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Diet, Environmental Factors, and Lifestyle Underlie the High Prevalence of Vitamin D Deficiency in Healthy Adults in Scotland, and Supplementation Reduces the Proportion That Are Severely Deficient1–3

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Abstract

Vitamin D deficiency has recently been implicated as a possible risk factor in the etiology of numerous diseases, including nonskeletal conditions. In humans, skin synthesis following exposure to UVB is a potent source of vitamin D, but in regions with low UVB, individuals are at risk of vitamin D deficiency. Our objectives were to describe the prevalence of vitamin D deficiency and to investigate determinants of plasma 25-hydroxyvitamin D (25-OHD) concentrations in a high northern latitude country. Detailed dietary, lifestyle, and demographic data were collected for 2235 healthy adults (21–82 y) from Scotland. Plasma 25-OHD was measured by liquid chromatography-tandem MS. Among study participants, 34.5% were severely deficient (25-OHD ≤ 25 nmol/L) and 28.9% were at high risk of deficiency (25–40 nmol/L). Only 36.6% of participants were at low risk of vitamin D deficiency or had adequate levels (≥ 40 nmol/L). Among participants who were taking supplements, 21.3% had a May-standardized 25-OHD concentration ≥ 50 nmol/L, 54.2% had 25–50 nmol/L, and 24.5% had < 25 nmol/L, whereas this was 15.6, 43.3, and 41%, respectively, among those who did not take supplements (P < 0.0001). The most important sources of vitamin D were supplements and fish consumption. Vitamin D deficiency in Scotland is highly prevalent due to a combination of insufficient exposure to UVB and insufficient dietary intake. Higher dietary vitamin D intake modestly improved the plasma 25-OHD concentration (P = 0.02) and reduced the proportion of severely deficient individuals (P < 0.0001). In regions with low UVB exposure, dietary and supplement intake may be much more important than previously thought and consideration should be given to increasing the current recommended dietary allowance of 0–10 μg/d for adults in Scotland. J. Nutr. 141: 1535–1542, 2011.

Introduction

In recent years, there has been an increasing awareness of the possible negative health impacts of vitamin D deficiency. Previ-

1 Supported by grants from Cancer Research UK (C348/A2758, C348/A8896), the Scottish Government Chief Scientist Office (KOPR/2/2/D333, C2B/4/04); Medical Research Council (G0000657-53203), and a Centre Grant from CORE as part of the Digestive Cancer Campaign (http://www.corecharity.org.uk). E.T. is funded by Cancer Research UK Fellowship C31250/A10107.


3 Supplemental Tables 1–3 are available from the “Online Supporting Material” link in the online posting of the article and from the same link in the online table of contents at Journalofnutrition.org.

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5 These two authors contributed equally to this work.

6 Deceased.


individual with skin type III 12 would need to spend ~3–8 min in the sun at noon, with 25% of the body surface area exposed in Boston (42°N) or 3–6 min in Miami (26°N) (9). Alternatively, vitamin D can be obtained from dietary sources (1,7). Due to the scarcity of vitamin D in food, the typical diet in most populations is rarely a sufficient source. Therefore, the lack of skin synthesis is a serious shortcoming in maintaining adequate vitamin D status.

The Scientific Advisory Committee on Nutrition in Scotland put forward a Reference Nutrient Intake 13 for vitamin D of 10 μg/d for individuals over 65 y and zero for everyone else (excluding young children and pregnant and lactating women) (11). However, the Institute of Medicine 2011 report on dietary reference intakes proposed an RDA of vitamin D of 15 μg/d for ages 1–70 y and 20 μg/d for older persons and an Estimated Average Requirement 14 of 10 μg/d. This recommendation is based on conditions of minimal sun exposure.

Due to the high northern latitude of Scotland (53° to 59° North) and the prevalent cloudy weather, conditions rarely facilitate vitamin D synthesis (12). In addition, an indoor-oriented lifestyle, obesity, high alcohol intake, and poor diet adversely affect vitamin D status (13,14). As expected, in a nation-wide cohort of British adults, participants from Scotland were identified as the most likely to suffer from vitamin D deficiency (15).

