Strahlenschutzkommission

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Protection of man against the hazards of solar UV radiation and UV radiation in solaria

Recommendation of the German Commission on Radiological Protection

Adopted at the 280th meeting of the German Commission on Radiological Protection on 11 and 12 February 2016

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Schutz des Menschen vor den Gefahren solarer UV-Strahlung und UV-Strahlung in Solarien

Empfehlung der Strahlenschutzkommission

This translation is for informational purposes only, and is not a substitute for the official statement. The original version of the statement, published on www.ssk.de, is the only definitive and official version.

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1 Introduction

The German Commission on Radiological Protection (SSK) started publishing recommendations and information about protecting humans against UV radiation in 1990 as a reaction to the constantly increasing incidence of skin cancer among humans resulting from ultraviolet radiation. The recommendation "Protection of man against exposure from solar radiation and UV irradiation units" was issued in 1990. Following this and several other publications, the SSK issued the "Practical behaviour recommendation to protect against skin cancer resulting from ultraviolet radiation" in 1993 and the recommendation "Protection of man against solar ultraviolet radiation" in 1997. These documents were then added to in 2001 by issuing another recommendation "Protection of man against the hazards of UV radiation in solaria" (SSK 1990, SSK 1993, SSK 1997, SSK 2001).

Scientific annexes included in the above SSK recommendation texts referred to the current state of research at that time, on the basis of which recommendations were issued on how to handle solar and artificial UV radiation with little risk.

After having already classified solar UV radiation as being carcinogenic to humans, in 2009 the World Health Organization (WHO) also classified UV radiation from artificial sources as being carcinogenic to humans. A broader set of data and new scientific findings are now available that can be incorporated into existing recommendations on the protection of man against solar UV radiation and UV radiation from artificial sources.

This text embodies and represents the results of that work, and replaces the recommendations and scientific backgrounds from 1990, 1993, 1997 and 2001 described above. The remaining information, statements and recommendations the SSK has previously issued about UV radiation highlight certain individual aspects and therefore remain effective without change. This applies in particular to the SSK statement issued in 2006 titled "Hazards from UV exposures of children and adolescents" which investigates ways to protect children and adolescents from the risk of UV radiation (SSK 2006). In this statement, the SSK recommends that children and adolescents receive regular and more in-depth instruction on the potential risks of UV radiation. The SSK also called upon legislators to prohibit children and adolescents below the age of 18 from using solaria. The German government followed this request by enacting the "Act on the protection against non-ionising radiation used in humans" (NiSG 2009) in 2009.

As requested in the advisory mandate for this recommendation which was issued by the Federal Ministry for the Environment, Nature Conservation and Nuclear Safety (BMU, today known as the Federal Ministry for the Environment, Nature Conservation, Building and Nuclear Safety - BMUB), "the effect of UV radiation on the formation of endogenous vitamin D" has been investigated as it is a tricky topic due to publications from the last decade largely having been interpreted such that thorough UV protection could lead to a lack of vitamin D among the majority of the population. This also gives rise to discussions as to whether a vitamin D deficiency could be expedient to or may negatively affect the development of a number of illnesses, e.g. cancer, in various organs.

Discussions surrounding vitamin D deficiency have led to an increase in the number of people using solaria over the last few years, and to an overall increase in solar UV exposure. The assumption here is that potential biopositive effects of UV-induced vitamin D synthesis outweigh the known health risks of UV radiation.

The SSK is monitoring this development closely, which is why it has reviewed its UV recommendations based on new findings. As a result of this review and the debate surrounding the effects of UV-induced vitamin D on health, the SSK does not see any fundamental need to

change the previous recommendations issued with regard to UV protection and minimising the risk of skin cancer.

2 Recommendations

The following recommendations are issued for skin types I to IV.

2.1 General recommendations

In October 2014, the European Code against Cancer (ECAC) was published on behalf of the World Health Organization (WHO) (IARC 2015).

The SSK follows the general recommendations provided by the International Agency for Research on Cancer (IARC), which were jointly funded at an international level. The recommendations are as follows:

– Avoid excessive[1] UV exposure; this applies to children in particular,
– Application of sun protection measures,
– No use of solaria.

The SSK has taken these general recommendations into consideration while putting together its own recommendations for the general public on how to protect against UV radiation, with a particular focus on UV protection for children.

2.2 Recommendations to protect against solar UV radiation

A person's solar UV radiation exposure level depends on the time of day, the time of year, their geographical location, the given weather conditions, and their individual behaviour. The UV Index is used to measure the level of solar UV irradiation relevant to sunburn (see Chapter 4 of the scientific background). UV protection measures should be based on the UV Index level and the WHO recommendations provided for that level.

The following order is recommended when applying sun protection measures:

– Avoid strong solar UV exposure
– Wear suitable clothing and sunglasses
– Apply sunscreen

The following measures should be taken to avoid strong solar UV exposure:

– Take note of the UV Index forecast when planning outdoor activities
– Avoid getting sunburn at all costs when spending time in the sun
– Seek shade

[1] When defining "excessive" UV exposure, various factors need to be taken into consideration, including individual, constitutional skin cancer risk as well as the highly variable UV radiation intensity relevant to sunburn quantified by the UV Index. The photophysical and photobiological principles described in the scientific background, the UV Index description (see Chapter 4) and the representation of individual risk factors (see Chapter 3.2.1.4) should enable an individual, situational estimate of excessive UV exposure as well as the targeted use of protective measures recommended below.

- Go outdoors in the morning or evening rather than during the middle of the day
- Give the skin time to get used to the sun (e.g. in spring or when on holiday); when on holiday, take account of the risk of high UV exposure when close to the equator, high up in the hills, or near water
- Keep the amount of time spent outdoors to a minimum when there is a high UV Index level

The following points should be observed when wearing suitable clothing and sunglasses:

- Wear clothing with sufficiently dense fabric that covers the majority of the body
- Cover the head with a broad-brimmed hat
- Wear footwear that covers the instep
- Wear sunglasses bearing the UV-400 symbol that offer sufficient protection against UV radiation from the side and which carry the applicable standard (DIN-EN ISO 12312-1) (DIN 2013)
- Never look directly at the sun, even when wearing sunglasses

Bear the following points in mind when applying sunscreen:

- Any areas of skin not covered by clothing should be protected using sunscreen with a high sun protection factor (SPF)
- Sunscreen should be applied evenly and in sufficient quantity (2 mg/cm²)[2] in order to achieve the stated sun protection factor (SPF)
- Only sunscreen should be used that provides full details of the UV range filtered out (UVA and UVB) as well as full details of the ingredients in order to ensure comprehensive UV protection and prevent allergic reactions from taking place

When spending time in the sun, perfumes, deodorants and cosmetics should not be used.

Anyone who takes medication should read the included instructions before spending time in the sun and, if necessary, consult the physician who prescribed the medication or ask a pharmacist.

The behavioural prevention measures stated above should be accompanied by technical measures to reduce solar UV exposure in terms of situational prevention (creating shade by means of a canopy, awning, etc.). These technical measures should in turn be accompanied by organisational measures, especially around noon (e.g. when organising sports events).

2.2.1 Protection of children against UV radiation

The following points should also be taken into consideration when protecting children against UV radiation:

- Infants should not be directly exposed to the sun
- Children’s eyes should be protected by wearing suitable children’s sunglasses with the quality characteristics described above

The following situational prevention measures are highly recommended for children:

[2] European Commission, Enterprise and Industry Directorate-General. Commission recommendation of 22 September 2006 on the efficacy of sunscreen products and the claims made relating thereto, 2006/647/EC. J.Eur. Union L265, 39 - 43 (2007).

- Shaded areas should be provided at crèches, preschools and schools; a sufficient number of shaded areas of ample size should be provided and care should be taken to keep residual UV exposure (e.g. due to scatter radiation) to a minimum
- Technical measures (i.e. provision of shaded areas) should be accompanied by organisational measures to reduce UV exposure, especially around noon (e.g. when planning schedules and organising sports events)

2.3 Recommendations for the protection of man against the hazards of UV radiation in solaria

The German UV Radiation Protection Ordinance (UVSV 2011) provides out a number of requirements that must be met when operating solaria. One such example is the fact that since January 2012, only UV irradiation devices with a maximum erythema-weighted irradiance of 0.3 W/m² are permitted for use. The legal basis for this ordinance is the "Act on the protection against non-ionising radiation used in humans" (NiSG 2009). After its enactment in August 2009, solaria operators are no longer permitted to allow minors to use their solaria and may be subject to a large fine should they break the law.

The SKK welcomes this law as it can make a major contribution to reducing UV-induced risks, particularly the occurrence of skin cancer. However, the SSK is concerned that German legislation and concepts based on scientific findings and recommendations for protecting the public against the hazards of UV radiation are largely called into question by standardisation bodies at national and international level in order to safeguard economic interests. The SSK is particularly unsettled about the fact that solaria still claim to be able to treat certain illnesses (e.g. psoriasis, neurodermatitis, acne).

Solaria visits represent an additional avoidable exposure of the human body to UV radiation. In light of this and the fact that UV radiation has been clearly proven to be carcinogenic (El Ghissassi et al. 2009), the SSK recommends:

- No use of solaria
- Ensuring that legislation pertaining to solaria is strictly enforced. Solaria operators must be subject to regular inspections to ensure that they comply with the German UV Radiation Protection Ordinance (UVSV)
- Commercially operated solaria should be prevented from advertising their services for therapeutic purposes. The treatment of certain illnesses by using UV radiation requires medical indication and should only be performed under medical supervision in clinics or specialist practices

2.4 Special consideration of the vitamin D problem

Sufficient vitamin D synthesis is ensured if a person's uncovered face, hands, arms and lower legs are subject to half of the minimum UV dose relevant to sunburn (0.5 *MED*) two to three times per week without wearing any sunscreen. For people, e.g. with skin type II, who spend time in the sun every day, this corresponds to about 15 minutes at a UV Index of 7 (which may be reached in the middle of summer in Germany) or about 30 minutes at a UV Index of 3. During winter in Germany, i.e. from mid-October to mid-March, it is practically impossible to form vitamin D at a UV index < 3. Even if a person is suspected of having a vitamin D deficiency, the SSK strongly advises against the use of solaria for health reasons and/or due to a low level of solar UV exposure owing to the time of year because solaria visits are associated with an increase in the risk of skin cancer. A diagnosed vitamin D deficiency should be treated by a doctor using a suitable form of medication.

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Scientific background to the recommendation of the German Commission on Radiological Protection

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1 Introduction

Life on Earth would not be possible without sunlight. Parts of the visible light emitted by the sun are needed for photosynthesis and therefore influence our biorhythms. Infrared (IR) radiation emitted by the sun heats up the Earth. Around 4% of optical radiation from the sun that reaches Earth is ultraviolet (UV) radiation. Vitamin D production in humans requires a certain UV radiation level and dose that is absorbed via the skin. However, it has also been proven that UV radiation is mutagenic and has a carcinogenic effect on humans. This is due to the fact that UV radiation damages genetic material (DNA) and other cellular components of human skin cells and the eye. UV radiation also weakens the immune system, which leads to a number of UV-dependent illnesses whose aetiology is yet to be fully explained. However, UV radiation has been proven to be the environmental risk factor in the formation of skin cancer, the frequency of which has increased drastically in Germany over the last few decades. Knowledge of this main risk factor (UV radiation) allows for the development of behaviour and situational prevention (primary prevention) measures to combat the formation of skin cancer. When still at an early stage, skin cancer can be easily cured, thus also making it a target cancer for early detection (secondary prevention).

2 Solar and artificial UV radiation

2.1 Definition of optical radiation/UV radiation

2.1.1 Optical radiation – a form of energy within the electromagnetic spectrum

Ultraviolet radiation is an optical radiation range within the electromagnetic spectrum.

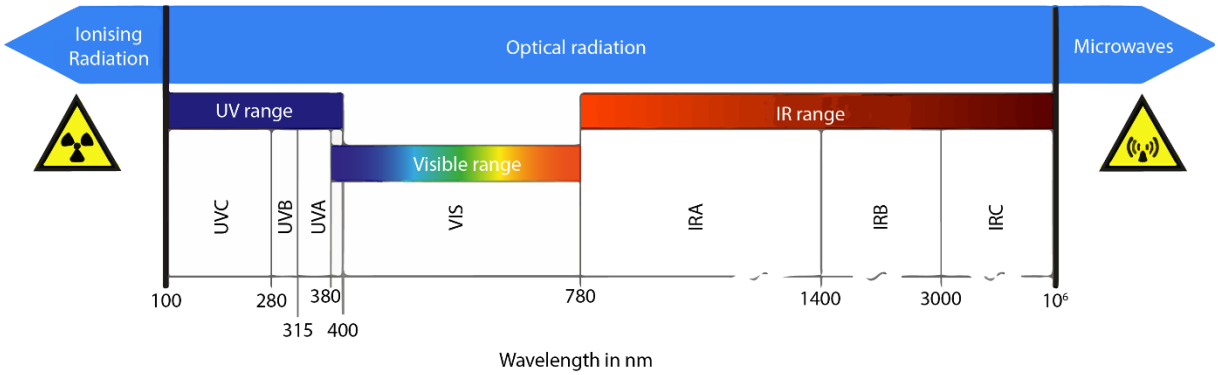

Fig. 1: UV range as part of optical radiation within the electromagnetic spectrum (© BAuA Dortmund 2011)

Ultraviolet (UV) radiation consists of an electromagnetic wavelength ranging from 100 nm to 400 nm (see Fig. 1). The UV radiation range is subdivided further, and the boundaries for these subdivisions are provided in Table 1. There are differing definitions for the UVB and UVA boundaries (315 nm or 320 nm) and for the UVA and visible light boundaries (380 nm or 400 nm). The values provided in Table 1 follow those set out in the "International lighting vocabulary" issued by the International Commission on Illumination (CIE 1987) and are therefore used in this scientific background.

Table 1*: UV radiation ranges*

Range	λ **in nm**
UVA	from 315 to 400
UVB	from 280 to 315
UVC	from 100 to 280

2.1.2 Emission spectrums of UV radiation sources

Depending on the radiation source, UV radiation sources may have continuous or discontinuous emission spectrums. A discontinuous spectrum may be a "line spectrum" composed of discrete emission lines, but it could be a band spectrum in which radiation is emitted at certain wavelength bands. A monochromatic spectrum is a special line spectrum with just one single emission wavelength.

2.1.3 Characterisation of UV radiation – relations, quantities, symbols, units

The quantity used to measure the intensity of optical radiation hitting a surface is known as irradiance *E*. The exposure of the irradiated object is calculated by multiplying exposure time Δt_{exp} (in s) by irradiance *E* (in W/m²) and is expressed as exposure *H* ("dose" in J/m²):

$$H = E \cdot \Delta t_{exp}. \tag{1}$$

The following equations apply in the event of varying irradiance:

$$H = \sum_{\Delta t_{exp}} E(t)\Delta t \qquad \text{or} \qquad H = \int_{\Delta t_{exp}} E(t)\mathrm{d}t. \tag{2}$$

The emission of an optical radiation source is characterised by the spectral radiance distribution. The wavelength-dependent radiation intensity is characterised by the spectral distribution of the irradiances per wavelength interval and expressed as spectral irradiance $E\lambda(\lambda)$ [W/(m²·nm)].

Photobiological effects triggered by UV radiation are largely dependent upon the wavelength and therefore upon the photon energy (see Chapter 2.2.3). This wavelength-dependent biological effectiveness is known as the impact spectrum or action spectrum. In order to express this mathematically and perform measurements for evaluation purposes, this photobiological effect action spectrum is approximated using a spectral weighting function $s_{biol}(\lambda)$ for UV radiation – so for UV erythema it is approximated using $s_{er}(\lambda)$.

The photobiologically weighted irradiance E_{bio} takes account of the spectral irradiance $E\lambda(\lambda)$ by means of integration across every wavelength interval, multiplied by the spectral weighting function $s_{biol}(\lambda)$ of the photobiological effect within the observed range of λ_1 to λ_2.

Expressed mathematically, this results in the following photobiologically weighted illuminance:

$$E_{biol} = \int_{\lambda_1}^{\lambda_2} E_\lambda(\lambda) \cdot s_{biol}(\lambda)\mathrm{d}\lambda\,. \tag{3}$$

This formula is also used to determine erythemally effective irradiance from solar UV radiation during a day with the aim of determining the UV Index (see Chapter 4). UV radiation with a weighted irradiance E_{biol} over an exposure time of t_{exp} results in a biologically effective exposure H_{biol} (also often known as "UV dose"):

$$H_{biol} = \int_{\Delta t_{exp}} E_{biol}(t)\mathrm{d}t \tag{4}$$

The photobiologically weighted irradiance on a person may change over time: $E_{biol}(t)$. This may be caused by a change to the distance from the UV radiation source or due to intermittent periods of time spent in the radiation field, or by a change to the UV radiation source emission itself.

Annex 1 contains an overview of photobiological quantities that serve to characterise UV radiation and exposure. It also provides a description of the quantities, symbols and units as well as references for further information.

Unweighted physical quantities are not a suitable unit for photobiological assessments. The results of unweighted measurements take equal account of all of the wavelengths. The photobiological effect on structures also consisting of microorganisms or organic tissue is, to a large extent, often dependent upon the radiation wavelength across several orders of magnitude. The photobiological weightings described above take account of this, with said weightings differing from one another depending on the observed effect.

2.2 Physical basis of the photobiological effects of UV radiation

2.2.1 Dependency of irradiance on incidence angle

Irradiance depends on the incidence angle θ. Maximum radiant flux per area occurs in the event of vertical incidence (θ = 0°). As the incidence angle increases up to the point where it merely glances an object, the irradiated area increases while irradiance decreases. The dependency of irradiance on the incidence angle is based on the cosine law

$$E = E_0 \cos\theta \tag{5}$$

where

E_0 is irradiance E on a flat surface for $\theta = 0°$

θ is the angle between the incident beam and perpendicular on the irradiated area

Depending on the anatomic region of the body or the body's momentary position, the skin will be irradiated by the sun at various angles of incidence. Due to the angle dependency described above, the so-called "sun terraces" (e.g. nose, shoulders) will first reach the sunburn threshold dose when exposed for the same period of time because they have the steepest angle of incidence for solar UV radiation.

2.2.2 Wavelength-dependent penetration depth of UV radiation into tissue

The penetration depth of UV radiation depends on the wavelength (see Fig. 2) and is influenced by the absorption behaviour of so-called chromophores (light-absorbing molecules) as well as reflection in the tissue. Skin thickness and thickness of the individual skin layers vary significantly and depend, among other things, on the localisation of the area of skin under consideration. As a result, in vivo transmission for the various skin layers can only ever be approximated. Fig. 3 contains a data table resulting from an investigation into skin penetration depth for various wavelengths. The penetration depth increases as the wavelength increases. At 250 nm (UVC, only emitted by artificial UV radiation sources), around 82% of the radiation is absorbed in the first 20 µm. At 297 nm (UVB), around 90% of the radiation is absorbed in the first 40 µm. At 365 nm, UVA radiation penetrates a depth of more than 20% of the epidermis. Within the transition range between UVA and visible light, around 60% of the radiation reaches the cutis (epidermis and dermis). Around 1% reaches the subcutis (Bruls et al. 1984).

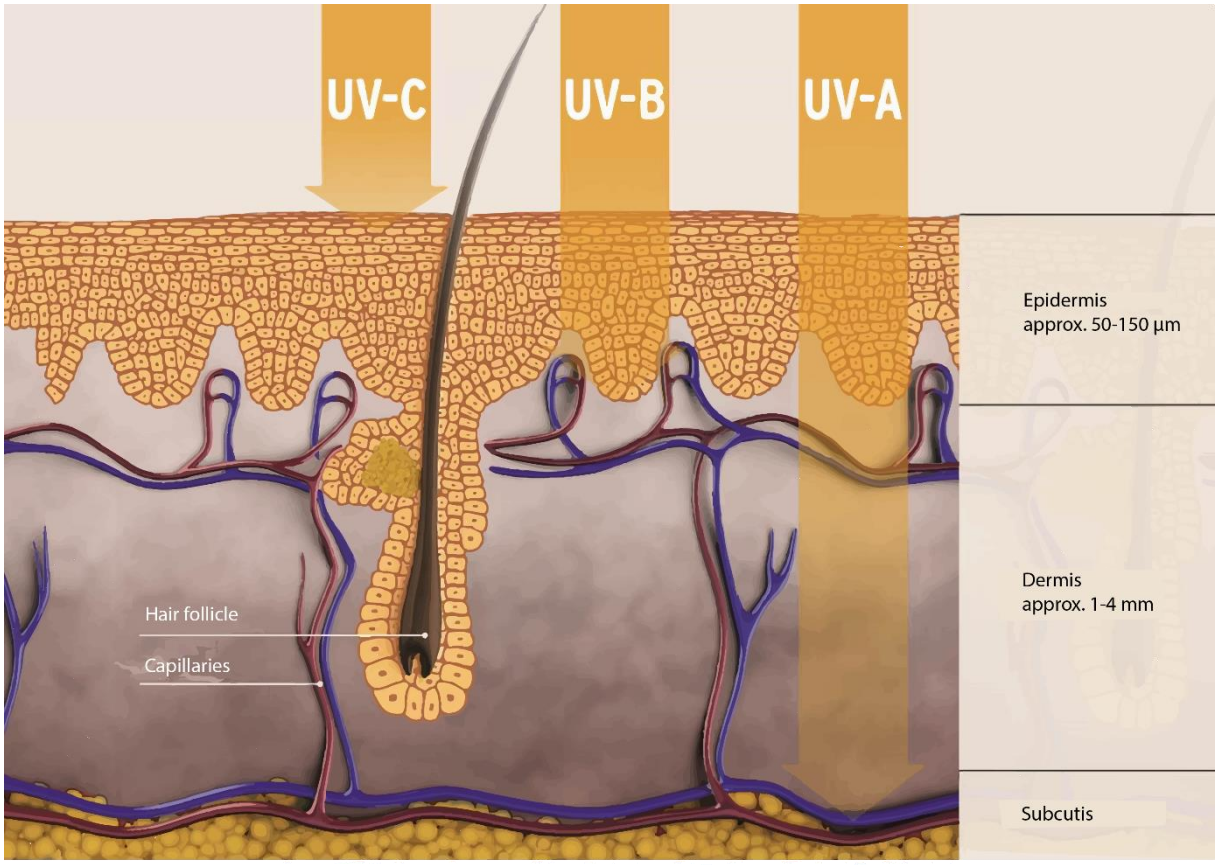

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Fig. 2: Penetration depth of UV radiation into the skin

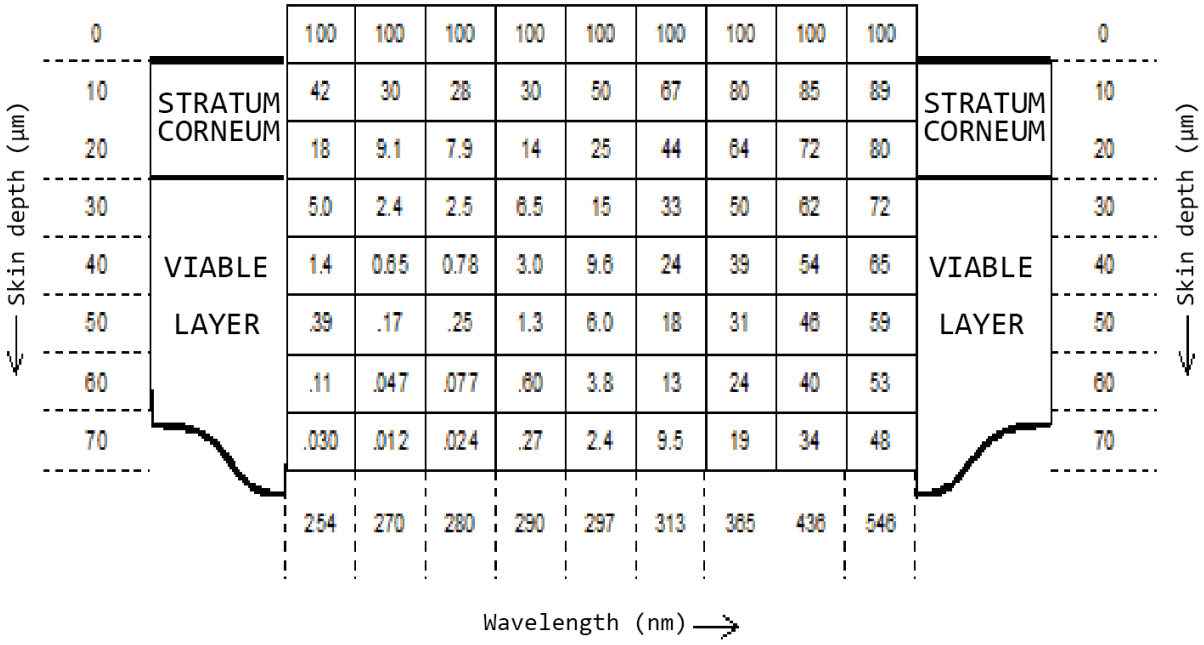

Fig. 3: Approximated in vivo transmission (in %) into various tissue depths for several wavelengths (according to Bruls et al. 1984)

The penetration depth of UV radiation into the eye also depends on wavelength (see Fig. 4). The penetration depth can be roughly expressed as follows (see Table 2; the transitions are smooth, however):

Table 2: Penetration depth of UV radiation into the eye (adult)

Radiation type	Penetrates the eye to the
UVC	Cornea/conjunctiva
UVB	Lens of the eye
UVA (98 – 99%)	Lens of the eye
UVA (1 – 2% > 365 nm)	Retina

UVB radiation is fully absorbed by the human lens of the eye. This also applies to UVA radiation with wavelengths below 365 nm. However, 1-2% of UVA radiation above 365 nm still reach the retina (Glickman 2011, Krutmann et al. 2014, Mainster und Turner 2010). A child's eye represents an exception to this (see Chapter 3.3.1).

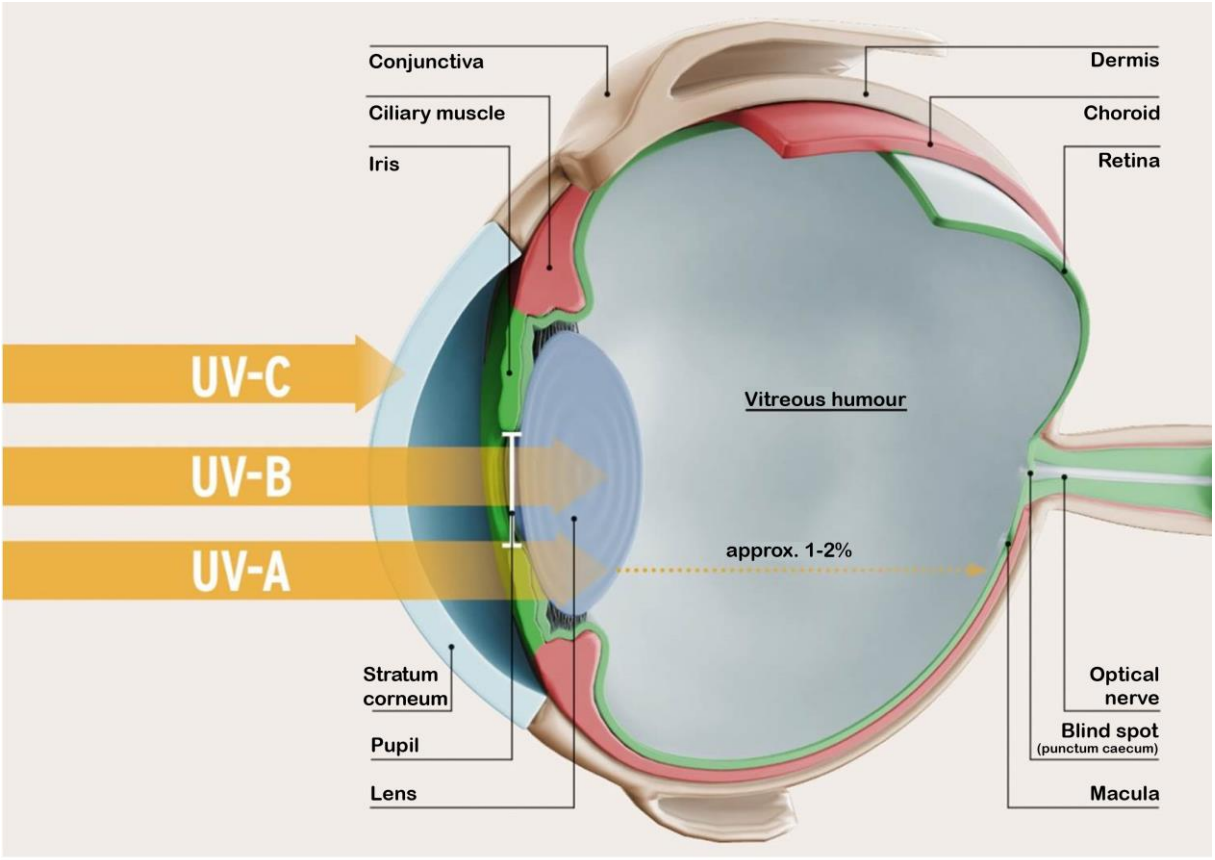

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Fig. 4: Penetration depth of UV radiation into the eye

2.2.3 Action spectra – Description of the radiation effect's dependency on wavelength

UV radiation affects microorganisms, plants, animals and humans due to the fact that its radiation energy is absorbed by certain molecules known as chromophores. These molecules in organisms or tissue (e.g. human skin or the eyes) absorb the radiation energy, generate photoproducts and induce biochemical changes that may lead to cell changes or cell death and hence to a reaction by the affected organ (see Chapter 3.1). DNA is a major chromophore whose spectral absorption drops by around five orders of magnitude (around 1/100,000) to up to 320 nm from the maximum (around 265 nm). Due to the limited penetration depth into human tissue, UV radiation generally only affects two organs: the eyes and the skin (see Chapter 3).

The UV-induced cascade of biochemical reactions starts with wavelength-dependent absorption by the chromophores and ends with a wavelength-dependent organ reaction. An action

spectrum describes the efficiency of optical radiation (e.g. UV radiation) in order to trigger/achieve a fixed response/end point of each monochromatic wavelength interval for the observed photobiological effect. Standardised spectral weighting functions (see Fig. 5) are available to mathematically describe some photobiological end points (see Chapter 2.1.3). They are used to compare effectiveness between UV radiation sources rather than to mathematically determine physiological results in organisms/people.

When using action spectra, it should be noted that they are determined using monochromatic radiation and that the interactions of effects at various wavelengths/ranges are not taken into account.

