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Vitamin D status of middle-aged women at 65–71°N in relation to dietary intake and exposure to ultraviolet radiation

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Abstract

Objective: To determine the vitamin D status of middle-aged women living in the Norwegian arctic and its relationship with vitamin D intake and exposure to ultraviolet (UV) radiation.

Design: Cross-sectional study.

Subjects and setting: This study is based on measurements of 25-hydroxyvitamin D (25(OH)D) levels in a sub-sample of the Norwegian component of the EPIC biological bank, which consists of blood samples from a random selection of participants in the Norwegian Women and Cancer Study. From November 2001 until June 2002, 309 blood samples were collected from a total of 443 invited middle-aged women (44–59 years) in northern Norway (65–71°N) (crude response rate, 69.8%). Questionnaire data provided information on dietary sources of vitamin D and UV exposure.

Results: Median plasma 25(OH)D concentration for the whole group was 55.0 nmol l^{-1} (range 8.1–142.8 nmol l^{-1}). Vitamin D intake was a significant predictor of 25(OH)D status ($P = 0.0003$). The time of the year when the blood sample was collected significantly predicted plasma 25(OH)D level ($P = 0.005$). Levels of 25(OH)D were positively associated ($P = 0.0002$) with estimated hours per day of exposure to UV-B radiation. Residing in northern Norway during the summer prior to blood sampling was negatively associated with 25(OH)D concentration ($P = 0.001$). The prevalence of moderate hypovitaminosis D was highest in January–February, when a quarter of the participants had 25(OH)D concentrations ≤ 37.5 nmol l^{-1}.

Conclusions: Increased ingestion of marine food items that provide vitamin D should be promoted and further studies should be carried out to investigate vitamin D status in arctic populations in relation to both UV exposure and traditional food sources.

Keywords
Vitamin D status
UV-B exposure
Arctic diet
Arctic health
'Vitamin D winter'

Vitamin D is essential for natural bone metabolism and for calcium and phosphorus homeostasis[1]. It is obtained either from the skin upon exposure to ultraviolet (UV) radiation or through dietary sources. Fatty fish, fish liver, cod-liver oil, egg yolk and mushrooms contain vitamin D, as do fortified foods like milk, butter and margarine. Since natural sources of vitamin D are limited, UV exposure has long been recognised as the principal source of vitamin D_3 for humans[2]. However, dramatic influences of seasonal and latitudinal changes of solar UV-B radiation on vitamin D_3 synthesis have been demonstrated by Webb *et al.*[3]. Below a certain threshold of UV radiation, corresponding to cloudless conditions in Boston, USA (43°N) in mid-February, no photoconversion of provitamin D to previtamin D was detected[3]. Because of this seasonal variation in UV-B radiation, cutaneous vitamin D production is absent during part of the winter and the length of the 'vitamin D winter' increases with latitude.

In northern Norway (65–71°N) the sun is below the horizon for up to 2 months during the winter. Owing to this high latitude and limited exposure to sunshine, the population living in this area is likely to be dependent on dietary sources of vitamin D to meet their biological needs during a considerable part of the year. Historically, high amounts of fish liver and fresh fish-liver oil were consumed among people who lived in the northern coastal areas of Norway[4]. The fish liver eaten was mainly from cod and saithe, and consumption followed their seasonal harvests. The season for cod liver was connected with the spawning time, starting around mid-December and lasting until April. Saithe liver was consumed from late summer until September/October. Studies from the first part of the 20th century showed that this food, which is rich in fat-soluble vitamins, constituted the most important source of vitamin D and prevented rickets in the coastal population[5]. Even though there still are areas where fish

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liver is consumed frequently[4], fatty fish and fortified margarine are today considered the main dietary sources of vitamin D in the Norwegian population[6].

In recent years some epidemiological studies have found high prevalences of hypovitaminosis in the Nordic countries[7–10]. Only two studies have examined determinants for vitamin D status in northern Norway[11,12] and both were based on small sample sizes (17 and seven subjects, respectively). To our knowledge, no previous population-based assessment of vitamin D status with random sampling, where both UV exposure and intake have been accounted for, has been conducted in arctic regions of Norway or corresponding latitudes.

