Prevalence of Cardiovascular Risk Factors and the Serum Levels of 25-Hydroxyvitamin D in the United States

Data From the Third National Health and Nutrition Examination Survey

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Background: Results of several epidemiologic and clinical studies have suggested that there is an excess risk of hypertension and diabetes mellitus in persons with suboptimal intake of vitamin D.

Methods: We examined the association between serum levels of 25-hydroxyvitamin D (25[OH]D) and select cardiovascular disease risk factors in US adults. A secondary analysis was performed with data from the Third National Health and Nutrition Examination Survey, a national probability survey conducted by the National Center for Health Statistics between January 1, 1988, and December 31, 1994, with oversampling of persons 60 years and older, non-Hispanic black individuals, and Mexican American individuals.

Results: There were 7186 male and 7902 female adults 20 years and older with available data in the Third National Health and Nutrition Examination Survey. The mean 25(OH)D level in the overall sample was 30 ng/mL (75 nmol/L). The 25(OH)D levels were lower in women, elderly persons (≥60 years), racial/ethnic minorities, and participants with obesity, hypertension, and diabetes mellitus. The adjusted prevalence of hypertension (odds ratio [OR], 1.30), diabetes mellitus (OR, 1.98), obesity (OR, 2.29), and high serum triglyceride levels (OR, 1.47) was significantly higher in the first than in the fourth quartile of serum 25(OH)D levels ($P<.001$ for all).

Conclusions: Serum 25(OH)D levels are associated with important cardiovascular disease risk factors in US adults. Prospective studies to assess a direct benefit of cholecalciferol (vitamin D) supplementation on cardiovascular disease risk factors are warranted.

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CARDIOVASCULAR DISEASE (CVD) is a major cause of mortality and morbidity in the United States.[1] Hypertension and diabetes mellitus are among the leading risk factors for CVD.[2] Several epidemiologic and clinical studies[3-5] have suggested that there is an excess risk of hypertension and diabetes mellitus among persons with suboptimal intake of vitamin D. Dietary sources of vitamin D are very few and are limited to fatty fish liver and fortified food sources, such as cereals and milk. The synthesis of vitamin D in the skin after exposure to type B UV light remains a major source of vitamin D in humans. The primary circulating form of vitamin D is 25-hydroxyvitamin D (25[OH]D), formed in the liver by the hydroxylation of vitamin D. The active form of the vitamin is 1,25-dihydroxyvitamin D, formed by a second hydroxylation of vitamin D, primarily in the kidneys, and is responsible for the physiologic functions of vitamin D. The nutritional status of vitamin D has always been assessed by the circulating level of 25(OH)D, but the data for the historical reference range for the circulating level of 25(OH)D originated from sun-deprived human populations with suboptimal vitamin D intake and may have underestimated the physiologic demands for vitamin D.[6,7] Recommended optimum levels of vitamin D have been established without accounting for the ubiquitous nature of the vitamin D receptor and the possible salutary affects of vitamin D on other organ systems that may affect CVD.[8,9] Indeed, even the adequacy of present recommendations for vitamin D to prevent osteomalacia has been questioned.[10]

We hypothesize that individuals with reduced 25(OH)D levels will exhibit an excess of CVD risk factors, and, therefore, we examined the association between serum 25(OH)D levels and CVD risk factors in US adults using data from the Third National Health and Nutrition Examination Survey (NHANES III).

METHODS

SURVEY AND SAMPLE

This study used data from the NHANES III, a national probability survey conducted by the National Center for Health Statistics at 89 survey locations between January 1, 1988, and December 31, 1994.[11] The survey was designed to estimate the prevalence of common chronic conditions and associated risk factors for disease control and prevention.[12] As described previously,[13] the sample for the survey was obtained through a complex multistage cluster design, with oversampling of persons 60 years and older, non-Hispanic black individuals, and Mexican American individuals to enhance the precision of prevalence estimates in these groups.

We examined interview and laboratory data from an initial cohort of 18 825 adult participants (aged ≥20 years) not taking cholecalciferol (vitamin D) except that contained in multivitamins. Racial/ethnic grouping for the purpose of this study was by self-identification as white, African American, and Hispanic. Participants who self-identified as "other" were excluded from this analysis owing to low sample size (n=715). We also excluded pregnant participants (n=225) and those with missing serum levels of 25(OH)D (n=2797), leaving a final sample of 15 088 adult participants.

