

# Vitamin B-complex deficiency, supplementation and management

G Schellack<sup>1</sup>, P Harirari<sup>2</sup>, N Schellack<sup>2</sup>

<sup>1</sup>Pharmaceutical Industry (Clinical Research)

<sup>2</sup>School of Pharmacy, Faculty of Health Sciences, Sefako Makgatho Health Sciences University

**Corresponding author:** Prof Natalie Schellack (natalie.schellack@smu.ac.za)

## Abstract

Vitamins are either fat-soluble or water-soluble micronutrients that are derived from a healthy, well-balanced diet. The B-complex vitamins are well-known examples of such water-soluble nutrients that are readily absorbed from a healthy gut, and easily eliminated via renal excretion. They are required for their vital physiological functions and are significant contributors towards maintaining optimal health. Multiple B-vitamin deficiencies are quite commonly seen and therefore a balanced diet, including a full spectrum of B-vitamins, is usually needed when any of them is found to be deficient. Conversely, their therapeutic value will be limited to supplementation during states of deficiency, since they have no additional benefits in the presence of an adequate dietary intake. In general, their active supplementation should only be used to correct deficiencies. This article provides an overview of such B-complex vitamin deficiencies and their supplementation.

**Keywords:** vitamin B, B-complex vitamins, thiamine, riboflavin, niacin, pyridoxine, pantothenic acid, biotin, inositol, folic acid, folate, cobalamin, Wernicke-Korsakoff syndrome, beriberi, pellagra, pernicious anaemia, megaloblastic anaemia, intrinsic factor, haematinic, peripheral neuropathy

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## Introduction

Vitamins are essential nutrients that may be sourced from food. They are required for their vital physiological functions and are significant contributors towards maintaining optimal health.<sup>1</sup> The B-group (or B-complex) vitamins, specifically, assist with the production of energy and the biosynthesis of many physiologically vital molecules in cells. They are water-soluble and are not stored in the body, therefore requiring daily replenishment from dietary intake.<sup>1</sup> Cereals, mainly in their purest and unrefined form, are a common source of these vitamins.<sup>2</sup> However, the commonly-encountered practice of excessively refining and polishing cereals does strip them of significant amounts of vitamin B.<sup>3</sup> Multiple B-vitamin deficiencies are quite commonly seen and therefore a balanced diet, including a full spectrum of B-vitamins, is usually needed when any of them is found to be deficient.<sup>4</sup> In other words, if at least one B-vitamin is found to be deficient, the full spectrum should be considered to be deficient (unless proven otherwise).

## What are vitamins?

The vitamins are organic compounds that are characterised by high levels of potency and are therefore only required in very small amounts. They are classified as nutrients, and most of them are obtained via dietary intake (hence the reason why several vitamins may become depleted in patients with a poor nutritional status). Vitamin B<sub>12</sub>, for example, may be viewed as the most potent nutrient known to us.<sup>5</sup> There are some noteworthy exceptions

that may, however, be synthesised in the human body, such as vitamin D, K and a few other examples. Some of the B-vitamins may also be synthesised by intestinal bacteria (but in insufficient quantities). The vitamins are typically categorised as either being water-soluble or fat-soluble, as shown in Table I.<sup>5,6</sup>

The vitamins are essential for normal growth, metabolism and reproduction. However, their therapeutic value will be limited to supplementation during states of deficiency, since they have no additional benefits in the presence of an adequate dietary intake. In general, their active supplementation should only be used to correct deficiencies, after which a well-balanced diet should be able to provide all the necessary nutrients.<sup>5</sup>

As already mentioned, the B-complex vitamins are water-soluble, and as for the other vitamins in this group, they are easily eliminated from the body via renal excretion. The typical members of this complex are thiamine (vitamin B<sub>1</sub>), riboflavin (vitamin B<sub>2</sub>), niacin (vitamin B<sub>3</sub>), pantothenic acid (vitamin B<sub>5</sub>), pyridoxine (vitamin B<sub>6</sub>), biotin (vitamin B<sub>7</sub>), folic acid (vitamin B<sub>9</sub>) and cobalamin (vitamin B<sub>12</sub>).<sup>6</sup> Some authors also include inositol, para-aminobenzoic acid and choline in this group.<sup>7</sup> Amongst a diverse array of functions, they act as important co-factors or co-enzymes in a wide variety of metabolic processes in the body (see text below). It is noteworthy that many different vitamins play a role in the formation and normal functioning of red blood cells.<sup>8</sup> Furthermore, vitamin C and E act as antioxidants, whilst vitamin A and D act as hormones.<sup>5</sup>

