Subject: Comment to SCCP Opinion UV and sunbeds

Dear Sirs,

The Standing Committee on Radiation Hygiene of the Health Council of the Netherlands has taken notice of request for comments on the Preliminary Opinion on Biological Effects of Ultraviolet Radiation Relevant to Health with Particular Reference to Sun Beds for Cosmetic Purposes of the SCCP. Please find below our comments, drafted by dr FR de Gruijl:

Following a notification by the Spanish authorities to the European Commission on an omission in the European harmonisation standard EN 60335-2-27, the Commission Services requested a scientific opinion from the “Non-Food Scientific Committees”. The omission concerned inadequate design safety aspects of tanning devices, in particular limiting the UV irradiance. The scientific committee was asked to answer six questions relating to health aspects of UV exposure; the fifth question pertained to restricting UV irradiances.

General comments

The composition of the committee is somewhat remarkable as only one member is a reputable expert on UV health effects, and proper expertise on ophthalmology appears to be lacking. This composition probably explains some imbalances in the document (see below). Also, the restriction to external experts from the UK is rather surprising (again lacking ophthalmologic expertise), and appears to be reflected in the references.

Standard UV dose limits for indoor workers exposed to artificial sources (ACGIH, ICNIRP/WHO) are mainly dictated by effects to the eye for radiation of wavelengths below 300 nm. A ban from tanning devices of (‘unnatural’) radiation of wavelengths below 290 nm would appear advisable considering the exquisite susceptibility of the eye. In addition, the importance of proper eye
protection in 'unnatural' exposure conditions – as with artificial tanning – should be ardently emphasized.

Sunburn is clearly related to various adverse health effects, ranging from suppression of cellular immunity to increased risk of cutaneous malignant melanoma. And there is no indication of any beneficial effect to a healthy individual. Based on current experimental and epidemiologic data, there should be a strong directive to avoid sunburn! This SCCP document is too reserved on this issue, and fails to relay this important message sufficiently clearly (e.g. pg 21 line 6: “The avoidance of sunburn may also reduce the risk of melanoma”).

Beneficial versus adverse effects of sub-erythemal exposures have recently become much under debate, and may therefore require some comments; e.g., would the (non-cosmetic) use of artificial UVB sources be advisable to counter winter-time lows in vitamin D levels?

Type I skin does not tan and therefore has little or no cosmetic gain from UV exposure. People with this skin type mainly increase their risk of adverse health effects and should be strongly advised against using tanning devices for cosmetic purposes. The SCCP document fails to relay this message clearly.

The IEC classification of, and requirements for, tanning devices incorporated into EN 60335-2-27, are directed at (CIE erythemally weighted) UV irradiances of wavelengths below and above 320 nm. It is a glaring omission that the SCCP document does not refer to these IEC/EN 60335-2-27 standards (mentioned in the supporting document http://www.icnirp.org/documents/sunbed.pdf), and does not comment on their (in-)adequacy (it is equally surprising that the Terms of Reference from the Commission Services do not include explicit reference to the EN 60335-2-27 requirements).

The 0.7 W/m² CIE-weighted UV irradiation limit (about 2 SED in 5 minutes) proposed in the SCCP document appears to be more lenient than the IEC requirement for short-wavelength UV sources (with a ban on sources with > 0.15 W/m² output < 320 nm; adopted in EN 60335-2-27). A rationale for this is missing.
Basing the irradiance limit solely on the prevention of UV-induced erythema within 5 minutes of exposure is a clear simplification, which may be largely defendable, either because other biologic effects appear to be closely related to sunburn or because the wavelength dependence of other effects is unknown. However, this restriction ignores known differences between eye and skin sensitivities (see comments above on ocular effects and UVC).

Boundaries between UVA, -B and –C are fairly arbitrary and may historically be related to physical characteristics of filters, but the suggestion that it has no bearing on biology is clearly without grounds – the long term persistence of this nomenclature attests to some degree of usefulness (note that there were plenty of other UV filters which were not used to define these boundaries). Even when limited to skin erythema, there are significant distinctions between these bands: efficacy is orders of magnitude higher in the UVB band than in the UVA band and there are considerable differences in dose and time dependence between UVB and UVC-induced erythema. Also the predominant photochemistry in cells and skin changes from UVC to long wavelength UVA. This can lead to differences in wavelength dependence between erythema and other biologic effects.

