
UNIT 4 NUTRITIONAL PROBLEMS-II

Structure

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4.1 INTRODUCTION

In the previous unit, we learnt about protein energy malnutrition and the commonly occurring micronutrient deficiencies of vitamin A, iron and iodine. These nutritional deficiencies are widely prevalent in India and other developing countries. They cause illness and death in a large number of people, especially in women and children. Other micronutrients found in food, including vitamins such as thiamine, niacin, riboflavin, folate, vitamin C and D can also significantly affect health when dietary deficiencies exist. As a public nutrition professional, it is very important for us to know about these problems. In this unit, we will learn about the deficiency diseases caused when there is a lack of these vitamins in the diet. We will also learn about fluorosis and lathyrism. However, these are not vitamin deficiency diseases. Fluorosis is caused by *excess of fluoride in water*. Lathyrism is caused by *neurotoxin present in kesari dal*. These diseases cause many complications in our body. So it is important for us to learn about them.

Objectives

After studying this unit, you should be able to:

- describe the significance of common deficiencies of vitamin B-complex, C and D;
- understand the problem of fluorosis and lathyrism in Indian population;
- identify cases of these nutritional problems;
- enumerate their causes and consequences; and
- educate the families and communities about the methods of their prevention.

4.2 VITAMIN DEFICIENCIES

We already know that vitamins are very essential to support growth and development in our body. They are not synthesized by our body and so need to be supplied in the daily diets in small quantities to satisfy the requirements and maintain

good health. B-complex vitamins and Vitamin C being water-soluble are not stored in the body, are easily excreted and hence, their deficiencies are generally encountered. In addition, deficiency of a fat soluble vitamin – vitamin D is also encountered in some areas. Box 1 lists clinical deficiency related to each vitamin.

Box 1	Clinical Deficiency Related to the Vitamins
Vitamins	Clinical Deficiency
Thiamine (B ₁)	Beriberi
Riboflavin (B ₂)	Ariboflavinosis
Niacin	Pellagra
Folic Acid and B ₁₂	Megaloblastic and Pernicious anaemia
Ascorbic acid (vitamin C)	Scurvy
Vitamin D	Rickets and Osteomalacia

Let us elaborate upon each of the vitamin deficiency in detail. We shall begin with deficiency of thiamine i.e. beriberi. We will study about the manifestations, cause, prevention and treatment of thiamine deficiency.

4.2.1 Beriberi

Beriberi, which is caused by the deficiency of vitamin B₁ (i.e. thiamine), was once a major disease problem in many parts of the developing world, including India. Today, the prevalence of beriberi has been greatly reduced. Beriberi occurs in people whose staple diet consists mainly of polished white rice, which contains little or no thiamine. Therefore the disease has been seen traditionally in people in Asian countries (especially in the nineteenth century and before) and in chronic alcoholics with impaired liver function.

Bontius (1642) and *Nicolaas Tulp* (1652) were the Dutch physicians, who gave the first clinical descriptions of beriberi. *Tulp's* description of beriberi was a detailed one, but interestingly he had no clues that it was a dietary deficiency disease. This discovery came more than two hundred years later. In fact, thiamine deficiency, which causes damage to central and peripheral nervous system and the heart, has been known well before even the thiamine (vitamin B₁) was discovered in the year 1926. The disease is now rather rare. Let us learn about the manifestations of this disorder.

Manifestations

A brief review of the manifestations of thiamine deficiency is also presented in the Advance Nutrition Course (MFN-004) in Unit 8. We suggest you look up the unit now. You would realize that beriberi is of different types described as *cardiac beriberi* (*wet beriberi*), *dry beriberi* and *infantile beriberi*. The severity of deficiency depends upon the degree and duration of deficiency. The early clinical features are anorexia and dyspepsia, associated with heaviness and weakness of the legs. There is tenderness of the calf muscles on pressure with complaints of 'pins and needles' pain and numbness in the legs. The knee jerks are usually sluggish but occasionally slightly exaggerated. The subjects feel weak and get easily exhausted while working. A brief description of the different types of beriberi mentioned above follows.

Cardiac beriberi is the wet type and the signs and symptoms are of ventricular failure characterized by difficulty in breathing, particularly on physical exertion,

palpitation, cyanosis and oedema. Remember, *oedema* is the important feature of wet beriberi. It may develop rapidly and involve not only the legs but also the face, trunk and serous cavities.

Dry beriberi is milder form of the disease with polyneuropathy with clinical signs characterized by numbness, burning sensation – commonly referred to as ‘pins and needles’ in the limbs, tenderness of muscles, muscle cramps and weakness in limbs. The muscles become progressively wasted and weak and walking becomes difficult. The emaciated subject needs the help of sticks to stand and walk and finally becomes bed-ridden. If not treated, the patients will die.

Infantile beriberi, seen among breast-fed infants, perhaps, is due to low thiamine in mother’s milk. Two types of infantile beriberi are known. These are: (i) cardiovascular type, and (ii) neuritic type. Let us get to know about them.

