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The Sunlight Conundrum: UV in the 21st Century

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Introduction

The sun provides the light and heat that enables life on Earth. It has been worshipped by many civilisations as the Giver of Life, while skin pigmentation, lifestyles and traditional dress have evolved or developed to both use and protect from the sun in a wide range of climates.

Scientific and industrial advances have led to modern lifestyles that bear little resemblance to traditional ways. The Sun is no longer the main control and clock for the day and year as light and heat are available whenever required. It is easy to travel to places with significantly more or less sun than our native home, either for a holiday or permanently. This progress, and its influence on both the environment and lifestyles, has resulted in changing attitudes towards, and medical advice regarding, sun exposure.

In the early 20th century rickets, the bone deforming disease that results from a deficiency of vitamin D, was a scourge of industrial regions. Sunlight exposure (what we now understand to be the ultraviolet B (UVB) part of sunlight) was advocated as a cure [Hess and Unger, 1921]. A few decades later a year round tan became the symbol of those able to afford frequent foreign holidays, and is still considered a desirable "healthy" look by many. By the end of the century the over-riding medical concern was of too much sun exposure leading to skin cancer, a message underlined by ozone depletion [Solomon, 1999] and prognoses and observations of increased UV radiation at the surface [WMO, 2003].

In the early years of the 21st century the incidence of skin cancers is still rising, cancers resulting from both acute (sunburn) and cumulative lifetime exposure to ultraviolet radiation (UVB + UVA: UVB 280 – 315nm; UVA 315 – 400nm). For most people the only significant source of UV radiation to which they are exposed is the sun. At the same time there is evidence of a real risk of inadequate exposure to UV radiation (sunlight) in some populations, appearing as an insufficiency of vitamin D.

The primary source of vitamin D for most people is cutaneous synthesis, initiated by irradiation with UVB radiation. Vitamin D can also be obtained from the diet, mainly from fatty fish, or supplementation, but for the majority of the population the sun is the predominant source of the vitamin. From its synthesis in the skin, the vitamin is hydroxylated in the liver to 25 hydroxyvitamin D (25OHD) – the form that is measured in blood plasma to determine vitamin D status – and then again in the kidney to the active form 1,25 dihydroxyvitamin D (1,25OHD).

Certain population groups have an increased risk of vitamin D deficiency: the elderly [Hegarty et al., 1994; Janssen et al., 2002]; pregnant and lactating mothers and their babies [Hollis et al., 2004a,b]; and those with pigmented skin living at high latitudes, especially if custom dictates that they do not expose their skin [Henriksen et al., 1995; Pugliese et al., 1998; Serhan et al., 1999]. In addition to these groups there is now evidence that the general population is also failing to get enough vitamin D. Two thirds of the north Norwegian population had below the recommended levels of circulating 25OHD [Brustad et al. 2004], while amongst other possible examples vitamin D insufficiency has been found in British children [Davies et al., 1999], and healthy American adults [Tangpricha et al., 2002].

At the same time there is an increasing body of evidence that vitamin D may do more than maintain a healthy skeleton. A combination of epidemiological studies, animal models, and cellular work imply that vitamin D may: play a role in protecting against cancers, particularly those of the colon, breast and prostate [Schwartz et al., 1998; Garland et al., 1989; Garland et al., 1990; Grant 2002a,b; John et al., 1999; Robsahm et al., 2004]; reduce hypertension [Scragg, 1990; Krause et al., 1998; Zittermann et al., 2003]; help to reduce the risk of some autoimmune diseases such as diabetes and multiple sclerosis [Mathieu et al., 1994;Hypponen et al., 2001; Ponsonby,et al., 2002].

Exposing the skin to solar (UV) radiation has both risks (skin cancer, photoaging) and benefits (skeletal and potential additional benefits associated with vitamin D). Here we explore the optimum exposure regime to maximise the benefits and minimise the risks.

