

Appropriate Vitamin D3 level and safe daily intake?
(Compiled by Charles (Chuck) Maack – Prostate Cancer Advocate)

Below is what Dr. Myers has to say about Vitamin D3 supplementation as well as some lengthy writings by Dr. John Cannell of the Vitamin D Council and Professor Reinhold Vieth of Mount Sinai Hospital in Toronto.

Note that Dr. Myers specifically remarks that the Vitamin D3 level should be within a "broad range" of 50-100ng/ml. That's his "broad range" spread. He assigns his patients specific goals, and for most that goal is in the 65ng/ml-75ng/ml range with total Vitamin D3 intake of up to 10,000 IU daily to reach that range.

IMPORTANT TO REMEMBER: According to the following, the accuracy of laboratory readings of 25-hydroxy Vitamin D levels are faulty. When determining a more “true” 25-hydroxy Vitamin D level, subtract approximately 20% from the laboratory result. See:

<http://www.ncbi.nlm.nih.gov/pubmed/19756833>

Accordingly, we should strive for a laboratory 25-hydroxy Vitamin D level between 75ng/ml to 94ng/ml in order to have reached a “true” range between 60ng/ml and 75ng/ml.

ALSO IMPORTANT TO REMEMBER: Increased Vitamin D3 intake should be accompanied by regular monitoring of blood serum 25-hydroxy Vitamin D, blood serum and urine calcium levels, and parathyroid hormone level.

Since there have been varying reports of Dr. Myers recommendations regarding appropriate 25-hydroxy Vitamin D (25(OH)D), and particularly since his recent book "Beating Prostate Cancer: Hormonal Therapy & Diet" erroneously stated on page 158 his recommendation that the level be 50-80nmol/L, I asked him for clarification. Here is his reply:

"The book is a typo We use a broad goal of 50-100ng/ml. Most of what has been reported as Vitamin D toxicity is really toxicity from excessive calcium intake. From what I have seen, 25-hydroxyvitamin D appears to be very safe by itself. Clearly the literature is evolving rapidly and the direction is **(Note the following words:)** toward higher levels being also safe. In certain settings, hypercalcemia is

quite unlikely. For example, in men on Zometa, it is hard to keep the calcium in the normal range - it all too easily slips into an abnormally low range, triggering hyperparathyroidism."

Interesting information I found during an internet search were these effects that can occur with Vitamin D deficiency: In mammals, a deficiency can cause bone problems such as rickets (osteomalacia), skeletal deformities, metabolic bone disease, osteoporosis, accelerated bone turnover rate, swollen wrists or ankles, and other problems such as muscle weakness, improper gonadal function, hypocalcemic seizures, decreased memory and learning, and possibly the winter blues. A Life Extension Foundation paper remarked: "A study conducted by an international team of researchers, published in the July 12, 2010 issue of the American Medical Association journal [*Archives of Internal Medicine*](#), found a greater risk of cognitive decline in older individuals with vitamin D insufficiency compared to those with sufficient levels." It was also interesting to learn that hippocampal levels of mRNA's for both vitamin D receptor and calcium binding protein are reduced in patients with Alzheimers.

And here is a bundle of information regarding Vitamin D3 with trials conducted by a variety of physicians and scientists:

Vitamin D may help treat prostate cancer

22 Nov 2004

Oliver Wendell Holmes, Sr, the eminent 19th-century physician and poet, once admonished doctors saying, "Beware how you take away hope from another human being." Thanks to a discovery announced last week, millions of hopeless men around the world suffering from advancing prostate cancer now have reason for hope. A presentation at the conference on vitamin D and cancer, sponsored by the National Institutes of Health and the National Cancer Institute, gave them reason to hope. A group from the University of Toronto reported the first human trials of a deltanoid (vitamin D compound) that appear to both help fight prostate cancer and to fight that cancer without causing any side effects.

