

# Sunlight and Reptile UVB Tubes

The value of UVB exposure

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**Abstract:** The absorption of calcium from the food relies on the presence of a hormone called 1,25, hydroxy-vitamin D. In some animals, this vitamin is provided by the food. There is good evidence that iguanas do not absorb vitamin D from food and therefore must be provided with the means to make their own vitamin D. This begins with the exposure of skin to UVB radiation. Strong sunlight is well known to provide sufficient UVB. The paper discusses the relative strength of sunlight in different areas and at different times of year, and the strength of reptile UVB lights maintained at 12 inches from the iguana. It concludes that for people living in the far north or south (more than 40 degrees from the equator), reptile lights should be used for iguanas during the winter months.

It has been known since 1822 that exposure to sunlight cures the disease called rickets. In 1890, Palm collected observations from scattered parts of the British Empire and the Orient. He compared them with observations made in Britain and concluded that rickets was rare in impoverished city-dwelling children in China, Japan and India, whereas British children living in supposedly better conditions had a higher incidence of this disease. Thirty years later, Huldschinski discovered that rachitic (rickety) children could be cured by exposure of one arm to the rays of a mercury vapor lamp. This was clearly not a localized effect as the other bones of the children's bodies also benefited. This bloodstream-borne "anti-rachitic factor" was later named Vitamin D. [1]

A fatty substance, 7-dehydrocholesterol (provitamin D3), is the precursor of vitamin D. It is found in the skin, with concentrations varying with the likelihood of exposure to UVB. For instance, in the chicken, there is thirty times the concentration of 7-DHC in the leg skin as there is in the feather-covered back skin. [8]

7-dehydrocholesterol in the skin absorbs UVB photons with energies between 290 and 315 nm in its 5,7-diene region, forming previtamin D3. [1] This reaction is purely dependent on light, and takes place in all animals but is particularly important in vertebrates. If 7-DHC is placed in vitro (in quartz tubes), the reaction takes place in the same way. It is not dependent on enzymes or cells at all. [2][3] If this previtamin D3 remains in the skin, and is further exposed to light, it breaks down into inert bioproducts, lumisterol and tachysterol. [1] No more than 15% of 7-DHC in the skin can be transformed to previtamin D3 in humans, no matter how long the exposure to sunlight. [4] This may be the maximum in all systems as it is the maximum in vitro [2] [3] and the maximum in surgically obtained human skin [7]. This is a useful regulatory device, making it impossible for an animal to produce too much previtamin D3. If the stores of previtamin D3 are completely depleted, lumisterol and tachysterol then become able to be photo-isomerized to previtamin D3, so these compounds are useful storage devices. [5]

Previtamin D3 then thermally equilibrates to vitamin D3 by isomerization. Although this step is temperature-dependent in vitro, skin contains a mechanism which speeds up this step by as much as 1700% in iguana skin at 5 degrees C and 1100% in iguana skin at 25 degrees C. Otherwise, poikilotherms (cold blooded animals) would never have been able to make sufficient vitamin D to maintain their bony skeletons. [6] This step takes 1-2 days in humans at 37 degrees C. [1]

In another paper, [23] Webb and coworkers found that exposure to weak sunlight can degrade into inert compounds vitamin D3 that has already been made. They write: 'Sunlight exposure in Boston during the late fall and winter will not promote the cutaneous synthesis of previtamin D3 (referencing [2]), yet the same solar radiation can photodegrade vitamin D3 to 5, 6, transvitamin D3, suprasterol I and suprasterol II. At the same time, the higher energy 290-315 NM radiation responsible for photolyzing cutaneous 7-DHC to previtamin D3 was absorbed to a great extent by the ozone layer, as the distance the radiation must travel through the atmosphere to reach the earth's surface is increased in winter when the sun's angle is lower. The reason why [you can't make vitamin D3] in the late fall and winter in Boston is that there are too few solar photons reaching the earth's surface with sufficient energy ( $\lambda$  less than 315nm), to cause it to [be made], and [the precursor] cannot absorb UV radiation beyond 315nm. However, vitamin D3 can absorb the relatively large number of lower energy photons ( $\lambda$  315-330) that are present in winter sunlight, resulting in [destruction] of vitamin D3 throughout the

year.'