- i) *The cardiovascular type (wet)*: It manifests itself in babies between the ages of 2 and 4 months. The onset is acute with classical signs and symptoms of congestive cardiac failure, tachycardia (rapid heart beat), dyspnoea (difficulty in breathing), enlargement of the heart, elevated venous pressure, enlarged tender liver, dependent oedema and oliguria (infrequent urination). In some infants, cyanosis and pulmonary oedema may develop rapidly and death may ensue in a matter of few hours.
- ii) *The neuritic type (dry)*: It shows typical manifestations of peripheral neuropathy, tenderness of calf muscles, diminished tendon jerks, hyperaesthesia, is rare in children, but a pseudo-meningeal form, (cerebral or Wernicke’s syndrome) tends to occur in older infants between 8 and 10 months of age. The accent is predominantly on the C.N.S. with sensorial alteration (irritability, apathy, drowsiness and coma) signs of raised intracranial tension, staring expression and varying degrees of neurologic deficit.

Having studied about the manifestations, it is important to understand that if not attended to immediately, beriberi can lead to loss of speech, convulsions, coma and ultimately death. In chronic alcoholics, thiamine deficiency is characterized by encephalopathy (disease of the brain), which manifests as confusion, polyneuropathy, and certain changes in eyes. It may cause forgetfulness, depression and delirium. For your recapitulation Box 2 lists types of beriberi.

Box 2	Types of Beriberi
	Cardiac Beriberi
	Dry Beriberi
	Infantile Beriberi
	Poly neuropathies

Next, let us learn about the causes of beriberi.

Causes

Some of the important causes of beriberi are consumption of highly polished rice and improper cooking practices such as throwing away the excess water after cooking the rice. You must have read in the Advanced Nutrition Course (MFN-004) that the thiamine requirements are related to the quantity of calories consumed by an individual viz 0.5 mg per 1000 calories/day. Consumption of foods of higher energy with lower thiamine content leads to lower vitamin energy ratio. The at-risk groups include children, adolescents, athletes and elderly. Deficiency of thiamine is also very common among chronic alcoholics. So then what measures can we adopted to treat and prevent this disorder? Read the next section and find out.

The specific treatment of beriberi is the administration of thiamine. Parenteral administration of thiamine in doses of 10-20 mg twice or thrice a day gives dramatic results. Care is required as it can lead to anaphylactic (hypersensitivity) reactions. Oral administration of 5-10 mg of thiamine for longer durations is preferred. Larger doses are wasteful.

Let us next review how beriberi can be prevented.

Prevention

In the community, there are several possible approaches to the prevention of beriberi. Diversification of the diet or the encouragement of the use of parboiled or undermilled rice i.e. avoiding excess milling and the consequent high polishing of rice are logical approaches. Similarly, adopting proper cooking practices such as not using and throwing excess water for cooking of rice would help in the retention of thiamine. Parboiling and hand-pounded rice are good sources of vitamin B₁. The communities should be educated to consume foods regularly, which are rich in thiamine (such as whole grain cereals, raw and hand-pounded or parboiled rice, pulses, wheat germ etc.) and should be encouraged to avoid excessive consumption of alcohol.

After thiamine, next let us learn about their manifestation, cause, prevention and treatment of riboflavin deficiency i.e. ariboflavinosis.

4.2.2 Ariboflavinosis (Riboflavin Deficiency)

Riboflavin is one of the important B-complex vitamin, the deficiency of which is encountered in our communities frequently. Surveys carried out in different areas of the country indicated that it is prevalent among the poorer groups of population of all ages, particularly among children and pregnant and nursing women. It is also common in elderly population.

Riboflavin, we know, is involved as a cofactor in a number of the respiratory enzymes (flavin adenine dinucleotide (FAD), and flavin mononucleotide (FMN)), which are involved in energy metabolism. Thus it plays a major role in intermediary metabolism. The dietary deficiency of this vitamin, therefore, leads to a condition called *ariboflavinosis*, characterized by mouth lesions. Let us learn about its manifestations in greater details.

Manifestations

Lesions in mouth and tongue, skin, corneal and haematological changes, characterize the deficiency of riboflavin. The commonest signs are *angular stomatitis* (cracks at the angles of the mouth), *glossitis* (sore tongue) and *cheilosis* (ulcers on lips) as illustrated in Figure 4.1. *Angular stomatitis* may progress to fissures at the angles of the mouth. Sometimes, fungal infection may supervene. In *glossitis*, the tongue is acutely inflamed and papillae (projections) on the tongue become hypertrophic (prominent), sometimes, the papillae also get atrophic (decrease in size), producing bald tongue. The hypertrophic papillae produce the classical magenta red tongue and as the disorder advances, the papillae get atrophic. In *cheilosis*, one of the features of chronic deficiency, mucous membrane of the lips denudes and ulcers are formed. *Nasolabial dyssebaceae*, a seborrhic type of dermatitis involving facial skin is also often seen in ariboflavinosis. Rarely, eye symptoms like *photophobia* (inability to see brightness) are also reported. Corneal vascularization may also occur in riboflavin deficient. Box 3 summarizes the manifestations of riboflavin deficiency.