In this large study, which included 2235 Scottish adults, we estimated the prevalence of vitamin D deficiency in Scotland. The aim of this study was to establish the main risk factors for vitamin D deficiency that are related to lifestyle patterns and dietary factors and to identify groups most at risk of vitamin D deficiency. This study attempted to recognize the major sources of vitamin D and clarify the role of dietary intake in general and supplements in particular on 25-hydroxvitamin D (25-OHD) concentration at a time when vitamin D supplements are receiving increased attention as studies show the beneficial effect of vitamin D supplements on health and overall mortality (2,16). To our knowledge, this is one of the largest studies to date to gather detailed demographic, lifestyle, and dietary data in a high latitude country.

Participants and Methods

The study population comprised 2235 healthy adults identified through the Community Health Index and invited by their general practitioner to take part as controls in a national case control study of colorectal cancer in Scotland. This study has been described in detail elsewhere (17). All participants gave written informed consent and were recruited in the period from 1999 to 2006. The participation rate among those invited to take part in the study was ~57%. More than 99% of all recruited individuals were white Caucasian. Approval for the study was obtained from the MultiCentre Research Ethics Committee in Scotland and the local Research Ethics Committee.

Data collection and samples. Participants completed a lifestyle questionnaire, a semiquantitative FFQ (Scottish Collaborative Group FFQ, version 6.41), and a supplements intake questionnaire (18). The lifestyle questionnaire inquired about outdoor activities: walking; cycling; gardening; and jogging, swimming, fitness, or other aerobic exercise. These were recorded separately for winter and summer months and used as an estimate of the total number of hours spent outdoors per week as a proxy measure for sun exposure. The FFQ was used to calculate each participant’s dietary intake of food items and nutrients and to calculate the mean contribution of each food group to a participant’s dietary vitamin D intake (data available for 2056 participants). The main characteristics and validity of the FFQ were previously reported (19,20).

Using an in-house calculation program (21), nutrients were calculated from the consumption frequencies of specified portion size for each food item from the FFQ and were weighted for total energy intake. Participants were also asked to give details about supplements taken and nutrient information was collected from the manufacturer’s product information.

Area deprivation scores were estimated from the postcodes of the participants (Carstairs deprivation index) based on 2001 Census data and took values from 1 (very low deprivation) to 7 (very high deprivation).

Blood was collected from all participants and transferred to the research center within 72 h of sampling. Plasma was prepared by gentle centrifugation of sodium EDTA tubes and 1.5 mL of each participant’s plasma was stored at −80°C.

Measurement of plasma 25-OHD. The liquid chromatography-tandem MS (LC-MS/MS) method was used to measure 25-hydroxyergocalciferol and 25-hydroxycholecalciferol and the total of the 2 was used; however, most of our samples contained no 25-hydroxyergocalciferol. The lower limit of detection with the LC-MS/MS method was 10 nmol/L for 25-hydroxycholecalciferol (22). The LC-MS/MS method was performed following standard protocols and appropriate quality control procedures, including multiple measurements of the same sample from our cohort and standardization against standard reference material (SRM 972). Serum pools distributed by four Vitamin D External Quality Assessment Scheme distributions in 2010 (n = 20) were analyzed using the LC-MS/MS method with a mean negative bias of 10% compared with the method mean and 15% compared to the all laboratory trimmed mean. This method has been rated as the preferred 25-OHD measurement method for population studies by an international panel of experts (22,23). More details can be found elsewhere (22,24).

Vitamin D deficiency is defined in terms of circulating 25-OHD, which is a reliable marker of vitamin D status (25). Based on the evidence, the Institute of Medicine 2011 report judged that nearly all individuals (97.5%) meet their needs when their plasma 25-OHD concentration is >50 nmol/L and this corresponds to the RDA (the vitamin D intake that satisfies the needs of nearly everybody) (26). Participants with 25-OHD >50 nmol/L were considered to have an adequate 25-OHD concentration. On the other hand, we regarded individuals with 25-OHD <25 nmol/L as severely deficient in concordance with other recent studies (7,27).