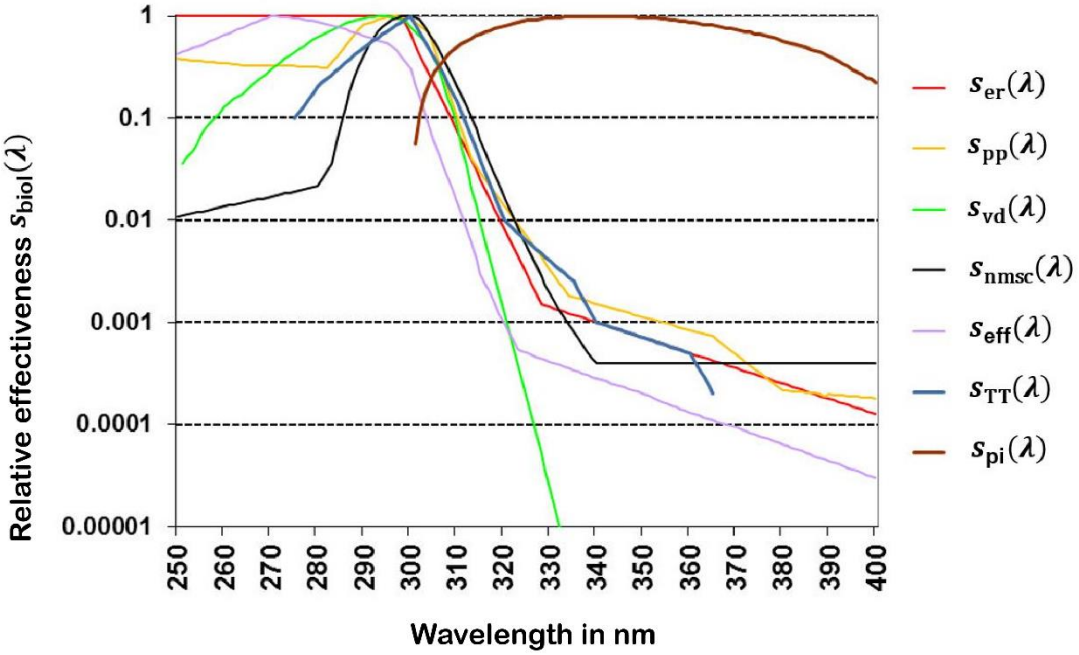


Fig. 5*: Standardised spectral weighting functions $s_{biol}(\lambda)$ for various photobiological processes*

s_{er}*: UV erythema (ISO 1999)*

s_{pp}*: Persistent pigmentation (largely corresponds to the erythema weighting function)*

s_{vd}*: Formation of pre-vitamin D3 from pro-vitamin D3 in the skin (CIE 2006)(data from CIE 2006a, CIE 2000b)*

s_{nmsc}*: Photocarcinogenesis of non-melanoma skin cancer - NMSC) (CIE 2006b)*

s_{eff}**: UV radiation hazard (ACGIH 2008, European Union 2006, ICNIRP 2004)*

s_{TT}: *Formation of thymine dimers (Freeman et al. 1989, Young et al. 1998)*

s_{pi} *Immediate pigment darkening from photooxidation of preexisting melanin*

**) The "artificial" action spectrum for the "harmful effects of UV on man" is used as a form of prevention when it comes to health and safety at work. It was derived as an envelope consisting of spectral threshold curves for triggering acute UV effects in the eyes and skin. This includes photokeratitis (corneal inflammation resulting from UV exposure) and photoconjunctivitis (conjunctivitis resulting from UV exposure) of the eye, and UV erythema of the skin (see Chapter 3).*

2.2.3.1 Illustration of superimposition of the UV radiation spectrum and action spectrum for photobiologically weighted irradiance

In order to assess photobiological effects and processes, the physical quantities have to be taken into account along with the wavelength dependencies of biological effects. The effectiveness of photobiological effects in the UVB range alone can vary by a factor of more than 100 (see Fig. 5). The photobiological effectiveness continually transitions from the UVA range to the UVB and UVC ranges. The values measured for physical UVA or UVB for characterising photobiological processes can only be potentially used for comparison if the action spectrum is identical (and measured using the same measuring device). Photobiologically weighted measurements require a highly precise UV sensor with adequate relative spectral sensitivity that corresponds to the weighting function of the investigated photo effect (broadband radiometer). Alternatively, irradiance is measured wavelength by wavelength using a spectroradiometer. Depending on the wavelength interval, these measured values are then multiplied by the accompanying effectiveness of the spectral weighting function of the observed effect and added to all of the individual interval values (integral convolution of the radiation spectrum and spectral action function: see Chapter 2.1.3 formula (4)).

As illustrated in Fig. 6, minor spectral deviations may occur between the UV radiation sources, as shown here for the summer and winter sun, which in turn lead to major changes in terms of photobiological effectiveness.

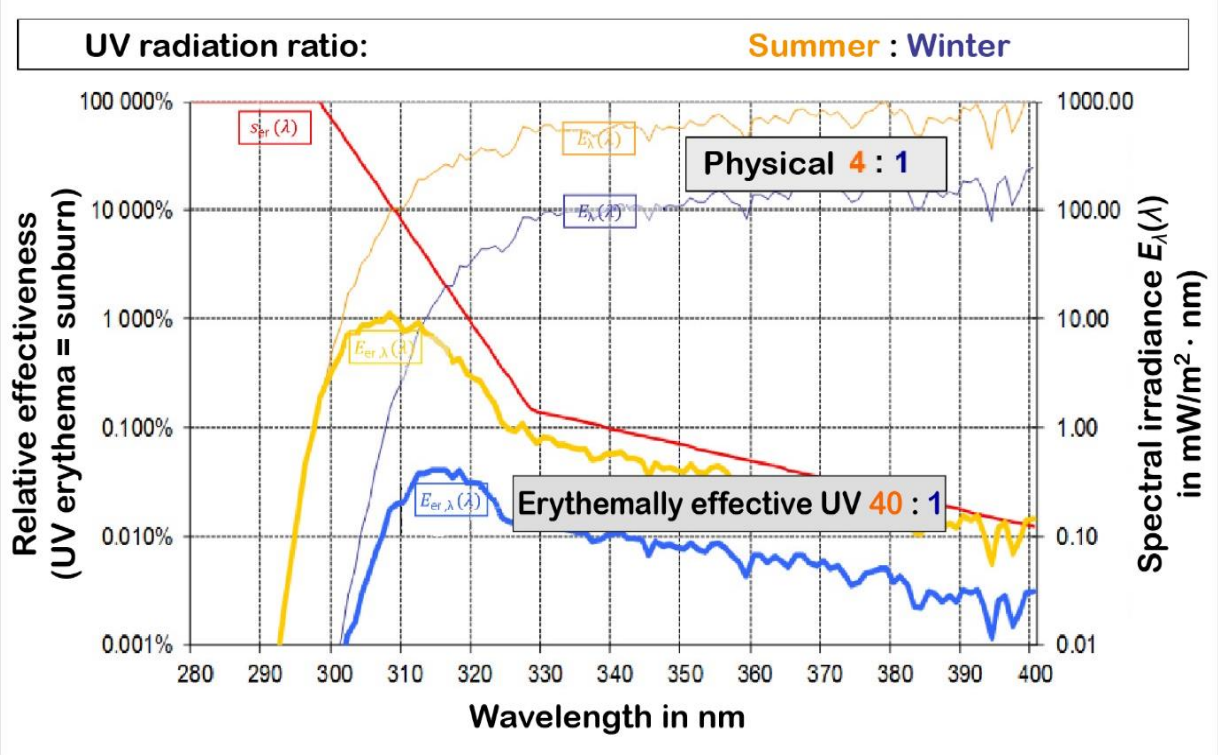


Fig. 6: UV radiation spectrum, spectral weighting function and photobiologically weighted UV effectiveness. The physical unweighted irradiance E for summer sun at noon is four times higher than in winter. Photobiological weighting (see Chapter 2.1.3 formula (4)) with a spectral weighting function for UV erythema $s_{er}(\lambda)$ shows that erythemally effective irradiance E_{er} is 40 times higher in summer (solar elevation angle $\gamma_s = 60°$) than in winter (solar elevation angle $\gamma_s = 15°$); see also the key for Fig. 5

As illustrated in Fig. 7, the photobiologically weighted UV exposure quantities E_{biol} and H_{biol} (see Chapter 2.1.3 formulae (3) and (4) and Annex 1) can be determined for each

photobiological effect (for which a weighting function is known) and for each UV radiation source triggering this effect.

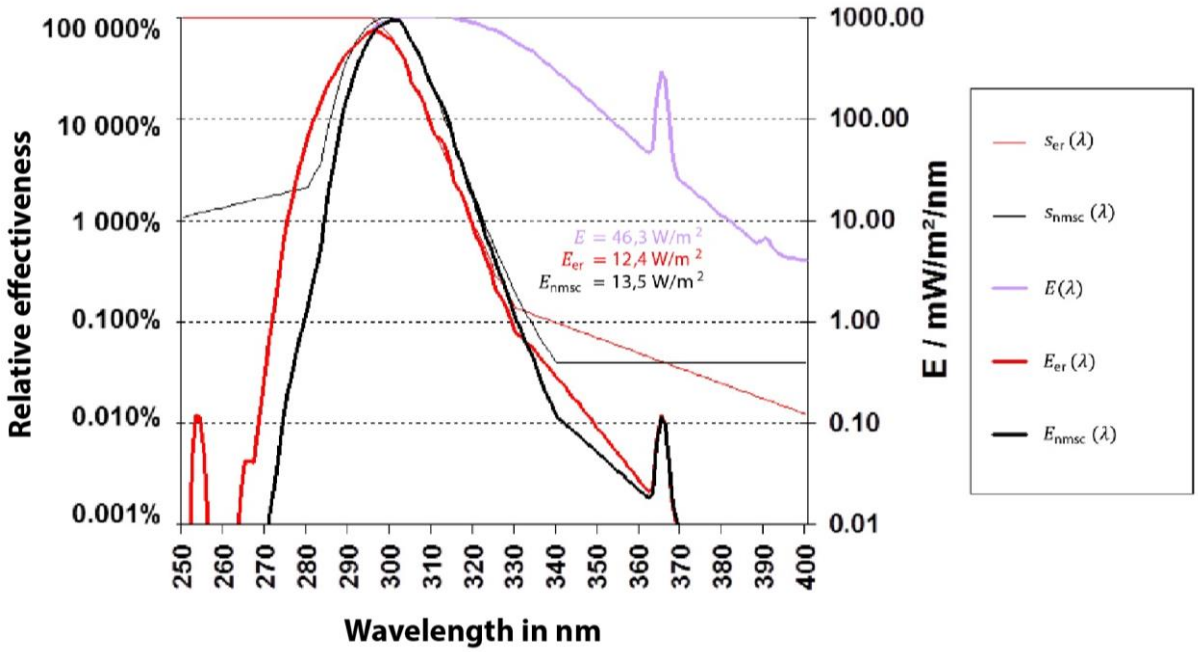


Fig. 7: UV radiation spectrum, spectral weighting function and photobiologically weighted UV effectiveness for a photodermatological broadband UVB emitter:
- *Relations between unweighted UV radiation (E(λ))*
- *Erythema-weighted UV radiation ($E_{er}(\lambda)$) and*
- *NMSC-weighted UV radiation ($E_{nmsc}(\lambda)$) (NMSC: Non-melanoma skin cancer)*

2.2.3.2 *MED*, SED – individual sensitivity and unit used to evaluate the effect of UV erythema

UV erythema (dermatitis solaris or "sunburn") is a traumatic reaction following overexposure of the skin to UV radiation (see Chapter 3.1.6). Reddening of the skin is a visible end point for deriving an action spectrum. Wavelength-dependent effectiveness depends on the extent of reddening (barely perceivable, mild, moderate, severe) selected as an end point. Standardised spectral weighting functions for UV erythema (e.g. ISO 1999) are based on mean values from various studies.

***MED* – Minimal erythema dose:**

Minimal erythema dose (*MED*) is the exposure value *H* (1 *MED*) which leads to the onset of highly localised skin reddening of an individual following a fixed control period (typically 24 hours). The *MED* is used as a unit to express individual momentary UV sensitivity of a single person.

MED depends on the following factors:

- Physiological and pathophysiological influences (see Chapter 3)
- Erythematous reaction over time: commences after 2 to 3 hours, reaches a maximum after 8 to 12 hours (up to 24 hours depending on the level of triggering UV dose)
- Time of reading following application of the UV test exposure: quasi-standard time of reading: 24 hours (when the erythematous reaction abates)
- Anatomic localisation: the *MED* for human skin varies depending on localisation (Table 3)

Table 3*: MED depending on anatomic localisation, determined using monochromatic UV radiation λ = 297 nm. Information is provided for 13 different, presumably light-skinned adults*

Part of the body	***MED* (297 nm) in J/m²**
Back	256 ± 111
Stomach	206 ± 56
Chest	204 ± 84
Forehead	204 ± 53
Cheek	211 ± 42
Neck	218 ± 93
Upper upper arm	380 ± 105
Lower upper arm	325 ± 101
Upper lower arm	424 ± 104
Lower lower arm	481 ± 191
Back of the hand	856 ± 177
Lower leg	691 ± 272

In order to uniformly evaluate and compare the radiation effect of UV sources of various spectral irradiance distributions in terms of their erythemal effectiveness and irrespective of individual sensitivity of human skin to erythema, a standardised spectral erythemal effectiveness curve was agreed upon (see below).

SED – Standard Erythema Dose:

The standard erythema dose (SED) is used as a unit for erythema-weighted exposure H_{er} (see Chapter 2.1.3 and Annex 1):

H_{er} = 100 J/m² = 1 SED.

Erythema-weighted exposure H_{er} is used to compare the erythemal effectiveness of UV radiation sources in relation to the "Erythema Reference Action Spectrum" (ISO/CIE 17166, 1999). Mean *MED* values for groups of individuals (e.g. UV skin types, see Chapter 3.1.4, Table 13) can be expressed in multiples of the SED unit (see Table 4).

The *MED* of an individual person can be provided in SED for a specific emitter spectrum. However, it should be noted that the individual *MED* provided in SED only also applies to a UV source with a significantly deviating spectral radiation distribution if the individual erythema sensitivity spectrum of the person largely matches that of the reference spectrum (see above). Given the existing variations for individual spectral erythema sensitivities, cases involving significant spectral differences between both UV radiation sources may see the *MED* – expressed in SED – deviate from the result of the first radiation source.

Table 4: Standardised constraints for the minimal erythema dose of the back for various skin types (DIN 2010), expressed as H_{er} in J/m² and SED.

UV skin type	Individual UV dose	Minimal erythema dose	
		in J/m²	in SED
I	1 *MED*	200	2.0
II	1 *MED*	250	2.5
III	1 *MED*	350	3.5
IV	1 *MED*	450	4.5

2.3 Solar UV radiation

2.3.1 Solar spectrum

The sun is a thermal radiation source. The level of radiation emission, radiation spectrum distribution and radiation spectrum maximum depend on the body's surface temperature as per Planck's distribution law. Planck's distribution law applies perfectly to ideal blackbodies (bodies that absorb and emit 100% of radiation). With real bodies, radiation emissions are lower than the theoretical maximum. Radiation emitted by hot bodies is also known as blackbody radiation or thermal radiation.

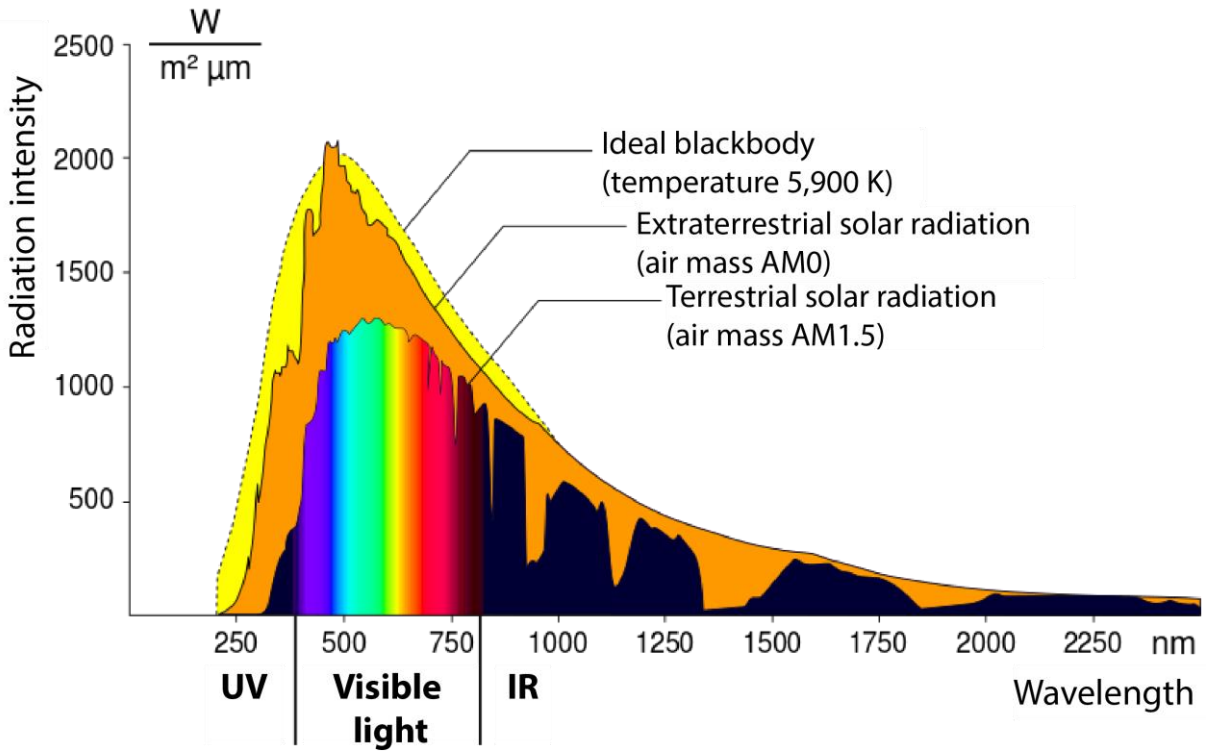


Fig. 8: Solar radiation spectrum (Source: Wikipedia (Wikipedia 2006), last visited on 11 April 2016). (AM0 = Air Mass 0: Solar radiation above the atmosphere, AM1.5: Solar radiation on the Earth's surface with a solar elevation angle of around 42° above the horizon)

Fig. 8 shows the solar radiation spectrum. It is the same as the spectrum of an ideal blackbody with a temperature of around 5,900 K (5,600 °C) (borderline of the yellow area). The borderline of the orange area indicates the solar spectrum outside of the Earth's atmosphere. There, solar radiation also contains visible and infrared wavelengths along with UVC, UVB and UVA wavelengths. The solar spectrum on the Earth's surface (borderline of the inner coloured area)

is much different to that of the extraterrestrial solar spectrum. Solar radiation is partially reflected, scattered and absorbed by constituents of the Earth's atmosphere such as ozone, oxygen, water vapour and carbon dioxide. Shortwave UV wavelengths are severely reduced by ozone, which is why solar radiation on the Earth's surface contains practically no wavelengths below 290 nm, i.e. it does not contain any UVC radiation (Fachverband für Strahlenschutz e.V. 2012). Clouds and aerosols are also highly variable attenuators of solar radiation in the Earth's atmosphere.

The solar spectrum on Earth varies depending on the sun's position above the horizon (solar elevation). Depending on the time of year, the time of day, and the observation location, solar radiation must pass through a layer of air of differing thickness before reaching the Earth's surface. This means that radiation is absorbed at different levels, which applies to total irradiance and selectively for various ranges. Total solar radiation of a horizontal, plane surface is known as global radiation and consists of direct radiation with a line of sight to the sun, radiation scattered in the atmosphere, and radiation scattered or reflected on the ground, and comprises UV radiation as well as visible and infrared radiation.

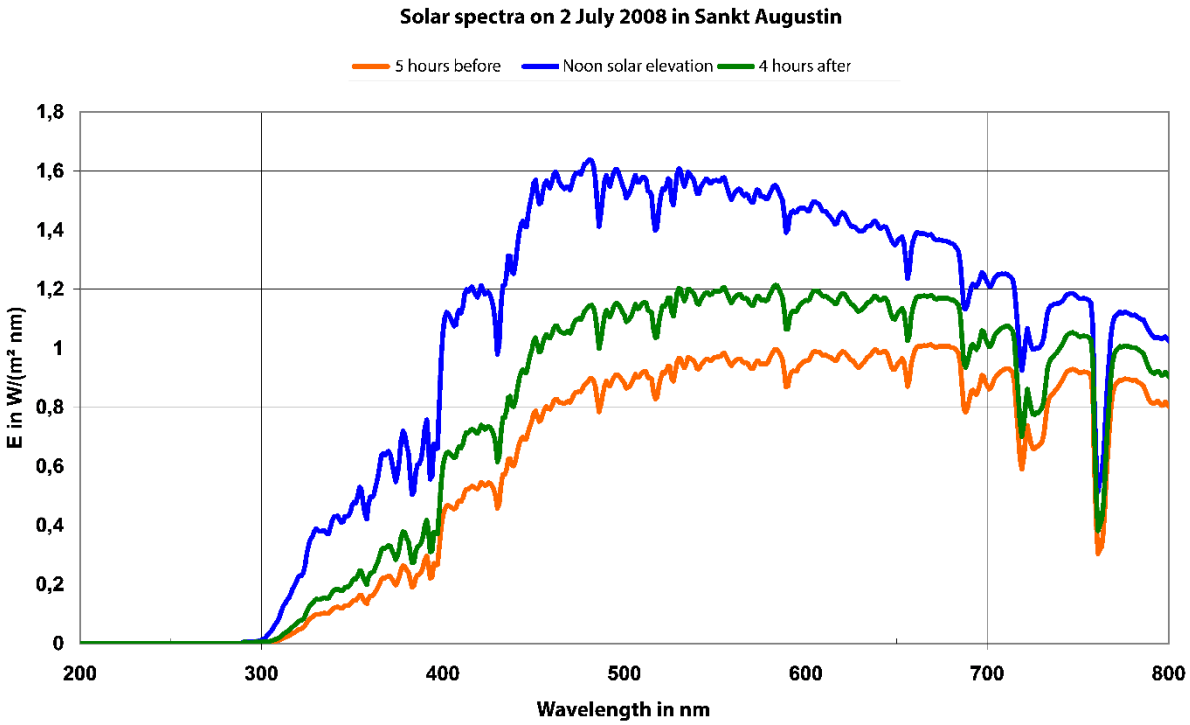


Fig. 9: Solar spectra measured on a cloudless summer day. Place of measurement: Sankt Augustin (N 50.8°; E 7.2°); Times of measurement (solar elevation angle): 08:30 (26.9°), 13:30 (62.2°) and 17:30 CEST (36.5°). Source: Institute for Occupational Safety and Health of the German Social Accident Insurance (IFA 2011)

Fig. 9 shows how the solar spectrum measured on the Earth's surface is dependent upon solar elevation (IFA 2011). The higher the sun above the horizon, the higher the total irradiance. At a higher solar elevation angle, the spectrum maximum shifts towards smaller wavelengths. Within the UV range, the spectrum changes significantly with solar elevation. Fig. 9 shows that there is no radiation at wavelengths below 290 nm. The UVB range (280 nm to 315 nm) and UVA range (315 nm to 400 nm) increase significantly as the solar elevation angle rises. The higher the sun above the horizon, the higher the level of UV radiation and hence the higher the risk when people are exposed to solar radiation without taking precautions. However, solar radiation also contains potentially harmful UV wavelengths, even at low solar elevation. UV

and other spectral wavelengths are only low enough not to cause acute eye and skin damage at either sunrise or sunset. Table 5 shows the level of UV radiation within the total ultraviolet and visible solar radiation range together with the ratio of UVA to UVB radiation depending on solar elevation.

Table 5*: Ratio of UVA to UVB and visible solar radiation with the sun in various positions, calculated using the spectra provided in Fig. 9*

Solar elevation angle [°]	UVB (280-315 nm): E [W/m²]	UVA (316-400 nm): E [W/m²]	UVB/UVA ratio	UV/(UV + visible radiation) ratio
26.9	0.22	14.1	0.015	0.069
36.5	0.37	20.8	0.018	0.049
62.2	1.27	42.7	0.030	0.076

If solar radiation penetrates the atmosphere, part of that radiation is scattered due to air molecules. This is known as Rayleigh scattering, which occurs when the radiation wavelength is higher than the diameter of the particles that are causing the scattering. The higher the level of scattered solar radiation, the lower the wavelength and the thicker the layer of air to be penetrated. Ultraviolet and blue solar radiation are therefore scattered more readily than other solar radiation wavelengths. This occurs most frequently at sunrise and sunset, i.e. with a solar elevation angle close to 0° when solar radiation has to penetrate the thickest layer of air. At higher solar elevation, the sun appears white due to the overlapping of many different wavelengths within the solar spectrum, whereas at sunrise and sunset, the sun appears red because the blue wavelengths within the visible light spectrum have largely disappeared due to scattering, meaning that the visible light spectrum is dominated by red wavelengths.

The scattered wavelengths of direct solar radiation can again be scattered by air molecules in the atmosphere, making them appear blue to observers on the Earth's surface. This is why the sky appears blue in every direction on a clear day. Rayleigh scattering increases as wavelengths become shorter, meaning that blue sky also contains UV wavelengths (UVA and UVB radiation), which are invisible to humans. If humans were able to see UV radiation, the sky would be dazzling.

In order to ensure protection against solar UV radiation, it is important to know that the global radiation to which a person is exposed outdoors consists of several components: direct solar radiation in the line of sight of the sun, radiation scattered by the sky, and radiation reflected by the ground. The ratio of reflected radiation to direct solar radiation is known as albedo (whiteness). UV radiation from the sky may constitute up to 50% of global radiation, meaning that it is not simply enough to use, e.g. a parasol, as a protective measure to shield against direct solar radiation. Radiation from the sky also requires shielding.

2.3.2 Solar elevation

Alongside several other factors, the level of solar UV radiation exposure on Earth largely depends upon the position of the sun in the sky. The higher the sun in the sky, the stronger the UV irradiance on Earth (if influencing factors such as the weather are excluded, although these may dominate). It is therefore interesting to know the solar elevation at a specific time and place. This in turn depends on the Earth's rotation and orbit around the sun. These factors are known and can therefore be calculated.

The position above the horizon as perceived by someone on Earth is stated as the elevation angle between the direction of the horizon and the direction in which the sun can be seen. At summer solstice (20/21 June) at the 50th degree of latitude (geographical latitude in Mainz, Germany), the sun rises to a maximum of 63.4° above the horizon. The Earth’s rotational axis

has an ecliptic of 23.4° in relation to the axis of the Earth's orbit around the sun, meaning that maximum daily solar elevation varies during the course of a year. The lowest noon solar elevation angle is 16.6°, which occurs during winter solstice (21/22 December). This is the reason why the solar UV radiation risk is higher in summer months than in winter months (see Chapter 2.2.3.1, Fig. 6). However, in winter, other factors such as snow-covered ground at high altitudes during the pursuit of winter sports may increase solar UV radiation risk. The right-hand chart in Fig. 11 shows outdoor UV radiation exposure fluctuations based on the time of year. It shows the monthly mean values for the daily maximum values of the UV Index, a measure for expressing erythemally effective irradiance of solar UV radiation measured across the UV measuring network (see Chapter 4). The chart shows that, weather permitting, between March and October outdoor UV exposure can be so high that protective measures are recommended. When doing winter sports at high altitudes, areas of the skin such as the face may need protection from strong solar radiation even between November and February.

Specification of time is linked to the sun's movement, but the local noon solar elevation can only be derived by corrections. This should be described in more detail due to its significance in terms of radiation exposure. In order to determine positions on Earth, the Earth's surface is divided up into 360 lines of longitude and 180 lines of latitude (starting at zero at the equator and counting up to 90 on either side of the equator, i.e. north and south). As the Earth performs one full axial rotation within a period of 24 hours, the sun passes 360 lines of longitude each day. The sun appears to move 15 lines of longitude from east to west every hour (it is in fact the Earth that rotates, but to people on the Earth's surface it appears as though the sun arcs across the horizon). The basis for measuring time on Earth is the prime meridian ($\lambda = 0°$ W/E) which passes through Greenwich in London (see Fig. 10). When noon solar elevation is reached in Greenwich, the UTC (Universal Time Coordinated, also formerly known as Greenwich Mean Time – GMT) is exactly noon (12:00). There are seasonal deviations, however. Germany and other European countries use Central European Time (CET), which refers to the 15^{th} meridian east. When the sun is at its highest in that location, it is by definition 12:00 CET. For reference: CET = UTC + 1 hour. During the summer months, the clocks are changed to Central European Summer Time (CEST): For reference: CEST = UTC + 2 hours. The 15th meridian east is located near Görlitz on the eastern border of Germany. Most places in Germany are west of the 15th meridian east, meaning that the time in Germany does not match the solar time determined by local noon solar elevation.

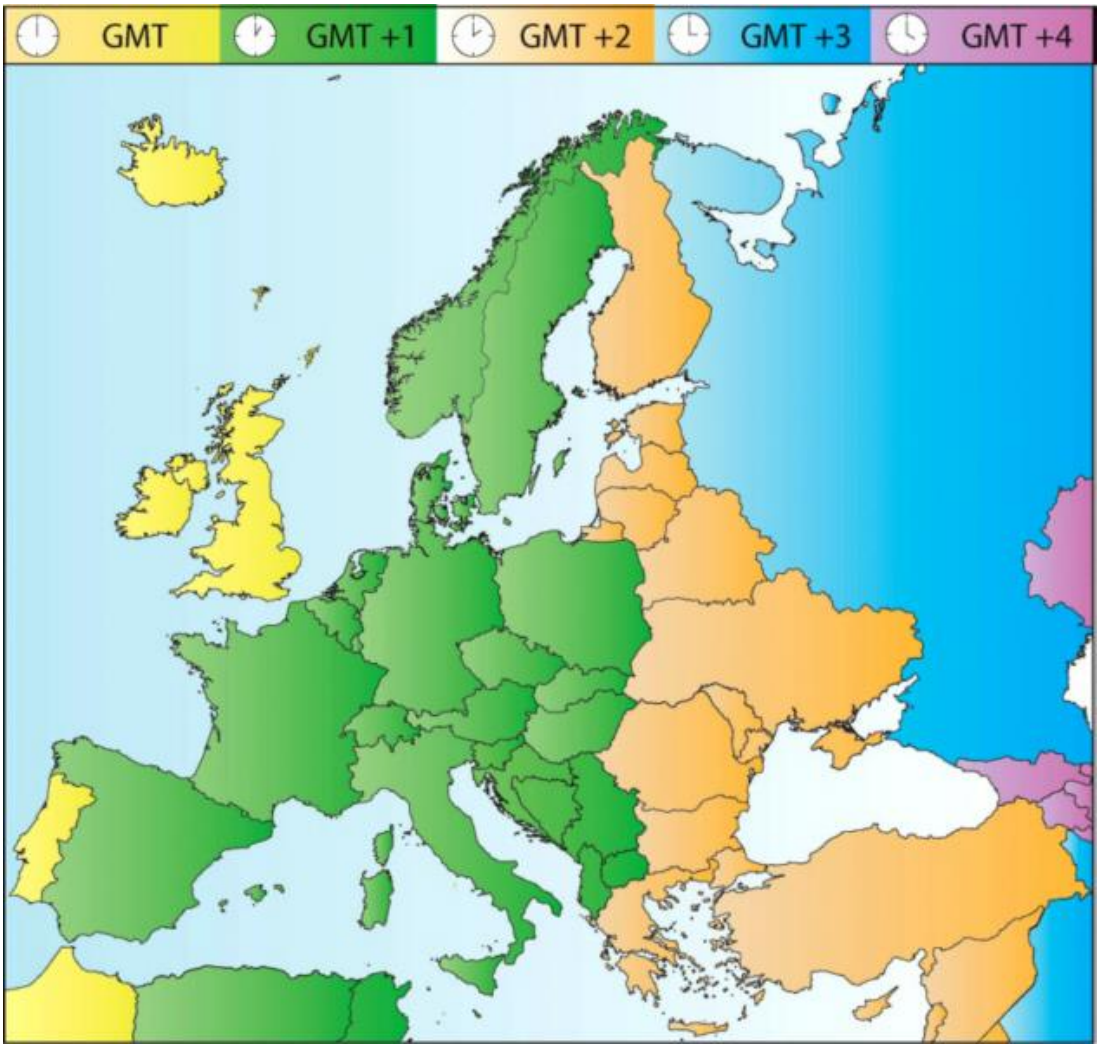


Fig. 10: *Time zones in Europe. The time zone coloured yellow represents Universal Time Coordinated (UTC) = GMT. The countries coloured green use Central European Time (CET - GMT + 1 hour) (from EC 2014)*

It is of interest to know when noon solar elevation (culmination) is reached during the day. The time of culmination depends on the location on Earth, on the time zone and on the season. There are 8.9 lines of longitude between Germany's eastern border near Görlitz and its western border near Aachen. This means that the sun needs 36 minutes on its apparent trajectory, which in turn means that due to the Earth's rotation, noon solar elevation varies in Germany between noon (12:00) and 12:36 CET during winter months and between 13:00 and 13:36 CEST during summer months. In addition to this, the Earth's elliptical orbit around the sun and the Earth's tilted axis (ecliptic) may cause the noon solar elevation for a given location to fluctuate within a range of -14 minutes to +16 minutes over the course of a year when compared to the average culmination time. The largest fluctuations can be seen during winter months. During summer (in 2014 this was from 30 March to 26 October), the fluctuations ranged from -7 minutes to +15 minutes. Depending on the date and location within Germany, noon solar elevation may occur between 11:46 and 12:52 CET in winter and between 12:53 and 13:51 CEST during summer. The exact culmination time and accompanying solar elevation angle can be calculated for any location and for each day using nautical tables (Köhne und Wößner 2005) and the Sun & Earth Applet (Giesen 2000).