Circulating 25-hydroxyvitamin D (25(OH)D) is considered the most sensitive clinical marker for determining nutritional vitamin D status[13]. A plasma 25(OH)D concentration of $\leq 37.5\,\text{nmol}\,\text{l}^{-1}$ has been used as an indicator for moderate hypovitaminosis D[14] and 25(OH)D concentration $>50\,\text{nmol}\,\text{l}^{-1}$ has been suggested as the recommended level[15,16].

The aim of this study was to investigate the vitamin D status of middle-aged women living in northern Norway (65–71°N) by determining 25(OH)D levels in blood plasma and assessing its relationship with vitamin D intake and UV exposure.

Method

Study sample

Subjects were recruited from the northern contingent of the Norwegian EPIC (European Prospective Investigation into Cancer and Nutrition) cohort, which has been described in detail elsewhere[17]. In short, the Norwegian EPIC cohort is a sub-sample of the nation-wide Norwegian Women and Cancer Study (NOWAC) consisting of 37 231 participants who, in 1991/1992 and 1998, answered and returned questionnaires regarding lifestyle and health. By random selection, 20 696 of these women were contacted by mail and asked to give blood samples to be included in the EPIC biological bank. These requests were sent out in batches from October 2000 until June 2002. During the period from November 2001 to June 2002, all participating women living in northern Norway were also asked to give an additional blood specimen for vitamin D determination. The blood specimens were collected at local health centres into vacutainer tubes 4.5 ml blue top; Becton-Dickinson, Plymouth, UK containing sodium citrate (3.2%) and returned by mail to the University of Tromsø, northern Norway. A total of 443 subjects were contacted, of whom 309 responded (response rate, 69.8%). Nine of these blood samples could not be used for analysis, leaving 300 subjects for this study.

Questionnaire

The participating subjects answered a two-page questionnaire covering various aspects of UV exposure including solarium use and dietary sources of vitamin D, including the consumption of cod and saithe liver, fresh fish-liver oil, vitamin D-fortified milk and butter/margarine, cod-liver oil supplements, dietary supplements and fatty fish. The questions on fatty fish and cod-liver oil supplements were equal to the questions applied in the NOWAC questionnaire, which has been evaluated and showed a significant correlation between data on $n-3$ fatty acids intake and phospholipid $n-3$ fatty acids in serum[18]. In the questions that focused on UV exposure, participants were asked to recall the number of hours they had spent in daylight last week (the week prior to collection of the blood sample), whether they had resided in northern Norway during the whole of summer 2001 (June–August, the summer prior to collection of the blood sample), whether they had been on a sun holiday abroad since September 2001, and whether they had used a solarium during the past month. In the questions regarding fish liver, the participants were asked to recall how often they had consumed cod and saithe liver separately during the 'fish-liver season'.

Determination of 25-hydroxyvitamin D

Blood plasma was collected and kept at $-80\,^{\circ}\text{C}$ until analysis for 25(OH)D according to a modified version of the method described by Aksnes[19]. Briefly, 0.25 ml plasma samples were spiked with ^{3}H-25(OH)D_3 (for calculation of recovery), and 25(OH)D was extracted with methanol and n-hexane. The n-hexane phase was collected, evaporated to dryness and injected into a reverse-phase high-performance liquid chromatography system. Elution of 25(OH)D was performed with methanol/water (85:15, v/v) and the eluate was monitored at 265 nm by a diode-array detector (UV6000; ThermoFinnigan, San Jose, CA, USA) equipped with a 5 cm detector cuvette. The mean recovery of 25(OH)D was 77.2% (standard deviation (SD) 3.9%) and the interassay variation was 6%, with a detection limit of $6.0\,\text{nmol}\,\text{l}^{-1}$. The method determined the sum of 25(OH)D_2 and 25(OH)D_3.

Biological effective UV dose rate

Based on the work by Webb *et al.*[3] and MacLaughlin *et al.*[20], we established a biological effective UV dose rate (BED) for photoconversion of 7-dehydrocholesterol (7-DHC) to previtamin D in skin. Webb *et al.*[3] found no detectable photoconversion of 7-DHC to previtamin D for mid-February and a small production of previtamin D at mid-March. For mid-February in Boston USA, they measured surface irradiances of 0.024, 1.0 and $10\,\text{mW}\,\text{m}^{-2}\,\text{nm}^{-1}$ with an Optronics 742 spectroradiometer at wavelengths of 300, 306 and 316 nm, respectively. The BED was established by adding the measured surface irradiances weighted by the relative efficiencies for converting 7-DHC to previtamin D_3. The weights are 0.92, 0.45 and 0 for 300, 306 and 316 nm, respectively[3,20]. Below the threshold $\text{BED}_{\text{threshold}} = 0.024 \times 0.92 + 1.0 \times 0.45 = 0.472$

