

# Recommended Summer Sunlight Exposure Levels Can Produce Sufficient ( $\geq 20 \text{ ng ml}^{-1}$ ) but Not the Proposed Optimal ( $\geq 32 \text{ ng ml}^{-1}$ ) 25(OH)D Levels at UK Latitudes

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Recommendations on limitation of summer sunlight exposure to prevent skin cancer may conflict with requirements to protect bone health through adequate vitamin D levels, the principal source being UVB in summer sunlight. We determined whether sufficient ( $\geq 20 \text{ ng ml}^{-1}$ ) and proposed optimal ( $\geq 32 \text{ ng ml}^{-1}$ ) 25(OH)D levels are attained by following UK guidance advising casual short exposures to UVB in summer sunlight, and performed the study under known conditions to enhance the specificity of future recommendations. During wintertime, when ambient UVB is negligible, 120 white Caucasians, aged 20–60 years, from Greater Manchester, UK (53.5°N) received a simulated summer's sunlight exposures, specifically 1.3 standard erythemal dose, three times weekly for 6 weeks, while wearing T-shirt and shorts. The baseline winter data predict that 5% (confidence interval (CI): 2.7–8.6) of Greater Manchester white Caucasians have deficient ( $< 5 \text{ ng ml}^{-1}$ ) 25(OH)D, 62.5% (CI: 55.2–69.4) have insufficient, and only 2.9% (CI: 1.4–5.6) have proposed optimal levels. After the simulated summer exposures, 90 (CI: 84.9–93.7) and 26.2% (CI: 20.1–33.2) reached 20 and 32  $\text{ng ml}^{-1}$  25(OH)D, respectively. Assuming midday UVB levels, sufficient but suboptimal vitamin D status is attained after a summer's short (13 minutes) sunlight exposures to 35% skin surface area; these findings will assist future public health guidance on vitamin D acquisition.

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## INTRODUCTION

Policy recommendations to limit summer sunlight exposure to prevent skin cancer have generated considerable international debate in recent years (Gillie, 2006; Wolpowitz and Gilchrest, 2006). Skin cancer has high incidence in countries with large populations of white Caucasians, including the United Kingdom, United States of America,

and Australia, and incidence continues to rise, with UVR being the principal etiological agent in the majority (Elwood and Jopson, 1997; National Radiological Protection Board, 2002). It is important that national and international authorities advise summer sunlight limitation in these populations (Ziegelberger *et al.*, 2006). However, UVB in sunlight triggers cutaneous synthesis of pre-vitamin D from 7-dehydrocholesterol, and this is the body's principal vitamin D source because usually only small amounts are obtained from diet (Hollis, 2005). Thus, public health policy on sunlight exposure should also consider vitamin D requirements.

Historically, vitamin D deficiency was defined as the circulating level of 25 hydroxyvitamin D (25(OH)D) associated with the development of the severe bone disorders, rickets, and osteomalacia, that is,  $< 5\text{--}10 \text{ ng ml}^{-1}$  (Berry *et al.*, 2002). The value  $5 \text{ ng ml}^{-1}$  is still used as the deficiency cutoff by national agencies such as the United Kingdom's Health Protection Agency and Department of Health (1998), although this is under reevaluation. Strong evidence exists showing that 25(OH)D levels  $< 20 \text{ ng ml}^{-1}$  are associated with secondary hyperparathyroidism, bone loss, fractures, muscle weakness, and reduced calcium absorption (Bischoff *et al.*, 2003; Heaney *et al.*, 2003; Zittermann, 2003; Bischoff-Ferrari *et al.*, 2004). Parathyroid

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Abbreviations: 25(OH)D, 25 hydroxyvitamin D; BMI, body mass index; HPA, Health Protection Agency; MED, minimal erythemal dose; PTH, parathyroid hormone; SED, standard erythemal dose

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hormone (PTH) is suppressed as the 25(OH)D level rises (Hollis, 2005), with a plateau being reached between 20 (Malabanan *et al.*, 1998) and 40 ng ml<sup>-1</sup> (Chapuy *et al.*, 1997; Thomas *et al.*, 1998). On the basis of these associations, some researchers now believe that 25(OH)D levels  $\geq 20$  ng ml<sup>-1</sup> are required to avoid vitamin D deficiency (Malabanan *et al.*, 1998; Wolpowitz and Gilcrest, 2006; Norman *et al.*, 2007; Holick, 2009), whereas it is proposed that an optimal level for health is reached at 30–32 ng ml<sup>-1</sup> or higher (Hollis, 2005; Bischoff-Ferrari *et al.*, 2006). Significant percentages of populations at northerly latitudes fall short of these levels (Webb and Engelsen, 2006; Hypponen and Power, 2007; Hirani *et al.*, 2009).

