Recommended summer sunlight exposure amounts fail to produce sufficient vitamin D status in UK adults of South Asian origin1–3

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ABSTRACT

Background: The cutaneous synthesis of vitamin D is dependent on UVB from sunlight, but melanin reduces the penetration of UVB and thus contributes to vitamin D insufficiency in individuals with darker skin. The national guidance provided on amounts of sunlight exposure in the United Kingdom is for the light-skinned population, and in the absence of dedicated information, darker-skinned people may attempt to follow this guidance.

Objectives: We determined the relative effect of a simulation of UK recommendations of summer sunlight exposure on the vitamin D status of individuals of South Asian ethnicity compared with that of whites.

Design: In a prospective cohort study, simulated summer sunlight exposures were provided under rigorous dosimetric conditions to 15 adults (aged 20–60 y) of South Asian ethnicity, and serum 25-hydroxyvitamin D [25(OH)D] was measured weekly. Dietary vitamin D intake was estimated. Outcomes were compared with those of 109 whites (aged 20–60 y) treated with the identical UV-radiation exposure protocol.

Results: At baseline (winter trough), all South Asians were vitamin D–insufficient [25(OH)D concentrations <20 ng/mL], and 27% of South Asians were vitamin D–deficient [25(OH)D concentrations <5 ng/mL]; although 25(OH)D concentrations increased postcourse (P < 0.0001), all South Asians remained vitamin D–insufficient. The mean increase in 25(OH)D was 4.3 compared with 10.5 ng/mL in the South Asian and white groups, respectively (P < 0.0001), and 90% of the white group reached vitamin D sufficiency postcourse. The median dietary vitamin D intake was very low in both groups.

Conclusions: Sunlight-exposure recommendations are inappropriate for individuals of South Asian ethnicity who live at the UK latitude. More guidance is required to meet the vitamin D requirements of this sector of the population. This study was registered at www.isrctn.org as ISRCTN 07565297. Am J Clin Nutr 2011;94:1219–24.

INTRODUCTION

Sunlight exposure is required for cutaneous vitamin D synthesis, but excessive exposure is the principal risk factor for the majority of skin cancers, which continue to rise in incidence in white populations (1). Hence, public health messages promoted by the UK Department of Health funded SunSmart campaign, and similar campaigns in other countries, recommended a limitation of summer sunlight exposure (2). National advice has also considered requirements for cutaneous vitamin D synthesis because the exposure of unprotected skin to UVB in sunlight is the principal source of vitamin D, with generally small amounts obtained from the diet (3, 4). Sunlight-exposure recommendations are geared predominantly at fair-skinned individuals but in the absence of dedicated information, darker-skinned people may attempt to follow these recommendations despite having much lower risks of skin cancer.

The active hormonal form of vitamin D is 1,25-dihydroxyvitamin D and is important for bone health because it is required for calcium absorption and bone mineralization (5). There is mounting evidence that vitamin D can convey other health benefits, including the potential prevention of colon cancer, diabetes, and multiple sclerosis (6–8). The circulating concentration of 25(OH)D3 is considered the best indicator of vitamin D status, with rickets in children and osteomalacia in adults shown at 25(OH)D concentrations <5–10 ng/mL (12.5–25 nmol/L). 25(OH)D concentrations <20 ng/mL (50 nmol/L) are now accepted by many authorities, including the US Institute of Medicine, to indicate vitamin D insufficiency, which is associated with bone loss, hyperparathyroidism, and muscle weakness (9, 10). On the basis of variables of bone health, a 25(OH)D concentration ≥32 ng/mL (80 nmol/L) was also proposed as optimal for health (11).
There is growing evidence of vitamin D insufficiency and deficiency in the United Kingdom (12–14). Low vitamin D status is particularly prevalent in darker-skinned people, with many reports of low concentrations and related health problems in South Asians (15–20). Like many countries at a similar latitude (50–60°N), the United Kingdom has a significant and rising population of individuals of sun-reactive skin type V (ie, with brown skin) who are particularly of South Asian ethnicity (21, 22). Although South Asians reportedly have the same capacity to synthesize vitamin D as do whites (sun-reactive skin types I–IV), pigmented skin requires greater sunlight exposure to raise circulating 25(OH)D as melanin absorbs a proportion of the incident UVB (23–25). Differences in diet and lifestyle may also contribute to low vitamin D status, but the relative risks attributable to skin color are ill defined.

