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REPORT

NEUROLOGICAL DISEASE

A Potential Breakthrough



Japanese scientists have identified a form of vitamin B12 that protects against neurological disease and aging by a unique mechanism that differs from current therapies. Some of the disorders that may be preventable or treatable with this natural vitamin therapy, called methylcobalamin, include Parkinson's disease, peripheral neuropathies, Alzheimer's disease, muscular dystrophy and, neurological aging. Americans have immediate access to this form of vitamin B12, and unlike prescription drugs, it costs very little and is free of side effects.

Vitamin B12 is a general label for a group of essential biological compounds known as cobalamins. The cobalamins are structurally related to hemoglobin in the blood, and a deficiency of vitamin B12 can cause anemia. The primary concern of conventional doctors is to maintain adequate cobalamin status to protect against anemia. The most common form of vitamin B12 is called cyanocobalamin.

However, over the last 10 years, a number of central and peripheral neurological diseases have been related to a deficiency of a very specific cobalamin, the methylcobalamin form, that is required to protect against neurological diseases and aging. The liver converts a small amount of cyanocobalamin into methylcobalamin within the body, but larger amounts of methylcobalamin are necessary to correct neurological defects and protect against aging.

Published studies show that high doses of methylcobalamin are needed to regenerate neurons, as well as the myelin sheath that protects axons and peripheral nerves.

Just how effective is methylcobalamin in treating acute disease? Lets take a look at some neurological diseases and other disorders where methylcobalamin has shown therapeutic results.

Bell's Palsy

Bell's palsy is non-lethal paralysis of the facial nerve. Any or all branches of the nerve may be affected, and, in fact, Bell's palsy victims may not be able to open an eye or close one side of the mouth. To assess the benefits of methylcobalamin, 60 patients with Bell's palsy were divided into three groups.

One group was given standard steroid drug therapy, the second group was given methylcobalamin plus steroid therapy, and the third group was given methylcobalamin by itself. The comparison among the three groups was based upon the number of days needed to attain complete recovery of nerve function, facial nerve scores, and improvement in symptoms.

The results: It took an average of 7.79 weeks for the group given the steroid drug to recover completely. In contrast, the group given the steroid drug and methylcobalamin took just 1.23 weeks to recover, and the group receiving the methylcobalamin by itself enjoyed complete recovery after just 5.1 days. The facial nerve score was significantly more severe in the steroid group compared with the methylcobalamin groups, and improvement in symptoms was better in the methylcobalamin groups compared with the group treated with the steroid drug.

The results of this study, published in *Methods and Findings of Experimental Clinical Pharmacology* (17[8]:539-44 1996 Oct), showed that methylcobalamin was 10 times more effective than the steroid drug approved by the Food and Drug Administration.

For those debilitated by Bell's palsy, a dose of 40 to 60 mg a day of methylcobalamin could be a safe and effective therapy.

Brain Aging

Unlike Bell's palsy, it is difficult to demonstrate methylcobalamin's rapid results when protecting against aging-related disorders. On the other hand, the mechanisms of action of methylcobalamin, however, are intriguing.

One cause of brain cell death is glutamate toxicity. Brain cells use glutamate as a neurotransmitter, but unfortunately glutamate is a double-edged sword in that it can also kill aging brain cells. The release of glutamate from the synapses is a usual means by which neurons communicate with each other.

Effective communication means controlled release of glutamate at the right time to the right cells, but when glutamate is released in excessive amounts, intercellular communication ceases. The flood of glutamate onto the receiving neurons drives them into hyperactivity, and the excessive activity leads to cellular degradation. The Life Extension Foundation has never recommended glutamine supplements for healthy people because of concern about glutamine-induced brain cell damage.

The good news is that it may now be possible to protect brain cells against glutamate toxicity by taking methylcobalamin supplements. In a study in the *European Journal of Pharmacology* (1993 Sep.7;7;241 (1):1-6), it was shown that methylcobalamin protected against glutamate-, aspartate- and nitroprusside- induced neurotoxicity in rat cortical neurons.

This study also showed that S-adenosylmethionine (S-AdoMet) protected against neurotoxicity. In a study in *Investigational Ophthalmology Visual Sciences* (1997 Apr; 38(5):848-854), a combination of methylcobalamin and S-AdoMet was used to protect against retinal brain-cell toxicity caused by glutamate and nitroprusside.

Researchers concluded that methylcobalamin protects against neurotoxicity by enhancing brain cell methylation. The Life Extension Foundation previously has recommended methylation-enhancing therapies such as vitamin B6, vitamin B12, folic acid and TMG (trimethylglycine) to protect against heart disease, stroke and other aging-related diseases.

The scientists who conducted the methylcobalamin studies emphasize that ongoing intake of methylcobalamin is necessary to protect against neurotoxicity. Thus, for methylcobalamin to be effective in protecting against neurological disease, daily supplementation may be required.

