NUTRIENT REQUIREMENTS AND RECOMMENDED DIETARY ALLOWANCES FOR INDIANS

A Report of the Expert Group of the
Indian Council of Medical Research

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The Director
National Institute of Nutrition
Jamai-Osmania PO, Tarnaka, Hyderabad-500 007
Andhra Pradesh, India

and

The Director-General
Indian Council of Medical Research
V Ramalingaswami Bhawan, Ansari Nagar
New Delhi-110 029

For information contact : The Director
National Institute of Nutrition
Jamai-Osmania PO, Tarnaka, Hyderabad-500 007
Andhra Pradesh, India
Email: icmrrda@gmail.com

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CONTENTS

Preamble	...	i
Members of the Expert Committee and Resource Persons	...	iv
Summary of Recommendations	...	vii
1. Introduction	...	1
2. General Considerations	...	6
3. Reference Body Weights	...	11
4. Energy	...	19
5. Protein	...	54
6. Fat	...	90
7. Dietary Fiber	...	112
8. Minerals	...	122
8.1. Calcium and Phosphorus	...	122
8.2 Magnesium	...	142
8.3 Sodium	...	148
8.4 Potassium	...	153
9. Iron	...	157
10. Zinc	...	174
11. Trace Elements	...	180
11.1. Copper, Manganese and Chromium	...	180
11.2. Selenium	...	184
11.3. Iodine	...	188
12. Water Soluble Vitamins	...	194
12.1 Thiamine	...	194
12.2 Riboflavin	...	198
12.3 Niacin	...	202
12.4 Vitamin B_6	...	205
12.5 Folate	...	212
12.6 Vitamin B_{12}	...	216
12.7 Ascorbic Acid (Vitamin C)	...	220

13. Fat Soluble Vitamins ... 230

13.1. Vitamin A ... 230
13.2. Vitamin D ... 233
13.3 Vitamin E (Alpha tocopherol) and Vitamin K ... 236

14. Antioxidants ... 244

15. Recommendations for Future Research ... 249

Appendices

Appendix 1: Food composition and nutrient content value of balanced diet for moderately active man ... 252

Appendix 2: Key Micronutrients in Vegetable and Animal Foods ... 253

Appendix 3: Summary of Recommended Dietary Allowances (RDA) for Energy, Protein, Fat and Minerals for Indians-2010 ... 254

Appendix 4: Summary of Recommended Dietary Allowances (RDA) for Water Soluble and Fat Soluble Vitamins for Indians–2010 ... 255

PREAMBLE

The Indian Council of Medical Research (ICMR) constituted an Expert Group in 2008, to revise and update the nutrient requirements and dietary allowances for Indians. This is indeed the Sixth Expert Group constituted by the ICMR. The first Recommendations on safe dietary intake by Indians were made in 1944 by the Nutrition Advisory Committee of Indian Research Fund Association (now ICMR). It was based on recommendations of the Health Committee of League of Nations in 1937 for desirable safe dietary intakes of nutrients for human health, adapted to Indian dietary habits and body weights of Indians of different ages. As our knowledge about human nutrient requirements improved, ICMR Nutrition Advisory Committee revised Recommended Dietary Allowances (RDA) for Indians on calories and proteins in 1960. In 1968, another Expert Group constituted by the ICMR revised nutrient requirements and RDA for Indians in respect of all nutrients except calorie. Subsequent revisions updating the nutrient requirements of RDA was done by the Expert Groups of ICMR in 1978 and 1988 also. While revising and updating nutrient requirements and RDA, the Expert Groups had based their recommendations on the knowledge generated by Indian Research and on International Reports especially by FAO, WHO and UNU.

The current revision of RDA has been undertaken after a gap of 20 years. There was a pressing need for a revision from all stakeholders of health and nutrition in view of the versatility of its implications in forming a basis for several national activities related to food nutrition and health like (a) fixing minimum wages of workers by the Planning Commission (b) planning food production through agriculture (c) planning import of food to meet the gap in the food needs of our population (d) guidance for national regulatory bodies like Food Safety Standards Authority of India (FSSAI).

The report currently arrived at is based on a series of deliberations on available evidence and considering the recent FAO/WHO/UNU recommendations, wherever adequate evidence from our own country is not available. The report included newer aspects of major nutrients like energy, fat and proteins, and also emphasized on a few trace minerals like zinc and selenium and newer dietary components like dietary fibre and antioxidants, which were not considered in the previous recommendations. Physical activity was given emphasis for deriving energy requirements of children while outdoor activity as a means of meeting vitamin D requirements for all age groups was recommended. Recent FAO/WHO guidelines were followed for fat requirements, considering the fact that during the past two decades nutritional and health consequences of dietary fat and its fatty acids have been shown to be

more varied and detrimental in humans than was understood hitherto. The group extensively reviewed the available scientific database to have a consensus on reference body weights for different physiological groups. The body weights of different physiological groups for the purpose of deriving RDA was computed from the National database on anthropometry. The 95th centile values of weights and heights for a given age and gender class were considered to be representative of well nourished normal Indian population. For children below 3 years median weights of WHO/MGRS growth standard was considered.

Resource Persons provided background papers on requirement and safe intakes of different nutrients. Some members of the Expert Group also contributed to the background papers on energy, protein and fat. The draft report was circulated among the committee members well in advance of the meeting. The Expert Group met at the NIN on 28-29 of April 2009. Each nutrient was considered one by one at the Expert Group meeting for discussing the specific comments of the members of the committee. The deliberations focussed on available evidence and proposed Indian RDA and discussed in the light of the existing international nutrient requirements. Detailed discussions were carried out on all nutrients especially energy, protein, fat and calcium. The second meeting was held on 3rd of November 2009 to deliberate on the revised document. The group agreed upon the RDA for all the nutrients. Comments from stakeholders of health and nutrition were elicited by publishing the draft document in ICMR website for a period of one month from 15th July to 16th of August 2010. The responses obtained were consolidated and circulated among the experts of the committee and appropriate modifications were incorporated into the document and finalized. The final document is being released to coincide with the centenary of ICMR (1911-2011).

I wish to place on record the wholehearted support and expert contributions of the members of the committee in revising and bringing out this report. The list of the members of the present group is given in the next page. The contribution from Dr. B. Sivakumar, former Director, NIN and Principal Coordinator of the Expert Group in preparing the background papers should be gratefully acknowledged. Many thanks are also due to Dr N Balakrishna, Scientist D, Division of Biostatistics, NIN and Dr. K. Damayanthi, Scientist 'C', Division of Extension and Training, NIN for providing valuable input. The editorial correction by Dr. V. Ramdas Murthy, Retired Senior Scientist, NIN and Mr. S. Devendran, Artist, NIN for his assistance in designing the cover page is gratefully acknowledged.

I hope that this document will be of immense value to all concerned in the area of health and nutrition. I would also recommend this valuable document

to form a part of curriculum, capable of creating human resource with a strong foundation in the important concepts governing nutrition as a science.

The committee is always receptive to constructive criticism and can be contacted at icmrrda@gmail.com.

Dr. B. S. Narasinga Rao
Chairman

Members of the Expert Committee and Resource Persons

Chairman

Dr. Narasinga Rao B.S
Former Director, NIN
D-1603, Mantrigreens Apartments,
No.1, 1st Main Sampige Road,
Bangalore- 560 003

Member Secretary

Dr. Sesikeran B
Director
National Institute of Nutrition
Jamai-osmania, Tarnaka P.O.
Hyderabad – 500 007

Principal Coordinator

Dr. Sivakumar B
Former Director, NIN
H.No.12-13-1231/7,
Pranitha Apartments
Street No. 9, Tarnaka, Hyderabad

Coordinator

Dr.Madhavan Nair K
Scientist E
Head, Micronutrient Research
National Institute of Nutrition
Hyderabad-500 007

MEMBERS

Dr. Anura V Kurpad
Dean, St. Johns Research Institute
Prof of Physiology, ST John's
Medical College
St. John's National Academy of
Health Sciences
Bangalore-560 034

Dr. Bhan, M.K
Secretary to Govt. of India
Ministry of Science & Technology
Department of Biotechnology
New Delhi-110 003

Dr. Ghafoorunissa
Former Dy. Director (Sr.Gr.), NIN
Res. Flat No.402
Maphar Comfortek Apt.
10-2-289/37/A, Shantinagar
Masab Tank, Hyderabad-500 028

Dr. Kamala Krishnaswamy
Former Director, NIN
H.No.2-98/2, Sriniketan
Kakateeyanagar Colony
Habsiguda, Hyderabad-500 007

Dr. Lokesh B.R
Editor-in-Chief
Journal of Food Science &
Technology
Dept. of Biochemistry & Nutrition
Central Food Technological
Institute
Mysore-570 013

Dr. Mahtab S Bamji
INSA Honorary Scientist
Dangoria Charitable Trust,
Hyderabad
Director-Grade-Scientist (Retd.),
NIN
211, Sri Dattasai Apartments
Charminar X Roads
Hyderabad-500 020

Dr. Prakash V
Director
Central Food Technological Institute, Mysore-580 013

Dr.Prakash Shetty
Professor of Human Nutrition
Head, Public Health Nutrition Unit
London School of Hygiene & Tropical Medicine
49/51, Bedford Square,
London WC 1B 3DP, U.K.

Dr. Prema Ramachandran
Director,
Nutrition Foundation of India
C-13, Qutab Institutional Area
New Delhi-110 016

Dr. Rajagopalan S
Distinguished Fellow
MS Swaminathan Research Foundation, 3rd Cross Street,
Taramani Institutional Area
Chennai-600 013

Dr. Ramachandran A
Diabetes Research Centre
4, Main Road, Royapuram
Chennai-600 013

Dr. Srinath Reddy K
President
Public Health Foundation of India
PHD House, 2nd Floor
4/2, Siri Fort Institutional Area
August Kranti Marg
New Delhi-110 029

Dr.Subhadra Seshadri
H.No.246, 5th Cross
RMV Extension, 2nd Stage,
2nd Block, Bangalore-560 094

Dr. Umesh Kapil
Professor, Public Health Nutrition
All India Institute of Medical Sciences
Ansari Nagar, New Delhi-110 029

Dr.Veena Shatrugna
Former Dy. Director, NIN
Tarnaka, Hyderabad-500 604

Dr.Vijayaraghavan K
Former Dy. Director (Sr. Grade), NIN
B 1/1, Kakateeyanagar,
1-2-99/B, Habsiguda
Hyderabad-500007

Dr. Vinodini Reddy
Former Director, NIN
Flat No. 503, Mount Meru Apt.
Road No.5, Banjara Hills
Hyderabad – 500 034

RESOURCE PERSONS

Dr. Brahmam G.N.V
Scientist F
National Institute of Nutrition
Hyderabad-500 604

Dr. Hanumantha Rao D
Former Dy. Director, NIN
C-12, Kakateeyangar, Habsiguda,
Hyderabad-500 007

Dr. Leela Raman
Former Dy. Director (Sr.Gr.), NIN
H.No.16, Shruti, Vayupuri
Secunderabad- 500 094

Ms. Little Flower Augustine
Senior Research Fellow (UGC)
Micronutrient Research
National Institute of Nutrition
Hyderabad- 500 007

Dr. Ramesh V Bhat
Former Dy. Director (Sr.Gr.), NIN
M-11, Kakateeyanagar,
Street No.3, Habshiguda,
Hyderabad-500 007

Dr. Raghu Ram T.C
Former Dy. Director (Sr.Gr.), NIN
H.No.26, Motilal Nagar
Begumpet, Hyderabad-500 016

Dr. Raghuramulu N
Former Dy. Director (Sr.Gr.), NIN
2-16-94, Road No.2,
Prashanthinagar
Uppal, Hyderabad-500 039

Dr. Sarma K.V.R
Former Dy. Director (Sr.Gr.), NIN
1-2-99/A., Kakateeyanagar,
Street No.2, Habsiguda
Hyderabad-500 007

Dr. Susheela A.K
Director
Fluorosis Research &
Rural Development Foundation
C-13, Qutab Institutional Area
New Delhi–110 016

Dr. Sushma Kashyap
Reader
Dept. of Foods and Nutrition
Lady Irwin College
Sikandra Road, New Delhi-110 001

Dr. Vajreswari A
Scientist F
National Institute of Nutrition
Hyderabad-500 007

1. INTRODUCTION

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 1944 (1.1). This followed the recommendations made by the Technical Committee of the Health Committee, League of Nations in 1936 (1.2), Food and Nutrition Board of the National Research Council, USA, 1944 (1.3) and Report of the Committee of Nutrition, British Medical Association 1933 (1.4). At that time, 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 man of 55 kg and a normal adult woman of 45 kg body weight (1.5). 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, being a country in developmental transition, faces the dual burden of 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. According to recent National Family Health Survey (1.6) and UNICEF reports (1.7), 46% of preschool children and 30% of adults in India suffer from moderate and severe grades of protein-calorie malnutrition as judged by anthropometric indicators. Currently, India is in nutrition transition with 10% rural adults and 20% urban adults suffering from overnutrition, leading to an emerging double burden of malnutrition (1.8).

Though severe clinical forms of PCM – kwashiorkor and marasmus have become rare, they persist in some less developed states like Uttar Pradesh and Orissa. 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 recent survey shows that milder grades of deficiency as judged by clinical signs like night blindness and Bitot spots and low serum vitamin A levels, are common (1.9). Deficiencies of other micronutrients like some B-complex vitamins particularly riboflavin, folic acid and perhaps vitamin B_{12} 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_2 exists in adults. This, besides low intake of calcium, may be

responsible for the high prevalence of osteoporosis particularly in women. Recently, ICMR conducted a Task Force Study on prevalence of osteoporosis in India. 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 implementation infirmities, 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 (1.10, 1.11) and protein (1.12) 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 time (1.13). In 1968, the requirements of all nutrients except energy were reviewed by an Expert Committee constituted by ICMR (1.14). In arriving at these new recommendations, the international data provided by the FAO/WHO Expert Group and those generated by then in India, were used. 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 revised (1.15). 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 1985 (1.16). In arriving at human energy requirement, this International Expert Group followed an entirely new set of guidelines.

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. This Indian Expert Group, while following the new guidelines of the Joint FAO/WHO/UNU Consultative Group of 1985(1.16), also considered the updated data on Indians that had accumulated after 1973 (1.17), to define the energy and protein requirements of Indians. This 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. This Expert Group included in its recommendations, several additional nutrients such as dietary fiber, electrolytes, 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 made

(1.18). The reference body weights of normal healthy adult man, woman and children were also altered based on body weight data on healthy normal adults and children then obtained by National Institute of Nutrition (NIN) (1.19, 1.20).

The FAO/WHO/UNU Expert Consultation considered the revision of human nutrient requirements again after 2000. One Committee revised the requirement of micronutrients in 2001 (1.21), energy in 2004 (1.22) and protein in 2007 (1.23). 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.

It is more than 15 years since nutrient requirements and RDA was recommended for Indians. In the meantime, there has been much change in the concept of human energy requirements based on actual measurements (double isotopic ratio methods) and the requirement of several micronutrients has also been reconsidered in the recent past. In view of these international developments, ICMR constituted an Expert Group to revise and upgrade the earlier RDA of nutrients for Indians.

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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 (2-3 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.

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 are being 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 retention consistent with satisfactory growth in infants and children, for satisfactory maternal and foetal growth during pregnancy, satisfactory output of breast milk 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 requirement (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 of turnover of nutrients in healthy persons, using isotopically labeled nutrients are employed in arriving at the requirement of certain nutrients. Requirements of vitamin A (2.1), vitamin C (2.2), iron (2.3) and vitamin B_{12} (2.4) have been determined employing this approach. Earlier, radioactive isotopes were used and currently stable isotopes, which are safer, are being increasingly used to determine the turnover of nutrients in the body (2.5). 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 B_1) riboflavin (vitamin B_2), and pyridoxine (vitamin B_6) 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.

Nutrient Requirement and Recommended Dietary Allowances (RDA)

Internationally used definitions (2.6)

Dietary standards, regardless of the name they go by – Recommended Dietary Allowances, Recommended Nutrient Intakes, Recommended Daily Amounts of Nutrients, or Safe Intakes of Nutrients – are the average daily amounts of essential nutrients estimated, on the basis of available scientific knowledge, to be sufficiently high to meet the physiological needs of practically all healthy persons in a group with specified characteristics.

Some internationally used terminologies are given below. The term Average Nutrient Requirement (ANR) refers to nutrient values that cover 50% of the population. ANR + 2SD would cover 98% of the population and refers to terms like RDI, RDA and Reference Nutrient Intake (RNI). In Korea, ANR is referred to as Estimated Average Requirement (EAR). For nutrients where scientific evidence is not available, the term average intake is used.

Recommended Dietary Allowance (RDA): The average daily dietary nutrient intake level sufficient to meet the nutrient requirement of nearly all (97 to 98 percent) healthy individuals in a particular life stage and gender group.

Adequate Intake: a recommended average daily intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group (or groups) of apparently healthy people, that are assumed to be adequate — used when an RDA cannot be determined. In the Indian context, this is referred to as acceptable intake.

Tolerable Upper Intake Level (UL): the highest average daily nutrient intake level that is likely to pose no risk of adverse health effects for almost all individuals in the general population. As intake increases above the UL, the potential risk of adverse effects increases.

Estimated Average Requirement (EAR): the average daily nutrient intake level estimated to meet the requirement of half of the healthy individuals in a particular life stage and gender group.

The RDA is derived from (i) the individual variability, and (ii) the nutrient bio-availability from the habitual diet.

Individual variability: Definition of RDA takes into account the variability that exists in the requirement of a given nutrient between individuals in a given population group. The distribution of nutrient requirement in a population group is considered normal and the RDA corresponds to a requirement, which covers most of the individuals (97.5%) in a given population. This corresponds to Mean + 2 SD. This is termed as a safe level of intake of a nutrient, that is, the chances of individuals having requirements above the RDA is only 2.5%. This principle is used in case of all nutrients except energy. In the case of energy, intakes either in excess or below, the actual requirement of energy are not safe. In respect of other nutrients, the RDA is 25% (+2SD) higher than the mean requirement, 12.5% being considered as the extent of individual variability in the requirements of all those nutrients.

Bioavailability: Bioavailability of a given nutrient from a diet, that is, the release of the nutrient from the food, its absorption in the intestine and bio-response have to be taken into account. It is the level of the nutrient that should be present in the diet to meet the requirement. This bioavailability factor is quite important in calcium and protein and trace elements like iron and zinc. In the case of iron, the amount to be present in the diet is 20-30 times higher than the actual iron requirement to account for the low bio-availability of iron from a given diet, particularly a cereal-based diet.

RDA represents the level of the nutrient to be consumed daily to meet all the requirements of most of the individuals in a given population. However, it must be recognized that RDA is not meant to be used as standard to determine whether or not a given individual requirement has been met, since it is a level above the requirement of most individuals in a given population. RDA value of a nutrient is valid only when all other dietary nutrient intakes are satisfactory.

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of high income group from different parts of our country, so that reliable reference standards can be drawn up for heights and weights of Indian children. However, this has not been accomplished till today.

As of now, data on age/gender, specific centiles for heights and weights of a sufficiently large population of rural Indians based on NNMB surveys carried out during 2000-01 in the States of Kerala, Karnataka, Tamil Nadu, Andhra Pradesh, Maharashtra, Madhya Pradesh, Gujarat, Orissa and West Bengal are available. In addition, we have data from District Nutrition Surveys carried out in the States of Assam, Haryana, Himachal Pradesh, Punjab (1996), Orissa (2001), Uttar Pradesh (including Uttaranchal) (2002) and West Bengal (2001) (3.6 & 3.7). The above surveys covering 16 states in different regions of India, except for Jammu & Kashmir and north-eastern States have more or less an all-India character. The 95th centile values of weights and heights for given age/gender can be taken to be representative of well- nourished normal population and considered as standard reference values for India (Table 3.1 and Annexures 3.1 & 3.2). These weights can be used to compute the RDA of nutrients (per kg body weight/day), for all age and gender groups except for children (0-3 years).

WHO Standard weights and heights of infants and preschool children

World Health Organization has recently published multi-centre growth reference standards for 0-60 month boys and girls, based on studies carried out among predominantly exclusively breastfed children in six countries viz., USA, Brazil, Ghana, Norway, Oman and India. The median weights of infants and preschool children (1-3 years) can be taken as reference values for Indian children also (Table 3.2).

Reference Indian adult man and woman

Reference man is aged between 18-29 years and weighs 60 kg with a height of 1.73 m and a BMI of 20.3; is free from disease and physically fit for active work. On each working day, he is engaged in 8 hours of occupation which usually involves moderate activity; while when not at work he spends 8 hours in bed, 4-6 hours in sitting and moving about, 2 hours in walking and in active recreation or household duties.

Reference woman is aged between 18-29 years, non-pregnant non-lactating (NPNL) and weighs 55 kg with a height of 1.61m and a BMI of 21.2, is free from disease and physically fit for active work. On each working day she is engaged in 8 hours of occupation which usually involves moderate activity, while when not at work she spends 8 hours in bed, 4-6 hours in sitting and moving about, 2 hours in walking and in active recreation or household duties.

Table 3.1

95th Centile values of weight (kg), height (cm) and BMI by age and gender: rural India (16 States)

Males			Age (years)	Females		
Weight (kg)	Height (cm)	BMI		Weight (kg)	Height (cm)	BMI
11.2	82.4	16.5	**1+**	10.7	81.6	16.1
13.0	90.7	15.8	**2+**	12.6	89.8	15.6
14.8	99.1	15.1	**3+**	14.4	98.2	14.9
16.5	105.7	14.8	**4+**	16.0	105.1	14.5
18.2	111.5	14.6	**5+**	17.7	111.0	14.4
20.4	118.5	14.5	**6+**	20.0	117.5	14.5
22.7	124.3	14.7	**7+**	22.3	123.6	14.6
25.2	130.1	14.9	**8+**	25.0	129.2	15.0
28.0	134.6	15.5	**9+**	27.6	135.0	15.1
30.8	140.0	15.7	**10+**	31.2	140.0	15.9
34.1	144.8	16.3	**11+**	34.8	145.3	16.5
38.0	151.1	16.6	**12+**	39.0	150.2	17.3
43.3	157.0	17.6	**13+**	43.4	153.8	18.3
48.0	163.0	18.1	**14+**	47.1	157.0	19.1
51.5	166.3	18.6	**15+**	49.4	158.8	19.6
54.3	168.3	19.2	**16+**	51.3	159.7	20.1
56.5	170.0	19.6	**17+**	52.8	160.2	20.6
58.4	171.3	19.9	**18-19**	53.8	161.1	20.7
60.5	172.5	20.3	**20-24**	54.8	160.7	21.2
62.0	172.3	20.9	**25-29**	56.1	161.0	21.6

References 3.6 and 3.7

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3. REFERENCE BODY WEIGHTS

Age, gender and body weight largely determine the nutrient requirement of an individual. Body weights and heights of children reflect their state of health, nutrition and growth rate, while weights and heights of adults represent what can be attained by an individual with normal growth. Anthropometric measurements of infants and children of well-to-do families having access to good health care and with no nutritional constraints are usually treated as reference values. The anthropometric parameters of most people in developed countries correspond to NCHS Standards of US and the reference standards of UK and other Western European countries.

The purpose of recommending nutrient requirements is to help in attaining these anthropometric reference standards. International Organizations like WHO have proposed reference standards applicable at the international level.

On the other hand, in developing countries where most people are affected by poverty and dietary constraints, meeting the nutrient requirements becomes a challenge. A majority of the population hence do not attain anthropometric measurements corresponding to reference standards, which forms the basis for recommending dietary allowances of nutrients. Normal anthropometric standards for population groups differ from country to country. Ideally, each country has to set up its own reference standards, since heights and weights of their population may be genetically determined. Members of well-to-do, elite population with no nutritional constraints and with good health care have to be identified and anthropometric measurements of that select population have to be collected to set up local reference standards. This may be a difficult task and most of the anthropometric surveys to identify nutritional and health problems cover overall population who might be suffering from several dietary constraints and show relatively lower anthropometric values (3.1). These measurements, therefore, cannot be used as standards for recommending dietary allowance of nutrients, since the objective of RDA is to aim at population with standard anthropometry. The same was realized while arriving at RDA in 1989 (3.2).

The Expert Committee of the ICMR (1989) used anthropometric data of elite population of India. These data were generated by NIN surveys on well-to-do children, those studying in public schools or in IITs in different parts of the country (3.3, 3.4). Although these anthropometric data were collected from well-to-do Indian children and were comparable to Western counterparts, they were still representing a segment of Indian population and did not have an all-India character. The Expert Committee recommended that

of high income group from different parts of our country, so that reliable reference standards can be drawn up for heights and weights of Indian children. However, this has not been accomplished till today.

As of now, data on age/gender, specific centiles for heights and weights of a sufficiently large population of rural Indians based on NNMB surveys carried out during 2000-01 in the States of Kerala, Karnataka, Tamil Nadu, Andhra Pradesh, Maharashtra, Madhya Pradesh, Gujarat, Orissa and West Bengal are available. In addition, we have data from District Nutrition Surveys carried out in the States of Assam, Haryana, Himachal Pradesh, Punjab (1996), Orissa (2001), Uttar Pradesh (including Uttaranchal) (2002) and West Bengal (2001) (3.6 & 3.7). The above surveys covering 16 states in different regions of India, except for Jammu & Kashmir and north-eastern States have more or less an all-India character. The 95th centile values of weights and heights for given age/gender can be taken to be representative of well- nourished normal population and considered as standard reference values for India (Table 3.1 and Annexures 3.1 & 3.2). These weights can be used to compute the RDA of nutrients (per kg body weight/day), for all age and gender groups except for children (0-3 years).

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16.5	105.7	14.8	**4+**	16.0	105.1	14.5
18.2	111.5	14.6	**5+**	17.7	111.0	14.4
20.4	118.5	14.5	**6+**	20.0	117.5	14.5
22.7	124.3	14.7	**7+**	22.3	123.6	14.6
25.2	130.1	14.9	**8+**	25.0	129.2	15.0
28.0	134.6	15.5	**9+**	27.6	135.0	15.1
30.8	140.0	15.7	**10+**	31.2	140.0	15.9
34.1	144.8	16.3	**11+**	34.8	145.3	16.5
38.0	151.1	16.6	**12+**	39.0	150.2	17.3
43.3	157.0	17.6	**13+**	43.4	153.8	18.3
48.0	163.0	18.1	**14+**	47.1	157.0	19.1
51.5	166.3	18.6	**15+**	49.4	158.8	19.6
54.3	168.3	19.2	**16+**	51.3	159.7	20.1
56.5	170.0	19.6	**17+**	52.8	160.2	20.6
58.4	171.3	19.9	**18-19**	53.8	161.1	20.7
60.5	172.5	20.3	**20-24**	54.8	160.7	21.2
62.0	172.3	20.9	**25-29**	56.1	161.0	21.6

References 3.6 and 3.7

Table 3.2

Median weights and lengths of 0-60 months children: WHO revised reference values (MGRS 2006)

Boys		Age in Months	Girls	
Weight (kg)	Length (cm)		Weight (kg)	Length (cm)
3.3	49.9	**0**	3.2	49.1
4.5	54.7	**1**	4.2	53.7
5.6	58.4	**2**	5.1	57.1
6.4	61.4	**3**	5.8	59.8
7.0	63.9	**4**	6.4	62.1
7.5	65.9	**5**	6.9	64.0
7.9	67.6	**6**	7.3	65.7
8.3	69.2	**7**	7.6	67.3
8.6	70.6	**8**	7.9	68.7
8.9	72.0	**9**	8.2	70.1
9.2	73.3	**10**	8.5	71.5
9.4	74.5	**11**	8.7	72.8
9.6	75.7	**12**	8.9	74.0
10.9	82.3	**18**	10.2	80.7
12.2	87.8	**24**	11.5	86.4
13.3	91.9	**30**	12.7	90.7
14.3	96.1	**36**	13.9	95.1
15.3	99.9	**42**	15.0	99.0
16.3	103.3	**48**	16.1	102.7
17.3	106.7	**54**	17.2	106.2
18.3	110.0	**60**	18.2	109.4

Reference 3.5

Table 3.3

Reference body weights of Indians employed for computing RDA, 2010

Group	Age	Reference body weight (kg)
Adult men	18-29 y	60.0
Adult women (NPNL)	18-29 y	55.0
Infants	0 - 6 m	5.4*
	6 – 12 m	8.4*
Children	1 – 3 y	12.9*
	4 – 6 y	18.0
	7 – 9 y	25.1
Boys	10 - 12 y	34.3
	13 – 15 y	47.6
	16 – 17 y	55.4
Girls	10 – 12 y	35.0
	13 – 15 y	46.6
	16 – 17 y	52.1

References *3.5, 3.6, 3.7

The anthropometric values mentioned here are derived from Tables 3.1 and 3.2 for the age group 18-29y for both the genders. The reference body weights employed for computing the revised RDA are given in Table 3.3. The method adopted for computing reference body weights for different age categories is as follows:

Infants

The Committee decided to retain the approach adopted by the previous committee and used the average of birth weight and body weight at 6 months for computing the reference body weights of infants (0-6months). For 6-12 months, an average of body weights at 6 months and at 12 months was adopted.

Children

For children 1-3y, an average of bodyweights at 18m, 30m and 42m of WHO/MGRS median weights was taken.

Since WHO/MGRS data cover children till the age group of 1-5y, and the age category for computing RDA is 4-6y, it was decided to obtain the body weights from NNMB/India Nutrition Profile data from 4-6y onwards. Therefore, the reference body weights for children of 4-6y were obtained by

averaging the body weights of 4+, 5+ and 6+ years. Similarly for other age groups also the reference body were obtained from the 95th centile values of body weights of rural India (Annexures 3.1 & 3.2).

Adults

The average of values for age category of 18-19, 20-24 and 25-29 years was used (Table 3.1) for computing the reference body weights for adult man and woman.

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Annexure 3.1

Mean, median and 95th centile values of weight (kg) by age and gender: rural India (16 States)

Males				Age	Females			
Mean	SD	Median	95th	(years)	Mean	SD	Median	95th
8.6	1.5	8.5	11.2	**1+**	8.1	1.5	8.0	10.7
10.3	1.6	10.3	13.0	**2+**	9.9	1.6	9.9	12.6
11.9	1.7	12.0	14.8	**3+**	11.5	1.7	11.5	14.4
13.3	1.9	13.3	16.5	**4+**	12.9	1.8	13.0	16.0
14.8	2.0	14.8	18.2	**5+**	14.3	2.0	14.3	17.7
16.3	2.4	16.2	20.4	**6+**	15.8	2.3	15.7	20.0
18.2	2.7	18.0	22.7	**7+**	17.7	2.7	17.5	22.3
20.1	3.1	20.0	25.2	**8+**	19.7	3.1	19.7	25.0
22.2	3.4	22.0	28.0	**9+**	21.9	3.5	21.5	27.6
24.3	4.0	24.0	30.8	**10+**	24.1	4.1	23.8	31.2
26.4	4.4	26.0	34.1	**11+**	26.6	4.7	26.3	34.8
29.2	5.2	28.7	38.0	**12+**	29.6	5.4	29.4	39.0
32.6	6.0	32.1	43.3	**13+**	33.6	5.8	33.5	43.4
36.7	6.6	36.4	48.0	**14+**	37.2	6.0	37.2	47.1
41.1	6.6	41.2	51.5	**15+**	39.8	5.8	40.0	49.4
44.2	6.3	44.4	54.3	**16+**	42.0	5.7	42.0	51.3
47.1	5.8	47.5	56.5	**17+**	43.2	5.5	42.9	52.8
48.9	5.7	49.2	58.4	**18-19**	44.1	5.7	44.0	53.8
50.8	5.8	50.8	60.5	**20-24**	44.5	5.9	44.4	54.8
51.5	6.1	51.4	62.0	**25-29**	44.8	6.4	44.5	56.1
52.1	6.5	52.0	63.5	**30-34**	45.4	7.0	45.0	58.2
52.3	7.1	52.0	65.2	**35-39**	45.9	7.7	45.2	60.1
52.6	7.6	52.1	66.0	**40-44**	46.2	8.2	45.3	61.3
52.2	7.8	51.7	66.2	**45-49**	45.8	8.5	45.0	61.8
51.6	8.0	50.5	66.6	**50-54**	45.2	8.6	44.4	61.0
50.6	8.0	50.0	65.7	**55-59**	44.1	8.4	43.0	59.7
49.3	7.8	48.6	64.1	**60-64**	42.9	8.5	41.8	60.0
48.4	7.9	47.7	63.4	**65-69**	42.6	8.6	41.0	59.4
48.2	8.1	47.6	62.8	**70-74**	41.3	8.2	40.0	57.8
47.7	8.6	46.9	64.4	**75-79**	41.1	8.6	40.0	56.2
47.4	8.5	47.0	62.4	**80-84**	40.8	8.2	39.8	56.8
46.2	7.8	45.2	60.7	**≥ 85**	40.1	8.1	39.4	57.3

Annexure 3.2

Mean, median and 95th centile values of height (cm) by age and gender: rural India (16 States)

Males				Age	Females			
Mean	-SD	Median	95th	(years)	Mean	SD	Median	95th
73.7	5.3	73.7	82.4	**1+**	72.5	5.5	72.4	81.6
81.7	5.6	81.9	90.7	**2+**	80.6	5.7	80.7	89.8
88.9	6.3	89.0	99.1	**3+**	87.7	6.3	87.7	98.2
95.2	6.5	95.3	105.7	**4+**	94.4	6.7	94.8	105.1
101.1	6.7	101.3	111.5	**5+**	100.3	6.8	100.5	111.0
107.0	7.1	107.1	118.5	**6+**	106.2	7.0	106.2	117.5
113.4	7.0	113.3	124.3	**7+**	112.4	7.0	112.3	123.6
118.6	7.2	118.7	130.1	**8+**	118.0	7.1	118.4	129.2
123.8	6.9	124.0	134.6	**9+**	123.4	7.1	123.6	135.0
128.2	7.4	128.5	140.0	**10+**	127.9	7.5	128.2	140.0
132.7	7.3	132.7	144.8	**11+**	132.7	7.8	132.8	145.3
137.4	8.1	137.4	151.1	**12+**	137.6	7.9	137.9	150.2
142.7	8.3	142.8	157.0	**13+**	142.7	7.3	143.2	153.8
148.5	8.8	148.9	163.0	**14+**	146.4	6.7	146.8	157.0
153.8	8.2	154.1	166.3	**15+**	148.5	6.3	148.7	158.8
156.9	7.4	157.6	168.3	**16+**	150.0	6.0	150.1	159.7
159.7	6.9	160.1	170.0	**17+**	150.6	5.7	150.3	160.2
161.4	6.5	161.8	171.3	**18-19**	151.3	5.8	151.3	161.1
162.5	6.3	162.5	172.5	**20-24**	151.3	5.6	151.2	160.7
162.5	6.0	162.5	172.3	**25-29**	151.4	5.6	151.3	161.0
162.4	6.0	162.5	172.3	**30-34**	151.3	5.6	151.2	160.5
162.4	6.1	162.5	172.3	**35-39**	151.4	5.7	151.3	161.0
162.3	6.0	162.5	172.3	**40-44**	151.2	5.6	151.2	160.6
162.2	6.1	162.4	172.1	**45-49**	150.8	5.7	150.7	160.3
161.9	6.2	162.1	171.8	**50-54**	150.3	5.9	150.1	160.2
161.4	6.3	161.6	171.4	**55-59**	149.7	5.9	149.6	159.6
160.9	6.3	161.1	171.4	**60-64**	149.2	6.0	149.1	159.2
160.7	6.6	160.9	171.0	**65-69**	148.6	6.4	148.6	159.2
160.7	6.7	160.8	171.4	**70-74**	148.2	6.4	148.3	158.5
160.2	6.7	160.5	170.3	**75-79**	147.0	6.9	147.6	158.9
159.6	7.2	160.1	170.5	**80-84**	146.8	6.4	147.0	157.0
159.5	7.0	159.3	169.9	**≥ 85**	146.5	7.0	146.3	158.6

4. ENERGY

INTRODUCTION

Body needs energy for maintaining body temperature and metabolic activity and for supporting physical work and growth. The energy allowances recommended are designed to provide enough energy to promote satisfactory growth in infants and children and to maintain constant appropriate body weight and good health in adults. The factors which influence energy needs are age, body size, physical activity and, to some extent, climate and altered physiological status such as pregnancy and lactation.

To maintain energy balance, input must equal the output, which corresponds to a steady state. The logical extension of this concept is that if 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 (say pregnancy and lactation) will be taken care of by specific additional intakes. 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 man should be in energy balance on a day-to-day basis. However, over a period of a week or a fortnight, he can be in energy balance, that is, his 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.

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, dietary allowance of protein and other nutrients is the safe allowance, which covers additional allowances demanded by intra-individual and inter-individual variations. In the case of energy, however, RDA represents only the average daily requirements corresponding to daily average energy expenditure of an individual.

4.1. Units of energy

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 1meter by a force of 1 Newton acting on it (One Newton is the force needed to accelerate one kg mass by 1 meter per sec^2).

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. Definition of energy requirement

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 or secretion of milk at rates consistent with good health (4.1).

4.3. Assessment of energy requirements: current approach

Currently, it is recommended that energy requirement must be assessed in terms of energy expenditure rather than in terms of energy intake. Energy intake may vary from day to day; on some days, it may be above the energy expenditure and sometimes, below it. Body energy reserves (viz., 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.

The importance of using energy expenditure to arrive at an estimate of energy requirement cannot be over emphasized. An analysis of energy intake data is not helpful since it is possible to have a grossly inadequate intake by individuals who, for maintaining normal obligatory energy expenditure, lose weight and become substantially underweight. This is particularly so among large population groups in underdeveloped and developing countries. Such populations may be able to maintain their body weight at a low level by reducing the metabolic activity of their body tissues. On the other hand, energy intake far 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 where plenty of food is available.

Recommended dietary intake for energy is intended for a healthy, well nourished and active population. Assessment of energy expenditure is therefore a more 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 and 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 made in the case of energy but only the **Average** requirement is defined.

4.4. Requirements of energy for Indians

The recommended dietary allowances of energy for Indians were first proposed by the Nutrition Advisory Committee of the Indian Research Fund Association (IRFA) in 1944 (4.2). 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 Indians (4.2) were revised subsequently by the Nutrition Advisory Committee (NAC) of the ICMR in 1958 (4.3). The factorial approach 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 Requirements (4.4). For this purpose the normal man and woman were defined as having 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 and activity break-up approach. These energy

requirements of Indian adult man were computed using both Indian and Western data. While the available data on BMR (4.5, 4.6) of Indians were used for computing the energy cost of sleep, the activity component from different daily activities was however, computed from Western data on energy cost of different activities which were extensive, 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 requirements (4.3).

The 1958 RDA for energy for Indians suffers from a number of shortcomings. Dr.V.N.Patwardhan, the author of the 1958 Report on Energy Requirements of Indians (4.3) 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 calorie 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 appears to be rather high due to the use of higher values for some of the non-occupational activities; no distinction has been made in this respect between sedentary individual 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 appears to be an overestimation. This is because the average energy cost of heavy activity used in the computation of energy expenditure (i.e.) 5 kcal/kg/h corresponds 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 miners, it cannot be higher than 4.5 kcal/kg/h (4.7). It is also reported that the sustained activity involving heavy work can be carried out only at 35% VO_2 max (4.8).