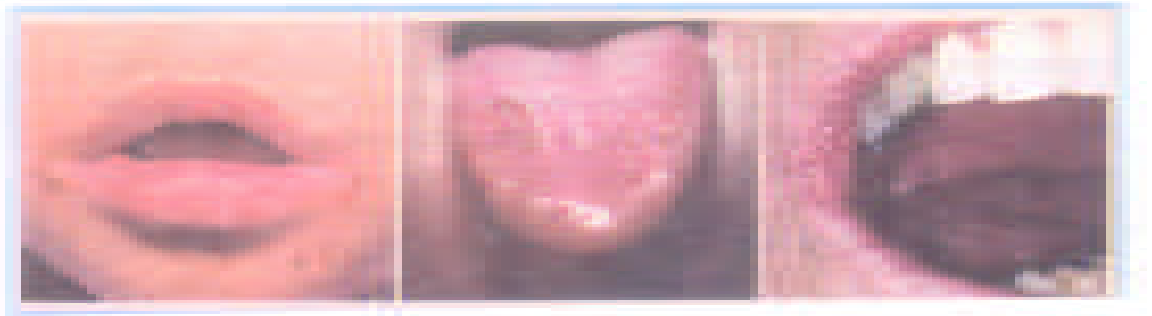


Figure 4.1: Manifestations of riboflavin deficiency

Box 3	Manifestations of Riboflavin Deficiency
	Angular stomatitis Glossitis Cheilosis Nasolabial Dyssebaceae

Let us learn about the causes of riboflavin deficiency, next.

Causes

Dietary inadequacy is usually the cause of riboflavin deficiency. Inadequate consumption of pulses, nuts and milk products, which the households belonging to the low socioeconomic groups cannot afford, is the main reason for the wide spread riboflavin deficiency in the country. Alcoholism, malabsorption, tuberculosis, hyperthyroidism and chronic infections are also associated with ariboflavinosis. Certain drugs can also induce it.

So, then how can ariboflavinosis be treated? Let us find out.

Treatment

Oral administration of 5-10 mg of riboflavin daily is often satisfactory to treat riboflavin deficiency. In subjects suffering from malabsorption, parenteral riboflavin may be given.

Let us learn how we can prevent riboflavin deficiency.

Prevention

Improvement of diets to ensure adequate riboflavin daily is the most rational solution to prevent riboflavin deficiency. For poorer populations, foods providing riboflavin like pulses, nuts and milk products are expensive. Supplements of riboflavin to vulnerable segments like pregnant women are often recommended.

Next let us move on to the deficiency of niacin i.e. pellagra. We will study about the manifestations, cause, prevention and treatment of pellagra.

4.2.3 Pellagra

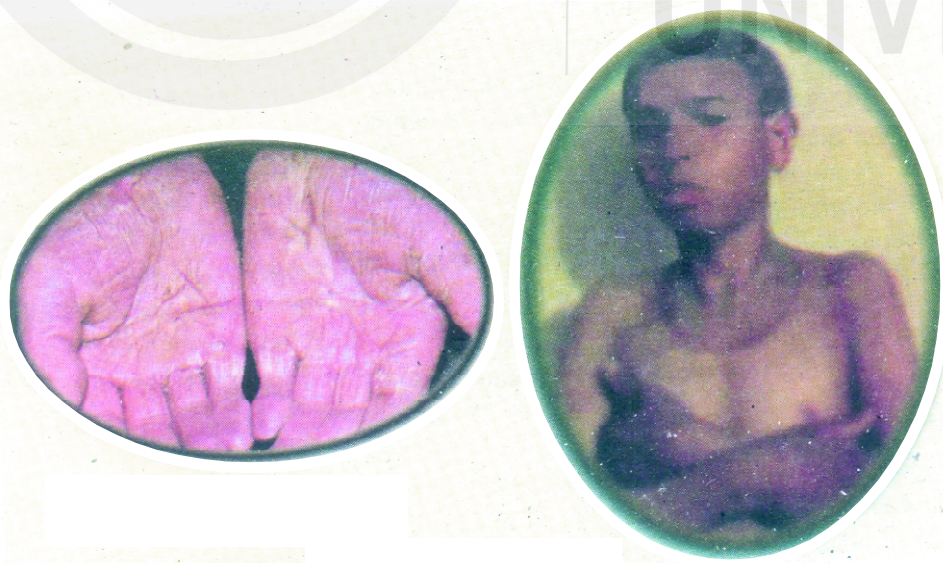
Pellagra was considered to be an infectious disease until the early 20th century. It was only in 1917 that *Joseph Goldberger* succeeded in proving that the disease was caused by nutritional deficiency.

Pellagra is a disease that occurs when a person does not get enough niacin (one of the B complex vitamins) or tryptophan (an amino acid) in their diet. It can also occur if the body fails to absorb these nutrients. Pellagra, due to niacin deficiency, was very common in countries like Mexico where maize was the staple. Niacin was demonstrated to be anti pellagra factor in 1937. What are the manifestations of pellagra? The next section focuses on this aspect.

Manifestations

Pellagra is seen generally in individuals in the age group of 20 and 50 years, in both the sexes. To start with, it may manifest with nonspecific symptoms like weakness, limited capacity for work, loss of appetite, nausea, early fatigue and some gastrointestinal disturbances, anxiety and sleeplessness. It is sometimes reported that considerable proportion of patients attending mental hospitals may be suffering from pellagra. The classical manifestations of niacin deficiency are *dermatitis*, *diarrhoea* and *dementia* (commonly referred to as 3 Ds) and can lead to death (the fourth D).

