

Chapter 8 Neuropsychiatric disorders, including dementia – prevention is better than cure

I look up to the hills, but where will my help really come from?

My help will come from the Lord, the Creator of heaven and earth.

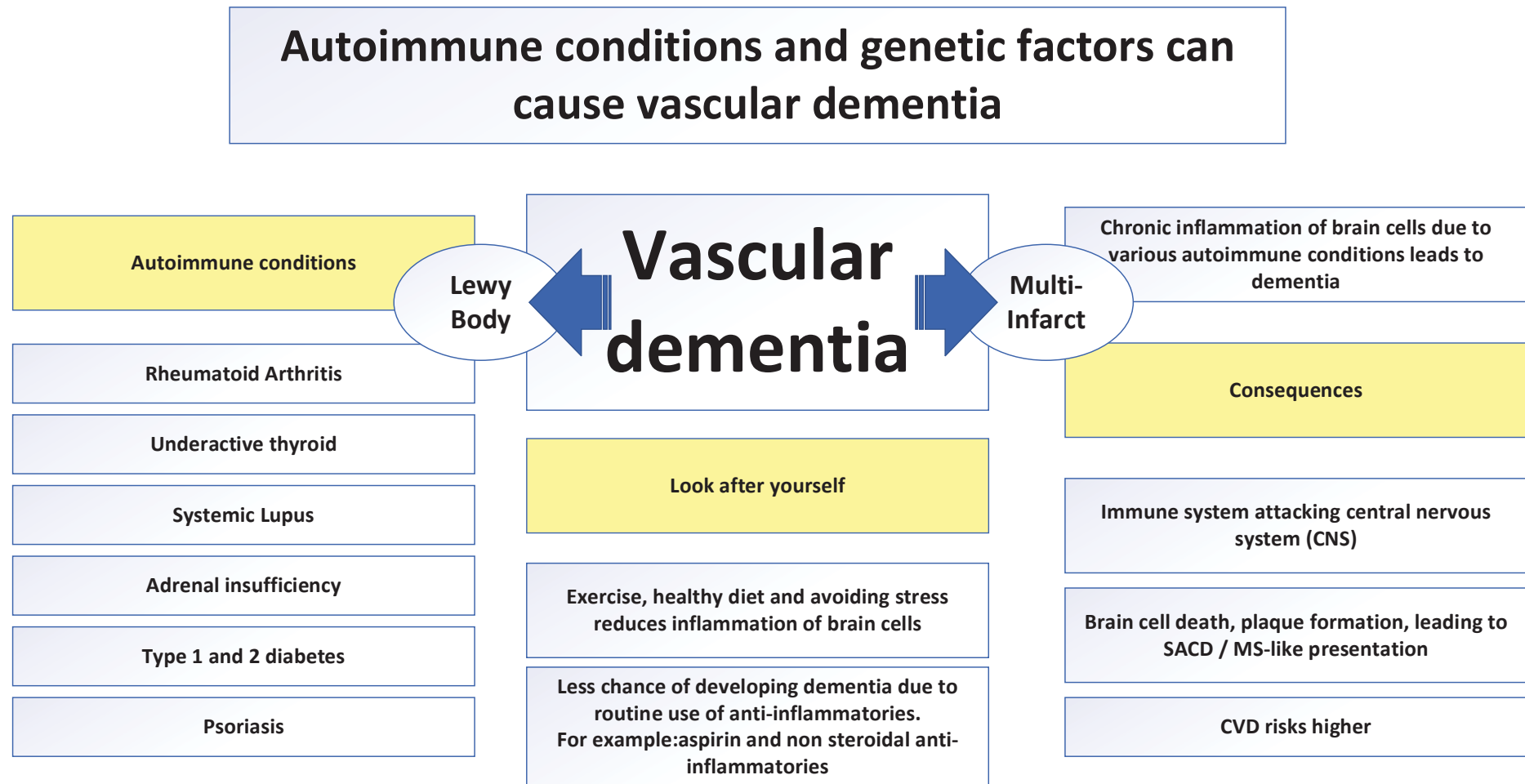
He will not let you fall. Your Protector will not fall asleep. Israel's Protector does not get tired. He never sleeps. The Lord is your Protector. The Lord stands by your side, shading and protecting you.

