21 Vitamins

Summary

Vitamins are organic compounds essential for normal growth and metabolic processes. They cannot be synthesized by humans in adequate amounts and, therefore, must be obtained from the food eaten. Vitamins A, D, E, and K are fat-soluble and are present in animal and plant fats and oils. They can be stored in body tissues, and if excessive amounts are consumed toxicity can occur. The water-soluble vitamins, vitamin C and vitamins of the B complex, are normally excreted in the urine if excessive amounts are consumed. A single vitamin deficiency seldom occurs in isolation, normally a multi-vitamin deficiency occurs. Vitamin supplementation is really not necessary for individuals who consume a wide variety of foods.

Introduction

A vitamin is an organic compound that occurs in small concentrations in food. Each of the 13 known vitamins have such specific and vital functions in the cell and tissues of the body that one vitamin cannot replace, or act for, another. The lack or deficiency of one vitamin can also interfere with the function of another and consumption of excessive amounts of one vitamin can lead to vitamin imbalances.

The body needs only small amounts, measured in micrograms or milligrams each day, of each vitamin to sustain the normal metabolic processes of life. Vitamins cannot be synthesised by the body (except vitamin D, which can be made in the skin upon adequate exposure to sunlight) and the continued lack of one vitamin in the diet, or it's improper absorption or utilisation by the body, results in a vitamin deficiency disease. Bacteria in the human intestine can synthesise certain vitamins but usually not in quantities large enough to meet the body's needs, with the exception of vitamin K; bacteria, however, represent an exogenous or 'outside source', rather than an endogenous source. An organic compound must be required in the diet to prevent a deficiency disease (such as scurvy, rickets or beriberi) in

order to be considered as a vitamin. In fact, vitamins were discovered while searching for the cause of such diseases.

Frank vitamin deficiency diseases are rare in Australia today. Primary deficiency states, due to dietary inadequacies, are probably less common than secondary deficiency states, which may be induced by some other factor or disease in the face of an apparently adequate dietary intake. The possibility of an excessive and potentially harmful intake of certain vitamins has also been identified with the consumption of large doses of certain vitamins by some individuals—such a condition is referred to as hypervitaminosis*. Healthy individuals usually meet their vitamin requirement by eating a varied diet. The extent to which the diet meets the average person's needs is often measured against the recommended dietary intakes (see chapter 26, food composition tables and dietary allowances) prepared by the Nutrition Committee of the NH & MRC (National Health and Medical Research Council) in Australia.

Hypervitaminosis: a toxic condition that occurs as a result of the excessive consumption of quantities of certain vitamins.

Vitamin classification

Vitamins are usually classified according to their solubility in either fat or water. The four vitamins A, D, E and K are soluble in fat, whereas vitamin C and B-complex vitamins are water-soluble. They may also be classified according to their function in the body, for example: some of the B-complex vitamins (vitamins B_1, B_2, niacin, B_6, pantothenic acid and biotin) function as co-enzymes* in many and varied metabolic reactions involving the release of energy for cellular activity; vitamin B_{12} and folate are involved in the synthesis of the gene-containing material of the cells, deoxyribonucleic acid (DNA).

Co-enzyme: a small molecule that combines with a particular protein molecule to make an enzyme.

Enzyme: a protein molecule that acts as a catalyst to facilitate chemical reactions.

As vitamins were discovered they were designated by letters assigned in alphabetical order. After they had been chemically identified, it became apparent that the vitamins were not single compounds but were mixtures of compounds. For example, several different compounds all with vitamin A activity have been isolated, and in the case of vitamin B, numerical subscripts were added to distinguish between individual vitamins as they were discovered (vitamin B_1, B_2, B_6 etc.).

Many vitamins exist in food in several different forms, some of which (precursors* and provitamins*) require conversion into vitamin active compounds in the body before they can function. Some vitamins may not be completely available for absorption from the human gut and therefore have reduced bioavailability. The term biological activity, when applied to a nutrient, takes into account factors that may affect its absorption and utilisation and it is therefore a measure of the nutritional effectiveness of the particular nutrient.

Precursor: a compound that can be converted to a nutrient in the body.

Provitamins: chemical compound closely related to vitamins in their structure. In the body the inactive provitamin is converted to the active form of the vitamin, e.g. β-carotene is a provitamin of vitamin A.

Fat-soluble vitamins

Fat soluble vitamins are present in a wide variety of foods. The mechanism of digestion and absorption follows a similar pathway to the dietary fats and any condition that hinders the function of the intestine or interferes in any way with fat absorption (such as the malabsorption syndrome*) will also limit the absorption of fat soluble vitamins. They can be stored, mainly in the liver, and consequently the clinical symptoms of deficiency develop more slowly than for water-soluble vitamins which are rapidly excreted or metabolised but which may also have effective long-term storage mechanisms. Some fat-soluble vitamins, if taken in excessive amounts, accumulate in the body and may produce undesirable toxic effects. In contrast to water soluble vitamins, fat soluble vitamins are not easily destroyed by usual cooking methods and they do not dissolve into the cooking water.

Malabsorption syndrome: a condition in which there is faulty absorption of nutrients.

Vitamin A (retinol)

Vitamin A is present in food in two main forms: as preformed vitamin A (the vitamin itself); and more commonly as a precursor of vitamin A, namely provitamin A (the carotenes and similar substances, the carotenoids). Vitamin A is the generic description for at least 7 different active forms of the vitamin. It is found almost exclusively in animal sources, usually in association with fats in such foods as dairy products and liver. Most of the vitamin A in a mixed diet is supplied in the form of carotenoids the most commonly present being α, β and γ carotene. These substances are found mainly in deep yellow and green coloured plants. The body can convert a considerable proportion of this carotene in food into vitamin A.

Beta carotene has the highest vitamin A activity of the carotenoids (and is the most abundant in food), but it is less efficiently utilised by the body than is preformed vitamin A (also called retinol). β-carotene is estimated to have only one-sixth of the biological activity (or usefulness) of a microgram of retinol. This difference is partly attributable to the poor absorption of carotenes. Vitamin A activity used to be measured in International Units (I.U.)* but nowadays Retinol Equivalent (R.E.)* are used because of the need to take into account not only the absorption of carotenoids but also the degree of conversion to vitamin A. The equivalents are listed in table 21.1.

Clinical deficiency of vitamin A is virtually never seen in Australia, probably because vitamin A is retained in the body more extensively than most other vitamins and the average amount stored (mostly in the liver) can satisfy the normal requirement for between one and two years. Toxicity from excessive and prolonged intakes of vitamin A, usually in the form of supplements, is rare but has been reported both in adults and children owing to the mistaken belief that large

International Units: the measure traditionally used for vitamins A and D. The amount of the vitamin comprising a unit was determined by its biological activity in rats, i.e. the amount required to cure or prevent the specific vitamin deficiency disease.

