III B.Sc NUTRITION, FSM &

DIETETICS

Community Nutrition

SUBJECT CODE: CNU53

UNIT-2

Nutritional problems confronting our country - PEM - Prevalence, classification - Kwashiorkor and Marasmus - etiology, symptoms, pathological changes, biochemical changes. Prevalence, etiology, symptoms, prophylaxis programmes - Anaemia, IDD and Vitamin A deficiency

- describe methods of prevention of vitamin A deficiency, iron deficiency anaemia and Iodine deficiency disorders, and
- educate the families and communities on prevention of PEM, vitamin A deficiency, iron deficiency anaemia and iodine deficiency disorders.

We will first start with the deficiency of macronutrients and then go over to learn about deficiency of micronutrients. Let us begin with protein energy malnutrition - the deficiency of macronutrients.

3.2 PROTEIN ENERGY MALNUTRITION (PEM)

Protein Energy Malnutrition (PEM) is the deficiency of macronutrients or energy and protein in the diet and forms the most important nutritional deficiencies of public health significance. It is a nutritional disorder, which affects all the segments of population like children, women and adult males particularly from the backward and downtrodden communities.

There are many different forms of PEM. Let us learn about these.

3.2.1 Different Forms of PEM

The term PEM is used to describe a wide range of clinical conditions ranging from the very clinically detectable florid forms to the mildest forms in which growth retardation is the major manifestation. It is widely prevalent in the developing countries of Asia and Africa. According to estimates, there are about 200 million children suffering from various forms of PEM in the world. India contributes almost 40% to the total malnourished population in the world.

PEM occurs in three clinically distinguishable forms, viz. kwashiorkor, marasmus and marasmic-kwashiorkor. In addition, a large number of children suffer from various sub-clinical forms of PEM like underweight, stunting (short stature) and wasting (thinness). In fact, the proportion of clinical cases of PEM in a given community reflects only the proverbial "tip of iceberg". In other words, for every clinical case there are many more children suffering from sub-clinical PEM. Box 1 lists different types of PEM.

Box 1	Different Types of PEM
Clinical forms	**Sub-Clinical forms**
• Kwashiorkor • Marasmus • Marasmic kwashiorkor	• Underweight • Wasting • Stunting

We will first study about the clinical forms of PEM and then go over to sub-clinical PEM. Let us start with the first clinical form of PEM i.e. Kwashiorkor

3.2.1.1 Kwashiorkor

Kwashiorkor is an African word, meaning a "disease of the displaced child", who is deprived of adequate nutrition. It is one of the most important florid forms of PEM occurring mostly in children between the ages of 1 and 3 years, when they are completely weaned (taken off the breast). The three essential manifestations or signs of kwashiorkor are:

- Oedema (swelling of feet),
- Growth failure, and
- Mental changes.

In addition, there may be changes in hair and skin associated with infection and micronutrient deficiencies. Refer to Figure 3.1 which illustrates the clinical forms of PEM. Frequent infections, particularly diarrhoea and respiratory infections, aggravate the condition. Most of the children with severe PEM would have recovered from a recent attack of measles.

Let us review the above clinical signs of kwashiorkor in detail:

- *Oedema:* Oedema refers to accumulation of fluid in the tissues and usually begins with a slight swelling in feet gradually spreading up the legs. Later, hands and face may also have oedema. If oedema is present, a depression is formed when you apply pressure with your thumb on the lower part of shin or the dorsal part of foot for about half a minute.

- *Poor growth:* Growth retardation is the earliest manifestation of kwashiorkor. The child will be lighter and shorter than its normal peers of same age. The children with kwashiorkor weigh about 80% or less of their normal peers. This is usually verified by comparing the body weight of the child with that of normal children of same age group. Sometimes, in cases of gross swelling, the body weight may be relatively higher. The child will also be wasted (thinner), which sometimes could be masked in the presence of extensive swelling of the body. The child's arms and legs will appear thin as a result of wasting.

- *Mental changes:* You would find a kwashiorkor child to be unusually apathetic with absolutely no interest in the surroundings. The child will also be irritable and prefers to stay at one place and in one position.

The signs discussed above are essentially present in a child suffering from kwashiorkor. Other signs which may be present are:

- *Hair changes:* In kwashiorkor, the hair loses its healthy sheen and becomes silkier and thinner. It takes coppery red colour (referred to as 'discoloured hair'). You could easily pluck small tufts of hair without causing any pain (referred to as 'easy pluckability') just by passing your hands through the hair.

- *Skin changes:* In many cases, dermatosis (changes in skin) is seen. Such changes are common in areas of friction. Dark pigmented patches, akin to sun-baked and blistered paint, are, at times, present (known as 'flaky-paint dermatosis'). These desquamated patches may peel off leaving bleeding patches resembling sunburns.

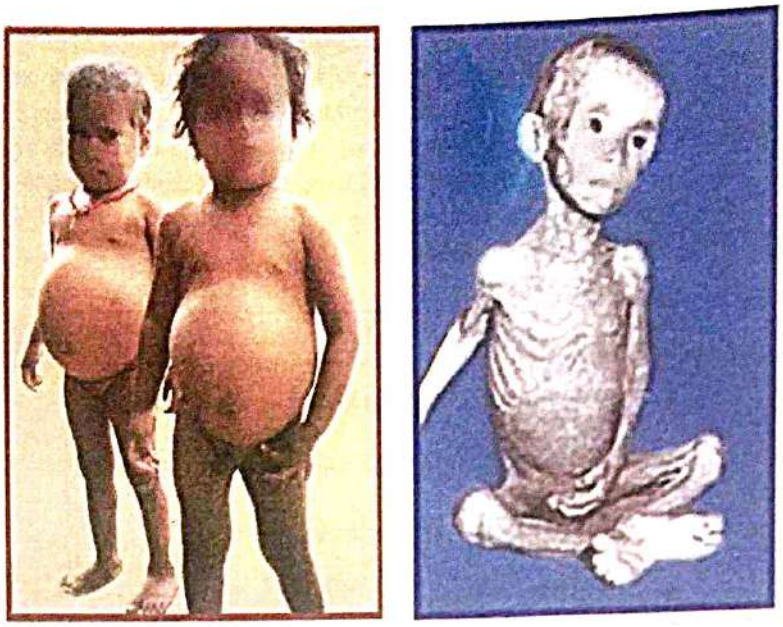
- *Moon face:* The cheeks may seem swollen with fluid or fatty tissue and often be slightly sagging. You should not mistake with the chubby cheeks of a normal and healthy child.

- *Micronutrient deficiencies:* Almost all the children manifest anaemia (due to iron deficiency) of some degree. Eye signs of vitamin A deficiency are also common in more than a quarter of children with kwashiorkor. Manifestations of vitamin B complex defiency are also noted in many cases.

- *Water and electrolyte imbalance:* The total body water and especially the extracellular fluid volume are increased in all forms of PEM. At the same time, there may be clinical signs of dehyadration, particularly sunken eyes, loss of skin turgor, dry mucosa. As for the electrolytes, its total sodium is increased although in some cases the serum sodium and osmolarity are seen to be reduced. This is obvious in patients who have oedema and signs of dehyadration. As for potassium it is usually deficient and magnesium deficiency is reported.

- *Infections:* There may be lower respiratory tract infections associated with diarrhoea/dysentry.

Figure 3.1(a) illustrates a kwashiorkor child. Look up Box 2, as well which summarizes the various signs of kwashiorkor.



(a) A typical case of kwashiorkor (b) A marasmic child

Figure 3.1: Clinical forms of PEM

Box 2	Signs of Kwashiorkor
	• Oedema • Underweight (<80% of normal weight for age) • Apathy and irritability • Moon face • Hair and skin changes • Micronutrient deficiencies

Let us now move on to the next clinical form of PEM i.e. marasmus

3.2.1.2 Marasmus

Marasmus, the other end of the same spectrum as kwashiorkor, is common in children below the age of 2 years. The characteristic manifestations, as illustrated in Figure 3.1(b) are:

- Severe growth retardation
- Extreme emaciation
- Old man's or monkey's face, and
- Loose and hanging skin folds over arms and buttocks.