STUDY VARIABLES

The diagnosis of diabetes mellitus was based on interview questions and fasting blood glucose levels. Participants who reported having ever been told by a physician that they have diabetes mellitus or sugar diabetes or who reported taking insulin or pills to lower blood glucose levels were classified as having diabetes mellitus. A fasting blood glucose level less than 110 mg/dL (<6.1 mmol/L) was considered normal. Participants with fasting blood glucose levels between 110 and 125.9 mg/dL (6.1-7.0 mmol/L) were classified as having impaired glucose tolerance, whereas those with levels of 126 mg/dL or greater (>7.0 mmol/L) were considered diabetic.

Hypertension status was established by history and blood pressure (BP) level. A certified technician performed BP measurements using a mercury sphygmomanometer and a standardized procedure.[14] A cuff size appropriate for the participant's arm circumference was used. Four BP readings were taken, with the average of the last 3 readings used for these analyses. Hypertension was defined as an average systolic BP of 140 mm Hg or greater, an average diastolic BP of 90 mm Hg or greater, or reported use of antihypertensive medications.[15]

Weight and height data were captured electronically from the measuring instruments to minimize potential data entry errors. Body mass index was calculated as weight in kilograms divided by the square of height in meters. Overweight was defined as a body mass index of 25 to 29.9, and obesity as a body mass index of 30 or greater.[16] Serum total cholesterol concentration was measured enzymatically at the Lipoprotein Analytical Laboratory at The Johns Hopkins Hospital, Baltimore, Md, which is certified by the Lipid Standardization Program of the Centers for Disease Control and Prevention. Individuals with total cholesterol concentrations of 240 mg/dL or greater (≥6.21 mmol/L) or who used cholesterol-lowering medications were considered to have high total cholesterol levels, whereas those with total cholesterol concentrations of 200 to 239 mg/dL (5.18-6.18 mmol/L) were considered to have above-optimal cholesterol levels. Participants with serum triglyceride levels of 150 mg/dL or greater (≥1.7 mmol/L) were considered to have hypertriglyceridemia.

Serum albumin levels were measured using an albumin test system (Boehringer Mannheim Diagnostics, Indianapolis, Ind) with bromcresol purple reagent. Bromcresol purple binds selectively with albumin and eliminates many of the nonspecific reactions with other serum proteins.[17] Participants with serum albumin levels less than 3.5 g/dL were considered to have low levels for the purposes of this analysis. Albuminuria was assessed by means of the urinary albumin–creatinine ratio and was evaluated at 2 levels defined as microalbuminuria, with a ratio of 30 to 300, and macroalbuminuria, with a ratio greater than 300. Glomerular filtration rate (GFR) was estimated from the serum creatinine (SCr) concentration using the Modification of Diet in Renal Disease[18] formula: GFR (in mL/min per 1.73 m^2) = 175 × SCr (exp[−1.154]) × Age (exp[−0.203]) × (0.742 if female) × (1.21 if black), with adjustments for differences in creatinine measurements between the NHANES III and the Modification of Diet in Renal Disease laboratories. Participants with estimated GFRs (eGFRs) less than 60 mL/min per 1.73 m^2 were considered to have significant chronic kidney disease for the purposes of this study.

Serum levels of 25(OH)D were measured using an INCSTAR 25(OH)D assay with a coefficient of variation of less than 10%. The first step in the assay procedure involves the rapid extraction of 25(OH)D and other hydroxylated metabolites from the serum or plasma using acetonitrile. After extraction, the treated sample was assayed by means of equilibrium radioimmunoassay. The radioimmunoassay method is based on an antibody with a relative specificity to 25(OH)D. The sample, antibody, and tracer were incubated for 90 minutes at 20°C to 25°C. Phase separation was accomplished after 20 minutes of incubation at 20°C to 25°C with a second antibody-precipitating complex.[19] The 25(OH)D levels reported usually represent the summation of ergocalciferol 25(OH)D_2 and cholecalciferol 25(OH)D_3. The radioimmunoassay method tends to overestimate the level of 25(OH)D because the antibody also recognizes 24,25-dihydroxyvitamin D, which comprises typically 10% to 15% of the value of the 25(OH)D assay.[20]

STATISTICAL ANALYSIS

Statistical analysis was based on 15 088 adults 20 years and older with data available in the NHANES III. The analysis sample was stratified by age, race/ethnicity, sex, BP level, history of hypertension, blood glucose level, history of diabetes mellitus, body mass index, triglyceride level, total cholesterol level, non–high-density lipoprotein cholesterol level, serum albumin level, eGFR, and albuminuria. Mean levels of serum 25(OH)D were computed and compared between groups using the 2-tailed *t* test or analysis of variance where appropriate.