Retinol equivalents: a measure of vitamin A activity that has replaced I.U., and accounts for dietary variances in retinol and its precursor, carotene. To calculate the retinol equivalents in a diet or food item, the following formula is used:

$$\mu g \text{ retinol} + \frac{\mu g \text{ β-catorene}}{6} + \frac{\mu g \text{ other caroteniods}}{12} = \text{retinol equivalents}$$

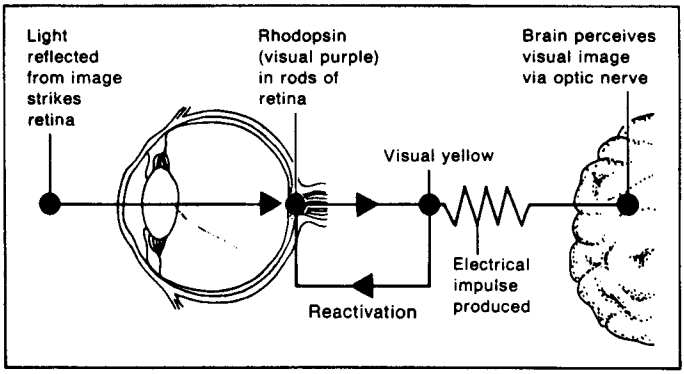

Figure 21.1 The function of retinol in black and white (dim light) vision is as a component of rhodopsin (visual purple) which is bleached to visual yellow by contact with light arriving at the retina. This reaction triggers an electrical impulse that is conducted through the optic nerve to the brain; this signal is recognised as part of a visual image. Rhodopsin can be regenerated so that the process can be repeated.

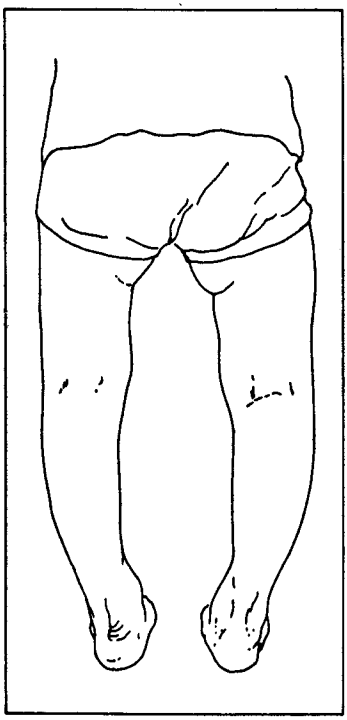

Figure 21.2 Bow legs characteristic of rickets.

Table 21.1 Conversion factors for retinol equivalents

1 retinol equivalent	= 1 microgram retinol
	= 6 micrograms β-carotene
	= 12 micrograms other provitamin A carotenoids
	= 3.33 I.U. vitamin activity from retinol
	= 10 I.U. vitamin activity from β-carotene

amounts are beneficial and safe. Vitamin A taken in excessive and repeated doses can be hazardous—especially during pregnancy. (See table 21.2 and figure 21.1.)

Vitamin D

Of the several forms of vitamin D that exist in nature, the two most important to man are vitamin D_2 (ergocalciferol), and vitamin D_3* (cholecalciferol). Both vitamins D_2 and D_3 are formed by the ultraviolet irradiation of two provitamins: provitamin D_2 (ergosterol), found in yeasts and fungi, and provitamin D_3 (7-dehydrocholesterol), found in the skin. Both forms seem equally effective in man. Only a few natural food sources of vitamin D are known to exist—small and often insignificant amounts are found in fatty fish, butter, eggs and fortified foods such as margarine. Australians, receiving adequate exposure to sunlight, do not need additional dietary vitamin D. The amount of vitamin D_3 synthesis in skin is determined by the length and intensity of exposure to sunlight and the amount of melanin (color pigment) in the skin. There is a risk of vitamin D deficiency in infants living in cold climates, especially during the winter months. This can cause rickets (figure 21.2). The elderly and those institutionalised for long periods are also at risk. (See figure 21.3 and table 21.3.)

Vitamin D_3: could be classified as a hormone rather than as a vitamin because although it is an essential nutrient for humans, it can be produced by the body.

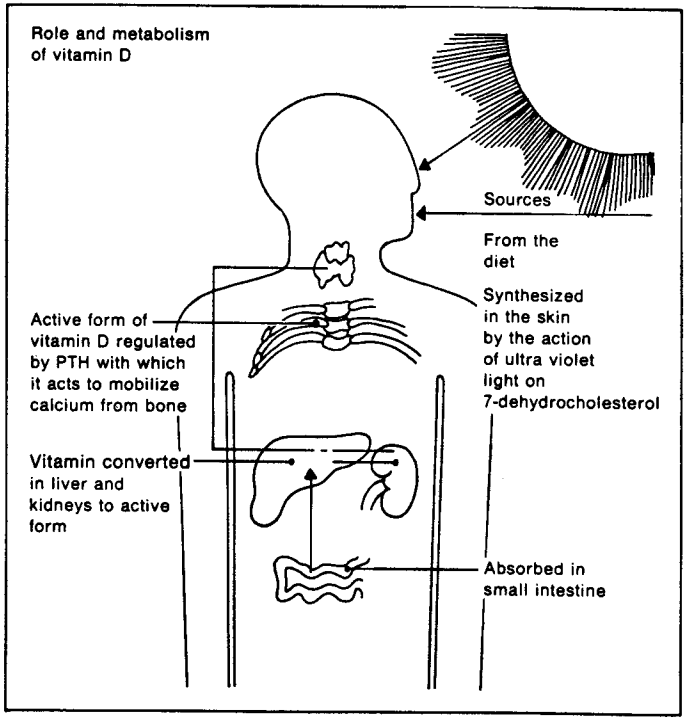

Figure 21.3 The role and metabolism of vitamin D are complex processes. The vitamin may be available in food or formed in the skin by the action of sunlight. It acts in conjunction with parathyroid hormone (PTH) to control calcium and phosphorus balance. The activated vitamin has hormone-like properties and acts on the intestine and kidney as well as on bone.

Vitamin E

Vitamin E exists in at least eight different tocopherol* and tocotrienol* forms. The compound d-alpha tocopherol is the most widely distributed and has the highest biological activity. The fundamental characteristic of vitamin E is as an antitoxidant (i.e. preventing or inhibiting oxidation).

The richest dietary sources of vitamin E are the commercially available vegetable oils—these are also the richest sources of polyunsaturated fatty acids (PUFA), which vitamin E protects from oxidative breakdown. Nuts are the next best source, while almost all vegetables and meats have small amounts.

The requirement for vitamin E in the body is difficult to estimate since it is influenced by the amount and type of fat eaten in the diet; that is, a high PUFA intake increases the vitamin E requirement. However, since foods that are good sources of PUFA are also generally good sources of vitamin E, the intake of the vitamin automatically increases as the intake of unsaturated oils increases.

It seems almost impossible to induce a vitamin E deficiency through the consumption of a vitamin E deficient diet. However, evidence of vitamin E deficiency is sometimes seen in intestinal malabsorption syndromes and in low birth weight infants whose blood levels are low for the first month or so of life. However, because these conditions are rare it is assumed that under normal circumstances enough vitamin E

Tocopherol: an oily liquid compound of the vitamin E group, found in seeds and fish oils.

Tocotrienol: the structure of vitamin E consists of two carbon rings joined together and a carbon side-chain. The side-chain of the tocotrienol is different to that of the tocopherol.

One way of calculating the total vitamin E activity of a varied diet is to multiply the alpha-tocopherol value (in milligrams) by 1.2 (this accounts for the other tocopherols present). This calculation gives an approximation of total vitamin E activity expressed as 'milligrams of alpha-tocopherol equivalents', but is not a complete statement about the vitamin E content.

is supplied by the normal diet and that there is no need for a supplement. (See table 21.4.)