This year, about 400,000 American men will learn they have prostate cancer, making it the leading cancer among men. Early diagnosis is important as surgery or radiation can be curative. Early diagnosis is helped by measuring PSA, a blood marker for possible prostate cancer. If a biopsy is positive, then surgery or radiation leads to falling PSA levels and hope for a cure. After such treatment, urologists then use PSA as a marker for tumor reoccurrence and progressive disease. If the treatment is not curative and the cancer continues to grow, the PSA often goes up again leading to castration, either surgical or chemical. Then the PSA often falls again indicating a remission. Castration can delay the cancer from spreading for several years, but then, if the cancer grows again, the PSA again marches steadily upwards and treatment options become quite limited, leading to hopelessness. Throughout the battle with prostate cancer, PSA is the standard clinical marker for progressive disease. This year, thousands of American men go down the hopeless road marked by progressive rises in PSA. About 40,000 will die, making prostate cancer the second leading cancer killer of American men.

Dr. Woo and his colleagues in Toronto have finally given these men and their families reason to hope. The senior investigator of the study, Dr. Reinhold Vieth, presented the first human trial of cholecalciferol ever reported in any cancer, as well as the best clinical results of any vitamin D compound ever tested in any human cancer. Woo, Vieth and colleagues studied **15 men with biopsy proven prostate cancer** who had **undergone surgery or radiation** but who had started down that hopeless road marked by **three progressive increases in PSA levels**. The researchers gave all the patients **2,000 units of cholecalciferol (the plain, cheap, simple, natural, available at any health food store, form of vitamin D) every day for up to 21 months**. In the majority of men, **their PSA either went down or stopped going up, suggesting their prostate cancer either started to regress or remained quiescent!**

The Toronto researchers found evidence of a clinical response in **14 of the 15 men** as confirmed by a **prolongation of their PSA doubling time**. What's more, none of the men had any side effects, of any kind. Again, this is the best result yet obtained for any vitamin D compound in any human cancer trial. For example, let's compare Vieth's result to any earlier trial of a vitamin D compound reported by a Stanford University Group in 1998. The Stanford group gave a very different form of vitamin D [activated vitamin D (calcitriol), a dangerous form of vitamin D known to cause high blood and urine calcium] to seven men in the same stage of prostate cancer. They also used PSA as a marker of tumor responsiveness. After getting the calcitriol, the PSA of the men continued to increase, although more

slowly. One of the patients showed a transient decrease in PSA but he had to be withdrawn from the study due to side effects from calcitriol. **Unlike the Toronto study, none of the Stanford patients showed a prolonged flattening or a prolonged decrease in their PSA.** All of the Stanford patients developed side effects such as high urine calcium, and one developed a kidney stone.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=9598513

(See end of document for this reference (# 1) & its abstract)

Therefore, the University of Toronto beat Stanford University, hands-down. Moreover, they did so with much lower comparable doses of deltanoids (deltanoids are a generic term for vitamin D compounds). The Stanford group gave increasing doses of the most potent and most dangerous deltanoid (the steroid hormone, calcitriol). They gave as much as the patients could tolerate until their urine calcium got too high. The Toronto group gave a fixed low dose (2,000 units) of the prehormone, cholecalciferol, a very safe compound **that never causes high calcium in the doses used. In fact, the lowest dose of cholecalciferol known to cause high blood calcium is more than 20,000 units.** Therefore, the Toronto group got better results with one-tenth the comparable dose of deltanoids! Vieth wanted to use more cholecalciferol but widespread ignorance about the physiology and pharmacology of vitamin D remains and he could not get adequate dosing past the various review committees.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=10232622

(See end of document for this reference (# 2) & its abstract)

In fact, 2,000 units of cholecalciferol a day will raise the “average” calcidiol [25(OH)D] level **to about 40 ng/ml, now considered by most authorities to be the lower limit of adequate vitamin D nutrition.** “Average” means half above and half below, which also means 50% of the Toronto men were still below 40 ng/ml; that is, one-half of the Toronto men with prostate cancer may still have been vitamin D deficient after treatment. Robert Heaney proved that adequate treatment of vitamin D deficiency **requires about 4,000 units of vitamin D every**

day. Said another way, it takes 4,000 units of cholecalciferol to assure that 97.5 % of healthy subjects will become vitamin D replete.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=12499343

(See end of document for this reference (# 3) & its abstract)

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=11157326

(See end of document for this reference (# 4) & its abstract)