Evidence continues to grow of wider health benefits conveyed by vitamin D, with much of this being indirect in nature. Mortality from prostate, colon, and breast cancers is inversely associated with ambient UVB, and this has been attributed to low vitamin D status (Berwick and Kesler, 2005; Garland *et al.*, 2006; Holick, 2006), whereas the International Agency for Research on Cancer (2008) supports a protective role for vitamin D in colon cancer. Further evidence exists for a beneficial effect of vitamin D on other diseases, including multiple sclerosis, tuberculosis, diabetes, hypertension, and other cancers (Zittermann, 2003; Holick, 2004; Vieth, 2006; Lipworth *et al.*, 2009), although epidemiological data are conflicting regarding protection against melanoma (Millen *et al.*, 2004; Ingraham *et al.*, 2008; Asgari *et al.*, 2009). Mechanisms may involve immunomodulatory and chemopreventive properties of 1,25-dihydroxyvitamin D, now known to be metabolized from 25(OH)D by many extra-renal tissues (Millen *et al.*, 2004; Ingraham *et al.*, 2008; Asgari *et al.*, 2009).

The UK Department of Health-funded SunSmart campaign is in line with similar campaigns in many countries in its recommendations to limit personal summer sunlight exposure (<http://info.cancerresearchuk.org/healthyliving/sunsmart>), whereas the United Kingdom's Health Protection Agency, in keeping with countries positioned at similar latitude, advises that casual exposures to summer sunlight, containing the requisite UVB, are sufficient for attaining vitamin D (National Radiological Protection Board, 2002). However, there is uncertainty regarding the specification and impact of following this guidance, as it is based on data derived from theoretical and *in vitro* models (National Radiological Protection Board, 2002; Ziegelberger *et al.*, 2006). To address this, we designed a study to examine the impact of following these recommendations on vitamin D status. A total of 120 white Caucasian subjects received simulated summer sunlight exposures mimicking UK guidance; sample size was selected for estimation of population variation with adequate precision. The aims of this study were to (i) determine whether recommended brief casual summer sunlight exposures can achieve 25(OH)D levels  $\geq 20$  ng ml<sup>-1</sup> and the proposed optimal, that is, 25(OH)D  $\geq 32$  ng ml<sup>-1</sup>, vitamin D status; and (ii) provide specific information to assist future guidance on vitamin D acquisition, by performance under known conditions of UV dose and skin surface area.

## RESULTS

### Volunteer characteristics

Of 120 recruited subjects, five were subsequently excluded because of vitamin D supplement use, and six failed to continue UVR treatment, resulting in 109 subjects (68% female, 32% male) completing the study. Baseline characteristics of the 109 volunteers, including minimal erythral dose (MED), 25(OH)D, PTH, and serum biochemistry values are shown in Table 1. The average daily oral vitamin D intake was low; intakes during the first and last weeks of the study are shown in Table 1.

### Circulating 25(OH)D levels rose significantly in the volunteer group and posttreatment levels were associated with pretreatment levels

The 6 weeks of UVR exposures caused the mean 25(OH)D value to rise significantly by 10.4 ng ml<sup>-1</sup> (95% confidence interval (CI): 9.1–11.8), from 17.6 ng ml<sup>-1</sup> (SD 7.6; range: 3.1–38) before treatment to 28.0 ng ml<sup>-1</sup> (SD 6.3; range: 10.8–50.9) after treatment (Figure 1). The increase in 25(OH)D levels after 6 weeks of UVR treatment varied among individuals (interquartile range: 5.4–14.5; range: 2.1–31.9 ng ml<sup>-1</sup>). Multiple linear regression analysis identified that posttreatment 25(OH)D levels were significantly associated with pretreatment 25(OH)D levels ( $P < 0.0001$ ), consistent with the results of a previous study (Moan *et al.*, 2009), whereas other factors were not significantly associated (Table 2).