we assumed that no photoconversion to previtamin D took place. For each day in the period November 2001 to June 2002, we simulated the maximum BED and the amount of time the BED exceeded the UV radiation threshold for vitamin D production described above for all municipalities in northern Norway. We applied the fast yet accurate UV simulation tool FastRT to simulate surface irradiances at 300 and 306 nm as would be measured by a spectroradiometer with an Optronics 742 spectral response function (http://zardoz.nilu.no/~olaeng/fastrt/fastrt.html). The ozone layer thickness was fixed at a typical level (300 Dobson Units). Clouds, altitude and snow were not accounted for in the simulations.

Statistical analysis

SAS version 8.02 (SAS Institute, Cary NC, USA) was used for nutrient calculations and statistical analysis. To assess predictors for plasma 25(OH)D level, general linear models were used. In these analyses the dependent variable 25(OH)D was log-transformed since it was not normally distributed. Daily vitamin D intake was calculated based on the Norwegian Food Composition Table[21] and on vitamin D determination of boiled fish liver and fresh fish-liver oil[4]. Dietary supplements, except for cod-liver oil, were not included in the estimation of daily vitamin D intake owing to limited information on nutrient content in the various products used, but instead were treated as a dichotomous variable. Subjects who reported that they had been on a sun holiday abroad or had used a solarium were excluded from some of the analyses in order to focus on predictors for vitamin D security at high latitudes. A variable named 'UV-hours', representing hours per day with exposure to UV radiation above the threshold for vitamin D production in skin, was devised. It combined questionnaire data on reported hours spent in daylight the week prior to the blood sampling with information on total hours when the simulated BED was above 0.472 (estimated threshold value for photoconversion of vitamin D). For each woman, the quantity was identical to the reported hours spent in daylight the week prior to the blood sampling. However, the time was not allowed to exceed the total hours at which vitamin D production could occur at a participant's geographical location (municipality) in the week the blood sample was collected. For missing values on hours spent in daylight, the 'UV-hours' variable was treated as missing except for women whose blood samples were drawn when BED < 0.472. For the latter, the 'UV-hours' variable was coded as zero. The variable for time of the year when the blood specimen was collected was separated into four 2-month categories. In the season-stratified analysis, only three groups were examined, since November–February were collapsed into a single dark season. The model's ability to explain the variation in plasma 25(OH)D level is given by the adjusted coefficient of determination (R^2_{adj}, adjusted for the number of degrees of freedom). When selecting independent variables to be included in each statistical model, the variance inflation factor (VIF) was applied to determine if collinearity was present. In the statistical tests, the 5% significance level (two-sided) was chosen.

Results

Some characteristics of the study sample are summarised in Table 1. The participants' age ranged from 44 to 59 years. The 5th, 10th, 90th and 95th percentiles for daily vitamin D intake were 0.95, 1.6, 16.9 and 22.8 μg, respectively. The median vitamin D intake among subjects with 25(OH)D concentrations ≥ 37.5 and < 37.5 nmol l^{-1} was 6.8 and 3.2 μg day^{-1}, respectively (Table 2). The main dietary sources of vitamin D were salmon/trout (30.2%), cod-liver oil supplements (23.4%), margarine/butter on bread (23.0%), and fish liver and fresh fish-liver oil (9.4%). There was no significant difference in calculated vitamin D intake by calendar month the women were recruited into the study ($P = 0.46$, Kruskal–Wallis test).

