (6.5 months old) in West Bengal reported a correlation between poor early growth and high salt intake which could be a risk factor for arterial hypertension²⁰.

A recent survey conducted in participants from North and South India showed high consumption of salt (~11 g/day) and most of it came from added salt²¹. A community based cross-sectional study was conducted among women aged 20 to 59 years in North India, where knowledge, attitude, and behavior questionnaire given by the World Health Organization and 24h dietary recall were used²². The results show that approximately, 80% of the participants believed that high salt diet causes serious health problems, and only 5% of the participants were aware of the existence of a recommendation for daily salt intake. Vegetable-based dishes were found to be the major contributors to the daily salt intake followed by pulse-based and cereal-based dishes, because of the high quantity in which they are consumed²². The INTERSALT study is a standardized, worldwide epidemiologic study of large sample size (n = 10079 men and women aged 20-59y from 32 countries) that tested both within and cross-population 24h sodium excretion and blood pressure. For individuals, a significant, positive, independent linear relation between 24h sodium excretion and systolic blood pressure (SBP) was found²³.

The National Nutrition Monitoring Bureau (NNMB) has conducted a large and comprehensive urban nutrition survey in 2016 [using a one day 24-hour dietary recall method] for the first time in 16 states in India, where the information on dietary intakes was collected from about 13,000 households. The consumption level of salt through dietary recall, were assessed for individual subjects in the study. The state wise salt consumption among rural communities was done by NNMB rural survey in 2011-12. The salt intake was below the recommended level (5 g/day) in rural adults in both the genders, while higher than the recommended level in the urban adult population. The sodium and potassium intakes were computed from the 24h dietary recall method by using the IFCT tables [*for urban population*] and NVIF tables [*for rural population*]. The median sodium intakes appear to be higher than the WHO recommended intake (2000 mg/day) in the adult [18-60y] urban population and much lower in the adult rural population in both the genders.

The NNMB urban survey data in respect of salt, sodium and potassium intakes was classified further into subjects who are non-hypertensive and hypertensive of both the genders. The data suggests that on an average the salt or sodium intake was higher in males compared to females among the non-hypertensive adult population, while the potassium intakes were lower in females compared to males irrespective of whether they were hypertensives or non-hypertensives. Higher percentage of subjects were found to be consuming more amount of salt (>5 g/day) and sodium (>2 g/day) irrespective of whether they were hypertensives or non-hypertensives in both the genders. Lower percentage (21.1%) of females consuming <2 g of sodium and >3.5 g of potassium were hypertensive compared to males (39.7%) who were hypertensive. Also, lower percentage [5-11%] of subjects were consuming more than the recommended amount of potassium [i.e. 3.5 g/day] in both the genders.

POTASSIUM (K^+):

INTRODUCTION

Potassium is the most abundant intracellular cation in the body. It is an essential nutrient needed for maintenance of total body fluid volume, acid-base and electrolyte balance, and normal cell function. Its total body content in a 60 kg man approximates 3000 to 4000 mmol (50 mmol/kg of body weight). Eighty percent of this amount is in the skeletal muscles. Of the total body stores of potassium, about 98% is in the intracellular fluid (ICF) at concentrations of 140 to 150 mmol/L, whereas the extracellular fluid (ECF) contains only 2% [60-80mmol] of total body potassium. The normal serum potassium concentration is maintained within a narrow range of 3.5 to 5.0 mmol/L over a wide range of potassium intakes. Potassium content decreases with age and adiposity. Total body potassium corresponds closely to lean body mass. Enzymes involved in glycolysis and oxidative phosphorylation are potassium-dependent.

Dietary Sources

Natural diets usually provide around 50-150 mmol of potassium per day (5-10 g of KCl). The major sources of potassium are plant foods such as cereals, pulses, fruits and vegetables. Vegetables such as avocado, broccoli, carrots, peas, lentils, tomatoes, potatoes, sweet potatoes, fruits like apricots, bananas, citrus fruits, milk, dairy products and nuts, meat and fish (clams, halibut, salmon and sardines) are good sources of potassium. Coconut water is a rich source which contains 1400mg of K^+ /liter. Diet can provide around 4 - 7.5 g (50-100 mmol/day) of potassium chloride. Potassium losses from cooking may be significant. Food processing reduces the amount of potassium in many food products, and a diet high in processed foods and low in fresh fruits and vegetables is often lacking in potassium.

Absorption and Excretion

In a normal person 90% of potassium consumed is absorbed by diffusion and is readily distributed in aqueous media throughout the body²⁴. Similar to Na^+ , approximately 80% to 95% of the daily potassium intake is excreted in the urine, and 5% to 20% is excreted in the stool. Renin-angiotensin-aldosterone and cortisol increase urinary loss of potassium. The kidney's ability to conserve potassium is less than that of sodium and serves as the major chronic protective mechanism against toxicity. Even with zero potassium intake urine continues to excrete potassium reducing it to a minimum of 5-20 mmol/day. Ninety to ninety five percent is reabsorbed by the proximal renal tubules and loop of Henle and on a normal dietary intake; potassium is secreted by distal tubules.

Adequate Intakes for Potassium

Potassium content of human milk is greater in early milk (19 mmol/L or 741 mg/L) than mature milk (15 mmol/L or 585 mg/L)²⁵. Based on the National Academy of Sciences, USA "Dietary Reference Intakes for Sodium and Potassium (2019)" the following adequate intakes [AI] have been proposed for potassium for the different age groups. The adequate intake [AI] for potassium is established at 400 mg/day for 0-6 month old infants, while the AI for 7-12 month old infants is 860 mg/day which includes complementary foods²⁶. The AI for 1-3 years is 2000mg/day and for 4-8 years is 2300 mg/day. The AI for 9-13y and 14-18y for male children is fixed at 2500 mg/day and 3000 mg/day, while for females of 9-18y it is 2300 mg/day. For >19y-70y of age males it is 3400mg/day, and for females of the same age group it is 2600 mg/day. In the absence of a specific indicator of potassium adequacy or deficiency, Adequate Intakes were derived using two nationally representative surveys. The highest median potassium intake across the two surveys was selected for each DRI group in children and adolescents, for adult females, and for adult

males. For adults, the data that informed the potassium AIs were from normotensive males and females without a self-reported history of cardiovascular disease. For infants, the AIs were derived from estimates of potassium intakes in breastfed infants. Potassium requirement in pregnant and lactating women has been recommended to be around 4g and 4.4g respectively from a recent German study²⁷. This amount is likely to be supplied by an increase in cereals and pulses intake.

Deficiency and Excess

Low blood potassium levels leads to potassium deficiency also known as ‘hypokalemia’. Normal blood potassium levels are typically in the range of 3.5-5.0 mmol/L. Among the many causes of hypokalemia are diarrhea, chronic kidney disease, folic acid deficiency, excessive sweating and use of diuretics. Potassium deficiency symptoms are often mild and may include nausea, muscle cramps, bloating, constipation and depression. When there is too much potassium in the blood, it is called high potassium, or ‘hyperkalemia’. Increase in blood potassium can be caused by dehydration, kidney disease, uncontrolled diabetes and excessive bleeding.

Potassium intake data from India

Potassium present in vegetarian food is probably enough to meet the daily requirement. WHO recommends an increase in potassium intake in adults from food to reduce blood pressure and risk of cardiovascular disease, stroke and coronary heart disease. WHO suggests a potassium intake of at least 90 mmol/day (3510 mg/day) for adults²⁸. Data with regard to potassium intakes in the Indian population is lacking. The intake of potassium in the NNMB urban population survey in 16 states [2016] and rural population survey in 10 states in India by the dietary recall method was computed. The potassium intakes appear to be much lower than the WHO recommended (3510 mg/day) in both the genders in the adult urban as well as rural populations. This could be attributed to the consumption of lower amounts of vegetables and fruits than the recommended servings/day.

Recommended Intakes for Sodium and Potassium

Hypertension is considered a major risk factor for cardiovascular diseases, particularly coronary heart disease and stroke. Therefore, the burden of morbidity and mortality from hypertension and related non-communicable diseases [NCDs] is currently one of the most urgent public health problems globally. The basis for the current guidelines of WHO for reduction in sodium intake and increase in potassium intakes is to prevent adverse health outcomes, i.e. reduction in blood pressure and thereby reduction in risk of cardiovascular disease, stroke and coronary heart disease in adults. If the WHO guidelines on both sodium and potassium are followed the molar ratio of sodium: potassium would be approximately one to one which is the desirable ratio. Due to lack of systematic balance studies [intake vs excretion] for both sodium and potassium in the normal Indian population, it is proposed to follow the WHO (2012) guidelines for these two electrolytes for the current RDA. The recommended intakes for sodium and potassium for the different physiological groups in the ICMR (2020) based on WHO (2012), are shown below in Table 8.3.1. The sodium and potassium intakes for infants and children were calculated based on the energy requirement [kcal] of a particular group taking the energy requirement of sedentary work for adult man [i.e. 2110 kcal] as the reference [Energy Chapter – RDA 2020].

Table 8.3.1: Recommended intakes of sodium and potassium (mg/d)

Age Group		Molar ratio	Sodium [mg]	Potassium [mg]
Adult [18-60y]	Men	1:1	2000	3500
	Women	1:1	2000	3500
Infants	0-6 m	1:1	500	900
Infants	7-12 m	1:1	650	1100
Children	1-3 y	1:1	1000	1750
	4-6 y	1:1	1300	2250
	7-9 y	1:1	1600	2825

References

1. Gleibermann L. Blood pressure and dietary salt in human populations. *Ecology of food and nutrition.* 1973 Jan 1; 2(2):143-56.
2. Morgano MA, Souza LA, Rondó PH. Mineral composition of human bank milk. *Ciênc Tecnol Aliment.* 2005; 25:819-24.
3. Cook NR, Obarzanek E, Cutler JA, Buring JE, Rexrode KM, Kumanyika SK, Appel LJ, Whelton PK. Joint effects of sodium and potassium intake on subsequent cardiovascular disease: the Trials of Hypertension Prevention follow-up study. *Archives of internal medicine.* 2009 Jan 12; 169(1):32-40.
4. Yang Q, Liu T, Kuklina EV, Flanders WD, Hong Y, Gillespie C, Chang MH, Gwinn M, Dowling N, Khoury MJ, Hu FB. Sodium and potassium intake and mortality among US adults: prospective data from the Third National Health and Nutrition Examination Survey. *Archives of internal medicine.* 2011 Jul 11; 171(13):1183-91.
5. Misra A, Singhal N, Sivakumar B, Bhagat N, Jaiswal A, Khurana L. Nutrition transition in India: Secular trends in dietary intake and their relationship to diet-related non-communicable diseases. *Journal of diabetes.* 2011 Dec; 3(4):278-92.
6. Mozaffarian D, Fahimi S, Singh GM, Micha R, Khatibzadeh S, Engell RE, Lim S, Danaei G, Ezzati M, Powles J. Global sodium consumption and death from cardiovascular causes. *New England Journal of Medicine.* 2014 Aug 14; 371(7):624-34.
7. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, AlMazroa MA, Amann M, Anderson HR, Andrews KG, Aryee M. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *The lancet.* 2012 Dec 15; 380(9859):2224-60.
8. Sarma PS, Sadanandan R, Thulaseedharan JV, Soman B, Srinivasan K, Varma RP, Nair MR, Pradeepkumar AS, Jeemon P, Thankappan KR, Kutty RV. Prevalence of risk factors of non-communicable diseases in Kerala, India: results of a cross-sectional study. *BMJ open.* 2019 Nov 1; 9(11).
9. Mohan B, Verma A, Singh K, Singh K, Sharma S, Bansal R, Tandon R, Goyal A, Singh B, Chhabra ST, Aslam N, Wander GS, Roy A, Prabhakaran D. Prevalence of sustained hypertension and obesity among urban and rural adolescents: a school-based, cross-sectional study in North India. *BMJ Open.* 2019; 8, 9(9):e027134.
10. Smina TP, Kumpatla S, Viswanathan V. Higher dietary salt and inappropriate proportion of macronutrients consumption among people with diabetes and other co morbid conditions in South India: Estimation of salt intake with a formula. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews.* 2019 Sep 1;13(5):2863-8.
11. World Health Organization. Sodium intake for adults and children: guideline. Geneva: World Health Organization. 2012.
12. Johnson C, Santos JA, Sparks E, Raj TS, Mohan S, Garg V, Rogers K, Maulik PK, Prabhakaran D, Neal B, Webster J Sources of Dietary Salt in North and South India Estimated from 24 Hour Dietary Recall. *Nutrients.* 2019; 1; 11(2). pii: E318.
13. World Health Organisation, Draft comprehensive global monitoring framework and targets for the prevention and control of non-communicable diseases. 2013.
14. Johnson C, Mohan S, Rogers K, Shivashankar R, Thout SR, Gupta P, He FJ, MacGregor GA, Webster J, Krishnan A, Maulik PK. Mean dietary salt intake in urban and rural areas in India: a population survey of 1395 persons. *Journal of the American Heart Association.* 2017 Jan 6; 6(1):e004547.
15. Ravi S, Bermudez OI, Harivanjan V, Chui KH, Vasudevan P, Must A, Thanikachalam S, Thanikachalam M. Sodium intake, blood pressure, and dietary sources of sodium in an adult South Indian population. *Annals of global health.* 2016 Mar 1;82(2):234-42.

16. Radhika G, Sathya RM, Sudha V, Ganesan A, Mohan V. Dietary salt intake and hypertension in an urban south Indian population-[CURES-53]. *Journal of Association of Physicians of India*. 2007 Jun 1; 55(6):405-11.
17. Smina TP, Kumpatla S, Viswanathan V. Higher dietary salt and inappropriate proportion of macronutrients consumption among people with diabetes and other co morbid conditions in South India: Estimation of salt intake with a formula. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*. 2019 Sep 1; 13(5):2863-8.
18. Nair S, Bandyopadhyay S. Sodium intake pattern in West Indian population. *Indian journal of community medicine: official publication of Indian Association of Preventive & Social Medicine*. 2018 Apr; 43(2):67.
19. Bhansali A, Dhandania VK, Deepa M, Anjana RM, Joshi SR, Joshi PP, Madhu SV, Rao PV, Subashini R, Sudha V, Unnikrishnan R. Prevalence of and risk factors for hypertension in urban and rural India: the ICMR-INDIAB study. *Journal of human hypertension*. 2015 Mar; 29(3):204-9.
20. Genovesi S, Antolini L, Orlando A, Brahmochary S, De Servi A, Capelli S, Giussani M, Nava E, Agostoni C, Gallieni M. Poor early growth and high salt intake in Indian infants. *International journal of food sciences and nutrition*. 2017 May 19; 68(4):467-72.
21. Johnson C, Santos JA, Sparks E, Raj TS, Mohan S, Garg V, Rogers K, Maulik PK, Prabhakaran D, Neal B, Webster J. Sources of Dietary Salt in North and South India Estimated from 24 Hour Dietary Recall. *Nutrients*. 2019 Feb 1; 11(2). pii: E318.
22. Aparna P, Salve HR, Anand K, Ramakrishnan L, Gupta SK, Nongkynrih B. Knowledge and behaviors related to dietary salt and sources of dietary sodium in north India. *Journal of Family Medicine and Primary Care*. 2019 Mar; 8(3):846.
23. Stamler J. The INTERSALT Study: background, methods, findings, and implications. *The American journal of clinical nutrition*. 1997 Feb 1; 65(2):626S-42S.
24. Agarwal R, Afzalpurkar R, Fordtran JS. Pathophysiology of potassium absorption and secretion by the human intestine. *Gastroenterology*. 1994; 107(2):548-571.
25. Wack RP, Lien EL, Taft D, Roscelli JD. Electrolyte composition of human breast milk beyond the early postpartum period. *Nutrition*. 1997; 13(9):774-777.
26. National Academies of Sciences, Engineering, and Medicine. Dietary Reference Intakes for sodium and potassium. National Academies Press; 2019 Aug 26.
27. Strohm D, Ellinger S, Leschik-Bonnet E, Maretzke F, Heseker H. Revised reference values for potassium intake. *Annals of Nutrition and Metabolism*. 2017; 71(1-2):118-24.
28. Guideline WH. Potassium intake for adults and children. Geneva: World Health Organization (WHO). 2012; 48.

9. IRON

INTRODUCTION

Iron is an essential micronutrient required as a component for number of proteins, including enzymes and haemoglobin. The latter is important for transport of oxygen to tissues throughout the body for metabolism. It is also required for many critical functions in human body such as – energy metabolism, mixed function oxidase systems, neurodevelopment, connective tissue synthesis and hormone synthesis.

The iron content of the body is tightly regulated and in absence of bleeding through menstruation and during pregnancy, only small amount of iron is lost from the body. Therefore, the physiological requirement is relatively low, particularly for men. The dietary requirement depends on the bioavailability of iron from the diet, and Indian diets typically are inhibitory to the absorption of iron. Thus, the bioavailability of iron through non-heme sources can increase the requirement for iron.

9.1 Iron deficiency anemia (IDA) and its prevalence

Anemia is a serious public health problem in India, affecting all segments of the population (50-70%), especially infants and young children, adolescent boys and girls, women of childbearing age and pregnant women. The national surveys conducted in India known as National Family Health Survey (3 and 4) shows that there is continuing high prevalence of anemia among Indians^{1,2} (Table 9.1). Despite supplementation programs being in operation for decades, there has been no perceptible decrease in the prevalence of anemia. Its prevalence is similar in both urban and rural areas; however, gender differences exist from the age of 15 years, as females become more vulnerable to this malady.

It has been thought that iron deficiency in the diet is the main cause of anemia in all age groups³⁻⁶. However, in the recent analysis by Ghosh *et al*⁷ showed that the median risk of inadequate intake of iron was in the range of 48-78% concluding that the risk of inadequate intake of iron among Indians is lower than previously thought. It was also emphasized that simultaneous implementation of supplementation and fortification program can increase the risk of increasing tolerable upper limit (TUL; 45 mg/d) for iron⁷. The iron deficiency is often seen to coexist with other causes of anemia in India. In an urban slum, 75% of children suffering from anemia were seen to respond to iron administration and 22% of anemic children also had biochemical vitamin B₁₂ deficiency¹. In pregnant women, studies with serum transferrin receptor or TfR which is used as a marker of tissue iron deficiency⁸⁻¹⁰ suggested low iron stores and widespread iron deficiency. A study carried out by NIN in semi-urban school children showed prevalence of multiple sub-clinical micronutrient deficiencies¹¹.

9.2 Changes from the ICMR 2010 recommendation for Iron requirement:

The following changes were made:

- a. The requirement provided by previous committee was the recommended dietary allowance (RDA), which by definition is the 97.5th percentile of the distribution of requirements, and is specifically meant to define the requirement of an individual, where the risk of an inadequate intake is low. However, for public health and the definition of the risk of dietary inadequacy of iron in a population, the estimated average requirement (EAR) is required. Using the RDA for this purpose will overestimate the risk of deficiency. Further, the EAR is used in formulating food fortification and supplementation guidelines¹².

Table 9.1: Prevalence (%) of anemia among Indians^{1,2}

Category/ anemia prevalence	NFHS 3				NFHS 4			
	Mild	Moderate	Severe	Any anemia	Mild	Moderate	Severe	Any anemia
Adult men	13.5	10.3	1.3	25.1	12.4	10.0	1.1	23.5
WRA (15-49y)	38.6	14.8	1.8	55.3	39.8	12.3	1.1	53.2
Pregnant women	25.8	30.6	2.2	58.7	24.5	24.6	1.3	50.4
Lactating women	44.8	16.6	1.7	63.2	44.5	12.6	0.9	58.0
Adolescent boys (15-19y)	16.7	12.1	1.4	30.2	15.5	12.5	1.2	29.2
Adolescent girls (15-19y)	39.1	14.9	1.7	55.8	41.2	11.9	1.0	54.1
Children								
6-11m	27.6	51.1	2.0	80.7	29.1	37.7	1.7	68.5
12-23m	23.9	54.7	4.5	83.0	27.5	40.3	2.6	70.5
24-35m	26.6	44.1	3.9	74.6	28.6	31.9	1.8	62.3
36-47m	27.3	33.1	2.7	63.0	28.1	23.2	1.1	52.3
48-59m	26.9	24.9	1.2	53.0	26.4	17.6	0.7	44.7

WRA – Women of reproductive age

- b. The factorial approach to determine the requirement requires the mean value of each factor, as well as its variation. The basal loss considered in the previous recommendation was based on the mean iron loss per kg body weight, but did not consider the variance of the iron loss, nor of the body weights of different age groups of Indians. In the present recommendation, the variance of iron loss¹³ was combined with the variability in the body weights for Indians.
- c. As described below in section 9.3.1, the distribution of iron losses due to menstruation was positively skewed; therefore, simple addition of this factor and its variance, within the factorial method is not possible. In this situation, Monte Carlo simulations were applied to obtain an approximated distribution of iron requirement. Further, the estimate for iron loss in menstruation was not clearly specified: this is now presented.

9.3 Iron requirement

The daily physiological requirement for iron was calculated using factorial method, sums the components of iron loss from the body, for all the age groups. This physiological requirement was then adjusted to iron bioavailability to provide EAR. The iron requirement for different age group is as follows:

9.3.1 Adult

The requirement for iron is different for males and females as latter has additional iron loss through menstruation.

Males

In adult males, the daily iron is required to replace the basal iron lost through desquamated gastrointestinal cells, bile, urine and sweat. The distribution of basal iron loss was derived from the data reported on adult males from Seattle, Venezuela and South African Indians¹³. The average basal loss from the study was 14 µg/kg body weight/d with CV of 29.2%. The distribution of standard body weight for adult male was derived from NNMB urban survey (2016)¹⁴. The mean

body weight of adult male was reported as 65 kg with SD 4.3 kg. Assuming a symmetric shape of the distribution of iron requirement, the mean and SD of the distribution were derived by the product of mean body weight and mean basal loss per kg and the square root of combined variance of the product of body weight and basal loss per kg, respectively. Further, the physiological requirement for iron was adjusted for iron absorption. While a study conducted on adult men in 1983 reported iron absorption of 5% from a cereal based diet¹⁵, a more extensive analysis of data from several recent studies in women (see below), indicated an absorption of 8% from similar cereal-based diets. There is no strong evidence suggesting that iron absorption in men will be lower than females, when both are iron replete. Therefore, the present EAR was adjusted for 8% iron absorption. The EAR of iron for adult Indian male was estimated to be 11.4 mg/d and RDA as 18.8 mg/d (Table 9.6).

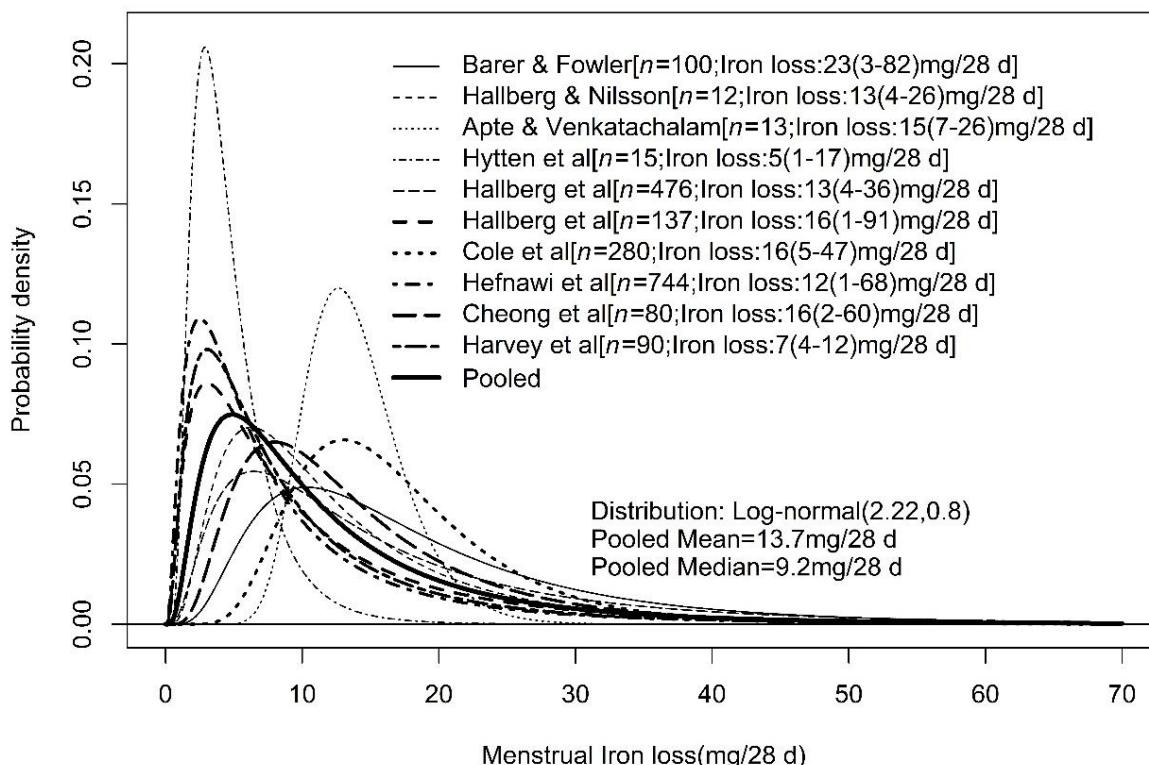
Females

Ghosh *et al*⁷ have recently analyzed the requirement for iron in WRA. For non-pregnant, non-lactating WRA, components of iron loss are the daily basal iron loss and menstrual loss of iron. This summation uses the average value for each factor and yields the daily estimated average physiological requirement for iron. The EAR is then derived as the ratio of the physiological requirement and the bioavailability of iron from the diet.

First, the distribution of the basal loss of iron was derived from the data reported on adult males from Seattle, Venezuela and South African Indians¹³. The average basal iron loss as a relation to the body weight was 14 µg/kg body weight/d with coefficient of variation (CV) 29.2%. Since data were not available for WRA, the basal iron loss reported for adult male was considered for this population. This is a reasonable assumption as there is no evidence to conclude that basal iron losses are different in women. To estimate variability in basal loss in WRA, the CV of basal loss per kg body weight was combined with the average CV of body weight in WRA between 18-49 years of age which was estimated to be 15.6%, from national anthropometric data², by variance of the product of two random variables assuming independence. For the reference body weight for WRA of 55 kg, an average daily iron loss of 0.77±0.25 (SD) mg/d. The probability distribution of basal loss was assumed to be normal.

Second, for the distribution of iron losses due to menstruation, studies reporting menstrual blood loss or iron loss were considered¹⁶⁻²⁵. There was only one study conducted in Indian women, therefore all relevant studies were included in the calculation of menstrual iron loss. Where blood loss was reported, the iron loss was derived as the product of the daily blood loss assuming an average of 28 days in a cycle²⁶, the Hb concentration taken as 135 g/L unless otherwise reported²⁷ and the iron content of Hb taken as 3.39 mg/g²⁸. In a validation of this approach, within the studies that reported iron loss, the mean bias of the calculated iron loss was only 0.05 mg/d. From the extracted literature, only one study was conducted on Indian WRA and due to limited data, all relevant studies were included in the analysis. Since the distribution of the menstrual iron loss appeared to be positively skewed in many studies, its probability distribution was taken as lognormal. Ghosh *et al*⁷ has reported the detailed calculation for estimating iron loss due to menstruation (Figure 9.1). Briefly, the mean and variance at the log scale were estimated from reported dispersion for each study. The reported range from the study was assumed to be from 0.5th to 99.5th percentile. The pooled estimates of mean and standard deviation were finally derived as weighted (based on the sample size) mean of the estimates obtained from all the studies.

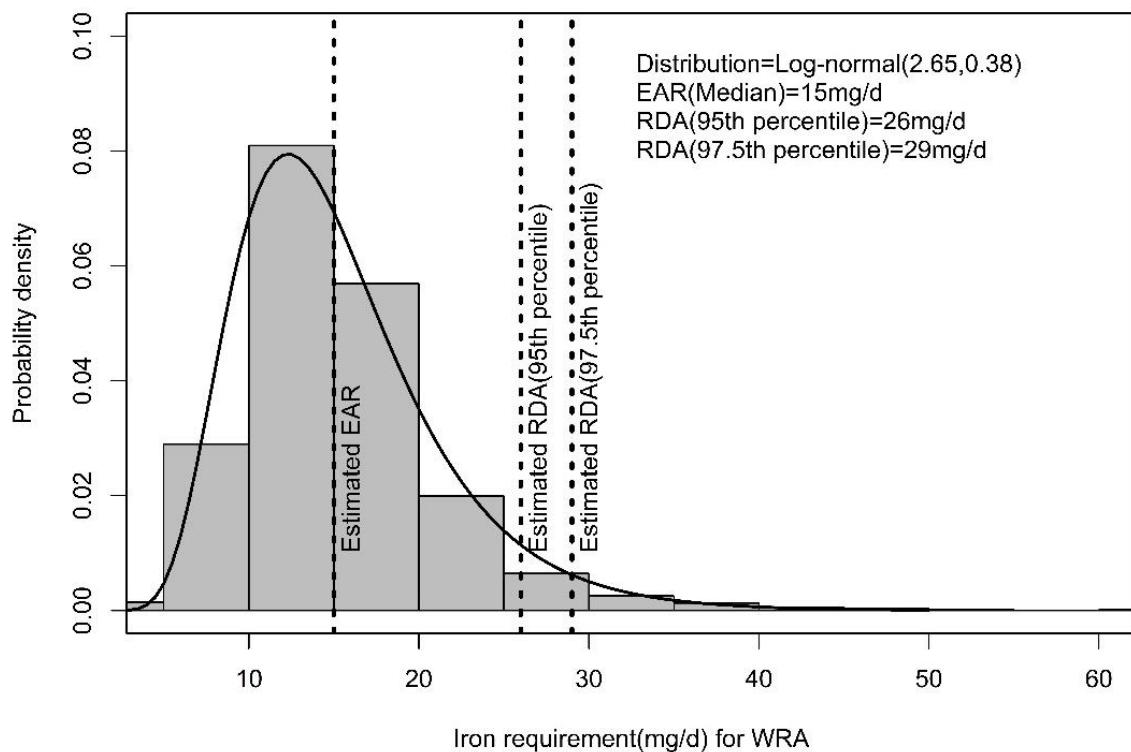
Figure 9.1: Estimated distribution of menstrual iron loss from different studies
[Adapted methodology from Ghosh *et al*⁷ and modified]



Third, the absorption of dietary iron from different cereal-based Indian meals was determined from published reports on Indian WRA and adolescent girls²⁹⁻³². The iron absorption from different cereal-based Indian meals was considered as the National Sample Survey Organization (NSSO) 68th round showed that nearly 70% of the iron consumed is from cereals and only about 1% from heme sources³³. The studies selected have used accurate iron absorption methods, which measured the incorporation of a stable isotope of iron provided in a common pool of iron from a meal into hemoglobin, and reported the mean and SD of iron absorption in anemic and normal WRA. An average absorption was calculated using weighted inverse of standard error of each study, the proportion of anemic and normal WRA in survey data², and the proportion consumption in weight of rice, wheat and millet in the total cereal intake in the Indian population³³. The average absorption of dietary iron was 8.7% which is similar to the value of 8%, used for WRA previous expert committee¹², and this value was used to adjust the physiological requirement of iron to obtain the EAR.

The distribution of iron requirements was obtained by convolution of the probability distribution of daily basal and menstrual iron loss. As no close form of the convolution of lognormal and normal distribution exists, Monte Carlo simulations were performed to obtain an approximated distribution of iron requirement. Finally, the median and 95th or 97.5th percentile were derived from estimated iron requirement distributions to represent the physiological EAR and RDA. These values were corrected for a dietary iron absorption of 8% to yield an EAR of 15 mg/d and RDA of 29 mg/d for the 97.5th percentile (Figure 9.2; Table 9.6).

Figure 9.2: EAR and RDA of iron for Indian WRA
 [Methodology adapted from Ghosh *et al*⁷ and modified]



Risk of inadequate dietary iron intake in Indian WRA

In order to define the risk of dietary inadequacy of iron, EAR is applied (for details, refer chapter-2; section 2.3) to the distribution of iron intake in the population. Either probability approach or EAR cut-point method can be used to assess dietary iron inadequacy. The use of RDA in estimating inadequate intake of iron in population is inappropriate as RDA is the intake level that exceeds the requirements of the large population and meant to define the requirement of an individual.

Ghosh *et al*⁷ have recently analyzed the risk of dietary iron inadequacy among Indian WRA. To estimate the risk of inadequate intake of iron among WRA, nationally representative survey data from NSSO were considered³³. The quantities of different foods purchased by a household were converted to nutrients of interest using the Indian food composition tables³⁴ and adjusted for the number of members in the household, to obtain the daily per capita iron intake. The maximum likelihood estimation (MLE) technique was applied to estimate the appropriate parametric distribution of usual intake of dietary iron for each state and union territory. The distribution of population risk of inadequate intake of iron was derived by using the probability approach, using the EAR of 15 mg/d⁷. The interquartile range of the risk lay between approximately 48% to 78%.

Validity of factorial method to estimate EAR in WRA

To validate the present estimate of EAR, the risk of inadequate dietary iron was estimated by the probability approach and the EAR estimated here, and compared with biomarker-based (α -glycoprotein and C-reactive protein-adjusted serum ferritin) measurements of iron deficiency in WRA. In a recently conducted survey in a third of the districts in Uttar Pradesh (UP)³⁵, the prevalence of iron deficiency was 51% in WRA, which compared well with the present new estimate of the risk of dietary iron inadequacy of 42% in all districts in UP⁷.

9.3.2 Pregnancy

Iron requirements during pregnancy were calculated using factorial method. During pregnancy, iron is required to meet the basal iron loss, iron deposited in fetus and related tissues and iron utilized in expansion in hemoglobin mass. Since there is no menstrual blood loss, the iron loss due to menstruation was not included.

Table 9.2: Additional iron requirement during pregnancy

Compartments	Iron requirement during entire pregnancy (mg)	
	10 kg GWG	12 kg GWG
Foetus	179.3±43.6 mg	179.3±43.6 mg
Expansion of maternal red cell mass	246.1±14.3 mg	295.3±17.2 mg
Total	425±45.9 mg	474.6±46.9 mg

GWG - Gestational Weight Gain

Blood lost during parturition considered to be taken care of by the contraction of maternal red cell mass and therefore not accounted for.

Based on the available data^{36,37} for Indian women, the additional iron required during the entire pregnancy period (includes requirement for fetus + expansion of maternal red cell mass) for Indian women having a pre-pregnancy body weight of 55 kg (CV=15.6%) and considering a gestational weight gain of 10 and 12 kg is provided in Table 9.2. A detailed derivation is given in Table 9.3. Total iron required per day during the three trimesters is given in Table 9.4.

Table 9.3: Iron requirement during pregnancy and for trimesters: basis and detailed calculation

Trimester	Mid-trimester weight gain (kg)	Body weight ^a (kg)	Basal loss ^b (mg/d)	Blood volume expansion ^c (mg/d)	Fetal growth ^d (mg/d)	Total (mg/d)
For 10 kg total weight gain (CV of weight=5.8%)						
First	1.8	56.6	0.79	0.88	0.64	2.31
Second	5.3	60.3	0.84	0.88	0.64	2.36
Third	8.6	63.6	0.89	0.88	0.64	2.41
For 12 kg total weight gain (CV of weight=5.8%)						
First	2.1	57.1	0.80	1.05	0.64	2.49
Second	6.3	61.3	0.86	1.05	0.64	2.55
Third	10.4	65.4	0.91	1.05	0.64	2.61

^aPre-pregnancy weight 55 kg

^bBasal loss – 14 µg/kg

^cBlood volume expansion: at the rate of 66ml / kg weight gain, Hb concentration 110 g/L and iron content of 3.47 mg/ g of Hb

^dBased on the reported fetal iron³⁵

9.2.3 Lactation

Iron requirement during lactation is the sum of the requirement of the mother and that required for making up the iron lost in breast milk. Since there is amenorrhea during lactation, only requirement beyond loss due to breast milk is due to the basal loss, which will be same as in the adult woman, i.e., 14 µg/kg/d.

According to a study conducted in India^{38,39}, iron content of breast milk from the study was 14 µmoles/L or 0.78 mg/L. Assuming average milk volume is around 700 ml/d, the amount of iron needed works out to be around 0.5 mg/d.

The iron content of breast milk was combined with basal iron loss from WRA to arrive at physiological requirement for iron. This requirement was then adjusted to 8% iron absorption to arrive at an EAR of 15.97 mg/d (Table 9.6).

9.3.4 Infants and children

The estimates of iron requirement for children and also for adolescent was analyzed by Ghosh *et al*⁴⁰. The physiological requirement for iron during infant and children was estimated using a factorial method. It was based on a) replacing the basal iron lost from the body and b) providing additional iron required for growth and storage (for increased mass of hemoglobin, non-storage tissue iron deposition and iron storage). The addition of mean values of these factors yielded the average physiological requirement of iron that must be absorbed into the body. The EAR was then derived after adjusting the physiological requirement for the bioavailability of dietary iron. To derive the RDA, the variance of each of the factors noted above were also estimated to provide a summed variance that allowed for the determination of the 97.5th percentile of the distribution of requirements.

The distribution of basal loss was assessed. The weight standardized basal iron loss reported in adult men from Seattle, Venezuela and South African Indians was used¹³, since data on basal iron loss were not available for children. There is no reason to consider that iron losses, standardized for body weight, would be different in children. The mean basal loss of iron was 14 µg/kg body weight/d with a standard deviation (SD) of 4.1 µg/kg body weight/d.

Second, the increase in Hb mass was calculated as the product of the annual increase in blood volume, Hb concentration and the iron content of Hb. To estimate the increment of blood volume, sex- and age-specific blood volume data were extracted for children from earlier studies^{41,42}, and regressed on age, ignoring sex for children <10y, since sexual dimorphism before adolescence is likely to be low. The specific average requirement of iron due to the increase in blood volume for any given age was derived by multiplying it into the minimum required Hb concentration at that age to be classified as non-anemic, and the iron content in Hb. The minimum Hb concentration was taken as provided in Table 9.5 and the iron content of Hb was assumed to be 3.39 mg/g²⁸.

Third, the non-storage tissue iron was calculated as the product of weight gain/d and iron deposited within that weight gain. The mean weight gain/d and its variance were estimated from

Table 9.4: Iron requirement during pregnancy (trimester-wise)

Trimester	Requirement (mg/d)			
	10 kg GWG		12 kg GWG	
	EAR	RDA	EAR	RDA
First	28.9	36.5	31.2	38.9
Second	29.5	37.5	31.9	40.0
Third	30.1	38.4	32.6	41.1

Table 9.5: Optimum haemoglobin concentration for different age and gender groups⁴³

Group	Haemoglobin g/L
Children 0.5-5 y	>110
Children aged 5-11 y	>115
Children aged 12-13 y	>120

annual body weights as described above and converting the yearly change of weight (mean and variance) to a daily change. The value for non-storage tissue iron deposition in the weight gain, for children up to 9y, was assumed to be 5 mg/kg. This was calculated as the product of the total body iron content (38 mg/kg) in children aged 1-9y⁴⁴ and the proportion of this total body iron content, which is non-storage content, and assumed to be 14%⁴⁵.

Fourth, the mean daily requirement for storage iron for children and adolescents was calculated based on the assumption that 12% of the increment in tissue iron deposition and Hb mass enters into body storage²⁷.

The values for each factor were summed to arrive at the distribution of the physiological requirement. Finally, estimates for the EAR in young children and adolescent boys were obtained by adjusting this value for a bioavailability from cereal-based meals, of 15% for infants (6-12 months) and 6% for children aged 1-9y⁴⁶. The physiological requirement, EAR and RDA is provided in Table 9.6.

9.3.5 Adolescents

In case of adolescents, the iron requirement was calculated as described for children. However, for adolescent girls, additional iron lost due to blood loss in menstruation was considered.

First, for younger children, the basal iron loss was estimated from weight standardized basal iron loss reported in adult men from Seattle, Venezuela and South African Indians¹³.

Second, the increase in Hb mass was calculated as the product of the annual increase in blood volume, Hb concentration and the iron content of Hb. To estimate the increment of blood volume, sex- and age-specific blood volume data were extracted for children from earlier studies^{41,42}, and regressed on age. For adolescents, sex-specific regression coefficients were estimated and applied in subsequent equations. The specific average requirement of iron due to the increase in blood volume for any given age was derived by multiplying it into the minimum required Hb concentration at that age to be classified as non-anemic, and the iron content in Hb. The minimum Hb concentration was taken as provided in Table 9.5 and the iron content of Hb was assumed to be 3.39 mg/g²⁸.

Third, the non-storage tissue iron was calculated as the product of weight gain/d and iron deposited within that weight gain. The mean weight gain/d and its variance were estimated from annual body weights as described above and converting the yearly change of weight (mean and variance) to a daily change. From 10y onward, since muscle development is significant, the muscle content of iron was taken to represent non-storage tissue iron deposition (as myoglobin). This has been found to be 26 mg/kg and independent of the dietary iron intake⁴⁷. It was assumed that 40% of the increment in body weight is due to skeletal muscle growth⁴⁸, since there is no evidence to suggest that in normal children, the proportion of muscle mass in the body is lower than elsewhere in the world. A value of 10 mg/kg body weight increase was assumed for non-storage tissue iron deposition in adolescents.

Fourth, the mean daily requirement for storage iron for children and adolescents was calculated based on the assumption that 12% of the increment in tissue iron deposition and Hb mass enters into body storage²⁷.

Finally, for adolescent girls, additional iron is required to replace that lost during menstruation. Studies reporting blood and iron loss during menstruation among women (15-50y) were included in this estimation, as it has been concluded that menstrual blood loss during adolescence does not differ from that of WRA group⁴⁹. Iron loss was calculated as the product of the daily blood loss over a 28d cycle, the hemoglobin (Hb) concentration and the iron content of

Hb. Details on the calculation are provided in section 9.3.1. Since the menstrual iron loss distribution was positively skewed, its probability distribution was taken as lognormal (see section 9.3.1 for details).

The values for each factor were summed to arrive at the distribution of the physiological requirement. For adolescent girls, in whom the menstrual iron loss was positively skewed, a convolution of normal and log-normal was obtained as the probability distribution of the physiological requirement. As there is no close form available for this convolution, a Monte-Carlo simulation technique was applied to obtain the distribution of the total physiological iron requirement.

Estimates for the EAR in adolescent were obtained by adjusting the physiological iron requirement for diet bioavailability from cereal-based meals of 8% for adolescent children aged 10-17y³². The RDA (97.5th percentile) was estimated from the pooled variance of the factors, assuming a normal distribution. The physiological requirement, EAR and RDA for adolescent is presented in Table 9.6.

Validity of factorial method to estimate EAR in children and adolescents

The current estimate of EAR for children and adolescents were validated by estimating the risk of inadequate dietary iron was estimated using current EAR and compared with biomarker-based (α -glycoprotein and C-reactive protein-adjusted serum ferritin) measurements of iron deficiency in children 1-3y. The risk of dietary iron inadequacy was determined to be 49%. In another recent study on <5y old children in 25 districts of UP³⁵, the prevalence of iron deficiency (based on AGP and CRP corrected serum ferritin levels), was 62%, which is reasonably close to the calculated prevalence of risk of dietary inadequacy (49%) in a similar population. Similarly, the prevalence of risk of dietary iron inadequacy was 54% for adolescent girls from UP, while the prevalence of iron deficiency based on serum ferritin was 41% (in UP and Bihar girls residing in the slums of Delhi)⁵⁰. Also, the sensitivity analyses were also performed to evaluate differences in the estimate of the physiological iron requirement and the EAR when the assumptions used were varied by 10%.

9.4 Tolerable Upper Level

The Tolerable Upper Intake Level (TUL) is the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects for almost all individuals (refer chapter-2 for details). Acute toxicity can occur due to overdose of supplemental iron and symptoms may include gastric upset, constipation, nausea, abdominal pain and vomiting^{27,54}. These symptoms can be observed with the doses between 20 and 60 mg/kg. Many studies have been done on children^{55,56} and pregnant women⁵⁷ who reported adverse effects of iron supplementation. The studies showed no gastro-intestinal (GI) side effects with supplementation of 20 to 30 mg/d iron in children, while 100 to 200 mg/d in pregnant women was associated with GI side effects such as abdominal pain, severe nausea, vomiting, constipation and diarrhoea. High intakes of iron supplements have also been associated with reduced zinc absorption⁵⁸. In severe cases (e.g., one-time ingestions of 60 mg/kg), overdoses of iron can lead to multisystem organ failure, coma, convulsions, and even death⁵⁹. Most of the studies reporting adverse events failed to have a control group. According to Institute of Medicine (IOM), the TUL was based on the level at which lowest adverse events were observed (60 mg/d)²⁷ and an extrapolation was done using uncertainty factor of 1.5 to arrive at level which shows no adverse effect. In case of infants and children, this uncertainty factor was 1. Using 60 mg/d level and an uncertainty factor, the TUL was set at 40 mg/d for infants and children up to 13 y, while for adolescents (14-18 y) and adults, the TUL was set at 45 mg/d (Table 9.7).

Table 9.6: Current EAR and RDA of iron for various physiological groups

Category / Age		Body weight ^a (kg)	Physiological Iron Requirement (mg)	Absorption assumed %	ICMR 2020 ^b	
					EAR (mg/d)	RDA (mg/d)
Men		65.0	0.91	8	11	19
Women (WRA)		55.0	1.20	8	15	29
Pregnant women		55.0 ^c	2.55	8	30	38
Lactating women (0-6m)		55.0	1.28	8	16	23
Infants	0-6 m	5.4	--	--	--	--
	6-12 m	8.5	0.33	15 ^d	2	3
Children	1-3 y	11.7	0.38	6	6	8
	4-6 y	18.3	0.49	6	8	11
	7-9 y	25.3	0.61	6	10	15
Adolescents						
Boys	10-12 y	34.9	0.93	8	12	16
Girls	10-12 y	36.4	1.27	8	16	28
Boys	13-15 y	50.5	1.23	8	15	22
Girls	13-15 y	49.6	1.39	8	17	30
Boys	16-18 y	64.4	1.45	8	18	26
Girls	16-18 y	55.7	1.46	8	18	32

^aReference body weights for Indians in various physiological groups^{14,51,52}

^bEAR and RDA are rounded off to whole number;

^cPre-pregnancy weight; EAR and RDA is given as average of all 3 trimester for a woman having 12kg GWG (refer Table 9.4 for details);

^dIron absorption reported for infants was 15%⁵³.

Table 9.7: Summary of EAR and RDA for Iron

Age category	Body weight kg	EAR mg/day	RDA mg/day	TUL mg/day
Adult Men	65.0	11	19	45
Adult Women	55.0	15	29	45
Pregnant	55.0 ^a	32	40	45
Lactating	55.0	16	23	45
Infants (0-6 mo)	5.4	--	--	40
Infants (6-12 mo)	8.5	2 (AI)	3 (AI)	40
Children (1-3y)	12.9	6	8	40
Children (4-6y)	18.3	8	11	40
Children (7-9y)	25.3	10	15	40
Boys (10-12y)	34.9	12	16	40
Girls (10-12y)	36.4	16	28	40
Boys (13-15y)	50.5	15	22	45
Girls (13-15y)	49.6	17	30	45
Boys (16-18y)	64.4	18	26	45
Girls (16-18y)	55.7	18	32	45

^aPre-pregnancy weight; EAR and RDA is given as average of all 3 trimester for a woman having 12kg GWG (refer Table 9.4 for details)

References

1. International Institute for Population Sciences. National Family Health Survey (NFHS-3), 2005-06: India. International Institute for Population Sciences; 2007.
2. International Institute of Population Science. National Family Health Survey India (NFHS-4), 2015-2016: International Institute for Population Sciences; 2016.
3. Gomber S, Kumar S, Rusia U, Gupta P. Prevalence & etiology of nutritional anaemias in early childhood in an urban slum. Indian Journal of Medical Research. 1998 Jun 1; 107:269.
4. Verma M, Chhatwal J, Kaur G. Prevalence of anemia among urban school children of Punjab. Indian Pediatrics. 1998 Dec; 35(12):1181-6.
5. Raman L, Pawashe AB, Ramalakshmi BA. Iron nutritional status of preschool children. The Indian Journal of Pediatrics. 1992 Mar 1; 59(2):209-12.
6. Vasanthi G, Fawashe AB, Susie H, Sujatha T, Raman L. Iron nutritional status of adolescent girls from rural area and urban slum. Hemoglobin (g/dl). 1994 Feb 1; 12(2.25):13-0.
7. Ghosh S, Sinha S, Thomas T, Sachdev HS, Kurpad AV. Revisiting dietary iron requirement and deficiency in Indian women: Implications for food iron fortification and supplementation. The Journal of nutrition. 2019 Mar 1; 149(3):366-71.
8. Bhaskaram P, Nair KM, Balakrishna N, Ravinder P, Sesikeran B. Serum transferrin receptor in children with respiratory infections. European journal of clinical nutrition. 2003 Jan; 57(1):75-80.
9. Nair KM, Bhaskaram P, Balakrishna N, Ravinder P, Sesikeran B. Response of haemoglobin, serum ferritin and transferrin receptor during iron supplementation in pregnancy. Nutrition 2004; 20:896–899.
10. Rusia U, Flowers C, Madan N, Agarwal N, Sood SK, Sikka M. Serum transferrin receptors in detection of iron deficiency in pregnancy. Annals of Hematology. 1999 Aug 1; 78(8):358-63.
11. Sivakumar B, Nair KM, Sreeramulu D, Suryanarayana P, Ravinder P, Shatrugna V, Kumar PA, Raghunath M, Rao VV, Balakrishna N, Kumar PU. Effect of micronutrient supplement on health and nutritional status of schoolchildren: biochemical status. Nutrition. 2006 Jan 1; 22(1):S15-25.
12. ICMR, Nutrient requirements and recommended dietary allowances for Indians. A report of the expert group of the Indian Council of Medical Research National Institute of Nutrition Hyderabad, 2010.
13. Green R, Charlton R, Seftel H, Bothwell T, Mayet F, Adams B, Finch C, Layrisse M. Body iron excretion in man: a collaborative study. The American journal of medicine. 1968 Sep 1; 45(3):336-53.
14. NNMB. (NNMB). Diet and nutritional status of urban population in India and prevalence of obesity, hypertension, diabetes and hyperlipidemia in urban men and women. Technical Report No. 27, National Nutrition Monitoring Bureau, National Institute of Nutrition, ICMR, Hyderabad, 2016.
15. Rao N, Vijayasarathy C, Prabhavathi T. Iron absorption from habitual diets of Indians studied by the extrinsic tag technique. Indian journal of medical research. 1983.
16. Barer AP, Fowler WM. The blood loss during normal menstruation. American Journal of Obstetrics and Gynecology. 1936 Jun 1; 31(6):979-86.
17. Hallberg L, Nilsson L. Determination of menstrual blood loss. Scandinavian Journal of Clinical and Laboratory Investigation. 1964 Jan 1; 16(2):244-8.
18. Apte SV, Venkatachalam PS. Iron losses in Indian women. Indian journal of Medical research. 1963; 51:958-62.
19. Hytten FE, Cheyne GA, Klopper AI. Iron loss at menstruation. BJOG: An International Journal of Obstetrics & Gynaecology. 1964 Apr; 71(2):255-9.
20. Hallberg L, Högdahl AM, Nilsson L, Rybo G. Menstrual blood loss—a population study: variation at different ages and attempts to define normality. Acta obstetricia et gynecologica Scandinavica. 1966 Jan 1;45(3):320-51.

21. Hallberg L, Hogdahl AM, Nilsson L, Rybo G. Menstrual blood loss and iron deficiency. *Acta medica Scandinavica*. 1966; 180:639-50.
22. Cole SK, Billewicz WZ, Thomson AM. Sources of variation in menstrual blood loss. *The Journal of Obstetrics and Gynecology of British Commonwealth*. 1971; 75:933-9.
23. Hefnawi F, El-Zayat AF, Yacout MM. Physiologic Studies of Menstrual Blood Loss. *International Journal of Gynecology and Obstetrics*. 1980; 17:343-52.
24. Cheong RL, Kuizon MD, Tajaon RT. Menstrual blood loss and iron nutrition in Filipino women. *Southeast Asian Journal of Tropical Medicine and Public Health* 1991; 22(4):595-604.
25. Harvey LJ, Armah CN, Dainty JR, Foxall RJ, Lewis DJ, Langford NJ, Fairweather-Tait SJ. Impact of menstrual blood loss and diet on iron deficiency among women in the UK. *British Journal of Nutrition*. 2005; 94(04):557-64.
26. Beaton GH, Thein M, Milne H, Veen MJ. Iron requirements of menstruating women. *American Journal of Clinical Nutrition*. 1970; 23:275-83.
27. Food and Nutrition Board. Dietary reference intakes for vitamin A vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc: A report of the Panel on Micronutrients. Washington, DC: The National Academies Press; Institute of Medicine; 2001.
28. Smith NJ, Rios E. Iron metabolism and iron deficiency in infancy and childhood. *Advances in Pediatrics*. 1974; 21:239-280.
29. Thankachan P, Walczyk T, Muthayya S, Kurpad AV, Hurrell RF. Iron absorption in young Indian women: the interaction of iron status with the influence of tea and ascorbic acid. *American Journal of Clinical Nutrition*. 2008; 87(4):881-6.
30. Kalasuramath S, Kurpad AV, Thankachan P. Effect of iron status on iron absorption in different habitual meals in young south Indian women. *Indian Journal Medical Research*. 2013; 137(2):324.
31. Herter-Aeberli I, Thankachan P, Bose B, Kurpad AV. Increased risk of iron deficiency and reduced iron absorption but no difference in zinc, vitamin A or B-vitamin status in obese women in India. *European Journal of Nutrition*. 2016; 55(8):2411-21.
32. Nair KM, Brahmam GN, Radhika MS, Dripta RC, Ravinder P, Balakrishna N, Chen Z, Hawthorne KM, Abrams SA. Inclusion of Guava Enhances Non-Heme Iron Bioavailability but Not Fractional Zinc Absorption from a Rice-Based Meal in Adolescents. *Journal of Nutrition*. 2013; 143(6):852-8.
33. National Sample Survey Office. Nutritional Intake in India, 2011-12. 560, NSS 68th Round. National Statistical Organization. Government of India. 2014.
34. Longvah T, Ananthan R, Bhaskarachary T, Venkaiah K. Indian food composition tables. National Institute of Nutrition, Indian Council of Medical Research Hyderabad, Telengana, India; 2017.
35. Larson L, Thomas T, Kurpad A, Martorell R, Hoddinott J, Swaminathan S, et al.(P12-065) Anemia in women and children in Uttar Pradesh: a path analysis of the associations between nutritional, environmental, infectious, and genetic determinants. Poster presented in Annual meeting of American Society of Nutrition, 2018. Available from: <https://www.eventsphere.com/2018/posters/ASNScientific/SplitViewer.asp>.
36. Bothwell HT, Robert WC, Cook JD and Finch CA. Iron metabolism in man. Blackwell Scientific Publications, London. 1979, Chapter 1: 20-21,
37. Apte SV, Iyengar L. Composition of the human foetus. *British journal of Nutrition*. 1972 Mar; 27(2):305-12.
38. Neville MC, Keller R, Seacat J, Lutes V, Neifert M, Casey C, Allen J, Archer P. Studies in human lactation: milk volumes in lactating women during the onset of lactation and full lactation. *Am J Clinical Nutrition*. 1988; 48: 1375-1386.

