

The Need for Vitamin D

by Dave Siever

Introduction

The winter of 2005/2006 was a real challenge for me. For no apparent reason, I had developed severe restlessness and insomnia in December. By January, I began feeling mildly depressed and was often very “fuzzy-headed” and lethargic. I was waking up at 2, 3 and 4 am, and because of that, I would sleep in until 9:30 to 10:30 am the next day to try and make up for my lack of sleep. I was craving chocolate like mad – and simple carbs (candy, cake, etc.) as well - but mostly chocolate. By February, I was feeling like a tired, old man. I had no physical energy and felt very weak. I quit exercising on the weights because it took just too much effort. By March, I was having severe muscle spasms all through my neck, shoulders, back and chest and I found myself constantly rubbing out the painful knots and sometimes using the MET (Micro-current Electro Therapy) with both the CESTa and OASIS II on various muscles, trying to get the knots out. One day, in late March, while standing in the kitchen, my left knee gave out and I collapsed to the floor. I struggled with walking.

By April, my lower right molar and gums were quite painful when I ate, making it difficult to chew. Later in April my upper left molar also began aching. My elbows, knees and ankles began to ache. This was of great concern for me, as I am typically a healthy and physically strong person.

In early April of 2006, just before attending the AAPB conference, I went for an 8-minute tan at a local tanning center and noticed that later in the day, I was feeling better, but by the next day I was back in the slumps. A few days later, I went back and tanned to the point of getting mildly pink. The next day there was no change but the following three days I was feeling better and my head was clearer, even though my joints and mouth continued to hurt.

Suspecting a connection, I took time off from work every day at noon for the first two weeks in May, to take in at least 30 minutes of tanning in the warm, spring sun. Never have I had so much sun so early in the year and fortunately, this spring was the warmest and sunniest ever, with leaves sprouting and flowers blooming a full three weeks ahead of schedule.

By May 15th, 2006, my mind was sharp and all of my body pain had vanished. I was suddenly bursting with energy and strength and biked 25 miles (40 km) on Mothers’ Day! Best of all, my wit and humor returned and I enjoyed being the life of the party again. On May 20th, I went to Jasper National Park and participated in the annual SCUBA diving aquathlon – feeling GREAT!

Some Basic Vitamin D Facts

Those who live above 30 degrees north latitude or below 30 degrees south latitude are likely to become vitamin D deficient throughout the winter (Cannell & Hollis, 2008). Over one billion people worldwide have become deficient in vitamin D including many Americans who work all day in office buildings. It is estimated that somewhere between 21% to 58% of adults and adolescents in the US are vitamin D deficient, and this is based on the older, lower standard based on 30 nl being an adequate level. Caucasians originated in latitudes far from the equator as their lighter skin produced vitamin D faster because of the long winters with low UV-light. (Pritchard, 2010). African Americans living at latitudes far from the equator are 10 times more likely to be deficient in vitamin D as compared to Caucasians (Sorenson, 2006).

The Power of Vitamin D

Vitamin D is a potent andro-steroid, much like testosterone (Dowd, 2008; Canell & Hollis, 2008). It is essential for the metabolizing of calcium (Cannell & Hollis, 2008) (which is also essential for generating the voltage potentials in the neurons). This explains the emotional collapse and the subsequent depression, anxiety, restlessness, and insomnia for those who are deficient. Vitamin D targets over 200 genes, essential for everyday metabolism (Cannell & Hollis, 2008). Vitamin D also plays a key role in muscle strength and the integrity of connective tissue and the maintenance of bones and cartilage as well as playing a part in controlling swelling and in tissue repair (Dowd, 2008; Sorenson, 2006).

Vitamin D is involved in brain function. Genes encoding the enzymes involved in the metabolism of vitamin D are expressed in brain cells. Vitamin D is important in the production of serotonin, which prevents depression, anxiety and improves sleep. Studies of sun-tanning in both natural sunlight sun and in tanning beds have shown increases in brain levels of serotonin, endorphins and dopamine, which boosts mood, improves sleep and reduces anxiety and depression (Sorenson, 2006). A study published in the New England Journal of Medicine reported that vitamin D significantly reduces, type I and type II diabetes, multiple-sclerosis, depression and schizophrenia (Holick, 2007; Sorenson, 2006). Autism is also affected by seasonality, and has tripled since the introduction of sunscreen and drug-company induced paranoia of sunshine, which may be related to vitamin D deficiency during pregnancy (Cannell & Hollis, 2008).