For nearly half of the subjects (45.7%), the 'UV-hours' metric was zero. Among the women who participated in November–February, 23.8% had been on a sun holiday or had used a solarium; this was the case for 34.3% of the

Table 1 Descriptive characteristics of the study sample ($n = 300$)*

Characteristic	Mean (SD) or *n*	Median or %
Age (years)	51.6 (4.2)	51.6
Body mass index (kg m^{-2})	25.1 (3.8)	24.4
Dietary vitamin D intake (μg day^{-1})†	8.1 (7.0)	6.2
Hours in daylight last week prior to blood sampling	10.8 (11.8)	7.0
Latitude		
65–67°N	105	35
68–69°N	153	51
70–71°N	42	14
UV-hours (h day^{-1})		
0	127	46
0.1–1.0	48	17
1.1–2.0	44	16
2.1 +	59	21
Cod-liver oil supplement users (once per week or more)	120	40
Fish liver (cod or saithe) consumption (twice per season or more)	176	59
Sun holiday	51	17
Solarium use	45	15
Dietary supplements other than cod-liver oil	171	58
Residing in northern Norway during the previous summer	157	53

SD – standard deviation.
* Subgroups may not total to 300 due to missing values.
† Contribution to vitamin D from dietary supplements except cod-liver oil was not included.

Table 2 Selected characteristics of the study sample by plasma 25-hydroxyvitamin D (25(OH)D) concentration ($n = 300$)

Characteristic	Plasma 25(OH)D (nmol l^{-1})		*P*-value
	<37.5 ($n = 41$)	≥37.5 ($n = 259$)	
Age (years), mean	51.0	51.7	0.09
Body mass index (kg m^{-2}), mean	26.2	24.9	0.13
Dietary vitamin D intake (μg day^{-1}), median	3.2	6.8	<0.0001*
Hours in daylight last week prior to blood sampling, mean	8.2	11.3	0.08
Latitude (°N), mean	67.9	67.8	0.91
UV-hours (h day^{-1}), median	0.0	0.57	0.02*

* Wilcoxon two-sample test.

subjects enrolled in March–June. Only 2% of the subjects had been both to a solarium and on a sun holiday.

The median 25(OH)D concentration for the whole group was 55.0 (range 8.1–142.8) nmol l^{-1} (Table 3). The prevalence of subjects with 25(OH)D concentrations below 37.5 nmol l^{-1} was 13.7%. When excluding subjects who had been on a sun holiday abroad or to a solarium (90 subjects in total), 16.4% had 25(OH)D levels below 37.5 nmol l^{-1} and the mean plasma concentration of 25(OH)D dropped to 53.1 (SD 17.6) nmol l^{-1}, which is significantly lower ($P = 0.03$, Student's *t*-test) than the mean for the whole group. The median 25(OH)D concentration for the subjects with no sun holiday/solarium and no 'UV-hours' ($n = 97$) was 45.9 nmol l^{-1}. Table 3 also presents plasma 25(OH)D levels by different time intervals for the blood sampling. The prevalence of moderate hypovitaminosis D was highest in January–February, at 23.3%. In March–April the prevalence was around a third of the January–February level. When excluding subjects who had been to a solarium or on a sun holiday, in January–February about a quarter of the subjects had 25(OH)D concentrations below 37.5 nmol l^{-1} and almost two-thirds had levels below 50 nmol l^{-1}.

The adjusted mean values of 25(OH)D concentration in relation to daily duration of vitamin D-productive light ('UV-hours') increased up to 1.1–2.0 UV-hours per day and then seemed to level off (Fig. 1).

As denoted in Table 4, in the multiple linear regression model including all subjects significant predictors for variation in 25(OH)D level were age, dietary vitamin D intake, time of year when the blood sample was collected, sun holiday, solarium use and residing in northern Norway the summer prior to the blood sampling. When stratifying the analysis by subjects who had been on a sun holiday or to a solarium and those who had not, only age was significantly associated for the sun holiday/solarium use group ($P = 0.01$). For the no sun holiday/no solarium use group, dietary vitamin D intake, time of year when the blood sample was collected and residing in northern Norway during the previous summer were highly significant. In further analysis where subjects who had been on a sun holiday or used a solarium were excluded, dietary vitamin D intake was significantly associated with plasma 25(OH)D concentrations throughout the whole time period, but most significantly in November–February ($P = 0.006$) (Table 4). The variable 'residing in northern Norway during the previous summer' significantly predicted 25(OH)D levels in blood in the November–February strata ($P = 0.05$).