Vitamin K

Two forms of vitamin K occur naturally: vitamin K_1 or phylloquinone which is found in green plants, and vitamin K_2 or menoquinone which is synthesised by the intestinal bacteria and is also found in small amounts in animal tissue. A third form of the vitamin, vitamin K_3, menadione is a synthetic product.

It has been estimated that approximately half of the vitamin K needed by the body can be obtained from the bacteria inhabiting the gut, whilst the remainder can be obtained from the diet. Vitamin K is widely distributed in food—good sources include green leafy vegetables, soya beans and wheat bran. Fruit and most animal products contain little vitamin K.

There is no recommended dietary intake of vitamin K in Australia, it is difficult to establish the amount required from food since the amount produced by gut bacteria is likely to vary. Deficiency is extremely rare, however, except under the conditions indicated in table 21.5. An adequate vitamin K status is probably ensured both because the intestinal bacteria constantly produce a supply and the amount the body needs is very small. A diet consisting of a wide variety of foods would provide approximately 300 to 500 μg of vitamin K daily, and this compares well with the estimated safe and adequate range of uptake which is from 70–140 μg for men and women.

Water-soluble vitamins

Thiamin (vitamin B_1)

Thiamin (vitamin B_1) is widely distributed in a large variety of animal and vegetable sources, but is present only in relatively small amounts in most of them. Yeast and Vegemite are the richest food sources, but are usually eaten in small amounts. Because refining cereal products removes much of the thiamin content, restoration of thiamin to bread and cereals is permitted in Australia to ensure that daily requirements are met.

Since thiamin is a water-soluble vitamin, storage in the body is limited and this leads, therefore, to a constant requirement for the vitamin; compared with fat-soluble vitamins, only short periods of deprivation will lead to deficiency. The principal role of thiamin in the body is as a co-enzyme in reactions that release energy from carbohydrates and trap it in the energy-laden compound, ATP*. The daily thiamin requirement, therefore, is proportional to the amount of energy consumed (particularly from carbohydrate sources). The recommended daily intake is usually expressed in terms of energy intake, i.e. 0.1 mg/1000 kJ for

ATP (adenosine triphosphate): the energy storehouse of the body.

all age groups. There are bacteria in the large intestine with the ability to synthesise thiamin but its absorption into the body is minimal.

The group most likely to develop a thiamin deficiency in Australia is chronic alcoholics (a regular daily intake of at least 40 g alcohol) who eat little or no food for extended periods of time. (See table 21.6.)

Riboflavin

Riboflavin is found in small amounts in a wide variety of animal and vegetable food sources; the most important in the Australian diet are milk and dairy products. Other good sources include breakfast cereals, organ meats and nuts.

Riboflavin, once absorbed from the small intestine must be converted (by the addition of phosphate) into its active form. It is a constituent of all metabolising cells but, as with thiamin, there is limited storage in the body; excess riboflavin is excreted by the kidneys in the urine, a possible explanation for its low toxicity. (See figures 21.4, 21.5 and table 21.7).

Niacin

Two forms of this vitamin have been identified—niacin (nicotinic acid) and niacinamide (nicotinamide). The best dietary sources of niacin are foods rich in protein, such as organ and muscle meats, poultry, legumes and peanuts. Niacin can also be made in the body from tryptophan, an amino acid obtained from animal and some vegetable protein sources. It has been estimated that approximately 60 mg of the precursor tryptophan is needed to produce 1 mg of niacin in the body. This relationship between niacin and tryptophan has been taken into account in the development of the unit known as the niacin equivalent. The niacin equivalent of a diet is calculated as follows:

Dietary niacin + (0.16 × dietary protein in grams) expressed as milligrams = Niacin equivalent (mg).

The roles of niacin and riboflavin in cell metabolism are closely related and clinical evidence of both deficiencies is often seen in the same individual. (See table 21.8.)

Vitamin B_6

Vitamin B_6 occurs naturally in food in three forms, pyridoxine, pyridoxal and pyridoxamine and it is widely distributed in a variety of animal and plant food sources: legumes, potatoes and bananas are among the best sources of pyridoxine; pork and organ meats are the richest sources of pyridoxal and pyridoxamine.

The amount of vitamin B_6 required is small, and deficiency is rare. Symptoms of deficiency resemble those of pellagra and can lead to niacin deficiency—since vitamin B_6 is required for both the conversion of tryptophan to niacin (as well as for folate metabolism).

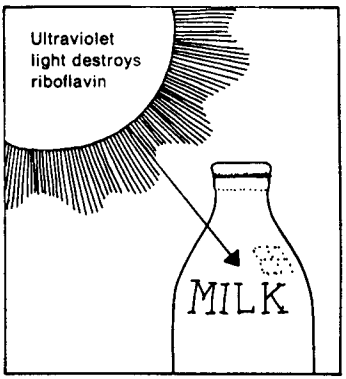

Figure 21.4 Riboflavin is one of the B-vitamins that is susceptible to destruction by the action of the ultra-violet rays in sunlight. Milk left in sunlight may lose a significant proportion of its riboflavin content.

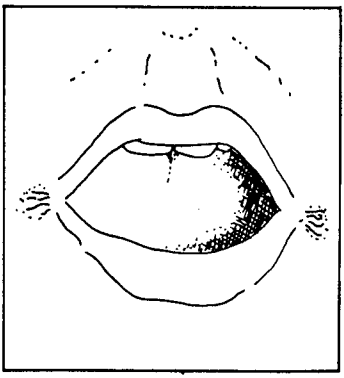

Figure 21.5 Angular stomatitis caused by riboflavin deficiency.

As vitamin B_6 is involved in protein metabolism, the need for it varies with protein intake—consumption of a high protein diet increases the need for vitamin B_6. The present recommendation for Australian adults allows for an intake of 100 grams or more of protein daily.

It is not safe to consume large amounts of this vitamin. The recommended upper limit in Australia should probably not exceed 10 mg daily on a regular basis. (See table 21.9.)

Pantothenic acid

Pantothenic acid (or pantothenate) is very widely distributed in food; liver, kidney and eggs (especially the yolk) are among the best sources. Because of this dietary deficiency of panthothenic acid has not been reported in humans, and only experimental deficiency, using a vitamin antagonist, has been described. A recommendation for dietary intake of pantothenic acid has therefore not been made in Australia and an average varied diet of 19 MJ/day, which is estimated to contain between 10–20 mg of pantothenic acid, provides the 5–10 mg which is regarded as being adequate for most children and adults.

Intestinal bacteria synthesise this vitamin but the availability to the body from this source is unknown. (See table 21.10.)

Biotin

There are eight isomers of biotin, but only one has vitamin activity. It is present in a variety of foods and good sources include egg yolks, brewers yeast, soya beans and liver; lesser amounts occur in meat, fruit and vegetables. Significant amounts of biotin are produced by intestinal bacteria which makes the dietary requirement uncertain. An average, varied diet is likely to contain between 50 and 300 mg of biotin per day, and appears to meet the needs of most healthy adults. (See table 21.11.)

Folacin (folic acid)

Folacin is the generic name for many compounds that exhibit the biological activity of folic acid (pteroylglutamic acid or PGA). Folacin is a provitamin which must be converted to its biologically active or enzyme form by the body—folic acid is one of the active forms. The different forms of folacin vary widely in biological activity, stability and availability to the body.