Vitamin D has been shown to significantly enhance the genetic expression of antimicrobial peptides in macrophages (our up-front immune system), thus improving up-front ability to attack and destroy a broad spectrum of invasive microbes, spanning both viruses and bacteria. Flu season occurs in the winter of both the northern and southern hemispheres, and it is not attributed to more people staying indoors as previously thought. Vitamin D has been shown to have neuro-protective and immune-modulatory effects and helps us resist bacterial (Matheson, et. al., 2010) and viral infections such as colds and flues, including H1N1 (Urashima, et. al., 2010; Cannell & Hollis, 2008; Mercola, 2009; Sorenson, 2006), and reduces asthma (Brehm, et al., 2010).

Most forms of cancer, including breast, colon, pancreatic, lymph, brain, skin and prostate cancer are attributed to low vitamin D levels (Falloon, 2007; Lappe, et al., 2007; Drake, 2009; Jenab, et al., 2010; Sorenson, 2006). Increases in melanoma are closely correlated with sales of sunscreen and has tripled since 1972 (Sorenson, 2006). Vitamin D also reduces migraine headache (Gandey, 2010), dementia & Alzheimer's in seniors (Llewellyn et. al., 2009; Llewellyn, et. al., 2010; (Llewellyn, et. al., 2010; Annweiler 2010), Parkinson Disease (Evatt et. al., 2008) and Multiple Sclerosis (Ramagopalan (2009). Hip fractures are 6-fold higher in those who don't suntan (Sorenson, 2006). Several studies show that vitamin D sharply reduces chronic pain (Plotnikoff & Quigley, 2003).

Research indicates vitamin D deficiency is a causal factor in all facets of human health, as shown below.

Brain and Mind

Alzheimer's Anxiety Autism Brain birth-defects Dementia	Depression Insomnia Multiple Sclerosis Fatigue and malaise Parkinson's	Insomnia Irritability Reduced IQ Schizophrenia Psychosis
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Body

Aneurysm Arthritis and pain Asthma Chronic pain Diabetes Fibromyalgia Heart disease	Hypertension Hip fractures Hypothyroid Hemorrhoids Migraines Multiple sclerosis Muscle weakness	Muscle wasting Osteoarthritis Osteoporosis Periodontal disease Rickets Seizure Stroke
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Immune System

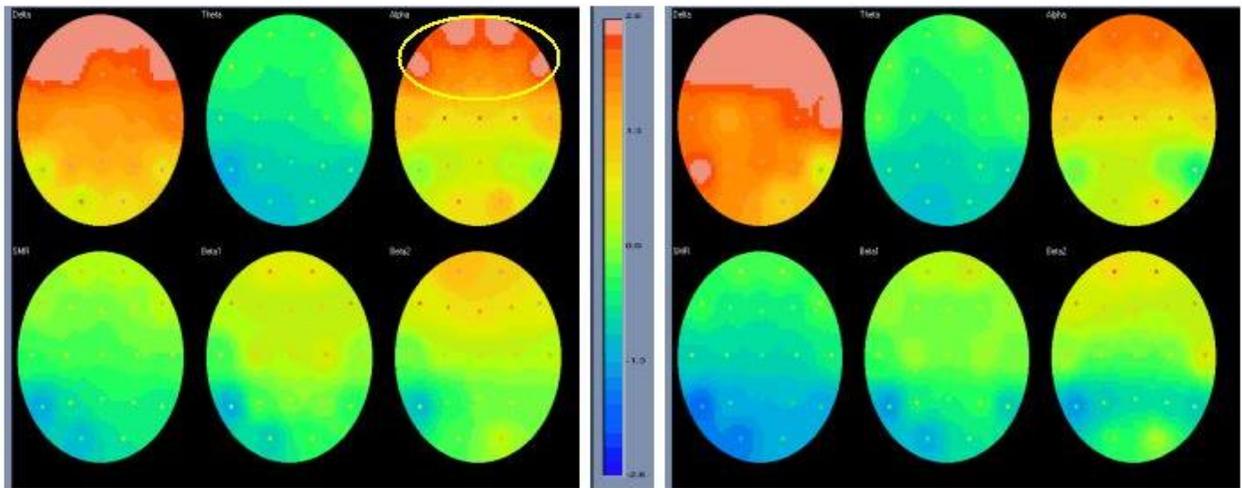
17 varieties of cancer	Common colds	Common flus & H1N1
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The best kind of vitamin D is vitamin D3 (cholecalciferol) which comes from the sun. Most vitamin supplements are vitamin D3. Calcidiol (25-hydroxy vitamin D) is a prehormone in your blood that is directly made from cholecalciferol. When being tested for vitamin D deficiency, calcidiol is the only blood test that should be drawn. When doctors refer to vitamin D blood levels, they are referring to calcidiol levels, but the lab will know calcidiol as 25-hydroxy vitamin D. The only natural sources of vitamin D are sunshine and fatty fish such as salmon and mackerel. Milk is fortified with vitamin D, but at very low levels – only enough to prevent rickets.