The dermatological changes, called “pellagra”, are usually the most prominent. *Dermatosis* in pellagra is seen typically in areas exposed to sun (photosensitive). It is seen on the exposed parts of the body like the upper and lower extremities, face and neck as can be seen in Figure 4.2(a). It may be symmetrical and bilateral (on both the sides). The lesions are aggravated by exposure to skin. The lesion starts with erythema resembling sunburn, which is symmetrically distributed on the parts of the body exposed to direct sunlight—the backs of the hands and forearms up to the rim of the sleeves (“pellagra gloves”), the feet and legs up to the edge of the trousers or skirt, the forehead, and on the nose and cheeks in a butterfly distribution. The skin lesions on the neck appear in the form of necklace, generally referred to as “Casal’s necklace” as illustrated in Figure 4.2(b).



(a) Skin lesions on the hand

(b) Skin lesions on the neck

Figure 4.2: Manifestations of pellagra

Pellagra patients usually complain of nausea, excessive salivation, a burning sensation in the epigastrium, and diarrhoea. *Diarrhoea*, due to inflammation of gastrointestinal tract could be bloody in nature. In fact, the mucous membrane of the gastrointestinal

tract is inflamed causing enteritis and gastritis. Signs of B-complex deficiency like glossitis are very common in pellagra. The mouth is sore and the tongue is brilliant or beef red in colour and swollen. Cheilosis and angular stomatitis are seen in niacin deficiency, though these may, in part, be a result of a simultaneous riboflavin deficiency.

As described above, early neurological symptoms associated with pellagra include anxiety, depression, and fatigue. Later symptoms include apathy, headache, dizziness, irritability and tremors. In early cases the manifestations are psychoneurotic, later, lesions affect the nerves. *Dementia*, where due to derangement of mental functions, the patient suffers from insomnia, disorientation, confusion and even delirium. There may be changes in electroencephalogram.

Box 4 summarizes the clinical manifestations of pellagra.

Box 4	Clinical Manifestations of Pellagra in Adults
Body System	Typical Lesions
Skin	Initial changes: temporary redness like that of sunburn Hyper pigmentation and thickening of skin Dark red or purplish eruptions followed by desquamation Lesions are bilateral or symmetrical involving areas of friction and exposure i.e. face, neck, hands and feet
Mouth	Gingivitis, stomatitis and glossitis, tongue is swollen and beefy red in colour
Gastrointestinal tract	Diarrhoea
Central Nervous System	Progressive dementia with apprehension and confusion in the early stages progressing to severe derangement.

Let us now learn how pellagra is caused.

Causes

Pellagra is a diseases closely associated with poverty, a low standard of living and poor environmental sanitation. The disease is associated with poorer communities whose staple is either maize or jowar. Maize has low tryptophan content and relatively low niacin content which, in addition, is in the bound form so that only about 30% is bioavailable. Tryptophan, the amino acid, is a precursor of nicotinic acid. You may recall studying in the Advance Nutrition Course (MFN-004) that 60 mg of tryptophan is equivalent to 1mg of nicotinic acid. The daily requirement of niacin are thus affected by the quantity and quality of protein in the diet, particularly the tryptophan content. In communities depending on jowar as staple, pellagra is attributed to metabolic changes caused by excess of amino acid *leucine*. Pellagra is also observed in alcoholics and those suffering from malabsorption.

Let us learn how pellagra can be treated.

Treatment

The patients with pellagra can be treated with a diet containing adequate amounts of protein, amino acid tryptophan or niacin. Oral administration of 100-300 mg of nicotinic acid every day is adequate except in cases with severe diarrhoea. Patients should also receive other B-complex vitamins, particularly riboflavin and pyridoxine to take care of neurological manifestations. The response to treatment is dramatic in the case of mental symptoms, which show improvement in 2-3 days. Three to four weeks' treatment is required for curing skin changes. However, prevention is better than cure. So, then let us study how we can prevent pellagra.

Replacement of the jowar or maize with cereals containing good quality protein can prevent pellagra. Development and propagation of strains of jowar that are low in leucine could be one of the approach. In areas which are endemic to pellagra, fortification of foods with niacin is another alternative. Fortunately, with changes in the quality of diet, particularly reduction in the consumption of maize and jowar, pellagra has been averted, to a large extent, in India.

Let us now learn about the deficiency of folic acid and Vitamin B₁₂ deficiency. We will study about the manifestations, cause, prevention and treatment of these deficiencies.

4.2.4 Folic Acid and B₁₂ Deficiency

Folic acid and vitamin B₁₂ are essential for the synthesis of nucleic acids and amino acids. In the recent past, folic acid is considered to be important to prevent neural tube defects in foetus. In this context it is important for us to study about the deficiency conditions associated with this vitamin. On the other hand nutritional deficiency of B₁₂ is rare. Let us review the symptoms, causes, prevention and treatment of folic acid and Vitamin B₁₂ deficiency.

Clinical Manifestations of Folic Acid and Vitamin B₁₂ Deficiency

The deficiency of folic acid the water-soluble vitamin of B-complex group leads to *megaloblastic anaemia*. Megaloblastic anaemia, you may recall studying earlier in the Applied Physiology Course (MFN-001) in Unit 2, is a blood disorder characterized by anaemia, with red blood cells that are larger than normal, usually resulting from a deficiency of folic acid or of vitamin B. Though, not as common as iron deficiency anaemia, folic acid deficiency is observed in as high as 40-50% of anaemia in pregnant women. Both peripheral smears of blood and bone marrow show macrocytes (larger RBC) as shown in Figure 4.3. The white cell count may also be less.

