Available online at www.sciencedirect.com
ScienceDirect
Nutrition Research 31 (2011) 48 – 54
Nutrition Research
www.nrjournal.com

Prevalence and correlates of vitamin D deficiency in US adults

Kimberly Y.Z. Forrest*, Wendy L. Stuhldreher
Department of Public Health & Social Work, Slippery Rock University of Pennsylvania, Slippery Rock, PA 16057
Received 8 September 2010; revised 1 December 2010; accepted 7 December 2010

Abstract

Mounting evidence suggests that vitamin D deficiency could be linked to several chronic diseases, including cardiovascular disease and cancer. The purpose of this study was to examine the prevalence of vitamin D deficiency and its correlates to test the hypothesis that vitamin D deficiency was common in the US population, especially in certain minority groups. The National Health and Nutrition Examination Survey 2005 to 2006 data were analyzed for vitamin D levels in adult participants (N = 4495). Vitamin D deficiency was defined as a serum 25-hydroxyvitamin D concentrations ≤20 ng/mL (50 nmol/L). The overall prevalence rate of vitamin D deficiency was 41.6%, with the highest rate seen in blacks (82.1%), followed by Hispanics (69.2%). Vitamin D deficiency was significantly more common among those who had no college education, were obese, with a poor health status, hypertension, low high-density lipoprotein cholesterol level, or not consuming milk daily (all $P < .001$). Multivariate analyses showed that being from a non-white race, not college educated, obese, having low high-density lipoprotein cholesterol, poor health, and no daily milk consumption were all significantly, independently associated with vitamin D deficiency (all $P < .05$). In summary, vitamin D deficiency was common in the US population, especially among blacks and Hispanics. Given that vitamin D deficiency is linked to some of the important risk factors of leading causes of death in the United States, it is important that health professionals are aware of this connection and offer dietary and other intervention strategies to correct vitamin D deficiency, especially in minority groups.

Keywords: Vitamin D; Adults; Prevalence rate; Risk factors; Minority groups; Obesity; Cholesterol
Abbreviations: CI, confidence interval; NHANES, the National Health and Nutrition Examination Survey; BMI, body mass index; HDL, high-density lipoprotein.

1. Introduction

Vitamin D has been traditionally considered as important in skeletal health. However, during the past decade, numerous research findings have revealed that vitamin D produces beneficial effects on extraskeletal tissues as well [1-3]. Some evidence suggests that vitamin D helps to regulate cell growth and prevent cancer progression [4-6]. Epidemiological studies have reported that higher vitamin D levels were associated with reduced cancer incidence and decreased cancer-related mortality [7-9]. Vitamin D was found to be involved in controlling the production of renin, one of the most important hormones for regulating blood pressure [10]. Thus, Vitamin D deficiency might contribute to development and progression of hypertension and cardiovascular disease [11-14]. Furthermore, vitamin D deficiency has been linked to the development of type 1 diabetes [1-3,15], multiple sclerosis [16,17], rheumatoid arthritis [18], and other autoimmune conditions [1-3,19,20].

Vitamin D can be synthesized by the skin through exposure to ultraviolet light of wavelength 290 to 315 nm that stimulates the conversion of 7-dehydrocholesterol to

* Corresponding author. Tel.: +1 724 738 2258; fax: +1 724 738 4559.
E-mail address: kimberly.forrest@sru.edu (K.Y.Z. Forrest).

0271-5317/$ – see front matter © 2011 Elsevier Inc. All rights reserved.
doi:10.1016/j.nutres.2010.12.001

previtamin D [21]. The other source of vitamin D is from the diet. Vitamin D from food undergoes hydroxylation in the liver to 25-hydroxyvitamin D—the major circulating form—and then in the kidney to 1,25-dihydroxyvitamin D, which optimizes calcium and phosphate absorption from the intestine, as well as having direct effects on bone cells [1,3]. The recommended adequate intake of vitamin D used to be 200 IU/d for all children and adults 50 years or younger, 400 IU/d for people aged 51 to 70 years, and 600 IU/d for those older than 70 years [22]. However, due to its beneficial effects, the amounts of vitamin D needed for optimal health are probably higher than previously thought [2,23]. On November 30, 2010, the Institute of Medicine released updated recommendations regarding vitamin D intake: 600 IU/d for people aged 1 to 70 years and 800 IU for people aged 71 and older [24].