**Table I.** The water-soluble *versus* fat-soluble vitamins<sup>6,7</sup>

Water-soluble vitamins	Fat-soluble vitamins
B-complex: Vitamin B <sub>1</sub> (thiamine) Vitamin B <sub>2</sub> (riboflavin) Vitamin B <sub>3</sub> (niacin) Vitamin B <sub>5</sub> (pantothenic acid) Vitamin B <sub>6</sub> (pyridoxine) Vitamin B <sub>12</sub> (cobalamin) Folic acid (vitamin B <sub>9</sub> ) Para-aminobenzoic acid (PABA) Choline Inositol (vitamin B <sub>8</sub> ) Biotin (vitamin B <sub>7</sub> ) Vitamin C (ascorbic acid)	Vitamin A (retinol) Vitamin D (ergocalciferol, cholecalciferol) Vitamin E ( $\alpha$ -tocopherol) Vitamin K (phytonadione)
<b>Of note:</b> The water-soluble vitamins are readily absorbed from the GIT and merely require passage in conjunction with water (the notable exception is vitamin B <sub>12</sub> , which also needs to bind to <i>intrinsic factor</i> that is derived from the stomach). These vitamins are excreted in the urine and are not really stored in the body in any significant quantities. Hypervitaminosis is therefore quite rare.	<b>Of note:</b> These vitamins require binding to ingested lipids to facilitate their absorption from the GIT. It is important to note that any condition that therefore interferes with fat absorption, will necessarily also interfere with the absorption of the fat-soluble vitamins. These vitamins are stored in the body (with the noteworthy exception of vitamin K), and toxicities are well-known (e.g. hypervitaminosis A).

[GIT: gastrointestinal tract]

## The various B-vitamins

### Vitamin B<sub>1</sub> (thiamine):

Thiamine is essential for glucose metabolism in the body.<sup>9</sup> It functions as a co-enzyme in the metabolism of carbohydrates and branched-chain amino acids.<sup>2</sup> It is also essential for normal appetite and plays a crucial role in maintaining proper functioning of the nervous system.<sup>1</sup>

A thiamine deficiency may manifest itself in any of the following<sup>2,10</sup>:

- Beriberi, which can be wet (oedematous) or dry (paralytic)
- Wernicke's encephalopathy, which is characterised by confusion, ataxia and nystagmus
- Korsakoff's psychosis, which is characterised by confabulation, lack of insight, retrograde and anterograde amnesia, and apathy.<sup>10</sup> The latter two are also referred to as the Wernicke-Korsakoff syndrome. Korsakoff's psychosis is associated with chronic alcoholism, following a thiamine deficiency in these patients.<sup>5</sup>

Treatment would invariably be via thiamine supplementation (additional details are shown in Table II).<sup>11</sup>

### Vitamin B<sub>2</sub> (riboflavin):

Riboflavin functions as a co-enzyme in several oxidation and reduction reactions in the body.<sup>2</sup> It is essential for the release of energy from food and the conversion of the amino acid tryptophan into niacin.<sup>1</sup>

A riboflavin deficiency may lead to cheilosis (fissuring and dry scaling of the vermilion surface of the lips and angles of the mouth), angular stomatitis (inflammation of the corners of the mouth, associated with a wrinkled or fissured epithelium that

does not involve the mucosa), and dermatitis (inflammation of the skin), mucosal crusts and even a magenta-coloured tongue.<sup>2,5</sup>

Treatment is usually via riboflavin supplementation (also refer to Table II).

### Vitamin B<sub>3</sub> (niacin):

Also known as nicotinic acid, niacin is a vitamin that is mainly involved in energy production, normal enzyme functioning, digestion, the promotion of normal appetite, healthy skin, and nerves.<sup>1</sup> It functions as a co-substrate or co-enzyme for hydrogen transfer with several dehydrogenases.<sup>2</sup>

A niacin deficiency may result in pellagra, which is characterised by dermatitis, diarrhoea and dementia (the so-called three D's of pellagra).<sup>5,12</sup> For treatment, nicotinamide is usually used, because unlike nicotinic acid (the most common form of niacin), it does not cause flushing, itching, burning, or tingling sensations (due to intense vasodilatation). (Also refer to Table II.)<sup>4,5</sup>

### Vitamin B<sub>5</sub> (pantothenic acid):

Pantothenic acid is important for the production of energy, hormone synthesis and the metabolism of fats, proteins, and carbohydrates.<sup>1</sup> It functions as a component of co-enzyme A and phosphopantetheine, and is involved in fatty acid metabolism.<sup>2</sup>