An appropriate dose to protect against neurological aging might be 1 to 5 mg a day taken under the tongue.

Parkinson's Disease

At its current rate, Parkinson's disease strikes one in every 100 people over the age of 65. Almost every human suffers Parkinson's-like symptoms as they age. Methylcobalamin may help to prevent Parkinson's disease and slow the progression in those who already have it. Here's how:

Dopamine is a neurotransmitter that controls motor functions. Dopamine transmits messages through different regions of the brain and along nerve pathways in order to coordinate muscle movement.

Proper dopamine metabolism also is required to maintain a state of psychological well-being. Aging humans suffer a progressive disruption of dopamine metabolism that can cause muscle weakness, loss of coordination, and depression. Parkinson's disease is caused by the premature destruction of specialized brain cells that produce dopamine.

When 80 percent of dopamine-producing brain cells have died, Parkinson's disease is usually diagnosed. It is therefore desirable to protect dopamine-producing brain cells and maintain youthful dopamine metabolism throughout life.

Dopamine is formed from the amino acid L-dopa. The more L-dopa that enters the brain, the more dopamine is produced, but the problem is that L-dopa itself is toxic to brain cells and is a direct cause of cell death.

The mechanism of L-dopa toxicity is excessive release of glutamate from neurons (*Brain Research* 1997 Oct 10; 771[1]: 159-162), which injures and kills brain cells. This could be why the drug Sinemet, which provides significant amounts of L-dopa to the brain, only works for several years before its effects wear off and the Parkinson's patient deteriorates rapidly.

The types of brain cells that are most vulnerable to glutamate-induced toxicity are the very cells involved in dopamine metabolism and neural-motor control. Methylcobalamin has been shown specifically to protect against glutamate- induced neural toxicity caused by L-dopa.

This means that supplementation with methylcobalamin could protect those patients with Parkinson's disease from glutamate-induced toxicity caused by the high amount of L-dopa they are putting into their brains by taking Sinemet. If brain cells that control motor function were protected against L-dopa-induced glutamate toxicity, it could mean that Parkinson's patients who take methylcobalamin could continue benefitting from the dopamine-enhancing effects of Sinemet for a much longer period of time.

Late-stage Parkinson's patients for whom Sinemet therapy no longer works may have already suffered too much glutamate-induced brain cell damage to benefit from methylcobalamin. The Parkinson's patients who are still benefitting from Sinemet may be able to protect their striatal neurons by taking 5 to 20 mg a day of methylcobalamin sublingually (under the tongue), along with Sinemet.

(Additional therapies are outlined in the Foundation's Parkinson's Disease Protocol. Call the Foundation at 1-800-544-4440 for a free copy, or refer to the Foundation's book, *Disease Prevention and Treatment Protocols*.)

The combination of methylcobalamin and Sinemet therapy could be a medical breakthrough, but this can only be proven by controlled studies. Today's Parkinson's patients cannot wait for the completion of clinical studies and may want to start sublingual intake of 5 to 20 mg a day of methylcobalamin immediately.

For Parkinson's disease prevention, 1 to 5 mg a day of sublingually administered methylcobalamin may be sufficient.

Alzheimer's Disease

A study in *Clinical Therapeutics* (1992 May;14(3):426-437) showed that the intravenous administration of large doses of methylcobalamin to Alzheimer's patients improved the patients' intellectual functions such as memory, emotions and communication with other people. The scientists concluded that methylcobalamin is a safe and effective treatment for psychiatric disorders in patients with Alzheimer-type dementia.

This is the only clinical study the Foundation could find on using methylcobalamin to treat Alzheimer's disease. It could be that 40 to 80 mg a day of sublingually administered methylcobalamin would be an effective adjuvant (assisting) Alzheimer's therapy. To obtain a referral to sources of intravenous methylcobalamin drugs, Foundation members should call our technical support line at 1-800-226-2370.

Multiple Sclerosis

A study in the journal *Internal Medicine* (1994 Feb. 33(2):82-86) investigated the daily administration of 60 mg of methylcobalamin to patients with chronic progressive multiple sclerosis (MS), a disease that has a poor prognosis and features widespread demyelination in the central nervous system.

Although motor disability did not improve, there were clinical improvements in visual and auditory MS-related disabilities. The scientists stated that methylcobalamin might be an effective adjunct to immunosuppressive treatment for chronic progressive MS. Those with less serious forms of MS may consider adding methylcobalamin to their daily treatment regimen.

The effects of methylcobalamin were studied on an animal model of muscular dystrophy. This study, published in *Neuroscience Letters* (1994 Mar 28; 170[1] 195-197), looked at the degeneration of axon motor terminals.

In mice receiving methylcobalamin, nerve sprouts were more frequently observed and regeneration of motor nerve terminals occurred in sites that had previously been degenerating.