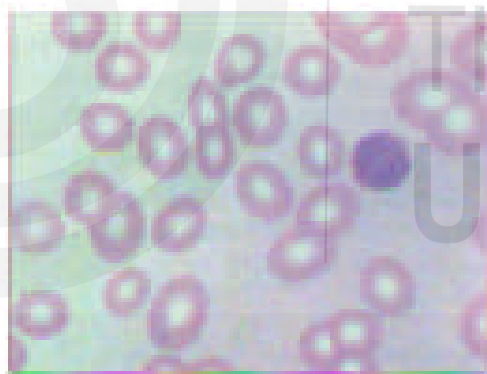


Figure 4.3: Large oversized red blood cells seen in megaloblastic anaemia

Vitamin B₁₂ deficiency, on the other hand, leads to what is known as *pernicious anaemia*, which is a type of megaloblastic anaemia, which could be considered as genetic in nature. *Pernicious anaemia* is caused by a lack of intrinsic factor, a substance needed to absorb vitamin B₁₂ from the gastrointestinal tract. Vitamin B₁₂, in turn, is necessary for the formation of red blood cells. Inadequate vitamin B₁₂ gradually affects sensory and motor nerves, causing neurological problems to develop over time. Because vitamin B₁₂ is needed by nerve cells and blood cells for them to function properly, deficiency can cause a wide variety of symptoms, including fatigue, shortness of breath, tingling sensations, difficulty in walking, and diarrhoea. In adults, it may lead to peripheral neuritis and some psychotic changes. In children, who are breastfed for prolonged periods, anaemia occurs as a result of dietary deficiency of the vitamin. Growth retardation and mental apathy are some of the manifestations.

Let us review what causes folic acid and B₁₂ deficiency.

Causes of Folic Acid and Vitamin B₁₂ Deficiency

Dietary deficiency is the main reason for folic acid deficiency. Folic acid is available in green leafy vegetables, liver, meat and pulses. However, considerable destruction of the vitamin occurs during cooking. Its deficiency can occur when there is impairment of absorption of folic acid like in pregnancy. Increased demands during infancy due to growth and pregnancy, prolonged use of anticonvulsants, infections and infestations may be important causes.

On the other hand, Vitamin B₁₂ deficiency occurs due to the absence of intrinsic factor in gastric mucosa. Intrinsic factor is a protein the body uses to absorb vitamin B₁₂. When gastric secretions do not have enough intrinsic factor, vitamin B₁₂ is not adequately absorbed, resulting in pernicious anaemia and other problems related to low levels of vitamin B₁₂. In addition to pernicious anaemia, other causes of vitamin B₁₂ deficiency include :

Nutrition (since vitamin B₁₂ is available only in animal foods, its deficiency is possible in pure vegetarians i.e. vegans. In countries like India, the vitamin appears to be derived mostly from faecal contamination of foods. Further poor diet in infant or poor maternal nutrition during pregnancy can be a cause for this deficiency)

Infection (intestinal parasites, bacterial overgrowth)

Gastrointestinal disease (stomach removal surgery, celiac disease (sprue), Crohn's disease)

Drugs (neomycin, tuberculosis, treatment with para amino salicylic acid etc.)

Metabolic disorders (methylmalonic aciduria, homocystinuria)

Let us next learn how these deficiencies can be prevented.

Prevention of Folic Acid and Vitamin B₁₂ Deficiency

Supplementation with folic acid along with iron is one of the strategies being adopted by the government to prevent and control anaemia due to folic acid deficiency. The details of the programme, you may recall studying in Unit 3 earlier under the micronutrient deficiencies – iron deficiency anaemia. The most rational approach to prevent folic acid deficiency is to improve the daily diets by ensuring foods rich in folic acid like green leafy vegetables, pulses and meat products. As for vitamin B₁₂ deficiency, consumption of as little as 250 ml of milk every day would suffice to prevent vitamin B₁₂ deficiency.

Let us get to know about the deficiency of Vitamin C i.e. scurvy. We will discuss about manifestations, causes, treatment and prevention here as we have done for the other deficiency diseases above.

4.2.5 Scurvy

Scurvy was endemic during the Middle Ages causing damage to armies in Europe and is, perhaps, one of the oldest diseases known to the humanity. It was considered to be due to poor intakes of fresh foods. What are the characteristic features of scurvy? Let us look at the features of scurvy next.

Manifestations

The characteristic clinical features of scurvy are spongy-bleeding gums (refer to Figure 4.4(a), petechial haemorrhages, joint pains, fatigue, depression and tenderness of bones. Common symptoms include pinpoint bleeding around hair follicles, along the gums, and under the nails, as seen in Figure 4.4 (b). In neonates, vitamin C deficiency is characterized by tenderness of lower extremities and haemorrhages in costochondral cartilages, fever and irritability. Bleeding into muscles and nail beds is

observed. Radiological evaluation (X ray) confirms the diagnosis. Vitamin C deficiency can lead to reduced ability to fight infections, reduced capacity for healing and mild anaemia.



