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Vitamin D Deficiency and Seasonal Variation in an Adult South Florida Population

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Hypovitaminosis D is associated with impaired neuromuscular function, bone loss, and fractures. If a person is not taking a vitamin supplement, sun exposure is often the greatest source of vitamin D. Thus, vitamin D deficiency is not uncommon in the winter, particularly in northern latitudes. Our goal was to establish the prevalence of vitamin D deficiency in south Florida (U.S.), a region of year-round sunny weather. At the end of the winter, 212 men and women attending an internal medicine clinic at a local county hospital were enrolled for measurements of 25-hydroxyvitamin D [25(OH)D], 1,25-dihydroxyvitamin D, and PTH; 99 participants returned at the end of summer. The mean (±SD) winter 25(OH)D concentration was 24.9 ± 8.7 ng/ml (62.3 ± 21.8 nmol/liter) in men and 22.4 ± 8.2 ng/ml (56.0 ± 20.5 nmol/liter) in women. In winter, the prevalence of hypovitaminosis D, defined as 25(OH)D less than 20 ng/ml (50 nmol/liter), was 38% and 40% in men and women, respectively. In the 99 subjects who returned for the end of summer visit, the mean 25(OH)D concentration was 31.0 ± 11.0 ng/ml (77.5 ± 27.5 nmol/liter) in men and 25.0 ± 9.4 ng/ml (62.5 ± 23.5 nmol/liter) in women. Seasonal variation represented a 14% summer increase in 25(OH)D concentrations in men and a 13% increase in women, both of which were statistically significant. The prevalence of hypovitaminosis D is considerable even in southern latitudes and should be taken into account in the evaluation of postmenopausal and male osteoporosis. (*J Clin Endocrinol Metab* 90: 1557–1562, 2005)

ALTHOUGH SEVERE VITAMIN D deficiency leads to osteomalacia and rickets, mild to moderate deficiency can result in osteoporosis and increased fracture risk. In the elderly, hypovitaminosis D is also associated with increased impaired neuromuscular function and increased falls (1, 2). Common causes of vitamin D deficiency include a diet low in vitamin D and reduced exposure to sunlight (3). Very few foods, such as fish oils, fatty fish, and eggs, naturally contain significant amounts of vitamin D, and in the United States, only some foods are fortified with vitamin D, mostly milk and cereals (4). Thus, in individuals not taking vitamin supplements, the attainment of adequate vitamin D levels is believed to be mostly due to sufficient sun exposure (5, 6). Solar radiation is weaker and hours of sunlight are shorter in winter compared with summer as well as in high latitudes compared with low latitudes. Thus, in adults living in the northern United States and Europe, there is not only a higher prevalence of hypovitaminosis D compared with regions closer to the equator, but also a large seasonal variation, with higher levels at the end of summer and lower levels at the end of winter (7–10).

Based on the important contribution of sunlight exposure to vitamin D levels, it has been commonly assumed that hypovitaminosis D is negligible and vitamin D seasonal variation is minimal in populations living in lower latitudes. Our goal was to establish the prevalence of hypovitaminosis D and vitamin D seasonal variation in an ambulatory adult population of south Florida, one of the southernmost regions of the continental United States (latitude 25.46°N) and an area of year-round sunny, warm weather. If in northern latitudes, long-term low vitamin D concentrations result in increased bone loss, fractures, and neuromuscular decline, we could hypothesize that individuals with year-round sunlight exposure, such as those living in low latitudes, should be chronically vitamin D replete and, therefore, not only have preserved neuromuscular function, but also experience low rates of bone loss and fractures. Such a finding could have enormous social and financial implications.

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Abbreviations: MARS, Multivariate adaptive regression splines; $1,25(OH)_2D$, 1,25-dihydroxyvitamin D; 25(OH)D, 25-hydroxyvitamin D.

