HEALTH ISSUES OF ULTRAVIOLET TANNING APPLIANCES USED FOR COSMETIC PURPOSES

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The International Commission on Non-Ionizing Radiation Protection*

BACKGROUND

Consumer demand for a cosmetic tan is the economic basis of the "suntanning industry," which develops and distributes equipment for commercial suntanning and markets suntanning services to consumers.

Suntanning is caused by ultraviolet radiation (UVR).† Exposure from sunbeds and other tanning appliances has the same potential risks as exposure to the UVR in solar radiation. The term "sunbed" is frequently used to describe all tanning appliances consisting of either a single UVR-emitting lamp (emitting UVA and/or UVB radiation) as in some facial tanners or a number of such lamps incorporated into a bed, canopy, panel, or any combination thereof.

Potential adverse health effects of exposure to UVR are well documented and reasonably well quantified (WHO 1994). The purpose of this document is to summarize the potential adverse effects of exposure to ultraviolet radiation from tanning appliances and to provide recommendations to minimize the risks of such effects. Recommendations in this document apply o ly to the use of sunbeds for cosmetic purposes.

TANNING APPLIANCES: TYPES AND EMISSION CHARACTERISTICS

There are two distinctly different categories of ultraviolet appliances used in tanning applications, each with different UVR emission characteristics and different requirements for filtering to eliminate undesirable wavelengths:

- Low-pressure fluorescent tubes, emitting mostly UVA or mostly UVB, with broad-band or narrowband emission; and
- Filtered high pressure and high intensity discharge lamps, emitting virtually only UVA or a mixture of UVA and UVB.

Tanning appliances are currently the subject of an international standard established by the International Electrotechnical Commission (IEC 1995), which has regulatory status in some countries. Four types of appliances are recognized in this standard and defined by this standard as follows:

- UV type 1 appliances are those that emit UV radiation such that the biological effect is caused by radiation having wavelengths longer than 320 nm and characterized by a relatively high irradiance (≥ 0.15 W m^{-2})' in the range 320 nm to 400 nm. The emission at wavelengths less than 320 nm is limited to 0.5 mW m^{-2},
- UV type 2 appliances are those that emit UV radiation such that the biological effect is caused by radiation having wavelengths both shorter and longer than 320 nm and characterized by a relatively high irradiance (≥ 0.15 W m^{-2};) in the range 320 nm to 400 nm. The it-radiance at wavelengths less than 320 nm is in the range 0.5-150 mW m^{-2};
- UV type 3 appliances are those that emit UV radiation such that the biological effect is caused by radiation having wavelengths both shorter and longer than 320 nm and characterized by a limited it-radiance (≤ 0.15 W m^{-2}) in each UV radiation band; and
- UV type 4 appliances are those that emit UV radiation such that the biological effect is mainly caused by radiation having wavelengths shorter than 320 nm (at an irradiance greater than 0.15 W m^{-2}, and in the wavelength range 320-400 nm, the it-radiance is limited to 0.15 W m^{-2}).

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† The International Commission on Illumination (CIE) defines UVR as optical radiation between 100 and 400 nm, and this spectral region is divided into three photobiological spectral regions: UVC (l00-280 nm), UVB (280-315 nm) and UVA (315-400 nm).

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‡ All it-radiances are erythemally weighted. In their international standard, the International Electrotechnical Commission (IEC), has used the wave length ranges from 320-400 nm and <320 nm to characterize the UV radiation.

The emission characteristics and the health risks associated with the use of each type of appliance are different (Gies et al. 1986). Type 4 appliances, associated with high levels of emission of UVB (280-3 15 nm), are intended to be used following medical advice and should not be used for tanning purposes, mainly because of the publicized association between UVB and skin cancer. Most of the appliances that are in use today are UVA-emitting (315-400 nm) appliances, UV types 1, 2, and 3. The term "sunbeds" in this document refers to UV-emitting appliances of UV type 1, 2, and 3, although UV type 4 appliances are still marketed to commercial suntanning establishments.