The sun cannot harm you during the day, and the moon cannot harm you at night. The Lord will protect you from every danger. He will protect your soul.

The Lord will protect you as you come and go, both now and forever.

Psalms 121: 1-8 ERV

Figure 8-1 Relationships between autoimmune diseases and dementia



8.1 Vitamin B12 deficiency and neuropsychiatric symptoms – an overview

Neuropsychiatric symptoms are among the most common presenting signs of vitamin B12 deficiency yet the diagnosis is often missed because of lack of awareness of this condition among clinicians (Lachner et al., 2012). In this chapter we begin by giving a brief overview of the importance of vitamin B12 for brain health and the many varied neuropsychiatric symptoms which may occur in vitamin B12-deficient patients. The remainder of the chapter focusses on the relationship of vitamin B12 to dementia, an illness of increasing worldwide concern, particularly in Western countries with ageing populations.

8.2 Historic evidence

Over sixty years ago, the neuropsychiatric effects of vitamin B12 deficiency (in the form, at that time, of pernicious anaemia) were documented in an article in the *British Medical Journal*. The author (Smith, 1960), quoting a number of authorities, stated:

“The occurrence of mental symptoms in association with pernicious anaemia has been known for many years and been commented upon by various authors (McAlpine, 1929; Samson et al., 1952; MacDonald Holmes, 1956; Wiener and Hope, 1959). Langdon (1905) drew attention to a group of cases in which nervous and mental symptoms preceded the onset of anaemia and described a great variety of neurotic and psychotic manifestations. Latterly (Wiener and Hope, 1959) the extreme variability of the symptoms has been stressed, and it is obvious that anything from a mild mood disorder to grossly psychotic behaviour may be encountered.”

Neuropsychiatric disturbances frequently occur in vitamin B12-deficient patients in the absence of anaemia or macrocytosis which have been considered the classic symptoms of B12 deficiency (see Chapter 4). In the 1980s, researchers showed that almost one-third of patients with the neuropsychiatric symptoms of vitamin B12 deficiency had no anaemia or macrocytosis (Lindenbaum et al., 1988).

8.3 Range of neuropsychiatric symptoms

More recently, researchers have described the neuropsychiatric symptoms of vitamin B12 deficiency as including “confusion, stupor, apathy, memory and judgment disorders or even psychoses, depressions and dementia” (Gröber et al., 2013). Others have listed: psychosis (with reported symptoms including suspiciousness, persecutory or religious delusions, auditory and visual hallucinations, tangential or incoherent speech, and disorganised thought-process); depression; mania; cognitive impairment (as in dementia) and delirium (fluctuating level of consciousness with attention deficits) (Lachner et al., 2012).

The close relationship between vitamin B12 deficiency and neuropsychiatric illness was also recently investigated in a retrospective study of patients who attended a specialised neuropsychiatric hospital in South India for a period of a year. Researchers found that of 259 patients who had vitamin B12 deficiency (defined as a B12 blood level less than 220 pmol/mL), 60 had neuropsychiatric symptoms. Twenty-one were diagnosed with Posterior dementias, 20 with frontotemporal dementia, seven with schizophrenia, four each with Parkinson's disease and alcohol-dependent syndromes (ADS), three with bipolar affective disorder, and one with Creutzfeldt-Jakob

disease. Eight patients also had hypothyroidism. The presenting symptoms included behavioral disturbances in 30 (50% of those with neuropsychiatric symptoms), memory loss in 20 (33.9%), and sensorimotor and movement disorders in nine (15.3%) (Issac et al., 2015).

It has also been our experience that neuropsychiatric symptoms are among the foremost clinical signs of vitamin B12 deficiency (see, for example, Case 8-1). It can be noted that many of the patients in the cases described in this book presented with psychiatric disorders. For this reason, the presence of such symptoms should alert clinicians to the possibility of vitamin B12 deficiency. If diagnosed soon enough these conditions are treatable and the symptoms reversible, as others have also found (Lachner et al., 2012).