The risk of deficiency increases gradually as 25-OHD concentration decreases from 50 nmol/L (adequate) to 25 nmol/L (severely deficient). The concentration of 40 nmol/L is associated with Estimated Average Requirement, a value that represents the median of the required intake in the population. Approximately one-half of individuals will have their needs met at this concentration; individuals below this cutoff (40 nmol/L) are more likely to be vitamin D deficient and those above are less likely to be deficient. Therefore, we consider individuals with a 25-OHD concentration between 25 and 40 nmol/L to be at high risk of deficiency and those with 40–50 nmol/L at low risk of deficiency.

Statistical analysis. For the analysis of factors affecting vitamin D status, 25-OHD measurements were standardized to the month of May to remove the prominent effect of the sampling month on the 25-OHD concentration. May-standardized values are free of the sampling month effect and represent the expected values for the sample as if taken in May. Mean values in May (33.2 nmol/L) are close to the yearly mean (mean of monthly means is 36.7 nmol/L) and can therefore be taken as a proxy of the individual’s yearly mean of 25-OHD in plasma. Our dataset was imbalanced due to different numbers of participants sampled in each calendar month. Therefore, the dataset was balanced to calculate corrected percentages in each category of vitamin D status, which is equivalent to having sampled the same number of participants in each month.

Values in the text are presented as mean ± SD. Most of the statistical analysis was done using R software version 2.6.1 (Hornik (2009), The R

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12 On the Fitzpatrick Scale, skin type III is beige in color, sometimes mildly burns, and tans gradually.

13 RDA is the amount that will cover the needs of 97.5% of the population. It is defined as the level of nutrient required, which is two standard deviations above the Estimated Average Requirement: assuming a normal population distribution, this covers the needs of at least 97.5% of the population and is therefore more than most people will actually need. This is equivalent to UK Reference Nutrient Intake (10).

14 Estimated Average Requirement (EAR) is the average requirement for a nutrient by a particular group of people. Individual requirements will vary from this mean, some people requiring more and others less than EAR (10).
We investigated plasma 25-OHD concentrations in 2235 healthy individuals (988 females) from Scotland aged 21–82 y (61.3 ± 10.5 y). The plasma 25-OHD concentration measured in our samples was 35.9 ± 22.3 nmol/L (Supplemental Table 1), almost identical to the mean of the balanced dataset (35.0 ± 22.1 nmol/L).

The most striking finding was the very high prevalence of severely deficient individuals (34.5%) and of those at high risk of vitamin D deficiency (28.9%). Extremely low concentrations (<12.5 nmol/L) were measured in 264 participants (11.8%).

The distribution of vitamin D sufficiency categories for each month is presented in Figure 1 and Supplemental Table 1. Sample month was strongly associated with 25-OHD concentration (P < 0.0001) (Table 1). The proportion of individuals who were severely deficient or at high risk of vitamin D deficiency (25-OHD <40 nmol/L) was extremely high throughout the year. From December to May, 69–83% were deficient, but an improvement occurred from June to November, when the prevalence was 33–69% (P < 0.0001).

Plasma 25-OHD concentration was higher in younger (≤61.3 y) participants (P = 0.002) and in participants with lower BMI (≤26.8) (P < 0.0001) (Table 2), but there was no difference between males and females (P = 0.49). Plasma 25-OHD concentrations were affected by participants’ vitamin D intake from food (P = 0.005), supplements (P < 0.0001), and overall (P = 0.02). Participants who consumed >0.9 fish servings/d had a higher plasma 25-OHD concentration than those who consumed less (P = 0.006).