Methods

The solar spectral UV irradiance on a flat horizontal surface has been calculated as a function of latitude, season and time of day for the Northern Hemisphere. Similar results are obtained for the Southern Hemisphere, except, of course, the time axis is shifted half a year. These irradiances were then used to assess the potential for benefit (vitamin D synthesis) and harm (erythema), and the relation between the two.

The radiative transfer model used was the fast and accurate FastRT UV simulation tool [Engelsen and Kylling, 2005]. The calculations assume a cloudless atmosphere over a non-reflecting surface with an ozone layer thickness fixed at a typical level (350 Dobson Units) and a rural aerosol [Shettle, 1989]. Aerosol optical depth is given by $\tau = \beta * \lambda^{-\alpha}$ where the Ångström coefficient α was set to 1.3, and the wavelengths (λ) are in micrometers. The Ångström coefficient β was related to 25 km visibility $R_m[km]$ using the parameterization of Iqbal [1983], i.e.,
$\beta = 0.55^{1.3}\,(3.912/\,R_m[km]-0.01162)[0.02472*(\,R_m[km]-5)+1.132]$.
In all other respects the model uses a US standard atmosphere [Anderson et al., 1986].

FastRT was used to compute CIE erythema [MacKinley and Diffey 1987] and vitamin D effective UV doses. The former were expressed in standard erythemal units (SED = 100 Jm^{-2} erythemally effective UV). The latter were computed using the action spectrum for conversion of 7-DHC to previtamin D in human skin [MacLaughlin et al., 1982] with an exponential decay extrapolation. The two action spectra are shown in figure 1.

The Standard Vitamin D Dose (SDD) is defined here as the UV equivalent of an oral dose of 1000 IU vitamin D, i.e. the dose recommended to gain all the possible health benefits of vitamin D [Holick, 2004]. Vitamin D status is measured in the blood (plasma 25OHD) and is a net response to all skin exposure (and dietary intake), so the SDD must be qualified by the conditions of skin exposure. Holick [2004] estimates that 1000 IU equivalent vitamin D can be gained from exposure to ¼ of personal MED on ¼ skin area (hands, face and arms) and these criteria were used in defining the SDD. The UV spectrum for a mid-latitude midday in spring in Boston (15 February, 42.2º N, ozone = 350DU) was calculated, and from this the time to acquire a ¼ MED (assumed = ½ SED) for a fair skinned person. Using the same solar exposure we then calculated the vitamin D effective dose acquired over the same time interval. This is then the SDD based on exposure of ¼ body surface area, and is equivalent to 29.2 Jm^{-2} vitamin D effective UV in the cloudless conditions described above, corresponding to about 32

minutes of exposure. A fair skinned person would now make sufficient vitamin D with 1 SDD, and would suffer a minimal erythema after 1 MED (2 SED). Darker skinned people will require both multiple SDDs and a greater number of SEDs to achieve the same effects.

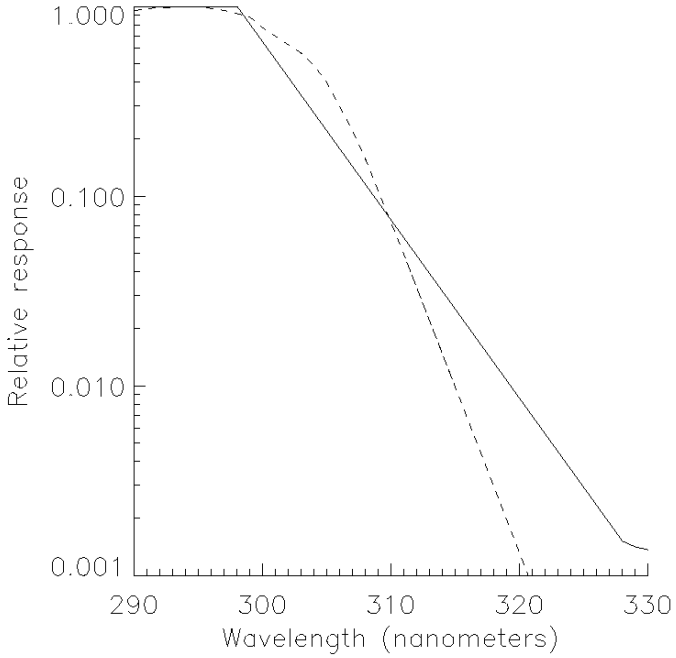

Figure 1 Action spectra for erythema (solid) and vitamin D (dashed)