Subjects and Methods

Study design and subjects

The study was conducted at the end of two seasonal periods: the first visit occurred in March 2000, at the end of winter, and the second visit occurred in September 2000, at the end of summer. Subjects were recruited in the outpatient internal medicine clinic at Jackson Memorial Hospital, the county hospital in Miami, FL. Patients attending outpatient clinics at this facility tend to be indigent or have low socioeconomic status. All consecutive ambulatory patients 18 yr of age or older attending the clinic in March 2000 were offered participation in the study. Our exclusion criteria included pregnancy and diseases or medications known to affect vitamin D, calcium, or bone metabolism. Enrollment was voluntary, and all participants signed a consent form previously approved by the hospital's medical sciences subcommittee

TABLE 1. Characteristics of the participants at baseline

Characteristic	Value
Age [yr (mean ± SD, range)]	54.6 ± 13.1, 18–88
Men [no. (%)]	77 (36.3)
Women [no. (%)]	135 (63.7)
White non-Hispanic [no. (%)]	8 (3.8)
White Hispanic [no. (%)]	157 (74.1)
Black [no. (%)]	45 (21.2)
Other [no. (%)]	2 (0.9)
Vitamin D intake [IU/d (mean ± SD, range)]	326.5 ± 252, 0–1568
Taking vitamin supplements [no. (%)]	207 (97.6)
Calcium intake [mg/d (mean ± SD, range)]	1152.0 ± 667, 40–3429
1–1000 mg/d [no. (%)]	96 (45.3)
>1000 mg/d [no. (%)]	116 (54.7)
Sun exposure [no. (%)]	
Mild	117 (55.2)
Moderate	30 (14.2)
Extensive	65 (30.6)

for the protection of human subjects in research. Participants were asked to return for a second, end of summer visit in September 2000.

Medical history and vital signs were obtained at the first visit. All subjects were assessed for vitamin D and calcium intake, sunlight exposure, and current medications at both visits. Also at both visits, blood was drawn for determination of fasting levels of 25-hydroxyvitamin D [25(OH)D], 1,25-dihydroxyvitamin D [1,25$(OH)_2$D], and intact PTH.

Dietary and sun exposure assessment

Dietary calcium and vitamin D intakes were estimated by a validated food frequency questionnaire containing 107 questions that focused on both calcium- and vitamin D-containing foods consumed over the past month (11). Mean total calcium and total vitamin D intake were calculated from the mean dietary plus the mean supplement intake for both the end of summer and end of winter visits.

A sun exposure questionnaire was administered to determine the amount of sunlight exposure received in the week before the study visit. The questionnaire developed by Hodkinson *et al.* (12) and adapted by Salamone *et al.* (13) has been supported by film badge measurements. It asks the number of times spent outdoors in the previous week, duration of time outdoors, type of clothing, and use of sunscreen. Travel to northern locations in the previous 6 months is also recorded. Two subjects who traveled out of state in the previous 6 months were excluded from the sun exposure analysis.

Laboratory tests

Samples were stored at −70C until tested. All samples were tested in the same run at the end of the study. Serum concentrations of 25(OH)D and 1,25$(OH)_2$D were determined by Dr. Hollis using an RIA from DiaSorin (Stillwater, MN). These assays have been well characterized and recognize both the D_2 and D_3 forms of the vitamin, as recently reported (14–16). The intraassay coefficient of variation is 10% for 25(OH)D and 15% for 1,25$(OH)_2$D. Serum intact PTH was measured by immunometric assay (Immulite, Diagnostic Products Corp., Los Angeles, CA). Intra- and interassay coefficients of variation are 5.4% and 5.0%, respectively.

Statistical analysis

Descriptive statistics were performed separately for winter and summer. A *t* test or one-way ANOVA was used to compare means between or among demographic groups for continuous variables, including 25(OH)D, 1,25$(OH)_2$D, and PTH concentrations. χ^2 tests were used for categorical variables such as sun exposure, vitamin D, and calcium intake. If the expected number of any cell in a contingency table was less than 5, Fisher's exact probability test was used instead. For seasonal variation, only those subjects who had both winter and summer visits were included in the analysis. A paired *t* test was used to compare differences between winter and summer.

