

## Short Communication

## Sun exposure, UV irradiance and serum 25-hydroxycholecalciferol in pregnant women in rural north India

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Submitted 29 July 2016: Final revision received 14 September 2016: Accepted 11 October 2016: First published online 19 December 2016

**Abstract***Objective:* To document the effect of season and environmental pollution on UVB irradiance; and to estimate cutaneous vitamin D synthesis in village women in different seasons.*Design:* Radiant UVB energy was measured by a spectroradiometer in different seasons and, in April and May, on successive days in open areas at the city outskirts, at a crowded inner-city area and the villages of our participants. Clothing, outdoor activity pattern and serum 25-hydroxycholecalciferol (25(OH)D) levels were documented.*Setting:* Rural north India, latitude 26.8°N.*Subjects:* Pregnant women (*n* 139, aged 20–40 years).*Results:* UVB irradiance ranged from 56 μW/cm<sup>2</sup> in January to 470 μW/cm<sup>2</sup> in June. Proportion of skin exposed was 18.5% in summer and 9.5% in winter. Mean (SD) daily duration of sun exposure was 3.2 (0.2) h during winter and 2.1 (0.4) h during summer. Cutaneous vitamin D synthesis was estimated to be 19.25 μg (770 IU) during winter and 37.25 μg (1490 IU) during summer. Mean (SD) serum 25(OH)D was 28 (15) nmol/l during winter (92% of participants with <50 nmol/l) and reached 56 (20) nmol/l during late summer (60% with >50 nmol/l). Mean (SD) UVB irradiance at peak summer was significantly higher at the open areas and in the villages than at the inner-city location (340 (45) and 310 (60) *v.* 250 (50) μW/cm<sup>2</sup>, *P*=0.03).*Conclusions:* In our population, at latitude 26.8°N, poor skin exposure is a limiting factor in all seasons. During winter, low UVB radiation energy also contributes. Particulate pollution limits UVB irradiance. Vitamin D supplementation during winter may be necessary.**Keywords**UV radiation energy  
Standard erythemal dose  
Season  
Pollution

There is a high prevalence of vitamin D deficiency reported in India: ranging from 75 to 90% of the population, in all age groups including pregnant women and their newborns, infants, adolescents and adults<sup>(1–4)</sup>; from north<sup>(1,2,5–7)</sup> and south India<sup>(3,5)</sup>; in urban<sup>(1–3,6)</sup> and rural<sup>(4)</sup> populations; and higher as well as lower socio-economic populations<sup>(6)</sup>. The reasons for this high prevalence, despite India being a tropical country, include low body surface area exposed to sun (due to the traditional, modest pattern of clothing and poor outdoor activity), skin pigmentation, dietary Ca deficiency and, possibly, particulate pollution. Goswami *et al.*<sup>(8)</sup>

emphasized the role of duration of sunshine exposure and skin pigment in their study of 25-hydroxycholecalciferol (25(OH)D) levels among soldiers, physicians, nurses, pregnant women and people with depigmented skin. All groups had suboptimal 25(OH)D levels except the soldiers and people with depigmented skin.

There is no study on rural subjects in India, correlating their direct UVB irradiance exposure in all seasons of the year and serum 25(OH)D levels. Such a study would help public health policy planners to know whether sunshine can be realistically expected to be an adequate source of

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vitamin D for Indians and, if so, in which months of the year.

We documented some of the limiting factors for cutaneous vitamin D synthesis in a group of pregnant rural women by measuring the magnitude of UVB energy exposure and serum 25(OH)D in different seasons throughout the year. We also analysed the differences in radiant UVB energy in regions with differing amounts of particulate pollution.