As you may have noticed in Figure 3.1(b), a typical case of marasmus can be described as a bonny cage having nothing but "skin and bones". You would notice that

the marasmic children are so weak that they may not have even energy to cry, wh
most often is barely audible. The child is extremely wasted with very little subcutane
fat with the skin hanging loosely particularly over the buttocks. In fact, when you h
the marasmic child in a standing position, you can see the loose skin folds hangi
prominently, unlike in any normal child. For the given age, the children will be genera
below 60% normal or < Median - 3SD of the standards. We will learn about these
standards later in this units. Unlike in kwashiorkor, oedema is absent and there are no
skin and hair changes. However, frequent diarrhoeal episodes leading to dehydration
and micronutrient deficiencies of vitamin A, iron and B-complex are common. Box 3
lists the signs and symptoms of marasmus.

Box 3	Signs and Symptoms of Marasmus
•	Extreme muscle wasting - "skin and bones"
•	Loose and hanging skin folds
•	Old man's or monkey faces
•	Absolute weakness

Let us now move on to the third clinical form of PEM- Marasmic kwashiorkor.

3.2.1.3 Marasmic Kwashiorkor

Sometimes, in areas where PEM is common, malnourished children exhibit the features of both kwashiorkor and marasmus. Such changes could occur during the transition from one form of severe PEM to another. For example, a marasmic child can develop oedema after a severe bout of infection or a kwashiorkor child, when loses oedema may develop this condition. Such a child is considered as suffering from 'marasmic kwashiorkor'. These children will have extreme wasting of different degrees (representing marasmus) and also oedema (a sign of kwashiorkor). They may also manifest some hair changes and often diarrhoea. Box 4 lists the signs and symptoms of marasmic kwashiorkor. So it must be evident that there is a continuous spectrum of signs from oedematous kwashiorkor through varying degree of marasmus associated with oedema. For your reference we have included the main features of PEM in children in Table 3.1.

Look up Box 4 for the signs of marasmic kwashiorkor

Box 4	Signs and Symptoms of Marasmic Kwashiorkor
•	Extreme muscle wasting - "skin and bones"
•	Loose and hanging skin folds
•	Old man's or monkey's face
•	Absolute weakness
•	Oedema

Besides the specific and essential features discussed in Table 3.1 a number of biochemical changes have been discussed in the blood, urine, gastrointestinal secretions, endocrine functions and tissue composition in PEM. The changes that are most important in diagnosis and treatment are summarized in Table 3.2

Table 3.1: Features of PEM in children

Features	Marasmus	Kwashiorkor
Essential Features		
1. Oedema	None*	Lower legs, sometimes face, or generalized*
2. Wasting	Gross loss of sub cutaneous fat, "all skin and bones"*	Less obvious; sometimes fat, blubbery
3. Muscle wasting	Severe*	Sometimes
4. Growth retardation in terms of body weight	Severe*	Less than in of Marasmus
5. Mental changes	Usually none	Usually present
Variable features		
1. Appetite	Usually good	Usually poor
2. Diarrhoea	Often (past or present)	Often (past or present)
3. Skin changes	Usually none	Often, diffuse pigmentation; occasional "flaky-paint"* or "enamel" dermatosis
4. Hair changes	Texture maybe modified but no dyspigmentation	Often sparse-straight and silky; dyspigmentation grayish or reddish
5. Moon face	None	Often
6. Hepatic enlargement	None	Frequent, although it is not observed in some areas.

**The most characteristics or useful distinguishing features.*

Table 3.2: Biochemical signs specific to PEM

Biochemical Changes	Marasmus	Kwashiorkor
Serum albumin	Normal or slightly decreased	low*
Urinary urea per g of the creatinine	Normal or decreased	Low*
Urinary Hydroxyproline Index	Low	Low*
Serum free amino acid ratio	Normal	Elevated*
Anaemia	May be observed	Common iron and folate deficiency may be associated
Pancreatic secretions	Reduced enzymatic activity	Reduced enzymatic activity

** The most characteristic or useful distingushing features*

As you may have noticed in Table 3.2, serum albumin and also serum total protein are markedly decreased in kwashiorkor. It is important to note that serum albumin level is one of the most useful biochemical indicators of PEM. We will learn more about this biochemical indicator later in Unit 9. As for the changes in urine, the hydroxyproline excretions in urine, is proposed as an indicator of the rate of growth in children with PEM. The index is essentially constant between the age of 6 months and about 5 years. It is low in malnourished children. Further the urinary excretion of creatinine decreases in relation to the reduction in the muscle mass. Hence, in both kwashiorkor and marasmus the creatinine excretion is low.

We have learnt about clinical forms of PEM. Now let us learn about sub-clinical PEM.

3.2.1.4 Sub-clinical PEM

You have already learnt that clinical forms of PEM represent only a small proportion of the total cases of PEM in a community in rural India. Growth retardation is not only an important and objective manifestation of PEM, but is also the first response to rehabilitation in such cases. Anthropometry (body measurement) is extensively used to detect various degrees of sub-clinical forms of PEM. Body weight is, by far, the most sensitive and frequently used parameter of nutritional status particularly in preschool children (1-5 years). Several methods have been suggested for classification of PEM in children based either on body weight alone or in combination with standing height/ recumbent length.

The following classifications based on body weight are commonly used in India.

1. Gomez classification,
2. Indian Academy of Pediatrics (IAP) classification, and
3. Standard deviation classification.

Let us elaborate on each of these now.

1) *Gomez classification*

It will be of interest to you to learn that in Mexico, a child specialist named *Gomez* and his co-workers proposed a classification expressing the body weights of children as percentage of normal values (standards) for age. They proposed that young children could be divided into 4 grades of malnutrition. They observed, based on their hospital observations, a marked difference in mortality of children suffering from second and third grades of malnutrition. The advantage is that it would be possible to prioritize actions based on the classification given in Table 3.3.

Table 3.3: Gomez classification for weight for age

% Weight for age of NCHS	Type of undernutrition	Grade of undernutrition
≥ 90	Normal	Normal
75 - 89.9	Mild	I
60 - 74.9	Moderate	II
< 60	Severe	III

All those with severe degree of malnutrition need the attention of a trained nutrition worker. Children in second and third grades of malnutrition require nutrition supplementation.

2) *Indian Academy of Pediatrics (IAP) Classification*

In India, the classification of children, which is extensively used to group children into various grades of malnutrition is the one proposed by the Indian Academy of Pediatrics. Growth charts based on this classification are used in the largest national nutrition

intervention programme, Integrated Child Development Services (ICDS), for growth monitoring of children. The nutrition subcommittee of IAP considered that children with body weights more than 80% of NCHS standards should be as normal and suggested the classification given in Table 3.4.

Table 3.4: IAP classification for weight for age

Weight for age (NCHS standard)	Grade of undernutrition
> 80%	Normal
70 - 79.9	I
60 - 69.9	II
50 - 59.9	III
< 50	IV

According to this classification, all the children in grades II, III and IV are included as beneficiaries in ICDS supplementary feeding programme.

3) *Standard Deviation (Z score) classification*

Statistics teaches us that when normal values of any variable are distributed as per their frequency of occurrence, they follow normal distribution encompassing values within two standard deviations (SD) of mean/median (Average). On this premise, all the children with weights less than median -2SD of normal weight for age (NCHS standards) are considered as suffering from undernutrition. The following criteria given in Table 3.5 are used to classify children into various degree of undernutrition based on mean/median and SD.

Table 3.5: SD classification for weight for age

Weight for age criterion	Grade of undernutrition
>Median - 2SD	Normal
<Median - 2SD to Median - 3SD	Moderate
< Median - 3SD	Severe

You have learnt about different forms of PEM. You must be now wondering how common is the occurrence of PEM in our communities. i.e. what is the prevalence of the problem. We will now discuss that in the next section.

3.2.2 What is the Prevalence of PEM?

The extent of a disease is measured in terms of *prevalence rate*, which indicates *the number of individuals with a particular disease (numerator) at a particular point of time in a specified number (usually per 100 population of a community) (denominator)*. The prevalence of kwashiorkor and marasmus, which was about 4% in the early sixties, has declined substantially over the period. As per the recent surveys conducted by the National Nutrition Monitoring Bureau (NNMB), the prevalence of kwashiorkor and marasmus is about 1%. However, as judged by weight for age, about a half of children under the age of 5 years suffer from sub-clinical PEM. About 60% of these children had stunting (short stature). National Family Health Survey results reveal that about 47% of the children under the age of 3 years, who are considered as at risk of developing PEM, are underweight. Thus, the problem of PEM in India is widespread and requires immediate intervention.

Since the problem of PEM in India is widespread and requires immediate intervention, next thing which must be coming to your mind is what causes it. Let us now learn what are its causes.