### The majority of the Greater Manchester, UK, white Caucasian nonelderly adult population is predicted to have insufficient (<20 ng ml<sup>-1</sup>) levels of 25(OH)D during wintertime

Assuming normality, the mean and SD values of the study group were used to calculate the percentage of the Greater Manchester population predicted to have 25(OH)D values, indicating deficient, sufficient, and proposed optimal status (Table 3). Baseline winter data predict that 5% (95% CI: 2.7–8.6) of the population has 25(OH)D levels  $< 5$  ng ml<sup>-1</sup>, 62.5% (CI: 55.2–69.4) has levels  $< 20$  ng ml<sup>-1</sup>, whereas 37.5% (CI: 30.6–44.8) has levels  $\geq 20$  ng ml<sup>-1</sup> and only 2.9% (CI: 1.4–5.6) has levels  $\geq 32$  ng ml<sup>-1</sup>.

### The majority of the Greater Manchester, UK, white Caucasian nonelderly population is predicted to reach sufficient ( $\geq 20$ ng ml<sup>-1</sup>) but less than the proposed optimal (<32 ng ml<sup>-1</sup>) levels of 25(OH)D after summer sunlight exposures according to national recommendations

After a summer's short sunlight exposures, 90% (95% CI: 84.9–93.7) of the Greater Manchester, white Caucasian population is predicted to reach sufficient ( $\geq 20$  ng ml<sup>-1</sup>) levels of 25(OH)D, whereas 26.2% (CI: 20.1–33.2) reaches 32 ng ml<sup>-1</sup> or higher (Table 3).

### The time to acquire an equivalent vitamin D-weighted dose at North American and European locations ranges from 9 to 16 minutes

Given the same conditions as seen in the volunteers in this study, including white skin, dressed to reveal 35% skin surface area, and receiving a regimen of regular short midday exposures, the exposure time taken to acquire the same vitamin D-weighted dose across a range of European and

**Table 1. Participant information**

Participants	109					
Sex: male, female (%)	35 (32.1), 74 (67.9)					
Skin type: I, II, III, and IV (%)	8 (7.3), 60 (55.0), 40 (36.7), 1 (0.9)					
	<b>Minimum</b>	<b>Lower quartile</b>	<b>Median</b>	<b>Upper quartile</b>	<b>Maximum</b>	
Height (m)	1.49	1.63	1.68	1.78	1.98	
Weight (kg)	45.4	62.6	72.1	79.8	112.0	
BMI (kg m <sup>-2</sup> )	17.7	21.8	24.5	27.5	43.7	
Age (years)	20	27	35	47	60	
MED (mJ cm <sup>-2</sup> )	16.0	28.0	34.0	51.0	82.0	
	<b>Minimum</b>	<b>Lower quartile</b>	<b>Median</b>	<b>Upper quartile</b>	<b>Maximum</b>	<b>Normal range</b>
Serum biochemistry						
25(OH)D (ng ml <sup>-1</sup> )	3.1	11.9	15.6	22.3	38	See text
Parathyroid hormone (pmol l <sup>-1</sup> )	0.6	1.2	1.6	2.7	7.5	0.8–3.9
Sodium (mmol l <sup>-1</sup> )	133	140	141	143	148	132–144
Potassium (mmol l <sup>-1</sup> )	3.0	4.1	4.3	4.6	5.5	3.5–5.5
Urea (mmol l <sup>-1</sup> )	2.6	3.7	4.5	5.2	6.9	3.5–7.4
Creatinine (µmol l <sup>-1</sup> )	44	63	70	80	128	62–106
Calcium (mmol l <sup>-1</sup> )	2.09	2.19	2.24	2.28	2.44	2.15–2.65
Inorganic phosphorus (mmol l <sup>-1</sup> )	0.78	1.12	1.21	1.32	1.56	0.7–1.4
Alkaline phosphatase (U l <sup>-1</sup> )	25	49	57	69	172	35–105
Albumin (g l <sup>-1</sup> )	40	44	45	47	53	34–48
Alanine transaminase (U l <sup>-1</sup> )	4	9	11	16	64	5–40
Total Protein (g l <sup>-1</sup> )	67	72	75	77	91	60–80
Bilirubin (µmol l <sup>-1</sup> )	2	4	6	8	24	0–22
Iron (µmol l <sup>-1</sup> )	5.4	13	18.5	23.2	59	7–29
Average daily vitamin D intake (µg)	0.2	1.4	2.2	3.1	9.9	—

Abbreviations: BMI, body mass index; MED, minimal erythemal dose.

