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## Review Article

# Vitamin D, sun, sunbeds and health

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### Abstract

*Objective:* To review the health effects of solar radiation, sunbeds and vitamin D.

*Design:* The literature was searched in the electronic database MEDLINE to identify published data between 1981 and 2011. Studies were included if they reported relative risk for cutaneous malignant melanoma (CMM) associated with sunbed use, vitamin D and UV effects on human health.

*Setting:* Data from different time periods for populations at different latitudes.

*Subjects:* Persons of different ages and ethnic groups.

*Results:* UV from sun and sunbeds is the main vitamin D source. Young people with white or pigmented skin in northern Europe have a low vitamin D status. A number of health benefits from sufficient levels of vitamin D have been identified. However, UV exposure has been suspected of causing skin cancer, notably CMM, and authorities warn against it.

*Conclusions:* The overall health benefit of an improved vitamin D status may be more important than the possibly increased CMM risk resulting from carefully increasing UV exposure. Important scientific facts behind this judgement are given.

### Keywords

Cutaneous malignant melanoma  
Solar radiation  
UV radiation  
Vitamin D

Vitamin D in the human body comes from two sources: diet and exposure to ultraviolet B (UVB; 280–315 nm) radiation. The systemic effect of vitamin D produced by UVB action on the skin was demonstrated by the fact that UVB exposure of one arm of a child with rickets could cure rickets in the other arm<sup>(1)</sup>. Vitamin D is formed by UVB action on 7-dehydrocholesterol (7-DHC) in skin, where the product previtamin D is transformed to vitamin D and transported to the liver bound to vitamin D-binding protein in the blood, as is dietary vitamin D. In the liver vitamin D is hydroxylated to 25-hydroxyvitamin D (25(OH)D), which is transported to the kidneys and to many other tissues and hydroxylated to 1,25-dihydroxyvitamin D (1,25(OH)<sub>2</sub>D), the active hormone, known for its classical role in bone ossification. A level of 25(OH)D in the blood of 20–30 nmol/l is needed to avoid rickets<sup>(2)</sup>. Vitamin D gives many health benefits<sup>(2)</sup>, beyond bone and muscle health, but they require higher blood levels<sup>(3)</sup>. Sun exposure produces vitamin D with high efficiency, making it the main source of vitamin D even at high latitudes. One minimal erythral dose of UV radiation (a slight skin pinkness 24 h after exposure<sup>(3)</sup>) gives about 250–625 µg of vitamin D<sup>(4)</sup>.

Due to the fear of skin cancer<sup>(5–7)</sup>, health authorities warn against sun and sunbed exposure. This policy, as well as the recommended vitamin D doses, may need revision.

### UV from sun and sunbeds

The sun and sunbeds emit UVB and ultraviolet A (UVA). Sunbed tubes with high fluence rates of UVA are allowed for two reasons: (i) UVA wavelengths are not significantly absorbed by DNA and do not affect DNA directly<sup>(8)</sup>; and (ii) UVA produces skin tanning, both immediate pigment darkening (IPD) and delayed tanning. Tanning is thought to protect DNA and reduce carcinogenesis as indicated by the low skin cancer risk of dark-skinned people<sup>(9)</sup>. The positive effects of UVB are not known, only its carcinogenic potential. Thus, UVB levels are restricted to those in solar radiation which are sufficient to increase the vitamin D levels in the blood<sup>(10–13)</sup>: 10 min of exposure to sunbeds, twice weekly, give similar vitamin D levels as a daily intake of 50 µg of vitamin D<sup>(11)</sup>, or 5 teaspoons (25 ml) of cod-liver oil, and can bring a winter level of vitamin D up to a summer level (70–90 nmol/l), which may be optimal<sup>(3)</sup>.

### Sun and cutaneous malignant melanoma

Sun exposure is commonly supposed to be the main cause of cutaneous malignant melanoma (CMM) in most populations<sup>(9)</sup>. However, the matter is disputed<sup>(14)</sup>, and we have reviewed the arguments for and against a causation<sup>(7)</sup>.

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Several factors are probably involved, as exemplified by a relationship sometimes found between gross domestic product and CMM incidence<sup>(6)</sup>.