Vitamin D Effects on My Brain

Figure 1 shows an eyes-closed brain map from the SKIL database at a scale of ± 2.6 SD before and after two weeks of sun tanning. Notice the exceptionally high alpha (circled in yellow) when deficient in vitamin D. The high delta is primarily artifact. If this was a case of true SAD, it would have resolved itself by the time the post QEEG was taken on May 15, as eye/pineal exposure is significant by May.

Figure 1. Vitamin D Deficiency vs Sunlight Exposure



Seasonal Affective Disorder

The prevailing belief is that 6% of northern populations are affected with Seasonal Affective Disorder (SAD) and another 14% have a milder form of SAD, called the “Winter Blues” (Rosen et al., 1990; Swedo, Leonard, 1996; Kasper, et al., 1989). Surprisingly, SAD may occur at any time of year and even in equatorial regions although the ratio of northerners with SAD compared to those living in the tropics is about 10 to 1. People in the southern USA experience SAD in the summer from staying indoors where air conditioning allows them to escape the summer heat. People have also experienced SAD moving into a basement suite or an office on the north side of a building or after painting the interior of their home a darker shade of color. People have experienced SAD following the development of cataracts or after wearing sunglasses for an extended period of time and during overcast, rainy periods (Rosenthal, 1993).

The common symptoms are depression, anxiety, extreme fatigue, hypersomnia, carbohydrate cravings, and weight gain. Women between the ages of 20 to 40 (their sexually reproductive years) are most susceptible (Rosenthal, 1993). The first controlled study using light therapy to treat SAD was published in 1984 and SAD was officially accepted as a clinical malady in 1987 by the American Psychiatric Association. Since that time, a great number of studies on SAD have been completed. While research on the behavioral effects of SAD are abundant, the literature on brain wave effects of SAD is inconsistent, ranging from increased broad alpha/theta to increased left frontal alpha activity as seen in depression.

The “Captain and Pineal”

All species studied to date have been observed to have a biological clock. This clock is essential for survival by regulating various types and levels of arousal to provide cues for alertness, eating, sleeping and the release of hormones. Light waves striking the retina activate electrical output that is sent down the optic nerve to the brain for visual processing. A secondary, smaller nerve tract from the retina, originating from specialized cells that utilize a light detecting pigment called melanopsin, also carries signals to the suprachiasmatic nucleus (SCN) of the hypothalamus. The SCN, in turn, sends nervous outputs to various parts of the brain including the pineal gland. Four genes that govern circadian cycles in flies, mice and humans have been discovered to not only reside within the SCN, but in all cells of the body. When cultured in a petri-dish under constant lighting, these cells continue with gene activity, hormone secretion and energy production in a 24-hour cycle that varies less than 1% (Wright, 2002).