39. Kumar A, Rai AK, Basu S, Dash D and Singh JS. Cord blood and breast milk iron status in maternal anaemia. *Pediatrics* 2008; 121:e673-e677.
40. Ghosh S, Sinha S, Shivakumar N, Thomas T, Sachdev HS, Kurpad AV. Daily iron requirements in healthy Indian children and adolescents. *Indian pediatrics*. 2019 Jul 1; 56(7):551-5.
41. Morse M, Cassels DE, Schlutz FW. Blood volumes of normal children. *American Journal of Physiology*. 1947; 151(2):448-58.
42. Russell SJ. Blood volume studies in healthy children. *Archives of disease in childhood*. 1949 Jun; 24(118):88.
43. WHO. Assessment, Prevention and Control – A Guide for Programme Managers. 2001.
44. Poskitt PME. Mineral deficiencies. In: Practical Pediatric Nutrition. London: Butterworths & Co.; 1988. p 115.
45. Domellof M, Hernell O. Iron-deficiency anaemia during the first two years of life. *Scandanavian Journal of Nutrition*. 2001; 46(1): 20-30.
46. Kodkany BS, Bellad RM, Mahantshetti NS, Westcott JE, Krebs NF, Kemp JF, et al. Biofortification of Pearl Millet with Iron and Zinc in a Randomized Controlled Trial Increases Absorption of These Minerals above Physiologic Requirements in Young Children. *Journal of Nutrition*. 2013; 143(9):1489-93.
47. Torrance JD, Charlton RW, Schmaman A, Lynch SR, Bothwell TH. Storage iron in muscle. *J Clinical Pathology*. 1968; 21: 495–500.
48. McCarthy HD, Samani-Radia D, Jebb SA, Prentice AM. Skeletal muscle mass reference curves for children and adolescents. *Pediatric obesity*. 2014 Aug; 9(4):249-59.
49. Hallberg, L. Iron requirements, iron balance and iron deficiency in menstruating and pregnant women. In: Hallberg L & Asp N G. editors. Iron Nutrition in Health and Disease. John Libbey & Co.; 1996. p 165–182.
50. Bansal PG, Toteja GS, Bhatia N, Vikram NK, Siddhu A, Garg AK, Roy AK. Deficiencies of Serum Ferritin and Vitamin B. *Int. J. Vitam. Nutr.* 2015; 85(1-2):14-22.
51. World Health Organization. WHO child growth standards - Length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age – Methods and development; 2006.
52. World Health Organization. Development of a WHO growth reference for school-aged children and adolescents; 2007.
53. Domellöf M, Lönnadal B, Abrams SA and Hernell O. Iron absorption in breast-fed infants: effects of age, iron status, iron supplements, and complementary foods. *American Journal of Clinical Nutrition*. 2002; 76:198–204.
54. Aggett PJ. Iron. In: Erdman JW, Macdonald IA, Zeisel SH, eds. Present Knowledge in Nutrition. 10th ed. Washington, DC: Wiley-Blackwell; 2012:506-20.
55. Burman D. Haemoglobin levels in normal infants aged 3 to 24 months, and the effect of iron. *Archives of disease in childhood*. 1972 Apr 1; 47(252):261-71.
56. Farquhar JD. Iron supplementation during first year of life. *American Journal of Diseases of Children*. 1963 Aug 1; 106(2):201-6.
57. Rybo G, Sölvell L. Side-effect studies on a new sustained release iron preparation. *Scandinavian journal of haematology*. 1971 Aug; 8(4):257-64.
58. Solomons NW. Competitive interaction of iron and zinc in the diet: consequences for human nutrition. *Journal of Nutrition*. 1986; 116:927-35.
59. Chang TP, Rangan C. Iron poisoning: a literature-based review of epidemiology, diagnosis, and management. *Pediatric emergency care*. 2011 Oct 1; 27(10):978-85.

10. ZINC REQUIREMENTS

INTRODUCTION

Zinc is an essential micronutrient for animal and human health. Adequate intake of Zn has been found necessary to enhance physical growth and decrease morbidity and mortality among children. Supplementation with Zn was found to lower frequency and severity of infections like diarrhoea, pneumonia, and reduce mortality¹. It is estimated that globally 2 billion people are at risk of zinc deficiency. Zinc supplementation has been shown to improve linear growth in stunted and underweight children at young age, but its impact in low and middle income settings remains uncertain^{2,3}. WHO/UNICEF recommends zinc supplementation during diarrhoeal infection and for treatment of severe malnutrition⁴. Zinc is considered a type-2 nutrient whose deficiency leads to conservation of nutrient in the body at the expense of growth, and thus clinical symptoms such as reduced growth precedes the decline in blood zinc levels⁵.

10.1 Zinc Homeostasis

Zinc is ubiquitously present in all tissues, with highest levels found in muscle and bone followed by liver. The whole-body zinc content is stable over a wide range of dietary zinc intakes indicating efficient homeostatic mechanisms^{6,7}. Homeostatic regulation of zinc metabolism is orchestrated through increased absorption during deficiency and excretion during repletion⁸. Zinc absorption and excretion in the gastrointestinal tract are the primary mechanisms for maintaining zinc homeostasis. Zinc absorption takes place throughout the small intestine with the highest rate of absorption occurring in the jejunum, but duodenum contributes to the maximal zinc absorption owing to its exposure to higher zinc concentration after a meal⁷. Thereafter, excess endogenous zinc can be secreted into the intestine through pancreatic juice and excreted in faeces⁶. The balance of intestinal absorption and endogenous losses of zinc through faeces are thus two important pathways that regulate the zinc homeostasis. During zinc deficiency or limited dietary zinc intakes, faecal zinc excretion falls with concurrent increase in intestinal absorption, thus conserves the zinc concentration in the tissues/plasma^{5,6}. On the other hand, during zinc excess, faecal zinc excretion increases while the absorption is not affected. Therefore, an exquisite balance of endogenous losses regulates the whole-body zinc homeostasis, such that the plasma zinc levels remain at steady state except under severe zinc deficiency^{6,7}.

In addition to the active intestinal excretion, zinc is also excreted through sweat, urine, hair, seminal fluid and menstrual cycle^{6,9-11}. Systematic zinc repletion/depletion studies in humans demonstrated that faecal zinc excretion increases as a function of dietary zinc intake and/or absorbed zinc but the losses in urine and sweat remained constant over a wide-range of zinc intakes^{5,6,12}. Therefore, entero-pancreatic axis is the major pathway of zinc excretion, and regulates the whole body zinc homeostasis.

10.2 Zinc Deficiency in India

Zinc deficiency is manifested with symptoms like growth failure, depressed immunity, anorexia, diarrhoea, altered skeletal function and reproductive failure. Diagnosis of zinc deficiency is more difficult because of the nonspecific clinical features. International zinc consultative group (IZiNCG) suggested that low serum zinc (<70 µg/dL) as suitable indicator to assess the prevalence of zinc deficiency at the population level, though it cannot be a diagnostic tool for individuals. Studies in different physiological groups indicated that the prevalence of serum zinc deficiency in

India is >20%, which indicates a public health significance¹³⁻²⁰. Recent comprehensive national nutrition survey (CNNS 2016-18)²¹ conducted by UNICEF-MoHFW revealed that 18.9% (1-4y), 16.8 % (5-9 y) and 31.7% (10-19 y) children in India are zinc deficient, based on low serum zinc levels. Girls tended to show higher prevalence of deficiency compared to boys at an early age (18% vs. 20.1%) but the trend reversed at adolescence (35.1% vs. 28.4%). Considering the high prevalence of stunting, anemia, high dietary phytate intakes together with observed high prevalence of serum zinc deficiency among Indian children and adolescents, suggest that Indian population is at risk of zinc deficiency, particularly adolescents.

10.3 Changes from the ICMR 2010 recommendation for Zinc requirement:

The following changes were made:

- a. The RDA 2010 provides only a single value of RDA and is adopted from WHO/FAO recommendation, and did not provide EAR. Since EAR is more relevant in population context, an effort was made to systematically derive the EAR and RDA using factorial approach.
- b. The factorial computation of zinc requirements was done considering all the average losses of zinc through bodily fluids and additional requirements due to growth (tissue and blood volume expansion), lactation and pregnancy needs. The RDA was then derived by multiplying the EAR with a factor of 1.2 (10% CV).

10.4 Strategy adopted for deriving Dietary requirements EAR and RDA: The EAR and RDA were derived by factorial approach that considered losses in 1. Sweat 2. Urine 3. Seminal fluid 4. Menstrual losses 5. Endogenous intestinal zinc excretion 6. Additional requirements during pregnancy, lactation and growth, all of which are summed and adjusted for bioavailability to derive estimated average requirements. The RDA was then computed by multiplying the EAR with a factor of 1.2 (assuming 10% CV).

10.4.1 Urinary zinc losses: A thorough literature search was conducted in Pubmed (search terms: zinc loss, zinc excretion, urinary zinc), out of which 20 studies that reported the sweat and urinary zinc loss/day of men^{11,12,22-37} and 10 studies reported the urinary loss in non-pregnant adult women^{10,11,27,37-41}. The mean and SD of urinary zinc loss/day reported in these studies was pooled separately for men and women. The mean urinary loss for men was 614 ± 238 µg/day for men and 443 ± 282 µg/day. Except under severe depletion, both sweat and urinary zinc losses are found to be constant, and thus these values are considered directly for the purpose of factorial computation^{5,12}.

10.4.2 Sweat zinc loss: Two studies reported the sweat zinc losses for men; 442.5 ± 190 µg/day²⁶ and 163.42 ± 39.41 µg/day²³. The pooled mean \pm SD sweat loss for men was 275 ± 122 µg/day. However, no data on sweat loss of zinc for women could be found, therefore, it is derived by dividing the men sweat loss with a factor of 0.86 to account for differences in surface area⁹, and amounts to 236 ± 104 µg/day.

10.4.3 Seminal zinc loss: Three studies reported the seminal zinc loss; 630 ± 540 µg/day²⁶, 139.91 ± 103 µg/day¹¹ and 111 ± 10 µg/day²⁴. Since the study by Baer *et al.*, 1984, reported very high seminal zinc content it was not considered for computation. The mean seminal loss from other two studies was 125.52 ± 65 µg/day.

10.4.4 Menstrual loss: Only one study reported the zinc loss through the menstrual fluid¹⁰. This study found the loss of 60 g menstrual fluid on an average, per period; Menstrual fluid zinc

concentration averaged about 3 µg/g of fluid that accounts of 154 µg/period or 5 µg zinc loss per day.

10.4.5 Bioavailability of zinc: Bioavailability of zinc is influenced by dietary phytic acid akin to iron. Two Indian studies measured the absorption of zinc in adolescents (girls 26.9% \pm 6; boys 32.5% \pm 16.6) and young children (17% \pm 8) from rice and millet based diets respectively^{42,43}. The bioavailability of zinc from whole wheat flour studied in Mexican adult women (20% \pm 5) was also considered⁴⁴. The mean and SD of these studies were pooled to derive a common bioavailability factor of 23.57% \pm 9.85, and is rounded off to 23% for computation of requirements.

A study in young (20-40 years) and elderly (70-80 y) Caucasian men found no differences in bioavailability of zinc from either high or low phytate meals^{45,46}. Similarly no effect of age was found on the bioavailability of zinc either in young (22-24 y) or elderly (66-75 y) women in Korea⁴⁶. The absorption of zinc was higher in adolescent's girls or women compared to boys and men, but the difference in pooled analysis of these studies was only 2-3%⁴⁷. In contrary, studies in India showed a trend of higher absorption in adolescent boys compared to girls, but the results remained insignificant⁴³. Based on these observations, 23% bioavailability was assumed across the all age/ gender groups.

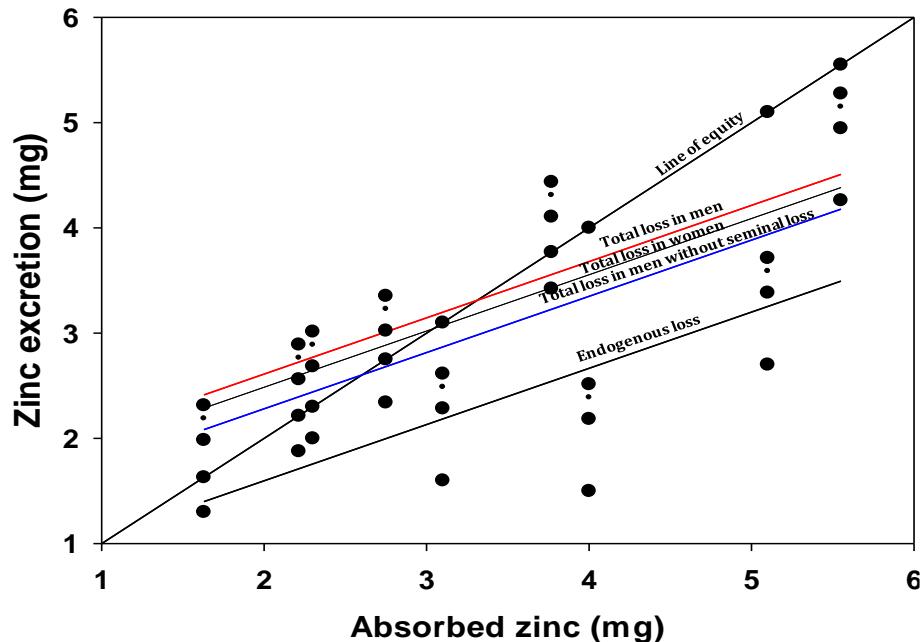
The absorption of zinc during pregnancy though showed a trend of increased absorption, the results remained insignificant⁴⁸. In contrast, many studies that measured the zinc absorption during lactation reported a significant increase in zinc bioavailability⁴⁸⁻⁵⁰. The fractional zinc absorption zinc during lactation (25%, 7-9 weeks postpartum) was found to be higher compared to preconception (14%)⁴⁹. A longitudinal study among Guatemalan women demonstrated increased fractional zinc absorption at 2nd trimester, at first two months of lactation followed by decline to basal levels at 6 months of lactation, but the extent of increase was higher in low phytate foods compared to habitual foods with higher phytic acid content⁵¹. The fractional zinc absorption was 22% at first trimester, 32% at 2nd trimester, 34% at 2 months of lactation and 26% at 6 months of lactation. Therefore, considering the high phytic acid content of cereal/pulse/millet based diets in India, a conservative estimate of 25% (23+2%) and 30% (23+7%) bioavailability were considered for pregnant and lactating women, respectively.

10.4.6 Endogenous zinc excretion and total zinc losses in adult men, and women (WRA) and children: As described above, endogenous zinc excretion in to the faeces is the major route of zinc excretion from the body and thus is of immense importance for deriving dietary requirements. Endogenous zinc excretion is dependent on the absorbed zinc, therefore it is essential to establish a relationship between these two variables to derive a factor that represents an endogenous loss as a function of absorbed zinc. To derive this factor, literature search was conducted in Pubmed (search terms: zinc loss, endogenous zinc loss, endogenous zinc excretion), out of which 9 studies that reported the endogenous zinc loss and absorbed zinc for men^{11,12,25,28,36,52} and 3 studies for non-pregnant adult women^{53,54} were identified for data extraction. The endogenous zinc excretion through intestine is assumed to be independent of gender, and thus the data was pooled.

As shown in Figure 10.1, the endogenous zinc excretion as measured by isotope dilution studies is strongly associated with absorbed zinc (Slope 0.62, $r^2=0.69$). Therefore we have summed the other mean losses of men (urinary, sweat and seminal losses) and women (urinary, sweat and menstrual losses), to the endogenous excretion and regressed over the intake. The intersection point of this line, with line of equity (absorbed zinc regressed over absorbed zinc) was then considered the absolute amount of zinc requirement which is 3.0 mg/day and 3.3 mg/day for adult women and men, respectively (Figure 10.1), which were then adjusted for mean body weight (65 kg) of all considered studies. This amounts to the absolute absorbed zinc requirements of 0.05 mg/kg body weight /day and 0.046 mg/kg body weight /day for adult men and women respectively⁵⁵. The

absolute zinc requirement without seminal and menstrual losses was 0.04 mg/kg body weight/day, and is extrapolated to compute requirements of children.

Figure 10.1: Endogenous and total loss of zinc as a function of absorbed zinc in adult men and women (WRA)



The endogenous zinc excreted in to the faeces was regressed over absorbed zinc; the urinary, sweat, seminal and menstrual losses were then summed with endogenous excreted zinc and regressed over absorbed zinc for male (total loss in men) and female (WRA; total loss in women). The absorbed zinc level at the intersection of line of equity (regression line of absorbed vs excreted zinc) with male and female regression curves was considered absolute amount of zinc required/day. The absorbed zinc level of men without seminal losses was considered for computing zinc requirements of children.

10.5 Zinc requirements

10.5.1 Adult men and women (WRA)

The absolute requirements of zinc derived from previous section (10.4.6), were then adjusted for body weights and 23% bioavailability, to arrive at 11 and 14.13 mg EAR for adult women (WRA) and men, respectively. The RDA was then derived by multiplying the respective EAR with 1.2 to arrive at 16.69 and 13.2, respectively (Table 10.1).

10.5.2 Zinc requirements of infants, children and adolescents

The absolute zinc requirements of adults (without reproductive losses) was extrapolated to compute the requirements, after excluding the contribution of seminal losses. 0.04 mg/kg body weight/day obtained for adult men without seminal losses was extrapolated for computing the requirement of infants (6-12 months) and children (1-9 years). The respective adult male and female zinc requirements were extrapolated to compute the requirement of adolescents.

Table 10.1: EAR and RDA of zinc for various physiological groups and comparison between ICMR 2020 and 2010

Category / Age	Body weight (kg) ^a	Physiological Zinc requirement (mg/day)	Absorption assumed (%)	ICMR 2020 (mg/day)		ICMR 2010 (mg/day)	IOM 2011 EAR-RDA (mg/day) ^d	EFSA 2017 (mg/day) ^d	IZiNCG 2004 ^d (mg/day)	TUL (IOM, 2011&ICMR 2018) (mg/day)
				EAR	RDA ^b					
Men	65	3.25	23	14.1	17	12	9.4 - 11	12.7*	≥ 19y	19
Women (WRA)	55	2.53	23	11.0	13.2	10	6.8 - 8	10.2*	≥ 19y	9
Pregnant women^c	55	3.0	25	12.0	14.5	12	9.5 - 11	11.5*	≥ 19y	13
Lactating women (0-6m)	55	3.53	30	11.8	14.1	12	10.4 - 12	12.6*	≥ 19y	10
Infants										
(0-6m)	5.8	-	-	-	-	-	2 (AI)	--	--	4
6-12m	8.5	0.486	23	2.1	2.5	-	2.5 - 3	2.4	6-11m	5
Children										
1-3 y	11.7	0.59	23	2.5	3.0	5	2.5 - 3	3.6	1-3y	3
4-6 y	18.3	0.85	23	3.7	4.5	7	4 - 5	4.6	4-6y	5
7-9y	25.3	1.13	23	4.9	5.9	8	4 - 5	6.2	7-8y	5
Adolescents										
Boys 10-12 y	34.9	1.62	23	7.0	8.5	9	7 - 8	8.8 (11-14y)	9-13y	9
Girls 10-12 y	36.4	1.64	23	7.1	8.5	9	7 - 8	8.9 (11-14y)	9-13y	9
Boys 13-15 y	50.5	2.75	23	11.9	14.3	11	8.5 - 11		14-15y	14
Girls 13-15 y	49.6	2.46	23	10.7	12.8	11	7.3 - 9		14-15y	11
Boys 16-18 y	64.4	3.38	23	14.7	17.6	12	8.5 - 11	11.8(15-17y)	16-18y	14
Girls 16-18 y	55.7	2.72	23	11.8	14.2	12	7.3 - 9	9.9 (15-17y)	16-18y	11

^aBody weight ICMR 2020

^bRDA was computed by multiplying EAR with a factor of 1.2.

^cPre-pregnancy weight; EAR and RDA is given as average of all 3 trimester for a women having 10 kg GWG (refer to Table 10.2)

^dHigh phytate diets

The average blood volume expansion and tissue growth with age were adopted from recent computation for iron requirements⁵⁶, the same is also adopted for computing infant requirements. The zinc requirements due to blood volume expansion were computed considering the average zinc content of whole blood (pooled for men and women) extracted from literature 5.74 µg/mL⁵⁷. The zinc requirement for tissue growth was computed considering 20 µg/g tissue, as analyzed by chemical analysis^{9,58}.

10.5.3 Lactation requirements

Breast milk zinc content varied depending on the duration of lactation. Four studies reported the breast milk concentration at different lactation periods in India⁵⁹⁻⁶², but the study by Sazawal *et al.*, 1996⁶² is published as an abstract and reported median zinc levels, and thus is not considered further. The mean zinc levels in colostrum 4.66 ± 5.55 µg/mL; transitional milk 3.61 ± 3.79 µg/mL; 1-3 months 1.95 ± 0.47 µg/mL; 3-6 months 1.49 ± 0.46 ; 6-12 months 1.28 ± 0.082 µg/mL. Since breast milk zinc levels are declined rapidly during the first one month, the average breast milk content (1.57 µg/mL) of zinc during 1-3 and 3-6 months of lactation was considered. Assuming average milk volume is around 700 mL/d, the amount of zinc needed works out to be 1.09 mg/day, rounded off to 1 mg/day. Therefore, an additional 1 mg/day zinc was added to the absolute zinc requirement and adjusted for bioavailability for computing the requirements.

10.5.4 Pregnancy requirements

During pregnancy, zinc is required to meet the basal iron loss, iron deposited in foetus, tissues and blood volume expansion. The zinc requirements for gestational weight gain of 10 and 12 kg at different gestational ages were computed, and are essentially adopted from computation of iron requirements (Chapter 9). Briefly, the absolute requirement of 0.04 mg/kg bodyweight/day (without seminal or menstrual loss), blood volume expansion (66 mL/kg weight gain) and actual weight gain (actual weight gain-blood weight), were computed for both 10 and 12 kg gestational weight gain (Table 10.2). The additional zinc requirements due to blood volume expansion were computed considering 5.75 µg/mL, 20 µg/g Zn in blood and tissue, respectively.

Table 10.2: Zinc requirement during pregnancy and for trimesters

Tri mester	Mid- trimester weight gain (kg)	Body weight ^a (kg)	Basal loss ^b (mg/d)	Blood volume expansion ^c (mg/d)	Foetal growth ^d (mg/d)	Total (mg/d)	EAR ^e (mg/d)	RDA ^f (mg/d)
For 10 kg total weight gain								
First	1.8	56.6	2.264	0.0076	0.37	2.64	10.56	12.67
Second	5.3	60.3	2.412	0.0147	0.72	3.15	12.6	15.12
Third	8.6	63.6	2.554	0.014	0.68	3.25	13.0	15.6
For 12 kg total weight gain								
First	2.1	57.1	2.284	0.0075	0.37	2.66	10.64	12.76
Second	6.3	61.3	2.452	0.0175	0.87	3.34	13.36	16.0
Third	10.4	65.4	2.616	0.071	0.85	3.48	13.92	16.7

^aPre-pregnancy weight 55 kg

^bBasal loss – 0.046 µg/kg body weight

^cBlood zinc computed assuming 66 mL blood/kg body weight gain and 5.75 µg Zn/mL blood

^dThe zinc content 20 µg/g of tissue is considered

^eEAR computed assuming 23% bioavailability

^fRDA was computed by multiplying EAR with a factor of 1.2

References

1. Yakoob MY, Theodoratou E, Jabeen A, *et al.* Preventive zinc supplementation in developing countries: impact on mortality and morbidity due to diarrhea, pneumonia and malaria. *BMC Public Health* 2011; 11 Suppl 3(Suppl 3): S23.
2. Gera T, Shah D, Sachdev HS. Zinc Supplementation for Promoting Growth in Children under 5 years of age in Low- and Middle-income Countries: A Systematic Review. *Indian pediatrics* 2019; 56(5): 391-406.
3. Radhakrishna KV, Hemalatha R, Geddam JJ, Kumar PA, Balakrishna N, Shatrujan V. Effectiveness of zinc supplementation to full term normal infants: a community based double blind, randomized, controlled, clinical trial. *PLoS One* 2013; 8(5): e61486.
4. WHO/UNICEF. World Health Organization/UNICEF, Clinical Management of Acute Diarrhoea . , 2004.
5. King JC. Zinc: an essential but elusive nutrient. *The American journal of clinical nutrition* 2011; 94(2): 679S-84S.
6. King JC, Shames DM, Woodhouse LR. Zinc homeostasis in humans. *The Journal of nutrition* 2000; 130(5S Suppl): 1360S-6S.
7. Kondaiah P, Yaduvanshi PS, Sharp PA, Pullakhandam R. Iron and Zinc Homeostasis and Interactions: Does Enteric Zinc Excretion Cross-Talk with Intestinal Iron Absorption? *Nutrients* 2019; 11(8).
8. Vallee BL, Falchuk KH. The biochemical basis of zinc physiology. *Physiological reviews* 1993; 73(1): 79-118.
9. IOM. Institute of Medicine (US) Panel on Micronutrients. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington (DC): National Academies Press (US): National Academies Press (US); 2001; 2001.
10. Hess FM, King JC, Margen S. Zinc excretion in young women on low zinc intakes and oral contraceptive agents. *The Journal of nutrition* 1977; 107(9): 1610-20.
11. Hunt CD, Johnson PE, Herbel J, Mullen LK. Effects of dietary zinc depletion on seminal volume and zinc loss, serum testosterone concentrations, and sperm morphology in young men. *The American journal of clinical nutrition* 1992; 56(1): 148-57.
12. Jackson MJ, Jones DA, Edwards RH, Swainbank IG, Coleman ML. Zinc homeostasis in man: studies using a new stable isotope-dilution technique. *British Journal of Nutrition*. 1984; 51(2): 199-208.
13. Kapil U, Bhadaria AS, Sareen N. Correlates of zinc deficiency among children in age group of six to sixty months belonging to the low-income group. *Journal of family & community medicine* 2013; 20(2): 139-40.
14. Pathak P, Kapil U, Kapoor SK, Dwivedi SN, Singh R. Magnitude of zinc deficiency among nulliparous non-pregnant women in a rural community of Haryana State, India. *Food and nutrition bulletin* 2003; 24(4): 368-71.
15. Kapil U, Jain K. Magnitude of zinc deficiency amongst under five children in India. *Indian journal of paediatrics* 2011; 78(9): 1069-72.
16. Pathak P, Kapil U, Kapoor SK, *et al.* Prevalence of multiple micronutrient deficiencies amongst pregnant women in a rural area of Haryana. *Indian journal of paediatrics* 2004; 71(11): 1007-14.
17. Pathak P, Kapil U. Role of trace elements zinc, copper and magnesium during pregnancy and its outcome. *Indian journal of paediatrics* 2004; 71(11): 1003-5.
18. Pathak P, Kapil U, Dwivedi SN, Singh R. Serum zinc levels amongst pregnant women in a rural block of Haryana state, India. *Asia Pacific journal of clinical nutrition* 2008; 17(2): 276-9.
19. Kapil U, Pathak P, Singh P, Singh C. Zinc and magnesium nutriture amongst pregnant mothers of urban slum communities in Delhi: a pilot study. *Indian pediatrics* 2002; 39(4): 365-8.

20. Kapil U, Toteja GS, Rao S, Pandey RM. Zinc deficiency amongst adolescents in Delhi. Indian pediatrics 2011; 48(12): 981-2.
21. CNNS. Ministry of Health and Family Welfare (MoHFW), Government of India, Comprehensive National Nutrition Survey (CNNS) National Report. New Delhi; 2019.
22. Wada L, Turnlund JR, King JC. Zinc utilization in young men fed adequate and low zinc intakes. The Journal of nutrition 1985; 115(10): 1345-54.
23. Milne DB, Canfield WK, Mahalko JR, Sandstead HH. Effect of dietary zinc on whole body surface loss of zinc: impact on estimation of zinc retention by balance method. The American journal of clinical nutrition 1983; 38(2): 181-6.
24. Johnson PE, Hunt CD, Milne DB, Mullen LK. Homeostatic control of zinc metabolism in men: zinc excretion and balance in men fed diets low in zinc. The American journal of clinical nutrition 1993; 57(4): 557-65.
25. Lee DY, Prasad AS, Hydrick-Adair C, Brewer G, Johnson PE. Homeostasis of zinc in marginal human zinc deficiency: role of absorption and endogenous excretion of zinc. The Journal of laboratory and clinical medicine 1993; 122(5): 549-56.
26. Baer MT, King JC. Tissue zinc levels and zinc excretion during experimental zinc depletion in young men. The American journal of clinical nutrition 1984; 39(4): 556-70.
27. Hallfrisch J, Powell A, Carafelli C, Reiser S, Prather ES. Mineral balances of men and women consuming high fiber diets with complex or simple carbohydrate. The Journal of nutrition 1987; 117(1): 48-55.
28. Turnlund JR, King JC, Keyes WR, Gong B, Michel MC. A stable isotope study of zinc absorption in young men: effects of phytate and alpha-cellulose. The American journal of clinical nutrition 1984; 40(5): 1071-7.
29. Spencer H, Asmussen CR, Holtzman RB, Kramer L. Metabolic balances of cadmium, copper, manganese, and zinc in man. The American journal of clinical nutrition 1979; 32(9): 1867-75.
30. Snedeker SM, Smith SA, Greger JL. Effect of dietary calcium and phosphorus levels on the utilization of iron, copper, and zinc by adult males. The Journal of nutrition 1982; 112(1): 136-43.
31. Mahalko JR, Sandstead HH, Johnson LK, Milne DB. Effect of a moderate increase in dietary protein on the retention and excretion of Ca, Cu, Fe, Mg, P, and Zn by adult males. The American journal of clinical nutrition 1983; 37(1): 8-14.
32. Johnson MA, Baier MJ, Greger JL. Effects of dietary tin on zinc, copper, iron, manganese, and magnesium metabolism of adult males. The American journal of clinical nutrition 1982; 35(6): 1332-8.
33. Behall KM, Scholfield DJ, Lee K, Powell AS, Moser PB. Mineral balance in adult men: effect of four refined fibers. The American journal of clinical nutrition 1987; 46(2): 307-14.
34. Coudray C, Bellanger J, Castiglia-Delavaud C, Remesy C, Vermorel M, Rayssignier Y. Effect of soluble or partly soluble dietary fibres supplementation on absorption and balance of calcium, magnesium, iron and zinc in healthy young men. European journal of clinical nutrition 1997; 51(6): 375-80.
35. Holbrook JT, Smith JC, Jr., Reiser S. Dietary fructose or starch: effects on copper, zinc, iron, manganese, calcium, and magnesium balances in humans. The American journal of clinical nutrition 1989; 49(6): 1290-4.
36. Turnlund JR, Durkin N, Costa F, Margen S. Stable isotope studies of zinc absorption and retention in young and elderly men. The Journal of nutrition 1986; 116(7): 1239-47.
37. Hunt JR, Matthys LA, Johnson LK. Zinc absorption, mineral balance, and blood lipids in women consuming controlled lactoovo vegetarian and omnivorous diets for 8 wk. The American journal of clinical nutrition 1998; 67(3): 421-30.

38. Colin MA, Taper LJ, Ritchey SJ. Effect of dietary zinc and protein levels on the utilization of zinc and copper by adult females. *The Journal of nutrition* 1983; 113(8): 1480-8.
39. Taper LJ, Hinnars ML, Ritchey SJ. Effects of zinc intake on copper balance in adult females. *The American journal of clinical nutrition* 1980; 33(5): 1077-82.
40. Swanson CA, King JC. Zinc utilization in pregnant and non-pregnant women fed controlled diets providing the zinc RDA. *The Journal of nutrition* 1982; 112(4): 697-707.
41. Wisker E, Nagel R, Tanudjaja TK, Feldheim W. Calcium, magnesium, zinc, and iron balances in young women: effects of a low-phytate barley-fiber concentrate. *The American journal of clinical nutrition* 1991; 54(3): 553-9.
42. Kodkany BS, Bellad RM, Mahantshetti NS, *et al*. Biofortification of pearl millet with iron and zinc in a randomized controlled trial increases absorption of these minerals above physiologic requirements in young children. *The Journal of nutrition* 2013; 143(9): 1489-93.
43. Nair KM, Brahmam GN, Radhika MS, *et al*. Inclusion of guava enhances non-heme iron bioavailability but not fractional zinc absorption from a rice-based meal in adolescents. *The Journal of nutrition* 2013; 143(6): 852-8.
44. Rosado JL, Hambidge KM, Miller LV, *et al*. The quantity of zinc absorbed from wheat in adult women is enhanced by biofortification. *The Journal of nutrition* 2009; 139(10): 1920-5.
45. Couzy F, Kastenmayer P, Mansourian R, Guinchard S, Munoz-Box R, Dirren H. Zinc absorption in healthy elderly humans and the effect of diet. *The American journal of clinical nutrition* 1993; 58(5): 690-4.
46. Kim J, Paik HY, Joung H, Woodhouse LR, Li S, King JC. Effect of dietary phytate on zinc homeostasis in young and elderly Korean women. *Journal of the American College of Nutrition* 2007; 26(1): 1-9.
47. Armah SM. Fractional Zinc Absorption for Men, Women, and Adolescents Is Overestimated in the Current Dietary Reference Intakes. *The Journal of nutrition* 2016; 146(6): 1276-80.
48. King JC. Effect of reproduction on the bioavailability of calcium, zinc and selenium. *The Journal of nutrition* 2001; 131(4): 1355S-8S.
49. Fung EB, Ritchie LD, Woodhouse LR, Roehl R, King JC. Zinc absorption in women during pregnancy and lactation: a longitudinal study. *The American journal of clinical nutrition* 1997; 66(1): 80-8.
50. Donangelo CM, Zapata CL, Woodhouse LR, Shames DM, Mukherjea R, King JC. Zinc absorption and kinetics during pregnancy and lactation in Brazilian women. *The American journal of clinical nutrition* 2005; 82(1): 118-24.
51. Hambidge KM, Miller LV, Mazariegos M, *et al*. Upregulation of Zinc Absorption Matches Increases in Physiologic Requirements for Zinc in Women Consuming High- or Moderate-Phytate Diets during Late Pregnancy and Early Lactation. *The Journal of nutrition* 2017; 147(6): 1079-85.
52. Taylor CM, Bacon JR, Aggett PJ, Bremner I. Homeostatic regulation of zinc absorption and endogenous losses in zinc-deprived men. *The American journal of clinical nutrition* 1991; 53(3): 755-63.
53. Hunt JR, Mullen LK, Lykken GI. Zinc retention from an experimental diet based on the U.S.F.D.A. total diet study. *Nutrition Research* 1992; 12(11): 1335-44.
54. Sian L, Krebs NF, Westcott JE, *et al*. Zinc homeostasis¹⁸⁸ during lactation in a population with a low zinc intake. *The American journal of clinical nutrition* 2002; 75(1): 99-103.
55. Brown KH, Rivera JA, Bhutta Z, Gibson RS, King JC, Lönnnerdal B, Ruel MT, Sandström B, Wasantwisut E, Hotz C, International Zinc Nutrition Consultative Group (IZiNCG). International Zinc Nutrition Consultative Group (IZiNCG) technical document# 1. Assessment of the risk of zinc deficiency in populations and options for its control. *Food and nutrition bulletin*. 2004 Mar; 25(1 Suppl 2):S99-203..

56. Ghosh S, Sinha S, Shivakumar N, Thomas T, Sachdev HS, Kurpad AV. Daily Iron Requirements in Healthy Indian Children and Adolescents. *Indian paediatrics*. 2019; 56(7): 551-5.
57. Dawson JB, Walker BE. Direct determination of zinc in whole blood, plasma and urine by atomic absorption spectroscopy. *Clinica chimica acta; international journal of clinical chemistry* 1969; 26(3): 465-75.
58. Widdowson EM, and J. W. T. Dickerson. Chemical composition of the body. *Mineral Metabolism*. Edited by Comar CL, Bronner F; 1964.
59. Hemalatha P, Bhaskaram P, Kumar PA, Khan MM, Islam MA. Zinc status of breastfed and formula-fed infants of different gestational ages. *Journal of tropical paediatrics*. 1997; 43(1): 52-4.
60. Rajalakshmi K, Srikantia SG. Copper, zinc, and magnesium content of breast milk of Indian women. *The American journal of clinical nutrition*. 1980; 33(3): 664-9.
61. Sharda B, Bhandari B, Bhandari LM. Copper, zinc, magnesium and cadmium levels of breast milk of Indian women. *Transactions of the Royal Society of Tropical Medicine and Hygiene*. 1983; 77(2):201-3.
62. Sazawal S, Jalla S, Dhingra P, Krebs N, Black RE, Bhan MJFJ. Impact of zinc supplementation on breast milk zinc levels among low socioeconomic Indian women. *FASEB Journal*. 1996; 10(3).

11. TRACE ELEMENTS

INTRODUCTION

Among the trace elements other than Fe, Zn and I, the requirements of chromium (Cr), copper (Cu), manganese (Mn), selenium (Se) have been studied in more detail and their recommended dietary intakes were suggested by different agencies. However, these recommendations are based on data available from clinical experimental studies with small sample size or based on dietary intake data from the population surveys. Data needed for direct assessment of the requirement of these trace elements are unavailable and thus the recommendations have been only provisional.

Some information on the intakes of copper, manganese and chromium in adults has been reported in a few community-based studies in India. However, data on absorption and excretion of these nutrients needed for estimating their EARs and RDAs are not yet available in the Indian population. International Agencies including Institute of Medicine in the USA and European Food Safety Authority have provided recommendations on adequate intake of these trace elements. RDA for Zn and I have been considered separately in view of their importance and a brief account of relevant information on the nutritional significance and suggested safe dietary intakes for Cu, Cr, Mn and Se for adults are provided in this chapter.

11.1. COPPER, MANGANESE AND CHROMIUM

11.1.1. Copper (Cu)

The adult body contains approximately about 80 mg Cu mainly stored in liver, followed by brain and muscle. Sea foods, legume seeds and oilseeds like sesame, sunflower and nuts are rich sources of Cu. Fruits and vegetables are moderate sources. Cu is transported in the form of ceruloplasmin in blood/plasma. Zn is well known to be antagonistic to Cu bioavailability in terms of its competition with metallothionein binding and thus enhancing the requirement of Cu. Cytochrome C oxidase, superoxide dismutase (SOD), lysyl oxidase and tyrosine oxidase are the major Cu containing metalloenzymes of which the SOD is also dependent on Zn. Deficiency signs of copper include anemia, vascular complications, osteoporosis and neurological manifestations. Lysyl oxidase is decreased in Cu deficiency leading to diminished collagen and elastin cross-linking. Anemia may result from deranged iron metabolism.

In fact, blood levels of hemoglobin were found to correlate significantly with the status of copper apart from other nutrients and relative significance of copper was more than that of ascorbic acid in iron-deficiency anemia¹. Copper deficiency has also been found in special conditions such as premature infants fed milk formulas, in infants recovering from malnutrition associated with chronic diarrhea and fed cow's milk², and in patients with prolonged total parenteral nutrition. On the other hand, excessive copper accumulation or copper overload has been recorded in Indian childhood cirrhosis (ICC). However, it has been concluded that though copper has a role to play in ICC, it may also be attributed to the genetic predisposition³.

A number of indicators for Cu status in humans have been considered by previous studies. Institute of Medicine (IOM) has used a combination of indicators, including plasma copper and ceruloplasmin concentrations, erythrocyte superoxide dismutase activity, and platelet copper concentration in controlled human depletion/repletion studies suggested Estimated Average Requirement (EAR) for copper for the US population⁴. However, all of these indicators have some limitations. Serum copper concentration falls in copper deficient individuals and returns to normal

within a few days of copper supplementation. But it does not reflect dietary intake except when intake is below a certain level. Similarly, Ceruloplasmin does not respond to dietary intake, unless intake is very low⁵. Moreover, ceruloplasmin is an acute phase protein and its levels may elevate with use of contraceptives and steroids. Therefore, although low ceruloplasmin concentration denotes copper depletion or deficiency, its use as a bio-marker of copper is limited in determining the copper overload. The dietary copper intake at which ceruloplasmin concentration ceases to increase in response to increased dietary copper may be considered the copper requirement for ceruloplasmin synthesis. Erythrocyte superoxide dismutase (SOD) activity is suggested to be a more specific indicator of copper status. However, it can increase in situations that produce oxidative stress and also in alcoholism. Two additional indicators - platelet copper concentration and platelet cytochrome c oxidase activity - have been demonstrated to respond more rapidly to low dietary copper in a few studies⁴.

Copper balance studies in humans are problematic because copper balance can be achieved over a broad range of dietary copper intakes and reflects prior dietary intake⁴. Studies with long duration are thus needed due to long adaptation period but such studies are not available. Moreover, the copper losses, although small, are very difficult to quantify. Therefore, balance studies have not been used by the IOM for estimating copper requirements.

Another important consideration is that the bioavailability of copper is influenced markedly by the amount of copper in the diet. Bioavailability was found to range from 75% of dietary copper absorbed when the diet contains only 400 µg/day to 12% absorbed when the diet contained 7.5 mg/day^{6, 7}. In addition, copper homeostasis is very efficient. As more copper is absorbed, turnover is faster and more copper is excreted into the gastrointestinal tract⁷. This excretion is probably the primary point of regulation of total body copper and helps to protect against copper deficiency and toxicity.

Copper status in Indians: A few studies from India have assessed serum copper concentrations and majority of these were conducted in disease conditions like liver cirrhosis, cystic fibrosis, visceral leishmaniasis etc. Additionally, studies have estimated serum copper when the intervention included zinc supplementation. One study has reported low serum copper levels (<80 µg/dL) among a tribal population in Jharkhand State with prevalence about 35%⁸.

Dietary copper intake: Indian diets showed high variation in copper intakes (1.6-5.2 mg/day). Data from NNMB rural and urban surveys shows dietary intakes of copper within the above range (Table 1). High variation was also observed in absorption (7-37%) and these values were lower in case of vegetarian diet compared to 40-50% absorption reported from meat based diets in the western countries⁴.

Deriving Dietary requirements EAR and RDA for Cu: The factorial computation of Cu requirements is not possible due to lack of data on the obligatory copper losses in healthy people and estimates of additional requirements due to growth (tissue and blood volume expansion), lactation and pregnancy needs.

Pregnancy: IOM has estimated EAR for pregnant women based on estimates of the amount of copper that must be accumulated during pregnancy (18 mg) to account for the fetus and products of pregnancy. Over the course of pregnancy, this additional requirement is approximately 67 µg/day of absorbed copper or 135 µg/day of dietary copper, a value based on 50 % bioavailability and rounding. Copper absorption is likely to be more efficient during pregnancy⁹.

Lactation: The EAR for lactation is determined on the basis of the amount of copper secreted in breast milk (approximately 200 µg/day) plus the EAR for NPNL women. Considering copper bioavailability to be 50%, an additional 400 µg/day of copper is recommended for lactating women.

Infants, children and adolescents: Data for computation of EARs / RDAs for copper for infants, children or adolescents are not available. Therefore, these have been estimated by extrapolation from the adult recommendations in the IOM guidelines (Table 3).

As there is no evidence to revise the acceptable intake (2 mg/d) suggested by the previous committee, the same value has been retained (Table 2).

Table 1: Dietary intakes (Median) of trace elements

NNMB Urban data 2016								
Age (years)	Copper (mg/day)		Chromium (µg/day)		Manganese (mg/day)		Selenium (µg/day)	
	Male	Female	Male	Female	Male	Female	Male	Female
	Median							
1-3	0.59	0.58	21.61	22.03	1.89	1.74	17.91	15.68
4-6	0.89	0.87	28.16	27.90	3.01	2.85	32.23	29.05
7-9	1.07	1.00	32.64	30.47	3.73	3.45	42.40	36.07
10-12	1.28	1.15	36.23	33.99	4.66	4.10	51.36	45.49
13-15	1.43	1.30	41.32	36.81	5.12	4.63	58.23	52.43
16-17	1.57	1.30	43.71	38.12	6.06	4.74	71.10	54.88
18-60	1.80	1.54	50.68	46.27	6.63	5.41	76.30	59.28
60 & Above	1.73	1.37	51.32	43.34	6.32	4.78	70.39	52.59
NNMB Rural data 2011-12								
1-3	0.62	0.52	12.45	12.61	1.35	1.34		
4-6	0.97	0.81	19.95	20.27	2.08	2.16		
7-9	1.23	0.97	25.13	23.22	2.65	2.49		
10-12	1.35	1.07	28.54	27.15	3.01	2.83		
13-15	1.54	1.19	32.55	31.08	3.46	3.16		
16-17	1.67	1.28	36.49	33.34	3.80	3.33		
18-60	1.87	1.38	41.43	36.83	4.41	3.70		
60 & Above	1.65	1.16	37.98	30.64	3.85	3.12		

11.1.2. Manganese (Mn)

Plant foods like wheat, barley, rice bran are rich in Mn. Fruits and vegetables are moderate sources and animal foods like eggs, beef and chicken contain low levels. About 10-20 mg of Mn is present in the body. Bone, liver, pancreas and kidney are important tissues containing Mn. Mn are the cofactor for the enzymes SOD, arginase, and glycosyl transferase which are involved in carbohydrate, lipid and amino acid metabolism¹⁰. There are other enzymes like phosphoenol pyruvate carboxy kinase and glutamine synthetase, which are activated by Mn ions. Growth failure, skeletal abnormalities and impaired reproductive function have been reported to be caused by Mn deficiency. Abnormal insulin metabolism and glucose tolerance are the important effects attributed to Mn deficiency. Though, there are reports on adverse effects from exposure to manganese (occupational hazard), most of them are inhalational. ATSDR (Agency for Toxic substances and Disease Registry) report says that oral exposure to contaminated water sources can equally be hazardous as inhalational exposure leading to neurological disorder known as manganism¹¹. However, threshold levels to cause these effects have not been established yet. Hence, Mn is established as no observed adverse effect level and an adequate intake has been suggested. Isotopic turnover and chemical balance studies have revealed Mn requirement of an adult to be between 2-5 mg/d (Table 2).

Table 2. Absorption and acceptable intakes of Cu, Mn, Cr and Se for Indian adults

Trace element	Indian adults	
	Mean and range Absorption %	Acceptable intake*
Cu (mg/d)	18 (7-37)	1.7
Mn (mg/d)	14 (2-24)	4.0
Cr ($\mu\text{g}/\text{d}$)	79 (63-94)	50
Selenium ($\mu\text{g}/\text{d}$)	≈ 90	40

*Reference 18

Table 3

Trace elements	IOM Dietary reference intakes [#]													
	Infants		Children		Adolescent				Adult		Pregnancy	Lactation		
					0-6 months	7-12 months	1-3 years	4-8 years	Boys	Girls	Boys	Girls	Men	Women
Cu ($\mu\text{g}/\text{d}$)	200	220	340	440	700	700	-	-	900	900	1000	890		
Mn ($\mu\text{g}/\text{d}$)	3	600	1200	1500	1900	1600	2200	1600	2300	1800	2000	2600		
Cr ($\mu\text{g}/\text{d}$)	0.2	5.5	11	15	25	21	35	24	35	25	30*	45*		
Se ($\mu\text{g}/\text{d}$)	15**	20**	20	30	40	40	55	55	55	55	60	70		

*1 unit less in case if less than 18 years

** Adequate Intake (AI)

Reference 4

Men have been found to have significantly less manganese absorption than women and that this difference may be related to iron status¹². It has also been demonstrated that high ferritin concentrations were associated with reduced Mn absorption¹³.

Wide range of manganese intakes can result in manganese balance and therefore, balance data are not useful to set an EAR. IOM has used dietary intake data from the population surveys to set an AI for manganese⁴.

In the Indian context, there is no evidence to revise the acceptable intake (4 mg/d) of Mn suggested by the previous committee. Therefore, the same value has been retained (Table 2).

11.1.3. Chromium (Cr)

Chromium is found to be distributed in nature in a way similar to that of Cu. Trivalent chromium has been postulated to be necessary for the efficacy of insulin in regulating metabolism of carbohydrates, lipids and protein. Sea foods (oysters), meat and whole grain products are good sources, followed by egg, butter and tubers like potato. Cheese is a concentrated source of Cr. Fruits and vegetables, in general, are not good sources of Cr. In chromium deficiency too, impaired glucose tolerance and weight loss along with peripheral neuropathy were observed. Cr deficiency attributable to its lack in the body was reported in total parenteral nutrition. In such cases Cr supplementation reversed symptoms of glucose intolerance and insulin requirement. There has been preponderance of evidence for Cr-potentiating insulin action both *in vitro* and *in vivo*. Cr was shown to be part of the ‘Glucose tolerance factor’ and thus has an impact on glucose tolerance.

Chromium deficiency has been reported in three patients who did not receive supplemental chromium in their total parenteral nutrition (TPN) solutions^{14,15,16}. There has not been any evidence or adverse effects reported on chromium overload¹⁷.

Published data to set an EAR for chromium is not available. IOM has therefore, set an Adequate Intake (AI) based on the estimated mean intakes from dietary surveys. The AI is 35 µg/day and 25 µg/day for young men and women, respectively.

In view of these limitations, the recommendation by the previous committee on ‘acceptable intake’ of chromium (50 µg/d) for adults has been retained (Table 2).

Further research is needed to determine absorption and metabolism of chromium to derive EAR for adults and requirements during pregnancy and lactation.

11.1.4. Requirements of Cu, Mn and Cr for Indian adults

Most of the data on nutrient requirements and dietary intake level of these three trace elements was generated during the years 1980-81 by Rao and Rao *et al*^{18,19}. Subsequently, Singh *et al*²⁰ reported the mean daily intake by rural and urban population, respectively, for Cu (mg): 2.01, 1.85; Mn (mg): 6.5, 8.7; Cr (µg): 60.5, 75.5, from North India. Other studies of Pathak *et al*²¹ and Kapil *et al*²² showed that the daily intakes of rural underprivileged communities for Cu 2.7 mg, Mn 9.6 mg /d, are far more than the average requirement reported here. Another study by Agte *et al*²³ from Western part of India reported that the absorption of Cu ranged from 10.2-21.7% at intakes of 2.7-5.2 mg/d; (the mean absorption was 17.8%) confirming the data of Rao and Rao ¹⁸. Also, the apparent absorption of Cu (16.4%) and Mn (12.2%) were reported by them in ileostomized human volunteers²⁴. Most data on dietary content of these trace elements generated in recent times ^{20,21,22}, though sparse, agree with the content first described for typical diets representing different regions of the country by Rao and Rao¹⁹.

R. Raghunath *et al.* (2006) observed the mean (SD) intake of Cu and Mn among adult population of Mumbai as 1.0 (1.6) and 2 (1.63), respectively. Vegetarian and non-vegetarian diet

were compared and a significant difference was established between these groups in case of Manganese²⁵. Patra *et al.* (2009) reported a mean intake of Cu to be 3.3 (1.2)²⁶. Gupta *et al.* (2017) reported copper and chromium was not included in diet adequately by 81% and 89%, respectively of the Geriatric population²⁷.

The earlier data pertain to the chemical balances conducted on adult male volunteers for Cu, Mn and Cr. The Indian Food Composition Tables published in 2017 provide information on these trace elements in more than 400 foods. Based on the NNMB data on the dietary intakes of the above trace elements in Indians and their homeostatic regulation, nutritional deficiency of these elements appears to be unlikely.

11.2. SELENIUM

11.2.1. Background and dietary sources

Importance of Selenium (Se) in biology has been intimately connected with that of the “trinity” of antioxidants, the remaining two being cysteine and vitamin E. The discovery of selenium as an important nutrient by Schwarz and Foltz²⁸ can be traced to the prevention of nutritional liver necrosis in vitamin E deficient rats when Se in trace amounts were supplemented to them. Selenium in food is present in at least two forms - as Selenomethionine in plant foods and selenocysteine in animal foods²⁹.

The selenium content of food varies depending on the selenium content of the soil where the animal was raised or the plant was grown. Organ meat and sea foods are rich sources of selenium, their content in the diet being 0.10-1.3 µg/g. Bioavailability of Se from sea foods may be low because of high concentration of heavy metals like Cd, Hg etc³⁰. There are certain plant foods like mustard, and to a lesser extent garlic and broccoli, which accumulate Se from soil. Cereals and grains are major dietary sources of Se (<0.1 µg/g to 0.8 µg/g). Bioavailability from common foods is reported to be in the range of 70-90%³⁰.

11.2.2. Functions

Glutathione peroxidase is the only selenoprotein enzyme well studied for the biological role of Se. Deiodinase isoforms that are involved in thyroid hormone metabolism are also selenium-containing proteins. Enzymes like thiolase and glycine reductase are the other less known sources of selenoproteins. Selenomethionine cannot be synthesized in the body. It can substitute for methionine in proteins or it can be converted to selenocysteine (SeCys). Inorganic selenium is incorporated into selenoproteins by condensation with serine-bound to tRNA, forming a tRNA- Se-Cys complex that is inserted into selenoproteins by the unique UGA codon sequence. Apart from its antioxidant protection against free radicals, Se was found to be functional in detoxification and immune potentiation^{30,31}. Inorganic selenium enters a reductive pathway to form reduced selenide through a complex formation with glutathione-selenodiglutathione (GSSeSG) and glutathione selenopersulfide (GSSeH). Selenium disulfide may undergo methylation in the presence of S-adenosylmethionine and get excreted in breath or in urine.