Case 8-1 Neuropsychiatric symptoms prominent

This patient, Eddie Rooney, presented in 2011, aged 52, with dermatitis. He was referred to a dermatologist but the treatment given did not solve the problem. He also complained of foot pain and had a number of neuropsychiatric symptoms, including longstanding depression, psychosis, hallucinations and fatigue due to lack of sleep. His condition meant that he was at risk of losing both his wife and his job. Because of these symptoms and the fact that his mother was being treated with vitamin B12, I suspected B12 deficiency. Tests indicated a very low B12 level of 145 ng/L. He was started on vitamin B12-treatment therapy and all his symptoms, including the dermatitis, foot pain and neuropsychiatric problems, disappeared within a few weeks.

8.4 Vitamin B12 deficiency and the human brain

Lachner et al. (2012) explain that vitamin B12 deficiency can cause “not only brain dysfunction, but structural damage, causing neuropsychiatric symptoms via multiple pathways” and that the possible mechanisms for this include: derangements in monoamine neurotransmitter production; derangements in DNA synthesis; vasculotoxic effects and myelin lesions associated with secondary increases in homocysteine and methylmalonic acid levels, respectively. They point out that vitamin B12 deficiency may also indirectly cause a functional folate-deficiency, resulting in high homocysteine levels, decreased monoamine production, decreased S-adenosylmethionine (SAME) production, and abnormal methylation of phospholipids in neuronal membranes.

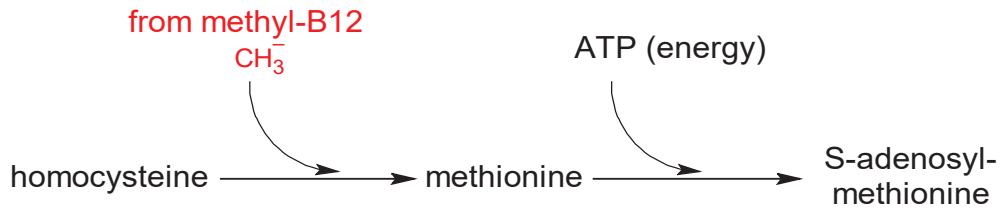
The medical textbook *Harrison’s Principles of Internal Medicine* (2018) states:

“Psychiatric disturbance ... is common in both folate and cobalamin deficiencies. This, like the neuropathy, has been attributed to a failure of the synthesis of SAM [S-adenosyl methionine], which is needed in methylation of biogenic amines (e.g., dopamine) as well as that of proteins, phospholipids, and neurotransmitters in the brain. Associations between lower serum folate or cobalamin levels and higher homocysteine levels and the development of decreased cognitive function and dementia in Alzheimer’s disease have been reported”
(Hoffbrand, 2018, p. 701)

Vitamin B12 deficiency is understood to cause demyelination of nerve axons (at least, vitamin B12 is required in order to facilitate the natural re-myelination of axons during the regular cycle of bodily repair). Demyelination may be related to a deficiency of SAME (which is required for methylation of

myelin phospholipids) which is a direct result of B12 deficiency, causing abnormal substrates for fatty acid synthesis in myelin.