In some countries using Central European Time, noon solar elevation fluctuations occur to an even greater extent than in Germany (see Fig. 10). Countries that experience extreme

fluctuations are Norway, Poland, Hungary, Serbia and Macedonia in the east and Spain in the west. Table 6 shows the time ranges within which maximum solar elevation may vary during the course of a year.

Table 6*: Varying maximum solar elevation times over the course of a year in various countries using Central European Time*

Country	Daily maximum solar elevation range	
	in winter	in summer
Germany	11:46 - 12:52 CET	12:53 - 13:51 CEST
Norway	10:42 - 12:58 CET	11:49 - 13:57 CEST
Poland	11:10 - 12:20 CET	12:17 - 13:19 CEST
Spain	12:33 - 13:53 CET	13:40 - 14:52 CEST

The time at which the sun is at its highest is of importance in terms of protection against UV radiation. One of the recommendations is to avoid spending time outdoors during the period around noon if there are no or only a few clouds in the sky so as to avoid overexposure to solar UV radiation. As can be seen in Fig. 11, solar UV radiation varies significantly depending on the time of day and season. The left-hand chart shows the UV Index (see Chapter 4) over the course of a day (measured by the German Weather Service in Lindenberg on 23 June 2005). It shows that solar UV irradiance increases rapidly following sunrise and peaks at the place of measurement at 13:00 CEST. After that time, solar UV irradiance decreases rapidly until sunset. If a person spends time outdoors, the level of potential solar UV dose to the skin is the product of the level of UV irradiance and exposure time (see Chapter 2.1.3). The area below the curve in the left-hand chart in Fig. 11 is a quantity for the UV dose. As can be seen in the curve in the left-hand chart in Fig. 11, around half of the area below the curve falls between -2 hours (around 11 a.m.) and +2 hours (around 3 p.m.) of the maximum. Around a quarter of the area below the curve falls between sunrise (around 5 a.m.) and 2 hours before the maximum (around 11 a.m.), while another quarter falls between 2 hours after the maximum (around 3 p.m.) and sunset (around 9 p.m.). In order to reduce the potential UV dose, no time should be spent outdoors starting 2 hours before and up to 2 hours after the maximum daily extreme UV exposure period. Based on this, the WHO recommends wearing suitable clothing, sunglasses and sunscreen to protect against the sun, and to seek shade when the UV Index ranges from 3 to 7. If the UV Index reaches 8 or more, the WHO recommends avoiding spending time outdoors. However, high UV irradiance values can still be assumed both before and after such peaks. The example in the left-hand chart in Fig. 11 shows a maximum UV Index of just over 8. However, two hours prior to the maximum, the UV Index has already reached 6 to 7, and two hours after the maximum, the UV index still provides a reading of 6 to 7. These are also values that require corresponding UV protection (see the UV Index and protective measures in Chapter 4). The WHO recommends using protective measures with a UV Index of 3 or more (WHO 2002). On the day used in the example in the left-hand chart of Fig. 11, protective UV measures are recommended between 9 a.m. and 5 p.m.

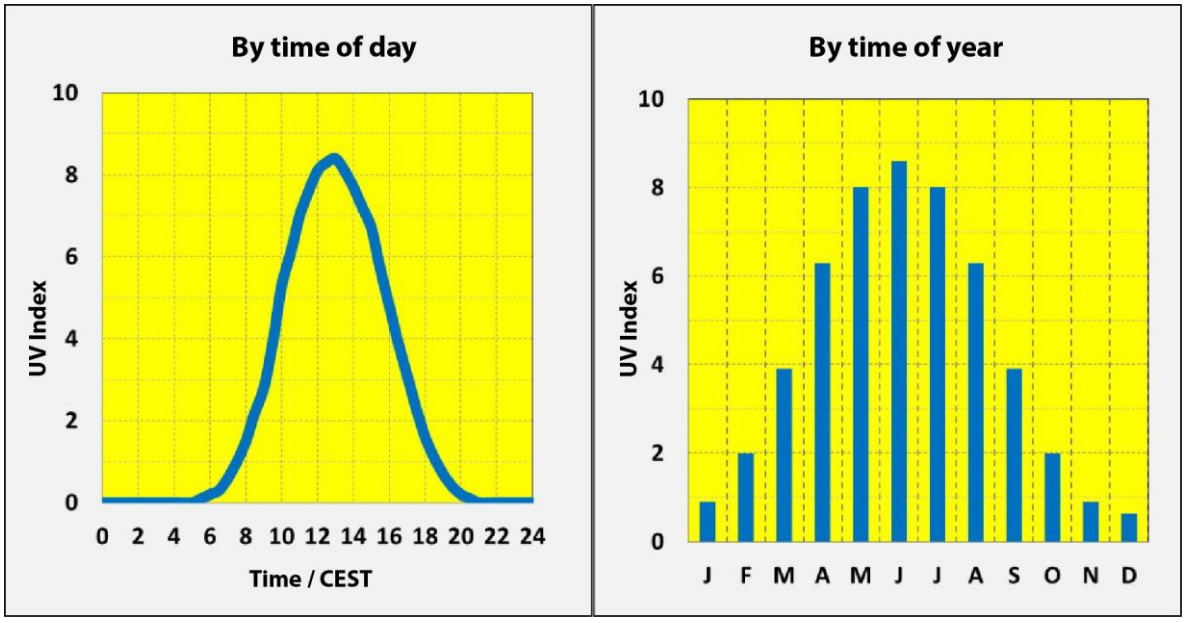


Fig. 11: *Left: Typical UV Index curve over the course of a day measured by the German Weather Service in Lindenberg on 23 June 2005. Curve smoothed, time of day converted to CEST. Right: Typical UV Index curve over the course of a year. Calculated maximum UV Index values on the 21st day of every month in 2014. Radiation transfer model used: Quick TUV Calculator (NCAR 2016), Parameters: Location: N 50° E 10° (Near Arnstein am Main), Sky: clear, Ozone: 300 DU, Altitude: 0.0 km, Albedo: 0.1*

Such recommendations should be adjusted to avoid overexposure when on holiday abroad. Table 7 provides examples of time periods during which maximum UV exposures are to be expected based on geographical location, the east-west longitudes of various countries, and the variation of daily noon solar elevation (Table 6). High solar UV irradiance (UV Index ≥ 3) may also occur before and after, thus rendering the need for corresponding protective measures when spending time outdoors.

Table 7: *Time periods of potentially higher solar exposure in different countries using Central European Time; the data take account of geographical location, east-west longitudes and variation of time of noon solar elevation over the course of a year.*

Country	High solar exposures over time
Poland, Slovakia, Hungary, Serbia, Macedonia	10:30 - 15:30 CEST
Germany	11:00 - 16:00 CEST
France	11:30 - 16:30 CEST
Spain (mainland)	11:30 - 17:00 CEST

2.3.3 Solar UV radiation and personal dose equivalent

2.3.3.1 Methods used to determine photobiologically effective UV exposure

Reliable data about solar UV radiation exposure need to be collected and made available in order to clarify scientific issues and inform the general public about the risks of solar UV radiation.

An overview and stipulations of methods used to measure or estimate and assess solar UV exposures are provided in DIN EN 14255-3 (DIN 2008). This standard includes simple methods for roughly estimating potential UV exposure, links to model data calculations for solar

immission, and measuring options for measuring solar irradiance on a plane surface and for performing measurements on people, and are described below.

Solar UV radiation immission can be measured using fixed spectroradiometers (with subsequent weighting using the necessary spectral weighting functions) (see Fig. 12(a)) or broadband radiometers (with UV sensors whose spectral response has been adjusted to the spectral weighting function) (see Fig. 12(b)). The UV Index can also be determined locally using such measuring techniques.

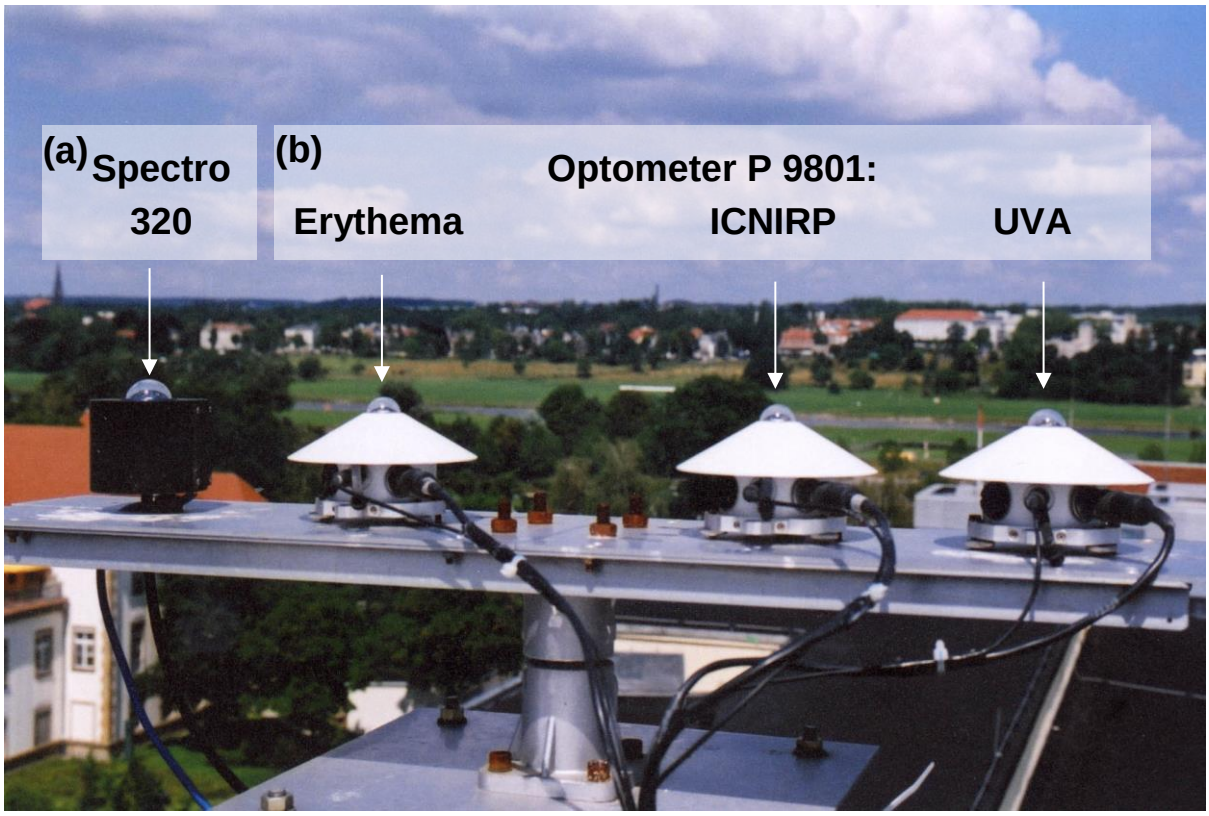


Fig. 12*: Measuring solar UV radiation using (a) spectroradiometers and (b) photobiologically weighted broadband radiometers*

UV immissions in Germany are determined and recorded by continually measuring terrestrial UV radiation in various locations (see Fig. 13) using a UV measuring network operated by the Federal Office for Radiation Protection (BfS), the Federal Environmental Agency (UBA) and other institutions.

Fig. 13: *Locations for measuring solar terrestrial UV radiation in Germany* ***(http://www.bfs.de/EN/topics/opt/uv/index/index_node.html)****, measurement performed on 4 June 2014*

More detailed measurements of individual UV exposures can be performed using mobile electronic data-logging dosimeters (see Fig. 14 A/B) or cumulatively measuring film dosimeters (see Fig. 14 C/D).

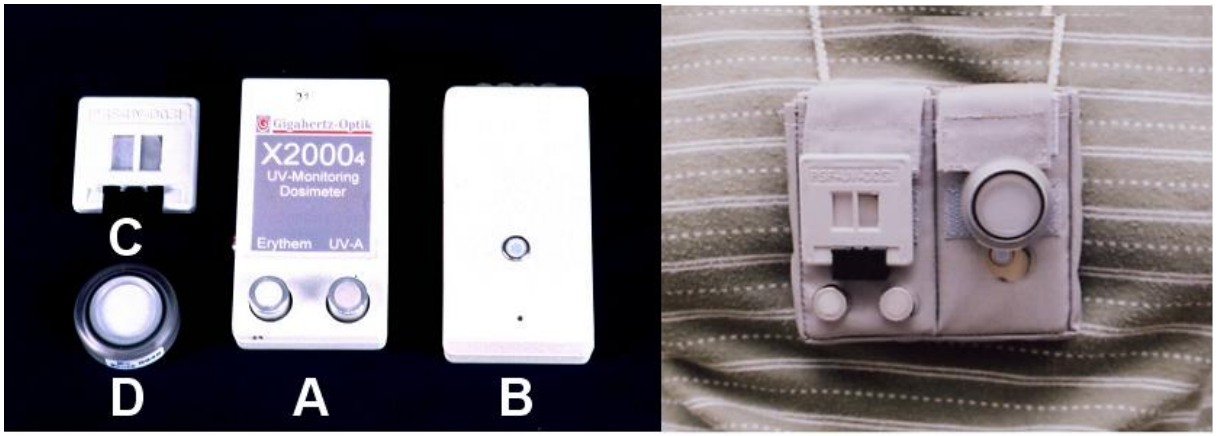


Fig. 14: *Personal UV dosimeters (left) and "4in1 dosimeter" (right) are used to perform intercalibration when collecting biologically weighted solar UV exposure. (A) X-2000 data-logging dosimeter, (B) UVDAN data-logging dosimeter, (C) Polysulphone film dosimeter, (D) VioSpor dosimeter (Basis: Biofilm)*

When affixed to certain places on the body, these light and small film dosimeters enable solar UV exposure to be measured all over the body and averaged for hours, days or weeks while taking account of the exposed person's movement. When comparing personal (erythema-weighted) UV exposures of parts of the body typically exposed to UV, such as the face, arms,

hands or chest, with the results of stationary measurements, it can be seen that up to 30% of local erythemally effective solar radiation may reach various parts of the body's surface (Knuschke et al. 2004, Knuschke et al. 2007).

When assessing the risk of solar UV radiation, the acute reaction of the skin to UV radiation – UV erythema – is used by drawing upon the reference erythemal action spectrum (ISO 1999) (see Fig. 5).

Precise determination of personal solar UV exposure is not always needed to protect against solar UV exposure. General information about the risk of spending time outdoors is often sufficient. The World Health Organization (WHO), the World Meteorological Organization (WMO), the United Nations Environment Programme (UNEP) and the International Commission on Non-Ionizing Radiation Protection (ICNIRP) introduced the UV Index as a standard measure to inform the general public about solar UV irradiance (see CIE S 013/E:2003 (CIE 2003)). The solar UV Index (UVI) is a measure of the erythemally effective UV irradiance on the Earth's surface and is measured or predicted on the basis of prevailing or anticipated weather conditions on a horizontal surface. As the UV Index is generally determined for larger regions, local solar UV exposure may differ depending on cloud cover and other reasons. Chapter 4 provides more information about the UV Index, its properties and its limits, and how it should be used as a guide for estimating the risk of solar UV radiation.

A similar approach involves the determination of skin and ocular exposure factors (Diffey 1984, ICNIRP 2007) as provided in Annex 2. It enables a rough estimate of local solar UV exposure by taking local factors (cloud cover, albedo) and individual factors (clothing and protective measures) into consideration. As this is not based on measurements, it may give rise to greater uncertainty than an estimate based on the UV Index.

The "shadow rule" is seen as an option for roughly estimating whether there is a risk of local solar exposure without using any measuring equipment and then determining whether protective measures should be implemented. "If the shadow is shorter than the height of the object in question, then the sun has an elevation angle of > 45° and thus protective measures should be implemented." It should be noted that this guidance may well be practical, but it does not correspond with the UV Index-based protection concept since a UV Index of 5 can be reached with a solar elevation angle of 45°. In extreme cases, a UV Index of 3, above which level protective measures are recommended, can even be reached at a solar elevation angle of 32° or more if the sky is clear (Knuschke et al. 2015). This is why, whenever possible, it makes sense to take account of UV Index values and forecasts for locations all over the world, such as those provided on http://www.wetter-online.de. On top of that, if the sky is clear, adjustment factors (Annex 3) can be applied to the current local UV Index using the length of shadows.

When planning precautions to be taken to protect against solar UV radiation while travelling, it may make sense to calculate exposure to global solar radiation depending on the time of year, the time of day, geographical location, etc. There are programs available to perform such calculations:
(http://cprm.acom.ucar.edu/Models/TUV/Interactive_TUV/, http://nadir.nilu.no/~olaeng/fastrt/fastrt.html).

In some cases, solar UV immission and personal solar UV exposure must be determined more accurately, and can be achieved by measuring the erythemally effective exposure, the irradiation covered by ICNIRP due to its detrimental health effects, and/or the irradiation covered by NMSC as set out in DIN EN 14255-3 (DIN 2008) together with the corresponding spectral weighting functions (see Fig. 5). This data can be used to determine individual risks.

2.3.3.2 Extent of solar UV exposure of the general public while on holiday, at leisure or at work

The extent of personal solar UV exposure is determined by global factors influencing solar UV immission as well as individual behaviour. The main global factors are the time of year, time of day, geographical latitude, prevailing weather conditions, and altitude at which the person spends time (see Chapters 2.3.1 and 2.3.2). As a global factor, the meteorological situation influences solar UV irradiance on the ground, with cloud cover and turbidity having an effect along with conditions such as temperature and rainfall influencing whether a person spends time outdoors. Investigations into various characterised groups of people in Germany show that the amount of time spent outdoors and accumulated personal UV doses, averaged for the given group, correlate with daily maximum temperatures (Knuschke et al. 2004).

The part of the body and its orientation to the sun are both key factors when it comes to irradiance on the skin, and lead to a body distribution of solar exposure. Various methods such as model calculations for maximum UV exposures with fixed orientation of body parts to the sun, measurements on static or rotating mannequins fitted with personal dosimeters, short-term measurements involving people outdoors (2-3 hours) or cumulative measurements performed over several days under everyday circumstances can all be used for investigations into body distribution. When using these investigations as a guide, the chest, face and wrist accumulate roughly 30% of the exposure received by the crown of the head. Momentary irradiance that constantly changes due to movement is higher for “sun terraces” (e.g. 60-70% for the shoulders). Specific areas of the skin in a fixed position (e.g. when sunbathing) that receive vertical solar radiation are subject to higher irradiance than the irradiance that falls upon a plane surface. Tables relating to body distribution and to dependencies of solar elevation during the course of the year are available in a number of publications (Diffey et al. 1977, Herlihy et al. 1994, Hoeppe et al. 2004, Knuschke et al. 2004, Knuschke et al. 2015, Knuschke et al. 2007). This solar UV exposure body distribution must be taken into account if UV measurement data used to determine an individual’s UV exposure reflect the UV exposure on a plane surface, as is the case, e.g. when determining the UV Index (see Chapter 4).

There are few national (or international) investigations available to date that provide substantiated measured data for individual UV exposure among various groups of the population. Estimates of personal exposures or personal dosimetric measurements, such as those carried out by various working groups in the last 30 years, were used to estimate cumulative solar UV exposures over shorter or longer periods of time with the aim of arriving at conclusions for average annual UV exposures and possibly cumulative lifetime UV doses. The backdrop for this are epidemiological results that prove there is a link between, e.g. the risk of squamous cell carcinoma and the cumulative lifetime UV dose. The personal dosimetric measurements methodically interpreted to evaluate cumulative exposure data level out the brief yet very high UV exposures. This must be taken into account when evaluating the estimates and measuring data provided below. Within the scope of a review, Godar (Godar 2005) compiles personal UV doses for people who work indoors and outdoors, children and adolescents by expressing them as a percentage in relation to erythemally effective solar UV immission (based on a plane surface) (see Table 8). The review included studies with personal UV dose estimates gleaned from surveys (Airey et al. 1997, Boldeman et al. 2004, Diffey et al. 1996, Godar 2001, Godar et al. 2001) and studies where personal UV dosimeters were used (Challoner et al. 1976, Gies et al. 1998, Kimlin et al. 1998, Leach et al. 1978, Munakata et al. 1998, Parisi et al. 2000, Schothorst et al. 1985, Slaper 1987, Thieden et al. 2004). The data show a significant influence on the extent of individual solar UV exposure by including periods of time spent on holiday.

Table 8: *Percentage of personal UV doses (without or – if stated – with exposures while on holiday as well as the period under observation) of adults, children and adolescents in terms of solar immission on a horizontal surface (modified according to Godar 2005*

	Time period	Personal UV dose as a percentage of solar UV immission				Study
		Indoor workers	Outdoor workers	Children	Adolescents	
Total global estimate excluding holiday		3% (2-4%)	10% (6.6-17.7%)	3% (2-4%)		
Sweden 58-60°N	Summer	6%	10%			(Munakata et al. 1998)*
	Summer			6.4% (0-5 years)		(Boldeman et al. 2004)
Denmark 55,41°N	Full year incl. holiday	3.1% 4.2%	6.6% (gardener)	4.1% (4-15 years)	4.7% (16-19 years)	(Thieden et al. 2004)
England 50-55°N	Full year	3% (2-4%)				(Challoner et al. 1976), Leach et al. 1978)
	Full year		10%			(Challoner et al. 1976)
	Summer			6.1% girls 6.9% boys (9-10 years)	4% girls 4.2% boys (14-15 years) 4.7 % 16-19 years (incl. holiday)	(Diffey et al. 1996)
Netherlands 52.5°N	Full year	2.5%	7%			(Schothorst et al. 1985, Slaper 1987)
Japan 40° N	Full year			3.1% (9-12 years)		(Kimlin et al. 1998)**
USA 44°N		3.1% (> 21 years)		3.1%	2.6%	(Godar 2001, Godar et al. 2001)
USA 34°N		3.1% (> 21 years)		3.1% (0-12 years)	2.6% (13-19 years)	(Godar 2001, Godar et al. 2001)
Australia 21-28°S	Summer	4.2% (27.5°S)	9.8% (gardener 27.5°S)		4.7% (15-16 years, 27.5°S)	(Kimlin et al. 1998)
	Summer		14% (farmer, 27.5°S)			(Airey et al. 1997)
	Summer			4.1% (7-12 years)	4.5% (13-19 years)	(Parisi et al. 2000)
	3/4 of a year incl. holiday			8% (boys 12 years, 21-28°S); 4.9% (girls 12 years, 21-28°S)		(Gies et al. 1998)

*) Godar's original publication stated – evidently incorrectly – that a Japanese study (Munakata et al. 1998) was the source of the data for Sweden (indoor and outdoor workers). Presumably the source should be (Boldeman et al. 2004).

**) Godar's original publication stated – evidently incorrectly – that an Australian study (Kimlin et al. 1998) was the source of the data for Japan (children). Presumably the source should be (Munakata et al. 1998).

In order to be able to evaluate the extent of solar UV exposure, the relations of personal UV doses to UV immission expressed as percentages are required along with, in particular, absolute values such as mean daily UV exposures in SED/d for the various behaviour groups of the general public.

Tables 9, 10 and 11 show personal dosimetric measurements over the course of a year for indoor workers (n = 18, personal UV dosimeter on the wrist) in Denmark (Thieden et al. 2006) and for indoor workers (n = 120, personal UV dosimeter on the chest) and outdoor workers (n = 120, dosimeter on the chest) in Germany, but also for children in their final year of preschool and then their first year at primary school (n = 60, personal UV dosimeter on the chest) (Knuschke et al. 2004), each subdivided into UV exposures received at work and at leisure. The Danish study used the mean daily UV dose for each person from the individual monthly total (consisting of workdays and days at leisure) to arrive at the median values of the personal UV dose per day and per group of people. The German personal UV monitoring study determined the median values per group using measured cumulative personal UV dosimeter values over a period of 10 workdays and 6 days at leisure/weekend days for each person during the given month. The personal dose measurements provided are extremely complex and can therefore only by carried out in small numbers in relation to the total population (82 million, of which 40 million are workers and 20 million are children). The data from the investigated random test therefore only provide indications for the link between occupation, i.e. group of people, and solar UV exposure level.

Everyday exposures already show clear differences in the extent of personal UV doses received during workdays and those received during days of leisure. Individual behaviour in terms of frequency of time spent outdoors play a major role in personal UV dose level, particularly during days of leisure. Here it should be noted that a distinction must be made within groups of people, such as indoor and outdoor workers, between those who pursue few outdoor leisure activities, and those who regularly pursue outdoor leisure activities such as hiking, cycling, running, etc. (Knuschke 2011).

Table 9: Indoor workers: Median of personal UV doses received in SED*/d over the course of a year during workdays or days at leisure in Denmark (measured on the wrist) (Thieden et al. 2006) and in Germany (measured on the chest) with further subdivision as follows: A: few outdoor activities (80% of monitored indoor workers) and B: regular outdoor activities (20% of monitored indoor workers) (Knuschke et al. 2004)*

Indoor workers - personal UV dose in SED/d						
Month	**Workday**			**Day at leisure**		
	Denmark	Germany		Denmark	Germany	
		A	B		A	B
February/ March*	0.005/ 0.067	0.040	0.055	0.008/ 0.10	0.067	0.23
May	0.19	0.23	0.41	1.06	0.71	2.1
September	0.21	0.073	0.13	0.48	0.25	0.91
December	0.002	0.014	0.018	0.007	0.021	0.049

* Denmark: February/March; Germany: 19 February to 6 March

Table 10: Outdoor workers: Median of personal UV doses received in SED*/d over the course of a year during workdays or days at leisure in Germany (measured on the chest with further subdivision as follows: A: few outdoor activities (70% of monitored outdoor workers) and B: regular outdoor activities (including journey to work and after work) (30% of monitored outdoor workers). Occupations of those involved: Road construction worker, gardener and landscaper, postman, traffic warden (Knuschke et al. 2004).*

Outdoor workers - personal UV dose in SED/d						
Month	**Workday**			**Day at leisure**		
		Germany			Germany	
		A	B		A	B
February/March*		0.15	0.26		0.069	0.15
May		0.72	1.15		0.51	1.6
September		0.25	0.39		0.23	0.88
December		0.040	0.067		0.021	0.041

* 19 February to 6 March

Table 11: Children (A: Preschool last year, B: Primary school 1st grade): Median of personal UV doses in SED*/d over the course of a year during workdays or days at leisure (weekend days) in Germany (measured on the chest)(Knuschke et al. 2004).*

Children – Personal UV dose in SED/d						
Month	**Workday**			**Day at leisure**		
		Germany			Germany	
		A	B		A	B
February/March*		0.094	0.12		1.10	1.05
May		0.51	0.72		0.84	1.1
September		0.24	0.15		0.29	0.31
December		0.037	0.034		0.020	0.037

* 19 February to 6 March

The random sample of personal UV doses measured among 120 indoor workers and 120 outdoor workers during days at leisure shows that there are hardly any differences between the two groups of people in terms of their outdoor leisure behaviour. Both the median personal UV dose values (see Tables 9 and 10) and the statistic distribution of individual doses around the median are comparable among the two groups of people, even during different seasons (Knuschke et al. 2004).

Personal UV doses in groups of people follow a log-normal distribution (Knuschke et al. 2004). A factor of 15 to 20 is present for the exposure level between the 5^{th} and 95^{th} percentile of the distribution of personal UV doses within a group of people with the same characteristics. Median group values can be reproduced relatively well under comparable conditions, but there is a high level of personal influence on the individual UV dose (Knuschke et al. 2004). This must be taken into consideration when comparing the median values for different groups of people.

The median personal UV doses for a group of people with different outdoor activities (see above) are 2.5 to 3 times higher during summer and 3 to 4 times higher during winter than those of indoor workers. Irrespective of the season, groups of people with regular outdoor activities have a personal UV dose that is three times higher on days at leisure (see Tables 9 and 10) than that of groups of people with “normal”, low levels of outdoor activity.

The median personal UV doses recorded during personal dosimetric monitoring of indoor workers can, depending on assignment of outdoor activity, also be transferred to groups of

school pupils (aged 14 or more), students, housewives and nursing home residents (Knuschke und Krins 2000).

Groups of children from preschools who were monitored by personal dosimeters over the period of a year and then moved up to primary school the following year exhibited few differences in personal UV dose distributions for workdays (i.e. while attending either preschool or school) and days at leisure (in the company of their parents) (see Table 11). Here, the medians for the personal UV doses at weekends (days at leisure) are highly comparable with those of indoor workers with few outdoor leisure activities, i.e. with those of parents to whom this kind of behaviour can be largely attributed.

Cumulative personal UV doses over the course of a year enable annual UV doses and, if details of the person's lifetime are known, cumulative lifetime UV doses to be stipulated or estimated, which can then be investigated in relation to mean UV population exposures. Such estimates are also used, for example, when evaluating UV light-induced skin cancer as a potential occupational disease as described in BK 5103 (BKV 1997)[3] However, cumulative personal UV dose data level out individual UV exposure peaks that occur during the measurement periods as a result of various influencing factors (including behaviour and weather). UV exposures around the 95th percentile (even with cumulative measurement) are around 3 to 5 times higher than the median values, which are indicative of exposure peaks in the measurement intervals.