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 digging, manual agricultural labour, stone cutters etc.

Further, the energy requirement recommended in 1958 for heavy work is not borne out by subsequent observation on Indian stone cutters (4.9).

(iii) The 1958 computation of daily energy requirement assumes that the level of daily activity is 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) (4.4) suggested energy requirement in terms of average for a year.

Considerable additional information and newer approaches for deriving energy requirements have emerged after 1980. Revised approaches have been used in 1985 (4.10) and 2004 (4.11) by FAO/WHO/UNU. Expert Consultants of ICMR Expert Group revised energy requirement for Indians in 1989 (4.12).

In 1989, the ICMR Expert Group adopted the procedure of 1985 FAO/WHO/UNU Expert Consultations (4.10) that is, using BMR factors for arriving at the energy requirements of Indian Man and Woman. 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 in terms of PAR values is quite obvious.

(a) PAR values for activities performed in a day can be aggregated over that 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. 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 = (8x1) + (8x2) +(8x3) = 48 PAR-hours.

The PAL (for the day) = Total PAR-hours / Total time = 48/24 = 2.0

(b) A large proportion of the daily energy expenditure is accounted for by the Basal Metabolism.

(c) A detailed table of PAR values for different activities is available in the FAO/WHO/UNU 2004 report (4.11).

(d) The energy expenditure for specific tasks when expressed as ratio of BMR as PAR values is similar in men and women and individuals with different body weights and ages.

(e) There are more extensive data on BMR of different population groups than on energy cost of activities.

As shown in Table 4.1, a close comparison of work expressed in PAR values between Indian and international data suggests that international data on different types of activities expressed in PAR values can be employed to compute energy expenditure of Indians using their basal metabolism values.

Table 4.1

Comparison of energy cost of some common daily activities in terms of PAR values

Activities	Energy cost of daily activities in PAR values	
	Indian data	International data
Sitting quietly	1.20	1.25
Standing quietly	1.40	1.33
Sitting at desk	1.30	1.36
Standing + doing lab. work	2.0	1.95
Harvesting	3.6	3.5
Hand saw	7.4	7.5
Typing (sitting)	1.58	1.69
Walking 3 MPH	3.71	3.77

The available data on energy cost of certain activities, when expressed as PAR, compare well with data obtained from other population groups.

(f) Expressing daily energy requirements in terms of PAL values would automatically take care of the problem of lower BMR of certain population groups such as Indians.

(g) The average energy requirement of Indians can readily be computed by using international data on daily energy requirements in terms of PAL values based on different daily activities, if BMR and the type of habitual activity and time spent on it are known.

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 2001 (4.11). However, this expert consultation has 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 here briefly. These methods are used to measure total energy expenditure over a 24-hr period including the metabolic response to food and the energy cost of tissue synthesis for adults, which is equivalent to daily energy requirements. 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 and from the average volume and composition of breast milk.

4.5. Heart rate monitor (HRM) method

Several investigations on total energy expenditure (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. 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.

4.6. Doubly labelled water (DLW) method

The use of the doubly labelled water (DLW) i.e. $^{2}H_{2}{}^{18}O$ 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 animals (4.13). Its application was later validated in humans (4.14). 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 (4.15, 4.16; Table 4.2).

Table 4.2

DLW studies performed in India

Source	Group studied	n	BMI	TEE (MJ/d)	PAL
Reference 4.15	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
Reference 4.16	8-9 y boys Mysore	30	14.4	6.1	1.4
	8-9 y girls Mysore	28	14.9	5.6	1.4

The limitation of this method is the cost and availability of stable isotope labelled water $^2H_2O_{18}$. Many studies are available in western population, but only one validated study has been done on free-living Indian adults, with simultaneous validation of DLW method against whole body calorimeter (WBC) (4.15). This study has been conducted in three groups of subjects, well nourished, upper socio-economic students, undernourished slum dwellers and young rural farmers (Table 4.3). 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 adults. Since there are larger data sets 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 4.3

TEE measured by the DLW and WBC method along with BMR and BMI

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

Reference 4.16
Values are Mean ± SD
BMR – Basal Metabolic Rate
TEE- Total energy expenditure
DLW – Doubly labelled water method
PAL – Physical activity level

4.7. Energy requirements of infants and children

4.7.1. Energy requirements of infants

The 1988 ICMR Expert Group and the earlier Expert Groups had adopted FAO/WHO/UNU recommendations on energy requirement per kg for Indian infants also. The earlier International expert groups had adopted the energy intakes of normal infants from the industrial countries as their energy requirement. The 2001 Consultations of FAO/WHO/UNU have however derived the energy requirement of infants on the basis of DLW method and to this, the energy acquisition due to growth is added to arrive at the total daily energy requirement. This Consultation Group has used the body weights of infants from 0-12 months representing both the industrial countries and the developing countries.

The same values can be used for the energy requirements of Indian infants also as there are no separate studies on energy requirements of Indian infants using the DLW method or the HRM method. The equation to

derive energy requirement of infants as reported by FAO/WHO/UNU (2004) is as follows:

TEE (MJ/d) = -0.416 + 0.371 kg
n=320, r=0.85 and
Standard error of estimate = 0.456 MJ/d

TEE (kcal/d) = -99.4 + 88.6 kg
Standard error of estimate = 109 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 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 4.4 and 4.5.

If data are available for normal healthy Indian infants, another set of figures of energy requirement of Indian children can be computed using the equation given by FAO/WHO/UNU (4.11).

The current estimate of energy requirement of infants is 11-20% lower than the 1988 estimates.

Table 4.4
Protein, fat and energy deposition during growth in the first year of life

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

Reference 4.11

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

Table 4.5a
Energy requirements of infants during the first year of life
(Unit: MJ/day)

Age Months	Weight (kg)	Weight Gain (g/d)	Total energy expenditure[a] (MJ/d)	Energy deposition[b] (MJ/d)	Daily energy requirement[c] (MJ/d)	(KJ/kg /d)[d]
Boys						
0 - 1	4.58	35.2	1.282	0.884	2.17	470
1 - 2	5.50	30.4	1.623	0.764	2.39	430
2 - 3	6.28	23.2	1.912	0.582	2.49	400
3 - 4	6.94	19.1	2.157	0.224	2.38	340
4 - 5	7.48	16.1	2.357	0.189	2.55	340
5 - 6	7.93	12.8	2.524	0.150	2.67	340
6 - 7	8.30	11.0	2.661	0.069	2.73	330
7 - 8	8.62	10.4	2.780	0.065	2.85	330
8 - 9	8.89	9.0	2.880	0.057	2.94	330
9 - 10	9.13	7.9	2.969	0.089	3.06	340
10 - 11	9.37	7.7	3.058	0.087	3.15	340
11 - 12	9.62	8.2	3.150	0.093	3.24	340
Girls						
0 - 1	4.35	28.3	1.197	0.746	1.94	450
1 - 2	5.14	25.5	1.490	0.672	2.16	420
2 - 3	5.82	21.2	1.742	0.559	2.30	400
3 - 4	6.41	18.4	1.960	0.285	2.25	350
4 - 5	6.92	15.5	2.149	0.239	2.39	350
5 - 6	7.35	12.8	2.309	0.199	2.51	340
6 - 7	7.71	11.0	2.442	0.083	2.53	330
7 - 8	8.03	9.2	2.561	0.069	2.63	330
8 - 9	8.31	8.4	2.665	0.063	2.73	330
9 - 10	8.55	7.7	2.754	0.074	2.83	330
10 - 11	8.78	6.6	2.839	0.063	2.90	330
11 - 12	9.00	6.3	2.920	0.060	2.98	330

Reference 4.11

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

[a] TEE (MJ/d) = -0.416 + 0.371 kg

[b] Weight gain x energy accrued in normal growth (Table 4.4)

[c] Requirement = total energy expenditure + energy deposition.

[d] Rounded off to the nearest 10 KJ.

Table 4.5b

Energy requirements of infants during the first year of life (Unit: kcal/day)

Age Months	Weight (kg)	Weight Gain (g/d)	Total energy expenditure[a] (kcal/d)	Energy deposition[b] kcal/d	Daily energy requirement[c] kcal/d[d]	kcal/kg/d[e]
Boys						
0 - 1	4.58	35.2	306	211	520	115
1 - 2	5.50	30.4	388	183	570	105
2 - 3	6.28	23.2	457	139	600	95
3 - 4	6.94	19.1	515	53	570	80
4 - 5	7.48	16.1	565	45	610	80
5 - 6	7.93	12.8	603	36	640	80
6 - 7	8.30	11.0	636	17	650	80
7 - 8	8.62	10.4	664	16	680	80
8 - 9	8.89	9.0	688	14	700	80
9 - 10	9.13	7.9	710	21	730	80
10 - 11	9.37	7.7	731	21	750	80
11 - 12	9.62	8.2	753	22	780	80
Girls						
0 - 1	4.35	28.3	286	178	460	105
1 - 2	5.14	25.5	356	161	520	100
2 - 3	5.82	21.2	416	134	550	95
3 - 4	6.41	18.4	469	68	540	85
4 - 5	6.92	15.5	514	57	570	80
5 - 6	7.35	12.8	552	47	600	80
6 - 7	7.71	11.0	584	20	600	80
7 - 8	8.03	9.2	612	17	630	80
8 - 9	8.31	8.4	637	15	650	80
9 - 10	8.55	7.7	658	18	680	80
10 - 11	8.78	6.6	679	15	690	80
11 - 12	9.00	6.3	698	14	710	80

Reference 4.11

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.

[a] TEE (kcal/d) = -99.4+ 88.6kg

[b] Weight gain x energy accrued in normal growth (Table 4.4)

[c] Requirement = total energy expenditure + energy deposition.

[d] Rounded off to the nearest 10 kcal.

[e] Rounded off to the nearest 5 kcal.

4.7.2. Energy requirement of children

It has been found that the estimated energy requirement is lower than the value derived from food intake data for children 1-10 years, and thereafter, for adolescents, it is higher (4.11). The requirement is arrived at, through the estimation of energy expenditure (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.

It is reported (4.11) that energy requirement of children and adolescents were estimated using 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 : TEE kcal/day: $310.2 + 63.3\ kg - 0.263\ kg^2$
TEE MJ/day: $1.298 + 0.265\ kg - 0.0011\ kg^2$

Girls : TEE kcal/day: $263.4 + 65.3\ kg - 0.454\ kg^2$
TEE MJ/day: $1.102 + 0.273\ kg - 0.0019\ kg^2$

Boys : n: 801, $r=0.982$, $r^2 = 0.964$

Girls : n: 808, $r = 0.955$, $r^2 = 0.913$

Table 4.6 shows energy acquired during growth: energy equivalent for 1g protein = 5.65 K cal, 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 4.7a and 4.7b 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 evidence that the BMR of Indian children is lower than that of their Western counterparts (cf. adults where it has been found to be 5% lower), the FAO/WHO/UNU 2004 (4.11) BMR prediction equations were used to predict BMR, while TEE was predicted from the quadratic prediction equation given above (4.11).

Table 4.6

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

Age	Boys			Girls		
	Body Wt	Weight gain	kcal/day	Body Wt.	Weight gain	kcal/day
y	kg	kg/y		kg	kg/y	
1-2*	10.9	2.4	13.15	10.2*	2.5	13.70
2-3*	13.3	2.0	10.96	12.7*	2.3	12.60
3-4	15.3	1.7	9.32	15.0	1.6	8.77
4-5	16.5	1.7	9.32	16.0	1.7	9.32
5-6	18.2	2.2	12.05	17.7	2.3	12.60
6-7	20.4	2.3	12.60	20.0	2.3	12.60
7-8	22.7	2.5	13.70	22.3	2.7	14.79
8-9	25.2	2.8	15.34	25.0	2.6	14.25
9-10	28.0	2.8	15.34	27.6	3.6	19.73
10-11	30.8	3.3	18.08	31.2	3.6	19.73
11-12	34.1	3.9	21.37	34.8	4.2	23.01
12-13	38.0	5.3	29.04	39.0	4.4	24.11
13-14	43.3	4.7	25.75	43.4	3.7	20.27
14-15	48.0	3.5	19.18	47.1	2.3	12.60
15-16	51.5	2.8	15.34	49.4	1.9	10.41
16-17	54.3	2.2	12.05	51.3	1.5	8.22
17-18	56.5	1.5	8.22	52.8	0.6	3.29

Please refer *MGRS body weight in Table 3.2 and for the remaining body weights NNMB and India Nutrition Profile in Table 3.1

Energy cost of weight gain: 2 kcal/g weight gain (4.11)

It is clear from the Tables (4.7a and 4.7b) 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 4.7c depicts a typical lifestyle that would be required for an adolescent aged between 12-16 years, to have a PAL of 1.8.

Table 4.7a

Energy requirement and PAL of Indian children and adolescents (boys)

Age	Boys					
	Weight[a]	TEE[b]	PAL[c]	Energy cost of growth[d]	Total energy requirement[e]	Total energy requirement[f]
(y)	(kg)	(kcal/d)		(kcal/d)	(kcal/d)	(kcal/kg/d)
1-2	10.9	901.1[g]	1.46	13.15	910	85
2-3	13.3	1105.6	1.45	10.96	1120	85
3-4	15.3	1217.1	1.43	9.32	1230	80
4-5	16.5	1283.0	1.46	9.32	1290	80
5-6	18.2	1375.1	1.50	12.05	1390	80
6-7	20.4	1492.1	1.54	12.60	1510	75
7-8	22.7	1611.6	1.58	13.70	1630	70
8-9	25.2	1738.3	1.61	15.34	1750	70
9-10	28.0	1876.4	1.65	15.34	1890	70
10-11	30.8	2010.3	1.67	18.08	2030	65
11-12	34.1	2162.9	1.71	21.37	2180	65
12-13	38.0	2335.8	1.76	29.04	2370	60
13-14	43.3	2558.0	1.80	25.75	2580	60
14-15	48.0	2742.6	1.82	19.18	2760	60
15-16	51.5	2872.6	1.83	15.34	2890	55
16-17	54.3	2971.9	1.84	12.05	2980	55
17-18	56.5	3047.1	1.84	8.22	3060	55

Note: This Table reflects the energy requirement of boys who are healthy, have attained the 95th percentile of weight for age and have a moderate activity level.

[a] Weight from Table 4.6

[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 equations (4.11).

[d] From Table 4.6

[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 infants (4.11).

Table 4.7b

Energy requirement and PAL of Indian children and adolescents (girls)

Age	Girls					
	Weight[a]	TEE[b]	PAL[c]	Energy cost of growth[d]	Total energy requirement[e]	Total energy requirement[f]
(y)	(kg)	(kcal/d)		(kcal/d)	(kcal/d)	(kcal/kg/d)
1-2	10.2	820.5[g]	1.45	13.70	830	80
2-3	12.7	1019.5	1.44	12.60	1030	80
3-4	15.0	1140.8	1.44	8.77	1150	75
4-5	16.0	1192.0	1.47	9.32	1200	75
5-6	17.7	1277.0	1.51	12.60	1290	75
6-7	20.0	1387.8	1.56	12.60	1400	70
7-8	22.3	1493.8	1.59	14.79	1510	70
8-9	25.0	1612.2	1.62	14.25	1630	65
9-10	27.6	1719.8	1.64	19.73	1740	65
10-11	31.2	1858.8	1.67	19.73	1880	60
11-12	34.8	1986.0	1.71	23.01	2010	60
12-13	39.0	2119.6	1.75	24.11	2140	55
13-14	43.4	2242.3	1.76	20.27	2260	50
14-15	47.1	2331.9	1.76	12.60	2340	50
15-16	49.4	2381.3	1.76	10.41	2390	50
16-17	51.3	2418.5	1.78	8.22	2430	45
17-18	52.8	2445.6	1.80	3.29	2450	45

Note: This Table reflects the energy requirement of girls who are healthy, have attained the 95th percentile of weight for age and have a moderate activity level.

[a] Weight from Table 4.6

[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 equations (4.11).

[d] From Table 4.6

[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 infants (4.11).

Table 4.7c

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	9	9	9	9
School-related activities					
Walk to School	3	0	0	1	3
In class	1.5	6	9	6	9
Recess play time	3	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	2	6	2	6
Focused Games (football, basketball)	8	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 ref 4.11.

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.

To illustrate this point, actual measurements of TEE have been performed by the DLW method in Indian children aged 8-9 years (4.16). These values (in middle class South Indian children) were 1458 kcal/day and 1338 kcal/day in boys and girls respectively (Table 4.2). 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, (4.11) assumes a moderately active child as the reference. This means that for a child aged 8-9, PAL would be about 1.6 (Table 4.7), which is higher than what was observed in sedentary Indian children (4.16, Table 4.2). 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 4.7a and 4.7b, and that the activity should increase in the teens to a PAL of 1.8.

Table 4.7d below, gives some play activities and their PAR. These can be used as in Table 4.7c, to calculate PAL values in any given lifestyle pattern.

Table 4.7d

PAR values for some activities*

	Average PAR	
Activity	**Males**	**Females**
Sleeping	1.0	1.0
Sitting quietly	1.2	1.2
Reading	1.3	1.5
Standing	1.4	1.5
Dressing	2.4	3.3
Walking slowly	2.8	3.0
Walking briskly	3.8	3.8
Cycling	5.6	3.6
Running – sprint	8.2	8.3
Running- long distance	6.3	6.6
Basketball	7.0	7.7
Football	8.0	-
Swimming	9	-

Reference 4.11

* It must be stressed that these are average PAR values reported within a single study, or averaged across different studies. The range of reported values can be around greater than 10% on either side, especially at low PAR activities.

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 (Table 4.7), 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 4.7e and 4.7f give the requirements (including energy required for growth) for a child who is sedentary, as well as for children who are very active.

Table 4.7e

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[c]** (kcal/kg/d)	**PAL**
6-7	20.4	1270	60	1.3	1760	85	1.8
7-8	22.7	1340	60	1.3	1850	80	1.8
8-9	25.2	1530	60	1.4	2070	80	1.9
9-10	28.0	1610	60	1.4	2180	80	1.9
10-11	30.8	1700	55	1.4	2310	75	1.9
11-12	34.1	1920	55	1.5	2550	75	2.0
12-13	38.0	2020	55	1.5	2680	70	2.0
13-14	43.3	2160	50	1.5	3010	70	2.1
14-15	48.0	2280	50	1.5	3180	65	2.1
15-16	51.5	2530	50	1.6	3310	65	2.1
16-17	54.3	2600	50	1.6	3400	65	2.1
17-18	56.5	2660	45	1.6	3490	60	2.1

* To be studied in conjunction with Table 4.7a, 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 4.7a). Age and weights taken from Table 4.7a; 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 4.7c 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 practising 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 4.7f

Energy requirements and PAL of Indian girls 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[c] (kcal/kg/d)	PAL
6-7	20.0	1170	60	1.3	1610	80	1.8
7-8	22.3	1330	60	1.4	1710	75	1.8
8-9	25.0	1410	55	1.4	1910	75	1.9
9-10	27.6	1490	55	1.4	2010	75	1.9
10-11	31.2	1580	50	1.4	2140	70	1.9
11-12	34.8	1770	50	1.5	2350	65	2.0
12-13	39.0	1840	50	1.5	2450	65	2.0
13-14	43.4	1930	45	1.5	2570	60	2.0
14-15	47.1	2000	45	1.5	2660	55	2.0
15-16	49.4	2040	40	1.5	2720	55	2.0
16-17	51.3	2040	40	1.5	2730	55	2.0
17-18	52.8	2040	40	1.5	2860	55	2.1

* To be studied in conjunction with Table 4.7b, 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 4.7b). Age and weights taken from Table 4.7b; 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 4.7c 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

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 4.7a and 4.7b above, 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.

4.8. Energy requirements of adults

Energy requirements (i.e.) TEE of adults computed by DLW or HRM methods have been estimated on a large number of subjects mostly in European and North American countries. Some data have been collected in developing regions mostly in South American countries and some studies have been done in India too (4.15, 4.16).

Daily energy expenditure of adults depends on their occupational 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. It largely depends upon the body weight, from which the subjects' basal metabolism is predicted, and then to which energy spent during the activities of the day are related, as the PAL value. Therefore, TEE = Predicted BMR x PAL.

A set of equations relating to body weight of adults and their BMR was given by the 1985 recommendation of the FAO/WHO/UNU (4.10). A study on BMR of Indian subjects by Shetty and co-workers (4.18) indicated that BMR of Indians is 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 in1989 (4.12). It is proposed that the same set of equations as used by the previous ICMR Expert Group (4.12) can be retained in the present report.

Equations for predicting BMR proposed by FAO/WHO/UNU and those proposed for Indians are given in Table 4.8.

The BMR factors for occupational and non-occupational activities in the developing countries will be higher than in the industrialized Western Society. The ICMR Expert Group, 1989 (4.12) proposed the following factors while computing energy requirement of Indian adults. Further, the same factors were proposed for men and women as both are presumed to work with same intensity.

Table 4.8

Equations for prediction of BMR (kcal/24h): FAO/WHO/UNU

	Age (y)	Prediction equation		Correlation co-efficient	SD
		Proposed by FAO/WHO/UNU Consultation (1985)	Proposed by ICMR Expert Group for Indians (1989)		
Males	18-30	15.1xB.W.(kg)+692.2	14.5xB.W.(kg)+645	0.65	151
	30-60	11.5xB.W.(kg)+873	10.9xB.W.(kg)+833	0.60	164
	>60	11.7xB.W.(kg)+587.7	12.6xB.W.(kg)+463	0.79	146
Females	18-30	14.8xB.W.(kg)+486.6	14.0xB.W.(kg)+471	0.72	121
	30-60	8.1xB.W.(kg)+845.6	8.3xB.W.(kg)+788	0.70	108
	>60	9.1xB.W.(kg)+658.5	10.0xB.W.(kg)+565	0.74	108

References 4.10 & 4.12
BMR of Indians is 5% lower than the international values (FAO/WHO/UNU)
B.W. = Body weight

What was proposed during 1989 for heavy work was rather high as it reckoned around 8 hrs of heavy work with a 4.5 PAR value. It is difficult to maintain an activity with a PAR of 4.5 continuously for 8 h. Heavy work involving 4.5 PAR units is usually carried out only for 3-4 h and the rest of the time is spent in resting or performing lesser intense activity; therefore, on the average, the PAR value may be 3.8 units when considered for all 8 h. On this basis, the average PAL value for a day spent in doing such heavy work will be 2.3, while it will be 1.6 for sedentary and 1.9 for moderate activity. These values are similar to those obtained in free-living Indian adults, measured by the DLW method (4.15).

PAL values for different categories of work for Indian reference adult man and woman are given in Table 4.9 along with values recommended by FAO/WHO/UNU Consultation of 2002.

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 1996-97 (4.18) 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 4.9

PAL Values proposed by ICMR Expert Group (2009), ICMR 1989 compared to the figures proposed by FAO/WHO/UNU Consultation, 2004

Level of activity	ICMR 1989	ICMR 2010	FAO/WHO/UNU
Sedentary Work	1.6	1.53	1.40-1.69
Moderate Work	1.9	1.8	1.70 – 1.99
Heavy Work	2.5	2.3	2.0 – 2.40*

Reference 4.11 & 4.12
*PAL Value > 2.40 is difficult to maintain over a prolonged period.

4.9. Computation of energy requirement of Indian adults in terms of PAL units

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.

Factorial computation of energy expenditure by adult Indian population is given in Table 4.10.

Energy expenditure at the three levels of activity at different age groups and at different body weights are given in Annexure 4.1.

Table 4.10

Factorial computation of energy expenditure of adult Indian population

Main daily activities	Duration (h)	Major lifestyles, energy expenditure (PAR values)		
		Sedentary	Moderate active	Heavy or vigorously active
Sleep	8	1.0	1.0	1.0
Occupational activity	8	1.5	2.3	3.8
Non-occupational activity	8	2.1	2.1	2.1
Mean		**1.53**	**1.80**	**2.30**
Non-occupational activity details				
Personal care	1	2.3	2.3	2.3
Eating	1	1.5	1.5	1.5
Commuting to work by bus or by vehicle or by walk	1	2.0	2.0	2.0
General household or other activities	2	2.5	2.5	2.5
Walking at various speeds without load	1	3.2	3.2	3.2
Light leisure activity	2	1.4	1.4	1.4
Mean non-occupational activity	8	2.1	2.1	2.1
Men: body wt. (60kg), BMR (1515 kcal)	--	2318	2727	3485
Women: body wt (55kg), BMR (1241 kcal)	--	1899	2234	2854

4.10. Energy requirement during pregnancy

Energy requirement during pregnancy comprises the normal requirement for an adult woman and an additional requirement for foetal growth plus the associated increase in body weight of the woman during pregnancy, most of which occurs during the second and the third trimesters. The total energy requirement during pregnancy for a woman weighing 55 kg is estimated to be 80,000 kcal (4.19) of which 36,000 kcal is deposited as fat, which is utilized subsequently during lactation. A direct estimate of energy expenditure during normal pregnancy through indirect calorimetry is about 27,000 kcal (4.19). Based on these estimates, FAO/WHO Consultants during 1985 (4.10) recommended additional daily allowance of energy of 150 kcal/day during first trimester and 350 kcal/day during the second and the third trimesters.

The FAO/WHO/UNU 2004 Consultation on energy requirements, reviewed the recent data on maternal weight gain and foetal body weight and decided that a 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 body weight gain consisting of protein, fat and water. Protein is predominantly deposited in fetus (42%) but also in uterus (17%), blood (14%) placenta (10%) and breast (8%). 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 basal metabolic rate (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 respectively (4.11).

Energy requirement during pregnancy also depends upon any deviation from normal physical activity. Several studies in different societies 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 labour (4.20).

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 respectively.

The additional energy required during pregnancy (Table 4.11) can be estimated by the factorial method in two ways: either by assuming an increase in the TEE or in the BMR. When the estimate is made on the basis of the costs of protein and fat deposition as well as the increase in PAL & TEE, it is 76530 kcal. Alternatively, calculation of the additional energy required can be based on the increase in BMR (this also assumes that there is no change in the actual costs of activities) as well as the costs of protein and fat deposition (adjusted for the efficiency of energy utilization): this value is 77100 kcal. The average estimate of these two methods (depending either on using the increase in TEE or BMR) is about 77000 kcal, which is about 4% lower than the earlier estimate of 80,000 kcal made in 1985.

4.11. Energy requirement during pregnancy among Indian women

Additional energy required during pregnancy consists of firstly, increase in TEE during pregnancy, most of which occurs during 2nd and 3rd trimesters. This increase depends upon the pre-pregnancy body weight and pre-pregnancy BMR. The second component, contributing to the increase in BMR is due to tissue deposited during pregnancy as protein and fat in the fetus and the mother and depends upon the total weight gain 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 and NFHS 3 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.

Additional energy requirement of Indian women during pregnancy have been computed on the basis of the reference Indian women and pregnancy weight gain of 10 kg and 12 kg. Additional energy needed for tissue deposition will be 85 kcal, 280 kcal and 470 kcal during the three trimesters if the body weight gain is 12 kg. As is the practice, if only 2nd and 3rd trimesters are taken into consideration, the average energy requirement would be 375 kcal/day during that period. If the body weight gain is only 10 kg and assuming that body weight as well as increase in body weight has an influence on PAL, increase in energy requirement during the three trimesters would be 70 kcal, 230 kcal and 390 kcal or an average of 310 kcal during the 2nd and 3rd trimesters. Computation of additional energy needed during

pregnancy for an Indian woman of 55 kg pre-pregnancy body weight is given in Table 4.11.

Table 4.11
Additional energy cost of pregnancy with gestational weight gain of 12 kg

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

[a] Includes costs of efficiency of energy utilization
[b] Rounded off to nearest 5 kcal.
Figures within parentheses correspond to energy costs if gestational weight gain is 10 kg instead of 12 kg.

Based on the Table above, the following figures can be recommended as additional energy requirements of an Indian woman with pre-pregnancy weight of 55 kg. Note that the average value for the 2nd and 3rd trimesters is taken for a single recommendation value.

	12 kg increase	10 kg increase
1st trimester	85 kcal	70 kcal
2nd trimester	280	230
3rd trimester	470	390
During 2nd& 3rd trimesters	375 kcal	310 kcal

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 may be made.

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.

4.12. Energy cost of 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. 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. A study on energy expenditure of Indian lactating woman indicates a negative energy balance, which could be corrected by the fat deposited during pregnancy. The efficacy of utilization of milk energy is 80%, which has to be corrected. Milk output data collected from traditional countries can be used to compute energy cost of lactation of Indian women (Table 4.12).

Table 4.12

Energy expenditure during lactation in Indian women

Month postpartum	Mean milk output[a] (g/d)	Corrected output[b] (g/d)	Gross Energy content[c] (kcal/d)	Daily gross energy secreted (kcal/d)	Energy cost of milk production[d] (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 breast feeding					
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**

Reference 4.19
Insensible water losses assumed to be equal to 5% of milk intake.
2.8 KJ/g 0.67 kcal/g measured by macronutrient analysis
Based on energetic efficiency of 80%

Daily additional energy requirement of a woman doing exclusive breast feeding 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. This is in contrast to the value given in the ICMR 1989 Report where a recommendation of 550 kcal was made for the first six months and 400 kcal for the subsequent 6 months. A study of energy requirement of lactating women based on milk output and energy output computed from actual measurement showed that average energy utilization for average milk production of 624ml 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 4.13. The values reported on Indian women in NIN studies (4.19) are given in Table

Table 4.13

Milk production and energy utilization by Indian lactating women on whom total energy balance was conducted

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	2148.1	2228.9	666.7	553.4

Reference 4.19
[a] Subject unwell at times
[b] Only 2- month value, hence not considered

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 fruits (4.21).

Dietary fiber forms an indigestible and important component of plant foods and was never considered as sources of energy. But this 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

There is a need to recalculate energy field of various foods on the basis of their revised content of carbohydrates, proteins, fat and dietary fiber. Hitherto, dietary fiber was not determined directly.

Need to correct the carbohydrate content of foods by inclusion of total dietary fiber

The main source of energy in most of the Indian diets is carbohydrates derived largely from cereals present in them. These cereals constitute 80% of our diet and provide 50-80% of daily energy intake. However energy contribution from diets varies very widely. Those belonging to low income groups have only 5% fat in their diet whereas, affluent families derive as high as 30% of their dietary energy from fat. However, most families derive nearly 10-12% of energy from proteins.

There appears to be an overestimation of energy content of foods derived from plant sources in the diets of Indians. The carbohydrate content of foods listed in the Nutritive value of Indian foods (4.22) are not directly estimated but obtained by difference:

Carbohydrate content = 100 - moisture - protein - fat - minerals - crude fiber.

This computation does not take into account the total dietary fiber (15.7g) which are currently estimated to be nearly 10 times higher than crude fiber (1.6g) in many foods rich in carbohydrates: if total dietary fiber is considered in computing carbohydrate by difference, the carbohydrate content of cereals and other major foods would be 10-15% lower. Similar lower values for the carbohydrate content of cereals would be obtained if carbohydrates are directly estimated. If the lower values for carbohydrate and 2 kcal/g for dietary fiber content are included, the estimated energy content of foods particularly that of cereals would be lower. Corrected carbohydrate and energy content of some important carbohydrate sources of Indian foods are given in Table 7.3 (Chapter 7 on Fiber). This point to a need for estimating carbohydrate (starch+ sugar+ other oligosaccharides) as an energy source directly to be able to obtain an accurate estimate of our major food source.

4.13. Summary of recommended energy requirement for Indians

The final recommended energy levels at different ages are given in Table 4.14

Table 4.14

Energy requirements of Indians at different ages

Age Group	Category	Body weights	Requirement	
			(kcal/d)[a]	(kcal/kg/day)
Men	Sedentary work	60	2320	39
	Moderate work	60	2730	46
	Heavy work	60	3490	58
Women	Sedentary work	55	1900	35
	Moderate work	55	2230	41
	Heavy work	55	2850	52
	Pregnant	55 + GWG[b]	+ 350	
	Lactating	55 + WG[c]	+600 +520	
Infants	0-6 m	5.4	500	92
	6-12m	8.4	670	80
Children[d]	1-3y	12.9	1060	82
	4-6y	18.1	1350	75
	7-9 y	25.1	1690	67
Boys	10-12y	34.3	2190	64
Girls	10-12y	35.0	2010	57
Boys	13-15y	47.6	2750	58
Girls	13-15y	46.6	2330	50
Boys	16-17y	55.4	3020	55
Girls	16-17y	52.1	2440	47

[a] Rounded off to the nearest 10 kcal/d

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

[c] WG – Gestational Weight gain remaining after delivery

[d] Energy 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 4.7e), especially when computing the gap between energy requirement and actual intake that needs to be filled by supplementation programmes.

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Annexure 4.1

Energy Requirement of Indian Men and Women at Different Ages and Body Weights

Sex	Body Weight	18-30 y				30-60 y				> 60 y			
		BMR	Sedentary Work	Moderate Work	Heavy Work	BMR	Sedentary Work	Moderate Work	Heavy Work	BMR	Sedentary Work	Moderate Work
	kg	kcal	kcal	kcal	kcal	kcal	kcal	kcal	kcal	kcal	kcal	kcal
MALES	45	1298	1986	2336	2985	1324	2026	2383	3045	1039	1590	1870
	50	1370	2096	2466	3151	1358	2078	2444	3123	1103	1688	1985
	55	1443	2208	2597	3319	1433	2196	2579	3296	1167	1786	2101
	60	1515	2318	2727	3485	1487	2275	2671	3420	1231	1883	2216
	65	1588	2430	2858	3652	1542	2359	2736	3547	1295	1981	2331
	70	1660	2540	2988	3818	1596	2442	2873	3671	1359	2079	2446
	75	1773	2716	3191	4078	1651	2526	2971	3797	1423	2177	2565
FEMALES	40	1031	1577	1856	2371	1120	1714	2016	2576	965	1477	1737
	45	1101	1685	1982	2532	1162	1778	2092	2673	1015	1553	1627
	50	1171	1792	2108	2693	1203	1841	2165	2767	1065	1630	1917
	55	1241	1899	2234	2854	1245	1905	2241	2864	1115	1706	2007
	60	1311	2006	2360	3015	1286	1966	2315	2958	1165	1782	2097
	65	1381	2113	2486	3176	1328	2032	2390	3054	1215	1860	2187
	70	1451	2220	2612	3337	1369	2095	2464	3149	1265	1936	2277

PAR Values : Sedentary – 1.53, Moderate – 1.8; Heavy 2.3.

5. PROTEIN

Dietary proteins provide amino acids for the synthesis of body proteins, both structural proteins and biologically active enzymes and other biologically important nitrogenous compounds in the body. Adequate dietary protein is essential during growth when new tissue proteins are being synthesized. In the adult, dietary protein is essential for synthesis of new proteins to replace those, which are being broken down. During pregnancy, additional protein is necessary for synthesis of foetal, placental and body proteins that are increased in terms of increase in body weight during pregnancy. During lactation, additional protein is necessary for synthesis of proteins of the breast milk, which are secreted to feed the infant.

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

Human protein requirements are determined by several methods such as (a) N balance (b) Obligatory N losses and adjustment for efficiency of protein utilization (5.1). Currently, greater reliance is placed on the nitrogen 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 (5.2, 5.3) are based largely on N balance method.

There are considerable experimental data in India on N balance in adults which is useful in establishing their protein requirements. In recent years, there have been some systematic studies on N balance in children, pregnant and lactating mothers consuming their habitual diets, which are useful in arriving at the protein requirement of these physiological groups.

5.1. Protein Quality and Protein Requirements

An important factor in establishing human protein requirement on habitual diets is the quality of the habitual dietary proteins in terms of their essential amino acid content. Efficiency of utilization of dietary protein depends upon its digestibility and absorption of the released amino acids. Egg protein in particular is used as a reference protein against which the quality of other proteins is considered. Vegetable 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 limiting in one or more of the 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. In spite of such supplementation, however, Indian diets deriving their proteins from cereals and pulse combination (5:1) have a biological value of only 65% relative to egg protein. Currently, instead of egg protein as standard, human essential amino acid requirement, determined directly by newer and more accurate methods, gives a set of values for requirement that are higher than those determined by the earlier N balance method. The recent 2007 FAO/WHO/UNU Consultation (5.3) has recommended that directly determined amino acid pattern, based on requirements, should be used as a standard amino acid pattern for scoring quality of proteins and assessing human protein requirement. In the next section, currently recommended amino acids for humans for scoring protein quality are given.

5.2. Human Amino Acid Requirements: Recent Developments

Indispensable or essential amino acid requirements (IAA) of adult man recommended so far, as shown in Table 5.1, are quite low. Many proteins limiting in one or more IAA could supply the recommended IAA if adequate level of the protein or a combination of proteins is present in the diet. A reinvestigation of human IAA since 1990 has indicated that the earlier RDA of IAA are quite low. Recent investigations of human amino acid requirements based on turnover of amino acids using the stable isotope of carbon (^{13}C) labelled amino acids, have shown that human IAA requirements are 2-3 times higher than what was recommended earlier (5.2). These revised figures are also shown in Table 5.1, along with earlier figures for comparison. The Consultation of FAO/WHO/UNU of 2007 (5.3), which revised the human protein and amino acid requirements states 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.".

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, such that 'carbon balances' could be obtained. This was the so-called direct amino acid balance

method. However, technical difficulties in identifying the enrichment of the intracellular precursor pool from which amino acid oxidation was taking place, indicated that the 24-hour ^{13}C-labelled direct amino acid oxidation/balance method could be used definitively only in the case of leucine. When the same method was applied for lysine and threonine, with some assumptions, there were problems in arriving at a consensus over the merits of this direct carbon balance method.

Table 5.1

Essential Amino Acid Requirements: Adults

	FAO/WHO/UNU 2007		FAO/WHO/UNU 1985	
Amino acid	**mg/kg/d**	**mg/g protein**	**mg/kg/d**	**mg/g protein**
Histidine	10	15	8-12	15
Isoleucine	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 etc (see below).
Requirement, in terms of mixed or single dietary proteins = safe protein requirement / PDCAAS (see text)

A newer method of measuring amino acid requirement, which addressed several of the above methodological questions, including the precursor pool enrichment, was the indicator amino acid method. This method also relies on stable isotopes to measure amino acid oxidation, but it differs from the carbon balance approach in that the oxidation of an amino acid *other than* the test amino acid is measured. In this case, it was measured by determining

leucine balance, since the intracellular pool of leucine from which oxidation occurs (the precursor pool) was a-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. 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. 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, in essence, 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.

Owing to the complexity of the design, shorter indicator methods 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 $^{13}CO_2$ 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 similar, 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 children.

The implications of the new requirement are that protein quality (as discussed below) is an important issue in determining the requirement of proteins. In a cereal-based diet, lysine is the first limiting amino acid.