To determine the cut-off point to define hypovitaminosis D based on PTH levels, we performed adaptive regression of PTH on 25(OH)D level. Based on scatter plot, the relationship between PTH and 25(OH)D was not linear; however, it appears that the decrease in PTH levels ceases when vitamin D levels are beyond a turning point (inflection point). We used multivariate adaptive regression splines (MARS) to estimate the inflection point (17). MARS is an adaptive procedure for regression; it is a nonparametric regression procedure that makes no assumption about the underlying functional relationship of 25(OH)D with PTH. To adjust for potential confounding factors in the MARS regression analysis, age, gender, and race were treated as covariates. All statistical analyses were performed using the SAS System for Windows, V8 (18).

TABLE 2. Winter measurements of 25(OH)D, 1,25$(OH)_2$D, and PTH

Subjects	25(OH)D (ng/ml)	1,25$(OH)_2$D (pg/ml)	PTH (pg/ml)
All	23.3 ± 8.4 (6–53)	36.1 ± 8.0 (3–62)	45.9 ± 30.6 (6–232)
Women	22.4 ± 8.2 (6–53)	36.5 ± 8.4 (3–62)	46.4 ± 32.9 (8–232)
Men	24.9 ± 8.7 (10–53)	35.4 ± 7.2 (21–55)	45.0 ± 26.3 (6–184)
All age <45 yr	21.7 ± 7.2 (9–39)	39.4 ± 7.3 (23–62)	38.6 ± 31.0 (16–219)
All age 45–60 yr	24.0 ± 9.0 (6–53)	36.5 ± 7.8 (22–62)	46.2 ± 32.0 (8–232)
All age >60 yr	23.5 ± 8.5 (7–53)	33.9 ± 8.0 (3–51)	49.2 ± 28.7 (6–184)
Women age <45 yr	20.5 ± 7.2 (9–39)	39.2 ± 7.2 (23–52)	40.3 ± 37.2 (18–219)
Women age >45 yr	23.0 ± 8.4 (6–53)	35.8 ± 8.6 (3–62)	48.1 ± 31.6 (8–232)
Men age <45 yr	24.5 ± 7.2 (14–35)	39.9 ± 7.7 (29–55)	34.9 ± 14.9 (16–74)
Men age >45 yr	25.0 ± 9.1 (10–53)	34.2 ± 6.7 (21–49)	48.0 ± 28.2 (6–184)
White non-Hispanic	22.3 ± 9.0 (10–40)	34.5 ± 7.1 (26–47)	32.8 ± 11.0 (18–54)
White Hispanic	23.6 ± 7.8 (6–53)	36.2 ± 8.2 (3–62)	47.6 ± 33.4 (6–232)
Black	22.4 ± 10.3 (9–53)	36.4 ± 7.6 (21–55)	43.2 ± 21.8 (11–95)
Other	25.5 ± 16.3 (14–37)	32.5 ± 6.4 (28–37)	33.0 ± 18.4 (20–46)
Vitamin D intake >0 and <400 IU/d	23.0 ± 8.5 (6–53)	36.1 ± 8.3 (3–62)	49.1 ± 32.8 (6–232)
Vitamin D intake 400–800 IU/d	23.4 ± 7.8 (10–41)	36.5 ± 7.1 (23–59)	40.8 ± 26.7 (10–184)
Vitamin D intake >800 IU/d	26.9 ± 9.7 (12–46)	33.6 ± 6.7 (22–45)	32.1 ± 14.7 (11–55)
Calcium intake <500 mg/d	22.8 ± 8.4 (6–53)	36.2 ± 8.0 (3–55)	46.6 ± 25.3 (13–148)
Calcium intake >500 mg/d	23.7 ± 8.4 (8–53)	36.0 ± 8.4 (21–62)	45.4 ± 34.5 (6–232)
Sun exposure, mild	22.8 ± 8.7 (6–53)	36.0 ± 8.3 (3–59)	48.9 ± 34.4 (8–232)
Sun exposure, moderate	22.4 ± 6.1 (12–34)	37.1 ± 8.4 (21–62)	39.7 ± 22.4 (10–117)
Sun exposure, extensive	24.7 ± 8.9 (10–53)	35.8 ± 7.3 (21–52)	43.4 ± 26.1 (6–184)

Values are the mean ± SD, with the range in *parentheses.* Multiply by 2.5 to convert 25(OH)D from ng/ml to nmol/liter; multiply by 2.5 to convert 1,25$(OH)_2$D from pg/ml to pmol/liter; multiply by 1 to convert PTH from pg/ml to ng/liter.

