Vitamin D And Prostate Cancer Prevention And Treatment

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Vitamin D and Prostate Cancer | Helpline Questions ~~Vitamin D and Prostate Cancer~~ ~~Has a vitamin D deficiency been linked to prostate cancer?~~ ~~PCSS - Prostate Cancer and Vitamin D~~ ~~Vitamin D3 Supplementation Treats Prostate Cancer~~ *Vitamin D \u0026 Prostate Health Vitamin D \u0026 Prostate Cancer* **Results of a Prostate Cancer/Vitamin D Trial: Effectiveness Safety Recommendations** *How Vitamin D Stops Cancer Formation* ~~Vitamin D may keep low-grade prostate cancer from becoming aggressive~~ ~~The Role of Vitamin D in Prostate Cancer Prevention~~ *Diet and Prostate Cancer—Now What?* **What Are Vitamin D Deficiency Symptoms? | Dr. Josh Axe 7 Best Foods For Prostate Health (2020)** *4 Foods To Avoid For Prostate Health Vitamin D3 Benefits* Carnivore Diet Mistakes with Dr. Ken Berry: Dehydration, Fat/Protein \u0026 Fasting *Take Vitamin D Every Day? This Will Make You Think Twice!* ~~You Need This Much Vitamin D Every Day~~ **Can we eat to starve cancer? - William Li Best Vitamins for Prostate Health – Dr. David Samadi**

Reasons for Deficiency of Vitamin D - Dr.Berg**ZERO Prostate Cancer Summit 2020 - Nutrition** *Choosing Supplements for Prostate Cancer Part: 1* VITAMIN D: Your Missing Health Link? 2020 ~~Higher Vitamin D levels reduce the risk of 19 types of cancer - Dr William Grant~~ *Nutrition and Prostate Cancer What We Think We Know and What We Actually Know PCSS- Are Vitamin D3 Supplements Helpful in Treating Prostate Cancer? Psoriasis cured by vitamin D supplementation* ~~Choosing Supplements For Prostate Cancer: Part Two~~ *Vitamin D And Prostate Cancer*

You can take other steps to reduce your prostate cancer risk and your overall cancer risk. eating a healthy, low-fat diet rich in fruits and vegetables. eating more plant-based fats, such as olive oil, nuts, and seeds. eating more fish that contain omega-3 fatty acids, such as salmon and tuna. ...

Vitamin D and Prostate Cancer: Is There A Link?

Vitamin D has received attention for its potential to disrupt cancer processes. However, its effect in the treatment of prostate cancer is controversial. This study aimed to assess the effect of vitamin D supplementation on patients with prostate cancer. In the present study, PubMed, Scopus, ISI Web ….

The Effect of Vitamin D Supplementation on Prostate Cancer ...

Abstract. Signaling through the vitamin D receptor has been shown to be biologically active and important in a number of preclinical studies in prostate and other cancers. Epidemiologic data also indicate that vitamin D signaling may be important in the cause and prognosis of prostate and other cancers. These data indicate that perturbation of vitamin D signaling may be a target for the prevention and treatment of prostate cancer.

Vitamin D in prostate cancer - PubMed

Prostate cancer is the most common cancer in men in the United States. Some scientists think vitamin D may help protect against prostate cancer. Prostate cancer generally occurs in men over the age of 50; however, it is slow growing in most men and takes a lifetime to develop. Prostate cancer has been found in the prostate gland of more than 80 percent of men over the age of 70 who died from an entirely unrelated disease.

How Vitamin D May Protect Against Prostate Cancer - dummies

Over an average of approximately five years, about 6% of participants in both the vitamin D and placebo groups developed cancer, with cancers of the prostate, breast and lung being the most common,...

Study: Vitamin D reduces risk for metastatic cancer, death ...

WEDNESDAY, March 2, 2016 (HealthDay News) -- Prostate cancer may be more aggressive in men who are deficient in vitamin D, new research suggests. A study of nearly 200 men having their prostate...

Low Vitamin D May Mean Aggressive Prostate Cancer

There’s been a lot of buzz about the benefits of vitamin D, particularly in the prevention of prostate, colon, and breast cancers. We know that as men get older, the incidence of prostate cancer rises. Studies have shown that older people have lower levels of this important vitamin. The evidence is mixed

Vitamin D and Prostate Cancer, Wonder Drug or Not?

D'Amico stressed that men should not start taking vitamin D supplements in hopes of slowing or curing prostate cancer. Vitamin D, known as the "sunshine vitamin," is produced by the body when it's...