During the last decade, increasing evidence of long term UVA-induced risks for the skin and the eye has led the sun-tanning industry to increase the UVB content in the emission spectrum of tanning lamps in order to more closely simulate natural sun exposure. This change has also permitted shorter tanning exposures. It is known that the ratio of UVA to UVB in the solar spectrum changes during the day and undergoes large variations according to season and latitude. It is also important to recognize that there is no firm scientific evidence to indicate that tanning with either UVA-dominated or a UVB-dominated sources poses less risk; and, likewise, the use of a simulated solar spectrum is not necessarily "safer" than other artificial sources.

EFFECTS ON SKIN

Tanning

When skin is exposed to UVR, two distinct tanning reactions ensue:

Immediate pigment darkening (IPD). IPD begins immediately on exposure to UVR and is caused by the darkening of the pigment melanin that is already present in the skin; it is normally seen only in people who have at least a moderate constitutive tan. Such pigmentation begins to fade within a few minutes after cessation of exposure. Radiation between 320 and 400 nm is regarded as being most effective for IPD (Irwin et al. 1993).

Delayed tanning (neo-melanogenesis). The visible pigmentation takes at least 3 d to develop and results from exposure to UVA but is more effectively produced by UVB (Parrish et al. 1982; Gange et al. 1985). UVR-induced neo-melanogenesis is strongly dependent on cellular responses arising from UVR-induced DNA damage in cellular nuclei (Eller et al. 1996; Gilchrest et al. 1996). Delayed tanning is more persistent than IPD and results from an increase in the number, size, and pigmentation of melanin granules (Bech-Thomsen et al. 1994). Exposure to UVB results also in an increase in the thickness and scattering properties of the epidermis (outer layer of the skin) (Bech-Thomsen and Wulf 1995). Due, at least in part, to these processes, the tan obtained from a pure UVA appliance, while perhaps cosmetically acceptable, is not as effective in protecting against further exposure to solar UVR as the equivalent pigmentation induced by exposure to solar radiation.

The degree to which an individual successfully tans as the result of exposure on a sunbed depends critically on his/her skin phototype as judged by his/her ability to tan and the susceptibility to sunburn as a result of exposure to solar UVR. Different skin phototypes (classified as phototypes I through VI) are presented in Table 1. In principle, the reaction of a person to UVR with respect to tanning or sunburning is similar whether the exposure is on a sunbed or to solar radiation.

Among users of artificial suntanning devices, persons of skin phototypes I and II, who do not tan well and/or who sunburn easily, are likely to be disappointed with the cosmetic results of using a sunbed. It has also been recognized that many users experience minor adverse cutaneous effects such as mild erythema, itching, and skin dryness (Diffey 1986; Rivers et al. 1989; Diffey et al. 1990).

Individual users of tanning appliances and attendants at tanning salons may incorrectly evaluate individual skin sensitivity to UVR and underestimate the user's sensitivity.

Sunburn

Minor sunburn is a skin reddening (actinic erythema) that appears up to 12 h after UVR exposure.

Table 1. Classification of skin phototypes based on their susceptibility to sunburn in sunlight and their ability to tan (Fitzpatrick et al. **1995).**

Skin phototype	Sun sensitivity	Sunburn susceptibility[a]	Tanning ability	Classes of individuals
I	Very sensitive	Always sunburn (<2 SED)	No tan	Melano-compromized[b]
IT	Moderately sensitive	High (2-3 SED)	Light tan	Melano-compromized[b]
III	Moderately insensitive	Moderate (3-5 SED)	Medium tan	Melano-competent
IV	Moderately resistant	Low (5-7 SED)	Dark tan	Melano-competent
V	Resistant	Very low (7-10 SED)	Natural brown skin	Melano-protected
VI	Very resistant	Extremely low (> 10 SED)	Natural black skin	Melano-protected

a The ranges of SEDs in parentheses are only indicative.
b Melano-compromized individuals have a greater risk of developing skin cancers than melano-competent individuals.

Erythema gradually fades after a few days, replaced by some tanning in individuals with tanning capability. Severe sunburn is painful and results in inflammation, blistering, and peeling of the skin. Aside from photoimmunological effects (discussed later), systemic effects of severe sunburn are unknown except for transient fever. Sunburn severity depends critically on skin phototype (Table 1) and UV dose. For fair-skinned people (skin phototypes I and II, melano-compromised), the relative effectiveness of UVR for tanning and for erythema is approximately the same over the entire range of UVB and UVA wavelengths (Parrish et al. 1982). For people who tan well and who rarely sunburn (skin phototypes III and IV, melano-competent), the tanning efficacy of UVA is higher than its erythemal efficacy (Gange et al. 1985). For a given sunbed type, there is a range of radiant exposures which can be expressed in terms of Standard Erythemal Dose (SED) unit related to the Minimal Erythemal Doses (MEDs),[§] that will produce noticeable effects in people of different skin phototypes.