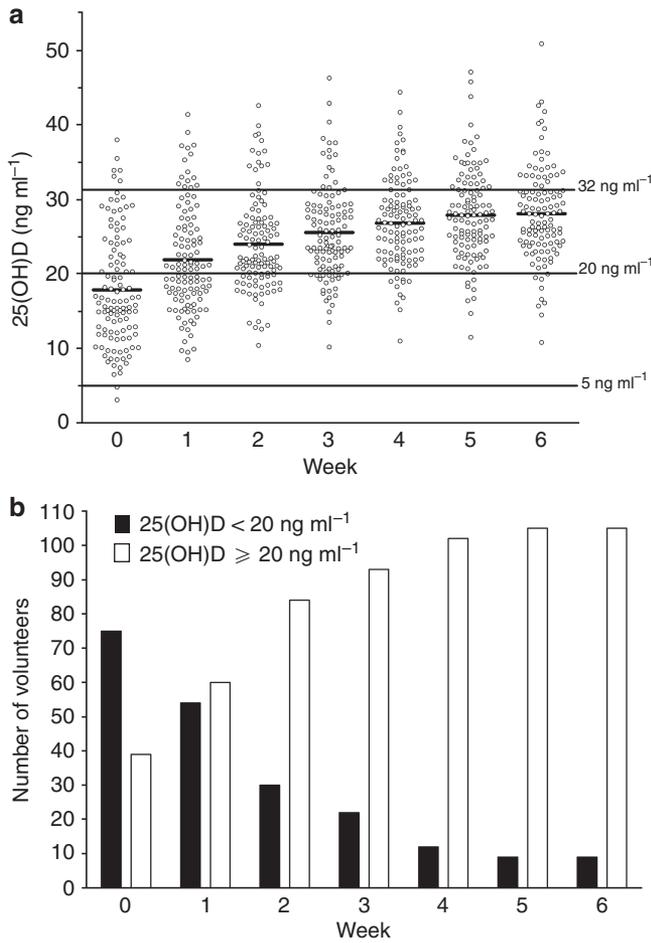
North American cities is calculated to be 9–16 minutes, compared with 13 minutes in Manchester, in midsummer (21 June; Table 4).

## DISCUSSION

Our data indicate that regular short midday exposures to summer sunlight while informally dressed would place 90% of the Greater Manchester, UK, white Caucasian nonelderly adult population in the vitamin D sufficiency range (25(OH)D  $\geq 20$  ng ml<sup>-1</sup>), and 26% in the proposed optimal range ( $\geq 32$  ng ml<sup>-1</sup>), whereas none would be in the range for deficiency that is currently defined by the UK Department of Health (<5 ng ml<sup>-1</sup>; 1998). Specifically, 13 minutes of midday sunlight exposure on a cloudless day, three times weekly, to 35% skin surface area over a 6-week summer period, is required to achieve these outcomes. These findings will apply across the majority of the UK population and to white Caucasian populations residing in countries positioned at similar latitude (50–60°N) when equivalent summer sunlight conditions prevail. Using knowledge of UVR action spectra for cutaneous vitamin D synthesis and of sunlight emission

spectra over different geographical conditions, the equivalent exposures required at a broader range of locations can also be estimated from our data (Table 4; Webb and Engelsen, 2006). Exposure times in the United Kingdom and elsewhere will vary with people's activities and the presence of shade.

An important consideration is the time of day at which individuals are exposed to summer sunlight, as this influences the amount of UVB available to generate vitamin D. Maximal amounts of UVB are available at solar noon, when the sun is directly overhead and solar radiation has the shortest path to the earth's surface, although in countries of mid latitude, such as the United Kingdom, UVB is insufficient to generate appreciable vitamin D even at midday from October to March (Webb and Engelsen, 2006). Thus, the amount of vitamin D generated from following current recommendations on summer sunlight exposure can range from maximal levels, such as in our study in which midday exposures were simulated, to minimal levels when people are exposed to sunlight only at other times during the day, as could arise depending on an individual's interpretation of the SunSmart advice. The SunSmart campaign, which is aware that UVB is



**Figure 1. Impact of simulated summer sunlight exposures on circulating 25(OH)D levels** (a) The 25(OH)D levels in the study group before the course of UVR, at weekly intervals during treatment, and at the end of the UVR course are displayed; mean values are displayed as horizontal lines. (b) Number of individuals displaying insufficient (<20 ng ml<sup>-1</sup>) and sufficient (≥20 ng ml<sup>-1</sup>) 25(OH)D levels.

**Table 2. Association of post-UV course 25(OH)D levels with participant baseline characteristics**

Factor	Estimate <sup>1</sup>	95% CI	P-value
Pre-treatment 25(OH)D	0.41	0.27–0.55	<0.0001
Age	-0.00	-0.10 to 0.10	0.94
Sex (F vs M)	0.32	-2.02 to 2.65	0.79
BMI	-0.18	-0.44 to 0.08	0.16
Log MED	-0.61	-3.15 to 1.93	0.63
Log dietary vitamin D	0.37	-0.28 to 1.03	0.22
Cohort (1 vs 2)	-1.31	-3.49 to 0.87	0.23

Abbreviations: BMI, body mass index; CI, confidence interval; MED, minimal erythral dose.