Vitamin D Supplements Might Slow Prostate Cancer, Study ...

Since vitamin D 3 levels decline in older men, the research suggests that supplementation of vitamin D 3 may slow or halt the progression of prostate cancer.

Vitamin D3 may be Latest Treatment on Prostate Cancer ...
Vitamin D is the name given to a group of fat-soluble prohormones (substances that usually have little hormonal activity by themselves but that the body can turn into hormones).Vitamin D helps the body use calcium and phosphorus to make strong bones and teeth. Skin exposed to sunshine can make vitamin D, and vitamin D can also be obtained from certain foods.

Vitamin D and Cancer Prevention - National Cancer Institute
Basically, vitamin D can help promote a normal prostate size and reduce prostate swelling like BPH and prostatitis. Vitamin D has proven properties that inhibit cellular proliferation, which is the process of cells increasingly growing or dividing.

The Link Between Vitamin D and Prostate Health - Dr Tracy ...
Among the more than 25,000 study participants, 1,617 were diagnosed with invasive cancer over five years, including breast, prostate, colorectal, lung and other forms of the disease.Of the nearly 13,000 participants who took vitamin D, 226 were diagnosed with advanced cancer compared with 274 who received the placebo.

Vitamin D And Cancer? | Vimitmans & Supplements | Andrew ...
Bruce W. Hollis, PhD, Medical University of South Carolina shares the results of a recent trial including identifying the vitamin D level needed to protect t...

Results of a Prostate Cancer/Vitamin D Trial ...
Our recent epidemiological study (Ahonen et al., Cancer Causes Control 11 (2000) (847-852)) suggests that vitamin D deficiency may increase the risk of initiation and progression of prostate cancer. The nested case-control study was based on a 13-year follow-up of about 19000 middle-aged men free of clinically verified prostate cancer.

Vitamin D and prostate cancer.
Nowadays, there are so many products of vitamin d and prostate cancer treatment in the market and you are wondering to choose a best one.You have searched for vitamin d and prostate cancer treatment in many merchants, compared about products prices & reviews before deciding to buy them. You are in RIGHT PLACE. Here are […]

Top 10 Vitamin D And Prostate Cancer Treatment - Home Design
A 2013 review for the U.S. Preventive Services Task Force found that taking vitamin D and/or calcium supplements showed no overall effect on rates of cancer or deaths from cancer, including prostate cancer.

Prostate Cancer, Nutrition, and Dietary Supplements (PDQ ...
Breaking News. Vitamin D protects against advanced cancer and metastases

Vitamin D protects against advanced cancer and metastases ...
Vitamin D has been studied in the treatment of breast cancer and colon cancer. Prostate cancer will affect 1 in 6 men. However, the disease will be fatal in only 1 in 36, due to its potential for...

"Sunlight, Vitamin D, and Prostate Cancer Risk" P. J. Hyde This science-based book is the first to demonstrate that in prostate cancer, insufficient access to the sun's short wavelength ultraviolet-B irradiance, necessary for photosynthesis of vitamin D, increases the risk of progression. The author surveys five populations with steeply elevated mortality from prostate cancer: African-North Americans, Norwegians, Swedes, Swiss and Danes. He finds that insufficient exposure to UV-B and inadequate photosynthesis of vitamin D is common to them all. Based on data extracted from World Health Statistics Annuals published in hard copy by the World Health Organization (WHO) in the 1980s, graphs depicting age-specific rates of prostate cancer mortality in Western European countries show that the Swiss rates are anomalously high for the country's latitude and may even surpass the rates for Norway and Sweden. Age-standardized (world) statistics published on-line by the WHO's International Agency for Research into Cancer (IARC) in its Globocan 2000 Cancer Epidemiology Database list identical rates for Norway, Sweden and Switzerland. The age-specific and age-standardized rates of prostate cancer mortality reported for other northern alpine countries are normal for the latitudes. Their more extensive surface areas and dispersed populations, with the great majority of their citizens residing far from the Alps, are consistent with this. In a population survey conducted by Swiss scientists from two institutions, blood levels of calcidiol (25-hydroxyvitamin D), the body's major circulating reservoir of the vitamin, were measured in a large representative sample of adults in all age groups. Subjects older than 65 were found to be spending less than 30 minutes a day outdoors in the summer. This is significant, because the capacity of exposed skin to photosynthesize vitamin D declines with age. Moreover, its inverse correlation with the intensiveness of pigmentation means that African-North Americans in particular need to spend longer periods of time in direct sunlight than may be possible for a variety of reasons. The book contends that many aging individuals residing far from the Equator can achieve year-round sufficiency in vitamin D through relatively frequent, brief periods of exposure to sunlight, reinforced in winter by adequate supplementation. Published research indicating how many international units of the vitamin may be needed daily is also discussed. There is a detailed description of the mechanism whereby minute quantities of a powerful steroid hormone (calcitriol) metabolized from calcidiol, bind

