# HCR

# 5

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5</SUBJECT><COMM>SHSS27</COMM></TARGET>

Alaska State Legislature

State Capitol, Room 102
Juneau, AK 99801
Phone: 465-2689
Fax: 465-3472
Toll Free (800) 665-2689
Representative_Paul_Seaton@legis.state.ak.us

345 W. Sterling Highway
Suite 102B
Homer, AK 99603
Phone: 235-2921
Fax: 235-4008

**REPRESENTATIVE Paul Seaton**
District 35

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## HCR 5
## Sponsor Statement

**Short Title: A resolution relating to prevention of disease and to vitamin D.**

HCR 5 urges the State of Alaska to adopt a disease prevention model of health and to increase the awareness of the preventative benefits of vitamin D supplementation, and increase vitamin D availability to reduce health impacts and costs.

Mounting scientific evidence suggests a correlation between vitamin D sufficiency and significant decrease in incidence of cancer, diabetes, heart disease, rheumatoid arthritis, chronic pain, Seasonal Affective Disorder, oral disease, influenza, upper respiratory illness, tuberculosis, multiple sclerosis, osteoporosis, fractures in the elderly, autism, rickets, pregnancy complications, and hepatitis C.

For seven months out of the year, the sun angle is too low for Alaskans to be able to produce vitamin D from sunlight. Partly due to this long "vitamin D winter," Alaska has some of the lowest blood serum levels of vitamin D in the country. Correspondingly, Alaska also has relatively high rates of chronic disease. For example, according to the Alaska Division of Public Health, chronic diseases (cancer, heart disease, respiratory disease, and stroke, respectively) make up four of the top five causes of death in the state. These diseases come at a great cost both socially and financially to our state.

Deficiency in vitamin D may contribute heavily to this steep social and financial cost. Two studies mentioned in HCR 5 demonstrate this. The first one, examining the economic burden of vitamin D deficiency, estimated that Canada could lower the death rate by 37,000 deaths and save $14.4 billion dollars per year by increasing the national average of vitamin D blood serum levels. The second, similar study showed that the United States could have 50,000—63,000 fewer deaths and save $40-56 billion per year with sufficient population levels of vitamin D.

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With higher rates of chronic disease in northern latitudes, and fewer and fewer federal dollars coming in to cover the escalating healthcare and Medicaid costs in Alaska, it is imperative to prevent—rather than just treat—chronic disease. Prevention of chronic disease is a matter of lifestyle: healthy diet, frequent exercise, and adequate levels of vitamins and nutrients. Most people understand this, but are unaware that vitamin D deficiency needs to be addressed in every community of this state.

HCR 5 asks Governor Sean Parnell to establish "prevention of disease" as a primary model of health care in Alaska, and for the Department of Health and Social Services and health care providers to increase attention to vitamin D deficiency and promote supplementation. It also asks the Department to provide vitamin D supplements to the elderly, children, and pregnant women. Vitamin D supplementation is a low-cost measure that could help save lives, and significantly improve the health of many Alaskans, while saving millions of dollars in health treatment costs.

# FISCAL NOTE

**STATE OF ALASKA**
**2011 LEGISLATIVE SESSION**

Fiscal Note Number
Bill Version: HCR 5
() Publish Date

Identifier (file name): HCR5-LEG-COU-03-02-2011
Title: Relating to Prevention of Disease and to Vitamin D
Sponsor: Representative Seaton
Requester: House Health and Social Services Committee

Dept. Affected: Legislature
Appropriation: Legislative Council
Allocation: Session Expenses
OMB Component Number: 782

**Expenditures/Revenues** (Thousands of Dollars)

Note: Amounts do not include inflation unless otherwise noted below.

| | Appropriation Required | Information | | | | | |
|---|---|---|---|---|---|---|---|
| **OPERATING EXPENDITURES** | **FY 2012** | **FY 2012** | **FY 2013** | **FY 2014** | **FY 2015** | **FY 2016** | **FY 2017** |
| Personal Services | | | | | | | |
| Travel | | | | | | | |
| Contractual | | | | | | | |
| Supplies | | | | | | | |
| Equipment | | | | | | | |
| Grants & Claims | | | | | | | |
| Miscellaneous | | | | | | | |
| **TOTAL OPERATING** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** |
| **CAPITAL EXPENDITURES** | | | | | | | |
| **CHANGE IN REVENUES** | | | | | | | |

**FUND SOURCE** (Thousands of Dollars)

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 1002 Federal Receipts | | | | | | | |
| 1003 GF Match | | | | | | | |
| 1004 GF | | | | | | | |
| 1005 GF/Program Receipts | | | | | | | |
| 1037 GF/Mental Health | | | | | | | |
| Other Interagency Receipts | | | | | | | |
| **TOTAL** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** |

**Estimate of any current year (FY2011) cost**

**POSITIONS**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Full-time | | | | | | | |
| Part-time | | | | | | | |
| Temporary | | | | | | | |

**Why this fiscal note differs from previous version**

Initial Version

Prepared by: Shane Miller, Finance Manager — Phone 465-6626
Division: Administrative Services Division — Date/Time 3/2/11 1:51 PM
Approved by: Pamela Varni, Executive Director — Date 3/2/2011
Legislative Affairs Agency

(Revised 12/29/2010 OMB)

STATE OF ALASKA
2011 LEGISLATIVE SESSION

BILL NO. HCR5

**Analysis**

This fiscal note has zero impact on the Legislative Affairs Agency.

# FISCAL NOTE

**STATE OF ALASKA**
**2011 LEGISLATIVE SESSION**

Fiscal Note Number
Bill Version **HCR005\M**
() Publish Date

Identifier (file name): HCR005-DHSS-PHN-03-09-11
Title Vitamin D Supplements
Sponsor Rep. Seaton
Requester House HSS

Dept. Affected Health and Social Services
Appropriation Public Health
Allocation Nursing
OMB Component Number 288

**Expenditures/Revenues** (Thousands of Dollars)

Note: Amounts do not include inflation unless otherwise noted below.

| | Appropriation Required | Information | | | | | |
|---|---|---|---|---|---|---|---|
| **OPERATING EXPENDITURES** | **FY 2012** | **FY 2012** | **FY 2013** | **FY 2014** | **FY 2015** | **FY 2016** | **FY 2017** |
| Personal Services | | | | | | | |
| Travel | | | | | | | |
| Services | | | | | | | |
| Commodities | | | | | | | |
| Capital Outlay | | | | | | | |
| Grants | | | | | | | |
| Miscellaneous | | | | | | | |
| **TOTAL OPERATING** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** |
| **CAPITAL EXPENDITURES** | | | | | | | |
| **CHANGE IN REVENUES** | | | | | | | |

**FUND SOURCE** (Thousands of Dollars)

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 1002 Federal Receipts | | | | | | | |
| 1003 GF Match | | | | | | | |
| 1004 GF | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| 1005 GF/Program Receipts | | | | | | | |
| 1037 GF/Mental Health | | | | | | | |
| Other (please identify) | | | | | | | |
| **TOTAL** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** | **0.0** |

**Estimate of any current year (FY2011) cost**

**POSITIONS**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Full-time | **0** | 0 | 0 | 0 | 0 | 0 | 0 |
| Part-time | | | | | | | |
| Temporary | | | | | | | |

**Why this fiscal note differs from previous version (if initial version, please note as such)**

Not Applicable. Initial version.

Prepared by Ward B. Hurlburt, M.D., MPH - Chief Medical Officer / Director
Division Public Health
Approved by Alison Elgee, Assistant Commissioner
DHSS Finance & Management Services

Phone 269-6680
Date/Time 3/4/11 12:00 AM
Date 3/9/2011

(Revised 1/27/2011 OMB)

FISCAL NOTE

STATE OF ALASKA
2011 LEGISLATIVE SESSION

BILL NO. HCR005

## Analysis

This House Concurrent Resolution relates to the prevention of disease and the benefits of Vitamin (Vit.)D.

The division is submitting a zero fiscal note because no new authorization is necessarily invoked by passage of this resolution. Should the Legislature decide to require the department to undertake activities through additional legislation, an additional appropriation will be necessary.

This analysis estimates the cost of implementing the four activities in the resolution if the division were to implement them, although the division has no plans to do so at this time. The assumptions used to calculate the fiscal note are detailed on page 3.

The resolution includes language that encourages or urges the Department of Health and Social Services to implement four activities in regards to Vitamin D supplements. The activities are

(1) to increase attention to Vit. D deficiency, Vit. D testing, and to promote awareness of the long-term health benefits of and increased chances of cancer survival with sufficient levels of Vit. D;
(2) investigate substituting Vit. D supplementation for influenza vaccination as a less costly method for preventing influenza;
(3) provide Vit. D supplements to the elderly to prevent bone loss, falls, fractures, and other age-related health problems; and
(4) provide Vit. D supplements to pregnant women and infants to prevent pregnancy complications, preterm births, type 1 diabetes, and rickets.

For the first activity, the department would contract for professional services to create an awareness campaign on the benefits of Vit. D. at an estimated cost of $50.0. This would be an ongoing cost.

For the second activity, the department would require $100.0 for a PhD-level epidemiologist (1 FTE, existing PCN, Range 20/A) plus $35.0 for overhead (rent, utilities, etc.). The epidemiologist would conduct a study investigating subsitution of Vit. D supplemention for influenza vaccination as a less costly method of preventing influenza. This study would take at least three years. This is a conservative estimate and could be double or triple the amount depending on the final study design.

For the third and fourth activities, the department would provide Vit. D supplements through Public Health Nursing Clinics at a cost of $211.2 annually. Elderly, pregnant women, and infants who go to the clinics would receive a limited office visit to determine if Vitamin D supplements were indicated. Of the approximately 17,200 public health clinic clients seen in 2011, it is assumed 3,225 (18.75%) could have received one or more 90-day supplies of Vit. D supplements. The client would receive a limited office visit each time the client came to the clinic for more supplements to determine if Vit. D was still medically necessary and evaluate their health condition. Public Health Nursing would report their results to the epidemiologist conducting the Vit. D study.

The total cost for Years 1 to 3 would be $396.2. Years 4 and 5 would be $261.2 since the second activity for a study would be completed by then.

(Continued on page 3.)

FISCAL NOTE

STATE OF ALASKA
2011 LEGISLATIVE SESSION

BILL NO. HCR005

**Analysis Continued**

**Assumptions:**

Clients
17,200 total clients served by Public Health Nursing, 2011
75% percent PHN clients who are elderly/pregnant women/infants
12,900 clients eligible for Vit. D supplements
25% percent clients eligible who receive Vit. D
3,225 clients eligible who receive Vit. D
806 clients (25%) visit 1/year
806 clients (25%) visit 2/year
806 clients (25%) visit 3/year
807 clients (25%) visit 4/year

Medical Supply Unit Cost
$ 1.20 Cost is $0.10 per Vit. D pill (over-the-counter, wholesale)
At 1 dose per week, with 12 doses dispensed per clinic visit (90-day supply)
$ 25.00 Cost of 15 min. limited office visit for each clinic visit (max. 4 /year)
$ 26.20 total cost per clinic visit for Vit. D

Total Annual Cost
$ 211,238 medical supplies for dispensing Vit. D
$ 100,000 personal services for 1 FTE epidemiologist (Range 20/A) to investigate Vit. D as substitute for influenza vaccine (Years 1-3)
$ 85.000 contractual costs for awareness campaign of benefits of Vit. D ($50.0); overhead for 1 FTE ($35.0)
$ 396,238 total annual cost Year 1

This analysis includes only the direct medical supply and administrative costs of implementing the resolution. The cost savings if patients opted for Vit. D supplements in lieu of influenza vaccine are not included in this analysis. Nor does it include the additional cost of treating patients who did not get vaccinated for influenza and later became ill.