<sup>1</sup>Estimates are modeled differences in post-treatment 25(OH)D levels per unit increase in factor.

“seek shade” between 1100 and 1500 hours (<http://info.cancerresearchuk.org/healthyliving/sunsmart/>). However, no specific advice is given on skin surface area or duration of exposure. The 2002 Health Protection Agency report advises that short exposures to summer sunlight containing the requisite UVB (by implication at midday), several times per week, to limited skin areas, are sufficient for avoidance of the vitamin D deficiency complications of rickets and osteomalacia in a fair-skinned person in the United Kingdom (National Radiological Protection Board, 2002). Given more recent definitions of vitamin D status (Malabanan *et al.*, 1998; Hollis, 2005; Wolpowitz and Gilchrest, 2006), along with suggestions based on theoretical grounds of the greater UVR requirements that may be needed to reach this more elevated status (Holick, 2004), agencies are aware of the need to assess the impact of following their general advice on vitamin D status, and to more accurately define how much sunlight exposure is required to achieve vitamin D sufficiency. Thus, our controlled conditions of known UVR dose and skin surface area exposure have provided a quantitative assessment that can more accurately inform future guidance.

Although the majority of our population reached 25(OH)D levels designated sufficient immediately after the simulated summer sunlight exposures, only a minority reached the proposed optimal level of 32 ng ml<sup>-1</sup>. The increase in 25(OH)D levels was less than that reported in some previous studies involving UVR exposures, but in these studies, volunteers received near total skin surface exposure (Varghese *et al.*, 1989; Holick *et al.*, 2007; Thieden *et al.*, 2008), whereas our volunteers wore casual clothing to reveal areas of skin commonly exposed during leisure activities; this is important as it cannot be assumed that different skin sites are equally efficient in synthesizing vitamin D. In addition, both oral vitamin D supplements and ambient UVB exposure were excluded to avoid confounding of UVR treatment outcomes. We observed the weekly incremental increase in mean 25(OH)D levels to reduce steadily during the course, with increases of only 0.8 and 0.2 ng ml<sup>-1</sup> during the last two weeks, suggesting that levels would plateau if UVR exposures continued, in agreement with previous smaller studies (Porojnicu *et al.*, 2008; Thieden *et al.*, 2008). This may be attributable to photoadaptation through UVR-induced epidermal thickening and melanization. In addition, the sufficient levels attained through summer exposures are anticipated to fall during the UK winter months (Hypponen and Power, 2007; Hirani *et al.*, 2009). Our study revealed low baseline 25(OH)D levels, with 5% of the population in the deficiency range, and only 37.5 and 3% reaching sufficiency and optimal levels, respectively. Thus, the Greater Manchester population was representative of the wider UK population, a substantial proportion of which does not currently achieve a pattern of UVB exposure and oral intake conferring vitamin D sufficiency during winter (Hypponen and Power, 2007; Hirani *et al.*, 2009).

Sunburn is a risk factor for malignant melanoma and non-melanoma skin cancer (Elwood and Jopson, 1997; National Radiological Protection Board, 2002), and has a greater probability of occurring after exposure during midday hours,

**Table 3. Estimated percentage (95% CI) of the Greater Manchester, UK population exhibiting deficient, sufficient, and the proposed optimal circulating 25(OH)D levels**

Week of study	Deficient < 5 ng ml <sup>-1</sup>	Sufficient ≥ 20 ng ml <sup>-1</sup>	Proposed optimal ≥ 32 ng ml <sup>-1</sup>
0	5.0 (2.7–8.6)	37.5 (30.6–44.8)	2.9 (1.4–5.6)
1	0.9 (0.3–2.2)	59.0 (51.7–66.0)	6.9 (4.0–11.2)
2	0.2 (0.04–0.6)	72.7 (65.7–78.9)	10.5 (6.7–15.7)
3	0.1 (0.01–0.3)	80.8 (74.3–86.1)	14.7 (10.1–20.6)
4	0.01 (<0.01–0.1)	87.5 (82.0–91.7)	19.3 (13.9–25.7)
5	0.01 (<0.01–0.1)	89.8 (84.7–93.5)	25.4 (19.3–32.3)
6	0.01 (<0.01–0.1)	90.0 (84.9–93.7)	26.2 (20.1–33.2)

Abbreviation: CI, confidence interval.