Although vitamin D deficiency is commonly defined as a 25-hydroxyvitamin D level ≤20 ng/mL (50 nmol/L) [20,25], published studies have used different definitions for vitamin D deficiency. By different cutoff points, vitamin D deficiency was found to be common in certain subpopulations, including elder adults (41% using 25-hydroxyvitamin D level ≤20 ng/mL) [20], African Americans (61% using 25-hydroxyvitamin D level ≤15 ng/mL) [26], and women with osteoporosis (64% using 25-hydroxyvitamin D level ≤30 ng/mL [27]. Given that low vitamin D levels are linked to all major health problems in populations, such as cardiovascular disease, cancers, and diabetes, it is of importance to identify how prevalent this condition is and what factors are associated with this condition in the US population. This study analyzed the data from the 2005 to 2006 National Health and Nutrition Examination Survey (NHANES) to describe the epidemiology of vitamin D deficiency in US adults, including prevalence patterns of vitamin D deficiency and its correlates. Based on the findings from the literature, it was hypothesized that vitamin D deficiency was common in the US population, especially certain minority groups. The objective of this study was to examine and compare the prevalence rate of vitamin D deficiency by age, race, other demographic factors, as well as by certain health conditions, using serum 25-hydroxyvitamin D level, the best indicator of vitamin D status.

2. Methods and materials

2.1. Study population

The NHANES is an ongoing program conducted by the National Center for Health Statistics to assess the health and nutritional status in the noninstitutionalized US population and track changes over time [28]. The survey combines interviews and physical examinations. The interview includes demographic, socioeconomic, dietary, and health-related questions. The examination component consists of medical and physiologic measurements, as well as laboratory tests.

The NHANES uses a stratified multistage probability sampling design and constructs sample weights to produce nationally representative data. The NHANES data are available in the public domain. The 2005 to 2006 NHANES oversampled certain subgroups of the US population, including low-income persons, older adults aged 60 years or older, African Americans, and Mexican Americans, to provide a more in-depth snapshot of these population groups. A total of 12 862 individuals were sampled into the 2005 to 2006 NHANES. Among the sampled individuals, 10 348 (80.5%) participated in the interview, 9950 (77.4%) were involved in the examination, and 8306 (65%) provided valid data on vitamin D measurement. The current analysis only included individuals who provided vitamin D data and were aged 20 years or older (N = 4495). The NHANES was approved by its institutional review board, and analyzing the public domain data from NHANES does not require additional institutional review board approval.

2.2. Measurements

2.2.1. Demographic variables

Age was recoded into 5-year age groups. Race was classified as white, black, Hispanic, and other races combined. Education level was dichotomized as yes or no for any postsecondary or college education.

2.2.2. Health-related variables

Smoking status was classified as never, former, and current. Overweight was defined by body mass index (BMI) (kg/m^2) between 25.0 and 29.9, and obesity was defined by BMI of ≥30.0. Total cholesterol is measured enzymatically in serum in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol [29]. A total cholesterol level that is ≥200 mg/dL was considered as elevated. High-density lipoprotein (HDL) was measured directly in serum [29], and a level less than 40 mg/dL was classified as low. Hypertension was assessed by self-reporting in answer to the question “has a physician ever told you that you have high blood pressure.” Milk consumption was coded as daily use yes or no. Health status was self-reported as good/excellent and poor/fair.

2.2.3. Vitamin D measures

Serum 25-hydroxyvitamin D concentration was measured at the National Center for Environmental Health, Center for Disease Control, using a radioimmunoassay kit (DiaSorin, Stillwater, MN) [29], and a level that was ≤20 ng/mL (50 nmol/L) was defined as vitamin D deficiency.

2.3. Statistical analyses

Data analysis was performed using SAS Release 8.2 (SAS Institute Inc, Cary, NC) and SUDAAN Release 9.0.1 (Research Triangle Institute, Research Triangle Park, NC). SAS was used for data management to sort data, recode variables, and run frequencies for examining the data.