## Materials and methods

### Study population and region

The study population was located in a rural area about 40 km from the city of Lucknow (latitude 26.8°N, longitude 80.9°E, altitude 128 m). Lucknow has three distinct seasons, namely winter (November–January), monsoon (July, August) and dry summer in the rest of the months. Pregnant women in the second trimester ( $n$  139; mean gestational age 22 (SD 5) weeks) were recruited consecutively over one year. They belonged to poor socio-economic status and resided in huts or poorly ventilated houses of one or two rooms. The daily activity included household chores which were mostly performed in the courtyard and farm work during the seasons of farming. Outdoor activity and clothing were uniform for a season and dictated by the weather and necessities of farming. The body surface area exposed during the winter included the face, neck and the hands, and during the rest of the year it also included the forearms. We compared the skin colour tone of the face with a skin shade chart based on the Fitzpatrick classification<sup>(9)</sup>. They all had skin type V. The mean daily Ca intake was low (214 (SD 150) mg) due to poor consumption of milk products (due to economic constraints), despite an effort made by families to provide more milk to pregnant women. A log of daily activities and clothing was recorded by the research staff once weekly throughout the year. The outdoor exposure was estimated between 10.00 and 14.00 hours, which included the peak hours of UV radiation exposure. Mean exposure time was 3.2 (SD 0.2) h/d during winter season (November–January) and 2.1 (SD 0.4) h/d during summer (February–October). The proportion of the body surface area exposed was 18.5% in summer and 9.5% in winter.

The study was approved by the Institutional Ethics Committee of Sanjay Gandhi Postgraduate Institute of Medical Sciences (2003). Verbal informed consent was obtained from all participants. Verbal consent was witnessed and formally recorded.

### Measurement of UVB radiant energy

A portable UVB light meter (a silicon carbide photodiode with a 290–320 nm UVB filter) was used to measure the UVB radiation in millivolts. (Surface UVB energy peaks at 310 nm while the UVB energy directly related to vitamin D production peaks at 300–305 nm.) The instrument was

designed, assembled and calibrated by one of the authors (P.U.). A silicon carbide detector made by Laser Components, UK (model JEC 0.3B) was used. The meter was calibrated by comparison with the integrated irradiance measured by a spectroradiometer which was calibrated by reference to a quartz–tungsten–halogen lamp irradiance standard against UK National Physical Laboratory standards. The calibration factor was 13  $\mu\text{W}/\text{cm}^2$  per mV. The estimated uncertainty corresponded to approximately  $\pm 15\%$ . The angular field of view was 180°. A total of 474 UVB readings were taken at three locations, namely our hospital campus situated in the outskirts of the city, at a crowded city centre and in the villages of residence of our participants. Measurements were taken at different times of day between 09.00 and 16.00 hours, in all seasons of the year. During April and May (a time of dry heat with no cloud cover), readings taken at any one location on a particular day were then immediately accompanied by readings at the other two locations on successive days, so as to have the same environmental conditions during each set of three days. Similarly, to document the effects of clouds, during the monsoon season of July and August, consecutive cloudy and sunny day readings were taken.

### Calculation of estimated cutaneous vitamin D formation

We employed the formula of Godar *et al.*<sup>(10)</sup>, which takes the following variables into account: standard erythemal dose (SED)<sup>(11)</sup>, a reflection of UVB energy received per hour; action spectrum conversion factor (ASCF)<sup>(12)</sup>, a correction factor for solar zenith angle; geometric conversion factor (GCF)<sup>(13)</sup>, a correction factor for geometrical body shape; skin type factor (STF), which accounts for relative efficiency of vitamin D synthesis for the given UVB energy depending upon the Fitzpatrick skin type<sup>(14)</sup>; age of the participants (AF); and fractional body surface area exposure (FBE)<sup>(15)</sup>. The formula employed is:

$$\begin{aligned} \text{Cholecalciferol (vitamin D}_3\text{) production (IU/d)} \\ = \text{SED/d} \times \text{ASCF} \times \text{GCF} \times 4900 \text{ IU for skin type II} \\ \times \text{STF} \times \text{FBE} \times \text{AF.} \end{aligned}$$