The conditions in Boston in February were chosen because they represent the time of year when vitamin D synthesis begins again after a period with insufficient solar UV [Webb et al., 1988]. The model also calculates the irradiance (and hence SED and SDD) for a flat horizontal surface. No account is taken of the shape and orientation of the human body. However, the relationship between SED and SDD is affected little since the same assumption has been applied to both.

Results and discussion

The time required to obtain the recommended UV dose for adequate vitamin D synthesis in human skin depends on the solar elevation, and the surface and atmospheric conditions. For fixed typical atmospheric conditions the changes in solar elevation with season and latitude lead to changes in both the solar UV spectrum and intensity, and produce significant variation in the time needed to acquire one SDD (figure 2). These calculations account for the changing solar elevation during the day, beginning with the noontime solar irradiance and integrating the changing vitamin D weighted irradiances until one SDD is reached. Note that for latitudes polewards of about 45 degrees exposure times are prolonged, and at higher latitudes unreasonable, for at least some periods of the year, indicating a “vitamin D winter” when cutaneous synthesis of the vitamin is impossible or at best highly impractical.

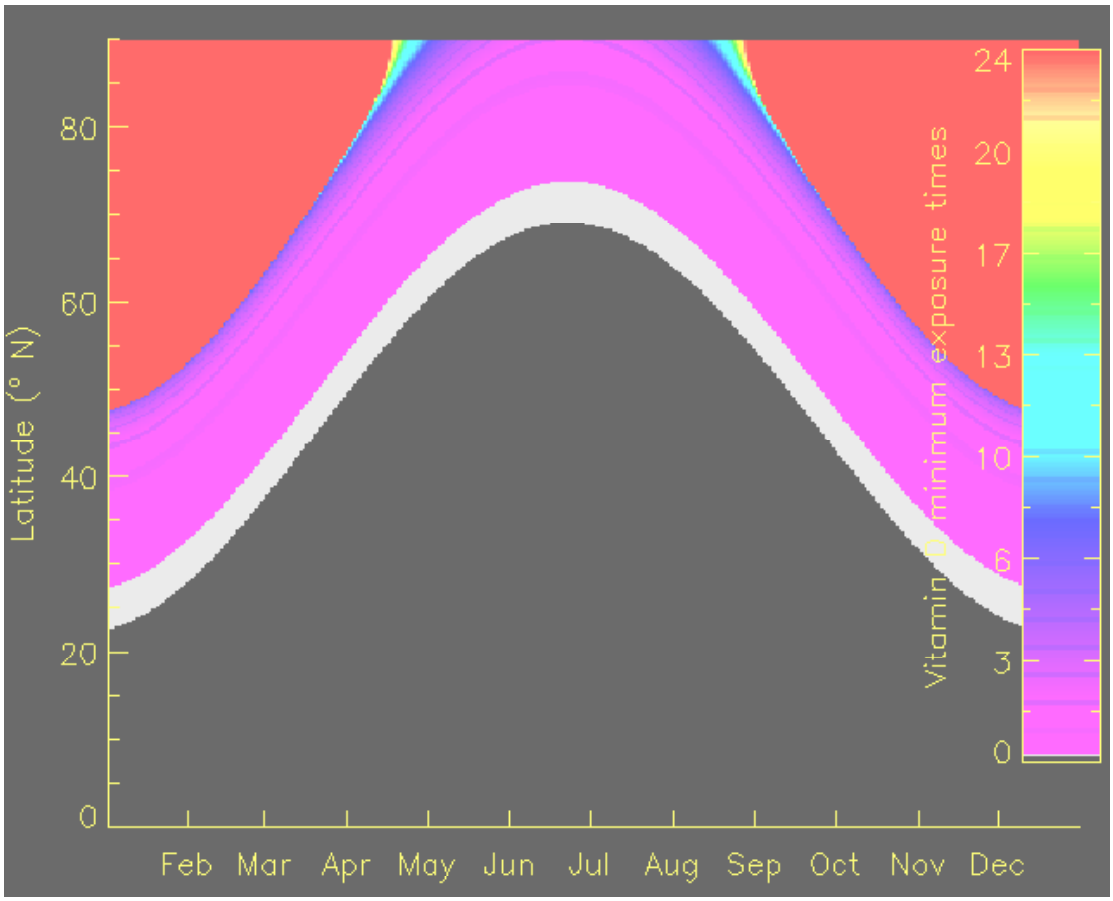

Figure 2 Exposure times in hours required to acquire one SDD. The region shaded black shows the times and places where exposures take minutes rather than hours: black < 16 min., grey 16-22 min., pink >22 min. The region shaded red shows where 1 SDD cannot be reached within daylight hours.