Vitamin D<sub>3</sub> is biologically inert, but once in the bloodstream it can reach the liver, where it undergoes its first hydroxylation and then to the kidney where it is further hydroxylated to 1,25(OH)<sub>2</sub> D. This is the active form, which acts as a hormone in the regulation of calcium metabolism.

Clearly, there must be a minimum amount of UVB that will produce measurable increases in previtamin D<sub>3</sub>. One major factor which affects the UVB intensity over the course of a year is solar zenith angle, as the Earth goes through the seasons. When the sun is low in the sky, light must travel through more atmosphere before reaching sea-level, and more UV is absorbed. Time of day also has an effect as the lower sun angle in the early morning and late afternoon increases the amount of atmosphere the sunlight must pass through. Finally, latitude (how far north or south of the equator you are) affects the angle. Additionally, it was, and still is being, suggested that the greater amount of clothing and the reduced time spent outdoors in winter is responsible for reduced bone density observed in high-latitude and low-latitude humans in winter. [9] In 1988, Webb, Kline and Holick set out to see whether the by-now-well-known seasonal fall in vitamin D concentrations in people was due to clothing or to reduced UVB irradiance in these latitudes and these months. [2]

Webb et al compared two systems of previtamin D<sub>3</sub> production. They dissolved 7-DHC in methanol and exposed it to varying light levels, and also exposed neonatal human skin (foreskins) to varying light levels. Both systems were then assayed for photoproducts of 7-DHC. As part of their controls, they divided each skin into two, keeping one half in the dark and exposing the other half. The two halves could then be directly compared. They found that the methanol system was a good model for the human skin, although the lack of cellular molecules competing for UVB photons (such as RNA, DNA and proteins) made it a little more sensitive to UVB light. [2]

Using the methanol-7-DHC method, Webb et al found that previtamin D<sub>3</sub> levels go up steadily for the whole three hours (in the Boston June samples), accompanied by a rise in lumisterol and a small rise in tachysterol. The greatest level of previtamin D<sub>3</sub> reached in these samples was 12%. Their method compensated for the fact that there are fewer sunny days in winter, by exposing the tubes only on sunny days. They found that skin samples exposed to sunlight in Boston (42 degrees north) from November and February for three hours between 1130 and 1430 local time did not convert 7-DHC to previtamin D<sub>3</sub>. It was not until March

17th that previtamin D3 was produced in the skin. Webb's figures for exposure show that 120 mJ/cm<sup>2</sup> are produced by Boston sunlight over 3 hours in summer (1130 to 1430 local standard time) and about 60 mJ/cm<sup>2</sup> of that is produced over one hour around solar noon. In winter, the figures are about 5 mJ/cm<sup>2</sup> in one hour around and very little more in the three hours 1130 to 1430. [2]

Webb et al went on to look at other cities. In Edmonton, Canada (52 degrees north), no previtamin D3 was produced from October until the beginning of April. Samples in Los Angeles (34 degrees north) produced previtamin D3 all year round, as did samples in Puerto Rico (18 degrees north). However, the amount produced in Los Angeles was significantly less than the amount produced in Puerto Rico. During January, the sun converted 3% of the 7-DHC in Los Angeles and 10% in Puerto Rico. [2]

Using a similar system (7-DHC dissolved in hexane), Pettifor and coworkers found that little D3 was formed in Cape Town (34 degrees south) from April through September (winter), but in Johannesburg (27 degrees south) it was significant all year round. Sufficient was formed in Cape Town to attribute vitamin D3 deficiency in humans to extra clothing rather than solar insufficiency. [9] In South America, Ladizesky et al exposed samples in Buenos Aires (34 degrees south) for four hours around midday, which produced significant vitamin D all year round, but in Ushuaia (55 degrees south) negligible amounts were formed from April to July (winter). [3]

