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Dispelling the Myths of Vitamin B6

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In 1942, pyridoxine (a form of vitamin B6) was determined to cause severe weakness and pathological changes in peripheral nerves and dorsal root ganglia in dogs and rats. Although the non-species-specific effects should have suggested human vulnerability to pyridoxine neuropathy, it was not until 1983 that human neuropathy was reported. In fact, further studies demonstrated that humans were even more sensitive to the neurotoxicity of pyridoxine than rodents (1). Despite these and other compelling studies regarding the toxicity of pyridoxine, it continues to be abused. The reason behind this abuse is the existence of unscientifically founded myths. In the following, the various myths and the startling evidence against them will be presented.

**MYTH #1: Vitamin B6 is water -soluble and hence not toxic in doses exceeding RDA recommendations.**

RDA recommendations of vitamin B6 range from 1.5 to 2.2 mg per day<sup>2</sup>. Extensive literature suggests that vitamin B6 is toxic in doses exceeding RDA values. The following outlines the findings of these studies (in chronological order):

- (1) Seven adults developed progressive sensory ataxia and profound limb impairment of position and vibration sense after consumption of large daily doses of pyridoxine (2 to 6 g) for 4 -40 months (3).
- (2) Daily doses as low as 500 mg of vitamin B6 may result in neuropathy (5).
- (3) Sensory neuropathy from low-dose pyridoxine occurred in individuals ingesting 0.1 to 4.0 g for 6 years (2).
- (4) Photosensitive lesions, vomiting, and peripheral neuropathy developed in children with Down's syndrome being treated with high doses of pyridoxine (1).
- (5) 103 women demonstrated impaired neurological function while attending a private clinic and ingesting an average of 117 + 92 mg of pyridoxine for a period ranging from 6 months to 5 years (3).
- (6) Acute sensory neuropathy-neuronopathy from pyridoxine developed in an individual ingesting 61 g for 3 days (2).
- (7) Excessive amounts of pyridoxine appear to cause degeneration of dorsal root ganglia (4).
- (8) Daily digestion of a high dose B-vitamin was associated with an acneiform eruption that promptly improved after discontinuation of the vitamin (6).
- (9) A woman exposed for 13 years to high daily doses of vitamin B6 (up to 10 g) developed sensory neuropathy with a slight motor component (2).

In general, most patients suffering from pyridoxine toxicity complain of progressive ataxia, particularly in the dark (loss of visual cues), accompanied by numbness of the feet and severe sensory dysfunction. This dysfunction is characterized by a decrease in joint function and vibratory sense, a decrease in the sense of touch in the distal symmetric distribution, and a decrease in the sensation of the lips and tongue. Standard electrical studies reveal degeneration of large axons and small unmyelinated fibers reflecting pathologic changes in the dorsal root and gasserian ganglia (5).

**MYTH #2: Vitamin B6 is a safe and effectual treatment for PMS.**

Vitamin B6 deficiency has been hypothesized to play a role in PMS via various proposed etiologies: (1) disrupted estrogen metabolism in liver; (2) lowered dopamine and/or serotonin concentrations; (3) disrupted production of essential fatty acids. The use of vitamin B6 in the treatment of PMS and other gynecological ailments began in the 1940s (7). In 1973, Adams et al. conducted a study that has often been cited as the rationale for using the vitamin to treat PMS. Their study sample consisted of women who became depressed after taking oral contraceptives with high amounts of estrogen and progesterone. Fifty percent of subjects also exhibited signs of vitamin B6 deficiency. Pyridoxine treatment was associated with improved symptoms of depression only in the vitamin B6 deficient group (7).

After 1973, several open or anecdotal trials reported using vitamin B6 for PMS in doses ranging from 40 to 800 mg/day with success (8). Double blind studies using 50-500 mg/day were also reported (7). An analysis of these studies revealed that most of these studies suffered from methodological flaws including inadequate subject selection, concurrent use of other medications for PMS or oral contraceptives, inappropriate statistical analysis, and high-placebo response rates (7).

In addition, no good evidence exists to support any of the proposed etiological factors involving vitamin B6, and women studied in protocols after 1973 did not appear to be deficient in the vitamin. Vitamin B6 values in women suffering from PMS do not appear to differ from symptom-free women (7).