3.2.3 What Causes PEM?

Some of the causes of PEM are elaborated herewith:

Low Birth Weight

The beginning of PEM in children starts in rural India from the time of their birth. At least a third of the Indian children are born with low birth weights (<2.5 kg) due to high maternal malnutrition (malnutrition in mother). You may be aware that the birth weight of a normal child is 3-3.4 kg.

Inadequate Breast Milk

Though prolonged breastfeeding of children is the rule in rural India, the amount of breast milk secreted in poor Indian mothers is lower than either normal women or those of developed countries. In other words, the infants may not be consuming adequate breast milk leading to inadequate nutrition.

Delayed Complementary Feeding

The mothers from poorer socioeconomic groups where PEM is more prevalent, delayed introduction of complementary foods (foods in addition to breast milk) usually until the infant completes one year of age is a common practice. Thus, when breast milk is not adequate, delaying complementary feeding further aggravates the dietary inadequacy among such infants leading to PEM. Rural Indian women, due to ignorance, firmly believe that children should be given complementary foods only when they are able to pick and eat. After weaning (completely stopping breast feeding), the children are not given any special diet other than the adult diet. Young infants cannot consume these diets in adequate amounts due to its bulk. Early and abrupt weaning and introduction of diluted milk formulae is one of the reasons for marasmus.

Primarily Energy Deficiency

Surveys on preschool children in different parts of the country reveal that PEM is primarily due to dietary energy deficiency arising as a result of insufficient food intake. The primary bottleneck in the dietaries of Indian children, who are given cereal-pulse based diets, is energy and not protein as, hitherto was believed.

Infections and Infestations

Childhood infections (viral/bacterial) and parasitic infestations are almost always associated with PEM. These cause anorexia (loss of appetite) leading to reduced food intake, interfere with nutrient absorption and utilization, and result in nutrient losses. The role of measles infection, frequent diarrhoea and acute respiratory infections in the onset of PEM is very important.

Ignorance and Poor Socioeconomic Status

Improper childcare, either as a result of lack of knowledge or lack of time for mother, could also contribute to the onset of PEM. PEM is mainly a disease of the poor and downtrodden. The mothers in these families are illiterate, work for their living and are largely influenced by the belief systems of the society, are superstitious and believe in spiritual healing etc. The families are generally large, and even if they spend their complete income on food with low purchasing power they would be unable to meet the requirements. Box 5 highlights causes of PEM.

Box 5	Causes of PEM
• Maternal malnutrition • Low birth weight • Faulty child feeding practices • Dietary inadequacy • Frequent infections	• Low purchasing power • Food taboos and superstition • Large families • High female illiteracy

We have seen that PEM is a nutritional disorder of public health significance. Let us now study what happens if PEM is not prevented and or treated. In other words, let us learn about the consequences of PEM.

3.2.4 What are the Consequences of PEM?

The consequences of PEM are most often long lasting and irreversible. The common consequences include:

- Irreversible growth retardation
- Increased susceptibility to infections
- Increased risk of mortality
- Low cognitive performance

As a result of extensive PEM since early childhood, in India, there is irreversible growth retardation leading to short stature among adults. While children of well to do communities, where the problems of inadequate diet and ill health are not common, are as tall and heavy as those from developed countries, poorer children suffer from stunting, wasting and underweight. Studies in different parts of India reveal that in children suffering from various grades of PEM, their immunity (ability to fight infections) is reduced and as a result, the incidence of childhood infections like diarrhoea and respiratory infections is high. The children with severe forms of PEM are usually brought to the hospital with complications arising as a result of severe infections. The immunity in these children is low leading to lowered resistance to infections. Therefore, respiratory and gastrointestinal infections are not only common but their severity is also higher. Severe diarrhoea might lead to dehydration. Septicemia and bronchopneumonia in children with kwashiorkor and marasmus could be fatal. It should also be recognized that such infections could increase the risk of PEM leading to a vicious cycle of malnutrition and infection. The risk of mortality in moderate and severe PEM is higher, particularly when they are exposed to frequent infections. The work output of adults who are lighter also has been shown to be lower affecting the productivity of the nation.

We have learnt about the causes and consequences of PEM. Next, let us learn how we can treat PEM.

3.2.5 How do we Treat PEM?

Major objective of the treatment of PEM is to provide adequate energy and protein intake and control infections, if any. Mild and moderate forms of PEM can be and should be managed at home under the supervision of health professional. Severe forms of PEM should be referred to a hospital particularly when associated with severe bronchopneumonia and diarrhoea. All the cases without any complications can be managed as outpatients in either a primary health center or a hospital. Here we will study the treatment of severe PEM. The key components of treatment are:

- *Diet*

Treatment of cases of kwashiorkor or marasmus involves mainly providing appropriate nutrition support. The child should receive a diet that provides adequate amounts of energy and protein. Both of these are required in larger quantities than normal requirements for rapid recovery. The child should be given the following concentrations:

Energy : 170 - 200 kcal per kg of body weight

Protein : 3 - 4 g/kg of body weight

Initially, milk based liquid diet, using either fresh milk or dry skimmed milk powder, is recommended. A milk formula could be prepared in one litre of clean water by adding dried skimmed milk powder: 90 g, sugar: 70 g and vegetable oil: 50 g. About 100 ml of reconstituted preparation would provide 100 kcal and 3 g of protein. The formula should be given to the children at the rate of 100-150 ml per kg of body weight. As dried skimmed milk powder does not contain any vitamin A, it should be enriched with vitamin A. Sugar or vegetable oil can be added to increase the energy content.

In the beginning, the child may refuse to accept any feeds due to loss of appetite. Under such situations, the diet could be given with a spoon. In extreme cases, gastric intubation (feeding through tube) may be resorted to. With improvement in appetite, the child would start eating the diet readily, at which time gradually solid foods can be introduced. In older children special diet based on cereal, pulse, dried skim milk powder and sugar/jaggery can be given. Addition of vegetable oil would increase the energy density of the recipe.

- *Vitamin and mineral supplements*

All cases of severe PEM require multivitamin preparation to meet the increased demands during recovery. Iron (60 mg) and folic acid (100 mg) may be given daily to correct anaemia.

- *Oral rehydration*

Since diarrhoea is very common in severe PEM, correction of dehydration is the first step in the treatment. Home made (salt-sugar mixture) or commercial oral rehydration solution (ORS) can be administered to correct dehydration. The WHO recommends that the ORS formula should contain sodium chloride: 3.5 g, sodium bicarbonate: 2.5 g, potassium chloride: 1.5 g and glucose: 20 g. It should be dissolved in a litre of clean water and given to the child in small quantities at frequent intervals at the rate of 70-100 ml/kg body weight. Intravenous fluids are required only in severe dehydration.

- *Control of infections and infestations*

Appropriate antibiotics should be started immediately since infections are the immediate cause of death in many children. Children with intestinal infestations like giardiasis and ascariasis should be treated.

- *Clinical progress*

Normally, clinical improvement is evident within a week with the disappearance of oedema. The appetite improves almost dramatically and the child starts gaining weight, after initial loss of body weight. The mother should be advised about the diet precisely. It would be better to involve her in the preparation of suitable recipes during the child's stay at the hospital.

We have learnt so far about the treatment of a child suffering from PEM. Have you heard the old proverb - "prevention is better than cure". So it becomes extremely important that we make sincere efforts to prevent and control PEM. Let us now see how this could be done.

3.2.6 How to Prevent and Control PEM ?

Any programme aimed at prevention of PEM should be holistic and comprehensive considering the family as a unit. Since the effects of undernutrition are cumulative, currently it is being emphasized that 'life cycle approach' as illustrated in Figure 3.2 should be adopted.

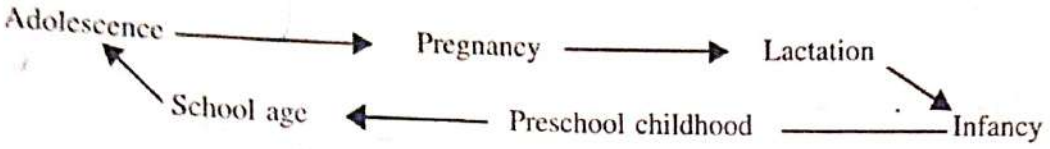


Figure 3.2: Life cycle approach

It is generally recommended that the entry point into the life cycle is the *adolescent girl*, who will be the future mother and should be given adequate attention. Their nutrition should be ensured and the family should be educated against the practices like adolescent marriages and pregnancies so that they would be prepared to be healthy women of tomorrow to be able to handle pregnancy, lactation and child care effectively. At all levels, the advice should include both health care and nutrition. Given below are some of the measures which should help in prevention of PEM.