**Table 4. Estimated time taken to acquire the same vitamin D-weighted dose as used in this study, at different North American and European locations at local noon on June 21 and December 21**

City	Latitude <sup>1</sup> (deg, min)	Summer <sup>2</sup> (minutes)	Winter <sup>2</sup> (minutes)
New Orleans	29, 57	9	39
San Diego	32, 42	9	49
Athens	37, 58	9	—
Washington	38, 53	9	—
Boston	42, 21	10	—
Vancouver	49, 13	11	—
Brussels	50, 52	12	—
Manchester	53, 30	13	—
Oslo	58, 57	16	—

<sup>1</sup>Latitude is given in degrees and minutes.

<sup>2</sup>Times are given to the nearest minute; times > 1 h are not shown.

that is, at the optimal time for vitamin D synthesis. We propose that future public health messages could promote regular short exposures to midday summer sunlight, their duration limited to below the sunburn threshold. Even suberythemal doses of UVR can cause DNA damage to skin cells (Young *et al.*, 1998); however, with sufficiently short exposures, the benefit/risk ratio is anticipated to be favorable for most individuals (Webb and Engelsen, 2006). Longer exposures do not provide further benefit, as cutaneous vitamin D production quickly reaches an equilibrium on UVR exposure due to photoisomerization of pre-vitamin D (Webb *et al.*, 1988), and vitamin D itself can be degraded (Webb *et al.*, 1989). However, attention to the skin surface area exposed is likely to be pivotal.

Individuals at high risk of developing skin cancer, including the immunosuppressed, and people with genetic susceptibility to, or previous history of, skin cancer will clearly need to continue to be advised to practice sunlight avoidance; oral supplements may be important in these individuals. Other sectors of the UK population known to be

at heightened risk of inadequate vitamin D status, including infants, the elderly, and people with pigmented skin, are currently advised to take vitamin D supplements (Department of Health, 1998).

In contrast, the recommended oral intake of vitamin D for healthy white nonelderly adults is low, that is, 5 µg (200 IU) per day, as advised by the WHO (2004) and by authorities in several European countries (Doets *et al.*, 2008), United States of America, and Canada (Institute of Medicine Food and Nutrition Board, 1997), whereas 0 µg day<sup>-1</sup> is advised in the United Kingdom (Department of Health, 1998), as the cutaneous route was assumed to fulfill vitamin D requirements. Evidence is growing that increased oral intake (food fortification/supplements) is required in the general population (Holick, 2006). This may apply even in situations in which people spend extended periods outdoors, as in a study of 30 men with extensive outdoor activities at 39–46.8°N, the elevated median 25(OH)D level of 50 ng ml<sup>-1</sup> at the end of summer was followed by wintertime levels of < 30 ng ml<sup>-1</sup> in half of the group (Barger-Lux and Heaney, 2002). The debate continues as to what represents a “normal” vitamin D status, with increasing literature supporting > 30–32 ng ml<sup>-1</sup> 25(OH)D as optimal (Bischoff-Ferrari *et al.*, 2006; Holick 2009), with proposals that levels below this could be defined as insufficient and < 20 ng ml<sup>-1</sup> as frankly deficient (Holick 2009). Should national/international authorities redefine 25(OH)D levels for vitamin D status, our data can be reinterpreted in the light of these, but it is evident that reevaluation of recommendations on oral vitamin D intake, in addition to sunlight exposure levels, would be required to provide 25(OH)D levels > 30–32 ng ml<sup>-1</sup> through the year in populations residing at higher latitudes.

In conclusion, we tested UK policy recommendations on vitamin D acquisition in healthy white Caucasian nonelderly adults, which advise that satisfactory vitamin D status is achieved solely through the cutaneous route, after casual short summer sunlight exposures. We found that the majority of the population reaches a 25(OH)D level that is designated as sufficient (≥ 20 ng ml<sup>-1</sup>), but not the proposed optimal level (≥ 32 ng ml<sup>-1</sup>), after a simulated summer's sunlight exposures, specifically 13 minutes of midday sun to 35% skin surface area, three times weekly for 6 weeks; these *in vivo*

findings will assist future public health advice on vitamin D acquisition. Further research should establish the 25(OH)D level required for optimal health, whether this should be maintained throughout the year, and if so, the best strategy (sunlight and dietary) for achieving this.

