Keywords B vitamin complex/Cellular metabolism/DNA synthesis/Anaemia

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In this article...

- The role of the vitamin B complex in cellular metabolism
- How B vitamins support nervous system function
- Effects on the body of vitamin B group deficiencies

Vitamin B complex: B group vitamins and their role in the body

Key points

The vitamin B complex comprises a large group of water-soluble vitamins

B vitamins play a vital role in energy production, DNA synthesis and central nervous system function

Although vitamin B deficiencies are relatively uncommon in the UK, certain populations are more at risk, such as people with some health conditions

Obtaining sufficient vitamin B12 can be challenging for people on vegan diets who may require vitamin supplements

Long-term vitamin B deficiency can cause diseases such pellagra, beriberi or anaemia

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Abstract Vitamin B complex plays a vital role in cellular metabolism and is discussed in this second article in a series on vitamins and minerals. A deficiency in B group vitamins can hamper efficient energy production in cells, leading to adverse health effects. Deficiency of vitamin B1, common in people with alcohol use disorder or AIDS, can cause beriberi, which affects the nervous system and heart. Vitamin B3 deficiency leads to pellagra, characterised by dermatitis, diarrhoea, depression and dementia. Deficiencies in vitamins B9 (folate) and B12 can lead to anaemia and neurological issues.

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The vitamin B complex consists of a large group of water-soluble B vitamins and their derivatives. This article, the second in a series on vitamins and minerals, examines the nature and physiological roles of the key B complex vitamins (Fig 1), as well as the pathological effects associated with their deficiencies.

An adequate supply of B vitamins is required for healthy metabolism and efficient energy release in cells as all the B vitamins except for vitamin B9 (folate) are involved in at least one step of the biochemical processes of cellular metabolism. Insufficient levels of these vitamins can become a rate-limiting factor for energy production, resulting in significant metabolic decline and negative health consequences (Tardy et al, 2020).

Cellular metabolism

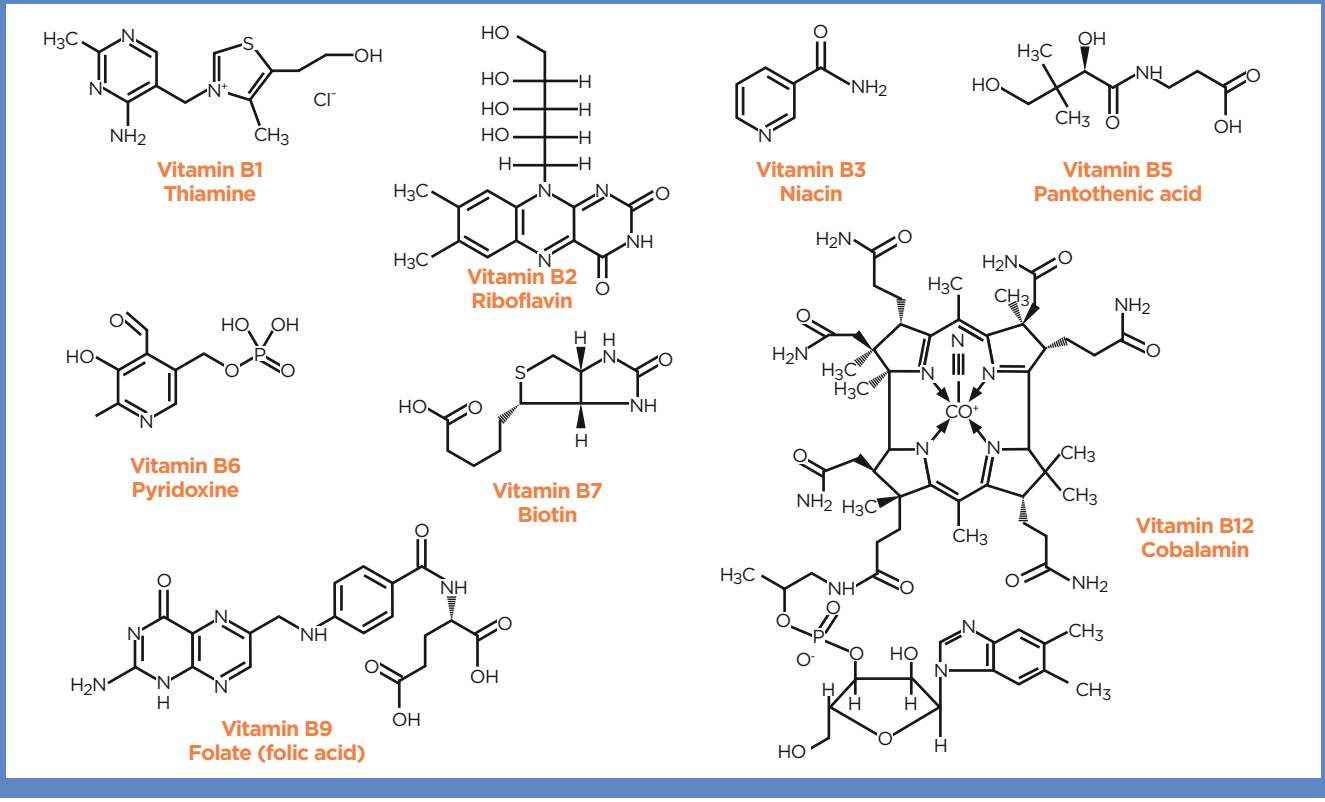
For cells to release the energy to function, they must absorb and use various organic macromolecules for fuel (primarily carbohydrates, fats and proteins) along with (vitamins and minerals), many of which act as essential cofactors for cellular enzymes. Once inside the cell, organic macromolecules undergo thousands of chemical reactions, known as cellular metabolism, which are facilitated by cellular enzymes. Metabolism can be divided into two types of reactions:

- Anabolism – building larger and more complex substances from simpler building blocks (for example, joining amino acids into proteins);
- Catabolism – breaking down larger, more complex substances into simpler substances (for example, breaking down complex food molecules in the gut, or cellular respiration involving metabolism of glucose to facilitate synthesis of adenosine triphosphate (ATP) for energy storage and use in cells.

Vitamin B1 (thiamine)

Vitamin B1 or thiamine (Fig 1) plays a role in cell metabolism and the functioning of the nervous system. The current recommended dietary allowance (RDA) is 1000µg a day for men and 800µg for women (NHS, 2023). Food sources of vitamin B1 are:

Fig 1. B complex vitamins



- Whole grains (for example, whole wheat, barley, brown rice, oats);
- Legumes (for example, lentils, beans, and peas);
- Nuts and seeds (for example, sunflower seeds, flaxseeds and peanuts);
- Pork;
- Enriched cereals.

Thiamine deficiency

Thiamine deficiency can be due to low intake, impaired intestinal absorption or increased excretion rate of thiamine; deficiencies are also common in people with alcohol use disorder and patients with acquired immunodeficiency syndrome (AIDS) (Martel et al, 2022). Fig 2 shows the signs of vitamin B1 deficiency.

Prolonged thiamine deficiency is associated with beriberi (Fig 2), a disease that can significantly affect the nervous system and heart. There are two manifestations of beriberi:

- Dry beriberi – characterised by nervous system-related symptoms, such as muscle weakness, tingling or numbness in the extremities, difficulty walking and loss of coordination. Severity correlates with the degree and duration of deficiency and can be accompanied by the neurological disorder Wernicke's encephalopathy and Korsakoff syndrome. These neurophathologies are characterised by confusion, poor memory, eye muscle paralysis (nystagmus) and problems with muscle coordination (Shible et al, 2019). Long-term thiamine deficiency is often associated with alcohol use disorder, which can lead to Wernicke's encephalopathy and Korsakoff syndrome.
- Wet beriberi – associated with cardiovascular symptoms that can lead to cardiomyopathy (diseases of the heart muscle). Clinical features of wet beriberi can include tachycardia (heart rate >100 beats/min), shortness of breath and fluid retention (Jankovic et al, 2021).

Thiamine supplements can be effective in preventing the onset of cognitive impairment in patients at risk of cognitive dysfunction due to thiamine deficiency. In one study of 10,000 patients diagnosed with alcohol use disorder, those who received thiamine therapy compared with a control group who received no therapy were at lower risk of developing dementia (Chou et al, 2019).

Early identification of thiamine deficiency as the cause of neurologic symptoms is essential as high-dose thiamine supplements offer an effective and inexpensive treatment (Wiley and Gupta, 2023).

Vitamin B2 (riboflavin)

Vitamin B2 or riboflavin (Fig 1) plays crucial roles in energy production, cell growth and metabolism (Ross et al, 2020). It has a key role in redox reactions (involving the transfer of electrons between two reactants), which play a crucial part in the production of energy during cellular respiration. It also acts as a protective antioxidant due to its association with the regeneration of glutathione, a potent free radical scavenger in cells (Vale et al, 2022).

The current RDA for vitamin B2 is 1,300µg a day for men and 1,100µg for women (NHS, 2023). Vitamin B2-rich foods include:

- Dairy products (for example, milk, yoghurt and cheese);
- Eggs;
- Lean cuts of red meat (for example, beef and pork);
- Organ meats (for example, beef liver);
- Leafy green vegetables (for example, spinach);
- Chicken breast;
- Salmon;
- Fortified cereal and bread;
- Almonds (Harvard TH Chan School of Public Health, 2023).