Given these results, what would evidence based medicine have us do? (Evidence based medicine is a new mantra which decries doctors from any treatment that is not rigorously scientifically proven). The evidence based medicine folks say one open study of 15 patients is hardly rigorous proof (the Vitamin D Council agrees); more trials are needed (the Vitamin D Council agrees); falling PSA does not prove the tumor is regressing (the Vitamin D Council agrees) and Vieth's study has only been presented, not published and should be viewed with scepticism (the Vitamin D Council agrees). Finally, the evidence based medicine folks say that doctors should not give cholecalciferol to prostate cancer patient until rigorously conducted scientific trials prove vitamin D helps cure prostate cancer. That is, the evidence based medicine folks say urologists should allow prostate cancer patients to die before treating them with cholecalciferol. The Vitamin D Council disagrees and we know some plaintiff attorneys who disagree as well.

You see, the question is not “Should men with prostate cancer be treated with vitamin D?” The question is, “Should men with prostate cancer be allowed to die vitamin D deficient?” The evidence based medicine folks say they should. We say they shouldn't. All patients with prostate cancer should have their vitamin D deficiency aggressively and immediately corrected and that requires up to 4,000 units of cholecalciferol every day. **Physicians, researchers, or scientists who say 4,000 units may be toxic are simply admitting their ignorance of current scientific literature.**

Physicians who have read the recent scientific literature and who understand the physiology and pharmacology of cholecalciferol **would be comfortable using up to 10,000 units of cholecalciferol a day while following the patient's PSA, urine and serum calcium, and 25(OH)D.** Thanks to the Toronto group, scientific evidence now exists that suggests such an approach may help prostate cancer

patients; only time will tell.

Many patients with prostate cancer are on the long hopeless road towards death. Not only may plain old vitamin D help men with prostate cancer, it is likely to give them back their hope. Physicians have many rights, but the right to take away hope is not among them.

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REFERENCES & ABSTRACTS

1: [Gross C](#), [Stamey T](#), [Hancock S](#), [Feldman D](#): **Treatment of early recurrent prostate cancer with 1,25-dihydroxyvitamin D3 (calcitriol).** [J Urol](#). 159:2035-9, 1998; discussion 2039-40. Erratum in: [J Urol](#) 160:840, 1998. [Related Articles, Links](#)

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PURPOSE: Substantial experimental and epidemiological data indicate that 1,25-dihydroxyvitamin D3 (calcitriol) has potent antiproliferative effects on human

prostate cancer cells. We performed an open label, nonrandomized pilot trial to determine whether calcitriol therapy is safe and efficacious for early recurrent prostate cancer. Our hypothesis was that calcitriol therapy slows the rate of rise of prostate specific antigen (PSA) compared with the pretreatment rate. MATERIALS AND METHODS: After primary treatment with radiation or surgery recurrence was indicated by rising serum PSA levels documented on at least 3 occasions. Seven subjects completed 6 to 15 months of calcitriol therapy, starting with **0.5 microg. calcitriol** daily and **slowly increasing to a maximum dose of 2.5 microg. daily** depending on individual **calciuric and calcemic responses**. Each subject served as his own control, comparing the rate of PSA rise before and after calcitriol treatment. RESULTS: As determined by multiple regression analysis, the rate of PSA rise during versus before calcitriol therapy significantly decreased in 6 of 7 patients, while in the remaining man a deceleration in the rate of PSA rise did not reach statistical significance. Overall the decreased rate of PSA rise was statistically significant ($p = 0.02$ Wilcoxon signed rank test). **Dose dependent hypercalciuria limited the maximal calcitriol therapy given (range 1.5 to 2.5 microg. daily).** CONCLUSIONS: This pilot study provides preliminary evidence that calcitriol effectively slows the rate of PSA rise in select cases, although dose dependent calciuric side effects limit its clinical usefulness. The development of calcitriol analogues with decreased calcemic side effects is promising, since such analogues may be even more effective for treating prostate cancer.

PMID: 9598513

Strum Comment: Vieth definitions of safe urinary calcium: a mean urinary calcium-creatinine ratio ≤ 1.0 (when calcium and creatinine are measured in mmol; ≤ 0.37 when measured in mg) during vitamin D supplementation. Gross et al used a threshold of 0.25 mg for the urinary calcium: creatinine ratio.