When the variables 'hours in daylight last week prior to blood sampling', 'latitude' and 'time of year when the blood sample was collected' (Table 4) were substituted by the variable 'UV-hours', the latter was strongly associated with plasma 25(OH)D concentration for both the whole group ($P = 0.0002$) and the no sun holiday/no solarium use stratum ($P = 0.0002$) (Table 5). However, for subjects who had been on a sun holiday or to a solarium, 'UV-hours' was not significantly associated ($P = 0.64$) with plasma 25(OH)D level. For the no sun holiday/no solarium use group, dietary intake of vitamin D as a

Table 3 Distribution of vitamin D measures in the study sample through the 8-month period ($n = 300$)

	All ($n = 300$)	November–December ($n = 36$)	January–February ($n = 86$)	March–April ($n = 111$)	May–June ($n = 67$)
25(OH)D concentration (nmol l^{-1})					
Mean (SD)	56.9 (18.8)	52.8 (16.0)	49.5 (15.6)	60.8 (18.4)	61.9 (21.5)
Median (range)	55.0 (8.1–142.8)	51.7 (22.6–100.1)	46.9 (26.2–98.3)	60.6 (8.1–125.4)	60.8 (22.6–142.8)
25(OH)D below 37.5 nmol l^{-1} (%)	13.7	13.9	23.3	8.1	10.5
Exclusion of subjects with solarium use or sun holiday	16.4	19.2	25.4	8.6	14.0
25(OH)D below 50.0 nmol l^{-1} (%)	38.0	44.4	58.1	24.3	31.3
Exclusion of subjects with solarium use or sun holiday	46.4	57.7	65.7	28.6	39.5

25(OH)D – 25-hydroxyvitamin D; SD – standard deviation.

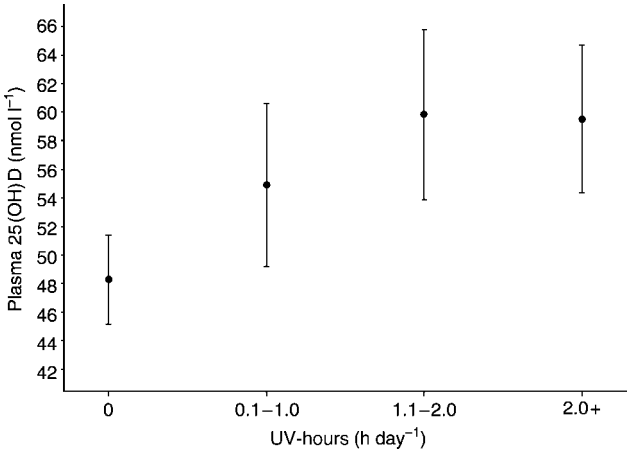

Fig. 1 Adjusted mean values (bars, 95% confidence interval) for plasma 25-hydroxyvitamin D (25(OH)D) by UV-hours (adjusted for age, body mass index, vitamin D intake, use of supplements and residing in northern Norway during the previous summer)

determinant of plasma vitamin D was highly significant when 'UV-hours' was used in the model ($P < 0.0001$). The same was found when looking at the subgroup of women with zero UV-hours and no sun holiday/no solarium use (data not shown).

To assess the association between plasma 25(OH)D and vitamin D contributions from fish-liver oil supplements and fish liver and fresh fish-liver oil separately, the variable 'total vitamin D intake' was split into three categories: 'vitamin D from cod-liver oil supplements', 'vitamin D from fish liver and fresh fish-liver oil' and 'vitamin D from all other sources'. These variables were then included as independent variables in the regression model to assess determinants for plasma 25(OH)D concentration together with age, body mass index, use of dietary supplements, UV-hours and whether or not residing in northern Norway the whole summer prior to blood sampling. This model explained 24% of the variance in plasma 25(OH)D concentration. Dietary vitamin D intake from fish liver and fresh fish-liver oil was positively associated with plasma 25(OH)D concentration, but of borderline significance ($P = 0.06$). The contributions from cod-liver oil supplements and from all other vitamin D sources were positively associated with 25(OH)D ($P = 0.0009$ and 0.02, respectively). (No collinearity between these three variables representing different vitamin D sources was found.)

Discussion

We found that the vitamin D status of subjects who stayed the entire year in northern Norway was dependent on dietary sources of vitamin D throughout the study period (November–June). The prevalence of moderate hypovitaminosis D was highest in January–February, when a quarter of the participants had 25(OH)D concentration $\leq 37.5\,\text{nmol}\,\text{l}^{-1}$. Subjects who had travelled to southern locations during the summer, prior to the blood sampling, had higher vitamin D levels in their blood compared with those who had remained in the north.