Figure 8-2 Role of vitamin B12 in the conversion of homocysteine to S-AdoMet



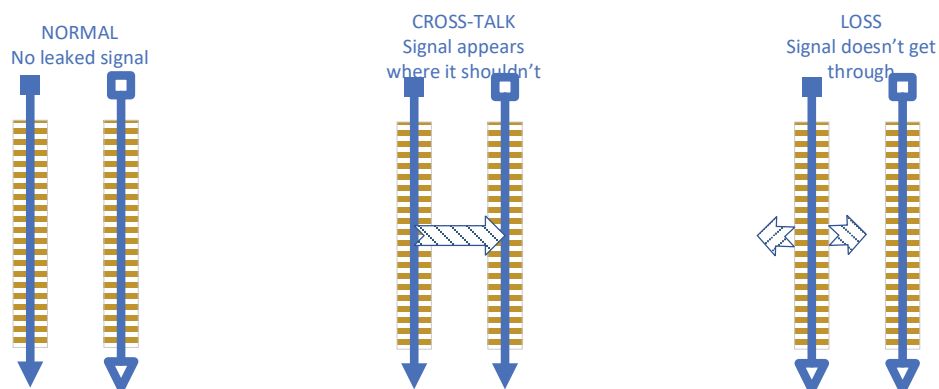
Graphic by Hugo Minney

In a discussion of the effect of B vitamins on the human brain Kennedy (2016) explains the integrated actions of B vitamins (including vitamin B12) and observes that brain concentrations of vitamin B-dependent neurochemicals are particularly high compared with amounts in the rest of the body. A deficiency is therefore likely to have a particularly strong impact on the brain which controls mood and cognition as well as most other body processes. Researchers from the US and Switzerland have found that some disorders, such as age-related cognitive and memory decline, autism and schizophrenia, could be linked to poor uptake of vitamin B12 from the blood to the brain (Zhang et al. (2016), reported in Wanjek (2016)). Further support for the effect of B vitamins on psychiatric behaviour is given in research by a team from the University of Manchester which showed that high doses (compared with low doses) of vitamins B6, B12 and biotin significantly reduced the symptoms of schizophrenia (Firth et al., 2017).

8.5 Categories of neuropsychiatric symptoms

The neuropsychiatric symptoms of vitamin B12 deficiency fall into several categories which may reflect the different functions of vitamin B12 in the human body. As described in Chapter 6, vitamin B12 is needed in both the Central Nervous System and (CNS) and Peripheral Nervous System (PNS) for the correct formation of the myelin sheath which insulates each nerve axon and ensures it sends its signal to the next neuron without “leaking” the signal to a neighbouring nerve.

Figure 8-3 nerve transmission when myelin sheath is damaged



8.5.1 Headache/confusion/psychosis

In the CNS (brain and spinal cord), symptoms that might be triggered by vitamin B12 deficiency include:

- Migrainous headache
- Tension headache
- Dizziness
- Confusion, memory disturbance/forgetfulness, fogginess
- Psychosis, hallucinations, delusion (including schizophrenia (Brown & Roffman, 2014))

8.5.2 Depression and/or anxiety

Another group of symptoms relates to depression and anxiety. These are almost polar opposites in terms of how a person feels: one induces a feeling of disconnectedness and low energy; the other makes a person feel uncomfortably alert, with heightened energy but fatigue. Patients may also experience panic attacks, mood swings and mental slowness.

One cause of depression is thought to be the build-up of homocysteine (Bottiglieri et al., 2000). As described in Chapter 1, homocysteine accumulates in the absence of vitamin B12 because of lack of availability of the B12-dependent co-enzyme methionine synthase required to convert homocysteine to methionine.

8.5.3 Irritability, snappy, disturbed sleep

Other neuropsychiatric effects of vitamin B12 deficiency include irritability and disturbed sleep. One of the first changes that patients' partners and families notice following vitamin B12-replacement therapy is that these symptoms reduce, sometimes within hours of the first injection of the loading dose.

When determining the cause of mental ill-health symptoms, the physician should take into account whether there are any obvious triggers or situations. For example, depression or anxiety may be the result of recent trauma, bereavement or stress. In order to resolve symptoms, it is often useful (although not vital) to understand the cause. If no obvious cause is present, then a physician should consider diet and in particular vitamin and mineral deficiencies, and ingestion of psychoactive (whether known or unknown) compounds.

8.6 Withdrawal of vitamin-B12 therapy may lead to psychosis

It has been our experience that people on lifelong vitamin B12-replacement therapy for neuropsychiatric conditions who stop the treatment themselves, perhaps because they think they are cured, then start to exhibit behaviours that could be considered psychotic, a "risk to themselves and to others" (Mind UK, 2017).

The result is frequently that people have their liberty taken away: they are moved from their own home to hospital, under one of the sections of the Mental Health Act 1983 (Mind UK, 2017). In some cases, Dr Chandy and the B12 Deficiency Support Group charity have been involved. The Chairman of another charity, the Pernicious Anaemia Society (PAS), Martyn Hooper, has been involved in Section 136 cases (where a person is considered to be in need of immediate care or control, in which case a police officer can take him/her to a place of safety) (Hooper, 2013, 2015).

Many other Sections of the Mental Health Act 1983 may also be invoked. The two charities have participated in giving evidence that the behavioural change may be associated with the withdrawal of vitamin B12, as this vitamin is vital for the proper functioning of the nervous system, including the brain. The charities try to have treatment restored, and where they are successful, in many cases the patient resumes their independent living and enjoyment of life.