Riboflavin deficiency

Vitamin B2 deficiency is relatively uncommon in developed countries, being

most frequent in poorer areas of Asia and Africa (Mahabadi et al, 2023). People at a higher risk for riboflavin deficiency include:

- Pregnant/lactating women since riboflavin crosses the placenta for use by the developing foetus and is also a water-soluble component of breast milk;
- Infants and children with inadequate levels of milk and meat in their diet;
- Patients with eating disorders such as anorexia nervosa;
- Older people and patients with malabsorption syndromes, such as coeliac disease and short bowel syndrome;
- People with alcohol use disorder;
- Athletes, as riboflavin is used in the metabolic pathways so can be depleted by vigorous exercise;
- Some women on birth control pills, which lead to poor absorption of vitamin B2 (Mahabadi et al, 2023; Peechakara and Gupta, 2022).

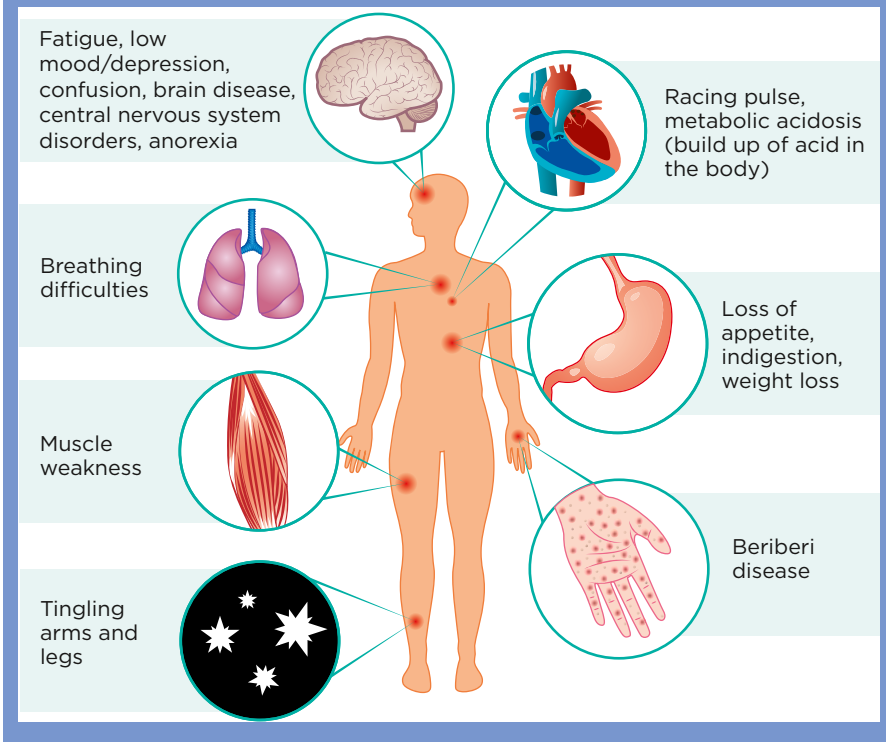
Riboflavin deficiency can cause skin disorders including: cracks and sores around the corners of the mouth (angular cheilitis); inflammation and redness of the tongue (glossitis); and scaly rash on the face and genitals (seborrheic dermatitis). It can also lead to sore throat, difficulty swallowing and swollen or purple-coloured tongue (magenta tongue). Eye problems may also occur, especially sensitivity to light (photophobia), blurred vision and itching and burning sensations in the eyes. In some cases, riboflavin deficiency can lead to anaemia, characterised by fatigue, weakness and reduced red blood cell production (Aljaadi et al, 2022).

Vitamin B3 (niacin)

Vitamin B3 or niacin is also essential for cellular metabolism, playing a key role in the Kreb's cycle (a key part of the aerobic respiration pathway) and the synthesis of many diverse molecules in the body (Makarov et al, 2019). Chemically, vitamin B3 is known as nicotinic acid and its amide as nicotinamide; both molecules are precursors of two important co-enzymes in metabolic reactions, as well as being involved in deoxyribonucleic acid (DNA) repair.

The recommended daily allowance of vitamin B3 is 16.5mg for men and 13.2mg for women (NHS, 2023). It can be obtained from a wide variety of foods, including meats (such as chicken and beef), fish and plant-based products (for example, nuts, legumes and grains) (National Institutes of Health (NIH), 2022a).

Fig 2. **Signs and symptoms of vitamin B1 deficiency**



Niacin deficiency

Severe niacin deficiency can lead to a condition called pellagra (sour skin); pellagra is characterised by varying degrees of dermatitis, diarrhoea, depression, seizures and dementia (National Institute of Diabetes and Digestive and Kidney Diseases (NIDDKD), 2021).

Historically, pellagra was endemic in populations reliant on corn-based diets, but today is a condition primarily associated with malnourished people with alcohol use disorder (NIDDKD, 2021). Although pellagra is rare in developed countries due to niacin being readily available from a balanced diet, the risk of niacin deficiency is higher in people with certain medical conditions, such as classical dermatitis and Crohn's disease, or who have dietary deficiencies (NIDDKD, 2021).