2: **Vieth R: Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety.** [Am J Clin Nutr.](#) 69:842-56, 1999. [Related Articles, Links](#)

Comment in: [Am J Clin Nutr.](#) 69:825-6, 1999. [Am J Clin Nutr.](#) 74:862-4, 2001.

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For adults, the 5-microg (200 IU) vitamin D recommended dietary allowance may prevent osteomalacia in the absence of sunlight, but more is needed to help prevent osteoporosis and secondary hyperparathyroidism. **Other benefits of vitamin D supplementation are implicated epidemiologically: prevention of some cancers, osteoarthritis progression, multiple sclerosis, and hypertension.** Total-body sun exposure easily provides the equivalent of 250 microg (10,000 IU) vitamin D/d, suggesting that this is a physiologic limit. Sailors in US submarines are deprived of environmentally acquired vitamin D equivalent to 20-50 microg (800-2000 IU)/d. **The assembled data from many vitamin D supplementation studies reveal a curve for vitamin D dose versus serum 25-hydroxyvitamin D [25(OH)D] response that is surprisingly flat up to 250 microg (10000 IU) vitamin D/d.** To ensure that serum 25(OH)D concentrations exceed 100 nmol/L (40ng/ml), a **total vitamin D supply of 100 microg (4000 IU)/d** is required. Except in those with conditions causing hypersensitivity, there is no evidence of adverse effects with serum 25(OH)D concentrations <140 nmol/L (**56 ng/ml**), which require a **total vitamin D supply of 250 microg (10,000 IU)/d to attain.** Published cases of vitamin D toxicity with hypercalcemia, for which the 25(OH)D concentration and vitamin D dose are known, all involve intake of > or = 1000 microg (40,000 IU)/d. Because vitamin D is potentially toxic, intake of >25 microg (1000 IU)/d has been avoided even though the weight of evidence shows that the currently accepted, no observed adverse effect limit of 50 microg (2000 IU)/d is too low by at least 5-fold.

PMID: 10232622

Strum Comment: I have added the equivalent value in ng/ml where Vit D levels are expressed in nmol/L.

3: [Heaney RP](#), [Davies KM](#), [Chen TC](#), [Holick MF](#), [Barger-Lux MJ](#): **Human serum 25-hydroxycholecalciferol response to extended oral dosing with cholecalciferol.**

[Am J Clin Nutr.](#) 77:204-10, 2003. Erratum in [Am J Clin Nutr.](#) 78:1047, 2003.

Comment in: [Am J Clin Nutr.](#) 78:496-7, 2003.

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BACKGROUND: The cholecalciferol inputs required to achieve or maintain any given serum 25-hydroxycholecalciferol concentration are not known, particularly within ranges comparable to the probable physiologic supply of the vitamin. **OBJECTIVES:** The objectives were to establish the quantitative relation between **steady state cholecalciferol input and the resulting serum 25-hydroxycholecalciferol concentration and to estimate the proportion of the daily requirement during winter that is met by cholecalciferol reserves in body tissue stores.** **DESIGN:** Cholecalciferol was administered daily in controlled oral doses labeled at 0, 25, 125, and 250 microg cholecalciferol for approximately 20 wk during the winter to 67 men living in Omaha (41.2 degrees N latitude). The time course of serum 25-hydroxycholecalciferol concentration was measured at intervals over the course of treatment. **RESULTS:** From a mean baseline value of 70.3 nmol/L (**28.1 ng/ml**), equilibrium concentrations of serum 25-hydroxycholecalciferol changed during the winter months in direct proportion to the dose, with **a slope of approximately 0.70 nmol/L (0.28 ng/ml) for each additional 1 microg cholecalciferol input.** The calculated oral input required to sustain the serum 25-hydroxycholecalciferol concentration present before the study (ie, in the autumn) was 12.5 microg (500 IU)/d, whereas the total amount from all sources (supplement, food, tissue stores) needed to sustain the starting 25-hydroxycholecalciferol concentration was estimated at approximately 96 microg (approximately 3800 IU)/d. By difference, the tissue stores provided approximately 78-82 microg/d. **CONCLUSIONS: Healthy men seem to use 3000-5000 IU cholecalciferol/d, apparently meeting > 80% of their winter cholecalciferol need with cutaneously synthesized accumulations from solar sources during the preceding summer months.** Current recommended vitamin D inputs are inadequate to maintain serum 25-hydroxycholecalciferol concentration in the absence of substantial cutaneous production of vitamin D. PMID: 12499343