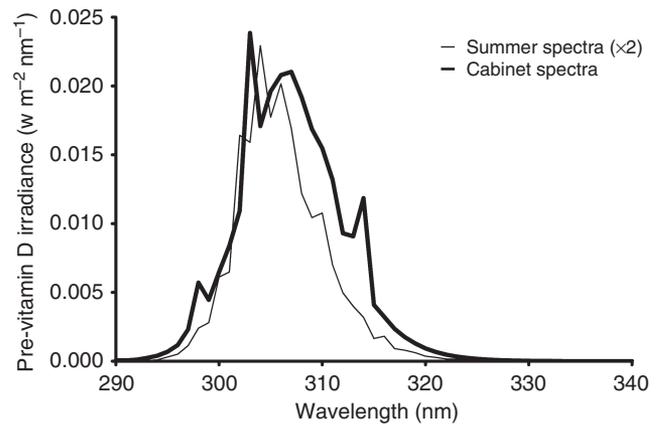
## MATERIALS AND METHODS

### Volunteers

Ethical approval was obtained from the North Manchester Research Ethics Committee (reference 06/Q1406/6). Written informed consent was obtained from the participants and the study adhered to the Declaration of Helsinki Principles. Volunteers ( $n=120$ ) were white Caucasians, sun-reactive skin types I–IV, aged 20–60 years, from Greater Manchester, UK. The exclusion criteria were pregnancy, breastfeeding, taking vitamin D supplements or photoactive medication, history of skin cancer, photosensitivity or systemic lupus erythematosus, and use of a sunbed or sunbathing in the 3 months before the commencement of the study or during the study. For logistical reasons, 60 subjects participated in January–February 2007 and 60 in January–February 2008.

### Simulated summer sunlight exposures

A 6-week course of UVR exposures was selected to be concordant with the length of the summer school holiday period when the population is most exposed to sunlight. Subjects were exposed to a constant UVR dose three times weekly using a whole body irradiation cabinet (Philips HB598, Eindhoven, The Netherlands) fitted with a combination of Arimed B (Cosmedico GmbH, Stuttgart, Germany) and Cleo Natural (Philips, Eindhoven, The Netherlands) fluorescent tubes, that is, lamps with UVR emission spectrum similar to sunlight (emission 290–400 nm, 95% UVA: 320–400 nm, 5% UVB: 290–320 nm). This was characterized using a Bentham DTM 300 spectroradiometer (Bentham, Reading, UK) and monitored using an Ocean Optics S2000 spectroradiometer (Ocean Optics, Dunedin, FL). Volunteers wore standardized T-shirts and knee-length shorts to expose approximately 35% skin surface area. The UVR course was given in January and February when ambient UVB is negligible at UK latitudes (50–60°N; Webb and Engelsens, 2006). A UVR exposure of 1.3 SED (Diffey *et al.*, 1997), equivalent to 1.1 SED in sunlight, was given to each subject at every visit, after a pilot study showed that this dose increased 25(OH)D levels without causing skin erythema. The time required to deliver the dose was found to be 6.5 minutes after accurate measurement of cabinet UV irradiance (Taylor *et al.*, 2002); a constant UVR dose was maintained throughout the study by adjusting for any decrease in irradiance by increasing delivery time. The pre-vitamin D irradiance dose for one exposure in the cabinet was equivalent to 13 minutes of sunlight exposure on a clear June midday in Manchester, UK (53.5°N). Although the UVR spectra of the treatment cabinet and that of a clear summer (June) midday differ, the pre-vitamin D irradiance of the lamps (calculated by multiplying the cabinet irradiance with the 7-dehydrocholesterol to pre-vitamin D conversion action spectrum; MacLaughlin *et al.*, 1982), that is, the biologically relevant quantity, is very similar to, although approximately twice, that of a clear June day in Manchester at noon (Figure 2). Thus, our simulation was designed to mimic short (13 minutes) unshaded midday exposures while wearing informal clothing, three times weekly, over the summer holiday period.



**Figure 2. Pre-vitamin D irradiance emission from the irradiation cabinet versus that of a clear Manchester, UK (53.5°N) summer day at noon.** The pre-vitamin D irradiance emission from the Phillips HB598 whole body cabinet (bold line) is spectrally comparable to, and approximately twice as high as, the solar emission (regular line, shown twice the value) on a clear Manchester, UK summer day at noon. At wavelengths > 340 nm, the radiation is not effective at producing pre-vitamin D and, therefore, is not a concern for this investigation.