The estimated total time spent on outdoor activities (h/wk) was strongly associated with plasma 25-OHD concentrations (P < 0.001). The strong association remained for each activity (walking, cycling, physical exercise, and gardening) when independently assessed both in summer and winter months (P < 0.03), with the exception of gardening in winter months (P = 0.14).

Plasma 25-OHD variance explained by total vitamin D intake and outdoor activity was independently calculated for winter (November to April) and summer months (May to October) and therefore, actual, nonstandardized measurements of plasma 25-OHD were used in this analysis.

Results

We investigated plasma 25-OHD concentrations in 2235 healthy individuals in Scotland aged 21–82 y (61.3 ± 10.5 y). The plasma 25-OHD concentration measured in our samples was 35.9 ± 22.3 nmol/L (Supplemental Table 1), almost identical to the mean of the balanced dataset (35.0 ± 22.1 nmol/L).

Next, participants were grouped into quartiles according to the total intake of vitamin D and further subdivided according to age in 4 age groups (<50, 50–60, 60–70, and >70 y). The number in each group, mean vitamin D intake, median plasma 25-OHD concentration, and percent of severely deficient individuals was assessed for each subgroup, by total vitamin D intake, and by gender.

Plasma 25-OHD variance explained by total vitamin D intake and outdoor activity was independently calculated for winter (November to April) and summer months (May to October) and therefore, actual, nonstandardized measurements of plasma 25-OHD were used in this analysis.

To illustrate the differences in plasma 25-OHD in a low- and high-risk environment, participants were divided into quartiles according to the outdoor activities and subdivided further according to daily supplement intake: none (<1 µg/d), low intake (1–5 µg/d), medium intake (5–10 µg/d), and high intake (>10 µg/d). The same was repeated by subdividing the groups according to only food and total (from food and supplements) vitamin D intake quartiles.

Next, participants were grouped into quartiles according to the total intake of vitamin D and further subdivided according to age in 4 age groups (<50, 50–60, 60–70, and >70 y). The number in each group, mean vitamin D intake, median plasma 25-OHD concentration, and percent of severely deficient individuals was assessed for each subgroup, by total vitamin D intake, and by gender.

Plasma 25-OHD variance explained by total vitamin D intake and outdoor activity was independently calculated for winter (November to April) and summer months (May to October) and therefore, actual, nonstandardized measurements of plasma 25-OHD were used in this analysis.

FIGURE 1 Monthly 25-OHD concentration distribution in adults in Scotland (n = 2235). Nonstandardized 25-OHD measurements were used.
in the lowest outdoor activity quartile (<7 h/wk), the percent of severely deficient individuals decreased with increasing supplementation level. Among those who took no supplements, 49.6% were severely deficient, followed by 34.4, 23.4, and 14.3% among those taking <5, 5–10, and >10 µg/d, respectively.

An analysis of total vitamin D intake by gender and age showed that men (9.8 ± 5.3 µg/d) had a higher intake than women (8.1 ± 4.8 µg/d) (P < 0.0001) (Table 5). Plasma 25-OHD concentrations were higher and the percent of severely deficient participants was lower in younger adult age groups of both genders. The percent that were severely deficient and the median plasma concentration of 25-OHD by vitamin D intake and age are shown in Table 5.

For plasma 25-OHD, the variance explained by total vitamin D intake was 5.05% and by outdoor activity was 0.24% in the quartile and age are shown in Table 5.

TABLE 1 Plasma 25-OHD concentrations in adults in Scotland1

<table>
<thead>
<tr>
<th>Month</th>
<th>n</th>
<th>Plasma 25-OHD, nmol/L</th>
<th>n %</th>
<th>Low risk &gt;40 and &lt;50 nmol/L</th>
<th>n %</th>
<th>High risk &gt;25 and &lt;40 nmol/L</th>
<th>n %</th>
<th>Deficient &lt;25 nmol/L</th>
<th>n %</th>
</tr>
</thead>
<tbody>
<tr>
<td>January</td>
<td>139</td>
<td>26.9 ± 16.4</td>
<td>12</td>
<td>8.6</td>
<td>46</td>
<td>33.1</td>
<td>69</td>
<td>49.6</td>
<td></td>
</tr>
<tr>
<td>August</td>
<td>216</td>
<td>51.9 ± 26.1</td>
<td>109</td>
<td>50.5</td>
<td>41</td>
<td>19.0</td>
<td>31</td>
<td>14.4</td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>2235</td>
<td>35.9 ± 22.3</td>
<td>503</td>
<td>22.5</td>
<td>646</td>
<td>28.9</td>
<td>772</td>
<td>34.5</td>
<td></td>
</tr>
</tbody>
</table>