Plasma Se and glutathione peroxidase (GSHPx) activity indicate the short-term status, whereas, red blood cell Se and GSHPX activity reflect long-term Se status. Platelet GSHPx activity is considered as a good indicator for assessing changes in selenium status. Urinary excretion is approximately twice the dietary intake and is highly variable with intakes of Se^{31,32}. Vitamin E and Se along with reduced glutathione (GSH) act in a complementary manner in oxygen radical scavenging activity and therefore spare the requirements of each other.

Selenium intake in adequate quantities might prevent from certain diseases like cancer, cardio-vascular diseases, cognitive decline and thyroid diseases as it plays a mighty role in development or the progression of the disease³³.

11.2.3. Deficiency and excess

Selenium deficiency has been associated with two childhood/adolescent endemic diseases, ‘Keshan’ (cardiomyopathy) and ‘KashinBeck’ (osteoarthritis) in China. These diseases are found to be prevalent in certain areas in China where the intake of Se is very low, 7-11 µg/d²⁹. A number of epidemiological studies suggest that poor intake of Se is associated with increased risk of cancer or heart disease, both related to its antioxidant function³¹. People undergoing long term hemodialysis and people living with HIV are at risk groups of selenium inadequacy³⁴.

Selenium toxicity (selenosis) is well known in livestock in seleniferous areas leading to blind staggers. Selenium poisoning has been observed in humans under occupational exposures or in seleniferous areas. Daily intakes above 700 µg/d or acute consumption of 1-7 mg Se/kg/d result in toxicity in humans. Dermatitis, depression and brittle finger nails, excessive tooth decay, numbness and hemiplegia are some of the non-specific symptoms of poisoning³¹.

11.2.4. Requirements

A broad range of Se requirements (9-80 µg) has been suggested based on the balance studies³². The requirements are also derived using saturation of blood or plasma glutathione peroxidase activity in people from endemic areas in China. After supplementing subjects suffering from *keshan disease* with different levels of selenium, dietary intakes beyond 40 µg/d did not produce any further increase in GPX activity^{29,30}. After adjustment for body weight and variations in bio-availability and dietary pattern, Se requirement was worked out to be 60-70 µg/d. Recent FAO/WHO Committee recommended a Se intake of 26 µg/d for adult women and 36 µg/d for adult men as normal requirement³⁵. These values are lower than the corresponding figures of 55 and 70 µg/d suggested by US Agency³⁶.

Based on the estimated accumulation of selenium by the fetus to saturate its seleno proteins of selenium [1,000 µg (12.6 µmol)], IOM has recommended an additional 4 µg (0.05 µmol)/day of selenium in pregnancy³⁶. As most selenium is highly bioavailable, no adjustment is made for absorption. Based on the amount of selenium in breast milk, requirement during lactation has been estimated by adding 14 µg (0.18 µmol)/day of selenium to the requirement for the non-pregnant and non-lactating (NPNL) women. Requirement for children and adolescents in the age group 1-18 years are estimated by extrapolation from the adult RDAs.

11.2.5. Situation in India

There have been very few studies on Se nutrition in the Indian population. Studies from NIN on staple foods from local markets near Hyderabad showed that the Se content ranged between 30-400 µg/100g for most cereals and pulses. Average estimated dietary intake was 41-51 µg/d in different income groups³⁷. NNMB data from the urban survey in 2016 show higher median intakes 76.3 µg/d in men and 59.2 µg/d women (Table 1). Extrapolating the analyzed data to the dietary pattern reported in 7 states, the mean Se intakes were found to vary from 71 to 163 µg/d³⁸. According to WHO RDA values, no state has lower than recommended intakes. Another estimate of intake of Se was 61 µg/day in rice-pulse based South Indian diets³⁹.

Based on the limited data available on blood values of Se or GSHPx activity in plasma, red cell or platelets, there was no evidence of inadequate Se status in Indian population. This was true both in children and adults^{38,40}. A recent report from Punjab describes the dietary Se intake and Se

content of hair, nails and urine in subjects investigated from two sets of villages- one with endemic selenosis and another control area. Based on the clinical symptoms and biochemical profile, an intake above 600 µg/d was associated with selenosis symptoms. The differences in mean values were 10-fold in Se intake, at least 20-fold in urinary excretion and more than 40-fold in hair and nail Se levels between villages with high and normal Se levels⁴⁰. The mean urinary Se excretion from subjects drawn from non-endemic villages (0.9-1.2 µg/100 ml) are slightly on the lower side of normal range reported by Novarro *et al*⁴¹ elsewhere (0.46-5.03 µg Se/100 ml) in persons consuming 55 µg dietary Se/d. Thus, from the available Indian data Se deficiency or depletion does not appear to be a problem and the dietary intake (71-163 µg/d) reported³⁸ is consistent with the RDA of 36 and 26 µg (for males and females, respectively) as suggested by FAO/WHO.

Due to paucity of new information, a level of 40µg/d recommended as the acceptable intake of Se for Indians is retained.

References

1. Chiplonkar SA, Agte VV, Mengale SS. Relative importance of micronutrient deficiencies in iron deficiency anemia. *Nutrition research*. 2003 Oct 1; 23(10):1355-67.
2. Shaw JC. Copper deficiency in term and preterm infants. In Nestle nutrition workshop series 1992.
3. Nayak NC and Chitlai AR. Indian Childhood Cirrhosis (ICC) and ICC like changing scenario of facts versus notions. *Indian Journal of Medical Research*. 2013;137:1029-1042.
4. Institute of Medicine (IOM): Dietary reference intakes of vitamin A, vitamin K, As, B, Cr, Cu, I, Fe, Zn. Food and Nutrition Board., National Acad. Press, Washington DC 2000.
5. EFSA NDA Panel (EFSA Panel on Dietetic Products, Nutrition and Allergies), 2015. Scientific Opinion on Dietary Reference Values for copper. *EFSA Journal* 2015;13(10):4253, 51 pp.
6. Turnlund JR, Keyes WR, Anderson HL, Acord LL. Copper absorption and retention in young men at three levels of dietary copper by use of the stable isotope ^{65}Cu . *The American journal of clinical nutrition*. 1989 May 1; 49(5):870-8.
7. Turnlund JR. Human whole-body copper metabolism. *The American journal of clinical nutrition*. 1998 May; 67(5):960S-4S.
8. Kapil U, Singh P. Serum copper levels among a tribal population in Jharkhand State, India: a pilot survey. *Food and nutrition bulletin*. 2005 Sep; 26(3):309-11.
9. Turnlund JR, Swanson CA, King JC. Copper absorption and retention in pregnant women fed diets based on animal and plant proteins. *The Journal of nutrition*. 1983 Nov 1; 113(11):2346-52.
10. EFSA NDA Panel (EFSA Panel on Dietetic Products, Nutrition and Allergies), 2013. Scientific Opinion on Dietary Reference Values for manganese. *EFSA Journal* 2013; 11(11):3419, 44 pp.
11. ATSDR (Agency for Toxic substances and Disease Registry, US Department of Health and Human Services), 2012. Toxicology profile for manganese.556pp.
12. Finley JW, Johnson PE, Johnson LK. Sex affects manganese absorption and retention by humans from a diet adequate in manganese. *American Journal of Clinical Nutrition*. 1994;60:949–955
13. Finley JW. Manganese absorption and retention by young women is associated with serum ferritin concentration. *American Journal of Clinical Nutrition*. 1999; 70:37–43.
14. Brown RO, Forloines-Lynn S, Cross RE, Heizer WD. Chromium deficiency after long-term total parenteral nutrition. *Digestive diseases and sciences*. 1986 Jun 1; 31(6):661-4.
15. Freund H, Atamian S, Fischer JE. Chromium deficiency during total parenteral nutrition. *Jama*. 1979 Feb 2; 241(5):496-8.
16. Jeejeebhoy KN, Chu RC, Marliss EB, Greenberg GR, Bruce-Robertson A. Chromium deficiency, glucose intolerance, and neuropathy reversed by chromium supplementation, in a patient receiving long-term total parenteral nutrition. *American Journal of Clinical Nutrition*. 1977; 30:531–538.
17. EFSA NDA Panel (EFSA Panel on Dietetic Products, Nutrition and Allergies), 2014. Scientific Opinion on Dietary Reference Values for chromium. *EFSA Journal* 2014; 12(10):3845, 25 pp.
18. Rao CN and Rao BSN. Absorption and retention of magnesium and some trace elements by man from typical Indian diets. *Nutrition Metabolism*. 1980; 24: 244-254.
19. Rao BSN and Rao CN. Trace element content of Indian foods and the dietaries. *Indian Journal of Medical Research*. 1981; 73: 904-909.
20. Singh RB, Gupta UC, Mittal N, Niaz MA, Ghosh S, Rastogi V. Epidemiologic study of trace elements and magnesium on risk of coronary artery disease in rural and urban Indian populations. *Journal of the American College of Nutrition*. 1997 Feb 1; 16(1):62-7.

21. Pathak P, Kapil U, Kapoor SK, Saxena R, Kumar A, Gupta N, Dwivedi SN, Singh R, Singh P. Prevalence of multiple micronutrient deficiencies amongst pregnant women in a rural area of Haryana. *The Indian Journal of Pediatrics.* 2004 Nov 1; 71(11):1007-14.
22. Kapil U, Verma D, Goel M, et al. Dietary intake of trace elements and minerals among adults in underprivileged communities of rural Rajasthan, India. *Asian Pacific Journal of Clinical Nutrition.* 1998; 7:29-32.
23. Agte V, Chiplonkar S, Joshi N, Paknikar K. Apparent absorption of copper and zinc from composite vegetarian diets in young Indian men. *Annals of nutrition and metabolism.* 1994; 38(1):13-9.
24. Nayak NC and Chitlai AR, 2013. Indian Childhood Cirrhosis (ICC) and ICC like changing scenario of facts versus notions. *Indian Journal of Medical Research,* 137, 1029-1042
25. Raghunath R, Tripathi RM, Suseela B, Bhalke S, Shukla VK, Puranik VD. Dietary intake of metals by Mumbai adult population. *Science of the Total Environment.* 2006; 356(1-3):62-68.
26. Patra AK, Wagh SS, Jain AK, Hegde AG. Assessment of daily intake of trace elements by Kakrapar adult population through ingestion pathway. *Environmental Monitoring and Assessment.* 2010; 169(1-4):267-272.
27. Gupta A, Khenduja P, Pandey RM, Sati HC, Sofi NY, Kapil U. Dietary Intake of Minerals, Vitamins, and Trace Elements Among Geriatric Population in India. *Biological Trace Elements Research.* 2017; 180(1):28-38. doi:10.1007/s12011-017-0972-8
28. Schwarz K, Foltz CM. Selenium as an integral part of factor 3 against dietary necrotic liver degeneration. *Journal of the American Chemical Society.* 1957 Jun; 79(12):3292-3.
29. Yang G, Ge K, Chen J, Chen X. Selenium-related endemic diseases and the daily selenium requirement of humans. *World review of nutrition and dietetics.* 1988.
30. Finley JW. Bioavailability of selenium from foods. *Nutrition reviews.* 2006 Mar 1; 64(3):146-51.
31. Levander OA. A global view of human selenium nutrition. *Annual review of nutrition.* 1987 Jul; 7(1):227-50.
32. Levander OA. Selenium. In: *Trace elements in human and animal nutrition*, 5thEdn. (W Mertz ed.), Acad. Press, Orlando, 1986.
33. National Institutes of Health. Selenium: fact sheet for health professionals. Source Updated March 2018;2.
34. EFSA NDA Panel (EFSA Panel on Dietetic Products, Nutrition and Allergies), 2014. Scientific Opinion on Dietary Reference Values for selenium. *EFSA Journal* 2014; 12(10):3846, 67 pp.
35. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.
36. Institute of Medicine (IOM): Dietary reference intakes of vitamin C, vitamin E, Selenium and carotenoids. Food and Nutrition Board, National Acad. Press, Washington, DC 2000.
37. Kumar A, Krishnaswamy K. Selenium content of common Indian cereals, pulses, and spices. *Journal of agricultural and food chemistry.* 1997 Jul 16; 45(7):2565-8.
38. Kumar AD and Krishnaswamy K. Selenium in human nutrition, *Nutrition News,* 24:1-6, 2003.
39. Srikumar TS. The mineral and trace element composition of vegetables, pulses and cereals of southern India. *Food chemistry.* 1993 Jan 1; 46(2):163-7.
40. Hira CK, Partal K, Dhillon KS. Dietary selenium intake by men and women in high and low selenium areas of Punjab. *Public Health Nutrition.* 2004 Feb; 7(1):39-43.
41. Navarro M, Lopez H, Lopez MC, Perez V. Determination of selenium in urine by hydride generation atomic absorption spectrometry. *Journal of AOAC International.* 1996 May 1; 79(3):773-6.

11.3. IODINE

11.3.1. Introduction

Iodine is an essential micronutrient, needed for synthesis of thyroid hormone for optimal physical growth and development of humans. The healthy human body contains about 20 mg of iodine, 70-80% of which is concentrated in the thyroid gland. Iodine deficiency leads to enlargement of thyroid gland, known as endemic goitre, as well as a wide spectrum of disorders, which are termed as iodine deficiency disorders (IDD) including abortion, stillbirths, low birth weight, cretinism, neonatal chemical hypothyroidism, psycho-motor defects, impaired coordination, mental retardation and hypothyroidism. Studies have revealed that in an iodine deficient environment, school children have about 13.5 IQ points lower and exhibit poor scholastic performance¹. Iodine deficiency is known to affect the livestock as well, including cattle, sheep, pigs and poultry in the form of abortions, stillbirths, low birth weight, inadequate growth, and functional disabilities². IDD is one of the most important micronutrient deficiency disorders of public health significance in India^{3,4}.

11.3.2. Aetiology

Environmental iodine deficiency is the major etiological factor leading to IDD. Sea water is the richest source of iodine on earth. Iodine evaporates due to sunlight into atmosphere along with water and then in the form of rain or snow, enriches the top layer of the soil. Plants assimilate the iodine from soil and thus form an important source of iodine for humans as well as animals. However, soil erosions caused by deforestation and flash floods, continued rain and snow fall, changing courses of rivers lead to depletion of iodine rich soil and thus making it iodine deficient. Foods that are grown in such soils are deficient in iodine, and communities solely subsisting on such foods get exposed to iodine deficiency.

11.3.3. The problem of IDD in India

In India, the classical endemic belt of IDD extends from the State of Jammu and Kashmir in the North, through parts of Punjab, Haryana, Himachal Pradesh, Uttar Pradesh, Northern part of Bihar, and West Bengal to North-Eastern states. Encouraged by the results of iodized salt supplementation experiment in *Kangra* valley of Himachal Pradesh, Government of India (GOI), in 1962, launched the National Goitre Control Programme (NGCP).

Nutrition Foundation of India, in 1981 evaluated National Goitre Control Programme⁵. In view of scientific reports of widespread problem of IDD, GOI in 1984, launched the programme of Universal Iodization of salt, with an objective of iodizing entire edible salt in the country in a phased manner, from 1986-87. In 1988, PFA Act was amended to specify that iodized salt should have iodine in the concentration of at least 30 ppm at production level and at least 15 ppm at the consumer level. Subsequently, the Indian Council of Medical Research Task Force study carried out during 1989, in 11 districts of nine States revealed goitre prevalence ranging from a low of 6.2% in Mirzapur to a high of 65.8% in Dibrugarh. Prevalence of cretinism (a severe form of IDD), ranged from 0.1% in Gorakhpur to a high of 6.1% in Central Manipur⁶.

A country-wide study was carried out by NIN and DGHS in 2003 in 40 districts to assess the impact on the prevalence of IDD, particularly in the districts with higher levels of endemicity before universal iodization was introduced. This study revealed that the overall prevalence of total goitre registered a significant decline from 14-69% during 1984-94 to 2-40% in 2003, especially in the north-eastern regions⁷. The prevalence of total goitre was $\geq 10\%$ in half of the districts and $\geq 5\%$ in 37 out of 40 districts surveyed. The median urinary iodine excretion level among 6-11 year children was

observed to be <100 µg/L only in 9 out of 40 districts. State level surveys have also indicated similar results suggesting existence of iodine deficiency throughout India⁸⁻¹⁰.

Recent scientific surveys further identified IDD endemic areas in almost all the States in peninsular India, which are termed as Extra-Himalayan foci of IDD. According to Director-General of Health Services, it has been observed that 263 out of 324 districts surveyed, and 5 Union Territories are endemic for IDD¹¹. Living on the sea coast does not guarantee iodine sufficiency and significant pockets of iodine deficiency have been reported from coastal regions in different parts of the country.

Unlike nutrients such as iron, calcium or the vitamins, iodine does not occur naturally in specific foods; rather, it is present in the soil and is ingested through foods grown on that soil. Iodine deficiency results when there is lack of iodine on the earth's crust. It cannot be eliminated by changing dietary habits or by eating specific kinds of foods grown in the same area.

Iodine deficiency remains the single greatest cause of preventable brain damage and mental retardation worldwide. Considering the magnitude of the problem, GOI decided to universalize the dietary intake of the specified quantity of iodine¹². The Prevention of Food Adulteration Act, Government of India was amended to ban the sale of un-iodized salt for human consumption with effect from May 17, 2006.

Sources of Iodine

About 90% of the iodine requirement is met through food, while the rest is obtained through drinking water. In India, added iodized salt is the largest contributor of salt intake and thereby iodine, in the Indian dietaries¹³. The NNMB data from rural (2011-12)¹⁴ and urban India (2014-15)¹⁵ report a higher average salt intake of 7g/day in adult men and women (18-60y), while it ranged from 2-6 g/d in the younger and older age groups. A more recent systematic review published in 2017¹⁶ that reviewed 20 studies conducted in different states in urban and rural areas of India from 1986-2014, report a mean weighted population salt intake of 10.98 g/day with no evidence of a change over time. Revised standards proposed by FSSAI¹⁷ on mandatory fortification of all edible salt with iodine at 15-30 ppm (15-30 mg/kg), as part of universal salt iodization strategy to prevent iodine deficiency disorders, is in place in India. This amount is close to the amount of daily salt intake considered by the previous committee. Therefore, the level of salt intake is retained in this report as well. Therefore, daily intake of 10 g of iodized salt should be able to provide about 150-300 µg of iodine (if adequately iodized and stored properly), in addition to iodine present in foods consumed. The studies carried out by Longvah *et al*¹⁸, on food, water and salt samples from 48 districts of 29 Indian states reported a mean iodine content of 32±24.1 µg/100g in 140 most commonly consumed representative recipes prepared using iodized salt, 31±16 ppm of iodine in 116 household iodised salt samples and 9±7.8 µg/L in 82 drinking water samples.

Stability of Iodine during cooking

It is estimated that iodine in foods is lost in varying amounts during cooking and depends on the type of cooking processes employed and the time of addition of salt. The studies carried out by Longvah *et al*¹⁸, on 140 most commonly consumed representative recipes prepared using iodized salt, from 48 districts of 29 states, reported a mean content of iodine of 32±24.1 µg/100g and an average retention of 60±21% though with a wide variation. Similar observations by Rana and Raghuvamshi¹⁹ from Gujarat and Dodd *et al*²⁰ from Mumbai revealed about 40 to 70% loss of iodine in various types of preparations wherein iodized salt was used. Goindi *et al*²¹ found that the mean losses of iodine during different cooking procedures were 20-40% depending on the type of

cooking (11.3.13 (20); 11.3.14(21)). Considering these studies a 40% loss of iodine on an average maybe more prudent to be considered.

Daily requirement of Iodine

The normal daily dietary intake of iodine by an adult in an iodine sufficient region is about 100 to 150 µg, which is readily absorbed from the gut. Kidneys excrete excess of iodine ingested. The levels of urinary excretion of iodine correlate with daily ingestion and hence it is used as an index/biomarker of iodine status of the community or the target population groups.

Considering the current salt intake as 10 g, intended to provide about 150-300 µg of iodine, cooking losses of about 40% and 70% absorption (as indicated in the previous RDA publication), about 63-126 µg of iodine will be provided in the usual diets in India. This quantity is broadly comparable to the daily physiological needs of the body.

Requirements for Iodine

Due to paucity of data from within the country, the present committee adopted the methodologies suggested by IOM²² and FAO/WHO²³ to estimate the requirements of iodine for Indians and are presented in Table 11.3.1.

Iodine requirements of Infants

The IOM recommended an adequate intake (AI) of 110 µg/day for 0-6 month children. This was based on an average daily intake of 780 ml human milk, median concentration of 146 µg/L of iodine and the average urinary excretion of 12.7 µg/kg/day observed in positive iodine balance studies among full term infants during early infancy. For Indians, considering an average intake of 700 ml/day of human milk and assuming a similar concentration of iodine in human milk, the iodine intake calculated would be 102 µg/day. FAO/WHO recommends an iodine intake of 15 µg/kg/day for 0-12 month old infants, based on positive iodine balance studies in term infants in order to achieve the required iodine stores in the thyroid gland. Considering an average weight of 5.8 kg in 0-6 months age category in India, the iodine requirement calculated is 87 µg/day. Therefore, considering the higher estimate of 102 µg/day, the AI suggested for 0-6 m old infants in India is set at 100 µg/day. The AI set for 7-12 month Indian infants is 130 µg/day, which has been extrapolated from the AI of the younger infants considering an average human milk intake of 600ml/day and using the metabolic weight ratio method suggested by IOM, i.e., AI for 7-12m infant = AI set for 0-6 m infant x F, where F = (reference weight of 7-12m infant / reference weight of 0-6m)^{0.75}.

Iodine requirements of Children and Adolescents

EARs for children have been estimated based on the methodology suggested by IOM and FAO/WHO, that is, by extrapolating down from the adult EAR using the metabolic weight ratio method including growth. EAR for child = EAR set for adult x F, where F = [(reference weight of child / reference weight of adult)^{0.75} x (1+growth factor (GF)]. The GF used were

GF = 0.57 for ages up to 1 years,
0.25 for ages 1 to 3,
0.06 for ages 4 to 6,
0.13 for ages 7 to 9,
In ages 10 to 15 – 0.11 for boys and 0.08 for girls,
In ages 16 to 18 – 0.08 for boys and 0.03 for girls.

The EARs calculated for children and adolescents are presented in Table 11.3.1. The RDA was computed considering a CV of 20%.

Positive iodine balance studies in 1.5-2.5y children reported in the IOM document, indicate a median iodine intake of 64 µg/day, while the requirement calculated for 1-3y Indian children, using the above formula, was 35 µg/day. The RDI for iodine suggested by FAO/WHO is 6 µg/kg/day, based on radio iodine uptake and urinary iodine excretion. Considering the average weight of 1-3 y Indian child as 11.7 kg, the RDA calculated would be 70 µg/day and EAR back calculated would be 50 µg/day. Considering the higher estimate, which indicates a positive iodine balance, the EAR set for Indian 1-3y children is 65 µg/day after rounding. The RDA set is 91 µg/day, rounded to 90 µg/day, considering a CV of 20%.

For the 4-6y and 7-9y children, iodine balance studies in the IOM document report that intake of 20-40 µg/day resulted in a negative iodine balance (-23 to -26 µg/day) which suggests that a minimum of 66 µg (40+26) rounded to 65 µg of iodine would be required to maintain balance. The requirement calculated using the formula referred above, adjusting for Indian weights and growth, was 40 and 60 µg/day respectively for 4-6y and 7-9y Indian children. As per recommendations of FAO/WHO, the calculated EAR was 78 µg/day for 4-6y child (based on 6 µg/kg/day) and 72 µg/day for 7-9y child (based on 4 µg/kg/day) considering the reference average weight of 18.3 kg and 25.3 kg for 4-6y and 7-9y Indian child respectively. Therefore, considering the higher estimates, the EAR for 4-6y Indian children is set at 78 rounded to 80 µg/day and the EAR for 7-9y old Indian child is set at 72 rounded to 80 µg/day. The RDA is set at 112 rounded to 120 µg/day for the two age groups, considering a CV of 20%.

For 10-12y adolescents, iodine balance studies indicate an intake of 55 µg/day, while the calculated intake based on extrapolation down from adults reported in the IOM document indicates 73 µg/day. When the Indian values were replaced in the formula, the calculated iodine intake requirement was 70 µg/day. As per the FAO/WHO requirement of 4 µg/kg/day and the reference Indian body weight, the EAR calculated is 102 rounded to 100 µg/day. Therefore considering the higher estimate, the EAR and RDA calculated for Indian 10-12y children is 100 and 140 µg/day (rounded to 150 µg/day) respectively.

With reference to 13-15y and 16-18y adolescents, the EAR recommended by IOM was 95 µg/day based on extrapolation down from adults, which was higher than that observed in the iodine balance studies (58 µg/day). The requirement based on IOM methodology (extrapolation from adult values) were calculated using Indian reference body weights of 13-18y age group and the EAR thus estimated was 100 µg/day for both the age groups i.e. 13-15y and 16-18y, independent of gender. Using the methods adopted by FAO/WHO (2 µg/kg/day), the EAR calculated was approximately 72 µg/day and 86 µg/day respectively for the two age groups. However, considering the higher estimate observed using IOM methodology, the EAR set for Indian 13-15y and 16-18y children is rounded to 100 µg/day and the RDA is set at 140 µg/day, which is rounded to 150 µg/day.

Iodine requirements of Adults

The EAR of 95 µg/day for adults was set by IOM, based on accumulation of radioiodine in the thyroid gland in turnover studies observed in euthyroid individuals and positive iodine balance studies in adults with normal thyroid function. Considering a CV of 20% due to variations in experimental design, the RDA calculated was 133 µg/day, rounded to 150 µg/day. The FAO/WHO recommended an intake of 150 µg/day for adults based on observations such as, the suggested intake value corresponded to the normal urinary iodine concentration value of 100 µg/L, the intake level was required to maintain iodine stores in the thyroid above the critical threshold of 10mg

required for optimal thyroid hormone synthesis, the intake level was necessary to maintain the plasma iodine above the critical limit of 0.1 µg/dL that was likely to be associated with onset of goitre. Observations from controlled studies considered in the FAO/WHO document revealed that the radioiodine uptake, thyroidal iodine content or the onset of goitre reached a steady state at an UIC of ≥ 100 µg/L that corresponds to an iodine intake of 150 µg/day, which is similar to the RDA value suggested by IOM. There is paucity of such data in the Indian population. Hence, based on the literature cited above, and assuming similarity even in the Indian context, the EAR for Indian Adults ≥ 19 y (Men and Women) is set at 95 µg/day and the RDA at 133 µg/day, rounded to 150 µg/day, considering the CV of 20%.

Iodine requirements of Pregnant and Lactating Women

The iodine EAR of 160 µg/day for pregnant women is suggested by IOM, based on information from iodine balance studies, iodine supplementation studies and thyroid iodine content of the new-borns and an RDA of 224 µg/day, rounded to 220 µg/day is recommended based on a CV of 20%. The technical committee of FAO/WHO recommended an iodine intake of 250 µg/day in pregnancy, based on normal median urinary concentration levels of iodine (150 µg/L), in an iodine sufficient population. These calculations were based on the average urinary volume of 1.5 L/day in pregnant women and assuming that 90% iodine ingested is excreted in urine, i.e., for every 100 µg of iodine ingested, 90 µg is excreted at a concentration of 60 µg/L and is closer to the RDA suggested by IOM. Therefore, for Indian pregnant women, the higher estimate of 250 µg/day for the RDA is considered. The EAR is back calculated to 179 µg/day, rounded to 180 µg/day, considering a CV of 20%.

The requirement of iodine for lactating women was fixed by IOM, based on the EAR of adult NPNL women (95 µg/day) plus loss of iodine in breast milk (114 µg/day) and the EAR calculated was 209 µg/day and considering a CV of 20%, the RDA was 292 µg/day rounded to 290 µg/day. The FAO/WHO suggested an iodine intake of 250 µg/day during lactation in order to bring the urinary iodine concentration back to normal level (≥ 100 µg/L) as well as ensure adequate secretion of iodine in human milk required by the infants. Therefore, for the Indian lactating women, the EAR and RDA is set at 197 µg/day rounded to 200 µg/day and 280 µg/day (considering 20% CV) respectively based on the higher estimates suggested by IOM after adjusting for the human milk intake by Indian infants (EAR of 95 µg/day of NPNL plus 102 µg/day from 700 ml of human milk).

The IOM also indicates that there is no data on differences in requirements between genders in adults or on any changes with advancing age.

The RDA suggested by the previous committee that has been adopted from WHO-UNICEF-ICCID²³⁻²⁶ is similar or closer to that suggested by the present committee.

Tolerable Upper Limit (TUL) of iodine intake

Excess of iodine ingestion can be harmful, which may inhibit the synthesis of thyroid hormones by the thyroid. This iodine-induced hypothyroidism is known as ‘Wolff-Chaikoff’ effect, the manifestation of which depends on level of iodine intake before exposure to iodine excess. A tolerable upper limit (TUL) of 1100 µg/day has been suggested for adult men and women by IOM based on elevated TSH (suggestive of thyroid dysfunction) observed in normal/euthyroidic adult men and women with an overall intake of 1700 µg/day (1500 µg from iodine supplements and 200 µg from diet)²². This TUL level is also adopted by the present committee due to lack of evidence from India. The TUL of 1100 µg/day is based on a Lowest Observed Adverse Effect Level (LOAEL) of 1700 µg daily intake of iodine both from diet and supplements per day and an uncertainty factor (UF) of 1.5 (1700/1.5=1133, rounded down to 1100 µg/day). The TUL values for children (1-9y) and adolescents (10-18y) are extrapolated from adult values after adjusting for reference body weights suggested in Table 3.6 of this document. TUL for child or adolescent =

(TUL for adult x Reference body weight of child or adolescent /reference average body weight of adults). The values have been rounded downwards to the nearest 100. Values similar to adult NPNL women are considered for pregnant and lactating women, as there is no current evidence on altered susceptibility to iodine excess. It was not possible to determine TUL for Infants below 1y, as there is no data on adverse effects.

Table 11.3.1 EAR and RDA of iodine for various physiological groups

Age Group	Reference Body Weight (Kg)	Category (ICMR 2020)	ICMR 2020 ($\mu\text{g}/\text{day}$)			RDA 2010 ($\mu\text{g}/\text{day}$)
			EAR	RDA	TUL**	
Men	65	>18y	95	150	1100	150
Women (NPNL)	55	>18y	95	150	1100	150
Pregnant women		>18y	180	250	1100	250
Lactating women		>18y	200	280	1100	250
Infants	5.8	0-6 m	100 (AI)	100 (AI)	ND	Breast milk
Infants	8.5	6-12 m	130 (AI)	130 (AI)	ND	90
Children	11.7	1-3y	65	90	200	90
Children	18.3	4-6y	80	120	300	90
Children	25.3	7-9y	80	120	400	120
Boys	34.9	10-12y	100*	150	600	120
Girls	36.4	10-12y	100*	150	600	120
Boys	50.5	13-15y	100*	150	900	150
Girls	49.6	13-15y	100*	150	900	150
Boys	64.4	16-18y	100*	150	1100	150
Girls	55.7	16-18y	100*	150	1100	150

* The values are pooled for gender; ** The TUL recommended by IOM, 2001 for adult men, adult NPNL women, pregnant women and lactating women was considered. Average adult reference body weight (men and NPNL women) of 60 kg was considered to extrapolate TUL values for children and adults based on reference body weights.

Status of iodine content of salt

The level of salt iodisation should provide a physiological intake of 100-150 $\mu\text{g}/\text{day}$, which would bring the median urinary iodine excretion (UIE) level within a range of 100-200 $\mu\text{g}/\text{L}$. Earlier, the recommended content of iodine in edible salt was ≥ 15 ppm, while it has been more recently revised to 15-30 ppm by the FSSAI¹⁷. NFHS 4²⁷ reported access to iodized salt (using spot test) in 93% of the households. Analysis of a total of 27,123 salt samples by iodometric titration method from 29 states and 7 Union territories²⁸ and six different geographical zones²⁹ revealed that 76% of the samples had >15ppm iodine and 41% had between 15-30ppm, indicating that the iodized salt manufacturers in the country were iodizing the salt with recommended quantity.

Status of Iodine intake of the population

The epidemiological criteria usually employed to assess iodine nutriture in different population groups is based on the iodine content of cooking salt/household coverage of adequately

iodized salt and the median urinary iodine concentration (mUIC) in the target groups. As per the revised standards recommended by FSSAI¹⁷, all forms of edible salt should have 15-30 ppm of iodine. According to WHO³⁰, the desired adequate mUIC should be between 100-200 µg/L for children, adolescents, adult men, adult women & lactating mothers and between 150-250 µg/L for pregnant women.

However, with respect to iodine content in salt, in most of the population based studies, salt has been tested using rapid testing kits that do not indicate an absolute value. At best it either indicates if the salt is iodized or not or gives a range with in which the iodine value is present. The two most recent large population based studies used the iodometric titration method to estimate iodine content in cooking salt. These studies conducted pan India in 29 states and 7 Union territories²⁸ or in six geographical zones²⁹ identified that almost 76% of households were using adequately iodized salt. Among them, 13 states reached universal salt iodization (i.e., more than 90% of households had access to iodized salt). Another recent study done in the rural, urban and urban slum areas in north and south India used the best preferred method to measure salt intake i.e., the 24Hr urinary salt excretion method and reported a mean weighted salt intake of 9-10 g/d³¹ which corroborates well with the level of intakes derived from the systematic review¹⁶.

According to mUIC, the biomarker used for iodine status, the India Iodine study²⁸, conducted during 2018-19, among 21,406 households from 29 states and 7 UTs, the mUIC in WRA women was 178 µg/L, while it was 173 µg/L in both the pregnant and lactating women, indicating adequate iodine status. In another recently (2019) completed CNNS study³², iodine status (mUIC) was adequate among preschool children (215 µg/L), school-age children (175 µg/L) and adolescents (173 µg/L), pan India, except in Tamil Nadu where mUIC was >300 µg/L in all the three age groups. The reason for this remains elusive.

References

1. Hetzel BS. The Story of Iodine Deficiency - An International challenge in Nutrition. Oxford Publications, 1989.
2. Pandav CS and Rao AR. Iodine deficiency disorders in livestock - Ecology & economics. Oxford Publications, 1977.
3. Sooch SS, Deo MG, Karmarkar MG, Kochupillai N, Ramachandran K, Ramalingaswami V. The Kangra valley experiment: prevention of Himalayan endemic goitre with iodinated salt. *Acta Endocrinologica Supplementum*. 1973 Oct; 179:110.
4. National Nutrition Policy, Government of India, Department of Women & Child Development, Ministry of Human Resource Development, New Delhi, 1993.
5. Gopalan C. The National Goitre Control Programme - A Sad Story, Bulletin of Nutrition Foundation of India, July 1981.
6. Epidemiological Survey of Endemic Goitre and Endemic Cretinism. An Indian Council of Medical Research Task Force Study, 1989.
7. Current status of IDD in select districts of different regions of the country. National Institute of Nutrition, Hyderabad, 2003.
8. Sankar R, Moorthy D, Pandav CS, Sangita Tiwari J, Karmarkar MG. Tracking progress towards sustainable elimination of iodine deficiency disorders in Bihar. *Indian Journal of Paediatrics*. 2006; 73(9):799-802.
9. Ategbo EA, Sankar R, Schultink W, van der Haar F, Pandav CS. An assessment of progress toward universal salt iodization in Rajasthan, India, using iodine nutrition indicators in school-aged children and pregnant women from the same households. *Asia Pacific Journal of Clinical Nutrition*. 2008;17(1):56-62.
10. Patro BK, Saboth P, Zodpey S, Shukla A, Karmarkar MG, Pandav CS. Tracking progress toward elimination of iodine deficiency disorders in Jharkhand, India. *Indian Journal of Community Medicine*. 2008;33:182-5
11. IDD & Nutrition Cell, Directorate General of Health Services, Ministry of Health & Family Welfare, Government of India, New Delhi (2006).
12. Universalization of access to iodized salt – A mid-decade goal. The Salt Department, Ministry of Industry, 1994.
13. Johnson C, Santos JA, Sparks E, Raj TS, Mohan S, Garg V, Rogers K, Maulik PK, Prabhakaran D, Neal B, Webster J. Sources of dietary salt in north and south India estimated from 24 hour dietary recall. *Nutrients*. 2019 Feb; 11(2):318.
14. NNMB. Diet and nutritional status of rural population, prevalence of hypertension and diabetes among adults and infant and young child feeding practices. Report of third repeat survey. NNMB Technical Report No: 26. National Nutrition Monitoring Bureau, Indian Council of Medical Research-National Institute of Nutrition (ICMR-NIN), Hyderabad, 2012.
15. NNMB. Diet and Nutritional status of Urban Population in India and prevalence of obesity, hypertension, diabetes and hyperlipidemia in urban men and women- a brief NNMB urban nutrition report. NNMB Technical Report No.27. National Nutrition Monitoring Bureau, Indian Council of Medical Research-National Institute of Nutrition (ICMR-NIN), Hyderabad, 2017.
16. Johnson C, Praveen D, Pope A, Raj TS, Pillai RN, Land MA, Neal B. Mean population salt consumption in India: a systematic review. *Journal of hypertension*. 2017 Jan 1;35(1):3-9.
17. FSSAI. (Food Safety Standards Authority of India). Food Safety and Standards (Fortification of Foods) Regulations, 2018. In: The Gazette of India: Extraordinary (PART 111, Section 4). Ministry of Health and Family Welfare (MoHFW), Govt. of India. New Delhi, India. 2018. https://fssai.gov.in/upload/uploadfiles/files/Gazette_Notification_Food_Fortification_10_08_2018.pdf.

18. Longvah T, Toteja GS, Bulliyaa G, Raghuvanshi RS, Jain S, Rao V, Upadhyaa A. Stability of added iodine in different Indian cooking processes. *Food chemistry*. 2012 Feb 15; 130(4):953-9.
19. Rana R, Raghuvanshi RS. Effect of different cooking methods on iodine losses. *Journal of food science and technology*. 2013 Dec 1; 50(6):1212-6.
20. Dodd NS and Swaroop Dighe. Iodine content of diets of the people of different regions living in Bombay. *Journal of Food Science & Technology*. 1993; 30(2):134-136.
21. Goindi G, Karmarkar MG, Kapil U, Jagannathan J. Estimation of losses of iodine during different cooking procedures. *Asia Pac J Clin Nutr*. 1995 Jun; 4(2):225-7.
22. Institute of Medicine (US) Panel on Micronutrients. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. 8, Iodine. 2001; 258-289.
23. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.
24. WHO/UNICEF/ICCIDD. Recommended iodine levels in salt and guidelines for monitoring their adequacy effectiveness. World Health Organization, United Nations Children's Fund and International Council for Control of Iodine Deficiency Disorders, 1997.
25. WHO Secretariat, Andersson M, de Benoist B, Delange F and Zupan J. Prevention and control of iodine deficiency in pregnant and lactating women and in children less than 2-years-old: conclusions and recommendations of the Technical Consultation. *Public Health Nutrition*. 10(12A):1606-11, 2007.
26. ICMR. A draft report of the expert committee on to examine the allowance of vitamins/minerals of more than one RDA in health or dietary supplements and nutraceuticals and safe upper limits. Indian Council of Medical Research, New Delhi, India. 2018.
27. International Institute for Population Sciences (IIPS) and ICF. National Family Health Survey (NFHS-4), 2015-16: India. Mumbai: IIPS. 2017.
28. Nutrition International, ICCIDD, AIIMS (New Delhi) and Kantar. India Iodine Survey 2018-19 National Report, New Delhi, India. 2019.
29. Pandav CS, Yadav K, Salve HR, Kumar R, Goel AD, & Chakrabarty A. High national and sub-national coverage of iodised salt in India: Evidence from the first National Iodine and Salt Intake Survey (NISI) 2014–2015. *Public health nutrition*. 2018; 21(16), 3027-3036.
30. WHO/UNICEF/ICCIDD. Assessment of iodine deficiency disorders and monitoring their elimination: a guide for programme managers, 3rd ed. Geneva: World Health Organization; 2007. (http://whqlibdoc.who.int/publications/2007/9789241595827_eng.pdf).
31. Johnson C, Mohan S, Rogers K, Shivashankar R, Thout SR, Gupta P, He FJ, MacGregor GA, Webster J, Krishnan A, Maulik PK. Mean dietary salt intake in urban and rural areas in India: a population survey of 1395 persons. *Journal of the American Heart Association*. 2017 Jan 6; 6(1).
32. Ministry of Health and Family Welfare (MoHFW), Government of India, UNICEF and Population Council. Comprehensive National Nutrition Survey (CNNS) National Report. New Delhi. 2019. Available from <https://nhm.gov.in/WriteReadData/1892s/1405796031571201348.pdf>.

12. WATER SOLUBLE VITAMINS

12.1. THIAMINE

INTRODUCTION

Thiamine is a water-soluble vitamin and occurs in three forms as thiamine monophosphate (TMP), thiamine pyrophosphate (TPP) and free thiamine. In animals, TPP is involved in oxidative decarboxylation of α -ketoacids like pyruvate, α -ketoglutarate and ketoacids derived from branched chain amino acids (leucine, isoleucine and valine). The products of these reactions - acetyl CoA, succinyl CoA and appropriate derivatives of branched chain amino acids play important roles in carbohydrate and lipid metabolism. TPP is also involved in the transketolase reaction- an important reaction of the pentose phosphate pathway. This pathway generates pentose phosphates needed for synthesis of nucleic acids and NADPH used for fatty acid synthesis, and generation of reduced glutathione^{1,2}.

Dietary sources

Rich sources of thiamine include whole grain cereals, nuts & oil seeds, legumes, green leafy vegetables (spinach, fenugreek leaves, gogu), beans, capsicum, peas, apricot, custard apple, green chillies, garlic, spices & condiments, organ meats, pork, liver, eggs, and milk & milk products³. Rice bran, wheat bran and wheat germ are also rich sources of thiamine. Thiamine losses occur during milling and polishing of grains, cooking and preservation of foods and processes like baking (29-30%), pasteurization of milk (10-20%) and ultraviolet irradiation. On an average, about 40-50% of the vitamin present in raw foods is lost during processing and cooking as practised in Indian homes. Tannic acid contained in foods like tea and betel nut destroys thiamine.

12.1.1. Thiamine intake and deficiency in India

Countrywide surveys conducted by the National Nutrition Monitoring Bureau (2012) in rural populations show that the median intake of the vitamin⁴ is comparable to earlier RDA⁵. About 77% households meet $\geq 70\%$ RDA, and only 10% consume less than 50% of the RDA. In urban population, the average intake of the vitamin is 1.2 mg/day for adult men and 1.0 mg/day for adult women (NNMB urban survey unpublished observations). Recently, a study among the apparently healthy urban adult population reported the median intake of thiamine as 1.2 mg/day (men) and 0.8 mg/day (women)⁶.

Inadequate intake is the major cause of thiamine deficiency in India. Relatively higher energy intakes from foods deficient in thiamine would reduce the vitamin/energy ratio below the requirement (0.5 mg/1000 kcal) and precipitate thiamine deficiency. Deficiency also occurs with the intake of raw fish containing the enzyme thiaminase that destroys thiamine. Secondary thiamine deficiency occurs in chronic diseases where there are several metabolic changes. Dry (paralytic) and wet beriberi (oedematous) is the ultimate disease manifestation. Alcoholism associated with poor quality diet, low in thiamine leads to Wernicke-Korsakoff syndrome. Milder deficiency may result in neuropathies manifested by tingling and numbness in the extremities. While beriberi used to be rampant in India in the early decades of this century, now its prevalence is rare. This change cannot be explained entirely on the basis of dietary improvement. Ban on excessive polishing of rice and dietary diversification may have contributed to it. In one study, young women from Hyderabad and Mumbai did not show any evidence of thiamine deficiency as judged by the erythrocyte transketolase activity coefficient (ETK-AC) test⁷. A recent study from the National Institute of Nutrition estimated the different forms of thiamine (TMP, TPP and thiamine) directly in whole blood by reverse-phase HPLC

method with fluorescence detection among the apparently healthy urban adult population. Considering TPP (the biological active form of thiamine) levels <66 nmol/L as deficient, the study showed that 10% women and 13% men were deficient⁶. Though, healthy adults have an estimated total body thiamine concentration of 25–30 mg, due to rapid metabolism and turnover of thiamine, and an estimated biological half-life of 9–18 days, regular recommended dietary intake is necessary⁸. Large single doses are absorbed poorly.

Thiamine status is often assessed by measuring the urinary excretion of the vitamin in a 24-h collection of urine or before and after the load of thiamine. Measurement of ETK activity and its *in vitro* stimulation (TPP effect) is more commonly used index for assessing thiamine status. TPP effect greater than 15% (ETK-AC >1.15) indicates thiamine deficiency. Blood thiamine levels have received little attention because of the difficulty in measuring small amounts of the vitamin.

12.1.2. Previous ICMR recommendations

a. Adults

Since the vitamin is needed for energy utilization, its requirement is related to calorie intake. Several early studies showed that beriberi is seen among populations whose thiamine intake is less than 0.12 mg/1000 kcal (4184 kJ)⁹. However, intakes above 0.3 mg/1000 kcal are consistent with no signs and symptoms of beriberi^{9,10}. Measurement of biochemical indices such as intake at which there is a steep increase in urinary excretion of thiamine (at low levels of intake very little intact thiamine is excreted in urine), and maximum levels of ETK activity, in systematic depletion-repletion studies done on human volunteers in India and elsewhere indicate 0.25–0.35 mg/1000 kcal as the minimum requirement of thiamine for adults^{9–11}. Similarly, Institute of Medicine (IOM, 1998), estimated the requirement based on metabolic studies (depletion-repletion studies) taking into account the ETK activity, urinary thiamine excretion and their dietary intakes¹². Making allowance for cooking losses, National Research Council (NRC, USA) has suggested a dietary allowance of 0.5 mg/1000 kcal⁹. In 1989, ICMR¹⁰ had made similar recommendation and in the absence of any new evidence, the same were retained in 2010 recommendations. However, minimum daily allowance of 1.0 mg of thiamine is recommended even if the calorie intake is less than 2000 kcal. While ICMR recommendations for adults are related to physical activity, FAO/WHO¹³ and NRC⁹ give a single figure for adult men and women (Table 12.1a).

b. Pregnancy and lactation

Earlier, ICMR Committee recommended 0.5 mg thiamine/1000 kcal (0.2 mg–300 kcal) assuming that the additional calorie allowance will take care of the extra thiamine requirement during pregnancy. The recommendations of FAO/WHO and NRC (1989) are slightly higher (Table 12.1a). FAO/WHO recommends additional 0.3 mg during pregnancy.

A well-nourished Western woman secretes about 0.2 mg thiamine per day in breast milk. Based on this, NRC⁹ has recommended additional thiamine allowance of 0.5 mg throughout lactation to accommodate thiamine secretion in milk and metabolism of additional calories. Indian mothers from low-income groups secrete only 15 µg thiamine/100 ml milk^{9,14} and after supplementation it goes up to 20 µg thiamine/100 ml. With a milk output of 700 ml, the maximum secreted through milk would be 0.14 mg. The lower level of milk thiamine even after supplementation may be because of inadequate utilization due to associated calorie and protein deficiency. The additional allowance recommended by the ICMR^{5,10} on the basis of additional calorie allowance is 0.3 mg for 0–6 months of lactation and 0.2 mg for 6–12 months. This level tends to be lower than the recommendations of FAO/WHO as well as NRC (1989) (Table 12.1a).

Table 12.1a: Comparison of ICMR, FAO/WHO and NRC Recommendations for Thiamine

Category / Age		ICMR 2010	FAO/WHO 2004	NRC 1989	IOM 2006
		mg/day			
Infants	0-6 m	0.2	0.2	0.3	-
	6-12 m	0.3	0.3	0.4	-
Children	1-3 y	0.5	0.5	0.7	0.5
	4-6 y	0.7	0.6	0.9	0.6 (4-8 y)
Boys	7-9 y	0.8	0.9	1.0 (7-10 y)	
	10-12 y	1.1	1.2	1.3 (11-14 y)	0.9 (9-13 y)
	13-15 y	1.4	1.2		1.2 (14-18 y)
Girls	16-18 y	1.5	1.2	1.5 (15-18 y)	
	10-12 y	1.0	1.1	1.3 (11-14 y)	0.9 (9-13 y)
	13-15 y	1.2	1.1		1.0 (14-18 y)
Men	16-18 y	1.2	1.1	1.3 (15-18 y)	
	Sedentary-1.2 Moderate-1.4 Heavy-1.7		1.3	1.7 (19-50 y) 1.4 (50+ y)	1.2
	Women NPML	Sedentary-1.0 Moderate-1.1 Heavy-1.4		1.3 (19-50 y) 1.2 (50+ y)	1.1
Pregnant		+ 0.2	1.4	1.5	1.4
Lactating	0-6 m	+0.3	1.5	1.6	1.4
	6-12 m	+0.2	1.5	1.6	1.4

c. Infants and children

The requirement for infants less than 6 months of age is generally computed from the quantity available through breast milk of healthy well-nourished mothers. Assuming a level of 20 µg/100 ml and 700 ml of breast milk output, the RDA for infants, 0-6 months is 0.3 mg/1000 kcal or 0.2 mg/day.

12.1.3. Current recommendations for thiamine

Estimation of thiamine requirement based on erythrocyte transketolase activity coefficient (ETK-AC)

The requirement of thiamine is estimated by plotting EKT-AC values versus dietary thiamine intake data obtained from healthy human adult population studies²⁸⁻³⁶. Average requirement (EAR) was calculated from regression analysis (Figure 12.1a). The acceptable cut-off used for ETK-AC is 1.15³⁷. The RDA is defined as equal to the EAR plus twice the standard deviation (SD). Standard deviation of requirements is calculated from the EAR and the Coefficient of Variation (CV). Assuming a CV of 10%, as the information is not available on the SD of the requirement for thiamine, the RDA for thiamine was then determined as EAR+2 (EAR*0.1). The simulated requirement distribution for thiamine based on the ETK-AC cut-off with assumed normal distribution for adult men and women is presented in Figure 12.1b, which shows an EAR of thiamine is 1.18 and corresponding RDA is 1.41 for adult men. Similarly, EAR of thiamine is 1.14 and corresponding RDA is 1.36 for adult women.

Figure 12.1a. The ETK-AC in relation to dietary intake in both genders

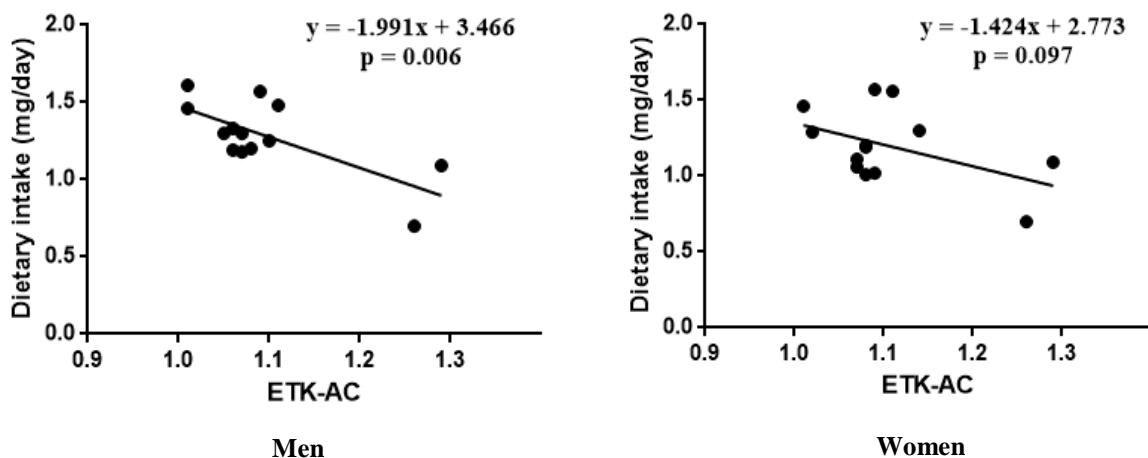
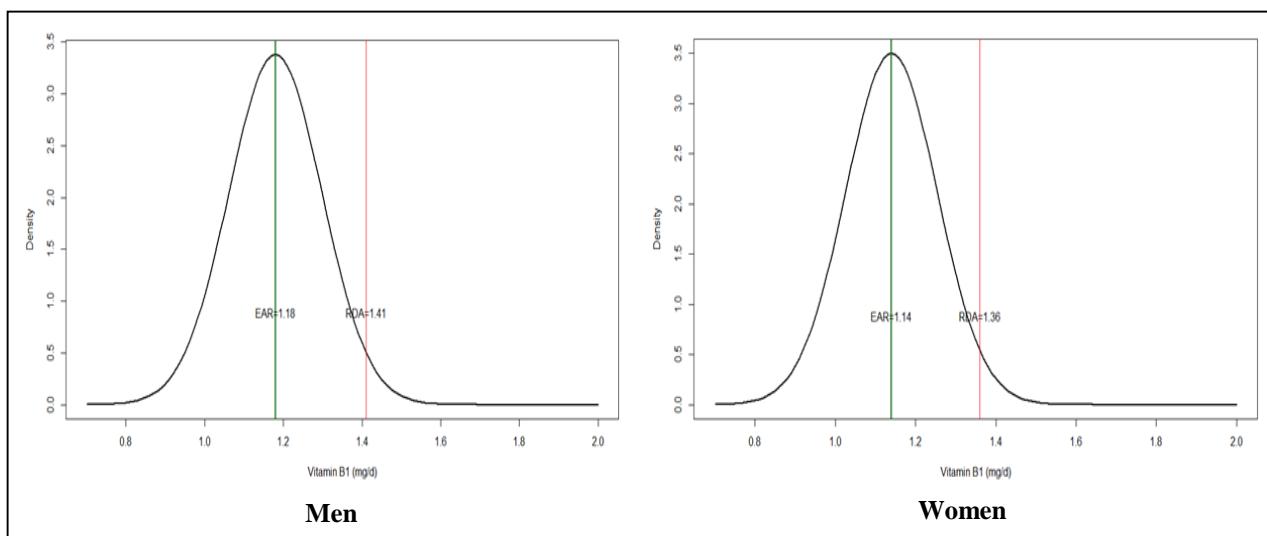


Figure 12.1b. Distribution of the requirements in both genders



During pregnancy, the additional allowance of 0.2 mg of thiamine based on the additional calorie allowance (350 kcal) of current recommendations would take care of maternal and foetal growth. During lactation, Indian mothers with a milk output of 700 ml may secrete a maximum of 0.14 mg of thiamine through milk. The additional allowance of 0.33 mg of thiamine for 0-6 months of lactation and 0.29 mg of thiamine for 6-12 months based on the additional calorie allowance [600 kcal (0-6 months), 520 kcal (6-12 months)] of current recommendations. The current recommendations for thiamine across age groups are given in Table 12.1b.