SUDAAN was designed to analyze survey data because this statistical software can account for the complex multistage sample design, such as NHANES. The PROC CROSSTAB procedure in SUDAAN was used for calculating prevalence rates of vitamin D deficiency presented as percentages (%) and 95% confidence intervals (CI). A prevalence rate was calculated as the percentage of the number of people who were classified as vitamin D deficiency by the total number of people in the corresponding category. Serum vitamin D concentrations were reported as mean ± SD. χ^2 Test was used to assess bivariate associations between vitamin D deficiency and other variables. Multiple logistic regression analyses (the PROC LOGISTIC procedure in SUDAAN) were used to examine the relationship between vitamin D deficiency and a set of explanatory variables to identify independent correlates of vitamin D deficiency and to calculate adjusted odds ratios and 95% CI. All variables with a significant bivariate association with vitamin D deficiency ($P < .05$) were examined in a multivariate logistic regression model. Dummy variables were created for each race to assess the individual minority group's risk for vitamin D deficiency. Taylor series linearization methods were used for variance estimation. All analyses were based on weighted data to adjust for nonresponse and to make the data representative of the US population. These adjustments were made by applying the examined sample weight variable provided by the NHANES. Weighted prevalence rate estimates were reported.

3. Results

A total of 4495 individuals aged 20 years or older were included in this study. About 17% of the study population were 65 years or older. Table 1 showed the characteristics of the study population. Fifty-two percent of the study participants were women. The race distribution was 72.5%, 11.1%, 11.3%, and 5.1% for white, black, Hispanic, and other races, respectively. More than 57% of study participants received at least some postsecondary education. Most individuals (83.8%) reported to have a good or excellent health status. Twenty-four percent of the study participants were current smokers, and 25% were former smokers. About one third of the study participants were overweight, and another one third were obese. Thirty percent of individuals had hypertension, 45.4% had a high total cholesterol level, and 15.5% had a low HDL cholesterol level. Less than half (43.3%) of the study population reported consuming milk products daily.

The mean ± SD of vitamin D levels were 19.9 ± 8.5 ng/mL in the study population and were 20.1 ± 7.9 ng/mL in men and 19.8 ± 9.0 ng/mL in women. The overall prevalence rate of vitamin D deficiency (≤20 ng/mL) was 41.6% (95% CI, 36.6%-46.8%). Examination of the prevalence rate of vitamin D deficiency by age showed insignificant variations between age groups (Fig. 1). The age groups of 55 to 59 and

Table 1
Characteristics of study population and prevalence rate (%) of vitamin D deficiency

Characteristics	n[a] (%)	Prevalence rate (%) (95% CI)
Age (y)		
<65	3432 (82.8)	41.7 (36.0-47.6)
≥65	1063 (17.2)	41.1(37.4-44.6)
Sex		
Male	2158 (48.0)	41.1 (36.6-45.8)
Female	2337 (52.0)	42.0 (36.2-48.1)
Race		
White	2271 (72.5)	30.9 (26.2-36.2)**
Black	1002 (11.1)	82.1 (76.5-86.5)
Hispanic	1049 (11.3)	62.9 (53.2-71.7)
Other	173 (5.1)	57.6 (46.9-67.4)
Any college education		
Yes	2182 (57.6)	36.7 (32.1-41.5)**
No	2308 (42.4)	48.2 (42.2-54.2)
Health status		
Good/excellent	3289 (83.8)	37.4 (32.4-42.7)**
Poor/fair	897 (16.2)	59.5 (51.3-67.2)
Smoking status		
Never	2365 (51.0)	42.4 (37.0-48.0)
Former	1147 (25.0)	37.8 (31.6-44.5)
Current	983 (24.0)	43.7 (37.9-49.7)
Body weight		
Normal	1413 (33.7)	33.0 (28.4-37.9)**
Overweight	1519 (32.5)	37.7 (32.1-43.7)
Obese	1563 (33.9)	53.8 (46.7-60.8)
Hypertension		
Yes	1482 (30.0)	46.3 (41.0-51.8)**
No	3054 (70.0)	39.5 (34.3-44.9)
High total cholesterol		
Yes	2043 (45.4)	40.3 (34.6-46.2)
No	2452 (54.6)	42.7 (37.5-48.0)
Low HDL cholesterol		
Yes	683 (15.8)	49.9 (43.1-56.7)*
No	3812 (84.2)	40.0 (34.8-45.5)
Consume milk products daily		
Yes	1967 (43.3)	33.2 (28.5-38.3)**
No	2528 (56.7)	48.0 (42.4-53.6)

[a] n indicates sample size.
* $P < .01$.
** $P < .001$ for difference between groups.