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

nan protein requirement should satisfy the currently established
ential amino acid requirements as recommended by FAO/WHO/UNU
nsultation of 2007 (5.3).

rogen balance data (both long term and short term) obtained in
mal, healthy well-nourished subjects should form the basis of fixing
imal N requirements.

ly protein requirement of different age groups should be expressed as
g. The safe level of requirement should be expressed as Mean + 1.96
while Mean – 1.96 SD should indicate the minimal requirement. These
extremes include 95% of the entire population within their limits,
ich extend to the 97.5 and 2.5 percentile of the populations
pectively.

daily requirement of protein for individuals of different age groups
uld be derived by a two-step process: First, the level of requirement
kg according to age has to be chosen and then the actual daily
uirement has to be computed for a given age by multiplying the
uirement per kg by the standard weight-for-age (as per the new
ndard weights chosen). For adolescents (10-18 years) and adults, the
uirement needs to be given according to gender.

the 1985 recommendations of FAO/WHO/UNU, the available long term
balance studies were considered, and the minimal protein requirement
adults, of 0.60 mg protein/kg/day was arrived at, whereas 2007
nsultation (5.3) considered all the now available N-balance studies,
ich totalled to nearly 235 studies. N requirements for zero balance were
nputed for each individual using simple linear regression, from which a
dian N requirement of 105 mg N/kg/day (equivalent to 0.66 mg
tein/kg/day), with 95% confidence intervals of 101 and 110 mg
g/day was derived. The median was estimated because the data were
tributed in a log normal distribution. In order to calculate the variability
these data, an ANOVA of cleaned data from all studies was performed,
ich indicated the variance to be distributed around 60% within studies
d 40% between studies. Where replicate measurements were available
the same individual (20 individuals in 4 studies), ANOVA of these data
owed that 2/3 of the variance was within-individual variance (temporal
experimental error) with the remaining representing true between-

individual variance. Combining these steps gave an estimate of 0.12 for the between-individual SD of the log requirement. Since the log requirements were normally distributed, the 97.5th percentile of the population requirement distribution was calculated as the log median requirement plus 1.96 multiplied by the SD of 0.12. The log median requirement was 4.65, and this, added to the product of 1.96 and 0.12, gave a value of 4.89. When exponentiated, it gave a value of 133 mg N/kg/day. Calculating an SD for this requirement value is not easily possible, because of the skewed distribution; however, calculating the cv from half the difference between the 16th and 84th percentiles would yield a value of about 12%.

6. These nitrogen values, in terms of protein requirements, would be 0.66 g/kg/day and 0.83 g/kg/day for the median and safe requirement respectively. The corresponding safe requirement recommended by FAO/WHO/UNU, 1985 Consultation was lower, i.e. 0.70 g/kg/day.

5.3. QUALITY OF PROTEIN

Another factor, which determines the daily protein requirement, is the absorption and the biological value of dietary proteins. The digestibility i.e., proportion of food protein which is absorbed, is computed from measurement of the nitrogen content of the food ingested and the nitrogen excreted in faeces, taking into account the extent to which faecal nitrogen is "endogenous" which, in turn, is measured as faecal nitrogen lost on a protein-free diet.

$$\text{Apparent protein (N) digestible} = \frac{I-F}{I} \times 100$$

$$\text{True protein (N) digestible} = \frac{I-(F-F_e)}{I} \times 100$$

Where I : Nitrogen intake
F : Faecal nitrogen lost on a test diet
F_e : Faecal nitrogen lost on a protein-free diet

The relative biological significance and practical importance of faecal as opposed to ileal digestibility has become a major issue. High level of dietary fiber in vegetables decreases protein digestibility. There may be differences between faecal and ileal digestibility. However, for the present, only faecal digestibility is used. It is possible that due to ileal fermentation by bacteria, some of the nitrogen is converted into bacterial amino acid, which eventually may be available for absorption. While this is possible, it is still not clear whether this contribution from recycled N is nutritionally significant. More studies are required in this area.

Amino acid scores of some food groups are presented in Table 5.2. 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 amino acid score, and hence the protein digestibility corrected amino acid score (PDCAAS, see below for a complete description of this index) will be less than the optimal score of 100, or less than the requirement for a high quality protein. The amino acid scores of pulses and animal protein remain above the optimal score of 100.

Table 5.2

Amino Acid Scores based on FAO/WHO/UNU 1985 and 2007 Consultation

Protein Source	Lysine Content mg/g protein	FAO/WHO/UNU 1985: Lysine score (16 mg/g protein)	FAO/WHO/UNU 2007: Lysine score (45 mg/g protein)
Wheat	27	>100	60
Rice	35	>100	78
Sorghum	24	>100	53
Millet	22	>100	50
Nuts / Seeds	35	>100	77
Vegetables	43	>100	96
Legumes	73	>100	>100
Animal Protein	82	>100	>100

References 5.2 & 5.3

In assessing the limiting amino acid and the PDCAAS of the protein, a diet simulating the habitual diet of any country, or a well-balanced diet to provide all nutrients at the recommended level has to be considered. 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 (5.4) 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 indispensable amino acid content of proteins from different sources in the diet, cereal, legumes, vegetables and milk were computed (see Table 5.13). Weighted averages of different food group proteins and their amino acid composition were computed, taken together and their protein digestibility corrected.

Biological value and amino acid score

The amino acid profile is assumed to determine the effectiveness with which absorbed dietary N can be utilized. This is usually defined in terms of biological value (BV) i.e.

$$\text{Apparent protein (N) biological value (\%)} = \frac{I-F-U}{I-F} \times 100$$

$$\text{True protein (N) biological value \%} = \frac{I-(F-F_e)-(U-U_e)}{I-(F-F_e)} \times 100$$

Where: I : Nitrogen intake
F : Faecal nitrogen loss on a test diet
F_e : Faecal nitrogen loss on a protein-free diet
U : Urinary nitrogen on test diet
U_e : Urinary nitrogen on protein-free diet

Protein quality can be determined by a purely biological method or by the amino acid profile as compared to that of a standard protein like egg or in comparison with that of requirement pattern. Earlier, 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 limiting amino acid content of the test protein should predict its quality or BV (i.e.) Amino acid score (AAS) is expressed as the ratio of mg of amino acid in 1 g of test protein to mg of amino acid in reference protein.

$$\text{Amino acid score (AAS)} = \frac{\text{mg of amino acid in 1 g of test protein}}{\text{mg of amino acid in 1 g reference protein}}$$

Then, Protein digestibility corrected amino acid score (PDCAAS) =
Protein Digestibility x Amino Acid Score

The protein digestibility on mixed vegetarian diets is usually about 85%. The PDCAAS value should predict the overall efficiency of protein utilization in terms of its two components, digestibility and biological value (BV) where BV is utilized N/digestible N, which should be related to its amino acid score. The principle behind this concept is that utilization of any protein will be first limited by digestibility. It determines the overall available amino acids nitrogen from the blood and BV describes the competence of the absorbed amino acids to meet the metabolic demand: For any diet, BV cannot exceed 100, since for the absorbed N the best that can be utilized cannot be more than the amount of requirement.

n any diet, the amount of rotein to meet the safe equirement = Safe level of protein (as derived above in section 5.2) / PDCAAS value of the diet

A key element in the understanding of the PDCAAS concept is that the lue is always truncated at 1 (equivalent to 100%). That is, even apparently jh quality proteins whose amino acid score is greater than 1, will have a orted PDCAAS of no greater than 1. In looking at the equation above, it is dent that if a PDCAAS value greater than 1 were used, it is possible that "requirement" of a high quality protein could be less than the safe uirement. Further, the PDCAAS value has been used in identifying teins that can be used to complement each other, in which case, non-ncated values of PDCAAS for each protein have been used. It must be ed that these considerations are only in relation to amino acid nposition. When considering single proteins, it is clear that considerations its quality are secondary to the amount absorbed after digestion, or its estibility. Thus, a protein such as soy protein, with a digestibility of 95%, an amino acid score of 1.04, cannot have a PDCAAS of 1.04 x 0.95 = 9. This would imply that the amino acid score could make up for the ino acids that were lost during digestion and absorption, and this is orrect. In such a case, PDCAAS would still be reported as 0.95, where the ino Acid Score is truncated to 1, such that PDCAAS of soy protein = 1.0 x 5; which is based on the first principle of how much of the protein was orbed after digestion.

While discussing PDCAAS of proteins in relation to fulfilling protein uirement of an individual, the quality of a mixture of proteins present in a has to be considered, instead of individual sources of protein. Proteins n different sources may be present in a diet and they may complement h other and a limiting amino acid in a diet may be different from individual tein sources present in the diet. For example, cereal proteins may be ting in lysine, but if a mixture of cereals and legumes or legumes and milk eins are present, lysine, deficiency may be reduced considerably. The ed proteins from cereals and legumes may complement each other and AAS of proteins from a cereal- pulse based diet will be more than that of real protein. It is important in these calculations to adjust for digestibility at is, the Amino Acid Score of these mixed proteins should be calculated from each protein's digestible amino acid content.

It would appear that the AA composition of protein from a well-balanced , deriving its protein from cereal, legume, vegetables and milk is more the recommended human requirement for adult man in case of all amino s except for lysine which is 97% of the requirement. The only other

limitation of vegetable protein is its digestibility. Several studies on the proteins from cereal-legume-milk based diets have shown that true (corrected for metabolic faecal N) digestibility is around 85%. Taking into consideration the amino acid index and digestibility, the PDCAAS for a cereal-legume-milk based diet is :

$$\text{PDCAAS} = \frac{97 \times 85}{100} = 82.5\%$$

The new value for protein requirement given by the 2002 FAO/WHO/UNU Consultation is 0.66 g $kg^{-1}d^{-1}$ and the safe requirement is 0.83 g $kg^{-1}d^{-1}$ respectively. When corrected for marginally lower amino acid score and lower digestibility, the protein requirement of healthy Indian (adult) on a well-balanced cereal-legume-milk (animal protein) diet in the ratio (8.0:2.4:1.0) is 0.8 g/kg/day and 1.0 g/kg/day respectively for mean and safe requirements. On the basis of current recommendations of FAO/WHO/UNU Consultation 2007, recommended daily safe protein allowance for an adult eating a standard Indian predominantly vegetarian diet would be 1.0 g/kg/day, a figure recommended by the ICMR Expert Group in 1989. Even if the digestibility on a cereal-pulse-milk diet is lower, at 80%, an almost similar safe requirement of 1.04 g/kg/day would be obtained.

5.4. Factorial method for arriving at RDA for proteins of Indians

Basal or obligatory N loss

The factorial method of assessing protein requirement of an individual can be in terms of obligatory loss of N through faeces, urine and skin. 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 obligatory loss, based on the zero intercept of the N balance to N intake relationship was found to be 48 mg N/kg/day. It was similar to the mean value of 47 mg N/kg/day obtained from 15 studies that had been designed to actually measure the obligatory requirement. There are several factors that can influence this loss, principally the energy intake. In terms of protein, this works out to a low value of 0.3 g protein/kg/day. However, the efficiency of utilization of protein being relatively low at about 47% (based on the meta-analysis of N balance studies in the 2007 Report), 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, obtained by individual zero N balance intakes in the meta-analysis above.

The directly observed obligatory N loss on a minimal protein diet in ians has been shown to be 20 mg/kg/d and 37mg/kg/d for faecal and ıary N excretion respectively. This value (total of 57 mg N/kg/d) was ıewhat higher than the average obligatory N loss of 48 mg/kg/d described ıve. However, this value was not significantly different from the values of gatory N loss described in Western studies, and close to the value ained in an African study. The Nigerian and Indian studies showed that cal losses of N were higher and accounted (not significantly) for the higher ıl obligatory N loss. In addition, these may have been due to differences in nature of the minimal protein diet provided in all these studies. Two key ments remain in the interpretation of these Indian values. First, the aneous loss of N, which was taken as 8 mg/kg/d, as suggested by)/WHO/UNU, 1985 Consultation. However, based on several studies in h area, the FAO/WHO/UNU, 2007 Consultation has suggested a loss of 11 /kg/d for subjects in tropics and 5 mg/kg/d for those in temperate climate. second element is the efficiency of utilization of protein. The body weight he low income group men who were studied ranged from 44.8 to 48.4 kg, l these body weights are among the lowest for subjects studied in all the dies of obligatory N loss. The efficiency of utilization of protein, while en to be 47% as an average, can vary, and can reach 100% in lnourished/ adapted individuals. It is likely that the efficiency of utilization ɔrotein may have been higher in the sets of Indian (and African) subjects, en their low body weight. Therefore, total obligatory N loss for Indian ılt subjects:

Urinary	37 mg/kg/d
Faecal	20 mg/kg/d
Cutaneous	11 mg/kg/d
Total	68 mg/kg/d = 0.4 g protein/kg/d

At the physiological level of requirement (assuming an additional uirement of 50%, which presumes a high degree of efficiency of utilization hese undernourished men), this works out to 0.64 g protein/kg/day.

e level: Mean+1.96 SD (assuming a cv of 12.5%) = 0.8 g/kg/d
rected for amino acid score and digestibility (PDCAAS of 82.5%) = 0.97 g/d.

refore, this method also yields a figure of nearly 1.0 g/kg/d.

sed on N Balance

rogen balance: Several studies on healthy, adult Indian subjects have wn that the minimum dietary protein intake for N equilibrium ranges from to 0.66 g protein/kg/day with a mean of 0.58 g/kg/day which, after

allowing for integumental loss of 11 mg N/kg/day, works out to 0.65 g protein/kg/day. Assuming a cv of 12.5%, the safe value of intake, (mean + 1.96 x SD) works out to 0.81 g protein/kg/day. The corresponding requirement based on a mixed Indian diet as shown in Annexure, will be 0.98 g protein/kg/d and this figure is also close to 1g/kg/d.

Thus, daily *safe level* (estimated average requirement + 1.96 SD) of protein requirement of Indian adults on a cereal-pulse-milk diet as defined in Annexure, *adjusted for quality* (PDCAAS) is about 1.0 g/kg/d, when computed by two methods : (i) Factorial method taking into account urinary, faecal as integumental N losses, at the physiological level after correcting for amino acid score and absorption and (ii) N balance after correcting for miscellaneous losses, all at the safe level (Mean+1.96 SD), in both instances, corrected for indispensable amino acid score and digestibility (PDCAAS).

There is no compelling evidence that suggests that protein requirements for the elderly are increased. There have been suggestions that the elderly require more protein intake, owing to a reduced absorptive capacity, but N balance studies do not provide enough evidence to make a recommendation for an increased protein intake. However, protein and energy are inter-related. In an elderly person whose activity is maintained, there is sufficient energy intake to ensure adequate protein intake. In the sedentary elderly person, with reduced energy requirement, there is a need to consider the protein energy ratio of the diet, since protein intake can reduce if the total diet is reduced.

5.5. Protein requirement during pregnancy

Protein requirement during pregnancy has been assessed by the factorial method as the additional requirement for foetal growth and expansion of maternal tissue.

This was done earlier by FAO/WHO/UNU 1985 Consultation and also by 1989 ICMR Expert Group on Recommended Dietary Allowances. 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 Consultation, while using the factorial method as as the Nitrogen balance method, has also considered newer data on ein deposition during pregnancy, using the total body potassium (TBK) hod, in which total body potassium accretion was measured in pregnant ien, by whole body potassium counting. Conversion of potassium etion to nitrogen accretion was based on the potassium nitrogen ratio 2.5 potassium / g N. These studies showed that the total protein deposited ng pregnancy was 686 g, but this was not deposited at a uniform rate ng pregnancy. In well-nourished women with a mean gestational weight (GWG) of 13.8 kg, this protein deposition was distributed as 1.9 g/day in second trimester and 7.4 g/day in the third trimester (5.3). Significantly, detailed measurements of FFM, based on TBK and total body nitrogen asured by prompt-gamma neutron activation) in women during and after nancy, showed that there was no net accretion or loss of protein during nancy, suggesting that during pregnancy protein was deposited only foeto-placental tissue (reported in 5.3).

herefore the FAO/WHO/UNU 2007 Report is based on more recent data, ugh it uses the same factorial approach as earlier. In that report, GWG based on a WHO collaborative study on gestational weight gain, which 12.0 kg (5.5). Based on the figures of GWG and protein deposition ssed in the paragraph above, if one assumed protein deposition to be ortional to GWG, in women with gain12 kg GWG, the total protein sited would be 597 g, distributed as 1:6 g/d and 6.5 g/d in the second the third trimesters respectively. In a woman gaining 10 kg GWG, in deposition figures would be 1.4 and 5.4 g/day for the second and trimesters respectively. While these computations have been based on eference women with a pre-pregnancy weight of 55 kg and gestational it gain of 10 kg, it is worthwhile considering that these may be even in the Indian pregnant woman whose mean pre-pregnancy weight is 47 d who has a GWG of 7-8 kg.

ie next question in the factorial estimate was the efficiency with which y protein could meet these deposition needs. This is discussed below.

gen balance and efficiency of utilization in pregnancy

all, 273 metabolic balance studies were available, the majority of which on women at or beyond 20 wk of gestation (5.6). The average N ion was 1.8 g/d from 20 wk onwards and 1.3 g/d before 20 wk. laneous N losses unaccounted for in these studies, were estimated to be l. The average theoretical retention (mean during whole pregnancy) .53g/d, but the observed N retention after correcting for miscellaneous es of 0.5 g/day was 1.1 g/day. The efficiency of N utilization calculated hese 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 figure used to calculate the extra protein requirement in Table 5.3). This figure was also reasonably close to 47% that was derived in non-pregnant adults.

Table 5.3

Recommended additional protein intake during Pregnancy, for a 10 kg gestational weight gain

Tri-mester	Mid-trimester weight gain[a] (kg)	Additional protein for maintenance g/d[b]	Protein deposition g/d	Dietary protein requirement for deposition[c] g/d	Mean extra protein requirement[d] g/d	Safe intake g/d[e]
1	0.6	0.4	0.0	0.0	0.4	0.5
2	3.5	2.3	1.4	3.3	5.5	6.9
3	8.0	5.3	5.4	12.9	18.2	22.7

[a] Women gaining 10 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% efficacy of utilization
[d] Sum of extra maintenance plus protein deposited
[e] Safe Intake = Mean extra protein requirement + (1.96 x SD) assuming CV of 12.5%. This requirement refers to high quality protein, with PDCAAS of 100. For a 12 kg gestational weight gain, see text.

Factorial approach to defining requirements during pregnancy

With the new data drawn from the observations made above, the additional protein intake needed during pregnancy was derived from the protein deposited (adjusted for the efficiency of utilization of dietary protein) and the maintenance costs of protein intake associated with increased body weight. The key question is the magnitude of the GWG for the average Indian woman. The present consultation decided that two values for GWG be presented in this report, that is, 12 and 10 kg. For the GWG of 12 kg, mean protein deposited was estimated from TBK accretion as described above (1.6 and 6.5 g/d in second and third trimesters). The efficiency of protein utilization was considered to be 42%. To this were added the maintenance costs, which were based on the mid-trimester body weight of pregnant woman and the adult maintenance value of 0.66 g/kg/d. For the GWG of 12 kg, mid-trimester weight gain was assumed to be 0.7, 4.1 and 9.4 kg in the first, second and third trimesters respectively. Finally, the safe level of

increased intake by trimester was derived from the estimated average extra requirement, assuming a coefficient of variation of 12.5%. Based on these calculations, the extra protein requirements (at safe levels) were 0.6, 8.1 and 27.0 g/day, during the first, second and third trimesters respectively, to support a 12 kg GWG.

If the GWG is only 10 kg, the same calculations would yield additional high quality protein requirement of 0.5, 6.9 and 22.7 g/day during the first, second and third trimesters respectively (Table 5.3). Given that surveys such as the NFHS and NNMB have reported pre-pregnancy weight of 47 kg, and GWG of only 8 kg, it is worth recording that the additional high quality protein requirements in such a pregnant woman gaining 8 kg during pregnancy, are 0.4, 5.6 and 18.1 g/day for the first, second and third trimesters respectively.

These requirements would increase further, if they were to be met only by protein in the cereal-pulse-milk based low cost Indian diet, which has a PDCAAS of 82.5%. In addition, given that the low cost Indian diet contains only about 10% protein calories, meeting the extra protein requirement in the third trimester, for example, would mean the consumption of far too much energy when only on the low-cost Indian diet; therefore, the consumption of high quality protein foods (such as milk or eggs) is recommended.

At first sight, 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, with a mid 3rd-trimester weight gain of 8 kg (GWG of 10 -12 kg), an energy allowance of 375 kcal /day, and an extra allowance of about 20 g protein/day, the calculated PE ratio of the required diet would be about 13%. This PE ratio would decrease to about 12% if the woman were moderately active. Therefore, the PE ratio (requirement) of the diet does not increase dramatically in spite of the higher protein requirement. Use of supplements very high in protein (greater than 34% PE ratio) may have some adverse events. It is therefore important that the higher intake of protein recommended during pregnancy should come from a normal, varied diet, 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 requirements and the safe limits will be lower and could easily be met from dietary sources such as cereal, pulse and milk-based vegetarian diets.

Rounded off requirements of high quality protein, for 10 kg gestational weight gain, are 1, 7 and 23 g/day in 1st, 2nd, and 3rd trimesters respectively.

It cannot be over-emphasized that protein supplements are not required to meet this additional requirement during pregnancy. One example of a typical Indian vegetarian high protein diet with a PE ratio of 12% is given in Annexure 5.2. In this example, protein intake was adjusted for its quality, through the use of the PDCAAS. These figures should be viewed in comparison to the low-cost well-balanced Indian diet for a non-pregnant non-lactating Indian woman of weight 55kg, following a moderate activity lifestyle (with a PE ratio of about 10%), based on the low-cost balanced Indian diet given in the Annexure 5.1. In the latter diet, the pulse:cereal ratio is less than 1:10, with a milk intake of 120 g/day. In the higher protein diet with extra milk, the pulse:cereal ratio is about 1:5. This would also automatically improve the PDCAAS of the mixed protein from the diet. The foods can also be varied, selecting foods with high protein content. For example, pulses or legumes, which have an individual PE ratio of 28%, can be added as a cup of lentils or whole gram at meal time, or even between meals. Similarly, a greater use of milk or milk-based products (with a PE ratio of 15%), or non-vegetarian foods such as eggs (PE ratio of 30%) or flesh foods can further increase protein intake. All these food groups also add high quality protein to diet.

A final consideration is the habitual activity of pregnant woman. Clearly, a sedentary lifestyle will need a higher PE ratio in the diet (about 13%). 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 600 g/day), pulse: cereal intake ratio is about 1:5, and root vegetables and visible fat are reduced. Non-vegetarian foods can help fill the requirement for high quality protein.

5.6. Protein requirements during lactation

Adopting the factorial approach to derive protein requirements during lactation, protein requirement during lactation has been computed on the basis of secretion of 9.4 g per day of protein in milk during 0-6 months and 6.6 g during 6-24 months. Earlier, FAO/WHO/UNU 1985 and ICMR Expert Group 1989 assessed the protein content of milk as N x 6.25. But in the 2002 FAO/WHO/UNU Consultation, mean concentration of protein and non-protein nitrogen in human milk were used to calculate the mean equivalent of milk protein and non-protein nitrogen output. Breast milk contains a relatively high concentration of non-protein nitrogen equivalent to 20-27% of total milk nitrogen, much of it being urea. However, in calculating requirements, protein requirement was assumed to be only for the protein component of the total N in milk. A factor of 6.25 is used to convert protein nitrogen in milk to protein equivalent. The efficiency of conversion of dietary nitrogen to milk protein equivalent can be assumed to be 47% on Indian diets, as in case of adults. Safe intake is calculated as mean + (1.96 SD) with 12.5% CV.

The additional mean and safe protein intake at different months of lactation are given in Table 5.4. Rounded off figures would be 19 g/d for safe allowance for a lactating woman during 1-6 months and 13 g from 6 to 12 months. The difference in the 1989 recommendations and present RDA is due to exclusion of NPN from total milk N while computing protein concentration of milk. Requirement of a lactating woman in terms of cereal-pulse-milk based dietary protein with PDCAAS of 82.5% would be 22.9 and 15.2 g/d during 0-6 months and 6-12 months respectively. However, as this would entail intake of a lot of energy, intake of high quality protein containing foods, with a high PE ratio is recommended. As noted above for pregnancy, these protein requirements can be met from a balanced diet with a PE ratio between 12-13%.

Table 5.4
Additional protein requirement during lactation

Months post-partum	Milk output g/day	Milk output corrected for IWL[a] (g/d)	Protein Conc. g/L [b]	NPN protein equi-valent (g/L)[b]	True protein secreted (g/d)	NPN protein equi-valent (g/d)	Mean dietary require-ment[c] (g/d)	Safe intake (g/d)
1	699	734	10.4	3.1	7.6	2.3	16.2	20.2
2	731	768	9.6	2.8	7.3	2.2	15.6	19.5
3	751	789	8.8	2.4	7.0	1.9	14.8	18.5
4	780	819	8.2	2.2	6.7	1.8	14.3	17.9
5	796	836	8.1	2.1	6.8	1.8	14.4	18.1
6	854	897	8.1	2.1	7.3	1.9	15.5	19.4
Mean 1-6	768.5	807.2	8.9	2.5	7.1	2.0	15.1	18.9[d]
Mean 6-12	550	578	8.2	2.1	4.7	1.2	10.0	12.5[e]

[a] Milk output corrected for insensible water loss during test weighing measurement(5%)
[b] Nitrogen converted to protein by using the factor 6.25
[c] Mean + 1.96 SD assuming coefficient of variation is 12.5%
[d] in terms of Indian dietary protein 22.9 g/d
[e] in terms of Indian dietary protein 15.2 g/d

Nitrogen balance during lactation

A nitrogen balance study in Indian lactating women (5.7) indicated a linear relationship between N balance and protein intake over a range of 60-100g a day. Minimum protein required to maintain N balance in the subjects studied was found to be 1.5 g protein/kg/day, which after allowing for a high faecal N excretion, was found to be 1.2 g/kg/day. These figures are close to the derived estimates of protein requirement during lactation, where, women

with a weight of about 60 kg after delivery would meet the additional 20 g required for lactation in the first 6 months, by increasing their protein intake to about 1.3 g/kg/day.

Overall, the higher level of protein required during pregnancy and lactation computed from N balance, when compared to normal requirement 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 must be quite low and not 70% as assumed.

5.7. Protein and amino acid requirements of infants and children

The 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, growth requirements are added. In the FAO/WHO/UNU 2007 Report, for the maintenance requirement, 10 studies were available, that examined the relationship between protein intake and nitrogen balance both above and below maintenance. Data from these multiple nitrogen balance studies among children were analyzed following a linear regression approach as described for adults. Individual data were fitted to the linear model.

Nitrogen Balance = A + (B x Nitrogen intake)

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

Studies from FAO/WHO/UNU 2007:

All individual estimates (7 studies) : 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, 3 other studies were available with estimation of slope and intercept (the latter to determine the obligatory N loss at a zero N intake), where 57 subjects were measured for N losses at zero or very low protein intake to directly estimate their obligatory N loss. In these latter studies, a value of 63 ± 12 mgN/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/kg/day (from the direct obligatory N loss studies) are quite similar. However, they are higher than obligatory N loss value of 47 mg N/kg/day in adult. In addition, the efficiency of utilization (slope) in studies on infant and child was on average 56-58% for all the N balance studies above, but 66% in the case of studies with milk/egg.

From the regression of N balance on N intake studies, a value for the maintenance requirement of N could be obtained, by calculating the N intake required for a zero N balance. The mean value for maintenance requirement was 108-110 mg N/kg/day in all studies, but lower at 93 mg N/kg/day in the animal protein (milk/egg) intake studies. This lower maintenance requirement could have been because of the better digestibility of animal proteins, as well as a better efficiency of utilization (66% vs. about 58% in the mixed protein intake diets).

Maintenance intake can also be calculated from the obligatory loss, divided by the efficiency of utilization. For all the N balance studies quoted above, this value would be 57/0.58 = 98 mg N/kg/day, and for the animal protein diets, this would be 62/0.66 = 94 mg N/kg/day. 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 (in the FAO/WHO/UNU 2007 Report) 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/day. This works out to a protein intake of 0.58 g protein/kg/day. For children with age higher than 6 months, the maintenance value chosen was 110mgN/kg/day. This was the mean maintenance value from all the N balance studies quoted above. It works out to 0.68 g protein /kg/day, and ultimately, since this was so close to the adult maintenance value, maintenance value for children above 6 months of age 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 the feeding of the infant changes to mixed diets on weaning.

These new values for the maintenance requirement are lower than 120 mg N/kg/d (or 0.75 g protein/kg/day) assumed by 1985 FAO/WHO/UNU Report. In addition, in that report, it was assumed that the maintenance requirement increased from 0.58 to 0.66 g protein/kg in proportion to fall in growth rate; as stated above, the maintenance requirement is now assumed to change once, at the age of 6 months.

Growth requirement and patterns of protein deposition during growth

Protein deposited for growth from 0.5 y to 18 y, together with an expression of amino acid composition of whole body protein allows a substantial improvement in the factorial estimates for overall protein and essential amino acid requirement during growth. The average daily rates of protein deposited have been derived from the measurement of whole body potassium (5.8, 5.9).

Children from age 0-2 y

In a longitudinal study, Butte *et al* (5.8) followed 76 individual infants from birth to 2 years with measurements taken every three 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:

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

The derivatives of these curves were fitted to power curves:

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

Next, individual weight data were fitted to power curves

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

Then, for each individual infant the ratio of these two equations estimated protein deposited per day per kg of body weight.

Children from age 4-18 y

Protein deposition needs of children (4-18 y) were reported by Ellis (5.9) in a cross sectional study (Tables 5.5). A single model was fitted to the entire data set for each gender: Data on protein (yearly cohort averages) were fitted into a single cubic curve for each gender.

Males: Protein (kg) = $5.46 - (1.285 \times \text{age}) + (0.166 \times \text{age}^2) - (0.00433 \times \text{age}^3)$, $r^2=0.992$

Females: Protein (kg) = $3.91 - (0.925 \times \text{age}) + (0.139 \times \text{age}^2) - (0.00428 \times \text{age}^3)$, $r^2=0.993$

These curves were differentiated to give protein deposition rate estimates.

Males: Protein deposited (kg/year) = $-1.285 + (0.332 \times \text{age}) - (0.0130 \times \text{age}^2)$

Females: Protein deposited (kg/year) = $-0.925 + (0.279 \times \text{age}) - (0.0128 \times \text{age}^2)$

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

Males: Weight (kg) = $5.42 - (15.0 \times \text{age}) + (1.89 \times \text{age}^2) - (0.0557 \times \text{age}^3)$, $r^2=0.991$

Females: Weight (kg) = $2.53 - (4.47 \times \text{age}) + (0.73 \times \text{age}^2) - (0.0198 \times \text{age}^3)$, $r^2=0.972$

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 years (FAO/WHO/UNU 2007). 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.

Table 5.5

Protein deposition in infants and children[a]

Age y	Protein deposition g/kg/d				
	Females	Males	Both genders	SD[b]	Group
0.5	0.266	0.266	0.266	0.035	Infant
1.0	0.168	0.1680	0.168	0.031	
1.5	0.108	0.108	0.0108	0.029	
2.0	0.076	0.073	0.075	0.026	Pre-school
3.0	0.044	0.034	0.039	0.022	
4.0	0.026	0.013	0.020	0.019	
5.0	0.022	0.009	0.016	0.017	
6.0	0.038	0.032	0.035	0.016	School children
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	
10.0	0.047	0.054	0.051	0.017	
11.0	0.043	0.050	0.047	0.018	Adolescents
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	
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* (5.8) Ellis *et al* (5.9)
[b] On average, the CV of protein growth was 24% (till 2 y) from Butte et al; and from longitudinal data on velocity of growth for older children (2-18 y).

Variability of these data is also an important consideration, as this variability is a part of the total variability of the requirement in a factorial model (below). 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. This variability was used in the factorial model for infants up to the age of 6 months (below, Table 5.6). 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 5.6

Safe level of protein intake for infants aged less than 6 months

Age (m)	Maintenance requirement[a]	Growth requirement[b]	Average requirement[c]	Safe level[d]	1985 Report[e]
1	0.58	0.83	1.41	1.77	2.25
2	0.58	0.65	1.23	1.5	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	0.4	0.98	1.14	1.3

[a] calculated from the maintenance requirement (from N balance studies with milk/egg).
[b] Protein deposition rates taken from Butte *et al* (6.8), adjusted for 66% efficiency of utilization (from N balance studies with milk/egg).
[c] Sum of maintenance and protein deposition rate (latter adjusted for efficiency of utilization).
[d] Mean + (1.96 x root mean square of SD values for protein deposition during growth, adjusted for efficiency of utilization, and the maintenance).
[e] Values from the 1985 FAO/WHO/UNU consultation.

Factorial estimates of protein requirements for 0-18y

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

APR = Maintenance + (Deposition / Efficiency of utilization)

Maintenance is calculated assuming that maintenance in young children is 0.58g/kg/d which increases at 6 months to the adult value of 0.66g/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 assumed to be 58%.

The safe level (exceeding the requirement of 97.5% of the population) is then estimated assuming that the requirement follows a log normal distribution i.e., safe level is the average level plus 1.96 standard deviation,

with total variability of maintenance and deposition calculated from the root mean square of CV of 12% for maintenance (as used in case of adults) and 24% for the protein deposition rates between 0 -2 y and observed values for the older children. 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.

Table 5.7

Safe level of protein intake for children over the age of 6 months up to 10 years (genders combined)*

Age (y)	g protein / kg Body weight /d				
	Maintenance[a]	Growth[b]	Total	Safe level[c] 1.96xSD (1985 values)	Safe level Indian diet[d]
0.5	0.66	0.46	1.12	1.31 (1.75)	1.69
1	0.66	0.29	0.95	1.14 (1.57)	1.47
1.5	0.66	0.19	0.85	1.03 (1.26)	1.33
2	0.66	0.13	0.79	0.97 (1.17)	1.25
3	0.66	0.07	0.73	0.90 (1.13)	1.16
4	0.66	0.03	0.69	0.86 (1.09)	1.11
5	0.66	0.06	0.69	0.85 (1.06)	1.09
6	0.66	0.04	0.72	0.89 (1.02)	1.15
7	0.66	0.08	0.74	0.91 (1.01)	1.17
8	0.66	0.09	0.75	0.92 (1.01)	1.18
9	0.66	0.09	0.75	0.92 (1.01)	1.18
10	0.66	0.09	0.75	0.92 (0.99)	1.18

Values in parentheses are based on 1985 FAO/WHO/UNU Consultations.

* 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 (taken from Table 4.6 in Energy Chapter). Then, the total protein requirement will be = 1.18 x 28 = 33.0 g/day. These calculations are presented for all ages and both genders in Table 5.15.

[a] From N balance studies

[b] From Table 5.5 adjusted for efficiency of utilization of 58% from N balance studies (see text)

[c] SD calculated as in text

[d] Corrected for protein from Indian cereal-pulse-milk based diet having PDCAAS of 77.4% for children up to 10 years, as calculated in Table 5.13.

After the age of 10 years, 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 although the principles of calculation remain exactly the same as for children up to the age of 10 years. These are given in Table 5.8, and safe values for protein derived from an Indian balanced diet are also provided.

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] 1.96SD (1985 values)	Safe level Indian[d] diet
Boys					
11	0.66	0.09	0.75	0.91 (0.99)	1.16
12	0.66	0.08	0.74	0.90 (0.98)	1.15
13	0.66	0.07	0.73	0.90 (1.00)	1.15
14	0.66	0.06	0.72	0.89 (0.97)	1.14
15	0.66	0.06	0.72	0.88 (0.96)	1.13
16	0.66	0.05	0.71	0.87 (0.92)	1.12
17	0.66	0.04	0.70	0.86 (0.90)	1.10
18	0.66	0.03	0.69	0.85 (0.86)	1.09
Girls					
11	0.66	0.07	0.73	0.90 (1.00)	1.15
12	0.66	0.06	0.72	0.89 (0.98)	1.14
13	0.66	0.05	0.71	0.88 (0.98)	1.13
14	0.66	0.04	0.70	0.87 (0.94)	1.12
15	0.66	0.03	0.69	0.85 (0.90)	1.09
16	0.66	0.02	0.68	0.84 (0.87)	1.07
17	0.66	0.01	0.67	0.83 (0.83)	1.06
18	0.66	0.00	0.66	0.82 (0.80)	1.05

Values in parentheses are based on 1985 FAO/WHO/UNU Consultations.
For total daily protein requirement based on attained body weight in each age band, see Table 5.15 below.

[a] From N balance studies

[b] From Table 5.5 adjusted for efficiency of utilization of 58% from N balance studies (above)

[c] SD calculated as in text

[d] Corrected for protein from Indian cereal-pulse-milk based diet having PDCAAS of 78.2% as calculated in Table 5.13, as an average for the entire age range of 10-18 years.

These protein requirements of children and adolescents have been based on systematic studies on protein deposition based on total body potassium (TBP) and maintenance requirements, extrapolated from infant to adult. 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 intake of protein 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 (vide Chap 3). Safe intakes of protein by Indian children in terms of good quality protein, proposed by FAO/WHO/UNU and values corrected for Indian cereal-pulse-milk based balance diets in case of children and adolescents are given in Tables 5.7 & 5.8. The values for infants up to 0.5 yrs are those proposed by FAO/WHO/UNU as they are based on breast milk or artificial feeding on milk.

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 5.9. These values were averaged from 3 sources (from FAO/WHO/UNU 2007). 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 5.9

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, 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 5.1) 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 used for multiplication by the protein deposited with growth (adjusted for efficiency of utilization of dietary protein), for each amino acid. The 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 5.10.

Table 5.10

Factorial calculation for daily amino acid requirement in children

			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
	Protein requirement (g/kg/d)		**Amino acid requirement (mg/kg/d)[d]**								
Age (y)	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	18	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 (Table 5.1)

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

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

The amino acid scoring pattern for this requirement is given in Table 5.11. This is in contrast to the 1985 FAO/WHO/UNU consultation, where the pattern for pre-school children was used generally for all ages. In general, the new FAO/WHO/UNU 2007 requirements are lower than the earlier values that were used for the pre-school child.

Table 5.11

Amino acid scoring pattern for children (mg/g protein)

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	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

The current recommendation of safe intake of protein during 0-18 years

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. Those values differ from the figures given by the 1985 Committee. Since the figures arrived at by the FAO/WHO/UNU Committee during 2007 were more methodologically sound and based on International research data, we can adopt these figures per kg weight for Indian population also. 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 amino acid score and digestibility (based on reported absorption of dietary protein from several Indian studies) of 85%, and the amino acid score 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. These computations are given in Table 5.12. Protein requirement from the Indian diet per kg/d can be calculated from the body weights of normal healthy Indians of different ages.

Table 5.12

Amino acid scoring pattern for requirement computation (mg/g protein)

Age groups (y)	His	ILe	Leu	Lys	SAA	AAA	Thr	Trp	Val
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 (adult)	15	30	59	45	22	38	23	6	39
Amino Acid composi-tion (mg/g protein)**	26.7	43.1	83.5	43.6*	37.6	86.7	36.3	11.1	54
Amino Acid Score									
3-10 y	100+	100+	100+	91	100+	100+	100+	100+	100+
11-14 y	100+	100+	100+	91	100+	100+	100+	100+	100+
15-18 y	100+	100+	100+	93	100+	100+	100+	100+	100+
>18 y (adults)	100+	100+	100+	97	100+	100+	100+	100+	100+

* Presuming wheat to be whole wheat. With refined wheat flour, this lysine value reduces by about 5-7%.