The age- and sex-adjusted prevalences of select CVD risk factors were determined across quartiles of serum 25(OH)D levels. The significance of the differences in the age- and sex-adjusted prevalence of select CVD risk factors across quartiles of serum 25(OH)D levels analysis were evaluated by calculating the odds ratio for select CVD risk factors in the first and fourth quartiles of serum 25(OH)D level.

A random sample of the total number of different vitamin supplements reported by the participants was taken for a sensitivity analysis to determine the average dose of cholecalciferol. Data analyses were conducted using SAS (version 8.0; SAS Institute Inc, Cary, NC) and SUDAAN (version 8.0; Research Triangle Institute, Research Triangle Park, NC) to account for the predesigned oversampling, nonresponse bias, and poststratification population totals. Statistical hypotheses were tested using $P<.05$ as the level of statistical significance.

Table 1. Serum 25(OH)D Levels in the Study Sample

Characteristic	Participants, No.*	25(OH)D, Mean, ng/mL	*P* Value
Overall	15 088	30	
Age, y			
20-39	5983	31	1 [Reference]
40-59	4070	29	<.001
≥60	5035	28	<.001
Race			
White	6618	32	1 [Reference]
African American	4254	19	<.001
Hispanic	4216	25	<.001
Sex			
M	7186	31	1 [Reference]
F	7902	28	<.001
Blood pressure, mm Hg			
<120/<80	5818	31	1 [Reference]
120-139/80-89	5378	29	<.001
≥140/≥90	3720	27	<.001
History of hypertension			
No	10 720	30	1 [Reference]
Yes	4246	28	<.001
Blood glucose level, mg/dL			
<110	12 642	30	1 [Reference]
110-125.9	1041	27	<.001
≥126	1166	25	<.001
History of diabetes mellitus			
No	13 794	30	1 [Reference]
Yes	1276	25	<.001
Body mass index†			
<25	5874	32	1 [Reference]
25-29.9	5308	29	<.001
≥30	3869	26	<.001
Triglyceride level, mg/dL			
<150	10 009	30	1 [Reference]
≥150	4866	29	<.001
Total cholesterol level, mg/dL			
<200	7095	30	1 [Reference]
200-239	4761	30	.009
≥240	3053	29	<.001
Non-HDL cholesterol level, mg/dL			
<150	7222	30	1 [Reference]
≥150	7587	29	<.001
ACR			
<30	12 821	30	1 [Reference]
30-300	1429	27	<.001
>300	320	25	<.001
Serum albumin level, g/dL			
≥3.5	14 321	30	1 [Reference]
<3.5	439	23	<.001
eGFR, mL/min per 1.73 m^2			
≥60	13 788	30	1 [Reference]
<60	971	27	<.001

Abbreviations: ACR, albumin-creatinine ratio; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; 25(OH)D, 25-hydroxyvitamin D.

SI conversion factors: To convert cholesterol to millimoles per liter, multiply by 0.0259; glucose to millimoles per liter, multiply by 0.0555; 25(OH)D to nanomoles per liter, multiply by 2.496; triglycerides to millimoles per liter, multiply by 0.0113.

*Numbers may not sum to total because data are missing for some of the participants.

†Calculated as weight in kilograms divided by the square of height in meters.

RESULTS

Most participants were young (<40 years, n=5983), white (n=6618), and female (n=7902). Mean serum levels of 25(OH)D were lower in participants with select CVD risk factors. Participants with low serum albumin levels (<3.5 g/dL) and reduced eGFR (<60 mL/min per 1.73m^2) also exhibited lower mean serum levels of 25(OH)D (**Table 1**). The prevalence of serum levels of 25(OH)D less than 30 ng/mL (<75 nmol/L) was higher in women, elderly persons, and racial/ethnic minorities (**Figure**) and in participants with select CVD risk factors, including obesity, hypertension, diabetes mellitus, hypertriglyceridemia, and hypercholesterolemia (Table 1). When the analyses were stratified by race and sex, mean serum levels of 25(OH)D were lower in women and in white and