### Minimal erythema dose assessment

Each subject's MED was assessed before treatment. A geometric series of 10 doses (13–128 mJ cm<sup>-2</sup>) of erythemally weighted UVR was applied over two horizontal rows to buttock skin using a Waldmann UV 236 B unit with Waldmann CF-L 36W/UV6 lamps (Waldmann GmbH, Villingen-Schwenningen, Germany; peak emission 313 nm, range: 290–400 nm). The MED value was defined as the lowest dose of UVR to result in visually discernible erythema at 24 h.

### Diet questionnaires

To estimate approximate oral vitamin D intake during first and last weeks of the study, volunteers completed daily diet questionnaires regarding vitamin D-fortified foods and six food categories: cheese; butter, margarine and other oily spreads; milk and milk-containing products; red meat; oily fish; and eggs and egg dishes. The vitamin D content of foodstuffs was obtained from the 5th edition and integrated data set of McCance and Widdowson's *The Composition of Food* (Holland *et al.*, 1991; The Food Standards Agency, 2002), and from food package labeling.

### Vitamin D, PTH, and serum biochemistry

Blood samples were taken weekly, and serum stored at -20°C until completion of the study. Serum 25(OH)D was measured by high-pressure liquid chromatography as reported previously (Berry *et al.*, 2007). The laboratory is accredited to ISO 9001:2000 and ISO 13485:2003 standards, and participates successfully in the national Vitamin D quality assurance scheme (DEQAS). Serum PTH level was measured before the commencement of course of UVR exposure, using the OCEIA immunoenzymometric assay, following the manufacturer's instructions (Immunodiagnostic Systems, Boldon, Tyne and Wear, UK), with sensitivity 0.06 pmol l<sup>-1</sup> and intra- and interassay coefficients of variation 4 and 6%, respectively. Serum biochemistry was measured using the Hitachi 917 autoanalyser (Hitachi, Tokyo, Japan) before UVR treatment, including renal and liver function tests.

### Definition of vitamin D levels

The definitions used in this study are as follows: circulating levels of 25(OH)D below  $5 \text{ ng ml}^{-1}$  ( $12.5 \text{ nmol l}^{-1}$ ) are defined as deficient; below  $20 \text{ ng ml}^{-1}$  ( $50 \text{ nmol l}^{-1}$ ) as insufficient; equal to or greater than  $20 \text{ ng ml}^{-1}$  as sufficient; below  $32 \text{ ng ml}^{-1}$  ( $80 \text{ nmol l}^{-1}$ ) as "sub-optimal"; and  $32 \text{ ng ml}^{-1}$  and above as optimal.

### Outcome measures

Our primary outcome measure was the percentage of the population reaching circulating 25(OH)D levels designated sufficient ( $\geq 20 \text{ ng ml}^{-1}$ ) after a simulated summer exposure. Other outcomes were the percentages of the population reaching 25(OH)D levels proposed to be optimal for health ( $\geq 32 \text{ ng ml}^{-1}$ ) after exposure, and percentages exhibiting deficient ( $< 5 \text{ ng ml}^{-1}$ ), sufficient, and proposed optimal levels during the baseline winter assessment.

### Calculation of the time to acquire the same vitamin D-weighted dose as used in this study, at different European and North American locations

Time taken to acquire the same vitamin D-weighted dose as used in this study was calculated for a range of European and US locations at local noon time on 21 June and 21 December. Calculations (<http://nadir.nilu.no/~olaeng/fastrt/fastrt.html>) used constant atmospheric conditions (cloudless sky, visibility 25 km, ozone 350 DU, albedo 0.05, and altitude 15 m). Although not exact for any site, these conditions are representative of those found at the locations. Dose rates were calculated using the CIE action spectrum for synthesis of pre-vitamin D in human skin (MacLaughlin *et al.*, 1982). To achieve the same change in vitamin D status as that in our volunteers, characteristics similar to this study, including skin type, manner of dress (skin surface revealed), and regimen of regular noontime exposures, are required.

### Sample size and statistical analyses

As the minimum recommended sample size for estimating the population SD value is 100 (Altman, 1991), we recruited 120 volunteers, allowing for a 15% dropout rate. Estimates and CI values for the proportions above and below the threshold values for population 25(OH)D levels were calculated from the weekly observed means and SD values by assuming normality. Factors associated with posttreatment 25(OH)D values were assessed using multiple linear regression. Where appropriate, variables were transformed to satisfy normality assumptions for regression.

### CONFLICT OF INTEREST

L.E.R. has acted as adviser to the SunSmart campaign. Other authors state no conflict of interest.

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