People exhibiting psychosis with no obvious psychotropic drug ingestion should be reviewed for vitamin B12 deficiency and treated accordingly. This is especially important where they have recently had vitamin B12-replacement therapy withdrawn.

8.7 What is dementia?

“Dementia: a generalised impairment of intellect, memory and personality with no impairment of consciousness” (Simon et al., 2014, p. 1012).

In the initial stages of dementia, a patient may present to their GP complaining of “being a bit forgetful”, or their relatives may complain about their behaviour. For most patients suffering from dementia, the early symptoms are loss of short-term memory and inability to perform what would normally be simple tasks. If early symptoms are not identified, patients present later with failure to cope at home or self-neglect. However, for a definitive diagnosis of dementia to be made there must be a clear history of progressive impairment of memory and cognition, often accompanied by personality change.

Memory is the most common cognitive ability lost in dementia; 10% of people under the age of 70 years and 20-40% of people aged over 85 years have clinically identifiable memory loss. In addition to memory, other mental faculties may be affected; these include language, visuospatial ability and skills of calculation, judgement and problem-solving.

Neuropsychiatric and social deficits also arise in many dementia syndromes, resulting in depression, apathy, hallucinations, delusions, agitation, insomnia and disinhibition. The most common forms of dementia are progressive but some are static and unchanging or fluctuate from day to day or even minute to minute. Most patients with Alzheimer’s disease (AD), the most prevalent form of dementia, suffer first from memory impairment. In other dementias, such as frontotemporal dementia, memory loss is not a presenting feature (Alzheimer's Society, 2017a).

There are several forms of dementia in addition to AD: Vascular dementia (Multi-infarct, caused by multiple strokes); Dementia with Lewy bodies; Frontotemporal dementia (FTD); Mixed dementia and Young-onset dementia (Alzheimer's Society, 2017a). The different forms may share similar outcomes for those suffering and require a similar response from their carers. The worldwide incidence of all types of dementia is estimated at 50 million, with 10 million new cases a year (WHO, 2017). This combined prevalence makes the search for causes and prevention all the more important. The World Health Organisation recognises dementia as a public health priority.

GPs have a particular duty to diagnose dementia because it is so widespread, and because specific actions can be taken to make life easier for people suffering from dementia and their carers once the diagnosis has been made. In the UK in 2018, an estimated 850,000 people were living with dementia of different types, a figure which is expected to exceed 2 million by 2051 (Alzheimer's

Society, 2017b). In the US, an estimated 5.7 million people were living with AD in 2018, expected to rise to nearly 14 million by 2050 (Alzheimer's Association, 2017).

The main factor which seems to contribute to dementia is advancing age (which we cannot “cure”). However, dementia is clearly not an inevitable consequence of age – the majority of people do not suffer from it and some centenarians have intact memory function and no evidence of clinically significant dementia.

Progression of dementia (cognitive and short-term memory decline) can also be traced through microscopic and larger changes to the brain, whether at autopsy or by CT and MRI studies, which gives hope for finding a cause and ultimately a cure.

There are generalised studies which indicate some connection with vitamin B12. For example, raised homocysteine was identified by a group of experts in 2018 as a modifiable risk factor in the development of cognitive decline, dementia and AD. The group published an International Consensus Statement declaring that the functional status of three B vitamins (folate, vitamin B12 and vitamin B6) in the body, which affects levels of homocysteine, has a significant impact on whole and regional brain atrophy and cognitive decline (Smith et al., 2018). Elsewhere, also, it has been shown that vitamin B12 deficiency can lead to cognitive decline (Clarke et al., 2007) and that across an elderly population, supplementation with vitamin B12 may delay the onset of dementia and improve the outcomes (Smith et al., 2010).