Intermittent sun exposure and severe sunburn in childhood are associated with an increased risk of CMM<sup>(15)</sup>. CMM incidence rates per unit skin area are larger on trunk (intermittently exposed) than on head and neck, while the opposite is true for basal cell and squamous cell carcinomas<sup>(7)</sup>. Occupational exposure (farmers, fishermen) and regular weekend sun exposure are associated with decreased risk of CMM<sup>(16,17)</sup>. Sun exposure may even protect against CMM on shielded skin sites<sup>(18,19)</sup>, and CMM arising on skin with signs of large UV exposure has the best prognosis<sup>(20)</sup>. UV exposure earlier in life is related to reduced overall and breast cancer<sup>(21)</sup>. It has also been observed that patients with the highest blood levels of vitamin D have thinner CMM and better survival prognosis from CMM<sup>(22)</sup>.

### Sunbeds and cutaneous malignant melanoma

A number of publications show conflicting results concerning the risk of CMM developing after sunbed use. Recent studies found that exposure to sunbeds has increased CMM risks<sup>(11,23–29)</sup>. These studies show that the use of sunbeds before 35 years of age significantly increases CMM risk. Some other studies show no increased CMM risk associated with sunbed use<sup>(30–34)</sup>.

Discrepancies between different studies may be related to differences between UVA/UVB ratios and intensities of the sunbeds. People who are using sunbeds frequently may also have higher than average sun exposure and it may be difficult to separate the effects of the two factors. There has been a significant increase in the number of sunbed exposures in Norway after 1990, but CMM incidence rates among persons younger than 50 years have stabilized<sup>(6,7,35,36)</sup>.

Figure 1 is an updated summary of the published studies on sunbed use and risk of CMM<sup>(23,25,31,37–40)</sup>. Some of the studies give conflicting results, such as an increased risk for women but not for men<sup>(37)</sup>. A recent study, including persons who were 18 years or younger between 1957 and 1977, gave among the highest odds ratios<sup>(28,39)</sup>. However, in this period there were very few sunbeds in Norway, so sunbed exposure cannot be the only risk factor for the increasing rates of CMM.

### Ultraviolet A and cutaneous malignant melanoma

UVA was reported to induce CMM with high efficiency in the small swordfish *Xiphophorus*<sup>(41)</sup>. The opossum *Monodelphis domestica* also develops CMM-like lesions after UVA exposure, but with low potency compared with UVB<sup>(42)</sup>. CMM are induced by UVB in a HGF/SF (hepatocyte growth factors/scatter factor) transgenic mouse model,

but not by UVA<sup>(43)</sup>. Furthermore, it has recently been noted that UVA did not induce melanomas in *Xiphophorus*<sup>(44)</sup>, so the UVA involvement in CMM generation is not solved experimentally.

Epidemiological investigations suggest that the use of sunscreens that absorb only UVB, but transmit UVA, may contribute to the risk of CMM<sup>(45,46)</sup>. Regular use of sunscreens absorbing both UVB and UVA perhaps reduces the CMM risk by approximately 50%<sup>(47)</sup>.

All of these findings have not resulted in any reduction of the allowed UVA fluence rates in sunbeds, which still may emit five to ten times more UVA than noon summer sun in southern Europe<sup>(46)</sup>. Most people get much more UVB and probably also more UVA from the sun than from sunbeds. This may not be true for frequent sunbed users.

The latitude gradient for CMM in Scandinavia, England, New Zealand and Australia is much lower than for non-melanomas<sup>(48)</sup>. Differences between Scandinavia and Australia are a factor of only two for CMM *v.* a factor of twenty to forty for non-melanomas<sup>(48)</sup>. The fact that the latitude gradient of ambient annual exposures is much smaller for UVA than for UVB (roughly a factor of 1.5 to 2.0 smaller) leads us to suggest that solar UVB is the main cause of non-melanomas and UVA may be CMM generating<sup>(49)</sup>.