In the mid 70s, Dr. Alfred Lewy of the National Institute of Mental Health (NIMH) discovered the neurotransmitter melatonin. The wake/sleep cycle in animals and humans is controlled by melatonin, which is produced by the pineal gland, a structure the size of a pea and located in the mid-brain. Every night, the pineal gland excretes melatonin into the bloodstream and continues to do so until dawn. However, under normal exposure to sunlight, secretions of melatonin follow the earth’s light/dark time frame and therefore more melatonin is typically released during the long dark hours of the winter months. Henceforth, the pineal gland is in charge or “captains” our wake/sleep arousal states.

Edmonton Winter 2006

Edmonton’s winter of 2006 was unusual. We normally get a foot of snow by Christmas, but this year, we didn’t get any snow until March 15! This meant a particularly severe year of SAD symptoms for many of my clients, likely due to a lack of reflection off of the snow. Edmonton is situated about 300 miles north of the USA/Canada border at 53 degrees north latitude. Edmonton is a fairly sunny place and the sun shines at roughly a 20-degree angle around the winter solstice. This means that plenty of sunlight bounces off of the snow and enters the eyes in addition to what the sun provides. This year however, without the snow, fewer people got out for their annual winter activities such as cross-country skiing and tobogganing, and the concurrent nourishment from the sun.

Treating SAD

A number of techniques are used to reduce the symptoms of SAD. These include long walks outside (without sunglasses), aerobic exercise, a diet rich in complex carbohydrates and protein, relocating to sunnier locations and winter vacations to tropical areas (which likely made vitamin D). Light-based clinical interventions include light box therapy and audio-visual entrainment.

Light box therapy has been successful in reducing the SAD symptoms for 60% to 80% of users. White light therapy, using intensities of 2,500 lux, requires exposure times from 2

to 6 hours, a considerable time investment for the user. Light exposures with an intensity of 10,000 lux for 30-minutes have been found to be more effective than 2,500 lux intensity with exposure times of several hours. Some people have reported that overuse of light therapy can leave them feeling “wired” and restless.

Audio-Visual Entrainment (AVE), which uses flashing lights and pulsing tones, has been shown to enhance EEG activity at the stimulation frequency. However, a lesser-known attribute of AVE lies in its inhibition effect at roughly the half-frequency of stimulation. In the QEEGs that we have collected from SAD clients, we have observed long spindles of 10 Hz alpha brain wave activity throughout the main cortex, with particularly increased alpha brain waves in the left frontal regions. We conducted an AVE study with 74 people struggling with SAD. AVE at 20 Hz inhibited the abnormally high alpha activity, which in turn produced profound reductions in anxiety, depression, carbohydrate cravings and body weight. Energy and quality of life increased.

A Comparison Between SAD and Vitamin D Deficiency

Although most anxiety and depression inventories could be used to detect SAD, one popular SAD test is the *Seasonal Pattern Assessment Questionnaire* or SPAQ, developed by Rosenthal and his colleagues at the NIMH. The SPAQ is a self-assessment questionnaire that evaluates one’s level of SAD. However, one big problem with the SAD test is that there is much overlap between the symptoms of SAD and vitamin D deficiency, and more than just the basic SAD type questions must be asked.

Symptoms of SAD

anxiety
depression
fatigue
carbohydrate cravings
hypersomnia

Symptoms of Vitamin D Deficiency

anxiety, restlessness and/or depression
fatigue and physical weakness
carbohydrate cravings
insomnia
connective tissue swelling

Could SAD have been Misdiagnosed for the Past 40 Years?

Given that only high levels of full-spectrum light-box therapy actually help treat SAD, could it be that vitamin D was being generated in the face and researchers missed that connection entirely? Could those suffering with SAD possibly comprise less than 0.6% of the population and not 6% as thought? Gloth (1999) completed a vitamin D study of 15 people diagnosed with SAD. Eight people were given a single injection of 100,000 IU (don’t take this quantity at home) and seven were given light-box therapy for SAD related depression. All of the people who received the vitamin D were depression-free on follow-up one-month later, whereas no one in the light-box group showed any improvement.

Treatment of Vitamin D Deficiency

The only treatment for vitamin D deficiency is by increasing vitamin D3, whether from food sources or from sun tanning. Many supplements are available. Because vitamin D is fat-soluble, it is difficult to become toxic from it. A wide variance of a suggested toxic limit exists in the literature, but recent research suggests that body-mass index is a critical factor. The more overweight a person is, the more vitamin D that person can tolerate before becoming toxic. An adult may have to consume 4000 to 8000 IU for more than four months in order to become toxic. An interesting observation is that arctic fish and seals have very high levels of vitamin D in their fat, and this has sustained the Inuit population since their existence.