The production of 122.5  $\mu\text{g}$  (4900 IU) vitamin D per SED was taken from experiments of Holick<sup>(16)</sup>. Furthermore, after certain duration of UVB energy exposure, there is cessation of cutaneous precholecalciferol formation and diversion to inactive metabolites such as lumisterol and tachysterol occurs. Various studies have shown a wide range of erythemal energy cut-off ranging from 0.5 to 1.5 minimal erythemal dose (MED), where cutaneous 7-dehydrocholesterol to precholecalciferol conversion plateaus off for various skin types<sup>(17–19)</sup>. Taking 1 MED (750 J/m<sup>2</sup> for type V skin) threshold for our calculation, we capped the SED threshold for maximum vitamin D production in type V skin as 7.5 SED/d<sup>(10,20)</sup> because 1 SED is equivalent to 100 J/m<sup>2</sup>.

### Serum 25-hydroxycholecalciferol

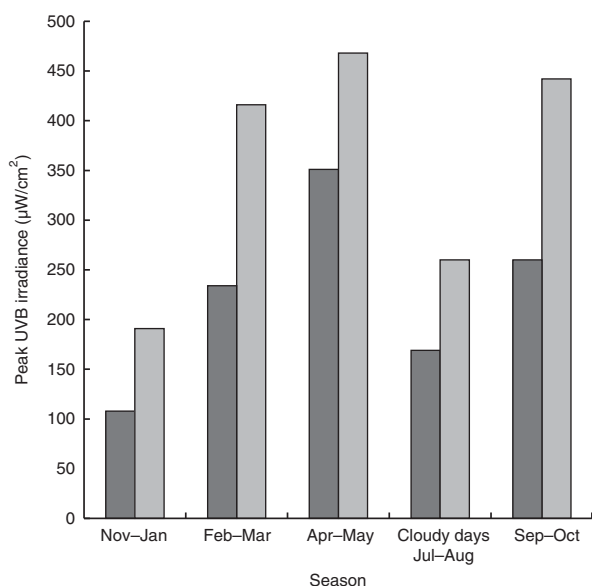
Participants were recruited during the different seasons throughout the year. Blood was sampled for analysis of serum 25(OH)D using a RIA kit (Diasorin, Stillwater, MN, USA; analytical sensitivity: 3.75 nmol/l, intra-assay CV: 8.6–12.5%, inter-assay CV: 8.2–11.0%). Vitamin D deficiency was defined as serum 25(OH)D < 50 nmol/l.

## Results

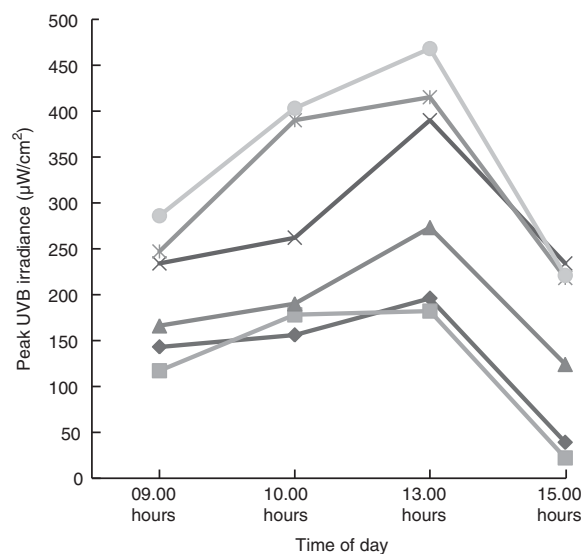
### Surface irradiance: variation with season, time of day and location

Variation in UVB energy with the time of day, season and cloud cover is shown in Figs 1 and 2. Peak readings (between 12.00 and 13.00 hours) were at their lowest during the winter season from November to January and were at their highest during the summer season in May and June. The presence of clouds in the sky during the rainy season was associated with reduced radiant UVB energy.