The recommended intake for folacin in Australia is now based on *total* folacin—this includes other forms combined with the amino acid, glutamic acid. Small amounts of folacin are synthesised by bacteria in the human intestine.

Folacin occurs widely in a variety of foods including leafy vegetables, liver, citrus fruit and nuts which are particularly good sources. Studies suggest that between 25 and 50% of folacin is absorbed from a diet containing a variety of foods. (See figure 21.6 and table 21.12.)

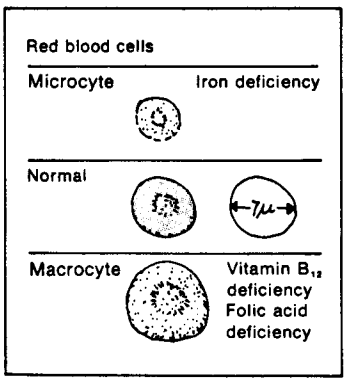

Figure 21.6 Folic acid deficiency.

Vitamin B_{12}

There are three predominant forms of vitamin B_{12} in food, methylcobalamin, adenosylcobalanum and hydroxycobalamin. Vitamin B_{12} is supplied almost entirely by animal foods—organ meats, eggs, seafood, dairy products and fermented foods being excellent sources. The occurrence of B_{12} in nature is the result of microbial synthesis but the amount available from the intestinal bacteria of man is not known.

Intestinal absorption of B_{12} requires the presence of a molecule called intrinsic factor which is secreted by the stomach and facilitates the transfer of vitamin B_{12} into the cells lining the ileum. Calcium is also thought to be necessary for this transfer.

The body has a good storage capacity for vitamin B_{12} as well as a very efficient method of recycling the vitamin so that the amount needed for normal metabolism appears to be small. The onset of deficiency symptoms due to depletion may be delayed by up to 10 years and is rarely seen within 2 years. (See table 21.13.)

Ascorbic acid (vitamin C)

Vitamin C exists in two forms, L-ascorbic acid (most of the vitamin exists in this form) and L-dehydroascorbic acid. The best sources of vitamin C include readily available fruits and vegetables. Considerable amounts of the vitamin are also added to our food supply in the preservation of food and in the process of nutrient restoration, for example ascorbic acid may be added to processed meats, flour and beverages such as fruit juices, drinks and cordials.

Ascorbic acid is easily destroyed by high temperatures and exposure to air, drying, alkali and prolonged storage. It is very soluble in water and losses during the preparation and cooking of fruit and vegetables can be considerable.

Clinical scurvy is a rare disease in this country and in most developed countries today. Subclinical deficiency represents a state of 'nutritional risk' but whether it impairs overall health is uncertain.

The controversy involving the usage of high doses of vitamin C, up to 1 gram or more per day, in the prevention and cure of the common cold remains unresolved. However, many of the original claims of beneficial effects of large doses of vitamin C have not been fully substantiated and since high dosages can have adverse effects, recommendations to the general public to increase intake of vitamin C to gram amounts is unjustified on the basis of the available evidence. (See table 21.14.)

Other vitamin-like substances

There are some substances, for example lipoic acid, choline, and inositol, which have vitamin-like functions, or which

enhance the activity of a vitamin. However, these substances do not meet the full criteria for the definition of a vitamin and at present are not considered to be vitamins.

Lipoic acid

Lipoic acid is a sulphur containing fatty acid. It is an important constituent of co-enzymes involved in biological oxidation and reduction, and is therefore essential for protein, fat and carbohydrate metabolism. Lipoic acid is essential for the growth of some micro-organisms, but it has not been demonstrated to be essential for growth in either animals or humans. Yeast and liver are sources of lipoic acid.

Choline

Choline plays a role in fat metabolism and prevents the accumulation of fat in the liver: because of this it is often referred to as a 'lipotropic factor'. Choline can be synthesised endogenously and no deficiency has been identified in humans. Choline is present as lecithin in a variety of foods including egg yolk, fish, cereal products and legumes.

Inositol

Brain, heart and skeletal muscles contain large amounts of inositol. The exact role of inositol is not known, but it is suggested that it is essential for growth of cells in tissue cultures. Glucose is a precursor of inositol and evidently enough can be synthesised from glucose to meet the metabolic needs of humans. Food sources include meats, milk and milk products, wholegrain cereals (present as phytic acid; see chapter 17, Dietary fibre), nuts, fruits and vegetables.

Carnitine

This was originally found to be essential for the growth of mealworms. It facilitates the transport of fatty acids within cells to sites where they are oxidised.

Vitamin supplementation in Australia

The idea that vitamins are endowed with miraculous properties still persists, despite all that is now known about their function. This misunderstanding, together with the belief that it is impossible to obtain all the vitamins we need from our food supply, has prompted many Australians to supplement their diets with additional vitamins.

This trend has increased significantly in recent years. In a survey carried out by the NHF in 1977–1978, 19% of respondents supplemented their diet with added vitamins. Results from the Australian Health Survey in 1980 reported this figure to be 12%, but in a recent South Australian survey (Worsley and Crawford, 1984) 37% of men and 53% of women had taken a nutrient supplement during the month preceding the survey. Of the vitamin supplements taken the most popular were multivitamins (16%) followed

by the B complex vitamins (11%) and vitamin C (10%). 'Supplement takers were endeavouring to prevent or ameliorate minor symptoms such as those arising from colds, stress, depression or hangovers as well as seeking more nebulous goals such as the general maintenance of health'.

The prevalence of group or family use suggested that there was considerable prophylactic rather than therapeutic use, with the consumption of vitamin supplements as a type of 'nutritional insurance'. Since most consumers are not aware of their vitamin status nor would they really know the amount of vitamins present in their diet they may be using vitamin supplements just to be sure. Vitamin supplementation for the prevention of diseases unrelated to vitamin deficiency also needs careful evaluation. If a person who takes supplements does not develop a certain disease he may attribute his good luck to the supplement, but this is far from proof of their efficacy.

In Australia, with its abundant food supply, frank vitamin deficiency diseases are seldom seen and when they do occur can be traced to a deficiency usually induced by poverty, ignorance or indifference about food selection, adaptation of bizarre eating habits or some disease condition. Vitamin deficiency as a result of poor eating habits is obviously best treated by correcting the diet to include food which contains the vitamin that is in low supply. Deficiencies arising from a disease condition should be diagnosed and evaluated by a qualified physician who will initiate treatment on an individual basis and may prescribe supplementary vitamins during the treatment of the precipitating disease. Hazards associated with the self treatment of diseases that require prompt medical attention should be evident.

If, however, 'nutrition insurance' is sought, then consult the following checklist before taking a vitamin supplement:

1. is there a clear indication of deficiency?;
2. try and correct any deficiency with an improved diet first;
3. use supplements in amounts as close to the RDI as possible; and
4. take supplements for the shortest possible time.

Promoters of vitamin supplements help to maintain the notion that it is difficult, if not impossible, to obtain all the vitamins we need from our current food supply alone. The 100 million dollars spent by Australians annually suggests that advertising to this effect has been particularly successful. Until vitamin supplement labelling is improved to reflect ingredients and the RDI, and until standards are uniform and adequate and advertising is accurate and honest, this situation is likely to continue.