This present investigation has several interesting features. First, this study is the only randomised, population-based assessment of vitamin D status measured as plasma 25(OH)D concentrations among middle-aged women in northern Norway. Second, the participants' broad geographical distribution throughout all latitudes in the north made it possible to investigate vitamin D status in a population living at some of the most northerly geographical locations of significant human settlement. Third, the extreme variation in sun exposure found at these high latitudes could be taken into account because the blood samples were collected throughout the dark season when the sun was below the horizon for 24 hours a day, and until summer when the sun never set. Fourth, the focused questionnaire aimed to collect information on both UV radiation-related issues and dietary vitamin D intake, and thus provided detailed data on all sources of vitamin D. Furthermore, the time- and geographically specific simulated UV-radiation measure devised for northern Norway made it possible to develop the 'UV-hours' variable to explain plasma 25(OH)D concentration. This new variable, 'UV-hours' (Table 5), was considered a better metric than the previous 'hours in daylight last week prior to blood sampling', 'time of year when the blood sample was collected' and 'latitude' (Table 4).

It would have been desirable to have a greater number of subjects participating in the study in light of the seasonal variation in plasma vitamin D level, especially for the stratified analysis, which had relatively few subjects assigned to each stratum. The reason for this limited number was that, during the data sampling period (November 2001–June 2002), among the randomly selected women asked to give blood for the Norwegian EPIC biological bank only 443 subjects were living in northern Norway. Further, in this nation-wide sampling, the blood collection was suspended during the summer months to avoid low response rates because of summer holidays. This introduced another limitation in our study because the blood sampling period did not cover the whole year, and it was therefore not possible to assess predictors for vitamin D status in northern Norway for all seasons.

A common problem in cross-sectional studies is that exposure data recalled are biased by the subject's knowledge of their own status with respect to the disease or outcome under investigation. In our study, the participants were unaware of their own plasma vitamin D level when they answered the questionnaire. Thus, food intake recalled in the questionnaire was most likely independent of the women's vitamin D status.

Several studies have concluded that vitamin D insufficiency is a common problem in Europe not only among the

Table 4 Regression models for predictors of log 25(OH)D concentrations in blood plasma*

			Stratified analysis									
			Sun holiday/solarium use				Season†					
	All ($n = 260$)		Yes ($n = 78$)		No ($n = 182$)		November–February ($n = 86$)		March–April ($n = 57$)		May–June ($n = 39$)	
Variable	β	*P*-value	β	*P*-value	β	*P*-value	β	*P*-value	β	*P*-value	β	*P*-value
Age (years)	0.010	0.02	0.023	0.01	0.004	0.42	−0.008	0.23	0.007	0.42	0.027	0.04
Body mass index (kg m^{-2})	−0.003	0.54	0.002	0.90	−0.004	0.48	−0.002	0.75	0.007	0.38	−0.030	0.07
Dietary vitamin D intake (μg day^{-1})	0.010	0.0003	0.002	0.66	0.013	0.0003	0.014	0.006	0.011	0.04	0.017	0.04
Supplement use‡ (yes/no)	0.022	0.56	−0.004	0.96	0.030	0.49	−0.033	0.58	0.110	0.12	−0.012	0.91
Residing in northern Norway during the previous summer (yes/no)	−0.107	0.004	−0.045	0.54	−0.144	0.0008	−0.118	0.05	−0.120	0.09	−0.159	0.15
Hours in daylight last week prior to blood sampling	0.002	0.36	−0.001	0.75	0.003	0.22	0.003	0.71	−0.004	0.44	0.005	0.09
Time of year when the blood specimen was collected	–	0.005	–	0.62	–	0.002	–	–	–	–	–	–
November–December	ref	ref	ref	ref	ref	ref	–	–	–	–	–	–
January–February	−0.032	0.61	−0.122	0.37	0.013	0.86	–	–	–	–	–	–
March—April	0.125	0.05	−0.046	0.74	0.204	0.005	–	–	–	–	–	–
May–June	0.120	0.12	0.032	0.84	0.139	0.11	–	–	–	–	–	–
Latitude (°N)	0.007	0.60	−0.024	0.31	0.015	0.350	–	–	–	–	–	–
Sun holiday (yes/no)	0.116	0.02	–	–	–	–	–	–	–	–	–	–
Solarium use (yes/no)	0.221	<0.0001	–	–	–	–	–	–	–	–	–	–
Model	$R^2_{adj} = 0.24$, $P < 0.0001$		$R^2_{adj} = 0.05$, $P = 0.21$		$R^2_{adj} = 0.23$, $P < 0.0001$		$R^2_{adj} = 0.10$, $P = 0.03$		$R^2_{adj} = 0.14$, $P = 0.03$		$R^2_{adj} = 0.26$, $P = 0.01$	

25(OH)D – 25-hydroxyvitamin D; ref – reference category.
*Due to missing values the total is less than 300.
†For the seasonal strata, subjects who had been on a sun holiday or used a solarium were excluded.
‡Cod-liver oil supplement not included.