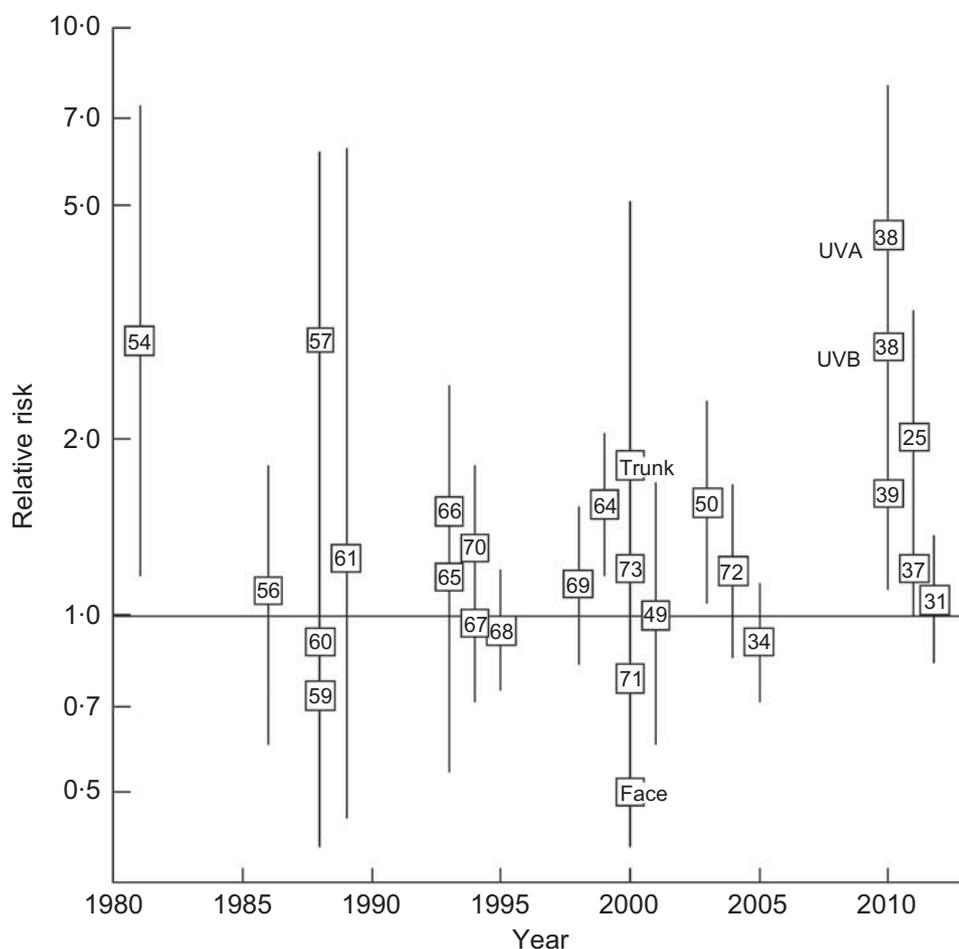
### Sun and vitamin D

There is a seasonal variation of vitamin D status as the sun is its main source<sup>(50)</sup>. Latitude gradients for blood levels of vitamin D have been recorded in the UK<sup>(51)</sup> and in France<sup>(52)</sup>, but international latitude gradients are not clearly documented. The reasons for this include varying methods of measuring vitamin D status<sup>(53)</sup>, varying skin types in different populations with less vitamin D produced in dark skin, and differences in intake of vitamin D-rich food in different countries. For example in Norway, people in the north eat more oily fish and consume more cod-liver oil than in the south<sup>(54)</sup>.

Young ethnic Norwegians and immigrants from southern countries in Norway have a low vitamin D status, notably in the winter<sup>(55)</sup>. This may be related to more indoor life. About 70% of 15-year-old persons spend more than 4 h daily in front of a computer or television<sup>(56)</sup>. They also spend more time on indoor activities and, therefore, less time out of doors during the day. Immigrants from South Asia usually cover their skin almost completely with clothes, and the women may cover their faces with veils. Compared with indigenous Norwegians they eat less vitamin D-containing oily fish and they have no tradition for cod-liver oil supplementation.

### Benefits *v.* risks of UV exposure

Using the relationship between CMM risk and UV exposure and the results published by Giovanucci *et al.*<sup>(57)</sup>,



**Fig. 1** Relative risks and 95% confidence intervals for cutaneous malignant melanoma associated with sunbed use in different studies. For results to 2005, numbers are references from the International Agency for Research on Cancer work<sup>(23)</sup>; for recent results (2010/2011), reference numbers are from the present study<sup>(25,31,37–39)</sup>

it can be estimated that increased sun exposure to the Norwegian population might at worst result in 200–300 more CMM deaths per year, but it would elevate the vitamin D status by about 25 nmol/l and might result in 4000 fewer internal cancers and about 3000 fewer cancer deaths overall<sup>(58)</sup>. The lack of sunlight exposure leads to more health problems than bone disease and increased risk for cancer<sup>(59)</sup>. Other benefits include protection against infectious diseases and non-cancerous diseases (diabetes, CVD, multiple sclerosis and mental disorders)<sup>(60)</sup>. New trials assessing higher doses of vitamin D supplementation are in progress and future research may more clearly demonstrate the benefits of vitamin D<sup>(59)</sup>.

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and J.M. had primary responsibility for the final content. All authors read and approved the final manuscript.

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