The UK Health Protection Agency advised that vitamin D deficiency can be avoided through casual exposures to summer sunlight that contains the requisite UVB (26). On the basis of an interpretation of this advice, we showed that a course of UVR that simulated sunlight exposure over the 6-wk school summer-holiday period produced 25(OH)D concentrations ≥20 ng/mL in 90% of the white adult population (27). To examine how skin pigmentation in South Asians influences vitamin D status outcomes, we examined the effect of the same course of UVR exposures under identical protocols in South Asian individuals.

SUBJECTS AND METHODS

Study subjects

Volunteers (n = 15) were of South Asian (Indian, Pakistani, and Bangladeshi) ethnicity, with Fitzpatrick sun-reactive skin type V (22), aged 20–60 y, and living in Greater Manchester, United Kingdom (53.5°N). Exclusion criteria were a history of skin cancer or a photosensitivity disorder, use of a sunbed or medication or supplements that contained vitamin D, and cur- rently pregnant or breastfeeding. Nine subjects participated in January–February 2009, and 6 subjects participated in January–February 2010. Ethical approval was obtained from the North Manchester Research Ethics Committee (references 08/H1006/24 and 09/H1014/73). White volunteers (n = 109), who were aged 20–60 y, had sun reactive skin types I–IV, and were from Greater Manchester, participated under the exact same protocols in January–February 2007 and January–February 2008 (27). Written informed consent was obtained from participants, and the study adhered to the principles of the Declaration of Helsinki. Heights and weights of subjects were measured, and BMI (in kg/m²) was calculated.

Simulated summer sunlight exposures

Subjects received a 6-wk course of UVR exposures, which represents the length of the summer school-holiday period in the United Kingdom when the population is most exposed to sunlight as previously described (27). Subjects were given a constant dose of UVR 3 times weekly with a whole-body irradiation cabinet (Philips HB598; Philips) fitted with a combination of Arimed B (Cosmedico GmbH) and Cleo Natural (Philips, Eindhoven, The Netherlands) fluorescent tubes, which produced a UVR emission spectrum similar to that of sunlight (emission: 290–400 nm; 95% UVA: 320–400 nm; 5% UVB: 290–320 nm). The emission spectrum was characterized with a Bentham DTM 300 spectroradiometer (Benthem) and monitored with an Ocean Optics S2000 spectroradiometer (Ocean Optics). Subjects wore standardized T-shirts and knee-length shorts to expose a skin surface area of ~35%, representing casual summer clothing. The UVR course was given in January and February when ambient UVB is negligible at UK latitudes (50–60°N) (28) and, hence, could not confound study outcomes. A UVR exposure of 1.3 SEDs (29) was given to each subject at every visit. The cabinet UV spectrum was matched to sunlight for vitamin D synthesis but differed slightly for erythema production such that 1.3 SEDs was equivalent to 1.1 SEDs in sunlight but the same in terms of vitamin D production. Therefore, the total dose received over the course was 23.4 SEDs, which was equivalent to 19.8 SEDs in sunlight. The irradiation cabinet simultaneously exposed dorsal and ventral surfaces, with the subject lying horizontal. With the use of radiative-transfer modeling to translate this to real-life exposures, it was estimated that a single 6.5-min (1.3-SED) UVR exposure in the cabinet approximated 26–30 min of unshaded sunlight exposure on a clear June midday in Manchester (30). This method took account of a range of possible activities, from walking around to lying down and exposing the front and then the back of the body, in turn, to sunlight. The UVR irradiance of the cabinet was accurately measured (31), and a constant UVR dose was maintained throughout the study with adjustment for any decrease in irradiance by increasing the exposure time.

MED assessment

The individual sunburn threshold (MED) of each subject was assessed before the exposure course. A geometric series of 10 doses (26.6–271 mJ/cm²) of erythemally weighted UVR was applied over 2 horizontal rows of buttock skin with a Waldmann UV 236 B unit with Waldmann CF-L 36W/UV6 lamps (peak emission: 313 nm; range: 290–400 nm; Waldmann GmbH). The MED value was defined as the lowest dose of UVR that produced a visually discernable erythema at 24 h.