1 Nonstandardized 25-OHD concentrations were used.
2 Months with the year’s lowest and year’s highest mean plasma 25-OHD concentration, respectively.

Discussion

A number of different 25-OHD thresholds to define categories of vitamin D deficiency have been published, but a final consensus on what plasma 25-OHD concentration is required for optimal health, skeletal and extra-skeletal, has not yet been reached (12,15,29–31). The Institute of Medicine 2011 report, based on available evidence, judged that 97.5% of all individuals meet their needs when their 25-OHD concentration is >50 nmol/L, whereas a concentration of 40 nmol/L is associated with the median requirement in the population (26). In a national sample, 63.4% of individuals had a 25-OHD concentration <40nmol/L and were at high risk of mild or severe vitamin D deficiency. Only 22.5% of the participants could be classified as having an adequate vitamin D status (25-OHD >50 nmol/L) based upon IOM guidelines.

Bone mineral density decreases with decreasing concentrations of 25-OHD in plasma (31) and osteomalacia and rickets can arise from low concentrations of plasma 25-OHD (22). A
concentration of 25 nmol/L is commonly used as the threshold and individuals with lower 25-OHD are at risk of skeletal problems and are regarded as severely deficient; for those, treatment with high doses of vitamin D should be considered (7,27). As many as one-third of adults in our sample were severely vitamin D deficient.

In Scotland, skin synthesis, normally a potent source of vitamin D, is greatly impaired due to the very low yearly quota of solar radiation (32) and a large additional loss of UVB in the atmosphere given the high latitude (33,34). Indeed, not even summer sunlight can produce optimal plasma 25-OHD concentrations at UK latitudes (12). These adverse environmental factors coupled with typical indoor lifestyle patterns contribute to the epidemic proportions of vitamin D deficiency in Scotland.

Epidemic hypovitaminosis D in Scotland has been previously reported. However, our study revealed an even higher proportion of vitamin D-deficient individuals. From May to November, Hyppönen et al. (15) reported 8.3, 27.5, and 74.9% of individuals with 25-OHD <25, 40, and 75 nmol/L, respectively, whereas we observed 25.2, 53.7, and 92.4%, respectively. The disparity is likely due to the age difference; their cohort included exclusively 45-y-old individuals, whereas the median age in our cohort was 62 y. It is apparent that levels in the Scottish population are very low relative to other populations, such as in Italy or France (35–37).

There are increasing reports of associations between low vitamin D levels and a variety of diseases of public health importance such as cancer (5,6), cardiovascular disease (2,3), and diabetes (4), as well as with increased total mortality rates (2,16). A recent study in the Scottish population (75 y and older) showed that vitamin D status at baseline was inversely related to mortality (38). Although it has not been proven that these associations are causal in nature, it is clear that this merits further research attention. If these associations are causal, then preventing vitamin D deficiency could have the potential to reduce the disease and health care burden from these conditions and this would also have to be considered when defining “optimal” vitamin D status (39).

Factors associated with 25-OHD. We observed a significant association between total dietary vitamin D intake and vitamin D status. The number of individuals who had higher levels of vitamin D intake was too low to attempt to define a daily intake that could maintain a sufficient vitamin D status in Scotland. In our study, supplement intake was significantly associated with an individual’s plasma 25-OHD and contributed most to the total dietary vitamin D intake. Fish intake was the single most important food source of vitamin D, in contrast to the US and Canada, where fortified dairy products contribute the most.