To explore the balance between harmful and beneficiary effects of UV exposure the number of MEDs (for a fair skinned person, = 2SED) acquired while obtaining one SDD has been calculated as a function of latitude and season (fig. 2). Since the solar spectrum changes with solar elevation and the action spectra for erythema and cutaneous vitamin D synthesis differ considerably (fig. 1), the 50:29.2 ratio of Erythemally effective dose:SDD is not a constant. This ratio (giving 0.25 MED while acquiring 1 SDD, by definition) is valid only for the same radiative regime used for defining SDD i.e. Boston in February with the standard model conditions. For other solar elevations, altitudes, aerosol, cloud and ozone amounts the solar spectrum and the ratio will change (fig. 3). Comparing figures 2 and 3 it is clear that an SDD can be obtained with solar exposures that are less than 0.5MED in all reasonable and practical circumstances. At latitudes and seasons where the exposure needed for 1 SDD would also give close to 1 MED the exposure times are unreasonably long, particularly given the likely temperatures, for exposing ¼ body surface area. The increase in fractional MED (for 1 SDD) with decreasing solar elevation is a consequence of the changing solar UV spectrum. As the solar elevation decreases the short-wavelength limit of the solar spectrum shifts towards longer wavelengths. This changes the ratio of UVB to

UVA radiation in favour of UVA. Since the erythemal action spectrum has a far greater UVA component than the vitamin D action spectrum (figure 1), the erythemal dose is enhanced relative to the vitamin D dose at low solar elevations. Thus, the optimum exposure regime for solar UV radiation would be a short period of exposure at the highest solar elevation available, rather than extended time at a lower solar elevation.