The annual cost per person for Vit. D supplements (assuming 52 doses, 4 visits) is $104.80. In comparison, the medical supply unit cost of influenza vaccine is approximately $33-$66 annual cost per person (assuming $8/dose plus $25 limited office visit for 1-2 doses/year).

The resolution notes that part of the budget of the Department of Health and Social Services is used to treat illnesses that could be prevented with adequate blood serum levels of Vit. D. Those potential costs savings are not included in this analysis as the amount of avoided costs are indeterminate and would likely not be realized in the time frame covered by the fiscal note.

# Breast cancer risk by 25(OH)D

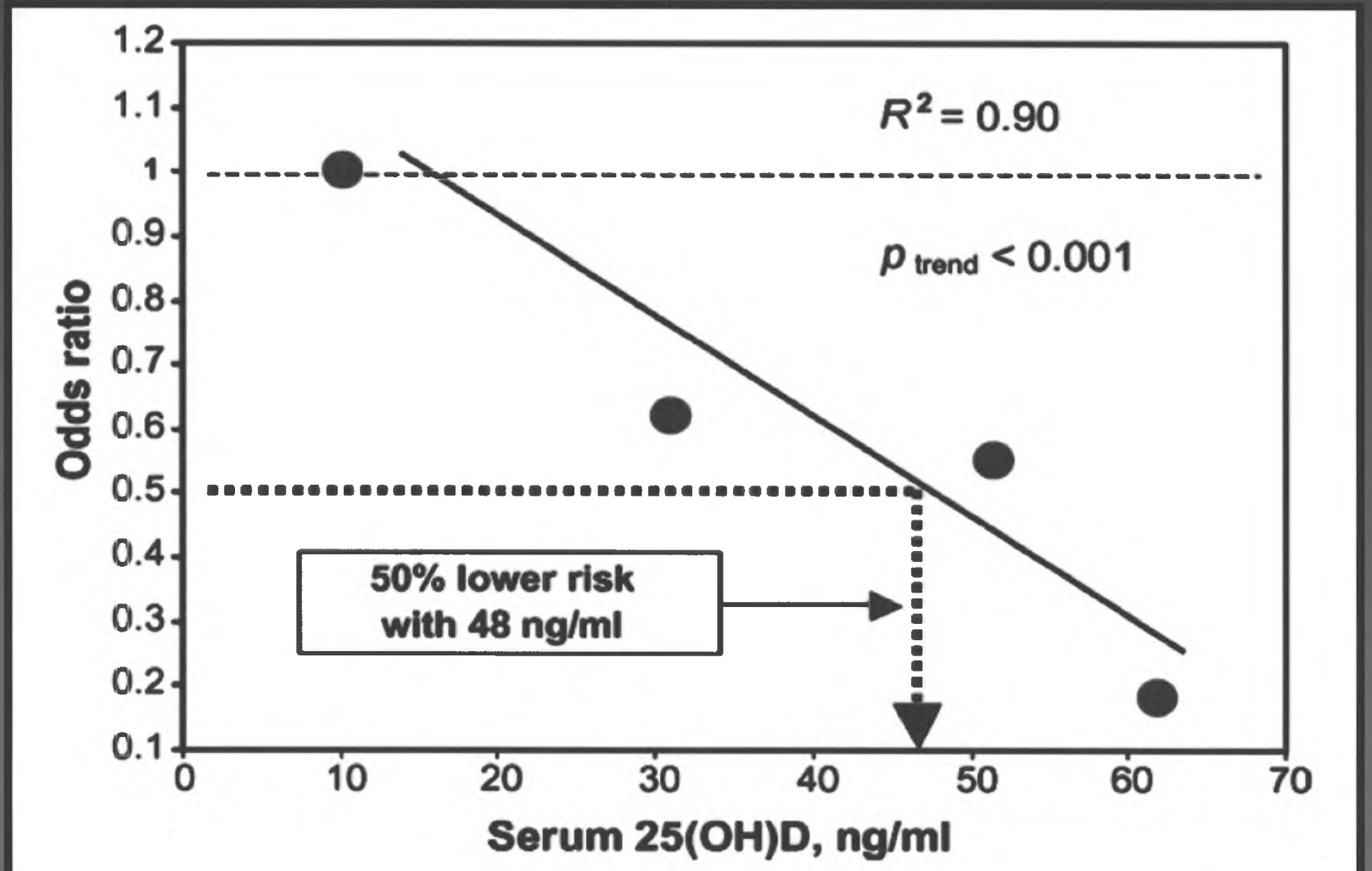
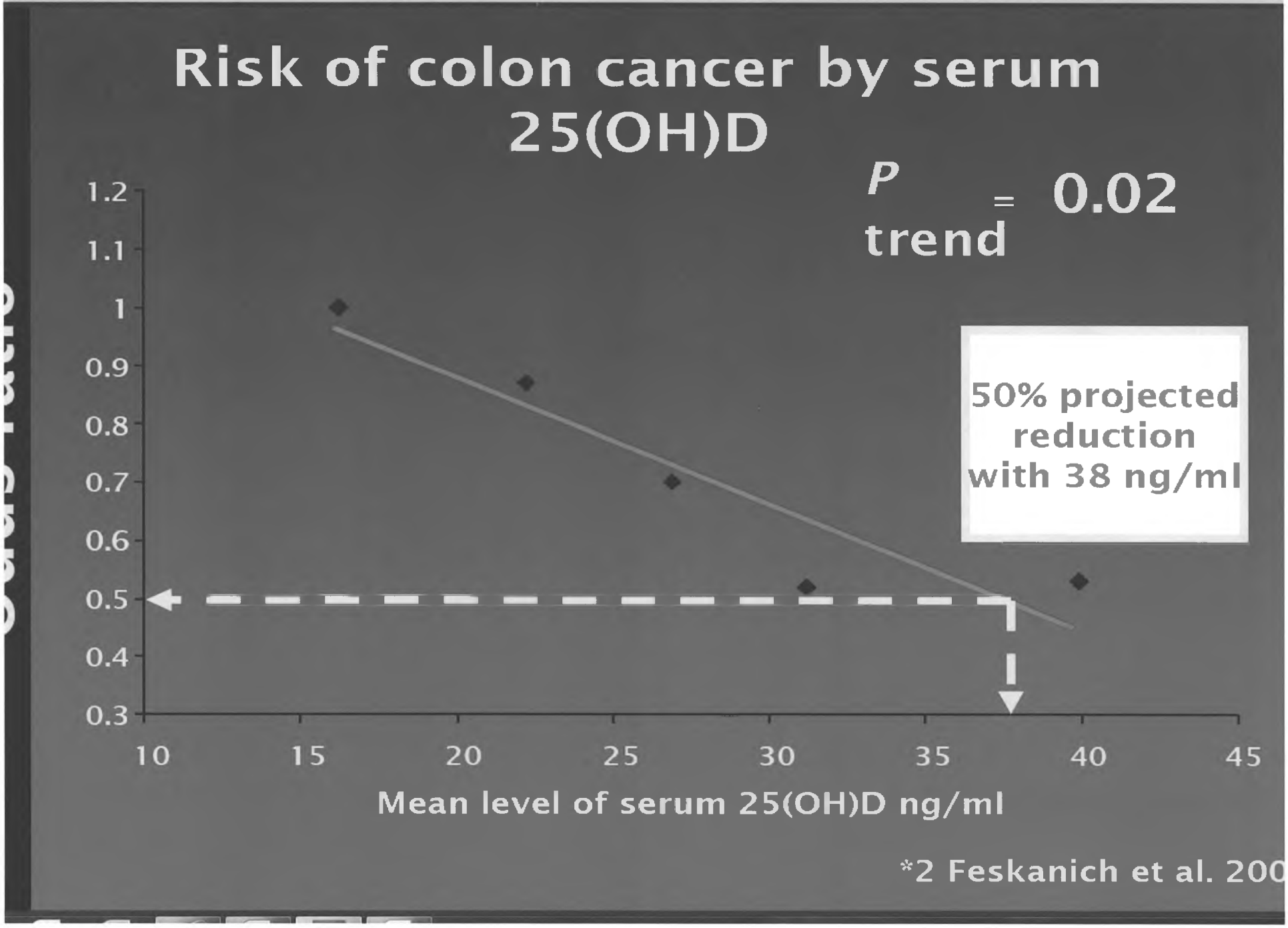



Fig. 2. Dose–response gradient of risk of breast cancer according to serum 25-hydroxyvitamin D concentration, St. George's Hospital, London