Niacin at high doses has been associated with the risk of significant liver injury (NIDDKD, 2021); a recent study has also suggested excessive amounts may increase risk of heart attacks and strokes, possibly by inflaming blood vessels (Ferrell M et al, 2024).

Vitamin B5 (pantothenic acid)

Vitamin B5 is also called pantothenic acid; this is derived from the Greek word "pantos" meaning everywhere. It is known as an "anti-stress vitamin" and acts as the precursor for biosynthesis of coenzyme A (CoA) and acyl carrier protein (ACP), both of which are involved in cellular metabolism (Gheita et al, 2020).

Vitamin B5 is found in varying amounts in most vegetables, whole grain foods and animal products, including eggs, beef, poultry, liver and kidneys (NHS, 2023). It is also artificially added to many fortified breakfast cereals. It cannot be stored in the body, so must be continually consumed as part of a well-balanced diet. Currently there is no established recommended daily intake of vitamin B5 in the UK (NHS, 2023). However, as implied by its Greek-derived name, its widespread availability in a diverse variety of foods normally means enough can be obtained from the daily diet.

Pantothenic acid deficiency

Pantothenic acid deficiency, although rare, may be present in individuals with eating disorders or with severe malnutrition due to starvation. Where present, a deficiency typically leads to fatigue, numbness, muscle cramps, and poor wound healing (Sanvictores and Chauhan, 2023).

People with a mutation in their pantothenate kinase 2 (PANK2) gene may also display pantothenic acid insufficiency. This is because the mutation potentially decreases the conversion of pantothenic acid to CoA, impeding cellular metabolism (Sanvictores and Chauhan 2023).

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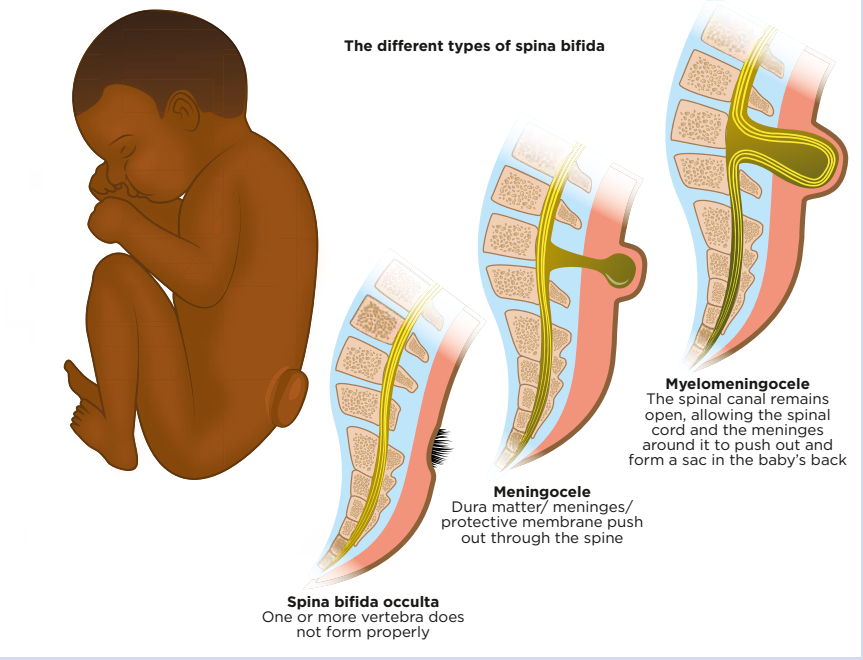
Pantothenate kinase associated neurodegeneration (PKAN) (previously known as Hallervorden-Spatz syndrome) is a genetic degenerative disease of the brain caused by mutation of the pantothenate kinase 2 (PANK2) gene. Individuals with PKAN display excess iron accumulation in the basal ganglia, which is apparent on MRI scans. In the transverse (horizontal) plane, this results as a tiger-like image with prominent eyes, known as the "eye-of-the-tiger" sign (Chang and Lin, 2011).

PKAN causes progressive motor dysfunction, cognitive decline and visual impairment. Neuropsychiatric manifestations include behavioural difficulties, obsessions, aggression, personality changes, intense emotions and rapid mood shifts. Informally, it is reported that vitamin B5 supplement in PKAN patients is well tolerated, with no known toxicity, and can lead to improved motor symptoms, speech, cognition and general well-being (Kurian and Hayflick, 2013).

Fig 3. **Spina bifida due to vitamin B9 (folate) deficiency**



Vitamin B6 (pyridoxine)

Vitamin B6 or pyridoxine plays a vital role in metabolism of carbohydrates, lipids, amino acids and nucleic acids. It is an antioxidant with the ability to reduce advanced glycation end products (AGEs). AGEs are harmful compounds that form when sugar reacts with proteins/fats in the blood stream and are seen as exacerbating many long-term conditions, such as atherosclerosis, renal disease and diabetes (Stach et al, 2021).