12.1.4. Tolerable upper limit for thiamine

There is no Tolerable Upper Limit (TUL) identified on the data available. While a few studies in India used 1-3 mg/ day of thiamine involving 5-15 year boys/ girls or women, other studies have used 100-600 mg/day on adults under various conditions. Further, the IOM (1998) also states that there is no adverse effect associated with excess thiamine intake from foods or supplements in healthy individuals¹². The evidence from the limited data is not sufficient for a quantitative risk assessment and therefore no TUL can be derived for thiamine.

Table 12.1b: Current recommendations for Thiamine across age groups

Age group (y)	EAR (mg/d)	RDA (mg/d)
Infants (0-6m) (6-12m)	- -	0.2(AI) 0.4(AI)
Children (1-3 y) (4-6 y) (7-9 y)	0.6 0.8 1.0	0.7 0.9 1.1
Boys (10-12 y)	1.3	1.5
Girls (10-12 y)	1.2	1.4
Boys (13-15 y)	1.6	1.9
Girls (13-15 y)	1.3	1.6
Boys (16-18 y)	1.9	2.2
Girls (16-18 y)	1.4	1.7
Adult Men		
Sedentary	1.2	1.4
Moderate	1.5	1.8
Heavy	1.9	2.3
Adult Women		
Sedentary	1.1	1.4
Moderate	1.4	1.7
Heavy	1.8	2.2
Pregnant	1.6	2.0
Lactating (0-6m) (6-12m)	1.7 1.7	2.1 2.1

References

1. Manzetti S, Zhang J and van der Spoel D. Thiamin function, metabolism, uptake, and transport. *Biochemistry*. 2014; 53, 821–835.
2. Combs. The vitamins: fundamental aspects in nutrition and health. Elsevier Academic Press, Boston. 2018.
3. Longvah T, Ananthan R, Bhaskarachary K, Venkaiah K. In: Longvah T, editor. Indian Food Composition Tables: National Institute of Nutrition Hyderabad; 2017.
4. NNMB. Diet and nutritional status of rural population. Prevalence of hypertension and diabetes among adults and infants and young child feeding practices. NNMB-Report of the third survey Technical Report No.26. National Institute of Nutrition, Hyderabad. 2012.
5. ICMR. Nutrient requirements and recommended dietary allowances for Indians. A report of the expert group of the Indian Council of Medical Research National Institute of Nutrition Hyderabad; 2010.
6. Sivaprasad M, Shalini T, Reddy PY, Seshacharyulu M, Madhavi G, Kumar BN, Reddy GB. Prevalence of vitamin deficiencies in an apparently healthy urban adult population: Assessed by subclinical status and dietary intakes. *Nutrition*. 2019 Jul 1; 63:106-13.
7. WHO Task Force on oral contraceptives. Impact of hormonal contraceptives vis-a-vis non-hormonal factors on vitamin status of malnourished women in India and Thailand. *Human Nutrition. Clinical Nutrition*. 40C: 205-200, 1986.
8. European Food Safety Authority. Dietary reference values for thiamine. *EFSA Journal* 2016; 14(12):4653. doi: 10.2903/j.efsa.2016.4653.
9. National Research Council. Recommended dietary allowances 10thed. National Academy Press Washington DC. FAO/WHO Expert Consultation on Human Vitamin and Mineral Requirements. 1989.
10. ICMR. Nutrient requirements and recommended dietary allowances for Indians. A Report of the Expert Group of the Indian Council of Medical Research. National Institute of Nutrition, Hyderabad, 1989.
11. Bamji MS. Transketolase activity and urinary excretion of thiamin in the assessment of thiamin-nutrition status of Indians. *The American Journal of Clinical Nutrition*. 1970 Jan 1; 23(1):52-8.
12. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline. National Academies Press (US); 1998.
13. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.
14. Belavady B and Gopalan C. Effect of dietary supplementation on the composition of breast milk. *Indian Journal of Medical Research*. 1960; 48:518-523.
15. Sauberlich HE, Herman YF, Stevens CO, Herman RH. Thiamin requirement of the adult human. *American Journal of Clinical Nutrition*. 1979; 32: 2237–2248.
16. E J van der Beek, W van Dokkuma, M Wedela, J Schrijvera and H van den Berga. Thiamin, riboflavin and vitamin B6: impact of restricted intake on physical performance in man. *Journal of the American College of Nutrition*, 1994, Vol. 13, No. 6, 629-640.
17. Wood B, Gijsbers A, Goode A, Davis S, Mulholland J, Breen K. A study of partial thiamin restriction in human volunteers. *American Journal of Clinical Nutrition*. 1980; 33:848–861.
18. Ziporin ZZ, Nunes WT, Powell RC, Waring PP, Sauberlich HE. Thiamine requirement in the adult human as measured by urinary excretion of thiamine metabolites. *Journal of Nutrition*. 1965; 85:297–304.
19. N Tasevska, SA Runswick, A McTaggart and SA Bingham. Twenty-four-hour urinary thiamine as a biomarker for the assessment of thiamine intake. *European Journal of Clinical Nutrition*. 2008; 62, 1139–1147.
20. Gontzea I, Gorcea V, Popescu F. Biochemical assessment of thiamin status in patients with neurosis. *Annals of Nutrition and Metabolism*. 1975; 19(3-4):153-7.
21. Oldham HG. Thiamine requirements of women. *NYASA*. 1962 Apr; 98(2):542-9.

22. Oldham HG, Davis MV, Roberts LJ. Thiamine excretions and blood levels of young women on diets containing varying levels of the B vitamins, with some observations on niacin and pantothenic acid. *Journal of Nutrition*. 1946; 32:163–180.
23. Hathaway ML, Strom JE. A comparison of thiamine synthesis and excretion in human subjects on synthetic and natural diets. *Journal of Nutrition*. 1946; 32:1.
24. Tomiko Tsuji, Tsutomu Fukuwatari, Satoshi Sasaki, Katsumi Shibata. Urinary excretion of vitamin B1, B2, B6, niacin, pantothenic acid, folate and vitamin C correlates with dietary intakes of free-living elderly, female Japanese. *Nutrition Research* 30 (2010) 171–178.
25. Leo L Hardt and Eugene U. Still. Thiamin in Sweat. *Experimental Biology and Medicine*. December 1, 1941.
26. Eric J Vander Beek, Wim van Dokkum, Jaap Schrijver, Michel Wedel, Anthony WK Gaillard, Anneke Wesstra, Henk Van de Weerd and Ruud J J Hermus. Thiamin, riboflavin, and vitamins B-6 and C: impact of combined restricted intake on functional performance in man. *American Journal of Clinical Nutrition*. 1988; 48:1451-62.
27. Fukuwatari T, Yoshida E, Takahashi K, Shibata K. Effect of fasting on the urinary excretion of water-soluble vitamins in humans and rats. *Journal of nutritional science and vitaminology*. 2010; 56(1):19-26.
28. Nicholas P. O'Rourke, Valda W. Bunker, Anita J. Thomas, Paul M. Finglas, Angela L. Bailey, Barbara E. Clayton. Thiamine status of healthy and institutionalized elderly subjects: analysis of dietary intake and biochemical indices. *Age and Ageing*. 1990; 19:325-329.
29. J. Mataix, P. Aranda, C. Sañchez, M. A. Montellano, E. Planells and J. Llopis. Assessment of thiamin (vitamin B1) and riboflavin (vitamin B2) status in an adult Mediterranean population. *British Journal of Nutrition*. 2003; 90, 661–6.
30. Itoh R, Suyama Y. Socio-demographic factors and life-styles affecting micronutrient status in an apparently healthy elderly Japanese population. *Journal of Nutrition for the Elderly*. 1995 Jun 8; 14(2-3):39-54.
31. Feili Lo Yang, Pei-Chun Liao, Yung-Ying Chen, Jui-Line Wang and Ning-Sing Shaw. Prevalence of thiamin and riboflavin deficiency among the elderly in Taiwan. *Asia Pacific Journal Clinical Nutrition* 2005; 14 (3):238-243.
32. M.J. Costa de Carvalho JC. Guilland D. Moreau V. Boggio F Fuchs. Vitamin status of healthy subjects in Burgundy (France). *Annals of Nutrition Metabolism*. 1996; 40:24-51.
33. Pascal Millet, Jean C Guilland, Francoise Fuchs and Jacques Klepping. Nutrient intake and vitamin status of healthy French vegetarians and non-vegetarians. *American Journal Clinical Nutrition*. 1989; 50:718-27.
34. McKay DL, Perrone G, Rasmussen H, Dallal G, Hartman W, Cao G, Prior RL, Roubenoff R, Blumberg JB. The effects of a multivitamin/mineral supplement on micronutrient status, antioxidant capacity and cytokine production in healthy older adults consuming a fortified diet. *Journal of the American College of Nutrition*. 2000 Oct 1; 19(5):613-21.
35. Mikael Fogelholm. Micronutrient status in females during a 24-week fitness-type exercise program. *Annals of Nutrition Metabolism*. 1992; 36:209-218.
36. Guilland JC, Penaranda TH, Gallet CO, Boggio VI, Fuchs FR, Klepping JA. Vitamin status of young athletes including the effects of supplementation. *Medicine and science in sports and exercise*. 1989 Aug; 21(4):441-9.
37. Bamji MS, Krishnaswamy K, Brahmam GN, editors. *Textbook of human nutrition*. Oxford & IBH; 2016.

12.2. RIBOFLAVIN

INTRODUCTION

Riboflavin-derived coenzymes, flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD) are cofactors of numerous enzymes involved in oxidation-reduction reactions and in energy production via the respiratory chain¹. Being a cofactor for the enzymes like glutathione reductase (GR), riboflavin is involved in antioxidant activity and it is also required for the metabolism of other vitamins like vitamin B₆, niacin and vitamin K. In addition, FAD is involved as a cofactor in one-carbon metabolism².

Dietary Sources

Rich dietary sources of riboflavin are milk, liver, kidney, heart, egg white, green leafy vegetables, legumes, mushrooms and almonds. Lean meat, beef, poultry, cheese, beans, brinjal, papaya, green chillies, garlic and spices & condiments contain fair amounts³. Major amount of vitamin in grains is present in the germ and bran. About 7% of flavin in flesh foods may be present as 8α-(amino acid) - FAD covalently attached to certain flavoproteins. The 8α-(amino acid) riboflavin is absorbed but biologically inactive. Although heat stable, riboflavin is destroyed in solution when exposed to light. Cooking losses of riboflavin in Indian preparations is about 20%⁴.

12.2.1. Riboflavin intake and deficiency in India

Dietary deficiency of riboflavin is rampant in India. Recent NNMB surveys show that the average (% RDA) intake of riboflavin is 47% (men) and 31.5% (women) in rural population⁵. In urban population, the average intake of the vitamin is 0.8 mg/day for adult men and 0.7 mg/day for adult women (NNMB urban survey unpublished observations). Recently, a study among the apparently healthy urban adult population reported the median intake of riboflavin as 0.9 mg/day (men) and 0.7 mg/day (women)⁶. Erythrocyte glutathione reductase activity coefficient (EGR-AC) test has been used in place of urinary excretion method and RBC levels for riboflavin deficiency. A study by Bamji *et al*, showed that more than 70% of women and children from low-income groups have biochemical evidence of riboflavin deficiency as judged by the EGR-AC test⁷. A recent study from the ICMR-National Institute of Nutrition estimated the different forms of riboflavin (FMN, FAD and riboflavin) directly in whole blood by reverse-phase HPLC method with fluorescence detection among the apparently healthy urban adult population. Considering FAD (the biological active form of riboflavin) levels <228 nmol/L as deficient, the study showed that 45% women and 54% men were deficient⁶.

While dietary inadequacy of riboflavin is the major factor, studies from Hyderabad showed that respiratory infections also contribute to tissue depletion⁸. Respiratory infections increase riboflavin utilization leading to mobilization of the vitamin from the tissues to blood and increase urinary excretion⁹. Negative nitrogen balance also results in excess urinary losses¹⁰. Exercise leads to increased riboflavin requirement as judged by EGR-AC values¹¹. Riboflavin deficiency is seen associated with prolonged fevers, trauma, malabsorption, hyperthyroidism and malignancy.

The well-established clinical signs of riboflavin deficiency are muco-cutaneous lesions of the mouth- angular stomatitis, glossitis, chelosis. In severe deficiency other areas like scrotum are also involved. The less recognized consequences of riboflavin deficiency are: impaired psychomotor performance¹²⁻¹⁵, reduced iron absorption due to lower conversion of ferric iron to ferrous iron, and risk of anemia¹⁶ and reduced skin collagen maturity (cross-linking)¹⁷. Diminished visual acuity can also be attributed to riboflavin deficiency.

12.2.2. Previous ICMR recommendations

Some of the earlier controlled, long-term studies in humans fed deficient or low-riboflavin diet suggest that on intakes close to 0.5 mg/1000 kcal, urinary excretion was only slightly higher than intakes seen in individuals with riboflavin deficiency signs^{8,9}. This has led to recommendation of 0.6 mg/1000 kcal^{8,9}. Similar value emerged through controlled depletion-repletion study in adult Indian volunteers¹⁸. The enzyme EGR showed linear increase with riboflavin intake and reached maximum levels at intakes close to 0.5 mg /1000 kcal. After allowing for cooking losses, the earlier Committee recommended 0.6 mg/1000 kcal⁴. A minimum intake of 1.2 mg/day was recommended even if the calorie intake is lower than 2000 kcal (Table 12.2a). Similarly Institute of Medicine (IOM, 1998) estimated the requirement based on metabolic studies (depletion-repletion studies) taking into account the EGR activity, urinary thiamine excretion and their dietary intakes¹⁹.

The lower recommendation of FAO/WHO²⁰ (Table 12.2a) is based on some earlier work indicating tissue saturation at daily intakes higher than 1.1 mg. The activity levels are not indicated. Neither WHO/FAO, nor NRC based their recommendations on energy intake as ICMR Committee does (Table 12.2a).

a. Riboflavin requirement in pregnancy and lactation

During pregnancy, there is an increase in the EGR-AC^{21,22}. In a depletion-repletion study, Kuizon *et al*²³ reported that 0.7 mg riboflavin/1000 kcal were required to lower EGR-AC in 4 of 8 pregnant women to 1.3, whereas 0.41 mg/1000 kcal was required for 5 of non-pregnant women. In a study of 372 pregnant women in USA²⁴, maternal riboflavin intake was positively associated with foetal growth.

NRC has recommended an additional intake of 0.3 mg/day during pregnancy to allow for additional demand for maternal and foetal tissues²⁵. ICMR has however retained the riboflavin/calorie ratio at 0.6, and recommended additional intake of 0.2 mg during pregnancy⁴.

The mean riboflavin content of milk of low-income Indian women is less than 30 µg/100ml^{20,26}. With supplementation, it can be raised to a maximum of 30 µg /100 ml²⁰. The earlier Committee felt that this loss could be compensated with an additional allowance of 0.3 mg for the first 6 months of lactation and 0.2 mg for the subsequent 6 months of lactation on the basis of the additional calorie allowance. This level is however lower than the WHO/FAO as well as NRC recommendation of additional 0.5 mg riboflavin during lactation (Table 12.2a). Their calculation is based on 0.26 mg and 0.21 mg riboflavin lost/day during first and second 6 months of lactation (milk volume 750 ml and 600 ml respectively), with utilization efficiency of 70%, and coefficient of variation of milk production as 12.5%. The earlier recommendation was retained in the absence of reliable new information on utilization efficiency of riboflavin for milk production.

b. Riboflavin requirement of infants and children

Riboflavin deficiency as judged by the EGR-AC values was seen in infants aged 1-6 months who received breast milk containing about 22 µg riboflavin/100 ml²⁶. Assuming milk output of about 700 ml during the first six months of lactation, it would appear that riboflavin intake of around 0.15 mg/day is inadequate for the infants. In the absence of any data on riboflavin requirement of older infants and children, the earlier recommendation of 0.6 mg /1000 kcal is retained. WHO/FAO and NRC have recommended additional 0.5 mg per day throughout lactation. FAO/WHO (1998) had recommended riboflavin on a daily basis, which is lower than ICMR Value (Table 12.2a).

c. Riboflavin requirement of the elderly

In a control study involving elderly subjects in Guatemala²⁷ in which measurement of urinary riboflavin excretion and EGR-AC were used, it was concluded that the requirement of healthy individuals aged above 60 years probably does not differ from that for individuals below 51.

Table 12.2a: Comparison of ICMR, FAO/WHO and NRC Recommendations for Riboflavin

Category / Age		ICMR 2010	FAO/WHO 2004	NRC 1989	IOM 2006
		mg/day			
Infants	0-6 m	0.3	0.3	0.4	-
	6-12 m	0.4	0.4	0.5	-
Children	1-3 y	0.6	0.5	0.8	0.5
	4-6 y	0.8	0.6	1.1	0.6 (4-8 y)
	7-9 y	1.0	0.9	1.2 (7-10 y)	
Boys	10-12 y	1.3	1.3	1.5 (11-14 y)	0.9 (9-13 y)
	13-15 y	1.6	1.3		1.3 (14-18 y)
	16-18 y	1.8	1.3	1.8 (15-18 y)	
Girls	10-12 y	1.2	1.0	1.3 (11-14 y)	0.9 (9-13 y)
	13-15 y	1.4	1.0		1.0 (14-18 y)
	16-18 y	1.5	1.0	1.3 (15-18 y)	
Men		Sedentary-1.4 Moderate-1.6 Heavy-2.1	1.3	1.7 (19-50 y) 1.4 (50+ y)	1.3
Women		Sedentary-1.1 Moderate-1.3 Heavy-1.7	1.1	1.3	1.1
Pregnant		+0.3	1.4	1.6	1.4
Lactating	0-6 m	+0.4	1.6	1.8	1.6
	6-12 m	+0.3	1.5	1.8	1.6

*Minimum intake of 1.2 mg/1000 kcal is recommended.

12.2.3. Current recommendations for riboflavin

Estimation of riboflavin requirement based on functionality [Erythrocyte glutathione reductase activity coefficient (EGR-AC)]

The requirement of riboflavin is estimated by plotting EGR-AC values versus dietary riboflavin intake data obtained from human studies^{31, 32, 34, 38, 39, 41-55}. Average requirement (EAR) was calculated from regression analysis (Figure 12.2a). The acceptable cut off used for EGR-AC is 1.2⁵⁶. The RDA is defined as equal to the EAR plus twice the standard deviation (SD). Standard deviation of requirements is calculated from the EAR and the Coefficient of Variation (CV). Assuming a CV of 10%, as the information is not available on the SD of the requirement for riboflavin, the RDA for riboflavin was then determined as EAR+2 (EAR*0.1).The simulated requirement distribution for riboflavin based on the EGR-AC cut-off with assumed normal distribution for adult men and women is presented in Figure 12.2b, which shows an EAR of riboflavin is 1.64 and corresponding RDA is 1.96 for adult men. Similarly, EAR of riboflavin is 1.56 and corresponding RDA is 1.87 for adult women.

Figure 12.2a: The EGR-AC in relation to dietary intake in both genders

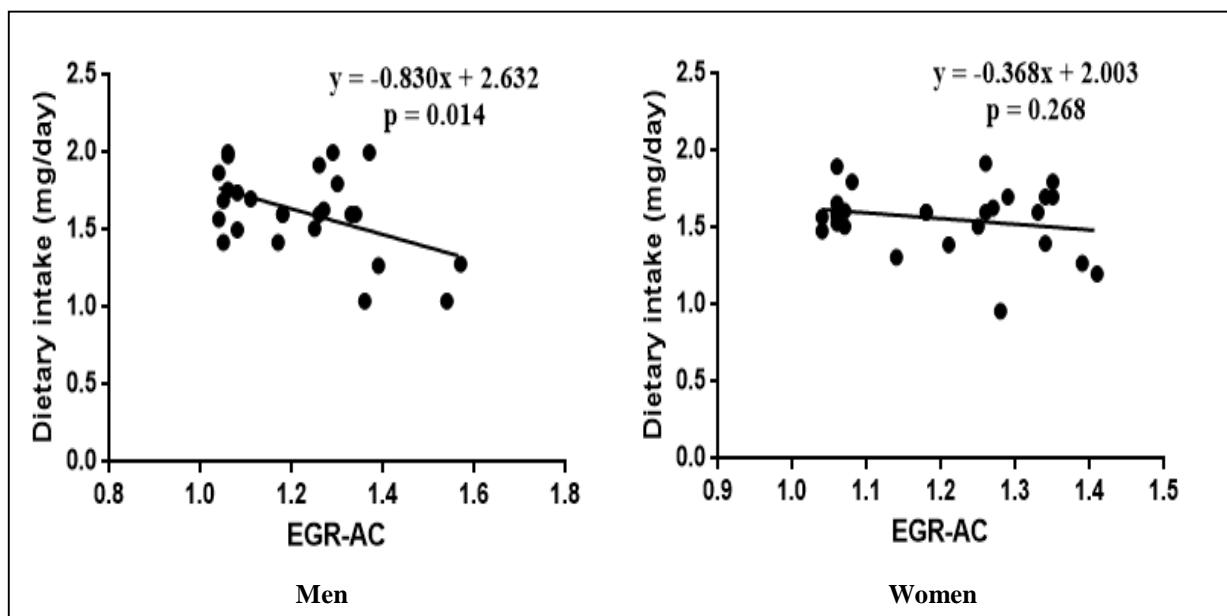
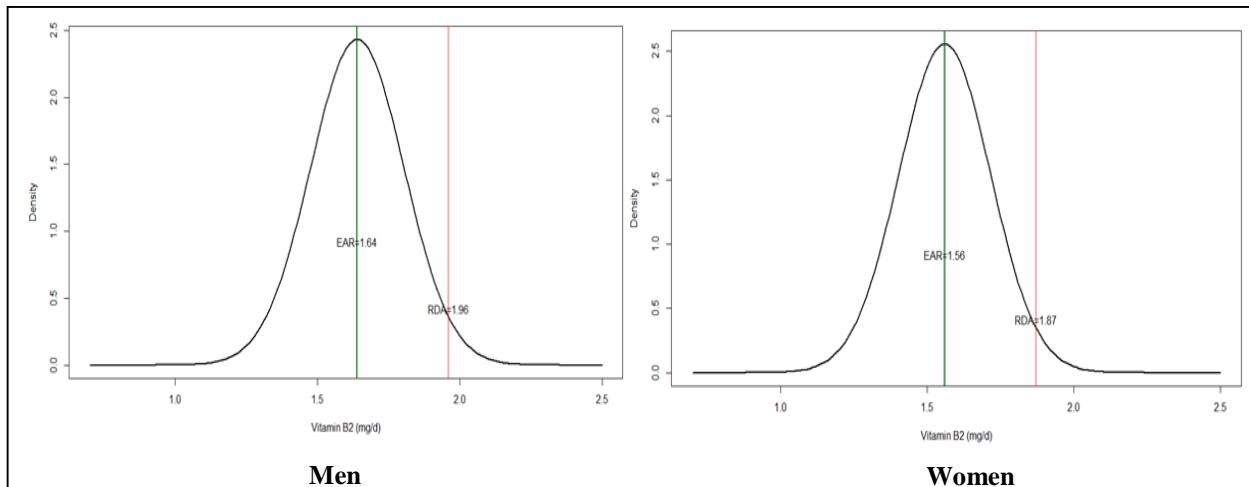


Figure 12.2b: Distribution of the requirements in both genders



During pregnancy, the additional allowance of 0.27 mg of riboflavin based on the additional calorie allowance (350 kcal) of current recommendations, would take care of maternal and foetal growth. During lactation, the additional allowance of 0.47 mg and 0.4 mg of riboflavin for 0-6 months and 6-12 months, respectively, based on the additional calorie allowance [600 kcal (0-6 months), 520 kcal (6-12 months)] of current recommendations. The current recommendations for riboflavin across age groups are given in Table 12.2b.

12.2.4. Tolerable upper limit for riboflavin

There is no Tolerable Upper Limit (TUL) identified on the data available. While a few studies in India used 1-3 mg/day of riboflavin involving 5-15-year boys/ girls or women, other studies have used 60-400 mg/day on adults under various conditions. Further, the IOM (1998) also states that the evidence on adverse effects is not sufficient to set a TUL for riboflavin¹⁹.

Table 12.2b: Current recommendations for Riboflavin across age groups

Age group (y)	EAR (mg/d)	RDA (mg/d)
Infants (0-6m) (6-12m)	- -	0.4(AI) 0.6(AI)
Children (1-3 y) (4-6 y) (7-9 y)	0.8 1.1 1.3	0.9 1.3 1.6
Boys (10-12 y)	1.7	2.1
Girls (10-12 y)	1.6	1.9
Boys (13-15 y)	2.2	2.7
Girls (13-15 y)	1.9	2.2
Boys (16-18 y)	2.5	3.1
Girls (16-18 y)	1.9	2.3
Adult Men		
Sedentary	1.6	2.0
Moderate	2.1	2.5
Heavy	2.7	3.2
Adult Women		
Sedentary	1.6	1.9
Moderate	2.0	2.4
Heavy	2.6	3.1
Pregnant	2.3	2.7
Lactating (0-6m) (6-12m)	2.5 2.4	3.0 2.9

References

1. Said HM and Ross AC. Riboflavin. In: Modern Nutrition in Health and Disease. Lippincott Williams & Wilkins, Philadelphia, USA, 2012; 1616 pp.
2. McKinley MC, McNulty H, McPartlin J, Strain JJ, Pentieva K, Ward M, Weir DG and Scott JM, 2001. Low-dose vitamin B-6 effectively lowers fasting plasma homocysteine in healthy elderly persons who are folate and riboflavin replete. *American Journal of Clinical Nutrition*, 73, 759–764.
3. Longvah T, Ananthan R, Bhaskarachary K, Venkaiah K. In: Longvah T, editor. Indian Food Composition Tables: National Institute of Nutrition Hyderabad; 2017.
4. ICMR. Nutrient requirements and recommended dietary allowances for Indians. A Report of the Expert Group of the Indian Council of Medical Research. National Institute of Nutrition, Hyderabad, 1989.
5. NNMB. Diet and nutritional status of rural population. Prevalence of hypertension and diabetes among adults and infants and young child feeding practices. Technical Report No.26. Report of the third survey National Nutrition Monitoring Bureau, National Institute of Nutrition (ICMR), 2012.
6. Sivaprasad M, Shalini T, Reddy PY, Seshacharyulu M, Madhavi G, Kumar BN, Reddy GB. Prevalence of vitamin deficiencies in an apparently healthy urban adult population: Assessed by subclinical status and dietary intakes. *Nutrition*, 2019 Jul 1; 63:106-13.
7. Bamji MS and Lakshmi AV. Less recognized micronutrient deficiencies in India. *NFI Bull* 19(2):5-8, 1998.
8. Bamji MS, Bhaskaram P and Jacob CM. Urinary riboflavin excretion and erythrocyte glutathione reductase activity in preschool children suffering from upper respiratory infections and measles. *Annals of Nutrition Metabolism*. 1987; 31:191-196.
9. Brijlal S, Lakshmi AV. Tissue distribution and turnover of [³H] riboflavin during respiratory infection in mice. *Metabolism*. 1999 Dec 1; 48(12):1608-11.
10. Pollack H, Bookman JJ. Riboflavin excretion as a function of protein metabolism in the normal, catabolic, and diabetic human being. *The Journal of laboratory and clinical medicine*. 1951 Oct 1; 38(4):561-73.
11. Soares MJ, Satyanarayana K, Bamji MS, Jacob CM, Ramana YV, Rao SS. The effect of exercise on the riboflavin status of adult men. *British journal of nutrition*. 1993 Mar; 69(2):541-51.
12. Bamji MS, Arya S, Sarma KVR and Radhaiah G. Impact of long-term, low dose vitamin supplementation on vitamin nutrition status and psychomotor performance of rural school boys. *Nutrition Research* 2. 1982; 147-153.
13. Prasad PA, Bamji MS, Lakshmi AV and Satyanarayana K. Functional impact of riboflavin supplementation in urban school children. *Nutrition Research* 10. 1990; 275-281.
14. Sterner RT and Price WR. Restricted riboflavin: Within subjects' behavioural effects in humans. *Am J Clinical Nutrition*. 1973; 26:150-160.
15. Bamji MS, Sarma KVR and Radhaiah G. Relationship between biochemical and chemical indices of B-vitamin deficiency. A study in rural school boys. *British Journal of Nutrition*. 1979; 41: 431.
16. Fairweather-Tait SJ, Powers HJ, Minski MJ, Whitehead J and Downes R. Riboflavin deficiency and iron absorption in adult Gambian men. *Annals of Nutrition Metabolism*. 1992; 36: 34-40.
17. Prasad R, Lakshmi AV and Bamji MS. Impaired collagen maturity in vitamin B₂ and B₆ deficiency - probable molecular basis of skin lesions. *Biochemical Medicine*. 1983; 30: 333-341.
18. Bamji MS. Glutathione reductase activity in red blood cells and riboflavin nutritional status in humans. *Clinica Chimica Acta*. 1969 Nov 1; 26(2):263-9.
19. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for thiamine, riboflavin, niacin, vitamin B6, folate, vitamin B₁₂, pantothenic acid, biotin, and choline. National Academies Press (US); 1998.
20. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.

21. Neela J and Raman L. The relationship between maternal nutrition status and spontaneous abortion. *Nutr Med J India* 10: 15, 1997.
22. Vir SC, Love AH and Thompson W. Riboflavin status during pregnancy. *American Journal of Clinical Nutrition*. 1981; 34: 2699-2705.
23. Kuizon MD, Natera MG, Alberto SP, Perlas LA, Desnacido JA, Avena EM, Tajaon RT, Macapinlac MP. Riboflavin requirement of Filipino women. *European journal of clinical nutrition*. 1992 Apr 1; 46(4):257-64.
24. Badart-Smook A, HOUWELINGEN AC, Al MD, Kester AD, Hornstra G. Fetal growth is associated positively with maternal intake of riboflavin and negatively with maternal intake of linoleic acid. *Journal of the American Dietetic Association*. 1997 Aug 1; 97(8):867-70.
25. National Research Council. Recommended dietary allowances 10thed. National Academy Press Washington DC. FAO/WHO Expert Consultation on Human Vitamin and Mineral Requirements. 1989.
26. Bamji MS, Prema K, Jacob CM, Ramalakshmi BA, Madhavapeddi R. Relationship between maternal vitamins B2 and B6 status and the levels of these vitamins in milk at different stages of lactation. A study in a low-income group of Indian women. *Human nutrition. Clinical nutrition*. 1986 Mar; 40(2):119-24.
27. Boisvert WA, Mendoza I, Castaneda *et al*. Riboflavin requirement of healthy elderly humans and its relationship to macronutrient composition of the diet. *Journal of Nutrition*. 1993; 123: 915-25.
28. European Food Safety Authority. Dietary reference values for thiamine. *EFSA Journal* 2016; 14(12):4653. doi: 10.2903/j.efsa.2016.4653.
29. Fukuwatari T, Yoshida E, Takahashi K, Shibata K. Effect of fasting on the urinary excretion of water-soluble vitamins in humans and rats. *Journal of nutritional science and vitaminology*. 2010; 56(1):19-26.
30. Tomiko Tsuji, Tsutomu Fukuwatari, Satoshi Sasaki, Katsumi Shibata. Urinary excretion of vitamin B1, B2, B6, niacin, pantothenic acid, folate and vitamin C correlates with dietary intakes of free-living elderly, female Japanese. *Nutrition Research*. 2010; 30: 171–8.
31. Belko AZ, Obarzanek E, Kalkwarf HJ, Rotter MA, Bogusz S, Miller D, Haas JD, Roe DA. Effects of exercise on riboflavin requirements of young women. *American Journal of Clinical Nutrition*. 1983; 37:509–517.
32. Belko AZ, Obarzanek E, Roach R, Rotter M, Urban G, Weinberg S, Roe DA. Effects of aerobic exercise and weight loss on riboflavin requirements of moderately obese, marginally deficient young women. *American Journal of Clinical Nutrition*. 1984; 40:553–61.
33. Belko AZ, Meredith MP, Kalkwarf HJ, Obarzanek E, Weinberg S, Roach R, McKeon G, Roe DA. Effects of exercise on riboflavin requirements: Biological validation in weight reducing women. *American Journal of Clinical Nutrition*. 1985; 41:270–7.
34. Soares MJ, Satyanarayana K, Bamji MS, Jacob CM, Ramana YV, Rao SS. The effect of exercise on the riboflavin status of adult men. *British Journal of Nutrition*. 1993; 69:541–51.
35. Davis MV, Oldham HG, Roberts LJ. Riboflavin excretions of young women on diets containing varying levels of the B vitamins. *Journal of Nutrition*. 1946; 32:143–61.
36. Winters LR, Yoon JS, Kalkwarf HJ, Davies JC, Berkowitz MG, Haas J, Roe DA. Riboflavin requirements and exercise adaptation in older women. *American Journal of Clinical Nutrition*. 1992; 56:526–32.
37. Brewer W, Porter T, Ingalls R, Ohlson MA. The urinary excretion of riboflavin by college women. *Journal of Nutrition*. 1946; 32:583–96.
38. Fu-Liu CS, Fujitaki C and Lewis JS. Riboflavin status: Dietary intake, urinary excretion and erythrocyte glutathione reductase coefficient activity of female university students. *Nutrition Research*, 1986; Vol.6, pp. 601-608.
39. Keith RE, and Alt LA. Riboflavin status of female athletes consuming normal diets. *Nutrition Research*, 1991; Vol.11, pp. 727-734.

40. Oldham H, Lounde E and Porter T. Riboflavin excretions and test dose returns of young women during periods of positive and negative nitrogen balance. *Journal of Nutrition*. 1947 Jul 10; 34(1):69-79.
41. Mataix J, Aranda P, Sa'nchez C, Montellano MA, Planells E and Llopis J. Assessment of thiamin (vitamin B1) and riboflavin (vitamin B2) status in an adult Mediterranean population. *British Journal of Nutrition*. 2003; 90: 661–6.
42. Millet P, Guilland JC, Fuchs F and Klepping J. Nutrient intake and vitamin status of healthy French vegetarians and non-vegetarians. *American Journal of Clinical Nutrition*. 1989; 50:718-27.
43. Itoh R and Suyama Y. Socio-demographic Factors and Life-Styles Affecting Micronutrient Status in an Apparently Healthy Elderly Japanese Population. *Journal of Nutrition for the Elderly*, Vol 14(2/3)1995.
44. Costa de Carvalho MJ, Guilland JC, Moreau D, Boggio V, Fuchs F. Vitamin Status of healthy subjects in Burgundy (France). *Annals of Nutrition Metabolism*. 1996; 40:24-51.
45. McKay DL, Perrone G, Rasmussen H, Dallal G, Hartman W, Cao G, Prior RL, Roubenoff R and Blumberg JB. The Effects of a Multivitamin/Mineral Supplement on Micronutrient Status, Antioxidant Capacity and Cytokine Production in Healthy Older Adults Consuming a Fortified Diet. *Journal of the American College of Nutrition*. 2000;19(5):613–21.
46. Fogelholm M. Micronutrient status in females during a 24-week fitness-type exercise program. *Ann Nutrition Metabolism*. 1992; 36:209-218.
47. Guilland JC, Penaranda T, Gallet C, Boggio V, Fuchs F, Klepping J. Vitamin status of young athletes including the effects of supplementation. *Med Sci Sports Exerc*. 1989 Aug; 21(4):441-9.
48. Yang FL, Liao P-C, Chen Y-Y, Wang J-L and Shaw N-S. Prevalence of thiamin and riboflavin deficiency among the elderly in Taiwan. *Asia Pacific Journal of Clinical Nutrition*. 2005; 14 (3):238-43.
49. Hustad S, McKinley MC, McNulty H, Schneede J, Strain JJ, Scott JM and Ueland PM. Riboflavin, flavinmononucleotide, and flavin adenine dinucleotide in human plasma and erythrocytes at baseline and after low-dose riboflavin supplementation. *Clinical Chemistry*. 2002; 48, 1571–1577.
50. Hughes CF, Ward M, Tracey F, Hoey L, Molloy AM, Pentieva K and McNulty H. B-Vitamin Intake and Biomarker Status in Relation to Cognitive Decline in Healthy Older Adults in a 4-YearFollow-Up Study. *Nutrients*. 2017; 9: 53.
51. McKinley MC, McNulty H, McPartlin J, Strain JJ and Scott JM. Effect of riboflavin supplementation on plasma homocysteine in elderly people with low riboflavin Status. *European Journal of Clinical Nutrition*. 2002; 56: 850–856.
52. Hoey L, McNulty H, Askin N, Dunne A, Ward M, Pentieva K, Strain JJ, Molloy AM, Flynn CA and Scott JM. Effect of a voluntary food fortification policy on folate, related B vitamin status, and homocysteine in healthy adults. *American Journal of Clinical Nutrition*. 2007; 86:1405–13.
53. Madigan SM, Tracey F, McNulty H, Eaton-Evans J, Coulter J, McCartney H and Strain JJ. Riboflavin and vitamin B₆ intakes and status and biochemical response to riboflavin supplementation in free-living elderly people. *American Journal of Clinical Nutrition*. 1998; 68:389–95.
54. Majchrzak D, Singer I, Männer M, Rust P, Genser D, Wagner K-H, Elmadfa I. B-Vitamin Status and Concentrations of Homocysteine in Austrian Omnivores, Vegetarians and Vegans. *Annals of Nutrition Metabolism*. 2006; 50:485–91. DOI: 10.1159/000095828.
55. Wright AJA, Southon S, Bailey AL and Finglas PM. Nutrient intake and biochemical status of non-institutionalized elderly subjects in Norwich: comparison with younger adults and adolescents from the same general community. *British Journal of Nutrition*.1995; 74: 453-75.
56. Bamji MS, Krishnaswamy K, Brahmam GNV. *Textbook of Human Nutrition*. 3rded: Oxford & IBH Publishing Co. Pvt. Ltd, New Delhi; 2009.

12.3. NIACIN

INTRODUCTION

Nicotinic acid (niacin) and nicotinamide (niacinamide) are the two forms of vitamin B3 (niacin). Niacin function as the precursor of nicotinamide nucleotide coenzymes NAD and NADP, which are involved in many metabolic processes including glycolysis, tissue respiration, fatty acid metabolism and synthesis of macromolecules.

Niacin can also be synthesised from the essential amino acid tryptophan as its metabolic end product and thus dietary tryptophan can spare the requirements of niacin. In considering the dietary adequacy of niacin, contribution of both is taken into account. Intakes of both energy and protein are known to regulate the efficiency of conversion of tryptophan to niacin^{1,2}. In well-nourished individuals 60 mg of tryptophan produce 1 mg of niacin and thus 60 mg tryptophan is considered as 1 mg niacin equivalents (NE)³. While computing dietary intake of niacin, tryptophan contribution as niacin equivalents is added to that of preformed niacin/ nicotinic acid as follows:

$$1 \text{ mg Niacin Equivalent (NE)} = 60 \text{ mg Tryptophan}$$

Dietary sources

Nicotinic acid is the basic precursor mainly available in the plant foods, particularly in nuts and oil seeds while nicotinamide is a degradation product of the pyridine nucleotides that are mainly absorbed from animal-based foods, especially liver.

12.3.1. Niacin intake and deficiency in India

Though whole grain cereals are satisfactory sources of niacin, mostly present in the esterified form, which is unavailable for absorption^{4,5}. In the recent study done at ICMR-NIN, niacin is reported to be present more in nuts and oil seeds among the plant foods⁶. Niacin is more stable than other B-vitamins in spite of some inevitable losses in cooking^{7,8}. Based upon studies carried out in India, an average loss of 25% in Indian cooking can be assumed.

Deficiency of niacin leads to development of pellagra (symptoms such as photosensitive dermatitis, skin lesions, tongue and mouth soreness, vomiting, diarrhoea, depression and dementia). This symptom was seen in endemic form in some parts of India where jowar (sorghum) was used as staple.

12.3.2. Previous ICMR recommendations requirements of adults

The recommendations of previous ICMR committee along with other global recommendation are given in table 12.3. Diet surveys from India show that the average intake of niacin is around 10 mg daily. Among predominantly rice eaters, intakes are much lower, the values ranging between 5 and 11 mg per day; together with tryptophan, such diets provide about 6 mg NE per 1000 kcal⁹. Pellagra is rarely seen among this population. Load tests using nicotinic acid have shown that subjects consuming 6.5 to 7.2 mg of NE per 1000 kcal have satisfactory niacin status¹⁰. Niacin intakes of subjects suffering from pellagra have, however, been found to be around 3.6 mg/1000 kcal - an apparently inadequate intake.

Joint FAO/WHO Expert Group¹¹ suggested an intake of 5.6 mg NE per 1000 kcal for adults. The earlier ICMR Committee recommended 6.6 mg/1000 kcal as adult RDA. The present committee have derived similar value by adding 10% CV (20% 2SD) to the EAR of 5.6 mg, providing an RDA of 6.7 mg/1000 kcal. EAR requirements for men, NPNL women, children aged 1-18 years are derived based on energy requirements (EAR=5.6 x energy in kcal/1000) and RDA of niacin was determined as EAR+2SD (20% CV).

Pregnancy and lactation

Information on the niacin requirements during pregnancy is scanty. Based on the observation that urinary excretion of metabolites of tryptophan is higher in pregnant women than in normal following an oral load of tryptophan, it has been suggested that conversion of the amino acid into niacin is more efficient during pregnancy. The extra allowance of niacin for the additional energy intake would cover the niacin needs during pregnancy and has been provided. The nicotinic acid content of breast milk of Indian women ranges between 100 and 150 µg per 100 ml and the amount lost in a day would thus be between 0.9 and 1.2 mg niacin in the mother¹². As in the case of pregnancy, niacin intake during lactation will be higher because of higher energy intakes which are recommended. The earlier ICMR Committee therefore recommended the same level (density) as in the diet of adult non-pregnant woman, to lactating woman and the present committee also used similar approach except an additional 1 mg of NE secreted in breast milk is added in addition to the additional energy requirements during lactation.

Infants and children

In the absence of any reports on the subject or any information on special needs in infants and children, the RDA was recommended on the basis of energy requirements by the earlier committees. The present committee recommends an AI of 2.0 mg of niacin for infants aged 0-6 months based on intake of 700 ml of breast milk which contain 1.5 mg/l of niacin¹¹. For infants aged 6-12 months, AI of 5.0 mg have been extrapolated based on the energy requirements in adults (Tables 12.4).

Table 12.3: Comparison of ICMR (2010), FAO/WHO and NRC Recommendations for Niacin

Group	Category / Age	ICMR 2010 (mg NE/d)	IOM (2006) (mg NE/d)	FAO/WHO (2004) (mg NE/d)	NRC 1989 (mg NE/d)
Infants	0-6 m	710 µg/kg	-	2	-
	6-12 m	650 µg/kg	-	4	-
Children	1-3 y	8	6	6	6
	4-6 y	11	8 (4-8 y)	8	8
	7-9 y	13		12	
Boys	10-12 y	15	12 (9-12 y)		12 (9-13 y)
	13-15 y	16	14 (14-18 y)		16 (14-18 y)
	16-18 y	17			
Girls	10-12 y	13	12 (9-12 y)	16 (10-18 y)	12 (9-13 y)
	13-15 y	14	12 (14-18 y)		14 (14-18 y)
	16-18 y	14			
Men	Sedentary	16	16	16	16
	Moderate	18			
	Heavy	21			
Women	Sedentary	12	14	14	14
	Moderate	14			
	Heavy	16			
	Pregnant	+2	18	18	18
	Lactating 0-6 m	+4	17	17	17
	Lactating 6-12 m	+3			

Table 12.4: Current recommendations for Niacin across age groups

Age Group	Category	Net Energy (Kcal/d)	EAR / 1000 kcal	RDA
Infants	0-6 m	550	2 (AI)	
	6-12 m	670	5 (AI)	
Children	1-3 y	1010	6	7
	4-6 y	1360	8	9
	7-9 y	1700	10	11
Boys	10-12 y	2220	12	15
Girls	10-12 y	2060	12	14
Boys	13-15 y	2860	16	19
Girls	13-15 y	2400	13	16
Boys	16-18 y	3320	19	22
Girls	16-18 y	2500	14	17
Men	Sedentary	2110	12	14
	Moderate work	2710	15	18
	Heavy	3470	19	23
Women	Sedentary	1660	9	11
	Moderate work	2130	12	14
	heavy	2720	15	18
	Pregnant	350	+2	+2.5
	Lactating 0-6 m	600	+4	+5
	7-12 m	520	+4	+5

12.3.3. Tolerable Upper Limit for Niacin

There is no evidence of adverse effects from excess consumption of diet based niacin. Study shows that, ingestion of a pharmacological dose ranging from 3-9 g of nicotinic acid daily results in various metabolic effects such as increased utilization of muscle glycogen stores, decrease in serum lipids and fatty acids mobilization in the adipose tissue during exercise¹³.

References

1. Satyanarayana U, Rao BN. Effect of dietary protein level on some key enzymes of the tryptophan-NAD pathway. *British Journal of Nutrition.* 1977 Jul; 38(1):39-45.
2. Satyanarayana U, Narasinga Rao BS. Effect of diet restriction on some key enzymes of tryptophan-NAD pathway in rats. *The Journal of nutrition.* 1977 Dec 1; 107(12):2213-8.
3. Horwitt MK, Harvey CC, Rothwell WS, Cutler JL, Haffron D. Tryptophan-niacin relationships in man: studies with diets deficient in riboflavin and niacin, together with observations on the excretion of nitrogen and niacin metabolites. *The Journal of Nutrition.* 1956 Oct 1; 60(suppl_1):1-43.
4. Wall JS, Young MR and Carpenter KJ, Transformation of niacin-containing compounds in corn during grain development - relationship to niacin nutritional availability. *Journal of Agricultural and Food Chemistry.* 1987; 35: 752-8.
5. Ball GFM, Niacin and tryptophan. In: Bioavailability and analysis of vitamins in foods. Ed Ball GFM. Chapman & Hall, London, UK, 319-354, 1998.
6. Longvah T, Ananthan R, Bhaskarachary K and Venkaiah K. Indian food composition tables. National Institute of Nutrition, Indian Council of Medical Research, 2017.
7. Basu KP, Malakar MC. Destruction of vitamin B₁ of some vegetables during cooking and the effect of cooking on tree and combined vitamin B₁ of some foodstuffs. *Indian Journal of Medical Research.* 1946; 34:39-43.
8. Rao PS, Ramasastri BV. Riboflavin and nicotinic acid content of some foods of plant origin. *Indian Journal of Nutrition and Dietetics.* 1969; 6:218-23.
9. Aykroyd WR, Swaminathan M. The Nicotinic-Acid Content of Cereals and Pellagra. *Indian Journal of Medical Research.* 1940; 27(3).
10. De HN, Banerjee KC. Nicotinic-acid requirements of Indian adult. *Indian Journal of Medical Research.* 1948; 36:335-9.
11. WHO/FAO (World Health Organization/Food and Agriculture Organization of the United Nations). (2004). Vitamin and mineral requirements in human nutrition: report of a joint FAO/WHO expert consultation, Bangkok, Thailand, 21–30 September 1998, 2004.
12. Deodhar AD, Ramakrishnan CV. Studies on human lactation. (Relation between the dietary intake of lactating women and the chemical composition of milk with regard to vitamin content.). *Journal of Tropical Pediatrics.* 1960; 6:44-7.
13. Darby WJ, McNutt KW and Todhunter EN. Niacin [Diet, disease]. *Nutrition Reviews.* 1975.

12.4 VITAMIN B₆

INTRODUCTION

The term vitamin B₆ includes three forms, pyridoxine (PN), pyridoxal (PL), pyridoxamine (PM), and their respective 5'-phosphates (PLP, PNP, and PMP). Pyridoxal phosphate (PLP), the physiologically active coenzyme form of vitamin B₆ is involved in 140 distinct enzymatic reactions including aminotransferases, decarboxylases, racemases, dehydratases and inside chain cleaving enzymes¹. Thus, vitamin B₆ is needed for important pathways like amino acid metabolism, glycogenolysis, heme biosynthesis, gluconeogenesis, synthesis of neurotransmitters like serotonin, dopamine, taurine, gamma-aminobutyric acid, norepinephrine and histamine. Along with folic acid, vitamin B₁₂ and riboflavin, vitamin B₆ is needed for the metabolism of homocysteine to cysteine. It is also involved in immune system and nucleic acid metabolism. In addition, vitamin B₆ plays a crucial role in antioxidant mechanism².

Dietary sources

As per Indian food composition table (IFCT) garlic, cloves, nuts and seeds (walnut, sunflower seeds, safflower and pistachio nut), poultry chicken, liver, sugarcane, Oyster mushroom are rich sources of vitamin B₆, while milk and milk products, potato, beet root, carrot and cherries, custard apple, musk melon are moderate sources³. Pyridoxine and PNP are the predominant forms of the vitamin present in the plant foods, sometimes in the form of a glucoside. Whereas in the animal foods the major form is pyridoxal and pyridoxal phosphate. The extent to which processing of foods and cooking practices destroy depend on the form in which vitamin B₆ is present and the method of processing. Considerable amounts of pyridoxal and pyridoxal phosphate are lost during cooking, whereas pyridoxine content of food is not affected. The approximate cooking loss of B₆ in daily meals consisting of various dishes was found to be 13%⁴.

About 5-80% of the naturally occurring vitamin B₆ in cereals, legumes, vegetables and fruits is present in glycosylated form, predominantly as pyridoxine-5'-D-glucoside. There appears to be an inverse relationship between pyridoxine glycoside content of the diet and the bioavailability of vitamin B₆. About 15% of the total vitamin B₆ content as glycosylated pyridoxine had no influence on vitamin B₆ status of lactating women⁵. The incomplete bioavailability of glycosylated pyridoxine may be a concern of nutrition in diets, which contain high proportion of pyridoxine glycosides and provide marginally adequate intake of total vitamin B₆. The glucosidal form in which pyridoxine is present in normal foods needs to be studied.

12.4.1 Vitamin B₆ intake and deficiency

Studies conducted by the National Nutrition Monitoring Bureau (NNMB) in urban population show that the average intake of vitamin B₆ is 1.27 mg/day for adult men and 1.13 mg/day for adult women (NNMB urban survey unpublished observations). In a recent study among the healthy urban adult population the median intake of vitamin B₆ was reported to be 1.2 mg/day for men and 1.0 mg/day for women⁶. In rural population, data is not available in Indian scenario.

Vitamin B₆ deficiency coprecipitates with other water-soluble vitamins than per se. Clinical signs and symptoms of pyridoxine deficiency include peripheral neuritis, epileptic form convulsions, anaemia, glossitis, and seborrhoeic dermatitis eczema, cheilosis, angular stomatitis, hyperirritability, convulsive seizures and abnormal electroencephalograms⁷. Deficiency of vitamin B₆ in infants is associated as hypochromic microcytic anaemia, failure to thrive, hyperirritability and convulsive seizures⁸. Biochemical as well as clinical (oral lesions) evidence of pyridoxine deficiency has been reported in young women of reproductive age in India particularly during pregnancy and in women

using oral contraceptives⁹⁻¹¹. Pregnant women and women using oral contraceptives show increased urinary excretion of xanthurenic acid after tryptophan load^{12,13}. More than 5-10 mg pyridoxine is needed daily to correct this abnormality, which is believed to be a hormonal effect on the enzyme kynureninase. Inadequate vitamin B₆ intake has also been reported to impair platelet function and clotting mechanisms^{14,15}. Indicators for assessment of vitamin B₆ status include direct measurement of urinary 4-pyridoxic acid (biologically inactive product of pyridoxine metabolism), blood pyridoxal phosphate levels, and indirect functional measurement of *in vitro* activation of erythrocyte aspartate amino transferase (α -EAST) or alanine amino transferase (α -EALT) with pyridoxal phosphate, and urinary excretion of xanthurenic acid after oral load of tryptophan. The increase in urinary cystathione after a methionine load is also used as an indicator of vitamin B₆ status¹⁶. Plasma pyridoxal phosphate (PLP) is the best single indicator, because it is believed to reflect tissue stores¹⁷.