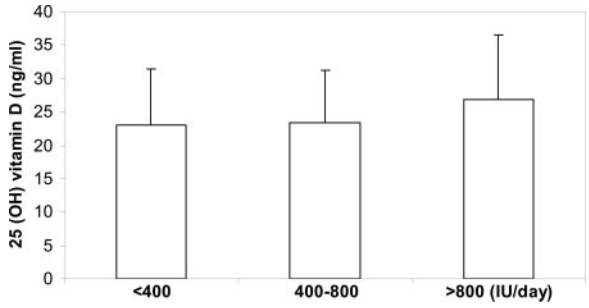

FIG. 1. 25(OH)D concentrations according to daily vitamin D intake.

Results

Clinical characteristics

We enrolled 212 patients in the study, 77 men and 135 women. The baseline characteristics of our sample are described in Table 1. Most of the subjects were women (64%) and Hispanic whites (74%). Sun exposure was mild in 55% of the individuals included in this study, and 97% reported taking vitamin supplements. Vitamin D and calcium intake was higher in men than in women and in subjects younger than age 45 yr than in those older than age 60 yr, but the difference was not significant in either group.

Table 2 shows winter 25(OH)D, $1,25(OH)_2D$, and PTH mean values in all subjects and in different subgroups. Higher vitamin D intake and greater sun exposure were associated with higher serum 25(OH)D concentrations (Figs. 1 and 2, respectively). Overall, 25(OH)D concentrations were lower in women than in men ($P = 0.02$) and in subjects with daily vitamin D intake less than 800 IU ($P = 0.08$). There was no relationship between age and 25(OH)D levels (Fig. 3). As expected, PTH increased with age (Fig. 4), and $1,25(OH)_2D$ decreased with age; levels were statistically significantly higher in individuals less than 45 yr of age than in older individuals ($P < 0.01$).

As shown in Fig. 5, PTH concentrations decreased as 25(OH)D serum levels increased. This negative relation ceased when 25(OH)D reached a certain level. Multivariate adaptive regression analysis, with age and gender as covariates, has indicated that the inflection point occurred at a 25(OH)D concentration of 20 ng/ml ($P = 0.024$). Therefore, in the present report, a serum concentration of 25(OH)D less than 20 ng/ml (50 nmol/liter) was used as the cut-off limit in defining hypovitaminosis D.

Prevalence of hypovitaminosis D in winter

The mean 25(OH)D concentration in winter for the entire study population was 23.3 ± 8.4 (±SD) ng/ml (58.3 ± 21 nmol/liter); it was 24.9 ± 8.7 (62.3 ± 21.8 nmol/liter) in men and 22.4 ± 8.2 ng/ml (56.0 ± 21.0 nmol/liter) in women. When hypovitaminosis D was defined as serum 25(OH)D levels less than 20 ng/ml (50 nmol/liter), the prevalence of hypovitaminosis D in winter in our 212 patients was 39–38% in men and 40% in women (Table 3). If the cut-off limit was set at 12 ng/ml (30 nmol/liter) or less, the prevalence was 9% (Table 3). Only six subjects (2.8%) had values of 8 ng/ml (20 nmol/liter) or less.

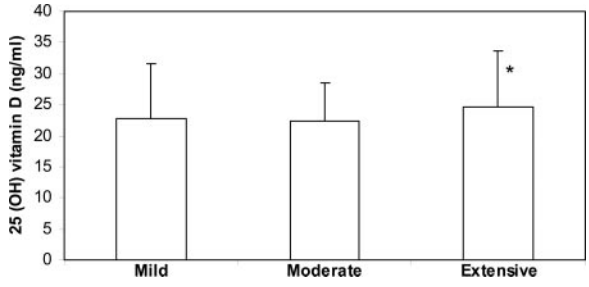

FIG. 2. 25(OH)D concentrations according to sun exposure.