The recommended dietary allowance for vitamin B6 is 1400µg a day for men and 1200µg for women (NHS, 2023). Good dietary sources are:

- Pork;
- Poultry;
- Many fish;
- Peanuts;
- Soya beans;
- Wheatgerm;
- Oats;
- Bananas;
- Milk;
- Some fortified breakfast cereals (NHS 2023).

Other sources include:

- Beef liver;
- Dark leafy greens;
- Chickpeas;
- Potatoes (Franco et al, 2022).

Pyrodoxine deficiency

Vitamin B6 functions as an essential cofactor for numerous enzyme systems, particularly those involved in amino acid biosynthesis, lipid metabolism, haeme synthesis, neurotransmitter synthesis and cellular metabolism (Franco et al, 2022). It also plays a role in supporting a healthy immune system; deficiency can impair immune function and increase susceptibility to infection. Severe pyrodoxine deficiency can result in significant neurological symptoms, such as confusion, depression, irritability, seizures, numbness or tingling in the extremities (Stach et al, 2021).

Although pyridoxine deficiency is rare, it can occur in people with significant malnutrition or reduced/poor pyridoxine absorption and may be seen in people with alcohol use disorder, where liver damage impedes the release of pyridoxal phosphate (PLP). PLP is the active coenzyme form and the most common measure of B6 blood level in the body (Franco et al, 2022).

Since pyridoxine is essential to haem synthesis, deficiency can contribute to the development of anaemia, with reduced oxygen transport leading to weakness and fatigue; other symptoms of deficiency include dermatitis, rashes, and dry cracked skin around the mouth (angular cheilitis) (Franco et al, 2022; NIDDKD, 2021).

Certain medications, such as isoniazid, L-dopa, penicillamine and cycloserine, interact with pyridoxine and can increase the risk of pyridoxine deficiency (Hemminger and Wills, 2023). Therefore, vitamin B6 supplements are frequently given with these drugs (typically 10-25mg daily) (NIDDKD, 2021). High doses of vitamin B6 (50-500mg daily) have been suggested to treat many diverse conditions, including carpal tunnel syndrome, schizophrenia, autism and diabetes (NIDDKD, 2021). However, the effectiveness of such doses for these conditions has not been scientifically proven.

Vitamin B7 (biotin)

Vitamin B7 is also known as biotin or vitamin H. It plays a key role in various metabolic processes, including the metabolism of carbohydrates, fats and proteins (Saleem and Soos, 2023), and is essential to maintaining a healthy skin and nervous system (Sirithanakorn and Cronan, 2021).

As mammals lack the ability to synthesise vitamin B7, it is an essential vitamin that must be acquired from the diet and/or intestinal microflora. Biotin-rich foods include:

- Egg yolk (especially when raw, as biotin is partially destroyed by cooking);
- Liver;
- Soybeans;
- Fish;
- Dairy products;
- Leafy vegetables (such as spinach)
- Rice.

A healthy gut microbiota can also synthesise significant amounts of biotin to supplement dietary intake (Saleem and Soos, 2023).

Although a formal recommended daily dietary intake for biotin has not yet been established, the required amount is generally small, with the average dietary intake

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in western populations estimated at 30-70µg daily (Saleem and Soos, 2023).

Biotin deficiency

As the dietary requirement appears so low, biotin deficiency is relatively rare due to biotin being widely available in various foods and being produced by intestinal bacteria. However certain conditions, such as genetic disorders, or prolonged consumption of raw egg whites (which contain a protein called avidin that binds to biotin, inhibiting its absorption) increase the risk of biotin deficiency (NIDDKD, 2021).

Significant deficiencies, although rare, may lead to growth retardation and skin and neurological disorders (Sirithanakorn and Cronan, 2021). Long-term treatment with oral antibiotics can also lead to biotin deficiency. This is caused by depletion of the biotin-producing gut microbiota and skewing of the microbial populations in favour of increased populations of biotin-consuming bacteria (Hayashi et al, 2017).

Skin and neural cells appear more sensitive to biotin deficiency than most other cell types (Suormala et al, 2002). Consequently, deficiency is commonly associated with hair loss, brittle hair, dry-scaly skin, and weak brittle nails (Patel et al, 2017). More rarely, it can also cause a diverse range of neurological symptoms, including depression, lethargy, hallucinations and tingling sensations in the extremities (Mikkelsen et al, 2016).