Strum Comment: 1 microgram (ug) of Vit D-3 = 40 IU; therefore, for each additional 1,000 IU of Vit D-3 taken, blood levels of D-3 should increase by 7 ng/ml. So far, in the patients that I have studied, the increment in D-3 levels has been far less than the above.

4: [Vieth R](#), [Chan PC](#), [MacFarlane GD](#): Efficacy and safety of vitamin D3 intake exceeding the lowest observed adverse effect level. [Am J Clin Nutr.](#) 73:288-94, 2001. [Related Articles](#), [Links](#)

Comments in: [Am J Clin Nutr. 74:862-4, 2001](#); [Am J Clin Nutr. 74:864-5, 2001](#) (author reply 866-7); [Am J Clin Nutr. 74:865-7, 2001](#).

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BACKGROUND: The Food and Nutrition Board of the National Academy of Sciences states that 95 microg vitamin D/d is the lowest observed adverse effect level (LOAEL). **OBJECTIVE:** Our objective was to assess the efficacy and safety of prolonged vitamin D₃ intakes of 25 and 100 microg (1000 and 4000 IU)/d. Efficacy was based on the lowest serum 25-hydroxyvitamin D [25(OH)D] concentration achieved by subjects taking vitamin D₃; potential toxicity was monitored by measuring serum calcium concentrations and by calculating urinary calcium-creatinine ratios. **DESIGN:** Healthy men and women (n = 61) aged 41 +/- 9 y (mean +/- SD) were randomly assigned to receive either 25 or 100 microg vitamin D₃/d for 2-5 mo, starting between January and February. Serum 25(OH)D was measured by radioimmunoassay. **RESULTS:** **Baseline serum 25(OH)D was 40.7 +/- 15.4 nmol/L (mean +/- SD) = 16.3 ng/ml +/- 6.16 ng/ml.** From 3 mo on, serum 25(OH)D plateaued at 68.7 +/- 16.9 nmol/L **in the 25-microg/d (4,000 IU/day) group (= 27.5 ng/ml +/- 6.7 ng/ml)** and at **96.4 +/- 14.6 nmol/L (= 38.6 +/- 5.8 ng/ml) in the 100-microg/d (4,000 IU/day) group.** Summertime serum 25(OH)D concentrations in 25 comparable subjects not taking vitamin D₃ were 46.7 +/- 17.8 nmol/L (= 18.7 +/- 7.1 ng/ml). The **minimum and maximum plateau serum 25(OH)D concentrations in subjects taking 25 (1,000 IU) and 100 microg (4,000 IU) vitamin D₃/d** were 40 (**16ng/ml**) and 100 nmol/L (**40ng/ml**) and 69 (**27.6 ng/ml**) and 125 nmol/L (**50.0 ng/ml**), respectively. **Serum calcium and urinary calcium excretion did not change significantly at either dosage during the study.** **CONCLUSIONS:** The 100-microg/d dosage (4,000 IU) of vitamin D₃ effectively increased 25(OH)D to high-normal concentrations in practically all adults and serum 25(OH)D remained within the physiologic range; therefore, we consider 100 microg vitamin D₃/d to be a safe intake.

PMID: 11157326

Recently we are hearing of a study or two claiming Vitamin D having little to do with reining in or preventing prostate cancer. Despite these claims, Vitamin D continues to be an extremely important requirement for other health issues. Read the following regarding Vitamin D deficiency and myocardial infarction (heart attack).

Deficient Vitamin D Levels? Greater Risk Of Myocardial Infarction (Heart Attack)!

http://www.lef.org/newsletter/2008/0610_reduced-vitamin-D-levels-associated-with-heart-attack-in-men.htm?source=eNewsLetter2008Wk24-1&key=Archive&l=0#article

IF ABOVE TOO LONG TO OPEN, TRY....