# Risk of colon cancer by serum 25(OH)D




*2 Feskanich et al. 200

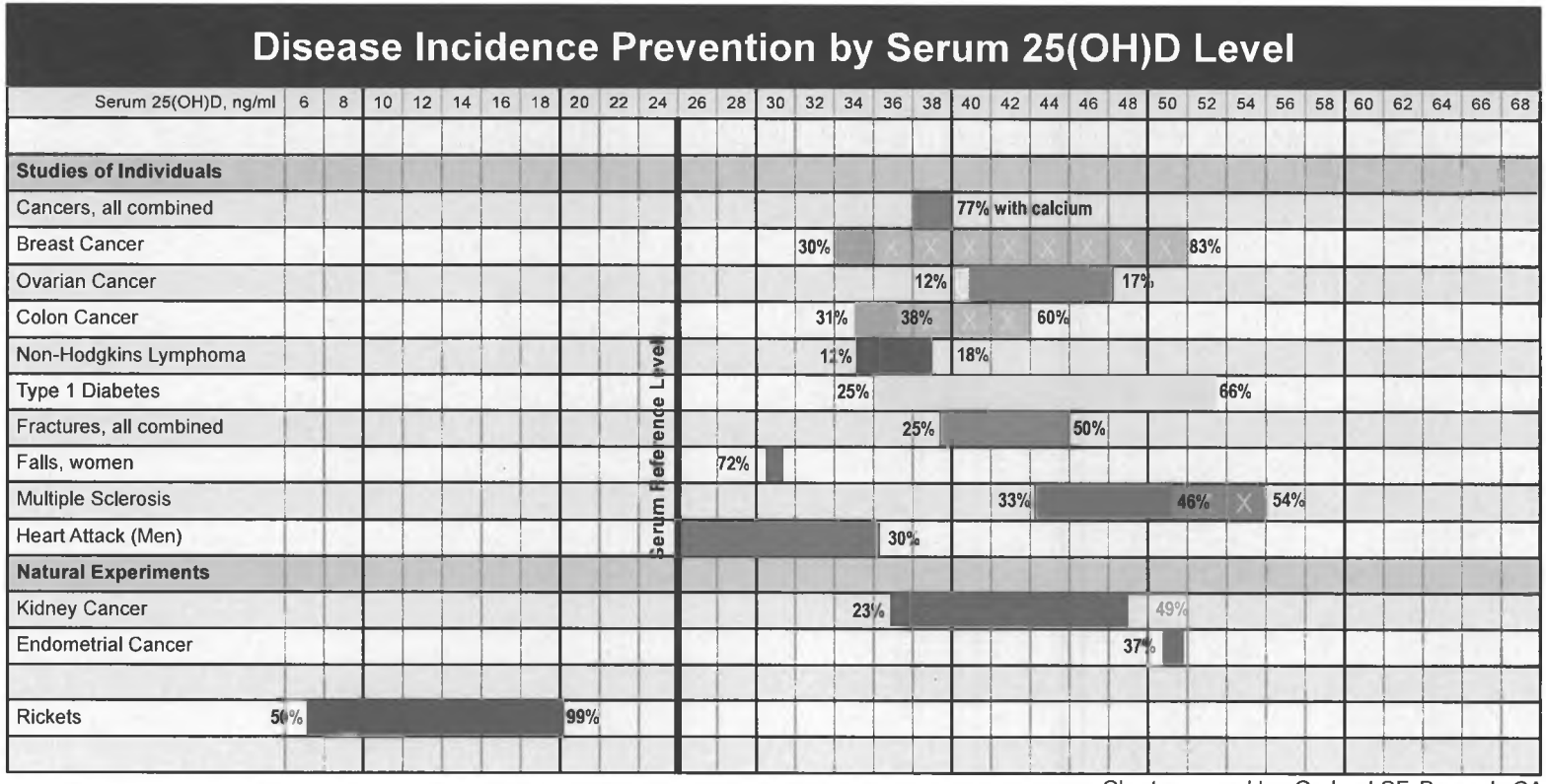



Chart prepared by: Garland CF, Baggerly CA

Legend:
All percentages reference a common baseline of 25 ng/ml as shown on the chart.
%'s reflect the disease prevention % at the beginning and ending of available data. Example: Breast cancer incidence is reduced by 30% when the serum level is 34 ng/ml vs the baseline of 25 ng/ml. There is an 83% reduction in incidence when the serum level is 50 ng/ml vs the baseline of 25 ng/ml.
The x's in the bars indicate 'reasonable extrapolations' from the data but are beyond existing data.

References:
All Cancers: Lappe JM, et al. Am J Clin Nutr. 2007;85:1586-91. Breast: Garland CF, Gorham ED, Mohr SB, Grant WB, Garland FC. Breast cancer risk according to serum 25-Hydroxyvitamin D: Meta-analysis of Dose-Response (abstract).American Association for Cancer Research Annual Meeting, 2008. Reference serum 25(OH)D was 5 ng/ml. Garland, CF, et al. Amer Assoc Cancer Research Annual Mtg, April 2008,. Colon: Gorham ED, et al. Am J Prev Med. 2007;32:210-6. Diabetes: Hyppönen E, et al. Lancet 2001;358:1500-3. Endometrium: Mohr SB, et al. Prev Med. 2007;45:323-4. Falls: Broe KE, et al. J Am Geriatr Soc. 2007;55:234-9. Fractures: Bischoff-Ferrari HA, et al. JAMA. 2005;293:2257-64. Heart Attack: Giovannucci et al. Arch Intern Med/Vol 168 (No 11) June 9, 2008. Multiple Sclerosis: Munger KL, et al. JAMA. 2006;296:2832-8. Non-Hodgkin's Lymphoma: Purdue MP, et al. Cancer Causes Control. 2007;18:989-99. Ovary: Tworoger SS, et al. Cancer Epidemiol Biomarkers Prev. 2007;16:783-8. Renal: Mohr SB, et al. Int J Cancer. 2006;119:2705-9. Rickets: Arnaud SB,

# SEASONAL VARIATION IN SERUM 25-HYDROXYVITAMIN D IN HEALTHY FAIRBANKS, ALASKA RESIDENTS: RELATION TO DIET AND SUNLIGHT EXPOSURE

A

THESIS

Presented to the Faculty of the University of Alaska

In Partial Fulfillment of the Requirements

for the Degree of

MASTER OF SCIENCE

By

Meredith Grant Tallas, B.S.

Fairbanks, Alaska

December 1986

## ABSTRACT

This study tested the hypothesis that lower UV radiation during the Fairbanks winter may cause seasonal vitamin D deficiencies. Forty-seven adult Caucasians (mean age, 34 years) donated monthly blood samples and gave 4-day food and sunlight exposure records during one year. There was a highly significant seasonal variation in serum 25-(OH)vitamin D (25-OHD), with the lowest mean mid-winter value above deficiency levels, and a yearly mean of 27 ng/ml for the full group. Analyses of variance indicated significant effects of vitamin D intake, sunlight exposure and sex on serum 25-OHD. Vitamin D intake appeared to be a more important factor determining year-round 25-OHD levels than sunlight exposure. Males had yearly mean 25-OHD levels 16% higher than females and 25% of the females, but none of the males, had yearly means less than 20 ng/ml indicating that females were at greater risk for the development of vitamin D deficiency.

4/2005 thru 3/2007
Ketchikan

RESEARCH

Research and Professional Briefs

# Vitamin D Deficiency in a Nonrandom Sample of Southeast Alaska Natives

JOSEPH T. FROST, MPH, RD; LANI HILL, FNP

**ABSTRACT**

Serum vitamin D has recently been inversely associated with risk for type 2 diabetes. Recent literature suggests that many more individuals than generally thought suffer from vitamin D deficiency. Southeast Alaskan Natives are at an increased risk due to limited sunlight exposure and possible inadequate vitamin D intake. Therefore, the relationship between vitamin D and glucose should be investigated specifically in the southeast Alaska Native population. A review of lab records yielded 83 charts of patients found to have a serum 25-hydroxyvitamin D during a 2-year period. Upon review of these charts, only nine of 83 vitamin D levels were found to exceed the 32 ng/mL (80 nmol/L) threshold. Age and vitamin D levels were associated in a positive linear relationship ($r=0.354$, $P=0.028$). The patients in the lowest vitamin D quartile were younger in age compared to the highest quartile (14.6 years, 95% confidence interval: 4.9, 24.29; $P=0.004$). The high rate of deficiency noted in this sample suggests this population should be further assessed for vitamin D deficiency. Future studies are needed to confirm the association between a vitamin D deficiency and diabetes incidence in this population.
*J Am Diet Assoc. 2008;108:1508-1511.*

Although traditionally associated with calcium absorption and bone health, expanded roles for vitamin D, including a relationship to diabetes, have recently been reported in the literature. Low serum 25-hydroxyvitamin D levels have been found in individuals with diabetes compared to controls (1,2). The Third Nutrition and Health Examination Survey revealed an inverse association between risk of diabetes and 25-hydroxyvitamin D levels (3). A similar association was seen in the Nurses' Health Study (4). Serum 25-hydroxyvitamin D levels were found to be inversely associated with pancreatic beta cell function, thus suggesting a cause and effect relationship with diabetes (5). Recent research suggests that many more individuals than generally thought, especially those in northern latitudes or with other sunlight restrictions, are deficient in vitamin D (6-9). Both the recommended levels of serum vitamin D and the Adequate Intake from food sources have recently been suggested to be inadequate. At these levels it is thought the beneficial effect of vitamin D cannot be achieved. (6,8,10-12). Despite being at an increased risk for vitamin D deficiency due to limited sunlight exposure (13,14) and lactose intolerance (up to 80%) (15), southeast Alaskan Natives have not been included in vitamin D deficiency studies. The objectives of this study were to investigate vitamin D deficiency in the southeast Alaskan Native population and its possible role in the incidence of diabetes.

## METHODS

A manual review of lab records for the 2-year time period of April 1, 2005, to March 30, 2007, was used to identify patients who had 25-hydroxyvitamin D tests completed as part of their care received at the Ketchikan Indian Community Tribal Health Clinic, a native health clinic on an Alaskan island that receives an annual rainfall of 12.5 feet per year. (Natives with varying degrees of native ancestry and who belong to one of 557 federally recognized tribes qualify for health care through Indian Health Service policies. In general, the term *American Indian* or *Alaskan Native* refers to those with native, although not necessarily exclusive, ancestry. Therefore, degree of Native blood varies in this population.)

This review yielded a nonrandom sample of 83 charts of Alaskan Natives. We conducted a retrospective electronic and manual review of these charts to investigate the possible relationship between serum vitamin D levels and abnormal glucose levels.

Electronic and paper charts of individuals known to have the test performed were reviewed, and test results for serum vitamin D and glucose, sex, age, and body mass index (BMI) were recorded. In the event of multiple vitamin D tests, only the initial test was used in data analysis. In all cases, the fasting glucose measured closest to the date of the initial vitamin D test was recorded. Glucose values obtained more than 1 year apart from the vitamin D test were excluded from the analysis. Of the 83 patients, fasting blood glucoses were recorded for 51.

*J. T. Frost is a Lieutenant Commander in the US Public Health Service; at the time of the study, he was the diabetes coordinator at the Ketchikan Indian Community Tribal Health Clinic, Ketchikan, AK, and a graduate student in the School of Public Health and Health Sciences, University of Massachusetts, Amherst. L. L. Hill is a family nurse practitioner, Ketchikan Indian Community Tribal Health Clinic, Ketchikan, AK.*

*Address correspondence to: Joseph Thomas Frost, MPH, RD, 692 Maquam Shore Rd, Swanton, VT 05488. E-mail: joefrostrd@yahoo.com*

*Manuscript accepted: February 29, 2008.*



*0002-8223/08/10809-0011$34.00/0*
*doi: 10.1016/j.jada.2008.06.427*

**Table.** Demographic data, 25-hydroxyvitamin D levels, and fasting glucose levels of patients with and without diabetes (n=83 except where otherwise noted)

| | Patients with diabetes (n) | Patients without diabetes (n) | *P* value |
|---|---|---|---|
| Sex | | | |
| Male | 12 | 16 | |
| Female | 16 | 39 | |
| | *mean±SD*[a] | | |
| Age[b] (y) | 56±14 | 45±16 | 0.004 |
| BMI[bc] | 38.9±9.2 | 31.9±7.2 | 0.001 |
| 25-hydroxyvitamin D (ng/mL) | 15.7±8.6 | 17.8±12.1 | 0.348 |
| Fasting glucose[ab] (mg/dL) | 139.8±49.9 (n=16) | 96.2±11.1 (n=35) | <0.001 |

[a]SD=standard deviation.
[b]Denotes category of statistical significance at the $\alpha=.05$ level.
[c]BMI=body mass index; calculated as kg/m$^2$.