(a) Bleeding gums

(b) Pinpoint bleeding under the nails

Figure 4.4: Manifestations of vitamin C deficiency

Having studied about the clinical manifestations of scurvy let us next get to know what causes scurvy.

Causes of Scurvy

The deficiency of ascorbic acid is not as common as it used to be before. However, vitamin C is heat-labile and water-soluble. Hence, faulty cooking practices, inadequate consumption of fresh vegetables and fruits are the major reasons for vitamin C deficiency. Citrus fruits, amla, guava and green leafy vegetables are good sources of ascorbic acid. You would note from the discussion that there are common causes of vitamin B complex and Vitamin C deficiency. These are listed in the Box 5.

Box 5	Causes of Vitamin B-complex and C deficiency
	Inadequate intakes
	Faulty cooking practices
	Malabsorption
	Prolonged use of drugs
	Alcoholism
	Increased demands

Let us learn how we can treat scurvy.

Treatment

Scurvy in children is cured by 10-25 mg of vitamin C, 2-3 times a day. It may take 2-3 weeks for complete treatment. Let us review how we can prevent scurvy.

Prevention

Scurvy is no more a public health problem in India. However, the role of vitamin C in promoting iron absorption and as a potent antioxidant has been recognized. Regular intake of fresh vegetables and fruits is the most rational and sustainable method of preventing vitamin C deficiency. The common sources of vitamin C that are readily available are: citrus fruits such as lemon, orange, guava, amla and tomato.

Let us learn about the deficiency of vitamin D i.e. Rickets and Osteomalacia. Here again, we will learn about manifestations, causes, treatment and prevention of vitamin D deficiency.

4.2.6 Rickets and Osteomalacia

Rickets has been known to be a nutritional disease for over 100 years and cod liver oil was known to prevent it. In India, rickets is not seen in community surveys, although it is seen in hospitals. Let us learn about the symptoms of rickets and osteomalacia.

Manifestations

Rickets occurs generally in growing children, who are not adequately exposed to sunlight. The signs and symptoms of the deficiency are mainly due to inadequate mineralization. While in the children it is known as *rickets*, in adults it is referred to as *osteomalacia*. The bones are soft and easily bend and therefore, deformities are common. In growing children, swollen and painful growing ends of long bones (particularly visible and palpable at the ribs, wrists and ankles) characterize it. The bones cannot stand even normal mechanical stress. As a result, when the children start walking, they develop bowlegs and *knock-knees*. In the ribs, at costochondral junction, swellings, which appear like rosary, referred to as *rachitic rosary* is found. Figure 4.5 illustrates some of these manifestations of vitamin D deficiency,

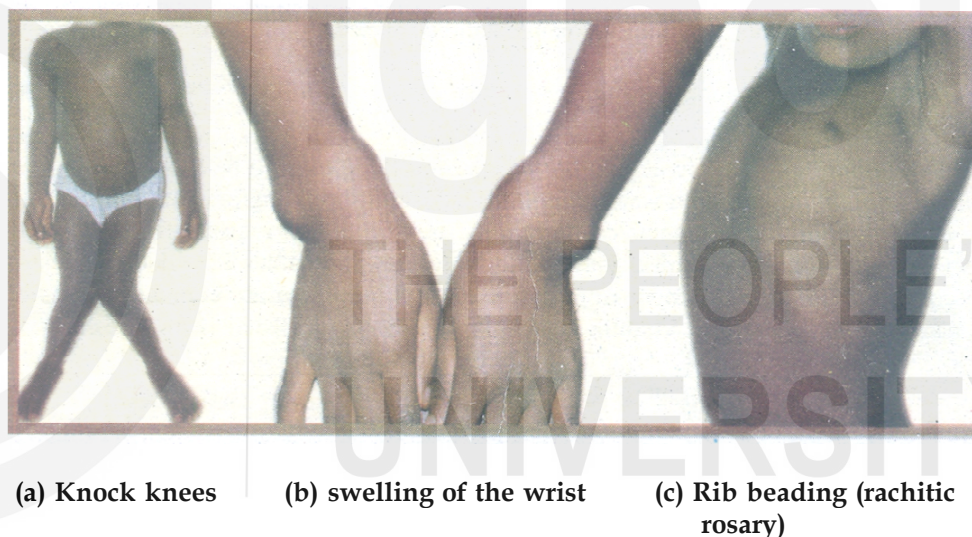


Figure 4.5: Manifestations of vitamin D deficiency

In adults, osteomalacia manifests due to undermineralization and excessive bone loss, resulting in the extreme cases, fractures. Osteomalacia occurs due to calcium depletion in women of childbearing age due to multiple pregnancies and particularly among those observing *purdah*, like in Muslim communities. The women may complain of pain in bones of lower extremities, back ache and difficulty in walking. Due to softness of bones, the bones become soft and can easily bend leading to bony deformities. Muscular hypotonia (low muscle tone), tetany and convulsions due to hypocalcemia occurs. Let us see what causes it.

Causes

It is due to inadequacy of vitamin D, considered as a prohormone. It gives rise to the hormone 1, 25-dihydroxy D₃, the primary function of which is to regulate serum calcium. One of the main causes is inadequate exposure to sun. Infants who are solely breastfed and not exposed to adequate sunlight and premature infants are more prone to rickets. Osteomalacia can occur due to some gastrointestinal disturbances and chronic renal diseases where there may be impairment of absorption of calcium and synthesis of vitamin D.