Table 5 Models for predictors of log 25(OH)D concentrations in blood plasma when 'UV-hours' replace 'hours in daylight last week prior to blood sampling', 'latitude' and 'time of year when the blood specimen was collected'

			Analysis stratified by sun holiday/solarium use			
	All ($n = 270$)		Yes ($n = 81$)		No ($n = 190$)	
Variable	β	*P*-value	β	*P*-value	β	*P*-value
Age (years)	0.009	0.03	0.025	0.004	0.002	0.61
Body mass index (kg m^{-2})	−0.003	0.57	0.004	0.75	−0.002	0.65
Dietary vitamin D intake (μg day^{-1})	0.011	<0.0001	0.001	0.79	0.014	<0.0001
Supplement use*	0.025	0.49	−0.001	0.99	0.027	0.52
Residing in northern Norway during the previous summer	−0.117	0.001	−0.066	0.35	−0.149	0.0003
UV-hours (h day^{-1})	–	0.0002	–	0.64	–	0.0002
0	ref	ref	ref	ref	ref	ref
0.1–1.0	0.100	0.04	0.010	0.92	0.128	0.03
1.1–2.0	0.181	0.0005	0.113	0.24	0.203	0.001
2.1 +	0.177	0.0002	0.059	0.53	0.201	0.0003
Sun holiday	0.113	0.02	–	–	–	–
Solarium use	0.224	<0.0001	–	–	–	–
Model	$R^2_{adj} = 0.25$, $P < 0.0001$		$R^2_{adj} = 0.06$, $P = 0.14$		$R^2_{adj} = 0.24$, $P < 0.0001$	

25(OH)D – 25-hydroxyvitamin D; ref – reference category.
*Cod-liver oil supplement not included.

elderly[22], but also for middle-aged[9] and younger populations[7,23]. Van der Wielen *et al.*[22] have shown that, among the elderly, sub-clinical vitamin D deficiency is surprisingly more common in southern Europe than in Scandinavia. This was explained by food fortification and vitamin D supplements that are common in Scandinavia. A recent study from Finland[24] indicated that one-third of a young Finnish population was vitamin D-deficient (< 25 nmol l^{-1}) during winter, and suggested that this could be a health concern for the whole of northern Europe. The prevalence of low vitamin D status in the Finnish study was higher than what we observed. This could be due to higher mean daily vitamin D intake, which for the Finnish compared with our northern Norwegian sample was 4.7 ± 2.5 μg versus 8.1 ± 7.0 μg, respectively.

A significant positive correlation between dietary vitamin D intake and 25(OH)D level has previously been reported in subjects with low sun exposure[25,26]. Our findings suggest that the middle-aged female population in northern Norway depends heavily on dietary sources of vitamin D due to insufficient UV exposure. Further, a considerable proportion is at risk of not consuming the amount necessary to cover their needs.

In contrast to Vik *et al.*[11] who did not find any seasonal variation in 25(OH)D level from November until May, our results show an obvious 'dark-season' effect on vitamin D levels. The low level of moderate hypovitaminosis D observed in March–April could be explained by Easter holidays, since there is a tradition in Norway to go skiing at this time in the mountains where reflection of UV radiation from the snow will increase vitamin D production in the skin.

Sun holidays and the use of a solarium were, not surprisingly, strong predictors of 25(OH)D level. In addition, when adjusting for time of year when the blood sample was taken, staying at more southern latitudes during the previous summer was also positively associated with vitamin D in the blood. This indicates a long-term impact of summer sunlight exposure on vitamin D status. The survey by Poskitt *et al.*[27] can be interpreted similarly as they found that, among children from the West Midlands, England, 25(OH)D concentrations were higher in those who had had a seaside holiday the year before blood sampling compared with those who had not.