- *Ensure proper diet*

PEM is preventable. It is a disease of the poor and the ignorant that suffer from social inequalities. Therefore, a holistic approach is necessary to prevent and control PEM. The most critical aspect is to ensure that the child is fed adequate quantities of diet containing all the nutrients daily. Therefore, the communities should be made aware that it is in their hands to ensure that their children and other members of the family consume adequate diets daily.

Increase purchasing power

The Government of India formulated the National Nutrition Policy and prepared National Plan of Action to bring down the prevalence of moderate and severe malnutrition. We will study about the National Nutrition Policy later in Unit 10. Here we should know that along with direct nutrition intervention, socio economic development, aimed at poverty alleviation to increase the purchasing power of the rural and urban poor, should become an important component of control programme.

The essential components of any control programme are: supplementary feeding, immunization, control of minor infections, promoting food security, nutrition communication, poverty alleviation, and empowerment of women. These components are described in details later in Unit 12 and 13 in this course. However a brief review is presented herewith.

- *Supplementary feeding*

Supplementary feeding has remained an important component to control undernutrition. Considering the dietary inadequacy in the diets of poor rural families, various programmes provide daily supplementary food providing about 300 kcal of energy and 8-10 g of protein per child under various feeding programmes. Of the direct intervention programmes of the government, Integrated Child Development Services (ICDS) is the largest being implemented by the Department of Women and Child Welfare of the Government of India in over 4000 projects all over the country with emphasis on backward and tribal areas. Supplementary feeding is an important component of ICDS. Other components are immunization, growth monitoring, and treatment of minor illness, non-formal preschool education, and nutrition education to the mothers. The Department of Health ensures immunization of children, distribution of six monthly massive doses of vitamin A and iron and folic acid tablets and treatment of minor ailments.

- *Promoting food security*

Public distribution of food grains through a network of ration shops, particularly to reach the population below the poverty line, so as to improve availability, access to food grains at affordable prices is an important step to improve food and nutrition security of the poor.

- *Poverty alleviation*

There are a number of development programmes aiming at employment assurance to the landless and other labour, with a focus on increasing the purchasing power. We will study about these programmes later in Unit 10, section 10.8.

- *Behavioural change communication*

One of the reasons for the widespread prevalence of PEM in our country is ignorance due to illiteracy, particularly among the females. Therefore, there is a need to change the behaviour in these women through effective communication programmes. Person-to-person communication, cooking demonstrations, and mass media like television and radio are some of the tools that are available. We should convince the community, particularly the mothers about the need for proper diet to children for normal growth and to prevent them developing PEM. She should be made aware that the main reason for PEM is shortage of food either as a result of poverty or due to inequitable intrafamily distribution of foods. You should be equipped to give advice on complementary feeding and be able to inform the mothers as to the types of foods that could be given to young children. Box 6 highlights essential components of prevention of PEM.

Box 6	Essential Components to Prevent PEM
➢	Supplementary feeding,
➢	Immunization,
➢	Control of minor infections
➢	Promotion of food and nutrition security
➢	Behaviour change communication
➢	Empowerment of women and
➢	Poverty alleviation

Eradication of PEM requires concerted efforts not only on the part of the government but also continuous and active community participation. Integration, convergence, commitment and community participation are the crucial pillars of any prevention strategy.

In this section, we learnt about signs, causes, prevalence, treatment and prevention of PEM. In the next section, we will discuss the micronutrient deficiencies. But before that, let us recapitulate what we have learnt so far. Answer the questions given in the check your progress exercise 1.

Check Your Progress Exercise 1

1. What are the different clinical forms of PEM?

.....

.....

Box 7	Common Micronutrient Deficiencies
	• Vitamin A deficiency • Iron deficiency anaemia • Iodine deficiency disorders • Zinc deficiency

We will now discuss these micronutrient deficiencies in detail. Let us begin with Vitamin A deficiency.

3.3.1 Vitamin A Deficiency

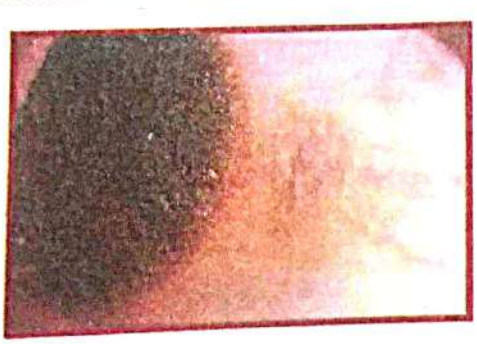
Vitamin A deficiency (VAD) is a major public health problem, and the most vulnerable are preschool children and pregnant women in low income countries. In children, VAD is the leading cause of preventable severe visual impairment and blindness. An estimated 250 000 to 500 000 VAD children world over become blind every year, and about half of them die within a year.

Vitamin A, we know, is essential for maintenance of healthy epithelium and normal vision. Deficiency of vitamin 'A' manifests in the form of eye lesions, which are grouped under 'xerophthalmia', can be either mild leading to night blindness and changes in conjunctiva (white of the eye) or in severe form causing damage to cornea (black of the eye) leading to irreversible blindness. We will learn about these ocular manifestations, prevalence, causes, treatment and prevention of vitamin A deficiency in this section.

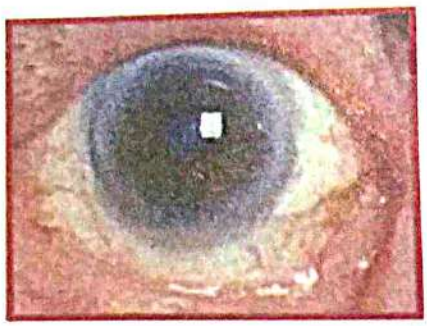
Let us begin with the signs and symptoms of vitamin A deficiency.

Clinical Features of Vitamin A Deficiency

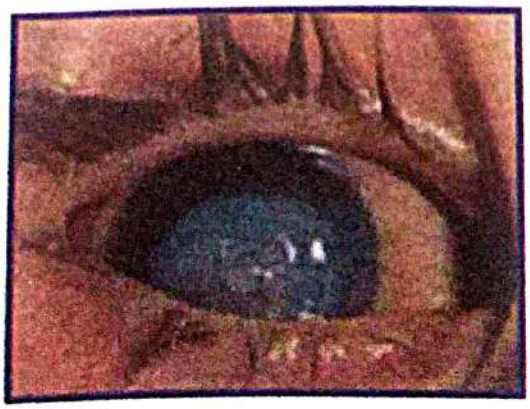
The clinical features or the occular manifestations specific to vitamin A deficiency are described here with and illustrated in Figure 3.3.



(a) Bitot Spot



(b) Bitot Spot with the xerosis of conjunctiva



(c) Keratomalacia

Figure 3.3: Clinical manifestations of xerophthalmia

Night Blindness

Night blindness is the earliest symptom of Vitamin 'A' deficiency. You may recall studying in the Nutritional Biochemistry Course (MFN-002) and the Advance Nutrition Course (MFN-004) that reduction in the supply of vitamin A aldehyde i.e retinal to the rod cells of the retina results in the impairment of dark adaptation. Under such situations, the affected child cannot see properly in sunlight particularly after the sunset. Often, an attentive mother can recognize the child's inability to see the plate of food or toys in ill-lit room. Pregnant women often experience deficiency symptoms, such as night blindness, that continues into the early period of lactation. In most part of the country, there is a local term for the condition, example in Hindi, it is called as "Rathaundi".

Bitot's Spots

As the deficiency progresses, dirty white, foamy and raised spots are formed on the surface of the conjunctiva, generally on the outer side of the cornea as you may observe in Figure 3.3(a). These spots are accumulation of denuded conjunctival epithelial cells. They stain black in the eyes when applied 'Kajal'. You would notice that the spot is quite superficial and more or less readly removed by direct inking or by lacrimination in a crying.

Conjunctival Xerosis

Conjunctiva in normal children is bright white, smooth and glistening. Conjunctival xerosis is characterized by dryness of the conjunctiva, after exposure to air for 10-15 seconds by keeping eyelids drawn back, which also becomes thick, rough and wrinkled. In case of an affected child, The changes associated with conjunctiva include: dryness (the literal meaning of "Xerosis"), unwettability, loss of transparency, wrinkling and pigmentation. Refer to Figure 3.3(b) which illustrates the conjunctival xerosis along with bitots spot.