Table 12: Number of cases per monitoring group with personal doses (measured on the wrist) of ≥ 10 SED *on at least one day for the number of data records per year for this group (Thieden et al. 2004)*

People	**Number**	**% of group**
Total	160 / 346	46
Children (5-15a)	38 / 68	56
Adolescents	17 / 22	77
Indoor workers	42 / 111	38
Sunbathers	27 / 49	55
Golfers	12 / 31	39
Gardeners	24 / 65	37

Measurements performed using personal dosimeters with data-logging can exhibit such increased individual UV exposures (see Table 12). UV exposure measurements taken on the wrist (see Table 12) can be roughly compared with facial exposures (Knuschke et al. 2015). In the absence of any measures to protect the skin from light, erythemally effective exposures ≥ 10 SED lead to a mean *MED* being exceeded five-fold for skin type I and twice for the non-adapted skin type IV. This random sample showed that such overexposures on one or more occasions during the year were observed in 40% to 50% of the monitored subjects.

These facts show – especially for children – the importance of preventive behaviour measures, such as providing shaded areas in general and, in particular, at crèches, preschools and schools; organisational measures should also be applied to avoid high UV exposure, e.g. suitable daily schedules, when organising work and schedules, and when organising sports events. Preventive behaviour measures are also required and may include information campaigns on the effects of

[3] With effect from 1 January 2015, multiple "actinic keratoses" – precursors to squamous cell carcinoma – and squamous cell carcinoma itself have been added to the so-called occupational disease list on account of solar UV radiation.

UV radiation as well as individual protective measures or the inclusion of UV Index information in daily schedules.

Personal UV doses are much higher when on holiday than during everyday exposures on workdays and days at leisure. For the majority of the population that does not work outdoors, holiday(s) constitute(s) up to 50% of annual UV exposure (Knuschke 2006). As Fig. 15 shows, the season and holiday destination play a major part in such data. Holidays spent in distant countries in winter, e.g. the Canary Islands, the Caribbean or Malaysia, accumulate an extremely high personal UV dose. However, one week of doing winter sports can lead to higher facial UV exposure than the weekly facial exposure of road construction workers in summer. It should also be noted that personal UV doses measured while spending summer holidays at the North Sea and Baltic Sea do not differ by more than an average of 5 SED per day from summer holidays spent at the Mediterranean Sea. One reason for this could be that comparably lower daytime temperatures at the North Sea and Baltic Sea lead to people often taking advantage of the midday warmth to visit the beach, while people generally tend to avoid going to the beach at noon when on holiday at the Mediterranean Sea because it is too hot. (Note: For a typical person with UV skin type II with non-adapted skin: 1 *MED* ≈ 2.5 SED.)

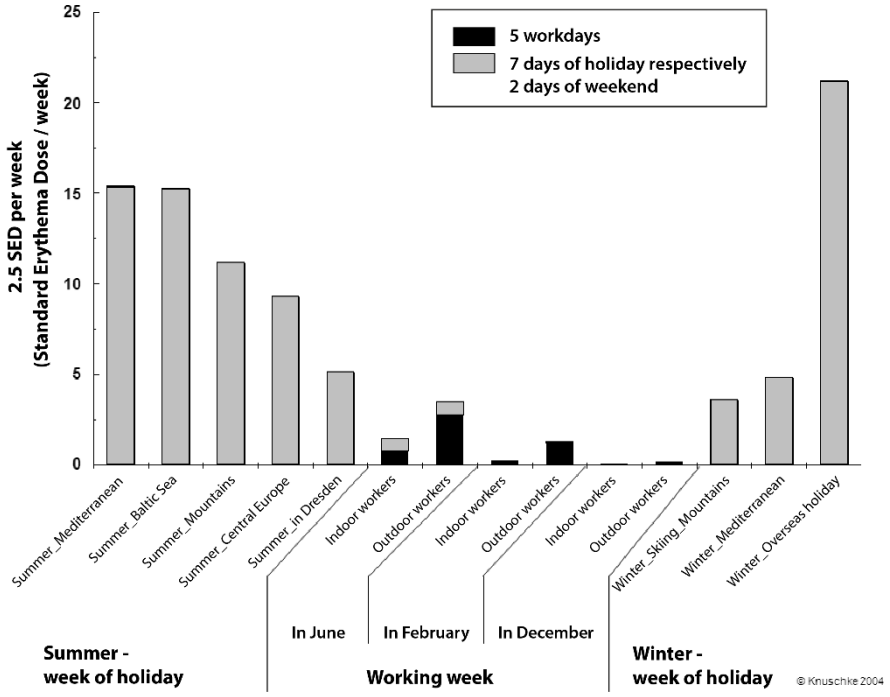


Fig. 15*: Mean personal UV dose over 7 days of holiday in relation to an everyday week (5 workdays with 2 days of weekend/days at leisure) for indoor and outdoor workers in summer and winter (Knuschke 2006)*

Individual UV exposure contributions received over the course of the year while at work, at leisure and on holiday provide the cumulative annual UV dose. Based on personal dosimetric measurements from the wrist over one (n = 340), two (n = 39) and three (n = 14) season(s), Thieden and employees in Denmark estimated an annual UV exposure of 132 SED/a for indoor workers (n = 89), 224 SED/a for gardeners (n = 47), 217 SED/a for golfers (n = 24), 181 SED/a for "sunbathers" (n = 53), 175 SED/a for female adolescents, 116 SED/a for male adolescents and 147 SED/a for children (Thieden et al. 2004). Comparable values were provided by continual personal UV monitoring of various occupational groups in Germany over a period of 52 weeks with a small test sample per group of n = 5. The measurements with dosimeters on the chest resulted in 353 SED/a for construction workers, 235 SED/a for farm workers (using

agricultural machinery), 266 SED/a for dustbin men, 173 SED/a for preschool staff, 148 SED/a for sports teachers, 133 SED/a for window cleaners (chest pressed against the facade) (neck in summer: 450% of chest UV exposure) and 114 SED/a for indoor workers (Knuschke et al. 2007, Unverricht et al. 2006).

Based on a database of personal UV doses from 1,100 person years involving a lot of different groups of people from the general public as well as data from the Federal Statistical Office, a mean annual UV exposure of 130 SED/a was estimated for the population in Germany in reference to exposure of the chest (Knuschke 2011). Previous measurements also provide higher values, e.g. 200 SED/a for children in Northern Europe (Diffey 1999, SSK 2006). It should be noted again that the recording of UV exposure using personal dosimeters is influenced by many factors such as the type of dosimeter used, the recording period, climatic fluctuations and the modelling sometimes used. In addition, individual UV exposure is greatly dependent upon individual behaviour, and UV exposures vary significantly among the general public. This cannot be sufficiently reflected by providing a single annual mean. A frequency distribution of UV exposures among the general public is required in order to provide a full picture.

2.4 Artificial UV radiation sources

UV radiation exposures from artificial radiation sources may occur both during a person's private life and at workplaces. Except for solaria visits and medical applications, such private exposures generally remain at a low level. Due to their relevance to UV exposure of the general public, solaria are covered in more detail in Chapter 5. Artificial radiation sources in private-life scenarios that may emit low levels of UV radiation include light bulbs, halogen light bulbs, fluorescent lights, energy-efficient light bulbs and light-emitting diodes (LED). Further details in this regard are available, e.g. in the SSK statement "Modern light sources" (SSK 2010). UV lamps are used cosmetically to cure gel nails. When compared to solar UV exposure of the hands, this kind of exposure is deemed insignificant (Diffey 2012, Markova und Weinstock 2013).

However, UV radiation exposure may reach high levels at workplaces with artificial UV emitters. Welding work leads to a high level of UV exposure. Depending on the technique and welding parameters used, electrical welding can lead to UV irradiance that is 1,500 times higher than the highest level of solar UV irradiance that occurs on Earth (IFA 2011). In extreme cases, the permitted UV exposure limits may be exceeded after just 0.3 s. Unlike the solar radiation spectrum on the Earth's surface, welding spectra include UVB, UVA and UVC radiation. Therefore, from a radiation protection perspective, welding is an extremely hazardous procedure. Sufficient radiation protection is imperative, and corresponding protection regulations have been in place for many years now. Further details about this can be found in the BGIA Report 3/2007 (UV radiation exposures at workplaces" (IFA 2007).

Other artificial UV emitters in the workplace include the following: gas flames when processing glass (IFA 2010), UVC sources for sterilisation purposes, UV emitters for crack-testing purposes, UV emitters for drying paint and adhesives, and UVA lamps to make markings visible.

As most artificial radiation sources are used at workplaces, risk assessments for such sources are performed in line with health and safety regulations. More information about this is available in the optical radiation handbook (Reidenbach et al. 2012)

3 Effect of UV radiation

As the UVC wavelengths in the solar UV spectrum cannot penetrate the ozone layer, only the UVA and AVB wavelengths of the solar UV spectrum are relevant to biological effects on Earth (see Fig. 16). Only these spectral ranges are used in solaria (which is also due to legislation). Some technical applications (e.g. sterilisation lamps, welding equipment) may also cause UVC emissions. The effect of UVC as a result of such applications must be taken into account (see Chapter 2.4), but is not covered in this recommendation.

The effect of UV radiation on man is conveyed via the skin and the eye. UV-dependent effects can be divided into short-term and long-term effects. The former occurs immediately or just a few minutes, hours or days after UV exposure, while the latter are characteristic of late effects (years or decades after exposure). Short-term effects include pigmentation (tanning) of the skin, reddening of the skin/sunburn (erythema), immunosuppression and vitamin D3 photosynthesis, corneal inflammation and conjunctivitis (photokeratitis/ photoconjunctivitis) and, at times, photochemical retina damage in the eyes, while late UV exposure effects are characterised by ageing of the skin, lens opacities (cataracts) and, above all, skin cancer. With the exception of vitamin D3 photosynthesis and, to some extent, immunosuppression, both acute skin reactions and late effects on the skin are usually caused by UV-induced DNA damage (Eaton 1994, Heenen et al. 2001, Melnikova und Ananthaswamy 2005, Phan et al. 2006, Tadokoro et al. 2005).

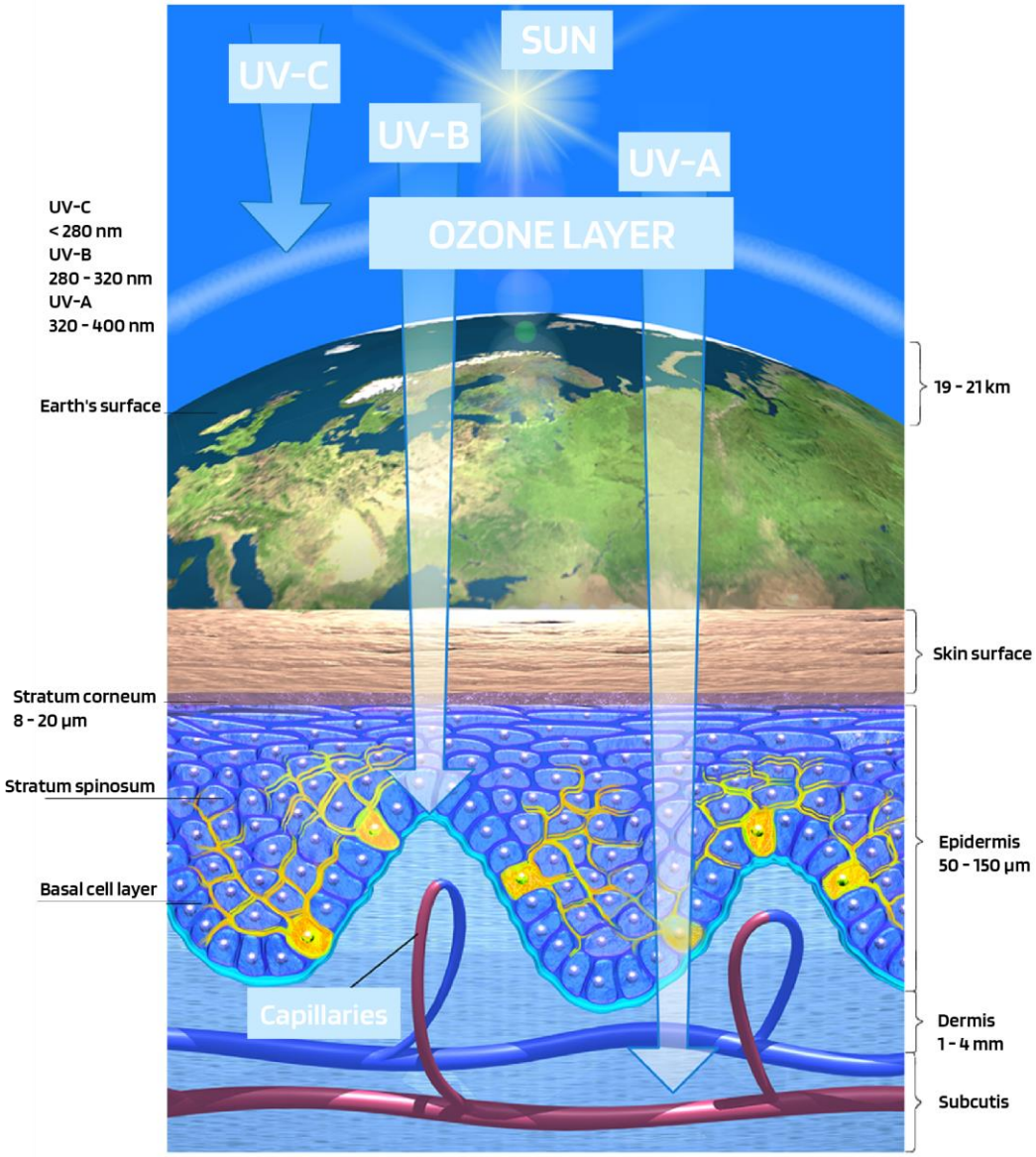


Fig. 16*: UV radiation on the Earth's surface. While UVC is fully absorbed by the ozone layer, UVB (partially) and UVA (almost fully) penetrate the atmosphere. UVA penetrates the skin further (as far as the subcutis) than UVB, which penetrates the epidermis to the basal cell layer. (German Association of Dermatological Prevention – ADP e.V.)*

3.1 Biological effect of UV radiation on the skin

The skin is man's largest organ; its structure (see Fig. 17) acts as a physical barrier against the effects of environmental noxae on the organism, yet it is itself affected as an organ by damaging influences such as UV radiation.

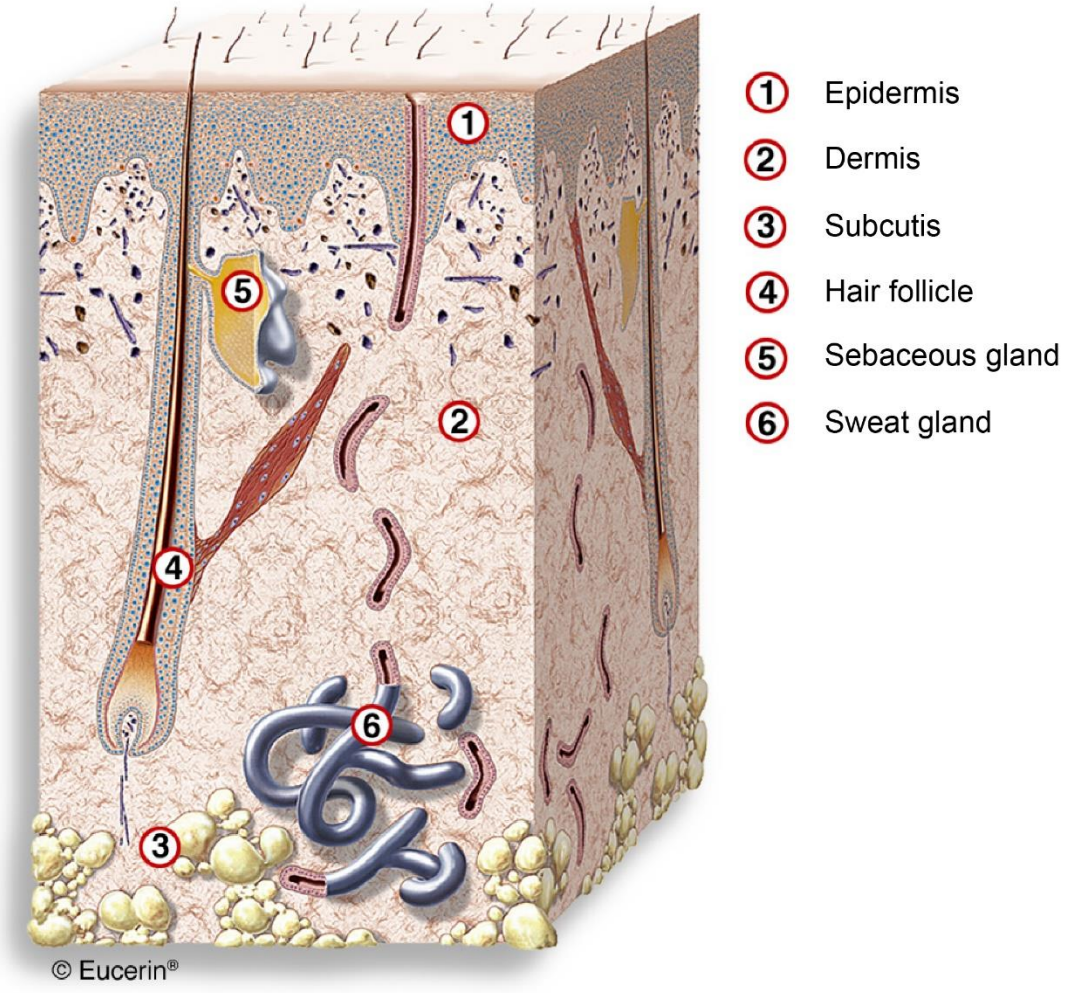


Fig. 17*: Diagram of human skin. The upper layer of skin is known as the epidermis (1), followed by the dermis and subcutis (3). Hair follicles (4) with the sebaceous gland (5) extend deep into the dermis. Here are the sweat glands (6). (Beiersdorf AG)*

Depending on the wavelength, UV radiation penetrates the skin to differing levels (see Chapter 2.2.2). Due to its interaction characteristics with molecular components of the skin (scattering, absorption, reflection), UVB radiation may penetrate the epidermis through to the basal cell layer. UVA radiation penetrates deeper into the skin and can reach the dermis and subcutis (see Figs. 2, 3 and 16). UV radiation may be absorbed by a range of different chromophores whose absorption spectra are within the UV range. These also include cellular components such as proteins and intracellular photosensitive molecules like flavins or porphyrins (Cadet et al. 2012). However, the absorption of UV photons by the DNA molecule is of key importance here.

3.1.1 Children's skin

Children's skin requires separate consideration when it comes to skin cell damage caused by UV radiation and the associated risk of skin cancer. This is particularly the case with epidemiological data where an increased occurrence of skin cancer during adulthood can be observed depending on UV exposure during childhood (see Chapter 3.2.1.3) (Autier und Doré 1998, Green et al. 2011a, Oliveria et al. 2006). Considering acute skin reactions following UV exposure, such as tanning or sunburn, there is no difference between adult and children's skin when it comes to UV sensitivity. Just a few weeks after birth, the ability for pigmentation is

already fully developed (Höger 2008, Loomis et al. 2001) and the minimum UV dose that may trigger sunburn (*MED*) is, on average, the same for adults and children (Cox et al. 1992). However, one difference can be seen in the structure of children's skin (Höger 2008) (see Fig. 18). Looking at the epidermis cones that extend into the dermis, epidermal thickness does not differ from that of adult skin. However, the dermal papillae, which together with the epidermis cones form the dermoepidermal junction, extend further into the epidermis. As a result, part of the basal cell layer in which the assumed target cells for UV-induced development of skin cancer (interfollicular epidermal stem cells and pigment-forming melanocytes) are localised is exposed to a higher level of penetrating UV radiation.

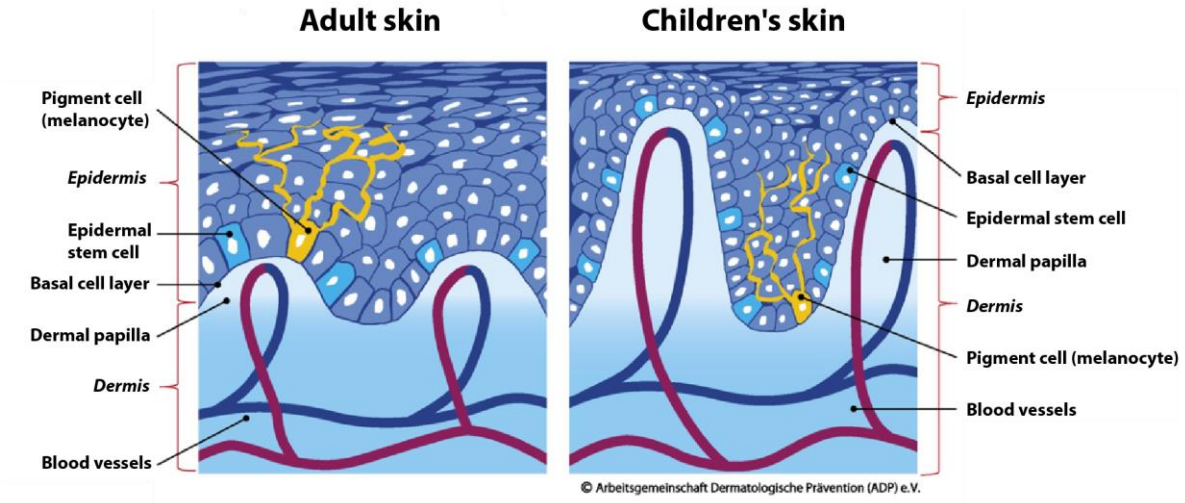


Fig. 18*: Diagram of adult skin and children's skin. In children's skin, the dermal papillae extend deep into the epidermis. Part of the epidermal stem cells in the basal cell layer are thus further below the surface of the skin than is the case with adult skin, hence increasing the level of exposure of UV radiation. (German Association of Dermatological Prevention – ADP e.V.)*

An additional stem cell pool associated with the development of skin cancer is the bulge region of the hair follicle (see Fig. 19). This also describes another difference between adult skin and children's skin. Children's skin has vellus hair which develops into terminal hair during puberty (Randall 2008). With vellus hair, the sensitive bulge region, which is also home to the precursors of pigment cells, is located further away from the surface of the skin than terminal hair, meaning that it is exposed to higher levels of damaging UV radiation (Garcia et al. 2011). The described phenomena indicate that epidermal stem cells may be exposed to higher levels of UV exposure due to their localisation in children's skin, thus increasing the risk of developing skin cancer during adulthood (Volkmer und Greinert 2011). Thorough protection from the sun during childhood should therefore be ensured in order to minimise this risk.

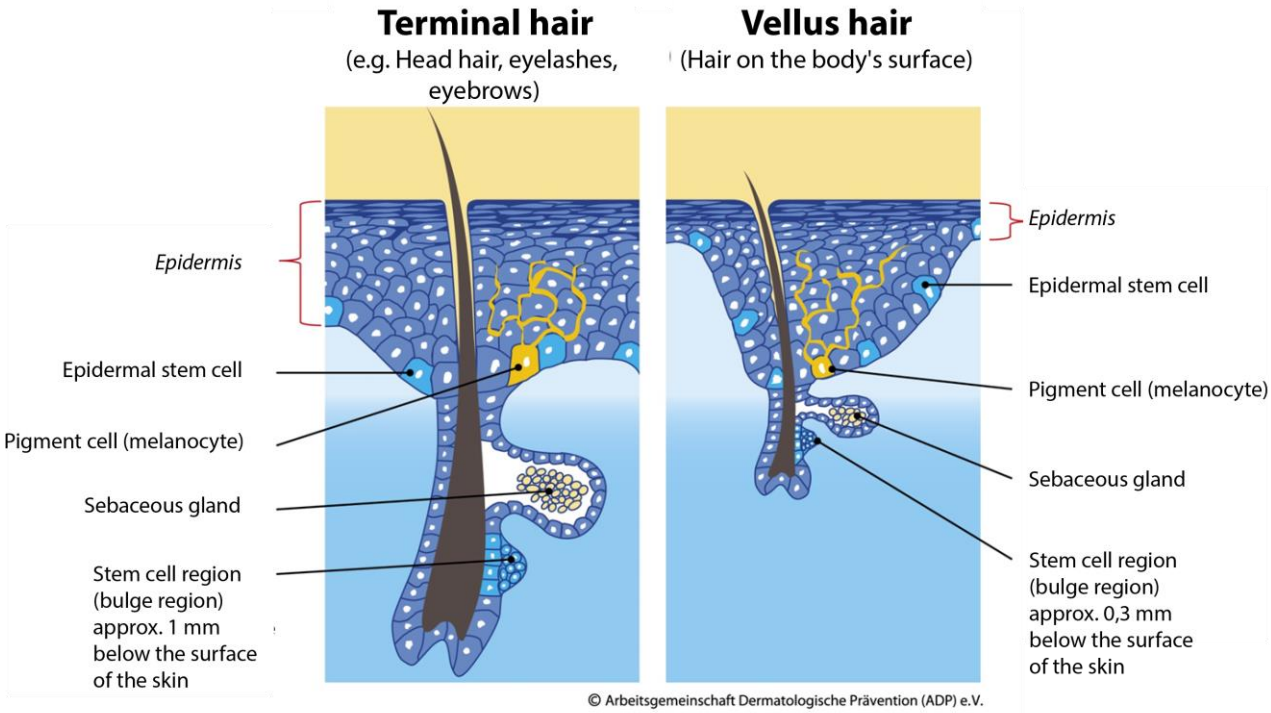


Fig. 19*: Localisation of the stem cell region (bulge region) in vellus hair and terminal hair. Children's skin mainly has vellus hair, which develops into terminal hair during puberty. The stem cell region (bulge region) of vellus hair is closer to the surface of the skin, and is thus exposed to higher levels of UV radiation. (German Association of Dermatological Prevention – ADP e.V, modified according to (Garcia et al. 2011))*

3.1.2 DNA damage

Induction - Direct reaction path (mainly UVB)

With this reaction path, primarily UVB photons are absorbed directly by the DNA molecule, and the absorbed energy is used for a photodimerisation reaction of neighbouring pyrimidine bases. Two UV-specific photoproducts generally occur as a result of this: cyclobutane pyrimidine dimers (CPD) and pyrimidine (6-4) pyrimidone photoproducts (6-4PP) (see Fig. 20). As can be seen in the action spectrum for the production of CPDs (see Chapter 2.2.3, Fig. 5), UVB radiation is around 1,000 times more effective than UVA radiation (Mouret et al. 2012). CPDs are induced at a ratio of 3:1 when compared to 6-4PP (Mouret et al. 2006). When irradiating cell cultures, a UVB dose that may trigger sunburn in man (skin type II, see Chapter 3.1.4, Table 13) is enough to generate several 100,000 CPDs in the genome of human ceratinocytes in vitro (Greinert et al. 2000).

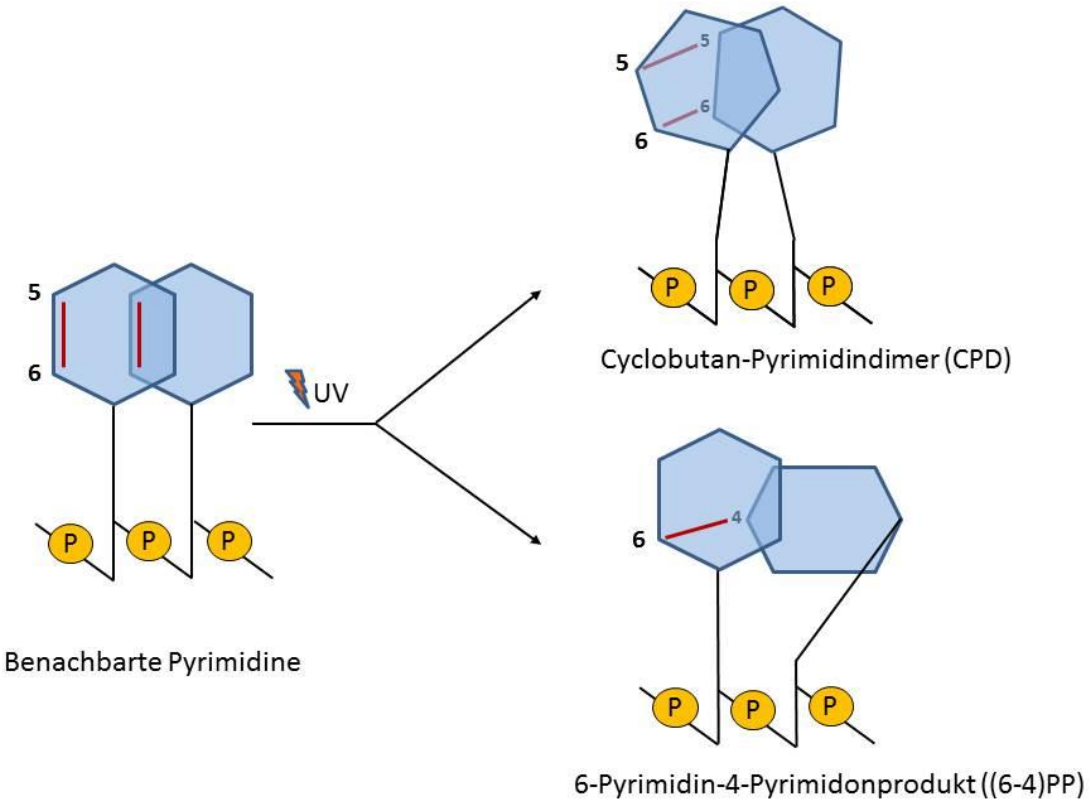


Fig. 20*: Diagram of the most common UV-induced DNA damage. Cyclobutane pyrimidine dimers (CPD) and pyrimidine (6-4) pyrimidone photoproducts (6-4PP) occur after exposure of neighbouring pyrimidines to UV radiation*

Induction - Indirect reaction path (mainly UVA)

UVA radiation mainly develops its damaging effect on the DNA molecule via indirect reaction paths. Here, the energy from UVA photons is absorbed by endogenous cellular photosensitive chromophors (photosensitisers) such as riboflavins and NADH. In an excited electronic (triplet) state, they can then interact with molecular oxygen via so-called type I and II photoreactions to form reactive oxygen species such as superoxide anions, H_2O_2, OH radicals or singlet oxygen. These reactive oxygen species may then lead to DNA single-strand breaks (SSB), DNA double-strand breaks (DSB), DNA protein crosslinking, alkali albile sites (ALS) or base modifications such as UVA-specific 8-oxoguanine (Matsumura und Ananthaswamy 2002a).

New works also show that UVA radiation is also in a position to produce CPDs (even if there is a smaller yield than UVB radiation). The mechanisms leading to the occurrence of CPDs following UVA exposure are yet to be fully established (Cadet et al. 2012, Mouret et al. 2012). However, premutagenic CPDs are the most common cause of UVA-induced DNA damage in human skin (Mouret et al. 2006). Given that 95% of solar UV radiation is within the UVA spectrum, this finding is of particular importance when assessing the risk of solar (and artificial) UV radiation.