A Vitamin B<sub>5</sub> deficiency occurs very rarely, but if it does occur, it is typically accompanied by deficits of other nutrients.<sup>4</sup> Deficiency is usually characterised by fatigue, sleep disturbances, impaired coordination, irascibility (bad temper), postural hypotension, rapid heart rate on exertion, epigastric distress with anorexia and constipation, nausea, as well as numbness and tingling of the hands and feet (so-called burning feet syndrome).<sup>2</sup> The recommended daily allowance has not been established with

certainty, but 5–10 mg daily (provided by a balanced diet) is probably adequate (also see Table II).<sup>13</sup>

### **Vitamin B<sub>6</sub> (pyridoxine):**

Pyridoxine, also known as pyridoxal phosphate or pyridoxamine, is required for the synthesis of the neurotransmitters, serotonin and noradrenaline, and for myelin formation.<sup>14</sup> It functions as a co-enzyme (as pyridoxal phosphate) in the metabolism of amino acids, glycogen, and sphingoid bases.<sup>2</sup>

A Vitamin B<sub>6</sub> deficiency is also rarely seen and is usually associated with a deficiency in other B-complex vitamins.<sup>14</sup> Deficiency is associated with anaemia, abnormalities on the electroencephalograph (EEG), dermatitis with cheilosis and glossitis (swollen tongue), naso-lateral seborrhoea, depression and confusion, weakened immune function and peripheral neuropathy (and seizures, especially in infants, which may be refractive to treatment with anticonvulsive agents); as well as normocytic, microcytic or sideroblastic anaemia.<sup>2</sup>

Treatment usually involves the elimination of risk factors, where possible, and administering pyridoxine (additional details are provided in Table II).

### **Vitamin B<sub>7</sub> (biotin):**

Biotin is required for the release of energy from carbohydrates and for the metabolism of fats, proteins and carbohydrates.<sup>1</sup> Vitamin B<sub>7</sub> functions as a co-enzyme in bicarbonate-dependent carboxylation reactions.<sup>2</sup> Deficiency presents with fatigue, conjunctivitis, alopecia, nausea, dermatitis (erythematous and seborrheic type), muscular pains and central nervous system abnormalities (such as hypotonia, lethargy and stunted development in children, and depression, hallucinations, and paraesthesia of the extremities in adults).<sup>2</sup>

Treatment is usually with biotin supplementation, and in cases where there is a secondary cause, e.g. eating raw eggs (due to the fact that avidin, a glycoprotein contained in raw egg white, binds to biotin and effectively prevents its absorption from the gastrointestinal tract) or anticonvulsant therapy (e.g. phenobarbital, phenytoin, primidone, and others), the secondary cause should be eliminated where possible or substituted, in the case of anticonvulsant therapy (also refer to Table II).<sup>15,16</sup>

### **Vitamin B<sub>8</sub> (inositol):**

Strictly speaking, inositol is not a true vitamin, because it is biosynthesised in the body.<sup>17</sup> It is involved in cell membrane synthesis, the maintenance of healthy hair, the control of oestrogen levels and in cholesterol metabolism.<sup>17</sup> Deficiency, although rare, may result in atherosclerosis, alopecia, eczema, increased blood cholesterol levels, skin disorders, eye disorders, declining brain function, mood swings and constipation.<sup>7</sup> Treatment is with the recommended dietary allowance (which is quite difficult to establish) of about 1 000 mg (also see Table II).<sup>18</sup>

### **Vitamin B<sub>9</sub> (folic acid):**

Folic acid, also known as folate or folacin, is a vitamin that assists with protein metabolism, the promotion of red blood cell formation and maturation (i.e. folic acid is a haematinic, together with iron, vitamin B<sub>6</sub>, vitamin B<sub>12</sub>, ascorbic acid and epoietin), and the synthesis of purines and pyrimidines.<sup>4,5</sup> It also lowers the risk for neural tube birth defects (e.g. spina bifida) and helps control homocysteine levels, thus potentially reducing the risk for coronary heart disease.<sup>1</sup> Deficiency causes megaloblastic anaemia, which cannot be distinguished from that caused by a vitamin B<sub>12</sub> deficiency.<sup>4</sup> Maternal deficiency increases the risk of neural tube birth defects (there is an increased demand for folate during pregnancy). Folic acid is converted to folinic acid, a metabolically-active folate, which may also be used to treat folic acid deficiency in patients taking dihydrofolic acid reductase inhibitors such as methotrexate, trimethoprim and pyrimethamine.<sup>5</sup> General treatment is with folate supplements (also refer to Table II).