MS patients can obtain a copy of the Life Extension Foundation's Multiple Sclerosis Protocol on-line. (Also, refer to *Disease Prevention and Treatment Protocols*.)

Regenerating Nerves

Few substances have been shown to regenerate nerves in humans with peripheral neuropathies. However, a study in the *Journal of Neurological Science* (1994 Apr. 122[2]:140-143) postulated that methylcobalamin could increase protein synthesis and help regenerate nerves. The scientists showed that very high doses of methylcobalamin produce nerve regeneration in laboratory rats.

The scientists stated that ultra-high doses of methylcobalamin might be of clinical use for patients with peripheral neuropathies. The human equivalent dose the scientists used is about 40 mg of sublingually administered methylcobalamin.

In humans, subacute degeneration of the brain and spinal cord can occur through the demyelination of nerve sheaths caused by a folic acid or vitamin B12 deficiency. In a study in the *Journal of Inherited Metabolic Diseases* (1993;16[4]:762-770), it was shown that some people have genetic defects that preclude them from naturally producing methylcobalamin.

The scientists stated that a deficiency of methylcobalamin causes demyelination disease in people with this in-born defect.

An early study published in the Russian journal *Farmakol Toksikol* (1983 Nov; 46[6]: 9-12) Nov 1983) showed that the daily administration of methylcobalamin in rats markedly activated the regeneration of mechanically damaged axons of motor neurons. An even more-pronounced effect was observed in laboratory rats whose sciatic nerves were crushed mechanically.

Two studies published in the Japanese journal *Nippon Yakurigaku Zasshi* (1976, Mar, 72,[2]: 269-278) showed that the administration of methylcobalamin caused significant increases in the in vivo incorporation of the amino acid leucine into crushed sciatic nerves, resulting in a stimulating effect on protein synthesis repair and neural regeneration.

Those suffering from peripheral neuropathies often take alpha lipoic acid. Based on our new understandings of peripheral neuropathy, we suggest that anyone using alpha lipoic acid also take at least 5 mg a day of sublingually administered methylcobalamin to ensure that alpha lipoic acid will be bioavailable to the peripheral nerves.

Cancer & Immune Function

A study in the journal *Oncology* (1987; 44[3]:169-173) examined the effects of methylcobalamin on several different kinds of tumors in mice. The administration of methylcobalamin for seven days suppressed liver, lung and ascites tumor growth. Mice receiving methylcobalamin survived longer than control mice. In mice irradiated before tumor cell inoculation, methylcobalamin did not improve survival.

The effects of methylcobalamin on human immune function was investigated in the *Journal of Clinical Immunology* (1982 Apr 2; [2]:101-109). The study showed that methylcobalamin showed remarkable T cell-enhancing effects when the T cells were exposed to certain antigens.

The scientists also showed that methylcobalamin improved the activity of T helper cells. The scientists concluded that methylcobalamin could modulate lymphocyte function by augmenting regulatory T cell activities.

Sleep

A study in the journal *Experientia* (1992 Aug;48[8]:716-720) indicates that those taking methylcobalamin also might want to take melatonin. In the study, it was detailed how nine healthy humans were given 3 mg of methylcobalamin a day for four weeks.

Among the results, it was found that melatonin levels were significantly lower in the group receiving methylcobalamin compared with placebo, although methylcobalamin did not adversely effect sleep patterns. On the contrary, previous reports of experiments show that vitamin B12 improves sleep patterns.

The Life Extension Foundation suggests that those taking methylcobalamin take at least 500 micrograms (½ mg) of melatonin at bedtime. In addition to its sleep-enhancing capabilities, melatonin has shown potent anti-cancer and immune-enhancing benefits.

A more recent German study appearing in *Neuropharmacology* (15[5]:456-464, 1996) showed that while methylcobalamin reduced the amount of time subjects slept, that sleep quality was better and subjects awoke feeling refreshed, and with better alertness and concentration. Part of this effect was apparently due to melatonin suppression during the daytime because methylcobalamin reduced drowsiness.

Most of the scientific studies cited in this article were conducted in Japan. Americans need to know about this important natural therapy that could extend the healthy human life span. A search of the scientific literature reveals 334 published studies on methylcobalamin. However, it would not be an exaggeration to say that virtually no American doctors know of it or are recommending it.

Methylcobalamin should be considered for the treatment of any neurological disease. For example, based on its unique mechanisms of action, methylcobalamin could be effective in slowing the progression of "untreatable" diseases such as ALS (Lou Gehrig's disease).

Since methylcobalamin is not a drug, there is little economic incentive to conduct expensive clinical studies on it, so it may be a long time before we know just how effective this form of vitamin B12 is in slowing the progression of common disorders like Parkinson's disease.

The sublingual intake of methylcobalamin is an affordable and effective natural therapy, and it is safe even when given in large doses. For prevention purposes, just 1 mg of methylcobalamin taken under the tongue every day could produce enormous anti-aging benefits at a very low price.

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