<http://tinyurl.com/5yv339>

The June 9, 2008 issue of the American Medical Association journal *Archives of Internal Medicine* published the finding of Harvard researchers that men who have deficient vitamin D levels have a greater risk of myocardial infarction (heart attack) than men whose blood levels of the vitamin are sufficient.

For the current study, Edward Giovannucci, MD of Harvard School of Public Health and his associates reviewed data from men aged 40 to 75 who participated in the Health Professionals Follow-up Study, a prospective cohort investigation designed to evaluate associations between chronic disease incidence and diet among male health care professionals. Blood samples collected from 1993 to 1995 were analyzed for plasma 25-hydroxyvitamin D (25-OHD), lipoprotein and triglyceride levels, and diet and lifestyle factors were ascertained through the use of questionnaires. Nine hundred participants without heart disease were matched for age, smoking status, and time of blood collection with 454 men aged 40 to 75 who had fatal coronary heart disease or non-fatal heart attack diagnosed between the time of blood sample collection through January, 2004.

Adjusted analysis of the data found a 2.42 times greater risk of heart attack among subjects with plasma vitamin D levels of 15 nanograms per milliliter or less compared with those whose levels were sufficient at 30 nanograms per

milliliter or higher. Even those whose vitamin D levels were intermediate had a 43 to 60 percent greater risk of heart attack compared to men with sufficient levels. Adjustment for a number of factors, such as family history of heart attack, failed to significantly reduce the association. Men with low levels of vitamin D were more likely to live in northern states, and less likely to be white or to use a multivitamin supplement, among other characteristics revealed by the analysis.

In their commentary, the authors cite vitamin D's effect on smooth muscle cell proliferation, inflammation, vascular calcification, and blood pressure via the renin-angiotensin system as possible protective mechanisms against myocardial infarction. Other potential mechanisms include protection against type 2 diabetes, inflammation, and seasonal respiratory tract infections (especially influenza), all of which can impact cardiovascular disease mortality.

“These results further support an important role for vitamin D in myocardial infarction risk,” the authors write. “If this association is causal, which remains to be established, the amount of vitamin D required for optimal benefit may be much higher than would be provided by current recommendations (200-600 IU per day), **especially in those with minimal sun exposure. Thus, the present findings add further support that the current dietary requirements of vitamin D need to be increased to have an effect on circulating 25(OH)D levels substantially large enough for potential health benefits.** (MY NOTE: Bold lettering and underlining my emphasis. Key importance is to have your Vitamin D level tested with a 25-hydroxy Vitamin D blood serum test and if deficient take action with increased but safe sun exposure as well as supplement consideration – in any event, include discussion with your physician). And keep in mind, in view of laboratory results actually found to be reading approximately 20% too low, we should strive for a laboratory 25-hydroxy Vitamin D level of between 75ng/ml to 94ng/ml in order to have reached a “true” range between 60ng/ml and 75ng/ml.

And even more:

<http://tinyurl.com/4ux995>

Lower vitamin D levels predict increased blood sugar and insulin resistance

The October, 2008 issue of the journal [*Diabetes*](#) published the outcome of a study of middle-aged men and women which found that lower serum vitamin D levels were associated with an increase in the risk of developing insulin resistance and elevated blood sugar over a ten year follow up period.

Researchers at the Institute of Metabolic Science in Cambridge, England followed 524 nondiabetic participants in the Ely Study, a prospective study established in 1990. Upon enrollment, the vitamin D marker serum 25-hydroxyvitamin D, serum parathyroid hormone, insulin-like growth factor-1 (IGF-1) and other factors were measured, and personal health habits such as smoking status and physical activity levels were ascertained. Weight, height, waist circumference, blood pressure, plasma glucose, lipids, and fasting insulin were measured during the initial and ten year follow-up visits.

At the end of the follow-up period, having a higher serum vitamin D level was associated with a lower adjusted ten-year risk of elevated blood sugar, insulin resistance, and high metabolic syndrome score. An association between increased IGF-1 levels and metabolic syndrome risk observed in an earlier study was not noted in the current research.