### **Vitamin B<sub>12</sub> (cobalamin):**

Cobalamin is another vital haematinic agent. It is also important in nucleic acid metabolism, methyl transfer, as well as myelin synthesis and repair.<sup>1</sup> Deficiency most commonly affects strict vegetarians, infants of vegan mothers, and the elderly.<sup>1</sup> It is characterised by fatigue, megaloblastic anaemia, neurological disorders and the degeneration of nerves, which results in peripheral neuropathy (numbness and tingling).<sup>4</sup> Treatment is with suitable cobalamin supplementation, for which hydroxocobalamin is preferred over cyanocobalamin, because the former is excreted more slowly than the latter (also refer to Table II).<sup>4,5</sup>

Table II provides a summary of B-complex vitamin functions, deficiencies and the treatment thereof. Also refer to Figure 1.

### **A few important points to ponder**

A specific B-vitamin deficiency is not likely to occur in isolation. Where megaloblastic anaemia is detected, both vitamin B<sub>12</sub> and folic acid should be supplemented. Drugs that could deplete folic acid include methotrexate, trimethoprim and pyrimethamine; those that could potentially hamper its absorption from the gut include cholestyramine and certain anticonvulsants (such as phenytoin). Folic acid is essential for normal cell proliferation and erythropoiesis, and an increased demand exists during pregnancy.<sup>5,8,13</sup>

In patients who develop pernicious anaemia, secondary to a deficiency in intrinsic factor (and therefore also in vitamin B<sub>12</sub>), life-long parenteral supplementation will be required. Lastly, vitamin B<sub>6</sub> prophylaxis is recommended for high-risk patients (i.e. high risk for the development of neurotoxicity) who need to take isoniazid (INH) to treat their tuberculosis (TB). Such patients include those who are malnourished, alcoholics, pregnant, or those who suffer from diabetes mellitus or HIV-infection. In these patients 10–25 mg per day may be sufficient; however, higher dosages need to be considered for those who already have an established neuropathy.<sup>5,13</sup>