The toxicity arising from the consumption of large amounts of certain vitamins (symptoms of which are well documented in tables 21.2 to 21.14) can be as hazardous as a deficiency. Vitamins, when consumed in amounts far exceeding those found in food, assume the properties of medications or drugs, and as such need careful monitoring. In addition, the

consumption of excessive amounts of one vitamin could well cause a deficiency of another vitamin despite the adequate consumption of the second vitamin. For example, vitamin C taken in large doses may decrease the absorption of vitamin B_{12}. Evidence of the potential danger associated with megadoses (amounts of vitamins at least 10 times the RDI) of vitamins is growing and there is no scientifically sound evidence to support the unrestricted use of megadoses of vitamins for healthy individuals.

Further reading

WAHLQVIST, MARK, HUANG, SOO and WORSLEY, ANTHONY. *Use and Abuse of Vitamins: Food Versus Pills*. Second edition, Sun Books, Melbourne, 1987.
WAHLQVIST, M. L. 'Nutrient Supplementation in Australia'. *Medical Journal of Australia* 140, pp. 573–574, 1984.
WORSLEY, A. and CRAWFORD, D. 'Australian dietary supplementation practices: health and dietary supplements'. *Medical Journal of Australia* 140, pp. 579–583, 1984.

Questions

1. Calculate the niacin equivalent of a diet which, on analysis, contained 90 g of protein and 14 mg of niacin.
2. Referring to chapter 26, Food composition tables and dietary allowances, how much of each of the following foods would you need to consume to obtain two-thirds of the recommended dietary allowance for thiamin, riboflavin, niacin, ascorbic acid, vitamin A:
(a) grilled pork chop (b) baked potato
(c) braised cabbage (d) sauteed carrots.
3. Identify in your diet the foods that are the best sources of vitamin E, thiamin, biotin, pyridoxine, niacin and folacin.
4. For a person who refuses to eat fruit and vegetables, what foods could be substituted to provide a dietary intake of 30 mg vitamin C a day?
5. In the UK (Department of Health and Social Security) and the USA (Food and Nutrition Board, National Academy of Sciences), it is considered that a daily intake of 10 mg vitamin E is adequate and safe. In what amounts, and why, might supplements be obtained from pharmacies or health food shops?

Table 21.2 Vitamin A

FOOD SOURCE	*Retinol (μg/100 g)*	*Carotene (μg/100 g)*	*Notes*	*RDI*
High			1. Maximum of 30–40% loss in cooking. 2. Both retinol and carotene are sensitive to light and oxygen.	Adults (over 11 years) 750 μg R.E.
Cod liver oil	28 400	1050		
Palm oil	0	7000		
Lambs liver (fried)	20 600	10		
Medium				
Carrots	0	2000		
Spinach	0	1000		
Sweet potato*	0	670*		
Egg yolk	660	100		
Low				
Bread	0	0		
Potato*	0	Trace		
Chicken	Trace	Trace		

*amount varies with variety

FUNCTION	1. Vision. An essential component of rhodopsin (visual purple) which is present in the retina of the eye and is responsible for vision in dim light. 2. Epithelial surfaces. Essential for the maintenance of specialised epithelial surfaces of the body. 3. Bone growth. Essential for normal bone formation.
DEFICIENCY (Hypovitaminosis A)	Clinical signs include: 1. Night blindness. The inability to see in dim light is usually the first symptom. 2. Keratinisation of epithelial surfaces: normal secretory epithelium is replaced by dry, hard keratinised epithelium which is more susceptible to infection. When this process occurs in the cornea of the eye it can lead to Xerophthalmia and if deficiency continues it can lead to blindness. 3. Poor dental health can be the result of deficiency. Deficiency is rare in Australia but may occur secondary to: 1. Malabsorption syndromes, including cystic fibrosis and celiac disease. 2. Severe liver disease can result in inadequate reserves. 3. Zinc deficiency, which can occur in alcoholics, may reduce utilisation of Vitamin A. In Australia zinc deficiency rather than deficiency of vitamin A can cause night blindness.

Table 21.2 cont'd

TOXICITY (Hypervitaminosis A)	Chronic intakes in excess of 1000 μg/kg body mass, (approx. 100 × R.D.I.) can induce symptoms of toxicity in adults — less for children. Clinically characterised by: loss of appetite, headache, blurred vision, irritability hair loss, general drying and flaking of skin, bony growths or exotoses, haemorrhages, bone fractures, liver damage and death. Congenital abnormalities in offspring observed when hypervitaminosis occurs in pregnancy. Carotene in large amounts is not toxic but causes a yellowing of skin which disappears when carotene is distributed.

Table 21.3 Vitamin D

FOOD SOURCE	*Vitamin D (μg/100 g)*	*Notes*	*RDI*
High		1. Maximum of 40% loss in cooking. 2. Stable to heating, aging and storage.	Infants and young children — 400 I.U. (10μg). No dietary recommendation is made for normal healthy adults in Australia because their needs seem to be satisfied by the action of sunlight.
Cod liver oil	210		
Medium			
Fatty fish	5–25		
Margarine	8		
Low			
Egg yolk	5		
Butter	1		
Cheese	0.2		
Milk	0.01		

*In the U.S.A. a recommendation for vitamin D is given of 5 μg per day for both men and women.

FUNCTION	1. *Absorption of calcium and phosphorus* aids the absorption of calcium from the small intestine and bone and reabsorption from the kidney; increases the permeability of the cell to both calcium and phosphorus. 2. *Bone formation.* Increases the rate calcium and phosphorous form and maintain bone. (*Vitamin A and C are also involved in bone formation.)
DEFICIENCY (Hypovitaminosis D)	Clinical signs include: 1. *Rickets* in children. Mineralisation of bone matrix is impaired and collagen synthesis is defective (largely due to inadequate absorption of calcium and phosphate). When this condition occurs in the adult it is known as *osteomalacia.* Deficiency rarely occurs in Australia since exposure to sunlight is not a problem, but *infants, young children, the elderly or invalids* may be at risk if not exposed to sunlight, especially in the winter months. In adults deficiency is usually secondary to: 1. *Malabsorption syndromes.* 2. *Gastrectomy* — osteomalacia can occur as a complication of removal of the stomach. 3. *Diseases of the liver and kidney* — activation of vitamin D requires a functioning liver and kidney. 4. *Skin disorders.* 5. *Medications* — prolonged treatment with phenobarbitone (sedative) may induce deficiency.
TOXICITY (Hypervitaminosis D)	Excessive intake can be hazardous. Most reports of toxicity induced in children range from 25 000 to 60 000 I.U./day for 1 to 4 months. *Clinically characterised by:* loss of appetite, headache, nausea, vomiting, intense thirst and polyuria, calcification of soft tissues such as lung and kidney, bone disease and death.

FOOD SOURCE	*Vitamin E* (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum 55% loss in cooking.	Adults and children 8 years and over 7–10 mg/day.
Wheatgerm oil	140	2. Sensitive to heat, oxygen and decomposes in sunlight.	(A daily diet consisting of a wide variety of food would provide 8–11 mg of α-tocopherol equivalents. An alternative recommendation taking into consideration the polyunsaturated fat content of the diet is 0.4 mg vitamin E/gram polyunsaturated fat.
Polyunsaturated vegetable oils	20–80	3. Some loss during frezer storage.	
Medium		4. Presence retards spoilage of unsaturated fats and β-carotene.	
Peanut oil	15–20		
Olive oil	5		
Nuts	1–20		
Low			
Milk	0.02		

*total vitamin E activity estimated to be 80% α-tocopherol and 20% other tocopherols.