** Amino acid content calculated from balanced diet presented in Annexure. For the purpose of calculation, cereals were presumed to be in the ratio 40:40:20 for rice:wheat:bajra. 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 green beans. Potato was considered to be representative of the roots and tubers food group. Fruits were considered similar to 'other vegetables'. Source of food composition: reference 5.13.

Taking data from Tables 5.12 and 5.13, it is evident that lysine is the limiting amino acid in all age groups, on a cereal-based diet.

Table 5.13

PDCAAS (assuming lysine as limiting) for different age groups

Age Group	Limiting AA	AA Score	PDCCAAS = AA Score X digestibility
3-10 y	Lysine	91	91 x 85/100 = 77.4
11-14 y	Lysine	91	91 x 85/100 = 77.4
15-18 y	Lysine	93	93 x 85/100 = 79.0
> 18 y (adult)	Lysine	97	97 x 85/100 = 82.5

Requirement in terms of mixed Indian diet protein (Annexure 5.1) = Requirement in terms of high quality protein/ (PDCAAS of mixed Indian diet protein /100)

5.8 Summary of recommended protein intakes for Indians

The final recommended safe protein intake at different ages is given per kg body weight as well as total requirement for a normal individual. These figures are given in Tables 5.14 for adults and in 5.15 for children. Total daily requirement of normal population is computed by multiplying the per kg value at different age levels with the corresponding normal standard body weights.

Table 5.14

Protein requirement for normal indian adults and allowances for pregnant and lactating women

Group	Body weight kg	Protein[a] g/kg/d	Daily additional require-ment[b] (g)	Total daily require-ment (g)
Adult				
Males	60	1.0		60
Females	55	1.0		55
Pregnant Women (3rd trimester, 10 kg GWG)			23	78
Lactating Women				
0-6 m			19	74
6-12 m			13	68

[a] In terms of mixed Indian diet protein (Annexure 5.1)
[b] High quality protein
GWG: Gestational Weight Gain

Protein requirement during pregnancy is the sum of adult requirement plus additional protein needed for tissue deposition and foetal growth. Protein

requirement during lactation is the adult requirement plus that needed for protein in breast milk secreted. These have been computed after correcting for the lower nutritive value of Indian diets. The daily requirement for Indian children, computed for their growth, is given below.

Table 5.15

Protein requirement and dietary allowances for infants, boys and girls

Age Group	Requirement[a,b] g protein/kg/d	Body weight (kg)	Total daily requirement (g protein/d)	Requirement[a,b] g protein/ kg/d	Body weight (kg)	Total daily Requirement (g protein/d)
Infants[c] (months)						
6-9	1.69	7.9	13.4			
9-12	1.69	8.8	14.9			
Pre-school children (y)		Boys			Girls	
1-2	1.47	10.3	15.1	1.47	9.6	14.1
2-3	1.25	12.8	16.0	1.25	12.1	15.1
3-4	1.16	14.8	17.2	1.16	14.5	16.8
4-5	1.11	16.5	18.3	1.11	16.0	17.8
School children (y)		Boys			Girls	
5-6	1.09	18.2	19.8	1.09	17.7	19.3
6-7	1.15	20.4	23.5	1.15	20.0	23.0
7-8	1.17	22.7	26.6	1.17	22.3	26.1
8-9	1.18	25.2	29.7	1.18	25.0	29.5
9-10	1.18	28.0	33.0	1.18	27.6	32.6
Adolescents (y)		Boys			Girls	
10-11	1.18	30.8	36.3	1.18	31.2	36.8
11-12	1.16	34.1	39.6	1.15	34.8	40.0
12-13	1.15	38.0	43.7	1.14	39.0	44.5
13-14	1.15	43.3	49.8	1.13	43.4	49.0
14-15	1.14	48.0	54.7	1.12	47.1	52.8
15-16	1.13	51.5	58.2	1.09	49.4	53.8
16-17	1.12	54.3	60.8	1.07	51.3	54.9
17-18	1.10	56.5	62.2	1.06	52.8	56.0

[a] In terms of mixed Indian vegetarian diet protein (Annexure 5.1; PDCAAS varying from 77.4 to 79.0 % for different age groups, see Table 5.13)
[b] Requirements for each age band taken as the protein requirement for the lower age limit at that age band, see Tables 5.7 and 5.8.
[c] For infants below 6 months, see Table 5.6

5.9. 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 nitrogen balance (NB). The slope of N balance with increase in N intake is steeper at a higher intake than at a lower energy intake. This has been demonstrated both in children (5.11) and adults in India (5.12). On the basis of international data, the relation of nitrogen balance (NB) to nitrogen intake (NI) and energy intake (EI) is shown by the following formula.

$$NB = 0.17 \times NI + 1.006 \times EI - 69.13$$

The slope of this curve indicates that NB improves by 1mg/kg/day per extra 1 kcal/kg/day. In a study among preschool children, it was shown that protein requirement for N equilibrium was 1.13 g/kg at an energy intake of 80kcal/kg while it was 0.98 g/kg at an energy intake of 100 kcal/kg. Similarly, at an energy intake of 80 kcal/kg protein intake for 40mg N retention/kg, protein requirement was 1.64 g/kg while at an energy intake of 100 kcal/kg, it was 1.33 g/kg. Similar relationships between energy intake and protein intake for N equilibrium were observed in adults engaged in heavy manual labour (5.12) in India. The effect of varying energy intakes on two levels of protein intake i.e., 40 g and 60 g per day showed that, at 40 g protein intake, the energy intake for N equilibrium was 2249 kcal while it was 2066 kcal at a protein intake of 60g/d. These studies on children and adults indicate that the increase in protein intake to meet N equilibrium criteria, when the energy intake is lowered by 20%, is of lower magnitude in children than in adults.

Therefore, it is useful to consider together the protein and energy requirements on habitual Indian diets. Protein requirement of different age groups can be expressed as ratio of protein energy to dietary energy requirement (PE ratio). This PE ratio will differ for different ages and also between Indian adults engaged in different activities (lifestyles). If the protein content of the habitual diet is expressed as PE ratio, PE ratio of a diet will indicate whether a diet will meet the protein requirement of any group if adequate energy is consumed on that diet. This concept is useful since in any population group, enough food is not eaten to meet energy requirement, resulting in energy deficits. In Table 5.16, 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 through that diet. It will also indicate the level of energy intake below which protein also becomes deficient.

Table 5.16

Protein energy ratio for different age groups

Group		Protein requirement g/kg/d[a]	Energy requirement kcal/kg/d	PE Ratio of requirement	PE Ratio after adjusting for PDCAAS[f]
Pre-school children[b]	1-5 years	0.94	81	4.6	5.9
School childen[b]	6-10 years	0.91	71	5.1[c]	6.6
Adolescents[b]	11-18 y (Boys)	0.88	60	5.8[d]	7.4
	11-18 y (Girls)	0.86	55	6.3[e]	8.1
Adults					
Men (Sedentary)		0.83	39	8.5	10.3
Women (Sedentary)		0.83	36	9.2	11.2
Men (Moderate active)		0.83	46	7.2	8.7
Women (Moderate active)		0.83	42	7.9	9.6

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

[a] Safe requirement of *high quality protein*

[b] Assuming moderately active children

[c] If sedentary (PAL of 1.4), then PE ratio increases to 5.9

[d] If sedentary (PAL of 1.4), then PE ratio increases to 6.7

[e] If sedentary (PAL of 1.4), then PE ratio increases to 7.1

[f] PE ratio of the requirement adjusted for the PDCAAS value of the dietary protein in a standard Indian vegetarian low cost diet. In this case, using an Indian balanced diet protein based on a cereal/pulse/milk mix, with a PDCAAS of 77.4% for children up to 10 years age, 78.2% for children up to 18 years of age, and 82.5% for adults.

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, while the energy requirement changes, the PE ratio also changes, becoming higher with reducing energy requirement, as in sedentary people. This is important, since the required level of protein in the food will then depend on the activity levels. In addition, the PE ratio only indicates the total amount of protein in the diet.

Given that protein quality is also an important consideration (based on the new amino acid requirements), it is important to adjust the PE ratio for

protein quality. This adjustment achieved as the ratio of protein energy to the total energy of the diet, is calculated using the PDCAAS as an index of protein quality (PDCAAS-adjusted PE Ratio). Then, it is evident that with protein derived from a largely cereal-based diet, which is lysine-limiting, the PDCAAS will be less than 100. This will mean the need for a higher intake of this protein. Consequently, the PE ratio, calculated for a protein intake that was corrected for its quality (by PDCAAS), would increase.

It is also important to note how even the PDCAAS-adjusted PE ratio of the requirement does not increase beyond about 11% in both children and adults. In children, the PE ratio is usually low, owing to the high energy needs. The PE ratio will only increase in all these age groups, if the person becomes more and more sedentary; this pattern of habitual activity is unhealthy. In the elderly, there has been some suggestion that the protein requirement is increased. While there is no evidence that 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 energy requirement will lead to an increased PE ratio of the required diet. However, even in these circumstances, the PDCAAS-adjusted PE ratio will not rise beyond 13-14%. The solution to an increasing requirement of a high PE ratio lies in increasing physical activity, which will raise the energy needs, and reduce the PE ratio.

Therefore, while protein quality of the 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 unnecessarily high protein diets (with a PE ratio of greater than 15%) be routinely advocated. The use of commercial high protein supplements for the elderly, or for pregnant and lactating women, is not to be 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.

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Annexure 5.1

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

Food composition	Amount g/d	Protein content (g)	Other nutrients	
Cereals & Millets	460	46.0	Calories (kcal)	2736
Pulses (legumes)	40	9.5	Proteins (g) [b]	65
Green leafy vegetables	50	4.0	Calcium (mg)	781
Other vegetables	100		Iron (mg)	17
Roots & tubers	100		Vitamin A (µg)	715
Fruits[a]	50		Riboflavin (mg)	1.15
Milk	150	5.7	Thiamine (mg)	2.45
Fats & oils	40		Vitamin C (mg)	74.8
Sugar & Jaggery	30		Niacin (mg)	15.7
			Total fat (g)	67

[a] Additionally included
[b] Protein content depends on the type of protein-containing foods

Annexure 5.2

Typical Indian vegetarian high protein diet with a PE ratio of 12% for an adult woman during pregnancy

Food Groups	Low cost Indian vegetarian diet (From Annexure 5.1)		High protein vegetarian diet	
	Amount (g/d)	Protein content (g)	Amount (g/d)	Protein content (g)
Cereals & Millets	375	38	375	38
Pulses (legumes)	32	8	75	18
Green leafy vegetables	50	4.0	50	4.0
Other vegetables	100		100	
Roots & tubers	100		100	
Fruits	50		50	
Milk, milk based products	120	4.6	500	19
Visible fats & oils	32		30	
Sugar & Jaggery	25		20	
Calories (kcal)	**2223**		**2578**	
Proteins (g)	**54**		**78**	
PE Ratio (%)	**9.7**		**12.1**	

Note: Total diet energy / protein values do not exactly match the recommendation for the moderately active 55 kg woman (2230 kcal and 55 g protein) in Table 4.14, because of rounding off of food group intakes, as well as of nutrient values.

Additional Note: The PDCAAS of mixed food protein in the higher protein diet is about 0.9, owing to a higher limiting amino acid (lysine) content and a better digestibility, because of additional milk intake. Additional energy provided by this diet, in comparison to the standard diet, was 355 kcal, while the additional protein was 24 g. This vegetarian diet, with a PE ratio of 12%, would meet energy and protein requirements in the third trimester of pregnancy. Milk intake could be substituted by intake of milk products or curds. In sedentary women, milk intake should be increased to 600 ml/day, while visible fats, root vegetables and sugar intake should be reduced.

Additional note: The key changes between the diets are reflected in a change in the pulse: cereal ratio from less than 1:10 in the standard diet, to about 1:5 in the higher protein diet, by maintaining cereal intake, but markedly increasing pulse intake. In addition, milk intake was increased 4-fold from the standard diet, and visible fats slightly reduced to compensate the fat from milk. In the urban context, it is advisable to use toned milk, which would reduce fat intake.

6. FAT

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. During the past two decades, nutritional and health consequences of dietary fat and its fatty acids have been shown to be more varied and detrimental in humans than was understood hitherto.

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) is the predominant LC n-6 PUFA} and LC n-3 PUFAs {eicosapentaenoic acid (EPA) docosapentaenoic acid (DPA) and docosahexaenoic acid (DHA)} respectively (6.1). These are incorporated into the membrane lipids (6.1). 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 LCPUFAs (6.1). 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 (6.1, 6.2), are essential for development of central nervous system(6.3), modulate lipoprotein metabolism and risk for diet-related non-communicable diseases (DR-NCD), namely, coronary heart disease (CHD), diabetes and cancers (6.4-6.8).

a) Modulation of membrane structure and functions

As 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 (6.1, 6.2). 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 neuroprotectins D1 (from DHA). Lipoxins and resolvins (from both EPA and DHA) have potent anti-inflammatory effects and neuroprotectin D1 has potent anti-inflammatory and neuroprotective effects. 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 recommended (6.9), emphasis should be on increasing the absolute levels of ALA and LCn-3 PUFAs (6.10).

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 neurotransmission (6.1-6.3). 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 (6.11, 6.12). 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 (6.4-6.8, 6.13-6.15). 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 animals (6.16) 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 (6.5-6.7, 6.15). 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 (6.5, 6.8, 6.13).

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 (6.4, 6.5, and 6.9).

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 (oryzanols and sesame lignans) lower serum LDL cholesterol (6.17, 6.18). Vitamin E, carotenoids, sesame lignans, oryzanols and phenols 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 diets (6.18).

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 Consultation (6.19) 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 Consultation (6.9) 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) 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/day (6.4).

- 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 (6.3, 6.5-6.8, 6.11, 6.20). Following the same criteria employed in WHO/ FAO to define the strength of evidence (6.4), the 2008 Consultation used evidence of sufficient strength to be 'convincing' or 'probable' to formulate a dietary recommendation (6.21). Some of their conclusions and recommendations are as follows:

a) 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 trans-fatty acids (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 (6.15).

b) Based on epidemiologic studies and randomized controlled trials of CHD events, 6% 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.

c) 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.

d) *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.*

e) *In promoting the removal of TFA, 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.*

f) *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*

[a] *To 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 Indians (6.22) considered the FAO/WHO 1977 recommendations (6.19) 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

The small amount of fat present as integral component in each and every item of food (invisible fat), the fat in processed and ready to eat foods (hidden fat) and visible fat (vegetable oil, ghee, butter and vanaspati), used as cooking fat together contribute to total fat intake.

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 MUFAs and PUFAs. In most cereals, millets, legumes and pulses, fat content ranges between 1.5-3% (higher contents in maize, bajra, bengal gram 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 ALA (6.23). 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 PUFAs (6.24). 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 (6.24, 6.25 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 foods (6.24-6.26 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 (6.16, 6.23, 6.27).

Visible fats

Vegetable oil used in cooking is the major type of visible fat consumed; vanaspati 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 (PHVO) 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

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 [l]
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]	DHA AI 0.1- 0.18 [l m] U-AMDR no upper value, within the human range upto 1.5% E
	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	DHA AI 10-12mg/kg [l]
Children	3-6 y	U-AMDR 8 [a]	By difference [b]	<1	U-AMDR 11		[n]	[n]	AI 100-150 mg
	7-9 y								AI 200-250 mg
Boys	10–12 y	U-AMDR 8 [a]	By difference [b]	<1	U-AMDR 11		[n]	[n]	AI 200-250 mg
	13–15 y								
	16–17 y								
Girls	10–12 y	U-AMDR 8 [a]	By difference [b]	<1	U-AMDR 11		[n]	[n]	AI 200-250 mg
	13–15 y								
	16–17 y								

References : 6. 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;
[f] minimum 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;
[j] including 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)

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 MUFAs 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 oil (6.19). 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 fats, vegetable oils contain non-glycerides which have specific health significance. The composition of non-glyceride components in dietary fats and oils is given in Table 6.3.

6.5 Fat intake in Indians: An update

Total fat intake in the Indian population is income-dependent and therefore highly skewed, the intake being low among rural and urban poor income groups. Diet surveys by the National Nutrition Monitoring Bureau (6.29) 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) (6.30). 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). Efforts to increase the dietary levels of total fat and n-3 PUFAs in the rural population would contribute to lifelong health and well being.

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]	46	49 [g]	4	-

Reference 6.28

* SFAs include < C10:0, C12:0 (lauric), C14:0 (myrisitc), C16:0 (palmitic), C18:0 (stearic)

< C 10:0- [a]15, [b]9 [c]15; C12:0 and C14:0- [d]65, [e]14

** Mainly cis C18:1 (oleic) other MUFAs when present indicated against superscripts

TFAs [f]5; [g]17 (range 5-38, data compiled between 2000-2009);

SFAs and MUFAs

- C20-24 [h]1 to 4, [i]~8; [j]C22:1 (erucic) ~2, [k]C 22:1 (erucic) ~50 and C 20:1(gadoleic) ~ 5

Table 6.3
Non-glyceride components in dietary fats and oils

Nonglyceride components	Oil	Biological and health function
Plant sterols Vitamins A, D, K	All vegetable oils Ghee/butter	Hypocholesterolemic Vitamin
Tocopherols	All vegetable oils	Vitamin , Antioxidant
Tocotrienols	Palm oil , Rice bran oil	Vitamin , Antioxidant
Carotenes	Red Palm oil	Provitamin, Antioxidant
Oryzanols	Rice bran oil	Hypocholesterolemic Antioxidant
Sesamin	Sesame	Hypocholesterolemic Anti-inflammatory
Sesamolin, Sesamol	Sesame	Antioxidant

Reference: 6.28

In the urban middle and upper income groups, the daily intake of visible fat ranges between 22-45g/p/d (6.31-6.34) and total fat in their diets furnish 20-33% E (6.32-6.35). 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 (6.23, 6.16, 6.33). It is necessary to increase n-3 PUFAs in the diets of the urban segments for providing fat quality consistent with good health (6.23, 6.16, 6.27).

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 (6.4, 6.9, 6.15, 6.19, 6.21), 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 (6.23, 6.16, 6.27), ii) unfavourable effects of low fat-high carbohydrate diets (6.5-6.8) 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 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 (6.11, 6.12, 6.21). To furnish 20% E total fat, diets of pregnant and lactating women should contain at least 30 g visible fat (Table 6.4).

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[l]		
	7- 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 – 17 y				50
Girls	10 – 12 y				35
	13 – 15 y				40
	16 – 17 y				35

[a] Reference 6.21
[b] gradually 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
[l] infant formulae/ milk substitutes should mimic contents of fat and fatty acids in human milk including arachidonic and docosahexaenoic acid.

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 (6.11, 6.12 and 6.20). 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 (6.20, 6.21). 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) (6.20). 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 calories (6.37). 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 vitamins (6.36). The provision of dietary sources of DHA to infants should come from human milk / infant formula and complementary foods (Table 6.1).

Children and adolescents (2-17y): Total fat intake below 25%E is considered to affect growth in children and adolescents (6.20). 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 (6.20, 6.21).

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 (6.23, 6.27).

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 (6.23, 6.27). A long term 'in home' study with oil combinations (which increase ALA) showed improvement of LC n-3 PUFA nutritional status in adults (6.33).

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 6.5

Recommendations on type of visible fat*

1.	Use correct combination / blend of 2 or more vegetable oils (1:1)##
	*Oil containing LA + oil containing both LA and ALA (Table 6.1, Columns 8 & 9)**
	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**
	*Oil containing high LA + oil containing moderate or low LA ***(Table 6.1 Column 8)*
	Sunflower / Safflower + Palmolein[c] / Palm oil[c] / Olive
	Safflower / Sunflower + Groundnut / Sesame[a] / Ricebran[b] / cottonseed
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. 6.16, 6.18, 6.27, 6.38, 6.39.

#Wide sources as part of a well balanced diet
All vegetable oils contain tocopherols and plant sterols

[a]Sesame lignans, [b]oryzanols + tocotrienols, [c]tocotrienols, [d]vitamin 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)

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 6.16, 6.23, 6.25, 6.27, 6.38, 6.39.

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

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

References: 6.16, 6.23-6.26.

[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, bhekti, 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 (Mystus nemurus), red pomphret hilsa, 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 pasteurs have higher n-3 content than grain fed

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

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7. DIETARY FIBER

7.1. Introduction

The meaning of the term "Dietary Fiber" went through a dramatic change over the past 30 years and the virtues of dietary Fiber as a nutrient has received enormous attention in recent years. Earlier, the definition of fiber included all components of plant material that are not digested by the enzymes of the mammalian digestive system. This definition was widely accepted at one time; in recent years, however, non-inclusion of non-polysaccharide components such as lignins, phytic acid and others such as resistant starch in the above definition was questioned. The current definition of dietary fiber "Dietary fiber is the remnants of the edible part of plants and analogous carbohydrates that are resistant to digestion and absorption in the human small intestine with complete or partial fermentation in the human large intestine". It includes polysaccharides, oligosaccharides, lignin and associated plant substances. Dietary fiber exhibits one or more of either laxation (fecal bulking and softening; increased frequency; and or regularity), blood cholesterol attenuation, and or blood glucose attenuation (7.1). Organic acids (butyric acid) and polyols (sorbitol) are also considered as part of fiber. Animal foods do not contain any fiber (7.2).

7.2. Structure

Starch is the major component in the digestible complex carbohydrates. Starch is formed of α-glucosidic chain and is found in cereals, potatoes, legumes and other vegetables. Two main constituents of starch are amylose (which has a non-branching helical structure) and amylopectin (which consists of branched chains of 24-30 glucose residues linked by 1→ 4 linkages with 1→ 6, linkages at branch points). Dextrins are formed in the course of breakdown of the starch molecule. Glycogen is more branched than starch and is an animal storage form of carbohydrate. Cellulose, the chief carbohydrate from plants is not soluble in ordinary solvents and consists of units of β D-glucopyronose in β 1→ 4 bonds and many parallel chains are cross-linked. β-glucan, on the other hand, is a linear, unbranched polysaccharide with β-(1→3)- and β-(1→4)-D-glucopyranose units. It occurs in the bran of grains such as barley, oats, and wheat. β-glucan and inulin are soluble polysaccharides forming important components of dietary fiber and are shown to be 'prebiotic' which promote microbial growth and control blood sugar and lipids (7.2).

7.3. Digestion of carbohydrates

Polysaccharides, like starch and cellulose, from plant foods and glycogen from foods of animal origin are the most abundant carbohydrates that human

beings eat. Starch and glycogen are completely hydrolyzed by enzyme action to yield free D-glucose. The process is started in the mouth on chewing and acted upon by salivary amylase which breaks many alpha (1→4) linkages in starch and glycogen, leading to a mixture of maltose, glucose and oligosaccharides. This process is continued in the small intestine by the action of pancreatic amylase secreted from pancreas into duodenum. Cellulose cannot be hydrolyzed by many mammals as they do not have the enzyme to break β(1→4) linkages. This undigested cellulose from plant foods forms 'roughage' or 'Fiber' and is essential for bowel movement (7.2).

7.4. Type and sources

Dietary fiber has been characterized by its source (cereal, vegetable and fruits) or its solubility in water (soluble, partly or fully and insoluble, both characters being essential for health promotion). Digestibility of fiber is determined by the physicochemical and structural properties of the dietary component and the process used (7.2). When exposed to longer duration of degradative conditions in the large intestine, more fiber is digested. It forms the substrate for fermentation by intestinal microbes. It is through this mechanism that part of the energy in carbohydrates, having β linkages resistant to human digestion (resistant starch) is rendered available. Apart from energy yielding reactions, in view of the solubility changes brought about in the intestine during digestion at different pH and other conditions and the produced intermediates of enzymatic digestion, dietary fiber not only promotes interactions between nutrients but also changes the pattern of microbes colonizing the colon and thus the metabolic products of such fermentation over time. Vegetarians may have different digestion pattern over that of the non-vegetarians and thus derive different health benefits. Thus the whole area of probiotics (characterized by helpful microbes) and prebiotics (substrates promoting the colonization of probiotic strains) opened up with this knowledge.

Original estimates of fiber covered all that was insoluble in boiling water or in dilute acid and alkali conditions (determined by chemical / gravimetric procedures). It was reported as 'crude fiber', which may include all structural fiber, cellulose, lignin and hemicellulose and was more a methodological result. On the other hand, dietary fiber is related to digestibility and has the property of holding water and swelling properties of the diet (it requires enzymatic and component analysis). It adds to the bulk of the food, favours satiety, increases transit time of the food in the gut and is an active substrate in the large intestine for release of important functional components like organic acids and nutraceuticals. Mostly complex carbohydrates, such as polysaccharides (cellulose, hemicellulose, pectin and a variety of gums, mucilages) form the fiber and enzymatic methods coupled with component analysis are used (7.2).

7.5. Nutritional and health significance

Though fiber has been the most important component of the Indian diets, its benefits have been overlooked for a long time. There has been overwhelming evidence that 'fiber' is essential to maintain body weight and composition, blood levels of sugar (low glycemic index), triglycerides and cholesterol (binding by fiber components and increased excretion). Regulatory bodies like FDA have approved health claim label of many sources of good fiber, the soluble fiber (7.3-7.5). Soluble fiber is derived from a variety of sources and the widely known ones are modified 'maltodextrins" (reduces blood glucose, promotes growth of healthy bacteria), inulin (from wheat, onions, banana and chicory or synthesized) a probiotic and laxative in nature or oligofructose (fructo oligisaccharides, shown to be associated with inulin or is formed as a byproduct of bacterial or fungal action on inulin. Insoluble fiber is composed of structural components of plant cells. Cereals, seeds, beans, many fruits and vegetables, bran and whole grain are food sources of insoluble fiber (7.2, 7.3, and 7.6). These fibrous compounds may also help to promote weight loss (7.7), reduce risk of colon cancer (7.8) and heart disease (7.9-7.10). One Indian study also found decreased risk of cardiovascular disease through better control of lipoprotein lipids in those supplemented with good fiber (7.11). Some foods containing different fractions of soluble and insoluble fibers favour slow release of sugar into small intestine and its absorption into blood (reduced peak and prolonged rate). They are therefore termed low glycemic index foods as compared to high glycemic foods with readily digestible and absorbable sugar, having practical utility in management of diabetes and control of obesity (7.12, 7.13) Table 7.1.

Dietary fiber has been shown to inhibit absorption of nutrients like minerals and some vitamins. However, the inhibitory effect was found to be confined to an insoluble fiber component (7.2, 7.6, 7.14). In fact, soluble fibers such as inulin and their oligopolysaccharides were shown to promote absorption of Mg, Ca, Fe and Zn by increasing permeability. Insoluble fiber binds the above 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 (7.2, 7.4).

Dietary fiber was found to be inversely related with incidence of colo-rectal cancers (7.4, 7.9). Direct interventional studies showed that soluble fiber could also reduce the risk of colon cancer development. Levels of harmful chemicals as markers of cancer in the digestive tract have been demonstrated. Vegetable and fruit fiber intake was found to have a big gradient in the incidence of laryngeal cancers (7.9).

Table 7.1

Examples of low glycemic index (GI)* foods

Classification	GI range	Examples
Low GI	55 or less	most fruit and vegetables (except potatoes, watermelon and sweet corn), whole grains, pasta foods, beans, lentils
Medium GI	56 – 69	sucrose, basmati rice, brown rice
High GI	70 or more	corn flakes, baked potato, some white rice varieties (e.g. jasmine), white bread, candy bar and syrupy foods

* Glycemic index of a food is defined by the area under the two-hour blood glucose response curve (AUC) following the ingestion of a fixed portion of test carbohydrate (usually 50 g) as a proportion (%) of the AUC of the standard (either glucose or white bread)

7.6. Requirements

Traditionally, fiber was considered as a non-nutrient component of food in the Indian context. Our emphasis on fiber has been confined to crude fiber content of foods and there was no evidence that Indian dietaries were deficient in crude fiber content (7.15, 7.16). Only recently Indian data base has been updated methodologically to include the total, insoluble and soluble fractions of dietary fiber and is reported in four publications (7.17-7.20). A brief summary of results on typical values for fiber component of cereals, pulses, vegetables and fruits is presented in Table 7.2. For comparison, the crude fiber values are given along with total fiber and percent soluble fiber data in these foods. In general, the total dietary fiber content is almost 2-3 fold higher than the crude fiber values of the foods. Thus there is no chance of Indian diets being deficient in fibers. While computing carbohydrates, only crude fiber was deducted, while total dietary fiber will be higher, giving a higher value for CHO; on the other hand soluble fiber undergoing fermentation in the colon would provide 2 Kcal per g of fiber that undergoes fermentation. Hence kcal content of food would be lower.

Apart from providing authentic information on fiber content, these data also revealed that the energy content of various foods was reported about 10% higher due to an underestimation of "fiber" as "crude fiber" content of cereals and pulses (Table 7.3). The new information on soluble and insoluble fiber levels in different foods has not been added to the original database

Table 7.2

Different dietary fiber fractions in selected Indian foods

Food Group	Food Item	Fiber content g /100g edible portion			Soluble (% TDF*)
		Crude Fiber	TDF*	Soluble	
Cereals	Rice	0.2	4.11	0.92	22.4
	Wheat	0.3	12.48	2.84	22.7
	Bajra	1.2	11.33	2.19	19.3
	Maize	2.7	11.54	1.65	14.2
	Jowar	1.6	9.67	1.64	17.0
	Ragi	3.6	11.85	0.89	7.50
Pulses, dhals	Lentil	0.7	10.31	2.04	19.8
	Chick pea	1.2	15.30	2.56	16.7
	Pigeon Pea	0.9	9.14	2.33	25.4
	Green gram	0.8	8.23	1.69	20.5
Vegetables	Cluster beans	3.2	5.7	1.6	28.0
	Brinjal	1.3	6.3	1.7	27.0
	Cabbage		2.8	0.8	28.6
	Cauliflower	1.2	3.7	1.1	30.3
	Bhendi	1.2	3.6	1.0	26.9
Roots and Tubers	Potato	0.4	1.7	0.6	33.5
	Carrot	1.2	4.4	1.4	30.6
	Onion	0.6	2.5	0.8	32.0
Green leafy Vegetables	Spinach	0.6	2.5	0.7	28.0
	Amaranth	1.0	4.0	0.9	22.5
Fruits	Orange	0.3	1.1	0.5	45.5
	Banana	0.4	1.8	0.7	38.9
	Apple	1.0	3.2	0.9	28.1
	Tomato	0.8	1.7	0.5	28.5

Reference 7.17-7.20
* Total dietary Fiber

(7.15). The energy values of the foods need to be re-evaluated. With economic transition, health transition too, has taken place in India, resulting in abdominal obesity, insulin resistance and hypertension and cardiovascular risk aggravating the burden of diseases among its people. With more and more proportion of them shifting to processed, refined and convenient foods, 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. A study by Joshi and Agte (7.21) estimated the digestibility of neutral detergent fiber of diets with wheat and rice as predominant cereals (Table 7.5). 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 data base 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.

Table 7.3

Reported and available energy (kcal/100 g) and carbohydrate (g/100 g) content of food grains

Food	Reported energy content[1]	Total fat[1]	Protein[1]	Total dietary fiber[1]	Carbohydrate available[2] and (total[1])	Available energy content[3] (% lower)[4]
Cereals						
Rice	345	0.5	6.8	4.1	74.3 (78.2)	329 (4.6)
Wheat	346	1.5	11.8	12.5	59.9 (71.2)	300 (13.2)
Sorghum	349	1.9	10.4	9.7	64.5 (72.6)	317 (9.2)
Ragi	328	1.3	7.3	11.5	63.8 (72.0)	296 (9.8)
Bajra	361	5.0	11.6	11.3	57.4 (67.5)	321 (11.0)
Maize, dry	342	3.6	11.1	11.9	57.4 (66.2)	307 (10.2)
Pulse/ dhals						
Red gram	335	1.7	22.3	9.1	50.0 (57.6)	305 (8.9)
Black gram	347	1.4	24.0	11.7	48.8 (59.6)	304 (12.4)
Green gram	348	1.2	24.5	8.2	52.5 (59.9)	318 (8.6)
Bengal gram	372	5.6	20.8	15.3	45.7 (59.7)	316 (15.1)
Average of all foods	347	2.5	15.1	10.5	57.4 (66.5)	311 (10.4)

[1] Reference 7.15
[2] Available carbohydrate = weight g/100 g [Food – (Moisture + Fat+ Protein+ Fiber+ Minerals)]
[3] Food energy conversion factors (kcal/g) : Fat= 9, protein= 4 and carbohydrate = 4
[4] Extent of overestimation in Food Table

Data from diets of the Western part of the country show that the dietary fiber content is about 30-40 g /d (7.22). Intake was increasing with increasing level of energy intake, 39 g -47 g/d in young men (7.23). The fiber intake is lower in women (15-30 g/d) and is much less in tribal population (15-19 g/d) (7.24) (Table 7.4). Another report from North India shows that the average total fiber intake per day is about 52 g (7.25). More data need to be generated in the Indian context to understand the phenomenon of health transition.

Table 7.4

Estimates of 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 7.23
LIG, MIG, HIG: Low, middle and high socioeconomic groups

Table 7.5

Digestibility of dietary fiber reported from Western India

Fiber component	Intake g/d	Digestibility (%)
Cellulose	18.2	30.0
Hemicellulose	12.8	53.4
Lignin	6.7	8.1
Neutral detergent fiber (total)	37.7	34.1

Reference 7.20

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 fibre 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 Agencies that a minimum intake of 20-35 g of fiber is conducive for long-term good health (7.2, 7.6) can be a positive guideline. Later, the amount of fiber was enhanced to 40g/d (7.4). Even the WHO Committee on chronic degenerative diseases recommended a daily intake of 30 g dietary fiber (7.27). 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 diarrhoea. Based on energy intake, a level of about 40g/2000kcal 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.

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8. MINERALS

8.1. CALCIUM AND PHOSPHORUS

The requirements of calcium 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 urine, stools, bile and sweat. It is estimated that in an adult man this loss may be 700 mg/day. However, the body can reduce this loss on a low Ca intake through a process of adaptation involving reduced excretion. 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 Ca in milk secreted.

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. Other rich sources of Ca among plant foods are the millet ragi (*Eleusine Coracana*), the pseudo cereal, rajkeera (*Amaranthus*) and the green leafy vegetables.

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 earlier Committee (8.1.1). The review of Nordin, 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 intake (8.1.2). 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 IOM Committee (8.1.3) and WHO Expert Group (8.1.4). 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 (Table given in Annexure 8.3).

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, and 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 DXA, 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 multicentric study supported by ICMR has generated large data base on bone health of Indian population using DEXA for the first time (8.1.5) but the data have not been published yet. Though these studies were not aimed at obtaining requirements of calcium,

results of a few aspects of that study which are relevant to the exercise of determining RDA of calcium are made available to the Expert Committee by the investigators/ ICMR and these have been considered in the present report.

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, the earlier Committee (1988) (8.1.1) suggested a lower level of calcium intake. However, after examining the evidence for Ca nutrition status of the Indian population the present Committee suggests an upward revision of calcium RDA. More than 50 % of the adult male and 45% female population are reported to be obtaining the RDA level of 400 mg/d (8.1.6). 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 group (8.1.7). There have been reports of moderately higher levels of circulating parathyroid hormone in Indians suggesting strains on calcium economy (8.1.8).

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 (8.1.9, 8.1.10). 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 activity (8.1.11) aggravating the situation. Current level of consumption providing less than 400 mg Ca/ d/ CU (8.1.12,) 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. As such, the previous Committee in 1988 (8.1.1) opted to reduce the range of earlier recommendations (1980) (8.1.13) in adults to a single lower figure of 400 mg/d assuming that it would be adequate as no adverse changes were reported on such lower intakes at that time. However, the present Committee, in view of the later evidences as indicated above, chose the upper value of calcium requirements for adults (600 mg/d), which also ensures that the values telescope well to those recommended for adolescents. A reevaluation of all the calcium balance studies carried out on Indian adults published earlier (8.1.14-8.1.18) studies involving a minimum 4 adult men or women at each dose level). Fig 8.1b 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 works out to 600 mg/d (Table 8.1.3).

A review of the available ICMR multicentric studies on bone health in Indian men and women is appropriate here (8.1.5). 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) and densities at 3 sites were obtained in 100 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.

Table 8.1.1

DEXA indicators of bone health in reference groups

Gender	n	BMI	Ca intake mg/d	Peak WB BMC g	Age for peak mass (y)	Peak bone mineral densities at sites g/cm²		
						hip	Fore-arm	spine
Male	104	23.0	1075	2472 ± 314	25	1.000 (-0.21)	0.618 (-0.78)	1.027 (-0.93)
Female	106	22.5	990	2026 ± 205	28	0.989 (-0.13)	0.547 (-0.03)	0.934 (-0.53)

Values in parentheses indicate the corresponding z scores.

The results show that WBBMC peaked (cross-sectionally) at age 25 and 28 years, respectively, in reference group of Indian men and women. 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.

The second part of the ICMR multicentric 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 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 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
1st	<344	2088 ± 22[a]	<326	1611 ± 20[a]
2nd	344-581	2137 ± 21[ab]	326-540	1675 ± 19[b]
3rd	581-872	2171 ± 21[b]	540-811	1730 ± 20[c]
4th	>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.

An update of the current recommendations of calcium requirements in adults may be separately considered at a later date when sufficient data are available or a suitable analysis of the ICMR muticentric data is carried out to draw firm conclusions.

Based on the current evidence from ICMR muticentric studies using DXA, the peak BMC attained is 2472 g in healthy normal reference males and 2026 g in females at ages 25 and 28 years, respectively. At the end of 15 years of age, the BMC was estimated to be 1800g in middle income semi-urban Indian children below 10 years age habitually consuming 600-800 mg Ca/d (cross-sectional) (8.1.19). From this age, the gap in BMC to achieve peak mass is 175g in girls/women in 13 years and 672g in boys/ men in 10years (i.e, 12

mg/d Ca retention in women and 62 mg/d in men). With a reported accretion from balance or from DEXA studies in Indian subjects, it is quite possible to attain such BMC status with intakes close to 600-800 mg/d in adults 20-30 years of age.

Unlike the past, recently there is a significant increase in the consumption of milk and milk products raising the level of calcium intake. Along with calcium intake, animal protein intake is also expected to increase. Therefore, the suggested intake is very practical and necessary for maintaining good bone health.

Table 8.1.3

RDA of calcium for various physiological groups

Group	Category / Age	Calcium* (mg/d)
Men		600
Women		600
Pregnant women		1200
Lactating women		
Post-menopausal women		800
Infants		500
Children	1 - 3 y	600
	4 - 6 y	600
	7 - 9 y	600
Boys	10-12 y	800
Girls		
Boys	13-15 y	800
Girls		
Boys	16-17 y	800
Girls		

* To achieve this level of intake, a minimum of 200 ml of milk/d would be essential on a cereal-legume diet.