Data from 2-hour oral glucose tolerance tests are not reported because only five were recorded. Random glucose levels were not analyzed due to multiple variables affecting their results. Vitamin D samples were drawn in-house and sent to a commercial laboratory for analysis using immunochemiluminometric assay. The threshold for the laboratory's vitamin D assay is 7.0 ng/mL (17.5 nmol/L); for data analysis all "undetectable" values were assigned 7.0 ng/mL (17.5 nmol/L).

Minitab version 15 (2007, Minitab Inc, State College, PA) was used for statistical analysis. Univariate regression calculations were done. Subjects were divided into quartiles based on vitamin D levels and comparisons were made using two-sample *t* tests with regard to glucose values, sex, age, and BMI. Results were considered significant at the $\alpha=.05$ level.

The protocol for this study was granted expedited status under 45 CFR 46.110 by the Alaska Area Institutional Review Board in Anchorage. It was also approved by Ketchikan Indian Community Tribal Health Clinic Administration staff, the Ketchikan Indian Community Health Board, and the Ketchikan Indian Community Tribal Council.

## RESULTS AND DISCUSSION

Of the 83 Native patients found to have received a 25-hydroxyvitamin D test during the 2-year period, 55 were female and 28 were male, with a mean age of 49 years (range=19 to 94 years) (Table). Twenty-eight of these patients were diagnosed with diabetes, of which all but two were vitamin D–deficient. Of the 55 without diabetes, only seven were vitamin D–sufficient (Figure). Subjects without diabetes were younger (−10.39, 95% confidence interval [CI]: −17.31, −3.47; $P=0.004$), had a lower BMI (−7.05, 95% CI: −11.06, −3.05; $P=0.001$), and had lower fasting blood glucose values (−43.63, 95% CI: −61.29, −25.97; $P=<0.001$). The mean (±standard deviation) 25-hydroxyvitamin D level was 17.1±11.0 ng/mL (42.8±27.5 nmol/L). Using two-sample *t* tests, no significant differences were found between sexes. Results indicated a significant positive linear relationship of age with respect to vitamin D levels ($r=0.354$, $P=0.028$).

Subjects were divided into quartiles based on 25-hydroxyvitamin D levels. These quartiles were compared in terms of sex, age, BMI, and fasting blood glucose values. With the exception of age, no statistically significant differences were observed between quartiles. The lowest quartile was on average 14.6 years younger compared with the highest quartile (95% CI: −24.29, −4.91; $P=0.004$). Patients known to have diabetes were then removed from the analysis (to control for confounding from the diabetes disease process or treatment, which may affect vitamin D), yielding similar results.

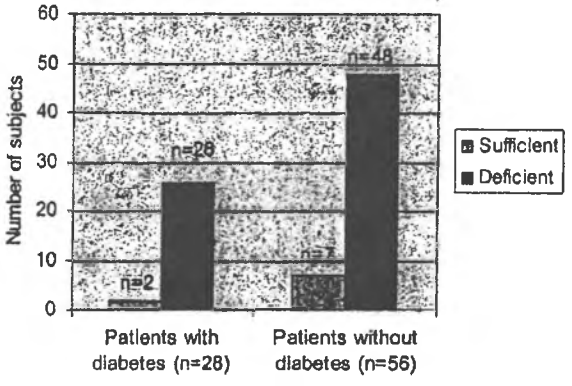



**Figure.** Vitamin D sufficiency of patients with and without diabetes using 32 ng/mL (80 nmol/L) as the minimum value for sufficiency.

No statistically significant relationships between vitamin D levels and blood glucose levels were observed. However, only nine of 83 vitamin D values were more than the recommended threshold of 32 ng/mL (80 nmol/L), and 18 of the vitamin D levels were so low that their actual value was undetectable. Although we cannot extrapolate these results to say that 89% of the population is vitamin D–deficient, the data suggest a basis for further investigation. It would seem unlikely to observe such a high rate of deficiency in this study, if there were not a high prevalence of vitamin D deficiency, even with perceptive clinicians ordering vitamin D tests for patients they thought to be at high risk for deficiency. The intent

of the study to compare serum glucose values between individuals of high and low serum vitamin D levels was somewhat undermined by the fact that so few normal values were observed. In fact, even the highest quartile of vitamin D values included values much less than the threshold of vitamin D deficiency.

The high prevalence of vitamin D deficiency found in this study is supported by studies reporting vitamin D deficiency in populations at higher latitudes or with otherwise limited sunlight exposure (although not at the magnitude found in this convenience sample) (7,8,12,13). The positive linear correlation between age and vitamin D levels found in this study differs from the common belief that vitamin D levels decrease with age. However, this positive correlation has been reported previously (7).

The finding related to subjects with known diabetes is also intriguing; only two of the 28 patients had 25-hydroxyvitamin D levels more than the 32 ng/mL (80 nmol/L) threshold. This is consistent with previous reports of vitamin D deficiency in patients with diabetes (1,2). Again, this is not a random sample but it does represent 20% of the known patients with diabetes per the Ketchikan Indian Community Diabetes Management System database. From a chart review we cannot determine that low vitamin D levels were a risk factor for diabetes in these individuals; however, this possibility deserves further investigation.

There are clear limitations to the design of this study. First, the study is retrospective in nature and therefore cannot determine causality. Second, this study did not assess known contributors to serum 25-hydroxyvitamin D levels, such as diet, supplementation, sunlight exposure, and medications that may interfere with vitamin D absorption and/or utilization. The results include 59 vitamin D levels measured during autumn and winter months, when vitamin D levels are thought to be lowest. Furthermore, the study consisted of a convenience sample of patients who were likely tested by providers on their suspicion that the patients had low vitamin D status. The study was not powered to detect differences; however, these data support the need for a larger study to investigate this association.

Despite these limitations, this study design offered a cost-effective opportunity to explore the possibility of vitamin D deficiency in this population and its relationship with diabetes. Given southeast Alaskan Natives' high risk for vitamin D deficiency, the increasing incidence of diabetes (100% to 125% in 14 years) (16), and recent research associating diabetes and vitamin D status, these results should not be dismissed. Although it is tempting to disregard vitamin D deficiency in this population as a genetic difference in normal values, it must be noted that African Americans also have lower vitamin D levels and have a 33% higher risk for cancer (17). Nor should Chiu and colleagues' conclusion that increasing a person's blood concentration of 25-hydroxyvitamin D from 10 ng/mL (25 nmol/L) to approximately 30 ng/mL (75 nmol/L) would improve insulin sensitivity by 60% be easily dismissed as not applicable to the native population (5).

Future efforts to explain the increase in incidence of diabetes should consider decreasing vitamin D–rich fish and fish oil consumption in the native population and the potential resultant decrease in serum vitamin D levels. It has been reported that in at least one group of Alaskan Natives that the consumption of traditional foods is much less common in younger natives compared with elders: 50% less in some categories of native food (18). Decreased traditional food consumption combined with additional lifestyle changes (eg, increased automobile use, more indoor activities, sedentary lifestyle) may be promote vitamin D deficiency.

## CONCLUSIONS

This study suggests southeast Alaskan Natives may be at risk for vitamin D deficiency. To the extent that vitamin D plays a role in the etiology of diabetes and other chronic disorders, Alaskan Natives with vitamin D deficiency may be at increased risk for these diseases. This study's finding of decreased vitamin D levels in younger individuals is of concern from a public health standpoint and should be further evaluated. Specifically, the possibility that elder natives consume more fish and fish oils and therefore have higher vitamin D levels should be considered. Registered dietitians should be aware of the emerging expanded role of vitamin D in chronic diseases such as diabetes, and should consider vitamin D status in their nutrition assessments, especially for patients with limited sun exposure.

The corresponding author is a commissioned officer in the United States Public Health Service but is submitting this manuscript as an individual and not on behalf of the Federal Government.

The authors thank Carolyn E. Ford, PhD, for her contribution to the conceptual design of the protocol, and the Ketchikan Indian Community/Official Village of Saxman Health Board and the Ketchikan Indian Community Tribal Council for their permission to conduct this research.

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# VITAMIN D VS BROAD SPECTRUM PHOTOTHERAPY IN THE TREATMENT OF SEASONAL AFFECTIVE DISORDER

F. MICHAEL GLOTH, III*, WASIF ALAM**, BRUCE HOLLIS***

* The Division of Geriatrics, The Department of Medicine, The Union Memorial Hospital and The Division of Geriatric Medicine and Gerontology, Depart-ment of Medicine, The Johns Hopkins University School of Medicine, Baltimore, Maryland. ** The Department of Medicine, The Union Memorial Hospital, Baltimore, Maryland. *** Department of Pediatrics, Children's Hospital, Medical University of South Carolina, Charleston, South Carolina. Correspondence to Dr. F. Michael Gloth III, Division of Geriatrics, The Union Memorial Hospital, 201 East University Parkway, Baltimore, Maryland 21218-2895. Phone: 410-554-2923; FAX: 410-554-6794.

**Abstract:** Seasonal Affective Disorder (SAD) is prevalent when vitamin D stores are typically low. Broad-spectrum light therapy includes wavelengths between 280-320 nm which allow the skin to produce vitamin D. This study was designed to test the hypothesis that vitamin D deficiency might play a role in SAD. A prospective, randomized controlled trial was conducted in a group of 15 subjects with SAD. Eight subjects received 100,000 I.U. of vitamin D and seven subjects received phototherapy. At the onset of treatment and after 1 month of therapy subjects were administered the Hamilton Depression scale, the SIGH-SAD, and the SAD-8 depression scale. All subjects also had serum levels of 25-hydroxyvitamin D (25-OH D) measured before and 1 week after intervention therapy. All subjects receiving vitamin D improved in all outcome measures. The phototherapy group showed no significant change in depression scale measures. Vitamin D status improved in both groups (74% vitamin D group, $p < 0.005$ and 36% phototherapy group, $p < 0.01$). Improvement in 25-OH D was significantly associated with improvement in depression scale scores ($r^2$=0.26; p=0.05). Vitamin D may be an important treatment for SAD. Further studies will be necessary to confirm these findings.

## Introduction

Because some evidence exists demonstrating a therapeutic effect of full spectrum light on people with a diagnosis of seasonal affective disorder (1,2), we hypothesize that depletion of vitamin D ("the sunshine vitamin") may have a role in the development of this syndrome. Similarly, it has been demonstrated that vitamin D status declines during winter (3,4), a time when Seasonal Affective Disorder is prevalent.

Poor nutritional habits, particularly in relation to dairy products and other foods supplemented with vitamin D, that many older adults may have, could obviate some of the obvious benefits of food supplementation (5,6). Liver disease, renal disease, and malabsorption, as well as some medications, also may affect vitamin D metabolism. These factors play a role in some form or another in many people.

Vitamin D deficiency has been associated with muscle weakness, abnormalities of bone metabolism, bone pain, fatigue, and various other metabolic abnormalities (7,8,9). There are many reasons to consider vitamin D deficiency as a source of Seasonal Affective Disorder (SAD) as well. First there is an intriguing temporal relationship between a nadir for vitamin D status and what, as the name of the disorder implies, is felt to be a peak occurrence of Seasonal Affective Disorder in the general population. Second, vitamin D receptors exist on many organs including the skin and brain (10, 11, 12, 13, 14, 15, 16). The vitamin D receptors in the brain and in many other sites thusfar have no clearly identified function. Third, phototherapy has been described as being useful in Seasonal Affective Disorder when full spectrum light is used. Coincidentally, the skin requires ultraviolet wavelengths in the range of 280 to 320 nm to convert 7-dehydrocholesterol to cholecalciferol (vitamin $D_3$). Full spectrum light includes ultraviolet wavelengths in the range of 280 to 320 nm.