Peak UVB readings during April and May, between 12.00 and 13.00 hours, were taken at the hospital campus, the crowded inner-city location and the village areas. A total of eighteen UV energy readings (six readings at each location) at similar times at all three locations were taken on consecutive days. Significantly higher surface UVB irradiance was recorded in the hospital campus (mean 340 (SD 45)  $\mu\text{W}/\text{cm}^2$ ) and in village locations (mean 310 (SD 60)  $\mu\text{W}/\text{cm}^2$ ) than in the crowded inner-city location (mean 250 (SD 50)  $\mu\text{W}/\text{cm}^2$ ;  $P=0.024$  for multiple group comparison,  $P=0.03$  on *post hoc* comparison between the hospital campus *v.* inner-city location).



**Fig. 1** Peak UVB irradiance measured at 10.00 hours (■) and 13.00 hours (□) at different seasons of the year in Lucknow, India (latitude 26.8°N)



**Fig. 2** Peak UVB irradiance measured at different times of day during different months of the year (—◆—, November; —■—, January; —\*—, April; —●—, May; —▲—, August (cloudy); —×—, August (sunny)) in Lucknow, India (latitude 26.8°N)

### Average daily vitamin D synthesis in rural women

Using the method of Godar *et al.*, the rural women's average daily vitamin D synthesis during the winter season was calculated to be 19.25  $\mu\text{g}/\text{d}$  (770 IU/d) and 37.25  $\mu\text{g}/\text{d}$  (1490 IU/d) during summer.

### Serum 25-hydroxycholecalciferol

Mean serum 25(OH)D was 28 (SD 15) nmol/l in women tested during winter ( $n=48$ ) and 92% of them had vitamin D deficiency (25(OH)D < 50 nmol/l). During the rest of the year, mean serum 25(OH)D was 42 (SD 20) nmol/l ( $n=91$ ) and 70% of the women had vitamin D deficiency (25(OH)D < 50 nmol/l). The highest 25(OH)D values were obtained in women sampled in the months of July and August (mean 56 (SD 20) nmol/l) and 60% had values > 50 nmol/l.

We did not find a significant difference in mean serum 25(OH)D levels between groups of women in early second trimester ( $\leq 20$  weeks; 37.5 (SD 20) nmol/l) and late second trimester ( $> 20$  weeks; 38 (SD 18) nmol/l;  $P=0.9$ ). The mean BMI of our participants was 21 (SD 2.2)  $\text{kg}/\text{m}^2$  and none of them had BMI  $\geq 27.0$   $\text{kg}/\text{m}^2$  (the obesity cut-off for Indians). Twenty-one participants had BMI  $\geq 23.0$   $\text{kg}/\text{m}^2$  (the overweight cut-off for Indians). There was no significant difference in mean serum 25(OH)D levels between groups of women with BMI < 23.0  $\text{kg}/\text{m}^2$  (39 (SD 18) nmol/l) and BMI  $\geq 23.0$   $\text{kg}/\text{m}^2$  (36 (SD 23) nmol/l;  $P=0.8$ ).

## Discussion

Our results demonstrate that in the summer months, the cutaneous production of vitamin D is equivalent to the daily ingestion of 37.25  $\mu\text{g}$  (1490 IU) of cholecalciferol. Going by

the findings of numerous studies in the literature as well as the recommendations of international expert groups, this amount should be sufficient to maintain serum 25(OH)D above 50 nmol/l<sup>(21,22)</sup>. Similar findings have been reported by other investigators studying *in vivo* cutaneous vitamin D production in South Asian individuals<sup>(23,24)</sup>. After 6 weeks of exposure of 35% body surface area, three times weekly, to 3.25 and 3.9 SED UVB energy, Farrar *et al.*<sup>(23)</sup> documented a rise in serum 25(OH)D to 23.8 and 31.5 nmol/l, respectively. Studying the effect of 30 min of natural sun exposure of 15–30% of body surface area in Indian children every day for 4 weeks, Marwaha *et al.*<sup>(24)</sup> documented a rise in serum 25(OH)D from 23 (SD 14) to 33 (SD 17) nmol/l. Extrapolating to a longer daily exposure and a longer duration of study would give a post-exposure value comparable to our summer peak 25(OH)D. A web-based calculator of vitamin D formation per skin type, latitude, body surface area exposed and exposure time also gives similar results of vitamin D production as in our study<sup>(25)</sup>.