Balance data from adults have indicated that intakes in the range of 500-600 mg/d are necessary for positive calcium balance. Using factorial method, calcium intake needed to maintain calcium adequacy was obtained for pregnant and lactating women using same rationale employed by the earlier Committee except that the basic requirements were raised by 200 mg/d. Information available from clinical trials in which additional calcium was given and then changes in bone mineral content were measured over time, was used for confirming the requirements in children. An example of calculating calcium requirements based on factorial method for adolescents during peak accretion by the IOM (8.1.3) is given in Table 8.1.4.

Table 8.1.4

Illustration showing factorial approach for determining calcium requirements (mg/d) during peak calcium accretion in the adolescents by IOM (Ref 8.1.10)

Compartments	Girls	Boys
Peak calcium accretion	212	282
Urinary losses	106	127
Endogenous fecal calcium	112	108
Sweat losses	55	55
Total	485	572
Absorption, percent	38	38
Adjusted for absorption	1,276	1,505

Children

Calcium balance studies (short-term) reported earlier were on children, mainly of 3-11 years of the pre-pubertal age (8.1.20). Two more balance studies had been carried out on children aged 3-7 years (n= 3 or 4). The levels of calcium studied were 76, 174, 450 and 610mg/d with energy intake varying between 945-1300 kcal/d. Data from each balance was utilized for interpretation. 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 Figs 8.1.1 and 8.1.2.

The data were used to generate two types of information as is evident from the figures. 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.

Where intake (X) and retention (Y) were related:

$$Y = -78.5 + 0.44\ X$$

Figure 8.1.1 Intake and excretion of Ca in adolescents

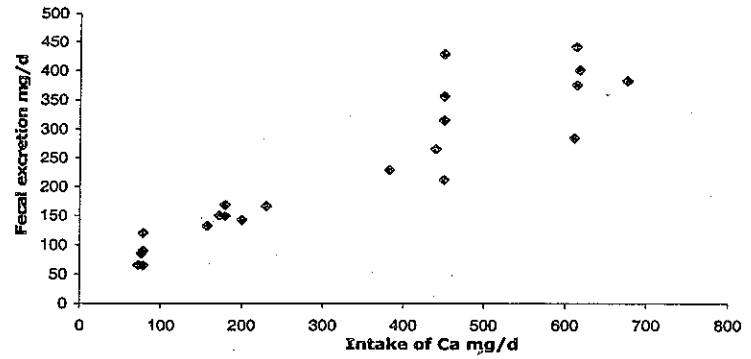
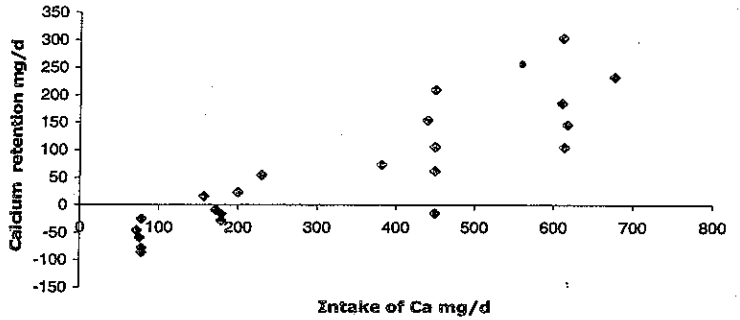


Figure 8.1.2 Intake and retention of Ca in adolescents



References : 8.1.20, 23, 24

At an intake of 200 mg, there was 0 mg retention as total losses are equal to the intake. The DRI Committee of IOM (8.1.3) recommended the calcium requirement of 3-9 year children to be 125-130 mg at the minimum level. Considering a retention value of 125 mg for full longitudinal bone growth and considering about 30mg/d for sweat losses, and taking mean + 2SD level (2 X15%= 30%), the total retained calcium needed to meet requirement is 200mg. To obtain this, the dietary intake is expected to be 600mg assuming 33% absorption. This level of intake would ensure the requirements of calcium for most of the pre-pubertal population (8.1.21).

The other relationship with intake (X) and fecal excretion (Y) is as: Y = 50+ 0.5154 X.

At 0 mg intake, 50 mg of fecal calcium excretion is predicted, which will be the endogenous fecal loss. The regression equation shows that about 40% dietary calcium is absorbed in the range of values tested. On the other hand, the urinary loss was not linearly, or otherwise, related to intake. On an average, 48 mg of calcium in urine was lost at any intake. Thus, the total obligatory loss works out to 100 mg for a child with mean age of 6+ years and expected body weight of 25 kg arriving at an obligatory loss of 3-4mg /kg body weight. This is close to the figure of 3 mg reported earlier for young adults and it can be expected to be a little more for young children (8.1.22). By using factorial approach, the requirement will be fecal (50) + urine (48) + sweat (30) +retention (125) = 253 mg. For 253 mg to be retained at a rate of 40% absorption noted, the dietary calcium needed is 600 mg, again arriving at the same figure as above.

The above dietary calcium requirement was confirmed from DEXA bone mineral content accretion data in children (discussed in the following section on adolescents).

Adolescents

Pubertal growth phase and peak bone velocity during the most rapid phase of skeletal development, particularly in the girls, has been the main focus of many DEXA studies in the west (8.1.25). It is calculated that in two years of this phase between 12-14 years, there is accretion of about 20% of the peak total bone mineral content found in adulthood. Many randomized clinical trials too have been carried out with supplemental calcium to determine optimum intake of calcium for building up the maximum bone mineral content during the growth phase so that they will be better protected from inevitable losses that occur later in life. IOM as well as WHO Expert Committees estimated the calcium requirements in pubertal age children based on intakes that ensure maximum peak velocity of bone accretion. Bone

accretion data from long term balance studies and DEXA have been used. The actual dietary intakes are calculated factorially from the calcium needs of bone growth and calcium losses (in urine, faeces and sweat etc) after adjusting for intestinal absorption. However both the Committees (8.1.3, 8.1.4) cautioned that desirable calcium retention figures vary according to populations studied and the data from West cannot be extrapolated to developing countries. The particulars of the comparative figures of estimates of calcium accretion in children along with an example of factorial calculation used by IOM are provided in Tables 8.1.6 and 8.1.7.

Unfortunately, there are no calcium balance data on pubertal boys or girls from India. An interesting exercise on calcium requirements for adolescents was made in a review article (8.1.26). The calcium deposited was arrived at theoretically considering the body weights at different ages of American girls and boys between the ages 10-18 years as reference and the longitudinal changes in their body composition, particularly, the increases in skeletal mass. Adding endogenous loss to it and using the true rate of calcium absorption rate as 60%, dietary calcium need was worked out by the author. The requirements varied between 487-701 mg/d for boys and 300-550 for girls in the age group of 11-16 years (Table 8.1.4).

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 supplementation (8.1.19).

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 8.1.5). 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 8.1.5 and 8.1.6. 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 (Table 8.1.7). 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 8.1.5 & 8.1.6).

Table 8.1.5

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

Source	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 (%)
8.1.27	94	11.9 ± 0.5	F	18	960	1,314	13.32	Total and Lumbar 10
8.1.21	48	9–13	F	12	728	1,200	35.52	Total and Lumbar 7-10
8.1.19	122	7-16 11.5	F M	14	745	1145	22.00	Femur neck 11
8.1.22	82	12.5± 0.3	F	18	600	1125	37.0	Total body 11
8.1.28	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 8.1.5 and 8.1.6). 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 8.1.5). 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 8.1.5 –8.1.7).

It is long 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 deficiency (8.1.4) 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 8.1.5 and 8.1.6). 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 8.1.3). It may also be noted that with increasing intake, the calculated net absorption also decreased in children (Table 8.1.6).

Table 8.1.6

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 used (8.1.19). 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.

In some studies, accretion rates are determined by a combination of two methods,- Balance and DEXA or Isotope and DEXA, particularly in the follow up studies after supplementation. In some cases, the accretion rate was at peak age and this cannot be applied at all other ages.

Table 8.1.7

Calcium accretion rates in adolescents

Source	Type of study	Age (Years)	Ca intake (mg/d)	Ca retention (mg/d)	Absorption (%)
8.1.26	Gain in body and skeletal mass and Ca kinetics	10-18 Male Female	500-700 500-550	292-421 305-330	60
8.1.29	Stable isotope	5-12	907	130	28
8.1.30	BMC, Suppl.	6-14	908 Placebo 1612 Suppl.	130	
8.1.31	Balance studies	12-15 Boys Girls	823-2164	282 212	
8.1.32	Stable isotope	11-14 Girls 19-31 Women	1330	494 283	38 22
8.1.33, 8.1.34	Balance	11-14 Girls 19-30 Women 12-15 Boys.	791 1019 700-2001	326 171 171	
8.1.25	DEXA Velocity	8-14 Boys Girls	1140 1113	359 284	36.5 29.6

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/WHO (8.1.4) 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 females (8.1.35). 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 (8.1.5) 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.

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 (1988) (8.1.1) 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 8.1.8).

Table 8.1.8

RDA of phosphorus for various physiological groups

Group	Category / Age	Phosphorus (mg/d)
Men		600
Women		600
Pregnant women		1200
Lactating women		
Post-menopausal women		600-800
Infants		750
Children	1 - 3 y	600
	4 - 6 y	600
	7 - 9 y	600
Boys	10-12 y	800
Girls		
Boys	13-15 y	800
Girls		
Boys	16–17 y	800
Girls		

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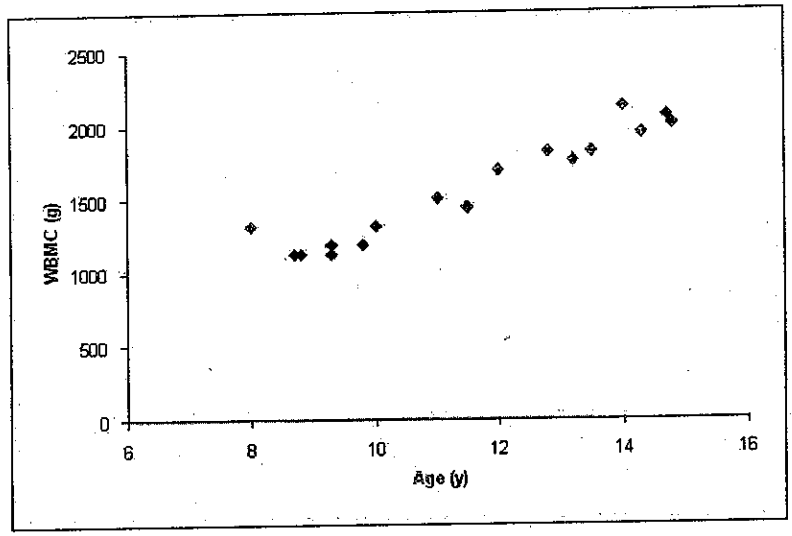
Age related changes in whole body mineral content of school children

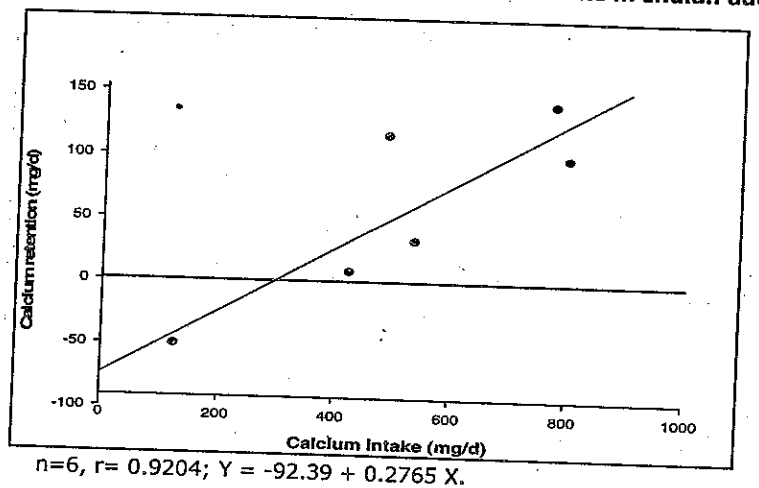


Whole body mineral content of middle-income, semi-urban school children is plotted against their mean age. The mineral content increased at a slower rate below the age of 11.5 y (with a slope of about 116 g/y) as compared to the pubertal spurt (with a slope of 170 g/y) during 11-16 y.
Reference: 8.1.19

found to be associated with other disease states mostly as inborn errors of

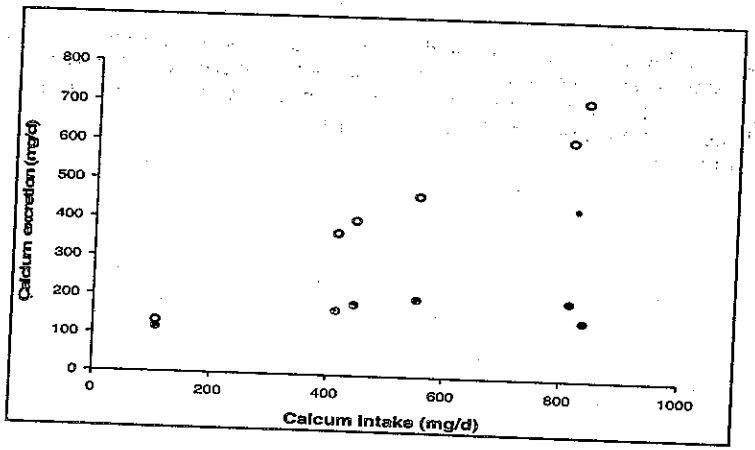
Annexure 8.2

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).
References: 8.1.14-8.1.18

muscle contraction. This also explains the key role of magnesium in

Annexure 8.3

Calcium requirements recommended by FAO/WHO (2004) and ICMR 1989

Category	FAO/WHO 2004		ICMR, RDA 1989 (mg/d)
	High protein intake, developed country (mg/d)	Low protein intake, less developed country (mg/d)	
Infants	400	300*	500
Children 1-3 y	500	500	400
4-6 y	600	550	400
7-9 y	700	700	400
Adolescents 10-18 y	1300	1000**	600
Adult females	1000	750	400
Pregnant women	1200	800	1000
Lactating women	1000	750	1000
Post menopausal women	1300	800	NA
Adult men 19-65 y	1000	750	400
Adult men 65+ y	1300	800	NA

References: 8.1.1, 8.1.4
* Human milk
** particularly during growth spurt

8.2. MAGNESIUM

Since the report of McCollum *et al* (8.2.1) 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, about 60-70% 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 on 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, absorption of magnesium ranged from 50-70% and around 25-30% in self-elected diet over long periods. 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%.

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, subjects suffer often from tetany and convulsions. Hypomagnesemia, hypocalcemia and hypokalemia are always associated with magnesium deficiency and are reversed by magnesium repletion. A syndrome of magnesium-dependent, vitamin D-resistant rickets was described in Indian child case reports (8.2.2).

8.2.3 Biochemical function

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 maintaining health of heart 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 relation to bone health and hypertension. A large number of interventional and clinical studies reveal variable effects of magnesium supplementation on essential hypertension (8.2.3) and pregnancy-induced hypertension and pre-eclampsia (8.2.4, 8.2.5), other than the associated changes that take place due to hypokalemia and its correction.

Lower levels of plasma magnesium and zinc and reduced zinc / copper ratio were associated with coronary artery disease in Indians (8.2.6). Dietary intakes of magnesium reported were 430 mg and 370 mg/d in rural and urban population respectively, confirming the earlier reports (8.2.7). Further studies in India have also reported similar intakes of magnesium. (8.2.8, 8.2.9).

8.2.4 Requirements

Requirement of magnesium has been worked out for adults based on the balance studies and tracer turnover studies. 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 on different regional diets in India. The absorption also varied between 20-50%. At any intake tested, retention was positive and was above 20-30 mg/d. (8.2.7).

These intakes suggest that there is no scope for Mg deficiency as the recommendations of FAO/WHO were 260 mg/d for adult males (8.2.10) and 420 mg/d according to US National Academy of Sciences (8.2.11).

Studies carried out in different physiological groups like infants using stable isotopic studies on absorption of Mg from maternal milk have indicated an absorption between 50 and 90 % (8.2.12). A 14-day balance study in 26 adolescent females showed that 52 ± 8 % Mg is absorbed from a diet containing 176mg of Mg/d (8.2.13). On the basis of 50% absorption, FAO/WHO has recommended desirable intake of Mg as 4 mg/kg/day for international use (8.2.10).

Table 8.2.1

RDA of Magnesium for various physiological groups

Group	Category/ Age	Magnesium (mg/kg/d)	Magnesium (mg/d)
Men	Sedentary work	5.7	340
	Moderate work		
	Heavy work		
Women	Sedentary work	5.7	310
	Moderate work		
	Heavy work		
	Pregnant		
	Lactating		
Infants	0-6 m	6.0	30
	6-12 m	5.5	45
Children	1-3 y	4.0	50
	4-6 y	4.0	70
	7-9 y	4.0	100
Boys	10-12 y	3.5	120
Girls	10-12 y	4.5	160
Boys	13-15 y	3.5	165
Girls	13-15 y	4.5	210
Boys	16-17 y	3.5	195
Girls	16-17 y	4.5	235

Figure 8.2.1
Correlation between magnesium intake and fecal excretion

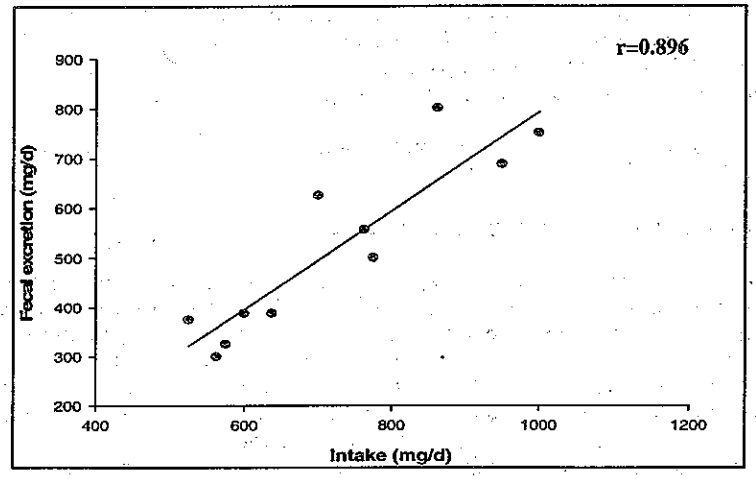
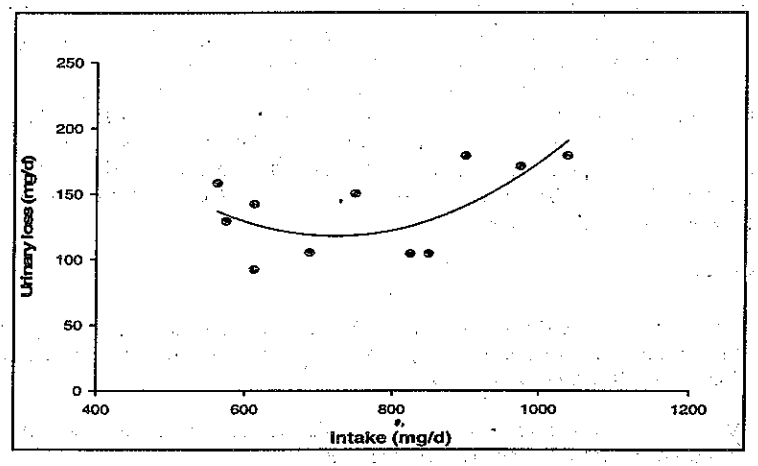


Figure 8.2.2
Relationship between magnesium intake and urinary loss



It has been observed that absorption of Mg among Indian adults from diets of different regions of the country and among different income groups (8.2.2) is close to 35%. Since FAO/WHO recommendations are applicable for international use, they can also be used for Indians after adjusting for lower absorption of dietary Mg, except for infants who derive their Mg through breast milk or formula milk. The Committee decided to use FAO/WHO recommendation for all age groups except adults where sufficient Indian data are available. These recommendations are given in Table 8.2.1.

There is a need for some renewed studies on Mg requirements for fixing RDA for Indians.

An exercise in determining Safe Intakes

The data of Rao and Rao (8.2.7) were reanalyzed for determining the safe intakes. 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. Computation of the requirements based directly on retention data obviously becomes difficult. However, there was a significant positive correlation between intake and faecal loss (Fig 8.2.1). 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 faecal loss was 184 mg at '0' intake and this, in principle corresponds to 'obligatory faecal loss'. Also there was a curvilinear relationship between intake and urinary excretion (Fig 8.2.2). 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. It 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.

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8.3. SODIUM

8.3.1. Introduction

Sodium is the principal cation of extracellular fluid and is involved primarily in the maintenance of osmotic equilibrium and extracellular fluid volume. Sodium functions as the "osmotic skeleton" of the extracellular fluid. The sodium-potassium gradient across the cellular membrane is maintained by a sodium-potassium pump - sodium potassium ATPase activity, which uses considerable energy in the body at rest. Sodium is essential for normal growth and survival. Sodium ingested in the diet is absorbed from the gastrointestinal tract. The body content of sodium and its concentrations in body fluids are under homeostatic 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. 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. Since potassium in the diet is high, animals/man have no such elaborate complicated mechanisms of potassium conservation.

Body needs of sodium are not great. Intakes of 1.1-3.3 g of sodium or 2.8-8.3 NaCl per day are considered to be safe and adequate for healthy adults, by the Food and Nutrition Board of National Academy of Sciences (8.3.1). Most dietary sodium is found in the form of sodium chloride, which is 40% sodium and 60% chloride. Excess sodium in the diet is said to contribute to hypertension in genetically prone individuals. Societies differ greatly in respect of the amount of sodium chloride consumed and most often this is culturally determined and is related to food habits. Therefore it is necessary to consider sodium requirements and the limits of safe intake to avoid metabolic consequences of excess intake of sodium chloride.

Salt, from time immemorial, has been used as a food preservative. Although salt is also used more with carbohydrates as a taste enhancer, salted fish and meat have been important components of the diet from very early times. However, epidemiological studies document certain populations, habitually on very low sodium intakes. Records of primitive people suggest that they did not use much salt and were free from hypertension (8.3.2).

8.3.2. Sodium sources

In Western countries major sources of sodium are processed foods including snacks where it is used as a flavour intensifier and curing agent.

Almost 1/3 of salt is present in food before processing, 1/3 is added during cooking and another 1/3 is added at the table. The salt content of natural diets, predominantly plant-based foods in India, does not exceed 300-400 mg of Na (1 g of NaCl). Diet provides 90% sodium from sodium chloride (salt) and only 10-15% originates from natural foods.

The NNMB data (1987) (8.3.3) indicate that salt consumption (added NaCl) ranges from <5 to 30 g in different states with almost 40% of families consuming around 10 g of NaCl/day. The amount of sodium ingested through water sources can differ from place to place. Sodium content of 20 mg-235 mg/L have been reported. Sodium content of water can be of a problem when severe salt-restricted diet is to be used.

8.3.3. Total body content of sodium

The normal distribution of sodium ion is in the extracellular fluid. Total body sodium is around 4000 mmol in a 75 kg man with more than half of it being in the extracellular fluid. About 300 mmol is in the intra-cellular compartment. About 1500 mmol is incorporated into the structure of crystals or minerals of bone and is not readily exchangeable. Concentration of sodium in the extracellular fluid is around 145mmol/L and in the intracellular fluid, about 10 mmol/L. The exchangeable sodium is around 2800 mmol.

8.3.4. Absorption and excretion

Sodium is rapidly absorbed from the gastrointestinal tract and the usual routes of excretion are urine, faeces and skin. Normally, balance is maintained on intakes of little more than minimal intake of 69 mg of Na in infants – 3 mmol (8.3.1).

8.3.5. Sodium output

Sodium output through urine and faeces amount to less than 1mol/day. Healthy human kidney has the capacity to excrete urine that is virtually sodium-free. Sodium losses through the skin are approximately 2-4 mmol/day under moderate climatic conditions but may increase to 350 mmol/day under conditions of hard physical labour at high temperatures (8.3.4).

Thapar *et al* (8.3.5) studied sodium excretion in normal volunteers during exercise and observed that sodium losses were around 66 mg/dl of sweat during exercise. Individuals accustomed to heat usually conserve sodium by secreting more dilute sweat 30 mmol/L, even during periods of hard physical work. However, when the water intake is beyond 3 litres, and there is excessive sweat secretion, additional sodium may have to be provided; the amounts needed may vary from 2 g NaCl to 7 g NaCl/L for extra water losses depending upon severity of losses and acclimatization.

8.3.6. Sodium intake in hypertension

Hypertension is a worldwide epidemic and its control is expensive. Several observations point to kidney as a primary factor and sodium as the main culprit. However, sodium salt is the most controversial subject in the causation of hypertension. Variation in salt consumption is greater than with any other nutrient. The relationship between salt and hypertension is based on epidemiological, patho-physiological, experimental pharmacological and clinical observations. Literature evidences suggest that among various cultures, salt intake correlates with prevalence of hypertension (8.3.2).

Epidemiological evidence

Gleibermann (8.3.2) tried to arrive at some trends and conclusion from data obtained from several sources (24 population groups) and compiled data on blood pressure versus salt intake. The age of the populations ranged between 45-65 years of either gender. A strong correlation for blood pressure and dietary salt was observed both in males and females. In fact in the same geographical areas with similar genetic influences and similar cultural trends such as in Polynesians and Brazilian Indians, distinct differences in salt consumption were observed and blood pressure tended to be higher with higher dietary intake. Based on his observations and statistical computations, salt intakes higher than 10 g of NaCl/d can be considered to have a definite tendency to increase blood pressures.

8.3.7. Hypertension in India

In India, 10% of the attributable deaths are due to hypertension and this appears to be an escalating disorder. Chronic disease risk factors are a leading cause of death and disability in all countries and the important risk factor is raised blood pressure. Around 7.1 million deaths occur as a result of hypertension and therefore it is essential to have dietary approaches at a population level to decrease hypertension, salt being one of the important factors in the genesis of blood pressure (8.3.6). It may be pertinent to note that an epidemiological study involving nine states of India revealed a prevalence of 24-25% hypertension in rural adult population (8.3.7).

8.3.8. Human needs of sodium

a. Infants

The main determinants of sodium requirements of infants are rate of growth, composition of feaces and losses through skin. Sodium concentration of fat-free tissues after birth is 90 mmol/kg of lean body mass. Obligatory urinary losses and cutaneous and faecal losses are 2 mmol/day and added growth needs to arrive at an estimate of minimal sodium requirements of 2.5

mmol/day in infants and young children (8.3.1). Dahl (8.3.9) estimated the infant needs and replacement losses of sodium could be generously met with intakes of 4-8 mmol/day. 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. Thus breast milk is adequate for infant growth, even if dermal losses are higher. Formula-fed infants in general have a high intake (10-50 mmol) from 2 months to 10-12 months. Proprietary formulations contain 7-17 mmol/L. Cow's milk contains 21 mmol/L.

b. Sodium requirement during pregnancy

In early pregnancy, there is an increase in the glomerular filtration and filtered sodium loss. An increase in progesterone also can inhibit sodium reabsorption. However, an intact renin-angiotensin-aldosterone system, with an increase in aldosterone in pregnancy increases sodium reabsorption. Further as the extra-cellular fluid volume increases and osmotic pressure falls, additional increase in production of renin and aldosterone accelerates sodium reabsorption. The regulatory mechanisms are not exhausted until the sodium intake is severely restricted.

On the basis of average weight gain of 11 kg, of which 70% is water, sodium requirement in pregnancy is calculated to be about 750 mmol (Sodium in ECF 145 mmol/L). Therefore only 3 mmol of sodium/day above the non-pregnant requirement is needed and is easily met by usual diets of pregnant women. In Indian situation, with a gain in body weight of about 6.5 kg of which 70% is water, the sodium requirement will be much less and there is no need for extra sodium.

c. Toxicity

Apart from its relationship to hypertension, at intakes of 590-680 mmol daily, healthy individuals can develop fluid retention.

8.3.9. Recommended dietary intake of sodium

Globally, the most common cause of sodium deficit is acute diarrhoea. The other disease, where it has a vital role to play, is hypertension. In general, one can easily manage with less than 1 mmol of sodium. A safe and adequate level is 1100-3300 mg/day (8.3.6). The minimum requirement for a healthy person is 500 mg of sodium for adults and 58 mg/day for infants and children. Maximum daily intake of sodium chloride should not exceed 5 g per day. Recommended intake of sodium is given in Table 8.3.1.

From the literature review it is evident that undesirable effects are seen over 100 mmol/day. As the natural diet in India provides less than 1 g of

NaCl, cooking salt can be reduced from 10 g to 5-6 g/day to obviate long term consequences of excess sodium intake. The recent WHO Consultation on Global Strategy recommends 5 g of salt /day to prevent chronic diseases particularly, hypertension, which is a major problem in India (8.3.8).

Table 8.3.1

Recommended intakes of sodium and potassium (mg/d) and molar ratio

Group		Sodium	Potassium	Molar ratio
Adult	Men	2100	3750	1.1
	Women	1900	3225	1.1
Infants	0-6 m	410	630	1.1
Children	1 – 3 y	590	1100	1.1
	4- 6 y	1010	1550	1.1

1 g of sodium chloride contains 39% sodium

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8.4. POTASSIUM

There is every reason to believe that early in the evolution of life on earth, the relative concentration of potassium compared to sodium, was much higher (8.4.1). Potassium is therefore the most abundant intracellular cation in the body. Potassium content of the extracellular fluid is about 80 mmol, whereas in the tissue it is around 3500 mmol. Eighty percent of this amount is in the skeletal muscles. The potassium content of bones is negligible (7%). Concentration of potassium in the extracellular fluid is around 5 mmol/L while the cells have about 150 mmol/L. Potassium content decreases with age and adiposity. Total body potassium corresponds closely to lean body mass and to the body nitrogen content (8.4.2). Potassium contributes to intracellular osmolality. Enzymes involved in glycosis and oxidative phosphorylation are potassium-dependent. It is involved in the maintenance of acid-base balance. Maintenance of extracellular potassium concentration is complex and is the result of intake, excretion and distribution. Aldosterone and cortisol increase urinary loss of potassium. Renin and angiotensin have a similar effect. 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.

8.4.1. Potassium sources

The major sources of potassium are plant foods. Vegetable foods contain more potassium than sodium. In the diet, good sources are cereals, pulses, fruits and vegetables. Nuts and oilseeds can also contribute significant amounts. Potassium intake in our diet depends on the consumption of cereals, pulses and fruits. Diet can provide around 4-7.5 g (50-100 mmol/day) of potassium chloride. Processed foods in general have their potassium washed out during processing and is replaced by sodium. There is only one form of potassium and there is no evidence that potassium is better utilized from some foods than from others.

8.4.2. Absorption and excretion

Natural diets usually provide around 50-150 mmol of potassium per day (5-10 g of KCl). Potassium is absorbed by diffusion and is readily distributed in aqueous media throughout the body. Potassium deficiency/excess rarely arises primarily as a result of dietary deficiency/excess. Urinary excretion of potassium is around 40-90 mmol/day and depends on dietary intake. Potassium excretion in urine increases whenever the tissues break down. The ratio of potassium to nitrogen losses during wasting is around 2.7 mmol

potassium per gram of nitrogen. Potassium is also lost through faeces and sweat; loss of 5-10 mmol in faeces and 3-5 mmol in sweat are reported. Under normal circumstances, only minimal amounts are excreted through the intestine. Sweating at normal rates produces sweat containing 9.5-60 mmol/L. Potassium is lost in excess in patients treated with diuretics and can lead to depletion if not properly supplemented. Converse is true in cases of renal failure where potassium accumulates. Diarrhoea can result in excess loss of potassium in the stools. In severe diarrhoeas, 90 mmol/24 hr (< 4 g of potassium) can be lost in stools and in infants suffering from protein energy malnutrition, potassium deficiency occurs when 10-30% of body potassium is lost. Such a situation can also occur in adults with very acute diarrhoea. The main effects of potassium deficiency are muscular weakness and mental confusion. Potassium depletion in cardiac muscle can be associated with changes in ECG and sudden deaths can also occur. Paralytic ileus can be precipitated particularly in children. Potassium deficiency due to drugs and diarrhoeas usually requires supplements rather than dietary treatment.

8.4.3. Infant needs of potassium

Newborn infants contain 50 mmol of potassium per kg fat-free body weight. Obligatory urinary and cutaneous losses are the major determinants of potassium needs. Need for growth has led to a minimal potassium requirement of 2.3 mEq/day (90 mg) for infants and children. Potassium content of human milk is greater in early milk (19 mmol/L) than mature milk (15 mmol/L). The ratio of sodium to potassium is 1:2 in mature milk. Cow's milk contains 38 mmol/L and commercial formulae have around 18 mmol/L.

8.4.4. Potassium needs during pregnancy

Supply of potassium is abundant for needs of all and is available in plenty in balanced plant-based diets. Potassium requirement in pregnancy has been assessed to be around 12 g. This amount is likely to be supplied by an increase in cereals and pulses intake.

8.4.5. Potassium and blood pressure

Potassium is vaso-active, increases blood flow and sustains metabolic needs of the tissue. Potassium is released by endothelial cells. Potassium supplements lower blood pressure, although the response is slow. A number of observations in humans suggest a primary role for dietary potassium in the genesis of arterial hypertension. Much of the information regarding potassium and blood pressure is always in relation to dietary sodium. High dietary sodium, low dietary potassium have been implicated in the etiology of hypertension as evidenced by epidemiological clinical and animal studies. Dustan reviewed the historical perspectives in primitive people (8.4.3). People

consuming primarily plant foods with large intakes of potassium and low intakes of sodium, rarely exhibit hypertension. Blacks and other hypertensives residing in the United States in general have lower excretion of potassium and higher incidence of hypertension. Low intakes of potassium were implicated in it. Even in Japan, low blood pressure was identified with high potassium intake (8.4.4).

8.4.6. Clinical studies on potassium and hypertension

In young borderline hypertensives, increase in blood pressure induced by sodium was prevented by potassium administration. In normotensives lowering the sodium intake and increasing potassium intake decreased blood pressure. Thus potassium seems to have a protective effect on hypertension. It is often not recognized that decline in blood pressure observed with high fruit / rice diet could probably be due to high potassium intake. Salt sensitive hypertension responds particularly well to potassium intake.

8.4.7. Toxicity of potassium

It is possible to induce toxicity by giving a potassium salt (10-18 g of KCl). Hyperkalaemia can be fatal and result in cardiac arrest. Individuals with sub-clinical/clinical renal failure are at risk of hyperkalaemia. The upper limit of salt intake is 140 mmol of potassium/day (10 g of KCl).

8.4.8. Recommended dietary allowances of potassium

Enough potassium should be present in the diet to balance sodium intake. As losses of potassium are around 30 mmol/day, an intake of 50 mmol is suggested at the lower end. Dietary intake of 140 mmol/day at the upper end is considered to be safe. The ideal desirable sodium : potassium ratio in the diet is 1:1 (in mmol). The recommended intake viewed as safe and adequate is 1875-5625 mg daily (10-28 g of KCl) while in infants of 0-0.5 years it is 350-925 mg (1.8g of KCl) and 0.5-1 year, 475-1275 (2.5 g of KCl). In children of 1-3 years, it is 550-1650 mg (3.2g of KCl) and of 4-6 years it is 775-2325 mg (4.5 g of KCl) (8.4.5).

For recommended intake of potassium for different age groups refer to Table 8.3.1.

8.4.9. Safe sodium and potassium intake in the tropics

The safe sodium and potassium intakes in a tropical country like India have to be determined in terms of a higher loss of sodium in sweat and lower content of Na and higher content of K in normal diets based predominantly on plant foods. Since not much of processed food containing added salt is

consumed in India, sodium content of normal diet predominantly based on plant foods is low. The added sodium chloride (NaCl) intake must be higher in India than in Western countries.

There is a need for research on turnover of sodium and potassium employing stable isotopes in sedentary and moderately active workers and dietary intake and its relation to hypertension in rural and urban areas. Data from such studies may help to arrive at more precise values of sodium and potassium intake.

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9. 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. Recent surveys conducted by the NNMB and NFHS-3 show high prevalence of anemia (24.3 %) even in men aged more than 20 years (9.1; 9.2; Table 9.1). Despite supplementation programs being in operation for decades, there has been no perceptible decrease in the prevalence of anemia (Table 9.2). 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.

Preponderant evidence indicates iron deficiency as the main cause of anemia in all age groups (9.3-9.7). Even higher income groups are victims of widespread anemia. Iron deficiency is 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 B_{12} deficiency (9.3). In pregnant women, studies with serum transferrin receptor or TfR which is used as a marker of tissue iron deficiency (9.8-9.10) suggested low iron stores and widespread iron deficiency. A recent study carried out by NIN in semi-urban school children showed prevalence of multiple sub-clinical micronutrient deficiencies (9.11).

Table 9.1: Prevalence (%) of anemia among Indians

Category/ anemia prevalence		Mild	Moderate	Severe	Any anemia
Children	1-< 5y	23.7	41.1	2.1	66.9
Men					
	20-49y	44.7	6.6	0.3	51.6
	≥50 y	56.2	17.0	1.2	74.4
Girls	12-14y				
	15-17y	46.8	20.8	1.1	68.7
Women (NPNL)	20-49y	47.0	20.9	1.8	69.7
	≥50y	41.9	30.3	2.9	75.1
Lactating	<6m	52.6	26.5	1.1	80.2
Pregnant	≥6m	43.0	31.9	3.2	78.1
		24.4	45.9	4.3	74.6

References 9.1 and 9.2

9.2 Iron requirements and basis for deriving requirements

Iron requirements for different groups were derived using factorial approach by the previous Committee (9.12). Basal iron loss formed the fundamental requirement unit for all. Additional requirements for menstrual loss in women, foetal growth and blood volume expansion for pregnant women, loss through milk for lactating women and for skeletal growth and expansion of stores in children, were accounted for, in this approach.

Table 9.2

Prevalence (%) of anemia among children (6m-5 y), adolescents (15-19y), pregnant and lactating women

Category/ anemia prevalence	NFHS 3				NFHS 2			
	Mild	Moderate	Severe	Any anemia	Mild	Moderate	Severe	Any anemia
Pregnant women	25.8	30.6	2.2	58.7	21.8	25.4	2.5	49.7
Lactating women	44.8	16.6	1.7	63.2	38.9	15.8	1.6	56.4
Adolescent boys (15-19y)	16.7	12.1	1.4	30.2	-	-	-	-
Adolescent girls (15-19y)	39.1	14.9	1.7	55.8	36.2	17.9	1.9	56.0
Children								
6-11m	27.6	51.1	2	80.7	27.0	41.5	3.2	71.7
12-23m	23.9	54.7	4.5	83.0	22.0	49.5	6.3	77.7
24-35m	26.6	44.1	3.9	5	21.9	44.5	5.6	72.0
36-47m	27.3	33.1	2.7	74.6	-	-	-	-
48-59m	26.9	24.9	1.2	63.0	-	-	-	-
				53.0				

-Values not reported
References 9.2 and 9.3

Basis for deriving iron requirements

Basal loss: The iron requirement for adults is equivalent to bioavailable iron sufficient to replace daily endogenous losses referred to as obligatory or basal loss. Body iron loss and excretion in man have been determined by turnover studies (9.13). The basal loss is proportional to the body size or surface area and thus in a 60 kg man it accounts for about 0.86 mg/day (Table 9.3).