8.8 The connection between vitamin B12 deficiency and dementia

There is a close relationship between symptoms of vitamin B12 deficiency and those of dementia and some cases of vitamin B12 deficiency have been mistaken for dementia. In describing one such case, the researchers note: “Along with the other cases reported in the literature, our case also proves there are some cases of vitamin B12 deficiency that can manifest with the symptoms of frontotemporal dementia and that they are completely reversible after substitution therapy” (Blundo et al., 2011). Other clinicians have reported similarly:

“Although in our patients, as well as in previously reported cases, the effects of vitamin B12 substitution cannot be positively distinguished from the effects of comedication, supporting therapeutic measures, and retest improvement, there is substantial evidence supporting the crucial involvement of vitamin B12 in several pathophysiological conditions affecting the CNS, reaching from myelination to transmitter function. Even though the causal relationship between cobalamin deficiency and dementia in individual patients is hard to prove and may often remain circumstantial, subclinical vitamin B12 deficiency, which today can be unambiguously identified, is a common condition in the elderly population. Considering the devastating impact of dementia on the quality of life of the individual and also the vast costs this often incurable condition causes, the proper diagnosis and inexpensive treatment of cobalamin deficiency should not be missed, especially in the early phases of cognitive decline (Goebels & Soyka, 2000).

We therefore thought it appropriate to compare the symptoms of the two conditions (Table 8-1), and consider whether dementia might be caused by vitamin B12 deficiency, either directly or indirectly, and therefore avoided by vitamin B12 supplementation.

Table 8-1 Comparison of characteristics of dementia and vitamin B12 deficiency

	DEMENTIA	VITAMIN B12 DEFICIENCY
<i>Symptoms</i>	Generalised impairment of intellect and personality with no impairment of consciousness. Often characterised by loss of short-term memory, confusion, impairment of cognition which can result in being unable to cope with everyday life.	Loss of short-term memory. Forgetful. Irritable. Confused. Impairment of cognition. Unable to cope.
<i>Prevalence</i>	Increases with age, although there is an early-onset form. > 60 years: prevalence from 5% > 65 years: prevalence 20%	Increases with age: more prevalent in >75 years. Vitamin B12 deficiency can be present even as early as the embryo stage unless the pregnant vitamin B12-deficient mother is prescribed both vitamin B12 and folic acid (see Chapter 5).
<i>How the brain is affected</i>	<u>Vascular (Multi-infarct) dementia</u> Causes problems with the thinking and planning faculties. Often occurs after the blood supply to the brain has been interrupted e.g. in vasculitis or after a stroke (Alzheimer's Society, 2017c). <u>Dementia with Lewy bodies</u> Symptoms often include fluctuating alertness, hallucinations and problems with movement. <u>Alzheimer's disease</u> Most common form. The main cause is still being studied. Both early onset and late onset occur.	Lack of vitamin B12 appears to cause a build-up of homocysteine and brain atrophy, accompanied by loss of cognitive function and short-term memory, and vasculitis. Supplementing with vitamin B12 for a population aged 70+ has been shown to reduce the risk (Douaud et al., 2013; Smith et al., 2010).
<i>Familial factor</i>	The main factor is age. However, Alzheimer's is known to have a genetic component (National Institute on Ageing, 2017). For example, the apolipoprotein E (APOE) gene on chromosome 19 is thought to increase risk of late-onset. APOE comes in different forms (called alleles). The most common allele,	Predisposition to vitamin B12 deficiency can be inherited – there are at least eight different transport and metabolism proteins that may be affected by variant genes. It is possible that normal function of the genes can be restored with vitamin

Table 8-1 Comparison of characteristics of dementia and vitamin B12 deficiency

	DEMENTIA	VITAMIN B12 DEFICIENCY
	APOEε3, is thought to be neutral to Alzheimer's. However, APOEε4 increases risk and APOEε2 may be protective.	B12 supplementation over one or two generations
<i>Reversible decline</i>	Hard to reverse but early diagnosis and effective treatment of coexisting autoimmune conditions can slow down further deterioration of this condition.	Periodic screening in suspected cases results in early diagnosis. Commencing replacement treatment at the outset prevents further deterioration and in many cases maximum reversal can be achieved.
<i>Central Nervous System (CNS) link</i>	Dementia is defined by the effect on the CNS (brain and cognitive ability).	Both the CNS and the PNS are affected by vitamin B12 deficiency.
<i>Triggers</i>	Genetic factors are likely to be important. However, inflammation, particularly inflammation caused by autoimmune disease, may be a major trigger (Coghlan; Wotton & Goldacre, 2017). People on anti-inflammatory medication (e.g. for arthritis) have reduced levels of dementia.	Vitamin B12 deficiency may cause autoimmune conditions (see Chapter 7) and therefore resulting inflammation.
<i>Associations</i>	Link between autoimmune disease and heart and circulatory problems.	Vitamin B12 deficiency causes elevated homocysteine levels which are strongly associated with heart and circulatory disorders.