If type V skin can produce such a generous amount of vitamin D, why do 70% of the women in our study still have 25(OH)D < 50 nmol/l in serum? First, it must be borne in mind that daily doses of vitamin D, when ingested orally, take about 3 months to reach a plateau level in blood<sup>(26)</sup>. A similar observation was documented with *in vivo* production in South Asians in response to UVB exposure for 6 weeks. Weekly measurements of 25(OH)D showed rising 25(OH)D even at 6 weeks, especially in the higher SED exposure group<sup>(23)</sup>. These studies support our observation that the highest 25(OH)D values were obtained in the months of July and August (mean 56 (SD 20) nmol/l; 60% of participants having values > 50 nmol/l). It suggests that the severe depletion in our women during the winter months (mean 25(OH)D being 28 (SD 15) nmol/l) takes time to be compensated during the summer months of April, May and June. Just after it reaches a plateau, the cutaneous vitamin production is again hampered by the monsoon season and the following winter season, thus rendering our participants vitamin D-deficient for many months of the year. Marwaha *et al.*<sup>(27)</sup> found a significant decrease in mean 25(OH)D levels during winter despite sun exposure. Second, a very important role is that of degradation of precholecalciferol into suprasterol I and II on prolonged exposure to sunlight<sup>(28)</sup>. This is nature's mechanism of preventing vitamin D toxicity. This mechanism of degradation of precholecalciferol on prolonged UVB exposure in our participants may partly explain their low observed 25(OH)D levels. All these studies reaffirm that it is the body surface area exposed which is the major limiting factor in our population. Third, our participants had an extremely low dietary Ca intake, which has been shown in both in human subjects and animal models to be associated with secondary hyperparathyroidism leading to depletion of vitamin D stores<sup>(29–31)</sup>. Fourth, the recent finding of genetic variation in the level of

vitamin D-binding protein in those of African ancestry suggests that this should be explored in Indians<sup>(32)</sup>.

Our studies on the comparison of the UVB irradiance in polluted *v.* non-polluted locations corroborate the findings of Agarwal *et al.*, who also found lower irradiance in a crowded location full of vehicular traffic as compared with the countryside<sup>(33)</sup>. Particulate pollution is well known to block penetration by solar UV rays.

Our study has some limitations. The study was not longitudinal as each participant was not tracked through a whole year. Second, we did not have an objective measure of a personal UV meter to track the exposure time of each individual. The serum 25(OH)D values were not measured in all trimesters (although it must be mentioned that serum 25(OH)D levels have not been found to be statistically different between trimesters in a previous publication from north India<sup>(34)</sup>). Furthermore, our analysis may not be extrapolated to non-pregnant women. Despite these limitations, however, we think our study allows us to conclude that cutaneous vitamin D synthesis at latitude 26.8°N in our rural women is limited more by body surface area exposed to the sun than by duration of exposure, especially during winter when it is compounded by poorer UVB irradiance. The degree of vitamin D deficiency can be expected to be more pronounced in urban people as they have even less outdoor exposure and equally poor body surface area exposure. Particulate pollution plays an important role in limiting UVB irradiance in crowded city centres in India. It may be necessary for policy makers to recommend universal preventive supplementation at least during the winter months.