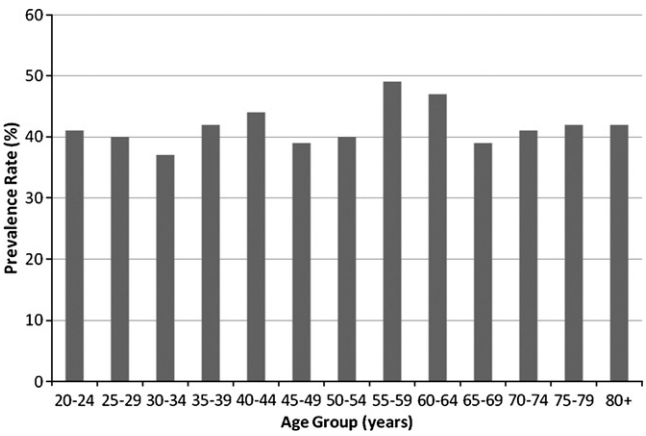

Fig 1. Prevalence rate (%) vitamin D deficiency by age.

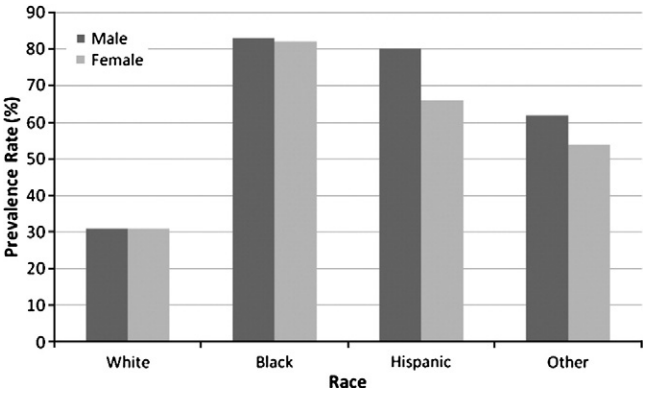

Fig 2. Prevalence rate (%) vitamin D deficiency by race and gender.

60 to 64 years had the highest prevalence rate of vitamin D deficiency (49.0% and 47.0%, respectively). The age patterns of vitamin D deficiency between men and women were not significantly different. Fig. 2 shows the prevalence rate of vitamin D deficiency by race and sex. Black adults had the highest prevalence rate of vitamin D deficiency (82.1%; 95% CI, 76.5%-86.5%), followed by Hispanic adults (62.9%; 95% CI, 53.2%-71.7%). Among Hispanic and other races, men were significantly more likely to have vitamin D deficiency than women ($P < .001$). When examining by other factors (Table 1), the prevalence rate of vitamin D deficiency was significantly more common among those who had no college education, were obese, with a poor health status, hypertension, low HDL cholesterol level, or not consuming milk products daily (all $P < .001$).

A comparison between individuals who had vitamin D deficiency and those who had a normal vitamin D level revealed several significant differences (Table 2). In demographic aspects, individuals with vitamin D deficiency were significantly more likely to be black (21.9% vs 3.4%; $P < .0001$) or Hispanic (17.1% vs 7.2%; $P < .0001$), without any college education (49.2% vs 37.6%; $P < .0001$), and not in good health (23.6% vs 11.2%; $P < .0001$). For cardiovascular disease–related risk factors, individuals with vitamin D deficiency were more likely to be overweight (73.3% vs 61.4%; $P < .0001$) or obese (43.9% vs 26.8%; $P < .0001$), have hypertension (33.5% vs 27.6%; $P < .001$), and have a low HDL cholesterol level (18.9% vs 13.5%; $P < .01$). A high percentage of the individuals with vitamin D deficiency did not consume milk products daily (65.4% vs 50.5%; $P < .0001$).

Table 3 shows the results from multivariate analyses. After adjusting for all related factors, the multivariate model revealed that being black or Hispanic, no college education, not in a good health status, being obese, low HDL cholesterol, and not consuming milk products daily were all significant independent correlates of vitamin D deficiency. Among the independent correlates, being a minority was the strongest indicator for vitamin D deficiency; compared with whites, blacks had 9.6 times and Hispanics had 3.2 times increased risk for vitamin D deficiency. Obese individuals showed nearly double the risk for vitamin D deficiency than nonobese individuals.