Table 9.3

Obligatory loss/basal loss of iron in a 60 kg man

Basal loss (60 kg man)	Amount mg/day
Sweat/exfoliated skin	0.17
Desquamated gastrointestinal cells	0.43
Bile	0.17
Urine	0.09
Total loss	0.86

From the data furnished above, the basal loss of iron estimated is 14 µg/kg/day (rounded off) and is used to compute the basal iron requirement of Indians belonging to different age groups, employing actual body weights. The optimum body weight suggested by the present Committee is given in Table 9.4.

Table 9.4
Desirable weights of Indians of different physiological groups

Age group		Body Wt kg	Body Wt gain kg/y
Men		60	---
Women (NPNL)		55	---
Infants	0-6 m	5.4*	0.73/ m
	6-12 m	8.4*	0.28/m
Children	1-3 y	12.9*	2.3
	4-6 y	18.0	2.0
	7-9 y	25.1	2.7
10-12 y	Boys	34.3	3.9
	Girls	35.0	3.6
13-15 y	Boys	47.6	3.0
	Girls	46.6	4.1
16-17 y	Boys	55.4	2.2
	Girls	52.1	1.5

*Values are from WHO (MGRS) 2006
Source: Chapter 3, Tables 3.1, 3.2

Requirement for an adult man (60 kg):

The basal loss of iron in an adult man is around 14 µg/kg body weight/ day and therefore, an adult male weighing 60 kg requires 0.84 mg of iron/ day.

Optimum haemoglobin: For calculating requirement of iron needed for blood volume expansion during growth phase and menstrual loss, normal haemoglobin concentration of the particular group is used. Table 9.5 provides the normal haemoglobin value used for the calculation.

Table 9.5
Optimum haemoglobin concentration for different age and gender groups

Group	Haemoglobin g/L
Children 0.5-5 y	>110
Children aged 5-11 y	>115
Children aged 12-13 y	>120
Men	>130
Non-pregnant women	>120
Pregnant women	>110

Reference 9.14

Requirement for an adult woman (55 kg)

In pre-menopausal women, besides basal loss of 14 µg/kg body weight, iron is required to replace blood lost in menstruation. Menstrual loss is computed from the reported range of blood loss (20-62 ml per cycle). A blood loss of 30 ml results in a loss of 12.5 mg iron per 28 day menstrual cycle which is equivalent to 0.45 mg iron/d. The range of iron loss is 0.3 – 0.9 mg or 8 µg/kg body weights during the adolescent period and 16 µg/kg after 18y of age (9.15, 9.16). Considering the basal loss and loss due to menstruation, the requirement for an adult woman works out to 14+16 =30 µg/kg or 1.65 mg/day.

Pregnancy

Iron requirements during pregnancy can be calculated taking into consideration the iron needs for fetal growth, expansion of maternal tissue including the red cell mass, iron in the placental tissue and the blood loss during parturition. These additional requirements should be added to the basal requirement. However, there is a saving of menstrual loss during pregnancy. Data are not available for these additional requirements in well-to-do Indian women.

Based on the available data (9.17-9.19) for Indian and Western women, an additional 760 mg of iron is required during the entire pregnancy period (includes requirement for fetus + expansion of maternal red cell mass + placenta and cord + obligatory loss) for Indian women having a pre-pregnancy body weight of 55 kg and considering a gestational weight gain of 10 and 12 kg (Table 9.6). A detailed derivation is given in Annexure 9.1.

Table 9.6
Iron requirement during pregnancy

Compartments	Iron requirement during entire pregnancy (mg)	
	10 kg GWG	12 kg GWG
Foetus	190	230
Expansion of maternal red cell mass	250	296
Placenta + cord	90	100
Obligatory losses[1]	230	234
Total	760	860

GWG is gestational weight gain
[1] @ 14 µg/kg
Blood lost during parturition considered to be taken care of by the contraction of maternal red cell mass and therefore not accounted for.
References 9.17 and 9.19

Value of total iron requirement (mg/day) is used for deriving iron required during the three trimesters, (Annexure 9.1). Total iron required during the three trimesters is given in Table 9.7.

Table 9.7
Iron requirement during pregnancy (trimester-wise)

Trimester	Requirement mg	
	10 kg GWG	12 kg GWG
First	130	138
Second	320	372
Third	310	351
Total	760	861

Detailed calculations are given in Annexure – 9.1.

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, the basal requirement will be the same as in the adult woman, i.e., 14 µg/ kg/d or 0.77 mg/d. According to a recent study in India (9.20, 9.21), iron content of breast milk is around 14 µmoles/L or 0.78 mg/L. Assuming that the average milk volume is around 650 ml/d, amount of iron needed works out to about 0.5 mg/d corresponding to 9 µg/kg/d. The total requirement during lactation is about 1.27 mg/d, which is lesser by about 0.4 mg/d, compared to non-pregnant, non-lactating woman. However, considering the losses during pregnancy, the requirement was fixed to that of NPNL woman i.e., 30µg/kg/d.

Iron requirement for growth

Iron requirement for growth refers to the iron needed for expansion of blood volume and the need for increase in lean body mass (Table 9.8). The factor for blood volume expansion has been calculated, based on the increase in volume of blood for a given increment in body weight (66 ml/kg for all except for boys 13-18 y where increase is assumed to be 75 ml/kg), the normal haemoglobin concentration (Table 9.5) and 3.47 mg/g towards the iron content of haemoglobin.

For example, from 10 to 12 y of age, body weight gain is about 3.5 kg. Using approximate estimates of blood volume in adolescents of 66 ml/kg body weight, the 3 y growth (10-12+) increment (3.5 kg/y x 66ml) (9.21) is associated with an increase in blood volume of 230 ml/y. Estimates of the quantity of iron needed for extra blood (assuming a blood haemoglobin concentration of 120 g/L and an iron concentration of 3.47 mg/g Hb) is ~100 mg/y or 0.27 mg/d.

Increase in lean body mass also requires iron, primarily for muscle myoglobin and also non-heme iron. Iron content of skeletal muscle is approximately 0.026 mg/g wet weight. If half of the increase in body weight is associated with greater muscle mass, the requirement would be 47 mg/y or 0.13 mg/d in this age group.

Thus, the additional requirement during adolescence for the growth spurt (expansion of blood volume, increase in haemoglobin concentration and increase in muscle mass) would be 12 µg/kgfor boys and 8 µg/kg for girls (based on average of all groups). Girls would require an additional 8 µg/kg to compensate for menstrual blood loss.

Body iron stores

During growth spurt, it is necessary to consider allowances for iron store. In males, during adolescence, about 6 mg/kg of iron is stored, which increases to around 12-15 mg/kg subsequently. A 60 kg man would have about 810 mg of iron as storage iron; therefore, an additional requirement of 0.4 mg of iron/d (810 mg spread over a period of 5 y) is required during 13-18 years of age to build up this store.

In women, the stores are very low and about 1/8th of that found in males. Considering that there is a constant drain of the nutrient, an allowance of 5 mg/kg had been added for maintenance of stores throughout adolescence in girls. However, no separate allowance for building up of stores is being considered while deriving the RDA in adults due to reclaiming of RBC iron and its re-utilization. The iron requirement during adolescence is given in Table 9.8.

Table 9.8

Iron requirement during adolescence

Age group (y)	Body Wt (kg)	Gain in body weight (kg/y)	Basal loss (mg/d)	Blood volume (mg/d)	Muscle mass (mg/d)	Store (mg/d)	Blood loss (mg/d)	Total Requirement (mg/d)
10-12								
Boys	34.3	3.5	0.49	0.27	0.13	0.16	------	1.05
Girls	35.0	3.7					0.28	1.33
13-15								
Boys	47.6	4.2	0.66	0.39	0.15	0.40	----	1.60
Girls	46.6	1.7	0.65	0.13	0.06	0.15	0.37	1.36
16-17								
Boys	55.4	1.5	0.78	0.14	0.05	0.40	-----	1.37
Girls	52.1	-	0.73	----	---	0.15	0.42	1.30

Requirement during infancy and childhood

There is not enough evidence available to establish RDA for iron for infants from birth through 6 months of age. Recommended iron intake for this age group is based on the average iron intake of healthy infants fed breast milk (Table 9.9). Iron in human breast milk is well absorbed by infants. It is therefore recommended that infants be exclusively breast fed for the first six months of life. It is estimated that infants can use greater than 50% of the iron in breast milk (9.23) and healthy full-term infants are born with a supply of iron that lasts for 4 to 6 months. Considering the breast milk intake to be around 600 ml during this period with iron content of 0.78 mg/L, the iron intake per day is about 0.47 mg/d. During this period small change occurs in storage iron and haemoglobin levels. Thus, a full term infant of 3.2 kg body weight needs only 0.23 mg/d to maintain his/her haemoglobin at the normal concentration of 110 g/L and to replace excretory losses. Iron requirements increase markedly, especially in relation to body size and energy intake during later 6 months of life; therefore, iron-enriched solid foods should complement breast milk from 7 to 12 months of age.

Table 9.9
Iron requirement during infancy

Age of infant	Wt (kg)	Basal loss (mg/d)	Blood volume expansion (mg/d)	Skeletal mass	Total (mg/d)	Require-ment (µg/kg/d)
0-6 m	5.4	Equal to milk iron			0.23	46
6-12m	8.4	0.12	0.4	0.21	0.73	87

During pre-school years (1-3 y), when growth rate slows down and the body mass increases by 1.9 kg/y, there is virtually no reserve store of iron between the ages 6 months and 2 years. Added to basal losses, the total requirement during this period is about 0.434 mg/d. (Table 9.10).

Table 9.10
Iron requirement for pre-school children (1-3 y)

Body Wt	12.9 kg
Basal loss	0.181 mg/d
For growth	0.270 mg/d
Total	0.451 mg/d

Iron requirement during childhood (4-9 y)

In childhood, the mean increase in body weight is 2.8 kg/y, which necessitates an iron requirement of 0.7 mg/d (Table 9.6). The average iron requirement for growth would be about 17 µg/kg/d. During childhood (4-9+y) body store of iron builds up to 5 mg/kg, which is maintained in girls until menarche (Table 9.11).

Table 9.11
Iron requirement during childhood (4-9y)

Age group y	Body Wt kg	kg/y	Basal loss mg/d	Blood volume mg/d	Muscle mass mg/d	Store mg/d	Body requirement mg/d
4-6	18.0	2.8	0.253	0.2	0.1	0.08	0.633
7-9	25.1	2.8	0.353	0.2	0.1	0.12	0.773

Comparison of existing and proposed requirement of iron for all physiological groups is given in Table 9.12.

Table 9.12
Comparison of previous (1989) and present (2010) requirement of iron for all physiological groups

Group		Previous (1989)		Present (2010)	
		Body weight (kg)	Require-ment (mg/d)	Body weight (kg)	Require-ment (mg/d)
Men		60	0.84	60	0.84
Women (NPNL)		50	1.50	55	1.65
Pregnant women		50	3.0	55*	2.80
Lactating women (0-6m)		50	1.50	55	1.65
Infants 0-6m		5.4		5.4	46µg/kg/d
6-12m		8.6		8.4	87µg/kg/d
Children	1-3 y	12.2	0.354	12.9	0.45
	4-6 y	19.0	0.551	18.0	0.63
	7-9 y	26.9	0.780	25.1	0.77
Adolescents					
Boys	10-12 y	35.4	1.02	34.3	1.05
Girls	10-12 y	31.5	0.945	35.0	1.33
Boys	13-15 y	47.8	1.243	47.6	1.60
Girls	13-15 y	46.7	1.401	46.6	1.36
Boys	16-17 y	57.1	1.485	55.4	1.37
Girls	16-17 y	49.9	1.497	52.1	1.30

*Pre-pregnancy weight

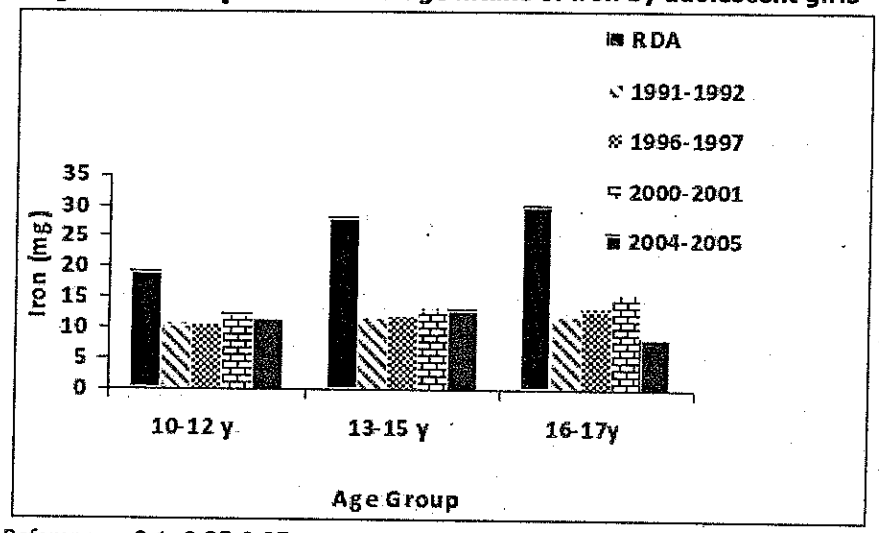
9.3 Dietary iron intake

Diet surveys carried out by NNMB (9.1) are the major source of information to calculate the intake of iron. There is a good agreement in the magnitude of differences in iron content of diets when it is chemically analyzed or computed. This implies that estimates from diet surveys reflect actual intakes (9.24). Periodic diet surveys have shown that there is an upward trend in the iron density of Indian diets but the intake of energy has got reduced over the years, implying that increased cereal intake does not contribute to increased iron density.

The NNMB survey revealed that intake of dietary iron is grossly inadequate in most of the states, meeting less than 50% of RDA set by the previous Committee. This deficit (in women) is highest in AP (72%) and lowest (23%) in Gujarat. These differences in iron intakes are attributable to regional differences in the consumption of staple foods, especially rice and millets. The extent of anemia prevalence is not correlated with the current

intake of iron, with Gujarat showing 55% anemia prevalence upon 23 mg/d iron intake and Kerala showing only 33% anemia prevalence upon 11 mg/d iron intake. A similar scenario of correlation emerges with lack of iron density.

Figure 9.1: Time trend in average intake of iron by adolescent girls



References: 9.1, 9.25-9.27

9.4 Iron absorption

It is important to have an accurate measure of iron content as well as its bioavailability from the Indian diets, to suggest RDA. The present Committee reviewed critically all the data available from India on iron absorption and its utilization from different dietary sources. Broadly, two types of methods - chemical balance and isotopic method, were used in determining the absorption and retention of iron (9.28 - 9.30).

Based on the recommendations in 1989 (9.10), iron density of daily diet was set at about 13 mg /1,000 kcal for all socioeconomic and age/gender categories and very few Indians would meet their iron RDAs with the current patterns of food consumption (working out to just half of the recommended value in each group). Besides this, it is pertinent to note that the figures of RDA did not smoothly slide into different age and gender groups, particularly those for girls of adolescent age groups (26, 19 and 28 mg for transition from under 10-12, 13-15 and 16-17 age groups) seem to be out of place and edgy.

9.5 Re-examination of iron absorption data

In the very first chemical balance studies carried out on Indians, absorption of iron from various Indian diets was found to vary from 7-20% (median-10%) (9.31). Using chemical balance method, Apte and Iyengar (9.18) demonstrated that in pregnancy, iron absorption increased from a mean of 7 % to 30 % and to 33 % at gestational weeks 8 - 16, 27 - 32 and 36 - 39 respectively. Absorption of iron was better among those with low per cent transferrin saturation than in women with high per cent transferrin saturation. As much as 58% of 30 mg of dietary iron ingested per day could be absorbed (17.5 mg) by an iron deficient full-term pregnant woman. However, the magnitude of difference in iron absorption between non-pregnant and pregnant Indian women is striking even when the same balance method is used. This fact was not considered by the earlier Committee for recommending RDA for iron during pregnancy. The balance methods were reported to yield, on an average, 7.4% more iron absorption than the extrinsic tag methods.

In 1983, detailed iron absorption studies with habitual Indian diets involving a single staple (wheat, rice, ragi or sorghum) were performed using extrinsic tag technique on adult men (9.30). Mean iron absorption from single meal ranged from 0.8 to 4.5 % depending on the type of staple used. The extent of absorption was the lowest (0.8 - 0.9 %) with millet-based diets, highest (4 - 5%) with rice-based diets and intermediate (1.7 - 1.9 %) with wheat-based diets. Based on the studies done in adult men, a uniform 3% absorption value of iron from a mixed cereal-pulse vegetarian diet was considered for deriving the RDA for iron, whereas it was 5% for women.

Recommendations based on the above absorption data now appear to be unrealistic for the following reasons:

i) The fact that the diets should provide an average of 14.2 mg of iron/1,000 kcal (range 8.8-21mg) with lowest iron density recommended for children (1-6 y) and adult males and the highest for boys aged 7-18 y.

ii) Even if an iron density of 10.8 mg/1,000 kcal is assumed for all socio-economic groups and age/gender categories, very few Indians would satisfy RDAs for iron and energy with the present patterns of food consumption.

These current considerations make it impossible for the Indian population to meet iron requirements through normal diet alone.

iii) Further review of literature on iron absorption in the Indian context reveals that iron absorption is not that poor as was reported in the very early studies. Absorption studies with wheat based meal among Indian

housewives (9.32) showed a non-heme iron absorption of 5.8%. In another study, iron absorption values were variable (4-12%) depending on the content of carotene or vitamin A added to the meal (9.33). Absorption studies among 7 human ileostomy volunteers on different cereal-pulse-vegetable based diet were reported to be 9.8%. (9.34)

iv) A more recent iron absorption study was carried out using state-of-the-art stable isotopes in normal and iron deficient women consuming single rice-based meal containing a total of 4.3 mg iron (9.35). The mean fractional absorption in iron-deficient subjects was 17.5% and it was 7.3% in normal women, which is greater than absorption values (5%) used earlier for calculating iron RDA for adult women.

v) Also, the recommendations for dietary iron for Indians (30 mg) are the highest in the world. These are at least two-to-three folds higher than those suggested for advanced countries like US and Canada and much higher than those suggested for other Asian countries, perhaps, due to poor bioavailability. This implies that measures should be taken to enhance the bioavailability of iron and not lay stress merely on the aspects related to density or content.

The present Committee accepts the basis and principle of obtaining the factorial requirement of iron in different age and physiological groups as reported by the earlier Committee. However, it differs on the issue of applying the factors of bioavailability.

9.6 Estimate of Indian RDA

i) The iron density of Indian diet is around 8.5 mg/1000 kcal based on diet survey records and is around 9 mg/1000 kcal based on chemical analyses which are lower than previously estimated figures due to 30% contaminant iron (14.2 mg/1000 kcal).

ii) Considering the fact that iron absorption is inversely related to body iron stores and that Indians have reduced iron stores compared to their peers in developed world, a realistic estimate of iron absorption would be 5% for all physiological groups except in the case of adult women where it can be in the range of 8-10%. These figures are in agreement with the recommendations of WHO/FAO, which for didactic reasons, lists three bio-availability levels of 5, 10, and 15% (9.36).

Unlike the earlier Committee which used three tier absorption for adjustment of dietary iron - 3%, 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 (9.36). They recommended using more realistic figures of 5% and 10% based on bioavailability. In the

Indian context, absorption of iron from a cereal-pulse based diet in adult male is 5% and a conservative figure of 8% is considered in women who are expected to have better absorption due to deficient iron store. However in infants 6- 12 months, absorption of 15% is derived based on stable isotopic studies carried out recently (9.37).

The RDA for iron after multiplying with the bioavailability factor of 5% and 8% is given in Table 9.13.

Table 9.13
RDA of iron for various physiological groups

Category / Age		Body weight kg	Require-ment µg/kg/d	Absorption assumed %	Iron mg/d
Men		60	14	5	17
Women (NPNL)		55	30	8	21
Pregnant women		55[a]	51	8	35
Lactating women	(0-6m)	55	30	8	21
Infants	0-6m	5.4	46	---	---
	6-12m	8.4	87	15[b]	5
Children	1-3 y	12.9	35	5	9
	4-6 y	18.0	35	5	13
	7-9 y	25.1	31	5	16
Adolescents					
Boys	10-12 y	34.3	31	5	21
Girls	10-12 y	35.0	38	5	27
Boys	13-15 y	47.6	34	5	32
Girls	13-15 y	46.6	29	5	27
Boys	16-17 y	55.4	25	5	28
Girls	16-17 y	52.1	25	5	26

[a] Pre-pregnancy weight
[b] Reference 9.37
Indian diet contains ~ 7-9 mg/1000 kcal (recalculation based on revised iron values from Nutritive Value of Indian Foods). It is recommended that the density of ascorbic acid should be at least 20 mg/1000 kcal (3 times by weight to achieve 1:2 molar ratio of iron to ascorbic acid) to ensure 5% iron absorption.

One fundamental point that the Committee would consider is that the current pattern of vegetarian diets may not entirely meet the requirements of iron and it is imperative that non-milk animal foods should be consumed to obtain heme iron (Annexure IV at the end of the document). If the conditions are not conducive to implement these changes, then, stress should be placed

on including adequate amounts of vitamin C in the diets for enhancing iron absorption (These aspects have been discussed in the Chapter on Ascorbic acid and its requirement and RDA).

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Iron requirement during pregnancy and for trimesters: basis and detailed calculation

Trimester	Body weight gain kg[a]	Body weight[b] kg	Basal loss[c]	Blood volume expan-sion[d]	Fetal growth[e]	Placenta and cord[f]	Total mg/d
First	1.3 @ 14g/d (17g/d)	56.3 (56.5)	0.79 (0.79)	0.36 (0.42)	0.27 (0.32)	-	1.42 (1.53)
Second	4.5 @ 50g/d (60g/d)	60.8 (61.9)	0.85 (0.87)	1.26 (1.51)	0.95 (1.15)	0.5 (0.6)	3.56 (4.13)
Third	4.1@ 45g/d (54g/d)	65 (67)	0.91 (0.94)	1.15 (1.36)	0.87 (1.04)	0.5 (0.6)	3.43 (3.94)

[a] Table 4.1
[b] Pre-pregnancy weight 55 kg
[c] Basal loss@14µg/ kg
[d] Blood volume expansion: at the rate of 66ml / kg weight gain, Hb concentration 110 g/L and iron content of 3.47mg/ g of Hb
[e] Table 9.6
[f] Placenta and cord: 90 mg (Table 9.6) spread over a period of 180 days considering negligible amount in the first trimester.

10. ZINC

10.1 Introduction

Zinc is an essential metal element for animal and human health. Adequate intake of Zn has been found necessary to reduce childhood illness, enhance physical growth and decrease morbidity and mortality in poor children (10.1). In developing countries, supplementation with Zn was found to lower frequency and severity of infections like diarrhea and pneumonia and reduce mortality. It is estimated that globally 2 billion people are at risk of zinc deficiency (10.2). Adequate zinc was shown to increase linear growth and weight gain in stunted and underweight young age Indian children (10.3). In fact, WHO recommends zinc supplementation during diarrhoeal infection and for treatment of severe malnutrition (10.4).

Zinc is an integral component of many enzymes and is widely distributed in the body; skeletal tissue, and muscle and soft tissues are rich sources. Zinc has a role in stabilizing macromolecular structure and synthesis. The role of the metal ion in the DNA and RNA synthesis is well documented and both DNA and RNA polymerases are zinc-dependent enzymes. Zinc was shown to suppress free radical formation and regulate cellular signaling.

10.2 Deficiency

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 non-specific clinical features. Association of low levels of circulating zinc may confirm the deficiency as an indicator. Disappearance of the symptoms with Zn treatment and improvement in the indicator confirms deficiency.

The available data on growth failure and morbidity in children is indicative of widespread zinc deficiency. There has been no supportive evidence of lowered blood zinc levels in Indian population. Also, supplementation with zinc did not yield improvement in growth except in children exposed to recurrent diarroheal episodes (10.5). In a few studies, the plasma levels of zinc were found to be low in rural women indicating isolated instances of biochemical deficiency (10.6). There are many other studies where Zn level in blood was not found to be low. There have been no large-scale studies yet on sub-clinical zinc deficiency in India. The symptoms assumed to be due to Zn deficiency are manifested by other nutrient deficiencies which are also prevalent in Indian population; hence it is difficult to attribute them to Zn deficiency specifically.

10.3 Dietary sources and absorption

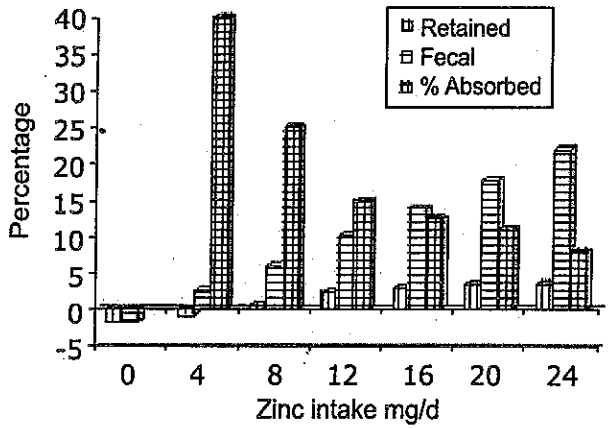
Flesh foods, liver, fish and milk are very good sources of zinc (Appendix 2). All food grains are good sources of zinc. Like iron, zinc is lost on milling and processing of the grains. Pulses and nuts are relatively rich sources of Zn.

As in the case of deficiency, only sporadic studies are reported in India on the dietary zinc content. In general, dietary intake appears to range from 7-12 mg/d (10.7), which is low as compared to the intakes reported from the Western countries. It is well known that intestinal absorption of zinc is markedly inhibited by the phytate and tannin content of diet. Habitual Indian vegetarian (mixed cereal/ pulse) diets are rich in phytate and thus the bio-availability of Zn is expected to be poor (10.8). There have been different recommendations and guidelines by the International Expert Committees like WHO, IOM, FNB and IZiNCG. All of these were reviewed by the IZiNCG (10.9). In general, the higher Zn-Phytate molar ratio of >15 occurring in Indian diets is supposed to cause low bio-availability (less than 15% absorption) as against 20-25% seen with diets low in phytate and rich in animal food. Thus, more than the total content of zinc, bio-available zinc is important to maintain adequate Zn status.

10.4 Requirements

Estimates of Zn requirements are mostly obtained from data using chemical balance methods or from the turnover studies employing radio or stable isotopes in adults. Factorial approach is used to extend the requirements to other groups. Rao and Rao worked out the first estimates of requirements of some trace elements including Zn in Indian adults (10.7 and 10.10). At least two chemical balance studies were conducted in adult males with Zn intake in the range of 7-20 mg/d. Both studies have shown equilibrium in adults with a Zn intake of around 9-11 mg. Intestinal absorption found in one of the above studies with a typical cereal and lentil-based diet is 36%, while in the second study using typical diets consumed in the four regions of the country, absorption was found to vary between 10-25%, with a mean value around 20%. This level of absorption is in tune with the data generated by FNB with unrefined cereal diet. Technical problems were encountered in the interpretation of the results of balance study due to non-linear relationships between intake and absorption of zinc and further determination of zinc requirements based on these balance studies was not attempted (10.10). However, a thorough re-evaluation was carried out later combining the data from two balance studies using natural food zinc and the findings is depicted in Fig 10.1.

Fig 10.1 Zinc Balance in Indian adults



References 10.7 & 10.10

Table 10.1

US/WHO/FAO RDA for zinc (mg/d) in different age and physiological groups (adopted values)

Group		USA*	FAO/ WHO**
Young children (1-3 y)		10	5.5 (3.3-11)
Pre-adolescents (11-14 y)	Males	15	9.3-12.1
	Females	12	8.4-10.3
Adults (25-50 y)	Males	15	9.4
	Females	12	6.5
Pregnant women			7.3-13.3
Lactating women			12.7-9.6

References * 10.18, **10.19

Thus Indian populations seem to be exposed to only a marginal risk of inadequacy of Zn at the intakes of 9-11 mg/d as referred to above. Also all the subjects studied including those showing very low absorption of 6-8% on regional diets, had positive balance at their customary intakes of Zn (as high 25mg). On the other hand all the Expert Committees assumed very low absorption with unrefined, mixed/ vegetarian diets with high phytate, based on the data generated on Western population consuming low phytate foods. Such data base used by the International Committees seems to result in higher recommended dietary intakes than those indicated from actual observations. The present Committee recommended that up to the

computation of requirements, the criteria of IZiNCG be followed and the correction for bioavailability should be based on the actual data with 20-25% absorption observed in Indian population at habitual levels of intake.

The available reports on zinc content of diet and biochemical Zn deficiency in India also do not indicate any widespread zinc inadequacy in the population (10.2) – pregnant women (10.11), school children (10.13) and adults (10.14). However, there are studies focusing Northern Region that indicate high levels of biochemical inadequacy in pregnant (Delhi slum) (10.15) and non-pregnant (rural Haryana) women (10.6). Dietary intakes of Zn were reported to be about 13 mg/d in rural Rajasthan and are adequate as per current recommendations (10.16).

Recent studies by Agte *et al* (10.17) have a novel and interesting suggestion to explain the relatively better absorption rates of Zn even at high molar ratios of dietary phytate and Zn in typical Indian foods. It was found that cooking in the Indian style results in partial hydrothermic degradation of phytates (chemically, inositol 6 phospahte or IP6) to lower phosphate derivatives like IP5, IP4 etc. In addition, the amount of IP6 was only 45% as against the much higher 48-54% in meals from other countries. Further, the ratios of small size molecules to large size molecules, viz., IP1/ IP6 and IP1+ IP2/ IP5+ IP6 for Indian meals were higher than those of meals from European and American regions of the globe.

Table 10.2

RDA of zinc for various physiological groups

Category / Age		Zinc (mg/d)
Men		12
Women		
	NPNL	10
	Pregnant	12
	Lactating 0-6 m	12
	Lactating 6-12 m	12
Children	1-3 y	5
	4-6 y	7
	7-9 y	8
Boys	10-12 y	9
Girls	10-12 y	9
Boys	13-15 y	11
Girls	13-15 y	11
Boys	16-17 y	12
Girls	16-17 y	12

After obtaining the zinc requirements and RDA based on balance and factorial data, and finding that the values closely correspond to those of WHO recommendations, the committee decided to adopt the figures recommended for different age and physiological groups on the lines of WHO Expert Committee. The RDA is given in Table 10.2.

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11. TRACE ELEMENTS

INTRODUCTION

Among the trace elements other than Fe, Zn and I, the requirements of chromium, copper, manganese, molybdenum, selenium have been studied in more detail and their recommended dietary intakes were suggested by different agencies. Still the available data were mainly experimental and based on clinical studies and thus the recommendations have been only provisional. Large scale intake data and food composition particulars are not yet available. Some information on the intakes and requirements of copper, manganese and chromium in Indian adults available to the previous Committee formed a small part of the report in their recommendations (11.1.1). However, it was decided by the present Committee that as more definitive information supported by community studies is available globally, more attention needs to be paid to these nutrients. Also, International Agencies worked out some more guidelines on the role of these nutrients and their requirements. RDA for Zn, I and Se 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, and Mn for adults are provided in this report.

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. 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 anemia (11.1.2). The RDA of Cu is about 2 mg/d (Table 11.1).

Table 11.1

Dietary intake, absorption and safe intakes of Cu, Mn and Cr for Indian adults

	IOM*	Indian adults		
Trace element	**Dietary reference intakes**	**Dietary intake range tested**	**Mean and range Absorption %**	**Acceptable intake****
Cu (mg/d)	0.9	1.6- 3.9	18 (7-37)	1.7
Mn (mg/d)	2.5	5.4-17.5	14 (2-24)	4.0
Cr (µg/d)	35	76-215	79 (63-94)	50 -

References *11.1.11 **11.1.3

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 form important tissues containing Mn. Mn is the cofactor for the enzymes SOD, arginase, and glycosyl transferase. 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 in Mn deficiency. Abnormal insulin metabolism and glucose tolerance are the important effects attributed to Mn deficiency. Isotopic turnover and chemical balance studies have revealed Mn requirement of an adult to be between 2-5 mg/d (Table 11.1).

11.1.3. Chromium (Cr)

Chromium is found to be distributed in nature in a way similar to that of Cu. 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. Estimated requirements of the adult male range between 50-200 µg Cr/d.

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* (11.1.3, 11.1.4). Subsequently, Singh *et al* (11.1.5) 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 Påthak *et al* (11.1.6) and Kapil *et al* (11.1.7) 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* (11.1.8) 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 (11.1.3). Also the apparent absorption of Cu (16.4%) and Mn (12.2%) were reported by them in ileostomized human volunteers (11.1.9). Most data on dietary content of these trace elements generated in recent times (11.1.5 – 11.1.7), though sparse, agree with the content first described for typical diets representing different regions of the country by Rao and Rao (11.1.4).

Very little information on requirement was added later. Also the earlier data pertain to the chemical balances conducted on adult male volunteers for Cu, Mn and Cr. The major national data base Nutritive value of Indian Foods is not complete and provides information on these trace elements only in major and some minor foods. In view of these limitations, the Committee decided to restrict the recommendations as 'acceptable intake' for adults and compare the values with those of WHO or other agencies (Table 11.1). The data may be extrapolated to women on body weight basis.

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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 Foltz (11.2.1) 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 foods. (11.2.2).

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 etc (11.2.3). 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% (11.2.3).

11.2.2. Functions

Glutathione peroxidase is the only selenoprotein enzyme well studied for the biological role of Se. Deiodinase isoenzymes 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 (11.2.3, 11.2.4). Inorganic selenium enters a reductive pathway to form reduced selinide through a complex formation with glutathione (selenodiglutathione (GSSeSG) and glutathione selenopersulfide (GSSeH)).Selenium disulfide may undergo some methylations in the presence of S-adenosylmethionine and get excreted in breath or in urine.

Plasma Se and 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 (11.2.4, 11.2.5). 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.

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/d (11.2.2). 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 function (11.2.4).

Selenium toxicity 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 are some of the non-specific symptoms of poisoning (11.2.4).

11.2.4. Requirements

A broad range of 9-80 µg Se requirements has been suggested based on balance studies (11.2.5). 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 (11.2.2, 11.2.3). 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 females and 36 µg/d for adult males as normal requirement (11.2.6). These values are lower than the corresponding figures of 55 and 70 µg/d suggested by US Agency (11.2.7).

11.2.5. Situation in India

There have been very few studies on Se nutrition in the Indian population. Most of the available information is from NIN. Many staple foods collected from local markets close to Hyderabad in Andhra Pradesh were analyzed for Se and the Se content ranged between 30-400 µg/g for most cereals and pulses. Average estimated dietary intake was 41-51 µg/d in different income groups (11.2.8). 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/d (11.2.9). 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 diets (11.2.10).

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 (11.2.9, 11.2.11). 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 levels (11.2.11). 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* (11.2.12) elsewhere (0.46-5.03µg 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) reported (11.2.9) is consistent with the RDA of 36 and 26 µg (for males and females, respectively) as suggested by FAO/WHO. Until further information is obtained, a level of 40 µg/d can be recommended as the acceptable intake of Se for Indians.

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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 performance (11.3.1). 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 disabilities (11.3.2.). IDD is one of the most important micronutrient deficiency disorders of public health significance in India (11.3.3, 11.3.4).

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 Programme (11.3.5). 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 Manipur (11.3.6).

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 regions (11.3.7). The prevalence of total goitre was ≥10% in half of the districts and ≥ 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 India (11.3.8-11.3.10).

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 IDD (11.3.11). 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 iodine (11.3.12). 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. Daily intake of 10 g of iodized salt having iodine at a minimum level of 15 ppm provides about 150 µg per day, in addition to iodine present in foods consumed.

Cooking losses of iodine

It is estimated that iodine in foods is lost in varying amounts during the process of cooking. The studies carried out by Dodd et al in Mumbai revealed about 40 to 70% loss of iodine in various types of preparations wherein iodized salt was used. Goindi et al found that the mean losses of iodine during different cooking procedures was 20-40% depending on the type of cooking (11.3.13; 11.3.14).

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 of iodine status of the community.

Since iodine, when ingested in large amounts, is easily excreted in the urine, iodine intake even at very high levels (milligram amounts) can be safe. It is documented scientifically that through adaptive mechanisms, normal people exposed to excess iodine remain euthyroid and free of goitre. The average daily salt intake in India is 10 g. Consumption levels are within the 5-15 g/day range for children and adults. From the average daily intake of 10 g iodine fortified salt (ie 150µg of iodine) of which about 30% is lost during cooking. The remaining 105 µg ingested with 70% absorption will work out to 70µg of iodine per day available from the diet. This quantity is broadly comparable to the daily physiological needs of the body.

Recommended dietary allowances for Iodine

Most of the countries in the World follow the WHO-UNICEF-ICCIDD recommendations (11.3.15, 11.3.16). Recently, WHO has reconsidered the recommendations for pregnant and lactating women including children below 2y old (11.3.17). The technical committee proposed to increase the current FAO/WHO recommended nutrient intake for Iodine during pregnancy from 200 to 250 µg /day. For children less than 2 years old the previously recommended Iodine intake of 90µg /day is retained. The present Committee decided to adopt for India the recommendations suggested by WHO-UNICEF-ICCIDD with the recent modifications of WHO (Table 11.3.1).

Table 11.3.1

RDA of iodine for various physiological groups

Category / Age	RDA of iodine		Upper limit (µg/kg/d)
	(µg/kg/d)	(µg/d)	
Infants (0-6m)	6-30	Breast milk	Breast milk
7-12m		90	140
Young children (1+ to 5 y)		90	50
School age children (6 to 12 y)	4	120	50
Adolescents and adults (≥ 13 y)	2	150	30
Pregnant women	4.5	250	40
Lactating women	4.5	250	40

Upper limit 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.

Status of iodine content of salt

The level of salt iodisation should provide a physiological intake of 100-150 µg/day, which would bring the median urinary iodine excretion (UIE) level within a range of 100-200 µg/L. Analysis of a total of 24,798 salt samples collected from more than 160 districts in 13 states of the country during 1994 – 2002 revealed that only 96% of the salt samples had iodine content of 15 ppm or more and only 0.73% had more than 75ppm, indicating that the iodized salt manufacturers in the country were iodizing the salt with recommended quantity. Also, the present level of salt iodization in India is within the safe limits (11.3.18).

Status of iodine intake of the population

It is recommended that the median UIE levels in a community with optimal iodine nutriture should be in the range of 100-200 µg/L. Analysis of 21,546 urine samples from 116 districts during 1994 to 2002 revealed that more than 75% of the districts had UIE level less than 200 µg/L (11.3.19).

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12. WATER SOLUBLE VITAMINS

12.1. THIAMINE

Functions

Thiamine pyrophosphate (TPP) is the coenzyme form of thiamine. In animals, TPP is involved in oxidative decarboxylation of α-ketoacids like pyruvate, á 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.