Skin color–change assessment

To assess for a potential increase in skin pigmentation attributable to the UVR course, skin lightness [International Commission on Illumination (CIE) L*] was measured on a scale from 0 (black) to 100 (white) with a spectrophotometer (CM2500d; Konica Minolta). Triplicate readings were taken from exposed buttock skin at baseline and at weekly intervals during the UVR course. Measurements were taken immediately before the third UVR exposure of the week.

Log of vitamin D–containing foods

Subjects completed a daily log of vitamin D–containing foods for 7 d during the first and last weeks of the study to estimate approximate daily oral vitamin D intakes. The log comprised vitamin D–fortified foods and 6 food categories: cheese; butter, ghee, margarine, and other oily spreads and oils; milk and milk-containing products; red meat; oily fish; and eggs and egg dishes. The vitamin D content of foodstuffs was obtained from the sixth edition
and integrated data set of McCance and Widdowson’s *The Composition of Food* (32) and from food-package labeling. The log requires validation against gold-standard nutritional assessment methods in this population. Thrice-weekly volunteer attendance for UVR exposures permitted a regular check of log completion.

### 25(OH)D, PTH, and serum biochemistry

Blood samples were taken weekly, and serum was stored at −20°C until completion of the UVR course. Serum 25(OH)D was measured by HPLC as previously reported (33) in a laboratory that participates in the national Vitamin D Quality Assurance Scheme and is accredited to International Organization for Standardization 9001:2000 and 13485:2003 standards. Serum PTH was measured before commencement and after completion of the UVR course by using the OCTEIA immunoenzymometric assay according to the manufacturer’s instructions (Immunodiagnostic Systems). The assay had a sensitivity of 0.06 pmol/L and intraassay and interassay CVs of 4% and 6%, respectively. Serum biochemistry was measured with a Hitachi 917 autoanalyser (Hitachi) before the UVR course and included renal- and liver-function tests.

#### Definition of 25(OH)D concentrations

In this study, circulating concentrations of 25(OH)D 5 ng/mL (12.5 nmol/L) were defined as deficient, <20 ng/mL (50 nmol/L) were defined as insufficient, ≥20 ng/mL were defined as sufficient, and ≥32 ng/mL (80 nmol/L) were defined as optimal.

#### Outcome measures

The primary outcome measure was the serum 25(OH)D concentration reached after the UVR-exposure course. The adequacy of this concentration was related to the categories of vitamin D status previously defined. Secondary outcomes were differences in final 25(OH)D concentrations and in the rise in concentrations between South Asian and white subjects and the concentration of PTH and oral vitamin D intake in South Asians.

#### Statistical analyses

Data were analyzed with StatsDirect software (v2.7.7; StatsDirect). Inter- and intragroup differences were tested by the unpaired or paired t test, respectively, with significance accepted at $P \leq 0.05$. Where appropriate, variables were transformed to satisfy normality assumptions.

### RESULTS

#### Subject characteristics

Fifteen subjects of South Asian ethnicity (12 men and 3 women) were recruited and completed the course of UVR exposures. Baseline characteristics of volunteers, including the MED, serum 25(OH)D, PTH, and biochemistry values are shown in Table 1. Data from vitamin D diet logs completed in the first and last weeks of the study indicated the average daily oral intake of vitamin D to be low with a median intake of 1.79 µg (72 IU). There was no significant difference in intake between weeks, with a median intake of 1.5 µg vitamin D/d in week 0 and 1.26 µg vitamin D/d in week 6. Serum 25(OH)D was undetectable, which reflected a low dietary vitamin D intake. At baseline, all subjects had a 25(OH)D concentration, $<20$ ng/mL, with 4 subjects (27%) who had a 25(OH)D concentration defined as deficient (<5 ng/mL).