Table 2
Univariate association between vitamin D deficiency and other variables

	Vitamin D deficiency		P [a]
	Yes (n = 2,257) % (95% CI)	No (n = 2,238) % (95% CI)	
Being female	52.5 (50.1-54.9)	51.6 (49.8-53.4)	.5584
65 years or older	17.0 (13.9-20.6)	17.4 (14.1-21.3)	.7721
Being black	21.9 (15.3-30.3)	3.4 (2.2-5.2)	<.0001
Being Hispanic	17.1 (12.8-22.5)	7.2 (5.3-9.7)	<.0001
No college education	49.2 (45.4-32.0)	37.6 (33.3-42.1)	<.0001
Poor/fair health status	23.6 (21.1-26.3)	11.2 (9.2-13.4)	<.0001
Being current smoker	25.2 (22.8-27.7)	23.1 (19.8-26.7)	.1204
Being former smoker	22.7 (20.7-24.9)	26.6 (23.7-29.8)	.0902
Being overweight	73.3 (70.2-76.2)	61.4 (57.6-65.1)	<.0001
Being obese	43.9 (40.2-47.6)	26.8 (23.4-30.6)	<.0001
Hypertension	33.5 (30.6-36.5)	27.6 (25.0-30.3)	.0009
High total cholesterol	44.0 (40.3-47.7)	46.4 (44.0-48.9)	.2291
Low HDL cholesterol	18.9 (16.4-21.7)	13.5 (11.3-15.8)	.0047
Not consume milk products daily	65.4 (61.2-69.4)	50.5 (47.1-54.0)	<.0001

[a] P values are for the difference between the 2 groups of vitamin D deficiency status.

4. Discussion

From the results of the current study, the hypothesis was accepted that vitamin D deficiency was common in US adults, especially among minority groups. Although

Table 3
Independent correlates of vitamin D deficiency

	Odds ratio	95% CI	P
Being black			
No (white)	1.0		
Yes	9.6	6.3-14.5	<.001
Being Hispanic			
No (white)	1.0		
Yes	3.2	2.1-4.9	<.001
No college education			
Yes	1.0		
No	1.3	1.1-1.5	.01
Poor/fair health status			
No	1.0		
Yes	1.8	1.4-2.3	<.001
Being obese			
No	1.0		
Yes	1.9	1.6-2.3	<.001
HDL Cholesterol <40 mg/dL			
No	1.0		
Yes	1.4	1.1-1.8	.03
Not consuming milk products daily			
No	1.0		
Yes	1.6	1.4-1.9	<.001

Note: Other covariates adjusted but not significant in the model included hypertension, health status, and education level.

different cutoff points have been used to define vitamin D deficiency, several studies have reported a high prevalence rate of vitamin D deficiency in non-Hispanic blacks [30-33]. Using the definition of serum 25-hydroxyvitamin D concentrations ≤20 ng/mL, we found that over 80% of black adults, both men and women, would be categorized as vitamin D deficient. Compared with white adults, other minorities were also at a higher risk for vitamin D deficiency, especially Hispanic men, which confirmed the results from other studies [31,34-36]. Because the skin pigment melanin absorbs sunlight [37], an important source of erethymal vitamin D, people of color are at particularly high risk for vitamin D deficiency [2,38]. The association between race and vitamin D deficiency may be related to several factors. Sun exposure is the primary determinant of vitamin D status [3] and non-whites require more sunlight exposure to obtain adequate vitamin D levels because of skin pigmentation. Another possible explanation could be because of the different dietary patterns, particularly the intake of dairy products in different population groups. Lower socioeconomic status among minority populations could also impact on food choices, for example, less likely to purchase fish that provide a good source of vitamin D. Kakarala et al [39] reported that underserved individuals, who were not medically insured and were mostly non-whites, were 3 times more likely than whites to have vitamin D deficiency and had a low-dietary vitamin D intake. Our analysis confirmed the finding that lower education level, a marker of low socioeconomic status, was associated with vitamin D deficiency.

Several important cardiovascular disease risk factors were found to be significantly associated with vitamin D deficiency in this study, including obesity and low HDL level. Obesity has been shown to be independently correlated with vitamin D deficiency in other studies [20]. Our analyses confirmed that the risk of vitamin D deficiency was almost double among obese adults compared with those who had a normal weight. Similar patterns have been reported worldwide. Research on 3100 women in northeast Scotland found that those with an average BMI of 34 produced 10% less vitamin D than those of average weight [40]. A Spanish study showed that over half of the morbidly obese patients (BMI ≥ 40) were diagnosed with vitamin D deficiency [41]. In an Italian study, BMI was also significantly correlated with 25-hydroxyvitamin D concentration after adjusting for insulin-sensitivity, HDL cholesterol, LDL cholesterol, total cholesterol, and triglycerides [42]. The relationship between vitamin D deficiency and obesity is still unknown, and the temporal relationship is not clear, for example, vitamin D deficiency causes obesity or the other way around. Excess body fat tissue could absorb and retain vitamin D, and thus, circulatory vitamin D is decreased and unavailable to the body [43]. The absence of vitamin D could create interference with the functioning of a hormone called leptin, which signals the brain when the stomach is full, therefore, stop eating [44]. In addition, overweight people may tend to spend more time indoors and receive less ultraviolet rays of the sun that spur the production of vitamin D [45].