A question remains about this seasonal effect. If no vitamin D can be produced from its precursors in large areas of the globe, is this reflected in signs of disease? Formal studies have not been completed for iguanas. The seasonal incidence of rickets and osteomalacia in cities in the northern US and Europe was well documented at the turn of the century. [1] Upwards of 40% of patients with acute hip fractures are found to be vitamin D deficient. In New England during the winter, a marked loss of bone can be detected, with the spine losing up to 3.6 percent and the hip losing up to 3.0 of its density in the fall-winter months. [10] In a Spanish study, premenopausal women showed a significant bone loss in winter. [13] In a similar study in the subtropical Taipei region, no such loss of bone occurred. [11] Snakes in France showed a similar annual cycle but no bone-loss markers or UVB exposure were studied in the paper cited. [12] There is also some evidence that summer-born infants have lower bone density than winter-born infants, suggesting that maternal vitamin D status is important during the first few months of gestation, a Chicago study showed. [14] Two studies using artificial UVB light showed that the

circulating vitamin D<sub>3</sub> doubled or more than doubled one day after exposure, but was almost back to baseline level after 7 days, and was below baseline level by day 15. [16, 17] Since there is little evidence that iguanas benefit from dietary D<sub>3</sub> supplementation, these data on humans suggest that iguanas would benefit from artificial UVB radiation during the winter months, if they are kept at latitudes above 40 degrees north or below 40 degrees south during the winter months.

How much UVB exposure is needed to supplement weak sunlight? The amount of UVB needed to mount a significant response is still in question for iguanas. For humans, three types of measurements have been used which are not always fully interchangeable. One such measure is the Minimal Erythremal Dose or MED, which is the amount of UV light needed to turn the skin pink (the pinkness is not related to vitamin D production; it is just a marker of exposure to intense UVB). The intensity needed to produce one MED varies with the individual, but one study found that the range for their subjects was 40 to 140 mJ/cm<sup>2</sup>. [15] Diffey [4] gives a MED estimate of 34mJ/cm<sup>2</sup> (range 14-84) for whites in the North East of England. This is a measure of energy received by the skin. The power of a lamp reaching a target such as skin is measured in μW/cm<sup>2</sup>. Multiplying this figure by the time of exposure gives the mJ/cm<sup>2</sup> figure.

In one study of the human elderly, 1000cm<sup>2</sup> of skin was irradiated with one-half MED three times a week for 12 weeks, using 3 Philips TL12s (sun-bed fluorescent tubes) at 45 cm. The irradiations of one half MED took from 3 to 7 minutes. The result was a significant rise in 25(OH)D from a mean of about 18 nmol/l to almost 60 nmol/l. The treatment significantly decreased circulating levels of the hormone PTH, showing that the increased vitamin D in the circulation was able to affect markers of bone resorption. The effects of this treatment was similar to the effects of 400 IU of vitamin D supplementation daily, as a simultaneous clinical trial of oral vitamin D showed. Four weeks after discontinuing treatment, the 25(OH)D level had fallen by 16 nmol/l. [15]

Another study showed significant increases in serum vitamin D concentration in Caucasians after exposure to a whole body dose of 54 mJ/cm<sup>2</sup> and some change after a similar irradiation of black subjects. A second exposure of one of the black subjects to 320 mJ/cm<sup>2</sup> gave a greater response. [18] The chickens in [8] were irradiated with 500 mJ/cm<sup>2</sup>, this large amount of radiation producing up to an average of 54 ng of previtamin D<sub>3</sub> per square centimeter of skin. Another study using adult volunteers showed that vitamin D concentrations rose from 2 ng/ml to 24 ng/ml 24 hours after exposure to 1 whole body MED [20], or about 50

mJ/cm<sup>2</sup>. It was calculated that this represents 30 ug of vitamin D<sub>3</sub> released per square meter of body surface. For reference, a multivitamin supplement for humans contains about 10 ug of vitamin D, or twice the recommended daily amount. So exposure to one whole-body MED provides about 12 days of vitamin D<sub>3</sub>.