12.4.2 Previous ICMR recommendations

a. Adults

Pyridoxine requirement is linked to protein content of the diet. Study suggests that pyridoxine intake may vary from 1.2-3.3 mg per day¹⁸. At an intake of 1.2 mg per day, biochemical evidence of 4-pyridoxine deficiency as judged by excretion of pyridoxic acid and xanthurenic acid after tryptophan load was seen. Similar evidence of biochemical deficiency was not seen in subjects consuming 1.9 mg per day. In a controlled depletion-repletion study, it was observed that when dietary protein was increased from 30 g to 100 g, pyridoxine requirement increased from 1.25-1.4 mg per day¹⁸. Based on this, the earlier committee of ICMR recommended 2.0 mg per day, especially since cooking losses of the vitamin are negligible. NRC (1989) has recommended 2.0 mg/day for adult males and 1.6 mg /day for adult females. This may however be an overestimate, since dietary vitamin B₆ ratio of 0.02 mg/g protein has been reported to ensure normal biochemical status for most parameters¹⁹. On the other hand, there is some evidence that availability of pyridoxine from vegetable foods may be less than that from animal foods. The former has some amount of pyridoxine in bound form as glycoside²⁰. Institute of Medicine (IOM, 1998), has recommended 1.3 mg /day for adult males and females²¹. FAO/WHO in 2004 recommended lower levels of B₆ intake for adult male and female and other groups²² (Table 12.4.1a).

b. Pregnancy and lactation

Pregnant women show biochemical evidence of pyridoxine deficiency, which gets corrected, only with high doses of pyridoxine. Earlier ICMR committee had recommended an additional 0.6 mg/day during pregnancy. FAO/WHO, NRC also recommend the same during pregnancy (Table 12.4.1a). Breast milk of well-nourished American mothers has been reported to contain 0.1-0.25 mg vitamin B₆/L. The NRC has recommended additional allowance of 0.5 mg of vitamin B₆ per day during lactation. ICMR had earlier recommended the same level (Table 12.4.1a), even though vitamin B₆ content in the breast milk of Indian women has been reported to be much lower^{23,24}. FAO/WHO has recommended an intake of 2.2 mg/d and 2.1 mg/day in pregnancy and lactation, respectively (Table 12.4.1a).

c. Infants and children

Evidence of vitamin B₆ deficiency has been reported in infants who consumed less than 0.1 mg vitamin B₆ through breast milk. The average intake of 0.3 mg was found to protect healthy babies against abnormal tryptophan metabolism. Based on this limited information, NRC has recommended an intake of 0.3 mg /day for infants aged 0-6 months. This level seems abnormally high when the amount of the vitamin available through breast milk is considered. Vitamin B₆ content of breast milk of American mothers has been reported to be about 0.13 mg/L or 0.1 mg/700 ml. Based on this, WHO/FAO recommend 0.1 mg for infants 0-6 months, but a higher level of 0.3 mg for 6-12 months

infants (Table 12.4.1a). The breast milk vitamin B₆ content of Indian mothers was found to be only 60-80 µg/L²⁴. At this level of intake, no evidence of enzymatic vitamin B₆ deficiency was detected in exclusively breastfed, 1-6 months old infants²⁴. Earlier ICMR committee recommends the retention of the earlier value, which is close to the FAO/WHO recommendation for infants (Table 12.4.1a).

Table 12.4.1a: Comparison of ICMR, FAO/WHO and NRC recommendations for vitamin B₆

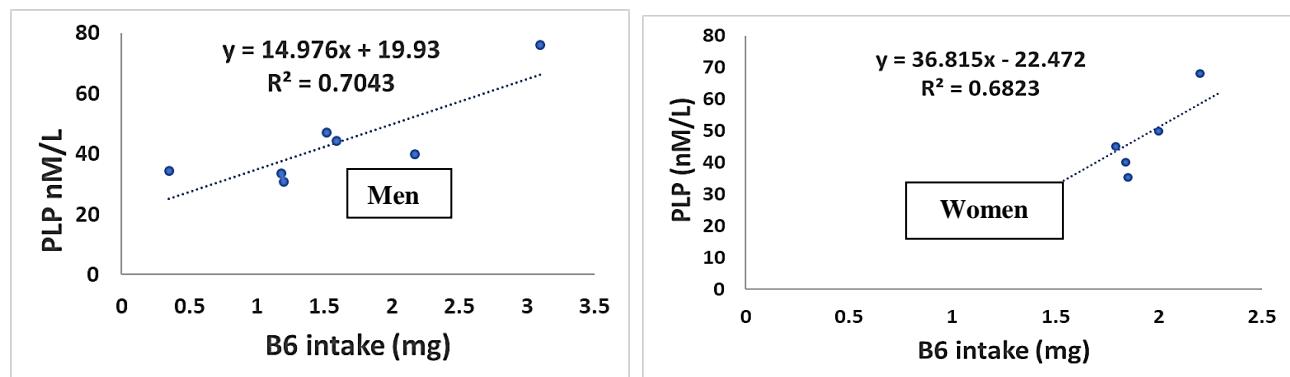
Category / Age		ICMR 1989	ICMR 2010	FAO/WHO 2004	NRC 1989	IOM 2006
		mg/d				
Infants	0-6 m	0.1	0.1	0.1	0.3	0.1
	6-12 m	0.4	0.4	0.3	0.6	0.3
Children	1-3 y	0.9	0.9	0.5	1.0	0.5
	4-6 y	0.9	0.9	0.6	1.1	0.6
	7-9 y	1.6	1.6	1.0	1.2	1.0
Boys	10-12 y	1.6	1.6	1.3	1.7 (11-14 y)	1.0
	13-15 y	2.0	2.0	1.3	2.0 (15-18 y)	1.3
	16-18 y	2.0	2.0	1.3		1.3
Girls	10-12 y	1.6	1.6	1.2	1.4 (11-14 y)	1.0
	13-15 y	2.0	2.0	1.2	1.5 (15-18 y)	1.2
	16-18 y	2.0	2.0	1.2		1.2
Men		2.0	2.0	1.3 (19-50 y)	2.0 (19-50 y)	1.3 (19-50 y)
				1.7 (>50 y)	(> 50 y)	1.7 (>50 y)
Women		2.0	2.0	1.3 (19-50 y)	1.6 (19-50 y)	1.3 (19-50 y)
				1.5 (50+ y)	(>50 y)	1.5 (>50 y)
Pregnant		2.5	2.5	1.9	2.2	1.9
Lactating	0-6 m	2.5	2.5	2.0	2.1	
	6-12 m	2.5	2.5	2.0	2.1	

12.4.3. Current recommendations for vitamin B₆

12.4.3.1 Estimation of Vitamin B₆ requirement based on Plasma PLP [Pyridoxyl-5-phosphate].

Plasma PLP correlates well with tissue stores. PLP concentration of 30 nmol/L is indicative of adequate vitamin B₆ status (IOM). Estimated average requirement was calculated from the linear relationship seen by plotting plasma PLP concentrations and the amount of vitamin B₆ intake (data obtained from human studies²⁵⁻³⁰) as given in the figure below.

Figure 12.4.1a: The relationships between plasma PLP and B₆ intake in both genders



Assuming a CV of 10% for the distribution of requirements, RDA for vitamin B₆ was then determined as the 97.5th percentile of this distribution.

$$\text{RDA} = \text{EAR} + 2\text{SD} \text{ where } (\text{SD} = \text{EAR} * \text{CV})$$

Based on this approach, the requirement (EAR) of vitamin B₆ for adult men to maintain PLP is 1.0 mg/day and for women it was 1.4 mg/day. As an alternative approach, measurement of erythrocyte aspartate aminotransferase activity coefficient (EAST-AC) was done as a functional marker, as it reflects long term status of vitamin B₆ adequacy.

Table 12.4.1b: Vitamin B₆ requirement to maintain Plasma PLP

Men	EAR	1.0 (mg/day)
	RDA	1.2 (mg/day)
Women	EAR	1.4 (mg/day)
	RDA	1.7 (mg/day)

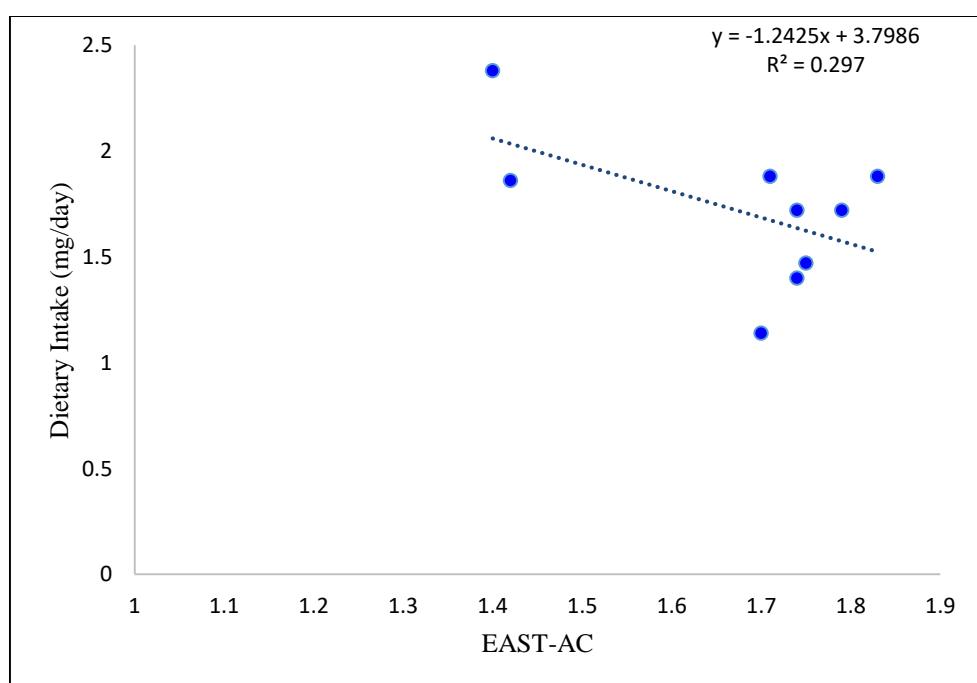
12.4.3.3 Estimation of Vitamin B₆ requirement based on functionality [Erythrocyte aspartate aminotransferase activity coefficient (EAST-AC)]

The functional activity of erythrocyte aspartate aminotransferase (EAST) which requires PLP as a cofactor, was used for estimating the estimated average requirement (EAR), and regression analysis was performed by plotting EAST-AC values versus dietary vitamin B₆ intake data obtained from human studies³¹⁻³⁶ for both men and women (Figure 12.4.1b). As the data on EAST-AC was very limited, data of both men and women were combined for the regression analysis. For the current recommendation EAR of B₆ was calculated using EAST-AC cut-off of 1.8 as suggested by EFSA. Assuming a CV of 10% for the distribution of requirements, RDA for vitamin B₆ was then determined.

$$\text{Requirement} = \text{Constant} + \text{slope} * \text{Cut-off value of EAST-AC}$$

$$\text{RDA} = \text{EAR} + 2\text{SD} \text{ where } (\text{SD} = \text{EAR} * \text{CV})$$

Figure 12.4.1b: EAST-AC in relation to dietary vitamin B₆ intake in both genders



12.4.3.4 Current recommendations for vitamin B₆ across different age groups

The dietary intake of B₆ was 1.6 mg/day for an EAST-AC less than 1.8. Considering 1.6 mg as the requirement (EAR) of B₆/day, the RDA was calculated by considering CV of 10%, and RDA of 1.9 mg/day was arrived at for an adult man. Since the energy requirement for a sedentary adult man (ICMR, 2020) is 2110 kcal/day, the vitamin B₆ requirement for 1000 kcal works out to be $1000 \times 1.6/2110 = 0.76$ mg (0.76mg B₆, EAR/1000 Kcal). The EAR and RDA thus derived for adults based on EAST-AC calculation was extrapolated for other physiological and age groups based on the energy requirements. Vitamin B₆ requirement for different groups based on their energy requirement is as follows:

Table 12.4.1c: Recommended vitamin B₆ intake in adults (mg/day) based on EAST-AC

		Applied 1.8 as cut-off for EAST-AC from EFSA
Men	EAR	1.6
	RDA	1.9
Women	EAR	1.6
	RDA	1.9

Table 12.4.1d: Current recommendations for vitamin B₆ across different age groups based on EAST-AC

Age group (Y)	EAR (mg/day)	RDA (mg/day)
Infants (0-6m) (6-12m)	- 0.5	0.1 (AI) 0.6
Children (1-3 Y) (4-6 Y) (7-9 Y)	0.8 1.0 1.3	0.9 1.2 1.5
Boys (10-12 Y)	1.7	2.0
Girls (10-12 Y)	1.6	1.9
Boys (13-15 Y)	2.2	2.6
Girls (13-15 Y)	1.8	2.2
Boys (16-18 Y)	2.5	3.0
Girls (16-18 Y)	1.9	2.3
Adult Men-Sedentary	1.6	1.9
Moderate	2.1	2.4
Heavy	2.6	3.1
Adult Women-Sedentary	1.6	1.9
Moderate	1.6	1.9
Heavy	2.1	2.4
Pregnant	1.9	2.3
Lactating (0-6m) (6-12m)	+0.22 +0.16	+0.26 +0.17

12.4.3.6. Tolerable upper limit for vitamin B₆

There is a paucity of data to arrive at upper tolerable limits. However, few studies in India used 1-10 mg/ day of pyridoxine for 5-15 year boys/ girls and 100 mg/day of pyridoxine for adults. Severe toxicity was observed in adults at doses of 500 mg/day³⁸. No adverse effects were observed in pregnant women taking up to 200 mg/day of pyridoxine orally and up to 100 mg/day of pyridoxine parenterally³⁹⁻⁴⁴. Further, the IOM (1998) also states that 100 mg/day of pyridoxine as TUL for adults.

References

1. Percudani R, Peracchi A. A genomic overview of pyridoxal-phosphate-dependent enzymes. *EMBO reports.* 2003 Sep; 4(9):850-4.
2. Hsu CC, Cheng CH, Hsu CL, Lee WJ, Huang SC, Huang YC. Role of vitamin B6 status on antioxidant defences, glutathione, and related enzyme activities in mice with homocysteine-induced oxidative stress. *Food & Nutrition Research.* 2015 Jan 1; 59(1):25702.
3. Longvah T, Ananthan R, Bhaskarachary K, Venkaiah K. In: Longvah T, editor. *Indian Food Composition Tables:* National Institute of Nutrition Hyderabad; 2017.
4. Shibata K, Yasuhara Y, Yasuda K. Effects of cooking methods on the retention of vitamin B6 in foods, and the approximate cooking loss in daily meals. *Journal of Home Economics of Japan.* 2001 Dec 15; 52(12):1187-97.
5. Andon MB, Raynolds RD, Moser-Veillon P and Pat Howard M. Dietary intake of total and glycosylated vitamin B₆ and the vitamin B₆ nutritional status of unsupplemented lactating women and their infants. *American Journal of Clinical Nutrition.* 1989; 50: 1050-8.
6. Sivaprasad M, Shalini T, Reddy PY, Seshacharyulu M, Madhavi G, Kumar BN, Reddy GB. Prevalence of vitamin deficiencies in an apparently healthy urban adult population: Assessed by subclinical status and dietary intakes. *Nutrition.* 2019 Jul 1; 63:106-13.
7. Sauberlich HE. Vitamin B6 status assessment: past and present. In: Leklem JE and Reynolds RD (ed.). *Methods in Vitamin B₆ Nutrition: Analysis and Status Assessment.* Plenum Press, New York, USA. pp. 203–239. 1981.
8. Borschel MW. Vitamin B6 in infancy: requirements and current feeding practices. In: Raiten DJ (ed.). *Vitamin B₆ Metabolism in Pregnancy, Lactation and Infancy.* CRC Press, Boca Raton, FL, USA. pp. 109–124.1995.
9. Joshi UM, Virkar KD, Amatayakul K, Singkamani R, Bamji MS, Prema K, Whitehead TP, Belsey MA, Hall P, Parker RA. Impact of hormonal contraceptives vis-à-vis non-hormonal factors on the vitamin status of malnourished women in India and Thailand. World Health Organization: Special Programme of Research, Development and Research Training in Human Reproduction. Task Force on Oral Contraceptives. *Human nutrition. Clinical nutrition.* 1986 May; 40(3):205-20.
10. Satyanarayana U and Narasinga Rao BS. Effect of dietary protein level on some key enzymes of tryptophan-NAD pathway. *British Journal of Nutrition.* 1997; 38: 39.
11. Ahmed F, Bamji MS and Iyengar L. Effect of oral contraceptive agents on vitamin nutrition status. *Am J Clinical Nutrition.* 1975; 28: 606-15.
12. Vijayalakshmi R, Bamji MS and Ramalakshmi BA. Reduced anaerobic glycolysis in oral contraceptive users. *Contraception.* 19883; 8: (1)-91-7.
13. Vijayalakshmi R and Bamji MS. Altered glucose metabolism in female rats treated with sex steroids: reversal by excess pyridoxine. *Indian Journal of Biochemistry and Biophysics.* 1988; 24(6), pp. 329-35.
14. Brattstrom LE, Israelsson B, Norrvig B, Bergkvist D, Thorne J, Hultberg B, Hamfelt A. Impaired homocysteine metabolism in early-onset cerebral and peripheral occlusive arterial disease. Effects of pyridoxine and folic acid treatment. *Atherosclerosis.* 1990; 81:51–60.
15. Subbarao K, Kakkar VV. Thrombin induced surface changes of human platelets. *Biochem Biophys Res Commun.* 1979; 88:470–476.
16. Park YK and Linkswiler H. Effect of vitamin B₆ depletion in adult man on the excretion of cystathionine and other methionine metabolites. *Journal of Nutrition,* 100, 110–116. 1970.

17. Lui A, Lumeng L, Aronoff GR, Li TK. Relationship between body store of vitamin B6 and plasma pyridoxal-P clearance: metabolic balance studies in humans. *The Journal of laboratory and clinical medicine*. 1985 Nov 1; 106(5):491-7.
18. ICMR. Nutrient requirements and recommended dietary allowances for Indians. A Report of the Expert Group of the Indian Council of Medical Research. National Institute of Nutrition, Hyderabad, 1990.
19. Hansen CM, Leklem JE and Miller LT. Vitamin B₆ status of women with a constant intake of vitamin B₆ changes with three levels of dietary protein. *Journal of Nutrition*. 1996; 126: 1891.
20. Van den Berg H. Bioavailability of vitamin D. *European journal of clinical nutrition. Supplement*. 1997; 51(1):S76-9.
21. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B₆, folate, vitamin B₁₂, pantothenic acid, biotin, and choline. National Academies Press (US); 1998.
22. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.
23. National Research Council. Recommended dietary allowances 10thed. National Academy Press Washington DC. FAO/WHO Expert Consultation on Human Vitamin and Mineral Requirements. 1989.
24. Bamji MS, Premakumari K, Jacob CM. Pyridoxine status and requirement of breast-fed infants: relationship with maternal status and milk pyridoxine levels. *Nutrition reports international*. 1987; 35(1):171-7.
25. Young-Nam Kim and Youn-Ok Cho. Evaluation of vitamin B₆ intake and status of 20- to 64-year-old Koreans. *Nutrition Research and Practice*. 2014; 8(6):688-694.
26. Ashima KK, Phylis BM, and Robert DR, Effect of age on changes in plasma, erythrocyte, and urinary. *American Journal of Clinical Nutrition*. 1988; 48: 1284-90.
27. Lindberg AS, Leklem JE, Miller LT. The effect of wheat bran on the bioavailability of vitamin B-6 in young men. *The Journal of nutrition*. 1983 Dec 1; 113(12):2578-86.
28. Daphne LE. Henk vanden Berg, Klaas R. Westerterp. The Influence of Protein Intake on Vitamin B₆ Metabolism Differs in Young and Elderly Humans. *American institute of Nutritin*.1994.
29. James EL, Lorraine TM, Anne DP and Diane EP. Bioavailability of Vitamin B₆ from Wheat Bread in Humans. *J. Nutr.* 110: 1819-1828, 1980.
30. Christine M Hansen, James E Lekiem, and Lorraine T Miller. Changes in vitamin B₆ status indicators of women fed a constant protein diet with varying levels of vitamin B6. *Journal of Clinical Nutrition*. 1997;66:1379-87.
31. Planells, CSa'nchez, MA Montellano, J Mataix and J Llopis. Vitamins B₆ and B₁₂ and folate status in an adult. *Mediterranean population*. *European Journal of Clinical Nutrition*. 2003; 57: 777–85.
32. D. Majchrzak I. Singer M. Männer P. Rust D. Genser K.-H. Wagner Elmadfa. B-Vitamin Status and Concentrations of Homocysteine in Austrian Omnivores, Vegetarians and Vegans. *Ann Nutrition Metabolism*. 2006; 50:485–91.
33. Guillard JC, Berekci-Reguig B, Lequeu B, Moreau D, Klepping J, Richard D. Evaluation of pyridoxine intake and pyridoxine status among aged institutionalised people. *International journal for vitamin and nutrition research. Internationale Zeitschrift fur Vitamin-und Ernahrungsorschung. Journal international de vitaminologie et de nutrition*. 1984; 54(2-3):185.
34. Kretsch MJ, Sauberlich HE, Skala JH, Johnson HL. Vitamin B-6 requirement and status assessment: young women fed a depletion diet followed by a plant-or animal-protein diet with graded amounts of vitamin B-6. *The American journal of clinical nutrition*. 1995 May 1; 61(5):1091-101.
35. Christine M. Hansen, Terry D. Shultz, Ho-Kyung Kwak, H. Sara Memon and James E. Leklem. Assessment of Vitamin B₆ Status in Young Women Consuming a Controlled Diet Containing Four Levels

of Vitamin B₆ Provides an Estimated Average Requirement and Recommended Dietary Allowance. American Society for Nutritional Sciences 2001.

36. Kant AK, Moser-Veillon PB, Reynolds RD. Effect of age on changes in plasma, erythrocyte, and urinary B-6 vitamers after an oral vitamin B-6 load. *The American journal of clinical nutrition*. 1988 Nov 1; 48(5):1284-90.
37. European Food Safety Authority. Dietary reference values for Vitamin B₆. *EFSA Journal* 2016; 14(6):4485.
38. Sashi G Kiran, Raj K Dorisetty, Malathi R Umrani, Sesikeran Boindala, Ramesh R, Maniprabha C, Singh H, Venkatesan V. Pyridoxal 5 phosphate protects islets against streptozotocin-induced beta-cell dysfunction - *in vitro* and *in vivo*. *Experimental Biology and Medicine*. 2011; 236:456-65.
39. Dalton K, Dalton MJ. Characteristics of pyridoxine overdose neuropathy syndrome. *Acta neurologica Scandinavica*. 1987 Jul; 76(1):8-11.
40. ELLIS J. Treatment of carpal tunnel syndrome with vitamin B6. *Southern Medical Journal*. 1987 Jul; 80(7):882-4.
41. Weinstein BB, Wohl Z, Mitchell GJ, Sustendal GF. Oral administration of pyridoxine hydrochloride in the treatment of nausea and vomiting of pregnancy. *Amur. J. Obstet. GynecoL*. 1944; 47:389-94.
42. Dorsey CW. The use of pyridoxine and suprarenal cortex combined in the treatment of the nausea and vomiting of pregnancy. *American Journal of Obstetrics & Gynecology*. 1949 Dec 1; 58(6):1073-8.
43. Dorsey CW. The use of pyridoxine and suprarenal cortex combined in the treatment of the nausea and vomiting of pregnancy. *American Journal of Obstetrics & Gynecology*. 1949 Dec 1; 58(6):1073-8.
44. Hart BF, McConnell WT. Vitamin B factors in toxic psychosis of pregnancy and the puerperium. *American Journal of Obstetrics and Gynecology*. 1943; 46.

12.5. FOLATE

INTRODUCTION

Folate is one of the water soluble B-complex vitamins and is the natural form of vitamin B9 present in food. The primary biochemical function of folate is to transfer single carbon units required in the biosynthetic reactions of nucleic acids and amino acids. In addition, adequate folate supply is required for rapidly growing and multiplying cells in our body. The folate found in foods consists of a mixture of unsubstituted polyglutamyl tetrahydrofolates and various substituted one carbon forms of tetrahydrofolate (e.g. 10-formyl-, 5, 10-methylene-, and 5-methyl-tetrahydrofolate). Along with vitamin B₁₂, folate is essential for the synthesis of nucleic acids. The other functions include methylation of DNA as part of epigenetic regulation. It also ensures supply of methionine for synthesis of various proteins. Synthetic form of folate is folic acid which is used in supplements and in fortified foods. After supplementation, folic acid is absorbed unchanged and reduced to the dihydro and tetrahydro forms in cells in the post-absorptive phase.

12.5.1. Food folate and bioavailability

Folate is present in a wide variety of foods of both plant and animal origin. The rich sources of folate in animal foods include marine and fresh water fish and shellfish, chicken liver, beef liver and pork liver, whereas the rich plant-based sources of folate are pulses & legumes, green leafy & other vegetables and cereals. Pulses usually contain two folds of folate as compared to cereals¹. A recent study also reported different forms and total folate content in selected traditionally prepared foods in Southern India². Folate is present in the free form or as conjugated form of polyglutamates with variable number of glutamate residues. The proportion of free and conjugated folate varies from food to food. Before absorption, polyglutamates are broken down by the folate conjugase in the intestine. The extent of absorption of dietary folate has not been well studied, while free folic acid is well absorbed. Reduced folate derivatives are less bioavailable than oxidized conjugates. Though there has been a controversy, it is generally believed that the polyglutamate and monoglutamate derivatives of folic acid have similar bioavailability.

The matic, dietary folate equivalent (DFE unit) was introduced to rationalize/express the total folate intake from natural food sources as well as folic acid intakes from fortified foods and supplements as an integrated measure of both. DFEs are defined as: μg of natural food folate + 1.7 x μg of synthetic folic acid. The 1.7 multiplier was arrived based on assumptions that extent of bioavailability of added folic acid was 85% and that of food folate bioavailability was only 50%. The 85/50 ratio also inferred that the bioavailability of food folate was about 60% relative to added folic acid³. The bioavailability of folic acid (both supplements and fortified foods) is virtually higher than the net bioavailability of naturally occurring food folate⁴.

The studies on actual availability of food folates are few in India. Over 70 % of the folates from egg, 60-70% from pulses and green leafy vegetables and 50% from vegetables are absorbed⁵. Several dietary factors like fibre are known to affect the availability of folate from foods. Studies with radio-labelled synthetic polyglutamates showed that more than 50% of it is in the active coenzyme form, irrespective of the chemical form in which it was ingested⁶. It has been assumed that 50% of the folate present in Indian diets is absorbed. A detailed study involving different Indian recipes demonstrated that the average loss of folates during cooking is 32% and the folate density in Indian meal is about 50 $\mu\text{g}/1000 \text{ kcal}$ ^{7,8}. A recent study gives an exhaustive account on bioavailability of dietary folates from South Indian urban diets for the first time, using advanced methodology [triple enzyme- LCMS for folate analysis and double stable isotope folate bioavailability using 5-methyl tetrahydrofolate (5MTHF) as reference]⁹. Investigators reported that the actual content of folates appears much more than what has been reported so far using

conventional methods and the mean bioavailability of the diets is around 45%, based on normalized red cell folate. There have been other reports too showing higher levels of food folate by refined methodology. It was calculated that a value of 429 µg of dietary folate by LCMS methodology (of which 188 µg is bioavailable) was found to be 277 µg/d by microbiological method (121 µg, bioavailable). It corresponded to only 100-170 µg/d on the basis of conventional food table computation. This study emphasizes the need for generating more reliable data on food folates (bound and free) and their absorption.

12.5.2. Deficiency

In recent years, both folic acid and vitamin B₁₂ deficiencies were found to be associated with elevated blood homocysteine levels, which in turn were reported to be a major etiological risk factor of cardiovascular disease. A recent study demonstrated that supplementation of folic acid alone or in combination with vitamin B₁₂ to Indian patients with vascular disease had reduced the observed elevated homocysteine levels¹⁰. Further, chronic and severe forms of folate deficiency also lead to abnormal hemopoiesis and megaloblastic anaemia (MBA), which promptly responds to treatment with the vitamin. This anaemia remains indistinguishable from the one produced by vitamin B₁₂ deficiency. MBA is relatively common in all age groups¹¹. A recent hospital based study has reported 3.6% MBA in anaemic subjects and the prevalence of vitamin B₁₂ and folic acid deficiencies in these MBA subjects were found to be 40% and 25% respectively, while combined deficiency was 35%¹². The other studies also reported a very low level of folate and/or Vitamin B₁₂ and also hyper-homocysteinemia in MBA cases compared to non-megaloblastic anaemia in both the genders^{13,14}.

MBA also occurs occasionally in pregnant women particularly from poor income groups. It is possible that the widespread iron deficiency (microcytic anaemia) could mask megaloblastic anaemia. An earlier study from south India showed biochemical deficiencies of both iron and folic acid occurring to the same extent in adult women drawn from a slum, though the proportion of each deficiency (45% showed inadequate folate status) may vary with progression of pregnancy¹⁵. In a previous study, sub-clinical folate deficiency was found to be about 26.3% in pregnant women from rural North India¹⁶. A different level of sub-clinical folate deficiency was also reported in young children¹⁷, under five year's children¹⁸, school-age children¹⁹, adolescents²⁰, adults and elderly²¹.

In addition, lack of folate at critical stages of conception was found to be an important cause of neural tube defects (NTD) in the neonates. According to the recent review, the overall prevalence of NTD in India was around 4.5 per 1000 total births²². However, an earlier multi-centric study had observed a very high prevalence of open NTD in India and in this study prenatal folate supplementation to mothers with proven record of delivering one such baby supported this observation demonstrating a drop in the recurrence rate of NTD (2.92%) in supplemented as compared to those receiving placebo control (7.04%)²³.

12.5.3. Dietary intakes in India

Inadequate dietary intake of folate may cause folate deficiency in India. A previous survey conducted by National Nutrition Monitoring Bureau (2000) had showed low intakes of dietary folate in all age groups⁸. A micronutrient survey in Eastern and North eastern part of the country reported that though the mean intakes of folate are comparable to the previous RDA of 100 µg/d (as suggested by the previous committee in 1990), yet dietary deficiency of folate was observed in 20-70% of subjects, thus, the mean values are perhaps masking the extent of dietary deficiency²⁴. Even discounting the effect of skewed distribution of folic acid intakes, there has been a considerable biochemical inadequacy of folic acid. In a study done among the middle income school age children at NIN, it was found that almost all the children in the age group 6-16 years had low RBC folate

though their diet contained a mean intake of 155% RDA for folic acid²⁵. It is obvious that either there were losses in cooking that were not accounted for or the RDA of 100 µg is just not adequate. Losses during cooking were not found to be high, leaving the low RDA as a possible reason.

Further, National Nutrition Monitoring Bureau survey (2012)²⁶ in 10 states of rural populations shows that the median intake of folate is less than the recommended RDA of 2010 in all age groups²⁷. Only about 32-41% of adults meet ≥70% RDA and about 39-46% were consuming less than 50% of the recommended folate RDA of 200 µg/day for adults. A recent secondary analysis of the NNMB 2012 dataset reported a high risk of dietary inadequacy for folate among adolescents (10-19y) and adult women of reproductive age (20-49y), assessed based on estimated average requirement (EARs) suggested by the World Health Organization (2004)²⁸. A subsequent survey in urban population has shown that about 69-79% of adults intake of total folate was ≥70% RDA and about 13-18% consumed ≤50% of RDA²⁹.

12.5.4. Dietary folate intakes and status in Adults

A recent cross sectional study reported the prevalence of folate deficiency at 4.4% and 9.5% based on plasma folate cut-off of <3 ng/ml among 20-40 year and 41-60 year old healthy urban adults, respectively. The corresponding median dietary folate intake was 154 µg/day and 162 µg/day in these two age groups respectively. However the cut-off level considered for deficiency is only <3 ng/ml which is lower than the WHO cut-off of <4 ng/ml. Also the intake in this study was below the current recommended RDA of 200 µg/day²¹. Another study carried out by the same group, consider the cut-off of plasma folate <10 nmol/L or <4ng/ml (suggested by the WHO), the prevalence of folate deficiency was 32% & 29% among 20-40 and 41-60 years of age groups respectively, while the median dietary folate intake in these two age groups was same as 152 µg/day and 162 µg/day, respectively³⁰.

A study carried out by Naik *et al* among young adult vegetarians has reported that there was no folate deficiency, considering a lower cut-off of <2.0 ng/ml and the median intake of folate was 268 and 275 µg/day among male and female study participants respectively³¹. Another study by the same group again showed no folate deficiency among young vegetarian Indian adult, whereas the median dietary intake of dietary folate was 355 µg/day³². Based on the above studies^{21,30-32}, if we take into consideration the WHO cut off of plasma folate of 4 ng/ml (10 nmol/l), intake of dietary folate of 200 µg/day may be insufficient and may result in folate deficiency.

12.5.5. RDA for adults: Previous ICMR recommendations

The reference values set for the folate vary from country to country and most of these values were arrived mainly from controlled metabolic studies, where in their primary indicator is Red blood cell (RBC) folate as it reflects tissue folate stores and secondary indicators are serum/plasma folate (reflects recent folate intakes) and total homocysteine (functional indicator). This requirement estimate was mainly based on the amount of folate essential to maintain normal levels of red cell folate.

In India, based on the earlier studies showing that an intake of 75 µg /d folic acid may ensure adequate blood folate levels, the previous ICMR Committee in 1990 made a recommendation of 100 µg folic acid per day for adult man and women³³. Another study revealed that daily intakes ranging between 200-250 µg are necessary to maintain satisfactory status. These studies had dose-response relationship built into them³⁴. A recent review among adults and elderly revealed that for every doubling in intake of folate, the changes in RBC folate, plasma/serum folate and plasma homocysteine levels were increased by 21%, 22%, and decreased by 16% respectively³⁵. Earlier studies reported that about 32% of the dietary folate is lost in Indian style of cooking⁷ and around

45-50% of the folate is absorbed. It is reasonable to assume that the bioavailable folate is about 1/3 of the total folate in Indian foods/diets and the advisable physiological requirement of folic acid (75 µg) can be obtained from 200 µg of dietary folate.

Considering the earlier recommendation of 100 µg/day³³ along with the recommendations of 200 µg/day suggested by International Union of Nutritional Sciences (1983) and 400 µg by Food and Nutrition Board of the USA (1974), the previous committee constituted in 2010 made a recommendation of 200 µg dietary folate per day for adult men and women²⁷. In view of the proven role of folic acid deficiency as an etiological factor for elevated blood homocysteine and CVD, and congenital NTD, there is a concern for upward revision of RDA for folic acid. There have been suggestions to increase intake of folate to at least 400 µg per day to prevent chronic disease, particularly cancer and CVD³⁶. However, considering the intake and the prevalence of sub-clinical deficiency, a level of intake that was found essential in previous studies, i.e; 200 µg/d is recommended as the intake of dietary folate for adult males and females²⁷.

12.5.6. Revision of RDA for adults

Since there is lack of data with respect to dietary folate intake and RBC folate status among Indian adult subjects, we have considered plasma folate as primary indicator and analysed pooled data of three cross sectional studies published from NIN^{21, 30, 37} to find out the dietary folate that is required to maintain plasma folate (>10nmol/L) and homocysteine at normal levels among adults (21 to <50 years).

Based on the descriptive statistics, the amount of folate (median intake) required to maintain normal plasma folate for the male subjects was ~250 µg/day and for female it was ~180 µg/day. Considering both the genders together, the median intake was 220 µg/day and the 95th centile was 260 µg/day (Table 12.5.1). The range of intake of dietary folate in this category was from 124 µg/day to 657 µg/day in both the genders.

Table 12.5.1: Median and mean dietary folate intake of adults (based on the dietary folate required to maintain normal plasma folate levels)

Group	Median (P25-P75) dietary folate intake (µg/day)	Mean (SD) dietary folate intake (µg/day)	Median +10%CV
Men (n=17)	250 (211-278)	285 (150)	300
Women (n=27)	180 (152-269)	233 (131)	220
Both the genders (n=44)	214 (171-273)	253 (140)	260

P25: 25th percentile, P75: 75th percentile.

SD: Standard Deviation.

Similarly, the amount of dietary folate required to maintain plasma homocysteine at normal levels (<15 µmol/L), for male subjects was median of ~424 µg/day and for females it was found to be ~200 µg/day. When analysed for both genders together, the median intake was ~220 µg/day and the 95th centile was 260 µg/day (Table 12.5.2). The intake of dietary folate in this category ranged from 117 µg/day to 687 µg/day in both the genders.

Table 12.5.2: Median and mean dietary folate intake of adults (based on the dietary folate required to maintain normal plasma homocysteine levels)

Group	Median (P25-P75) dietary folate intake ($\mu\text{g}/\text{day}$)	Mean (SD) dietary folate intake ($\mu\text{g}/\text{day}$)	Median +10%CV
Men (n=4)	424 (321-573)	447 (164)	508
Women (n=18)	200 (177-229)	221 (82)	240
Both the genders (n=22)	217 (192-319)	262 (131)	260

P25: 25th percentile, P75: 75th percentile.

SD: Standard Deviation

Further, the amount of dietary folate required to maintain both plasma folate and homocysteine at normal levels was also analysed and the median intake of folate for females was found to be ~205 $\mu\text{g}/\text{day}$. As for males, only one male subject had normal plasma folate and homocysteine level (Table 12.5.3). When analysed for both genders together, the median intake was ~217 $\mu\text{g}/\text{day}$ and the 95th centile was 260 $\mu\text{g}/\text{day}$ (Table 12.5.3). The intake of dietary folate in this category ranged from 124 $\mu\text{g}/\text{day}$ to 657 $\mu\text{g}/\text{day}$ in both the genders.

Table 12.5.3: Median and mean dietary folate intake of adults (based on the dietary folate required to maintain normal plasma folate and homocysteine levels)

Group	Median (P25-P75) dietary folate intake ($\mu\text{g}/\text{day}$)	Mean (SD) dietary folate intake ($\mu\text{g}/\text{day}$)	Median +10%CV
Men (n=1)	657	657	---
Women(n=12)	205 (162-274)	231 (114)	245
Both the genders(n=13)	217 (177-319)	264 (132)	260

P25: 25th percentile, P75: 75th percentile.

SD: Standard Deviation

Based on the data shown in Table 12.5.1 to 12.5.3 and also the number of subjects and variations in the mean/median data, we have considered dietary folate required for maintaining normal folate levels (Table 12.5.1) for calculating the RDA. The amount of folate (median intake) required to maintain normal plasma folate levels for male subjects was ~250 $\mu\text{g}/\text{day}$ (EAR) and considering CV as 10%, the RDA (EAR+2SD) was arrived at 300 $\mu\text{g}/\text{day}$, while for female, the EAR was ~180 $\mu\text{g}/\text{day}$ and the RDA was arrived at 216 (220) $\mu\text{g}/\text{day}$ (Table 12.5.4).

The EAR and RDA values for children and adolescence have been extrapolated from the adult values based on growth factors and body weights as described earlier in IOM, 1998³. While calculating RDA for pregnant women, the gestational weight (~12 kgs) was added. The present committee has agreed with the previous committee²⁷ over the conceptual basis on which additional requirements of 300 $\mu\text{g}/\text{day}$ and 100 $\mu\text{g}/\text{day}$ were added respectively during pregnancy and lactation for meeting the factorial extra needs. While for infants (0-6 months), the previous committee²⁷ recommended values were retained, and for 6-12 months age EAR and RDA was calculated based on the adult values (Table 12.5.4).

Table 12.5.4: RDA of dietary folate for various physiological groups

Group	Category/Age	Body weights (kg)	EAR	RDA 2020	TUL (μg)
			Dietary folate (μg/d*)		
Men	Sedentary	65.0	250	300	1000
	Moderate	65.0			
	Heavy	65.0			
Women	Sedentary	55.0	180	220	1000
	Moderate	55.0			
	Heavy	55.0			
	Pregnant	55.0	480	570	1000
	Lactating 0-6 m 6-12 m	55.0	280 280	330 330	1000
Infants	0-6 m	5.8	---	25 (AI)	
	6-12 m	8.5	71 (AI)	85 (AI)	
Children	1-3 y	11.7	90	110	6-9 y 300
	4-6 y	18.3	111	135	
	7-9 y	25.3	142	170	
Boys	10-12 y	34.9	180	220	9-17 y 600-800
Girls	10-12 y	36.4	186	225	
Boys	13-15 y	50.5	238	285	
Girls	13-15 y	49.6	204	245	
Boys	16-18 y	64.4	286	340	
Girls	16-18 y	55.7	223	270	

*1 μg of food/dietary folate = 0.5 μg of synthetic folic acid taken on empty stomach or 0.6 μg folic acid taken with meals.

AI: Adequate intake

12.5.7. Tolerable upper limit for folate

According to the IOM, 1998³, Food Safety and Standards Authority of India, the tolerable Upper Limits (TUL) for folate is 300 μg/day for 6-9 years; 600-800 μg/day for 9-17 years; While a few studies in India used 500 μg/day involving adolescent boys and girls; 5 mg for urban healthy pregnant women of 17-40 years of age; 10 mg or 30 mg/week by rheumatoid arthritis patients (18-75 yrs.) with no adverse effects. However, high maternal folate intakes coupled with low B₁₂ intakes were associated with a higher risk of delivering a small-for-gestational age infant. The deleterious effect of high folate intakes with low B₁₂ intakes needs to be explored further.

References

1. Longvah T, Ananthan R, Bhaskarachary K, Venkaiah K. In: Longvah T, editor. Indian Food Composition Tables: ICMR-National Institute of Nutrition Hyderabad; 2017.
2. Vishnumohan S, Pickford R, Arcot J. Naturally occurring folates in selected traditionally prepared foods in Southern India. *Journal of food science and technology*. 2017 Dec 1; 54(13):4173-80.
3. Institute of Medicine (US). Dietary Reference Intakes for Thiamine, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. National Academy Press, Washington, DC, 1998.
4. Wright AJ, King MJ, Wolfe CA, Powers HJ, Finglas PM. Comparison of (6 S)-5-methyltetrahydrofolic acid v. folic acid as the reference folate in longer-term human dietary intervention studies assessing the relative bioavailability of natural food folates: comparative changes in folate status following a 16-week placebo-controlled study in healthy adults. *British Journal of Nutrition*. 2010; 103(5):724-9.
5. Babu S, Srikantia SG. Availability of folates from some foods. *American Journal of Clinical Nutrition*. 1976; 29(4):376-9.
6. Godwin HA, Rosenber. IH. Absorption of synthetic cold and tritium-labeled pteroylheptaglutamic acid. In *Journal of clinical investigation* 1970 Jan 1 (vol. 49, no. 6, p. A35);Rockefeller Univ Press.
7. Agte V, Tarwadi K, Mengale S, Hinge A, Chiplonkar S. Vitamin profile of cooked foods: how healthy is the practice of ready-to-eat foods?. *International journal of food sciences and nutrition*. 2002 Jan 1; 53(3):197-208.
8. NNMB Special Report, Report on Food and Nutrient intake of Individuals, Report No 20: pp 67-95, Hyderabad, NIN. 2000.
9. Vishnumohan S. Natural folates - Method development, analysis and bioavailability of the most predominant 5-methyl tetrahydrofolate in mixed diets in humans. A Thesis Submitted to the School of Chemical Sciences and Engineering for the Degree of Doctor of Philosophy, The University of New South Wales, 2008.
10. Bhargava S, Ali A, Bhargava EK, Manocha A, Kankra M, Das S, Srivastava LM. Lowering homocysteine and modifying nutritional status with folic acid and vitamin B12 in Indian patients of vascular disease. *Journal of clinical biochemistry and nutrition*. 2011;1202080140-.
11. Khanduri U, Sharma A. Megaloblastic anaemia: prevalence and causative factors. *National Medical Journal of India*. 2007; 20(4):172-5.
12. Kaur N, Nair V, Sharma S, Dudeja P, Puri P. A descriptive study of clinico-hematological profile of megaloblastic anemia in a tertiary care hospital. *Medical journal armed forces India*. 2018 Oct 1; 74(4):365-70.
13. Yadav MK, Manoli NM, Madhunapantula SV. Comparative Assessment of Vitamin-B₁₂, Folic Acid and Homocysteine Levels in Relation to p53 Expression in Megaloblastic Anemia. *PLoS One* 2016; 11(10):e0164559.
14. Yadav MK, Manoli NM, Vimalraj S, Madhunapantula SV. Unmethylated promoter DNA correlates with p53 expression and apoptotic levels only in Vitamin B9 and B₁₂ deficient megaloblastic anemia but not in non-megaloblastic anemia controls. *International Journal Biomedical Macromolecules*. 2018; 109:76-84.
15. Raman L, Subbalaxmi PV, Vasumathi N, Rawal A, Vasanthi G, Parvathi CH, Adinarayana K, Pawashe AB, Rao KV. Iron and folic acid nutritional status of women in slum. *Nutrition reports international (USA)*. 1989.
16. Pathak P, Kapil U, Kapoor S, et al. Prevalence of multiple micronutrient deficiencies amongst pregnant women in rural area of Haryana. *Indian Pediatrics*. 2004; 71:1007-1014.
17. Houghton LA, Trilok-Kumar G, McIntosh D, Haszard JJ, Harper MJ, Reid M, Erhardt J, Bailey K, Gibson RS. Multiple micronutrient status and predictors of anemia in young children aged 12-23 months living in New Delhi, India. *PLoS One*. 2019; 14(2):e0209564.
18. Kapil U, Toteja GS, Bhaduria AS. Cobalamin and folate deficiencies among children in the age group of 12-59 months in India. *Biomed J*. 2015 Mar 1; 38(2):162-6.

19. Kapil U, Toteja GS, Bhadaria AS. Cobalamin and folate deficiencies among children in the age group of 12-59 months in India. *Biomed J.* 2015 Mar 1; 38(2):162-6.
20. Jani R, Salian N, Udupi S, Ghugre P, Lohia N, Haas J, Boy E. Folate status and intake of tribal Indian adolescents aged 10 to 17 years. *Food and nutrition bulletin.* 2015 Mar; 36(1):14-23.
21. Sivaprasad M, Shalini T, Balakrishna N, Sudarshan M, Lopamudra P, Suryanarayana P, *et al.* Status of Vitamin B12 and folate among the urban adult population in South India. *Annals of Nutrition Metabolism.* 2016; 68:94-102.
22. Allagh KP, Shamanna BR, Murthy GV, Ness AR, Doyle P, Neogi SB, Pant HB; Wellcome Trust- PHFI Folic Acid project team. Birth prevalence of neural tube defects and or ofacial clefts in India: a systematic review and meta-analysis. *PLoS One* 10(3):e0118961, 2015.
23. ICMR Central Technical Co-ordinating Unit. Multicentric study of efficacy of peri-conceptional folic acid containing vitamin supplementation in prevention of open neural tube defects from India. *Indian Journal of Medical Research.* 2000; 112:206-11.
24. Chakravarty I and Sinha RK. Prevalence of micronutrient deficiency based on results obtained from the National Pilot Program on Control of Micronutrient Malnutrition. *Nutrition Reviews.* 2002; 60: (II) S53-S58.
25. Sivakumar B, Nair KM, Sreeramulu D, *et al.* Effect of micronutrient supplement on health and nutrition status of school children: biochemical status. *Nutrition.* 2006; 22: Suppl No1, 15-25.
26. NNMB. Diet and nutritional status of rural population. Prevalence of hypertension and diabetes among adults and infants and young child feeding practices. -Report of the third survey NNMB Technical Report No.26. National Institute of Nutrition, Hyderabad. 2012.
27. ICMR. Nutrient requirements and recommended dietary allowances for Indians. A report of the expert group of the Indian Council of Medical Research National Institute of Nutrition Hyderabad; 2010.
28. Radhika MS, Swetha B, Kumar BN, Krishna NB, Laxmaiah A. Dietary and non-dietary determinants of nutritional status among adolescent girls and adult women in India. *Annals of the New York Academy of Sciences.* 2018 Mar; 1416(1):5-17.
29. NNMB. Diet and nutritional status of urban population in India and prevalence of obesity, hypertension, diabetes and hyperlipidemia in urban men and women. National Nutritional Monitoring Bureau brief report on urban nutrition. Technical Report No.27. National Institute of Nutrition, Hyderabad. 2017.
30. Shalini T, Sivaprasad M, Balakrishna N, Madhavi G, Radhika MS, Kumar BN, Pullakhandam R, Reddy GB. Micronutrient intakes and status assessed by probability approach among the urban adult population of Hyderabad city in South India. *European journal of nutrition.* 2019 Dec 1; 58(8):3147-59.
31. Naik S, Mahalle N, Bhide V. Identification of vitamin B 12 deficiency in vegetarian Indians. *British Journal of Nutrition.* 2018 Mar; 119(6):629-35.
32. Naik S, Bhide V, Babulkar A, Mahalle N, Parab S, Thakre R, Kulkarni M. Daily milk intake improves vitamin B-12 status in young vegetarian Indians: an intervention trial. *Nutrition journal.* 2013 Dec 1; 12(1):136.
33. ICMR. Nutrient Requirements and Recommended Dietary Allowances for Indians. A Report of the Expert Group of the Indian Council of Medical Research, National Institute of Nutrition, Hyderabad, 1990.
34. Babu S. Studies on folic acid. PhD Thesis. 1976. Osmania University, Hyderabad, India.
35. Novaković R, Geelen A, Ristić-Medić D, Nikolić M, Souverein OW, McNulty H, Duffy M, Hoey L, Dullemeijer C, Renkema JM, Gurinović M. Systematic review of observational studies with dose-response meta-analysis between folate intake and status biomarkers in adults and the elderly. *Annals of Nutrition and Metabolism.* 2018; 73(1):30-43.
36. Krishnaswamy K, Nair KM. Importance of folate in human nutrition. *British Journal of Nutrition.* 2001 May; 85(S2):S115-24.
37. Sivaprasad M, Shalini T, Reddy PY, Seshacharyulu M, Madhavi G, Kumar BN, Reddy GB. Prevalence of vitamin deficiencies in an apparently healthy urban adult population: Assessed by subclinical status and dietary intakes. *Nutrition.* 2019; 63-64:106-13.

12.6 VITAMIN B₁₂

INTRODUCTION

Vitamin B₁₂ is a water-soluble micronutrient and is important in the diet as it is required for formation of genetic materials in the cells and helps to keep the body's nerve and blood cells healthy. It is a key nutrient associated with one carbon metabolic pathways related to substrate metabolism. The major source for vitamin B₁₂ (henceforth B₁₂) is animal source foods like milk and other dairy products, fish, poultry and meat¹. The deficiency of B₁₂ is characterized by pernicious anaemia, weakness, fatigue, loss of appetite, constipation, and weight loss^{2,3}. The neurological symptoms of B₁₂ deficiency include depression, confusion, cognitive decline, dementia, failure to thrive and developmental delays⁴. Adequate intake of the nutrient ensures optimal functioning of the body.

12.6.1 Vitamin B₁₂ intake and deficiency in India

Based on analyses of household food expenditure from the 68th round of National Sample Survey Organization (NSSO), the average adult Indian population consumed 1.13 µg B₁₂/d⁵. The consumption was high in the states which consumed more fish. These were estimates of intake based on consumer units of household intake, and biomarker based evaluations of B₁₂ status are better indicators. The more recent Comprehensive National Nutrition Survey (CNNS) 2019 survey of children and adolescents in India showed that there was a relatively low prevalence of biomarker based B₁₂ deficiency in all age groups of children⁶. The prevalence of deficiency (based on serum B₁₂ concentration) was 13.8%, 17.2% and 30.9% in 1-4, 5-9 and 10-19y, respectively. Corroborating this, a study conducted on adults from Tamil Nadu found that only 19.9% were deficient based on total serum homocysteine concentration (tHcy)⁷. Another study in Hyderabad showed that 34% females and 42% males were deficient based on tHcy⁸. It is important to note that the prevalence of B₁₂ deficiency measured in these studies can vary because it is based on the type of biomarker used and there is no consensus regarding the cut-off used for defining the deficiency.

12.6.2 Previous ICMR recommendations

The B₁₂ requirement provided by ICMR expert committee of 1989 was retained by ICMR 2010 expert committee^{9,10}. The 1989 requirement was based on daily B₁₂ loss from the body, which was estimated from studies conducted on Western populations using radioactive labelled B₁₂ isotope to measure the loss by whole body counting. The reported loss daily from the body was 0.1%. Since the liver store contains 1 mg of B₁₂, therefore, 1 µg/d (0.1% of 1 mg B₁₂ store) was required to replenish the amount lost from the body. However, absorption was considered to be 100%. Another approach based on B₁₂ turnover studies which reported that the losses can be between 0.5 – 1.0 µg/d was also used to derive the daily requirement.

Eventually, the daily B₁₂ requirement was estimated after correcting for cooking losses and bioavailability. However, there was no clarity on the exact amount lost during cooking. There was also uncertainty regarding the bioavailability or absorption of B₁₂. The variability or distribution of daily losses and absorption was not considered while deriving the requirement, and thus, it was not clear that if the stated requirement was an Estimated Average Requirement (EAR) or Recommended Dietary Allowance (RDA).

12.6.3 Factorial approach for estimating vitamin B₁₂ requirement

The requirement for B₁₂ can be defined using a factorial approach where daily loss of B₁₂ from the body stores are estimated after which, the amount required to replace the daily loss is adjusted for bioavailability of B₁₂ from the usual diet. Studies conducted on Western populations have measured

the daily rate of B₁₂ loss in humans using radioactive labelled B₁₂. The loss was measured as the mean decay in radioactivity after distribution of administered labelled B₁₂ throughout the body, in both healthy subjects and subjects with low serum B₁₂ concentrations, as well as those with pernicious anemia. From these studies, the daily B₁₂ loss, expressed as a percentage of total body stores, was 0.14% (Table 12.6.1).

Table 12.6.1: Studies reporting daily B₁₂ losses using radioactive labelled vitamin B₁₂ measured through whole body counting method¹¹⁻¹⁴

Author and year	n	Health status, age, n	Rate of loss (% loss/day)		
			Mean	Range	%CV
Bozian <i>et al</i> , 1963	16	Healthy, 25-41 y, n=3 Patients without pernicious anemia, 40-65 y, n=2 and with pernicious anemia, 57-85 y, n=11	0.14	0.09 – 0.15	27
Heyssel <i>et al</i> , 1966	14	Healthy, n=3 Patients with pernicious anemia, n=11	0.13	0.09 – 0.17	15
Boddy and Adams, 1968	14	Healthy, 42 y, n=1 Low vitamin B ₁₂ , 34-74 y, n=13	0.13	0.09 – 0.19	21
Adams and Boddy, 1968	6	Healthy, n=6	0.17	0.15 – 0.20	10

The total body stores have also been studied in Western populations, using a tracer dose of radioactive B₁₂, and showed an average body B₁₂ store of 2629 µg (Table 12.6.2).

Table 12.6.2: Total body stores of B₁₂ reported in literature¹⁵⁻¹⁸

Author and year	n	Subjects	Method of measurement	Total store (µg)	
				Mean	Range
Grasbeck <i>et al</i> , 1958	4	Autopsies	Sum of B ₁₂ content in liver, spleen, kidneys, heart, brain, muscles	3900	790 – 11100
Reizenstein <i>et al</i> , 1966	3	Healthy	Radioactivity measured using whole body counting	3030	-
Adams <i>et al</i> , 1970	18	Patients for surgery	Radioactivity and B ₁₂ content of liver biopsy after dosing with radioactive labelled B ₁₂	2528	960 – 5984
Bessent <i>et al</i> , 1980	4	Controls	Clearance of radioactive B ₁₂ from serum and whole body was used to estimate total body content of B ₁₂	1060	780 - 1350

Using the reported values of daily percentage loss of B₁₂ from the reported total body store, the average daily loss is estimated be 3.7 µg/d. Next, a bioavailability factor should be applied to the intake required to replace this loss, to provide the EAR. Further, the coefficient of variation (CV%) of daily loss and bioavailability should be used to provide the RDA.