Dosage

The only way to know the amount of vitamin D in your body is with a 25-hydroxy-vitamin D (25OHD) test. This is the circulating form of vitamin D and routinely used to diagnose vitamin D deficiency. The Vitamin D Council recommends 25OHD levels between 50 and 80 ng/ml, year around. These levels assure vitamin D metabolism is normalized. Furthermore, levels of 50 to 80 ng/mL are “natural” levels, that is, levels normally achieved by people who work in the sun.

Levels are measured in one of two ways: nanograms per milli-litre or nanomoles per litre. Levels should be between 50 to 80 ng/ml (125 to 200 nmol/L) year-round, in both children and adults. Heaney and his colleagues found that the body does not reliably begin storing vitamin D3 (cholecalciferol) in fat and muscle tissue until 25OHD levels get above 50 ng/ml (125 nmol/L). The average person starts to store cholecalciferol at 40 ng/ml (100 nmol/L), but at 50 ng/ml (125 nmol/L) virtually everyone begins to store it for future use. At levels below 50 ng/ml (125 nmol/L), the body uses up vitamin D as fast as you can make it, or take it, indicating chronic D3 starvation in fat with no reserves—not a good thing. All recent research suggests that healthy men require upwards from 3000 to 7000 IU per day of quality vitamin D3 in order to maintain these levels (Cannell & Hollis, 2008; Sorenson, 2006).

My Recommendations

I suggest using a high-quality liquid vitamin D. My suggested dose is 2500 IU per day per 100 lbs (44 kgs) of healthy, lean weight. Fat will store vitamin D, but does not metabolize it, so you have to base your dosage on what your “healthy” lean weight would be. For example, an average sized woman (120 - 150 lbs) should take 10,000 IU each day for the first week and then reduce to 3000 to 4000 IU per day after that. An average sized man (160 – 200 lbs) should take 10,000 IU each day for the first two weeks before reducing to 4000 to 5000 IU per day.

Being that vitamin D releases serotonin in the brain, I take 2000 IU at bedtime and another 2000 IU when I go for my bathroom break at 3 or 4 am. I hold the dropper about 6 inches above my tongue so I feel the drops hit my tongue. Because vitamin D is a steroid, I recommend that you swallow it back with a bit of water to avoid getting “dry-

mouth.” Vitamin D appears to be an effective sleep aid for those deficient in serotonin.

Even though vitamin D is an effective sleep aid, too much can trigger instant insomnia. When I first began using Pure Encapsulation vitamin D (a very high-quality brand), I began sleeping like never before, which was fabulous! I took 20,000 IU for eight weeks and at the end of the eighth week, I woke up at 1 am and was wide awake. Further investigation revealed that too much vitamin D causes a flooding of calcium into the blood-stream. This throws the calcium/magnesium ratio off balance (relative magnesium becoming low) which can cause a person to be wide awake. To resolve this, stop taking vitamin D for a few days and then resume to a lower dosage.

Once you’ve figured out your optimum vitamin D intake and you are still having problems with your sleep, I have also found that taking a calcium/magnesium powder named CALM can also help to quickly restore sleep. If I find myself waking in the middle of the night, I sometimes mix a teaspoon of CALM in a half-glass of water and it will put me back to sleep. If I wake up with nagging issues on my mind, I will also take L-tryptophan (Life Choice or Source Naturals) and St. John’s Wort (FLORA) to help me get a good sleep.

I enjoy tanning, as I find that endogenous (self-made) vitamin D works best and tanning-based vitamin D is self-regulating, so I can’t make too much. I generally don’t take any vitamin D for a couple of days after sun-tanning outdoors, particularly in summer, when I’m getting abundant sun. If I haven’t tanned for a week, I start taking 2000 IU each day for one week and increase to 3000 IU for the second week, and finally take 4000 IU per day from the third week on. When I begin indoor sun-tanning, I have to reduce my intake to 2,000 IU per day or I develop insomnia as it seems that indoor tanning twice a week is roughly equivalent to 2000 IU of vitamin D per day. The body knows when to stop making vitamin D through tanning, but it doesn’t seem to know how much oral D is in the body – so tanning while supplementing can produce an excess of D in the body.