Repairing UV-induced DNA damage

In mammal cells, bulky lesions such as CPD and 6-4 PP can be eliminated from the genome by means of nucleotide excision repair (NER). This is a complex interaction involving a number of proteins (up to 30) that are responsible for detecting damage, unravelling DNA, cutting DNA, processing DNA and the like. In the end, the damage is enzymatically cut out of the double helix in a 28 to 31-nucleotide long, single-strand DNA section before the resulting gap is filled again (with the opposite strand as a matrix) (Lagerwerf et al. 2011, van der Wees et al. 2007).

UV-induced oxidative damage such as 8-oxoguanine and other oxidative DNA damage may, depending on the kind of damage and its processing, be eliminated from the genome by a number of enzymatic repair paths (Peterson und Côté 2004), including base excision repair (BER) (Dianov und Parsons 2007, Robertson et al. 2009), DNA single-strand break repair (SSB repair) (Dianov und Parsons 2007, Horton et al. 2008, Wilson 2007), DNA double-strand break repair (DSB repair) (Shrivastav et al. 2008, Valerie und Povirk 2003, Weinstock et al. 2006), mismatch repair (Skinner und Turker 2005, Slupphaug et al. 2003) as well as nucleotide excision repair (NER) (Lindahl 1993, Satoh et al. 1993, Wang 2008).

Overall, these briefly outlined repair systems are able to effectively eliminate DNA damage from the genome (within a period of minutes through to days, depending on the kind of damage and quantity of induced damage). However, taking into account the fact that minimal erythemal dose (see Chapter 2.2.3.2) generates several 100,000 CPDs per genome of each individual exposed cell (Greinert et al. 2000), it becomes clear how effective, e.g. NER works, and how dysfunctions (non-repair) can occur, as is the case with overloading, deficiency and/or limited repair, which can then lead to mutations.

The DNA repair mechanisms are determined genetically. Xeroderma pigmentosum is a genetic disorder that leads to the affected person being much more sensitive to UV radiation and up to 1,000 times more at risk of developing skin cancer (Emmert et al. 2011, Kraemer et al. 2007, van Steeg und Kraemer 1999).

UV-specific mutations

If UVA-induced or UVB-induced CPDs in a cell's genome are not rectified by cellular repair mechanisms, they lead to the occurrence of C→T or CC→TT transition mutations known as UV signature mutations because they generally only occur in the broad spectrum of all potential DNA mutations (Matsumura und Ananthaswamy 2002a, Pfeifer und Besaratinia 2012, Sage et al. 2012). The majority of mutations identified in skin tumours are UV-specific (Aszterbaum et al. 1999, Benjamin und Ananthaswamy 2007, Brellier et al. 2004, de Gruijl et al. 2001). Mutations occur in a range of tumour suppressor genes and oncogenes (e.g. patched, p16, ras, p53), which play a key part in particular in the etiology of basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). CPDs represent the main UV-induced DNA damage in terms of skin cancer. In addition, UVA-specific DNA lesions can contribute to the occurrence of mutations such as T→G transversions as a result of 8-oxoguanine, which can be made responsible for the development of skin cancer (de Gruijl et al. 1993, Dumaz et al. 1997, Matsumura und Ananthaswamy 2002a).

3.1.3 Immunosuppression

One of the main effects of UV radiation is its ability to suppress the body's immunological response to antigens. In vivo experiments involving mice show that (UV-induced) skin tumours following subcutaneous transplantation only grow in animals who have been immunosuppressed in advance with UV radiation, while the skin of non-exposed mice rejected the tumours (Norval 2000). UV exposure triggers a series of reactions in the skin that may lead

to local, but possibly also systemic immunosuppression. These reactions are a complicated interaction involving a wide range of different cell types such as keratinocytes, antigen presenting cells, Langerhans cells and T-helper cells (T_h cells) as well as a number of cellular messengers and mediators such as interleukins (IL-1, IL-2, IL-10) or interferons such as interferon γ (INF-γ). The precise mechanisms are yet to be fully understood. Although a number of immunosuppressive experiments have been performed on animals, it is now assumed that local and systemic suppression of the human immune system is triggered by comparable mechanisms (Bataille et al. 2000, Duthie et al. 1999, Kelly et al. 1998, Moyal und Fourtanier 2002), and has also been proven in the course of human studies (Ruegemer et al. 2002). As one of the immune system's tasks is to identify and eliminate degenerate cells, immunosuppression resulting from UV radiation can also be considered an additional risk factor for the development of skin cancer in man (Yoshikawa et al. 1990). In connection with this, the increased risk of skin cancer should also be associated with (transplant) patients receiving immunosuppression therapy.

3.1.4 Tanning

Tanning is the result of UV-induced melanogenesis (Maddodi et al. 2012), which can be split into two wavelength-dependent mechanisms (Sklar et al. 2013):

1. UVA-induced pigment darkening (PD)
2. UVB-induced delayed tanning (DT)

Melanin is the chromophore for both radiation qualities (UVA and UVB) that absorbs UV radiation and, following UV exposure, is released via melanocytic dendrites to neighbouring keratinocytes in order to protect their nucleus from the damaging effects of UVA and UVB radiation.

UVA radiation only generates an intense PD following extremely high doses (>100,000 J/m^2) and with particular effectiveness at wavelengths of around 340 nm. Here, UVA causes the oxidation of melanin precursors, which are stored in the melanocytes' small cell organelles, the melanosomes. The oxidised form of melanin is then released via the melanosomes to neighbouring keratinocytes and spreads itself over the nucleus as a protective barrier. The UVA-induced pigmentation change occurs dose-dependent in two phases. At doses > 10,000 J/m² to 20,000 J/m², immediate pigment darkening (IPD) is induced within minutes, but fades again after a period of a few minutes to a few hours. If the UVA doses exceed values 100,000 J/m² to 200,000 J/m², a more intense IPD is induced that fades again within hours and leaves behind persistent pigment darkening (PPD) that may last for more than 24 hours and result in UVA-induced delayed tanning (DT) (Sklar et al. 2013). In contrast to IPD and PPD, DT occurs due to photochemical reactions that generate a resynthesis of melanin. The UVA-dependent melanin accumulation depends on the wavelength. At a range of 340 nm to 400 nm, there is an increase in melanin density in the basal cell layer of the epidermis. In contrast, an increase in melanin granules can be seen in cell layers closer to the surface at a range of 315 nm to 340 nm (due to the low penetration depth of the epidermis). However, it was shown that the IPD, PDD or DT generated by UVA does not provide any (or only very little) protection against subsequent UV exposure (Miyamura et al. 2011). As a result, literature on erythema formation provides a sun protection factor (SPF) of 1.5 for UVA-included tanning, and is thus relatively low (Gange et al. 1985).

UVB radiation results in delayed tanning (DT). It initially occurs around 24 hours after exposure and reaches maximum intensity 3 to 6 days later. Without renewed exposure, this tanning fades within about 3 to 4 weeks as a result of the cell proliferation of human skin. UVB-induced tanning is the result of a de novo synthesis of melanin and an increase in the number

of melanosomes in the melanocytes subsequently transported to neighbouring keratinocytes. Photoprotection from tanning acquired in this way is heavily dependent upon the genetically determined ability to form melanin, which determines skin type (see Table 13). The ratio between pheomelanin and eumelanin (two possible forms of melanin) is, in particular, seen as a key indicator of the presence of weak melano-protection (skin type I, pheo/eu = large) or effective melano-protection (skin type IV, pheo/eu = small) (Kadekaro et al. 2003). Literature provides SPF values of 2 to 3 for UVB/SSR-induced tanning (Gange et al. 1985, Sheehan et al. 2002). However, these values refer to erythema formation and do not provide any information about the influence of DT on DNA damage relevant to skin cancer.

Table 13*: Definition of skin types*

	Properties	**Acute reaction to high UV exposure**	**Ability to form melanin**
Skin type I (Caucasian)	– Extremely fair skin – Generally has freckles – Extremely sensitive skin – Light eyes – Sandy-red hair	Never tans Generally burns	Limited melanogenesis
Skin type II (Caucasian)	– Fair skin – Often has freckles – Sensitive skin – Light eyes – Light hair	Slowly tans Often burns	Limited melanogenesis
Skin type III (Caucasian)	– Medium skin – Light or dark eyes – Brown hair	Slowly tans uniformly Sometimes burns	Normal melanogenesis
Skin type IV (Caucasian)	– Olive skin, generally not sensitive – Dark eyes – Dark brown or black hair	Tans quickly and well Rarely burns	Normal melanogenesis
Skin type V (e.g. Asian)	– Dark skin, generally not sensitive – Dark eyes – Black hair	Rarely burns	Protected by melanin
Skin type VI (Negroid)	– Black skin, generally not sensitive – Dark eyes – Black hair	Very rarely burns	Protected by melanin

3.1.5 Hyperkeratosis

Hyperkeratosis is a UV-induced increase in cell division within the epidermis, represented histologically as an acanthosis, which is then followed by increased horn formation, i.e. thickening of the horny layer of the skin (stratum corneum). Both UVB and UVA can generate hyperkeratosis, although UVB does this to a greater extent (Gambichler et al. 2005, Pearse et al. 1987). UV-induced proliferation of epidermal keratinocytes occurs 24 to 48 hours after exposure (Matsumura und Ananthaswamy 2002b). Here, activation of the epidermal growth factor receptor (EGFR) plays a major part as it is a key signal path via which proliferation, differentiation and survival of the cell are controlled (El-Abaseri et al. 2006). As EGFR activation is a major factor in a number of cancer entities, this mechanism can also be linked to the development of skin cancer. On the other hand, UV-induced hyperkeratosis and the epidermis thickened as a whole provide epidermis cells, particularly the basal cell layer and the cutis below that, a certain level of natural UV protection. However, hyperkeratosis only occurs

with UV exposures that correspond to or exceed a momentary, individual minimal erythema dose, as shown in experiments into the dose-response relationship (Knuschke et al. 2010). Everyday exposures received while avoiding sunburn (which also includes workers who always perform activities outdoors) do not lead to any demonstrable hyperkeratosis. As a result, the protective effect is negligible in terms of erythema sensitivity (Knuschke et al. 2010). On average, repeat extreme UV exposures over prolonged periods of time (daily over a period of 20 days resulting in UV erythema) lead to a 40-fold increase in the *MED*. A similar procedure involving an albino increased the *MED* by a factor of 4.5, which was confirmed by the histology and attributed to the reactive acanthosis that was triggered. This led to the conclusion that following excessive UV exposures, Caucasian skin increases individual *MED* by a factor of 40. The reactive acanthosis triggered thereby accounts for around a quarter of the *MED* increase (Jung und Anton-Lamprecht 1971).

3.1.6 Sunburn (inflammation, solar erythema)

Inflammatory early effects largely triggered by UVB have been investigated thoroughly, both in vitro and in vivo. They are characterised by a complex regulated change in the concentration of molecules which maintain the intercellular communication network between different cell types (keratinocytes, melanocytes, mast cells, granulocytes and macrophagocytes) in the human epidermis (Black et al. 1980, Clydesdale et al. 2001, Krutmann und Grewe 1995, Urbanski et al. 1990). UV-induced changes to these molecule class concentrations can be determined 2 to 72 hours after exposure. Histamine release of mast cells appears to represent an extremely early post-exposure event, and vasodilation is a clinical parameter that can be determined at an early stage which eventually leads to clinically perceivable erythema. From a morphological perspective, this involves a (subjectively) barely perceivable reddening of the skin that occurs 24 hours after exposure and subsides after a number of days. This kind of slight reddening represents the mildest form of sunburn. However, as the level of UV exposure increases, severe sunburn may occur together with blistering. The occurrence of erythema remains the dermatological quantity for overexposure of the skin to UV radiation. Nevertheless, DNA damage of relevance to skin cancer may in fact occur before the erythema threshold is reached.

3.1.7 Vitamin D

3.1.7.1 Cutaneous vitamin D3 synthesis

Vitamin D3, the precursor to naturally occurring, biologically active vitamin D metabolite 1α,25-dihydroxyvitamin D3 (1α,25(OH)2D3), occurs in human skin when UVB radiation (280 nm to 315 nm) acts on 7-dehydrocholesterol (7-DHC) (provitamin D3) (Holick 2007, Reichrath 2009) (see Fig. 21). 7-DHC is formed from cholesterol. Based on living conditions in Germany and a number of other countries with similar solar exposure, it is estimated that 80% to 90% of vitamin D present in the body is formed due to endogenous synthesis in the skin, while around 10% to 20% of vitamin D comes from the consumption of food (vitamin D3 from animal foodstuffs, vitamin D2 from vegetable foodstuffs and fungi) (Holick 2007).

UVB-triggered synthesis of vitamin D3 from its precursors takes place within a complex network of photochemical reactions which, according to the current state of knowledge, is characterised by conversion rates in four photoreversible and one non-reversible phototransformation, and involves a total of nine different biochemical equilibrium reactions (Lehmann et al. 1999, Schlumpf et al. 2010). As well as other factors such as UV exposure time period and exposed skin surface, vitamin D3 formation in the skin largely depends upon wavelength and irradiance of the UV radiation as well as the ratio of UVB (280 nm to 315 nm) to UVA (315 nm to 400 nm) (Holick et al. 1980). If previtamin D3 is formed in the skin due to UVB exposure, a few minutes later an equally UVB-dependent photoisomerisation of

previtamin D3 occurs via an additional reaction path along with the (in terms of calcium metabolism) inactive isomers tachysterol and lumisterol (Holick 1981, Wacker und Holick 2013). This results in a stable equilibrium. Vitamin D3 also absorbs UVB and is broken down into various photoproducts such as 5,6-transvitamin D3 or suprasterol as a result of a photodegradation process (Wacker und Holick 2013). Photoisomerisation and photodegradation prevent vitamin D3 from being overproduced (see Chapter 3.2.2).

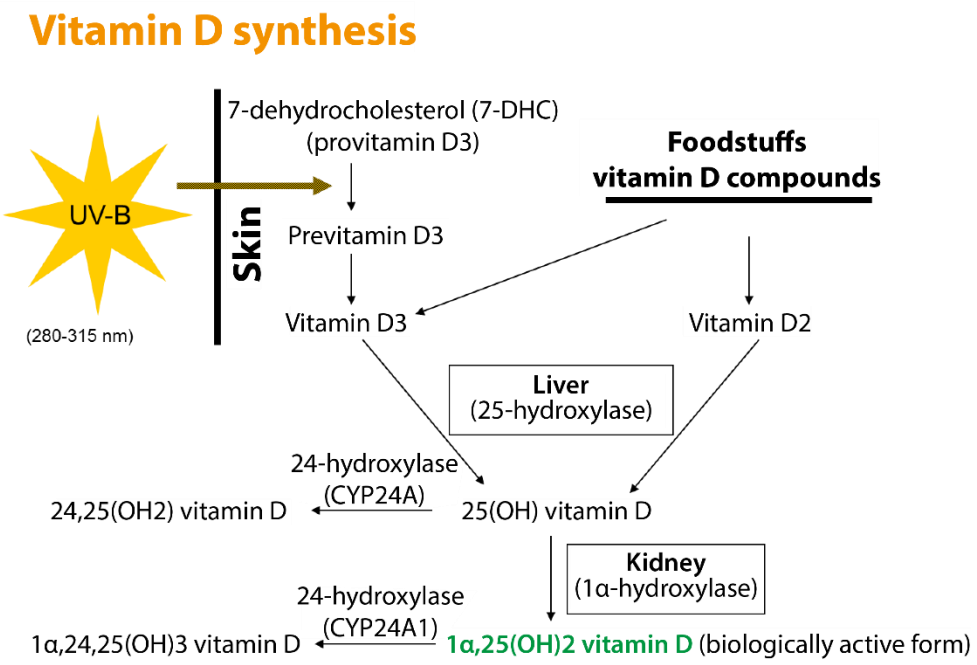


Fig. 21: Diagram of vitamin D synthesis (according to Asmuß and Baldermann 2012)

3.1.7.2 Human vitamin D metabolism

Vitamin D formed in the skin or consumed via foodstuffs passes through the blood stream bound to vitamin D-binding protein (DBP or GC) and on to the liver where it is hydroxylated for the first time in C-25 position by the cytochrome P450 enzyme (vitamin D-25 hydroxylation, CYP27A1; CYP2R1) (Holick et al. 1980) (see Fig. 21). 25-hydroxyvitamin D [25(OH)D] is the main vitamin D metabolite in blood, where it is generally bound to DBP. A second hydroxylation in C-1 position occurs in the renal tubule and involves another cytochrome P450 enzyme, namely 25-hydroxyvitamin D-1α-hydroxylase (CYP27B1). This results in 1α,25(OH)2D3, the biologically active metabolite of vitamin D. While C-25 hydroxylation in the liver does not appear to be impaired later in life (Harris und Dawson-Hughes 2002), the ability for C-1 hydroxylation is assumed to decline due to age-related functional renal impairment, although renal function does need to be extremely limited for this to occur (Armbrecht et al. 1980, Gallagher et al. 2007).

The concentration of 1α,25(OH)2D3 in blood is regulated by a feedback mechanism involving 1α,25(OH)2D3 itself (via induction of the 1α,25(OH)2D3-metabolising enzyme [1α,25(OH)2D3-24-hydroxylase, CYP24A1]), and also by parathormone (parathyrin, parathyroid hormone - PTH), calcium and various cytokines such as interferon γ (IFNγ) and the tumour necrosis factor α (TNFα) (Holick 2003, Holick 2007).

It was long assumed that only the kidney is able to convert 25(OH)D to the active form 1α,25(OH)2D3. However, in vitro experiments as well as studies on nephrectomised patients have shown that a number of extrarenal cells such as keratinocytes, monocytes, macrophages, osteoblasts, prostate cells and colon cells are also able to convert 25(OH)D to the active form

1α,25(OH)2D3 by expression of 1α-hydroxylase (Bikle et al. 1986, Holick 2007, Lehmann et al. 1999). In keratinocytes, both 1α-hydroxylase (CYP27B1) and 25-hydroxylase (CYP27A1) were proven (Fu et al. 1997, Lehmann et al. 1999, Seifert et al. 2009), meaning that keratinocytes possess the enzymatic requirements to fully synthesise 1α,25(OH)2D3 from 7-DHC. This was also confirmed by Lehmann et al. (Lehmann et al. 2001) using an in vivo skin model. Based on the current state of knowledge, the 1α,25(OH)2D3 formed in extrarenal tissue is not passed on to the bloodstream and is not involved in regulating bone and calcium metabolism; instead it regulates local proliferation, differentiation and a number of different tissue-specific functions (Holick 2007).

In the target cells and renal tubule cells, the breakdown of 1α,25(OH)2D3 is initiated by CYP24A1 via a third hydroxylation in C-24 position (Holick 2007). This results in 24,25-dihydroxycholecalciferol, which only has a weak biological effect (Henry 2001, Holick 2007). Based on the current state of knowledge, 1α,25(OH)2D3 has a 100-fold to 1,000-fold higher level of biological activity than other known natural vitamin D metabolites (Holick 2007).

These metabolic steps apply in equal measure to vitamin D2 and vitamin D3. However, the biological effectiveness of vitamin D2, which humans only absorb through foodstuffs or substitution, may be lower than that of vitamin D3 (Trang et al. 1998).

3.2 Biological effect of UV radiation and long-term effects on the skin

As described in Chapter 3.1, UV radiation penetrates the skin and, once there, leads to immediately measurable effects such as DNA damage. Hours later it often leads to local or systemic reactions by the body such as tanning, sunburn or immunosuppression. Over the course of years, repeat acute damage, indicated for example by UV-induced ageing of the skin, may lead to late health effects such as skin cancer. Vitamin D3 synthesis (see Chapter 3.1.7) is the only positive effect of UV radiation and is in stark contrast to all of the other damaging effects of UV radiation. Vitamin D3 synthesis is indeed a positive effect as a lack of vitamin D can cause negative biological effects in terms of bone structure and preservation.

A distinction must be made between deterministic and stochastic effects when describing the health effects of UV radiation. Deterministic effects always occur after a dose threshold has been exceeded (as is the case with sunburn, for example), where the higher the exposure, the higher the effect that occurs beyond the dose threshold. With stochastic effects, only the probability of occurrence depends on the dose (as is the case with skin cancer, for example). The extent (clinical progression in the event of skin cancer) of the effect is not determined by the dose. Based on the current state of knowledge, a dose threshold is not known for the development of skin cancer.

3.2.1 Skin cancer

Skin cancer represents the most severe late effect due to excessive exposure to solar and/or artificial UV radiation. As can also be seen globally, the number of people developing skin cancer in Germany has been growing significantly for many years now (see Figs. 22 and 23). This rise is attributed to increased solar exposure during leisure time and increased use of solaria. The temporally and locally limited thinning of the ozone layer in Europe may also have an impact on this observation. The introduction of skin cancer screening for people with statutory health insurance in 2008 also led to the expectation of an increase in skin cancer incidence. The German Institute for Cancer Epidemiology regularly provides skin cancer incidence extrapolations for Germany. As skin cancer data collection in the German state of Schleswig-Holstein is nearing completeness, this data will be used as a basis for calculation. The age-specific incidence rates of various types of skin cancer (source: Cancer Register

Schleswig-Holstein, http://www.krebsregister-sh.de) are assigned and totalled for the corresponding age groups of Germany's general population (source: Federal Statistical Office). The extrapolation provides the skin cancer incidence in Germany, based on the assumption that incidence rates for Schleswig-Holstein are representative of Germany as a whole. This assumption appears to be justified as additional estimates (Association of Population-based Cancer Registries in Germany – GEKID) provide similar results for malignant melanoma. New skin cancer incidence rates for 2012 collected as described above are presented in Table 14. By observing all three types of skin cancer, i.e. basal cell carcinoma, squamous cell carcinoma and malignant melanoma, skin cancer was the most common form of cancer in Germany with around 260,000 new registered cases in 2012. For many years now, efforts have been made on a regular basis to warn the general public in Germany about the risks of UV radiation. Any resulting reduction in UV exposure should lead to a decline in the number of new cases of skin cancer. Such a trend appears to be occurring with malignant melanoma (see Fig. 22), but at present it cannot be ruled out that this is due to a random fluctuation.

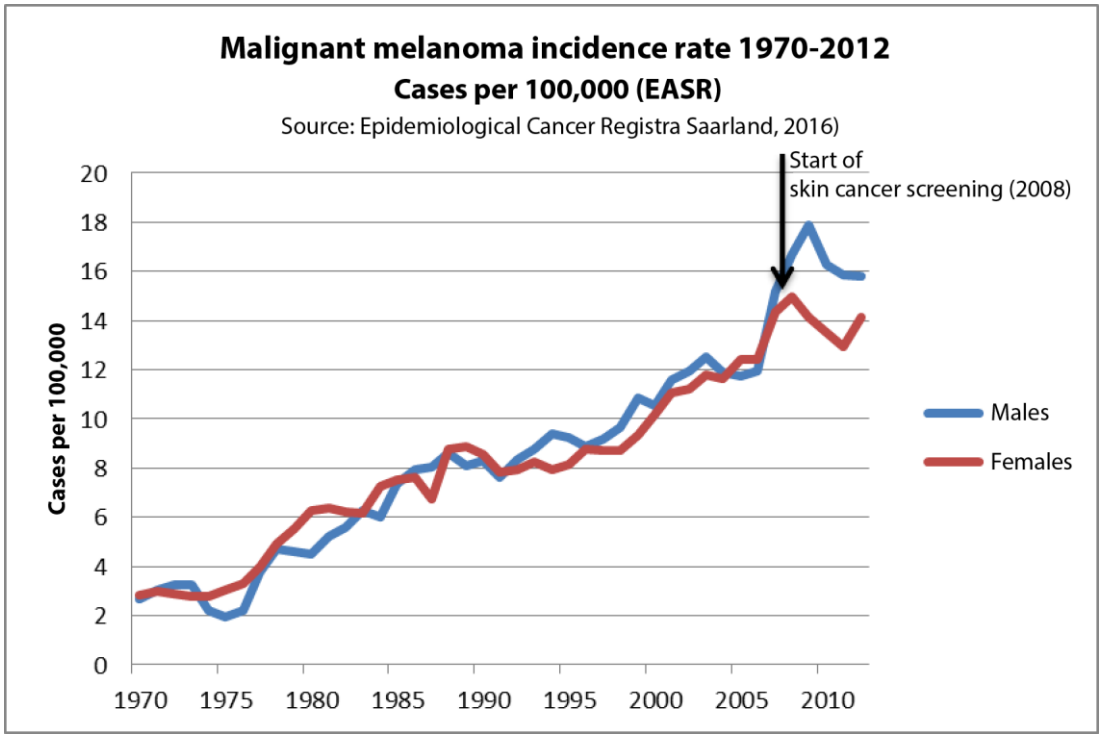


Fig. 22: *Development of age-standardised incidence rates over time for malignant melanoma in Germany, European standard (EASR) (Epidemiologisches Krebsregister Saarland 2016, GEKID 2014)*

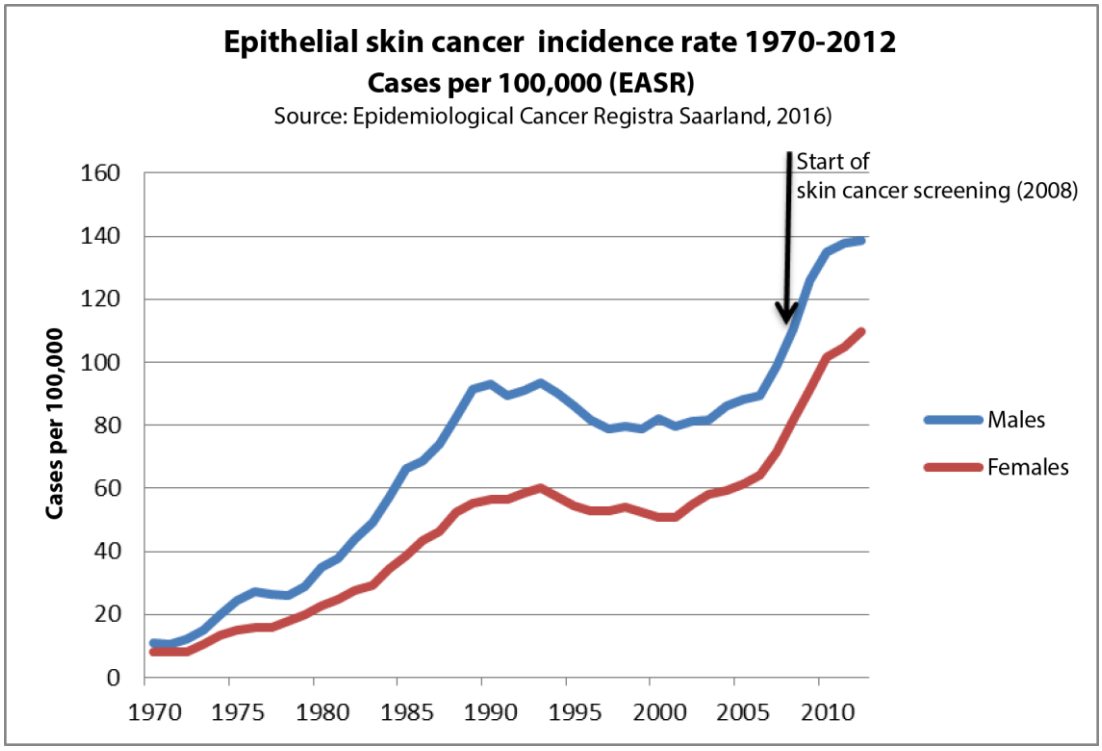


Fig. 23: Development of age-standardised incidence rates over time for non-melanocytic skin cancer (squamous cell carcinoma and basal cell carcinoma) in Germany, European standard (EASR) (Epidemiologisches Krebsregister Saarland 2016, GEKID 2014)

Table 14*: Extrapolation of new skin cancer incidences in Germany in 2012 (age-standardised)*

Skin cancer entity	Total:	Male	Female
Malignant melanoma, invasive	21,180	10,640	10,540
Malignant melanoma, in situ	9,420	4,780	4,640
Malignant melanoma, total	30,600	15,420	15,180
Basal cell carcinoma	149,920	74,970	74,950
Squamous cell carcinoma, invasive	45,140	25,200	19,940
Squamous cell carcinoma, in situ	38,210	17,410	20,800
Squamous cell carcinoma, total	83,350	42,610	40,740
Skin cancer, total	263,870	133,000	130,870

Source: Cancer Register Schleswig-Holstein, 2015 (Katalinic 2015)

3.2.1.1 Basal cell carcinoma (BCC)

Basal cell carcinoma is a tumour that grows slowly and generally starts out as a small nodule that destroys local tissue (see Fig. 24) and only metastasises in a few exceptional cases. It is assumed to originate in the epithelium of hair follicles and generally occurs on the head and throat region, which is constantly exposed to UV radiation. It can, however, also occur in other parts of the body normally covered by clothing. Basal cell carcinoma has no precursor. Depending on its localisation, the consequences can be severe and may have a substantial impact. The risk of developing BCC is influenced both by the lifetime dose of UV radiation and by intermittent UV exposures such as holidays in southern countries and sunburn which occurred during childhood or as adolescents (Armstrong und Kricker 2001, Gallagher et al. 1995b, Kricker et al. 1991, Kricker et al. 1995b, Kricker et al. 1995a, Rosso et al. 1996, Vitasa et al. 1990, Wu et al. 2014a, Wu et al. 2014b, Zanetti et al. 1996).

In up to 100% of all occurring BCCs, mutations (often UV signature C→T transition mutations) are described as occurring in genes of the key Sonic-Hedgehog-Patched-Smoothened signalling pathway involved in hair follicle growth and morphogenesis in the skin (Aszterbaum et al. 1999, Brellier et al. 2004). A number of experiments indicate that this signalling pathway's disorder represents the main reason for developing BCC, and that BCCs arise from hair follicle cells (Boehnke et al. 2012, Boukamp 2005).

Fig. 24*: Basal cell carcinoma*

BCC represents the most common form of skin cancer. According to Table 14, there were 74,970 new cases of BCC in men and 74,950 new cases of BCC in women in Germany in 2012. The 6th and 7th decade of a person's lifespan is the peak age for BCC. Metastasis is extremely

uncommon (Di Lernia et al. 2013). The rate of mortality is low: < 0.6 per 100,000 male inhabitants and 0.3 per 100,000 female inhabitants for non-melanocytic skin cancer (BCC and SCC) (GEKID 2014).

3.2.1.2 Squamous cell carcinoma (spinocellular carcinoma - SCC)

Squamous cell carcinomas destroy local tissue during growth and occur in 90% of cases in areas exposed to UV such as the face, ears, lower lip and back of the hand (see Fig. 25). The risk of developing SCC increases with the UV lifetime dose (Armstrong und Kricker 2001, Wu et al. 2014a, Wu et al. 2014b). SCCs generally occur in skin that has been chronically damaged by UV (e.g. solar elastosis) and in which actinic keratosis forms as a precursor with sharply delimited reddening and a raw, sandpaper-like surface. Over time, firmly adhering, brownish-yellow cornifications that look like a scab will develop. In up to 10% of cases, the changes will result in squamous cell carcinoma. Based on the cornification, the squamous cell carcinoma infiltrates the skin, which subsequently becomes nodular and grows to a point where it may split like an ulcer. This change may take place very quickly, perhaps even within a few days or weeks.