In an alternative to the factorial approach, Institute of Medicine (IOM)², 1998 estimated the requirement based on a dose-response approach, where the amount of B₁₂ required to maintain an adequate hematological status (stable Hb, normal mean cell volume, normal reticulocyte response and serum B₁₂ levels) in persons with pernicious anemia was considered. The EAR was obtained from the estimated amount of B₁₂ required for adequate hematological status, which was then adjusted for a bioavailability of 50%, which is a conservative value obtained from dairy foods and most forms of meat and fish. Assuming a CV of 10% for the distribution of requirements, the RDA for B₁₂ was then determined as the 97.5th CI of this distribution. The recommendation based on this approach is still under development and there is still no strong consensus on the appropriate B₁₂ cut-off levels in the blood to determine adequate status. Additionally, the serum B₁₂ biomarker which was used by IOM for estimating requirement, assesses the total circulating B₁₂ and ~80% of this is bound to haptocorrin and not available for cellular uptake. Therefore, measuring serum B₁₂ status will be unreliable to reflect cellular B₁₂ status and the functional deficiency of B₁₂ cannot be assessed using serum B₁₂ alone. Therefore, a factorial approach serves as an alternative and reliable approach to estimate the requirements for B₁₂.

12.6.3 – Current recommendations for vitamin B₁₂

Given data available, the factorial approach, where daily loss of B₁₂ from the body stores is estimated and then adjusted for bioavailability of B₁₂ from usual diet. However, there are no studies which measured losses of B₁₂ from the body among Indians. Recently, a stable isotope labelled B₁₂ kinetics approach has been conducted at St. John's Research Institute, Bengaluru. In this approach, 12 fasted healthy adult subjects were administered a dose of 2.5 µg of ¹³C-labelled B₁₂ (cyanocobalamin) to estimate its kinetics over 9 hours¹⁹.

The principal labelled B₁₂ moiety that appeared in the plasma was ¹³C-methylcobalamin (meaning very efficient decyanation of cyanocobalamin), measured by a high-resolution analytical platform consisting Vanquish Flex Binary UHPLC coupled to a Q Exactive (LC-HRAM-MS, Thermo Scientific). Kinetic modelling of the plasma enrichment curve, using a two-compartment (central extracellular fluid and storage) model was performed to estimate the absorption and clearance (excretion) terms. The mean estimated bioavailability was 46% (SD±12%). Using the mean daily excretion used in the previous ICMR 2010 recommendation, of 0.1% of a 1 mg store, or a daily excretion of 1 µg/d, and adjusting for rounded off mean bioavailability of 50% at intakes of B₁₂ that are close to 1 µg/d, would provide an EAR of 2 µg/d for adults. The CV of absorption was ~20%, and this value was used to calculate the 97.5th percentile of the distribution of requirement, to define a RDA of 2.5 µg/d. The same value is proposed for school children and adolescents. For infants and pre-school children, this value should be reduced in relation to body size, since there are no data available on the requirement. A conservative EAR value of 1 µg/day based on a lower daily excretion, and RDA of 1.2 µg/day is suggested here until specific requirement data are available. This value is also consonant with the low levels (14%) of vitamin B₁₂ deficiency observed in 1-4y old children in the Comprehensive National Nutrition Survey (CNNS)²⁰. For school children and adolescents, there are also no data describing the daily B₁₂ loss and since no TUL is established (see section 12.6.4), the adult requirement is suggested for these age groups. (Table 12.6.3).

During pregnancy, studies have shown that the human foetus accumulates 0.1 µg B₁₂/d. These estimates were based on liver content of B₁₂ of infants born to mother with adequate B₁₂ status. The livers of still born foetuses at different gestational age were also studied for their B₁₂ content, and in vitamin B₁₂ deficient mothers, fetal liver stores greatly reduced²¹. Studies have shown that as the foetal age increased the accumulation of B₁₂ also increased reaching a maximum amount of 20-25 µg B₁₂²². In another study, the liver of still births and neonates that died within 72 hrs of delivery were studied for their B₁₂ content and this study showed that a foetus with normal weight had a liver B₁₂

content of 19.4-31.3 µg²³. In another study, B₁₂ and folate content of foetuses showed that at term, the content was 27.3 µg B₁₂²⁴. Based on these studies, and a simple division of accumulated B₁₂ by the number of days of pregnancy, it is suggested that an additional mean requirement of 0.1 µg B₁₂/d for adequate foetal growth, and when adjusted for 50% absorption, is 0.2 µg/d. When adjusted for an assumed variability of 10%, this yields an additional pregnancy RDA of 0.25 µg/d.

During lactation, the additional B₁₂ requirement was based on the B₁₂ content of milk. On average, breast milk contains 0.6-0.7 µg B₁₂/L and the breastmilk output in Indian women is 700 ml/d in first 6 months²⁵. Therefore, the additional loss of B₁₂ to the body during lactation is about 0.4 µg/d, and the requirement during lactation, adjusted for 50% absorption, is 0.8 µg/d. When adjusted for an assumed variability of 10%, this yields an additional pregnancy RDA of 1.0 µg/d.

12.6.4 – Tolerable Upper Limit for vitamin B₁₂

There is limited evidence to suggest a level at which adverse events can be observed. In the Norwegian Vitamin (NORVIT) intervention trial, patients with acute myocardial infarction received 400 µg of B₁₂ daily for 3 years and reported no serious adverse events²⁶. In another study (HOPE 2), patients with vascular disease or diabetes aged >55 y received 1 mg B₁₂ daily for 5 years and reported no serious adverse event with this treatment²⁷. Additionally, the IOM (1998) also states that there is no adverse effect associated with excess B₁₂ intake from foods or supplements in healthy individuals². This could be due to limited absorption from gastrointestinal tract with high doses, and therefore, IOM did not establish an upper limit for B₁₂. The evidence from the limited data is not sufficient for deriving Tolerable Upper Limit (TUL). Further, the Indian population mostly consume vegetarian diets which do not provide B₁₂, except milk and its product. Therefore, high intakes of B₁₂ from foods are unlikely.

Table 12.6.3: Current recommendations for vitamin B₁₂ across age groups

Age group (y)	EAR (µg/d)	RDA (µg/d)
Infants (0-6 m)	-	Breast milk
Infants and pre-school children (6 m - 5 y)	1.0	1.2
School children and adolescents (5-17 y)	2.0	2.5
Adults	2.0	2.5
Pregnant (Additional)	0.20	0.25
Lactating (Additional)	0.8	1.0

References

1. Watanabe F. Vitamin B₁₂ sources and bioavailability. *Experimental biology and medicine*. 2007 Nov; 232(10):1266-74.
2. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. *Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B₆, folate, vitamin B₁₂, pantothenic acid, biotin, and choline*. National Academies Press (US); 1998.
3. Heaton EB, Savage DG, Brust JC, Garrett TF, Lindenbaum J. Neurological aspects of cobalamin deficiency. *Medicine* 1991; 70:229-44.
4. Bottiglieri T. Folate, vitamin B₁₂, and neuropsychiatric disorders. *Nutrition Reviews*. 1996; 54:382-90.
5. National Sample Survey Office. Nutritional Intake in India, 2011-12. 560, NSS 68th Round. National Statistical Organization. Government of India. 2014.
6. Ministry of Health and Family Welfare (MoHFW), Government of India, UNICEF and Population Council. Comprehensive National Nutrition Survey (CNNS) National Report. New Delhi, 2019.
7. Jayashri R, Venkatesan U, Rohan M, Gokulakrishnan K, Rani CS, Deepa M, Anjana RM, Mohan V, Pradeepa R. Prevalence of vitamin B₁₂ deficiency in South Indians with different grades of glucose tolerance. *Acta Diabetol*. 2018; 55(12):1283-93.
8. Sivaprasad M, Shalini T, Reddy PY, Seshacharyulu M, Madhavi G, Kumar BN, Reddy GB. Prevalence of vitamin deficiencies in an apparently healthy urban adult population: Assessed by subclinical status and dietary intakes. *Nutrition*. 2019 Jul 1; 63:106-13.
9. 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, ICMR, New Delhi, 1989.
10. ICMR. Nutrient requirements and recommended dietary allowances for Indians. A report of the expert group of the Indian Council of Medical Research National Institute of Nutrition Hyderabad; 2010.
11. Bozian RC, Ferguson JL, Heyssel RM, Meneely GR, Darby WJ. Evidence concerning the human requirement for vitamin B12. Use of the whole body counter for determination of absorption of vitamin B₁₂. *American Journal of Clinical Nutrition*. 1963; 12: 117–129.
12. Heyssel RM, Bozian RC, Darby WJ, Bell MC. Vitamin B₁₂ turnover in man. The assimilation of vitamin B₁₂ from natural foodstuff by man and estimates of minimal daily dietary requirements. *American Journal of Clinical Nutrition*. 1966; 18: 176-184.
13. Boddy K, Adams JF. Excretion of cobalamins and coenzyme B₁₂ following massive parenteral doses. *American Journal of Clinical Nutrition*. 1968; 21: 657–664
14. Adams JF, Boddy K. Metabolic equilibrium of tracer and natural vitamin B12. *The Journal of laboratory and clinical medicine*. 1968 Sep 1; 72(3):392-6.
15. Grasbeck R, Nyberg W, Reizenstein P. Biliary and Fecal Vit. B12 Excretion in Man. An Isotope Study. *Proceedings of the Society for Experimental Biology and Medicine*. 1958 Apr; 97(4):780-4.
16. Reizenstein P, Ek G, Matthews CM. Vitamin B12 kinetics in man. Implications on total-body-B12-determinations, human requirements, and normal and pathological cellular B12 uptake. *Physics in Medicine & Biology*. 1966 Apr; 11(2):295.
17. Adams JF, Tankel HI, MacEwan F. Estimation of the total body vitamin B12 in the live subject. *Clinical science*. 1970 Jul; 39(1):107-13.
18. Bessent RG, Watson WS, MacDonald CM, Adams JF. Application of the occupancy principle in studies of the metabolism of vitamin B12 in man. *Clinical Science*. 1980 Feb; 58(2):169-71.
19. Devi S, Pasanna RM, Shamshuddin Z, Bhat K, Sivadas A, Mandal AK, Kurpad AV. Measuring vitamin B-12 bioavailability with [13C]-cyanocobalamin in humans. *The American Journal of Clinical Nutrition*. 2020 Aug 25.

20. Comprehensive National Nutrition Survey, 2016-2018. Ministry of Health & Family Welfare, Govt of India. 2019.<https://nhm.gov.in/WriteReadData/1892s/1405796031571201348.pdf>
21. Baker SJ, Jacob E, Rajan KT, Swaminathan SP. Vitamin-B₁₂ deficiency in pregnancy and the puerperium. *BMJ*. 1962; 1(5293):1658.
22. Loria A, Vaz-Pinto A, Arroyo P, Ramírez-Mateos C, Sánchez-Medal L. Nutritional anemia. VI. Fetal hepatic storage of metabolites in the second half of pregnancy. *Journal of Pediatrics*. 1977; 91(4):569-73.
23. Vaz Pinto A, Torras V, Sandoval JF, Dillman E, Mateos CR, Cordova MS. Folic acid and vitamin B₁₂ determination in fetal liver. *American Journal of Clinical Nutrition*. 1975; 28(10):1085-6.
24. Pawlak R, Vos P, Shahab-Ferdows S, Hampel D, Allen LH, Perrin MT. Vitamin B₁₂ content in breast milk of vegan, vegetarian, and non-vegetarian lactating women in the United States. *American Journal of Clinical Nutrition*. 2018; 108(3):525-31.
25. Madhavapeddi R and Narasinga Rao BS. Energy balance in lactating undernourished Indian women. *European Journal of Clinical Nutrition*. 1992; 46: 349-354.
26. Bønaa KH, Njølstad I, Ueland PM, Schirmer H, Tverdal A, Steigen T, Wang H, Nordrehaug JE, Arnesen E, Rasmussen K. Homocysteine lowering and cardiovascular events after acute myocardial infarction. *New England Journal of Medicine*. 2006 Apr 13; 354(15):1578-88.
27. Lonn E. Heart Outcomes Prevention Evaluation (HOPE) 2 Investigators. Homocysteine lowering with folic acid and B vitamins in vascular disease. *Nat Clin Pract Cardiovasc Med*. 2006;3:414-5.

12.7 ASCORBIC ACID (VITAMIN C)

12.7.1. Function

Ascorbic acid or vitamin C is a six-carbon lactone which is synthesized from glucose by many animals, but not by man. It is an electron donor and therefore a reducing agent or antioxidant. All of its biochemical and molecular functions can be accounted for, by this function. Ascorbic acid acts as an electron donor for 8 enzymes. e.g. hydroxylation of collagen, biosynthesis of carnitine, and catecholamines¹. Ascorbic acid in gastric juice has been shown to prevent the formation of N-nitroso compounds, which are potentially mutagenic². High intakes of ascorbic acid correlate with reduced gastric cancer risk³. Ascorbic acid protects low-density lipoproteins *ex vivo* against oxidation and may function similarly in the blood.

There are reports that the antioxidant properties of ascorbic acid may stabilize folate in food and in plasma, and cause increased excretion of oxidized folate derivatives in human scurvy⁴. Similarly, Ascorbic acid promotes absorption of soluble non-heme iron possibly either by chelation or simply by maintaining the iron in the reduced (ferrous, Fe²⁺) form or both. This effect can be achieved with the amounts of ascorbic acid obtained in foods. However, the amount of dietary ascorbic acid required to increase iron absorption ranges from 25 mg upwards and depends largely on the amount of inhibitors, such as phytates and polyphenols present in the meal⁵.

Recent studies have shown potential health effects of ascorbic acid in decreasing risk of cardiovascular accidents and asthma, cancer morbidity and mortality, prevention of cataract and its impact on common cold⁶.

12.7.2. Overview of significant scientific information

The amount of ascorbic acid required to prevent or cure early signs of deficiency is between 6.5 mg and 10 mg/day. In many developing countries, limitations in the supply of ascorbic acid are often determined by seasonal factors as well as food handling and culinary practices.

Dietary sources

Good sources of ascorbic acid include citrus fruits, tomatoes, berries and green vegetables. Potato is an important staple food in many countries that provide the required ascorbic acid even though its ascorbic acid concentration is low. The average ascorbic acid content of raw foodstuffs is given in Table 12.7.1

Stability of ascorbic acid during processing

Ascorbic acid is sensitive to heat, light and oxygen and interacts with copper, iron and tin⁷. In the dry state, ascorbic acid is reasonably stable in air, but in solution, it is rapidly oxidized. During storage, losses have been found to vary (8-35%). It may also darken on exposure to light, moisture and heat. Cooking typically destroys ascorbic acid by accelerating the oxidation reaction. Exposure to low pH value enhances its stability while high pH is deleterious⁸. Oxidative destruction of ascorbic acid

occurs as a consequence of two factors, temperature increase and length of exposure. Heat processing methods have different impacts on ascorbic acid stability. After sterilization of milk at 131°C for 5

Table 12.7.1: Average ascorbic acid content (raw) per 100g

Food stuffs	Ascorbic acid (mg)*
Cereals	0.00
Pulses and Legumes	0-3
Leafy vegetables	60-250
Roots and Tubers	10-40
Other vegetables	20- 80
Nuts & Oilseeds	0-7
Condiments & Spices	0-50
Fruits	45-600
Fish	10-30
Meat and Poultry	2.00-20
Milk and milk products	1-6

*Cooking reduces ascorbic acid content by more than 60-75%.

minutes, retention was 35% while ultra-high temperature processing at 141°C for 4 seconds retains 57% of the original ascorbic acid content⁹. Baking seems to have a more destructive effect on ascorbic acid than boiling. Retention of ascorbic acid in sweet potato after 30 min of boiling was 45.6% while the value after the same time period under baking condition was 30.3%¹⁰. Boiling causes losses to an average 10% higher than with steaming¹¹. Overcooking leads to a major loss of nutrient.

Ascorbic acid was found to be more stable during frying than on pressure cooking and boiling, since the temperature inside the food never exceeds 100°C. In addition, frying is usually for very short periods and there is no leaching of water-soluble vitamins¹². Iron absorption studies have shown a reduction or lack of effect on ascorbic acid when the vitamin was added to meals before cooking, baking, or even warming for prolonged duration^{13,14}. Traditional household processing method used in Punjab was found to retain about 74% and provided 46 mg of ascorbic acid per 100 g¹⁵. Also the retention of ascorbic acid was better when GLV were mixed with other food ingredients and the prepared dishes had much higher ascorbic acid (losses of only 33%) than when the main ingredient vegetable alone was cooked¹⁶.

12.7.3. Requirement

For more than a century, the essentiality of ascorbic acid in preventing scurvy has been undisputed; yet, the actual requirement remains widely debated, with different approaches, the result of which has been the disparities among various countries across the globe in fixing the RDA, ranging from 40 to 220 mg/day. In the USA, the RDAs for males have been successively changed from 75mg/day in 1943¹⁷, 60mg/day in 1968¹⁸, 1980¹⁹ and 1989²⁰; 45mg/day in 1974²¹, while the most recent revision in 2000 was fixed at 90 mg/day⁶. Countries like UK²², Australia²³, New Zealand²³ and India²⁴ have based their RDA on FAO/WHO recommendations at 40-45 mg/day and have been countries with the lowest RDAs for ascorbic acid. In contrast, the RDA in Netherlands²⁵ is 75 mg/day and Germany, Austria and Switzerland²⁶ is 110 mg/day. The Chinese have adopted 2 recommendations -100 mg/day to prevent scurvy and 200 mg/day for decreasing non communicable diseases risk²⁷.

The cause for such disparities lies in the criteria and estimates used in deriving EARs and RDAs. Estimates of the values of body pool and absorption efficiency vary which contributes to the variations in RDAs. Four distinct criteria are used globally: Intakes sufficient for –

- i. Preventing scurvy
- ii. Partly saturating immune cells (neutrophils) with ascorbic acid while limiting its excretion
- iii. Replacing daily turnover to maintain adequate plasma levels
- iv. Attempting optimization of individual's health by using optimal rather than sufficient amount of vitamin C.

Scurvy can be effectively prevented with relatively little vitamin C – some have estimated as little as 10 mg/day²⁸. Estimating EARs using this as criteria is potentially erroneous as they are based on body pool estimations from a time when the complexity of vitamin C pharmacokinetics was not fully understood²⁹.

The criterion chosen for the USA and Canadian EAR estimation⁶ was “the vitamin C intake that maintains near-maximal neutrophil concentrations with minimal urinary excretion of ascorbate” However, this has been criticized over choosing extrapolated values on graphs rather than actual data points, concentrations used for determining antioxidant protection and the rationale of using emerging urinary excretion as an indicator for saturation of body stores³⁰. Body pool estimations are basis for calculating EARs by EFSA and also by WHO/FAO. WHO/FAOs recommendations come from the calculation based on a value half way between body pool saturation in men (1500 mg) and the point where deficiency occurs (about 300–400 mg), i.e. a value of 900 mg. Multiplying with the rate of

turnover (2.9% per day) and correcting for absorption efficiency (85%) and twice the estimated coefficient of variation (estimated at 20%) results in 44 mg/day rounded up to the recommendation of 45 mg/day. The EFSA on the other hand, used the following rationale: the body pool in men (1500 mg) is multiplied by the metabolic loss (2.9% per day) and compensated for the urinary loss (25% per day) and absorption efficiency (80%). Adding twice the estimated coefficient of variation (estimated at 10%) results in the recommendation of 110 mg/d for men³⁰.

Methodology for calculating EAR and RDA of Ascorbic acid

Males

The present recommendation is based on the rationale that the body pool saturation of ascorbic acid was 900mg. Considering metabolic loss of 2.9% per day, compensating for the urinary loss of 25% per day, and taking absorption efficiency of 50% in Indian foods³¹, the EAR was calculated as 65 mg/day for males. A CV of 10% was used and RDA of 78 mg/day was arrived at, which was rounded off to 80 mg per day.

Females

As no value for metabolic losses was available in women, the EAR for women was extrapolated from the EAR for men. On the basis of differences in reference body weight, EAR was set at 55 mg/day for women, and using a CV of 10% the RDA was calculated as 65 mg/day.

Children

EARs for children have been estimated based on relative body weights, as a function of reference weights and growth, using the formula⁶:

$$\begin{aligned}\text{EAR}_{\text{child}} &= \text{EAR}_{\text{adult}} \times F, \text{ where} \\ F &= (\text{Weight of child}/\text{weight of adult})^{0.75} \times (1+GF) \\ GF &= 0.57 \text{ for ages up to 1 years,} \\ &\quad 0.25 \text{ for ages 1 to 3,} \\ &\quad 0.06 \text{ for ages 4 to 6,} \\ &\quad 0.13 \text{ for ages 7 to 9,} \\ &\quad \text{In ages 10 to 15} - 0.11 \text{ for boys and } 0.08 \text{ for girls,} \\ &\quad \text{In ages 16 to 17} - 0.08 \text{ for boys and } 0.03 \text{ for girls.}\end{aligned}$$

Pregnancy

For pregnant women, in the absence of precise data regarding transfer of maternal vitamin C to the fetus, evidence that intakes of 7 mg/day of vitamin C will prevent young infants from developing scurvy, the EAR for pregnancy was estimated to increase by 10 mg/day over the vitamin C requirement for the non-pregnant woman. Subsequently, 15 mg/day additional requirements were added to RDA⁶.

Lactation

To estimate the EAR for lactation, the average vitamin C produced in milk, 40 mg/day during the first 6 months of lactation, was added to the EAR of the nonlactating women. Accordingly, an additional allotment of extra 50 mg/day was added over and above the regular RDA of pre-pregnant state⁶.

12.7.4. TUL for ascorbic acid

The Tolerable Upper Intake Level (UL) is the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects in almost all individuals. Although members of the general population should be advised not to exceed the UL routinely, intake above the UL may be appropriate for investigation within well-controlled clinical trials. In light of evaluating possible benefits to health, clinical trials of doses above the UL should not be discouraged, as long as subjects

participating in these trials have signed informed consent documents regarding possible toxicity and as long as these trials employ appropriate safety monitoring of trial subjects. Also, the UL is not meant to apply to individuals who are receiving vitamin C under medical supervision.

Reviews of high vitamin C intakes have indicated low toxicity; adverse effects have been reported primarily after very large doses (greater than 3 g/day)³². Saturable intestinal absorption and renal tubular reabsorption data suggest that overload of ascorbic acid is unlikely in humans³³. Possible adverse effects associated with very high intakes have been reviewed and include: diarrhea and other gastrointestinal disturbances, increased oxalate excretion and kidney stone formation, increased uric acid excretion, pro-oxidant effects, systemic conditioning (“rebound scurvy”), increased iron absorption leading to iron overload, reduced vitamin B12 and copper status, increased oxygen demand, and erosion of dental enamel^{34,35}.

Gastrointestinal disturbances such as nausea, abdominal cramps, and diarrhoea are the most common adverse effects of high vitamin C intake³⁶. These effects are attributed to the osmotic effect of unabsorbed vitamin C passing through the intestine. Based on considerations of causality, relevance, and the quality and completeness of the database, osmotic diarrhoea and related gastrointestinal disturbances were selected as the critical endpoints on which to base a UL.

Human data suggest that an intake of vitamin C greater than 3 g/day is likely to cause osmotic diarrhoea in many individuals, although some reports involving a few individuals suggest this may occur at 3 g/day. Thus, the 3-g/day intake is considered a LOAEL. An uncertainty factor (UF) of 1.5 was selected to extrapolate the LOAEL to a NOAEL. Thus, the 3 g/day intake is considered a LOAEL, and a NOAEL or TUL of 2 g/day is estimated for adult humans⁶.

For children, TULs were adjusted to weight at the rate similar to adults, i.e., 31 mg/kg/day and as 36 mg/kg/day in case of girls. The values were rounded off to the nearest 50mg. Since no teratogenicity is recorded in ascorbic acid overdose, the TULs in pregnant and lactating women remain same as in non-pregnant state.

12.7.5. Summary of RDA and TUL for ascorbic acid

Effect of ascorbic acid on non-heme iron absorption in human

Ascorbic Acid with its reducing and chelating properties is the most efficient non-heme iron absorption enhancer, when its stability in the food vehicle is ensured³⁷. An adequate intake of ascorbic acid is however required in the context of fixing the requirement of iron from vegetable foods. The committee on RDA (2010) examined the possibility of ensuring adequate iron absorption from typical meals consumed in India, which is considered to be high in inhibitors and low in promoters of iron absorption. Absorption studies have confirmed that increase in iron absorption is observed only when the two are consumed together and not when ascorbic acid was administered several hours before meal³⁸. In the context of instability of ascorbic acid during food processing (60-70%) and storage, obtaining sufficient ascorbic acid to improve iron availability is a difficult proposition. Also the molar proportion of ascorbic acid to iron to be present in the food for an effective improvement in iron absorption has not been established with Indian diet.

A number of research studies have been carried out on this aspect, covering both semi-synthetic meal and natural meal.

Effect of different ascorbic acid: iron ratios on iron absorption

Promotion of iron absorption in the presence of ascorbic acid is more pronounced in a composite meal containing inhibitors of iron absorption. The effect of ascorbic acid and other enhancers or inhibitors of iron absorption was examined in several studies using the single meal

technique in which the non-heme iron component of the meal is labelled either with a radioisotope or stable isotope of iron.

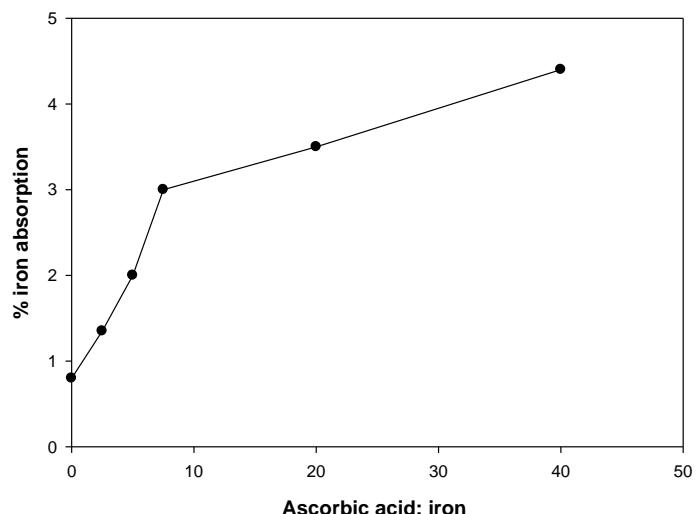
Table 12.7.2: RDA of ascorbic acid (vitamin C) for various physiological groups

Physiological Group	Body weight (Kg)	RDA 2020		TUL (mg/day)	H-AR (mg/day)	IOM RDA (mg/day)
		EAR (mg/day)	RDA (mg/day)			
Adult Men	65	65	80	2000	90	90
Adult Women	55	55	65	2000	80	75
Pregnant	55+GWG	55+10	65+15	2000	80	85
Lactating	55+	55+40	65+50	2000	145	120
Infants (0-6 mo)	5.8	-	20 (AI)	-	-	40 (AI)
Infants (6-12 mo)	8.5	-	27 (AI)	-	-	50 (AI)
Children (1-3 y)	11.7	22	27	350	15	15
Children (4-6 y)	18.3	27	32	550	25	25
Children (7-9 y)	25.3	36	43	800	40	25 (7-8) 45 (9)
Boys (10-12 y)	34.9	45	54	1050	60	45
Girls (10-12 y)	36.4	44	52	1300	60	45
Boys (13-15 y)	50.5	60	72	1550	60	75
Girls (13-15 y)	49.6	55	66	1800	60	75
Boys (16-18 y)	64.4	69	82	1950	85	75
Girls (16-18 y)	55.7	57	68	2000	75	75

H-AR: Harmonized average requirement

A meal containing low-to-medium levels of inhibitors requires the addition of ascorbic acid at a molar ratio of 2:1 (e.g., 20 mg ascorbic acid: 3 mg iron)³⁹. To promote absorption in the presence of high levels of inhibitors, ascorbic acid needs to be added at a molar ratio in excess of 4:1, which may be impractical⁴⁰. The dose-response relationship obtained from a semi-synthetic meal containing 4:1 mg iron and ascorbic acid in doses ranging from 25 to 1000 mg is best described by a steep linear response up to a 7.5 molar ratio of ascorbic acid to iron, followed by a less pronounced linear dose-response for molar ratios above 7.5, as shown in figure 12.7.1^{38,39}. All such data advocating enhanced ascorbic acid intake were of short term duration or mainly confined to meals and not whole diets. Later data by Cook and Reddy⁴¹ demonstrated that the beneficial effect of high levels of ascorbic acid on dietary non-heme iron absorption was lost on long term consumption, raising doubts about the efficacy of such step to improve iron absorption. Data within India show that a dietary intake of 40 mg of ascorbic acid can be easily achieved and such consumption was associated with good iron absorption and good iron status¹⁵. Thus, at least 40 mg of ascorbic acid should be available in the small intestine at the time of iron absorption from a day's meal.

Figure 12.7.1: The effect of ascorbic acid on iron absorption from a semi-synthetic meal



References

1. Levine M. New concepts in the biology and biochemistry of ascorbic acid. *New England Journal of Medicine.* 1986 Apr 3; 314(14):892-902.
2. Mirvish SS. Effects of vitamins C and E on N-nitroso compound formation, carcinogenesis, and cancer. *Cancer.* 1986 Oct 15; 58(S8):1842-50.
3. Liu and Russel RM. Nutrition and gastric cancer risk: An update. *Nut Rev* 66(5):237-249, 2008.
4. Stokes PL, Melikian V, Leeming RL *et al.* Folate metabolism in scurvy. *American Journal of Clinical Nutrition.* 1975; 28(2):126-9.
5. Siegenberg D, Baynes R.D, Bothwell TH, *et al.* Ascorbic acid prevents the dose-dependent inhibitory effects of polyphenols and phytates on nonheme-iron absorption. *American Journal of Clinical Nutrition.* 1991; 53: 537-41.
6. Monsen ER. Dietary reference intakes for the antioxidant nutrients: vitamin C, vitamin E, selenium, and carotenoids. *Journal of the Academy of Nutrition and Dietetics.* 2000 Jun 1; 100(6):637.
7. Bauernfeind JC. Antioxidant function of L-ascorbic acid in food technology. *International journal for vitamin and nutrition research. Supplement=Internationale Zeitschrift fur Vitamin-und Ernahrungsorschung. Supplement.* 1985; 27:307.
8. Quadri SF, Liang YT, Seib PA, Deyoe CW, Hoseney RC. Stability of 1-ascorbate 2-sulfate and 1-ascorbate in wheat foods and milk. *Journal of Food Science.* 1975 Jul; 40(4):837-9.
9. Raffier. The influence of added trace elements on the stability of vitamins in milk based infant formulas. Dissertation for Doctor's Degree. 1998.
10. Abdel-Kader ZM. Effect of boiling and baking on the content of some nutrients of sweet potatoes. *Food/Nahrung.* 1991; 35(3):321-4.
11. Paulus K. Changes in nutritional quality of foods in catering. *Journal of Nutritional Science and Vitaminology.* 1990; 36: Suppl (1), 35S-45S.
12. Fillion L, Henry CJ. Nutrient losses and gains during frying: a review. *International journal of food sciences and nutrition.* 1998 Jan 1; 49(2):157-68.
13. Sayer MH, Lynch SR, Jacobs P, Charlton RW, Bothwell TH, Walker RB, and Mayet F. The effects of ascorbic acid supplementation on the absorption of iron in maize, wheat and soy. *British Journal of Haematology.* 1973; 24: 209-18.
14. Hallberg L, Rossander L, Persson H. and Svahn E. Deleterious effects of prolonged warming of meals on ascorbic acid content and iron absorption. *American Journal of Clinical Nutrition.* 1982; 36: 846-50.
15. Gupta S, Bains K. Traditional cooked vegetable dishes as important sources of ascorbic acid and β -carotene in the diets of Indian urban and rural families. *Food and nutrition bulletin.* 2006 Dec; 27(4):306-10.
16. Agte V, Tarwadi K, Mengale S, Hinge A, Chiplonkar S. Vitamin profile of cooked foods: how healthy is the practice of ready-to-eat foods? *International journal of food sciences and nutrition.* 2002 Jan 1; 53(3):197-208.
17. Food and Nutrition Board of the National Research Council. Recommended dietary allowances. *Nutrition Reviews* 1. 1943; (6):164-8.
18. Staff of National Academy of Science. Recommended dietary allowance, 7th ed. Washington, DC: National Academy of Science. 1968.
19. Food and Nutrition Board of the National Research Council. Recommended dietary allowances, 9th ed. Washington, D.C.: National Academy of Sciences. 1980.

20. National Academy of Sciences. National Research Council Subcommittee on the Tenth Edition of the Recommended Dietary Allowances. Recommended Dietary Allowances, 10th ed. Washington (DC): National Academy of Sciences. 1989.
21. Committee on Dietary Allowances. Recommended dietary allowances, 8th ed. Washington DC: National Academy of Sciences. 1974.
22. Dietary reference values for food energy and nutrients for the United Kingdom. Report of the panel on dietary reference values of the committee on medical aspects of food policy. Report on Health and Social Subjects (London). 1991. 41: 1–210.
23. National Health and Medical Research Council. Nutrient reference values for Australia and New Zealand: Executive summary. In Department of Health and Ageing, editor, 89. Canberra: National Health and Medical Research Council. 2006.
24. Nutrient requirements and recommended dietary allowances for Indians. A report of the expert group of the Indian Council of Medical Research. Hyderabad: National Institute of Nutrition; Indian Council of Medical Research. 2010.
25. Health Council of the Netherlands. Dietary reference values for vitamins and minerals for adults. 2018.
26. German Nutrition Society (DGE). AÖaSS. D-A-CH reference levels for nutrient intake. Bonn. 2015b.
27. Chinese Nutrition Society. 2014. Chinese dietary reference intakes. Beijing: China Science Publishing House.
28. Hodges RE, Hood J, Canham JE, Sauberlich HE and Baker EM. Clinical manifestations of ascorbic acid deficiency in man. *The American Journal of Clinical Nutrition*. 1971; (4):432–43.
29. Lykkesfeldt J and Tveden-Nyborg P. The pharmacokinetics of vitamin C Nutrients. 2019;11(10):2412.
30. Carr AC, Lykkesfeldt J. Discrepancies in global vitamin C recommendations: a review of RDA criteria and underlying health perspectives. *Critical Reviews in Food Science and Nutrition*. 2020 Mar 28:1-4.
31. ICMR. Nutrient Requirements and Recommended Dietary Allowances for Indians. A Report of the Expert Group of the Indian Council of Medical Research, National Institute of Nutrition, Hyderabad, 1990.
32. Johnston CS. Biomarkers for establishing a tolerable upper intake level for vitamin C. *Nutrition reviews*. 1999 Mar 1; 57(3):71-7.
33. Blanchard J, Tozer TN, Rowland M. 1997. Pharmacokinetic perspectives on mega-doses of ascorbic acid. *American Journal of Clinical Nutrition*. 1997; 66:1165–1171
34. Hornig DH. The safety of high vitamin C intake in man. *Vitamin C*. 1981; 225.
35. Rivers, JM. Safety of high-level vitamin C ingestion. *Annals of New York Academy Sciences*. 1987; 498:445–454.
36. Hoffer A. Ascorbic acid and toxicity. *The New England journal of medicine*. 1971 Sep; 285(11):635-6.
37. Conrad ME and Schade SG. Ascorbic acid chelates in iron absorption: a role for hydrochloric acid and bile. *Gastroenterology*. 1968; 55: 35–45.
38. Cook JD and Monsen ER. Ascorbic acid, the common cold, and iron absorption. *American Journal of Clinical Nutrition*. 1997; 30: 235–241.
39. Hurrell R. How to ensure adequate iron absorption from iron-fortified food. *Nutrition reviews*. 2002; 60:S7-15.
40. Teucher, Olivares, Cori. Enhancers of iron absorption: ascorbic acid and other organic acids. *International Journal for Vitamin and Nutrition Research*. 2004 Nov 1; 74(6):403-19.
41. Cook JD, Reddy MB. Effect of ascorbic acid intake on nonheme-iron absorption from a complete diet. *The American journal of clinical nutrition*. 2001 Jan 1; 73(1):93-8.

13. FAT SOLUBLE VITAMINS

13.1 VITAMIN A

Significance

Vitamin A is an essential nutrient, must be provided as a part of the diet. Pro-vitamin A carotenoids such as β -carotene, α -carotene, and β -cryptoxanthin abundant in plant foods (vegetables and fruits) are the primary dietary source of vitamin A. Preformed vitamin A (retinyl esters) is obtained from animal foods such as milk, egg and meat products. In addition, fortified foods (milk and oil), health beverages and supplements also provide preformed vitamin A. Vitamin A plays a vital role in vision, maintenance of overall health due to the involvement in a wide variety of biological activities including the embryonic development and immune system.

Deficiency

One of the earliest manifestations of vitamin A deficiency (VAD) is night blindness and more severe deficiencies include ocular changes leading to blindness, particularly in young children. National Nutrition Monitoring Bureau surveys indicate that, during the past three decades there has been virtual disappearance of keratomalacia, and a sharp decline in the prevalence of clinical symptoms of VAD (Bitot's spots 0.4%) in India¹⁻². The prevalence of sub-clinical vitamin A deficiency among pre-school children reduced from 64% in the year 2012¹ to 17.4% in the year 2018³. Though there has been a significant reduction in severe Vitamin A deficiency over the last three decades, it remains a nutritional challenge in many sections of the populations. Poor dietary intakes imposed by poverty or knowledge, recurrent infections and concomitant deficiency of other nutrients (protein, zinc etc.,) are thought to be the major etiological factors of vitamin A deficiency in populations.

Vitamin A homeostasis

The dietary preformed retinol is absorbed in the intestinal cells via a saturable process, but the mechanisms remain unknown as yet⁴. The dietary pro-vitamin A carotenoids are assembled into micells during gastro-inestinal digestion and absorbed in enterocytes via a carrier mediated process. Within the intestinal cells⁴, part of the provitamin A carotenoids (β -carotene: equivalent to 2 retinol; while α carotene and β -cryptoxanthine: yields 1 molecule of retinol) undergoes central cleavage to yield retinol, a process that appears to depend on the actual vitamin A status of the host -lower the status; higher the conversion. The intestinal retinol (esterified) or carotenoids are then assembled in chylomicrons and secreted into lymphatic circulation⁵ taken up and stored by the liver. About 80% of the whole body retinol was found in liver, while the rest is in adipose (15%) and other tissues. The retinol is then excreted into the blood via a carrier protein called retinol binding protein (RBP), which in turn delivers it to the target tissues⁶. The eye, for its visual function and the skin are the two most important tissues that have high demands of retinol for their normal function. Predominant part of vitamin A, in the form of oxidized vitamin A metabolites are excreted in the urine⁷. In addition, the retinol is metabolized in liver into variety of metabolites, which are mainly excreted through bile⁷. Therefore, entero-biliary axis is the major pathway that regulates the whole body vitamin A homeostasis. Numerous other factors such as inflammation, protein and zinc deficiency are known to negatively influence the vitamin A utilization, independent of actual vitamin A status⁷.

Carotene conversion

In addition to preformed vitamin A from animal derived foods, the pro-vitamin A carotenoids forms the major source of vitamin A from green leafy vegetables and fruits. Theoretically each β -carotene molecule yields two retinol (RAE), while α -carotene and β -cryptoxanthine yield one molecule of retinol. However, multiple dietary and host related factors influence these ratios. The conversion of β -carotene (BC) to vitamin A is most debated, as it forms an important factor in setting the dietary requirements and in assessing the dietary vitamin A adequacy of the population. The knowledge on the carotenoid speciation, digestion, metabolism and its conversion expanded in both molecular and clinical fronts in the past decade, particularly with the cloning and characterization of β -carotene dioxygenase⁵ and application of stable isotopic tracer studies⁸⁻⁹. The important development that needs to be considered are carotenoid conversion related to the dose administered (higher the intakes, lower the conversion) and the vitamin A status of subjects (higher the status; lower the conversion). The intact carotenoids are both absorbed and converted to vitamin A post absorptively in multiple tissues.

The dietary guidelines of IOM considered 12:1 ratio as opposed to 6:1 by WHO, while the previous ICMR RDA committee (2010) considered an 8:1 ratio by triangulation to find the best conversion ratios that explains the subclinical vitamin A deficiency across different regions of the world. The carotene conversion ratios are mainly derived from studies that measured the plasma area under the curve (AUC) of a standard β -carotene, retinol and test food which varied from 2:1 to 55:1. These variations are primarily due to base line vitamin A status, food matrix and consumption of dietary fat⁹. Since, speciation and composition of carotenoids in food matrix is unique at least to food groups (GLV vs carrot, potato, fruits etc.), extrinsic labelling studies are not suitable to assess the conversion of endogenous carotenoids¹⁰. Literature search in PubMed with key words (carotene) OR ("green leafy vegetables") AND (conversion) AND (equivalence) retrieved 41 studies, of which 6 studies¹¹⁻¹⁵ reported the conversion estimates using intrinsic labelling of test foods, reported post absorptive conversion (≥ 21 days), dose administered and base line vitamin A status (plasma vitamin A) of the study subjects. Of these, one study measured the conversion of standard β -carotene in oil¹² and thus was not considered for inclusion. Another study¹⁶ that measured the bioconversion of carotene from kale in children reported 13 and 11:1 conversion at 1.5 mg of β -carotene intake, and was considered an outlier. This study also did not measure the sequential blood samples in all the children.

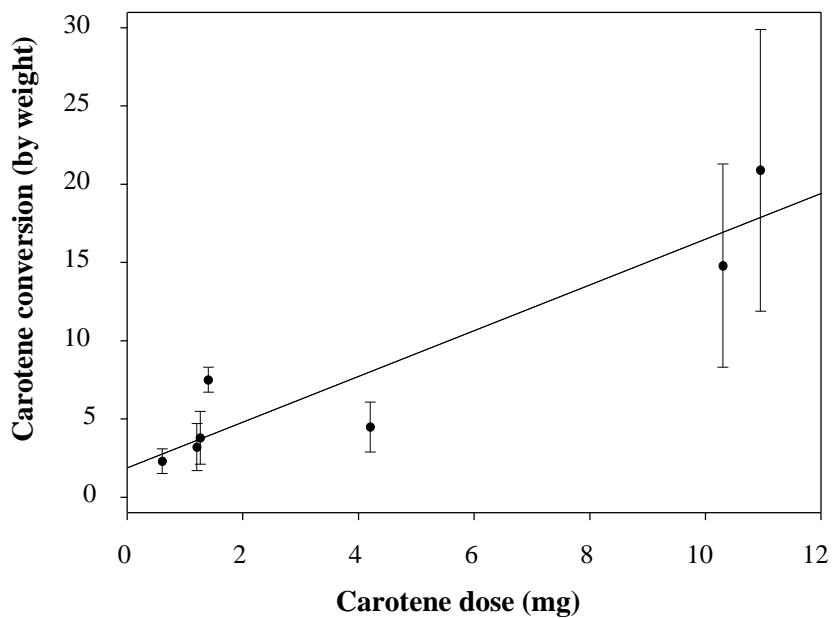
Table 13.1.1 shows the details of age, base line vitamin A status, food type, dose of carotene and reported conversion ratios by weight. The conversion ratio is strongly correlated (Pearson correlation) with dose of β -carotene administered (0.932; 0.002), but not with duration of study (0.085; 0.856), BMI (0.468; 0.290) or age of subjects (0.433; 0.332). Further, base line vitamin A status also did not correlate with conversion ratio (0.234; 0.613), which is rather unexpected. It is important to note that the base line status of all the study subjects was more than adequate (20 $\mu\text{g/dL}$), which might have hampered this relationship. Further, regression of β -carotene dose with conversion ratio (Figure 13.1.1) showed a linear relationship ($r^2=0.86$; slope: 1.46 and intercept: 1.8). These observations clearly indicate that the conversion ratio is strongly dependent on the dietary intake of carotene. A recent study in Indian urban adults using 3-day dietary recall¹⁷ found that the median dietary intake of β -carotene equivalents was 1.55 mg (n=897; IQR 0.73-2.9 mg). Therefore, assuming maximum intake of carotenoids in Indian adult population at ~3 mg/day (conversion= $1.46 \times 3 + 1.8 = 6.18$), a conservative conversion factor of 6:1 was considered for setting vitamin A requirements. The conversion factors for other carotenoids such as β -cryptoxanthine and α -carotene were set at 12:1, for computing the vitamin A equivalents of dietary carotenoids. It is

also worth mentioning here that the conversion factor could be more efficient in subjects with compromised vitamin A status and/or intakes below that were considered in this computation.

Table 13.1.1: Carotene conversion ratios from intrinsically labelled test foods

Study reference	Food type	Baseline s.retinol ($\mu\text{g}/\text{dL}$)	SD	Age (years)	SD	BMI (kg/m^2)	SD	Duration of study (days)	Carotene dose (mg)	Conversion by weight	SD
Guangwen Tang <i>et al.</i> , 2005.	Spinach	50.4	9.24	57.1	6.5	26.1	3.0	35.0	10.95	20.9	9.0
Guangwen Tang <i>et al.</i> , 2005.	Carrot	56.0	11.2	57.1	6.5	26.1	3.0	35.0	10.3	14.8	6.5
Jie Wang <i>et al.</i> , 2008.	Spirulina	47.9	9.24	48.1	5.7	23.4	3.1	48.0	4.2	4.5	1.6
Guangwen Tang <i>et al.</i> , 2012.	Golden rice	25.6	5.8	7.0	1.0	14.4	-	21.0	0.6	2.3	0.8
Guangwen Tang <i>et al.</i> , 2012.	Spinach	24.2	3.5	7.0	1.0	14.4	-	21.0	1.4	7.5	0.8
Guangwen Tang <i>et al.</i> , 2009.	Golden rice	62.7	14.8	59.0	11.0	26.0	2.5	36.0	1.26	3.8	1.7
Tawanda <i>et al.</i> , 2011.	Yellow maize	59.2	17.1	48.4	10	22.4	3.1	36.0	1.2	3.2	1.5

Figure 13.1.1: Relationship of β -carotene dose with efficiency of conversion



Derivation of average daily vitamin A (retinol) requirements in Indian adults and young children

Changes from the ICMR 2010 recommendation for vitamin A requirement

The following changes were made:

- Since EAR is more relevant in population context, an effort was made to systematically derive the EAR and RDA using factorial approach.

- b. The factorial computation of vitamin A requirements was done using a similar methodology, but several assumptions in the previous RDA were revised.
- c. The carotene conversion ratio was revised to account for tissue conversion, based on recent knowledge.

ADULTS

The factorial model for determining daily requirements

The dietary requirement for maintaining normal vitamin A status in healthy well-nourished Indian adults was calculated using the factorial approach described by Olson¹⁸⁻¹⁹. This approach defines the EAR of vitamin A as the daily intake required to balance its daily catabolic loss, which is calculated from an assumed daily loss from the body stores, the absorption and efficiency of storage, as described below^{7,19-20}. This method has been adopted by IOM and EFSA in their recommendations, with different assumptions for each of the factors^{7,20}.

In this factorial method, six factors are considered, with their assumed values as in the EFSA recommendation²⁰.

X = Percent of the vitamin A store lost per day (0.7%)

N = Minimum acceptable liver vitamin A reserve for health (20 µg/g liver tissue)

Y = Liver weight: body weight ratio (0.022 for adults and 0.034 for children 1-5y; derived)

W = Reference weight for specific age groups and genders

P = Ratio of total body vitamin A store to liver vitamin A store (1.25; assuming 80% of the total body store is in the liver)

Z = Efficiency of storage of ingested vitamin A (50%)

The assumption of numeric values and the CV for each of the factors is discussed below.

Daily catabolic loss (X)

The vitamin A lost through urine and faeces was reported to be 128 µg/d. In initial considerations by the IOM, this was based on the excretion of radio labeled vitamin A in subjects on a vitamin A-free diet, and calculated to be 0.5% of the pool/day²¹⁻²². In later tracer experiments, Cifelli and co-workers²³ found much higher catabolic rates in healthy Chinese and US adults with high vitamin A stores, but these rates were positively correlated with the store size. Cifelli and co-workers²⁴ also showed in rats that vitamin A losses are related to the vitamin A status. When the pool size is lower, as considered in the factorial model (see below), where the minimum pool size compatible with health is considered, the catabolic rates will be lower. Therefore, a daily pool loss of 0.7% was conservatively considered by EFSA²⁰, and this has been assumed to be relevant in Indian adults and children, until direct measurements are made here (Table 13.1.2). The CV for this factor was taken as 21%¹⁸⁻¹⁹. It is worth mentioning that the CV of the requirement is taken as 20% by IOM and EFSA⁷⁻²⁰, and probably comes from this single factor value.

Minimum acceptable liver stores (N)

The minimal acceptable liver reserve was based on the vitamin A concentration in liver tissue at which (i) no clinical signs of VAD are observed, (ii) adequate plasma retinol concentrations are maintained (iii) induced biliary excretion of vitamin A is observed, and (iv) protection against VAD is provided for approximately 4 months while on a vitamin A deficient diet. This was shown to be 0.07 µmol/g or 20 µg/g in rat models²⁵⁻²⁶. There have been recent reports that the acceptable liver store should be 0.1 µmol/g based on rat studies²⁷ or that liver fibrosis has been shown to occur at a value of 0.065 µmol/g in a Mongolian gerbil model²⁸. Equally, no liver abnormality (based on enzymes) was observed in Bangladeshi adult subjects who had liver stores ranging from 0.02 to 0.3

$\mu\text{mol/g}$ ⁸ and in Bengaluru, India, a stable isotope based measurement of total body retinol stores on 28 healthy NPNL young adult women with normal liver function, found that 3 (11%) of them had liver stores that were less than $0.07 \mu\text{mol/g}$. There is uncertainty in what the minimum acceptable store should be, but based on the earlier considerations, a liver retinol concentration of $0.07 \mu\text{mol/g}$ or $20 \mu\text{g/g}$ in a healthy person assumed to fulfill all physiological needs and to provide an adequate reserve for at least three months. This value is considered for both adults and children, and taken as a fixed factor.

Table 13.1.2: Average daily requirement of Vitamin A in Adults – using current statistical method

	Indian men	Indian women
Wt (Kg)	65	55
Liver: body weight ratio	0.022	0.022
Liver store concentration ($\mu\text{g/g}$)	20	20
Daily loss (% of total body store)	0.7	0.7
Total body store: Liver store ratio	1.25	1.25
Efficiency of storage of intake in store	50%	50%
EAR ($\mu\text{g RAE / day}$) ¹	464	392
RDA ($\mu\text{g RAE / day}$) ¹	998	844
EAR ($\mu\text{g RAE / day}$) ²	501	424
RDA ($\mu\text{g RAE / day}$) ²	1088	920

1. Multiplicative model assuming reported values of factors as mean (current method described, and recommended)
2. Multiplicative model assuming reported values of factors as median (IOM/EFSA method)

Liver to body weight ratio (Y)

To calculate the absolute value of liver reserve, the liver weight is required, which must be multiplied into the desirable (as above) liver retinol concentration. The IOM and EFSA used a body weight factor to estimate the liver size^{7,20}. However, a literature search revealed that the liver size varies by ethnicity, and is thought to be smaller in Indians/Asians. Therefore, liver/body weight ratio was derived for Indian population through a review of literature. A Pubmed search [("liver volume") OR ("liver weight") OR ("liver morphology") AND (India)] retrieved 26 articles, of which 4 articles reported both body weight and liver volume²⁹⁻³². An additional 5 articles were obtained through searching Google Scholar³³⁻³⁸.

For adults, the data on age, body weight and liver volume/weight were extracted. The liver volume was converted to weight by considering the density of liver³⁹⁻⁴⁰ to be 1.05 g/cm^3 . Some studies^{30,35} did not provide body weights and were not considered further in this ratio calculation. The age, gender, body weight and liver weight data from remaining 7 studies were given in Table 13.1.3. The mean liver to body weight ratio ranged from 0.0209 to 0.0239 with a mean of 0.023. The mean liver to body weight ratio computed from 4 Asian studies⁴¹⁻⁴⁴, from China (2 studies), Korea (1 study) and Singapore (1 study) was 0.021 (Table 13.1.3). Therefore, 0.022, as an average of all values was considered as the liver to body weight ratio to calculate liver weight. The CV was considered to be 9%⁴⁵.

Table 13.1.3: Adult liver/body weight ratios

Indian Studies								
		Sample size	Average Age	Body weight (Kg)	Liver weight (Kg)	Liver/body wt ratio	Study type	Female/Male ratio
1	Chandramohan <i>et al.</i> , 2012 ³⁰	366	49 (18-70)	58.7 (39-89)	1.409.51 (0.79-2.44)	0.0238	Autopsy	91/274
2	Chandramohan <i>et al.</i> , 2007 ²⁹	238	46.5 (10-70)	57.5 (22.7-99.7)	1.378 (805.2-27)	0.0238	CT scan	88/150
3	Gupta <i>et al.</i> , 2008 ³⁶	50	35.05 (19-65)	54.3 ± 9.48 (35-75)	1.140 ± 0.244 (0.800-1.72)	0.0209	Autopsy	-
4	Vijay <i>et al.</i> , 2013 ³⁸	33	45.2 ± 19.45	57.2 ± 12.48	1.334 ± 0.358	0.0232	Autopsy	14/19
5	Anitha <i>et al.</i> , 2019 ³⁴	100	39.15 (18-64)	60.27 ± 10.0	1.34 ± 0.23	0.0222	CT scan	50/50
6	Sharma <i>et al.</i> , 2016 ³⁷	100	48.33 ± 10.87	62.06 ± 8.84	1.45.46 ± 0.36	0.0233	CT scan	38/62
7	Agrawal <i>et al.</i> , 2011 ³³	337	49.34 ± 11.99	63.1 ± 9.77	1.517 ± 0.34	0.239	CT scan	176/161
Average liver/body weight ratio					0.0230			
Asian studies								
8	Yu <i>et al.</i> , 2004 ⁴⁴	652	42.4 ± 16.5	60.6 ± 13.3	1.396.0 ± 0.37	0.023	Autopsy	222/430
9	Chan <i>et al.</i> , 2006 ⁴¹	159	35.8 ± 10.5 (18-57)	56.3 ± 8.4 (41.0-78.5)	1.153.85 ± 0.19	0.020	CT scan	106/53
10	Feng <i>et al.</i> , 2017 ⁴²	244	48.8 ± 12.00 (18-88)	65.41 ± 10.92 (38-96)	1.253 ± 0.25	0.019	CT scan	106/138
11	Lui <i>et al.</i> , 2016 ⁴³	79	34 [22-57]	62 [39-117]	1.337.7 ± 0.264	0.021	CT scan	38/41
Average liver/body weight ratio					0.021			

Reference weight (W)

The reference weight taken for adult Indian man and woman are 65 and 55 kg, respectively (ICMR-RDA-2020, Chapter 3). The reference weight for girls and boys (1-6y) was taken as the age and gender-specific median value of the WHO growth curve weights⁴⁶. The CV of adult body weight was considered to be 14% and similar in children⁴⁷.