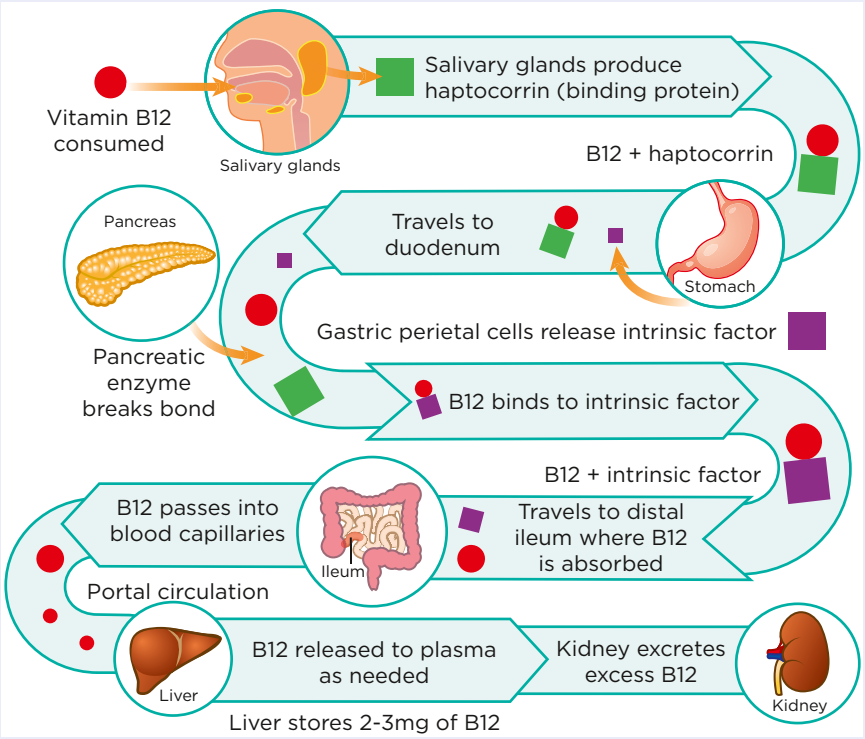
More severe neurological conditions associated with biotin deficiency primarily arise from genetic defects in biotin metabolism. One example is biotin-responsive basal ganglia disease. This is characterised by recurrent encephalopathy and dystonia along with more variable symptoms, including muscle spasticity, seizures, dysphagia (difficulty swallowing) and slurred speech. This condition predominantly affects children and adolescents, and potentially results in death if not effectively treated with high doses of biotin (Kassem et al, 2014).

Vitamin B9 (folate)

Vitamin B9, also known as folate (available synthetically as folic acid), is essential for the replication and synthesis of DNA, required for normal cell division (Franco et al, 2022). Folate is converted into tetrahydrofolic acid (THF), which plays a critical role in various transfer and methylation reactions vital to the synthesis of the nitrogenous bases of DNA and RNA (Franco et al, 2022).

Folate is present naturally in many food sources including: dark leafy green vegetables (such as spinach); asparagus and Brussels sprouts; fruit and fruit juices; nuts; beans; peas; seafood; eggs; meats; poultry; milk; and grains (NIH, 2022b).

Fig 4. **Transportation and absorption of vitamin B12 in gastrointestinal tract**



Folate needs to be regularly consumed as part of a balanced diet since humans lack the ability to synthesise it (Merrell and McMurry, 2022). The current RDA for vitamin B9 is 200µg a day for both men and women (NHS, 2023).

Folate deficiency

Folate deficiency is commonly due to poor dietary intake, alcohol use disorder, malabsorption disorders or by the diet not being adapted to compensate for increased folate requirements during pregnancy (Merrell and McMurry, 2022).

Deficiency can cause macrocytic megaloblastic anaemia due to the stem cell precursors of erythrocytes (red blood cells) no longer being able to divide efficiently, leading to much larger macrocytic cells. Symptoms classically include fatigue, weakness, shortness of breath and pale skin (Socha et al, 2020).

Adequate folate intake is vital for normal embryonic development to allow closure of the neural tube (the early precursor of the brain and spinal cord). Pregnant women may require additional folate supplementation (as advised by health professionals) to prevent congenital neural tube defects such as spina bifida (Fig 3).

Neural tube defects affecting the brain and the spinal cord due to folate deficiency may lead to lifelong disabilities (Pei et al, 2019). Deficiency in infants and children can cause delayed growth, poor weight gain and developmental issues. Folate also plays a key role in reducing levels of the amino acid homocysteine where these are elevated, which is important as elevated homocysteine is associated with increased risk of heart disease (Jakubowski, 2019).

Vitamin B12 (cobalamin)

Vitamin B12 or cobalamin plays a crucial role in DNA synthesis and the formation of red blood cells; it is also essential for a healthy, properly functioning nervous system (Calderón-Ospina and Nava-Mesa, 2020).

Cobalamin is primarily found in animal-derived foods, such as meat, fish, eggs and dairy products, making it more challenging for individuals on strict vegan diets to obtain adequate levels (Hariz and Bhattacharya, 2023). The current RDA for cobalamin is 1.5µg a day for both men and women (NHS, 2023). To reach these levels, individuals reducing or eliminating the consumption of animal products may require vitamin supplementation or consumption of fortified foods.