Skin cancer

The most serious long-term effects attributed to UVR exposure of the skin are the skin cancers (IARC 1992). Squamous and basal cell carcinomas are common, rarely fatal forms of skin cancer, which are often referred to collectively as non-melanoma skin cancers (NMSCs). Experimental studies clearly indicate that UVA and UVB can cause squamous cell carcinomas (SCCs) in mice. The same effects are likely in humans (Sterenborg 1987; van Weelden et al. 1988). The erythemal potential of a UVR source is gaining acceptance as a reasonable approximation for a quantitative measure of its carcinogenic potential (Cole et al. 1985). A series of publications (Berg et al. 1993; de Gruijl et al. 1993; de Gruijl and van der Leun 1994; de Gruijl and Forbes 1995) has been a source for the proposal of an action spectrum for photocarcinogenesis by CIE TC 6-32 (CIE 2000). It will be used to evaluate the maximal number of authorized yearly sessions as the basis of a recommendation in the next revision of the international standard, IEC 335-2-27. Efforts to estimate the increased risk of a series of tanning sessions have been made; however, these have been limited to non-melanoma skin cancer, and such estimates necessarily require a number of assumptions such as the dose-response function, the use of ecological rather than individual-based data to estimate the relation between UV exposure and risk, the extent of natural exposure, and neglecting its intermittency. For example, estimates of the risk of incidence of NMSC due to the use of UVA sunbeds suggest a doubling of risk for no more than 20 sessions per year over 30 y in Northern Europe population (Diffey 1987). Despite these health risks, if individuals insist on acquiring a tan, there are conclusions that can be drawn from the scientific data to minimize risk such as presented in the recommendation. Based upon a modeling of human skin-cancer risk (Diffey 1987), 10 sessions of 30 min per year will increase by 5% the risk of skin cancer compared with non-users of solaria. A "safe" level of solarium use does not exist.

Cutaneous malignant melanoma (MM), while much less frequent than NMSC, is much more serious and accounts for the majority of deaths from skin cancer. There are some mammalian data (Ley et al. 1989; Ley 1997) for MM that indicate a strong UVB melanoma etiology, but these data are not entirely consistent with data from a fish melanoma model that indicate a strong implication of UVA in addition to UVB in the induction of fish melanoma (Setlow 1993). The animal models demonstrate an increased impact of neonatal exposure on tumor induction including malignancy (Robinson et al. 2000; Noonan et al. 2001). The evidence for the indictment of sunlight as a causal agent is limited to epidemiological data. The data indicate that intermittent exposure to high levels of solar UVR, particularly at an early age, may be a contributing causal factor (for recent review see Armstrong and Kricker 1995; Gilchrest et al. 1999). The individual risk of MM is higher in people who have a large number of nevi (moles) and who sunburn readily and tan poorly with exposure to solar UVR. Some data suggesting an association between the use of sunbeds and an increased risk of MM have been published, but it is unclear as to the relative importance of UVB, UVA, and other factors (especially, general sun-seeking behavior) in causing this association (Swerdlow et al. 1988; Walter et al. 1990; Autier et al. 199 1; Higgins and du Vivier 1992; Westerdahl et al. 1994; Autier et al. 1994; Spencer and Amonette 1995; Stem et al. 1997; Miller et al. 1998; Swerdlow and Weinstock 1998; Westerdahl et al. 2000).

Taken collectively, both experimental and epidemiological data on skin cancer all indicate that cumulative exposure increases the risk for skin cancers. This includes childhood exposures. Therefore, the added exposure from UV tanning appliances is likely to add to the detrimental consequences of natural solar exposure.