Corneal Xerosis

This is a sign of severe vitamin 'A' deficiency, in which the cornea loses its normal smooth and glistening appearance and becomes dry and rough. The child tends to keep the eyes closed, particularly in bright light due to photophobia (inability to see in bright sun) and hence, the condition may be missed during the clinical examination, if not observant.

Corneal Ulcer

Corneal xerosis, if not treated promptly, leads to ulceration of the cornea. Initially, the ulcer may be shallow, and if it becomes deep, it may lead to perforation resulting in prolapse of contents of the eyeball. These lessions are more common in the lower central cornea.

Keratomalacia

This is a condition of rapid destruction and liquefaction of full thickness of cornea, leading to prolapse of iris, resulting in permanent blindness. Usually keratomalacia consists of characteristic softening of the entire thickness of a part, or more often the whole of the cornea leading to deformation or destruction of the eyeball. It is painless but the corneal structure just melts into a cloudy gelatinous mass, dead-white or dirty yellow in colour. Extrusion of the lens and loss of the vitreous may occur. In infective conditions, the eye will be red and swollen. Figure 3.3(c) illustrates keratomalacia.

Corneal Scar

The corneal ulcer, on healing, leaves a white scar, which may vary in size depending upon the size of the ulcer. When the scar is big or positioned centrally blocking the pupillary region, normal vision is affected.

In addition, to the above mentioned manifestations, thickening of the hair follicles (follicular hyperkeratosis) is a cutaneous manifestation of vitamin A deficiency.

From the description above it must be clear that xerophthalmia represents the ocular consequences of vitamin A deficiency that includes various manifestations, about which we have learnt above and the same are classified by WHO as given in Table 3.6.

Table 3.6: WHO classification for assessment of vitamin A status

Classification	Primary signs
XI A	Conjunctival Xerosis
XI B	Bitot's Spots
X 2	Corneal Xerosis
X 3A	Corneal Ulceration
X 3B	Keratomalacia
	Secondary signs
X N	Night blindness
X F	Fundal changes
X S	Corneal scarring

The classification presented above may be summarized as:

- Stage XN is the earliest stage, involving night blindness owing to impaired dark adaptation,
- Stage XI A is corneal xerosis caused by reduction of goblet cell mucus,
- The appearance of Bitot's spot, a foamy excrescence on the temporal surface of the conjunctiva, constitutes the advancing stages (XIB),
- X2 consists of simple drying of the cornea,
- When the cornea undergoes the liquification process of keratomalacia, corneal ulceration, or both, it is classified as X3,
- The situation is classified as X3A if <1/3 of the corneal surface is involved,
- Past involvement leaves a corneal scar (XS), and
- A globe destroyed by advanced keratomalacia is xerophthalmic fundus (XF).

The discussion above focused on the signs and symptoms of vitamin A deficiency. Let us now learn how common the problem of vitamin A deficiency is i.e. the prevalence of vitamin A in our country.

Prevalence of Vitamin A deficiency

The World Health Organization (WHO) has recommended a set of prevalence criteria (both clinical and biochemical) for defining the vitamin A deficiency (VAD) problem of public health significance among children under 6 years of age in the community. This criteria is given in Table 3.7. The prevalence of any one or more indicators signifies public health problem.

Table 3.7 : Prevalence criteria for assessing vitamin A deficiency problem of public health significance among children under 6 years of age

Indicator/Criteria	Minimum prevalence (%)
Clinical	
Night Blindness (XN)*	>1.0
Bitot's spot (X1B)	>0.5
Conjunctival xerosis with Bitot's spot (XI)	>0.5
Corneal xerosis/ulceration/keratomalacia (X2, X3A, X3B)	>0.01
Xerophthalmia related Corneal scars (XS)	>0.05
Biochemical	
Serum retinol (vitamin A) < 0.35µmol/l (<10µg/dl)**	>5.0

* Proposed prevalence of night blindness in pregnant women >5% (IVACG, 2001)

** Proposed to be > 15% with serum retinol < 7.0µmol/l (IVACG, 2001)

Surveys by National Nutrition Monitoring Bureau (NNMB) and the Indian Council of Medical Research (ICMR) reveal that about 0.7% of preschool children have Bitot spots. As you would note from the Table 3.4, that as per WHO, prevalence of Bitot spots more than 0.5% in children under the age of 6 years is an indication that vitamin A deficiency is a public health problem requiring intervention. During the last two decades, the extent of Bitot spots in children showed a decline from about 2% to about 0.7%. The contribution of vitamin A deficiency to total blindness has come down significantly during the last 4 decades.

Globally, data suggest that among the children under 5 years of age affected by VAD, some 3 million have ocular lesions of xerophthalmia and 100 to 140 million present only subclinical manifestations, yet live with a greater risk of mortality and of developing severe infections.

We have learnt about the signs and symptoms and the criteria for assessing the public health significance of xerophthalmia and vitamin A deficiency. Next, you must be wondering what is its etiology? Let's find out.

Causes of vitamin A deficiency

Some of the causes of vitamin A deficiency are given below:

- *Inadequate diet*

An Indian child is born with poor stores of vitamins and minerals due to maternal malnutrition. Diets of pregnant women are deficient in several nutrients, including vitamin A. The concentration of vitamin A in breast milk is low among undernourished mothers and the most poor mothers delay complementary feeding beyond the age of one year and foods containing vitamin A are seldom given. The daily intake of vitamin A is about 100 mg while the recommended intake is 400 mg of retinol. The exclusively vegetable based diets, therefore, contain β-carotene and little or none of preformed vitamin A, except from breast milk.

- *Poverty and ignorance*

Low purchasing power of the communities and their consequent inability to meet the nutrient requirements and traditional wrong beliefs and ignorance are also important causes. Low cost β carotene and iron rich foods like dark green leafy vegetables and fruits like papaya are not given to children and pregnant women due to the belief that consumption of green leafy vegetables leads to diarrhoea in children and papaya when consumed by pregnant women can cause abortions.

- *Infections*

During acute infections, vitamin A intake in preschool children is reduced due to impaired appetite and impaired vitamin A absorption as in acute diarrhoea and respiratory infection, and, consequently, serum levels of vitamin A are significantly reduced during acute infections. An infective episode of diarrhoea and respiratory infection and an attack of measles can aggravate vitamin A deficiency. Vitamin A deficiency is often associated with ascariasis and giardiasis.

You learnt about the causes of vitamin A deficiency. Let us now look at how we can treat vitamin A deficiency.

Treatment of vitamin A deficiency

All forms of vitamin A deficiency are treated with a massive oral dose of vitamin A in oil (200,000 IU), immediately after diagnosis. The health workers may refer all cases of corneal xerophthalmia, after first administering vitamin A, to medical doctor. Secondary infections should be controlled with suitable antibiotics. If necessary, a second dose may be given 48 hours after the first dose. Since more than 90% of the cases of keratomalacia are associated with severe clinical protein energy malnutrition (kwashiorkor or marasmus), the patients should also be treated for the same. A schedule recommended by WHO for treatment of individuals with corneal xerophthalmia is given in Table 3.8.

Table 3.8: Treatment of xeropthalmia in all ages

Timing of dose	Children (0-5 months)	Children (6-12 months)	Children over 12 months, male adolescent and male adults
Immediately on diagnosis	50 000 IU	100 000 IU	200 000 IU
The following day	50,000 IU	100 000 IU	200 000 IU
Subsequent contact (at least 2 weeks later)	50 000 IU	100 000 IU	200 000 IU

In women of reproductive age group with night blindness or bitots spots, a daily dose of 10 000 IU or a weekly dose of 25 000 IU of vitamin A for at least 4 weeks is the recommended treatment schedule. In population with a high prevalence of HIV infection (>10%), neonates should be given an extra dose of 50 000 IU at birth.

We have learnt that vitamin A deficiency is a condition of public health significance. Let us now understand what we can do to prevent vitamin A deficiency.

Prevention of vitamin A deficiency?

Since dietary inadequacy is the major cause for micronutrient deficiencies, the most rational approach to prevent these deficiencies would be to ensure adequate amounts of the nutrients in the daily diets of the population at risk. There are two basic

approaches to achieve this: i) long term programmes for promotion of adequate intakes of foods rich in micronutrients, and ii) supplementation of specific nutrients either as medicinal doses or through food fortification to meet immediate needs In view of the serious nature of the problems, many countries have been adopting short-term measures, which though are interim in nature. Some of these measures by which we could prevent vitamin A deficiency are highlighted next.