This study was designed to identify a population diagnosed with SAD and to determine their vitamin D status and their clinical and serologic response to either dietary vitamin D supplementation or exposure to full-spectrum light.

## Methods

Fifteen subjects diagnosed with Seasonal Affective Disorder (Seasonal Pattern Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition)(17) and belonging to a support group for patients with SAD were enrolled into the study after providing informed consent. The protocol, which was approved by the Institutional Review Boards at the Sheppard and Enoch Pratt Hospital and the Union Memorial Hospital, involved obtaining psychological testing for SAD and Depression and serum samples for the measurement of levels of 25 hydroxyvitamin D (25 OHD) in a fasting state at entry into the study. Serum 25-hydroxyvitamin D was measured using a modification of a previously reported technique involving a direct radioimmuno-assay employing an antibody to the 23,24,25,26,27-petanor-C(22)-carboxylic acid of vitamin D (18). Subjects were screened for liver disease, renal disease, and malabsorption, as well as certain medications, that may

100,000 IU single dose ≈ 3,300 IU/d
Full spectrum light at 2 hr./d



fect vitamin D metabolism. Subjects could have had no treatment changes for the prior 3 months and did not have any other treatment changes during the month of study. Subjects were then randomized using computer generated randomization to receive one of two interventions. Either 100,000 I.U. of vitamin D (ergocalciferol) once as a single dose or scheduled exposure to full spectrum light on a daily basis. After 1 week, a repeat serum measure of vitamin D metabolites was obtained. Vitamin D is fat soluble and a single large dose of vitamin D typically raises the vitamin D status into the normal range for months (19).

≈3300 IU/d

Phototherapy was provided by a Sol-Lux-I phototherapy Unit (Triple "H" Associates, Owings Mills, Maryland). This is a 58.125 cm x 33.125 cm x 10.625 cm portable unit that produces 2,500 Lux of full spectrum light at 120 cm. Subjects had 2 hours of exposure per day verified by entries into a logbook.

At the end of one month (all subjects were evaluated between February and March) repeat psychological testing (Hamilton Depression Scale, the Combined Hamilton/SAD-8 or SIGH-SAD, and the SAD-8)(20,21,22), was obtained by an investigator blinded to the intervention. One month was selected for follow up because it was a period of time in which vitamin D status would improve regardless of the intervention chosen, and it was felt that this would allow sufficient time for an end-organ response. Mean baseline scores were compared between the vitamin D and phototherapy groups using a t-test. A comparison of mean change in test scores between the two groups was also made using analysis of variance (ANOVA testing). Vitamin D status was also compared in a similar fashion. Vitamin D status was measured after 1 week of treatment to determine whether overall vitamin D status improved into the normal range with intervention.

Regression analyses were also done between the change in vitamin D status and the change in each of the pschological tests for the combined population to ascertain a potential relationship between improvement in vitamin D status and resolution of depression in SAD.

## Results

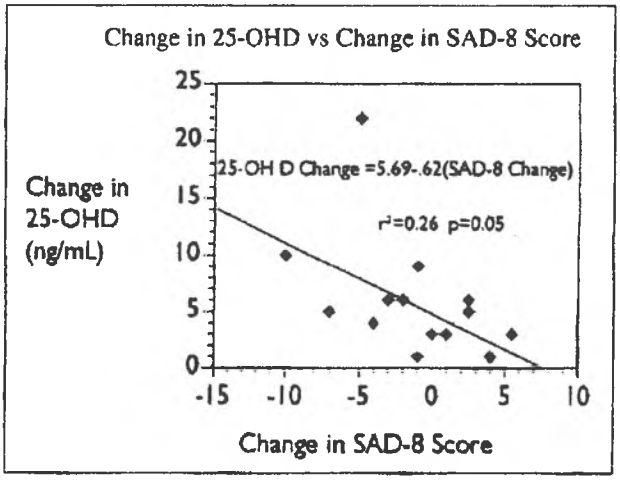
Baseline characteristics of the two groups were similar with regards to age (range 15-61 years), sex (14 females, 1 male), race (Caucasian), psychological test scores (Table 1 following page), and vitamin D status. The mean test scores for all psychological tests significantly improved in the group treated with vitamin D (Table I). None of the tests improved significantly in the group receiving phototherapy. The scores in the vitamin D supplementation group were also significantly better post-treatment when compared to the phototherapy post-treatment group (See Figure 1).

Vitamin D status in the groups was similar (11.0 ± 3.6 ng/mL vitamin D supplementation group vs 13.7 ± 3.1 ng/mL phototherapy group) at baseline. Following 1 week of treatment vitamin D status in the vitamin D supplementation group increased by 74% (19.1 ± 8.2 ng/ml; paired t-test p=0.003) compared to a 36% change (18.6 ± 2.5 ng/mL paired t-test p=0.007) in the phototherapy group.

A significant linear relationship was seen between improvement in vitamin D status and changes in SAD-8 scores before and 1 month after treatment in both groups combined (see Figure 1).

**Figure 1**

Improvement in vitamin D status was significantly associated with improvement in SAD-8 scores when both cohorts were examined together




## Discussion

Subjects identified with SAD all responded favorably to vitamin D supplementation while phototherapy did not consistently result in improvement. A combined analysis of the phototherapy group and the vitamin D supplementation group demonstrated a significant association between improvement in vitamin D status and SAD-8 scores. While the association was significant, one subject demonstrated an inordinately large increase in vitamin D (>20ng/mL). Removal of this potential outlier (analysis and regression not shown) only strengthened the relationship statistically.

Outside of abstracts, one other report has been published which pursues the role of vitamin D with SAD (23). This study selected women with only mild SAD-type symptoms and used 400 I.U. supplementation. Thus, the study cohort and the treatment regimen were substantially different. Serum levels of 25-hydroxyvitamin D were measured but not specifically reported in that study. That study did not show a benefit to the relatively small doses of vitamin D. Possible explanations for different outcomes in our study include: 1) The 400 I.U. supplement in the Harris study may have been inadequate to

substantially improve vitamin D status (24); 2) the outcome measures were different and, with milder symptoms, may not have had adequate sensitivity to detect changes; 3) baseline vitamin D status may not have been low, in which case supplementation would not be likely to have an effect.

It is of interest to note that, on average, our subjects had a vitamin D status below 15 ng/mL (38 nmol/L). Such levels have been associated with other physiologic aberrations, e.g. secondary hyperparathyroidism (5). Vitamin D supplementation also led to significant improvements in vitamin D status.

Vitamin D status seems to play a role in SAD and may play a role in the overall effect of phototherapy. By chance, the phototherapy group had higher baseline vitamin D status. Perhaps phototherapy responses in this study would have also been statistically evident if baseline and post-treatment differences in vitamin D status were of a greater magnitude. In this relatively modest study, the change in vitamin D status in the phototherapy group was less than the difference in the oral supplementation group which may explain the lack of response seen with phototherapy. This would support the hypothesis that inconsistent results of phototherapy may be associated with inadequate exposure to effectively alter vitamin D status or an incorrect diagnosis at the onset.

The present study design did not incorporate a placebo which may be a legitimate criticism in this limited study. Under the circumstances, however, the phototherapy control group may actually parallel the function of a placebo arm.

While these results are by no means conclusive, the findings are provocative enough to stimulate interest in additional studies with larger sample sizes. Such studies with similar results are necessary before the suggestion of a causal relationship can be confidently presented. Nevertheless, it is intriguing to consider that some of the depression seen in seniors who are confined indoors and likely to be vitamin D deficient may be attributable to SAD which may be amenable to simply replenishment with vitamin D.

*Acknowledgments:* The authors would like to acknowledge Dr. Henry Hyman, for assistance with the phototherapy boxes and all of the participants for their interest and dedication.

**Table I**

Depression scale differences before and after interventions and between cohorts (vertical)

| | Baseline | Post Treatment | Difference | p |
|---|---|---|---|---|
| Vitamin D (n=8) | | | | |
| Hamilton | 10.9 | 6.2 | -4.7 | 0.040 |
| SAD-8 | 6.0 | 2.8 | -3.2 | 0.003 |
| Comb'd (SIGH-SAD) | 16.9 | 9.1 | -7.8 | 0.008 |
| Phototherapy (n=7) | | | | |
| Hamilton | 12.6 | 11.3 | -1.3 | 0.49 |
| SAD-8 | 6.9 | 7.4 | 0.5 | 0.77 |
| Comb'd (SIGH-SAD) | 19.4 | 18.7 | -0.7 | 0.85 |
| Mean Difference | | | | |
| Hamilton | 1.7 | 5.0 | | |
| SAD-8 | 0.9 | 4.7 | | |
| Comb'd (SIGH-SAD) | 2.5 | 9.6 | | |
| p | | | | |
| Hamilton | 0.73 | 0.28 | | |
| SAD-8 | 0.73 | 0.07 | | |
| Comb'd (SIGH-SAD) | 0.69 | 0.14 | | |

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**Record: 1**

**Title:** Comparisons of estimated economic burdens due to insufficient solar ultraviolet irradiance and vitamin D and excess solar UV irradiance for the United States.

**Authors:** Grant WB; Garland CF; Holick MF

**Author Address:** Sunlight, Nutrition and Health Research Center (SUNARC), 2107 Van Ness Avenue, Suite 403B, San Francisco, CA 94109-2529, USA. wgrant@sunarc.org

**Source:** Photochemistry And Photobiology [Photochem Photobiol] 2005 Nov-Dec; Vol. 81 (6), pp. 1276-86.

**Publication Type:** Comparative Study; Journal Article; Research Support, N.I.H., Extramural; Research Support, Non-U.S. Gov't

**Language:** English

**Journal Information:** *Country of Publication:* United States *NLM ID:* 0376425 *Publication Model:* Print *Cited Medium:* Print *ISSN:* 0031-8655 (Print) *Linking ISSN:* 00318655 *NLM ISO Abbreviation:* Photochem. Photobiol. *Subsets:* MEDLINE

**MeSH Terms:** Cost of Illness*
Health Expenditures*
Ultraviolet Rays*/adverse effects
Vitamin D/*physiology
Cataract/economics; Dietary Supplements; Great Britain; Humans; Keratosis/economics; Melanoma/economics; Melanoma/etiology; Multiple Sclerosis/economics; Neoplasms/economics; Neoplasms/etiology; Osteoporosis/economics; Risk Reduction Behavior; Skin Neoplasms/economics; Skin Neoplasms/etiology; Sunlight; United States; Vitamin D/pharmacology

**Abstract:** Vitamin D sufficiency is required for optimal health, and solar ultraviolet B (UVB) irradiance is an important source of vitamin D. UVB and/or vitamin D have been found in observational studies to be associated with reduced risk for over a dozen forms of cancer, multiple sclerosis, osteoporotic fractures, and several other diseases. On the other hand, excess UV irradiance is associated with adverse health outcomes such as cataracts, melanoma, and nonmelanoma skin cancer. Ecologic analyses are used to estimate the fraction of cancer mortality, multiple sclerosis prevalence, and cataract formation that can be prevented or delayed. Estimates from the literature are used for other diseases attributed to excess UV irradiation, additional cancer estimates, and osteoporotic fractures. These results are used to estimate the economic burdens of insufficient UVB irradiation and vitamin D insufficiency as well as excess UV irradiation in the United States for these diseases and conditions. We estimate that 50,000-63,000 individuals in the United States and 19,000-25,000 in the UK die prematurely from cancer annually due to insufficient vitamin D. The U.S. economic burden due to vitamin D insufficiency from inadequate exposure to solar UVB irradiance, diet, and supplements was estimated at $40-56 billion in 2004, whereas the economic burden for excess UV irradiance was estimated at $6-7 billion. These results suggest that increased vitamin D through UVB irradiance, fortification of food, and supplementation could reduce the health care burden in the United States, UK, and elsewhere. Further research is required to confirm these estimates.