The association between vitamin D deficiency and weight might be also because of the link between vitamin D and the metabolic syndrome, as overweight is a major component of the metabolic syndrome. Previous studies have shown an inverse relationship between vitamin D concentrations and the prevalence of the metabolic syndrome, including insulin resistance, high total cholesterol and triglyceride levels, low LDL cholesterol level, and high blood pressure [46-48]. Observational studies have suggested an association between vitamin D deficiency and the onset of type 2 diabetes, a common consequence of the metabolic syndrome [46,48]. Vitamin D has important effects on insulin action and may impact on several pathways, which may be of importance in the development of type 2 diabetes [49]. Among obese individuals, vitamin D deficiency was associated with other metabolic syndrome risk factors [41]. Our findings also supported this association that low HDL cholesterol was independently and significantly correlated with vitamin D deficiency.

The strength of this study was the large sample size and the population-based data on vitamin D. There were some limitations recognized in this study. Cross-sectional data do not permit determination of the causative nature of the association between vitamin D deficiency and its correlates. This study was based on a one-time measurement of vitamin D, and it could not show the variation of vitamin D concentration during different seasons because there is a seasonal impact on vitamin D level. There was a lack of the information on sun exposure, such as time spent outdoors and sunscreen use. Different regions have different altitudes, which can affect the strength of ultraviolet rays, and this study did not have region-specific data on vitamin D deficiency. In addition, vitamin D supplement use can influence the vitamin D level, but this factor could not be evaluated in the current study.

In conclusion, vitamin D deficiency was common in both men and women across different age groups in the US adult population, especially among minority groups. Due to the link between vitamin D deficiency and major chronic diseases and all-cause mortality [50], it might underscore the necessity to identify vitamin D deficiency as part of screening for risk factors. It may be prudent for registered dietitians and other health professionals to advise clients on ways to increase dietary vitamin D or recommend supplementation. Traditional sources of vitamin D are fatty fish and their oils and fortified milk. The expansion of vitamin D fortified food products has given consumers a wider variety of sources for vitamin D, such as juices, ready-to-eat cereal, and yogurt. Although many of these food sources are low-cost solutions for vitamin D deficiency, even supplemental vitamin D is a relatively inexpensive source [51]. Correcting vitamin D deficiency by supplementation is less challenging than asking people to change dietary patterns. Given that minority populations experience a higher prevalence of

cardiovascular disease, hypertension, and metabolic syndrome, as well as vitamin D deficiency, proactive health professionals could be instrumental in correcting vitamin D deficiency in minority groups. Using diet, supplements, or even safe sun exposure could be a public health strategy, which is simple, effective, and low cost, for risk reduction of vitamin D deficiency.

Acknowledgment

This research was supported by the College of Health, Environment and Science at Slippery Rock University of Pennsylvania.