#### Change in 25(OH)D concentrations after the 6-wk UVR course

The 6-wk course of UVR exposures led to a significant rise in the mean (±SD) 25(OH)D concentration of 4.3 ng/mL, from

<table>
<thead>
<tr>
<th>TABLE 1</th>
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<tr>
<td><strong>Baseline characteristics of South Asian (sun-reactive skin type V) subjects ($n = 15$; 12 M, 3 F)$^{1}$</strong></td>
</tr>
<tr>
<td><strong>Minimum</strong></td>
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<tr>
<td>Age (y)</td>
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<td>Height (m)</td>
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<tr>
<td>Weight (kg)</td>
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<tr>
<td>BMI (kg/m$^2$)</td>
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<tr>
<td>MED (mJ/cm$^2$)</td>
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<tr>
<td>Average daily vitamin D intake (µg)</td>
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<td>Serum biochemistry</td>
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<tr>
<td>25(OH)D (ng/mL)</td>
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<tr>
<td>Parathyroid hormone (pmol/L)</td>
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<td>Sodium (mmol/L)</td>
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<td>Potassium (mmol/L)</td>
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<td>Urea (mmol/L)</td>
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<td>Creatinine (µmol/L)</td>
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<td>Calcium (mmol/L)</td>
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<tr>
<td>Inorganic phosphorous (mmol/L)</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/L)</td>
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<tr>
<td>Albumin (g/L)</td>
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<tr>
<td>Alanine transaminase (U/L)</td>
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$^{1}$ MED, minimal erythemal dose (ie, individual sunburn threshold); 25(OH)D, 25-hydroxyvitamin D.
6.4 ± 1.9 ng/mL (range: 2.7–11.0 ng/mL) before treatment to 10.7 ± 2.6 ng/mL (range: 5.8–14.8 ng/mL; P < 0.0001) after treatment (Figure 1). This was accompanied by an apparent but nonsignificant fall in the mean PTH from 3.2 ± 1.7 pmol/L (range: 1.1–6.4 pmol/L) to 2.4 ± 1.3 pmol/L (range: 1.1–5.6 pmol/L). After treatment, the 25(OH)D concentration of all 4 subjects defined as deficient at baseline increased to >5 ng/mL. However, none of the participating subjects achieved a sufficient concentration of 25(OH)D, with all concentrations at <20 ng/mL.

Change in skin pigmentation after the 6-wk UVR course

Skin lightness, which was measured as L*, was determined in a subset of South Asian subjects (n = 6) at baseline and at weekly intervals during the UVR course. Skin lightness was significantly decreased after the UVR course with a mean (±SD) L* value of 31.14 ± 15.64 compared with 41.11 ± 12.84 at baseline (P < 0.05; Figure 2).

Comparison of South Asian and white groups

In our previous study, 109 whites underwent an identical course of UVR treatment (27). The median MED of this group was 34 mJ/cm² (range: 16–82 mJ/cm²). At baseline, the white group had a significantly higher mean (±SD) 25(OH)D concentration of 17.6 ± 7.6 ng/mL (range: 3.1–38.0 ng/mL; P < 0.0001; Figure 1), and a significantly lower mean PTH concentration of 2.1 ± 1.3 pmol/L (range: 0.6–7.5 pmol/L; P < 0.01) than did the South Asian group. There was no significant difference in BMI between the 2 groups with a mean (±SD) of 25.0 ± 4.3 in whites and a mean (±SD) of 25.4 ± 4.7 in South Asians. The mean rise in 25(OH)D of 10.4 ng/mL for the white group after the UVR course was significantly greater than that of the South Asian group (P < 0.001). There was no significant difference in postcourse PTH concentrations between the 2 groups, with a mean (±SD) of 2.0 ± 1.2 pmol/L for whites and 2.4 ± 1.3 pmol/L for South Asians. The dietary intake of vitamin D was very low in both groups, with a median intake of 1.79 µg/d (range: 0.35–5.37 µg/d) in South Asians compared with 2.16 µg/d (range: 0.18–9.89 µg/d) in whites (P < 0.05; Table 2).

DISCUSSION

Our data showed that regular, short, midday exposures to summer sunlight at a latitude off 53.5°N while exposing ~35% of the body’s skin surface did not produce sufficient vitamin D status in individuals of South Asian ethnicity. There are limited data on vitamin D status after a controlled exposure to UVR in South Asians, and previous work has not attempted to simulate actual sunlight exposure but, instead, performed whole-body exposures and used lamps that mainly emitted UVB radiation (24). Our novel study was a more appropriate model of real-life sunlight exposure because we used casual summer clothing to reveal commonly exposed skin sites, as different skin sites could not be assumed to be equally efficient in vitamin D synthesis, and used fluorescent lamps selected to be a close as possible in UVR spectral emission to natural sunlight. Although vitamin D synthesis is attributable to UVB, UVA is involved in its degradation in the skin, and therefore its influence should be taken into account (34). Rigorously controlled dosimetry allowed for