In our study we found that age was a positive predictor of plasma 25(OH)D among subjects who had been on a sun holiday or spent time in a solarium. This is contradictory to what has been reported in other studies, where age has been associated with an increased risk of low levels of vitamin D in blood. This was thought to reflect the decreased capacity in the elderly of human skin to produce vitamin D_3[28]. However, Need *et al.*[29] found that 25(OH)D levels fell significantly after the age of 69. The age of our participants ranged from 44 to 59 years, and thus was below the age when the capacity of the skin to produce vitamin D is likely reduced. Thus it is probable that the increased 25(OH)D level with age observed in subjects who had been on a sun holiday or used a solarium was due to other factors associated with age that could not be controlled for in the present analysis, such as use of suntan lotion, time spent in direct sun or clothing worn.

When assessing sun exposure in relation to vitamin D status in populations, a 9-point scale has been employed in several studies[14,25,26,30] and has been considered valid for ranking subjects by sunshine exposure. Briefly, the ranking scale is based on recall of the time and degree of exposure to sunshine the week before administration of the questionnaire, sunscreen use, and travel to southern locations during the previous month. Our estimated 'UV-hours' variable correlated well with plasma 25(OH)D

level. This suggests that recall of hours spent in daylight the week prior to the blood sampling, weighed for estimated geographic- and time-specific UV radiation relevant for vitamin D photoconversion, can be considered a valid measure for UV-induced vitamin D production. Furthermore, this association indicates that, for middle-aged women living at 65–71°N, seasonal variation in vitamin D status can be explained by variation in UV exposure at these latitudes. The association between 'UV-hours' and vitamin D status does not necessarily mean that blood 25(OH)D concentrations reflect UV exposure the week prior to the blood sampling. Instead it could indicate that recall of the time spent in daylight for one week gives a representative estimate for usual sun exposure per week during the actual season. This interpretation is in line with the discussion above about the long-term impact of summer exposure to sunshine on vitamin D status.

Cod-liver oil supplements, margarine, butter and fatty fish have been established in dietary surveys conducted in Norway as the primary dietary sources of vitamin D[6]. However, the impact of the traditional fish-liver dish *mølje* and fresh fish-liver oil on vitamin D status is a phenomenon characteristic for the population of northern Norway[4,5]. Recently, the Norwegian Food Control Authority has recommended that all women of childbearing age, pregnant women and children do not to eat fish liver because of the risk of long-term negative health impact due to the presence of dioxins and other persistent organic pollutants (POPs) above tolerable limits[31]. Our study showed that consumption of fish liver was an important contributor to vitamin D intake for the population in the north. Uncertainties regarding the possible negative health effects of POPs versus the nutritional benefit of traditional foods have been discussed previously, in particular in relation to the marine diet among populations living in arctic areas, and has been named the 'Arctic dilemma'[32]. Studies from Barrow, Alaska (71.3°N) have, for example, found a high rate of vitamin D deficiency in adults especially among subjects who were not consuming a traditional diet[33]. From a public health perspective, a change in food habits away from traditional diets, that are rich in essential nutrients such as fat-soluble vitamins and polyunsaturated fatty acids, can cause increased risk of some diseases. For the northern Norwegian population, which is vulnerable to vitamin D deficiency due to living at high geographical latitudes, a reduction in liver consumption during winter can have an influence on vitamin D status and health. Thus, a balance between the nutritional benefits of traditional foods and the possible negative health outcomes caused by toxins such as POPs must always be considered when developing and promoting dietary guidelines.

In conclusion, dietary sources of vitamin D are of essential importance for the inhabitants of northern Norway. At these latitudes, skin synthesis of vitamin D cannot compensate for low intakes during winter. Traditional foods, like fish liver and fish-liver oils, constitute good sources of vitamin D. Travelling to southern locations can also improve vitamin D status and protect against deficiency throughout the winter. However, a considerable proportion of residents do not go abroad on a sun holiday or to the south during the summer, and are therefore at increased risk of low vitamin D levels if they fail to consume sufficient food items rich in vitamin D. Increased ingestion of marine foods that provide vitamin D should be promoted and further studies should be carried out to investigate vitamin D status in arctic areas, in relation to both UV exposure and traditional food sources. Study designs with repeated blood measurements are recommended to assess seasonal variation in vitamin D in combination with UV-exposure measurements.

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