Dietary sources

Rich sources of thiamine include whole grain cereals, nuts, legumes, green leafy vegetables, organ meats, pork, liver and eggs. Since the vitamin is water-soluble and heat labile in alkaline solutions, considerable amounts are lost during cooking. On an average, about 40-50% of the vitamin present in raw foods is lost during processing and cooking as practised in Indian homes.

Thiamine status assessment

Thiamine status is generally assessed by measuring the urinary excretion of the vitamin in a 24-h collection of urine or before and after load of thiamine. Measurement of erythrocyte transketolase activity and its *in vitro* stimulation (TPP effect) is the 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. However, with the advent of HPLC which can measure lower levels, this index may become important.

Thiamine deficiency

Countrywide surveys conducted by the National Nutrition Monitoring Bureau (1999) show that though the average intake of the vitamin is close to RDA, only 40 % households meet 100 % of the RDA, and 29% consume less than 60% of the RDA.

Inadequate intake is the major cause of thiamine deficiency in developing countries. Alcoholism is also an important factor particularly in the industrialized world. Dry or wet beriberi 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. Beriberi used to be rampant in India in the early decades of this century. However, now the disease is a rarity. 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 ETK-AC test (12.1.1).

Thiamine requirement and allowance

a. Adults

Thiamine stores in the body are small and regular intake is necessary to maintain its metabolic functions. Large single doses are absorbed poorly. 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) (12.1.2). However, intakes above 0.3 mg/1000 kcal are consistent with good health. (12.1.2, 12.1.3). 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 erythrocyte transketolase activity, in systematic depletion-repletion studies done on human volunteers in India and elsewhere also indicate 0.25-0.35 mg/1000 kcal as the minimum requirement of thiamine for adults (12.1.2, 12.1.3, 12.1.4). Making allowance for cooking losses, National Research Council, USA, has suggested a dietary allowance of 0.5 mg/1000 kcal (12.1.2). In 1989, ICMR (12.1.3), had made similar recommendation and in the absence of any new evidence, this recommendation can be retained. However, minimum daily allowance of 1 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/WHO (12.1.5) and NRC (12.1.2) give a single figure for adult male and female. (Table 12.1a).

b. Pregnancy and lactation

In the absence of direct studies, the earlier ICMR Committee recommended the same ratio of 0.5 mg thiamine/1000 kcal assuming that the additional calorie allowance will take care of the extra thiamine requirement during pregnancy. Accordingly, the Committee recommended additional daily allowance of 0.2mg (300 kcal) 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. This amount would take care of maternal and foetal growth and the additional calories. Information on thiamine status of pregnant women in India is needed.

Table 12.1a

Comparison of ICMR, FAO/WHO and NRC Recommendations for Thiamine

Category / Age		ICMR 1989	FAO/ WHO 2004	NRC 1989
		mg/day		
Infants	0-6 m	0.3 (0.5 mg/1000 kcal)	0.2	0.3
	7-12 m	0.4	0.3	0.4
Children	1-3 y	0.6	0.5	0.7
	4-6 y	0.9	0.6	0.9
	7–9 y	1.0	0.9	1.0 (7-10 y)
Boys	10-12 y	1.1	1.2	1.3 (11-14 y)
	13-15 y	1.2	1.2	
	16-18 y	1.3	1.2	1.5 (15-18 y)
Girls	10-12 y	1.0	1.1	1.3 (11-14 y)
	13-15 y	1.0	1.1	
	16-18 y	1.0	1.1	1.3 (15-18 y)
Men		1.4-1.9 Sedentary Heavy	1.3	1.7 (19-50 y) 1.4 (50+ y)
Women NPNL		0.9–1.2 Sedentary Heavy	1.1	1.3 (19-50 y) 1,2 (50+ y)
Pregnant		+ 0.2	1.4	1.5
Lactating	0-6 m	+0.3	1.5 *	1.6
	7-12 m	+0.2	1.5	1.6

* Reference 12.1.5

A well-nourished Western woman secretes about 0.2mg thiamine per day in breast milk. Based on this, NRC (12.1.2) 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 (12.1.2, 12.1.6). After supplementation with thiamine the level goes up to maximum 20 µg thiamine/100 ml. With a milk output of 700 ml, the maximum secreted through milk would be 0.14 mg. This is lower than the figure of 0.2 mg reported for western, well-nourished women. The lower level of milk thiamine

even after supplementation may be because of inadequate utilization due to associated calorie and protein deficiency. In the studies quoted (12.1.6), nothing is mentioned about providing calories and proteins along with vitamin supplements. The additional allowance recommended by the ICMR (12.1.3), 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) for the sedentary and the moderate activity categories. According to the former, an additional intake of 0.4 mg thiamine during lactation would compensate for the loss through milk, and the increased energy cost of lactation. More studies are needed to determine optimum thiamine secretion in breast milk of healthy well-nourished Indian women.

Table 12.1b

RDA of Thiamine for various physiological groups

Group	Category / Age	Body weights (kg)	Thiamine (mg/d)
Men	Sedentary	60	1.2
	Moderate	60	1.4
	Heavy	60	1.7
Women	Sedentary	55	1
	Moderate	55	1.1
	Heavy	55	1.4
	Pregnant	55	+0.2
	Lactating 0-6 m 6-12m	55	+0.3 +0.2
Infants	0-6 m	5.4	0.2
	6-12 m	8.4	0.3
Children	1-3 y 4-6 y 7-9 y	12.9 18.0 25.1	0.5 0.7 0.8
Boys	10-12 y	34.3	1.1
Girls	10-12 y	35.0	1.0
Boys	13-15 y	47.6	1.4
Girls	13-15 y	46.6	1.2
Boys	16-17 y	55.4	1.5
Girls	16-17 y	52.1	1.0

c. Infants and children

The requirement of 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. Considering current recommendation of energy requirement of 92 kcal /kg and body weight of 5.4 for 0-6m, the current requirement would be 5.4X 92= 496.8 rounded off to 500 (based on actual energy content of milk, 722mL output, it is 594 kcal). With around 600 kcal, 140/600 x 1000 gives 0.233 mg of thiamine. The current recommendations of the Committee are given in Table 12.1b.

12.2. RIBOFLAVIN

Functions

Riboflavin-derived coenzymes, FMN and FAD are cofactors of numerous enzymes involved in oxidation-reduction reactions. Several of these enzymes are involved in the process of energy transduction. Riboflavin is also involved in antioxidant activity, being a cofactor for the enzymes like glutathione reductase and is required for the metabolism of other vitamins like vitamin B_6, niacin and vitamin K.

Rich dietary sources of riboflavin are flesh foods, poultry, dairy products, legumes, nuts and green leafy vegetables. About 7 per cent of flavin in flesh foods may be present as 8∝-(amino acid) FAD covalently attached to certain flavoproteins. The 8∝-(amino acid) riboflavin is absorbed but biologically inactive. Although heat stable, riboflavin is destroyed in solutions when exposed to light. Cooking losses of riboflavin in Indian preparations is about 20% (12.1.3).

Assessment of riboflavin status

In recent years, the erythrocyte glutathione reductase activation (EGR-AC) test has replaced methods based on urinary excretion and RBC levels. However, with the advent of sensitive HPLC method, measurement of blood levels has become relatively easy and precise and merits adoption.

‘boflavin Deficiency

tary deficiency of riboflavin is rampant in India. Recent NNMB surveys at the average (% RDA) intake of riboflavin is only 43% of the t (NNMB, 2006). Studies from Hyderabad show that more than

70% of women and children from low-income groups have biochemical evidence of riboflavin deficiency as judged by the EGR-AC test (12.2.1).

While dietary inadequacy of riboflavin is the major factor, studies from Hyderabad show that respiratory infections also contribute to tissue depletion (12.2.2). Respiratory infections increase riboflavin utilization leading to mobilization of this vitamin from the tissues to blood and increase urinary excretion (12.2.3). Negative nitrogen balance also results in excess urinary losses (12.2.4). Exercise leads to increased riboflavin requirement as judged by EGR-AC values (12.2.5).

The well-established clinical signs of riboflavin deficiency are muco-cutaneous lesions of the mouth- angular stomatitis, glossitis, chelosis etc. In severe deficiency other areas like scrotum are also involved. The less recognized consequences of riboflavin deficiency are: impaired psychomotor performance (12.2.6 - 12.2.9) reduced iron absorption due to lower conversion of ferric iron to ferrous iron, and risk of anemia (12.2.10), reduced skin collagen maturity (cross-linking) (12.2.11), impaired wound healing (12.2.12) (animal studies), and reduced phagocytosis (animal studies) (12.2.13). Interruption of the transfer of riboflavin from the mother to the fetus by active or passive immunization against riboflavin binding protein results in interruption of pregnancy in animals. This points out to the importance of riboflavin for successful outcome of pregnancy (12.2.14).

Riboflavin requirement and allowance

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 (12.2.2, 12.2.3). This has led to recommendation of 0.6 mg/1000 kcal (12.2.2, 12.2.3). Similar value emerged through controlled depletion-repletion study in adult Indian volunteers (12.2.15). The enzyme erythrocyte glutathione reductase showed linear increase with riboflavin intake and reached maximum levels at intakes close to 0.5 mg /1000 cal. After allowing for cooking losses, the earlier Committee recommended 0.6 mg/1000 kcal. (12.1.3). In the absence of any new evidence, this recommendation can be retained (Table 12.2a). However, a minimum intake of 1.2 mg/day may be recommended even if the calorie intake is lower than 2000 kcal.

The lower recommendation of FAO/WHO (12.1.5) (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 base their recommendations on energy intake as ICMR Committee does. Riboflavin supplementation studies conducted on rural Indian children suggest that

riboflavin requirement based on tissue saturation (RGR-AC values) may be greatly increased during respiratory infections due to excessive urinary losses (12.2.6). FAO/WHO Consultation in 1998 (12.1.5) have not recommended on energy basis. Their recommendations are given in Table 12.2a.

Table 12.2a
Comparison of ICMR, FAO/WHO and NRC Recommendations for Riboflavin

Category / Age		ICMR 1989	FAO/WHO 2004	NRC 1989
		mg/d		
Infants	0-6 m	0.35 (0.6 mg/1000 kcal)	0.3	0.4
	7-12 m	0.52	0.4	0.5
Children	1-3 y	0.7	0.5	0.8
	4-6 y	1.0	0.6	1.1
	7-9 y	1.2	0.9	1.2 (7-10 y)
Boys	10-12 y	1.3	1.3	1.5 (11-14 y)
	13-15 y	1.5	1.3	
	16-18 y	1.6	1.3	1.8 (15-18 y)
Girls	10-12 y	1.2	1.0	1.3 (11-14 y)
	13-15 y	1.2	1.0	
	16-18 y	1.7	1.0	1.3 (15-18 y)
Men		1.4-1.9 Sedentary-Heavy*	1.3	1.7 (19-50 y) 1.4 (50+ y)
Women		1.1-1.5 Sedentary-Heavy*	1.1	1.3
Pregnant		+ 0.2	1.4	1.6
Lactating	0-6 m	+0.3	1.6	1.8
	7-12 m	+0.2	1.5	1.8

*Minimum intake of 1.2 mg/1000 kcal is recommended.

a. Riboflavin requirement in pregnancy and lactation

During pregnancy, there is an increase in the EGR-AC (12.2.16, 12.2.17). In a depletion-repletion study, Kuizon *et al* (12.2.18) 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 USA (12.2.19), 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 tissues

(12.1.2). ICMR has however retained the riboflavin/calorie ratio at 0.6, during pregnancy and recommended additional intake of 0.2 mg during pregnancy (12.1.3). More information is required about riboflavin requirement of Indian women during pregnancy.

The mean riboflavin content of milk of low-income Indian women is less than 30 µg/100ml (12.1.5, 12.2.20). With supplementation, it can be raised to a maximum of 30 µg /100 ml. (12.1.5). 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 can be retained In the absence of reliable new information on utilization efficiency of riboflavin for milk production. Recommendations of FAO/WHO (2004) are higher on daily intake basis during pregnancy and lactation.

b. Riboflavin requirement of infants and children

Riboflavin status of infants of low-income group mothers has been observed to be better than that of their mothers, suggesting that infants are protected against riboflavin deficiency. Nevertheless, 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 ml (12.2.20). 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. 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 Guatemala (12.2.21) 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.

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. RDA for riboflavin for Indians is given in Table 12.2b.

Table 12.2b

RDA of riboflavin for various physiological groups

Group	Category / Age	Body weights (kg)	Riboflavin (mg/d)
Men	Sedentary	60	1.4
	Moderate	60	1.6
	Heavy	60	2.1
Women	Sedentary	55	1.1
	Moderate	55	1.3
	Heavy	55	1.7
	Pregnant	55	+0.3
	Lactating 0-6m 6-12m	55	+0.4 +0.3
Infants	0-6 m	5.4	0.3
	6-12m	8.4	0.4
Children	1-3y 4-6y 7-9 y	12.9 18.0 25.1	0.6 0.8 1.0
Boys	10-12y	34.3	1.3
Girls	10-12y	35.0	1.2
Boys	13-15y	47.6	1.6
Girls	13-15y	46.6	1.4
Boys	16-17y	55.4	1.8
Girls	16-17y	52.1	1.2

12.3. NIACIN

Nicotinic acid (niacin) and nicotinamide (niacinamide) are generally termed as niacin. Niacinamide is a part of the coenzymes connected with glycolysis, tissue respiration and synthesis of macromolecules.

Niacin is also derived 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 (12.3.1, 12.3.2). In well nourished individuals, 60 mg tryptophan is taken as equivalent to 1 mg niacin (12.3.3). While computing dietary intake of niacin,

tryptophan contribution as niacin equivalents is added to that of preformed niacin/ nicotinic acid as follows:

$$\text{Niacin equivalent in mg} = \text{Niacin mg} + \frac{\text{Tryptophan (mg)}}{60}$$

Foods of animal origin are rich sources of both tryptophan and niacin. Cereals are satisfactory sources of niacin in Indian diets; although in some foods like maize, the vitamin is present in "bound" or unavailable form. Niacin is more stable than other B-complex vitamins, in spite of some inevitable losses in cooking (12.3.4, 12.3.5). 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 which was seen in endemic form in some parts of India where jowar (sorghum) was used as staple.

Requirements of adults

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 mg and 11 mg per day; together with tryptophan, such diets provide about 6 mg niacin equivalents per 1000 kcal (12.3.6). Pellagra is rarely seen among this population. Load tests using nicotinic acid have shown that subjects consuming 6.5 to 7.2 mg of niacin equivalents per 1000 kcal have satisfactory niacin status (12.3.7). Niacin intakes of subjects suffering from pellagra have, however, been found to be around 3.6 mg/1000 kcal - an apparently inadequate intake.

Recent FAO/WHO Expert Group (12.1.5) recommended an adult allowance of niacin and the above Indian data are in consonance with this. The earlier ICMR Committee recommended 6.6 mg/1000 kcal as adult RDA and the present Committee accepts the same figure of 16 mg and 14 mg niacin equivalent per day for males and females, respectively.

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 normals 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.

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 mother (12.3.8). 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 also and the present Committee does not see any need to change that.

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 too retains the same figures as in the earlier report (Table 12.3).

Table 12.3

RDA of niacin for various physiological groups

Group	Category / Age	Body weights (kg)	Niacin equivalents (mg/d)
Men	Sedentary	60	16
	Moderate	60	18
	Heavy	60	21
Women	Sedentary	55	12
	Moderate	55	14
	Heavy	55	16
	Pregnant	55	+2
	Lactating 0-6m 6-12m	55	+4 +3
Infants	0-6 m	5.4	710µg/kg
	6-12m	8.4	650µg/kg
Children	1-3 y 4-6 y 7-9 y	12.9 18.0 25.1	8 11 13
Boys	10-12 y	34.3	15
Girls	10-12 y	35.0	13
Boys	13-15 y	47.6	16
Girls	13-15 y	46.6	14
Boys	16-17 y	55.4	17
Girls	16-17 y	52.1	14

12.4. VITAMIN B_6

The term vitamin B_6 includes three vitamins, pyridoxine, pyridoxal, pyridoxamine and their phosphorylated derivatives. Pyridoxal phosphate (PLP) is the coenzyme for a variety of enzymes like aminotransferases, decarboxylases, and side chain cleaving enzymes. Thus vitamin B_6 is needed for important pathways like gluconeogenesis, synthesis of neurotransmitters like serotonin, dopamine, taurine, γ-aminobutyric acid, norepinephrine and histamine. Along with folic acid, vitamin B_{12} and riboflavin, vitamin B_6 is needed for the metabolism of homocysteine. It is also involved in immune system and nucleic acid metabolism.

Assessment of vitamin B_6 status

Methods for assessment of vitamin B_6 status include measurement of urinary pyridoxic acid (biologically inactive product of pyridoxine metabolism), blood pyridoxal phosphate levels, *in vitro* activation of erythrocyte aspartate amino transferase or alanine amino transferase with pyridoxal phosphate, and urinary excretion of xanthurenic acid after oral load of tryptophan. Plasma pyridoxal phosphate is considered to be the best single indicator, because it is believed to reflect tissue stores.

Pyridoxine deficiency

The clinical signs and symptoms of pyridoxine deficiency include peripheral neuritis, epileptiform convulsions, anemia, glossitis, and seborrheic dermatitis. Since these signs and symptoms are seen in other B- vitamin deficiencies as well, it is difficult to assess the magnitude of clinical pyridoxine deficiency in Indians. Deficiency in infants is associated with neurological symptoms and abdominal distress. 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 contraceptives (12.1.1, 12.3.1, 12.4.1). Pregnant women and women using oral contraceptives show increased urinary excretion of xanthurenic acid after tryptophan load. 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. Health implications of this metabolic abnormality and the wisdom of administering high doses of pyridoxine to correct it are not clear. Pregnant women also show lower levels of PLP in plasma and enzymatic evidence of pyridoxine deficiency. There is some evidence of vitamin B_6 deficiency in the elderly.

Dietary sources

Information on the vitamin B_6 content of foods is not as complete as for many other B-vitamins. Meat, fish, poultry, pulses, nuts and wheat are known to be rich sources of the vitamin, while other cereals, potato and banana are moderate sources. The extent to which processing of foods and cooking practices destroy the vitamin depends on the form in which vitamin B_6 is present and the method of processing.

Pyridoxine is the predominant form of the vitamin present in the plant foods, whereas in the animal foods the major form is pyridoxal and pyridoxal phosphate. Considerable amounts of pyridoxal and pyridoxal phosphate are lost during cooking, whereas pyridoxine content of food is not affected.

About 5-80% of the naturally occurring vitamin B_6 in cereals, legumes, vegetables and fruits is present in glycosylated form, predominantly as pyridoxine-5'-α-D-glucoside. There appears to be an inverse relationship between pyridoxine glycoside content of the diet and the bioavailability of vitamin B_6. About 15% of the total vitamin B_6 content as glycosylated pyridoxine had no influence on vitamin B_6 status of lactating women (12.4.2). 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 B_6. This aspect needs further studies. The glycosidal form in which pyridoxine is present in normal foods needs to be studied.

Vitamin B_6 requirement and allowance

a. Adults

Pyridoxine requirement is linked to protein content of the diet. In the absence of information on the pyridoxine content of all the Indian foods, it is difficult to assess its dietary intake by Indians. Limited information available from Hyderabad suggests that pyridoxine intake may vary from 1.2-3.3 mg per day (12.1.3). 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 g per day (12.1.3). 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 B_6 ratio of 0.02-mg/g protein has been reported to ensure normal biochemical status with regard to most parameters (12.4.3). 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 glycoside (12.4.4).Until more definite information is available; the present recommendation of 2.0 mg/day for adults can be retained. The FAO/WHO (12.1.5) in 2004 has recommended lower levels of B_6 intake for adult male and female and other groups (Table 12.4 a).

b. Pregnancy and lactation

As mentioned earlier, pregnant women show biochemical evidence of pyridoxine deficiency, which gets corrected, only with high doses of pyridoxine. In the absence of more information, the earlier Committee suggested using the recommendation made by the NRC, namely on additional 0.6 mg/day. This recommendation can be retained. FAO/WHO also recommend additional 0.6 mg during pregnancy (Table 12.4a).

Table 12.4a

Comparison of ICMR, FAO/WHO and NRC recommendations for vitamin B_6

Category / Age		ICMR 1989	FAO/WHO 2004	NRC 1989
		mg/d		
Infants	0-6 m	0.1	0.1	0.3
	6-12 m	0.4	0.3	0.6
Children	1-3 y	0.9	0.5	1.0
	4-6 y	0.9	0.6	1.1
	7–9 y	1.6	1.0	1.2
Boys	10-12 y	1.6	1.3	1.7 (11-14 y)
	13-15 y	2.0	1.3	2.0 (15-18 y)
	16-18 y	2.0	1.3	
Girls	10-12 y	1.6	1.2	1.4 (11-14 y)
	13-15 y	2.0	1.2	1.5 (15-18 y)
	16-18 y	2.0	1.2	
Men		2.0	1.3 (19-50 y)	2.0 (19-50 y)
			1.7 (>50 y)	(> 50 y)
Women		2.0	1.3 (19-50 y)	1.6 (19-50 y)
			1.5 (50+ y)	(>50 y)
Pregnant		2.5	1.9	2.2
Lactating	0-6 m	2.5	2.0	2.1
	6-12 m	2.5	2.0	2.1

Breast milk of well-nourished American mothers has been reported to contain 0.1-0.25 mg vitamin B_6/L. The NRC has recommended additional allowance of 0.5 mg of vitamin B_6 per day during lactation. ICMR had earlier recommended the same level, and this can be retained (Table 12.4b), even though vitamin B_6 content in the breast milk of Indian women has been reported to be much lower (12.2.20, 12.4.5). FAO/WHO (12.1.5) has recommended an intake of 2.2 mg/d and 2.1 mg/day in pregnancy and lactation respectively (Table 12.4a).

Table 12.4b

RDA of vitamin B_6 for various physiological groups

Group	Category / Age	Body weights (kg)	Vitamin B_6 (mg/d)
Men	Sedentary Moderate Heavy	60	2.0
Women	Sedentary	55	2.0
	Moderate		
	Heavy		
	Pregnant	55	2.5
	Lactating 0-6 m 6-12 m	55	2.5 2.5
Infants	0-6 m	5.4	0.1
	6-12m	8.4	0.4
Children	1-3 y 4-6 y 7-9 y	12.9 18.0 25.1	0.9 0.9 1.6
Boys	10-12 y	34.3	1.6
Girls	10-12 y	35.0	1.6
Boys	13-15 y	47.6	2.0
Girls	13-15 y	46.6	2.0
Boys	16-17 y	55.4	2.0
Girls	16-17 y	52.1	2.0

c. Infants and children

Evidence of vitamin B_6 deficiency has been reported in infants who consumed less than 0.1 mg vitamin B_6 through breast milk (12.1.2). In one study, 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 B_6 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 12.4a). The breast milk vitamin B_6 content of Indian mothers was found to be only 60-80 µg/L (12.4.5). At this level of intake, no evidence of enzymatic vitamin B_6 deficiency was detected in exclusively breastfed, 1-6 months old infants (12.4.5). Till more information is available, this Committee considers the retention of the earlier recommendation which is close to the FAO/WHO recommendation for infants (Table 12.4b).

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12.5. FOLATE

The primary biochemical function of folic acid is to transfer a single carbon in the synthesis of a number of metabolites in the body. The folates found in food consist of a mixture of unsubstituted polyglutamyl tetrahydro folates and various substituted one carbon forms of tetrahydrofolate (e.g. 10-formyl-, 5, 10-methylene-, and 5-methyl-tetrahydrofolate). Along with vitamin B_{12}, folic acid is concerned with 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 absorbed unchanged and reduced post-absorptively to the dihydro and tetrahydro forms in cells.

Food folate and bioavailability

Folate is present in a wide variety of foods, both of plant and animal origin. Leafy vegetables, fruits and yeasts are particularly rich sources. Cereals and pulses contain fair amounts and pulses usually contain twice as much as cereals do. Folic acid 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 folic acid 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.

Dietary folate equivalent (DFE unit) was introduced to rationalize the mixed dietary folate and folic acid intakes (sourced from fortified foods) prevailing in developed countries as an integrated measure of both. DFEs are defined as: µg natural food folate + 1.7 x µg synthetic folic acid. The 1.7 multiplier was based on assumptions that extent of availability of added folic acid was 85% and that of food folate was only 50%. The 85/50 ratio also inferred that the bioavailability of food folate was about 60% relative to added folic acid (12.5.1).

Studies on actual availability of food folates are few. Over 70 % of the folates from egg, 60-70% from pulses and green leafy vegetables and 50% from vegetables are absorbed. 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 active coenzyme form ultimately, irrespective of the chemical form in which it was ingested (13.5.2). It has been assumed that 50% of the folate present in Indian diets is absorbed. A detailed study involving different Indian recipes

recently demonstrated that the average loss of folates during cooking is 33% and the density of Indian meal is about 50 µg/1000 kcal (12.5.3 or 12.5.4).

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) (12.5.5). Investigations report that the actual content of folates appears much more than what has been so far reported 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 microbial 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.

Deficiency

Chronic and severe forms of folic acid deficiency lead to abnormal hemopoiesis and megaloblastic anemia, which promptly responds to treatment with the vitamin. This anemia remains indistinguishable from the one produced by vitamin B_{12} deficiency. In recent years, both folic acid and B_{12} deficiency were found to be associated with elevated blood homocysteine level, which in turn was reported to be a major etiological risk factor of cardiovascular disease. 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. A very high prevalence of open NTD was reported in India. A recent multicentric study (12.5.6) with 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%).

On the other hand, mean dietary intake data consistently show low intakes of dietary folate in all age groups (12.5.4). A micronutrient survey in Eastern and North eastern part of the country suggested intakes of folate close to, or more than 100 µg /d. One of the striking features is that the proportion of subjects receiving less than RDA of 100 µg /d was 20-70 % (12.5.7) and, thus, the mean values are perhaps masking the extent of dietary lack. 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

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folate though their diet contained a mean intake of 155% RDA for folic acid (12.5.8). 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.

Megaloblastic anemia is not encountered frequently in general population, but it occurs occasionally in pregnant women from poor income groups. It is possible that the widespread iron deficiency (microcytic anemia) could mask megaloblastic anemia. 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 pregnancy (12.5.9). In a recent study, sub-clinical folate deficiency was found to be about 30% in pregnant women from rural North India (12.5.10). A high level of sub-clinical folate deficiency was also reported in semi urban school children (12.5.10). There are some sporadic reports indicating its prevalence in adults.

RDA for adults

Diet survey data indicate that the average Indian diet provides about 50-70 µg/d dietary folate computed as folic acid, and 50-60% of the adults do not get the current recommended allowance of 100 µg folic acid. Based on the first studies from India that an intake of 75 µg /d folic acid may ensure adequate blood folate levels, the previous ICMR Committee (12.5.11) made a recommendation of 100 µg folic acid per day. 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 them. In recent years, there are studies to show that about 33 % of the dietary folate is lost in Indian style of cooking (12.5.3) and 45-50% of the folate is absorbed. Till such time adequate data are available (on the lines indicated above), it is reasonable to assume that the bioavailable folic acid is about 1/3 of the total folate in Indian foods/diets. This factor accounts for 33% losses from cooking and assumes 50% absorption from the remaining. It is advisable to retain 75 µg physiological requirement of folic acid which can be obtained from 200 µg of dietary folate.

Revision of intakes for adults

Almost 30 years back, iron and folate supplements were demonstrated to be necessary for pregnant women to reduce anemia and increase the birth weights of newborn. A national Iron-folic acid supplementation program is in operation for over 30 years to prevent anemia in pregnant women and reduce prevalence of low birth weight. However, anemia continues to be a major problem and low birth weight is still unacceptably high and about 35% did not

receive the folate tablets altogether (NFHS 3study, 2005-06, Report published 2007). It is imperative that folate reserves are built up in women during non-pregnant, non-lactating state so that the risk of adverse outcome of pregnancy is minimized. The current recommendation of100 µg/d is extremely low as compared to the 200 µg/d suggested by FAO/WHO which is about 400µg as dietary folate. It is pertinent to note that these recommendations were based on the data on dietary folate generated by modern methods which yield a higher amount of folates than the conventional methods and the latter cannot be considered unless Indian dietary data is updated.

Table 12.5

RDA of dietary folate for various physiological groups

Group	Category/Age	Body weights (kg)	Dietary folate (µg/d*)
Men	Sedentary	60	200
	Moderate	60	
	Heavy	60	
Women	Sedentary	55	200
	Moderate	55	
	Heavy	55	
	Pregnant	55	500
	Lactating 0-6 m	55	300
	6-12 m		
Infants	0-6 m	5.4	25
	6-12m	8.4	
Children	1-3 y	12.9	80
	4-6 y	18.0	100
	7-9 y	25.1	120
Boys	10-12 y	34.3	140
Girls	10-12 y	35.0	
Boys	13-15 y	47.6	150
Girls	13-15 y	46.6	
Boys	16-17 y	55.4	200
Girls	16-17 y	52.1	

* 1 µg of food folate = 0.5 µg of synthetic folic acid taken on empty stomach and 0.6 µg folic acid taken with meals.

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 CVD (12.5.12). From the nutritional standpoint, such a steep hike does not seem to be justified. Taking the current level of intake and the prevalence of sub-clinical deficiency into consideration, 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 females (Table 12.5).

This Committee agrees with the previous Committee (1989, 12.5.11) over the conceptual basis on which additional requirements were added to the basic needs to obtain the dietary intakes necessary for different groups. Therefore, an additional requirement of 300 and 100 µg respectively during pregnancy and lactation was decided to be added for meeting the factorial extra needs. Similarly for children of 1-6y and 7-12 y, a factorial extra need of 40-50 µg and 60-70 µg respectively was added to the minimum daily intake to meet the RDA.

12.6. VITAMIN B_{12}

In the quest for finding the reasons for "pernicious anemia", vitamin B_{12} was discovered to be the 'extrinsic factor' necessary to react with the "intrinsic factor" of stomach to be absorbed and thereby prevent the disease. Vitamin B_{12} is a component of several coenzymes like that of folic acid. It also shares and complements the functions catalyzing the same set of metabolic reactions. Along with folate derivatives, Vitamin B_{12} coenzymes are involved in one carbon metabolism and ultimate synthesis and transfer of methyl groups essential for synthesis of nucleic acids and proteins.

Vitamin B_{12} is a co-enzyme for two enzymes, methionine synthase and methyl malonyl coenzyme mutase. The former uses methyl cobalamine and the latter uses 5' deoxyadenosylcobalamine or coenzyme B12. In nature, there are two other forms of vitamin B12 known as hydroxycobalamine and aquacobalamine and the synthetic form is knowm as cyanocobalamine. The above three forms of B12 are enzymatically activated to methyl or deoxyadenosyl cobalamines in all mammalian cells.

Food source

All the vitamin B_{12} found in nature is synthesized by microorganisms. The vitamin is absent in plant foods except when contaminated with microbes. Liver, meat, egg and milk are good sources. Microflora in the large intestine

produce vitamin B_{12} but the contribution from this source to the body needs is not evident. Since populations subsisting essentially on foods of vegetable origin do not show evidence of widespread vitamin B_{12} deficiency, it is speculated that polluted environment and unhygienic practices could be providing the necessary minimal vitamin B_{12}.

Deficiency and intake

Deficiency of vitamin B_{12} leads to abnormal hemopoiesis, leading to megaloblastic anemia, as in the case of folic acid deficiency. In addition, neurological manifestations due to sub-acute combined degeneration of spinal cord or demyelination of nerve fibres may also occur. However, this is rarely seen in India.

While clinical deficiency of B_{12} is not manifested, sub-clinical deficiency is reported to exist in the country. Recent studies from India indicate an appreciable extent of vitamin B_{12} deficiency occurring in women (>50%) (12.6.1, 12.6.2). Reports from other parts of the country also show that vitamin B_{12} deficiency could be more than 30% in adults and in children. According to a study done in Hyderabad, sub-clinical deficiency of vitamin B_{12} was about 45% in school-age children (12.5.8). It is not at all surprising that blood levels of vitamin B_{12} are low since a large proportion of the population depends on plant foods for nutrients. A possible link of maternal vitamin B_{12} deficiency coupled with folate excess to offspring adiposity and insulin resistance is attracting attention (12.6.1 and 12.6.3).

Requirements

It was established by three different methodologies based on dose-response, liver stores and radioisotopic turnover, that the amount of absorbed vitamin B_{12} is only 0.1 µg/d. The recommended allowance by WHO of 1 µg/d allows enough margins of safety for cooking losses, uncertainties in absorption and a small amount for storage. The previous ICMR Committee (1989, 12.5.11) accepted these observations and recommended an intake of 1 µg vitamin B_{12}/day. The Committee also estimated the mean intake to be around 0.7 µg when modest amounts of milk products and flesh foods are included. Thus there is no reason for revising the requirements of vitamin B_{12} and this Committee accepts the current value of 1 µg/d. It is encouraging that the animal food intake is showing an upward swing in recent years (NSSO, 12.6.4). However, recommendations should reflect the need for improving the content of healthy animal foods, including milk, in general, to improve the quality of diets.

Recent FAO/WHO Consultation (12.6.5) on B_{12} requirements recommended an intake of 2.4 µg/d for adult and 2.6 and 2.8 µg/d for

pregnancy and lactation, respectively. The recommendations reflected the estimated average requirement based on the intake by general populations subsisting mainly on animal foods, in contrast to predominantly vegetarian foods that Indians consume. In view of the differing criteria, the present ICMR Committee also retains the earlier recommendation (Table 12.6).

Table 12.6

RDA of vitamin B_{12} for various physiological groups

Group	Vitamin B_{12} (µg/d)
Men	1.0
Women (NPNL)	1.0
Pregnant women	1.2
Lactating women	1.5
Infancy	0.2
Children 1-9 y	0.2-1.0
Adolescents 10-12 y	0.2-1.0
Adolescents 13-17y	0.2-1.0

It is very essential to assess the dietary intake of vitamin B_{12} of different segments of the population and also the estimates of deficiency in the country as well as measures to improve vitamin B_{12} content of milk, which is the widely consumed animal food.

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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 many enzymes. e.g. hydroxylation of collagen, biosynthesis of carnitine , and catecholamines (12.7.1). Ascorbic acid in gastric juice has been shown to prevent the formation of N-nitroso compounds, which are potentially mutagenic (12.7.2). High intakes of ascorbic acid correlate with reduced gastric cancer risk (12.7.3). Ascorbic acid protects low-density lipoproteins *ex vivo* against oxidation and may function similarly in the blood.

Anemia is a well known feature of ascorbic acid deficiency in man and animals. 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 scurvy (12.7.4). Similarly, Ascorbic acid promotes absorption of soluble non-heme iron possibly either by chelation or simply by maintaining the iron in the reduced (ferrous, Fe^{2+}) 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 meal (12.7.5).

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 7.1

Stability of ascorbic acid during processing

acid is sensitive to heat, light and oxygen and interacts with nd tin (12.7.6). 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 deleterious (12.7.7). Oxidative destruction of ascorbic acid occurs as a consequence of two factors, temperature increase and length of exposure.

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%.

Heat processing methods have different impacts on ascorbic acid stability. After sterilization of milk at 131°C for 5 minutes, retention was 35% while ultra high temperature processing at 141°C for 4 seconds retains 57% of the original ascorbic acid content (12.7.8). 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% (12.7.9). Boiling causes losses to an average 10% higher than with steaming (12.7.10). 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 vitamins (12.7.11). 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 (12.7.12, 12.7.13). Traditional household processing method used in Punjab was found to retain about 74% and provided 46 mg of ascorbic acid per 100 g (12.7.14). 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 cooked (12.7.15).

12.7.4. Requirement

Human ascorbic acid requirement has been determined by depletion and repletion studies. Minimum daily intake of ascorbic acid to cure scurvy is as low as 6.5 mg. The minimum intake to saturate leucocytes after depletion of adult volunteers was found to be between 10 and 20 mg. However, the daily intake of ascorbic acid has been fixed at 40 mg by the previous ICMR Committees based on the presumption that 50% of ascorbic acid may be lost on cooking or storage of foods rich in ascorbic acid (12.7.16). While advanced countries like USA and UK revised the ascorbic acid requirements upward of 60 mg based on its antioxidant and other health benefits, WHO recommendations continued to be held around 45 mg from the nutritional standpoint.

Adults

The minimum requirement of ascorbic acid has been determined by controlled human experiments. Studies carried out by the Medical Research Council, UK, have shown that the minimum amount of ascorbic acid needed to prevent or cure scurvy is about 10 mg daily(12.7.17). Studies in human volunteers have confirmed these findings (12.7.18, 12.7.19). These studies have also shown that although urinary levels of ascorbic acid fall rapidly after ascorbic acid intakes are drastically lowered, clinical signs of scurvy do not appear until the body pool of the vitamin is reduced to less than 30 mg. Administration of measured amounts of ascorbic acid showed that 6.5 mg/day was the absolute minimum needed to cure the disease.

There are several studies on the urinary excretion of ascorbic acid and response to load test in Indian subjects. Although these tests provide some data on the adequacy of ascorbic acid intakes, they do not provide adequate information to formulate requirements (12.7.20).

Concentrations of ascorbic acid in plasma or blood are poor indicators of ascorbic acid status. It is now accepted that concentration of the vitamin in circulating leukocytes reflects tissue concentrations. Ascorbic acid requirements of Indian adults were determined based on intakes necessary to maintain the vitamin in leukocytes at saturation level (12.7.21). After saturating the leukocytes with large doses of the vitamin, it was observed that in some subjects, intake of 10 mg/day was sufficient to maintain these levels while in others it tended to decline. When the intake was raised to 20 mg, however,

concentrations of ascorbic acid in leukocytes could be maintained at saturation level for as long as 100 days. On the basis of these studies, the Committee considered that a daily intake of 20 mg of ascorbic acid is sufficient to maintain a satisfactory level of ascorbic acid in leukocytes. Taking into account that about 50% of ascorbic acid is lost in cooking, a dietary allowance of 40 mg per day is recommended (12.7.16).

Pregnancy and lactation

There are no new data to indicate that the ascorbic acid requirement is increased during pregnancy. Foetal requirement is too small to justify an additional intake. Therefore, extra allowance may not be necessary since the allowance for normal adults includes a sufficient safety margin. However, the Committee accepts the recommendation made by FAO/WHO (12.7.30) of an extra 10 mg/day throughout pregnancy which should enable reserves to accumulate to meet the extra needs of the growing foetus in the last trimester. Considering the cooking loss of 50%, the RDA of ascorbic acid has been set at 60mg/day.

Additional needs during lactation are calculated on the basis of the ascorbic acid secreted in milk. Assuming a daily milk secretion of 700 ml with an ascorbic acid content of 3 mg/dl by well-nourished women, additional requirement during lactation will be 20 mg and therefore the Committee recommends an intake of 80 mg per day during lactation.

Infants and children

On the basis of ascorbic acid ingested by breast fed infants in well-nourished populations, the earlier Committee recommended an intake of 25 mg ascorbic acid per day for infants. The present Committee also recommends a daily intake of 25 mg ascorbic acid for infants and 40 mg for children.