8.9 Links with other conditions

8.9.1 Cardiovascular Disease (CVD) and dementia

AD is more prevalent in patients suffering from conditions such as autoimmune inflammation, diabetes, other heart disease and vascular damage, and neurone/nervous-system damage (Coghlan, 2017; Wotton & Goldacre, 2017).

CVD is more prevalent in patients with autoimmune inflammation. Since vitamin B12 deficiency can cause autoimmune disorders (as described in Chapter 7) it may lead indirectly to dementia through this route. Supplementation with vitamin B12 throughout later years may play an important part in reducing the prevalence of all of these conditions (Douaud et al., 2013; Smith et al., 2010).

Vascular damage, especially of the micro blood vessels, is particularly closely related to dementia. Cerebral amyloid angiopathy (CAA)³⁴ is a major cause of lobar intracerebral haemorrhage (ICH) and cognitive impairment in the elderly and CAA is present in the brains of most people suffering from AD (Viswanathan & Greenberg, 2011).

Since vitamin B12 deficiency is associated with CVD, through the accumulation of homocysteine (Amer et al., 2015; Esteghamati et al., 2014; Gilfix, 2005; Harvard Health Publishing & Harvard Medical School, 2014; Mahalle et al., 2013; Ntaios et al., 2009) and its known causal effect on the cardiovascular system (Ganguly & Alam, 2015; Ueland et al., 2000), a greater role for vascular damage in causing dementia could indicate a greater importance for maintaining vitamin B12 levels in the prevention of dementia.

8.9.2 Autoimmune conditions and dementia

In Chapter 7 we showed that there is a link between many autoimmune glandular conditions and vitamin B12 deficiency. These conditions include Type I diabetes which is well known to be an autoimmune disorder, and also Type II diabetes which recent research suggests might also be an autoimmune condition (Diabetes.co.uk, 2018; Hemminki et al., 2015). It is known that people suffering from diabetes are at increased risk of developing dementia (Alzheimer Society Canada, 2018) so it is conceivable that vitamin B12 therapy, by reducing the autoimmune tendency, or by reducing the effects of diabetes, might help to reduce dementia risk through this route.

8.9.3 Neurological damage

Dementia is usually associated with some form of neurological damage in the brain. A population study of supplementation with three B vitamins (vitamin B12 in the form of cyanocobalamin, folic acid and pyridoxine), found that in elderly patients not taking the supplement, brain atrophy occurred more quickly and was associated with cognitive and memory decline (Smith et al., 2010). Images of Subtraction MRI scans included in their study report show the contrast between the brain of an elderly participant who was given active treatment with high doses of supplementary B vitamins (folic acid, and vitamins B12 and B6) over two years to lower homocysteine and that of an elderly participant in the placebo group who was given no treatment. The latter shows brain tissue changes and an atrophy rate of 2.5% per year compared with the former where there was no significant pattern of atrophy.

The study concluded: “Since accelerated brain atrophy is a characteristic of subjects with mild cognitive impairment who convert to Alzheimer's disease, trials are needed to see if the same treatment will delay the development of Alzheimer's disease” (Smith et al., 2010). The exact form of neurological damage is not well understood in dementia, possibly because “dementia” describes the symptom of cognitive decline, which may be caused by a huge number of independent mechanisms.

Many of the symptoms of vitamin B12 deficiency also relate to neurological decline in the CNS and brain. “Brain fog” is described as a feeling of being apart from people, able to hear voices but unable to understand why they might be trying to communicate with one. What better description of the early stages of cognitive decline relating to dementia?

³⁴ Cerebral amyloid angiopathy (CAA) is a condition in which proteins called amyloid build up on the walls of the arteries in the brain. CAA increases the risk for stroke caused by bleeding and dementia.

8.10 Is dementia inherited?

As described in Table 8-1, several genes play important pathogenic roles in at least some patients with AD. For example, adults with the genetic condition trisomy 21 (Down Syndrome) who survive beyond age 40 consistently show typical neuropathologic hallmarks of AD.

The role of vitamin B12 in ensuring correct gene transcription and therefore in preventing the occurrence of gene variants which might lead to illness, is described in Chapter 9.