Fig. 25*: Squamous cell carcinoma*

For SCC etiology there is a relatively well described model in which early UV-specific mutations in the p53 gene during tumour initiation favour the development of a precancerous precursor of SCC, actinic keratosis (AK). It is assumed that AK only initially involves a mutation of a single allele of the p53 gene which prevents the p53-dependent apoptosis of some of the UV-damaged cells (sunburn cells). As “neighbouring” cells exhibit normal apoptosis at the same time, p53-mutated cells have a “selection advantage” and can expand clonally to the AK. If the second p53 allele is mutated during the tumour promotion phase, the p53-dependent cell cycle checkpoint function is deactivated. This leads to uncontrolled cell growth and, as a result of induction, to further (possibly UV-induced) mutations in other genes (e.g. ras) during the tumour progression phase, which in turn leads to the formation of invasive SCCs (Brash 1997, Cleaver und Crowley 2002, Ziegler et al. 1994). In more than 90% of patients with in situ SCC (i.e. not yet invasively growing SCC), UV signature mutations were proven in the p53 gene (Ortonne 2002).

SCC is the second-most common skin cancer. There were 42,610 new cases of SCC in men and 40,740 new cases of SCC in women in Germany in 2012 (see Table 14). The 6th and 7th decade of a person’s lifespan is the peak age for SCC, although young people can also develop it as well. The rate of mortality is low: 0.6 per 100,000 male inhabitants and 0.3 per 100,000 female inhabitants for non-melanocytic skin cancer (BCC and SCC). Depending on the extent of the

tumour, around 0.5% to 5% of patients with SCC in the skin experience metastasis (Lund 1965, Møller et al. 1979), although this figure rises to around 16% for patients with extremely advanced tumours (Brantsch et al. 2008). The occurrence of metastasis is associated with a poor prognosis.

3.2.1.3 Malignant melanoma (MM)

Malignant melanoma (see Fig. 26) generally involves a pigmented skin tumour that is responsible for around 90% of all mortalities from skin cancer. Among other reasons, this is due to the fact that metastasis often occurs (in 25% of all cases) (Garbe und Lasithiotakis 2006)). MM occurs in various clinical manifestations. Some melanomas grow slowly into the afflicted area, whereas others spread out and penetrate the skin very rapidly. MM can occur anywhere on the skin, and the areas of the head covered with hair, the mucous membranes and the skin underneath fingernails and toenails may also be afflicted. The link between UV exposure and induction of MM in the skin is regularly called into question as MM also occurs in parts of the body that are not generally exposed to UV. However, most cases of MM (94%) occur in parts of the body that may be regularly or intermittently exposed to UV radiation (face, other parts of the head, neck, chest, back, upper arm, lower arm, hand, thigh, lower leg, foot) (Blum et al. 2004).

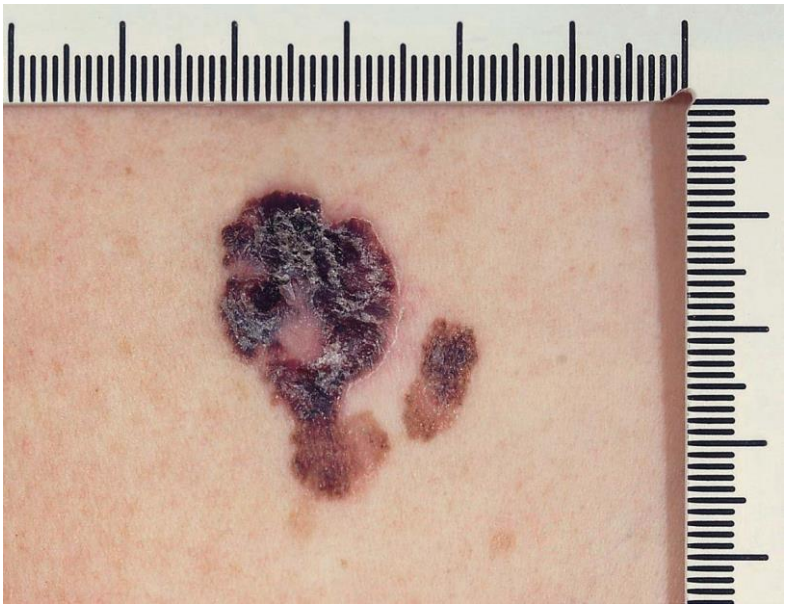


Fig. 26*: Malignant melanoma (scale in mm)*

There are a number of indications that MM occurs as a result of intermittent UV exposure and severe sunburn during childhood and as adolescents (Armstrong und Kricker 2001, Blum et al. 2004, Dulon et al. 2002). This is also supported by so-called immigrant studies which investigate the risk of developing MM by migrating to a country with a high risk of disease when compared to the person's place of birth. The study data involving Europeans with a light skin type who migrated to Israel or Australia showed that the risk of developing MM only increased in the country of immigration if migration occurred during the person's childhood (Cooke 1985, Katz et al. 1982, Khlat et al. 1992, Levine et al. 2013, McCredie und Coate 1989, Steinitz et al. 1989). Light-skinned individuals (skin type I; see Chapter 3.1.4, Table 13) with red or blonde hair, who tend to develop freckles, do not tan, and burn easily in the sun, are at high risk of developing MM. There are indications that this is linked to the formation of the pigment pheomelanin, which has a photosensitising effect as damaging reactive oxygen species

occur after this molecule absorbs UVA radiation (Ranadive et al. 1986). More recent works also indicate that the formation of pheomelanin also increases the level of reactive oxygen species in the cell without the influence of UV radiation (Morgan et al. 2013), which is why pheomelanin can be said to have a potentially mutagenous effect (Mitra et al. 2012). In contrast to SCC and BCC, UV-induced mutations in the p53 gene appear to be of minor importance. Only around 20% of malignant melanomas exhibit p53 mutations (Zerp et al. 1999).

Mutations in the BRAF gene are of major importance to the development of MM (Daniotti 2004, Sasaki et al. 2004). These mutations lead to permanent activation of the important MAPK (mitogen-activated protein kinase) reaction path (Sondak und Smalley 2009). In 2010, Pleasance and staff catalogued for the first time ever the entire spectrum of somatic mutations in the entire genome of a melanoma metastasis (Pleasance et al. 2010). Their catalogue showed that the majority (around 70%) of detected single-base substitutions were type C→T and around 70% of dinucleotide substitutions were type CC→TT. As these are known to be UV-specific mutations (signature mutations – see Chapter 3.1.2), this finding creates a strong link between malignant melanoma and UV exposure (Pfeifer und Besaratinia 2012).

For malignant melanoma there are indications that the predisposition is autosomal dominant as around 3% to 5% of afflicted patients have one or more direct relatives who have also had malignant melanoma (Fallah et al. 2014, Olsen et al. 2010). Mutations in the $p16^{INK4A}$ gene play an important part in this regard (Hussussian et al. 1994, Kamb et al. 1994).

There were 15,420 new cases of MM in men and 15,180 new cases of MM in women in Germany in 2012 (see Table 14), with around 3,000 people dying as a result of their illness. The peak age for MM is around 50, although young people can also develop it as well.

3.2.1.4 Risk factors for skin cancer

The description of risk factors is based on the current S3 guideline on prevention of skin cancer with systematic literature research ranging from January 1995 to April 2012 (AWMF 2014)(AWMF 2014)(AWMF 2014)(AWMF 2014)(AWMF 2014).

When it comes to risk factors, a distinction must be made between constitutional (congenital), acquired and exposure risk factors for each skin cancer type.

Skin type is a constitutional risk factor for non-melanocytic skin cancer (BCC and SCC) (see Chapter 3.1.4, Table 13) (Gallagher et al. 1995a, Gallagher et al. 1995b) (see Table 15), while acquired risk factors include chronically UV-damaged skin (Moon und Oh 2001), the presence of actinic keratoses (Dodson et al. 1991), a previous affliction of BCC or SCC (past medical history) (Marcil und Stern 2000), immunosuppression (e.g. within the context of an organ transplantation) (Berg und Otley 2002, Dantal et al. 1998, España et al. 1995, Jensen et al. 1999, Otley 2002, Preciado et al. 2002) or skin damaged by radiation therapy (Karagas et al. 1996, Lichter et al. 2000, Travis und Arndt 1986).

Pertinent literature incorporating various studies provide values for relative risks (RR) and lifetime risks for the given acquired risk factors for non-melanocytic skin cancer. Some of the values for non-melanocytic skin cancer are provided below by way of example:

The presence of multiple actinic keratoses over a 10-year period with a lifetime risk of developing squamous cell carcinoma (SCC) is stated as being within a range of 6% to 10%. There is a 30% risk of developing an additional SCC within 5 years, and around a 40% risk of developing a basal cell carcinoma (BCC) within 5 years. There is a 44% risk of developing an additional BCC within 3 years, and around a 6% risk of developing a SCC within 5 years. SCCs occur up to sixty-five times more often in immunosuppressed transplant patients than in control patients. Immunosuppressed transplant patients develop more SCCs than BCCs (4:1).

SCC and BCC differ in terms of exposure risk factors. While the probability of developing an SCC correlates with an increasing UV dose acquired over a lifetime (cumulative dose), the UV dose-response relationship for BCC is yet to be fully established. With BCC, cumulative UV exposure only appears to be of minor importance, with intermittent UV exposure playing a great part (Armstrong und Kricker 2001, Khalesi et al. 2013) (see Table 17).

Table 15: Relative risks for non-melanocytic skin cancer (SCC, BCC): Constitutional risk factor "skin type" (see Chapter 3.1.4, Table 13) (Gallagher et al. 1995a, Gallagher et al. 1995b)

Risk factor	**Relative risk Numbers in brackets: 95% CI**
Skin type I vs. IV (BCC)	5.1 (1.4-11.3)
Skin type II vs. IV (BCC)	5.3 (1.7-10.6)
Skin type I vs. IV (SCC)	1.4 (0.5-3.0)
Skin type II vs. IV (SCC)	2.2 (0.7-3.8)

Constitutional risk factors of MM are skin type (see Chapter 3.1.4, Table 13) (Gandini et al. 2005c) and the number of hereditary, in particular large and very large pigmentations (DeDavid et al. 1997, Greeley et al. 1965, Grob et al. 1990, Hendrickson und Ross 1981, Illig 1986). The main acquired risk factors for MM include the number of acquired pigmentations (Bataille et al. 1996, Breitbart et al. 1997, Gandini et al. 2005a, Naldi et al. 2000), the number of clinically atypical pigmentations (Gandini et al. 2005a, Halpern et al. 1991), a previous history of developing MM (Tucker et al. 1985) and a direct relative (parent, child) developing MM (Ford et al. 1995, Hemminki et al. 2001). The risk factors for malignant melanoma identified in a large meta-analysis are set out in Table 16. Intermittent UV exposures (e.g. holidays involving high levels of solar exposure) and sunburn represent the largest exposure risk factor for MM (Armstrong und Kricker 2001, Gandini et al. 2005b) (see Table 17). Exposure in solaria is also a risk factor, and the risk of developing MM due to regular solaria use (once per month) is 60% higher among people below the age of 35 (Boniol et al. 2012a, Boniol et al. 2012b).

Table 16: Relative risks for malignant melanoma (constitutional and acquired) (Gandini et al. 2005a, Gandini et al. 2005b, Gandini et al. 2005c)

Risk factors	**Relative risks (95% CI)**
Skin type I (see Chapter 3.1.4, Table)	2.09 (1.67-2.58): I vs. IV
Number of acquired nevi	6.89 (4.63-10.25): 101-120 vs. < 15
Number of atypical nevi	6.36 (3.80-10.33): 5 vs. 0
Melanoma in family history	1.74 (1.41-2.14)
Lots of freckles	2.10 (1.80-2.45)
Skin colour: light vs. dark	2.06 (1.68-2.52)
Eye colour: blue vs. dark	1.47 (2.80-2.55)
Hair colour: red vs. dark	2.02 (1.24-3.29)
Precursor and skin cancer lesions*	4.28 (2.8-6.55)
Actinic damage**	2.02 (1.24-3.29)

* Actinic keratosis, squamous cell carcinoma, basal cell carcinoma, ** Solar lentigines, elastosis

Table 17: Relative risks of developing BCC, SCC and MM based on various solar exposure samples (Armstrong und Kricker 2001). The relative risk is determined by comparing the lowest and highest UV exposure within the investigated groups with various exposure samples.

Exposure type	BCC - RR (95% CI)	SCC - RR (95% CI)	MM - RR (95% CI)
Total (cumulative):	0.98 (0.68-1.41)	1.53 (1.02-2.27)	1.20 (1.00-1.44)
Occupational	1.19 (1.07-2.27)	1.64 (1.26-2.13)	0.86 (0.77-0.96)
Non-occupational or intermittent	1.38 (1.24-1.54)	0.91 (0.68-1.22)	1.71 (1.54-1.90)
Sunburn at every age	1.40 (1.29-1.51)	1.23 (0.90-1.69)	1.91 (1.69-2.17)

3.2.2 Importance of vitamin D

3.2.2.1 The health discussion surrounding UV-induced vitamin D

Over the last 10 years, experts and, increasingly, the general public, have been discussing whether UV-induced vitamin D synthesis in the skin and the resulting bio-availability of vitamin D in the body can improve a person's health beyond the scope of the known positive influence it has on bone structure and preservation. This is contrasted by the need for thorough UV protection in order to minimise the risk of developing skin cancer, and thus requires a thorough risk-benefit analysis.

The discussion was initiated by epidemiological findings from geographical correlation studies (ecological studies) and experimental studies which support the hypothesis that the development of cancer and other illnesses could be related to reduced vitamin D production owing to reduced UV exposure (Deeb et al. 2007, Garland und Garland 1980, Grant 2009a, Grant und Mohr 2009). The two major analyses conducted by the International Agency for Research on Cancer and the Institute of Medicine (IOM 2011a, IARC 2008), and a review by Lucas et al. 2015 (Lucas et al. 2015) provide a good overview of the literature published on this topic.

Over the last few years, this data has led to increased calls (e.g. by the WHO (Lucas et al. 2006a)) for more balanced messages on radiation protection to be issued (to the public) which, besides pointing out the risks of UV radiation, also indicate the positive health effects of UV radiation (particularly in relation to vitamin D production).

When evaluating the data available on the health effects of UV radiation that are considered "healthy", particularly in terms of vitamin D production and vitamin D levels (in blood or serum), there are at least three points that need to be taken into account:

A Action spectra of vitamin D production / development of skin cancer

B Ideal 25(OH)D concentration and necessary UV exposure

C Study design – study quality

These three points are described below.

A Action spectra of vitamin D production / development of skin cancer:

The action spectra for the occurrence of UV-induced DNA lesions (e.g. thymine dimers, see Chapter 2.2.3, Fig. 5), for the formation of (non-melanocytic) skin cancer, for the induction of erythema, and for the UV-dependent synthesis of previtamin D in the skin are almost identical (see Fig. 27) (Wolpowitz und Gilchrest 2006).

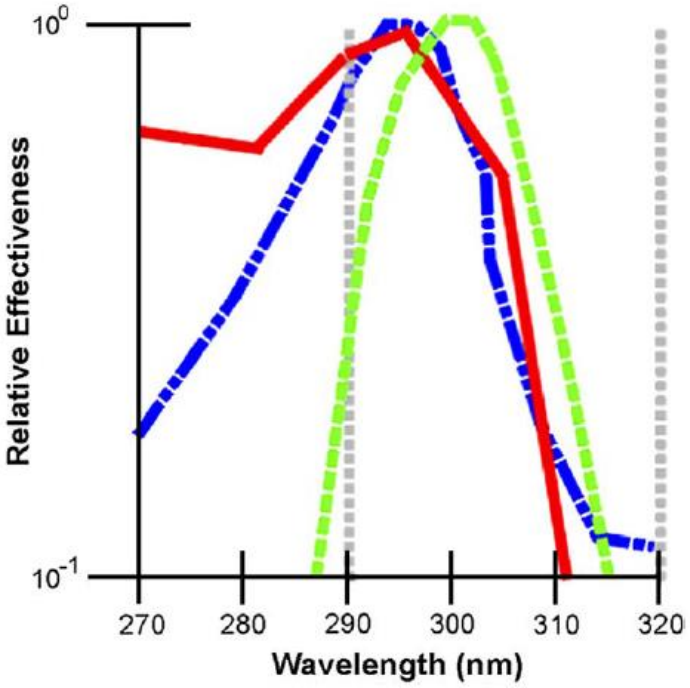


Fig. 27: Cutaneous vitamin D synthesis cannot be considered independently of the damaging effects of UV radiation because the spectral action spectra overlap one another. The diagram shows the action spectra for cutaneous synthesis of previtamin D (blue) (Matsuoka et al. 1987), for induction of squamous cell carcinomas, and for DNA damage (green) (de Gruijl 1999) as well as for induction of erythema (red) (Adams et al. 1982). The maximum effectiveness for all end points is within the UVB range. Diagram from Wolpowitz and Gilchrest 2006

This means that, in principle, UV-induced vitamin D synthesis cannot occur without an increase in the risk of developing premutagenic DNA lesions and skin cancer (de Gruijl 1999, IARC 2008, Wolpowitz und Gilchrest 2006). This fact is important in terms of radiation protection and must be duly taken into account when issuing recommendations to the general public.

B Ideal 25(OH)D concentration and necessary UV exposure:

There is still a lot of uncertainty regarding the quantitative link between UV dose, vitamin D production in the skin, and the extent to which factors such as age, skin pigmentation level, use of sunscreen, areas of exposed skin on the body, ratio of UVA and UVB depending on the time of day and year, and other factors that influence vitamin D synthesis (see e.g. (Fioletov et al. 2009, Fioletov et al. 2010, Godar et al. 2011, Godar et al. 2012, Tsiaras und Weinstock 2011)). Due to a large interindividual variability of measured serum concentrations of 25(OH)D, there is also no evidence of a certain value of vitamin D serum concentration that characterises healthy people in general. Differing methods of determination can also lead to different results (e.g. (Roth et al. 2008)).

Due to large studies (primarily on the risk of falling and risk of fracturing) and the reference values published in a large review conducted by the Institute of Medicine (IOM 2011a) and in agreement with, e.g. the German Nutrition Society (DGE 2011), a 25(OH)D serum concentration of at least 50 nmol/l (20 ng/ml) is considered sufficient for meeting the general public's vitamin D requirements. The DGE considers a deficit to be present at a concentration of less than 30 nmol/l (12 ng/ml), (DGE, ÖGE und SGE 2013).

In light of this, data collected by the Robert Koch Institute that was published in 2008 generated a large amount of public interest. The representative study conducted Germany-wide by the

Robert Koch Institute determined the 25(OH)D concentrations in the serum of 3,917 adults (participants of the nutrition survey conducted between October 1997 and March 1999 and integrated in the national health survey of 1998 (Mensink et al. 1999)) aged between 18 and 79 (RIA testing kit, DiaSorin, Stillwater, USA). The 25(OH)D values in the serum of 10,015 children and adolescents aged between 1 and 17 (participants of the German Health Interview and Examination Survey for Children and Adolescents, KiGGS) were also measured. Children and adolescents from a migrant background were represented in line with their numbers among the general population.

Table 18: 25(OH)D concentrations in serum, annual average by age and gender (Source: Statement issued by the German Nutrition Society (DGE 2011) (according to Hintzpeter et al. 2008a, 2008b)

25-hydroxyvitamin D		All (age: 1-17)	Boys (age: 1-17)	Girls (age: 1-17)	All (age: 18-79)	Men (age: 18-79)	Women (age: 18-79)
[nmol/l]	[ng/ml]	n = 10,015	n = 5,107	n = 4,908	n = 3,917	n = 1,706	n = 2,211
< 12.5	< 5.0	3.8%	3.60%	4.00%	2.0%	2.20%	1.9%
12.5 to < 25	5.0 to < 10	15.50%	15.60%	15.40%	14.3%	13.40%	15.1%
25 to < 50	10 to < 20	43.70%	42.90%	44.50%	41.0%	41.20%	40.8%
50 to < 75	20 to < 30	22.80%	23.3%	22.30%	20.8%	22.60%	19.1%
> 75	> 30	14.20%	14.60%	13.8%	21.9%	20.60%	23.1%

Around 62% of boys and 64% of girls aged 1 to 17 and 57% of men and 58% of women aged 18 to 79 exhibited suboptimal 25-hydroxyvitamin D concentrations of less than 50 nmol/l (20 ng/ml) when averaged over the course of a year (see Table 18). When evaluating and interpreting the results of the study, a value of 25 nmol/l (10 ng/ml) was used as the reference level for determining a deficit (Hintzpeter et al. 2008a), and was also provided 2011 as a reference level for a vitamin D deficit (DGE 2011) in the statement issued by the DGE. According to that study, 19% of boys and girls, and 16% of men and 17% of women aged 18 to 79 exhibited a vitamin D deficit with 25(OH)D values of less than 25 nmol/l (10 ng/ml) (see Table 18).

25(OH)D concentrations in the serum are highly dependent upon the seasons with the average general population exhibiting lower levels in winter and higher levels in summer. In winter, 25(OH)D concentrations of less than 50 nmol/l (20 ng/ml) in the serum were seen in around 50% of infants aged 1 to 2, in over 60% of people aged 18 to 79, and in over 80% of adolescents aged 11 to 17. Even in summer, the majority of women aged 65 to 79 (75%) and migrants aged 3 to 17 (65%) had 25(OH)D concentrations of less than 50 nmol/l (20 ng/ml) in the serum (Hintzpeter et al. 2008a, Hintzpeter et al. 2008b).

Assuming a 25(OH)D deficit at a level of less than 25 nmol/l (10 ng/ml), this was seen to be the case in winter among 31% of women in this category aged between 65 and 79, and among 23% of women in this category aged between 65 and 79 during summer. With adolescent males aged 11 to 17 and from a migrant background, a deficit was seen among 51% of the group during winter and 15% in summer, while with adolescent females aged 11 to 17 and from a migrant background, a deficit was observed among 50% during winter and 17% in summer (DGE 2011).

This data was partially taken as an opportunity to counter a suboptimal vitamin D status (< 50 nmol/l, < 20 ng/ml 25(OH)D correspondingly) among large parts of the general population (more than 50% according to the results of the RKI studies) by recommending a

more intense UV exposure involving both solar and artificial UV radiation. The assumption was also made that thorough UV protection aimed at minimising the risk of skin cancer actually contributes to a lack of vitamin D and should therefore be associated with an increased risk of developing various other illnesses, including cancer. A number of publications issued by a working group posited that this leads to a significant increase in the number of deaths from cancer as a whole, and that the economic burden on the health services due to a lack of vitamin D is much higher than that incurred due to UV-induced skin cancer (Grant 2002, Grant 2008, Grant 2009b, Grant 2011, Grant und Garland 2006, Grant et al. 2005, Grant et al. 2010).

Up until recently, there were no validated results available for UV-dependent vitamin D synthesis in human skin in vivo. Age is known to play a part in vitamin D synthesis of the skin as it declines significantly with age and can drop to a rate of less than 50% (MacLaughlin und Holick 1985). As well as the decreasing thickness of skin as a person ages (Need et al. 1993), a reduced level of 7-DHC is considered to be a potential cause of the reduction in vitamin D synthesis in older skin (MacLaughlin und Holick 1985). Holick et al. (Holick et al. 1989) showed, albeit with very low numbers of test subjects (n), that a single UV full-body exposure of younger test subjects (aged 20 to 30, n=6) leads to a far greater increase in 25(OH)D serum concentration [from 6.5 nmol/l (2.6 ng/ml) to 75 nmol/l (30 ng/ml)] than among older test subjects [aged 62 to 80, n = 6; an increase from 3.2 nmol/l (1:5 ng/ml) to 19 nmol/l (7.6 ng/ml)]. More recent experiments fail to confirm these age-dependent trends (up to the age of 65), both in terms of 7-DHC levels in the skin and when it comes to 25(OH)D_3 formation effectiveness (Knuschke et al. 2012).

A series of experiments and their resulting estimates, particularly those provided by the working group headed by M. Holick (on a small group of test subjects), suggest that in many parts of the world, brief and limited UV exposures to the sun are enough to ensure sufficient cutaneous vitamin D synthesis (Holick 2007). These estimates show that cutaneous production of vitamin D by exposing the body to the sun in a bathing outfit, thus resulting in a minimal erythema dose (1 *MED*), i.e. the UV dose that causes visible reddening of the skin, roughly corresponds to the oral consumption of 10,000 to 25,000 IE (250 to 635 µg) of vitamin D (Matsuoka et al. 1987). As a result, some authors consider an exposure of less than 18% of the body's surface (e.g. hands, arms and face) two to three times per week with a dose of up to a third or half of the *MED* in spring, summer and autumn as being enough for ensuring a sufficient vitamin D supply (Holick 2007, Holick 2008).

Recent investigations which, for the first time ever, determined the vitamin D serum concentration following UV exposure under controlled conditions involving a number of test subjects in "summer clothing", are now able to confirm these estimates by way of experiment:

Rhodes et al. (Rhodes et al. 2010) and Webb et al. (Webb et al. 2011) exposed Caucasians (skin types I, II and III, see Chapter 3.1.4, Table 13), dressed only in a T-shirt and shorts (≈ 35% skin) and with no protection, to an UV irradiation device emitting solar spectrum UV (95% UVA, 5% UVB) totalling 1.3 SED (approx. 0.5 *MED* for skin type II) three times per week for a period of 6 weeks. In the described temporal exposure sample, the 1.3 SED were sufficient to build up and maintain a 25(OH)D serum concentration of 50 nmol/l (20 ng/ml). Taking into account the fact that the study involved the exposed person lying down while receiving UV exposure from a device, Webb et al. corrected the exposure period for a more realistic case where people are upright (standing). Under these conditions, a period of 33 minutes three times per week or 17 minutes per day of solar UV exposure of 35% of the skin (T-shirt and shorts) to the noon sun in summer in Manchester, England, is enough to maintain a sufficient level of 25(OH)D. As Rhodes et al. and Webb et al. show, this period of exposure is a mean for the investigated skin types I - III. It should be noted that exposure of the "sun terraces" (Hoeppe et

al. 2004) may be much higher, meaning that there is a risk of UV erythema being triggered among skin types I – III.

These figures prove that brief outdoor exposures wearing summer clothing and involving UV indices typical during summer are enough to ensure sufficient 25(OH)D serum concentrations.

As vitamin D production depends on the level of the skin's UV exposure (i.e. the product of irradiance and length of exposure) (see Chapter 4), a shorter period of exposure at higher levels of UV irradiance (see above), which may occur during the period around noon in summer in Manchester, are enough to ensure sufficient 25(OH)D serum concentrations, while lower UVI values require a longer period of exposure.

These prolonged UV exposures over relative short periods of time do not lead to an increase in vitamin D formation. In its 2008 report, the IARC (IARC 2008) points out that maximum previtamin D3 synthesis for people with light skin is reached quickly (within minutes) when exposed to sub-erythemal UV doses (Holick 1981) and that an equilibrium between photoisomerisation products (tachysterol, lumisterol), photodegradation and previtamin D3 already occurs after brief UVB exposures (Holick 2004). If this exposure range is exceeded, it only leads to an increase in UV-induced DNA damage and an increase in the risk of developing skin cancer, but not to an increase in previtamin D3 in the epidermis (Gilchrest 2008, Wolpowitz und Gilchrest 2006).

Use of sunscreen and vitamin D production

In order to prevent skin cancer, it is recommended to avoid extreme UV exposure, wear clothing with UV protection, and to use sunscreen. When used correctly (i.e. by applying 2 mg/cm² to the body), modern sunscreens can reduce UVB and UVA exposure and thus limit the risk of skin cancer (Green et al. 2011b). As a result, there have been frequent discussions as to whether sunscreen contributes towards vitamin D deficiency and potential health risks (see above and Chapter 3.2.2.2).

Smaller experimental and patient studies conducted by Matsuoka et al. do in fact show that vitamin D production is limited by the use of sunscreen (Matsuoka et al. 1987, Matsuoka et al. 1988, Matsuoka et al. 1990). The same working group performed a case-control study which reported that long-term users (1 year) of sunscreen exhibited 25(OH)D concentrations of around 40 nmol/l (16 ng/ml) compared to about 90 nmol/l (36 ng/ml) in the control group, i.e. long-term sunscreen users exhibited a lower vitamin D serum level, but no vitamin D deficiency (Matsuoka et al. 1988). These results were also confirmed in a prospective randomised control study conducted by Marks et al. which showed that the use of sunscreen with SPF 17 was enough to avoid the onset of actinic keratoses without causing a vitamin D deficit (Marks et al. 1995). This finding was supported by several other studies (Farrerons et al. 1998, Sollitto et al. 1997). More recent works are contradictory. Applying 2 mg/cm² of sunscreen reduces and prevents vitamin D production in terms of its concentration (Faurschou et al. 2012), while a recent Australian study shows that applying sunscreen has no influence on vitamin D status (Jayaratne et al. 2012). These contradictory findings were reflected in a large investigation carried out within the scope of the US National Health and Nutrition Examination Survey (NHANES 2006 to 2009) and generally linked to spending time in the shade and wearing full-length clothing (Linos et al. 2012). The studies take account of the influence of potentially different behaviour exhibited by sunscreen users who tend to spend prolonged periods in the sun (Autier et al. 2000).

In spite of these somewhat contradictory findings, it is assumed that the use of sunscreen does not lead to a vitamin D deficit (Wolpowitz und Gilchrest 2006). This is, on the one hand, due to the fact that even when using sunscreen with SPF 20, 5% of UVB radiation still reaches the

skin. On the other hand, it is also known that people often do not apply sunscreen as recommended and required to ensure ideal UV protection as per the SPF. Studies show that sunscreen is often applied unevenly and in an insufficient quantity (only about 50% of the required 2 mg/cm²) (Bech-Thomsen und Wulf 1992, Wolpowitz und Gilchrest 2006). However, it should be noted that the use of sunscreen does not provide any protection against skin cancer (IARC 2001).

Vitamin D supplements

At certain times of the year (e.g. in winter), insufficient UV irradiance means that it is practically impossible to synthesise sufficient concentrations of vitamin D or any vitamin D at all by means of cutaneous photosynthesis (Hanwell et al. 2010, Thieden et al. 2009, Webb et al. 2010).

However, a number of major studies conducted over the last few years have shown that vitamin D supplements are enough to maintain or build up sufficient vitamin D concentrations in the serum (DGE 2011, IOM 2011a, IARC 2008). Vitamin D supplements therefore represent one, if not the main exit from the ongoing debate on UV skin cancer risk and vitamin D deficit. International studies assume that a daily vitamin D supplement of 200 IE to 600 IE (depending on age) is enough to ensure sufficient vitamin D supply (Weinstock und Moses 2009). However, other studies state that up to 5,000 IE are required on a daily basis (Aloia et al. 2008, Heaney et al. 2003). This excludes a toxic effect resulting from a vitamin D supplement of up to 4,000 or even 10,000 caused by hypercalcemia and/or hypercalciuria (Hathcock et al. 2007)

There is currently no agreement as to which quantity (IE) of vitamin D should be taken as a supplement (DGE 2011, IOM 2011b, IARC 2008, Zeeb und Greinert 2010).

C Study design

The validity of a study depends on the study design. Generally speaking, interventions studies are associated with a high level of evidence, while ecological studies are linked to a low level of evidence.