Treatment

Treatment of both rickets and osteomalacia requires administration of vitamin D and ensuring adequate calcium intake. Let us know about the prevention.

Prevention

Adequate exposure to sunlight is absolutely essential for prevention of rickets and osteomalacia. Simultaneously, it should be ensured that the diets provide adequate amounts of calcium daily. The awareness among the communities should be increased so that the diets contain foods, which provide calcium. Among the foods, milk is the best food.

We will now learn about fluorosis and lathyrism in the next section. But first let us recall what we have learnt so far.

Check Your Progress Exercise 1

1. Match the following:

Column A

Vitamins

1. Thiamine (B₁)
2. Riboflavin (B₂)
3. Niacin
4. Folic Acid
5. Ascorbic acid
6. Vitamin D

Column B

Clinical Deficiency

- a) Rickets/Osteomalacia
- b) Scurvy
- c) Beriberi
- d) Megaloblastic anaemia
- e) Pellagra
- f) Ariboflavinosis

2. List causes of:

- a. Thiamine deficiency

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.....

- b. Niacin deficiency

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3. How can we prevent:

- a. Folic acid deficiency

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- b. Vitamin D deficiency

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4.3 FLUOROSIS

Fluorosis, a chronic public health problem, is caused by excess intake of fluoride through drinking water or food products or industrial pollutants over a long period.

Fluorosis is endemic in several parts of India like in the States of Uttar Pradesh, Madhya Pradesh, Bihar. An estimated 11.5 million people are at risk and 62 million people in India suffer from dental skeletal and non-skeletal fluorosis. (NPPCF, 2016). What is fluorosis? *It is a crippling and painful bone disease caused due to consumption of excessive fluoride in water.* According to scientific surveys, skeletal fluorosis in India occurs when the fluoride concentration in water exceeds 1 part per million (ppm), and has been found to occur in communities with only 0.7 part per million. Interesting isn't it! Let us look at the manifestations of fluorosis.

Manifestations

Fluorosis manifests mainly as *dental fluorosis, skeletal and non-skeletal fluorosis*. A review of these manifestations follows.

Dental Fluorosis: It occurs in children of both the sexes exposed to high fluoride intake even before the dental mineralization is complete. It is characterized by mottling of teeth, which appears as yellowish or brown streaks or spots, as can be seen in Figure 4.6, particularly evident on the incisors. Sometimes, pitting (deeper depressions) may occur on these teeth. Dental mottling is irreversible and is confined to permanent teeth.



Figure 4.6: Dental Fluorosis

Skeletal Fluorosis: This is a bone disease caused by an excessive consumption of fluoride. This is a slowly progressing condition and is not as clinically obvious as dental fluorosis. The clinical features include joint pains, musculoskeletal dysfunction, restricted mobility of spine and deformities of flexion type. The disorder starts with vague nonspecific symptoms like pain in the joints, followed by stiffness and restriction in the movements of spine. In the later stages there may be spinal deformities with vertebral column becoming rigid with inability even to bend. In the recent past, severe deformities known as *genu valgum*, an adult form of exaggerated knock-knees has been described in endemic fluorotic areas. Experts suggest that crippling skeletal fluorosis might occur in people who have ingested 10-20 mg of fluoride per day for 10-20 years isn't that alarming.

Non-Skeletal Fluorosis is an earlier manifestation of fluorosis seen as a gastrointestinal complaint etc. and may overlap with other diseases leading to misdiagnosis.

So, it is clear that fluorosis can affect young and old, men and women alike. Let us then learn what causes fluorosis?

Causes

It is mainly due to very high content of fluorides in drinking water. Foods also contribute significantly to fluoride content of the diet. Cereals and vegetables grown

in areas, which are endemic for fluorosis, contain higher amounts of fluoride. It is reported that as much as 85% of the total fluoride intake is contributed by food. Poor socioeconomic conditions and poor nutritional status may be associated with fluorosis. Remember, fluoride can enter the body through drinking water, food, toothpaste, mouth rinses and other dental products; drugs, and fluoride dust and fumes from industries using fluoride containing salt and or hydrofluoric acid.

Let us now learn how we can prevent fluorosis.

Prevention

Ensuring that the drinking water has safe levels of (below 1 ppm) fluoride is the best solution for controlling endemic fluorosis. Supply of water from rivers, dams, canals and other sources of surface water is one of the methods adopted extensively in areas, which are highly endemic. Another method is *defluoridation* (removal of fluoride) of water by appropriate treatment of water. Several domestic methods are suggested of which 'Nalgonda technique' and 'Prashanti Technique' are perhaps simple and acceptable. While in the first method, lime and alum are added to water, in the latter, activated alumina is used for passing water. Commercial defluoridation is very expensive and is not practiced frequently. Education of communities to avoid use of fluoride rich toothpastes, pesticides and fertilizers is important.

Finally, we move on to lathyrism.