8.11 Nutrition and lifestyle

8.11.1 Alcoholism

Dementia can accompany chronic alcoholism. This may be a result of associated malnutrition: alcohol destroys B vitamins, including thiamine (vitamin B1). Chronic alcoholism may also cause cerebral damage in other ways. For example, a rare idiopathic syndrome of dementia and seizures with degeneration of the corpus callosum (Marchiafava-Bignami Disease) has been reported in male Italian red-wine drinkers and other cases of alcoholism (Lyford et al., 2017). It is commonly treated with thiamine or B-complex vitamins (combining folate, thiamine and B12) (Nemlekar et al., 2016; Parmanand, 2016).

8.11.2 Dietary or induced B vitamin and trace mineral deficiencies

Many vitamins and trace elements are required for mental health. Thiamine (vitamin B1) deficiency is well known to cause the neuropsychiatric disorder Wernicke's encephalopathy.³⁵ This is particularly significant in our study because it shows the dramatic neuropsychiatric effects that one vitamin can have. Although alcoholism is a frequent cause of this condition, the clinical presentation also features patients who are malnourished because of malignant disease, gastrointestinal disease and surgery, and vomiting due to hyperemesis gravidarum. Other causes include fasting, starvation, malnutrition and the use of unbalanced diets (Galvin et al., 2010). The patient displays varied neurocognitive symptoms, typically involving mental status changes and gait and oculomotor dysfunction (BMJ Best Practice, 2018e). Prompt administration of parenteral thiamine (100 mg intravenously for three days followed by daily oral dosage) may reverse the disease if given in the first days of symptom onset. However, prolonged untreated thiamine deficiency can result in an irreversible dementia/amnestic syndrome (Korsakoff syndrome) or even death.

Vitamin B6 and niacin as well as the trace minerals zinc, copper, manganese and magnesium have also all been shown to play important roles in maintaining mental health (Cornish & Mehl-Madrona, 2008).

8.12 Treating dementia and other neuropsychiatric conditions

The main goals for dementia management are to treat correctable causes, and provide comfort and support to the patient and caregivers.

It has been shown that supplementing with vitamin B12 in an elderly population reduces the incidence of increased levels of homocysteine, brain atrophy, and cognitive decline (all characteristics of dementia). It is therefore important that this age group, in which dementia is most

³⁵ This illness is frequently combined with Korsakoff syndrome. The two illnesses together are known as Wernicke-Korsakoff syndrome or Wernicke-Korsakoff psychosis.

prevalent, receives appropriate supplements of vitamin B12 to reduce the likelihood of dementia developing (Smith et al., 2010).

Once the damage has been done, it is generally considered that damaged axons or axons with no myelin sheath are not able to recover, and therefore that the dementia will persist. However, we have observed many other instances (not including dementia) where non-functioning axons have restored their function. Others have also observed the repair of scleroids in the spinal column at the same time as restoration of motor and sensory functions (Scalabrino, 2005, 2009; Scalabrino et al., 1995).

Possible restoration of nerve function in children diagnosed with autism has also been observed. We have experienced cases where children manifesting autistic-spectrum behaviour have had all the symptoms reversed rapidly following treatment for vitamin B12 deficiency. From being highly disruptive at school, or unable to cope with schoolwork, they have become sociable pupils with good school performance. Pacholok and Stuart also comment: “doctors are finding that many autistic children improve remarkably when they receive B12 injections” (Pacholok & Stuart, 2011, p. 135).

It is also likely that vitamin B12 deficiency may cause psychosis (see Case 8-1), and that B12 supplementation may alleviate psychosis (Berger, 2004; Blundo et al., 2011; Denson, 1976; Dogan et al., 2009; Roze et al., 2003). Vitamin B12 supplements may also have a beneficial impact on the early stages of Parkinson’s disease (Christine et al., 2018), epilepsy and palsies. Supporting evidence for this from our experience is provided by the fact that about 17 patients from the Shinwell Medical Group were taken off the epilepsy register following successful long-term vitamin B12-replacement therapy.

This leaves us with the hope that an appropriately balanced treatment with vitamins and hormones that are deficient may one day be available to reverse cognitive decline in people with dementia (Gröber et al., 2013).

