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VITAMIN D SUPPLEMENTATION

Recent Science Presents a Contrarian View
For Chronic Illnesses

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This report contains information regarding the most recent scientific evidence of Vitamin D and its effects on the human body. The contrast between current beliefs in public health policy and the latest in scientific evidence implicating Vitamin D as a causative agent in human illnesses and diseases are discussed. Although publicized studies have suggested a link between Vitamin D supplementation in palliation of autoimmune symptoms, a reduced risk of bone fractures and the prevention of certain types of cancers in specific subsets of the American population, scientific evidence supporting a contrarian view is emerging. By examining the data contained in peer-reviewed scientific journals and the results of several studies linking Vitamin D supplementation to adverse pathogenesis of several human diseases, including the aforementioned disorders, this project will explore the basic biochemical processes of Vitamin D in its various forms, investigate prevailing misconceptions about the nature and action of Vitamin D within the human body, and present evidence supporting a contrarian view of Vitamin D supplementation as it applies to the current trend in public health policy.

One of the most recently publicized topics in public health involves Vitamin D and its role in the prevention of human disease. Myriad articles suggest that Vitamin D is a useful tool in the fight against disorders ranging from cancer to osteoporosis to Alzheimer's disease. Proponents argue that the mounting evidence from these studies show a positive, dose-dependent correlation between Vitamin D intake and the reduction in symptoms of many types of human disease, particularly in illnesses of a chronic or rheumatoid nature. However, advances in technology particularly in-silica (computer modeling) experiments, are presenting a far different picture of Vitamin D and its effects in the human body than the recent publicity would suggest.

Vitamin D (also known as calciferol) is the generic name given to a broad number of fat-soluble secosteroid metabolites. Calciferol, discovered in 1922, was erroneously classified as a vitamin. Later research confirmed that the body synthesizes calciferon in the skin after exposure to ultraviolet or infrared radiation, eliminating the need for an ongoing source of the nutrient in the diet. Calciferol is found in several different forms in the human body, but for the purpose of this report, only three metabolites will be discussed; D₃, 25-D, and 1,25-D. The form found in food, supplements and as the result of exposure to light is known as Vitamin D₃. Vitamin D₃ is converted in the liver to 25-D, which according to recent data, is an inert compound that acts on the immune system as an immunosuppressive steroid (Marshall, 2006). The more important form, 1,25-D (sometimes referred to as "the master hormone"), is a biologically active hormone which is created in the kidneys and controls bone metabolism, thyroid regulation, and the immune system (Managing, 2005).

Both forms function as the native ligands which control the Vitamin D Receptor (VDR), one of the key nuclear receptors in the body. It is estimated that the VDR transcribes over 3% of the entire human genome, including antibacterial, antiviral and antimetastatic (anti-cancer) proteins (Albert, Proal & Marshall, 2009). Indeed, one of the most crucial roles of the VDR is in the regulation of the human immune system, but their actions within the VDR are very different: the regulation of the human immune system, but their actions within the VDR are very different: at normal clinical levels, 1,25-D serves as an activator while 25-D acts as an inhibitor (Wang, Nestel, & Bordeaux,

2004). Thus, when people ingest dietary sources or supplements of Vitamin D, the D₃ is converted into 25-D, which then binds into the VDR receptor and inhibits gene transcription of immune cells and natural defense proteins (Proal, 2007). To support this theory, Schaubert et al. (2007) performed a study in mice that showed high levels of 25-D inhibited wound healing response when the epidermis was exposed to injury.

People with autoimmune disease, rheumatism or other forms of chronic illness often display low levels of 25-D while 1,25-D is elevated. An explanation for dysregulated Vitamin D metabolism in chronically ill individuals can be found in capnine, a lipid produced by several type of L-form bacteria (bacteria which lack cell walls) implicated in autoimmune disease. Capnine, like 25-D, is known to block the VDR from transcribing an enzyme known as CYP24, which breaks down excess 1,25D (Marshall, 2007). Elevated levels of 1,25-D interfere with the body's ability to maintain normal levels of 25-D, and the chronically ill patient presents with low levels of 25-D, leading the physician to recommend Vitamin D supplementation. As the patient already shows evidence of an abnormal immune system, supplementation often leads to grave results, as low levels of 25-D in chronically ill people is actually an indication of the disease process, not a Vitamin D deficiency (Proal, 2007). The specific mechanism is proposed by Albert et al (2009).

“As the microbial (in-vivo L-form bacteria) continues to dysregulate the VDR, transcription of key enzymes is thwarted. VDR production of CYP24A1 decreases, allowing 1,25-D to rise without a feedback system to check it. As the hormone/secosteroid rises above a normal range, it down-regulates, via the PXR Nuclear Receptor, the amount of Vitamin D converted into 25-D. This results in the low levels of 25-D characteristic of autoimmune diagnoses.”

Vitamin D has long been considered an essential component for bone health. However, as advances in molecular continue, the prevailing wisdom may be changing. An interesting fact to note is that when the level of the 1, 25-D in the body rises above 43 pg/ml, osteoclasts are stimulated (Magolas, 2000). Osteoclasts are cells which dissolve bone material and deposit the minerals into the bloodstream. If the elevated level of 1,25-D is maintained over a period of years, the process will result in osteoporosis and calcium deposits in the kidneys and lungs, which lead to kidney stones (Proal, 2007). In fact a study by Adams, Song, & Kantoroich (199) on patients with osteoporosis and hypercalciuria (an elevated level of calcium in the blood) found that Vitamin D decreases bone mineral density. The patients in the study were instructed to discontinue Vitamin D supplementation over a period of three years, during which the scientists monitored their bone density. During this period all of the patients' hypercalciuria was eradicated, the high levels of 25-D were normalized, and every one of the patients showed annual increases in bone density. At the 3-year follow-up evaluation, one of the patients showed annual increases in bone density. At the 3-year follow-up evaluation, all patients had maintained the bone density gains made during the study. The authors concluded that “resolution of Vitamin D intoxication was associated with a rebound in bone mineral density.” As Prowl (2007) states, “The study was particularly valuable because (the

authors' three-year follow-up phase showed that the increase in bone mineral density existed after initial recovery.”