**Grant Information:** AR3696312 United States AR NIAMS NIH HHS; M01RR00533 United States RR NCRR NIH HHS

**Substance Nomenclature:** 1406-16-2 (Vitamin D)

**Entry Dates:** *Date Created:* 20051215 *Date Completed:* 20060609 *Latest Revision:* 20071114

**Record: 1**

**Title:** An estimate of the economic burden and premature deaths due to vitamin D deficiency in Canada.

**Authors:** Grant WB; Schwalfenberg GK; Genuis SJ; Whiting SJ

**Author Address:** Sunlight, Nutrition, and Health Research Center (SUNARC), San Francisco, CA 94164-1603, USA. wbgrant@infionline.net

**Source:** Molecular Nutrition & Food Research [Mol Nutr Food Res] 2010 Aug; Vol. 54 (8), pp. 1172-81.

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**Language:** English

**Journal Information:** *Country of Publication:* Germany *NLM ID:* 101231818 *Publication Model:* Print *Cited Medium:* Internet *ISSN:* 1613-4133 (Electronic) *Linking ISSN:* 16134125 *NLM ISO Abbreviation:* Mol Nutr Food Res *Subsets:* MEDLINE

**MeSH Terms:**
Health Care Costs*
Mortality*
Vitamin D/*administration & dosage
Vitamin D/*physiology
Vitamin D Deficiency/*economics
Vitamin D Deficiency/*physiopathology
25-Hydroxyvitamin D
2/blood; Adult; Calcifediol/blood; Canada/epidemiology; Child; Female; Humans; Infant; Male; Nutrition Policy; Pregnancy; Vitamin D Deficiency/epidemiology; Vitamin D Deficiency/prevention & control

**Abstract:** The objective of this work is to estimate the economic burden and premature death rate in Canada attributable to low serum 25-hydroxyvitamin D (25(OH)D) levels. Vitamin D deficiency has been linked to many diseases and conditions in addition to bone diseases, including many types of cancer, several bacterial and viral infections, autoimmune diseases, cardiovascular diseases, and adverse pregnancy outcomes. Canadians have mean serum 25(OH)D levels averaging 67 nmol/L. The journal literature was searched for papers reporting dose-response relationships for vitamin D indices and disease outcomes. The types of studies useful in this regard include randomized controlled trials, observational, cross-sectional, and ecological studies, and meta-analyses. The mortality rates for 2005 were obtained from Statistics Canada. The economic burden data were obtained from Health Canada. The estimated benefits in disease reduction were based on increasing the mean serum 25(OH)D level to 105 nmol/L. It is estimated that the death rate could fall by 37,000 deaths (22,300-52,300 deaths), representing 16.1% (9.7-22.7%) of annuals deaths and the economic burden by 6.9% (3.8-10.0%) or $14.4 billion ($8.0 billion-$20.1 billion) less the cost of the program. It is recommended that Canadian health policy leaders consider measures to increase serum 25(OH)D levels for all Canadians.

**Substance Nomenclature:**
1406-16-2 (Vitamin D)
19356-17-3 (Calcifediol)
21343-40-8 (25-Hydroxyvitamin D 2)

**Entry Dates:** *Date Created:* 20100810 *Date Completed:* 20101130

**Update Code:** 20101209

**PMID:** 20352622

**Database:** MEDLINE

Dr Emily A. Kane ND, LAc
Natural Healthcare
Juneau AK 99801

3-8-11

I strongly support any legislation or resolutions that would promote awareness of, and apply remedies to, Vitamin D deficiency which is rampant in Alaska.

We have become an indoor society. I have been checking Vit D levels routinely in my primary care practice for five years. Not one Alaskan who does not supplement with 3-6,000 IU's daily is replete. The fix is cheap: a year's worth of Vit D3 drops costs about $20.

Sincerely,
Dr Emily A. Kane

# The FNB Has Failed Millions

After 13 year of silence, on Nov. 30, 2010, the quasi-governmental agency the Institute of Medicine's (IOM) Food and Nutrition Board (FNB) recommended that a 3-pound premature infant take virtually the same amount of vitamin D as a 300-pound pregnant woman. While that 400 IU/day dose is close to adequate for infants, 600 IU/day in pregnant women will do nothing to help the three childhood epidemics most closely associated with gestational and early childhood vitamin D deficiencies: asthma, autoimmune disorders, and – as recently reported in the largest pediatric journal in the world – autism. Professor Bruce Hollis of the Medical University of South Carolina has shown that pregnant and lactating women need at least 5000 IU/day, not 600.

The FNB also reported that vitamin D toxicity might occur at an intake of 10,000 IU/day (250 micrograms/day), although it could produce no reproducible evidence that 10,000 IU/day has ever caused toxicity in humans and only one poorly conducted study indicating that 20,000 IU/day may cause mild elevations in serum calcium, but not clinical toxicity.

Viewed with a different measure, this FNB report recommends that an infant should take 10 micrograms/day (400 IU) and a pregnant woman 15 micrograms/day (600 IU). As a single, 30-minute dose of summer sunshine gives adults more than 10,000 IU (250 micrograms), the FNB is apparently also warning that natural vitamin D input – as occurred from the sun before the widespread use of sunscreen – is dangerous. That is, the FNB is implying that God does not know what she is doing.

Disturbingly, this FNB committee focused on bone health, just as it did 14 years ago. It ignored the thousands of studies from the last 10 years that showed higher doses of vitamin D help: heart health, brain health, breast health, prostate health, pancreatic health, muscle health, nerve health, eye health, immune health, colon health, liver health, mood health, skin health, and especially fetal health. Tens of millions of pregnant women and their breast-feeding infants are severely vitamin–D deficient, resulting in a great increase in the medieval disease rickets. The FNB report seems to reason that if so many pregnant women have low vitamin D blood levels then it must be OK because such low levels are so common. However, such circular logic simply represents the caveman existence (never exposed to the light of the sun) of most modern-day pregnant women.

Hence, if you want to optimize your vitamin D levels – not just optimize the bone effect – supplementing is crucial. But it is almost impossible to significantly raise your vitamin D levels when supplementing at only 600 IU/day (15 micrograms). Pregnant women taking 400 IU/day have the same blood levels as pregnant women not taking vitamin D; that is, 400 IU is a meaninglessly small dose for pregnant women. Even taking 2000 IU/day of vitamin D will only increase the vitamin D levels of most pregnant women by about 10 points, depending mainly on their weight. Professor Bruce Hollis has shown that 2000 IU/day does not raise vitamin D to healthy or natural levels in either pregnant or lactating women. Therefore supplementing with higher amounts – like 5000 IU/day – is crucial for the woman who wants her fetus to enjoy optimal vitamin D levels, and the future health benefits that go along with it.

For example, taking only two of the hundreds of recently published studies: Professor Urashima and colleagues in Japan gave 1200 IU/day of vitamin D3 for six months to Japanese 10-year-olds in a randomized controlled trial. They found that vitamin D dramatically reduced the incidence of influenza A as well as the episodes of asthma attacks in the treated kids, while the placebo group was not so fortunate. If Dr. Urashima had followed the newest FNB recommendations, it is unlikely that 400 IU/day treatment arm would have done much of anything and some of the treated young teenagers may have come to serious harm without the vitamin D. Likewise, a randomized controlled prevention trial of adults by Professor Joan Lappe and colleagues at Creighton University, which showed dramatic improvements in the health of internal organs, used more than twice the FNB's new adult recommendations.

Finally, the FNB committee consulted with 14 vitamin D experts and – after reading these 14 different reports – decided to suppress them. Many of these 14 consultants are either famous vitamin D researchers, like Professor Robert Heaney at Creighton or, as in the case of Professor Walter Willett at Harvard, the single best-known nutritionist in the world. So, the FNB will not tell us what Professors Heaney and Willett thought of its new report? Why not?

Today, the Vitamin D Council directed our attorney to file a federal Freedom of Information (FOI) request to the IOM's FNB for the release of these 14 reports.

Most of my friends, hundreds of patients, and thousands of readers of the Vitamin D Council newsletter (not to mention myself) have been taking 5000 IU/day for up to eight years. Not only have they reported no significant side effects, indeed, they have reported greatly improved health in multiple organ systems. My advice, especially for pregnant women: continue taking 5000 IU/day until your 25(OH)D is between 50 and 80 ng/mL (the vitamin D blood levels obtained by humans who live and work in the sun and the midpoint of the current reference ranges at all American laboratories). Gestational vitamin D deficiency is associated not only with rickets, but a significantly increased risk of neonatal pneumonia, a doubled risk for preeclampsia, a tripled risk for gestational diabetes, and a quadrupled risk for primary cesarean section.

The FNB has failed millions of pregnant women whose as-yet unborn babies will pay the price. Let us hope that the FNB will comply with the spirit of "transparency" by quickly responding to our Freedom of Information requests.

John Cannell, MD
Vitamin D Council
1241 Johnson Avenue, #134
San Luis Obispo, California 93401

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February 7, 2011

Sharon L. Norton, RN
Suicide Prevention Council Member
PMHNP Graduate Student
61025 Ohlson Mountain Road
Homer, AK 99603

Representative Paul Seaton
345 W. Sterling Hwy. Suite 102B
Homer, Alaska 99603

RE: Preventative Health - Vitamin D Resolution

I appreciate your efforts and agree that research links Vitamin D deficiency and insufficiency to depression, bone disorders, cancer, immune disease, diabetes, and coronary disease, at the very least. Therefore, I support your resolution.

Perhaps, an addendum to your resolution might address a list of micronutrients including Vitamins and Minerals be drawn annually by all primary healthcare providers and with all initial mental health assessments. I would suggest expanding, at the very least, to include Niacin, B12, folate (folic acid), thiamine, Vitamin D and trace minerals including iron, magnesium, and selenium. Scientific evidence links a variety of psychiatric symptoms with deficiency and insufficiency as components to psychiatric syndromes.

