# Vitamin D and ultraviolet radiation exposure – a photodermatologist's viewpoint.

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**Abstract**. Vitamin D is essential for bone health and possibly various other health outcomes. It is available following ultraviolet (UV)-B (280-315nm) irradiation of skin, or oral ingestion. Careful sunscreen use of sun protection factors (SPFs) 18-19 during such exposure reduces blood level increases on sunny holidays by up to about 50%, but these increases remain significant. Further, the maintenance of such levels requires unprotected exposures of only a fraction of the sunburn dose over small body areas. Finally, skin colour, whether fair or dark, has no effect on these levels. Unprotected UVB exposure induces potentially precancerous skin thymine dimers and is not advocated to maintain vitamin D status, but rather protected exposure or else oral supplementation. Minimal skin UVB exposure seems an innate requirement for not only vitamin D production, but also contact dermatitis and polymorphic light eruption avoidance, and anti-bacterial defensin induction.

## Discussion

Vitamin D, not in fact a vitamin but a corticosteroid hormone, is essential for the maintenance of calcium and phosphate homeostasis and optimal bone and muscle health. Low levels may lead to rickets in children and osteomalacia in adults, though probably not osteoporosis (Reid et al, 2014). Frequent claims of links between low vitamin D levels and other health problems such as colorectal cancer, cardiovascular disease and all-cause mortality are also made, but generally felt not adequately supported yet by research evidence. However, as vitamin D has important functions in most cells, such claims remain worthy of continuing investigation.

Vitamin D is acquired through the action of solar or artificially produced UVB (280-315nm) on human skin 7-dehydrocholesterol, or else by oral ingestion, followed by conversion in liver and kidney to its 1,25 dehydrocholesterol active form. To be effective, it also needs normal parathormone and calcitonin function along with adequate dietary calcium, of which it regulates the absorption. 25-hydroxyvitamin D is the major form of vitamin D in blood and there is good agreement that levels below 25nmol/L (10ng/mL) are deficient, although the evaluation of such levels has been said to be widely variable on the same sample between laboratories, making individual results unreliable if this is so, a matter needing rapid resolution Lai et al, 2012). In any case, it is not yet possible to nominate an optimal vitamin D status level, but 50nmol/L or more is probably ideal. Although no safe upper level has yet been identified, values above 125nmol/L are not recommended. In fact, average levels in normal subjects remain largely the same at all latitudes, as measured from about 25$^{o}$ (Northern Caribbean) to 70$^{o}$ (Northern Scandinavia) North, probably because regulated by homeostatic mechanisms (Zittermann et al, 2005), while only small exposures seem needed to increase low values rapidly (Bogh et al, 2010). Levels therefore appear not heavily dependent on constant or marked sun exposure, being slightly higher in summer and lower in winter everywhere, with values running between 40 and 80nmol/L.

Recent work (Professor Antony Young, St John's Institute of Dermatology, London, UK, personal communication, see Petersen et al, 2014) shows unprotected UVB exposure leads to potentially mutagenic skin thymine dimers, precursors to skin photoageing and cancer, but such exposure also enables normal subjects to synthesise adequate vitamin D. However, it would seem rational in evolutionary terms that such damaging exposure might not be needed and careful work has therefore been now undertaken to determine what degree of exposure is in fact required (Professor Antony Young, personal communication, see Petersen et al, 2014). In that study, two groups of twenty fair-skinned subjects were monitored for sun exposure and also full and regular application during exposure of sunscreens of SPFs 19 and 18 but high and low UVA protection respectively, during a week of very strong sun exposure in Tenerife, Canary Islands, and their 25-hydroxyvitamin D levels assessed before and after. A third similarly exposed group of twenty-two subjects did not use sunscreen. Both the sunscreen groups, the better UVA sunscreen somewhat less so, did demonstrate up to 50% lower vitamin D blood increases than the non-sunscreen group, who in fact also suffered important sunburn, but these increases were still significant clinically. Such sunscreen use is however known markedly to diminish skin thymine dimer production (Young et al, 2000), indicating that adequate vitamin production can be achieved with minimal skin damage. This outcome is essentially similar to that of an earlier study (Faurschou et al, 2012).

In addition, other work was undertaken during the same series of investigations to consider vitamin D levels in irradiated subjects of all depths of skin pigmentation (Professor Antony Young, personal communication). Whole body exposures of two standard erythema doses (SEDs) each except on underwear sites took place five times at three- to four-day intervals with an Arimed B solar simulator emitting radiation approximating clear London summer sunshine. This showed that skin colour, whether minimal or extremely dark, had no effect on the elevations achieved in blood 25-hydroxyvitamin D levels following solar simulated irradiation, a finding repeated elsewhere but with UVB (Bogh et al, 2010).

Further, in totally separate work, cows bearing normal hair manufactured vitamin D adequately, a response diminished by covering parts of the hide (Spradbow et al, 1987), and cows do not normally develop skin cancer on hair-covered areas, while hair, at least on the human scalp, gives sun protection of 5 to 17 times (Parisi et al, 2009).

This further suggests that just minimal ultraviolet exposure is sufficient to maintain adequate systemic vitamin D levels.

In fact there is further evidence in man that only minimal UVB exposure is required for the maintenance of adequate vitamin D levels, probably only about 0.5 weekly SEDs (though over about 90% body area) on unprotected skin are needed (Bogh et al, 2012), while only 0.75 SEDs over about 25% of the body area or 1.5 SEDs over about 5% every two to three days increases levels significantly (Bogh et al, 2011).

Therefore, there appears to be no need for any subject to expose the skin to the sun without adequate sun protection so as to diminish skin cancer risk, unless very possibly with the constant, obsessive use of thickly applied sunscreens of very high protection factor or virtually complete clothing cover during all sun exposure. In such cases, therefore, or in any case where concern exists, oral supplementation should be used instead, just as effective in maintaining vitamin D levels (Jones et al, 1998). For this, 600 IU/day (15μg) for adults, 800 for those over 70 years, and 4000 maximum, are recommended, though not generally in the presence of hypercalcaemia, hypervitaminosis D or renal osteodystrophy with hyperphosphataemia (Ministry of Health and Cancer Society of New Zealand, 2012). Care may also be needed in some subjects with atherosclerosis, cardiac impairment, hypersensitivity to vitamin D, renal function impairment, or sarcoidosis, when protected sun exposure as described above may be preferable.

There is in fact evidence that minimal, non-damaging UVB exposure is of value, arguably in evolutionary terms, in leading not only to adequate vitamin D production, but also to the prevention of constantly occurring contact dermatitis and certain photodermatoses, particularly the common polymorphic light eruption, through the reduction of adaptive immunity, and to the induction of bacterial defensins to minimise skin infections, through the enhancement of innate immunity (.Glaser et al, 2009)

Finally, it should again be stressed that excessive sun (or sunbed) exposure for the maintenance of adequate vitamin D levels is dangerous and unnecessary, and exposure with sun protection or else oral supplementation should always be preferred.

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