4.4 LATHYRISM

What is lathyrism? You may recall studying about lathyrism in the Food Microbiology and Safety Course (MFN-003) in Unit 7. Certain foods of *Leguminaceae* family contain toxic amino acids, which pose serious health problems to mankind. Of them, *Lathyrus sativus* (*Kesari dhal*) could be considered to be of public health significance, in view of the serious crippling consequences due to continued consumption of the pulse. The disease attributed to the consumption of this food is referred to as lathyrism. Lathyrism, causes upper motor neuron degenerative disease, leading to paralysis. Let us elaborate on symptoms of lathyrism.

Manifestations

The disease, seen among young adults in their most productive age, is insidious in nature. It is characterized by altered gait, severe pain in the lumbar region of the back, spasticity and paralysis. The earliest symptom is muscular spasms in the calf. This is followed by stiffness and heaviness in limbs, muscular cramps, involuntary tremors and ultimately typical stiff legged scissors gait. In the initial stages the affected may be able to walk with the help of a single stick, which progresses to two-stick stage and finally to crawling stage.

Let us learn what causes it.

Causes

The disease is caused due to exclusive consumption of *kesari dhal* over a long period of time. The pulse contains a toxic amino acid known as *beta-oxalyl amino alanine* or *BOAA*, which is a neurotoxin. In parts of Madhya Pradesh and contiguous areas of Uttar Pradesh and Bihar, the labourers receive *kesari dhal* as wages, particularly during drought seasons. *L. sativus*, which is grown as a mixed crop along with wheat, being a hardy, survives despite damage to wheat crop. As a result, the labourers solely depend on *kesari dhal rotis* for their survival, ultimately suffering from the crippling condition. Let us see how we can prevent it.

Prevention

In most of India, gradually, cultivation of *kesari dhal* crops has declined over time because of ban on its sale under Prevention of Food Adulteration Act. In addition,

distribution of cereals at affordable rates through public distribution system has helped in total dependence of the labour on *kesari dhal*. In the long run, development of genetically modified low toxin levels of *L. sativus* would help not only in controlling lathyrism but also in improving the availability of pulses. The toxin, being water soluble, can be removed by parboiling. At the domestic level, steeping of the pulse in boiled water and drying the same, removes most of the toxin. Education of the communities to adopt such simple household methods would help in the control of the irreversible paralytic condition.

With this, we end our study of the nutritional problems which are of concern and can be of concern to us. Do answer the questions given here under the check your progress exercise 2 and check your understanding about the nutritional problems learnt in the last section.

Check Your Progress Exercise 2

1. List key manifestations and causes of fluorosis.

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2. How can we prevent fluorosis?

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3. What is the cause of lathyrism?

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4. List the three stages of lathyrism.

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4.5 LET US SUM UP

In this unit we learnt that there are other nutritional problems which may occur in large number of people in developing countries either due to poor diet or other environmental conditions. These are deficiency of some B-complex vitamins like thiamine, riboflavin, niacin, folic acid and B₁₂ as well as deficiency of vitamin C and vitamin D. Fluorosis and lathyrism although not vitamin deficiency, cause many complications in the human body. Next, we studied that most of Vitamin B- complex deficiencies and Vitamin C deficiencies are due to faulty cooking practices, malabsorption, prolonged use of drugs, alcoholism and increased demands due to physiological changes in the body. Another nutritional disorder is fluorosis which is endemic in several parts of India like in the States of Andhra Pradesh, Gujarat, Karnataka, Maharashtra, Punjab, Rajasthan and Tamil Nadu. It is due to consumption

of excessive fluoride in water. Finally, we read about Lathyrism. Lathyrism is a disease which is caused due to exclusive consumption of *kesari dhal* over a long period. It can lead to paralysis. In India, there is a ban on the sale of *kesari dhal* under Prevention of Food Adulteration Act.

4.6 GLOSSARY

Cyanosis	: bluish discolouration of skin due to the presence of oxygen-deficit blood.
Delirium	: disordered state of mind involving incoherent speech and excitement.
Hyperaesthesia	: a state of enalted or morbidity increased sensibility of the body or a part of it.
Neurotoxin	: any poison that acts on nervous system.
Palpitations	: rapid strong or irregular heart beat.

4.7 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress Exercise 1

- 1 c, 2 f, 3 e, 4 d, 5 b, 6 a
- Thiamine deficiency is caused when people consume highly polished rice and adopt improper cooking practices such as throwing away the excess water after cooking the rice.
 - Niacin deficiency is generally found in poorer communities where people use either maize or jowar as staple food. Pellagra is also observed in alcoholics and those suffering from malabsorption.
- Folic acid deficiency can be prevented by supplementing the diet with folic acid along with iron tablets. This is one of the strategies being adopted by the governments to prevent and control anaemia due to folic acid deficiency.
 - Vitamin D deficiency can be prevented through adequate exposure to sun light daily and by providing recommended amount of calcium in diet daily.

Check Your Progress Exercise 2

- Fluorosis manifests mainly as dental fluorosis and skeletal fluorosis. It is caused mainly due to very high content of fluoride in drinking water. Foods also contribute significantly to fluoride content of the diet.
- Fluorosis can be prevented by ensuring that the drinking water has safe levels of below 1 ppm fluoride.
- Lathyrism is a disease which is caused due to exclusive consumption of *Kesari dhal* over a long period.
- Three stages of lathyrism are: i) Able to walk with the help of one stick, ii) Only possible to stand and walk with two sticks, and iii) Paralytic crawling stage.