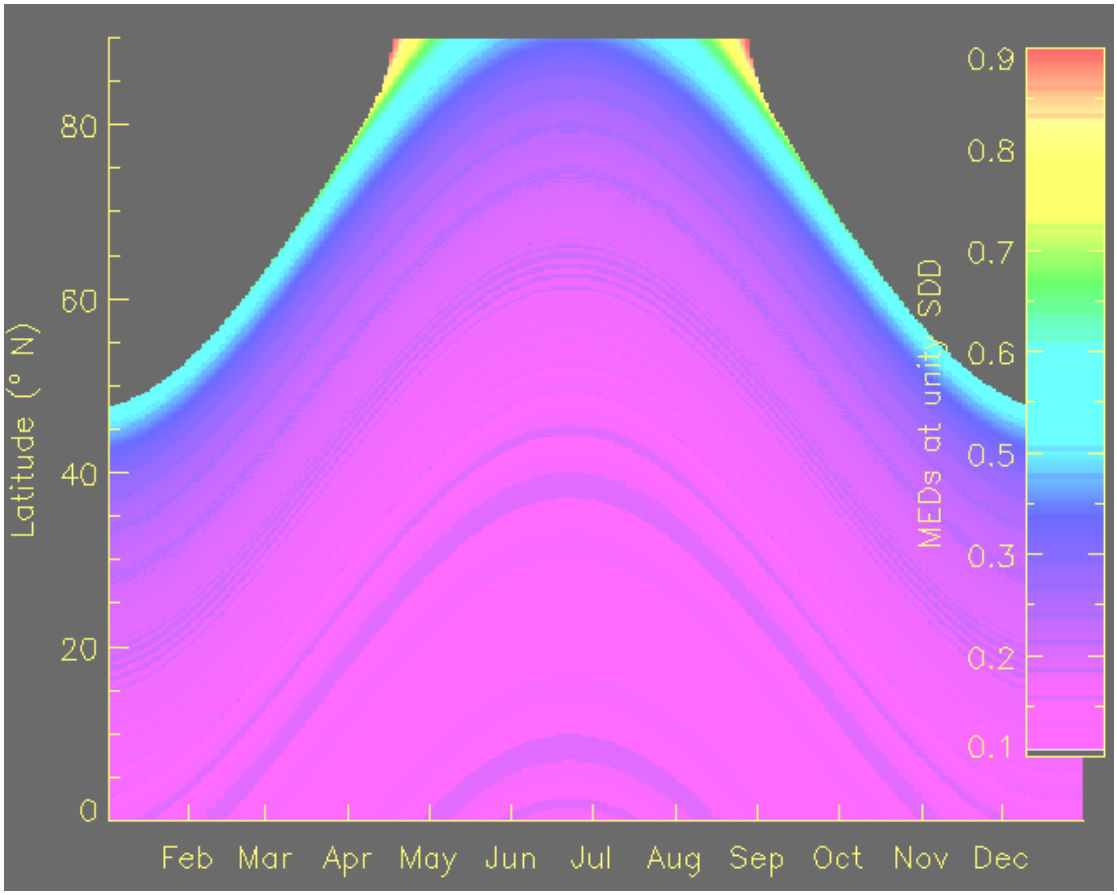

Figure 3 Latitudinal and seasonal dependence of the number of MEDs acquired in the time taken to get one SDD. The banding in the lower pink sections is an artefact of the programme.

Skin Type	Tan	Burn	Hair colour	Eye colour	Typical MED
I	Rarely	Always	Red	Blue	200 Jm^{-2}
II	Sometimes	Sometimes	Blond	Blue / green	250 Jm^{-2}
III	Usually	Rarely	Brown	Grey/ brown	350 Jm^{-2}
IV	Always	Rarely	Black	Brown	450 Jm^{-2}

Table 1 Skin Types and characteristics for a European population. Naturally pigmented skins (Types V and VI) have even higher MEDs and a correspondingly higher number of SDDs are needed to give the same vitamin D benefits.

The calculations illustrated in figures 2 and 3 represent a global uniform atmosphere. In reality there is enormous spatial and temporal variation in cloudiness, ozone, aerosols., surface reflection and altitude. These differences between the ideal model and the real world will result in a wide range of possible exposure times at any time and place, rather than the single values shown in figure 2. The latter are only a representative guide for specified conditions, and for fair skinned people. People with other skin types (table 1) will require longer exposures both to gain 1 SDD and suffer from 1 MED. The ratio of the two effects (figure 3) is more robust in that it is less affected by the details of the state of the atmosphere or the skin of the target person. Changes in ozone will affect the spectral composition of solar UV but cloud, aerosols, surface reflectivity and skin type have a minor influence on the incident spectrum and the skin's response. Note that in figure 3 MED refers to the 2SED at which a fair skinned would show erythema: if a person requires 4 SDD to acquire sufficient vitamin D then their personal MED will be correspondingly higher.