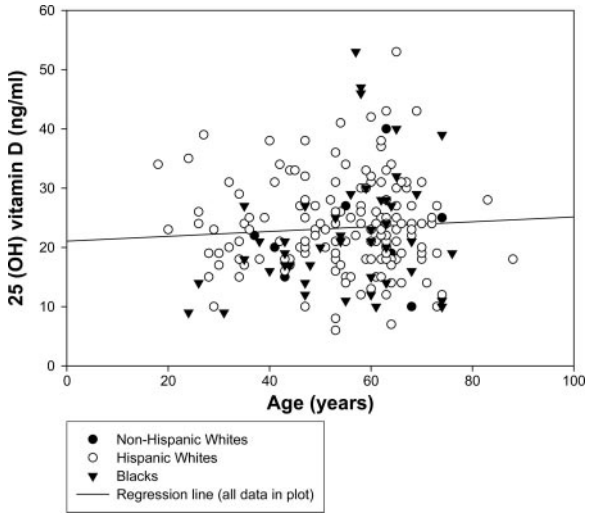

FIG. 3. 25(OH)D level according to age.

Among the 146 individuals who reported a daily vitamin D consumption of less than 400 IU, 57 (39%) were vitamin D deficient; of the 47 subjects consuming 400–800 IU vitamin D/d, 20 (43%) were deficient; and of the 16 individuals consuming more than 800 IU/d, 5 (31%) were deficient (Table 3).

When defined as concentrations below 12 ng/ml (30 nmol/liter), hypovitaminosis D was more prevalent in women than in men (10% *vs.* 5%; $P = 0.09$), as shown in Table 3. There was also a higher prevalence of hypovitaminosis D in individuals older than 45 yr of age than in younger individuals (9% *vs.* 6%), and in blacks (18%) and non-Hispanic whites (13%) than in Hispanic whites (6%).

Prevalence of hypovitaminosis D in summer

Only 99 (47%) subjects returned for the second visit at the end of summer. The subjects who returned for the second visit had characteristics similar to those who did not return (Table 4). However, it is noteworthy that older female patients were more likely to return for the end of summer visit. For the returning subjects, the mean 25(OH)D concentration in summer was 26.8 ± 10.3 (±SD) ng/ml (67.0 ± 25.8 nmol/liter); it was 31.0 ± 11.0 ng/ml (52.5 ± 27.5 nmol/liter) in men and 25.0 ± 9.4 ng/ml (62.5 ± 23.5 nmol/liter) in women.

At the end of the summer, the prevalence of hypovitaminosis D was 22%, if we define vitamin D deficiency as concentrations below 20 ng/ml (50 nmol/liter) and 6% if we define deficiency as concentrations below 12 ng/ml (30 nmol/liter; Table 5). At the 20 ng/ml cut-off point, summer prevalence was higher in women than in men (28% *vs.* 10%; $P = 0.07$; Table 5). With 12 ng/ml (30 nmol/liter) as the cut-off point, none of the men and 9% of the women had vitamin D deficiency.

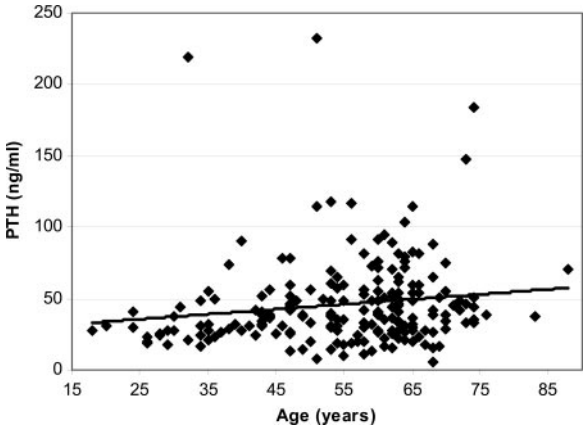

FIG. 4. PTH concentrations according to age.