If the sun in Boston in winter is too weak to create detectable previtamin D<sub>3</sub>, how can reptile lights manage? Ladizesky [3] says, quoting a paper I haven't read, that a whole body irradiance of 20 mJ/cm<sup>2</sup> is sufficient to maintain adequate vitamin D levels in humans. Holick's paper also gives the 20 mJ/cm<sup>2</sup> figure, and a close reading of this paper and several Holick reviews in Endocrinology textbooks shows that he believes that exposure of the hands and face only to this level is sufficient for vitamin D<sub>3</sub> maintenance (or a comparable size skin area - it doesn't have to be the hands and face). Bernard et al [24] exposed vitamin D deficient iguanas to UVB from a Sylvania 2096 fluorescent tube (not yet available) for 12 hours daily and found that so much previtamin D<sub>3</sub> was produced that the researchers wondered if the normal feedback mechanisms applied to iguanas.

How strong is a reptile light? A Vita-Lite at 12 inches produces about 1.1 μW/cm<sup>2</sup> [21], which would produce 20 mJ/cm<sup>2</sup> over any exposed skin in 5 hours. That should be sufficient to begin vitamin D<sub>3</sub> production. Gehrman [22], gives the Zoo Med Iguana light 5.0 at 10 μW/cm<sup>2</sup>. This would produce 20 mJ/cm<sup>2</sup> in less than an hour. (A reptile D-light 8% produces about 9 μW/cm<sup>2</sup>, and a Reptile D-light 3% produces 4 μW/cm<sup>2</sup>.) Winter sunlight in Boston is a non-vitamin-D-producing 5mJ/cm<sup>2</sup> over three hours; the Zoo Med 5.0 would beat this figure in just a few minutes and go on to vitamin D<sub>3</sub> producing levels, sustaining them for the rest of the day. Although this looks very strong on paper, I have personally found no problems with iguanas living about 12 inches away from Zoo Med tubes for 6-10 hours per day over the past two years."

It is my opinion that iguanas kept in areas where the natural sunlight is weak throughout the winter months (more than 40 degrees above or below the equator) should have access to reptile UVB lighting for several hours each day. Iguana owners unable or unwilling to provide natural sunlight in other areas or at other times should also provide artificial UVB exposure. If this exposure is provided, the vitamin D<sub>3</sub> produced in the iguana will provide the means for the iguana to absorb calcium from the diet and also to perform the other valuable functions of this vitamin.

Obtained from <http://www.anapsid.org/uvbanne.html>

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**Evolutionary importance for the membrane enhancement of the production of vitamin D3 in the skin of poikilothermic animals.**

Holick MF; Tian XQ; Allen M; Department of Medicine, Boston University Medical Center, MA 02118, USA. *Proc Natl Acad Sci U S A* 1995 Apr 11;92(8):3124-6

ABSTRACT: The photoproduction of vitamin D in the skin was essential for the evolutionary development of terrestrial vertebrates. During exposure to sunlight, previtamin D3 formed in the skin is isomerized to vitamin D3 (calcitriol) by a temperature-dependent process. Since early land vertebrates were poikilothermic, the relatively slow conversion of previtamin D3 to vitamin D3 at ambient temperature put them at serious risk for developing vitamin D deficiency, thus leading to a poorly mineralized skeleton that could have ultimately halted further evolutionary development of vertebrates on land. We evaluated the rate of isomerization of previtamin D3 to vitamin D3 in the skin of iguanas and found the isomerization rate was enhanced by 1100% and 1700% at 25 degrees C and 5 degrees C, respectively. It is likely that the membrane entrapment of previtamin D3 in its s-cis,s-cis conformation is responsible for the markedly enhanced conversion of previtamin D3 to vitamin D3. The membrane-enhanced production of vitamin D3 ensures the critical supply of vitamin D3 to poikilothermic animals such as iguanas.

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