[§] A minimum erythemal dose (MED) is defined as the UVR exposure that will produce a "just noticeable" erythema on previously unexposed skin of an individual. If the exposure is spectrally weighted by the CIE erythemal action spectrum, the MED corresponds to an effective radiant exposure expressed in Standard Erythemal Dose (SED) units, depending on individual skin phototype (Table 1). Erythemally effective it-radiances (W m^{-2} eff.), expressed in SEDs per hour, can be calculated for any source of UVR using spectral k-radiance data for the source, at the point of interest, and the relative spectral weighting factors for erythema, promulgated by the CIE (McKinlay and Diffey 1987; CIE/ISO 1999). One SED (Diffey et al. 1997) is a CIE/ISO official unit, and effective radiant exposure of 100 J m^{-2}.

Premature skin aging

There is considerable evidence that cumulative UVR (UVA and UVB) exposure results in premature skin aging characterized by a dry, coarse, leathery, and wrinkled appearance. It has been clearly demonstrated that UVA causes skin damage in mice (Kligman et al. 1987; Bissett et al. 1989). Similar effects might be expected in humans as a result of excessive use of sunbeds.

Daily exposures to suberythemogenic purely UVA within the spectral region 320-400 nm for 8 d or exposure to longer UVA wavelengths between 340-400 nm for 2 mo result in cumulative morphological skin alterations, which are indicative of tissue injury (Lavker et al. 1995; Lowe et al. 1995; Seité et al. 1998). In a 5-year longitudinal study of women who used or did not use tanning salons (Piérard 1998), serious modifications of skin elasticity and extensibility were found in the tanning salon user group. In that group, the severity of skin disorders was inversely correlated with their natural pigment capacities. It has been concluded from the study that the unremitting use of sunbeds induces a functional decline of the dermis resembling premature aging.

Other skin effects

People who have excessively used UVA sunbeds have exhibited increased skin fragility and blistering (Farr- et al. 1988; Murphy et al. 1989) and atypical melanocytic lesions (Jones et al. 1987; Williams et al. 1988; Roth et al. 1989; Salisbury et al. 1989; Kadunce et al. 1990).

Photodermatosis and photosensitivity

Polymorphic light eruption (PLE) is a common photodermatosis readily produced in some people by exposure to UV sunbed radiation (Rivers et al. 1989). Other photoaggravated dermatoses such as systemic lupus erythematosus are also exacerbated by the use of UV sunbeds (Stern and Docken 1986). Certain medicines and chemical and topical products (Table 2) such as perfumes and lotions may cause skin photosensitization in sunbed users.

PHOTOIMMUNOLOGICAL EFFECTS

UVR exposure causes localized skin and systemic modifications due to photoimmunological reactions (Noonan and De Fabo 1990), also a particular concern with UVA sunbed radiation (Hersey et al. 1988; Rivers et

Table 2. Agents producing photosensitivity.

Agents	Incidence	Type of reaction	Effective wavelength range
Agents producing photosensitivity at local administration			
Sulphonamides and related chemicals (sunscreens, blancophores)	n.a.[a]	phototoxic and photoallergic	290-320 nm
Disinfectants (salicylanilide compounds in soaps and deodorants)	n.a.	phototoxic and photoallergic	290-400 nm
Phenothiazines (creams, dyes and insecticides)	n.a.	phototoxic and photoallergic	320-visible
Dyes	n.a.	phototoxic hyperpigmentation	Visible
Coal tar and derivatives (phenolic compounds)	n.a.	phototoxic	340-430 nm
Essential oils (perfumes and colognes)	n.a.	phototoxic hyperpigmentation	290-380 nm
Furocoumarines compounds (psoralens)	n.a.	phototoxic hyperpigmentation	290-400 nm
Cadmium sulphide (tattoos)	n.a.	phototoxic	380-445 nm
Agents producing photosensitivity after oral or parenteral administration			
Amiodarone	High	phototoxic	300-400 nm
Thiazide diuretics	Medium	photoallergic	300-400 nm
Chlorpromazine and related phenothiazines	Medium	phototoxic and photoallergic	320-400 nm
Nalidixic acid	High	phototoxic	320-360 nm
Non *steroid anti-inflammatory drugs*	Low	phototoxic photoallergic	310-340 nm
Protriptyline	High	phototoxic	290-320 nm
Psoralens	High	phototoxic	320-380 nm
Sulfonamides (bacteriostatic and antidiabetic)	Low	photoallergic	315-400 nm
Tetracyclines (antibiotics)	Medium	phototoxic	350-420 nm