Supplementation

Administration of large doses of vitamin A to children at risk has been the most popular approach to control nutritional blindness. Extensive field trials carried out by NIN, Hyderabad have demonstrated the feasibility and effectiveness of this approach. The Government of India has launched vitamin A supplementation programme on a national scale, as early as in 1970. The programme is now in operation in all the States in the country, targeted to about 30 million preschool children. Under this programme, sponsored by the Department of Health and Family Welfare, Government of India, one teaspoonful of oil-miscible vitamin A syrup containing 200,000 IU of vitamin A is given once every 6 months to children between the ages of 9-36 months. The programme is implemented through the sub-centre – primary health centre complex of the States. Paramedical personnel (ANM/MPHW), under the supervision of the PHC Medical Officer, carry out the actual distribution of vitamin A.

Food Based Approach

Although nutrient supplementation is a simple and effective intervention, it is only a short-term measure. It must be combined with dietary intervention or food based approaches for long lasting effects. What are these food based approaches? A detail discussion on this approach is presented later in Unit 12 in this course. Here in the context of vitamin A deficiency, fortification as a food ba[illegible] strategy has been used. Fortification of sugar has been in operation in Central American countries with reasonable success. In India, sugar may not be the suitable vehicle for the most needy segments of population who are very poor and cannot afford the same. Home gardening, another food based approach, has been found to be a feasible long-term strategy, to increase production and consumption of leafy and other vegetables and fruits by the community to control vitamin A deficiency. The Departments of Agriculture and Social Forestry are making efforts in this direction. The Indian Council of Agricultural Research (ICAR) has established 101 Krishi Vigyan Kendras or Farm Science Centres so far in various parts of the country to impart training in agriculture technologies to farmers. In the past, the major thrust was on cereal and millet production. It is only in the recent years that horticulture production is receiving emphasis. Women Extension Workers are trained not only in agriculture technologies, but also in home gardening and preparation of recipes based on locally available nutritious foods.

Nutrition Education

Ignorance, you may recall studying earlier, is an important determinant of vitamin A deficiency. There is, therefore, a need to increase the awareness of the community about the significance of proper diet in the prevention of vitamin A deficiency. Although education is a component of all health and nutrition programmes, this has been one of [illegible]e weakest links. The health functionaries are either not properly oriented or do not have the necessary audio-visual tools to impart nutrition education. Multi-media approach involving communication experts will have to be adopted for success of nutrition education efforts. Food and Nutrition Board, through its network of 67 centres has been imparting education and training in nutrition, as well as, on home-scale preservation [illegible] fruits and vegetables. However, efforts made, so far, have not been adequate. [illegible]ation programmes adopting social marketing (by applying marketing principles to [illegible]on campaigns) approach have been shown to be effective in changing the [illegible]ur of community. Box 8 highlights in brief different strategies to prevent [illegible] A deficiency.

Box 8	Prevention of Vitamin A Deficiency
•	Supplementation with large doses of vitamin A
•	Treatment of infections and infestations
•	Home gardening
•	Behavioural change communication

In the above section, we learnt about signs, prevalence, causes, consequences, treatment and prevention of vitamin A deficiency. We will now move on to iron deficiency anaemia. But first let us review what we have learnt so far.

Check Your Progress Exercise 2

1. Which are the three major micronutrient deficiencies affecting large segments of population.

   .....

   .....

2. List the importance of Vitamin A in our body.

   .....

   .....

3. List the manifestations of mild and severe forms of vitamin A deficiency.

   .....

   .....

4. List three causes of Vitamin A deficiency.

   .....

   .....

5. List four different strategies to prevent Vitamin A deficiency.

   .....

   .....

We will now learn about signs, prevalence, causes, consequences, treatment and prevention of iron deficiency anaemia.

3.3.2 Iron Deficiency Anaemia (IDA)

Iron deficiency anaemia (IDA) is the most common micronutrient deficiency in the world, particularly in the developing countries like India. Estimates suggest that as many as 4-5 billion people i.e. 66-80% of the world's population, may be iron deficient; 2 billion people i.e. over 30% of the world's population are anaemic.

Anaemia occurs when haemoglobin (a pigment that gives red colour to the red blood cells) production is considerable reduced, leading to a fall in its level in the blood. Mostly anaemia is due to iron deficiency. The other causes of anaemia may include folate and vitamin B_{12} deficiency or anaemia of chronic diseases. Iron deficiency and anaemia reduce the work capacity of individuals and entire populations, bringing serious economic consequences and obstacles to national development. For children, health consequences include premature birth, low birth weight, infections and elevated risk o

eath. Later physical and cognitive development is impaired, resulting in lowered school performance. For pregnant women, anaemia contributes to 20% of all maternal deaths. Iron deficiency affects more people than any other condition, constituting a public health condition of epidemic proportions. So then let us get to know about iron deficiency and anaemia. We begin with the signs and symptoms of IDA.

Signs and symptoms of iron deficiency anaemia

Since the level of haemoglobin is reduced in the blood, it causes paleness (pallor) on certain parts of the body. Initially, such paleness can be seen in conjunctiva and in the roof of the mouth. Since haemoglobin is important for carrying oxygen in the body, anaemic individuals develop breathlessness even on milder exertion. These manifestations exist among adults, especially in pregnant and lactating women. The nails of finger and toes become papery thin and bend upwards to assume shape of spoon. This condition is known as "koilonychia". In severe cases of anaemia particularly among pregnant women, oedema (swelling of feet) is also present. Blood examination for haemoglobin estimation is the best way for the diagnosis of anaemia. Box 9 gives the manifestations of iron deficiency anaemia.

Box 9	Manifestations of Iron Deficiency Anaemia
• Paleness of conjunctiva • Paleness of tongue • Paleness of mucosa of soft palate • Low haemoglobin • Swelling of feet in severe anaemia • Koilonychia	

We have reviewed the signs and symptoms of iron deficiency anaemia. Let us now learn how common the problem of iron deficiency anaemia i.e. the prevalence is?

Prevalence of iron deficiency anaemia

We can find out about the prevalence of anaemia if we know what percentage of population is suffering from anaemia. The WHO has recommended different cut-off levels of haemoglobin below which an individual is considered as anaemic. These are indicated in Table 3.9. These values are dependent on age, sex and physiological status.

Table 3.9: WHO haemoglobin cut-off criteria

Group	Cut-off for Haemoglobin (g/100 ml)
Children < 6 years	11
Children > 6 years Adolescents Non-pregnant and non-lactating adult women	12
Pregnant women	11
Lactating women	12
Adult males	13

sessment of anaemia is based on estimation of these criteria for cut-off values for emoglobin. You probably know that women of child bearing age, including adolescent ls, are at the highest risk of developing anaemia followed by preschool children, hool children and adult men. A number of sample surveys carried out recently in

our country showed that 60-80% of pregnant women had haemoglobin le
11 g/dl and about 10 percent had severe anaemia with less than 8 g/dl. Eve
preschool children, about 40-50% were anaemic. In the villages near Kolkata,
hookworm infestation was common, more than 90% of the population was anae
These data clearly indicate that in India there is a need to cover the entire population in the intervention programme designed to control anaemia.

So far we have looked at the signs and symptoms and prevalence of iron deficiency anaemia. Let us elaborate on what causes iron deficiency anaemia.

Causes of iron deficiency anaemia

Anaemia is a condition in which the blood cannot carry enough oxygen. This may be because there are fewer red blood cells than normal, or because, as mentioned above, there is not enough haemoglobin in each cell. Iron is the main component of haemoglobin. Lack of dietary iron is the world's leading nutritional deficiency and the most common cause of anaemia. Let us get to know about the causes in greater details.

- *Inadequate dietary intake*

The commonest cause of anaemia is dietary inadequacy of iron. The dietary intakes are usually half of the recommended dietary allowances in every age and physiological group. In Indian communities, since cereals form the major source of iron, poor bioavailability of iron from the habitual diets is an important cause of iron deficiency. Isotope studies have shown that iron absorption ranges between 2-6 percent, depending upon the type of cereal in the diet. Phytates and tannins present in Indian diet interfere with iron absorption to a significant extent. The chemically determined iron content of the Indian diets is apparently high (15mg/1000 calories), but 30% of it is unabsorbable contaminant iron. The true dietary iron content is, therefore, only 10 mg/1000 calories, which can meet the iron requirement of adult men and children less than 6 years, provided their dietary intake meets the energy requirements. However, in order to meet the iron requirements of women in the reproductive age group, either the bioavailability of dietary iron should be improved or additional iron must be supplemented.