I receive healthcare through Indian Health Services and have had few of these labs drawn to my knowledge as late as age 60. My Father is 83, a diabetic, with hypertension, hyperlipidemia, skin cancer, and situational depression who receives services through the VA. The labs listed below were not drawn, any time recently that I am aware of, *until* I made a request of a local practitioner October of 2010. Which found the labs to be: Vitamin D 25 OH Total level of 21, optimum levels range from 30-100 with <20 deficient and 20-30 insufficient, B12 was 127, low was indicated at 180 and high 914, and his Iron was 23, low indicated at 45 and high 182. I plan to request a recheck at his next VA visit this month. His diet has improved and he has been taking supplements I would like to evaluate any improvement or lack thereof. Additionally, I am awaiting my own lab results as we speak.

A large majority of Alaska's citizens would not know to request these labs. It is, therefore, up to the State of Alaska to protect the physical & mental

health and wellbeing of Alaska's people, of all ages. We seek as healthcare professionals and legislative representative's to address the prevention of medical and mental illness; not only personal and family short and long term impact, but as our obligation to address and curb the economic impact on Alaska as a whole.

Additionally, I would support, as an indication of need, an Alaskan research study to evaluate levels of vitamins and minerals, using a very large sample group of both rural and urban Alaskans (not less than 20,000), with a variety of ages, listing medical and psychiatric diagnoses, and comparing regions, those consuming the traditional Native diet compared to Western diet, and those taking supplements with those not. *Including,* those individuals of all ages having threatened and attempted suicide. In order to discover whether levels may or may not suggest a relationship to depression and other psychiatric symptoms potentially contributing to suicide. With a follow-up study evaluating diet changes and vitamin and mineral supplements provided over time.

I appreciate your addressing the preventative health of Alaskans.

With Kindest Regards,

Sharon Norton

Sharon Norton, RN

[Reference links attached.]

References links that support my response [sorry I did not have time to format them correctly].

Page 844 http://books.google.com/books?id=u-ohbTtxCeYC&pg=PA844&lpg=PA844&dq=vitamin+deficiency+and+depression+scientific+evidence&source=bl&ots=9fBDwji4xU&sig=Q8Su0TNEtpPOa3s6WUahWQmyj0Q&hl=en&ei=vFFOTYbeA42isQPL6onCg&sa=X&oi=book_result&ct=result&resnum=10&ved=0CGAQ6AEwCQ#v=onepage&q=vitamin%20deficiency%20and%20depression%20scientific%20evidence&f=true

Mayoclinic http://www.mayoclinic.com/health/vitamin-d/NS_patient-vitamind/DSECTION=evidence suggests among many other diagnoses that:
**Seasonal affective disorder (SAD) is a form of depression that occurs during the winter months, possibly due to reduced exposure to sunlight. In one study, vitamin D was found to be better than light therapy in the treatment of SAD. Further studies are necessary to confirm these findings.**

**NYU Medical Center** http://www.med.nyu.edu/content?ChunkIID=21566 Other micronutrients are also commonly deficient in elderly populations. A small study among nursing home residents found that low levels of the mineral selenium was associated with depression. Moreover, 8 weeks of mineral supplementation tended to improve the mood of the most seriously depressed patients with low selenium levels.

Science Direct: http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WN2-4C0D0FP-YT&_user=10&_coverDate=02/28/1991&_rdoc=1&_fmt=high&_orig=search&_origin=search&_sort=d&_docanchor=&view=c&_searchStrId=1633643654&

Lance Armstrong Foundation: http://www.livestrong.com/article/309794-what-vitamin-mineral-deficiencies-cause-anxiety/

**National Institute of Health: Vitamin D in fibromyalgia depression and anxiety**
http://www.ncbi.nlm.nih.gov/pubmed/16850115

National Institute of Health: Vitamin D in over weight and obese:
http://www.ncbi.nlm.nih.gov/pubmed/18793245

National Institute of Health: Vitamin D and Women with Depression in the Winter
http://www.ncbi.nlm.nih.gov/pubmed/19616172

National Institute of Health: Vitamin D and Depression in Japanese
http://www.ncbi.nlm.nih.gov/pubmed/19690578

National Institute of Health: Vitamin D and Depression in Young Adults
http://www.ncbi.nlm.nih.gov/pubmed/21067618

**Crystal Rogers**

**From:** Rep. Paul Seaton
**Sent:** Tuesday, February 15, 2011 9:19 AM
**To:** Crystal Rogers
**Subject:** FW: thanks for HCR 5

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**From:** Joy Lyon [mailto:jlyon@aeyc-sea.org]
**Sent:** Monday, February 14, 2011 6:21 PM
**To:** Rep. Paul Seaton
**Subject:** thanks for HCR 5

Dear Representative Seaton,

I just read the resolution on promotion of Vitamin D in Alaska, and I would like to personally thank you for your leadership in this area.
I told a group of pediatricians at a meeting today, and we are all so pleased that this important health issue is getting attention thanks to your resolution.
This is an example of a low or no-cost health care solution.

Thanks again, and if testimony on this is needed please let me know and I will be happy to help get the word out.

**Joy Lyon, M.A.**
Executive Director
AEYC Southeast Alaska
Main 907.789-1235 or 888-785-1235
Fax 907.789-1238
jlyon@aeyc-sea.org
www.aeyc-sea.org

www.threadalaska.org
connecting early care and education to Alaska

**Crystal Rogers**

**From:** Rep. Paul Seaton
**Sent:** Tuesday, April 05, 2011 8:46 AM
**To:** Crystal Rogers
**Subject:** FW: HCR 5 - Vitamin D

**From:** Kirstie Shirey [mailto:kirstie_shirey@yahoo.com]
**Sent:** Friday, April 01, 2011 9:47 PM
**To:** Rep. Paul Seaton
**Subject:** HCR 5 - Vitamin D

To Paul Seaton,

Hello,
My name is Kirstie Shirey. I'm twenty years old and am in college to become a early childhood teacher. I'm writing this e-mail for both myself, and class. I was given a list of things to choose from, this was one that was easy and something I believe in. I am low in Vitamin D, and my father is also is low.

Vitamin D, something that everyone needs. I think that a lot of people don't know how little they are getting. Even those who go outside everyday and are active don't get a lot of sun. It's to cold, with clouds, snow, and bundled up with little skin showing for the little sun Alaska gets in the winter.

support bill HCR 5.

Thank you for taking the time to read my little e-mail.

Sinisterly
Kirstie Shirey

March 9, 2011

The Honorable Wes Keller
Chair, House Health & Social Services Committee
Alaska Capitol, Room 427
Juneau, Alaska 99801

Dear Representative Keller,

On behalf of the more than 12,000 U.S. members of the American Academy of Dermatology Association (AADA), I am writing to point out several inaccuracies contained in HCR 5 regarding vitamin D. HCR 5 misrepresents several findings of the cited scientific literature, and as such endangers the health of Alaska's citizens by overemphasizing the role of vitamin D in disease prevention.

The AADA would not want HCR 5 to be used as grounds to legitimize use of indoor tanning beds as a means for increasing vitamin D levels. In 2010, the Institute of Medicine (IOM) released an exhaustive review of scientific literature and revised its previous recommendations on vitamin D intake. Of particular note, the IOM stated that individuals should not increase their exposure to UV radiation in order to increase their vitamin D levels, because of the risk of developing skin cancer and melanoma.

The AADA would like to point out specific omissions (**noted in bold**) in the cited data in HCR 5, which erroneously make the case for substantial vitamin D supplementation:

- A 2007 article published in the American Journal of Clinical Nutrition reported that a study that compared cancer rates of a group ***of post menopausal women*** taking 1100 IU of vitamin D supplements in combination with calcium to cancer rates of a group taking a placebo found the risk of developing any cancer after four years was 60 percent lower in the group taking vitamin D supplements *(page 1, line 16 – page 2, lines 1-4);*
- A 2007 article published in the American Journal of Preventative Medicine reported that a study found blood serum levels of vitamin D of at least 33 ng/ml ***to be associated with a 50 percent lower risk of colorectal cancer incidence*** compared with blood serum levels of vitamin D of less than 12 ng/ml *(page 2, lines 9-12)*;
- A 2001 study published in the Lancet found that children in Finland who received 2,000 IU a day of vitamin D for the first year of life 78 percent reduced risk of type 1 diabetes ***over the ensuing 31 years*** compared to children receiving 400 IU a day of vitamin D *(page 2, lines 23-26)*;
- The Centers for Disease Control and Prevention report that influenza vaccine effectiveness varies greatly ***based on the age and immunocompetence of the vaccine recipient and the degree of similarity between the viruses in the vaccine and those in circulation*** *(page 3, line 22-23)*;

American Academy of Dermatology Association
Excellence in Dermatology™

1445 New York, Ave., NW,
Suite 800
Washington, DC 20005-2134

Main: 202.842.3555
Fax: 202.842.4355
Web site: www.aad.org

- A 2010 article published in the American Journal of Clinical Nutrition reported that a study of a group of Japanese school children who received ***1200 IU*** of vitamin D ***had a decreased incidence of influenza compared to children receiving placebo. The reduction was more prominent in specific subgroups of children who had not been taking other vitamin D supplements and who started nursery school after age 3*** *(page 3, lines 27-30)**; and***
- A 2010 article in The Lancet reported that the risk of multiple sclerosis increases with latitude ***~~and with low blood serum levels of vitamin D~~*** *(page 4, lines 3-4).*

The key information missing from the above whereas clauses misrepresent the scientific data regarding vitamin D health benefits by neglecting to include critical information on the populations involved in the studies and the intent of the research.

While there are epidemiologic studies that show a statistical relationship between lower vitamin D levels and a higher incidence of some of these diseases, there are also multiple studies that have suggested an inverse association between vitamin D intake and cancer. There is some evidence that too much vitamin D may be harmful. Further, well-designed prospective studies of vitamin D intake and its association to diseases are needed to better assess this ambiguous association.

The AADA recommends vitamin D should be obtained through a healthy diet which includes drinking milk, eating foods which are good sources of vitamin D, and taking vitamin supplements. Intentional exposure to ultraviolet light from indoor tanning beds or the outdoor sun to produce optimum levels of vitamin D is not recommended, as ultraviolet (UV) radiation exposure is associated with increased risk of skin cancer and melanoma.

> **FURTHER RESOLVED** that the Alaska State Legislature urges the Department of Health and Social Services to provide vitamin D supplements to the elderly to prevent bone loss, falls, fractures, and other age-related health problems;

Studies have demonstrated that calcium and vitamin D are two essential nutrients, long known for their joint role in bone health. The AADA supports use of vitamin D supplements taken orally (according to the Institute of Medicine recommended dosage) along with calcium for the elderly to prevent bone loss, falls, and fractures. It is critical for overall health, that individuals follow the recommendations of their physician and abide by the guidelines of the Institute of Medicine as there is emerging evidence that too much of these nutrients may be harmful.