12.7.5. RDA for ascorbic acid

The Committee considered the beneficial effects of ascorbic acid on non-heme iron absorption. To derive this benefit, however, ascorbic acid and iron would have to be consumed at the same time in the same meal. It is imperative that the RDA of ascorbic acid may have to be at a level that enhances absorption of non-heme iron. The Committee has evaluated all the evidence available on this subject, and decided to retain the earlier recommendations of RDA for ascorbic acid for all age groups except for pregnancy (Table 12.7.2).

Table 12.7.2

RDA of ascorbic acid (vitamin C) for various physiological groups

Group	Category / Age		Vitamin C (mg/d)
Men			40
Women (NPNL)			40
Pregnant women			60
Lactating women			80
Infants	0 – 6 m		25
	6 – 12 m		
Children	1 – 3 y		40
	4 – 6 y		
	7 – 9 y		
Adolescents	Boys	10-12 y	40
	Girls	10-12 y	
	Boys	13-15 y	40
	Girls	13-15 y	
	Boys	16-17 y	40
	Girls	16-17 y	

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 ensured (12.7.22). An adequate intake of ascorbic acid is however required in the context of fixing the requirement of iron from vegetable foods. The present Committee 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 meal (12.7.23). 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

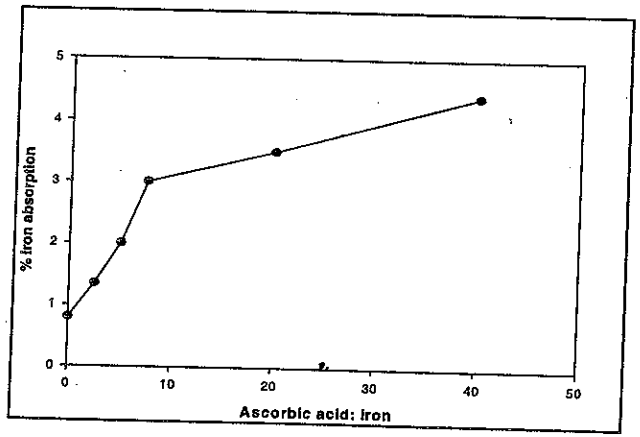
Promotion of iron absorption in the presence of ascorbic acid is more pronounced in a composite meal containing inhibitors of iron absorption. The effect of ascorbic acid and other enhancers or inhibitors of iron absorption

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.

A meal containing low-to-medium levels of inhibitors requires the addition of ascorbic acid at a molar ratio of 2:1 (e.g., 20mg ascorbic acid : 3mg iron) (12.7.24). 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 impractical (12.7.25). 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. (12.7.23,12.7.24). 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 Reddy (12.7.26) 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 status (12.7.14, 12.7.27, 12.7.28). Thus, 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



12.7.6. Increased intake of food rich in ascorbic acid

Increased ascorbic acid intakes through habitual consumption of ascorbic acid rich foods represent an important strategy to meet the iron requirement of Indian population. Ascorbic acid intake well below the RDA has been reported in recent survey of NNMB (12.7.29). Intake of ascorbic acid at household level was only 70% of RDA (RDA=40 mg). An increase in intake, either by increasing the total intake of ascorbic acid or by changing meal pattern, may therefore represent a sustainable way of improving the iron status in such populations. Feasibility of this approach will depend on sustained behavioural changes and access to ascorbic acid-rich foods. An intervention trial carried out in India found a significant improvement in hemoglobin levels in young working women receiving ascorbic acid -rich gooseberry juice (extra 20 mg ascorbic acid per day) and with lunch (total ascorbic acid intake to 49 mg/day) at their work place for 6 months (12.7.27).

There is enough evidence from recent studies that the average bioavailable density of Fe in Indian diets is 0.45 mg/1000 kcal, which is enough to meet iron requirements in normal physiological states when the energy intake is adequate. This view is supported by the latest WHO recommendation (12.7.34), in fixing the ascorbic acid RDA at 40 mg/d. Thus, the present Committee comes to the conclusion that there is no need of increasing the requirements and RDA of ascorbic acid to enhance dietary iron absorption. However, the importance of ensuring adequate intake of ascorbic acid and other related nutrients like folic acid is the most important measure to control anemia in Indian population.

Ascorbic acid fortification

Absorption of iron from fortified cereals can be increased two-fold to three-fold if the cereals are also fortified with about 5 mg of ascorbic acid per mg of iron (12.7.31, 12.7.32). Some of the effectiveness of iron-fortified infant formulas in preventing iron deficiency has been attributed to their fortification with this vitamin (12.7.33).

12.7.7. Desirable level of ascorbic acid in a meal to improve iron absorption among Indians on a vegetarian diet

The above review of studies on the effect of ascorbic acid on iron absorption from meals has shown that a molar ratio above 2:1 of ascorbic acid to iron in the meal can bring about a remarkable improvement in iron absorption. Based on current intake, it is suggested that at least 20 mg of ascorbic acid should be distributed in all meals in 2:1 molar ratio to improve iron absorption from a typical Indian meal.

Indian diet contains about 7 mg of iron per 1000 kcal (refer Chapter 9). It is conditional that density of ascorbic acid should be at least 20 mg/1000 kcal (3 times by weight of iron to achieve 1:2 iron to ascorbic acid) to ensure iron absorption of 5% in all groups.

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13. FAT SOLUBLE VITAMINS

13.1. VITAMIN A

Significance

Vitamin A (Retinol) is essential for normal vision, for maintaining the integrity of epithelial tissues and for a wide variety of metabolic functions. Considerable amounts of vitamin A can be stored in the liver and made available for use as the need arises. Although the role of vitamin A in the visual process is well known, its mechanism of action in other metabolic processes is, as yet, not well understood.

Deficiency

One of the earliest manifestations of vitamin A deficiency is night blindness and more severe deficiencies include ocular changes leading to blindness, particularly in young children. Though there has been a significant reduction in severe clinical forms of Vitamin A deficiency over the last three decades, vitamin A deficiency is still a public health problem with 40-60% child population in developing countries showing inadequate biochemical status of vitamin A. Very low intakes, poor bio-availability of pro-vitamin A from the predominantly vegetarian diet and recurrent infections are thought to be the main reasons for widespread prevalence of vitamin A deficiency.

Dietary sources

Foods provide vitamin A either in the preformed state (directly as retinol or retinyl esters of fatty acids) from animal sources such as milk, butter, egg and fish or its precursor carotenoids, especially β carotene, derived from leafy vegetables and yellow- and orange-coloured fruits and vegetables. β-carotene and other carotenoids are variably converted into vitamin A in the body. Since β-carotene forms a major source of dietary vitamin A in many developing countries including India, the efficiency with which it is absorbed and utilized, becomes important in translating β-carotene values into retinal equivalents (13.1.1). Although the enzymatic conversion of one molecule of β-carotene to retinol should theoretically result in two molecules of retinol, because of physiological inefficiency, the maximum conversion that has been shown in experimental animals is around 50%. In addition, the efficiency of β-carotene absorption is variable, depending upon the food source. Red palm oil contains easily absorbable carotenes. Based upon available evidence, the FAO/WHO Expert Group (13.1.2) assumed that about one-third of dietary β-carotene is absorbed. As a result, a factor of 0.16 was used to convert β-carotene to retinol. In two studies carried out in India, one in adults (13.1.3)

and the other in children (13.1.4), absorption of β-carotene from green leafy vegetables was found to be much higher, ranging from 50 to 99%. Lower figures were obtained for absorption of β-carotene from other sources such as carrots and papayas.

Requirements for adults

The previous ICMR Committee (13.1.5) considered the evidence produced by Olson (13.1.6) from vitamin A turnover data, the liver levels of vitamin A in free living, well-nourished population and their dietary intakes, proposing requirements of 600 RE for a 65 kg man which works out to 9.3 RE/kg body weight. This fundamental concept was accepted by the previous Committee which worked out the dietary requirements (as RE, assuming 1:4 ratio for different age groups). The present Committee modifies only the extent of conversion efficiency to 1:8 and retains the recommendations on retinol requirements for all groups except pregnant women (Table 13.1.1). The Committee recommends that a minimum of 50% RE be drawn from animal sources to ensure adequacy at least in vulnerable groups like pregnant and lactating women. It should be considered essential to consume other carotene sources than only green leafy vegetables to overcome the adverse conversion ratio associated with them. Detailed considerations for employing a conversion ratio of 1:8 is described in Annexure 13.1.

Table 13.1.1

RDA of Vitamin A for various physiological groups and its comparison with FAO/WHO (2004)

(Retinol Equivalents, RE, µg/ d)

Group	Age	FAO/WHO 2004	Present Committee	
		Vitamin A	Vitamin A	β-carotene*
Men		600	600	4800
Women (NPNL)		500		
Pregnant women		800	800	6400
Lactating women		850	950	7600
Infants	0-6 m	375	350	---
	6-12 m	400		2100
Children	1-6 y	400/450	400	3200
	7-9 y	500	600	4800
Adolescents	10-17 y	600	600	4800

* A conversion ratio of 1:8 is used.

Pregnancy and lactation

There have been no direct studies to quantitate the increase in vitamin A requirements during pregnancy and lactation. Vitamin A requirements during pregnancy have been calculated on the basis of the vitamin content of livers of the newborn. Additional intake of vitamin A required for this purpose is about 25 µg/day throughout pregnancy. Since this constitutes a very small fraction of the recommended allowance for normal women, no additional dietary allowance during pregnancy was suggested earlier.

Recently, a series of studies (13.1.7, 13.1.8) on pregnant women were carried out at NIN, correlating the duration of vitamin A supplementation that was required to prevent the fall of plasma vitamin A invariably occurring in the last few weeks of pregnancy, of those who did not receive any supplement. It was found that a daily dose of 780 RE was able to not only prevent the fall in plasma level but also increase neonatal status. Such amounts of intake were associated with improved feto-placental function reversing the changes in protein catabolism and oxidative stress. Thus, the Committee accepts the requirement level suggested for pregnancy of 800 RE on the basis of these studies.

The additional needs during lactation are calculated on the basis of vitamin A secreted in milk. The FAO/WHO Expert Group (13.1.2) used an average milk secretion of 700 ml/day with a retinol content of 50 µg/dl (a value observed in well-nourished population) and recommended an additional intake of 350 µg vitamin A per day during lactation. This intake of 350 µg/day of vitamin A by Indian nursing mothers is in keeping with the intake allowance for infants aged below 6 months. The mean vitamin A content of breast milk of Indian women has been reported to be 21 µg/100 dl. These women, however, were known to consume diets which were deficient in vitamin A.

Infants and children

In the absence of direct evidence, recommended intakes of infants and children are calculated on the basis of vitamin A intake through breast milk for infants and the values extrapolated for children. Daily intake of vitamin A by Indian infants through breast milk is about 140 µg during the first six months of life (13.1.9). Such infants grow well and do not show any deficiency. It is however possible that they are drawing on their reserves. This possibility is supported by the observation that children of such communities do develop deficiency signs during early childhood. Intake of 140 µg/day thus appears to be inadequate. On the basis of vitamin A ingested by breast-fed infants in well-nourished communities, the Expert

Group (13.1.2) has recommended a daily allowance of 350 µg retinol up to the age of six months. In the absence of specific data on the needs of infants from 6-12 months of age, the same intake of 350 µg/day has been suggested during the latter half of infancy too. Vitamin A requirements of the children of other ages have been computed from the requirement/d figures for infants (50 µg/kg) and adults (9.3 µg/kg) taking into account growth rates at different ages (13.1.2). On this basis, vitamin A requirement of children will be 300-400 µg retinol/day. Incidence of vitamin A deficiency signs is high and serum vitamin A levels are generally low among Indian children whose dietary intake is less than 100 µg. In a group of children receiving food supplements, which provided a total of 300 µg of vitamin A per day over a period of 6 months, serum vitamin A levels were found to be around 30 µg/dl and clinical signs of vitamin A deficiency were rarely seen (13.1.10).

A health and nutrition survey conducted on tribals in three ecological zones of Madhya Pradesh had shed light on vitamin requirement in 1-6 yr old children. (13.1.11). The results on 1401 pre-school age children selected from the three zones showed that though the milk intake was very low in all districts, the people consumed a variety of pulses, leafy vegetables, roots and tubers, and wild fruits. This tribal sample had low clinical forms of protein energy malnutrition and vitamin A deficiency compared to others in the rural districts. Bitot's spots were not at all seen in Sarguja and Bastar areas with vitamin A intakes (µg/d using 1: 4 conversion of dietary β-carotene) ranging from 216 to 419 µg in 1-6 year age children. It was seen in 0.3% in Jhabua area with an intake of 170-244 µg. The Committee therefore recommends a daily allowance of 400 µg retinol for preschool children and 500 µg for school age children and 600 µg (the adult figure) for adolescents.

The requirements suggested for infants, children and pregnant and lactating women and for the bodily conversion factor of β-carotene to vitamin A have been taken into account in the recommended vitamin A intakes for various groups as given in Table (13.1.1). The figures are similar to what were recommended by the 1988 Expert Committee. However, the pro-vitamin A values are twice that recommended earlier.

13.2. VITAMIN D

Vitamin D is a unique vitamin and its availability in the body largely depends on its synthesis in the skin when exposed to sunlight and hence, its dietary requirement is usually very small especially in the Indian context. Apart from its classical calcitropic etiological role in the development of rickets (in children) and osteomalacia (in adults), leading to weak bones and physical deformation, vitamin D has been found to have many non-calcitropic health promoting effects. Diseases such as osteoporosis, diabetes, psoriasis, hypertension, arthritis, multiple sclerosis and CVD are known to have the

involvement of vitamin D leading to the suggestion that the above spectrum of deficiency diseases be called vitamin D deficiency diseases (VDDD) as in the case of other micronutrients (13.2.1).

Diet

As indicated above, vitamin D (D_3, cholecalciferol) can be photolytically formed from 7-dehydrocholesterol (as provitamin D_3) in the skin when exposed to sunlight (UV-B rays). It can be also obtained from the diet both through plant sources as provitamin D_2 (ergosterol) and get converted to D_2 (ergocalciferol) in the skin or through animal foods as preformed vitamin D_3 or its provitamin. It is estimated that about one tenth of the body's requirement is derived from dietary sources in the Indian context; mainly the exposure to sunlight being the limiting factor (Table 13.2.1).

Table 13.2.1

Vitamin D status and exposure to sunlight

Group	Max. Exp. time to sunlight (min/d)	25 OH D* (nmol/l)	PTH level* (ng/l)
Soldiers	370	47	17
Physicians/ nurses	25	8	39
Depigmented	5	18.2	35.5

* Differences between all groups were significant.

Deficiency

Vitamin D deficiency 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 and 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* (13.2.2). In recent years, in endemic areas of fluorosis, excess of fluoride in drinking water was found to be associated with rachitic bone deformities and VDD in young children (13.2.3).

Role

Vitamin D is activated in two series of hydroxylation reactions in liver and kidney to active hormones endogenously which mediate increased intestinal absorption and renal tubular resorption of calcium and contribute to an increase in the availability of calcium. The first hydroxylated compound, 25 hydroxy cholecalciferol (25HCC) and the next 1, 25 dihydroxy CC (1,25 HCC) are the well known metabolites of vitamin D. The first one is the circulating and storage form and represents the status of vitamin D. 1,25 DHCC is the active hormone metabolite which induces the formation of calcium transport protein involved in calcium absorption. Secondarily to calcium, phosphorus absorption takes place in the intestine. Blood calcium 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 thus normalize blood calcium. Lack of vitamin D will reflect in alterations in the calcitropic hormones and not always in serum calcium.

Requirement

Vitamin D is now considered more as a pro-hormone, than as a vitamin. It can be synthesized in the body in adequate amounts by simple exposure to bright sunlight even for 5 min per day. Habitual Indian diets do not provide even 10% of the requirement. 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 Committees recommended 100 Units (2.5 µg) /d for adult males in 1988 (13.2.4) and increased them later in 2004 to 200 Units (5 µg)/d (13.2.5). This is obviously due to progressive decrease in the exposure to sunlight and the need to obtain the requirements from dietary and supplement sources. Hence, many foods such as milk and vegetable oils are subjected to mandatory fortification with vitamin D in the developed countries.

RDA comments

Recommendations of advanced 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. Also, populations from many advanced 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. It is pertinent to note that even in India, young growing children and adults, particularly in urban areas, are physically less active and are not being exposed outdoors. This reduces the chances of vitamin D formation (Table 13.2.1). This is being increasingly felt as more and more studies are reporting reduced circulating levels of 25 HCC in children of school age (13.2.6) and also in some adults (13.2.7). Another preliminary report of Rege *et al* (13.2.8) demonstrated graded lowering in mean 25 HCC levels (ng/ml) in men in rural (25.5), urban slums (16.9) and urban middle class (12.20) in consonance with their expected outdoor activity. It is, therefore, felt that increasing the RDI is not the solution but ensuring adequate exposure to sunlight is. Whiting and Calvo reviewed more than 80 studies to draw similar inference and emphasized that food supply cannot be a substitute for vitamin D supply in place of sunlight (13.2.9). Therefore, the Committee agrees that outdoor physical activity is a means of achieving both adequate vitamin D status and controlling overweight and obesity in the population. This is reflected in the recommendations and no specific suggestions are made on the intakes of different groups. However, under situations of minimal exposure to sunlight, a specific recommendation of a daily supplement of 400 IU (10 µg) is retained.

13.3. VITAMIN E (ALPHA TOCOPHEROL) AND VITAMIN K

There are very limited data on vitamin E (alpha tocopherol) and vitamin K intake as well as requirements. They occur widely in vegetable oils and plant foods and dietary deficiencies of these two vitamins are not normally encountered.

Available information indicates that blood levels of alpha tocopherolamong Indians is satisfactory (13.3.1, 13.3.2). Alpha tocopherol content of vegetable oils and invisible fat in cereals and other foods is adequate. Requirement is limited to essential fatty acid intake.

Alpha 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 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 alpha tocopherol levels of 0.5 mg/kg/ml which is regarded as satisfactory. Alpha tocopherol requirements are related directly to the levels of essential fatty acids (linoleic and linolenic acids). The suggested requirement of alpha 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/WHO (13.1.27) suggested an RDA of 7.5-10 mg α-tocopherol and 55 µg of vitamin K /d for adults. In the Indian context, limited data-base 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/or WHO are accepted. The safe intakes of different groups may be considered on the basis of uniform energy density / density of PUFA energy.

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There are some very recent stable isotopic data in Bangladeshi population

Annexure 13.1

13.1. Vitamin A

Retinol equivalents- Validation

To verify which of the five different conversion factors, 1: 4, 6, 8, 12 and above 15 is closer to the reality in the populations, UNICEF data on dietary intake of vitamin A (both preformed and carotene-derived from different regions of the globe) were compared with the corresponding biochemical VAD (less than 20 µg/dl in plasma) figures of the populations. Table below provides the data source used for obtaining the comparative information. All figures provide a comparison of the regionwise prevalence of sub-clinical deficiency and the dietary level of retinol derived from intake records available with International Agencies (13.1.12 and 13.1.13). Each figure adopts carotenoids conversion according to the different ratios suggested by ICMR 1989 (1:4), WHO (RE, 1:6), IOM (RAE, 1:12) or and the other studies (1:8) (13.1.5, 13.1.2, 13.1.14, 13.1.8 & 13.1.15). This could help to pick up the best for the association and compatibility of sub-clinical and dietary deficiency.

Table: Dietary retinol values (µg/d)* with (preformed retinol, %) and sub-clinical vitamin A deficiency reported in different regions of the world

Region	Sub-clinical VAD % prev.	Diet vitamin A ratio 1:4[1]	Diet vitamin A ratio 1:6[2]	Diet vitamin A ratio 1:12[3]	Diet vitamin A ratio 1:8[4]
Africa	49	1102 (11)	776 (16)	449 (27)	612 (19)
South Asia	47	617 (12)	435 (16)	253(28)	344 (21)
SEAR	69	619 (9)	431 (12)	242 (22)	337 (16)
East Europe	22	971 (28)	738 (37)	504 (54)	621 (44)
East Medit	22	1231 (28)	936 (37)	640 (54)	788 (44)
Americas	20	1073 (27)	814 (36)	554 (53)	684 (43)
West Pacif	27	1388 (15)	997 (22)	606 (36)	802 (27)
MENA	29	1258 (27)	951 (36)	644 (52)	797 (42)
WORLD	30	1065 (21)	785 (29)	506 (45)	645 (35)
Correlation Coefficent		-0.7025 P<0.05	-0.7629 P<0.02	-0.6616 NS	-0.8115 P<0.01

* Total retinol = retinol µg derived from dietary carotene intake (plant sources) + preformed retinol from animal sources. A prevalence rate of more than 20% indicates public health magnitude of vitamin A deficiency.

References: 13.1.8, [1]13.1.5, [2]13.1.2, [3]13.1. 14, [4]13.1.15

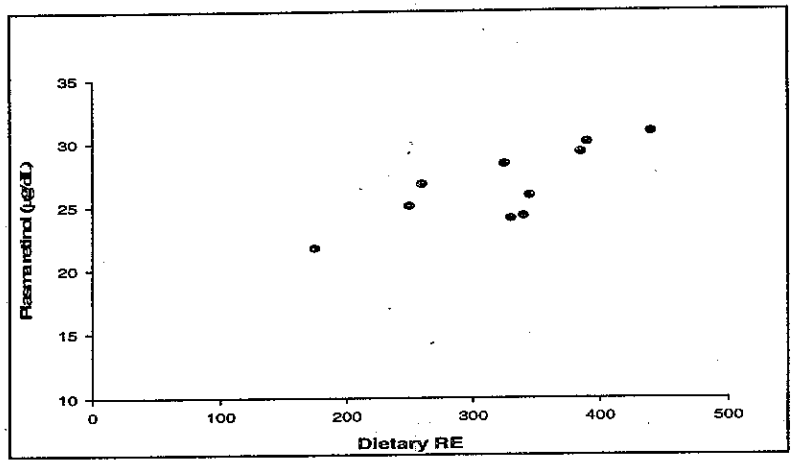
The rationale is that regions with total retinol intake of the diets above RDA (600 µg RE), should be associated with absence or least sub-clinical deficiency. A prevalence rate of 20% and above was globally accepted as a measure of public health magnitude of vitamin A deficiency. Earlier, clinical and sub-clinical deficiency were found to be associated with populations mostly deriving their daily vitamin A from vegetable sources. It is interesting to note that a ratio of 1:4 is too high and 1: 12 and above is too low to correlate with the data. At a ratio of 12, most regions had inadequate intake (less than RDA of 600 µg RE) and in addition, those regions with an inadequate intake <600 RE have both higher (> 40%) or lower (< 20-22%) prevalence rates. On the other hand, at a ratio of 1:6, levels of intake of 1000 and 780 RE were associated with 27 and 40% prevalence of sub-clinical deficiency. However a ratio of 1:8 seems to be adequate as the concurrence level seems to be the best between intake and deficiency reported in different regions. Also, the main aim of promoting horticultural approach and home gardening for greens by WHO and other agencies to control vitamin A deficiency in developing countries and the successes achieved so far in this regard in the past, will be kept in a shadow of doubt if one were to agree with the poor bio-availability and conversion of 1: 21 or 27 as proposed by some reports (13.1.16). It is also evident that along with total intake as retinol equivalents, the proportion contributed by preformed vitamin A >40% is associated with lower level of pre-clinical vitamin A deficiency. This observation may explain some part of the discordance observed between the efficacy of dietary carotene conversion and sub-clinical deficiency and would suggest that a higher proportion of preformed vitamin A from animal food sources may be recommended for groups requiring higher amounts of vitamin A.

The other way to look at the data is to plot the % prevalence of sub-clinical deficiency of vitamin A against the intake of total retinol derived from the above table. The set of data that shows best fit with highest negative correlation coefficient is considered to be the one closest to the real situation existing in the population. It is clear that retinol data obtained with a conversion ratio of 1: 12 is not significantly related to the deficiency state and thus, may not be practically correct. On the other hand, the correlations were significant with the other set of conversions data and the correlation coefficient of 0.81 was the highest with 1:8 conversion ratio as demonstrated in the table above.

In a recent study, Chiplonkar *et al* (13.1.17) compared the dietary retinol intake (RE, using a conversion ratio of 1:4 as per ICMR committee) and vitamin A inadequacy as reflected by plasma levels of vitamin A in healthy adult men and women drawn from both rural and urban setting classified into 5 different socio-economic strata. These results are plotted in Fig 13.1.1. Results show a significant positive correlation between the two variables

(n=10, r= 0.794, P< 0.01; Y= 18.3 + 0.0224 X) as expected. To obtain a mean plasma vitamin A of 30 µg/dL, which is considered absolute normal, the median intake is calculated to be 520 RE. The recommended adult value of 600 RE is expected to provide enough margin of additional retinol to cover the variation and be adequate for substantial population.

Figure 13.1.1
Dietary retinol (median) and plasma retinol in different groups



Reference 13.1.16
n=10, r= 0.794, P< 0.01; Y= 18.3 + 0.0224 X

Evaluation of green leafy vegetables and spirulina as dietary source of β-carotene

There are some very recent stable isotopic data in Bangladeshi population (13.1.18) suggesting that conversion of dietary β-carotene from leafy vegetable could be better than 12 (low about 10). Other studies from developing countries and particularly from India showed that long-term consumption of green leafy vegetables and other carotene-rich sources like spirulina were compatible with adequate or improved vitamin A status in target groups and the increase in plasma vitamin A was close to that expected at a ratio of 6 (13.1.19-13.1.25). All these studies point out that conversion is not very poor as suggested by earlier stable isotope data and as recommended in Western countries. Discordance in the data may be related to the reported higher efficiency of intestinal conversion of carotene to vitamin A in at-risk population. Animal experiments also provide similar

evidence. There have been also doubts that modern methods involving the use of stable isotope have not been sufficiently validated and there could be errors both in equilibration of the stable isotope and pool size estimates in such studies. Some of these aspects have been recently reviewed (13.1.26). The Committee has recommended a conversion ratio of 8 for beta-carotene to vitamin A. The recent FAO/WHO Expert Group (2004) however, has recommended a higher ratio of 1: 14 for beta carotene (13.1.27).

Finally, there are reports to indicate that a minimum daily dose of 50µg retinol (50 RE) is necessary to show a detectable increase in plasma vitamin A in 2-5 y children. Thus, where a significant change in circulating vitamin A is shown after consumption of carotene-rich foods as in the above reports (13.1.19-13.1.24), the obvious extra intake should be at least 50 RE. This amount of retinol as per retinol conversion ratio equivalent is 480 µg β-carotene per day (13.1.8, 13.1.15, 13.1.28). This quantity of carotene is closer to the amounts actually supplemented as in relative dose response (RDR) test and is more practical than the figure of >700 µg β-carotene, if the ratio had been as high as 12 or more.

14. ANTIOXIDANTS

14.1. Introduction

The role of antioxidants (AO) in oxidant or free radical scavenging is of great current interest to the biomedical community and the common public.

Antioxidants are substances which are both nutrients, viz. vitamins E, C, β-carotene, selenium and non-nutrients, viz. plant phenols, flavonoids, coumarins, benzyl isothiocyanates, caffeic, ferrulic, 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 result in oxidant 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 beverages such as tea and wine and cereals are excellent sources of AO, scientific evidence for their protective role is available only for vegetables and fruits in several chronic disorders (14.1, 14.2). None of the randomized clinical trials however conducted so far with nutrient AO supplements has demonstrated a significant benefit in community trials barring one or two major trials in high-risk populations. The following paragraphs review briefly the published studies and examine the question whether at all specific recommended dietary allowances for antioxidants could be fixed now.

14.2. Basic scientific studies

Experimental studies have amply indicated that both pro-oxidant and AO have a fundamental role in pathogenesis of diseases (14.3, 14.4). 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, neuro-degenerative disorders and age-related masculopathy. 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 isothiocynides, caffeic, ferulic, gallic and ellagic acids (14.5, 14.6). Polyphenols are more complex and of great diversity in structure, bio-

availability and functions (14.5, 14.6). 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 anti-oxidant 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 disappointing (14.7).

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.

14.3. 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 has provided the necessary evidence to increase the intake of antioxidants (14.8). The Cambridge Heart Antioxidant study provides some support for vitamin E (400-800mg) supplements for decreasing mortality in patients with myocardial infarction (14.9). On the other hand, studies on cancer (14.10, 14.11) in smokers where β-carotene supplements were given in two separate studies, increased risk of cancer and fatal cardiac incidence were noted (14.12). There were no effects on colo-rectal 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) (14.13, 14.14) and gastric cancer mortality in China (selenium, β-carotene and zinc) reported regression of lesions and 13% reduction in mortality respectively (14.15). 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 cancer (14.16). 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 (14.17, 14.18). 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 relapse(14.19). 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 mortality (14.20). A Cochran database review of well-controlled studies on vitamin-mineral supplements to control age-related mascular degeneration (AMD) found a beneficial effect of antioxidants (β-carotene, vitamins C and E and zinc) on progression to advanced stage.

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 cancer (14.21) ,CVD and other disorders (14.22). 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.

14.4. 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 (400 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.

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15. RECOMMENDATIONS FOR FUTURE RESEARCH

1. A National survey of body weights of healthy adequately nourished population of different age groups, by gender, residing in different urban and rural areas of the country should be taken up. Currently it is obtained from 95th percentile of body weights and heights collected from 16 States.

2. A National survey of adult population of urban and rural areas and their occupational level is essential to obtain for calculation of PAL

3. Currently, we are using the FAO/WHO equation adjusting for 5% lower BMR for Indians. Therefore, there is a need to generate National database on the physical activity level (PAL) of adults, children and pregnant women can be measured along with body weights and heights among different segments of healthy and well-nourished population in different parts of the country. Such large data base will help in setting up equations for deriving PAL and BMR.

4. Direct determination of energy requirement of Indian infants, preschool children, children up to 10 years and adolescents employing the DLW and HRM methods is an important area that needs to be addressed. Estimation of energy requirement of adults engaged in different life-styles employing the factorial method and comparing the results with those obtained with DLW or HRM methods.

5. Nutrient requirements of special groups such as those engaged in heavy physical work, and sports persons need to be examined.

6. There is a need to estimate accurately carbohydrate content of our major food source. This can be done as a part of the ongoing activity of revising food composition tables. Concurrently characteristics of carbohydrates from our major foods and their role in the control of diabetes need to be addressed.

7. Dietary fiber as conceived today needs to be assessed.

8. Fatty acid composition of breast milk is an area of interest. ALA to DHA and EPA conversion ratios in the context of Indian diet should be investigated.

9. More systematic calcium balance and vitamin D status accompanied by bone density (DEXA) studies have to be done in normal subjects (adolescents, postmenopausal women and men post 65 y).

10. Studies on sodium and potassium balance among the different population considering their activity pattern is an area of research.
11. A broader data base on the iodine content of Indian foods collected from different regions of the country should be created for computation of intake.

12. Research to assess the bioavailability of iron, zinc and their requirements in Indians on typical Indian diets is necessary.

13. More systematic studies have to be carried out to assess the trace element (Cu, Mn and Cr) content of different foods and their excretion, absorption and bioavailability from Indian diets among Indian population.

14. Dietary content of Selenium, status and excretion need to be carried out to understand selenium nutriture.

15. The current estimates regarding requirement and allowances of thiamine, riboflavin and vitamin B_6 are based on rather limited experimental evidence and hence are at best tentative. Neither NRC nor FAO/WHO base their recommendations for thiamine and riboflavin allowance on physical activity, and corresponding calorie allowance. Therefore there is a need for more research on these vitamin status among different segments of population, their diet and optimum needs.

16. Extensive data need to be generated on folate in Indian foods, absorption and status. The extent of folate deficiency also needs to be mapped.

17. Based on the emerging data on deficiency of vitamin B_{12} indicated by the available research, there is a need to map the deficiency across the country, including its possible implications and to define RDA. There is a need to understand the requirement of this vitamin in Indians who are on a predominantly vegetarian diet. The implications of gut health and its possible relation to vitamin B_{12} requirement need to be assessed. This data is important to have a policy on vitamin B_{12} fortification also.

18. 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.

19. Studies are necessary to determine antioxidant potential of foods.

20. Whether prevention of micronutrient deficiency is synonymous with positive health or the requirement for the latter goes beyond the present recommendations needs to be considered, especially in the elderly where some micronutrients are believed to delay the process of ageing.

APPENDICES

Appendix 1

Food composition and nutrient content value of balanced diet for moderately active man

Food Composition	Amount (g /day)	Nutrient	Vegetarian diet	Non-vegetarian diet	RDA
Cereals & Millets	400	Energy (kcal)	2730	2730	2730
Animal foods[1]	60	Protein (g)	80	80	60
Pulses (Legumes)[1]	80	Visible fat (g)	30	30	30
Green leafy vegetables	50	Calcium (mg)	850	850	600
Other Vegetables	150	Iron (mg)	20.0	20.0	17.0
Roots & Tubers	100	Zinc (mg)	12	12	12
Fruits	100	Magnesium (mg)	600	600	340
Milk	300	Vitamin A (µg)	120	220	600
Fats & Oils	30	β-Carotene (µg)	4800	4000	4800
Sugar & Jaggery	40	Thiamine (mg)	2.0	2.0	1.7
Nuts & oil seeds	25	Riboflavin (mg)	1.5	1.6	1.6
		Niacin (mg)	20	20	18
		Vitamin B_6 (mg)	2.0	2.0	2.0
		Vitamin C (mg)	80	80	40
		Folate (µg)	250	250	200
		Vitamin B_{12} (µg)[2]	0.40	1.7	1.0

[1] Protein content depends on the type of protein containing foods. Pulses can be replaced with animal foods (egg, meat, fish and chicken) for non-vegetarians to meet the requirements.
[2] Only 40% of the RDA of vitamin B_{12} can be met in a vegetarian diet.

Appendix 2

Key micronutrients in vegetable and animal foods

(All values are for 100g edible portion)

Micronutrients	Leafy vegetables	Other vegetables	Pulses	Egg	Chicken	Mutton	Beef	Fish	Liver (sheep, goat, lamb)	Buffalo's Milk[d]	Cow's Milk[d]
Iron (mg)	7[a]	3.4[a]	4.7[a]	2[a]	0.7[b]	2.5[b]	2.4[b]	0.7[b]	6.3	0.2	0.2
% RDA*	42	20	28	12	4	15	14	4	37	1	1
Zinc (mg)	0.2	0.5	2.3	1.3	1.2	3.3	3.3	0.6	4.7	NA	NA
% RDA*	2	4	19	11	10	27	27	5	39		
Vitamin A (µg)	1072[c]	155[c]	15.5[c]	190	11	9	20	23	6690	48	53
% RDA*	179	26	3	32	2	1	3	4	1115	8	9
Riboflavin (mg)	0.2	0.08	0.2	0.4	0.14	0.14	0.2	0.2	1.7	0.1	0.19
% RDA*	12	5	12	23	8	8	12	12	100	6	11
Dietary folate (µg)	50	5	112	78	19	6	19	5.4	180	5.6	8.5
% RDA*	25	2	56	39	9	3	9	3	90	3	4
Vitamin B_{12} (µg)	Nil	Nil	Nil	2	NR	2.0	2.0	4	90	0.14	0.14
% RDA*				180		200	200	450	9000	14	14

* % RDA for an adult man; [a] Low absorption of non heme iron can be improved by consuming more vitamin C rich foods in raw form as much as possible.
[b] Meat , liver and poultry contains high bio-available heme iron and also increases absorption (meat factor) of non-heme iron (including fish); [c] Carotenoid conversion to retinol equivalents; [d] Good source of bioavailable calcium; NA= Not available; NR=Not reported

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Appendix 3

Summary of Recommended Dietary Allowances (RDA) for Energy, Protein, Fat and Minerals for Indians - 2010

Group	Category/Age	Body Weight (kg)	Net Energy (kcal/d)	Protein (g/d)	Visible Fat (g/d)	Calcium (mg/d)	Iron (mg/d)	Zinc (mg/d)	Magnesium (mg/d)
Men	Sedentary work	60	2320	60.0	25	600	17	12	340
	Moderate work		2730		30				
	Heavy work		3490		40				
Women	Sedentary work	55	1900	55.0	20	600	21	10	310
	Moderate work		2230		25				
	Heavy work		2850		30				
	Pregnant		+350	78	30	1200	35	12	
	Lactating 0-6 m		+600	74	30	1200	21		
	6-12 m		+520	68	30				
Infants	0 - 6 months	5.4	92 kcal/kg/d	1.16 g/kg/d	--	500	46µg/kg/d	---	30
	6 – 12 months	8.4	80 kcal/kg/d	1.69 g/kg/d	19		05	---	45
Children	1-3 years	12.9	1060	16.7	27	600	09	5	50
	4-6 years	18.0	1350	20.1	25		13	7	70
	7-9 years	25.1	1690	29.5	30		16	8	100
Boys	10-12 years	34.3	2190	39.9	35	800	21	9	120
Girls	10-12 years	35.0	2010	40.4	35	800	27	9	160
Boys	13-15 years	47.6	2750	54.3	45	800	32	11	165
Girls	13-15 years	46.6	2330	51.9	40	800	27	11	210
Boys	16-17 years	55.4	3020	61.5	50	800	28	12	195
Girls	16-17 years	52.1	2440	55.5	35	800	26	12	235

Appendix 4

Summary of Recommended Dietary Allowances (RDA) for Water Soluble and Fat Soluble Vitamins for Indians - 2010

Group	Category/Age	Body Weight (kg)	Vitamin A (µg/d)		Thiamine (mg/d)	Riboflavin (mg/d)	Niacin eqivalent (mg/d)	Vitamin B_6 (mg/d)	Ascorbic Acid (mg/d)	Dietary folate (µg/d)	Vitamin B_{12} (µg/d)
			Retinol	β-carotene							
Men	Sedentary work	60	600	4800	1.2	1.4	16	2.0	40	200	1.0
	Moderate work				1.4	1.6	18				
	Heavy work				1.7	2.1	21				
Women	Sedentary work	55	600	4800	1.0	1.1	12	2.0	40	200	1.0
	Moderate work				1.1	1.3	14				
	Heavy work				1.4	1.7	16				
	Pregnant		800	6400	+0.2	+0.3	+2	2.5	60	500	1.2
	Lactating 0-6 m		950	7600	+0.3	+0.4	+4	2.5	80	300	1.5
	6-12 m				+0.2	+0.3	+3	2.5			
Infants	0 - 6 months	5.4	350	----	0.2	0.3	710 µg/kg	0.1	25	25	0.2
	6 –12 months	8.4		2800	0.3	0.4	650 µg/kg	0.4			
Children	1-3 years	12.9	400	3200	0.5	0.6	8	0.9	40	80	0.2-1.0
	4-6 years	18.0			0.7	0.8	11	0.9		100	
	7-9 years	25.1	600	4800	0.8	1.0	13	1.6		120	
Boys	10-12 years	34.3	600	4800	1.1	1.3	15	1.6	40	140	0.2-1.0
Girls	10-12 years	35.0			1.0	1.2	13	1.6			
Boys	13-15 years	47.6			1.4	1.6	16	2.0	40	150	0.2-1.0
Girls	13-15 years	46.6			1.2	1.4	14	2.0			
Boys	16-17 years	55.4			1.5	1.8	17	2.0	40	200	0.2-1.0
Girls	16-17 years	52.1			1.0	1.2	14	2.0			