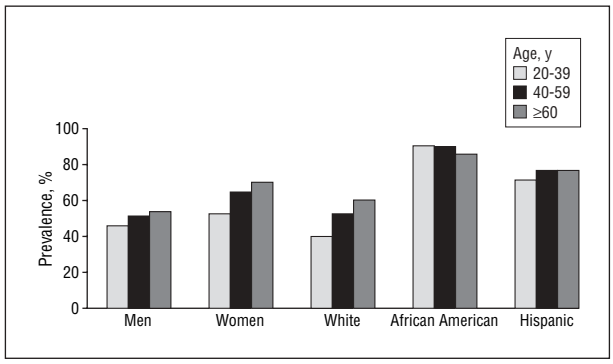

Figure. Prevalence of insufficient 25-hydroxyvitamin D levels (<30 ng/mL [<75 nmol/L]) by sex and race/ethnicity across age groups.

Hispanic participants with select CVD risk factors (**Table 2**). The relationship did not hold for African American participants, for whom the mean serum level of 25(OH)D was 19 ng/mL (47 nmol/L).

The age-, sex-, and race-adjusted prevalences and odds ratios were higher in the first than in the fourth quartile of serum 25(OH)D levels and were statistically significant for all of the select CVD risk factors except for reduced eGFR and elevated serum total and non–high-density lipoprotein cholesterol levels (**Table 3**). There was an inverse relationship between obesity, hypertension, and diabetes mellitus and serum levels of 25(OH)D in the overall population, but total cholesterol level was unrelated to serum levels of 25(OH)D.

Albuminuria and eGFR were included in all the analyses as indices of renal function to ensure that the association of vitamin D and CVD risk factors is not merely a function of abnormal mineral metabolism or other factors associated with CVD. Serum albumin concentration was included in all the analyses as a marker of nutritional status to mitigate the effect of malnutrition as a confounder of the association between serum vitamin D level and CVD risk factors. Low serum albumin levels were associated with low serum 25(OH)D levels in univariate and multivariate analyses but did not affect the association with CVD risk factors. The interaction term for race × serum albumin level was not statistically significant ($P = .08$). A random sample of 15 of the nearly 200 different vitamin supplements identified from the NHANES III medication list (6.7% of the sample) revealed that the average dose of cholecalciferol was only 297 IU/d. The Institute of Medicine[21] recommends an adequate daily intake level of 5 µg (200 IU) for individuals 1 to 50 years old, 10 µg (400 IU) for individuals 51 to 70 years old, and 15 µg (600 IU) for those older than 70 years.

COMMENT

This is the first study, to our knowledge, to demonstrate a significant association between low vitamin D levels and CVD risk factors in a nationally representative sample. Previous studies suggesting similar associations between low serum vitamin D levels and CVD risk factors were limited to subpopulations and small study samples.[3,22] Several plausible biological mechanisms that link vitamin D with CVD and CVD risk factors have been identified.

The administration of 1,25-dihydroxyvitamin D_3 has been shown to prevent the development of type 1 diabetes mellitus in animal models.[23,24] Serum levels of 25(OH)D less than 20 ng/mL (<50 nmol/L) have been associated with decreased β-cell function, and insulin sensitivity is as much as 60% higher in individuals with serum levels of 25(OH)D of 30 ng/mL (75 nmol/L) vs 10 ng/mL (25 nmol/L).[5] The doubling of the odds ratio for diabetes mellitus among the participants in the first quartile compared with the fourth quartile is consistent with the established literature and suggests a potential role for the serum level of 25(OH)D in the promotion of insulin sensitivity and the prevention of diabetes mellitus.

Vitamin D deficiency has been associated with congestive heart failure,[25] whereas incresed blood levels of 25(OH)D in response to UV-B irradiation have been associated with decreased BP.[26] The association of higher serum levels of the active vitamin D metabolite (1,25-dihydroxyvitamin D_3) with lower BP and plasma renin activity has led to the implication of vitamin D in the regulation of the renin-angiotensin system.[27-29] This finding is further supported by studies in the vitamin D receptor knockout mouse, an animal model emulating vitamin D deficiency, which displays increased BP, serum angiotensin-converting enzyme levels, and tissue renin content. In vitro studies[30] using a juxtaglomerular cell model have shown that 1,25-dihydroxyvitamin D_3 and other vitamin D analogues directly suppress renin expression via a vitamin D response element present in the renin gene. The administration of an activated vitamin D analogue has recently been shown to reduce proteinuria, suggesting a direct vascular effect of vitamin D[31] that is consistent with recent findings of 1-hydroxylase activity in vascular smooth muscle cells.[32] These basic studies provide plausible pathobiologic mechanisms for the association between low serum vitamin D levels and hypertension in this study.