FUNCTION	1. *In the body:* (a) Acts as a tissue antioxidant protecting the lipid membrane of cells against oxidant damage. In the same way it preserves the integrity of red blood cells and protects β-carotene, and unsaturated fatty acids from oxidative breakdown. (b) May be involved in the formation of haemoglobin. 2. *In food.* It acts as an antioxidant by preventing the oxidation and breakdown of polyunsaturated fatty acids (PUFA). Effective synthetic antioxidants appear to eliminate the need for vitamin E as an antioxidant.
DEFICIENCY (Hypovitaminosis E)	There is the possibility of *haemolytic anaemia* in low birth-weight infants and in malabsorptive states. Central nervous system involvement. Deficiency has not been reported in Australian adults but may occur in: 1. *Low birth weight infants* given formula feeds low in vitamin E. They may develop a form of haemolytic anaemia associated with low blood levels of vitamin E. 2. Possibly in *Malabsorption syndromes* such as cystic fibrosis
TOXICITY (Hypervitaminosis E)	Relatively non toxic, though some adverse effects have been observed with daily intakes of 300 mg of synthetic α-tocopherol. Symptoms include: severe influenza, malaise, fatigue, and minor gut disturbances. An unbalanced ratio of vitamins E and K may lead to impairment of blood coagulation in humans.

Table 21.5 Vitamin K

FOOD SOURCE	*Vitamin K (μg/100 g)*	*Notes*	*RDI*
High		1. Maximum of 5% cooking loss. 2. Sensitive to light, oxygen and both acid and alkali conditions.	No recommendation for vitamin K intake has been made in Australia. Estimate of daily requirement is approximately 2 mg/kg body mass (half of this may be supplied by gut bacteria).
Spinach	240		
Soya beans	190		
Cabbage	100		
Medium			
Bran (wheat)	80		
Green beans	20		
Pork liver	20		
Low			
Oranges	less than		
Apples	5		

*In the U.S.A. the safe and adequate range of intake for vitamin K is estimated to be between 70–140 μg/day for men and women.

FUNCTION	1. *Clotting of blood.* (a) Involved in the synthesis of certain blood clotting factors in the liver (clotting factors II, VII, IX and X are vitamin K dependant proteins). (b) Through the action of these clotting factors, vitamin E may influence calcium metabolism in various parts of the body.
DEFICIENCY (Hypovitaminosis K)	*Haemorrhage* — the time that blood takes to clot is delayed and may result in a tendency to bleed; if this occurs in the infant it is called *harmorrhagic disease of the newborn.* Deficiency in Australian adults is rarely seen but may occur in: 1. *Newborn and low birth weight infants* have limited stores of vitamin K (due to low vitamin K stores in the mother) and since the intestinal tract is sterile, haemorrhage may occur during the immediate post-natal period. 2. *Malabsorption syndromes* such as diseases of the pancreas, intestines or alcohol abuse may prevent adequate absorption. 3. *Liver diseases* may reduce synthesis of clotting factors despite adequate vitamin K. 4. *Medications* — certain antibiotics (neomycin) can reduce the content of bacteria in the gut (but is rare as a cause of deficiency). 5. Anticoagulant therapy (warfarin) can lead to a bleeding tendency if dietary vitamin K is poor or supply from the gut bacteria is reduced or both.
TOXICITY (Hypervitaminosis K)	Potentially toxic if given in large doses over a prolonged period of time.

Table 21.6 Thiamin

FOOD SOURCE	*Vitamin B_1 (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum loss during cooking is 80% (usual loss in cooking of mixed diet is about 25% but if cooking water is discarded the loss is greater). 2. Sensitive to heat, light and alkali conditions — in refining cereal products some of the vitamin can be lost. 3. Some foods contain thiamin antagonists (tea, coffee, etc.) and enzymes that break down thiamin (raw fish).	Expressed in terms of energy intake: 0.1 mg/1000 kJ for all ages. It varies from 0.4 mg per day for infants to 1.1 mg per day for adults.
Yeast (brewers)	12.0		
Vegemite	9.7		
Medium			
Lean pork (raw)	0.9		
Legumes	0.4		
Bread			
wholemeal	0.26		
white	0.18		
Low			
Polished rice	trace		

FUNCTIONS	Functions as a *co-enzyme* in at least 24 enzyme systems, many of which are involved in the release of energy from carbohydrate, protein and fat.
DEFICIENCY (Hypovitaminosis B_1)	Frank deficiency is known as beriberi. Clinical effects are reflected in: 1. *Gastrointestinal symptoms*, such as loss of appetite, indigestion, vomiting, loss of muscle tone. 2. *Nervous system symptoms* called '*dry beriberi*'. Results in impairment of sensation, loss of reflexes, difficulty in movement, partial paralysis. When symptoms are accompanied by oedema it is termed 'wet beriberi'. If the brain is affected *Wernicke's encephalopathy* develops with symptoms of: involuntary eye movement, muscular unco-ordination, mental deterioration, and may progress on to *Korsakoff's disease* if untreated. This is characterised by memory defect and confabulation. 3. *Cardiovascular symptoms*: cardiac failure unlike that caused by other forms of heart disease. Deficiency in Australia occurs mainly among *chronic alcoholics* and is probably one of the most common vitamin deficiencies. Other causes of deficiency include: 1. *Kidney disease* (renal dialysis patients in particular). 2. *Long-term intravenous feeding*. 3. Consumption of large amounts of thiamin antagonists or large amounts of food containing thiaminase (e.g. raw fish).
TOXICITY (Hypervitaminosis B_1)	Large doses taken orally appear safe but large doses administered intravenously have produced symptoms of shock.

Table 21.7 Riboflavin

FOOD SOURCE	*Riboflavin (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum loss during cooking is 75%. 2. Sensitive to light and alkali conditions. Milk, an important source, if exposed to light for about 2 hours may lose at least half of its riboflavin content.	Expressed in terms of energy intake: 0.12 mg/ 100 kJ for all ages. Varies from 0.5 mg/day for infants to 1.4 mg/day for adults.
Vegemite	14.3		
Lambs Liver (fried)	4.4		
Cornflakes	2.1		
Medium			
Milk	0.2		
Cheese (Cheddar)	0.5		
Low			
Potato (boiled)	0.03		
Apple	0.02		
FUNCTION	Functions as part of a group of enzymes called *flavoproteins* which are involved in: 1. Cell respiration; 2. Release of energy from carbohydrate, protein and fat.		
DEFICIENCY	Deficiency causes damage to a variety of body tissues, clinically characterised by: 1. Pallor and cracking of the lips (*cheilosis*) and at the angles of the mouth (*angular stomatitis*). 2. Swelling of the tongue (glossitis). 3. Failure to grow in children. 4. Eye and visual disturbances. 5. Conjunctivitis and greasy, scaling dermatitis affecting nasolabial folds. These features often occur in multiple deficiencies of the B complex and are not specific to riboflavin deficiency. There is no evidence of riboflavin deficiency in Australia. (Dietary deficiency of riboflavin is usually associated with a deficiency of other B group vitamins.)		
TOXICITY	Large doses taken orally appear safe.		