References

[1] Holick MR. Vitamin D: importance in the prevention of cancers, type 1 diabetes, heart disease, and osteoporosis. Am J Clin Nutr 2004;79: 362-71.
[2] Holick MF. Sunlight and vitamin D for bone health and prevention of autoimmune diseases, cancers, and cardiovascular disease. Am J Clin Nutr 2004;80(Suppl):1678S-88S.
[3] Holick MF. Vitamin D deficiency. N Engl J Med 2007;357:266-81.
[4] Chen TC, Holick MF. Vitamin D and prostate cancer prevention and treatment. Trends Endocrinol Metab 2003;14:423-30.
[5] Cross HS, Bareis P, Hofer H, et al. 25-Hydroxyvitamin D_3-1α-hydroxylase and vitamin D receptor gene expression in human colonic mucosa is elevated during early cancerogenesis. Steroids 2001;66: 287-92.
[6] Tangpricha V, Flanagan JN, Whitlatch LW, et al. 25-Hydroxyvitamin D-1α-hydroxylase in normal and malignant colon tissue. Lancet 2001; 357:1673-4.
[7] Freedman DM, Looker AC, Chang SC, Graubard BI. Prospective study of serum vitamin D and cancer mortality in the United States. J Natl Cancer Inst 2007;99:1594-602.
[8] John EM, Schwartz GG, Dreon DM, Koo J. Vitamin D and breast cancer risk: the NHANES I epidemiologic follow-up study, 1971-1975 to 1992: National Health and Nutrition Examination Survey. Cancer Epidemiol Biomarkers Prev 1999;8:399-406.
[9] Zhao XY, Feldman D. The role of vitamin D in prostate cancer. Steroids 2001;66:293-300.
[10] Li YC. Vitamin D regulation of the renin-angiotensin system. J Cell Biochem 2003;88:327-31.
[11] Dobnig H, Pilz S, Scharnagl H, et al. Independent association of low serum 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D levels with all-cause and cardiovascular mortality. Arch Intern Med 2008;168: 1340-9.
[12] Rostand SG. Ultraviolet light may contribute to geographic and racial blood pressure differences. Hypertension 1997;30:150-6.
[13] Wang TJ, Pencina MJ, Booth SL, et al. Vitamin D deficiency and risk of cardiovascular disease. Circulation 2008;117:503-11.
[14] Zittermann A, Schleithoff SS, Tenderich G, Berthold HK, Körfer R, Stehle P. Low vitaminD status: a contributing factor in the pathogenesis of congestive heart failure? J Am Coll Cardiol 2003; 41:105-12.
[15] Casteels K, Waer M, Bouillon R, et al. 1,25-Dihydroxyvitamin D3 restores sensitivity to cyclophosphamide-induced apoptosis in non-obese diabetic (NOD) mice and protects against diabetes. Clin Exp Immunol 1998;112:181-7.
[16] Hernan MA, Olek MJ, Ascherio A. Geographic variation of MS incidence in two prospective studies of US women. Neurology 1999; 53:1711-8.
[17] Munger KL, Zhang SM, O'Reilly E, et al. Vitamin D intake and incidence of multiple sclerosis. Neurology 2004;62:60-5.
[18] Merlino LA, Curtis J, Mikuls TR, et al. Vitamin D intake is inversely associated with rheumatoid arthritis: results from the Iowa Women's Health Study. Arthritis Rheum 2004;50:72-7.
[19] Holick MF. Vitamin D: important for prevention of osteoporosis, cardiovascular heart disease, type 1 diabetes, autoimmune diseases, and some cancers. South Med J 2005;98:1024-7.
[20] Holick MF. High prevalence of vitamin D inadequacy and implications for health. Mayo Clin Proc 2006;81:353-73.
[21] Bouillon R, Carmeliet G, Daci E, Segaert S, Verstuyf A. Vitamin D metabolism and action. Osteoporosis Int 1998;8(Suppl 1):S13-9.
[22] Holick MF. Vitamin D requirements for humans of all ages: new increased requirements for women and men 50 years and older. Osteoporos Int 1998;8(Suppl):S24-9.
[23] Dawson-Hughes B, Heaney RP, Holick MF, et al. Estimates of optimal vitamin D status. Osteoporos Int 2005;16:713-6.
[24] Institute of Medicine of the National Academies. Dietary Reference Intakes for Calcium and Vitamin D. http://www.iom.edu/~/media/Files/Report%20Files/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D/Vitamin%20D%20and%20Calcium%202010%20Report%20Brief.pdf. Accessed Dec. 1, 2010.
[25] Bischoff-Ferrari HA, Giovannucci E, Willett WC, Dietrich T, Dawson-Hughes B. Estimation of optimal serum concentrations of 25-hydroxyvitamin D for multiple health outcomes. Am J Clin Nutr 2006;84:18-28.
[26] Tseng M, Giri V, Bruner DW, Giovannucci E. Prevalence and correlates of vitamin D status in African American men. BMC Public Health 2009;9:191-7.
[27] Lips P, Hosking D, Lippuner K, et al. The prevalence of vitamin D inadequacy amongst women with osteoporosis: an international epidemiological investigation. J Intern Med 2006;260:245-54.
[28] National Health and Nutrition Examination Survey. Centers for Disease Control and Prevention Web site. http://www.cdc.gov/nchs/nhanes.htm. Accessed June 28, 2010.
[29] Centers for Disease Control and Prevention, National Center for Health Statistics. National Health and Nutrition Examination Survey. Internet: http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/lab05_06.htm. Accessed Nov. 23, 2010.
[30] Looker AC, Pfeiffer CM, Lacher DA, Schleicher RL, Picciano MF, Yetley EA. Serum 25-hydroxyvitamin D status of the US population: 1988-1994 compared with 2000-2004. Am J Clin Nutr 2008;88: 1519-27.
[31] Yetley EA. Assessing the vitamin D status of the US population. Am J Clin Nutr 2008;88(suppl):558S-64S.
[32] Tangpricha V, Pearce EN, Chen TC, Holick MF. Vitamin D insufficiency among free-living healthy young adults. Am J Med. 2002;112:659-62. 50.
[33] Nesby-O'Dell S, Scanlon KS, Cogswell ME, et al. Hypovitaminosis D prevalence and determinants among African American and white women of reproductive age: third National Health and Nutrition Examination Survey, 1988-1994. Am J Clin Nutr 2002; 76:187-92.
[34] Zadshir A, Tareen N, Pan D, Norris K, Martins D. The prevalence of hypovitaminosis D among US adults: data from the NHANES III. Ethn Dis 2005;15(Suppl5):S5–97-S5-101.
[35] Araujo AB, Travison TG, Esche GR, Holick MF, Chen TC, McKinlay JB. Serum 25-hydroxyvitamin D and bone mineral density among Hispanic men. Osteoporos Int 2009;20:245-55.
[36] Jacobs ET, Alberts DS, Foote JA, et al. Vitamin D insufficiency in southern Arizona. Am J Clin Nutr 2008;87:608-13.
[37] Clemens TL, Henderson SL, Adams JS, Holick MF. Increased skin pigment reduces the capacity of skin to synthesise vitamin D_3. Lancet 1982;319:74-6.
[38] Holick MF, Chen TC. Vitamin D deficiency: a worldwide problem with health consequences. Am J Clin Nutr 2008;87(suppl):1080S-6S.
[39] Kakarala RR, Chandana SR, Harris SS, Kocharla LP, Dvorin E. Prevalence of vitamin D deficiency in uninsured women. J Gen Intern Med 2007;22:1180-3.