I have tested twelve brands of vitamin D, eight of which were in tablet form. I didn’t respond to any tablet at all. Most of my clients also do not respond to the tablets either. There are some gel-cap brands that also do not work and possibly a couple of cheap liquid brands also. These are the brands that I recommend:

Pure Encapsulations vitamin D
 Carlson vitamin D in 1000 and 2000 IU.
 Carlson D drops (USA) are available at all higher-end health-food stores.
 D-drops

If you can’t find a supplier in your area, the Pure Encapsulations and Carlson brands may be ordered from:

Optimum Health
 #2-7115-109 St
 Edmonton, Alberta, Canada
 T6G 1B9
 Ph: 866-874-HEAL (4325)

Local: 780-432-5464

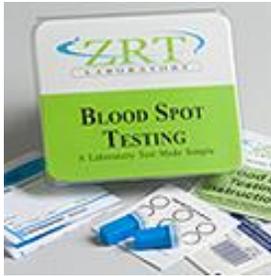
Email: info@optimumhealthvitamins.com

Other Optimum Health locations:

Downtown Edmonton: 116 St & 104 Ave

Sherwood Park: 110-101 Granada Blvd.

Check out: www.vitamindcouncil.org for excellent information on vitamin D. The council provides a vitamin D test-kit, which may be ordered through their website.



References

Annweiler, C., Schott, A., Allali, G., Bridenbaugh, S., Kressig, R., Allain, P., Herrmann, F., Beauchet, O. (2010) Association of vitamin D deficiency with cognitive impairment in older women. *Neurology*, Vol. 74, 1, 27-32.

Brehm JM, Schuemann B, Fuhlbrigge AL, Hollis BW, Strunk RC, Zeiger RS, Weiss ST, Litonjua AA; Childhood Asthma Management Program Research Group. (2010). Serum vitamin D levels and severe asthma exacerbations in the Childhood Asthma Management Program study. *Journal of Allergy and Clinical Immunology*, 126(1), 52-58.

Cannell, J. Go to: www.vitamindcouncil.org.

Cannell, J., Hollis, B. (2008). Use of vitamin D in clinical practice. *Alternative Medicine Review*, Vol 13, (1), 6-20.

Dowd, J., Vitamin D Cure. (2008). John Wiley & Sons, Inc., New Jersey.

Drake, M., Maurer, M., Link, B., Micallef, I., Habermann, T., Kelly, J., Macon, W., Nikcevic, D., Colgan, J., Allmer, C., Slager, S., Weiner, G., Witzig, T., Cerhan, J. (2009). Vitamin D deficiency is associated with inferior event-free and overall survival in diffuse large B-Cell lymphoma. *Blood* (ASH Annual Meeting Abstracts) 2009 114: Abstract 1952 © 2009. American Society of Hematology. 51st Annual Meeting, December 5 - 8, 2009; New Orleans, Louisiana.

Evatt, M., DeLong, M., Khazai, N., Rosen, A., Triche, S., Tangpricha, V., (2008). Prevalence of Vitamin D Insufficiency in Patients With Parkinson Disease and Alzheimer Disease. *Arch Neuro*, 65, (10), 1348-1352.

Faloon, W. (2007). Should the president declare a national emergency? *Life Extension*, 13, (10), 7-16.

Gandey, A. (2010). Vitamin D Low in Patients With Headache and Migraine. *American Headache Society (AHS) 52nd Annual Scientific Meeting*: Poster 51. Presented June 26, 2010.

Gloth, F., Alam, W., Hollis, B. (1999). Vitamin D versus broad spectrum phototherapy in the treatment of seasonal affective disorder. *Journal of Nutrition, Health and Aging*, 3, 1, 5-7.

Godfroid, I.O. (1998). Placebo II. Psychiatria and the brain organization. *Annales Medico-Psychologiques*, 156, 2, 108-114.