![Figure 1](https://example.com/f1.png)

**FIGURE 1.** Mean and full range of serum 25(OH)D concentrations in South Asians (black squares; n = 15) and in whites (open squares; n = 109) before the course of UV radiation (mean ± SEM: 6.4 ± 0.5 and 17.6 ± 0.7 ng/mL, respectively) at weekly intervals during treatment and at the end of the UV-radiation course (mean ± SEM: 10.7 ± 0.7 and 28.0 ± 0.6 ng/mL, respectively). ***P < 0.001 (2-tailed paired t test) compared with baseline (week 0). 25(OH)D, 25-hydroxyvitamin D.

![Figure 2](https://example.com/f2.png)

**FIGURE 2.** Mean (±SEM) values of skin lightness (0 = black; 100 = white) in a subset of South Asian subjects (n = 6) before the course of UV radiation at weekly intervals during treatment and at the end of the UVR-radiation course. *P < 0.05 (2-tailed paired t test) compared with baseline (week 0). L*, lightness.

| TABLE 2 | Dietary sources of vitamin D for South Asian and white groups
<table>
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<tbody>
<tr>
<td>Dietary source</td>
<td>South Asians (n = 15)</td>
</tr>
<tr>
<td>Fortified foods</td>
<td>0 (0–0.72)</td>
</tr>
<tr>
<td>Red meat</td>
<td>0.08 (0–0.60)</td>
</tr>
<tr>
<td>Cheese</td>
<td>0.01 (0–0.13)</td>
</tr>
<tr>
<td>Eggs and egg dishes</td>
<td>0.6 (0–1.65)</td>
</tr>
<tr>
<td>Milk</td>
<td>0.04 (0–0.12)</td>
</tr>
<tr>
<td>Fats and oils</td>
<td>0.01 (0–0.78)</td>
</tr>
<tr>
<td>Oily fish</td>
<td>0 (0–2.92)</td>
</tr>
<tr>
<td>All foods</td>
<td>1.79 (0.35–5.37)</td>
</tr>
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</table>

*All values are medians; ranges in parentheses.*
a definitive comparison of vitamin D status in South Asian and white adults after the same exposure protocols, which approximated recommended amounts of summer sunlight exposure in the United Kingdom. Winter baseline concentrations of 25(OH)D were low, with a concentration of <20 ng/mL in all South Asian subjects, 27% of whom were deficient (<5 ng/mL). Moreover, although a circulating 25(OH)D concentration <5 ng/mL has been defined as the cutoff for deficiency (26), some authorities regard 25(OH)D concentrations below approximately <10 ng/mL as a reflection of risk of deficiency (ie, where rickets and osteomalacia may occur) (10, 35), and all but one (93%) of our subjects had 25(OH)D concentrations <10 ng/mL. However, there is still controversy regarding cutoffs, with studies that showed high proportions of apparently healthy individuals with 25(OH)D concentrations <10 ng/mL (36). The concentrations we observed were consistent with previous reports of 25(OH)D concentrations in larger samples of South Asian adults resident in the United Kingdom (15, 16, 18–20). Although 25(OH)D concentrations rose significantly after the course of UVR exposures, the mean concentration achieved was only 10.7 ng/mL, and concentrations remained significantly lower than those of white individuals who underwent identical protocols (27), with none of the South Asian subjects having attained sufficiency (20 ng/mL). The weekly gain in 25(OH)D steadily decreased during the UVR course with concentrations that reached an apparent plateau after 6 wk. We observed a similar plateau in 25(OH)D concentrations in white individuals, and it is uncertain whether a continuation of these low-exposure doses past 6 wk would have increased 25(OH)D concentrations further. Photodadaptation through UV-induced epidermal thickening and increased melanin synthesis are mechanisms that reduce 25(OH)D production in fair-skinned individuals (27, 37). Greater constitutive (basal) pigmentation reduces UVR penetration into the skin (38), and the significant increase in skin darkness (ie, facultative pigmentation) that we observed in our South Asian subjects will serve to reduce UVR penetration further on repeated sunlight exposures as occurs during the summer.