## Acknowledgements

*Acknowledgements:* Technical help in the hormone laboratory from P.K. Awasthi and in the rural area from Arjun Singh is gratefully acknowledged. *Financial support:* This work was supported by the Department of Biotechnology, Government of India (V.B., grant number DBT/PR/3552/SPD/11/349/2002). The Department of Biotechnology, Government of India, had no role in the design, analysis or writing of this article. *Conflict of interest:* The authors declare no conflict of interest. *Authorship:* S.S. acquired the data, analysed the data and wrote the manuscript; P.U. conceptualized the study, standardized the methodology, analysed the data and approved the manuscript; M.S. planned the study, acquired the data and approved the manuscript; V.R. acquired the data and approved the manuscript; V.B. conceptualized, planned and executed the study, analysed the data and approved the manuscript. *Ethics of human subject participation:* This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects were approved by the ethics committee of Sanjay Gandhi Post-graduate Institute of Medical Sciences. Verbal informed



consent was obtained from all participants. Verbal consent was witnessed and formally recorded.

## References

- Sachan A, Gupta R, Das V *et al.* (2005) High prevalence of vitamin D deficiency among pregnant women and their newborns in northern India. *Am J Clin Nutr* **81**, 1060–1064.
- Seth A, Marwaha RK, Singla B *et al.* (2009) Vitamin D nutritional status of exclusively breast fed infants and their mothers. *J Pediatr Endocrinol Metab* **22**, 241–246.
- Balasubramanian S, Shivbalan S & Kumar PS (2006) Hypocalcemia due to vitamin D deficiency in exclusively breast fed infants. *Indian Pediatr* **43**, 247–251.
- Sahu M, Bhatia V, Aggarwal A *et al.* (2008) Vitamin D deficiency in rural girls and pregnant women despite abundant sunshine in northern India. *Clin Endocrinol (Oxf)* **70**, 680–684.
- Harinarayan CV (2005) Prevalence of vitamin D insufficiency in postmenopausal south Indian women. *Osteoporos Int* **16**, 397–402.
- Marwaha RK, Tandon N, Reddy DR *et al.* (2005) Vitamin D and bone mineral density status of healthy schoolchildren in northern India. *Am J Clin Nutr* **82**, 477–482.
- Zargar AH, Ahmad S, Masoodi SR *et al.* (2007) Vitamin D status in apparently healthy adults in Kashmir Valley of Indian subcontinent. *Postgrad Med J* **83**, 713–716.
- Goswami R, Gupta N, Goswami D *et al.* (2000) Prevalence and significance of low 25-hydroxyvitamin D concentrations in healthy subjects in Delhi. *Am J Clin Nutr* **72**, 472–475.
- Fitzpatrick TB (1988) The validity and practicality of sun-reactive skin types I through VI. *Arch Dermatol* **124**, 869–871.
- Godar DE, Pope SJ, Grant WB *et al.* (2012) Solar UV doses of young Americans and vitamin D<sub>3</sub> production. *Environ Health Perspect* **120**, 139–143.
- International Commission on Illumination (1999) *Erythema Reference Action Spectrum and Standard Erythema Dose*. Joint ISO/CIE Standard, ISO 17166:1999(E)/CIE S 007-1998. Vienna: CIE.
- Pope SJ, Holick MF, Mackin S *et al.* (2008) Action spectrum conversion factors that change erythemally weighted to previtamin D<sub>3</sub>-weighted UV doses. *Photochem Photobiol* **84**, 1277–1283.
- Model C, Pope SJ & Godar DE (2010) Solar UV geometric conversion factors: horizontal plane to cylinder model. *Photochem Photobiol* **86**, 457–466.
- Godar DE, Pope SJ, Grant WB *et al.* (2011) Solar UV doses of adult Americans and vitamin D<sub>3</sub> production. *Dermatoendocrinology* **3**, 243–250.