Seasonal variation

In the seasonal variation analysis, we took into account only the 99 subjects seen at both the winter and summer visits. For this subgroup, the winter 25(OH)D concentration was 27.0 ± 9.8 ng/ml (67.5 ± 24.5 nmol/liter) in men and 22.0 ± 8.3 ng/ml (55.0 ± 20.8 nmol/liter) in women. In the summer, the 25(OH)D concentration was 31.0 ± 11.0 ng/ml (77.5 ± 27.5 nmol/liter) in men and 25.0 ± 9.4 ng/ml (62.5 ± 23.5 nmol/liter) in women (Fig. 6). Seasonal variation was statistically significant for both men ($P = 0.0033$) and women ($P = 0.0009$). This seasonal change represents a 14.8% summer increase in 25(OH)D concentrations in men and a 13% increase in women.

Discussion

Vitamin D plays a major role in calcium and bone metabolism. It increases intestinal absorption of calcium and phosphate and is an essential factor in the mineralization of the skeleton. In addition, vitamin D seems to play an important part in the preservation of neuromuscular function and in the prevention of certain cancers (2, 19, 20). Although rickets and osteomalacia are rare, less severe vitamin D deficiency appears to be quite common, particularly in certain populations, such as homebound individuals and the elderly. Hypovitaminosis D can result from inadequate exposure to sunlight and/or poor dietary intake. Because of concerns arising from extended winter periods of weak sunlight, most studies assessing the prevalence of hypovitaminosis D have taken place in high latitude regions.

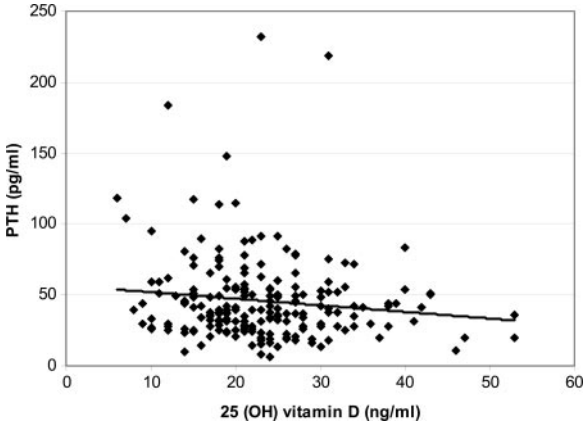

FIG. 5. PTH concentrations according to 25(OH)D levels.

TABLE 3. Winter prevalence of hypovitaminosis D according to different set-points for vitamin D deficiency in all subjects and among various subgroups (n = 212)

	25(OH)D ≤12 ng/ml (30 nmol/liter)	25(OH)D <20 ng/ml (50 nmol/liter)
All subjects	18 (9)	83 (39)
Age		
<45 yr	3 (6)	24 (51)
45–60 yr	7 (9)	27 (34)
>60 yr	8 (9)	32 (37)
Gender		
Female	14 (10)	54 (40)
Male	4 (5)	29 (38)
Race		
Hispanic whites	9 (6)	4 (50)
Non-Hispanic whites	1 (13)	57 (36)
Black	8 (18)	21 (47)
Other	0	1 (50)
Vitamin D intake		
<400 IU/d	14 (10)	57 (39)
400–800 IU/d	2 (4)	20 (43)
>800 IU/d	1 (7)	5 (31)
Calcium intake		
<500 mg/d	4 (11)	11 (31)
>500 mg/d	14 (8)	72 (41)
Sun exposure		
Mild	12 (10)	49 (42)
Moderate	1 (3)	11 (37)
Extensive	5 (8)	23 (35)

Values are numbers of subjects (percentage).