Our findings indicated that skin-pigmentation differences have a profound effect on the sunlight-exposure vitamin D response. It is also pertinent to ask whether people of these different ethnic backgrounds have the same capacity to synthesize vitamin D, and in particular, whether there may be genetic differences that influenced this capacity. Differences in cutaneous 7-dehydrocholesterol concentrations and/or in the efficiency of conversion of vitamin D to 25(OH)D are possible factors, but firm evidence is lacking (15, 39). Although additional studies are required to address this lack of evidence, an early UVR intervention study indicated that the capacity for biosynthesis of vitamin D did not show a substantial difference between South Asians and whites (24). Similar vitamin D outcomes were seen when individuals were given personalized UVR doses that related to their sunburn threshold (MED) to adjust for differences in UVR penetration attributable to pigmentation, which meant that the absolute UVR-exposure doses given to subjects with pigmented skin were higher (24). In contrast, we used identical protocols, such that the difference in vitamin D outcomes could be evaluated between these different pigmentation groups, with doses that could be acquired through recommended short summer sunlight exposures in the United Kingdom. Moreover, all of our subjects had sun-reactive skin type V (brown skin), whereas only one subject in the study reported by Lo et al (24) apparently fell in this category. The doses we gave (each dose was equivalent to 30 min of sunshine exposure) led to vitamin D sufficiency in whites (27). With consideration of the effect of constitutive skin pigmentation alone, South Asians might achieve sufficiency with exposures that are 4 times longer (ie, equivalent to 2 h summer sunlight exposure 3 times/wk) because the median South Asian MED (134 mJ/cm²) in this study was 4 times that of whites (34 mJ/cm²); however, this conclusion makes several assumptions and requires testing as discussed below.

We previously showed that the amount of sunlight exposure achieved by the UK white population was very similar to that of the simulated summer exposures in the current protocols (14). The actual exposure patterns of South Asians in the United Kingdom are currently unreported, but if they attempt to follow national guidance, insufficient vitamin D status will be attained. Moreover, the potential for a more conservative dress in South Asian individuals and lower sun-seeking behavior (ie, less desire to acquire a tan through exposure to sunlight) may further limit 25(OH)D synthesis. We also showed a small but significant difference in oral vitamin D intake between South Asian and white groups. When sensitivity analysis was performed in which individuals with low BMI (<18.5) were excluded, all study conclusions were the same except for the loss of a significant difference in dietary vitamin D intake (P = 0.12). The very low oral intakes seen were similar to those previously reported (36) and compound the problem of a low cutaneous vitamin D source in South Asians. Intakes of vitamin D fall well below WHO recommendations of 5 µg/d (200 IU/d) for adults aged 19–50 y and 10 µg/d for adults aged 51–65 y (40). The UK Department of Health recommends people with pigmented skin take 10 µg oral vitamin D/d, which contrasts with no recommended intake for white subjects aged 4–64 y (26), but there is a lack of awareness of this advice in South Asian populations in the United Kingdom (41).

Our study used a simulation of short exposures on the basis of midday UVR amounts (ie, which provided maximal exposure and maximal vitamin D production). At other times of the day, longer exposures would be required to achieve the same dose. Because risk of UV-induced skin cancer is low in South Asian populations, increased sunlight exposure, which necessitates longer exposure times, may be an appropriate approach, in addition to encouragement to increase oral intakes, to raise 25(OH)D concentrations without being detrimental to skin health. Although the production of 25(OH)D relates to the UVB dose (42), it is currently unknown whether an increase of individual doses of UVR would produce sufficient 25(OH)D concentrations in South Asians. Thus, additional research is required to examine the UVR-25(OH)D dose-response relation for South Asian individuals and to observe actual amounts of sunlight exposure in the UK South Asian population. From the current study, we conclude that casual sun exposure at UK latitudes plays a much smaller role in maintaining vitamin D status in pigmented groups than in light-skinned groups. Public health advice should emphasize to people of pigmented skin that it is inappropriate for them to follow sun-exposure and sun-protection guidance geared for fairer skin types, and an increased oral vitamin D intake is needed.
The authors’ responsibilities were as follows—LER, ARW, and AV: designed the research; DA, JLB, MTD, RK, and SJF: conducted the research and analyzed data; MDF: performed statistical analyses and wrote the manuscript; LER: wrote the manuscript and had primary responsibility for the final content of the manuscript; and all authors: read and approved the final manuscript. None of the authors declared a conflict of interest.

REFERENCES