- *Poverty and ignorance*

Low purchasing power of the communities and their consequent inability to meet the nutrient requirements, even after spending 80-90% of their income on foods is an important factor contributing to prevalence of nutritional deficiencies. Animal foods help in increasing the bioavailability of iron, but their consumption is low due to the high cost. In addition, due to traditional beliefs and ignorance, locally available inexpensive sources like green leafy vegetables are not fully utilized. Similarly, the utilization of medical and health services is also poor. Box 10 lists different causes of iron deficier anaemia.

Box 10	Causes of Iron Deficiency Anaemia
	• Dietary Inadequacy
	• Poor bioavailability of iron
	• Presence of absorption interfering substances in diet
	• Poverty and ignorance

Having studied about the causes, let us now learn what happens if iro anaemia is not prevented or controlled, that is, what its consequences are.

Consequences of iron deficiency anaemia

The consequences of anaemia, particularly in women and children, are quite s and have far reaching implications as already discussed above. Some of th listed as follows:

- *Maternal and perinatal mortality*

Severe anaemia in pregnancy is associated with increased risk of maternal and perinatal mortality and foetal wastage. It is estimated that at least 80,000 women die due to anaemia every year.

- *Low birth weight*

In addition, maternal anaemia contributes to high incidence of premature delivery and low birth weight and mortality.

- *Physical work and mental performance*

Generally, quite often, women neglect milder forms of anaemia, but there is now evidence showing that even a moderate reduction in haemoglobin can lower resistance to infection and reduce work capacity.

- *Poor cognitive performance in children*

Anaemia in infancy and childhood is associated with poor cognitive abilities and behavioural changes. Box 11 highlights various consequences of IDA.

Box 11	Consequences of Iron Deficiency Anaemia
	• Maternal and perinatal mortality • Low birth weight and prematurity • Reduced physical work capacity • Poor cognitive performance in children

We have learnt about consequences of iron deficiency anaemia. Next, how do we treat this problem. Read the next section and find out.

Treatment of iron deficiency anaemia

Oral iron is the preferred method of treatment of IDA. The dosage is decided depending on the severity of the condition. Generally, in moderate to severe anaemia, 2 tablets of fersolate (each equivalent to 100 mg of elemental iron) are given. In view of side effects like gastric irritation, constipation, black stools and at times joint pains, many patients discontinue treatment. They should, therefore, be advised to consume the tablets after food. In very severe anaemia with very low levels of haemoglobin (< 5-7 g/100 ml), packed cell transfusion is recommended. This mode of treatment should be considered only after proper evaluation of the subject. Sometimes, parenteral iron therapy is advised when oral iron is not tolerated or in late pregnancies. In view of the risk of some systemic and allergic reactions, this should be given preferably in hospitals.

2. Fill ..... ave now seen that iron deficiency anaemia is a very common problem in women and children. It thus becomes very important that we learn about different measures to prevent it. The next section focuses on this aspect.

Prevention of iron deficiency anaemia?

of As in the case of vitamin A deficiency, correction and prevention of dietary inadequacy of iron are important sustainable methods of prevention of iron deficiency anaemia. However, this is a long-term strategy requiring not only improvement in increasing availability of iron in the diets but also changing behaviours of community. In view of the widespread extent of iron deficiency anaemia, alternate methods are required to control anaemia. A mix of approaches is necessary. The available methods of prevention and control of anaemia are:

- Supplementation
- Food fortification

- Dietary diversification
- Education (behaviour changes)
- Health care

Let us review each of these in detail.

Supplementation

Supplementation with low doses of iron is necessary to prevent anaemia in particula groups of people. Fortified foods and a good diet are not enough if a person is iro deficient and anaemic. Consumption of supplement in the form of pills and syrup wil. raise iron levels and normalize a person's iron stores. Thereafter, dietary improvement and consumption of fortified foods will prevent iron deficiency. Taking cognizance of the wide spread prevalence of nutritional anaemia, the Government of India launched the 'National Nutritional Anaemia Control Programme' in 1970 to prevent and control nutritional anaemia. A detailed discussion on this programme is presented later in Unit 10. You will learn that the beneficiaries are pregnant women, lactating women, preschool children and family planning acceptors. Under the programme, all the beneficiaries receive one tablet, containing iron and folic acid commonly referred to as folifer tablets, daily for 100 days. While the adult beneficiaries get tablets containing 100 mg of elemental iron and 0.5 mg (500 mg) of folic acid, the children receive 20 mg of elemental iron and 0.1 mg (100 mg) of folic acid. Each beneficiary should receive a total of 100 tablets. In the case of children, each year, 100 tablets are given.

Although the national programme has been in operation for over 30 years, the prevalence of anaemia continues to be very high due to poor implementation of the programme due to the following reasons:

- inadequate and irregular supplies,
- poor coverage due to lack of supervision,
- orientation of health functionaries, and
- absence of nutrition education to the illiterate community.

Fortification

Food fortification is one of the alternatives that ensure consumption of the nutrient regularly in daily diet. Fortification is addition of iron to food items that are regularly consumed by at-risk groups of population. However, the food item should be: centrally produced, inexpensive, consumed in uniform quantities daily, should not alter either the cooking quality of the food item or the taste or colour of the food. At present, of all the food items, salt satisfies these criteria and, hence, could be a suitable vehicle for fortification with iron. Studies conducted at the National Institute of Nutrition clearly indicate the feasibility of fortification of salt as a simple method to prevent and co iron deficiency anaemia. Other food items that are being fortified are wheat flour and breakfast cereals. Infant weaning foods are also fortified with iron, as milk is a poor source of iron. In India, the national nutrition policy recommends implementation of food fortification as a method of control of anaemia. Since iodized salt is already being distributed in different parts of the country, the technology of fortification of salt with both iodine and iron has been successfully developed at the National Institute of Nutrition, Hyderabad. Field trials are in progress.

Dietary diversification

It aims to ensure that deficient populations have access to foods rich in iron and also foods rich in vitamin C (since vitamin C helps the body absorb iron). Since the deficiencies of micronurients are common, what is needed is a strategy, which is self-sustaining, and provides multiple nutrients at a cheaper cost to the needy population. Home gardening and horticulture is an important strategy, which could be easily adopted by the population to whom raising gardening is a daily practice. It does not require

3. Answer the following briefly:

a) List four main consequences of iron deficiency anaemia.

b) Why does prevalence of iron deficiency anaemia continues to remain high in India?

Let us now learn about Iodine Deficiency Disorders.

3.3.3 Iodine Deficiency Disorders (IDD)

Iodine is an essential micronutrient required for the normal mental and physical growth and development of man. Iodine deficiency is a naturally occurring ecological phenomenon that is present in many parts of the world. IDD affects over 740 million people, 13% of the world's population. 30% of the remainder are at risk. About 60 million people in our country are estimated to suffer from goitre and another 3 million from cretinism. About 200 million populations are at risk of developing iodine deficiency disorders. Traditionally the endemic goitre belt in our country stretches across the entire sub-Himalayan belt extending from Jammu and Kashmir to Arunachal Pradesh. In addition, a number of new regions have been identified in Andhra Pradesh, Karnataka, Kerala, Maharashtra and Madhya Pradesh.

The term iodine deficiency disorders (IDD) includes *a spectrum of disabling conditions affecting the health of human beings starting from foetal life through adulthood resulting from inadequate dietary intake of iron.* We will explain this term in more detail in the next few paragraphs. Let us begin our study on IDD by getting to know about the signs and symptoms of this disabling condition.

Signs and symptoms of iodine deficiency disorders

Before we discuss the signs and symptoms of iodine deficiency disorders, let us first understand why we need iodine in our body. Iodine is required in our body for the synthesis of thyroxin, which you may already know is the hormone produced by the thyroid gland. When iodine intake falls below the recommended levels, the thyroid gland is no longer able to synthesize sufficient amounts of thyroxine hormone. One of the well-recognized features of iodine deficiency disorders is *goiter.* Thyroid gland in its efforts to produce the required thyroxine, in the presence of iodine deficiency, swells up leading to enlargement of the thyroid gland as illustrated in Figure 3.4. This condition is known as *goitre*, which is more a cosmetic problem. The real health problems are because of functional failure of thyroid gland in different stages of individual development. Let us get to know about these problems.