The link between UV exposure and the development of skin cancer was confirmed in a number of ecological studies, various experimental studies, and by clarifying interaction mechanisms. For this reason, the SSK performed an evaluation comparing the evidence of cancer risks due to radiation (SSK 2011) in which it concluded that the evidence in favour of a link between UV exposure and skin cancer is convincing. Based on this scientific evidence, the IARC has classified UVA and UVB radiation as a class I carcinogen (carcinogenic for humans) (El Ghissassi et al. 2009).

When it comes to the health effects of vitamin D, convincing evidence gleaned from extensive intervention studies present the positive effect that oral vitamin D substitution has on the risk of older people falling over (see Chapter 3.2.2.2) (DGE 2011). Many of the studies used as a basis over the last few years when discussing a link between a lack of vitamin D (due to low UV exposure) and an increase in cancer mortality (involving various types of cancer) are of an ecological nature (see, e.g. (Grant und Mohr 2009)). Available ecological studies generally involve the collection of cancer mortalities in different geographical regions in which certain approaches were used for UV exposure of the general population. As the IARC emphatically shows, these approaches can only be used to generate hypotheses on the potential effects of UV exposure (IARC 2008) as ecological studies exhibit known limitations when it comes to proving causal relationships. These limitations are predominantly given if associations for groups of individuals (e.g. in different geographical regions) are not the same as for individuals themselves. As a result, some studies assume that every person in a certain region is subject to the same UV exposure as everyone else. Given the known influences of personal behaviour on,

e.g. accumulated UV dose for the given UV Index, this is a misleading generalisation. In view of this, in its report "Vitamin D and cancer" (IARC 2008), the IARC concludes with the estimate that "...ecological studies have many potential biases that affect the ability to draw conclusions from them about causality at the level of the individual person".

The SSK has shared this view for some time now: "Due to the considerable problem with controlling confounders and including interactions at an aggregated data level, the SSK considers ecological studies to be generally unsuitable for investigating epidemiological relationships. Risk estimates that are only or primarily based on ecological studies only provide convincing evidence in a few exceptional cases (radiation exposure is the main risk factor, a large number of small-scale exposure measurements are available, etc.)" (SSK 2007b, SSK 2007a).

3.2.2.2 Risk of illness in connection with vitamin D status

In order to clarify the link between vitamin D status, risk of illness, and the occurrence of various illnesses (incl. cancer), a number of studies (of varying quality) have been conducted over the last few years which mainly look into the influence of vitamin D supplementation under controlled conditions on incidence, morbidity and mortality rates of illnesses. The German Nutrition Society (DGE) used available literature as a basis for compiling the evidence in favour of certain illnesses as well as falls and fractures to show whether or not there are any links (see Table 19).

There is compelling evidence that vitamin D supplements or sufficient vitamin D status in older people is associated with a reduced risk of falls and fractures. With probable evidence, sound vitamin D supply reduces the risk of old people suffering limited range of motion (power, mobility, balance) and reduces the risk of premature death. This evaluation is based on the results of intervention studies and meta-analyses of intervention studies. With all other investigated illnesses, the evidence in favour of vitamin D having a preventive effect was only deemed to be possible or insufficient (see Table 19). This also applies to ecological studies on cancers, which led to interpretations that there is a link between UV exposure, vitamin D production and reduced cancer incidence and mortality.

Table 19: Summary of the results on the potential preventive effect of vitamin D. Table from (DGE 2011)

	Evidence			
	compelling	**prob-able**	**possible**	**insufficient**
Falls	↓ (older people)			
Limited range of motion		↓ (older people)		
Fractures	↓ (older people)			
Cancers, total				Ø
Colorectal carcinoma			↓	
Breast cancer			○	
Prostate cancer		○		
Malignant tumours of the endometrium, oesophagus and stomach, kidneys, ovaries and pancreas, and Non-Hodgkin lymphoma		○		
Pancreatic cancer		○	(↑)*	
Diabetes mellitus type 2				Ø
High blood pressure			○** (↓)***	
Cardiovascular illnesses			↓	
Total mortality		↓ (older people)		

↓ Reduction in risk due to vitamin D supplementation (in intervention studies) and with increasing 25(OH)D serum concentrations (in observational studies),
↑ Increase in risk with increasing 25(OH)D serum concentrations (in observational studies),
○ No correlation,
Ø Insufficient evidence;
* At serum concentrations > 100 nmol/l;
** In normotonics, i.e. people with normal glucose tolerance;
*** In hypertonics

The results of the summary provided by the German Nutrition Society therefore match those of large overview studies presented over the last few years by the IARC and IOM (IOM 2011b, IARC 2008). Among others, the IARC points out that large, randomised controlled trials (RCTs) must first be able to determine whether there is a link between vitamin D supply and a reduction in cancer risk. The IARC also emphasises the substantial fact that there is no evidence to ascertain whether a low vitamin D status could perhaps be due to developing cancer.

Based on the result of these large, recently compiled overview analyses, a group of experts met up at a workshop organised by the ICNIRP, the WHO, and BfS at the start of December 2011. During the course of this workshop, the experts recommended not changing the behaviour suggestions for the general public to avoid increasing the risk of skin cancer when exceeding certain UV Index values (Allinson et al. 2012) (see Chapter 4).

3.2.3 Ageing of the skin

As is the case with all other organs, human skin is subject to a natural ageing process. Unlike other (inner) organs, the skin forms a barrier between the body and the environment, and

protects against the influence of natural (environmental) and artificial exposures such as UV radiation, which could lead to ageing. Premature ageing depends on cumulative UV exposure and UV skin sensitivity, and often occurs in people with a light skin type (skin types I and II, see Chapter 3.1.4, Table 13). Increased solar elastosis occurs due to the deposition of elastin directly below the dermis-epidermis boundary layer, and is responsible for the resulting visible UV-induced premature ageing of the skin (photoageing) which is exhibited in the form of clear, deep folds and wrinkles (see Fig. 28) as well as "leathery" skin. Other manifestations of aged skin include increased pigmentation, dryness, and the onset of telangiectasia (visible venules) (Yaar et al. 2002). When compared to skin exhibiting photageing, aged skin that has been protected from the sun is often thinner, with even pigmentation, looser, and generally only with small, fine folds and wrinkles (Yaar et al. 2002).

Reactive oxygen species (ROS) in skin cells are considered to be a primary mechanism initiated as a molecular response in human skin to UV radiation (UVB and UVA). ROS predominantly consist of superoxide anions (O_2^-), peroxides (H_2O_2), hydroxyl radicals (OH•) and singlet oxygen ($^1O_2$). The exact mechanisms that cause these ROS, e.g. by activating receptors and transmitting signals to the cell, remain unclarified. What is clear, however, is that UV exposure damages the skin's connective tissue. The induction of matrix metalloproteinases (MPPs) such as collagenase MMP-1, which destroys the cellular matrix by breaking down collagen, plays an important part in this process (Angel et al. 2001, Karin et al. 1997). Procollagen expression is also inhibited in the skin's fibroblasts (Fisher et al. 2002), which also has a negative effect on net collagen production following UV exposure (Fisher et al. 2000). This eventually leads to characteristic, UV-exposure-induced (not age-induced) premature folds and wrinkles in the skin caused by photoageing.

Fig. 28*: Skin with chronic UV damage*

3.2.4 Photodermatoses

3.2.4.1 Polymorphous light eruption

Polymorphous light eruption (PLE) accounts for 90% of all genuine photodermatoses (Berg 1989, Ros und Wennersten 1986). PLE is often referred to as a "sun allergy", although it is not actually an allergic reaction since it has not been possible to furnish proof of a relevant photoallergen to date. A number of investigations and experiments into this have already been performed, yet the exact action spectrum of polymorphous light eruption still remains unknown. Indirect findings, such as the fact that patients in whom PLE was also induced when exposed

to UV through panes of glass (through which only UVA can pass), indicate that UVA radiation is the prevailing factor here (Diepgen et al. 1989, Hönigsmann und Ortel 1988, Lindmaier und Neumann 1991).

PLE causes itching, reddening, red nodules (papular) or red patches (plaque), but only in areas of the skin exposed to solar radiation. Nodules with vesicles (papular vesicular) may also occasionally occur. Since all of these skin changes can occur, light eruption is considered to be polymorphous. Patients will only ever exhibit one of the above indications, meaning that the skin is monomorphous. Changes to the skin often occur on the throat, chest, arms and back of the hand. Sometimes they occur on the face, and occasionally around the edges of the ear during childhood. These skin reactions occur several hours after solar exposure and recede within a few days, even without administering any form of treatment. However, they may reappear after being exposed to the sun again.

3.2.4.2 Phototoxic, photoallergic reactions

Phototoxic and photoallergic reactions represent additional potential outcomes of UV exposure. With phototoxic dermatitis, UV radiation triggers photochemical reactions in substances, which in turn lead to inflammations. Photoallergic reactions are where a substance is chemically activated by UV radiation such that the substance is bound to a macromolecule and then triggers sensitisation. This sensitisation can, in turn, lead to allergic reactions.

Phototoxic reactions occur a few hours after exposure and appear in the form of sunburn in exposed parts of the skin. Sunburn involves reddening of the skin, often with swelling and blistering, which are mainly limited to exposed areas of the skin. As is usually the case with sunburn, the skin will subsequently tan (melanin-induced pigmentation). This is quite pronounced, especially with phototoxic reactions caused by external contact, and often lasts for several months. Cosmetics/perfumes and other scents containing bergamot oils with furocoumarins as photosensitive molecules may in particular trigger a phototoxic reaction if applied to the skin and the person then exposes those parts of the skin to the sun. Various medications, the number of which should not be underestimated, can also trigger phototoxic reactions. Such medications include antibiotics, diuretics, retinoids, antiepileptics, antihistamines, antifungals, antidepressants, etc. (Cosa 2004, Lehmann 2004, Lugović et al. 2007, Schauder 2005, Svensson et al. 2001). A list of potentially photosensitising medications is provided in Annex 4. Other lists of photosensitising medications are available in international literature or on the internet (Aronson 2004, Litt 2000, Malone et al. 2003, Moore 2002).

Photoallergic dermatoses occur in areas of the skin exposed to solar or artificial UV radiation. They can expand to neighbouring areas of the skin and spread out to distant regions of the skin. Expansion and spreading out are developments that distinguish photoallergic dermatoses from phototoxic dermatoses, which are limited to the area exposed to UV. Changes observed in the skin are similar to those exhibited with typical allergic eczematous contact dermatitis: Reddening, nodules, vesicles; desquamation during regression. Photoallergic contact dermatitis, which is triggered by a UV-modified contact allergen applied to the skin, can thus be distinguished from a photoallergic reaction to medication where the allergen reaches the skin via the blood stream.

3.3 Ocular damage

The effect of UV radiation on the eye depends, among other things, on the depth to which the radiation penetrates, the intensity and duration of exposure, and the resulting effects over time. UV radiation causes ocular effects in areas where the radiation is absorbed.

3.3.1 Child's eye

The eye's UV transmittance depends on age (see Chapter 2.2.2). In contrast to the anatomy of the adult eye (see Chapter 2.2.2), the transmission spectrum of a child's lens includes an additional narrow window at 320 nm, which allows part of this range to penetrate the lens of a child's eye immediately after birth (Glickman 2011, Söderberg 2011). This phenomenon decreases constantly with age. By the age of 14, only about 1% of UV radiation containing this range reaches the retina. From the age of 30, this transmission window is fully closed (Glickman 2011, Mainster und Turner 2010). Correspondingly, the child's eye may be more sensitive to UV radiation and therefore requires special protection against high levels of UV exposure in order to prevent against damage as described below (Söderberg 2011).

3.3.2 Corneal inflammation and conjunctivitis

UV radiation with a high level of intensity can damage the front of the eye within a matter of hours or even minutes. It can cause photokeratitis (corneal inflammation) and photoconjunctivitis. Both types of damage are caused by photochemical reactions and their resulting oxidative stress in epithelial cells (Black et al. 2011, Cejka et al. 2012). Here, the outermost cells of the cornea and conjunctiva are destroyed, with the effects of the damage presenting themselves in the form of severe eye pain six to eight hours after exposure. The afflicted person feels like he/she has sand in his/her eyes. New epithelial cells are formed constantly in the cornea and conjunctiva, meaning that such damage is reversible, with the healing process taking around one to two days to complete following initial manifestation. Action spectra and thresholds for triggering damage are provided as a standard in DIN 5031 Part 10 (DIN 2012). The action spectra and photoconjunctivitis are provided in Figure 29. The above standard provides a threshold dose for triggering photoconjunctivitis of $H_{s,ko}$ = 50 J/m². DIN 5031-10 sets a value of $H_{s,ke}$ = 100 J/m² as the threshold dose for photokeratitis.

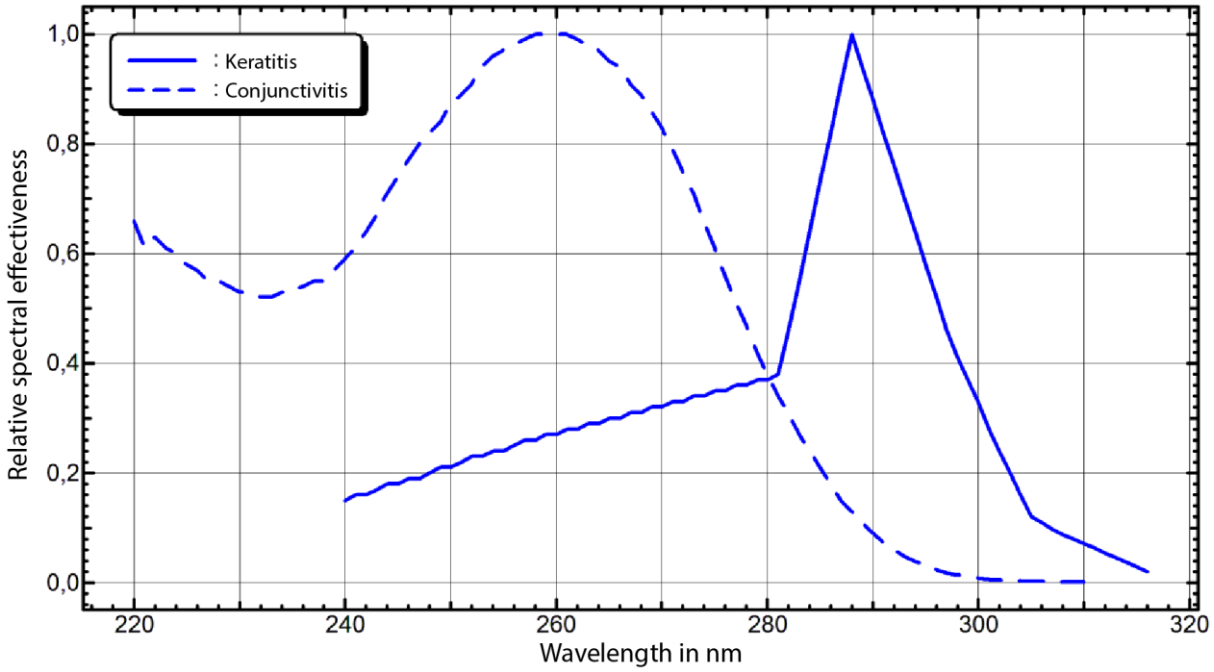


Fig. 29: *Standardised action spectra for photoconjunctivitis and photokeratitis (from DIN 2012*)

Photoconjunctivitis and photokeratitis predominantly occur in situations where the eyes are exposed to high UV irradiance. This is the case, e.g. when spending time on snow-covered mountains when the sky is clear, or when using an electric welder without wearing any eye

protection. Mountaineers call this relatively frequently occurring illness “snow blindness”, while welders call it “welder’s flash”.

3.3.3 Clouding of the eye lens (cataract) due to UV radiation

Long-term or frequent UV exposures can trigger a clouding of the eye lens (cataract). This is caused by a change to crystalline proteins in the lens cells as a result of photochemical reactions. In conjunction with other factors, such as diabetes, this may lead to cell pigmentation and clouding of the eye lens. This process continues over time until the person’s vision is severely impaired or the person goes completely blind. In contrast to other tissue in the body, the eye lens is not able to produce new cells, thus rendering such damage irreversible. Clouding of the eye lens may occur for several reasons: as a result of the effect of ionising, infrared and ultraviolet radiation, prolonged consumption of certain kinds of medication, and certain illnesses such as diabetes (Werdermann 1998). With UV-induced cataracts, UVA and UVB radiation can have a damaging effect (Müller-Breitenkamp et al. 1999), while more recent experiments indicate that UVB plays the more dominant part (Okuno et al. 2012). An exact action spectrum for clouding of the human eye as a result of the long-term effects of UV radiation remains unknown. To date, only action spectra for the short-term effects were determined during animal experiments (NRPB 2002, Okuno et al. 2012).

Cataracts represent a serious health problem worldwide. Risk factors for a cataract include strong genetic components, smoking and UV exposure (Brian und Taylor 2001). According to estimates, around 5% of all cataracts can be attributed to the effects of UV radiation (Neale et al. 2003). Cataracts often lead to blindness in large numbers of people in developing countries where medical provisions are often in short supply and solar radiation is generally high. As long ago as 1990, it was assumed that more than 3 million people go blind each year in India as a result of cataracts (Minassian und Mehra 1990). More recent epidemiological investigations from China present a convincing link between solar UV exposure and cataract frequency (Wang et al. 2012). Several other authors have also highlighted this problem (Lucas 2011).

The UV irradiance level that causes such an illness is far below the level that leads to acute photokeratitis or conjunctivitis. The most important factor here is a long exposure time, generally over several decades. Cataracts can be triggered by artificial UV radiation sources, as was the case with a doctor’s assistant who treated small children with a sunlamp for many years without wearing any eye protection. In this case, it was possible to determine a damaging UV dose (Siekmann et al. 1997). However, cataracts can also be caused by solar UV radiation. This affects people who frequently work outdoors, e.g. farmers or sailors. Cataracts can in fact affect anyone. An age-related cataract occurs among people approaching the age of eighty and above (Werdermann 1998). The number of people among the general public who develop cataracts increases significantly with age. Cataracts are an extremely prevalent illness in general. However, modern operation procedures mean that the clouded eye can be replaced with a plastic lens. Around 700,000 cataract operations are performed every year in Germany (Werdermann 1998). Due to insufficient health care, cataracts remain the world’s most common cause of loss of sight (Bourne et al. 2013, Thylefors 1998).

There is currently no explanation as to whether the development of cataracts is a stochastic or deterministic process (SSK 2009).

3.3.4 Other eye illnesses potentially dependent upon UV

Cataracts are not the only illness where UV radiation is or is considered to be the likely cause. Other such illnesses include pterygium (conjunctivae), pinguecula and squamous cell carcinoma of the eye lens/conjunctiva, and ocular melanoma.

Pterygium (surfer's eye) is a degenerative, mostly bilateral, generally nasal conjunctival hyperplasia that is wing-shaped and grows from the nasal side of the conjunctiva. In a few rare cases, the pterygium grows to such an extent that the pupil is covered, thus limiting the afflicted person's sight. Along with the repeat influence of foreign objects (sand, dust, etc.) UV radiation is also thought to be responsible for triggering pyterygium (Threlfall und English 1999).

Pinguecula is a fibrous degenerative change to the interpalpebral conjunctiva. It involves pathological changes similar to those of actinic elastosis of the skin, hence why UV radiation is discussed as being a possible trigger. However, there is still not a great deal of epidemiological evidence in support of this connection (Norn 1979, Norn 1982).

There is, however, strong evidence in favour of UV-induced skin tumours on the eyelid. In 90% of cases these are BCCs, while in 9% of cases they involve SCCs (Yam und Kwok 2014).

Neoplasia can also occur in the eye due to the effects of UV radiation. Squamous cell carcinoma of the eye lens and conjunctiva typically occur in the eyelid fold (Sun et al. 1997), i.e. a localisation exposed to UV. These neoplasia are rare, but increasingly occur among xeroderma pigmentosum (XP) sufferers. XP is characterised by extreme sensitivity to UV radiation and is based on DNA repair defects for UV-induced DNA damage (Sun et al. 1997). Latitude-dependent experiments and animal studies show that there is a clear link between UV exposure and squamous cell carcinoma of the eye lens and conjunctiva (Kusewitt et al. 2000, Newton et al. 1996).

In terms of ocular melanoma, the WHO assumes in its Environmental Burden of Disease Series, No 13: "Solar Ultraviolet Radiation" from 2006 (Lucas et al. 2006b) that there is "insufficient" evidence for the link between natural UV exposure and the occurrence of ocular melanoma.

A meta-analysis published in 2005 (Shah et al. 2005) agrees in part with this statement, but notes that occupational exposures from welding (during which UV radiation is emitted) represent a risk factor for ocular melanoma.

In 2006, Stang and staff (Stang et al. 2006) pointed out that only little information is available about the incidence of uveal melanomas (i.e. melanomas that occur in the pigmented, melanocyte-rich uvea, the triple-layer structure of the eye, corneosclera-uvea-retina) as such cases are poorly registered or not registered at all. However, the authors of two case-control studies were able to show that uveal melanoma incidence is increasing at a low absolute level (2 to 10 cases per 1,000,000). A new study (Schmidt-Pokrzywniak et al. 2009) carried out by the same working group shows that there appears to be a positive link between light iris colours and UV exposure when it comes to the risk of uveal melanomas.

Epidemiological data gleaned from a more recent review only show minimal evidence of a link between natural UV exposure and the occurrence of uveal melanomas (Mallet et al. 2013). However, molecular experiments into uveal melanomas revealed UV-specific mutations in frequently affected genes of the uveal melanoma (GNAQ, GNA11 and RAC1). The same genes also saw non-UV-specific mutation patterns that also occur in the frequently mutated BRAF gene of the skin's malignant melanoma.

Future research will need to provide clear evidence of a link between UV exposure and the risk of uveal melanoma.

Macular degeneration is the term used to summarise a group of retina illnesses that affect the fovea (macula lutea) and cause gradual loss of function of the various retina cells. The most common form by far is age-related macular degeneration (AMD). As UV radiation is almost entirely absorbed by the cornea and lens in adults, a link between UV exposure and the occurrence of AMD is not to be expected. This was proved in a series of studies reviewed by Yam and Kwok (2014) (Yam und Kwok 2014). On the other hand, a recent meta-analysis

showed a 38% higher risk of AMD in relation to solar exposure (Sui et l. 2013). New molecular investigations show that cumulated deletions occur in the mitochondrial DNA of the macular region which can be linked to oxidative damage (Gendron et al. 2013) and also occur after UV exposure. The current assumption is that visible light and, in particular, the blue spectrum (440 nm to 500 nm) may lead to retina damage and hence the occurrence of AMD. However, it cannot be excluded that the low proportion of UV radiation that reaches the retina may also contribute to AMD.

3.4 Summarising evaluation

In view of the debate surrounding the (potentially) positive effects of UV-induced vitamin D on health, the SSK does not see any need to change the recommendations issued with regard to UV protection and in order to minimise the risk of skin cancer.

Given the current state of knowledge, sufficient vitamin D synthesis is ensured if a person's uncovered face, hands, arms and lower legs are subject to half of the minimum UV dose relevant to sunburn (0.5 *MED*) two to three times per week without wearing any sunscreen. Suitable medication administered under proper medical supervision can be used to treat a vitamin D deficiency that is having a negative effect on a person's health.

Additional solar or artificial UV exposure (e.g. in solaria) should be avoided due to the potential of damaging the eyes and the skin, and especially because of the associated increase in the risk of skin cancer. This also applies to elderly people as they must already be assumed to have sustained significant prior UV damage.

4 UV Index

The UV Index (UVI) was introduced in 1994 by the World Health Organization (WHO), the United Nations Environmental Programme (UNEP), the World Meteorological Organization (WMO) and the International Commission on Non-Ionizing Radiation Protection (ICNIRP). The UVI represents an internationally standardised indicator of the UV irradiance present in a given location. It can be used to estimate an individual's risk of sunburn when spending time outdoors.

The UVI is a way of measuring the erythema-weighted irradiance of solar UV radiation that reaches a horizontal surface, and is determined using equation (6) (Naldi et al. 2000):

$$\mathrm{UVI} = k_{er} \int_{250\,nm}^{400\,nm} E_{\lambda}(\lambda) s_{er,rel}(\lambda) \mathrm{d}\lambda = k_{er} E_{er} \qquad (6)$$

E_{er}: Erythemally effective irradiance [Wm^{-2}]

$E\lambda(\lambda)$: Spectral irradiance [$Wm^{-2}nm^{-1}$]

$s_{er,rel}(\lambda)$: Relative spectral sensitivity for UV erythema (CIE reference action spectrum; ISO 17166:1999/CIE S 007/E-1998))

The scaling factor is $k_{er} = 40\ m^2W^{-1}$. The UV Index is generally expressed as an integer.

The UV Index corresponds to a momentary quantity at a certain time as per equation (6). Weather forecasts often only provide the maximum expected UV Index value for the given day. The UVI represents a dimensionless number due to the introduction of constant k. According to equation (6), UVI 1 corresponds to an erythema-weighted irradiance of $E_{er} = 0.025\ Wm^{-2}$, while UVI 12 equates to an irradiance of $E_{er} = 0.3\ Wm^{-2}$.

An agreement was reached at international level to always state the UVI as an integer. The UVI forecasts provided by the Federal Office for Radiation Protection (BfS) and the German Weather Service[4] take this agreement into account. UVI diurnal cycles or current UVI values are also often published. In line with the recommendation issued by the German Commission on Radiological Protection in 2004, the daily maximum UVI should be clearly marked with the abbreviation "max." (SSK 2004).

UVI - a globally standardised scale

A UVI of 7 is to be understood the same way in Germany as in Kenya or Canada. At the equator, UVI levels of 12 or more may be reached. In Germany, UVI levels may reach 8 or 9 in summer, with some mountainous regions in southern Germany reaching even higher levels than that. The higher the UVI, the faster sunburn will occur with unprotected skin.

UVI - influencing factors

The UVI depends on meteorological factors and the position of the sun. It changes depending on the time of day, year and geographical latitude. The total ozone concentration in the atmosphere (not to be confused with the summer smog ozone concentration close to the ground) and the level of cloud cover and altitude also contribute to UVI levels. Major cloud cover can reduce the UVI, while the presence of just a few clouds hardly reduces the UVI at all. Certain situations involving clouds can in fact lead to the UVI being higher than with a clear sky due to the presence of additional scatter radiation.

UVI application

In the 20 years since the UV Index was introduced, the media have disseminated the UVI in order to let the general public know the daily solar UV exposure forecast, the associated risks and required protective measures (see Fig. 30).

The UV Index is an indicator of the risk from solar UV radiation when spending time outdoors. The UV Index is often used when attempting to determine the level of UV exposure to a person's skin, therefore equating the UV Index's given UV irradiance with skin exposure (UV Index = skin exposure). This can be used to calculate self-protection times, i.e. periods of time up to which people with a certain skin type (see Chapter 3.1.4, Table 13) can presumably be exposed before the onset of skin erythema. However, equating the UV Index with skin exposure is incorrect as personal exposures involving certain parts of the body may differ significantly from local immission. They may, in fact, be lower, equal to or higher than the immission.

UV irradiance may be higher than the UV irradiance provided by the UV Index for the following reasons:

- Areas of the skin on upward-facing parts of the body may be better aligned with the sun than horizontally laid-out measuring surfaces used to determine the UV Index. Depending on the prevalent environmental and weather conditions, UV irradiance on such surfaces may also be higher than on horizontal surfaces. This leads to increased UV exposure and shorter times until the onset of sunburn on the body's "sun terraces" (nose, forehead, shoulders, etc.).

[4] (http://http://www.bfs.de/EN/topics/opt/uv/index/forecast/forecast_node.html)
(http://kunden.dwd.de/uvi/max?uv_euro=ID1)

– UV reflected by a person's surroundings, e.g. when spending time around snow or water, may lead to higher UV exposure. Spending time at altitude also increases UV exposure.

– Clouds at the UVI measuring point may differ from a person's place of residence.

A self-protection time calculated on the basis of the UV Index and a standardised erythema dose threshold also fail to take account of the actual variation in skin sensitivity present from person to person. The above reasons mean that it is not possible to derive exact self-protection times purely from the UVI values.

The UV Index is a general indicator of the level of potential risk from spending time outdoors without protection. The higher the UV Index, the higher the potential UV burden, and hence the more necessary it is to implement sun protection measures to avoid sunburn and long-term damage such as skin cancer (see Fig. 30). When applied this way, the UV Index can be a useful aid in protecting against damage from solar UV radiation.

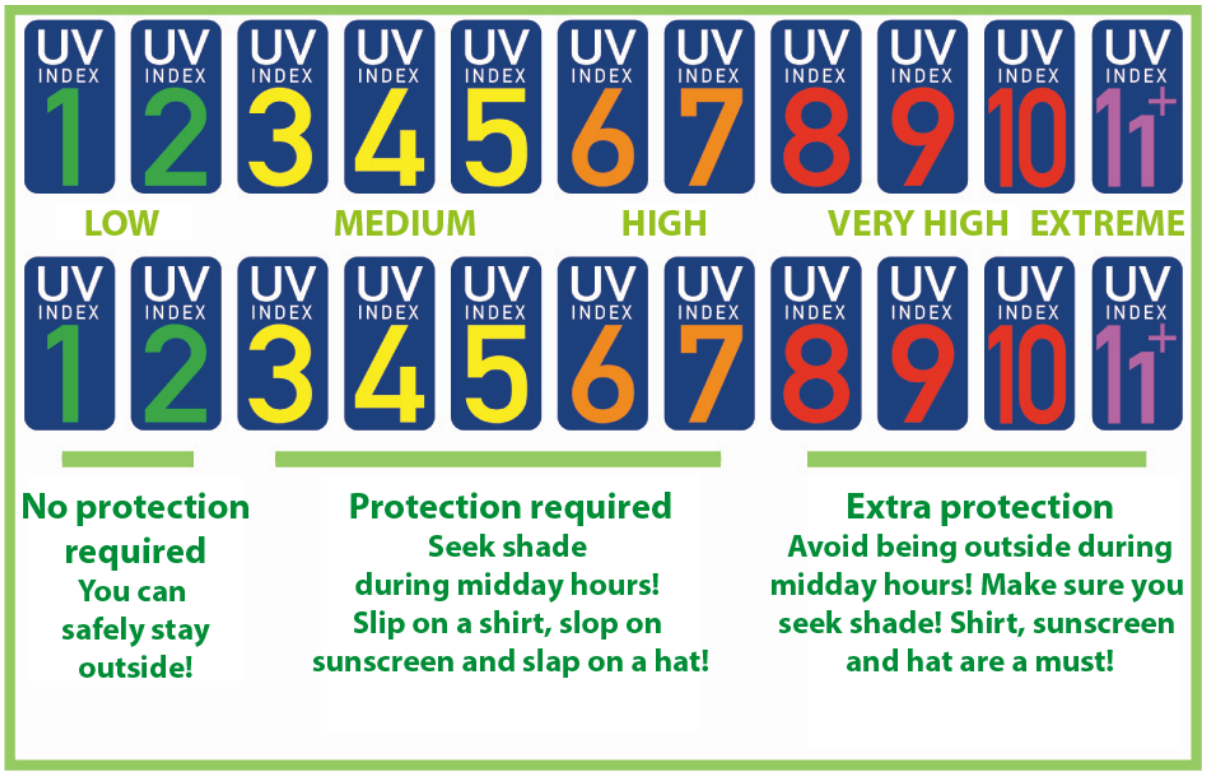


Fig. 30: *Internationally agreed UV protection recommendations depending on the UV Index (http://http://www.bfs.de/EN/topics/opt/uv/index/index_node.html, last visited on 21 July 2016)*

Back in 2005, an "international workshop on UV exposure guidance" and vitamin D looked into "balanced messages" aimed at avoiding skin cancer while also representing an ideal vitamin D status (ICNIRP 2006). This workshop established several gaps in scientific findings before moving on to define recommendations for future scientific investigations (McKinlay 2006).