This is the first report of the prevalence of vitamin D deficiency in an ambulatory adult population living in south Florida, a low latitude region (25.46°N) in the continental United States. In this group, we have shown a 39% prevalence of hypovitaminosis D at the end of winter, not very different from studies performed in northern regions of the United States and Europe. Comparable studies in Boston have found a 35–75% winter prevalence of hypovitaminosis D (21, 22). Prevalence in our Hispanic population, which consisted mainly of Hispanic whites, was similar to that reported for Hispanics living in Boston (50% *vs.* 48%) (22). The 14% seasonal variation in vitamin D concentrations we observed in south Florida residents seems to be smaller than that reported in northern latitudes (up to 38%) (23). As described in other populations, we found an inverse correlation between 25(OH)D and PTH levels. Also as expected, $1,25(OH)_2D$ levels declined with advancing age, probably reflecting an age-associated impairment in the renal hydroxylation of 25(OH)D.

Regular sunlight exposure has been considered an effective prophylaxis against vitamin D deficiency (24). However, studies in other regions of the world located at low latitude, such as the Middle East, have also shown a high prevalence of vitamin D deficiency, from 50–97%. These findings have been explained as being mostly due to the customary clothing that covers nearly the entire body (25, 26). Although clothing in south Florida commonly leaves arms and legs uncovered, other factors can impair dermal vitamin D production, including age, pigmentation of the skin, and sunscreen use (27). The higher than expected prevalence of vitamin D deficiency in south Florida could be explained by

TABLE 4. Baseline characteristics of the subjects who were seen only for a winter visit and of those who returned for a summer visit

Characteristic	Winter visit only (n = 113)	Winter and summer visits (n = 99)
Age [yr (mean ± SD, range)]	53.0 ± 13.8 (18–88)	56.5 ± 12.1 (20–83)
Males [no. (%)]	47 (41.6)	30 (30.3)
Females [no. (%)]	66 (58.4)	69 (69.7)
White non-Hispanic [no. (%)]	5 (4.4)	3 (3.0)
White Hispanic [no. (%)]	77 (68.1)	80 (80.8)
Black [no. (%)]	31 (27.5)	14 (14.2)
Other [no. (%)]	0	2 (2.0)
Vitamin D intake [IU/d (mean ± SD, range)]	317.7 ± 263.6 (9–1568)	336.5 ± 239.5 (0–1278)
Calcium intake [mg/d (mean ± SD, range)]	1109.7 ± 662.2 (40–3058)	1200.2 ± 672.5 (75–3429)

avoidance of sun exposure because of the heat and increased awareness of the risk of developing skin cancer. The small seasonal variation in 25(OH)D concentrations can be explained by the more even exposure to sun throughout the year in south Florida, compared with northern regions where sun exposure is minimal because of the cold weather and shorter hours of sunlight during the winter months.

We observed a good correlation between the use of vitamin supplements and serum 25(OH)D concentrations. However, although 97% of our subjects reported the use of vitamins, their intake of vitamin D did not make up for insufficient sun exposure. Mean vitamin D intake in our subjects was 326 IU/d; although higher than the current recommended daily allowance of 200 IU/d, it does not appear to be sufficient to maintain adequate serum levels in some individuals and supports recent calls for increasing the recommended daily allowance of vitamin D (28). The mean daily calcium intake in our population was 1152 mg, higher than that usually reported (29).

There is no consensus on what concentration of 25(OH)D defines vitamin D deficiency. Some have proposed a population-based estimate, and others define deficiency as the vitamin D concentration below which PTH concentrations begin to rise. The difficulty in defining vitamin D deficiency based on a population-based reference sample lies in the fact that the definition will change according to the criteria used to select the sample population, including age, race, health status, time of year, and geographical location (30). Although concentrations less than 8 ng/ml are usually considered severe vitamin D deficiency (31), different investigators have defined the normal lower limit as 25(OH)D levels below 12 ng/ml (30 nmol/liter) or below 20 ng/ml (50 nmol/liter). Low levels of 25(OH)D are associated with impaired calcium absorption and the compensatory increase in PTH concentrations, which, in turn, stimulates bone resorption and bone loss. This PTH-mediated bone loss has been shown to contribute to increased fractures (32). Thus, it would be reasonable to set the lower limit of normal at the concentration of 25(OH)D that suppresses PTH, thereby reflecting a biological response to a "normal" vitamin D level. Strong epidemiological data support this approach; patients with hip fractures have a higher prevalence of hypovitaminosis D than controls with normal or low bone mineral density (33), and studies in elderly women receiving vitamin D supplements show a 19–43% reduction in nonvertebral fractures (34, 35) and a 49% reduction in falls (2). In addition to the complexity of defining the normal range of vitamin D concentrations and then estimating the true prevalence of vitamin D deficiency, taking into account geographical and seasonal differences, one must also consider the variation in vitamin D determinations among assays (36). Similar to the reports of others, we have defined vitamin D deficiency as concentrations less than 20 ng/ml (50 nmol/liter), because below this level PTH began to rise (37–39).