The association of low serum vitamin D levels with obesity is less likely to be a direct effect of vitamin D. It has been shown that UV light exposure and time spent outdoors are better predictors of 25(OH)D levels than dietary vitamin D intake.[33] Diminished exposure to UV light associated with reduced outdoor activities and likely physical inactivity may account, in part, for the lower level of serum vitamin D in overweight and obese participants, who are more likely to be sedentary in their lifestyle. In addition, the lipid solubility of vitamin D also modifies its bioavailability and may contribute to the lower serum levels of vitamin D in overweight and obese participants.[34-36] The effect of vitamin D on adiposites and adipokines is unclear.

Vitamin D may affect CVD and its risk factors through other pathways, such as its immunosuppressive effects to reduce the proliferation of lymphocytes and the production of cytokines,[37] which have recently been identified as having an important role in atherogenesis.[38] Vitamin D receptors are present in T and B cells, monocytes, macrophages, dendritic cells, and natural killer cells.[39] Vitamin D analogues have been shown to inhibit the production of several proinflammatory cytokines while stimulating the effects of T_H2 lymphocytes, leading to a reduction in matrix metalloproteinase and, thereby, reducing

Table 2. Serum 25(OH)D Levels by Race and Sex

Variable	25(OH)D, Mean, ng/mL					
	White Participants		African American Participants		Hispanic Participants	
	Male	Female	Male	Female	Male	Female
Age, y						
20-39	35	34	21	18	28	23
40-59	32*	29*	21	21	26*	21*
≥60	31*	26*	22*	20*	26*	22
Blood pressure, mm Hg						
<120/<80	34	32	21	18	28	23
120-139/80-89	33*	29*	20	18	27	21*
≥140/≥90	32*	26*	21	19	26*	21*
History of hypertension						
No	33	31	21	18	27	23
Yes	32*	27*	21	18	26	21*
Blood glucose level, mg/dL						
<110	33	31	21	18	28	23
110-125.9	31*	25*	19	18	25*	20*
≥126	29*	25*	21	20*	22*	20*
History of diabetes mellitus						
No	33	31	21	18	27	23
Yes	29*	25*	22	19*	23*	21*
Body mass index†						
<25	35	33	21	19	28	24
25-29.9	33*	29*	21	18	27	23
≥30	31*	26*	20*	17*	25*	20*
Triglyceride level, mg/dL						
<150	34	31	21	18	28	22
≥150	31*	28*	21	18	26*	22
Total cholesterol level, mg/dL						
<200	34	31	21	18	27	22
200-239	33*	30*	21	18	27	22
≥240	32*	28*	21*	19*	27	22
Non-HDL cholesterol level, mg/dL						
<150	34	32	21	18	28	22
≥150	32*	28*	21	19*	27*	22
ACR, male/female						
<20/<30	33	31	21	18	27	22
20-200/30-300	32*	28*	20	19	26*	22
>200/>300	29*	25*	21	19	23*	20
Serum albumin level, g/dL						
≥3.5	33	30	21	18	27	22
<3.5	23*	27*	17*	16*	21*	21
eGFR, mL/min per 1.73 m^2						
≥60	33	31	21	18	27	22
<60	31*	26*	23*	21*	22*	21

Abbreviations: ACR, albumin-creatinine ratio; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; 25(OH)D, 25-hydroxyvitamin D.

SI conversion factors: To convert cholesterol to millimoles per liter, multiply by 0.0259; glucose to millimoles per liter, multiply by 0.0555; 25(OH)D to nanomoles per liter, multiply by 2.496; triglycerides to millimoles per liter, multiply by 0.0113.

*$P<.05$ compared with the reference group (first row).

†Calculated as weight in kilograms divided by the square of height in meters.

plaque production or instability. Disruption of the nuclear vitamin D receptor gene, simulating vitamin D deficiency, has also been associated with increased thrombogenicity in mice.[40] Furthermore, low vitamin D states, which are associated with abnormal bone turnover, have been postulated directly and indirectly to affect CVD risk by increasing susceptibility to arterial calcification[41,42] and, subsequently, hypertension via increased arterial resistance.[43,44]

The results of this study originated from the analysis of a representative sample of the US population and are likely to have broad implications with implicit limitations. Although NHANES III provides some of the best available estimates of the prevalence and treatment of chronic diseases in the United States, its cross-sectional design does not allow for direct causal inference.