- Lund CC & Browder NC (1944) The estimation of areas of burns. *Surg Gynecol Obstet* **79**, 352–358.
- Holick MF (1995) Environmental factors that influence the cutaneous production of vitamin D. *Am J Clin Nutr* **61**, 3 Suppl., S638–S645.
- Holick MF, MacLaughlin JA & Doppelt SH (1981) Regulation of cutaneous previtamin D<sub>3</sub> photosynthesis in man: skin pigment is not an essential regulator. *Science* **211**, 590–593.
- Matsuoka LY, Wortsman J, Haddad JG *et al.* (1989) *In vivo* threshold for cutaneous synthesis of vitamin D<sub>3</sub>. *J Lab Clin Med* **114**, 301–305.
- Holick MF (1981) The cutaneous photosynthesis of previtamin D<sub>3</sub>: a unique photoendocrine system. *J Invest Dermatol* **76**, 51–58.
- Tejasvi T, Sharma VK & Kaur J (2007) Determination of minimal erythral dose for narrow band-ultraviolet B radiation in north Indian patients: comparison of visual and dermaspectrometer readings. *Indian J Dermatol Venereol Leprol* **73**, 97–99.
- Vogiatzi MG, Jacobson-Dickman E & DeBoer MD (2014) Vitamin D supplementation and risk of toxicity in pediatrics: a review of current literature. *J Clin Endocrinol Metab* **99**, 1132–1141.
- Holick MF, Binkley NC, Bischoff-Ferrari HA *et al.* (2011) Evaluation treatment and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab* **96**, 1911–1930.
- Farrar MD, Webb AR, Kift R *et al.* (2013) Efficacy of a dose range of simulated sunlight exposures in raising vitamin D status in South Asian adults: implications for targeted guidance on sun exposure. *Am J Clin Nutr* **97**, 1210–1216.
- Marwaha RK, Sreenivas V, Talwar D *et al.* (2015) Impact of solar ultraviolet B radiation (290–320 nm) on vitamin D synthesis in children with type IV and V skin. *Br J Dermatol* **173**, 604–606.
- Webb AR & Engelsen O (2006) Calculated ultraviolet exposure levels for a healthy vitamin D status. *Photochem Photobiol* **82**, 1697–1703.
- Heaney RP, Davies KM, Chen TC *et al.* (2003) Human serum 25-hydroxycholecalciferol response to extended oral dosing with cholecalciferol. *Am J Clin Nutr* **77**, 204–210.
- Marwaha RK, Yenamandra VK, Sreenivas V *et al.* (2016) Regional and seasonal variations in ultraviolet B irradiation and vitamin D synthesis in India. *Osteoporos Int* **27**, 1611–1617.
- Webb AR, de Costa B & Holick MF (1989) Sunlight regulates the cutaneous production of vitamin D<sub>3</sub> by causing its photodegradation. *J Clin Endocrinol Metab* **68**, 882–887.
- Clements MR, Johnson L & Fraser DR (1987) A new mechanism for induced vitamin D deficiency in calcium deprivation. *Nature* **325**, 62–65.
- Thacher TD, Fischer PR, Pettifor JM *et al.* (1999) A comparison of calcium, vitamin D, or both for nutritional rickets in Nigerian children. *N Engl J Med* **341**, 563–568.
- Harinarayan CV, Ramalakshmi T, Prasad UV *et al.* (2007) High prevalence of low dietary calcium, high phytate consumption, and vitamin D deficiency in healthy south Indians. *Am J Clin Nutr* **85**, 1062–1067.
- Powe CE, Evans MK, Wenger J *et al.* (2013) Vitamin D binding protein and vitamin D status of black Americans and white Americans. *N Engl J Med* **369**, 1991–2000.
- Agarwal KS, Mughal MZ, Upadhyay P *et al.* (2002) The impact of atmospheric pollution on vitamin D status of infants and toddlers in Delhi, India. *Arch Dis Child* **87**, 111–113.
- Marwaha RK, Tandon N, Chopra S *et al.* (2011) Vitamin D status in pregnant Indian women across trimesters and different seasons and its correlation with neonatal serum 25-hydroxyvitamin D levels. *Br J Nutr* **106**, 1383–1389.