**Table II.** Summary of B-complex vitamin functions, deficiencies and their treatment<sup>4,5,11,13,15,18</sup>

Vitamin	Functions	Results of a deficiency	Treatment of deficiency
Vitamin B <sub>1</sub> (Thiamine)	Functions as a co-enzyme in the metabolism of carbohydrates and branched-chain amino acids in the body; it catalyses the decarboxylation of $\alpha$ -keto acids.	Beriberi, polyneuritis, and Wernicke-Korsakoff syndrome.	Thiamine is given as follows: <ul style="list-style-type: none"> <li>For mild polyneuropathy: 10–20 mg orally, once daily for two weeks.</li> <li>For moderate or advanced neuropathy: 20–30 mg once daily, continued for several weeks after symptoms disappear.</li> <li>For congestion and oedema due to cardiovascular beriberi: 100 mg intravenously once daily for several days.</li> <li>For Wernicke-Korsakoff syndrome: 50–100 mg intramuscularly or intravenously, twice daily, usually for several days, followed by 10–20 mg once daily until a therapeutic response is obtained.<sup>11</sup></li> </ul> <p><b>Note:</b> Neuropathy will not respond to treatment if the nerve cells have died off. Prophylactic thiamine should be used in patients with chronic diarrhoea and those that have undergone intestinal resection.<sup>5</sup> Give 100 mg of thiamine intravenously <b>before administering intravenous glucose*</b> to alcoholic patients, or any other patients that are at risk of having a thiamine deficiency.<sup>11</sup></p>
Vitamin B <sub>2</sub> (Riboflavin)	Functions as a co-enzyme in several oxidation and reduction reactions in the body.	Cheilosis, angular stomatitis, lip fissures and dermatitis (fissures may become infected with <i>Candida albicans</i> ), normochromic-normocytic anaemia.	Riboflavin 5–10 mg orally, once daily is given until recovery. Other B-vitamins should also be administered. <sup>11</sup> Dosages up to 50 mg orally or intravenously may need to be considered. <sup>5</sup>
Vitamin B <sub>3</sub> (Niacin/nicotinic acid/nicotinamide)	Functions as a co-substrate/co-enzyme for hydrogen transfer with several dehydrogenases.	Pellagra with dermatitis, diarrhoea and dementia; as well as secondary to diarrhoea, cirrhosis of the liver, alcoholism and carcinoid syndrome.	Nicotinamide 250–500 mg daily, orally, in divided dosages 3 to 4 times a day, or 100 mg 8-hourly. <sup>5,11</sup>
Vitamin B <sub>5</sub> (Pantothenic acid)	Functions as a component of co-enzyme A and phosphopantetheine and is involved in fatty acid metabolism.	Fatigue, sleep disturbances, impaired coordination and nausea.	The recommended daily allowance has not been established with certainty, but 5–10 mg daily (provided by a balanced diet) is probably adequate. <sup>13</sup>
Vitamin B <sub>6</sub> (Pyridoxine/pyridoxal phosphate/pyridoxamine)	Functions as a co-enzyme (pyridoxal phosphate) in the metabolism of amino acids, glycogen, and sphingoid bases.	Naso-lateral seborrhoea, glossitis, and peripheral neuropathy (and epileptiform convulsions, especially in infants, which may be refractive to treatment with anticonvulsive agents); as well as normocytic, microcytic or sideroblastic anaemia.	<ul style="list-style-type: none"> <li>For secondary deficiency, specific causes (e.g. the use of pyridoxine-inactivating drugs, such as isoniazid, malabsorption, etc.) should be corrected.<sup>4</sup></li> <li>Pyridoxine 50–100 mg orally, once daily.</li> <li>Dosages may, however, be as low as 2 mg per day.<sup>5</sup></li> <li>For deficiency due to increased metabolic demand, amounts larger than the daily recommended intake may be required.<sup>4</sup></li> <li>For most cases of inborn errors of metabolism, high dosages of pyridoxine may be effective.<sup>4</sup></li> </ul>
Vitamin B <sub>7</sub> (Biotin)	Co-enzyme functions in bicarbonate-dependent carboxylation reactions.	Fatigue, conjunctivitis, alopecia, nausea, dermatitis, muscular pains, depression, hallucinations and paraesthesia of the extremities. Patients that are receiving long-term TPN† may develop an exfoliative dermatitis if biotin is not supplemented as part of their total nutrition.	<ul style="list-style-type: none"> <li>Biotin 150 <math>\mu</math>g intramuscularly, once daily – symptoms should begin to resolve within 3–5 days and should basically be absent within 3–5 months.</li> <li>Biotin 5–20 mg per day – with larger dosages, symptoms may resolve much faster.<sup>15</sup></li> </ul>
Vitamin B <sub>8</sub> (Inositol)	Functions include cell membrane synthesis, maintenance of healthy hair, control of oestrogen levels and cholesterol metabolism.	Atherosclerosis, alopecia, eczema, increased blood cholesterol levels, skin disorders, eye disorders, declining brain function, mood swings and constipation.	Treatment is difficult to establish as inositol is not really recognised as a vitamin, but a recommended dietary allowance of about 1 000 mg can be given. <sup>18</sup>

Vitamin B <sub>9</sub> (Folic acid/ folate/folacin)	Functions include aiding protein metabolism, promoting red blood cell formation and maturation, synthesis of purines and pyrimidines. Folic acid is converted to folinic acid.	Megaloblastic anaemia and neural tube defects.	Folate 400–1000 µg orally, once daily, with the normal requirement being 400 µg per day. <sup>4</sup>
Vitamin B <sub>12</sub> (Cobalamin)	Functions include aiding in nucleic acid metabolism, methyl transfer, myelin synthesis and repair, as well as red blood cell production.	Megaloblastic anaemia, fatigue, neurological disorders and the degeneration of nerves (resulting in peripheral neuropathy)	<ul style="list-style-type: none"> <li>• Cobalamin 1000–2000 µg orally, once daily to patients who do not have severe deficiency or neurologic symptoms or signs.</li> <li>• For severe deficiency, cobalamin 1 mg intramuscularly, 1 to 4 times per week for several weeks until haematological abnormalities are corrected; then give once per month.<sup>4</sup></li> </ul>

[\*Intravenous glucose can worsen a thiamine deficiency; †Total parenteral nutrition.]