to the hormone's receptors (VDR) within the nuclei of the cells. By a signalling procedure involving "cross talk" between the hormone and the VDR, and resulting changes in the transcription of genes within the nuclei, proliferation of malignant cells can be inhibited. Besides prostate cancer, several other common cancers are reported to be associated with deficiencies of vitamin D. "Recommended reading for anyone interested in lowering their risk of prostate cancer"---Christopher Morash, MD, FRCSC, Chief of Urosurgical Oncology, Ottawa Regional Cancer Centre, Ottawa, Ontario. ISBN: 1-4010-8258-0 (paperback) and 1-4010-8259-9 (hardback). Surface: Xlibris Corporation at International Plaza II, Suite 340, Philadelphia, PA, U.S.A. 19113 Web: http://www.xlibris.com/bookstore/bookdisplay.asp'bookid=17092 Email: Orders@Xlibris.com Price (paperback): U

Background: The concept that cancer incidence and mortality are related to latitude was first suggested in 1930. Since then, there have been a plethora of studies addressing that connection. Studies of vitamin D have demonstrated its anti-proliferative, anti-angiogenesis and differentiating properties which are all anti-neoplastic. The relationship between prostate cancer and latitude has long been suspected. Prostate cells have vitamin D receptors and enzymes for hydroxylation of vitamin D metabolites and enabling anti-carcinogenetic effects. As a result of these findings, many ongoing studies are evaluating whether vitamin D deficiency impacts prostate cancer risk. This review summarizes the last two years of published studies on this topic. Methods: The search used Ovid/Medline, PubMed, CINAHL and Web of Science databases limited to include clinical studies, English language, major journals and including articles from 2007 to the present. Review of the abstracts produced the relevant studies, and the bibliographies of these articles led to other sources not found in the search. Keywords: prostate cancer, risk, vitamin D and vitamin D deficiency. Results: Five studies reviewed from the end of 2007 to the present were germane to the research topic. One study was an observational study, one a prospective study and the remainder were case control studies from much larger randomized controlled trials. Only one case reviewed the relationship between solar radiation and prostate cancer risk and all the relationships were positive for low solar radiation and higher prostate cancer risk. Three studies reviewed the relationship of serum levels of vitamin D and prostate cancer risk relative to polymorphisms of the VDR and two found that low levels of serum vitamin D increase prostate cancer risk, especially aggressive prostate cancer risk. One large study examining serum concentrations of only one vitamin D metabolite found no association with PCa. Conclusions: Vitamin D deficiency is a widespread health issue. There is an increased risk of prostate cancer with low vitamin D levels. The risk is greater with more aggressive disease. Conflicting results regarding PCa risk and VDR polymorphisms in light of low vitamin D levels, and it may be a one-time serum sample which causes these discrepancies. There is an association between solar radiation and PCa risk, need to follow the serum levels or solar exposure of patients much younger. Both metabolites need to be measured to understand the impact on prostate cancer risk, especially since the data shows an increased risk with aggressive disease with the one typically not measured. One serum sample does not provide information needed for understanding the relationship between vitamin D and PCa. More prospective studies beginning at an earlier age with more serum samples and solar radiation studies are needed in order to better understand the relationship between prostate cancer and vitamin D.

Genes may play a strong role in prostate cancer etiology but epidemiological studies suggest that prostate cancer risk is largely determined by gene and environmental interactions. In order to explore the effects of UV exposure, serum Vitamin D, and skin color on prostate cancer risk in African American men. Ninety affected AA men with histologically diagnosed adenocarcinoma of the prostate; PSA of> 2.5 ng/ml and a positive DRE were recruited under the direction of Dr. Mireku-Boateng from the division of Urology at the Howard University Hospital and forty age and ethnicity matched controls have been recruited through the monthly free screenings program at the Howard University Cancer Center. For each prostate cancer patient and matched control we have collected information on personal and family history, and blood samples for candidate gene testing. In order to measure the intake of dietary Vitamin D each subject completed the standardized food frequency questionnaire and the serum circulating levels of 25-OH Vitamin D have been measured by Enzyme Immunoassay for all participants. To elucidate their exposure to UV from childhood until current the UV exposure questionnaire has been completed. In addition their constitutive skin color (M-index) measured has been done by using the dermaspectrophotometer.