Melanoma risks from tanning devices are presented in Table 3 as a reduction in number of melanoma cases if people would not have used these devices, and by mathematical modelling of attributable cases in the UK (Diffey, 2003). Neither method is very solid: the estimates in table 3 are based on inadequate assessments of UV exposures, and the mathematical model is debatable in its premises on the wavelength dependence, effective dose and dose-time kinetics (e.g., compare with Slaper et al, 1996).

Guidelines on limiting UV exposure from artificial tanning devices were already given by the Health Council of the Netherlands in 1986 by comparing annual solar UV exposures of indoor and outdoor workers and estimates of corresponding risks of skin carcinomas. The advice was that the exposure of indoor workers should be well below the median exposure of outdoor workers, and that tanning should not add more than 200 SEDs (then 100 standard MEDs for type I-II skin) to the total exposure. Later recommendations, as the one cited from the BPG, became more restrictive but were based on similar, or less well-founded risk assessments. It is a step back that the SCCP document makes no effort to answer question 6 in a similar concrete or even more
substantial and up-to-date manner. Annual dose limits should not be arbitrary but based on acceptable levels of risk, which is a political decision, weighing costs and benefits.

**Detailed comments**

- Pg 6: with erythema the blood volume in the superficial capillaries is increased, which explains the red coloration.
- Pg 7: the selection of references on immunologic effects is somewhat biased and does not appear to give sufficient credit to original work on the topics: the proper reference for UV effects on HPV should be given.
- Pg 7, bottom 2 lines: “.. resolves, without long term consequences, ..” is a scientifically unjustified inserted comment.
- Pg 8: actinic keratoses are more than just biomarkers of SCCs! They are benign precursors of SCCs, but the majority never progress to SCC within a life time.
- Pg 8: the literature indicates that BCCs can be both related to intermittent (BCC at young ages) and chronic (BCC at old age) exposures, where the former etiology appears to be more prominent.
- Pg 8: the insertion of text on vitamin D in the Melanoma section is misplaced.
- Pg 9: MC1R determines skin types, and appears to be a better indicator of skin cancer risk than skin type.
- Pg 9: nevus number may be genetically determined, but is also affected by childhood UV exposure (Gallagher et al, 2000, 1990). Clearly, an individual’s genetic background cannot be influenced, but sun exposure can.
- Pg 10: emphasis on familial melanoma is interesting but it attributes <10 % to the total melanoma incidence.
- Pg 10: the cross sectional melanoma age-specific incidences are widely different from the longitudinal ones! The latter show clear steep increases with age whereas the former ones are consequently less steep. In other words, the “relatively flat” age distribution is largely due to a trend of increasing incidences (within successive birth cohorts) with time.
- Pg 10: by far most melanoma in fair skinned people occur on trunk and legs; these are intermittently exposed skin areas, and this would appear to be of predominant importance (many epidemiologic studies show lower melanomas risks in outdoor workers, i.e., a ‘protective effect’ of chronic sun exposure).
- Pg 11: the sun is evidently not primarily a UVA source: visible and infrared radiant energy is greater.
- Pg 12: medical use of UV does not necessarily amount to lower exposures than cosmetic exposures.
- Pgs 12-13: it should be mentioned that psoriatic patients have a basically different (skin) immunity than healthy volunteers which may well affect the skin cancer risk under UV exposure.
- Pg 13: does the Mann et al (2005) reference include a control group for BCC risk or is it based on population-based registration, which generally underestimates the incidence?
- Pg 13, bottom: how does the poor penetration of UVC, which does not reach basal epidermal cells, explain a much less steep dose dependence of the erythema? This is not at all self evident!
- Pg 14: the most up-to-date and state-of-the-art action spectrum for erythema (by Anders et al, 1995) is not referred to, which is a clear omission. The deviations from the standard CIE action spectrum are very notable: most specifically, a pronounced peak around 300 nm, a very steep drop in effectiveness at wavelengths > 300 nm, and a plateau in the UVA range. The approximation with straight line segments in log-action versus wavelength plots clearly does not represent a biologically plausible and true action spectrum.
- Pg 14: the Ptc ko and transgenic hedgehog mice show enhanced BCC development under UV exposure (Aszterbaum et al, 1999), which appears to represent an aspect of human BCCs (i.e. introducing p53 mutations).
- Pg 14, bottom paragraph, 2nd sentence: the lack of (p53) signature mutations pertains to melanoma – which is not mentioned explicitly, i.e. UVB may not be causal for melanoma!
- Pg 16: reciprocity does NOT hold in every case; it has been proven for erythema provoked by exposure times < 2 hours. But reciprocity does not hold for SCC induction in mice, and epidemiologic data appear to show the same for humans: if the daily dose is increased two fold, the median induction time of a first tumour is not halved, but reduced by about 35%. Reciprocity does, however, hold for daily doses with exposure times < 2 hours, and action spectra based on daily doses can be determined.
- Table 2 is therefore not correct with regard to tumorigenesis in mice: the reciprocity column should consistently show ‘no’; the reference of De Gruijl et al, 1982 is inappropriate and should be De Gruijl et al, 1983, with a range of daily doses varying 33-fold.
- Table 2: under immunosuppression the study by Sontag et al, 1997, on the outgrowth of inoculations of UV tumours could be included with ‘yes’ for reciprocity.
- Pg 20: the formula here is rather meaningless in defining a percentage erythemal output: a monochromatic source at 295 nm would come out with 100% whereas one at 310 nm may come around 10% - the latter source is also fully erythemally effective. These percentages are therefore not useful for determining the spectral similarity of two sources, as suggested in this document (two widely different spectra can result in exactly the same percentages). Only by direct comparison of spectral shapes can conclusive information be obtained.