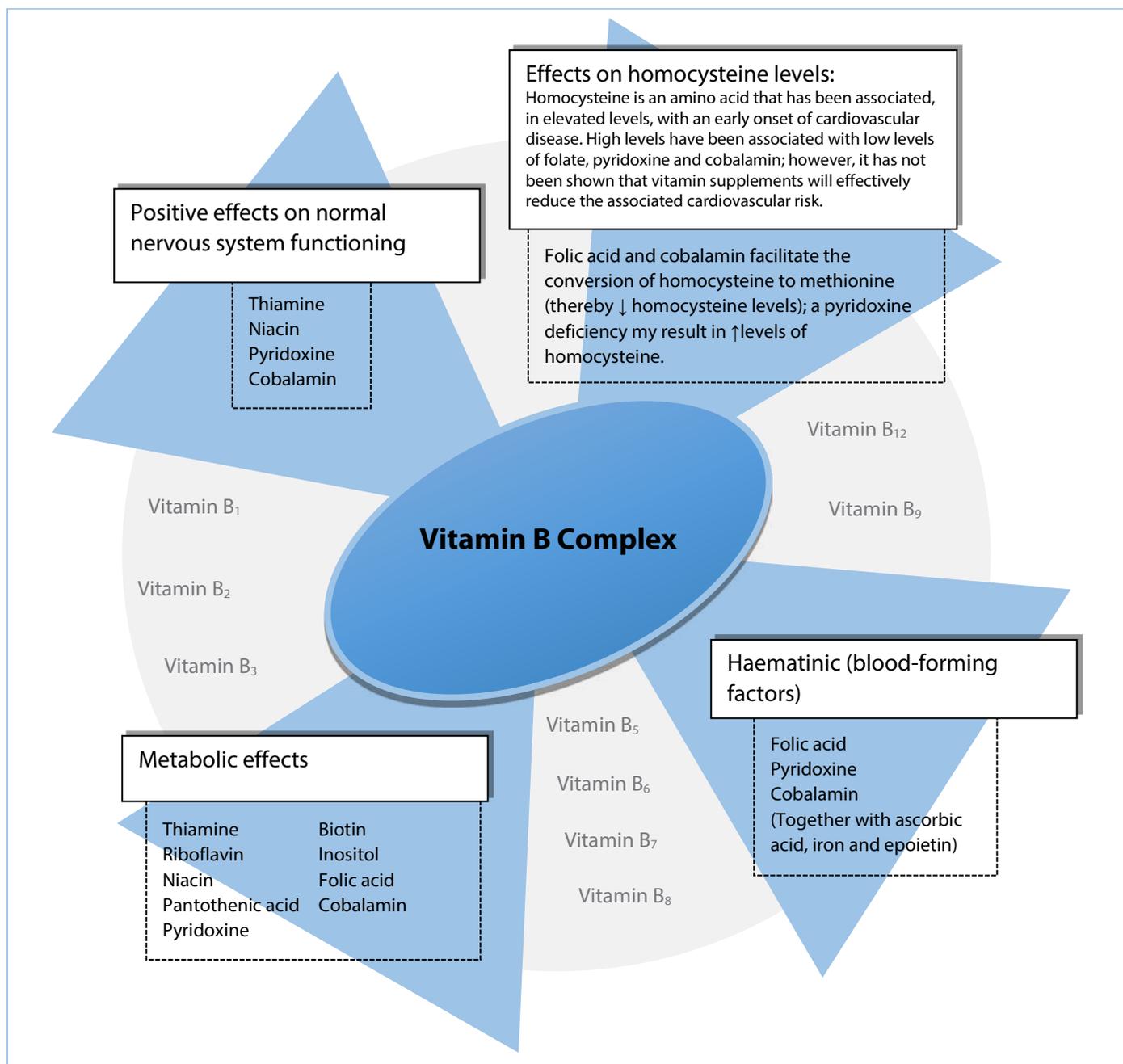


Figure 1. Simplified summary of the B-complex vitamins and their major areas of physiological functioning<sup>5,6,8</sup>

When having to decide how to treat B<sub>12</sub> deficiency with neuropsychiatric signs and symptoms, with or without anaemia or macrocytosis, it is important to remember the following<sup>19</sup>:

- B<sub>12</sub> deficiency is a multi-system, poly-glandular, multi-point poisonous syndrome. It is also called megaloblastic anaemia or pernicious anaemia.
- Where one to three body systems are affected, and blood serum B<sub>12</sub> is below the local lab threshold (200 ng/L usually) then treat as for B<sub>12</sub> deficiency without neurological involvement.
- Where one to three body systems are affected and blood serum is above the local lab threshold, then monitor the patient and review. Following specific medical history, prophylaxis may be initiated.
- When four to six body systems are affected, with a blood serum more than 350 ng/L (subtle deficiency) it is important to consider a therapeutic trial to confirm diagnosis. With blood serum less than 350 ng/L (moderate and severe deficiency), follow treatment for B<sub>12</sub> deficiency with neurological signs and symptoms.
- If more than six body systems are affected, commence treatment immediately without waiting for blood tests. Treat as B<sub>12</sub> deficiency with neurological signs and symptoms.