[40] Macdonald HM, Mavroeidi A, Barr RJ, Black AJ, Fraser WD, Reid DM. Vitamin D status in postmenopausal women living at higher latitudes in the UK in relation to bone health, overweight, sunlight exposure and dietary vitamin D. Bone 2008;42: 996-1003.

[41] Botella-Carretero JI, Alvarez-Blasco F, Villafruela JJ, Balsa JA, Vazquez C, Escobar-Morreale HF. Vitamin D deficiency is associated with the metabolic syndrome in morbid obesity. Clin Nutr 2007;26: 573-80.

[42] Muscogiuri G, Sorice GP, Prioletta A, et al. 25-Hydroxyvitamin D concentration correlates with insulin-sensitivity and BMI in obesity. Obesity 2010;10:1038.

[43] Gilsanz V, Kremer A, Mo AO, Wren TAL, Kremer R. Vitamin D status and its relation to muscle mass and muscle fat in young women. J Clin Endocrinol Metab 2010;95:1595-601.

[44] Menendez C, Lage M, Peino R, et al. Retinoic acid and vitamin D_3 powerfully inhibit in vitro leptin secretion by human adipose tissue. J Endocrinol 2001;170:425-31.

[45] Scragg R, Camargo CA. Frequency of leisure-time physical activity and serum 25-hyproxyvitamin D levels in the US population: results from the Third National Health and Nutrition Examination Survey. Am J Epidemiol 2008;168:577-86.

[46] Ford ES, Ajani UA, McBuire LC, Liu S. Concentrations of serum vitamin D and the metabolic syndrome among US adults. Diabetes Care 2005;28:1228-30.

[47] Chiu KC, Chu A, Go VLW, Saad MF. Hypovitaminosis D is associated with insulin resistance and beta cell dysfunction. Am J Clin Nutr 2004;79:820-5.

[48] Lind L, Hanni A, Lithell H, Hvarfner A, Sorensen OH, Ljunghall S. Vitamin D is related to blood pressure and other cardiovascular risk factors in middle-aged men. Am J Hypertens 1995;8:894-901.

[49] Ozfirat Z, Chowdhury TA. Vitamin D deficiency and type 2 diabetes. Postgrad Med J 2010;86:18-25.

[50] Lee JH, O'Keefe JH, Bell D, Hensrud DD, Holick MF. Vitamin D deficiency: an important, common, and easily treatable cardiovascular risk factor? J Am Coll Cardiol 2008;52:1949-56.

[51] Buckley LM, Hillner BE. A cost effectiveness analysis of calcium and vitamin D supplementation etidronate, and alendronate in the prevention of vertebral fractures in women treated with glucocorticoids. J Rheumatol 2003;30(1):132-8.