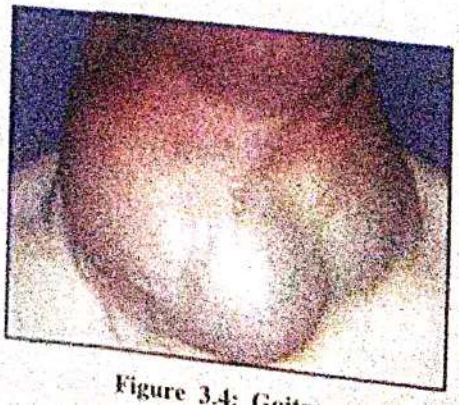


Figure 3.4: Goit

Iodine deficiency in the mother interferes with the development of the unborn child. In many cases, iodine deficiency can cause abortions, congenital abnormalities and increased perinatal mortality. The major effect of foetal iodine deficiency is *endemic cretinism*. It is characterized by growth failure, mental deficiency, deaf mutism and spastic paralysis of legs. Inadequate production of thyroid hormone leads to hypothyroidism. Hypothyroidism is the principal factor responsible for the damage done to the developing brain and the other harmful effects known collectively as *iodine deficiency disorders* (IDD). Populations residing in iodine deficient areas exhibit low intelligence, lack of initiative and poor decision making capacity. Box 13 highlights various signs and symptoms/manifestations of IDD.

Box 13	Manifestations of IDD
•	Goitre
•	Abortions, Congenital abnormalities,
•	Increased perinatal mortality
•	Cretinism

You have learnt about signs and symptoms of IDD. We hope having gone through the discussion above the whole spectrum of disabling conditions caused due to IDD must be clear. Let us now look at the prevalence of IDD.

Prevalence of IDD

We can determine the prevalence of IDD by conducting population surveys. Most of the population surveys are based on clinical assessment of goitre and cretinism, which are the two classical features of iodine deficiency. Before we discuss the prevalence of IDD, let us find out the WHO criteria for classification of goitre size. For clinical assessment of goitre, a standard technique based on palpation of thyroid is used through which goitre size can be assessed. Table 3.10 gives the WHO criteria for classification of goitre size. The sum of grades 1 and 2 provides Total Goitre Rate (TGR). IDD is considered to be a public health problem, if the TGR is more than in 10% of the children aged 6-12 years in an area.

Table 3.10: Classification of goitre

Grade '0'	No goitre (Neither palpable nor visible, palpable but the size is less than the distal phalange)
Grade 'I'	Not visible when neck is in normal position, but palpable (The size of the enlargement of the gland should be more than the size of the distal phalange of the thumb of the subject.
Grade 'II'	Visible from the minimum distance.

Now let us look at the prevalence of goitre and cretinism in India. Out of 208 districts surveyed by the Directorate General of Health Services of Government of India in the country, 182 districts have been found to be endemic for goitre, the prevalence ranging from 10% in Ranchi district to 96% in some districts of Mizoram. It is estimated that, today, among the 200 millions living in endemic areas, more than 54 million people in India are suffering from endemic goitre and 8.8 million from different grades of mental/motor handicaps. In the villages of Uttar Pradesh and Bihar where the goitre prevalence was high, deaf-mutism, mental retardation and other clinically detectable defects attributable to environmental iodine deficiency were found in 4% of the children.

Epidemiological assessment of IDD also requires a measure of dietary iodine, which is provided by urinary iodine excretion. Determination of iodine in random urine

samples, which is more convenient, provides a good indication of the level of iodine nutrition. Urinary iodine content of ≥ 10 mg/dl is normal. In some of the areas, despite prevalence, which is indicative of endemicity, urinary iodine levels are in the normal range.

It must be evident to you that IDD is widely prevalent in our country. Let us now review the causes of IDD.

Causes of iodine deficiency disorders

We have studied above that iodine deficiency is a naturally occurring ecological phenomenon that is present in many parts of the world. The main cause of iodine deficiency in soils is leaching by floods or high rainfall. Mountainous regions including the Himalayas therefore have some of the highest prevalence of iodine deficiency. Iodine deficiency also occurs due to flooding; for example, in India around the Ganges. In areas of endemic iodine deficiency, the water and foods (plants and animals grown there) have low iodine content. Let us get to know about the causes next.

- *Environmental deficiency of iodine:* The ultimate causative factor is deficient intake of iodine. Iodine occurs in soil and sea as iodide, the ions of which are oxidized by sunlight to elemental iodine (which is volatile). Iodine in the atmosphere returns to soil by rain. The return of iodine, however, is slow and small in amount compared with original loss. In hilly slopes, repeated flooding leaches out the iodine from soil or erodes topsoil causing iodine deficiency in the soil. All crops grown in this soil will, therefore, be iodine deficient. As a result, human and animal populations, which are totally dependent on food grown in such soil, become iodine deficient.

- *Goitrogens:* Certain chemical substances like thiocyanates, phenols, disuphides, flavanoids etc, found in the environment, can interfere with iodine metabolism. These substances are known as *goitrogens*, which could cause goitre. Common foods such as cabbage, sorghum, finger millets and mustard contain goitrogens. Although excessive intake of such foods can cause goitre, this appears to be of secondary importance in the etiology of endemic goitre, at least in India.

Let us now review the consequences of IDD.

What are the consequences of IDD?

As discussed earlier under signs and symptoms, the consequences of IDD include: mental retardation, other defects in the development of the nervous system, goitre, physical sluggishness, growth retardation, reproductive failure, increased childhood mortality and lowered economic productivity. Cretinism is the result of iodine deficiency during pregnancy, which adversely affects foetal thyroid function. Neurological cretinism is characterized by poor cognitive ability, deaf mutism, speech defects, and proximal neuromotor rigidity. It is much more prevalent than myxoedematous cretinism which includes hypothyroidism with dwarfism. Maternal iodine deficiency during pregnancy is associated with a higher incidence of stillbirths, abortions and congenital abnormalities. Iodine deficiency has been called the world's major cause of preventable mental retardation. So then what can be done to prevent this disabling condition? Read and find out.

Prevention of IDD

Many approaches to reduce iodine deficiency have been formulated. Some of these are reviewed herewith:

Iodized salt distribution: Since IDD is due to reduced uptake of iodine by human body from the environment, the control measures essentially aim to ensure sufficient intake of iodine by persons living in iodine deficient environment. The oldest and the commonest control measure is fortification of common salt with potassium

India, the efficacy of iodized salt in the control of endemic goitre was first established in Kangra Valley of Himachal Pradesh. Subsequently, the Government of India launched the National Goitre Control Programme, in 1962, to supply iodized salt in endemic areas. Although the programme has been in operation for the last three decades, it has gained momentum only recently. Available evidence indicates that iodized salt consumption is quite safe even in non-endemic areas.

Communication campaign: A mass communication campaign is needed to create awareness in the community about the consequences of IDD and the benefits of iodized salt. The community should be made aware of the ill effects of iodine deficiency and the advantages of iodized salt. They should be encouraged to consume iodized salt daily.

Double fortified salt: Since iron deficiency anaemia and iodine deficiency disorders often co-exist, the most effective approach to control these public health problems would be simultaneous fortification of salt with iron and iodine. The technology for double fortification of salt has been successfully developed at NIN. Laboratory studies have shown satisfactory results with respect to stability and bioavailability of iron and iodine. Large-scale community trials are underway for field-testing the double fortified salt.

Iodized Oil: The other approach employed as a specific measure for women and children in hyper-endemic areas is injection of iodized oil. Intramuscular injection of iodized oil has been used for tackling goitre and cretinism in hyper-endemic areas in many countries of the world. The advantage of the injection procedure is that a single dose of 1 ml will provide protection for 3-5 years. Though, it has been found to be effective, the high cost and the difficulty in reaching all the victims of IDD make this approach less practicable. The use of disposable syringes, as a result of the risk of hepatitis-B and HIV AIDS, is now mandatory. Box 14 highlights methods of prevention and control of IDD.

Box 14	Prevention and Control
•	Iodized salt
•	Iodized oil injection
•	Double fortified salt
•	Mass communication

We have just learnt about manifestations, prevalence, causes, prevention and control of iodine deficiency disorders. Finally, let us now learn briefly about zinc deficiency.

3.3.4 Zinc Deficiency

Evidence suggests that nearly one-third of preschool children in lower-income countries have stunted growth, and that a considerable proportion of this growth failure is likely attributable to zinc deficiency.

Zinc is a cofactor for a large number of 200 metalloenzymes, which regulate several cellular functions of the body. Zinc is essential for cell division and growth, stabilization of bio-membranes, protection against free radical damage, immune function and its possible role in testosterone production. Zinc, in the recent past, has attained an important place as an important trace element. We will briefly study here the signs and symptoms and the consequences of zinc deficiency and the recommended daily requirements for zinc.