TABLE 5. Summary prevalence of hypovitaminosis D according to different set-points for vitamin D deficiency in all subjects and among various subgroups (n = 99)

	25(OH)D ≤12 ng/ml (30 nmol/liter)	25(OH)D <20 ng/ml (50 nmol/liter)
All subjects	6 (6)	22 (22)
Age		
<45 yr	2 (12)	3 (18)
45–60 yr	4 (11)	9 (25)
>60 yr	0	10 (22)
Gender		
Female	6 (9)	19 (28)
Male	0	3 (10)
Race		
Hispanic whites	0	0
Non-Hispanic whites	5 (6)	18 (23)
Black	1 (7)	3 (21)
Other	0	1 (50)
Vitamin D intake		
<400 IU/d	3 (4)	18 (25)
400–800 IU/d	2 (10)	3 (21)
>800 IU/d	0	1 (50)
Calcium intake		
<500 mg/d	1 (6)	5 (31)
>500 mg/d	5 (6)	17 (21)
Sun exposure		
Mild	3 (8)	10 (27)
Moderate	2 (17)	2 (17)
Extensive	1 (2)	10 (20)

Values are numbers of subjects (percentage).

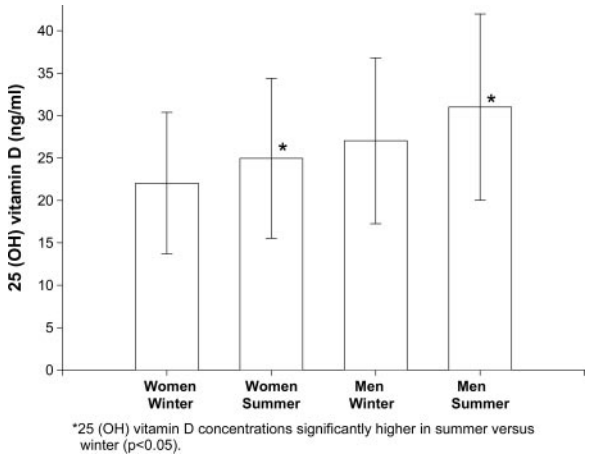

FIG. 6. Mean 25(OH)D concentrations by gender and season.

Our study has the following limitations: subjects were not randomly selected, but volunteered to participate, and the population consisted of patients attending the internal medicine clinic of a county hospital, who, although ambulatory and in apparent good health, could be sicker than the general population or not as well nourished. Also, because renal function was not routinely assessed in our study population, we cannot calculate the contribution of declining renal function associated with aging to their $1,25(OH)_2D$ and PTH levels. Finally, the degree of sun exposure and the daily intake of vitamin D and calcium were obtained by self-report and might not reflect the true exposure or intake.

Supplementation with vitamin D results in a significant increase in bone mineral density and a reduction in fracture rates (35). Although the prevalence of vitamin D deficiency among ambulatory adults living in south Florida is considerable, it is commonly overlooked in the evaluation and treatment of postmenopausal and male osteoporosis. Even in this southern region of year-round sunny and warm weather, health-care providers assessing patients for osteoporosis should consider vitamin D deficiency as a possible and reversible risk factor contributing to bone loss, even in those subjects taking small doses of vitamin supplements.

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