The timing of blood sample collections for NHANES participants occurred within communities at different latitudes, which may have affected the distribution of serum vitamin D levels. The staggering of blood sample collection minimized the impact of seasonal variation due to sunlight exposure on vitamin D levels as samples were

Table 3. Age-, Sex-, and Race-Adjusted Prevalence and ORs of Select Cardiovascular Disease Risk Factors Between the First and Fourth Quartiles of Serum 25(OH)D Levels

Risk Factor	Prevalence of Cardiovascular Risk Factor		OR (95% CI)	*P* Value
	1st Quartile (<21 ng/mL)	4th Quartile (≥37 ng/mL)		
Blood pressure ≥140/≥90 mm Hg	20.46	15.10	1.30 (1.13-1.49)	.001
Fasting blood glucose level, mg/dL				
110-125	6.96	3.25	2.15 (1.69-2.74)	<.001
≥126	6.85	3.38	1.98 (1.57-2.51)	<.001
History of diabetes mellitus	6.96	3.28	1.73 (1.38-2.16)	<.001
Body mass index ≥30*	24.69	11.50	2.29 (1.99-2.63)	<.001
Triglyceride level ≥150 mg/dL	32.86	23.84	1.47 (1.30-1.65)	<.001
Total cholesterol level ≥240 mg/dL	19.98	15.92	0.97 (0.85-1.11)	.65
Non-HDL cholesterol level ≥150 mg/dL	48.99	41.49	1.04 (0.93-1.16)	.50
Serum albumin level <3.5 g/dL	2.77	1.57	2.90 (1.89-4.46)	<.001
eGFR <60 mL/min per 1.73 m^2	5.12	4.27	1.08 (0.87-1.35)	.47
ACR ≥200 for males/≥300 for females	1.59	0.76	2.54 (1.65-3.48)	<.001

Abbreviations: ACR, albumin-creatinine ratio; CI, confidence interval; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; 25(OH)D, 25-hydroxyvitamin D; OR, odds ratio.

SI conversion factors: To convert cholesterol to millimoles per liter, multiply by 0.0259; glucose to millimoles per liter, multiply by 0.0555; 25(OH)D to nanomoles per liter, multiply by 2.496; triglycerides to millimoles per liter, multiply by 0.0113.

*Calculated as weight in kilograms divided by the square of height in meters.

collected during the warmer months in northern areas, when sunlight is more abundant. This NHANES III blood sampling method made it appropriate for assessing vitamin D levels in the general population.[45] The high prevalence of CVD risk factors in ethnic minorities, who have been found to be more likely to be vitamin D insufficient,[46] may have affected the results of these analyses despite the inclusion of race in the statistical modeling. Hispanic participants exhibited lower levels of 25(OH)D than white participants, and Hispanic participants with CVD risk factors exhibited lower levels of 25(OH)D than those without CVD risk factors. African American participants exhibited the lowest levels of 25(OH)D, with little or no correlation between 25(OH)D levels and CVD risk factors. The poor correlation between CVD risk factors and levels of 25(OH)D in African American participants is likely due to the very low levels of 25(OH)D in this subpopulation.

This study provides important information to support a reassessment of the current position on what levels of vitamin D constitute vitamin D insufficiency and necessitate vitamin D repletion. The current recommended levels of serum 25(OH)D are primarily based on levels needed to maintain optimum bone health and prevent rickets but do not address the levels of vitamin D that may be necessary to minimize the prevalence of CVD risk factors.[47] Our group[48] recently reported that mean serum 25(OH)D levels in the general population, and in particular in the elderly, women, and minority populations, were substantially below the recommended national goal. Although the implication of the present findings for the excess prevalence of CVD risk factors remains to be determined, note that the inverse relationship between the prevalence of several CVD risk factors (obesity, diabetes mellitus, and hypertension) and 25(OH)D levels continued well into the fourth quartile, suggesting that levels of 37 ng/mL or greater (≥92 nmol/L) may confer additional health benefits. Prospective studies are warranted to assess a direct effect of vitamin D on select CVD risk factors and to establish the optimum serum level of vitamin D.

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