The scientific rationale for this Idea Grant was to clarify whether modifiable, mainly nutritional, influence levels of IGF-l, IGFBP-3, 1,25 (OH)2 vitamin D (l,25(OH)2D), and 25(OH)vitamin D. High levels of IGF-l and low l,25(OH)2D have been shown to be related to elevated risk of prostate cancer. Some dietary factors that hypothetically impact on these serological factors, including total energy intake, calcium intake and protein intake, have been associated with prostate cancer risk. In the current study, none of the hypothesized factors showed correlations with l,25(OH)2D in 630 men. These results do not support our original hypothesis that 1,25 vitamin D is an important mediator of risk of several dietary risk factors of prostate cancer. We were able to demonstrate that intake of protein and minerals influence IGF-l levels moderately in 751 men, but men with high intakes of protein and minerals were not at higher risk of prostate cancer relative to men with low intakes of these. These findings suggest that the range of dietary influences on prostate cancer in generally well-fed populations is unlikely to have a major impact on risk of prostate cancer.

Substantial data indicate the broad importance of vitamin D-based signaling in normal human physiology and the broad effects of vitamin D deficiency. Vitamin D may play a role not only in the control of bone and mineral metabolism, but also appears to be involved in immune function, cardiovascular health, thrombosis and vasculogenesis and neuromuscular function. Considerable epidemiologic data demonstrate

that low vitamin D serum levels occur very commonly in normal adult populations and that vitamin D deficiency is associated with an enhanced risk of cancer death from lung, prostate, head & neck, colorectal and other gastrointestinal cancers. In addition, preclinical data provide evidence that calcitriol and other active analogues of calcitriol have anti-proliferative, pro-differentiative, pro-apoptotic and anti-angiogenic activity in numerous in-vitro and in-vivo models. It is quite clear that, while it requires high exposure to calcitriol to induce these effects, such exposure can be readily achieved when high dose intermittent therapy is given.

1,25-(OH)2 vitamin D3 inhibits cell proliferation of a variety of cancers including prostate. In the human prostate cancer cell line LNCaP, 1,25-(OH)2 vitamin D3-mediated growth inhibition is attributed to cell cycle G1 accumulation which correlates with a robust decrease of cyclin-dependent kinase 2 (CDK2) activity and pronounced relocalization of CDK2 into the cytoplasm. Nuclear targeting CDK2 blocks the 1,25-(OH)2 vitamin D3-mediated growth inhibition and cell cycle G1 accumulation. Further, the nuclear targeted CDK2 blocks 1,25-(OH)2 vitamin D3-mediated inhibition of CDK2 activity and nuclear exclusion in LNCaP cells. Therefore, CDK2 cytoplasmic relocalization is the key mechanism for 1,25-(OH)2 vitamin D3 effects. Since cyclin E is important for CDK2 nuclear localization and activation, 1,25-(OH)2 vitamin D3 may exert its effects through regulation of cyclin E. Cyclin E but not a cyclin E mutant deficient in CDK2 binding reverses 1,25-(OH)2 vitamin D3-mediated antiproliferation which suggests the involvement of cyclin E as a mechanism. However, the studies showed no effects of 1,25-(OH)2 vitamin D3 on cyclin E levels, intracellular localization or binding to CDK2. In order to develop a model for studying 1,25-(OH)2 vitamin D3-mediated antiproliferative effects, LNCaP vitD. R cell line, a vitamin D resistant LNCaP derivative, was generated by continuously culturing of LNCaP cells in medium supplemented with 10 nM 1,25-(OH)2 vitamin D3 for over 9 months. The initial characterization of this cell line showed complete resistance to 1,25-(OH)2 vitamin D3-mediated effects. Analysis of vitamin D regulation of VDR target gene expression revealed that vitamin D resistance in LNCaP vitD. R cells was not due to deregulation of VDR signaling. HDAC inhibitor Trichostatin A (TSA) did not confer sensitivity of LNCaP vitD. R cells to vitamin D treatment suggested the resistance to 1,25(OH)2 vitamin D3 effect of LNCaP vitD. R cells is not due to histone deacetylase remodeling of the chromatin structure which leads to inhibition of gene transcription. While the partial sensitization of LNCaP vitD. R cells to 1,25(OH)2 vitamin D3 effect by demethylation reagent 5-Aza-2-deoxycytidine treatment suggested a set of genes involved in 1,25(OH)2 vitamin D3-mediated antiproliferative effects is silenced via hypermethylation in LNCaP vitD. R cells. These results suggested LNCaP vitD. R cell line is a useful tool and further studies to elucidate the genes involved in this effect will help uncover the mechanisms of 1,25(OH)2 vitamin D3-mediated antiproliferative effects.