> **FURTHER RESOLVED** that the Alaska State Legislature urges the Department of Health and Social Services to investigate substituting vitamin D supplementation for influenza vaccination as a less costly method for preventing influenza;

Studies have not conclusively demonstrated than vitamin D supplements will prevent infectious diseases; yet, numerous studies have conclusively demonstrated that vaccines are very effective in preventing infectious diseases. Vigorous vaccination practices and healthy living conditions will lower the rates of preventable infectious diseases and also reduce morbidity and mortality from these preventable diseases.

HCR 5 would endanger patients by replacing a proven method of disease prevention with a vitamin supplementation program which has not been tested to protect the general population from infectious disease, such as influenza.

> **FURTHER RESOLVED** that the Alaska State Legislature urges the Department of Health and Social Services to provide vitamin D supplements to pregnant women and infants to prevent pregnancy complications, preterm births, type 1 diabetes, and rickets.

The AADA strongly cautions the Alaska Legislature to further review the scientific literature regarding vitamin D, and disease prevention before moving forward with a supplementation program aimed at specific, vulnerable segments of the population. Moreover, it is critical that the public be appropriately educated about vitamin D and be encouraged to consult their physician before taking any vitamin supplements. Lastly, Alaskans should be educated about the dangers of ultraviolet radiation (UV). As stated previously, UV radiation from the sun and indoor tanning beds is associated with a significant increased risk in the development of skin cancer and melanoma. The public should be educated about proper sun protection and urged to avoid UV exposure from indoor tanning devices.

Thank you for the opportunity to provide written comments on HCR 5. For further information, please contact Kathryn Chandra, Assistant Director of State Policy for the AADA, at kchandra@aad.org or (202) 712-2615.

Sincerely,

Ronald L. Moy, MD, FAAD
President, American Academy of Dermatology Association
RLM/kgc

CC: Alaska House Health & Social Services Committee Members
Jim Jordan, Executive Director, Alaska State Medical Association

PERSPECTIVE

JBMR

# Why the IOM Recommendations for Vitamin D Are Deficient

Robert P Heaney[1] and Michael F Holick[2]

[1]Creighton University , Omaha, NE, USA
[2]Department of Medicine, Division of Endocrinology, Boston University Medical Center, Boston, MA, USA

**ABSTRACT**

The IOM recommendations for vitamin D fail in a major way on logic, on science, and on effective public health guidance. Moreover, by failing to use a physiological referent, the IOM approach constitutes precisely the wrong model for development of nutritional policy.


**KEY WORDS:** VITAMIN D; NUTRITIONAL POLICY; EVOLUTIONARY PHYSIOLOGY

## Introduction

In the past two years, vitamin D supplement sales to consumers have increased by more than 100% per year.[1] Now, following publication of the report[2] on Dietary Reference Intakes (DRIs) for calcium and vitamin D by the Institute of Medicine (IOM), many physicians report that they are decreasing their vitamin D recommendations to patients. This change was explicitly proposed by members of the IOM panel in their various media statements. While a small fraction of consumers may well have all the vitamin D they need, on balance, we consider a general downward trend to be harmful to the health of the public.

Both the authors of this Perspective served as members of the panel that drafted the 1997 report of the IOM on the DRIs for calcium and vitamin D. That report was the first issued by the IOM under the then-new evidence-based guidelines for evaluating studies and making recommendations. We are thus familiar with the process and, most important, with vitamin D itself. On the basis of this experience, we respectfully dissent from many of the findings and recommendations in the current report, and we set forth here a small fraction of the reasons for that dissent.

The IOM report (and its presentation to the media) stressed that its recommendations for vitamin D were based primarily on the intake (and serum 25-hydroxyvitamin D concentration) needed to ensure skeletal health and that, in the panel's judgment, there was insufficient evidence to make any recommendations with respect to nonskeletal benefits, if any. Second, the report concluded that a serum level for 25-hydroxyvitamin D [25(OH)D] of 20 ng/mL was sufficient to ensure bone health. And third, the panel concluded that since the bulk of the American public had 25(OH)D values that were above 20 ng/mL, most individuals were getting all the vitamin D they needed and had no reason for further supplementation. These conclusions fail on three grounds: logic, science, and guidance.

First, logic. Since the panel, in its judgment, concluded that it did not know whether there might be nonskeletal benefits (or at what blood level they could be ensured), then it is patently incorrect to say that they know that people are getting enough. The most the panel could have said logically was, "Here's what you need for bone; most people get that much; we do not know whether more would confer possible nonskeletal benefits." That, at least, would have been an honest communication of the state of the issue as the panel apparently understands it. However, to state publicly that the general public does not need more goes well beyond what the panel admits it knows.

Second, science. The statement that skeletal health can be ensured at serum 25(OH)D levels of 20 ng/mL is simply incorrect. Without going into an exhaustive recital of all the evidence pointing to a skeletal need for higher levels, we cite here three illustrative observations that, in our collective judgment, indicate that instead of 20 ng/mL, a serum level of 30 ng/mL is closer to the bottom end of the acceptable range for skeletal health. First, there is the large randomized, controlled trial in the United Kingdom that raised serum 25(OH)D level from 21 to 29 ng/mL and produced a 33% reduction in all major osteoporotic fractures combined.[3] The fact that other trials, with less good compliance, failed to reproduce that effect does not negate the evidence of a well-conducted trial. Second, there are the many meta-analyses of Bischoff-Ferrari and colleagues[4,5] demonstrating that, taken overall, fracture reduction with vitamin D does not occur reproducibly below serum 25(OH)D

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Address correspondence to: Robert P Heaney, MD, Creighton University, 601 North 30th Street, Suite 4841, Omaha, NE 68131, USA. E-mail: rheaney@creighton.edu
For further discussion on this topic, please see Reid and Avenell (J Bone Miner Res. 2011;452–454. DOI: 10.1002/jbmr.327).

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DOI: 10.1002/jbmr.328

levels of 30 ng/mL and for some fractures even 40 ng/mL. Finally, there is the demonstration, in a large German autopsy series (strangely misinterpreted by the panel), that osteoid seam width—the histologic hallmark of vitamin D deficiency—does not reach fully normal values until serum 25(OH)D levels are above 30 ng/mL.[6] [N.B.: Of 33 patients with 25(OH)D values between 20 and 30 ng/mL, more than half (18) had elevated osteoid volume. A Recommended Daily Allowance (RDA), by definition, meets the need of 97.5% of the population.] In a closely related finding, investigators from South Australia[7] showed seasonal variation in osteoid seam width and mineral appositional rate, reflecting variations in serum 25(OH)D precisely within the 20 to 30 ng/mL range, that is, above the IOM panel's "adequate" level.

Additionally, there is an apparent inconsistency between the recommended intake (600 IU/day for all individuals up through age 70) and the bottom end of the acceptable 25(OH)D serum concentration range (let alone higher values). As virtually universal experience with vitamin D supplementation demonstrates, 600 IU/day, if the body's sole input of vitamin D, would not be enough to produce a value of even 10 ng/mL, let alone 20 ng/mL or above. There is a generally recognized "rule of thumb" to the effect that each additional 100 IU of vitamin D per day raises serum 25(OH)D concentration by approximately 1 ng/mL. That is, in fact, a "rounding up" for convenience of calculation. Several studies indicate that the response increment is closer to 0.7 ng/mL/100 IU.[8,9] Either way, 600 IU/day will not suffice without appreciable solar and dietary input. Furthermore, as is also widely recognized, 600 IU/day produces barely perceptible changes in individuals who are overweight or obese (now better than 50% of the US adult population). Hence the increase from the 1997 DRIs, while welcome, and certainly in the right direction, is simply inconsistent with current professional experience. It not only is inadequate, by itself, to meet even the panel's recommended serum levels, but this internal inconsistency detracts from the credibility of the whole report inasmuch as it flies in the face of the everyday experience of clinicians who recommend supplements to their patients and measure the resulting responses.

Finally, guidance. At already noted, the panel indicated that it was uncertain about extraskeletal benefits—benefits that might accrue at intakes above the new intake recommendations. At the same time, the panel raised the upper-level intake "TUIL" to 4000 IU/day. (The report acknowledges that intakes up to 10,000 IU/day are probably safe for everyone and applied an uncertainty factor[10] to that 10,000-IU figure to generate the 4000-IU TUIL. It is important to stress that the TUIL is not a limit but instead constitutes an assurance of safety for such an intake.) One should have thought that even a very simplistic game-theory approach would have led to a guidance statement such as the following: "We do not know whether taking more vitamin D than we are currently recommending will help you, but it could, and we can assure you that supplemental intakes up to at least 4000 IU/day are safe." Such a statement, couched, perhaps, in less straightforward language, nevertheless would provide guidance that both the public and governmental agencies could find useful. Instead, we now have only a confused public.

Beyond these errors and inconsistencies, though, serious as they are, lies a much deeper flaw in the approach taken by the panel, exemplified by a quote from one of the panel members to the *New York Times* at the time of release of the report.[11] The statement was simply that the "onus" (ie, burden of proof) fell on anyone who claimed benefits for intakes higher than the panel's current recommendations. This is an approach that is correct for drugs, which are foreign chemicals and which do carry an appropriately heavy requirement for proof. For drugs, the position of privilege is given to the placebo. And in the current IOM report, the privilege is given to a serum 25(OH)D level that is effectively the status quo. We judge that this is exactly backward for nutrients. The privilege instead must be given to the intake that prevailed during the evolution of human physiology, the intake to which, presumably, that physiology is fine-tuned. So far as can be judged from numerous studies documenting the magnitude of the effect of sun exposure,[12,13] the primitive intake would have been at least 4000 IU/day and probably two to three times that level, with corresponding serum 25(OH)D levels ranging from 40 to 80 ng/mL. The fact that primitive levels would have been higher than current IOM recommendations does not, of course, prove their necessity today. But such intakes should be given the presumption of correctness, and the burden of proof must be placed on those who propose that lower intakes (and lower serum levels) are without risk of preventable dysfunction or disease. The IOM, in its report, has utterly failed to recognize or meet that standard.

Finally, we commend the IOM panel for their concern about safety, certainly an appropriate posture for a body crafting public policy. However, the standards adopted by the panel for taking into evidence papers indicating possible risk were, we note, far lower than those the panel required to indicate benefit. Additionally, many of the purported risks were, on their face, implausible and inconsistent with the experience of population subgroups that routinely have serum levels in the range mentioned by the panel as possibly risky (eg, approximately 50 ng/mL). We note that one of the widely accepted Hill[14] criteria for acceptance of observational data is precisely biologic plausibility. Furthermore, we consider it highly implausible that serum levels such as prevailed during hominid evolution could carry more risk than benefit for the populations concerned. Had that been the case, one should have expected that natural selection would have eliminated those prone to such risks.

In this Perspective, we have deliberately avoided a mind-numbing laundry list of the vast number of factual inaccuracies and misinterpretations in the report. We are informed that there is a request, through the Freedom of Information Act, to obtain the external review comments submitted to the IOM in response to a prepublication draft. When those materials become available, those interested can review the many problems with the IOM report in detail. For now, our recommendation to the American public is that the IOM report should be taken with a grain of salt (another nutrient the IOM finds risky).

## Disclosures

Both authors state that they have no conflicts of interest.

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