Conclusion

A risk / benefit analysis of exposure to solar UV radiation has been explored with respect to erythema and vitamin D synthesis. The UV irradiance under a standard set of atmospheric conditions has been calculated globally as a function of time. The exposure time to achieve one SDD has been assessed, assuming a fair skinned person exposing ¼ body surface area (face, hands and arms), plus the associated fraction of an MED gained for the same radiation regime. There are significant periods of the year at mid - high latitudes when one SDD is not achievable during daylight hours and vitamin D status will decline unless diet and supplementation are used as alternative sources of the vitamin. In regions where one SDD can be acquired in practical exposure times the accompanying erythemal load is less, usually substantially less, then 0.5 MED. When solar elevations are low the SDD exposure times become very prolonged and the associated erythemal load approaches one MED. However, in these situations the exposure times are deemed impractical and very unlikely for the general population. When solar UV radiation is sufficient to enable cutaneous vitamin D synthesis the benefits of exposure can be gained at sub-erythemal doses of UV. This also holds for naturally pigmented people who require a larger absolute dose of UV for their vitamin D requirements, but whose minimal erythemal dose is also far greater than a fair skinned person. Nonetheless, pigmented people will have to spend longer in the sun to acquire 1000 IU equivalent of vitamin D so the latitudes and seasons where sufficient vitamin D synthesis is not possible will be more extensive than that shown in figure 2 for a fair skinned person.

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References

Anderson, G. P., S. A. Clough, F. X. Kneizys, J. H. Chetwynd and E. P. Shettle (1986) AFGL atmospheric constituent profiles (0-120 km). *Tech. Rep. AFGL-TR-86-0110*, Air Force Geophys. Lab., Hanscom AFB, Mass.

Banwell, C. M., R. Singh, P. M. Stewart, Uskokovic M. R. and M. J. Campbell (2003) Antiproliferative signalling by 1,25(OH)2D3 in prostate and breast cancer is suppressed by a mechanism involving histone deacetylation. *Recent Results Cancer Res.* **164,** 83-98.

Brustad, M., E. Alsaker, O. Engelsen, L. Aksnes, and E. Lund (2004a) Vitamin D status in middle-aged women at 65-71 ºN in relation to dietary intake and exposure to ultraviolet radiation. *Publ. Health Nutr.* **7**, 327-335.

Davies P.S. et al. (1999) Vitamin D: seasonal and regional differences in preschool children in Great Britain. *Eur J Clin Nutr* **55** (3), 195-198

Engelsen, O. and A. Kylling (2005) A fast simulation tool for ultraviolet radiation at the Earth's surface. Accepted for publication in the Journal of Optical Engineering. (http://nadir.nilu.no/~olaeng/fastrt/fastrt.html)

Garland, C. F., G. W. Comstock, F. C. Garland, K. J. Helsing, E. K. Shaw and E. D. Gorham (1989) Serum 25-hydroxyvitamin D and colon cancer: eight-year prospective study. *Lancet* **2,** 1176-1178.

Garland F.C., garland C.F. Gorham E.D. and Young J.F. (1990) Geographic variation in breast cancer mortality in the United States: A hypothesis involving exposure to solar radiation. *Preventive Medicine* **19**, 614-622

Grant, W.B. (2002a) An ecologic study of dietary and solar ultraviolet-B links to breast carcinoma mortality rates. *Cancer* **94,** 272-281.

Grant, W.B. (2002b) An estimate of premature cancer mortality in the U.S. due to inadequate doses of solar ultraviolet-B radiation. *Cancer* **94,** 1867-1875.

Hegarty, V., Woodhouse, P., and Khaw, K. (1994) Seasonal variations in 25-hydroxyvitamin D and parathyroid hormone concentrations in healthy elderly people. *Age and Ageing* **23**, 478-482

Henriksen C., Brunvand L., Stoltenberg C et al. (1995) Diet and vitamin D status among prenant Pakistani women in Oslo. *Eur.J.Clin.Nutr*. **49**, 211-218

Hess A.F. and Unger L.F. (1921) Cure of infantile rickets by sunlight. *JAMA* **77**, 33-41

Holick, MF (2004) *The UV Advantage*. ibooks inc., New York

Hypponen E., Laara E., Reunanen A., Jarvelin M.R., and Virtanen S.M. (2001) Intake of vitamin D and risk of type 1 diabetes: A birth cohort study. *Lancet* **358** (9292), 1500-1503

Iqbal, M. (1983) An Introduction to Solar Radiation, Academic, San Diego, CA.