When assessed together, the absolute level of increase in median plasma 25-OHD and the decrease in the proportion of severely deficient individuals were greater across observed levels of supplementation than across quartiles of outdoor activity. The effect of supplements was greatest in the lowest outdoor activity quartile, emphasizing the value of supplementation for individuals deprived of skin synthesis. With increasing total vitamin D intake, the largest reduction in severe deficiency occurred in the older age groups.

### TABLE 3

<table>
<thead>
<tr>
<th>25-OHD</th>
<th>Vitamin D supplements</th>
<th>Total (from food and supplements) vitamin D intake &gt;5 μg/d</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
</tr>
<tr>
<td>&gt;50 nmol/L</td>
<td>243</td>
<td>15.6</td>
</tr>
<tr>
<td>&gt;25 and ≤50 nmol/L</td>
<td>673</td>
<td>43.3</td>
</tr>
<tr>
<td>&gt;12.5 and ≤25 nmol/L</td>
<td>471</td>
<td>30.3</td>
</tr>
<tr>
<td>≤12.5 nmol/L</td>
<td>167</td>
<td>10.7</td>
</tr>
<tr>
<td>Total</td>
<td>1554</td>
<td>74.4</td>
</tr>
</tbody>
</table>

1 The total number of participants with 25-OHD and supplement intake data available was 2089. For 44 participants, information on vitamin D intake from food was not available.
Unlike in the US or Canada, vitamin D food fortification in Scotland is currently optional, except fortification of margarine, which is required (7.5–10 μg vitamin D/100 g end product). In recent years, a selection of vitamin D-fortified products, primarily milk, yogurt, orange juice, and breakfast cereals, became available on the market, but the amount added is highly variable. Current measures of fortification and dietary recommendations of 10 μg/d of vitamin D for individuals over 65 y and zero for everyone else (excluding children and pregnant or lactating women) do not seem to prevent hypovitaminosis D in Scotland, because very few individuals achieve the recommended 25-OHD concentration.40 nmol/L.

A growing body of evidence suggests that even higher supplement doses of vitamin D (20–30 μg/d) are safe and well tolerated (40). In the IOM 2011 report, the upper limit for vitamin D intake was set at 100 μg/d for individuals over the age of 14 y. Even doses of 250 μg/d given to breast cancer patients for 4 mo were reported to be safe (41). Our study found a high prevalence of deficiency even among participants in the highest quartile of total vitamin D intake and therefore supports calls for a higher RDA for vitamin D and for the consideration of food fortification. The RDA of 600–800 IU/d (15–20 μg/d) is required to achieve a plasma 25-OHD concentration of 50 nmol/L and is based on the assumption that there is no contribution from skin synthesis (26). This may also be applicable to Scotland given the complete lack of UVB during many months and low synthesis even in the summer months due to the weather. In addition, because food fortification with vitamin D in Scotland is not mandatory, individuals living in Scotland might be getting less vitamin D from food than those living in the US or Canada.

**Strengths and limitations of the study.** Plasma 25-OHD concentrations are very stable and unlikely to be affected by time in transport to research centers or low temperature storage (42). Analysis of multiple measurements of the same sample from our cohort was in agreement with laboratory internal quality control data. Because it is very difficult to measure diet, some measurement errors in FFQ-derived vitamin D intakes are expected. The correlation coefficients between FFQ-derived measures of vitamin D intake and weighted dietary record measurements for vitamin D were 0.51 for men and 0.39 for women (19). When using outdoor activity as a proxy for sun exposure, measurement error could arise from the fact that vitamin D synthesis depends strongly on numerous factors, such as atmospheric conditions and time of day, and also some of these activities may be performed indoors. Persons who agreed to participate might have had a

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**FIGURE 3** Mean contributions of different food products and supplements to daily vitamin D intake in adults in Scotland (n = 2056). The contribution of supplements was calculated for the 527 individuals who take >1 μg/d of vitamin D from supplements.