- Figure 3 is not entirely correct in showing a monotonous increasing probability with dose. The probability ranges between 0 and 1 and should therefore follow a sigmoid curve with dose, more like shown in the first panel for the severity of sunburn. It is rather the other way around: the severity is more likely to show a monotonous increase (if there were no saturation in red coloration). The cumulative hazard function (the average number of tumours per individual at risk) will show a monotonous increase with dose/age (until a saturation in this number occurs at doses well over the one at which 95 or 99% probability is reached).

Sub Q1:
MC1R is a better risk indicator than skin type.
Moles are also related to childhood sun (UV) exposure.

Sub Q2:
Most data indicate that sunburn and UVB radiation are determining the melanoma risk, however, more conclusive evidence is needed.

Sub Q3:
Here it is said that there are no quantitative data for long term effects, but on pg 23 the risk of melanoma is quantified (e.g. Table 3).

Ecological data on latitudinal and ambient UV in relation to skin cancer incidences constitute quantitative data on which crude population-based risk of skin cancer may be based (with additional data on percentage of outdoor workers etc). However, these estimates may not be reliable enough for far-reaching decisions on dose limits. Case-control studies on skin cancer risks provide no viable alternative because of the assessment of personal UV exposures by inadequate surrogates (mainly based on memory).

Sub Q4
Skin aging?
Sub Q5:
Ban UVC to protect eyes; emphasis on proper eye protection would be in order here.

Action spectra for various (suspected) effects in humans are not (well) known.

The erythemal action spectrum is used in dose limits to avoid adverse acute effects and related long term effects, but there is no claim to guarantee the adequacy in minimizing every kind of adverse effect.

Sub Q6:
Arguments are missing to limit additional exposure from artificial UV sources for cosmetic purposes to, for example, < 100 SED, following or criticising earlier guidelines.

An annual dose limit need not and should not be “arbitrary” but based on a political acceptable level of risk (compare with risks from ionizing radiation and imposed dose limits).

Additional references


Yours sincerely,

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Scientific Secretary
Standing Committee on Radiation Hygiene