Janssen,H., Samson,M., and Verhaar, H. (2002) Vitamin D deficiency, muscle function and falls in elderly people. *Am.J.Clin.Nutr*. **76**, 1454-6

John, E.M., G. G. Schwartz, D. M. Dreon and J. Koo (1999) Vitamin D and breast cancer risk: the NHANES I Epidemiologic follow-up study, 1971-1975 to 1992. National Health and Nutrition Examination Survey. *Cancer Epidemiol. Biomarkers Prev.* **8,** 399-406.

Krause R., Buhring M., Hopfenmuller W., Holick M.F. and Sharma A.M. (1998) Ultraviolet B and blood pressure. *Lancet* **352**(9129), 709-10.

MacKinley, A. F. and B. L. Diffey, (Eds.), A reference action spectrum for ultraviolet induced erythema in human skin, *CIE J.,* **6**(1), 17-22, 1987.

MacLaughlin, J. A., R. R. Anderson and M. F. Holick (1982) Spectral character of sunlight modulates photosynthesis of previtamin D_3 and its photoisomers in human skin. *Science*. **216**, 1001-1003.

Mathieu C., Waer M., Laureys J., Rutgeerts O. and Bouillon R. (1994) Prevention of autoimmune diabetes in NOD mice by 1,25 dihydroxyvitamin D_3 *Diabetologia* **37,** 552-8

Ponsonby A.L., McMichael A. And van der Mei I. (2002) Ultraviolet radiation and autoimmune disease: Insights from epidemiological research. *Toxicology* **181-182**, 71-78

Pugliese M.T., Blumberg D.L., Hludzinski J. Et al.(1998) Nutritional rickets in Suburbia. *J Am Coll Nutr* **17**(6) 637-641.

Robsahm T., et al. (2004) Vitamin D3 frm sunlight may may improbé the prognosis of breast, colon and prostate cancer. *Cancer Causes and Control* **15**

Scragg R., Jackson R., Holdaway I.M.,Lim T. And Beaglehole R. (1990) Myocardial infarction is inversely associated with plasm 25-hydroxyvitamin D3 levels: A community based study. *Int. J. Epidemiology* **19**, 559-563

Serhan E., Newton P., Ali H.A. et al. (1999) Prevalence of hipovitaminosis D in Indo-Asian patients attending a rheumatology clinic. **Bone** *25*, 609-11.

Schwartz G.G., Whitlatch L.W., Chen T.C., Lokeshwar B.L and Holick M.F. (1998) Human prostate cells synthesise 1,25-dihydroxyvitamin D_3 from 25-hydroxyvitamin D_3. *Cancer Epidemiology Biomarkers and Prevention* **7**, 391-395

Shettle, E. P. (1989) Models of aerosols, clouds, and precipitation for atmospheric propagation studies. *AGARD Conf. Proc.* **454**, 15-32.

Solomon, S., Stratospheric ozone depletion: A review of concepts and history, *Rev. Geophys.,* **37**(3), 275-316, 1999.

Tangpricha V., Pearce E.N., Chen T.C. and Holick .F. (2002) Vitamin D insufficiency among free-living healthy young adults. *Am J Med* **112**, 659 - 662.

Webb, A. R. , L. Kline and M. F. Holick (1988) Influence of season and latitude on the cutaneous synthesis of vitamin D_3: exposure to winter sunlight in Boston and Edmonton will not promote vitamin D_3 synthesis in human skin. *J. Clin. Endocrinol. Metab.* **67**, 373-378.

WMO (World Meteorological Organization), Scientific Assessment of Ozone Depletion: 2002, Global Ozone Res. and Monit. Proj., *WMO Rep. 47*, Geneva, 2003.

Zitterman A., Schulze Schleithoff S., Tenderich C., Berthold H., Koefer R. And Stehle P. (2003) Low vitamin D status: A contributing factor in the patogénesis of congestive heart failure? *J. American Colege of Cardiology* **41** (1), 105-112