Ratio of total body/liver store (P)

It is known that most of the body stores of total vitamin A (upto 80%) are stored in the liver⁴⁸, as assumed by EFSA. The ratio of total body to liver vitamin A store was taken as 80% (a multiplicative factor of 1.25 for the calculated liver store) among individuals with adequate vitamin A status. This value was considered as a fixed factor for both adults and children.

Efficiency of storage of ingested vitamin A (Z)

The efficiency of vitamin A storage depends on the pre-existing vitamin A status, physiological status and dose administered (dietary intake). Using a radio-isotopic method⁴⁹, whole-body retinol retention after 1 mg dose of labelled retinol in Indian children (2–10 years) found to be 82.2 ± 2.0% and 57.6 ± 6.0%, in healthy and infected children, respectively. However, at higher doses of retinol (6 mg), the retention reduced to 47%. In a subsequent study using similar methodology⁵⁰, retention was found to be 48–54% among healthy Indian children (n=17; 3–6 years). Liver stores of vitamin A were not measured in these studies in children where VAD was anticipated, which could improve the retention and storage. A study in Bangladesh men with normal vitamin A stores,

determined the efficiency of liver storage to be 40%⁴⁸. However, this study did not account for the oxidized metabolites of retinol during the experimental period. Keeping these uncertainties in mind, a 50% efficiency of storage is considered in computing the vitamin A requirements of Indian adults and children, and is similar to the value used by EFSA. In the Bangladesh study, the CV of deposition was 30%, and this was considered for both adults and children.

Statistical considerations in the factorial model

The Olson method (1-3) uses a simple multiplicative model, multiplying all these factors into each other. The distribution of the product of all these factors ($X \times N \times Y \times Z \times W \times P$), (where 'X', 'Y', 'Z' and 'W' are variables that are assumed to be fixed in their value) would produce a distribution of the requirement of daily dietary vitamin A to replace daily catabolic loss and maintain the vitamin A reserve in the body. However, the use of a simple product method assumes a Gaussian distribution of desired requirement, but this may not be true even if all factors derive from a Gaussian process.

In general, a wide variation in some of the factors estimates has been reported, with CV greater than 10%. The CV of the body weight (W), daily catabolic vitamin A loss (X), liver weight proportion of body weight (Y), and the efficiency of deposition into the store (Z) were taken from the literature as described for each factor above. The concentration of the liver vitamin A store (N), its relation to the total body vitamin A store (P) and the age-based growth factor in children was assumed to be constant.

The high factor CV makes it prudent to consider an alternate method for calculating the factorial estimate. To do so, the simple multiplicative model is converted into an additive structure by log transformation. It is already well known that the standard body weight is skewed as in the WHO standards⁴⁶, and the finding of a CV of 30% for the efficiency of vitamin A storage is also an indicator of a skewed distribution for that factor. Keeping these features in mind, it is assumed that the distribution of the factors 'X', 'Y', 'Z' and 'W' are symmetric in log scale, and that the average values of 'X', 'Y', 'Z' and 'W' for adults are the mean of a mild to moderate skewed distribution. However, the average of standard weight⁴⁶ for children are the median value with the same assumption.

The multiplicative model in log scale will be:

$$Z_R = \log(X) + \log(Y) + \log(Z) + \log(W) + \log(NP)$$

For simplicity, it is assumed that the probability distribution of 'X', 'Y', 'Z' and 'W' are normal in log scale, hence have Log normal distribution in anti-log scale. Following the features of log normal distribution, the mean and SD of the distribution of each factor in log scale can be derived by the following expression

$$\mu = \log(m) - \frac{1}{2} \log(1 + s^2) \quad \& \quad \sigma^2 = \log(1 + s^2)$$

Where m and s are the mean and CV of the factor reported in the literature. For the weight of the children $\mu = \log(m)$.

Finally the mean and variance of the requirement distribution in log scale is

$$\mu_R = E(Z_R) = \mu_x + \mu_y + \mu_z + \mu_w + \log(NP)$$

and

$$\sigma_R^2 = Var(Z_R) = \sigma_x^2 + \sigma_y^2 + \sigma_z^2 + \sigma_w^2$$

The EAR and RDA can then be derived from the median and 97.5th percentile of the distribution of the dietary requirement of vitamin A, which will be a log normal distribution in the above formula.

The following are the expressions for EAR and RDA

$$EAR = \exp(\mu_R)$$

and

$$RDA = \exp(\mu_R + 1.96 \times \sigma_R)$$

The calculation of the EAR and RDA standardized for the body weight (EAR/Kg body weight or RDA/Kg body weight) is performed by removing the body weight factor from the model described above.

Requirements for adult men and women

The derived/ assumed factors for adult men and women are given in Table 3 using the assumption of mean or median values for each factor. Using the IOM and EFSA method with mean values of factors, the EAR of vitamin A requirement in adult men and women (rounded) are 500 and 425 respectively. Using the statistically correct assumption of median values method described above, the EAR of men and women are (rounded) 460 and 390, respectively. The lower EAR for women is due to their lower body weight compared to men.

Pregnancy and Lactation

Vitamin A requirement during pregnancy has been calculated considering the additional requirement of 14 µg/day for gestational weight gain. For an adult woman with 55 kg weight, the EAR of retinol during pregnancy is 406 µg/day with an RDA of 900 µg/day.

The additional needs during lactation are calculated on the basis of vitamin A secreted in milk. Considering the average milk secretion of 700 ml/day with a retinol content of 46.4 µg/100mL, an additional EAR is 325 µg/day of retinol with an RDA of 720 µg/day.

Young Children

For children, it is known that the liver to body weight ratio is higher when compared to adults. Earlier studies reported that the liver weight increases linearly up to 20 kg of body weight followed by flattening thereafter. Studies in Japanese infants⁵¹ reported a liver to body weight ratio of 0.0358 in <2y children (Table 13.1.4). Further, from a meta-analysis of developmental changes in liver⁵², the mean liver to body weight ratio ranged from 0.0358 to 0.028 among 0-6 year children, with average of 0.034. This value was considered for computation of total vitamin A liver stores and its average requirement in Indian children aged 1-6y, with a similar CV as in adults.

Growth factors in children

An additional requirement is needed in children to account for the usage of retinol in growth. For this, a growth factor, based on the deposition of lean tissue in growing children at 1-3y and 4-6y was calculated and multiplied into the factorial estimate, based on tissue accretion rates at those ages. These were taken as a proportion of the protein requirement for growth to the maintenance requirement⁵³ at each age group. This was altered for the 3-6y age group, since in the WHO/FAO/ UNU report⁵³, protein deposition was calculated across ages from 2 datasets, one longitudinal for 0.5-2y, and the other cross-sectional for 4-18y. These estimates, made by K counting for protein deposition, meant that there were no data for the intervening ages, and also, there was an offset

between 2y (from the first dataset) and 4y (from the second). A quadratic equation was used to bridge this offset smoothly. However, using this equation meant that at 3-5y, the estimated protein deposition dropped to near zero, which might seem biologically implausible. Thus, in the EFSA and IOM (2, 3) estimates of the EAR, a low growth factor results, and is used at the 3-6y age group. In the present calculation, it was felt that an interpolated value for growth deposition should be used for the 3-6y age group (interpolated from the values at 1-3y and 6-9y). These were assumed to be fixed factors.

Table 13.1.4 Children liver/body weight ratios

		Sample size	Age	Body weight (Kg)	Liver weight (Kg)	Liver/body weight ratio	Study type
1	Urata <i>et al.</i> , 1995 ⁵¹	26	<2 years	8.0 ± 1.4	0.287± 0.556	0.0358	Autopsy
2	Johnson <i>et al.</i> , 2005 ⁵²	576	Neonates	3.4 – 3.75	0.075-0.168	0.034	Meta-analysis
		1518	Infants (<1 year)	4.1 – 10.1	0.129-0.39	0.037	
		1535	Young Children (<6 years)	9.65 – 21.9	0.231-0.639	0.028	
Average liver/body weight ratio						0.0337 (rounded to 0.034)	

Table 13.1.5: Average daily requirement of Vitamin A in boys using reported values of factors

Age group	0.5-1	1-3	3-6	6-9	9-12	12-15	15-18
Wt (Kg)	9.1	12.3	17.4	24.2	33.0	47.7	63.4
Liver: body weight ratio	0.034	0.034	0.034	0.031	0.028	0.024	0.022
Liver store concentration (ug/g)	20	20	20	20	20	20	20
Daily loss (% TBS*)	0.7	0.7	0.7	0.7	0.7	0.7	0.7
TBS:TLR** ratio	1.25	1.25	1.25	1.25	1.25	1.25	1.25
Efficiency of storage of intake	50%	50%	50%	50%	50%	50%	50%
Growth Factor	1.57	1.25	1.19	1.13	1.13	1.09	1.06
EAR (μg RAE / day)	167	183	242	294	358	433	481
RDA (μg RAE / day)	352	387	518	632	769	932	1036
EAR (μg RAE / kg / day)	18	14	13	12	10	9	8
RDA (μg RAE / kg / day)	36	29	27	24	21	18	16

*: Total body store

**: Total body store: Total Liver reserve

Table 13.1.6: Average daily requirement of Vitamin A in girls using reported values of factors

Age group	0.5-1	1-3	3-6	6-9	9-12	12-15	15-18
Wt (Kg)	8.4	11.6	17.2	23.8	34.2	48.3	55.4
Liver: body weight ratio	0.034	0.034	0.034	0.031	0.028	0.024	0.022
Liver store concentration (ug/g)	20	20	20	20	20	20	20
Daily loss (% TBS*)	0.7	0.7	0.7	0.7	0.7	0.7	0.7
TBS:TLR** ratio	1.25	1.25	1.25	1.25	1.25	1.25	1.25
Efficiency of storage of intake	50%	50%	50%	50%	50%	50%	50%
Growth Factor	1.57	1.25	1.19	1.13	1.11	1.06	1.02
EAR (μg RAE / day)	154	175	237	290	365	415	401
RDA (μg RAE / day)	329	372	511	625	785	894	863
EAR (μg RAE / kg / day)	18	14	13	12	10	9	7
RDA (μg RAE / kg/day)	36	29	27	24	21	17	15

*: Total body store

**: Total body store: Total Liver reserve

Table 13.1.7: Summary of EAR and RDA by age group and per kg body weight (Rounded off values)

Age Group	EAR (μg RAE/day)	RDA (μg RAE/day)	EAR (μg RAE/kg/day)	RDA (μg RAE/kg/day)
Adult Men	460	1000	7	15
Adult Women	390	840	7	15
Boys				
0.5-1y	170	350	18	36
1-3y	180	390	14	29
3-6y	240	510	13	27
6-9y	290	630	12	24
9-12y	360	770	10	21
12-15y	430	930	9	18
15-18y	480	1000	8	16
Girls				
0.5-1y	160	330	18	36
1-3y	180	370	14	29
3-6y	240	500	13	27
6-9y	290	630	12	24
9-12y	370	790	10	21
12-15y	420	890	9	17
15-18y	400	860	7	15

Requirements in young children (1-6 years)

The derived/ assumed factors for children are similar to that in adult men and women, except that a liver to body weight ratio of 0.034 was used for children from 0.5 to 6y of age. For the 15-18y age group, the liver ratio of body weight was assumed to be the same as in adults. For the in-between age groups, a linear interpolated value was used. The median value method was used to calculate EAR and RDA for each age group, as shown in Tables 13.1.5 & 13.1.6.

The rounded off EAR and RDA values, also adjusted for weight, for adults and children are given in Table 13.1.7. Due to the higher liver weight ratio, and the additional requirement to support growth deposition, the EAR and RDA/kg are higher in children. The fact is that the per kg body requirement of vitamin A is low with increase of age of the child. Summary and comparison with the previous RDA is presented in Table 13.1.8.

Table 13.1.8: Vitamin A daily requirements for various physiological groups and its comparison with ICMR RDA 2010 and IOM 2001

Physiological group	Body weight (kg)	RDA 2020		RDA 2010 (μg /day)	TUL (μg /day)	H-AR	IOM RDA
		EAR (μg /day)	RDA (μg /day)				
Adult-Male	65	460	1000	600	3000	570	900
Adult-Female	55	390	840	600	3000	490	700
Pregnant	55.0+GWG	406	900	800	3000	540	770
Lactating ^π	55.0	720	950	950	3000	1020	1300
Infants (0-6 mo)	5.8	-	350* (AI)	350	600 ^{\$}	-	-
Infants (6-12 mo)	8.5	170 (AI)	350 (AI)	350	600 ^{\$}	190	-
Children (1-3y)	11.7	180	390	400	600 ^{\$}	205	300
Children (4-6y)	18.3	240	510 (300)	400	900 ^{\$}	245	400
Children (7-9y)	25.3	290	630 (350)	600	900 ^{\$}	320	400
Boys (10-12y)	34.9	360	770	600	1700	480	600
Girls (10-12y)	36.4	370	790	600	1700	480	600
Boys (13-15y)	50.5	430	930	600	2800	480	900
Girls (13-15y)	49.6	420	890	600	2800	480	700
Boys (16-18y)	64.4	480	1000	600	2800	580	900
Girls (16-18y)	55.7	400	860	600	2800	490	700

RAE: Retinol Equivalents (μg/d); GWG: Gestational weight gain

* Adequate intake; # Extrapolated from infant and adult values; ^{\$}Adopted from IOM;

H-AR: Harmonized average requirement⁵⁵

^π Additional EAR required for lactating women: 325 (μg /day)

References

1. Laxmaiah A, Nair MK, Arlappa N, Raghu P, Balakrishna N, Rao KM, Galreddy C, Kumar S, Ravindranath M, Rao VV, Brahmam GN. Prevalence of ocular signs and subclinical vitamin A deficiency and its determinants among rural pre-school children in India. *Public Health Nutrition*. 2012 Apr;15(4):568-77.
2. Vijayaraghavan K. National control programme against nutritional blindness due to vitamin A deficiency: Current status & future strategy. *The Indian journal of medical research*. 2018 Nov;148(5):496.
3. Ministry of Health and Family Welfare (MoHFW), GoI., UNICEF and Population Council. Comprehensive National Nutrition Survey (CNNS) National Report. New Delhi. 2019.
4. Eroglu A, Harrison EH. Carotenoid metabolism in mammals, including man: Formation, occurrence, and function of apocarotenoids thematic review series: Fat-soluble vitamins: Vitamin A. *Journal of lipid research*. 2013 Jul 1;54(7):1719-30.
5. Harrison EH. Mechanisms involved in the intestinal absorption of dietary vitamin A and provitamin A carotenoids. *Biochimica et Biophysica Acta (BBA)-Molecular and Cell Biology of Lipids*. 2012 Jan 1;1821(1):70-7.
6. Raghu P, Sivakumar B. Interactions amongst plasma retinol-binding protein, transthyretin and their ligands: implications in vitamin A homeostasis and transthyretin amyloidosis. *Biochimica et Biophysica Acta (BBA)-Proteins and Proteomics*. 2004 Dec 1;1703(1):1-9.
7. Russell RM, Beard JL, Cousins RJ, Dunn JT, Ferland G, Hambidge KM, Lynch S, Penland JG, Ross AC, Stoecker BJ, Suttie JW. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. A Report of the Panel on Micronutrients, Subcommittees on Upper Reference Levels of Nutrients and of Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes Food and Nutrition Board Institute of Medicine. 2001.
8. Haskell MJ. The challenge to reach nutritional adequacy for vitamin A: β-carotene bioavailability and conversion—evidence in humans. *The American journal of clinical nutrition*. 2012 Nov 1;96(5):1193S-203S.
9. Tang G. Vitamin A value of plant food provitamin A—evaluated by the stable isotope technologies. *Int J Vitam Nutr Res*. 2014 Jan 1;84(Suppl 1):25-9.
10. CA VL, West CE, Van Breemen RB, Zhu D, Siebelink E, Versloot P, Hulshof PJ, van Lieshout M, Russel FG, Schaafsma G, Naber TH. Vitamin A equivalency of beta-carotene in healthy adults: limitation of the extrinsic dual-isotope dilution technique to measure matrix effect. *The British Journal of Nutrition*. 2008 Nov 24;101(12):1837-45.
11. Muzhingi T, Yeum KJ, Bermudez O, Tang G, Siwela AH. Peanut butter increases the bioavailability and bioconversion of kale beta-carotene to vitamin A. *Asia Pacific Journal of Clinical Nutrition*. 2017;26(6):1039.
12. Tang G, Qin J, Dolnikowski GG, Russell RM. Short-term (intestinal) and long-term (postintestinal) conversion of β-carotene to retinol in adults as assessed by a stable-isotope reference method. *The American journal of clinical nutrition*. 2003 Aug 1;78(2):259-66.
13. Tang, G, Qin J, Dolnikowski GG, Russell RM, Grusak MA. Spinach or carrots can supply significant amounts of vitamin A as assessed by feeding with intrinsically deuterated vegetables. *American Journal of Clinical Nutrition*. 2005; 82(4), 821-828. doi:10.1093/ajcn/82.4.821.
14. Tang G, Hu Y, Yin SA, Wang Y, Dallal GE, Grusak MA, Russell RM. Retracted: β-Carotene in Golden Rice is as good as β-carotene in oil at providing vitamin A to children. *The American journal of clinical nutrition*. 2012 Sep 1;96(3):658-64. doi:10.3945/ajcn.111.030775.

15. Wang J, Wang Y, Wang Z, Li L, Qin J, Lai W, Fu Y, Suter PM, Russell RM, Grusak MA, Tang G. Vitamin A equivalence of spirulina β-carotene in Chinese adults as assessed by using a stable-isotope reference method. *The American journal of clinical nutrition*. 2008 Jun 1;87(6):1730-7. doi:10.1093/ajcn/87.6.1730.
16. Muzhingi T, Gadaga TH, Siwela AH, Grusak MA, Russell RM, Tang G. Yellow maize with high β-carotene is an effective source of vitamin A in healthy Zimbabwean men. *The American journal of clinical nutrition*. 2011 Aug 1;94(2):510-9. doi:10.3945/ajcn.110.006486.
17. Shalini T, Sivaprasad M, Balakrishna N, Madhavi G, Radhika MS, Kumar BN, Pullakhandam R, Reddy GB. Micronutrient intakes and status assessed by probability approach among the urban adult population of Hyderabad city in South India. *European journal of nutrition*. 2019 Dec 1;58(8):3147-59. doi:10.1007/s00394-018-1859-y.
18. Olson JA. Recommended dietary intakes (RDI) of vitamin A in humans. *The American journal of clinical nutrition*. 1987 Apr 1;45(4):704-16.
19. Olson JA. (1987). Recommended dietary intakes (RDI) of vitamin A in humans. *Am J Clin Nutr*, 45(4), 704-716. doi:10.1093/ajcn/45.4.704
20. EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA). Scientific opinion on dietary reference values for vitamin A. *EFSA Journal*. 2015 Mar;13(3):4028.
21. Olson JA, Gunning D, Tilton R. The distribution of vitamin A in human liver. *The American journal of clinical nutrition*. 1979 Dec 1;32(12):2500-7. doi:10.1093/ajcn/32.12.2500.
22. Sauberlich HE, Hodges RE, Wallace DL, Kolder H, Canham JE, Hood J, Raica Jr N, Lowry LK. Vitamin A metabolism and requirements in the human studied with the use of labeled retinol. In *Vitamins & Hormones* 1975 Jan 1 (Vol. 32, pp. 251-275). Academic Press. doi:10.1016/s0083-6729(08)60015-1.
23. Cifelli CJ, Green JB, Wang Z, Yin S, Russell RM, Tang G, Green MH. Kinetic analysis shows that vitamin A disposal rate in humans is positively correlated with vitamin A stores. *The Journal of nutrition*. 2008 May 1;138(5):971-7. doi:10.1093/jn/138.5.971.
24. Cifelli CJ, Green JB, Green MH. Dietary retinoic acid alters vitamin A kinetics in both the whole body and in specific organs of rats with low vitamin A status. *The Journal of nutrition*. 2005 Apr 1;135(4):746-52. doi:10.1093/jn/135.4.746.
25. Hicks VA, Gunning DB, Olson JA. Metabolism, plasma transport and biliary excretion of radioactive vitamin A and its metabolites as a function of liver reserves of vitamin A in the rat. *The Journal of nutrition*. 1984 Jul 1;114(7):1327-33. doi:10.1093/jn/114.7.1327.
26. Loerch JD, Underwood BA, Lewis KC. Response of plasma levels of vitamin A to a dose of vitamin A as an indicator of hepatic vitamin A reserves in rats. *The Journal of nutrition*. 1979 May 1;109(5):778-86. doi:10.1093/jn/109.5.778.
27. Tanumihardjo SA, Russell RM, Stephensen CB, Gannon BM, Craft NE, Haskell MJ, Lietz G, Schulze K, Raiten DJ. Biomarkers of Nutrition for Development (BOND)-vitamin A review. *The Journal of Nutrition*. 2016;146:1816S–48S.
28. Tanumihardjo S, Gannon B, Kaliwile C, Chileshe J. Hypercarotenodermia in Zambia: which children turned orange during mango season? *European Journal of Clinical Nutrition*. 2015; 69, 1346–1349.
29. Chandramohan A, Eapen A, Govil S, Govil S, Jeyaseelan V. Determining standard liver volume: assessment of existing formulae in Indian population. *Indian Journal of Gastroenterology*. 2007 Jan 3;26(1):22.
30. Chandramohan A, Ramakrishna B, Venkatramani S. Formula for calculating standard liver volume in Indians. *Indian Journal of Gastroenterology*. 2012 Jan 1;31(1):15-9. doi:10.1007/s12664-011-0152-2.
31. Chaubal G, Borkar VV, Shetty G, Chattpadhyay S, Bahure U, Badhe R, Udare A, Shah S, Gupte P, Shukla A, Rela M. Estimation of liver volume in the western Indian population. *Indian Journal of Gastroenterology*. 2016 Jul 1;35(4):274-9. doi:10.1007/s12664-016-0662-z.

32. Singh NK, Prasad RC, Chaube HD, Shukla RC, Agarwal AK. The value of sonography for estimation of liver volume by using a simple geometric formula ascertained in cadaveric livers. *The Journal of the Association of Physicians of India*. 1998 Jun 1;46(6):521-4.
33. Agrawal D, Lalwani R, Asghar A, Sahai A, Sharma PK, Singh R. Assessment of liver volume with spiral computerized tomography scanning in North Indian Adults. *Internet J Radiol*. 2011;13(1).
34. JASPeR A. Liver and Splenic Volumes in the Indian Population: Is There a Single CT Measurement Correlate?. *Journal of Clinical & Diagnostic Research*. 2019 Aug 1;13(8).
35. Deepika K, Sushma M, Kumar V. Study of the weights of human heart and liver in relation with age, gender and body height. *IJRMS*. 2017;5:3469-73.
36. G Gupta M, Sodhi L, Yadav TD. Morphology of liver. *Indian Journal of Surgery*. 2008 Feb 1;70(1):3-7. doi:10.1007/s12262-008-0001-4.
37. Sharma M, Singh A, Goel S, Satani S, Mudgil K. Assessment of liver volume with spiral computerized tomography scanning: predicting liver volume by age and height. *Int J Res Med Sci*. 2016 Jul;4:3020-3.
38. Vijay K, Naidu CS, Godara R, Rao P, Sharma S, Vijayvergia V. Standard liver volume estimation in Indian population: Need for an accurate formula. *Indian Journal of Transplantation*. 2013 Apr 1;7(2):39-41.
39. <https://bionumbers.hms.harvard.edu/files/Density%20and%20mass%20of%20each%20organ-tissue.pdf>.
40. Overmoyer BA, McLaren CE, Brittenham GM. Uniformity of liver density and nonheme (storage) iron distribution. *Archives of pathology & laboratory medicine*. 1987 Jun;111(6):549.
41. Chan SC, Liu CL, Lo CM, Lam BK, Lee EW, Wong Y, Fan ST. Estimating liver weight of adults by body weight and gender. *World journal of gastroenterology: WJG*. 2006 Apr 14;12(14):2217. doi:10.3748/wjg.v12.i4.2217
42. Feng LM, Wang PQ, Yu H, Chen RT, Wang J, Sheng X, Yuan ZL, Shi PM, Xie WF, Zeng X. New formula for predicting standard liver volume in Chinese adults. *World journal of gastroenterology*. 2017 Jul 21;23(27):4968. doi:10.3748/wjg.v23.i27.4968.
43. Lui SA, Bonney GK, Kow WC, Iyer SG, Chang SK. Standard Formulae in Predicting Liver Volumes: A South East Asian Series of Adult Living Donors. *J. Transplant. Technol. Res*. 2016;6(1):1-4.
44. Yu HC, You H, Lee H, Jin ZW, Moon JI, Cho BH. Estimation of standard liver volume for liver transplantation in the Korean population. *Liver transplantation*. 2004 Jun;10(6):779-83. doi:10.1002/lt.20188.
45. Andersen V, Sonne J, Sletting S, Prip A. The volume of the liver in patients correlates to body weight and alcohol consumption. *Alcohol and Alcoholism*. 2000 Sep 1;35(5):531-2.
46. World Health Organization. WHO child growth standards: length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: methods and development. World Health Organization; 2006.
47. Adak DK, Gautam RK, Bharati S, Gharami AK, Pal M, Bharati P. Body mass index and chronic energy deficiency of adult males of Central Indian populations. *Human biology*. 2006 Apr;78(2):161-78. doi:10.1353/hub.2006.0032.
48. Furr HC, Amedee-Manesme O, Clifford AJ, Bergen 3rd HR, Jones AD, Anderson DP, Olson JA. Vitamin A concentrations in liver determined by isotope dilution assay with tetradecuterated vitamin A and by biopsy in generally healthy adult humans. *The American journal of clinical nutrition*. 1989 Apr 1;49(4):713-6. doi:10.1093/ajcn/49.4.713.
49. Reddy V, Sivakumar B. Studies on vitamin A absorption in children. *Indian pediatrics*. 1972 Jun;9(6):307-10.

50. Kusin JA, Reddy V, Sivakumar B. Vitamin E supplements and the absorption of a massive dose of vitamin A. *The American journal of clinical nutrition*. 1974 Aug 1;27(8):774-6. doi:10.1093/ajcn/27.8.774.
51. Urata K, Kawasaki S, Matsunami H, Hashikura Y, Ikegami T, Ishizone S, Momose Y, Komiyama A, Makuuchi M. Calculation of child and adult standard liver volume for liver transplantation. *Hepatology*. 1995 May;21(5):1317-21.
52. Johnson TN, Tucker GT, Tanner MS, Rostami-Hodjegan A. Changes in liver volume from birth to adulthood: a meta-analysis. *Liver transplantation*. 2005 Dec;11(12):1481-93.
53. Joint WHO/FAO/UNU. Re: Expert Consultation on protein and amino acid requirements in human nutrition, 2002. World Health Organization. Technical series No- 935. Geneva: WHO; 2007.
54. Tang G, Qin J, Dolnikowski GG, Russell RM, Grusak MA. Golden Rice is an effective source of vitamin A. *The American journal of clinical nutrition*. 2009 Jun 1;89(6):1776-83. doi:10.3945/ajcn.2008.27119.
55. Allen LH, Carriquiry AL, Murphy SP. Perspective: proposed harmonized nutrient reference values for populations. *Advances in Nutrition*. 2020 May 1;11(3):469-83.

13.2 VITAMIN D

Vitamin D₃ is synthesized in the skin from 7-dehydrocholesterol following exposure to ultraviolet B (UVB) radiation with wavelength 290 to 320 nm. The level of synthesis in skin is influenced by a number of factors including the season of the year, level of air pollution, skin pigmentation, latitude, use of sunscreen, type of clothing, and amount of skin exposed. Regions northern to 33° latitude have scanty UV-B with a possibility of limited photolytic vitamin D synthesis in individuals living in these regions¹. Moreover, the synthesis of vitamin D declines with increasing age partly due to fall in 7-dehydrocholesterol levels and alterations in skin morphology. Body fat composition may also play a role in low levels of circulating vitamin D due to its sequestration in the fat tissue. It has been suggested earlier that the de novo synthesis in the skin following sun exposure is the major source of vitamin D and its dietary requirement is very small in the Indian context. However, with the existing widespread vitamin D deficiency in the country which is partly related to the limited effective sun exposure due to indoor sedentary behaviour, clothing, air pollution, darker complexion, the role of dietary vitamin D needs to be re-examined.

This is particularly important with increasing recognition of many non-calcitropic health promoting effects important for chronic diseases such as diabetes, psoriasis, hypertension, arthritis, few neuropsychiatric illnesses, malignancies and CVD, apart from its well known classical calcitropic etiological role in the development of rickets (in children) and osteomalacia (in adults)².

Diet

Dietary vitamin D can be obtained in two forms: 1. vitamin D₂ (ergocalciferol) from sources such as plants, mushrooms and yeast and 2. vitamin D₃ (cholecalciferol) from animal foods such as fish and eggs. Recently published Indian Food composition data based on LC-MS methodology show that a number of foods, including cereals and pulses, contain vitamin D₂. Vitamin D₂ and vitamin D₃ are respectively metabolized to 25(OH)D₂ and 25(OH)D₃ and further to 1,25(OH)D₂ and 1,25(OH)D₃. Supplementation with vitamin D₂ was found to increase the level of 24,25 (OH)D₃, indicating activation of vitamin D₃ catabolic pathway via CYP24A1. In the absence of studies measuring the vitamin D₂ metabolites along with PTH and calcium homeostasis markers in Indian settings with likely substantial intake of vitamin D₂, it is difficult to ascertain the contribution of dietary sources of vitamin D to the overall status of vitamin D.

Deficiency

Vitamin D deficiency (VDD) leads to abnormal calcium homeostasis resulting in defective mineralization of the growing long bones (rickets in children) or a decrease in the mineral content of the matrix of the bones (osteomalacia in adults) ending with weakened bones. A poor calcium deposition during growth phase and faulty economy in adults lead to osteoporosis wherein vitamin D and calcium supplementation is shown to be beneficial. While the rabid forms of bone disease are no longer prevalent in children now, growing urbanization, reduced physical activity and low exposure to sunlight are believed to contribute to a spurt of VDD as evidenced from low circulating vitamin D levels. Emergence of sub-clinical vitamin D deficiency in the country has been reviewed by Goswami *et al*³.

Role

Vitamin D is activated in two series of hydroxylation reactions: first in liver to produce 25 hydroxy cholecalciferol (25 HCC) and the second in kidney to produce 1, 25 dihydroxy cholecalciferol (1, 25 DHCC), an active hormone which mediates intestinal absorption and renal tubular resorption of calcium for increasing the availability of calcium. Accumulated evidence over the past two decades has demonstrated that the active form i.e. 1, 25 DHCC is synthesized in a number of other tissues in the body indicating extra-renal synthesis. The extra-renal formation of

the active hormonal form is known to aid in paracrine and autocrine functions⁴. The level of 25 HCC is usually considered as an indicator of vitamin D status. Serum calcium level is maintained in a narrow range by the concerted actions of parathyroid hormone (PTH), thyrocalcitonin and vitamin D. A drop in calcium intake triggers an elevation in PTH levels which enhances the release of 1, 25 DHCC so as to increase calcium absorption and maintain serum calcium level. Vitamin D deficiency therefore results in alterations in the calcitropic hormones and not always in serum calcium. Hypocalcemia induced secondary hyperparathyroidism leads to bone resorption (brittle bones), in an attempt to maintain calcium homeostasis. Vitamin D induced calcium absorption from kidneys and intestine, prevents bone resorption due to secondary hyperparathyroidism.

Requirement

Vitamin D is considered more as a pro-hormone, than as a vitamin. It can be synthesized in the body by exposure to sunlight. The duration of sun exposure for adequate synthesis of vitamin D varies with the latitude, season of the year, time of exposure during the day and amount of skin exposed. The UV-B rays from sunlight responsible for vitamin D synthesis are maximal between 11 AM – 3 PM.

The requirements of vitamin D have been determined over the years based on the healing response in rickets, increased calcium absorption or circulating levels of 25 HCC and also turnover of isotopic vitamin D. The WHO Expert Committee recommended 100 International Units (IU) (2.5 µg) /d for adult males in 1988⁵ which was increased later in 2004 to 200 Units (5 µg)/d⁶. The Institute of Medicine (US) guidelines in 2011 recommend 600 IU for adult males assuming minimal sun exposure⁷. The increased requirement is attributed to progressive decrease in sunlight exposure necessitating dietary sources to meet the requirement. Hence, many foods such as milk and vegetable oils are subjected to mandatory fortification with vitamin D in many developed countries.

Estimating the vitamin D requirements in Indians is especially complicated due to following reasons:

1. Uncertainty about the contribution of sun exposure to overall vitamin D nutrition in Indians poses difficulty for estimating the dietary requirement of vitamin D.
2. Due to close interaction between calcium and vitamin D, the requirement of vitamin D varies with the dietary calcium intakes, especially for the bone health outcomes.

Moreover, data needed to estimate dietary requirements of vitamin D with associated SD/ CV values measured in Indian participants are extremely limited (Annexure 13.2).

RDA comments

Recommendations from high income countries and the international agencies for calcium and vitamin D are based on the results of large-scale clinical trials and are intended to ensure sufficient levels of nutrients for prevention of chronic diseases. However, populations from many high income countries live in conditions where exposure to sunlight is limited and therefore, need to obtain substantial amounts of vitamin D from the diets through fortification and if necessary, by supplementation. Despite opportunities for sunlight exposure throughout the year, high prevalence of vitamin D deficiency in different population groups including school children⁸ and adults⁹ has been demonstrated by studies from India. A preliminary report of Rege *et al*¹⁰ demonstrated gradient in mean 25 HCC levels (ng/ml) in men from rural (25.5), urban slums (16.9) and urban middle class (12.20) background in consonance with their expected outdoor activity. ICMR task force study at National Institute of Nutrition, Hyderabad (unpublished) showed that the prevalence of vitamin D deficiency was much higher in high and middle income group men and women engaged in mostly sedentary occupations (66.9% and 45.5%, respectively), compared to that in

urban slum dwellers who were mostly involved in manual labour (4.1%). Few studies from India have, however, reported inadequate vitamin D synthesis despite sunlight exposure^{11, 12}. A study by Marwaha *et al* (2015) included 71 school children (aged 10 – 15 years) from Delhi, who were categorised into 2 groups, one with 15% and the other with 30% of body surface area (BSA) exposed to sunlight for 30 minutes (11.15 AM– 11.45 AM). After follow up for 4 weeks, during the months of April and May, children with 30% of BSA exposure had increase of 4.9 ± 6.2 ng/ml in serum 25(OH)D while the group with 15% of BSA exposure exhibited relatively lower rise in serum 25(OH)D (3.4 ± 3.2 ng/ml)¹¹. Another study from geographically similar location (Delhi) cross sectionally estimated vitamin D levels in 88 outdoor labourers, 32 workers with partial outdoor activity and 74 individuals with predominantly indoor activity.

The study found that about 60% of the participants with outdoor activity and 97% of the individuals in the other 2 groups had vitamin D insufficiency (serum 25(OH) D <30 ng/dl)¹². A recent study from Pune which examined sun exposure using UV dosimeters, reported that 1 SED-UV exposure, which corresponded to more than one hour of midday casual sunlight exposure, was sufficient to maintain 25(OH)D concentrations over 20 ng/ml¹³. In a RCT from Pune in men aged 40-60 y with vitamin D deficiency at baseline, increased sun exposure for more than an hour daily, over a 6-month period significantly increased 25(OH)D levels. Interestingly, although the increase in vitamin D levels was lower than that observed with cholecalciferol supplementation, increased sun exposure also resulted in a significant reduction in serum total cholesterol¹⁴. These findings corroborate the results of a meta-analysis based on ten randomized clinical trials on the influence of Vitamin D supplementation on plasma lipid profiles, which concluded that Vitamin D supplementation could increase LDL-Cholesterol concentrations¹⁵. However, the findings of this study need to be confirmed with further studies especially the RCTs in different parts of the country to estimate the relative contribution of sun exposure to vitamin D status and additional dietary requirement of Vitamin D.

Recent study from NIN (unpublished) using satellite data on UV index (after accounting for cloud cover) from ten centres across the country, showed that around 20 minutes of direct sun exposure at solar noon in summer would be enough for adequate cutaneous synthesis of vitamin D assuming 10 % body surface area exposure in individuals with skin type V in almost all regions of the country except Srinagar. Data on the North East region is not available. The equivalent duration of sun exposure in other seasons is higher with maximum of around 45 minutes in monsoon season.

To summarize, the evidence regarding the adequacy of sun exposure for ensuring vitamin D sufficiency is mixed but suggests that ensuring adequate sun exposure during 11 am to 3 pm in all seasons is an effective and desirable way of improving vitamin D status. However, the duration of required sun exposure varies in relation to urban-rural residence, occupation, socioeconomic status, skin tone, pollution etc., and it is unclear what proportion of population would be able to achieve the sufficient sun exposure.

Under situations of minimal exposure to sunlight, the following recommendation of a daily dietary intake may be considered (Table 13.2.1). It is important to note that these values are valid if the dietary calcium intakes are adequate.

**Table 13.2.1: EAR and RDA of vitamin D in case of minimal sun exposure
(only with adequate dietary calcium intake)**

	Body weight kg	EAR Unit/day	RDA Unit/day
Adult Men	65	400 IU (10 µg)	600 IU (15 µg)
Adult Women	55	400 IU (10 µg)	600 IU (15 µg)
Pregnant	-	400 IU (10 µg)	600 IU (15 µg)
Lactating	-	400 IU (10 µg)	600 IU (15 µg)
Infants (0-6mo)	5.8	-	400 IU (10 µg) AI
Infants (6-12mo)	8.5	-	400 IU (10 µg) AI
Children (1-3y)	11.7	400 IU (10 µg)	600 IU (15 µg)
Children (4-6y)	18.3	400 IU (10 µg)	600 IU (15 µg)
Children (7-9y)	25.3	400 IU (10 µg)	600 IU (15 µg)
Boys (10-12y)	34.9	400 IU (10 µg)	600 IU (15 µg)
Girls (10-12y)	36.4	400 IU (10 µg)	600 IU (15 µg)
Boys (13-15y)	50.5	400 IU (10 µg)	600 IU (15 µg)
Girls (13-15y)	49.6	400 IU (10 µg)	600 IU (15 µg)
Boys (16-18y)	64.4	400 IU (10 µg)	600 IU (15 µg)
Girls (16-18y)	55.7	400 IU (10 µg)	600 IU (15 µg)

Reference from Institute of medicine⁷

Annexure 1

The following studies could be identified by systematic search of studies on vitamin D intake in Indians using PubMed search engine:

George et al 2014 investigated the median vitamin D consumption among apparently healthy Asian - Indians living in Johannesburg and reported a median dietary intake of vitamin D as 1·17 (IQR 0·50–2·11) µg/day with a prevalence of vitamin D deficiency (<30 ng / dl in serum) in 28.6% of the Asian-Indian population. The study also describes the sun exposure patterns and seasonal variations¹⁶.

Tergistina et al 2014 identified an increased incidence of vitamin D deficiency among preterm infants who otherwise were normal at the time of birth following 400 IU/D vitamin D supplementation over 6 weeks¹⁷.

Khadgawat et al 2013 study in 10 - 14 year apparently healthy school pupils reported final vitamin D levels of 22.87 ± 6.75 ng/dL as compared to baseline levels of 11.42 ± 5.24 ng/dL, following 12 weeks of 600 IU / D of vitamin D supplementation via fortified milk¹⁸.

Kumar G T et al 2011 observed serum mean vitamin D levels of 55 ng/dl post 6 months of intervention with 1400 IU (35 µg) of vitamin D / week, since birth¹⁹.

Puri et al 2008 observed serum 25(OH)D levels of 31·87 (15·43) nmol/l in 8 - 14 year school girls with an average daily dietary consumption of 2·2 (1·5) µg of vitamin D²⁰.

13.3 VITAMIN E (ALPHA TOCOPHEROL) AND VITAMIN K

They occur widely in vegetable oils and plant foods and dietary deficiencies of these two vitamins are not normally encountered. There are very limited data on vitamin E (α -tocopherol) and vitamin K intake as well as requirements. No relevant studies in apparently healthy individuals could be identified by systematic search of studies on vitamin E and K intake in Indians using PubMed search engine. Available information indicates that blood levels of α -tocopherol among Indians is satisfactory^{21, 22}. α -tocopherol content of vegetable oils and invisible fat in cereals and other foods is adequate.

α -tocopherol requirement is related to its protective antioxidant property on essential fatty acid content of the diet and the suggested intake is 0.8 mg per g of essential fatty acid (EFA). Vegetable oils and invisible fat of cereals and other foods like nuts and vegetables contribute to adequate tocopherol content in Indian diets. There is limited information suggesting that Indians have blood α -tocopherol levels of 0.5 mg/kg/ml which is regarded as satisfactory. α -tocopherol requirements are related directly to the levels of essential fatty acids α -linoleic and linolenic acids. The suggested requirement of α -tocopherol is 0.8 mg/ g of dietary essential fatty acids. This roughly works out to 8-10 mg tocopherol /d, depending on the edible oil used.

Vitamin K deficiency is not usually encountered in India. Apart from its role in blood clotting mechanism, vitamin K is implicated in chemical modification of bone matrix and its turnover. FAO/WHO²³ suggested an RDA of 7.5-10 mg α -tocopherol and 55 μ g of vitamin K/d for adults. In the Indian context, limited database is available on the content of these nutrients in foods. Till more information is available on nutrient requirements in Indians and their habitual dietary intake, the values suggested by the earlier ICMR Committee and WHO are accepted. The safe intakes of different groups may be considered on the basis of uniform energy density / density of PUFA energy.

References

1. Wacker M and Holick MF. Sunlight and Vitamin D: A global perspective for health. *Dermato-Endocrinology* 2013; 5(1), 51–108.
2. McLaren DS. VDDD: A global threat to health; *Sight and Life Magazine* 2006; (3): 6-15.
3. Goswami R, Mishra SK and Kochupillai N. Prevalence and potential significance of vitamin D deficiency in Asian Indians. *Indian Journal of Medical Research*. 2008; 127: 229-38.
4. Bouillon R. Extra-Skeletal Effects of Vitamin D. *Frontiers in Hormone Research*. 2018; 50:72-88. doi: 10.1159/000486072.
5. World Health Organization. Calcium requirements. WHO Technical Report Series No 230, Geneva, WHO, 1988.
6. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Annexure 2, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.
7. Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium; Ross AC, Taylor CL, Yaktine AL, *et al.*, editors. Washington (DC): National Academies Press (US); 2011.
8. Sivakumar B, Nair KM, Sreeramulu D, *et al.* Effect of micronutrient supplement on health and nutritional status of school children: biochemical status. *Nutrition* 2006; 22: Suppl No1, 15S-25S.
9. Goswami R, Gupta N, Goswami D, *et al.* Prevalence and significance of low 25-hydroxyvitamin D concentrations in healthy subjects in Delhi. *American Journal of Clinical Nutrition*. 2000; 72:472–475.
10. Rege SS, Raut KN, Joglekar CV, Bhat DS, Naik SS, Yajnik CS. Circulating vitamin D₃ in rural and urban Indian men, Proceedings of the Nutrition Society, India, 38th Annual Meeting, (Abstract: BNM 6.05), P 78, 2006.
11. Marwaha, R, Sreenivas V, Talwar D, Yenamandra V, Challa A, Lakshmy R, Sethuraman G. Impact of solar ultraviolet B radiation (290-320 nm) on vitamin D synthesis in children with type IV and V skin. *British Journal of Dermatology*. 2015; 173(2):604-6.
12. Goswami R, Saha S, Sreenivas V, Singh N and Lakshmy R. Vitamin D-binding protein, vitamin D status and serum bioavailable 25(OH)D of young Asian Indian males working in outdoor and indoor environments. *Journal of Bone Mineral Metabolism*. 2017; 35:177–184.
13. Patwardhan VG, Mughal ZM, Chiplonkar SA, Webb AR, Kift R, Khadilkar VV, Padidela R, Khadilkar AV. Duration of Casual Sunlight Exposure Necessary for Adequate Vitamin D Status in Indian Men. *Indian Journal of Endocrinology & Metabolism*. 2018; 22(2):249-255.
14. Patwardhan VG, Mughal ZM, Padidela R, Chiplonkar SA, Khadilkar VV, Khadilkar AV. Randomized Control Trial Assessing Impact of Increased Sunlight Exposure versus Vitamin D Supplementation on Lipid Profile in Indian Vitamin D Deficient Men. *Indian Journal Endocrinology & Metabolism*. 2017; 21(3):393-398.
15. Wang H, Xia N, Yang Y, Peng DQ. Influence of Vitamin D supplementation on plasma lipid profiles: A meta-analysis of randomized controlled trials. *Lipids Health Dis*. 2012; 11:42.
16. George JA, Norris SA, van Deventer HE, Pettifor JM, Crowther NJ. Effect of adiposity, season, diet and calcium or vitamin D supplementation on the vitamin D status of healthy urban African and Asian-Indian adults. *British Journal of Nutrition*. 2014;112:590-9.
17. Tergestina M, Jose A, Sridhar S, Job V, Rebekah G, Kuruvilla KA, Thomas N. Vitamin D status and adequacy of standard supplementation in preterm neonates from South India. *Journal of Pediatric Gastroenterology and Nutrition*. 2014; 58(5):661-5.

18. Khadgawat R, Marwaha RK, Garg MK, Ramot R, Oberoi AK, Sreenivas V, Gahlot M, Mehan N, Mathur P, Gupta N. Impact of vitamin D fortified milk supplementation on vitamin D status of healthy school children aged 10–14 years. *Osteoporosis International*. 2013 Aug 1; 24(8):2335-43.
19. Kumar GT, Sachdev HS, Chellani H, Rehman AM, Singh V, Arora H, Filteau S. Effect of weekly vitamin D supplements on mortality, morbidity, and growth of low birth weight term infants in India up to age 6 months: randomised controlled trial. *BMJ*. 2011; 342:d2975.
20. Puri S, Marwaha RK, Agarwal N, Tandon N, Agarwal R, Grewal K, Reddy DH, Singh S. Vitamin D status of apparently healthy schoolgirls from two different socioeconomic strata in Delhi: relation to nutrition and lifestyle. *British Journal of Nutrition*. 2008; 99(4):876-82.
21. Jagadeesan V, Reddy V. Interrelationship between vitamins E and A: a clinical study. *Clinica Chimica Acta*. 1978 Nov 15; 90(1):71-4.
22. Jagadeesan V and Prema K. Plasma tocopherol and lipid levels in pregnancy and oral contraceptive users. *British Journal of Obstetrics & Gynaecology*. 1980; 87: 903-907.
23. Joint FAO/WHO. Vitamin and mineral requirements in human nutrition. A report of the joint FAO/WHO expert consultation, Bangkok, Thailand, 21-30 September 1998, Second Edition, 2004.

14. WATER

INTRODUCTION

Water is biologically important and has a major role in metabolism. Water is essential for various metabolic activities such as transportation of nutrients and oxygen to cells, maintenance of blood volume, functioning of the cardiovascular and digestive systems, regulation of body temperature, maintenance of acid-base balance, elimination of toxins, lubrication of skin and tissues, maintenance of elasticity of tissues and muscles, cell shape and structural integrity, boosting energy metabolism, reduction of arterial pressure, hydration of brain cells and thereby, maintaining better cognitive function.

Total Body Water (TBW)

Human body contains approximately 60% of body water, if we consider, a 70 kg healthy man, TBW would be approximately 42 litres and varies with age, gender, body size and composition. Distribution of water in various body tissues and organs by weight ranges from 10% to 95%, wherein, 10% is found in adipose tissue, 25% in bone, 75% in skin, 75% in muscles, 80% in brain, 85% in lungs, 83% in kidneys, 94% in blood and 95% in eyes¹⁻².

Males have a higher TBW (43-75%) than females (41-63%) due to their higher proportion of muscle mass and lower body fat mass. Elderly (39-67%), consequent to the aging process have a lower TBW than children (49-75%) and new born (64-84%)³. Athletes have a higher TBW due to the higher fat-free mass, lower body fat, and higher skeletal muscle glycogen levels than non-athletes. Glycogen present in the skeletal muscle increases the water content of fat-free tissue to regulate osmotic pressure exerted by glycogen granules within the muscle sarcoplasm.

Total body water (TBW) is mainly distributed between extracellular fluid (ECF) and intracellular fluid (ICF). In adults, the ECF contributes to 1/3 or 33 % of TBW (Approx. 14 litres) and the ICF contributes to 2/3 or 67% of TBW (Approx. 28 litres). Whereas, in neonates the ICF contributes to 1/3 or 33 % of TBW and the ECF contributes to 2/3 or 66 % of TBW. ECF is further divided into the interstitial fluid (ISF) and the intravascular fluid (blood plasma and lymph). The ISF contains 2/3 of ECF or 25% of TBW (Approx. 11 litres) and the IVF contains 1/3 of ECF or 8% of TBW (Approx. 3 litres). The ISF provides the medium for the passage of nutrients and metabolic products from cells to plasma and plasma to cells. IVF represents the fluid portion of the blood. The cell membranes separate IVF and ICF and blood vessels separate the interstitial fluid and the plasma. Another compartment of fluids in the ECF is transcellular fluid (TCF) which includes cerebrospinal fluid, synovial fluid, liquids secreted to the gastrointestinal tract (digestive juices), fluids in pleural, peritoneal and pericardial cavity. TCF is normally not accounted as it is a fraction of the extracellular fluid i.e., 2.5% of TBW (1 to 2 litres).

Composition of Body Fluids

Electrolytes: Electrolyte is a scientific term for salts, specifically electrically charged ion, which moves to either a positive (cathode) or negative (anode) electrode. A wide range of electrolytes and solutes are dissolved in ICF and ECF in varying amounts, that play an important role in maintaining membrane potential of cells and carry electrical impulses across the cells in the body. The major electrolytes present in the body are Sodium (Na^+), Potassium (K^+), Chloride (Cl^-), Calcium (Ca^{++}), Magnesium (Mg^{++}), Bicarbonate (HCO_3^-), Phosphate (PO_4^{2-}), Sulphate (SO_4^{2-}) and Organic Acids. It is critical for the body to maintain electrolyte homeostasis, in order to maintain the cell membrane potential, osmolarity of body fluids, and body water distribution. The presence of primary cation sodium and anions chloride, and bicarbonates in the ECF contributes to its

osmolarity and ECF volume. The cations potassium and magnesium, and anions proteins contribute to ICF osmolarity. Thus, the presence of electrically balanced solutes in the ICF and ECF contributes to water balance and osmolarity. If the electrolyte concentration in the ICF is greater (hypertonic) than the ECF, water is moved inside the cell freely and if the electrolytes are low (hypotonic), water is moved from the cell. Water movement between extracellular and intracellular water compartments is dependent on osmotic, hydrostatic and oncotic pressures³⁻⁴.

Regulation of Water and Electrolyte Balance

Several hormones such as angiotensin, aldosterone, vasopressin (antidiuretic hormone or arginine vasopressin), renin and natriuretic peptides influence the renal system to correct the fluid and electrolyte imbalances. The major regulatory factor in controlling sodium levels in the body is renin-angiotensin-aldosterone system (RAAS). When blood pressure or plasma volume is low, the granular cells in the juxtaglomerular apparatus of the kidneys are innervated by the sympathetic nervous system to release renin, which hydrolyse angiotensinogen (inactive protein synthesized in the liver) to angiotensin-I. Angiotensin-I is subsequently hydrolysed to angiotensin-II by angiotensin converting enzyme (ACE) which is present in the capillaries of the lungs. Angiotensin-II increases blood pressure by vasoconstriction of arteries, stimulating thirst centre in the hypothalamus, vasopressin release by kidneys to reabsorb water by the kidneys and reducing glomerular filtration, aldosterone secretion by adrenal cortex to reabsorb fluid and sodium through kidneys and increased potassium and hydrogen ions excretion through urine⁵⁻⁶.

Natriuretic peptide (ANP), a peptide hormone synthesized in the atrial cells of the heart and B- type natriuretic hormone (BNP), a peptide hormone synthesized by myocytes in the ventricles of the heart have opposing effects to RAAS. When plasma volume is expanded, the ANP and BNP are released into the circulation to increase glomerular pressure and filtration rate to promote loss of sodium and lower blood volume and pressure.

Thermoregulation

Maintenance of optimum hydration status is critical to regulate the body temperature. Body temperature is maintained by controlling the rate of heat production from basal metabolic rate, muscular activities, hormones, thermic effect of food, environment and rate of heat loss through radiation, conduction, convection and evaporation. The temperature control centre in the hypothalamus initiate an increase in heat production when body temperature falls and an increase in heat loss when body temperature rises. Heat loss through sweat, is an important cooling mechanism in normal conditions as well as in hot climates and in physical activity. Sweat losses if not compensated with fluid intake, can lead to a hypo-hydrated state, with concomitant increases in core body temperature. The magnitude of core temperature elevation ranged from 0.1°C to 0.23°C for every per cent body weight loss. Sweat production is dependent upon age, gender, physical activity, fitness levels, body composition, hydration status, acclimatization, environmental temperature and humidity, and the type of clothing worn. The combination of impaired thermoregulation and environmental heat stress can lead to heat related disorders such as heat cramps, heat exhaustion and heat stroke⁷⁻⁸.