**MYTH #3: Vitamin B6 is an anti-neurotoxic, anti-edemal, stress reducing, and energy producing "super" drug (that also relieves PMS and asthmatic symptoms)!**

Much of the abuse of vitamin B6 stems from confusion surrounding its properties and functions. For example, a 1986 article in Food and Fitness being distributed at a Californian health store in 1994 described the role of vitamin B6 as follows: "[Pyridoxine] plays several roles in health, helping in the chemical conversion of food to energy, as well as helping to maintain the central nervous system" (10). This is the type of definition proffered by many lay articles that the general population reads! It illucidates why many people begin self-medicating with very high doses of vitamin B6 that are furnished in numerous multi-vitamins. Most of the population is unaware of what Vitamin B6 comprises and how it actually functions. The actual biochemistry of Vitamin B6 is as follows: It comprises the compounds pyridoxine, pyridoxal, and pyridoxamine (which can interconvert within the body). Vitamin B6 is a coenzyme participating in numerous reactions (e.g., transaminations, deaminations, and decarboxylations) primarily related to protein metabolism. The functions of vitamin B6 range from glycine and serine interconversion; homocysteine conversion to cystathionine; niacin and serotonin from tryptophan, formation of delta-aminovulnic acid for heme synthesis, and the conversion of glycogen to glucose-1-phosphate. Scientific evidence fails to supports its role as an anti-neurotoxic, anti-edemal, stress -reducing, energy producing, and asthma and PMS treating agent (4).

**MYTH #4: More is better.**

Most people do not realize that excessive vitamin doses are likely to produce effects that differ from normal function of the vitamin (i.e., more vitamin does not equal "enhanced" vitamin function) (1). Indeed, these abnormal functions tend to be the injurious factors. Interestingly, the effects of pyridoxine toxicity resemble those of pyridoxine deficiency.

Rudman et al. (8), proposed three mechanisms to describe the mechanism of pyridoxine toxicity. They are as follows: (1) Pyridoxine is a member of the pyridine family which are neurotoxic. The sparing of the central nervous system in pyridoxine toxicity can be explained by the limited transport of pyridoxine across the blood-brain barrier, in contrast to an unlimited gastrointestinal transport. Many of the cell bodies of peripheral sensory fibers are located in the dorsal ganglia outside the blood-brain barrier, whereas the cell bodies of the motor fibers are located within the spinal cord. This may explain the selective toxicity of pyridoxine for the sensory fibers of peripheral nerves. (2) Although some water-soluble vitamins depend on carriers for gastrointestinal absorption and hence transport is saturable, pyridoxine moves via passive transport. Also, pyridoxine must be converted to its active form, pyridoxal phosphate, via two enzymes, pyridoxal kinase and pyridoxine phosphate oxidase. High circulating pyridoxine can saturate these enzymes and then act as a biologically inactive competitive inhibitor of pyridoxal phosphate. Therefore, a pyridoxal phosphate deficiency is mimicked. (3) Finally, an additional toxic factor may reside in the impurities of the pharmaceutical product. FDA regulations allow a 2% impurity which increases exponentially when an individual is ingesting megadose amounts (9). For example, a recent epidemic of eosinophilia-myalgia syndrome in individuals ingesting large amounts of tryptophan appears to be caused by a contaminant in the production of tryptophan (1).

Finally, a recent book suggests that daily supplements of 200 mg of pyridoxine over several months may result in a pyridoxine dependency when the supplement is discontinued (9). Little is known regarding the physiological or psychological basis of a vitamin B6 abuse-induced dependency. It is interesting to note that many individuals self-medicating with vitamin B6 began with innocuous amounts and increased them dramatically over time. This finding may indicate the development of tolerance and a need for a higher dose to produce the same effects, or perhaps the disruption of homeostasis. One can also consider the psychological dependence potential of vitamin B6 if individuals believe the vitamin to provide them with stress-relief and energy.

In conclusion, the scientific evidence regarding the dangers of vitamin B6 megadoses is overwhelming. Despite fifty years of such evidence, the abuse persists based on the existence of myths accepted both by the general population and members of the medical community. With the popularity of anti-oxidants and the general interest in vitamins as preventative health agents, it is imperative that these myths be dispelled.

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