Table 21.8 Niacin

FOOD SOURCE	*Niacin (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum loss of 75% during cooking. 2. Relatively stable to heat, light and alkali conditions. 3. Milk is a good source of precursor tryptophan. 4. Niacytin (a bound form of niacin) in wheat, corn and rye is unavailable to the body for biological purposes.	Expressed in terms of energy intake: 1.6 mg niacin equivalents/1000 kJ for all ages.
Vegemite	110		
Bran (wheat)	29.5		
Ovaltine	18		
Medium			
Peanut butter	15		
Lamb liver (fried)	15		
Beef	2.5–5.5		
Low			
Vegetables, Fruit & Milk	trace to 4.5		
Eggs	trace		

FUNCTION	Functions as part of the *nicotinamide adenine dinucleotide* enzyme systems and as such is involved in: 1. Cell respiration. 2. Metabolism of carbohydrate protein and fat synthesis. (The vitamin activity of niacin and riboflavin is closely related.)
DEFICIENCY	Frank deficiency is known as *Pellagra* and is characterised by the three Ds: 1. *Dermatitis* (pigmented and scaly skin) develops on areas of the body exposed to sunlight. 2. *Diarrhoea*. 3. *Dementia* (rare). Early symptoms include: loss of appetite, indigestion, weakness, anaemia, glossitis. (All of these can be found in association with other vitamin deficiencies.)
TOXICITY	Large doses of nicotinic acid (100–200 × R.D.I.) have pharmacological effects such as: Lowering of blood cholesterol and trighlyceride. 2. Increasing blood high density lipoprotein — HDL, (which appears to be protective against heart disease). Undesired side effects include: 'flushing', gastrointestinal disturbances, arrythmias, and liver toxicity.

Table 21.9 Vitamin B_6

FOOD SOURCE	*Vitamin B_6 (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum loss of 40% during cooking. 2. Sensitive to light, air or oxygen and alkali conditions.	Adults and children 8 years and over 1.0–2.2 mg/day based on: 0.015–0.020 mg/gram protein per day for all ages, from a mixed diet providing 10–15% of energy from protein.
Vegemite	3.0		
Walnuts	0.7		
Bananas	0.5		
Lamb liver (fried)	0.5		
Medium			
Chicken	0.2–0.5		
Egg yolks	0.3		
Potatoes	0.2		
Low			
Milk	.04		
Apples	.02		

FUNCTION

Functions as a co-enzyme for many different enzyme systems involved in:
1. *Synthesis and metabolism of proteins* such as serotonin* and gamma butyric acid (see chapter 35, Nutrition and the brain);
synthesis of niacin from tryptophan, and the formation and growth of red blood cells including haemoglobin.
2. *Release of energy from carbohydrate and fat.*

DEFICIENCY

Clinical symptoms of deficiency are not specific but may include:
1. *Central nervous system disturbances* such as irritability, mental depression, convulsive seizures, abnormal brain wave pattern, and conduction of nerve impulses.
2. *Dermatitis*, similar to that seen in pellagora.
3. *Cheilosis, angular stomatitis* and *glossitis.*
4. *Sideroblastic anaemia* is responsive to vitamin B_6.
5. *Immune deficiency* — impaired antibody formation.
6. *Renal calculi.*

Deficiency is rare in Australia but groups at risk include:
1. *Infants*, the ratio of vitamin B_6 to protein may be low in some breast and cow's milk.
2. *Alcoholics*, the incidence may be in the order of 20–30%.
3. *Women on oral contraceptives*, especially those containing oestrogen.
4. *Vitamin B_6 dependent syndromes*, genetic abnormality requiring large amounts of vitamin B_6 to prevent convulsions, seizures, mental retardation.
5. *Thyroid disease* (overactive thyroid gland).
6. *Certain medications* inactivate vitamin B_6; izoniazed and penicillamine.

Table 21.9 cont'd

TOXICITY	Toxicity is low but regular and prolonged intakes of large amounts can induce: 1. Dependency on vitamin B_6. 2. Interference with the action of certain drugs such as L-dopa (Parkinsonism). 3. Sensory neuropathy leading to problems in walking with doses down to about 200 mg per day.

Table 21.10 Pantothenic acid

FOOD SOURCE	*Pantothenic acid (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum loss of 50% during cooking. 2. Sensitive to heat and both acid and alkali conditions. 3. Considerable amounts are lost during the milling of cereal grains.	No recommendation has been made in Australia. 4–7 mg/day for adults have been estimated to be safe and adequate (U.S.A.) (intestinal bacteria synthesise small quantities).
Lambs liver (fried)	7.6		
Egg yolk	4.6		
Broad beans (boiled)	3.8		
Medium			
Lobster (boiled)	1.6		
Apricots	0.7		
Beef	0.5–0.8		
Milk	0.4		
Low			
Pear	.05		
French beans (boiled)	.07		

FUNCTION	Functions as a co-enzyme for Coenzyme A and acylcarrier protein (ACP) and is involved in many key chemical reactions in the body. Some of these include: 1. Energy release from the metabolism of carbohydrates, proteins and fats. 2. Synthesis of amino acids, fatty acids, sterols (such as cholesterol), steroid hormones and vitamin D. 3. Formation of red blood cells, haemoglobin. 4. Formation of acetylcholine (neurotransmitter).
DEFICIENCY	Clinical signs of deficiency have been recorded only under experimental conditions, and include: 1. personality changes. 2. irritability, restlessness. 3. fatigue, vomiting and muscle cramps. Evidence of dietary deficiency has not been recognised in man — though marginal deficiencies may exist along with deficiencies of other B-complex vitamins.
TOXICITY	Toxicity is low, however some studies indicate that daily doses of 10–20 gms may result in diarrhoea and fluid retention.

Table 21.11 Biotin

FOOD SOURCE	*Biotin (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum loss of 60% during cooking. 1. Sensitive to alkali conditions as well as air and oxygen. 3. Avidin, an anti-vitamin, found in raw egg white inactivate biotin. 4. Biotin in wheat is in a bound form and unavailable to the body.	No recommendation has been made in Australia. 100–200 mg/day has been estimated to be safe and adequate (U.S.A.) (intestinal bacteria produce significant amounts for absorption).
Brewers yeast	200		
Egg (yolk)	60		
Soya beans	60		
Liver	40		
Medium			
Wholemeal bread	6		
Fish	6–10		
Milk	2		
Low			
Potato	trace		
FUNCTION	Functions as a co-enzyme in: 1. The fixation of CO_2 into organic molecules. 2. Linking the metabolism of carbohydrate and fat. 3. Formation of glucose and fatty acids in the body. 4. May be involved in the metabolism of vitamin B_{12}.		
DEFICIENCY	Deficiency in man has been characteristically associated with the consumption of *avidin* which is rare. Symptoms include: lethargy, loss of appetite, nausea, vomiting, glossitis, mental depression, dry scaly dermatitis, muscle pain, hypercholesterolemia, and changes in the electrocardiogram (E.C.G.). Groups at risk of deficiency include: 1. *Infants* below 6 months of age may develop *seborrheic dermatitis* due to dietary deficiency. 2. *Leiner's disease* in children, a condition resulting in dermatitis responds to large doses of biotin. 3. *Long term intravenous feeding* (total parenteral nutrition).		
TOXICITY	Toxicity is low.		