Heaney, R. P., (2005). The Vitamin D requirement in health and disease. *Journal of Steroid Biochemistry & Molecular Biology*.

Holick, M. (2007). Vitamin d deficiency. *New England Journal of Medicine*, 357, (3), 266-81.

- Jenab, M., Bas Bueno-de-Mesquita, H., Ferrari, P., van Duijnhoven, F. (2010). Association between pre-diagnostic circulating vitamin D concentration and risk of colorectal cancer in European populations: a nested case-control study. *British Medical Journal*, 21 January 2010, doi:10.1136/bmj.b5500
- Kasper, S., Rogers, S., Yancey, A., Schulz, P., Skwerer, R., Rosenthal, N. (1989). Phototherapy in individuals with and without subsyndromal seasonal affective disorder. *Archives of General Psychiatry*, 46, Sept, 837-845.
- Lappe, J., Travers-Gustafson, D., Davies, K., Recker, R., Heaney, R. (2007). Vitamin D and calcium supplements reduces cancer risk: results of a randomized trial. *American Journal of Clinical Nutrition*, 85, (6), 1586-1591.
- Llewellyn, D., Langa, K., Lang, I. (2009). Serum 25-Hydroxyvitamin D Concentration and Cognitive Impairment. *Journal of Geriatric Psychiatry and Neurology*, Vol 22, 3, 188-195.
- Llewellyn, D., Lang, I., Langa, K., Muniz-Terrera, G., Phillips, C., Cherubini, A., Ferrucci, L., Melzer, D., (2010). Vitamin D and Risk of Cognitive Decline in Elderly Persons. *Archives of Internal Medicine*, 170(13), 1135-1141.
- Llewellyn DJ, Lang IA, Langa KM, Melzer D. (2010). Vitamin D and Cognitive Impairment in the Elderly U.S. Population. *J Gerontol A Biol Sci Med Sci*. Nov 2. [Epub ahead of print]
- Matheson EM, Mainous AG, Hueston WJ, Diaz VA, Everett CJ. (2010). Vitamin D and methicillin-resistant *Staphylococcus aureus* nasal carriage. *Scand J Infect Dis*. Mar 8.
- Mercola, J., (2009). Despite Anti-Vitamin D Bias, CDC Stumbles on Deficiency Link to H1N1 Deaths. Posted: www.mercola.com.
- Plotnikoff, G., & Quigley, J. (2003). Prevalence of severe hypovitaminosis D in patients with persistent, nonspecific musculoskeletal pain. *Mayo Clin Proceedings*, 2003;78:1463-1470
- Pritchard, J. (2010). How we are evolving. *Scientific American*, Vol 303, (4), 41-47.
- Ramagopalan, S., Maugeri, N., Handunnetthi, L., Lincoln, M., Orton, S., et al. (2009) Expression of the Multiple Sclerosis-Associated MHC Class II Allele *HLA-DRB1*1501* Is Regulated by Vitamin D. *PLoS Genetics*, 5(2): e1000369. doi:10.1371/journal.pgen.1000369
- Rosen, L.N., Targum, S.D., Terman, M., Bryant, M.J., Hoffman, H., Kasper, S., Hamovit, J.R., Docherty, J.P., Welch, B., & Rosenthal, N.E. (1990). Prevalence of seasonal affective disorder at four latitudes. *Psychiatry Research*, 31, 131-144.

Rosenthal, N.E. (1993). *Winter blues: what it is and how to overcome it*. New York: Guilford Press.

Shannon, S., Louie, J., et al. (2009). Surveillance for Pediatric Deaths Associated with 2009 Pandemic Influenza A (H1N1) Virus Infection - Center for Disease Control, United States, April--August 2009. *Website:*
www.cdc.gov/mmwr/preview/mmwrhtml/mm5834a1.htm.

Sorenson, M. (2006). *Solar Power for Optimum Health!* Self-published.

Swedo, S., Leonard, H. (1996). *It's not all in your head*. 1st ed. New York: Harper Collins Publishers Inc.

Urashima M, Segawa T, Okazaki M, Kurihara M, Wada Y, Ida H. (2010). Randomized trial of vitamin D supplementation to prevent seasonal influenza A in schoolchildren. *Am J Clin Nutr*. Mar 10.