Heat storage will be calculated before and during (at every chance of rest) exercise using the formula of Adams *et al.* (1992)⁹.

$$\text{Heat storage} = 0.965m\Delta Tb/\text{AD}$$

Where, 0.965 is the specific heat storage capacity of the body ($\text{W} \cdot \text{kg}^{-1} \text{C}^{-1}$), m is the mean body mass (kg), AD is the body surface area (m^2).

$$\text{Body Surface Area (BSA)}^{10} = (\text{W}^{0.425} \times \text{H}^{0.725}) \times 0.007184$$

Consequences of Over or Hyperhydration

Excess intake of fluid or decreased body's ability to excrete water or increased tendency to retain water by the body leads to overhydration. It is generally observed in disease conditions such as kidney disorders, liver diseases, congestive heart failure and hormonal imbalance. Under normal conditions, over hydration is not dangerous, however, in clinical conditions, it can be fatal. Over or hyper hydration reduces the concentration of sodium in the body and results in hyponatremia, this in turn leads to muscle cramps and weakness¹².

However, certain degree of hyperhydration before the event is recommended among athletes participating in long duration events such as distance running, cycling and rowing to improve the exercise performance or heat tolerance by lowering the body's core temperature.

Assessment of Hydration Status

Hydration can be measured using many techniques such as dilution technique, neutron activation analysis, bioelectrical impedance, saliva, urine, tears, serum/plasma indices, body mass changes, temperature and heat storage and perceptual indicators, but, each technique has its own pros and cons with regard to administration, cost, accuracy and reliability. It is required to determine the most accurate, realistic, convenient, and cost effective method for measuring hydration status depending on the degree of accuracy and facilities available.

Water Balance

Body water balance depends on the net difference between water gain and water loss. Water gain occurs from consumption of food and fluids and metabolic water production, while, water losses occur from renal, respiratory and skin surface.

Total Daily Water Losses

Majority of fluid is excreted via kidneys as urine and smaller amounts are excreted in the feces. Water losses occur via insensible losses such as respiratory tract and skin surface. Approximately 1 to 2 litres of water is lost through urine (60%), 50 to 200 ml through faeces (4%), and 250 to 350 ml through insensible losses such as evaporation from the lungs during exhalation (12%) and 600 ml (24%) through sweat and nonsweat diffusion from the skin. The water loss through urine depends on water intake, type of macronutrient consumed, and the amount of sodium. The loss of water through faeces depends on the amount of dietary fibre in the stool. The minimum urine output is approximately 500 ml/day and the maximum up to 20 L/day during diuretic condition. In dehydrated state, the output can be less than 500 ml/day. Exercise and heat strain will reduce urine output by 20 to 60 per cent, while cold and hypoxia will increase urine output.

The amount of insensible water loss through lungs is dependent on pprox. or volume and water vapour pressure gradient. Ventilatory volume is increased by physical activity, hypoxia, and hypercapnia, whereas the water vapour pressure is modified by the ambient temperature, humidity, and barometric pressure. The respiratory water loss is greatly influenced by physical activity than the environmental conditions. In sedentary people, respiratory water loss is approximately 250 to 350 ml/day, where as in active people, it may range from 500 to 600 ml/day. Exposure to high altitude may further increase respiratory water losses by 200 ml/day.

Respiratory water loss can be predicted from the equation given by Hoyt and Honig, 1996¹³.

$$\text{Respiratory water loss } y \text{ (mL/day)} = 0.107x + 92.2.$$

Where, x= metabolic rate (kcal/day)

Insensible non sweat losses of water from the skin is approximately 450 ml/day, and sensible dermal losses may range from 100 ml to 1000 ml. Water loss through sweat depends on the metabolic rate, duration and intensity of physical activity and environmental factors such as temperature, humidity, wind, radiation, type of clothing. Exercise in hot and less humid conditions can increase insensible water loss through lungs by 120 to 300 mL/day. In cold and less humid conditions, water losses at rest and heavy exercise can be approximately 5ml / hr and 15 to 45 ml/hr. At higher altitude levels, the losses can reach up to 250 ml/day above the base line. In armed forces (8 hours of rest, 12 hours of moderate activity, and 4 hours of moderate-heavy activity), the respiratory water losses increase by approximately 340 mL/day (breathing -20°C versus +25°C air)^{1,2}.

Sources of Total Daily Water Intake

Beverages (60-65%) and foods (25-30%) are the major contributors to total fluid requirements². Fluids other than water can be consumed in the form of dishes like rasam, sambar, porridges, kanji, soups, broth, tea, coffee, milk, buttermilk, fruit juices, coconut water, sugarcane juice and soda etc. Drinking fluids other than water can contribute to an intake of excess amounts of calories and other nutrients. Water content of different foods varies at a range of 10-95%. Water content of cereals, millets, nuts and oil seeds is less (5-14%) than fruits and vegetables (70-96%) milk and milk products (80-85%). The minor contributor to total fluid requirements is metabolic water (water produced during biochemical reactions) with a contribution of 10% to total fluid requirements. Daily metabolic water generation is about 200-300 ml depending on the macronutrient oxidation where 100 gm of each nutrient such as fats, carbohydrate and protein generates 110 ml, 55 ml and 41 ml of water^{2,14}. In case of athletes', the metabolic water production is high and may increase up to 500- 600 ml/day due to high oxidation of macronutrients¹⁵.

Water Requirement by Life Stage and Gender

Water requirement can be met from foods and fluids and the requirement varies based on age, gender, body size and composition, type of food, environment, and rate of metabolism, clothing type, and duration and intensity of physical activity. There are various approaches being adopted to estimate fluid requirements, however, the precise prediction of water requirements is impossible, except under controlled conditions^{1,2}. In India, so far no scientific research has been carried out and hence, there are no records available on adequate intake of water across various age-groups. To formulate age and gender specific water intake guidelines for Indians, recommendations given by World Health Organization (2005)¹⁶ have been adapted. The Food and Nutrition Board (FNB), a part of the National Research Council, was established in 1940, recommended 1-1.5 ml of fluid for each calorie ingested and further suggested additional fluid requirements depending on variation in physical activity level, heat stress, sweat losses and solute load.

Considering the lack of water balance studies among Indian population, the total water requirement across life stages was calculated using a factorial approach. It is based on the energy requirement (Estimated Average Ratio, 2020) and the WHO guidelines of 1.5 ml for each calorie ingested¹⁶. The water gained while consuming food and during metabolism was subtracted from the total water requirement in order to arrive at the water required from beverages, represented as ml per kg body weight, and the detailed rationale is provided under Table 14.1 – 14.4.

Infants	Total Water Requirement
0–6 months	0.6 L/day (from breast milk or formula)
7–12 months	1.0 L/day (from breast milk or formula, food, plain water and other beverages)

Rationale: Infants (0- 6 months) on exclusive breast milk do not require supplemental water and their requirement was calculated based on the average consumption of breast milk by Indian Infant (around 700 ml), corrected for the water content in breast milk (considering 88% in 100 ml)¹⁷. The total water requirement of Indian infants aged 7–12 months was calculated based on the estimated energy requirement (refer “Summary of EAR for Indians 2020”), multiplied with the amount of fluids recommended for each calorie (1.5 ml/cal) by WHO¹⁶ which amounts to 1 litre per day. Considering that the average intake of water from breast milk is around 530 ml/day (for an average of 600 ml of partial breast feeding), the remaining 470 ml/day (Approx. 0.5L) of water should be taken from complementary foods and fluids. Note that the total water requirements are rounded to the nearest 50.

Table 14.1: Water requirement for Children and Adolescents

Age Group	Body Weight (Kg)	Energy Requirement (Kcal)	Total Water Requirement* (ml)	Water from Food* (ml)	Water from Beverages (ml)	Water Requirement from Beverages (ml/Kg BW)
Children						
1-3 y	11.7	1010	1500	700	800	68
4-6 y	18.3	1360	2050	850	1200	66
7-9 y	25.3	1700	2550	1000	1550	61
Boys						
10-12 y	34.9	2220	3300	1200	2100	60
13-15 y	50.5	2860	4200	1300	2900	57
16-18 y	64.4	3320	4500	1500	3000	47
Girls						
10-12 y	36.4	2060	3050	1250	1800	49
13-15 y	49.6	2400	3500	1550	1950	39
16-18 y	55.7	2500	3700	1350	2350	42
Adult Man						
Sedentary	65.0	2110	3150	1100	2050	32
Moderate	65.0	2710	4050	1250	2800	43
Heavy	65.0	3470	5200	1450	3750	58
Adult Woman						
Sedentary	55.0	1660	2500	1000	1500	27
Moderate	55.0	2130	3200	1100	2100	38
Heavy	55.0	2720	4100	1250	2850	52
Pregnant	55.0+GWG ^a	+350	3700	1100	2600	-
Lactating	55.0+ ^b	+600	4100	1250	2850	-

*Values are rounded off to the nearest 50. BW=Body Weight

^aGestational weight gain; ^bGestational weight gain remaining after delivery

Rationale: The Total Water Requirement was calculated based on the Estimated Energy Requirement for Adult Man, Woman, adolescents and children (Represented in the Table 14.1 depicting “Summary of EAR for Indians 2020”) multiplied with the amount of fluid recommended per kilocalorie (1.5 ml/kcal)¹⁶. To arrive at the water from food, the total water gained (metabolic water and the moisture content in food) was estimated. Metabolic water was calculated using the Estimated Average Requirement (EAR) of macronutrients for Adult Man and Woman (Atwater general factor system was used for conversion of macronutrients in grams to calories, wherever applicable), multiplied with the water generated/gained during macronutrient oxidation (i.e. 100 grams of carbohydrate, protein and fat providing 55 ml, 41 ml and 110 ml of water, respectively)^{2,14}. The moisture content of food was estimated based on the age-specific recommended food group consumption of Sedentary and Moderate Adult Man (Appendix 1 and 2); For Heavy Man and Woman the Dietary Guidelines for Indian was used¹⁷, multiplied with the average per cent moisture content for cereals (10%), pulses (9%) and commonly consumed/selected roots and tubers (80%), green leafy vegetables (85%), other vegetables (89%) and fruits (84%) calculated from Indian Food Data Tables (2017)¹⁸. Considering that milk is classified under a beverage, it has been assumed that at least 250ml of 300ml recommended milk intake (RDA, 2020) may be consumed as a beverage and subtracted from the food group “milk and milk products”, to arrive at the moisture content from milk products (47%). Note that these calculations have not considered water content for animal foods due to lack of consistent data on its recommendations across age-groups¹⁸. Further, in hot and humid climate, water requirements are higher than the recommendations to maintain hydration status.

The energy and nutrient requirements of a pregnant woman is slightly higher than an adult woman because of expanding extracellular fluid space, the needs of the foetus and the amniotic fluid. Thus, there is an increased requirement of fluid in pregnancy. Water accounts for 88% of milk and the average milk production in the first six months of lactation is 700 ml/day and 7-12 months 500 ml/day. For production of milk lactating mother requires additional calories, nutrients and water. Therefore, the total water requirement was estimated from the estimated energy requirement for pregnancy and lactating mother (refer table depicting “Summary of EAR for Indians 2020”) multiplied with 1.5 ml of fluid recommended per calorie¹⁶. Metabolic water and water content in foods was considered as 30%¹⁶, due to lack of data on recommended food groups across the various categories of work during pregnancy and lactation, in order to obtain the amount of water intake from foods.

Table 14.2: Water requirement for elderly persons

Group	Body Weight (Kg)	Energy Requirement (Kcal)	Total Water Requirement* (ml)	Water from Food* (ml)	Water from Beverages (ml)	Water Requirement from Beverages (ml/kg BW)
Man						
Sedentary	60	1883	2800	850	1950	33
Moderate	60	2216	3300	1000	2300	38
Woman						
Sedentary	55	1706	2550	750	1800	33
Moderate	55	2007	3000	900	2100	38

*Values are rounded off to the nearest 50. BW= Body Weight

Rationale: Water requirements are higher in old age because of decreased thirst mechanisms. To maintain normal saliva flow rate, bowel movements, gastric functioning and emptying, metabolic, renal, cardiovascular functions and cognition, greater volume of fluids are recommended. To calculate the water requirement from beverages and the total water requirement, the ideal body mass and energy requirements for elderly were taken from the “Nutrient Requirements and Recommended dietary allowances for Indians, 2000²⁰” due to unavailability of other recent recommendations for Indians. It was estimated by multiplying the energy requirement with 1.5 ml of fluid recommended per calorie¹⁶. Foods which are high in water content are recommended. Metabolic water and water content in foods (fixed as 30% for men and woman)² were considered to obtain the amount of water intake from foods¹¹.

Athletes

Proper hydration should be ensured for an athlete for maintenance of health and improved performance²¹. Therefore, it is very important for an athlete to determine his/her individual fluid losses, in order to develop successful rehydration strategies. Adequate amounts of fluids, carbohydrates and electrolytes should be consumed before, during and after exercise. A basic plan with adjustments for changing environmental conditions and competition stress helps the athlete to be proactive in preventing and delaying dehydration and other nutrient related problems. In India, majority of the athletes are hypo hydrated due to various reasons such as lack of availability and accessibility of water during the practice sessions, ignorance, lack of awareness and knowledge and intentional restriction. The fluid recommendations for athletes is given in the Table 14.3.

Table 14.3: Fluid requirement for athletes

Exercise	Amount of Fluid Required	Amount of Carbohydrates, Sodium & Potassium	Type of Carbohydrate
Pre Exercise			
2-4 hrs prior	5-7 ml/kg/ BW	CHO: 1-4 g/kg/BW Na: 20-50 mmol/L	Moderate GI Foods
2 hrs prior	3-5 ml/kg/BW*	-	-
10- 20 min	200 -300 ml	-	
During Exercise			
<45 min	0.4-0.8 L/hr	CHO and Na: Not Required	-
45-75 min		CHO: Small amounts including mouth rinse	Single or multiple transportable carbohydrates
1-2.5 hrs		CHO: 30-60 g/hr; Na: 20-80 mmol/L; K: 4-8 mmol/L	Single or multiple transportable carbohydrates
>2.5 -3 hrs		CHO: 90 g/hr; Na: 20-80 mmol/L; K: 4-8 mmol/L	Only multiple transportable carbohydrates
Post Exercise			
<1 hr	1.25 -1.5 L/kg body weight lost	CHO:1-1.5g kg/BW for first 30 min, and again every 2 hours for 4-6 hours	Moderate to High GI foods

Source: Sawka *et al.* (2007)²². American College Sports Medicine Position Statement.

*if athlete is not hydrated.

Athletes can choose from a variety of fluids and each beverage has its own benefits and drawbacks. Water or isotonic solutions are best choices just prior to exercise. During exercise

isotonic carbohydrate and electrolyte drinks are most preferred. Milk, sugarcane juice, sports drinks and fruit juices are ideal after an exercise. However, no single strategy is best for all athletes and each athlete should find his or her own personalized plan. Coffee and tea are often preferred because of the claimed ergogenic effects of caffeine; however, it has diuretic effects. Athlete can assess their hydration status using simple Water Urine Thrust (WUT) method; water loss, urine colour and thirst. Athlete should consult physician in the case of Extreme Heat Illnesses (EHI) such as heat exhaustion, heat stroke and exertional hyponatremia.

Armed Forces

Soldiers are deployed in different climatic conditions such as hot deserts, hot humid jungles, snow bound high-altitude areas, underwater (submariners) etc. Fluid and electrolyte imbalances not only threaten the health of armed forces but also significantly impair performance and reduce combat effectiveness. Fluid requirements of various types of troops under different environmental conditions are calculated based on the energy expenditure estimated using indirect calorimetry and doubly labelled water. An intake of 1.5 ml was considered per each calorie expended. The requirement for sodium chloride is higher than the normal recommendations due to higher sweat loss. Approximately 15–16 g of salt normally taken in diet is quite adequate for acclimatized soldiers. Acclimatization to the environmental conditions for 3 days decreases the sodium losses through sweat^{23–25}. The Fluid requirements are presented in Table 14.4.

Table 14.4: Estimation of Fluid Requirements for Indian Troops based on their Energy Expenditure under Different Environments and Training

Type of troops and environment	Total Energy Expenditure (kcal/day)	Average Fluid Intake from Foods (ml/day)	Average Fluid Intake from Beverages (ml/day)	Average Total Fluid Requirement (ml/day)
Army				
Sea level- Combat and support	3511	1600	3650	5250
Desert -Combat and support	3304	1500	3450	4950
Training centre – Infantry	4670	2100	4900	7000
Training centre – Support*	3487	1600	3650	5250
Commando training*	4498	2050	4700	6750
HA warfare training*	4837	2200	5050	7250
HA(2,700–4,500m)- Combat and support	3880	1750	4050	5800
HA (>4,500 m) – Combat	4270	1900	4500	6400
Navy				
Ground crew	2900	1300	3050	4350
Officers	3615	1600	3800	5400
Air force				
Ship crew	3313	1500	3450	4950
Submariners	3168	1450	3300	4750
MARCOs and divers	4055	1850	4250	6100

Source: Total energy expenditure determined from Malhotra et al. (1975)²³; Force DotAaA (2003)²⁴; Singh et al. (2014)²⁵

HA- High altitude; Energy expenditure measured using oxygen consumption and *indicates where DLW method was used²². The energy expenditure and/or energy requirement was multiplied with 1.5 ml/kcal for estimation of total fluid requirements. Metabolic water and water content in foods (fixed as 30%)² were considered to obtain the amount of water intake from foods. Values were rounded to the nearest 50.

The guidelines provided by Force DotAaA (2003)²⁴ and Luippold *et al* (2018)²⁶ also can be adapted to recommend water intake during work by armed forces. Fluid recommendations were given based on the type of work involved, uniform configurations and work and rest cycles to maintain fluid and thermoregulation under five designated flag conditions (presented in Table 14.5). Military work was divided into easy, moderate and hard work; easy work involves weapon maintenance, walking on hard surface at 2.5 mph, <30 pound (lb) load, manual of arms marksmanship training, drill and ceremony; moderate work involves walking on loose sand at 2.5 mph, no load, walking hard surface at 3.5 mph, <40lb load, calisthenics, patrolling, individual movement techniques, defensive position construction; heavy work involves walking on hard surface at 3.5 mph, > or equal to 40 lb load and walking loose sand at 2.5 mph with load.

Table 14.5: Fluid Replacement and Work/Rest Guidance for Warm Weather Training Conditions and Refill Frequency for 3L Collapsible Drink System^{28,30}

Heat Category	WBGT Index (°F)	Easy work			Moderate Work			Hard Work		
		Work: Rest (min)	Water Intake (ml/hr)	Refilling Frequency (hr)	Work: Rest (min)	Water Intake (ml/hr)	Refilling Frequency (hr)	Work: Rest (min)	Water Intake (ml/hr)	Refilling Frequency (hr)
1 (White)	78-81.9	NL	475	4	NL	710	4	40:20	710	4
2 (Green)	82-84.9	NL	475	4	50:10	710	4	30:30	950	3
3 (Yellow)	85-87.9	NL	710	4	40:20	710	4	30:30	950	3
4 (Red)	88-89.9	NL	710	4	30:30	710	4	20:40	950	3
5 (Black)	>90	50:10	950	3	20:40	950	3	10:50	950	3

Source for classification of Heat Category: Institute of Medicine, 2005² and WHO, 2004¹⁶

*NL- No limit for work; WBGT- Wet-Bulb Globe Temperature; Rest- Minimal physical activity like sitting or standing in the shade.

Recommended fluid requirements and work: rest times will sustain performance and hydration for at least 4h of work in the specified heat category. Fluid requirements can vary based on individual differences (± 240 ml/h) and exposure to full sun or full shade (± 240 ml/h). If NBC (Nuclear, Biological, Chemical) clothing is worn (mission-oriented protective posture (MOPP 4)), then 10°F should be added to WBGT index for easy work, and 20°F should be added to WBGT index for moderate and hard work. The guidelines are viable for use with modern uniform configurations in primarily mild flag conditions.

References

1. Institute of Medicine (US). Panel on Dietary Reference Intakes for Electrolytes, Water. DRI, dietary reference intakes for water, potassium, sodium, chloride, and sulfate. National Academy Press; 2005.
2. Institute of Medicine. Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate. Washington, DC: The National Academies Press. 2005. <https://doi.org/10.17226/10925>.
3. Altman PL, Katz DD, editors. Blood and other body fluids. Federation of American Societies for Experimental Biology.
4. Barry MP, Kristen ED, Irwin HR. Water hydration and health. Nutrition reviews. 2010; 68(8):439-58. doi:10.1111/j.1753-4887.2010.00304.x.
5. Convertino VA, Armstrong LE, Coyle EF, Mack GW, Sawka MN, Senay Jr LC, Sherman WM. American College of Sports Medicine position stand. Exercise and fluid replacement. Medicine and science in sports and exercise. 1996 Jan 1; 28(1):i-vii.
6. Ganong, W.F. Review of Medical Physiology. 22nd edition, McGraw, Hill, New York. 2005.
7. Douglas J. C. Exercise in the Heat. I. Fundamentals of Thermal Physiology, Performance Implications, and Dehydration. Journal of Athletic Training. 1999; 34(3):246-252.
8. Hosseini A, Khamnei S, Zamanlu M. The effect of water temperature and voluntary drinking on the post rehydration sweating. International journal of clinical and experimental medicine. 2013; 6(8):683.
9. Adams WC, Mack GW, Langhans GW, Nadel ER. Effects of varied air velocity on sweating and evaporative rates during exercise. Journal of Applied Physiology. 1992 Dec 1; 73(6):2668-74.
10. Dubois D and Dubois EF. A formula to estimate the approximate surface area if height and weight be known. Archives of Internal Medicine. 1916; 17:863-871.
11. Sansevero, A.C. Dehydration in the elderly: strategies for prevention and management. Nurse Practices. 1997; 22:41–2:51–7:63–72.
12. Meyer F, Bar-Or O, Salsberg A, Passe D. Hypohydration during exercise in children: effect on thirst, drink preferences, and rehydration. International Journal of Sport Nutrition and Exercise Metabolism. 1994 Mar 1; 4(1):22-35.
13. Hoyt RW, Honig A. Energy and macronutrient requirements for work at high altitudes. Nutritional Needs in Cold and High-Altitude Environments: Applications for Military Personnel in Field Operations. 1996 May 16:379.
14. Mellanby, Kenneth. Metabolic water and desiccation. Nature. 1942; 150.3792: 21-21.
15. Bamji MS, Krishnaswamy K, Brahmam GN, editors. Textbook of human nutrition. Oxford & IBH; 2016.
16. Organisation mondiale de la santé, WHO--Work programme, Światowa Organizacja Zdrowia, World Health Organization, World Health Organisation Staff. Guidelines for drinking-water quality. World Health Organization; 2004 Aug 31.
17. Lawrence RA, Lawrence RM. Breastfeeding e-book: a guide for the medical professional. Elsevier Health Sciences; 2010 Sep 30.
18. Manual A. Dietary guidelines for Indians. National Institute of Nutrition. 2011; 2:89-117.
19. Longvah T, Anantaran R, Bhaskarachary K, Venkaiah K, Longvah T. Indian food composition tables. Hyderabad: National Institute of Nutrition, Indian Council of Medical Research; 2017.
20. Nutrient Requirements and Recommended Dietary Allowances for Indian, 2020. A report of the Expert Group. The Indian Council of Medical Research, New Delhi 110 029.
21. Potgieter S. Sport nutrition: A review of the latest guidelines for exercise and sport nutrition from the American College of Sport Nutrition, the International Olympic Committee and the International Society for Sports Nutrition. South African journal of clinical nutrition. 2013 Jan 1; 26(1):6-16.

22. Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American College of Sports Medicine position stand. Exercise and fluid replacement. Medicine and science in sports and exercise. 2007 Feb 1; 39(2):377-90.
23. Malhotra MS, Chandra U, Rai RM, Venkataswamy Y, Sridharan K. Food intake and energy expenditure of Indian troops in training. British Journal of Nutrition. 1976 Mar; 35(2):229-44.
24. Force DotAaA: Heat stress control and heat casualty management. Departments of the Army and Air Force Technical Bulletin Medical 507. Air Force Pam, 48–152(I). 2003.
25. Singh SN. Determining Nutritional Requirements of Indian Soldiers: An Outcome of Translational Research. In Translational Research in Environmental and Occupational Stress 2014 (pp. 109-116). Springer, New Delhi.
26. Luippold AJ, Charkoudian N, Kenefick RW, Montain SJ, Lee JK, Teo YS, Cheuvront SN. Update: efficacy of military fluid intake guidance. Military medicine. 2018 Sep 1; 183(9-10):e338-42.

15. ANTI-OXIDANTS

INTRODUCTION

Antioxidants (AO) are substances which are both nutrients, viz. vitamins E, C, β -carotene, selenium etc; and non-nutrients, viz. plant phenols, flavonoids, coumarins, benzyl isothiocyanates, caffeic, ferulic, gallic and ellagic acids, some enzymes like SOD, catalase superoxides mutase. These antioxidants reduce the adverse effects of reactive oxygen species (ROS) and nitrogen species, which are generated during physiological or pathological conditions and cause extensive oxidative damage. Literature is replete with evidence that ageing and several diet/nutrient related chronic disorders are due to chronic exposure to ROS. While it is well established that vegetables and fruits, legumes and spices and some beverages such as tea and wine are excellent sources of AO, scientific evidence for their protective role is available only for vegetables and fruits in several chronic disorders^{1,2}. None of the randomized clinical trials conducted so far with nutrient AO supplements have demonstrated a significant benefit in community trials barring one or two major trials in high-risk populations. Unfortunately, the term “antioxidant” is widely misused, for just about any molecule that can act as an antioxidant with an appropriate oxidizable substrate. Indeed, most antioxidants can become prooxidants under certain cellular circumstances; for instance, α -tocopherol (the most active form of vitamin E), can be pro-oxidant and initiate tocopherol-mediated peroxidation. Also, electron transfer to antioxidants can generate other free radicals that can have their own pathophysiological actions³. This chapter reviews the published studies and examines the question whether specific recommended dietary allowances for antioxidants could be fixed with available evidence.

Basic scientific studies

Experimental studies have amply indicated that both pro-oxidant and AO have a fundamental role in pathogenesis of diseases⁴. Reactive oxygen species (ROS) damage the bio-molecules such as DNA, protein, carbohydrates and lipids and affect the enzyme processes and genetic machinery. The oxidation products of bio-molecules accumulate with age. ROS can be derived from an environmental source also. There are several endogenous and exogenous sources of ROS, which play an important role in diseases such as cardiovascular, cancer, cataract, diabetes, neurodegenerative disorders and age-related maculopathy. Chronic infections aggravate the damage. Further, research in this field has highlighted the mechanistic details about the role of antioxidants in mitigating the damage. The phyto-chemicals (non-nutrients) have received considerable attention and are called the vitamins of the 21st century. Among the most investigated non-nutritive chemopreventors are plant phenols, flavonoids, coumarins, benzyl isothiocyanides, caffeic, ferulic, gallic and ellagic acids. Polyphenols are more complex and of great diversity in structure, bio-availability and functions^{5,6}. Free radicals produced during tissue metabolism and their consequent damage are reduced by nutrient antioxidants e.g. Vitamins E, C, β -carotene and selenium and non-nutrients such as polyphenols and flavonols and enzymes such as catalase and super-oxide dismutase. The AO, particularly vitamins E, C, co-enzyme Q and glutathione seem to be working in concert by recycling each other. *In vitro* studies have generated enough evidence for the antioxidant network concept with paucity of information for its validity *in vivo* particularly in relation to functional aspects of disease prevention, control or sustained therapeutic benefits. Though animal models of diseases suggest that natural and synthetic antioxidants can prevent development of clinical end points and there are correlations between circulating antioxidants and dietary intake, and beneficial effects have been demonstrated on surrogate bio-markers, randomized double blind control clinical trials have been disappointing⁷.

In healthy subjects, the dietary anti-oxidants from a balanced diet with adequate fruits and vegetables ranging from 500-600 g/d will probably be enough to take care of oxidant damage and repair cellular and tissue defects. However, certain groups of populations like pre-mature infants, smokers, alcoholics, and those exposed to environmental pollutants including carcinogens, individuals with chronic infections as well as those engaged in strenuous physical activity and geriatric population, are at high risk of oxidant damage.

Clinical trials

Role of antioxidants in reducing the risk of CVD has been a promising area of research. Experimental data does reveal that AO have a significant role to play from LDL oxidation and endothelial damage to platelet aggregation and thrombosis. Observational and analytical epidemiological studies on clinical end points are positive and the descriptive studies in general suggest a link between antioxidant nutriture and CVD. However, none of the randomized studies with enough power have provided the necessary evidence to increase the intake of antioxidants⁸. The Cambridge Heart Antioxidant study provides some support for vitamin E (400-800 mg) supplements for decreasing mortality in patients with myocardial infarction⁹. On the other hand, studies on cancer^{10,11} in smokers where β-carotene supplements were given in two separate studies, increased risk of cancer and fatal cardiac incidence were noted¹². There were no effects on colorectal adenoma or subsequent recurrence of cancers. On the other hand, studies in India and Canada on pre-cancerous lesions in oral cavity (vitamin A and β-carotene)^{13,14} and gastric cancer mortality in China (selenium, β-carotene and zinc) reported regression of lesions and 13% reduction in mortality respectively¹⁵. In US, patients with history of basal cell carcinoma intervention with selenium decreased only secondary end points such as total cancer mortality and incidence of lung, colo-rectal and prostate cancer¹⁶. A study in India with vitamin A, riboflavin, selenium and zinc on reverse smokers with pre-neoplastic palatal lesions exhibited a beneficial effect in terms of clinical remission^{17,18}. A recent meta-analysis of randomized trial of malignant transformation of oral leukoplakia as an outcome with vitamin A and retinoids and mixed tea and β-carotene supplements did not show any benefit. Even though clinical remission was better, there was a high rate of relapse¹⁹. A recent meta-analysis of antioxidant supplements on mortality due to cancer finds that they are of no benefit and infact seem to increase overall mortality²⁰. A Cochrane database review of well-controlled studies on vitamin-mineral supplements to control age-related muscular degeneration (AMD) found a beneficial effect of antioxidants (β-carotene, vitamins C and E and zinc) on progression to advanced stage.

Although there is a substantial body of evidence that a diet rich in plant foods (particularly fruit and vegetables) conveys health benefits, as do high plasma levels of several antioxidant nutrients found in these foods, a causal link between lack of antioxidants and disease occurrence or between antioxidant administration and disease prevention remains to be established. There is a lack of understanding of the mechanisms underpinning the apparent protective effect of plant foods and, as yet, no clear picture of which components are effective, and hence no way of predicting whether all or just some plant foods are important in this respect. Further evidence is required regarding the efficacy, safety and appropriate dosage of antioxidants in relation to chronic disease. The most prudent public health advice continues to be to increase consumption of plant foods²¹.

These studies in general show that subjects at risk may benefit from antioxidant supplements. The doses employed are relatively large and the effects may be pharmacological. It is not possible to extrapolate these results to general population to delay or prevent the onset of chronic diseases. Further, it is important to remember that antioxidants exert pro-oxidant effect towards other molecules under certain circumstances. The only positive statement that can be safely made is that a diet containing foods rich in several types of antioxidants helps in delaying ageing, reducing

cancer²², CVD and other disorders²³. The totality of scientific evidence from cells to animal models, from epidemiology to clinical trials needs to be consistent to formulate the recommended dietary allowances for antioxidants. Probably, the nutrient and non-nutrient antioxidants and their synergistic effect in food matrix is a cost-effective and sustainable solution. As far as strength of association and magnitude of effects are considered, there is enough hope for vegetables and fruits for all chronic disorders including neuro-degenerative disorders. Even in people subjected to strenuous physical activity like athletes or those practising recreational exercises, use of antioxidant supplements remains controversial. Only foods rich in antioxidants could be the recommendation.

Recommendations for dietary intake of antioxidants

People who run the risk of low intake of AO include economically poor, tobacco users and those who are perpetually on slimming diets or reduced intake of diet due to disease and related surgical interventions. In India, supra normal intakes are rare and therefore AO rich diets can be safely recommended to maximize potential health benefits and minimize toxicity. Liberal intake of vegetables, fruits, whole grains, legumes, nuts, seeds, spices, low fat dairy products to postpone ageing and fight diet-related chronic disorders and to promote better quality of life, should be recommended. Though short-term intervention studies in literature show biological effects, they will depend on the class of polyphenols. There are clear gaps. Therefore, it is not possible to fix dietary requirements for antioxidants till such time as co-ordinated research efforts are available on accurate biomarkers of risk for diseases and long-term functional benefits in terms of disease prevention and health promotion. There are two aspects to be considered. There is a wide range of non-nutrients, each of which can be exerting its AO activity. It is not possible to fix the AO activity from a food like fruits or vegetables. At present the amount of AO to be consumed daily to protect against risk factors cannot be quantitatively fixed. What can be recommended currently is consumption of a generous amount of fruits and vegetables (500 g/d) to protect against certain chronic disorders. Such a level of intake of fruits and vegetables also provides some of the vitamins, viz. vitamin A, vitamin E, etc., at higher than RDA levels.

References

1. National Academy of Science. Diet and Health. Implications for reducing chronic disease. Washington DC. National Academy Press, 1989.
2. Krishnaswamy K. Newer roles of vegetables in the prevention and control of problems of over nutrition and chronic degenerative diseases. Food security and vegetables-A global perspective. Bangalore, India: Dr Prem Nath Agricultural Science Foundation. 2004:162-80.
3. Dusting GJ, Triggle C. Are we over oxidized? Oxidative stress, cardiovascular disease, and the future of intervention studies with antioxidants. *Vasc Health Risk Manag.* 2005; 1(2):93-7.
4. Gey KF. Prospects for prevention of free radical disease, regarding cancer and cardio vascular disease. *British Medical Bulletien.* 1993; 49:679-99.
5. Rice-Evans CA, Miller NJ, Paganga G. Structure-antioxidant activity relationships of flavonoids and phenolic acids. *Free radical biology and medicine.* 1996 Jan 1; 20(7):933-56.
6. Proceedings of the 1st International Conference on Poly-phenols and Health. Dietary polyphenols and health. *American Journal of Clinical Nutrition.* 2005; S215-335.
7. Greig L, Maxwell S. Anti-oxidants-a protective role in cardiovascular disease? *Expert opinion on pharmacotherapy.* 2001 Nov 1; 2(11):1737-50.
8. Hennekens CH, Gaziano JM, Manson JA and Buring JE. Antioxidant vitamin cardiovascular disease hypothesis is still promising but still unproven: The need for randomized trials. *American Journal of Clinical Nutrition* 62: 1985; S1377-80.
9. Mitchension MJ, Stephens NG, Persons A *et al.* Mortality in the CHAOS trial. *Lancet.* 1999; 353: 381-2.
10. Alpha-Tocopherol Beta Carotene Cancer Prevention Study Group. The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. *New England Journal of Medicine.* 1994 Apr 14; 330(15):1029-35.
11. Omenn GS, Goodman GE and Thornquist MD. Effects of a combination of β-carotene and vitamin A on lung cancer and cardiovascular diseases. *New England Journal of Medicine.* 1996; 334 (18): 1150-5.
12. Rapola JM, Virtamo J, Ripatti S, Huttunen JK, Albanes D, Taylor PR, Heinonen OP. Randomised trial of α-tocopherol and β-carotene supplements on incidence of major coronary events in men with previous myocardial infarction. *The Lancet.* 1997 Jun 14; 349(9067):1715-20.
13. Stich HF, Hornby P and Dunn BP. A pilot β-carotene intervention trial with inuits using smokeless tobacco. *International Journal of Cancer.* 1985; 36:321-7.
14. Stich HF, Rosin MP, Hornby AP, Mathew B, Sankaranarayanan R, Nair MK. Remission of oral leukoplakias and micronuclei in tobacco/betel quid chewers treated with beta-carotene and with beta-carotene plus vitamin A. *International Journal of Cancer.* 1988 Aug 15; 42(2):195-9.
15. Blot WJ, Li JY, Taylor PR, Guo W, Dawsey S, Wang GQ, Yang CS, Zheng SF, Gail M, Li GY, Yu Y. Nutrition intervention trials in Linxian, China: supplementation with specific vitamin/mineral combinations, cancer incidence, and disease-specific mortality in the general population. *JNCI: Journal of the National Cancer Institute.* 1993 Sep 15; 85(18):1483-91.
16. Clark LC, Combs GF, Turnbull BW, Slate EH, Chalker DK, Chow J, Davis LS, Glover RA, Graham GF, Gross EG, Krongrad A. Effects of selenium supplementation for cancer prevention in patients with carcinoma of the skin: a randomized controlled trial. *Jama.* 1996 Dec 25; 276(24):1957-63.
17. Krishnaswamy K, Prasad MP, Krishna TP, Annapurna VV, Reddy GA. A case study of nutrient intervention of oral precancerous lesions in India. *European journal of cancer part B: oral oncology.* 1995 Jan 1; 31(1):41-8.
18. Prasad MP, Mukundan MA, Krishnaswamy K. Micronuclei and carcinogen DNA adducts as intermediate end points in nutrient intervention trial of precancerous lesions in the oral cavity. *European Journal of Cancer Part B: Oral Oncology.* 1995 May 1; 31(3):155-9.

19. Lodi G, Sardella A, Bez C, Demarosi F, Carrassi A. Systematic review of randomized trials for the treatment of oral leukoplakia. *Journal of dental education*. 2002 Aug; 66(8):896-902.
20. Bjelakovic G, Nikolova D, Simonetti RG, Gluud C. Antioxidant supplements for preventing gastrointestinal cancers. *Cochrane Database Syst Rev*, 2004, Oct., 18 (4): CD004183. *Evid Based Nurs*. 2005; 8(2):48.
21. Stanner SA, Hughes J, Kelly CN, Buttriss J. A review of the epidemiological evidence for the 'antioxidant hypothesis'. *Public health nutrition*. 2004 May; 7(3):407-22.
22. Evans JR, Lawrenson JG. Antioxidant vitamin and mineral supplements for slowing the progression of age-related macular degeneration. *Cochrane Database of Systematic Reviews*. 2017(7).
23. World Cancer Research Fund in Association with American Institute of Cancer Research. *Food nutrition and prevention of cancer: A global perspective*, 1997.

16. RECOMMENDATIONS FOR FUTURE RESEARCH

1. It is critical to get current and precise estimates of what is being eaten in India, at all ages, all socioeconomic levels and geographical areas. This was earlier done by the NNMB, but this needs to be expanded and restored.
2. Additional information on health (anthropometry, biomarkers of nutritional status and overnutrition) also needs to be collected simultaneously. This needs to be collected at the household level, powered adequately for district level estimates. Measurement of body composition also should be undertaken in subgroups.
3. Energy, protein and fat requirements of young children, and the response of body composition to feeding.
4. Evaluate appropriate and optimum feeding for SAM and MAM, along with body composition and blood biomarkers.
5. Evaluate biomarker cut-offs to define health in India. This requires a proper survey of anthropometry and nutritional biomarkers of healthy, adequately nourished population of different age and gender groups, residing in different urban and rural areas of the country.
6. Defining the extent of physiological adaptation to different nutrient intake levels, with a view to defining functional cut-offs for deficiency.
7. Direct estimation of energy requirements through measurement of energy expenditure and physical activity by the doubly labelled water (DLW) and heart rate monitor (HRM) methods, in urban and rural areas.
8. Direct determination of energy requirement of Indian infants, preschool children, children up to 10 years and adolescents employing the DLW method and HRM methods.
9. Longitudinal breast milk nutrient studies and its relationship to maternal nutritional status.
10. Nutrient requirements of special groups such as those engaged in heavy physical work, and sports persons.
11. Nutrient requirement during pregnancy and lactation, in relation to gestational weight gain.
12. More systematic calcium balance studies have to be done in normal subjects (adolescents, postmenopausal women and men post 65 y) on predominantly vegetarian diet with limited protein intake. This need to be accompanied by bone density study to match the two measurements.
13. Beneficial effects of vitamin D supplements during pregnancy, infancy and childhood and long-term effects of higher vitamin D intakes, differences in vitamin D₂, D₃ supplements and natural vitamin D, their kinetics and efficiency.
14. Zinc and iron bioavailability from whole meals needs to be assessed in different physiological groups, particularly pregnant and lactating women, using stable isotope labelling method.
15. Identifying reliable indicators and defining cut-off values for clinical and subclinical deficiency states or functional and biochemical end points for vitamins like A, D, B₁, B₂, B₆, B₁₂, folic acid etc. with specific biomarkers based studies.
16. More reliable data has to be collected on folate in Indian foods and their absorption.

17. Definitive studies are necessary to fix the beta carotene to vitamin A conversion ratio. This will help in fixing vitamin A requirements in terms of beta carotene intake.
18. Determine antioxidant potential of different Indian foods.
19. Studies to determine the requirements, lower and upper limits of macronutrient intakes carbohydrates including fiber, fats with special focus on EFA and proteins, keeping in view the current nutrition scenario.
20. Role of nutrients on epigenetic processes and application of nutrigenomics to develop personalized nutrition guidelines.
21. In view of current status of mandatory food fortification with certain nutrients, in the context of nutrient -nutrient interactions, it is essential to conduct periodic studies to evaluate the SUL, TUL, NOAEL etc.

APPENDICES

Appendix 1

A1. Food composition and nutrient content value of balanced diet for moderate active man

Food Composition	Amount (g /day)	Nutrient	Vegetarian diet	Non-vegetarian diet	EAR	RDA	
Cereals & Millets	360	Energy (kcal)	2690	2650	2710	2710	
		Protein (g)	87.5	81.7	43	54	
Pulses (Legumes)/ flesh foods ¹	120	Visible fat (g)	35	35	30	30	
Green leafy vegetables	150	Calcium (mg)	1084	1054	800	1000	
Other Vegetables	200	Iron (mg)	33.3	31.1	11	19.0	
Roots & Tubers (excluding potatoes)	100	Zinc (mg)	16.3	15.9	14.0	17	
Fruits	150	Magnesium (mg)	968	891	320	385	
Milk	300	Vitamin A (µg)*	1802	1796	460	1000	
Fats & Oils	30	β carotene	9842	9779	2760	6000	
Oil seeds & Nuts (gingely seeds & Pea nuts)	30	Thiamine (mg)	2.0	1.9	1.5	1.8	
		Riboflavin (mg)	1.9	1.9	2.1	2.5	
		Niacin (mg)	19	20.0	15	18	
		Vitamin B ₆ (mg)	2.4	2.4	1.7	2.1	
		Vitamin C (mg)	209	209	65	80	
		Total Folates (µg)	559	491	250	300	
		Vitamin B ₁₂ (µg) ²	1.5	2.4	2.0	2.5	

¹ Protein quality depends on the type of food.

¹Pulses can be replaced with animal foods (egg, meat, fish and chicken) for non-vegetarians to meet the requirements.

²Only 60% of the RDA of vitamin B₁₂ can be met by the vegetarian diet.

* Retinol derived from β carotene from diet was also added to the total

For cereals and millets, it is recommended to consume 50% as whole grains.

For green leafy vegetables (GLVs), it is recommended to consume 150g of GLVs, especially drumstick leaves, amaranth and bathua leaves to meet the requirements of all the vitamins and minerals.

Appendix 2

A2. Food composition and nutrient content value of balanced diet for sedentary man

Food Composition	Amount (g /day)	Nutrient	Vegetarian diet	Non-vegetarian diet	EAR	RDA
Cereals & Millets	275	Energy (kcal)	2130	2084	2110	2110
		Protein (g)	67.2	69.1	43	54
Pulses (Legumes)/ flesh foods ¹	80	Visible fat (g)	25	25	25	25
Green leafy vegetables	150	Calcium (mg)	968	958	800	1000
Other Vegetables	200	Iron (mg)	27.4	26.3	11.0	19.0
Roots & Tubers (excluding potatoes)	100	Zinc (mg)	13.5	13.2	14.0	17.0
Fruits	150	Magnesium (mg)	798	762	320	385
Milk	300	Vitamin A (µg)*	1768	1765	460	1000
Fats & Oils	25	B-carotene (µg)	9638	9605	2760	6000
Oil seeds& Nuts (gingely seeds & Pea nuts)	30	Thiamine (mg)	1.7	1.6	1.2	1.4
Spices	10	Riboflavin (mg)	1.6	1.6	1.6	2.0
		Niacin (mg)	15.6	16.0	12	14
		Vitamin B ₆ (mg)	2.0	2.0	1.3	1.6
		Vitamin C (mg)			65	80
		Total Folates (µg)	483	450	250	300
		Vitamin B ₁₂ (µg) ²	1.5	2.0	2.0	2.5

¹ Protein quality depends on the type of foods.

¹Pulses can be replaced with animal foods (egg, meat, fish and chicken) for non-vegetarians to meet the requirements.

²Only 60% of the RDA of vitamin B₁₂ can be met by the vegetarian diet.

* Retinol derived from β carotene from diet was also added to the total

For cereals and millets, it is recommended to consume 50% as whole grains.

For green leafy vegetables (GLVs), it is recommended to consume 150g of GLVs, especially drumstick leaves, amaranth and bathua leaves to meet the requirements of all the vitamins and minerals.

Appendix 3

A3. Food composition and nutrient content value of balanced diet for moderate active woman

Food Composition	Amount (g /day)	Nutrient	Vegetarian diet	Non-vegetarian diet	EAR	RDA
Cereals & Millets	300	Energy (kcal)	2135	2084	2130	2130
		Protein (g)	74.2	73.3	36.3	45.7
Pulses (Legumes)/ flesh foods ¹	90	Visible fat (g)	20	20	25	20
Green leafy vegetables	150	Calcium (mg)	999	989	800	1000
Other Vegetables	200	Iron (mg)	29.0	27.9	15.0	29.0
Roots & Tubers (excluding potatoes)	100	Zinc (mg)	14.0	13.6	11.0	13.2
Fruits	150	Magnesium (mg)	841	806	270	325
Milk	300	Vitamin A (µg) *	1804	1741	390	840
Fats & Oils	20	B-carotene (µg)	9489	9457	-	-
Oil seeds& Nuts (gingely seeds & Pea nuts)	30	Thiamine (mg)	1.75	1.70	1.4	1.7
spices	10	Riboflavin (mg)	1.65	1.64	2.0	2.4
		Niacin (mg)	16.3	16.8	12	14
		Vitamin B ₆ (mg)	1.96	1.97	1.3	1.6
		Vitamin C (mg)	187	187	55	65
		Total Folates (µg)	191	459	180	220
		Vitamin B ₁₂ (µg) ²	1.5	2.0	2	2.5

¹ Protein quality depends on the type of foods.

¹Pulses can be replaced with animal foods (egg, meat, fish and chicken) for non-vegetarians to meet the requirements.

²Only 60% of the RDA of vitamin B₁₂ can be met by the vegetarian diet.

* Retinol derived from β carotene from diet was also added to the total

For cereals and millets, it is recommended to consume 50% as whole grains.

For green leafy vegetables (GLVs), it is recommended to consume 150g of GLVs, especially drumstick leaves, amaranth and bathua leaves to meet the requirements of all the vitamins and minerals.

A4. Food composition and nutrient content value of balanced diet for Sedentary woman

Food Composition	Amount (g /day)	Nutrient	Vegetarian diet	Non- vegetarian diet	EAR	RDA
Cereals & Millets	200	Energy (kcal)	1690	1650	1660	1660
		Protein (g)	58.2	57.4	36.3	45.7
Pulses (Legumes) ¹	60	Visible fat (g)	15	15	20	20
Green leafy vegetables	150	Calcium (mg)	905	895	800	1000
Other Vegetables	200	Iron (mg)	23.8	22.8	15.0	29.0
Roots & Tubers (excluding potatoes)	100	Zinc (mg)	11.0	10.5	11.0	13.2
Fruits	150	Magnesium (mg)	684	649	270	325
Milk	300	Vitamin A (µg) *	1730	1727	390	840
Fats & Oils	15	B-carotene (µg)	9412	9380	-	-
Oil seeds& Nuts (gingely seeds & Peanuts)	30	Thiamine (mg)	1.36	1.30	1.1	1.4
spices	10	Riboflavin (mg)	1.5	1.48	1.6	1.9
		Niacin (mg)	13.0	13.5	9.0	11.0
		Vitamin B ₆ (mg)	1.7	1.7	1.3	1.6
		Vitamin C (mg)	187	187	55	65
		Total Folates (µg)	426	395	180	220
		Vitamin B ₁₂ (µg) ²	1.5	2.0	2	2.5

¹ Protein quality depends on the type of foods.

¹Pulses can be replaced with animal foods (egg, meat, fish and chicken) for non-vegetarians to meet the requirements.

²Only 60% of the RDA of vitamin B₁₂ can be met by the vegetarian diet.

* Retinol derived from β carotene from diet was also added to the total

For cereals and millets, it is recommended to consume 50% as whole grains.

For green leafy vegetables (GLVs), it is recommended to consume 150g of GLVs, especially drumstick leaves, amaranth and bathua leaves to meet the requirements of all the vitamins and minerals.

Appendix 5

A5. Food composition and nutrient content value of balanced diet for pregnant woman

Food Composition	Amount (g /day)	Nutrient	Vegetarian diet	Non-vegetarian diet	EAR	RDA
Cereals & Millets	250	Energy (kcal)	2060	2040	2010	2010
		Protein (g)	71.7	71.6	54	56
Pulses (Legumes) ¹	75	Visible fat (g)	15	15	30	30
Green leafy vegetables	150	Calcium (mg)	980	970	800	1000
Other Vegetables	200	Iron (mg)	27.2	26.0	32	40
Roots & Tubers (excluding potatoes)	100	Zinc (mg)	13.1	12.8	13.0	15.5
Fruits	150	Magnesium (mg)	786	747	320	385
Milk	400	Vitamin A (µg) *	1821	1818	406	900
Fats & Oils	15	B-carotene (µg)	9634	9597	-	-
Oil seeds& Nuts (gingely seeds & Pea nuts)	40	Thiamine (mg)	1.64	1.60	1.4	1.7
Spices	10	Riboflavin (mg)	1.8	1.84	2.3	2.19
		Niacin (mg)	16.0	16.7	14	14
		Vitamin B ₆ (mg)	2.0	2.0	1.6	2.0
		Vitamin C (mg)	210	210	65	70
		Total Folates (µg)	484	447	480	570
		Vitamin B ₁₂ (µg) ²	2.0	2.4	2.2	2.8

¹ Protein quality depends on the type of foods.

¹Pulses can be replaced with animal foods (egg, meat, fish and chicken) for non-vegetarians to meet the requirements.

²Only 80% of the RDA of vitamin B₁₂ can be met by the vegetarian diet.

* Retinol derived from β carotene from diet was also added to the total

For cereals and millets, it is recommended to consume 50% as whole grains.

For green leafy vegetables (GLVs), it is recommended to consume 150g of GLVs, especially drumstick leaves, amaranth and bathua leaves to meet the requirements of all the vitamins and minerals.

A6. Key micronutrients in different food groups
 (All values are for 100g edible portion)

Micronutrients	Cereals ^a	Pulses ^b	Leafy Vegetables ^c	Other vegetables	Roots & Tubers ^d	Fruits ^e	Milk (Buffalo') ^f	Milk (Cow) ^f	Egg	Chicken	Mutton	Beef	Fish	Liver (Sheep, Goat, Lamb)
Iron (mg)	3.00	5	8.5	2.12	0.6	0.56	0.2	0.2	1.82	1.5	1.3	2.2	0.6	6.3
Zinc (mg)	2.16	2.1	0.2	0.3	0.3	0.11	0.4	0.3	1.2	1.7	3	4.6	0.6	3.6
Vitamin A (µg)	2.43	8.6	259.1	22.4	70.0	32.36	49.8	58.3	198	21.4	9	15.5	5.6	6690
Riboflavin (mg)	0.15	0.14	0.1	0.07	0.0	0.01	0.13	0.11	0.2	0.09	0.16	0.12	0.03	0.36
Dietary folate (µg)	24.03	127.7	16.7	24.4	31.3	17.61	8.6	7	49.3	9.3	6.4	8.1	15.4	92
Vitamin B₁₂ (µg)	Nil	Nil	Nil	Nil	Nil	Nil	1.5	1.5	1.8	NA	2.8	1.7	1.4	91.9

^a Mean values of nutrients from commonly consumed cereals (67% weightage) such as rice and wheat were taken and 33% weightage was also given to millets such as Bajra, Jowar, Maiz and Ragi.

^b Mean values of nutrients from Lentils, Tur dhal, Bengal gram, Black gram, Cowpea, Green gram, Peas, Rajmah, Red gram and Soyabean were considered.

^c Carotenoid conversion to retinol equivalents.

^d Mean values of nutrients from Beetroot, Carrot, Colocasia, Onion, Radish, Tapioca and Yam were considered.

^e Mean values of nutrients from Amla, Apple, Banana, Cherries, Grapes, Guava, Jack fruit, Lemon, Lichi, Mango, Melon, Orange, Papaya, Pine apple, Pomegranate, Sapota, Custard apple, Strawberry were considered.

^f Good source of bioavailable calcium.

NA= Not available; NR=Not reported

- Low absorption of non heme iron can be improved by consuming more vitamin C rich foods (Amla, Lemon, Oranges, Guava, etc.) in raw form as much as possible.
- Meat, poultry and liver contains high bio-available heme iron and also increases absorption (meat factor) of non-heme iron (including fish).

References

1. Longvah T, Ananthan R, Bhaskara Chary K and Venkaiah K (2017). Indian Food Composition Tables – 2017, ICMR-National Institute of Nutrition.
2. HeimoScherz and Friedrich Senser : Food Composition and Nutrition Tables, Sixth Edition, Medpharm Scientific Publishers Stuttgart, CRC Press, 2000
3. Food Standards Agency (2002) McCance and Widdowson's The Composition of Foods, Sixth Summary Edition, Cambridge: Royal Society of Chemistry.
4. Gopalan C, Rama Sastry BV and Balasubramanian SC: Nutritive Value of Indian Foods. First Edition 1971. Revised and updated by Narasinga Rao BS, Deosthale YG and Pant KC 1989, National Institute of Nutrition, Hyderabad.