### The HIV and vitamin B<sub>12</sub> deficiency link

The first case of HIV was reported in 1985. The global burden of the disease is said to be about thirty-three million, with about sixty-seven percent of those affected living in sub-Saharan Africa, where malnutrition is widespread.<sup>20,21</sup> Deficiencies in water-soluble vitamins appear to occur less frequently than those of fat-soluble vitamins, and only cobalamine deficiency is associated with HIV disease progression.<sup>20,21</sup>

Vitamin B<sub>12</sub> (cobalamine), is a water soluble vitamin with a key role in the normal functioning of the brain and the nervous system, and for the formation of red blood cells. It is normally involved in the metabolism of every cell of the human body, especially affecting DNA synthesis and regulation, but also fatty acid synthesis and energy production.<sup>20,21</sup> A common manifestation of B<sub>12</sub> deficiency is macrocytic anaemia.<sup>20,21</sup>

The small intestine plays an important role in digestion and absorption of nutrients. It is lined with a thin epithelial layer known as the mucosa.<sup>20,21</sup> The mucosa has many small protrusions called *microvilli* which essentially increase the surface area inside the gut. This allows more of the digested food to come into contact with the lining and thus more of the nutrients can be absorbed as the food passes through the small intestine.<sup>20,21</sup>

HIV can cause many secondary diseases such as HIV enteropathy.<sup>20,21</sup> HIV enteropathy is a diseased state of the intestines that occurs with no recognisable infections. The most prominent feature of this diseased state is chronic and persistent diarrhoea.<sup>20,21</sup> The exact process of HIV enteropathy is not clearly understood but analysis at the microscopic level indicates a

condition known as subtotal villous atrophy, where the irritation and inflammation of the small intestines and some other possible factors, which remain to be fully elucidated, may cause the *microvilli* to shrink. They may not disappear completely but may be shorter than normal.<sup>20,21</sup> However, some of the intestinal tissue, including parts of the *microvilli*, may already be destroyed from previous recurring intestinal infections that are more likely to occur in HIV.<sup>20,21</sup> This eventually causes the reduction of the surface area of the small intestines.<sup>20,21</sup>

The reduced surface area of the small intestines will thus cause poor absorption of vitamin B<sub>12</sub>. The resultant deficiency in vitamin B<sub>12</sub> is therefore caused by malabsorption.<sup>20,21</sup>

### Conclusion

The B-complex vitamins play a vital role in the maintenance of an optimal health status in human beings. Included within this group of water-soluble vitamins, is a diverse group of micronutrients that are normally derived from the intake of a healthy and well-balanced diet. However, when a deficiency is detected or diagnosed, the likelihood exists that multiple B-vitamins are actually lacking. The B-complex vitamins are essential for normal growth, metabolism and reproduction. However, their therapeutic value will be limited to supplementation during states of deficiency, since they have no additional benefits in the presence of an adequate dietary intake. In general, their active supplementation should only be applied to correcting actual insufficiencies. The typical members of this complex are thiamine (vitamin B<sub>1</sub>), riboflavin (vitamin B<sub>2</sub>), niacin (vitamin B<sub>3</sub>), pantothenic acid (vitamin B<sub>5</sub>), pyridoxine (vitamin B<sub>6</sub>), biotin (vitamin B<sub>7</sub>), folic acid (vitamin B<sub>9</sub>) and cobalamine (vitamin B<sub>12</sub>), although a few others, such as inositol may also be included in the list.

Several disease states are associated with one or more B-vitamin deficiency, including nutritional deficits such as beriberi and pellagra, various forms of anaemia, suppressed metabolic and nervous system functioning and a weakened immune system. Their major areas of physiological functioning therefore include metabolism, including the normal metabolic conversion of homocysteine to methionine, as well as neurological and blood-forming effects. Furthermore, a number of well-known examples of drugs that could contribute to B-vitamin depletion also exist. For example, drugs such as methotrexate and trimethoprim (as well as cholestyramine and others) may cause a folic acid deficiency. Knowledge of the B-complex vitamins will contribute towards an understanding of their role, individual functions, deficiency states and the judicious use of their supplementation.