[a] n.a. = not available.

al. 1989). The clinical improvement of atopic dermatitis with sub-erythemal UVA exposure indicates that UVR is able to modify substantially normal and pathological immunological reactions. UVB radiation can promote the development of skin cancer, perhaps by suppressing the immune system allowing the tumor to escape immune surveillance (Duthie et al. 1999). The action spectra in the UVB for mixed lymphocyte reaction and mixed epidermal cell lymphocyte reaction were found to be similar to the induction of thymine dimers upon DNA irradiation (Hurks et al. 1995).

There is also evidence that exposure to UVR can activate and accelerate the growth of human viruses (Otani and Mori 1987; Perna et al. 1987), including human immunodeficiency virus (HIV) (Zmudzka and Beer 1990), and have effects on infectious disease (Halliday and Norval 1997). At present, the significance of these observations with respect to human health is unclear. The effects of UVA exposure on the immune system are even more uncertain (Schwarz 1998; Vermeer et al. 1998).

OCULAR EFFECTS

The cornea

The principal adverse effect of the absorption of UVR (UVC and UVB) by the cornea is termed photokeratitis ("welder's flash" or "snow-blindness"), and damage is generally limited to the epithelial (front surface) cells of the cornea (Sliney and Wolbarsht 1980). After a 6- to 12-h latent period that depends inversely on the severity of the exposure, there is severe cornea1 pain, photophobia, lacrimation, and eyelid spasm. These symptoms are terribly distressing (with incapacitation), but typically resolve in 24 h. There is some evidence of possible long-term effects of UVR absorption by the cornea (Taylor et al. 1989) and evidence of endothelial thinning (Pitts et al. 1987).

The lens

Transmission of UVA to the lens is much greater than that of UVB. Animal data indicate that threshold lenticular damage is limited mainly to UVR exposure in the 295 to 325 nm wavelength band. Experimental spectral efficiency for acute cataracts in laboratory animals has been measured only in this spectral region. Although UVA sunbeds produce limited 295-325 nm radiation, it should not be inferred that UVA is safe with respect to lens exposure. Crystalline lens aging is characterized in part by loss of elasticity and browning (brunescence), both of which may be caused partly by UVA. Certain medicines may act as UVA photosensitizers of the crystalline lens.

The retina

At least for the chronic effects of exposure to solar radiation and to radiation from conventional light sources, the most important retinal damage mechanism is photochemical injury from short-wavelength light (Sliney and Wolbarsht 1980). The gradual brunescence of the lens as it ages results in its decreased transmission of blue-light and of UVR thereby affording increased protection to the retina. Young children and people who have had a lens surgically removed (aphakes) are at a higher risk of retinal damage from UVR and blue-light. Until the last decade, many implanted artificial lens did not effectively absorb UVA. Macular injuries to two tanning booth users were mentioned in one study but not confirmed (Walters and Kelley 1987).

The crystalline lens and cornea serve in considerable measure to protect the retina from most UV tanning booth radiation, even without protective goggles. As discussed previously, the crystalline lens blocks UVR below 400 nm and the cornea blocks UVR below 300 nm, but trace amounts of UV-B radiation between 300 and 315 nm may reach the retina (Boettner and Wolter 1962; Sliney 1986).

Protective eyewear

The use of protective eyewear (goggles) will prevent exposure of the eyes to harmful levels of UVR and blue-light. This represents a very important issue for sunbed exposures since an individual is normally protected from most of the overhead solar UVR by geometrical shading by the brow ridge and upper lids. The exposure from sunbeds is geometrically greatly different. Furthermore, oblique rays can be focused into the nasal equatorial region of the lens (Coroneo et al. 1991). UVR can only reach the critically important germinative region of the lens by the focusing of oblique rays. Eyewear that does not incorporate side protection is not suitable to protect the eye from lateral UVR.