Table 21.12 Folacin

FOOD SOURCE	*Folacin (μg/100 g)*	*Notes*	*RDI*
High		1. One of the most unstable vitamins and considerable amounts are lost during cooking (up to 100%) either by leaking into the cooking water or destruction by heat. 2. Sensitive to light, air or oxygen. 3. Folacin antagonist in some foods may decrease its availability to the body.	200 μg *total* folacin/day for adults; 400 μg *total* folacin/day during pregnancy (bacteria in the intestines synthesise small amounts of folate for absorption).
Cabbage (boiled)	230–430		
Lambs liver (fried)	260		
Spinach (boiled)	140		
Peanuts	110		
Medium			
Peas	78		
Egg yolk	52		
Oranges	37		
Wholemeal bread	30		
Low			
Meat	3		
Milk	0.3		

FUNCTION

1. Functions as a co-enzyme in the transfer of carbon molecules to various compounds in the synthesis of key materials such as:
(a) *purines* (essential cell components involved in cell division transmission of inherited traits);
(b) *thymine* involved in DNA synthesis;
(c) *haemoglobin*;
(d) *choline*.
2. Involved in the metabolism of protein and fat (functions are closely linked with those of vitamin B_{12}).

DEFICIENCY

Characteristic and early result of deficiency is *macrocytic anaemia* in which the red blood cells are abnormally large. Any frequently dividing cell in the body can be affected:
1. *Small intestine defects* resulting in malabsorption and deficiency of other nutrients.
2. *White blood cells* and *platelets* or during periods of *rapid growth* such as pregnancy.
(Clinical manifestations of deficiency have been reported to develop in approximately 16 weeks on low folacin diets.)

Probably the most common vitamin deficiency in Australia and is seen in:
1. *Institutionalised elderly chronic alcoholics and pregnant women* (oral folacin supplementation appears desirable to maintain maternal stores and provide for increased needs of the foetus).
2. *Malabsorption syndromes*.
3. *Medications* such as the antitumour drug (methotrexate), antimalarial drug (pyrimethamine), antibiotic (trimethoprim), sedatives and barbituates.

TOXICITY

Large doses — up to 15 mg/day appear to be non-toxic.

Table 21.13 Vitamin B_{12}

FOOD SOURCE	*Vitamin B_{12} (μg/100g)*	*Notes*	*RDI*
High		1. Maximum loss of 10% during cooking. 2. Sensitive to alkaline conditions but stable to heat, light, oxygen and acid.	2.0 μg/day for adults and boys and girls from 11 and 18 years of age (intestinal bacteria produce vitamin B_{12}).
Lambs liver (fried	81		
Sardines	28		
Oysters	15		
Medium			
Egg yolk	4.9		
Fish	1.5		
Beef	1.2		
Cheese	1.2–1.5		
Low			
Vegetables	0		
Fruit	0		

FUNCTION

1. Functions as a co-enzyme in *protein metabolism* such as the synthesis of nucleic acids e.g. DNA and RNA. This is especially important in the:
(a) *bone marrow*, where the red blood cells are formed;
(b) *nerve tissue*, where vitamin B_{12} is involved in the breakdown of certain fats.
2. Vitamin B_{12} acts indirectly on red blood cell formation through the activation of folacin coenzymes.

DEFICIENCY

Characteristic result of deficiency is:
1. *Pernicious anaemia* — a chronic, niacrocytic type of anaemia (similar to that seen in folacin deficiency). Symptoms include lethargy, dypspnoea, loss of appetite and weight, pallour, lemon-yellow appearance of the skin.
2. *Neurological disturbances.*

Groups at risk of nutritional deficiency:
1. *Strict vegetarians* (vegans) who eat no animal products, milk or eggs.
2. *Newborn infant* of the *vegetarian (vegan)* mother, where the newborn has insufficient stores of the vitamin and the breast milk may be a poor source.
3. *Methylmalonic acidaemia*, an inherited defect in vitamin B_{12} metabolism.
4. *After surgical removal* of part of the *stomach* which produces the intrinsic factor or part of the *ilium* where absorption takes place.

TOXICITY

Very low toxicity in doses over 1000 times the R.D.I.

Table 21.14 Ascorbic acid

FOOD SOURCE	*Vitamin C (mg/100 g)*	*Notes*	*RDI*
High		1. Maximum loss of 100% during cooking. 2. Sensitive to heat, light, oxygen and alkali such as bicarbonate of soda. 3. Use of copper cookware can destroy vitamin C.	1. 30–40 mg/day for adults 2. 10 mg/day will prevent and cure clinical signs of scurvy.
Blackcurrents	200		
Green peppers	100		
Orange juice (fresh)	50		
Lemon	47		
Medium			
Brussel sprouts	35		
Cauliflower	20		
Cabbage	20		
Spinach (all cooked)	20		
Low			
Milk (cow's)	2		
Beef	0		

FUNCTION

1. *Maintenance of body connective tissue* — involved in the formation of hydroxyproline in the synthesis of *collagen*, a connective tissue conferring structures and form to body cells.
2. *Brain and nerve function* — involved in the formation and breakdown of noradrenalin and serotonin (neurotransmitters).
3. *Muscle function* through formation of carnitine is involved in energy release from fatty acids.
4. It may also be involved in:
(a) increased intestinal absorption of iron;
(b) cholesterol metabolism;
(c) formation of thyroid hormone;
(d) folic acid metabolism;
(e) reduction of nitrosamine formation in the stomach (see chapter 34).

DEFICIENCY
(Hypovitaminosis C)

Frank deficiency is known as *scurvy* and is characterised by defective collagen synthesis and a tendency of blood vessels to bleed. Other symptoms: *bruising, delay in wound healing, ulcers, drying of skin and mucous membranes, susceptibility to infections, anaemia, muscle weakness, and fatigue* may also develop.
It takes several months on a diet deficient in vitamin C for scurvy to develop

Scurvy is uncommon in Australia but may occur in:
1. *Newborn infants* of mothers who have ingested large doses of vitamin C adapt to the elevated level prenatally, and may suffer scurvy after birth.
2. *Young children* fed exclusively and for a prolonged period on cow's milk.

Table 21.14 cont'd

	3. *Institutionalised elderly* — recent studies indicate that 30% have low blood levels of vitamin C. 4. *Chronic alcoholics.* 5. *Cigarette smokers*, those on *large doses of aspirin* and *oral contraceptive users* have been reported to have low blood levels of vitamin C. 6. *Nutritional ignorance* combined with poor cooking and eating habits is usually associated with deficiency.
TOXICITY (Hypervitaminosis C)	Toxicity is low. Risks of megadose therapy include. 1. *Rebound scurvy* may occur after discontinuation of megadoses of vitamin C. 2. *Oxalate stone formation* in the kidney & bladder with daily intake in excess of 8 gms. 3. *Gastrointestinal disturbances* nausea/abdominal cramps/diarrhoea usually before meals. 4. *Blood clotting.* 5. *Vitamin B_{12} deficiency* may occur if intake of vitamin C is high. 6. *Toxic metal absorption* such as mercury may increase. 7. *Alteration of action of certain drugs* such as warfarin (anticoagulant), aspirin, antidepressants and the contraceptive pill. 8. *Increased absorption of iron* may cause problems in individuals susceptible to iron overload.

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