Rickets, (a disorder leading to the softening and deformation of bones, usually in children), was long thought to be the result of a Vitamin D deficiency. However, three separate studies by Graff, et al (2004), Deman (2006), and Demay, Sabbagh, & Carpenter (2007) confirmed that Rickets is in fact caused by deficient levels of calcium and phosphorus. Interestingly, the Demay (2006) study was on mice genetically engineered without a VDR proving that Rickets was not in any way associated with the Vitamin D metabolism. In light of these findings, the US Department of Agriculture website explicitly states the “Rickets in toddlers is a large problem in parts of Africa, especially Nigeria. It is not due to Vitamin D deficiency but is caused by not having enough calcium in the diet” (Proal, 2007).

Vitamin D is currently being touted as a beneficial supplement in the prevention of cancer, and again, the recent science does not support the claims. As Proal (2007) states:

“In the latest study by the National Cancer Institute – the first study to actually look at the relationship between measured Vitamin D in the blood and subsequent total cancer deaths – the data failed to show an association between baseline Vitamin D status and overall cancer risk in men, women, non-Hispanic whites, non-Hispanic blacks, Mexican Americans, and in person younger than 70 or 70 years or older”.

In correlation with the blocking of the VDR (and the resulting immunosuppression) by secreted caprine, L-form bacteria have been implicated in cancer pathogenesis. In a review article published by a research team, the group concluded the “overwhelming body of evidence has determined that relationships among certain bacteria and cancer exists,” and mechanisms of action include “chronic infection, immune evasion, and immune suppression.” Several strains of bacteria and the incidence of certain forms of cancer are discussed, including *Salmonella typhi* (gallbladder cancer) *Streptococcus bovis* and *Escherichia coli* in colon cancer, and *Chlamydia pneumoniae* (Mager, 2006). According to a study performed by the National Cancer Institute (Stolzenberg-Solomon, 2006), Vitamin D is implicated in pancreatic cancer, one of the fastest-spreading and most lethal cancers in humans. Male patients were tracked over the course of 16 years and Proal (2007) states that, “in the long term, high 25-D levels greater than 26 ng/ml were associated with a three-fold increased risk for pancreatic cancer, suggesting that individuals consuming high levels of Vitamin D were more likely to fall ill with the disease.” The findings led the team to conclude that, “contrary to expectations, subjects with high prediagnostic Vitamin D status had an increased pancreatic cancer risk compared with those with lower status.”

Data detailing the long-term effects of Vitamin D supplementation are beginning to emerge. Of particular interest to parents in Finnish study (Hypponen et al, 2004) which tracked the health effects of Vitamin D supplementation during the first year of life on nearly 8,000 children born in 1966. At the 31-year point of the study, the authors discovered that the “prevalence of atopy, allergic rhinitis, and asthma at age 31 years was

higher in participants who had received Vitamin D supplementation regularly during the first year compared to others, and had received Vitamin D supplementation regularly during the first year compared to others, and these associations persisted after adjustment for a wide range of behavioral and social factors.” Also, a Norwegian study of women 50-70 years of age discovered that “women who had not taken cod liver oil (a substance that contains high levels of Vitamin D) during childhood had higher bone mineral density compared to those who had ingested cod liver oil” (Proal, 2007). With data showing the effects of childhood supplementation persisting into the elderly years, it would seem that the intake of Vitamin D during childhood should be carefully considered by concerned parents and public health officials alike.

A debate has ensued over the levels of government-mandated supplementation of Vitamin D in the food supply. Scientists who oppose mandated supplementation point out that the current FDA standards suggesting that people maintain 25-D levels of 30-32 ng/ml is within the range of which 25-D acts as an immunosuppressant (Calvo & Whiting, 2006). The FDA is currently considering a proposal to raise the RDA (Recommended Daily Allowance) for vitamin D from 400 IU/day, the equivalent of 40 glasses of milk per day (Sullivan, 2002)..

It is clear that advances in technology, particularly in-silico molecular modeling, are uncovering the nature of the mechanisms of action of Vitamin D in the human body. Even as the scientific evidence against Vitamin D supplementation mounts, the US Food and Drug Administration is weighting data based on decades-old information as it considers raising the RDA of Vitamin D. If the data linking Vitamin D supplementation with Chronic Illness is overlooked, and the newly recommended guidelines for increased Vitamin D supplementation in the food supply are adopted by policy makers, the abundance of recent research suggests that it may well stand as one of the greatest public health policy blunders in recent history.

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Company Profile

Life Sources, Inc® is a Nevada Corporation with order fulfillment located in Fair Oaks, California and is a member of the NNFA, American Association of Nutritional Consultants, and the Citrus Heights, California Chamber of Commerce. The President and Founder is Andrea McCreery, PhD. Dr. McCreery is currently developing several new proprietary products to add to the Life Sources anti-aging and chronic illness system. Her combined talents represent 12 years of research in nutrition, and Targeted Nutritional Intervention™.

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