**FIGURE 4** Proportion of severely deficient individuals with plasma 25-OHD <25 nmol/L according to the outdoor activity and levels of supplement intake.

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**TABLE 4** Plasma 25-OHD concentrations in adults in Scotland by level of supplemental vitamin D intake and quartile of outdoor activity.1,2

<table>
<thead>
<tr>
<th>Supplemental vitamin D intake</th>
<th>Quartiles of outdoor activity, h/wk</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>25-OHD median, nmol/L</td>
</tr>
<tr>
<td>&lt;1 μg/d</td>
<td>n=385</td>
</tr>
<tr>
<td></td>
<td>381</td>
</tr>
<tr>
<td></td>
<td>371</td>
</tr>
<tr>
<td></td>
<td>361</td>
</tr>
<tr>
<td>1–5 μg/d</td>
<td>n=32</td>
</tr>
<tr>
<td></td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>40</td>
</tr>
<tr>
<td></td>
<td>31</td>
</tr>
<tr>
<td>≥5 and &lt;10 μg/d</td>
<td>n=64</td>
</tr>
<tr>
<td></td>
<td>73</td>
</tr>
<tr>
<td></td>
<td>78</td>
</tr>
<tr>
<td></td>
<td>79</td>
</tr>
<tr>
<td>≥10 μg/d</td>
<td>n=21</td>
</tr>
<tr>
<td></td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>32</td>
</tr>
</tbody>
</table>

1 May-standardized values of 25-OHD were used.  
2 In total, 245 participants were excluded from this analysis due to missing data regarding outdoor activity and supplements intake.

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15 Upper limit indicates a level above which there is risk of adverse events.
In view of the limited opportunity for the production of vitamin D in skin and restricted dietary vitamin D intake, most Scottish adults appear to be unable to attain an adequate vitamin D status. Although increasing vitamin D intake from food and supplements significantly but modestly increased the plasma 25-OHD concentration, the decrease in the proportion of severely deficient individuals is notable and important.

Given the epidemic proportions of vitamin D deficiency, the current RDA for vitamin D seems to be insufficient for Scotland and this is reflected in the high proportion of deficient individuals, even among those whose dietary intake is more than the current RDA. Because these results might reflect the situation in other regions with low UVB radiation, such as other high latitude countries (43), our results suggest that the recommended daily vitamin D intake in countries with low UVB exposure should be reviewed.

Acknowledgments

L.Z. and E.T. analyzed the data and wrote the manuscript; F.A., K.J., and G.M. analyzed the data; H.C. designed the study and wrote the manuscript; M.D. designed the study; S.K., S.F., R.C., M.W., H.C., M.D., and M.P. collected the data; and G.M., K.J., S.K., and A.M.W. provided consultation in their areas of expertise. All authors read and approved the final manuscript.

Literature Cited

10. In view of the limited opportunity for the production of vitamin D in skin and restricted dietary vitamin D intake, most Scottish adults appear to be unable to attain an adequate vitamin D status. Although increasing vitamin D intake from food and supplements significantly but modestly increased the plasma 25-OHD concentration, the decrease in the proportion of severely deficient individuals is notable and important.

Given the epidemic proportions of vitamin D deficiency, the current RDA for vitamin D seems to be insufficient for Scotland and this is reflected in the high proportion of deficient individuals, even among those whose dietary intake is more than the current RDA. Because these results might reflect the situation in other regions with low UVB radiation, such as other high latitude countries (43), our results suggest that the recommended daily vitamin D intake in countries with low UVB exposure should be reviewed.

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L.Z. and E.T. analyzed the data and wrote the manuscript; F.A., K.J., and G.M. analyzed the data; H.C. designed the study and wrote the manuscript; M.D. designed the study; S.K., S.F., R.C., M.W., H.C., M.D., and M.P. collected the data; and G.M., K.J., S.K., and A.M.W. provided consultation in their areas of expertise. All authors read and approved the final manuscript.

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