The scientific rationale for this Idea Grant was to clarify whether modifiable, mainly nutritional, influence levels of IGF-l, IGFBP-3, 1,25 (OH)2 vitamin D (l,25(OH)2D), and 25(OH)vitamin D. High levels of IGF-l and low l,25(OH)2D have been shown to be related to elevated risk of prostate cancer. Some dietary factors that hypothetically impact on these serological factors, including total energy intake, calcium intake and protein intake, have been associated with prostate cancer risk. In the current study, none of the hypothesized factors showed correlations with l,25(OH)2D in 630 men. These results do not support our original hypothesis that 1,25 vitamin D is an important mediator of risk of several dietary risk factors of prostate cancer. We were able to demonstrate that intake of protein and minerals influence IGF-l levels moderately in 751 men, but men with high intakes of protein and minerals were not at higher risk of prostate cancer relative to men with low intakes of these. These findings suggest that the range of dietary influences on prostate cancer in generally well-fed populations is unlikely to have a major impact on risk of prostate cancer.

Abstract: Prostate cancer cells contain vitamin D receptor for 1a,25-dihydroxyvitamin D3 [1a,25(OH)2D3], which is known to inhibit the proliferation and invasiveness of these cells. Normal prostate cells and several prostate cancer cell lines convert 25-hydroxyvitamin D 3 [25(OH)D3] to 10,25(OH)2 D3 . In addition, epidemiological evidence correlated an inverse relationship between vitamin D status and prostate cancer risk, suggesting that vitamin D and its metabolism may be important in the development and growth of prostate cancer. Using a human androgen-insensitive prostate cancer xenograft mouse model, the effect(s) of dietary vitamin D and calcium on tumor growth were evaluated. DU-145 cells were implanted in mice and monitored for 76 days. Serum, for 25(OH)D and calcium determinations, and tumors, for immunohistochemistry and gene expression analysis, were collected. Tumor growth was highest in mice fed a normal calcium, vitamin D deficient diet. Diets containing high calcium, with or without vitamin D, did not alter tumor growth compared to the normal calcium vitamin D sufficient diet. To elucidate the role of 1a,25(OH) 2 D3 production by 25-hydroxyvitamin D-1a-hydroxylase (CYP27131) on prostate cancer cell growth, LNCaP cells were stably transfected with CYP27B1 (1a-S cells). 1a-S cells converted 25(OH)D 3 to 1a,25(OH)2 D3 unlike untransfected LNCaP cells. There was a dose dependent decrease in 3 H-thymidine incorporation in 1a,25(OH)2 D3 treated LNCaP cells, not seen with 25(OH)D3 treatment, and in 1a-S cells treated with 25(OH)D3 . In DU-145 cells, the decrease in 3 H-thymidine incorporation seen with 25(OH)D3 treatment was diminished with suppression of CYP2781 with siRNA. 1a,25(OH)2 D3 treated LNCaP cells and 25(OH)D3 treated 1a-S cells demonstrated increased G1 phase accumulation and apoptosis while 25(OH)D3 treatment had no effect in LNCaP cells. 1a,25(OH) 2 D3 , but not 25(OH)D3 , in LNCaP cells and 25(OH)D 3 in la-S cells increased cell cycle regulatory gene expression; CDKN1A , CDKNIB and TP53 , and opposing apoptotic genes, BAX and BCL-2 , were induced and suppressed, respectively. 25-hydroxyvitamin D-24-hydroxylase (24-OHase) suppression enhanced 25(OH)D3 and 1a,25(OH)2 D 3 effects

in LNCaP and la-S cells. This study supports the hypothesis that local production of 1a,25(OH)2 D is important to inhibiting prostate cancer growth and suggests dietary vitamin D as a preventive agent in androgen-insensitive prostate cancer.

The Role of Vitamin D in the Prevention and Treatment of Prostate Cancer.