### References

1. Bellows L, Moore R. Water-soluble vitamins: B-complex and vitamin C. Fact Sheet No. 9.312. Food and Nutrition Series | Health. Colorado State University Extension. 2012.
2. World Health Organisation (WHO) Food and Agriculture Organisation (FAO). Thiamin, riboflavin, niacin, vitamin B6, pantothenic acid and biotin: Human Vitamin and Mineral Requirements. World Health Organisation (WHO) Food and Agriculture Organisation of the United Nations. 2002.
3. Chin RL. Neuropathy and the gastrointestinal system. Available at: <http://www.neuropathy.org/site/DocServer/nutritionGI-RussellCMD.pdf?docID=1601>. [Accessed 29 March 2019].
4. Porter RS, Kaplan JL. Nutritional disorders: The Merck Manual of Diagnosis and Therapy.

- 19th edition. Whitehouse Station, New Jersey: Merck, Sharp and Dohme Corp., a subsidiary of Merck and Co, Inc. 2011.
5. Sommers De K. Sommers' Pharmacology. 1st English ed. Pretoria: De K Sommers. 2000.
  6. Marieb EN. Human Anatomy and Physiology. 5th ed. San Francisco: Benjamin Cummings. 2001.
  7. Presser A. B-Vitamins: Literature education series on dietary supplements. Huntington College of Health Sciences. 2009. Available at: <http://www.hchs.edu/literature/B-Vitamins.pdf>. [Accessed 27 March 2019].
  8. Brenner GM, Stevens CW. 2013. Pharmacology. 4th ed. Philadelphia: Saunders.
  9. Ramsey D, Muskin PR. Vitamin deficiencies and mental health: How are they linked? *Current Psychiatry*. 2013;12(1):37-43.
  10. World Health Organisation (WHO). Thiamine deficiency and its prevention and control in major emergencies. The Department of Nutrition for Health and Development of the World Health Organization. 1999.
  11. Johnson LE. The Merck Manual Professional Edition. 2014. Available at: [http://www.merck-manuals.com/professional/nutritional\\_disorders/vitamin\\_deficiency\\_dependency\\_and\\_toxicity.html](http://www.merck-manuals.com/professional/nutritional_disorders/vitamin_deficiency_dependency_and_toxicity.html). [Accessed 30 March 2019].
  12. Rull G. Pellagra. 2011. Available at: <http://www.patient.co.uk/doctor/Pellagra.htm>. [Accessed 30 March 2010].
  13. Rossiter D. South African Medicines Formulary. Rondebosch: Health and Medical Publishing Group.
  14. National Institutes of Health (NIH). Dietary supplement fact sheet: Vitamin B6. 2011. Available at: <http://ods.od.nih.gov/factsheets/VitaminB6-HealthProfessional/>. [Accessed 30 March 2019].
  15. Scheinfeld NS, Freilich SB. Biotin deficiency treatment and management: Drugs and diseases. *Medscape*. 2015. Available at: <http://emedicine.medscape.com/article/984803-treatment>. [Accessed 31 March 2019].
  16. Biotin.com. Available at: <http://www.biotin.com/neurological/biotin-supplements-can-help-counteract-epilepsy-drug-side-effects-and-reduce-seizures>. [Accessed 07 Apr 2019].
  17. Miller D. Inositol for healthy nervous system. *Disabled World*. 2013. Available at: <http://www.disabled-world.com/medical/supplements/vitamins/inositol.php>. [Accessed 31 March 2019].
  18. Health Supplements Nutritional Guide. Inositol. Health Supplements Nutritional Guide: Nutritional Health Resource. 2010. Available at: <http://www.healthsupplementsnutritionalguide.com/Inositol.html>. [Accessed 01 April 2019].
  19. Chandy J, Minney H as part of the Vitamin B12 deficiency Support Group. Protocol for excluding B12 deficiency (Megaloblastic anemia/Pernicious Anaemia) from adult and child patient presentation.
  20. Dr Chris, HIV Enteropathy (HIV/AIDS) Small Intestine, Bowel Disease, 2017, Available at: <http://www.healthype.com/hiv-enteropathy-hiv-aids-small-intestine-bowel-disease.html> [Viewed 24 April 2019].
  21. McCutchan JA, et al. Cyanocobalamin (vitamin B12) supplementation does not prevent the haematologic toxicity of AZT. Fifth International Conference AIDS, Montreal, abstract MBP 325, 1989.

## Pharmacist Available:

Industry Pharmacist in **GAUTENG** with 44 yrs experience in all aspects of production, especially tableting, GMP, RP.

Available for contract, consulting or locum positions.

**Contact** Bernard on 083 288 2476