CONCLUSION AND RECOMMENDATIONS

A review of scientific evidence shows that solar UVR is a cause of squamous cell cancer, basal cell cancer, and cutaneous melanoma as well as causing accelerated skin aging and other adverse health effects. Because of this strong evidence on the adverse health effects of UVR, even though there is not conclusive direct evidence that sunbed exposure causes skin cancer, it is ICNIRP's view that any use of suntanning appliances is likely to raise the risk of cancer. This risk is particularly high for people having skin phototypes I and II and for children.

ICNIRP, therefore, recommends against the use of W-emitting appliances for tanning or other non-medical purposes. The following groups are at particularly high risk of incurring adverse health effects from UVR, and therefore should be particularly counseled against the use of tanning appliances:

- People who have skin phototypes I or II;
- Children (i.e., less than 18 y of age);
- People who have large numbers of nevi (moles);
- Persons who tend to freckle;
- Individuals who have a history of frequent childhood sunburn;
- People who have premalignant or malignant skin lesions;
- People who have sun-damaged skin;
- Those who are wearing cosmetics. These may enhance their sensitivity to UV exposure; and
- Persons taking medications. In this case they should seek advice from their physician to determine if the medication will make them UV-sensitive.

Although contrary to the above recommendation, if persons decide to use suntanning appliances, steps should be taken to minimize the risk. The recommendations provided in Appendix A have been made by several workshops, meetings, and publications which have pointed to the identification of optical radiation hazards associated with the development and popularity of the "suntanning industry" (Council on Scientific Affairs 1989; ICNIRP/CIE Measurements of Optical Radiation Hazards 1998; Miller et al. 1998; Moseley et al. 1998; USNIH 1998; Greinert et al. 2001). ICNIRP generally supports these recommendations as a likely means to reduce the risk of skin cancers from the use of UV tanning appliances and particularly to minimize the risks for high-risk groups. Suggestions for further reading can be found in Appendix B.

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APPENDIX A

If tanning devices are used, then the following specific recommendations from past workshops should apply:

- Claims of beneficial medical effects should not be made. Any therapeutic use of tanning devices should be done only in medical units;
- Tanning devices should comply with the requirements of the IEC standard (1995) and be limited to UV type 1, 2, or 3 as defined in that standard;
- Appropriate health warnings should be provided to the client prior to tanning exposure;
- Appropriate UV-protective goggles should be provided and worn during tanning exposures;
- Operator staff should be provided with appropriate approved training (receive appropriate certification);
- Professional operators are responsible for providing client information and guidance on the safe use of tanning devices;

- Minimize the number of sessions. For example, the French Regulations (J. 0. Republique Francaise, Annexe 2, 1997) require that regular exposure, for phototypes III and IV, melano-competent skin, should not exceed two sessions per week with a maximum of 30 sessions per year (erythemally effective exposure of 500 J m^{-2} per session). An occasional break from the regularity of exposure is advisable;
- Manufacturers or dealers must supply exposure schedules based on the tanning device lamp characteristics;
- Because the sensitivities of individuals vary greatly, it is advisable to limit the duration of the first session to about one-half of a regular session in order to establish the user's skin response. If following the first session any adverse reaction occurs, further use of the sunbed should be discouraged;
- Products designed to enhance or accelerate tanning should not be used;
- Any modifications, such as the replacement of lamps, filters or reflectors should not change the IEC classification of the device. Tanning devices should have an appropriate timer;
- Tanning devices in hotels or in recreational facilities should be subject to the same controls as noted above (as for any commercial outlet);
- Because of their possible misuse, unattended or coin-operated tanning devices should not be used;
- By the nature of their use, sunlamps in the home are not subject to the same degree of control as those used under proper supervision in commercial outlets, so additional safety information should be provided by the vendor or supplier of the tanning device. In these circumstances only IEC type 3 tanning devices should be used.
- Recognizing that different countries will have different ways of implementing and determining compliance with these recommendations, the tanning facilities should comply with these recommendations, and that compliance should be checked by the appropriate national authority where possible.

APPENDIX B

Further readings

- American Academy of Dermatology. Position Statement on Indoor Tanning. Dermatology World; March 1999.
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- NRPB Use of Sunbeds and cosmetic tanning. Statement by NRPB Advisory Group on Non-Ionising radiation. Radiological Protection Bulletin No. 2 18, 11-15; 1999.
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- Swedish Radiation Protection Institute: Regulatory code concerning sunbeds (SSI FS 1998: 2).
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