The authors remark that the study's findings add evidence to previously reported observations concerning vitamin D's effect on metabolic syndrome risk. Possible mechanisms of vitamin D include direct effects on pancreatic beta cell secretory function, and indirect effects involving inflammatory processes. Additionally, insufficient vitamin D can elevate serum parathyroid hormone, which is associated with a reduction in insulin sensitivity in healthy individuals.

In an accompanying editorial, Robert Scragg of the University of Auckland in New Zealand asks the question, "Are we ready for a prevention trial?" In light of the dramatically rising diabetes rates worldwide and the mounting evidence for a role of vitamin D in the prevention of the disease, well-designed clinical trials involving vitamin D supplementation are needed to confirm vitamin D's possible protective benefits. Dr Scragg notes that the dose of vitamin D administered in these trials needs to be at least 2,000 international units per day in order to raise serum 25-hydroxyvitamin D levels above 80 nanomoles per liter, a level at which diabetes risk is lowest. **(MY NOTE: 80nmol/L = 32ng/ml – the very low**

side of supposed “safe” level. One’s level should be much higher and at the very least, 50ng/ml/125nmol/L. To raise levels, likely no less than 4000 IU daily would be required). “If well-designed trials are carried out and confirm a protective effect from vitamin D, it could be used by the general population as a simple and cheap solution to help prevent the diabetes epidemic,” he concludes.

AND THIS FROM JOHNS HOPKINS:

[Can Vitamin D Prevent Prostate Cancer?](#)

If you thought vitamin D's main role was preventing rickets and strengthening bone, think again. Many researchers now believe that the "sunshine vitamin" may one day play a key role in preventing the growth of prostate cancer, and in killing rogue prostate cancer cells that have escaped into the body. The data are quite suggestive and vitamin D is a most promising area for prostate cancer research.

During the past decade, there's been a surge in research into the association between vitamin D and prostate cancer. Multiple studies have reported a link between sub-optimal levels of vitamin D and an increased risk of developing various cancers including prostate cancer, although not all studies have been confirmatory. While these findings are encouraging and could eventually lead to widespread screening for and treatment of vitamin D deficiencies, we still need a large, randomized, placebo-controlled trial to demonstrate whether vitamin D supplementation can actually prevent prostate cancer.

Vitamin D was first isolated by Adolf Windaus, who was awarded the Nobel Prize in 1928 for his work. Vitamin D is not actually a vitamin; it's a hormone. A vitamin is a substance you have to get from food. Vitamin D, however, is manufactured in the body -- the definition of a hormone. While researchers are still working to determine the effects of vitamin D on the prostate, here are some of the heart benefits of this vitamin:

- **Blood pressure regulation.** While there is no direct evidence that vitamin D supplementation will lower blood pressure, people with high blood pressure generally have low blood levels of vitamin D.
- **Heart attack, stroke, heart failure reduction.** A recent study in *Circulation* reported that events such as heart attacks, strokes, and heart failure were anywhere from 53% to 80% higher in people with low levels of vitamin D in their blood. That risk increased even more in people with high blood pressure.

Low blood levels of vitamin D may increase the risk of heart disease and stroke, especially for people with high blood pressure, according to researchers with the Framingham Heart Study. The scientists followed 1,739 men and women for more than five years and reported that participants with low blood levels of vitamin D were 62% more likely to develop cardiovascular disease than those with higher levels. For those with low vitamin D levels and high blood pressure, cardiovascular risk doubled.

- **Helps reduce inflammation.** Researchers speculate that more vitamin D could lead to less inflammation in the arteries. Until recently, most researchers believed that heart disease was essentially a "plumbing" problem caused by an accumulation of hardened fat and cholesterol in the coronary arteries, known as plaque. However, an increasing body of evidence now shows that this accumulation of plaque is actually the result of chronic, low-grade inflammation in the coronary arteries. Researchers also believe that in the battle against heart disease, damping down this inflammation is nearly as important as lowering cholesterol.

Disclaimer: Please recognize that I am not a Medical Doctor. I have been an avid student researching and studying prostate cancer as a survivor and continuing patient since 1992. **The comments or recommendations I make are not intended to be the procedure for you to now follow; rather, they are to be reviewed along with the comments or recommendations of others for your own further research, study, and discussion with the physician providing your prostate cancer care to come to your own, personal conclusion.**