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Cobalamin and the nervous system

The role of cobalamin in the functioning of the central nervous system includes conversion through the methionine synthase pathway of the amino acid homocysteine to methionine, an amino acid vital for DNA and RNA synthesis. A deficiency of cobalamin can impair DNA and RNA synthesis, therefore, causing homocysteine to accumulate at the expense of methionine. This leads to abnormalities in the myelin-producing oligodendrocytes of the nervous system (Bagur et al, 2017).

Transportation and absorption of cobalamin

Cobalamin binds to intrinsic factor (IF), a small glycoprotein produced by gastric parietal cells of the stomach (Fig 4). Binding of cobalamin to IF occurs within the duodenum and jejunum of the small intestine, before IF acts to facilitate the subsequent absorption of cobalamin in the ileum (Calderón-Ospina and Nava-Mesa, 2020) (Fig 4). The liver stores approximately 2-3mg of cobalamin, which is enough to sustain the body for 2-4 years.

Cobalamin is also absorbed and carried by complex mechanisms involving other transport proteins, such as haptocorrin (HC) (Fig 4) and transcobalamin (TC), and their respective membrane receptors (Guéant et al 2022).

Cobalamin deficiency

The primary cause of cobalamin deficiency is pernicious anaemia, an autoimmune disorder that leads to gastric atrophy and reduced production of IF (Toh, 2014). People with certain medical conditions, who have undergone gastrointestinal surgeries (gastrectomy, ilial resection) or have chronic inflammation (ileitis) that affect nutrient absorption may also have increased risk of cobalamin deficiency (Montoro-Huguet et al, 2021).

Other causes for impaired cobalamin absorption include Zollinger-Elliston syndrome, blind loop syndrome, fish tapeworm infestation and pancreatic insufficiencies (Hariz and Bhattacharya, 2023).

The classical treatment of pernicious anaemia includes administration of B12 through the intramuscular route every other day for 1-2 weeks, followed by weekly injections for a month, then adjusted to monthly injections for life.

As with vitamin B9 deficiency, vitamin B12 deficiency is also associated with megaloblastic anaemia due to the vitamin's vital role in DNA synthesis (Socha et al, 2020). Multiple sclerosis has also been linked with vitamin B12 deficiency due to impaired DNA synthesis in the myelin-producing oligodendrocytes (Calderón-Ospina and Nava-Mesa, 2020; Bagur et al, 2017).

Conclusion

Vitamin B complex includes key B vitamins that play important roles in the body. Generally, most people can get sufficient levels of B vitamins in their diet. However, certain conditions can increase the risk of deficiency, including dietary choices, medical conditions, medications, alcohol dependency and genetics. Vitamin B deficiencies, when they do occur, can cause significant health problems.

The next article in the series will examine vitamin D, commonly known as the 'sunshine vitamin'. **NT**

References

Aljaadi AM et al (2022) Riboflavin intake and status and relationship to anemia. *Nutrition Reviews*; 81: 1, 114–132.
Bagur MJ et al (2017) Influence of diet in multiple sclerosis: a systematic review. *Advances in Nutrition*; 8: 3, 463–472.
Calderón-Ospina CA, Nava-Mesa MO (2020) B vitamins in the nervous system: current knowledge of the biochemical modes of action and synergies of thiamine, pyridoxine, and cobalamin. *CNS Neuroscience & Therapeutics*; 26: 1, 5–13.
Chang CL, Lin CM (2011) Eye-of-the-tiger sign is not pathognomonic of pantothenate kinase-associated neurodegeneration in adult cases. *Brain and Behaviour*; 1: 1, 55–56.
Chou WP et al (2019) Thiamine for preventing dementia development among patients with alcohol use disorder: a nationwide population-based cohort study. *Clinical Nutrition*; 38: 3, 1269–1273.
Ferrell M et al (2024) A terminal metabolite of niacin promotes vascular inflammation and contributes to cardiovascular disease risk. *Nature Medicine*; 30:2, 424-434.
Franco CN et al (2022) Simplifying the B Complex: how Vitamins B6 and B9 modulate one carbon metabolism in cancer and beyond. *Metabolites*; 12: 10, 961.
Gheita AA et al (2020) The potential role of B5: a stitch in time and switch in cytokine. *Phytotherapy Research*; 34: 2, 306–314.
Guéant JL et al (2022) Vitamin B12 absorption and malabsorption. *Vitamins and Hormones*; 119: 241–274.
Hariz A, Bhattacharya PT (2023) Megaloblastic anemia. In *StatPearls*. StatPearls Publishing.
Harvard TH Chan School of Public Health (2023) Riboflavin – vitamin B2, *hsph.harvard.edu*, March (accessed 25 January 2024).
Hayashi A et al (2017) Intestinal dysbiosis and biotin deprivation induce alopecia through overgrowth of Lactobacillus murinus in mice. *Cell Reports*; 20: 7, 1513–1524.
Hemminger A, Wills BK (2023) Vitamin B6 toxicity. In: *StatPearls*. StatPearls Publishing.
Jakubowski H (2019) Homocysteine modification in protein structure/function and human disease. *Physiological Reviews*; 99: 1, 555–604.
Jankovic J et al (2021) *Bradley and Daroff's Neurology in Clinical Practice*. Elsevier.
Kassem H et al (2014) Biotin-responsive basal ganglia disease: neuroimaging features before and after treatment. *American Journal of Neuroradiology*; 35: 10, 1990–1995.
Kurian MA, Hayflick SJ (2013) Pantothenate kinase-associated neurodegeneration (PKAN) and PLA2G6-associated neurodegeneration (PLAN): review of two major neurodegeneration with brain iron accumulation (NBIA) phenotypes. *International Review of Neurobiology*; 110, 49–71.
Mahabadi N et al (2023) Riboflavin deficiency. In: *StatPearls*. StatPearls.
Makarov MV et al (2019) The chemistry of the vitamin B3 metabolome. *Biochemical Society Transactions*; 47: 1, 131–147.
Martel JL et al (2022) Vitamin B1 (Thiamine). In: StatPearls. StatPearls Publishing.
Merrell BJ, McMurry JP (2022) Folic Acid. In: StatPearls. StatPearls Publishing.
Mikkelsen K et al (2016) The effects of vitamin B in depression. *Current Medicinal Chemistry*; 23: 38, 4317–4337.
Montoro-Huguet MA et al (2021) Small and large intestine (I): malabsorption of nutrients. *Nutrients*; 13: 4, 1254.
National Institute of Diabetes and Digestive and Kidney Diseases (2021) Vitamin B. In: *LiverTox: Clinical and Research Information on Drug-Induced Liver Injury*. National Institute of Diabetes and Digestive and Kidney Diseases.
NHS (2023) B vitamins and folic acid, *nhs.uk*, (accessed 25 January 2024).
National Institutes of Health (2022a) Niacin – health professional fact sheet, *nih.gov* 18 November (accessed 25 January 2024).
National Institutes of Health (2022b) Folate – health professional factsheet, *nih.gov*, 30 November (accessed 25 January 2024)
Patel DP et al (2017) A review of the use of biotin for hair loss. *Skin Appendage Disorders*; 3: 3, 166–169.
Peechakara BV, Gupta M (2022) Vitamin B2 (Riboflavin). In *StatPearls*. StatPearls Publishing.
Pei P et al (2019) Folate deficiency induced H2A ubiquitination to lead to downregulated expression of genes involved in neural tube defects. *Epigenetics & Chromatin*; 12: 1, 69.
Ross AC et al (2020) *Modern Nutrition in Health and Disease*. Jones & Bartlett Learning.
Saleem F, Soos MP (2023) Biotin deficiency. In: *StatPearls*. StatPearls Publishing.
Sanvictores T, Chauhan S (2023) Vitamin B5 (pantothenic acid). In: *StatPearls*. StatPearls Publishing.
Shible AA et al (2019) Dry beriberi due to thiamine deficiency associated with peripheral neuropathy and Wernicke's encephalopathy mimicking Guillain-Barré syndrome: a case report and review of the literature. *American Journal of Case Reports*; 20: 330-334.
Sirithanakorn C, Cronan JE (2021) Biotin, a universal and essential cofactor: synthesis, ligation and regulation. *FEMS Microbiology Reviews*; 45: 4,
Socha DS et al (2020) Severe megaloblastic anemia: vitamin deficiency and other causes. *Cleveland Clinic Journal of Medicine*; 87: 3, 153–164.
Stach K et al (2021) Vitamin B6 in health and disease. *Nutrients*; 13: 9, 3229.
Suormala T et al (2002) Biotin-dependent carboxylase activities in different CNS and skin-derived cells, and their sensitivity to biotin-depletion. *International Journal for Vitamin and Nutrition Research*; 72: 4, 278–286.
Tardy AL et al (2020) Vitamins and minerals for energy, fatigue and cognition: a narrative review of the biochemical and clinical evidence. *Nutrients*; 12: 1, 228.
Toh BH (2014) Diagnosis and classification of autoimmune gastritis. *Autoimmunity Reviews*; 13: 4-5, 459–462.
Vale AHF et al (2022) Riboflavin did not provide anti-inflammatory or antioxidant effects in an experimental model of sepsis. *Brazilian Journal of Medical and Biological Research*; 55: e12107.
Wiley KD, Gupta M (2023) Vitamin B1 (thiamine) deficiency. In: *StatPearls*. StatPearls Publishing.