Since the workshop was held in 2005, there has not been a downturn in the scientific debate on the risk of skin cancer from UV radiation in contrast to the benefits of UV radiation in ensuring sufficient vitamin D status. Discussions have been held at scientific conferences as to whether the UV Index and its associated recommended protective measures need to be revised on the basis of new scientific findings.

For this reason, at the end of 2011, ICNIRP, WHO and BfS formed a working group of experts tasked with reviewing and evaluating the scientific basis for UVI. The working group put together an overview of new works on vitamin D and also investigated the role of UVA radiation when determining the UVI and in terms of photocarcinogenesis (Allinson et al. 2012).

The group of experts provided the following summarising evaluation:

– "Based on these gaps in knowledge and the note that only small amounts of UVB radiation, well below the minimum erythemal dose (Webb 2006) are needed for vitamin-D-synthesis, the expert group felt it was not warranted at this stage to extend the concept of the UVI to include vitamin D issues";

– "Therefore, although the contribution of UVA to carcinogenesis has been undoubtedly underestimated in the past, minor modification of the action spectrum to take this into account is not expected to have a significant impact on the UVI".

The following observation was made in terms of using the UVI in intervention campaigns for primary prevention:

– "... the impact of the UVI on sun protection behaviour is currently very limited and primary research is needed to improve the effectiveness of the UVI as an educational tool. The final goal of changing sun protection behaviour in the population might be reached by developing health promotion campaigns that account for personal determinants, such as attitudes, self-efficacy and self-affirmation".

5 Solaria

Despite the fact that UV radiation specifically generated in irradiation devices in solaria is classified as being "carcinogenic for humans" (class Ia carcinogen) by the IARC (El Ghissassi et al. 2009), such devices are frequently used all over the world (Bock et al. 2013, Diehl 2010, Dissel et al. 2009, Heckman et al. 2008, Køster et al. 2009, Schneider et al. 2013, Schneider und Krämer 2010, Schneider et al. 2009, Thomson et al. 2010). There is a trend indicating that solaria are being increasingly used by teenagers (Køster et al. 2009, Thomson et al. 2010) and young adults (Bock et al. 2013, Diehl 2010, Schneider und Krämer 2010).

A 2012 study from Germany shows that 39% of the German population aged 19 to 45 have visited a solarium at least once (ever user), while around 15% of the same age group visited a solarium in the last 12 months (current user). Around 5% of minors are current solarium users, while around 9% of people with skin types I and II are current solarium users. Positive determinants for using solaria are female gender, a migrant background, and full-time or at least part-time employment. The main reason given for using solaria are for relaxation purposes and to boost attractiveness (Schneider et al. 2013).

Unfortunately, there are only a few studies that look into spectral distribution and real irradiance of UV radiation in solaria. However, the few studies published on the subject (up until 2013) show that the erythema-weighted irradiance is often higher in many devices than that set out in recommendations and national/international classifications (Bonino et al. 2009, Facta et al. 2013, Gerber et al. 2002, Gies et al. 2011, Nilsen et al. 2011, Nilsen et al. 2008, Oliver et al. 2007). This appears to particularly apply to the UVA in the emitted UV spectrum of irradiation devices used in solaria. Nilsen et al. conducted a large-scale study where they performed spectral measurements on more than 190 UV irradiation devices in 78 solaria in Norway. This study showed that the mean UVA irradiance of the face was 5 times higher than the level of natural solar exposure (35°N, Crete during the period around noon). At the irradiation device's two points of emission (bed and lid), the UVA irradiance was 3.2 times and 3.4 times higher

respectively than in the sun. Peak UVA exposures in solaria were 19 times higher than the level of solar exposure. The mean UVB exposures in solaria were 0.7 to 0.8 times lower than regular solar exposure. The mean erythema-weighted irradiance in solaria corresponded to that of solar exposure in Crete during the period around noon (35°N) (Nilsen et al. 2011, Nilsen et al. 2012).

In 2009, Germany introduced the Act on the protection against non-ionising radiation used in humans to govern UV exposure and the use of solaria (NiSG 2009, Yam und Kwok 2014) along with the accompanying UV Radiation Protection Ordinance (UVSV 2011, Linos et al. 2012). This legislation prohibits the use of erythema-weighted UVA and UVB irradiance at levels above 0.3 W/m². Solaria operators are banned from letting adolescents below the age of 18 use their facilities. Since November 2012, solaria operators are required to employ trained personnel who are in turn required to offer their customers advice on tanning. Further details about UVSV are provided in List 1.

List 1: Contents of the German UV Radiation Protection Ordinance (UVSV) (selection)

- Maximum irradiance:
  - E_{er} (250 – 400 nm) ≤ 0.3 W/m²
  - E_{er} (200 – 280 nm) ≤ 0.003 W/m²

  These values are also stipulated in EN 60335-2-27:2008
- Equipment and organisation safety measures:
  - Provision of UV goggles (protection level 2-5 as per DIN EN 170)
  - Designed or marked minimum distance to be maintained from UV lamps (if the distance can be adjusted)
  - Emergency shutdown
  - Forced shutdown at 800 J/m²
  - Minimum selectable erythemally effective irradiation H_{er} ≤ 100 J/m²
- At least one qualified person (specialist) for the solarium's entire opening hours if the solarium has more than 2 devices:
- Instruct customers on how to use the equipment (emergency shutdown), offer advice in terms of UV skin type, and provide a dose plan
- Duty to inform customers of the following:
  - Maximum irradiation time of first irradiation for UV skin types I - VI
  - UV skin types I and II are exclusion criteria for using solaria
  - Risk of exposing the eyes to UV radiation and in conjunction with medication or cosmetics
  - Ban on children and adolescents below the age of 18 from using solaria
- Duty to keep records (incl. rolling sunbed and log book)

It should be noted that this – now legally binding – value of 0.3 W/m² for maximum permissible erythema-weighted irradiance in solaria still represents an UV Index of 12 (vertical irradiation), i.e. it is a value equivalent to the erythema-weighted solar irradiance present at noon at the equator with clear skies. This means that it is not possible to declare exposures received at solaria as being free of risk under these conditions. This was proven, e.g. in recent studies and meta-analyses that clearly indicate a link between solaria use and an increased risk of skin cancer (Berwick 2008, Coelho und Hearing 2010, Doré und Chignol 2012, Fears et al. 2011, Lazovich et al. 2010). However, the best evidence in support of this link comes from two large-scale meta-analyses published in the last 7 years. Back in 2007, IARC published a meta-analysis summarising works that looked into the link between solaria use and melanoma risk. The meta-analysis showed that regular solaria use (once per month) before the age of 30 led to a 75% increase in the risk of developing melanoma later in life (IARC 2007).

A recent meta-analysis from 2012 (Boniol et al. 2012a) now covering 27 studies uses a 1.20% (95% CI: = 1.08 to 1.34) increase in the relative risk of developing melanoma when comparing solaria "ever users" with "never users". This risk increases to 1.59% (95% CI: 1.36 to 1.85) if first solaria use takes place before the age of 35**.** A dose-response relationship was also calculated which showed that each additional solarium use per year leads to a 1.8% (95% CI: 0

to 3.5) increase in melanoma risk. It is also estimated that around 5% of all new melanoma cases in Europe can be attributed to solaria use (mainly among women). This meta-analysis also includes relative risks (RR) for the occurrence of non-melanocytic skin cancers (SCC, BCC) when comparing "ever users" with "never users"; SCC: RR=2.23 (95% CI: 1.39 to 3.57), BCC: RR=1.09 (95% CI: 1.01 to 1.18). These data emphatically support the recommendations against using solaria, and a number of countries worldwide have either imposed restrictions on solaria use or, as is the case in a few countries, banned solaria altogether (Sinclair und Makin 2013).

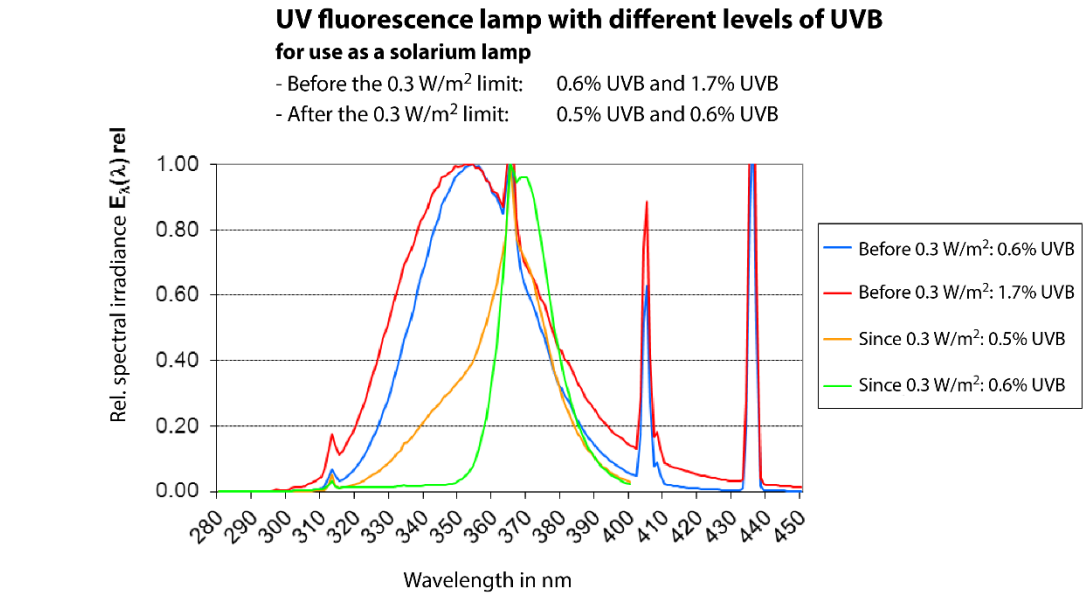
5.1 UV lamp types and lamp spectra

Solaria generally use low-pressure mercury lamps (fluorescent lamps) as their artificial UV radiation sources. Metal halide lamps are sometimes also used, especially for partial body tanning devices and integrated facial tanners in solaria. These lamps are fitted with preliminary filters to remove short-wave UVB, UVC and IR spectra.

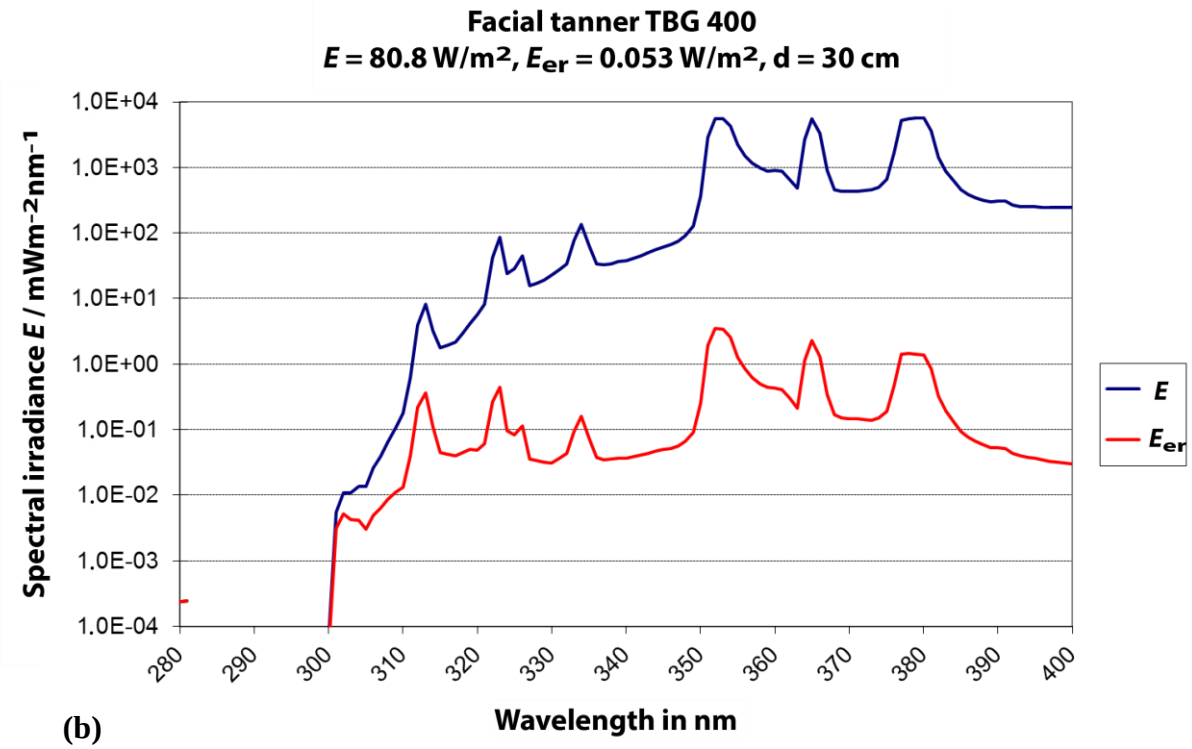
In Germany, the Act on the protection against non-ionising radiation used in humans (NiSG) and UV Radiation Protection Ordinance (UVSV) legally require the erythemally effective irradiance in solaria devices to be limited to a maximum of 0.3 W/m². The radiation spectra of fluorescent lamps typically used in solaria prior to and since the introduction of this legislation are illustrated in Fig. 31(a) together with the level of UVB and UVA in the spectra. The UV and VIS emitted within the radiation spectrum may vary depending on the composition of the fluorescent material. Solaria lamps must be designed such that their emission complies with the irradiance limit of E_{er}(250 nm to 400 nm) ≤ 0.3 W/m² on the user's body. The radiation spectra of solaria lamps as per the 0.3 W/m² limit have clearly shifted to the longer-wave UVA spectrum.

Metal-haloid lamps are medium-pressure mercury lamps with metal haloid added to increase the gas discharge resonance line density to a quasi-continuous spectrum. Fig. 31(b) shows the spectrum and spectral erythemal effectiveness of an UVA facial tanner fitted with a metal-haloid lamp.

It should again be noted that the legal limit for erythemally effective irradiance in solaria is 0.3 W/m² and the associated shift of the used UV spectra to the longer-wave UVA spectrum does not lead to risk-free solaria use. Both the remaining UVB spectrum required to obtain a lasting tan (see Chapter 3.1.4) and the now enhanced UVA range of the spectrum contribute to DNA damage and may ultimately lead to skin cancer (see Chapter 3.2.1).



(a)



(b)

Fig. 31: *Artificial UV radiation sources typically used in solaria (a) UV fluorescent lamp spectra prior to and after the introduction of the erythemally effective irradiance limit of 0.3 W/m² with a higher and lower UVB spectrum and (b) a metal-haloid lamp spectrum with spectral erythemal effectiveness*

5.2 Device design for cosmetic skin-tanning purposes

In Germany, sunbeds are used in 90% of cases. Upright tanning booths are hardly ever used in Germany (in contrast to the USA where they are the most commonly used type of device). The surface on which the person lays down is generally fitted with acrylic glass through which UVB

can pass. In order to determine the erythemally effective irradiance of ≤ 0.3 W/m², the resulting UV radiation of the fluorescent lamp and acrylic glass used need to be taken into account. The tunnel-like cover also contains fluorescent lamps, either with or without additional facial tanning lamps, which either use fluorescent lamps with a higher UVB/UVA spectrum or metal-haloid lamps. There are also sunbed designs where metal-haloid lamps are used for the entire upper body.

As is the case with facial tanners, partial-body tanning devices mainly used in people's homes use both fluorescent lamps and metal-haloid lamps. As stipulated in the German UV Radiation Protection Ordinance (UVSV), users should be informed of the shortest permissible irradiation distance for such lamps in order to prevent the E_{er}(250 nm to 400 nm) ≤ 0.3 W/m² limit from being exceeded.

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Annex 1

Table A1 Terminology of quantities and units used to characterise UV radiation (derived from EN 14255-4 2006, Table 1)

Symbole	Größe (Erklärung)	Maßeinheit	Definiert in
λ	Wellenlänge	nm	CIE 17.4 (1987) ref 845-01-14
λ_1, λ_2	Grenzen des Wellenlängenbereichs $\Delta\lambda$	nm	EN 14255-4 (2006)
Δt_{exp}	Expositionsdauer	s	EN 14255-4 (2006)
E	Bestrahlungsstärke	W/m² (10 W/m² = 1 mW/cm²)	CIE 17.4 (1987) 845-01-37
$E_\lambda(\lambda)$	spektrale Bestrahlungsstärke	W/(m²·nm)	EN 14255-4 (2006)
H	Bestrahlung	J/m² (10 J/m² = 1 mJ/cm²)	CIE 17.4 (1987) 845-01-42
$H_\lambda(\lambda)$	spektrale Bestrahlung	J/(m²·nm)	EN 14255-4 (2006)
$s_{biol}(\lambda)$	Wichtungsfunktion für einen photobiologischen Effekt	—	
$s_{er}(\lambda)$	Erythemwichtungsfunktion	—	ISO/CIE 17166 (1999)
$s(\lambda)$; $s_{eff}(\lambda)$ *)	Wichtungsfunktion für die Gefährdung durch Ultraviolettstrahlung ($s(\lambda) = s_{eff}(\lambda)$) *)	—	EN 14255-4 (2006) *) z. B. in ICNIRP (2004)
$s_{nmsc}(\lambda)$	Wichtungsfunktion für die Photokarzinogenese nicht-melanozytärer Hautkrebs (NMSC)	—	CIE S 019/E (2006a)
E_{biol}	biologisch effektive Bestrahlungsstärke	W/m²	
E_{er}	erythem-effektive Bestrahlungsstärke	W/m²	ISO/CIE 17166 (1999)
E_S; E_{eff} *)	effektive Bestrahlungsstärke für die Gefährdung durch Ultraviolettstrahlung ($E_S = E_{eff}$) *)	W/m²	EN 14255-4 (2006) *) e. g. ICNIRP (2004)
E_{nmsc}	Non-Melanoma Skin Cancer effektive Bestrahlungsstärke	W/m²	EN 14255-4 (2006)
H_{er}	erythem-effektive Bestrahlung	J/m²	ISO/CIE 17166 (1999)
H_S; H_{eff} *)	Effektive Bestrahlung für die Gefährdung durch Ultraviolettstrahlung ($H_S = H_{eff}$) *)	J/m²	EN 14255-4 (2006) *) e. g. in ICNIRP (2004)
H_{nmsc}	Effektive Bestrahlung bezüglich des NMSC-Risikos	J/m²	EN 14255-4 (2006)
SED	Standard-Erythemdosis (H_{er} = 100 J/m² = 1 SED)	—	ISO/CIE 17166 (1999)
MED	minimale Erythemdosis (individuelle UV-Response der Haut, gekennzeichnet durch eine gerade abgegrenzte zarte Rötung)	—	ISO/CIE 17166 (1999)
I_{UV}	solarer UV-Index ($I_{UV} = k_{er} \int_{250nm}^{400nm} E_\lambda \cdot s_{er}(\lambda) d\lambda$, k_{er} = 40 m²/W)	—	CIE S 013 (2003)
f_{SE}	Hautexpositionsfaktor	—	ICNIRP (2007)
γ_s	Sonnenhöhe (Winkel zwischen Horizont und der Sonne)	°	
SZA	solar zenith angle (Winkel zwischen der Sonne und der Normalen auf die Erdoberfläche) SZA = 90° - θ_s)	°	

*) The weighting function for the risk from ultraviolet radiation is expressed differently in various sources: $s(\lambda)$ in EN 14255-4 and $s_{\mathrm{eff}}(\lambda)$ in ICNIRP (2004). However, these different expressions refer to the same function. There are also different terms in use for spectrally weighted quantities: E_{s} or E_{eff} and H_{s} or H_{eff}.

Annex 2

Indicative estimate of solar UV exposure without measurement

From: ICNIRP, *Protecting workers from ultraviolet radiation*, in *ICNIRP 14/2007*, H.M. Vecchia P, Stuck BE, van Deventer E, Niu S. , Editor. 2007. p. 52-55.

The International Commission on Non-Ionizing Radiation Protection (ICNIRP), the International Labour Organization (ILO) and the World Health Organization (WHO) published a paper titled "Protecting Workers from UV Radiation" (ICNIRP, 2007). A skin exposure factor A 1 and ocular exposure factor A 2 were published so as to be able to provide indicative estimates of solar outdoor exposures for which no measurement data are available about the extent of solar UV exposure.

The skin exposure factor is determined by multiplying a series of risk assessment factors, f_1 to f_6, which influence individual solar UV exposure:

$$\text{Skin exposure factor} = f_1 * f_2 * f_3 * f_4 * f_5 * f_6 \qquad \text{(A 1)}$$

Table A2-2 compiles the set of risk assessment factors that influence the extent of outdoor skin exposures. These factors are not all strictly independent. Nevertheless, it is possible to provide an indicative estimate if at least basic facts such as localisation, time of year, intended length of time to be spent outdoors, surroundings and level of UV protection are known or can be estimated. Once the factors for each situation have been determined (see Table A2-2), they are multiplied to arrive at the skin exposure factor.

Table A2-1 is a classification of the determined skin exposure factors and their derived minimum protection levels. It contains the individual protection components recommended for the individual categories of skin exposure factors.

Table A2-1: Guide for determining the minimum level of protection required for the workplace (derived from ICNIRP 2007)

Haut-Expositionsfaktor	**benötigtes Hautschutzniveau**
< 1	nichts erforderlich
> 1 aber < 3	Hemd, Hut mit Krempe
> 3 aber < 5	Langärmeliges Hemd und Hosen, Hut mit Krempe, Lichtschutzsubstanz mit LSF 15+
> 5	Arbeitsumgebung und -gewohnheiten anpassen. Möglichst Schatten schaffen. Langärmeliges Hemd und Hosen, Hut mit Krempe, Lichtschutzsubstanz mit LSF 15+

Table A2-2: Risk assessment factors for solar-exposed skin (derived from ICNIRP 2007, Table 9)

Jahreszeit	**Geographische Breite (Faktor f_1)**		
	>50°N oder S	30°-50°N oder S	<30°N oder S
Frühling/Sommer	4	7	9
Herbst/Winter	0,3	1,5	5
Wolkenbedeckung	**Faktor f_2**		
Klarer Himmel	1		
Wechselhafte Bewölkung	0,7		
Bedeckter Himmel	0,2		
Expositionsdauer	**Faktor f_3**		
Ganztägig	1		
1-2 Stunden zur Mittagszeit	0,5		
Am frühen Morgen oder späten Nachmittag	0,2		
Bodenreflexion	**Faktor f_4**		
Frischer Schnee	1,8		
Trockener Sand, Meeresbrandung, Beton	1,2		
Alle anderen Oberflächen, auch offene Gewässer	1		
Bekleidung	**Faktor f_5**		
Oberkörper, Schultern und Arme ungeschützt	1		
Oberkörper geschützt, aber Arme und Beine exponiert	0,5		
Voll bekleidet, nur Hände und Gesicht exponiert	0,02		
Schatten	**Faktor f_6**		
Kein Schatten, z. B. offene Felder, Tundra, Strand, Meer	1		
Teilweise Schatten, z. B. lockere Bebauung, vereinzelte Bäume	0,3		
Guter Schatten, z. B. dichte Bebauung, Wald, Sonnendach	0,02		

The ocular exposure factor is also determined by multiplying a series of risk assessment factors, f_1 to f_6, which influence individual solar UV exposure (see Tables A2-3 and A2-4):

$$\text{Ocular exposure factor} = f_1 * f_2 * f_3 * f_4 * f_5 * f_6 \qquad \text{(A 2)}$$

Table A2-3: Guide for determining the minimum level of protection required for the workplace (derived from ICNIRP 2007)

Augen-Expositionsfaktor	**benötigtes Augenschutzniveau**
<1	nichts erforderlich
>1 aber <3	Hut mit Krempe
>3 aber <5	Hut mit Krempe, Brillengläser oder Sonnenbrille
>5	Rundum anliegender Augenschutz, Hut mit Krempe

Table A2-4: Risk assessment factors for solar-exposed eyes (derived from ICNIRP 2007, Table 10)

Jahreszeit	**Geografische Breite (Faktor f_1)**		
	> 50°N oder S	30° - 50°N oder S	< 30°N oder S
Frühling/Sommer	4	7	9
Herbst/Winter	0,3	1,5	5
Wolkenbedeckung	**Faktor f_2**		
Klarer Himmel	1		
Wechselhafte Bewölkung	1,5		
Bedeckter Himmel	0,2		
Expositionsdauer	**Faktor f_3**		
Ganztägig	1		
1-2 Stunden zur Mittagszeit	0,3 - 0,5		
Am frühen Morgen oder späten Nachmittag	0,2		
Bodenreflexion	**Faktor f_4**		
Frischer Schnee	1		
Trockener Sand, Meeresbrandung, Beton	0,1		
Alle anderen Oberflächen, auch offene Gewässer	0,02		
Augenschutz	**Faktor f_5**		
Kein Augenschutz	1		
Sonnenbrille ohne Hut	0,5		
Klare Brillengläser ohne Hut	0,5		
Sonnenbrille oder Brille und Hut mit Krempe	0,02		
Schatten	**Faktor f_6**		
Kein Schatten, z. B. offene Felder, Tundra, Strand, Meer	1		
Horizont hinter Bergen oder Häusern, vereinzele Bäume	0,3		
Horizont und teilweise Himmel durch Hochhäuser verdeckt	0,02		

Annex 3

Estimating the UV Index using the shadow rule

The current intensity of solar radiation in a person's given location determines whether or not measures to protect against the sun are necessary. The UV Index is an indicator of the level of solar UV irradiance. The WHO recommends the use of various measures to protect against the sun depending on the UV Index. If it is not possible to find out the current UV Index for the given location from the radio, TV or internet, the UV Index can be roughly estimated using the so-called shadow rule. If there are no clouds in the sky, the UV Index depends on solar elevation, i.e. the height of the sun above the horizon (solar elevation angle). As the length of the shadow of a vertical object also depends on solar elevation, the length of the shadow can be used to determine the UV Index.

Table A3-1: Estimating the UV Index based on solar elevation and the corresponding shadow length if there are no clouds in the sky. Calculation using the Quick TUV Calculator, inputs: Solar zenith angle = (90° - Solar elevation angle), date: 000630, ozone: 300 DU, albedo 0.1, altitude: 0.0 km, measurement altitude: 0.0 km. (http://cprm.acd.ucar.edu/Models/TUV/Interactive_TUV/)

Solar elevation angle [°]	Ratio of shadow length to height of a vertical rod	UV Index
37	1.33	3
42	1.11	4
45	1.00	4.6
47	0.93	5
52	0.78	6
56	0.67	7
61	0.55	8
66	0.45	9

Table A3-1 allows for a simple estimate of the UV Index if there are no clouds in the sky. Divide the length of the shadow of a vertical rod (or person) by the rod's (or person's) height. In the second column of Table A3-1, look for the value closest to the calculated ratio. Then move across one column to the right for the UV Index based on the given solar elevation angle. If the shadow length to rod length ratio is less than 1.3, the WHO recommends (Naldi et al. 2000) using protective measures for the estimated UV Index. If the shadow is shorter than the height of the rod (ratio ≤ 1.0), measures to protect against the sun must be implemented.

Calculation note: This calculation should enable a rough estimate of the current UV Index when in a given location so as to be able to take any necessary measures to protect against the sun as per the WHO recommendation. The UV Index in Table A3-1 calculated from the solar zenith angle using the Quick TUV Calculator applies to average latitudes on Earth (Central Europe), clear skies, sea level, normal albedo and a comparably low level of ozone in the atmosphere. This is on the safe side in terms of ozone content, i.e. higher UV Index levels are generally calculated than with a higher ozone content. The actual UV Index may be higher than the levels provided in Table A3-1 when at high altitudes, e.g. mountains. Table A3-1 should not be used for locations near to the equator. The relationship between solar elevation and UV Index also does not apply if there is a lot of cloud cover.

Annex 4

Medication used in Germany in 2005 for which photosensitisation reports are available[*1]

From: Schauder, S., Phototoxic reactions in the skin triggered by medication. Deutsches Ärzteblatt, 2005. 102: p. 2,314-2,319.

Diuretics
Hydrochlorothiazide, Furosemide
Bendroflumethiazide
Amiloride, etacrynic acid
Triamterene, Spironolactone
Xipamide[*2]

Non-steroidal anti-inflammatory drugs (NSAIDs)
Naproxen, Ketoprofen
Tiaprofenic acid
Piroxicam
Diclofenac
Phenylbutazone, mefenaminic acid
Indometacin, ibuprofen

Antimicrobial substances
Sulfamethoxazole/*Trimethoprim*, Sulfasalazine
Ciprofloxacin, enoxacin, lomefloxacin
Ofloxacin, norfloxacin
Oxytetracycline, tetracycline
Doxycycline, minocycline
Isoniazid
Gentamicin
Griseofulvin, nitrofurantoin

Antimalarial substances
Chloroquine
Quinine, pyrimethamine
Mefloquine
Hydroxychloroquine

Antipsychotics
Chlorpromazine
Thioridazine
Promethazine
Chlorprothixene
Perazine, fluphenazine, promazine
Haloperidol

Antidepressants
Amitriptyline, trimipramine
Nortriptyline, desipramine
Imipramine, Doxepin
Clomipramine[*2]

Substances with cardiovascular side effects
Amiodarone, nifedipine
Quinidine, captopril, enalapril
Fosinopril, ramipril, disopyramide
Hydralazine
Simvastatin

Antiepileptics
Carbamazepine, lamotrigine
Phenobarbital, phenytoin
Topiramate[*2], valproic acid[*2]

Antihistamines
Cyproheptadine
Diphenhydramine
Loratadine

Cytotoxic substances
Fluorouracil, vinblastine
Dacarbazine, procarbazine
Methotrexate
Azathioprine

Hormones
Corticosteroids, oestrogen, progesterone, spironolactone

Systemic dermatitis medication
Isotretinoin, *methoxsalen*
(= 8-Methoxypsoralen)

Other
Gold salts, Hematoporphyrin

[*1]Listed due to their therapeutic active group and generic name; medications within an active group are listed in order of the number of reports according to which they trigger photosensitisation. Medications with roughtly the same number of reports as one another are listed in the same row.
Active agents in *italics* also trigger photoallergic reactions.

[*2]Denotes medications that do not appear in the list provided by Moore (2002), but which meet the given criteria and are available on the German market.

The above list of around 90 medications approved for sale in Germany for which there are a number of reports on their photosensitising (phototoxic and photoallergic) effects are based on data from:

Schauder S and Ippen H 1988 Photosensitivity. In: Manuale allergologicum V, Fuchs E and Schulz KH (eds), Dustri-Verlag, Deisenhofen 1988, 1-30

Malone et al. 2003 Malone PM, Melville M, Staff DRE. Topical Drugs Photosensitivity, Greenwood Village: Micromedix, Colorado, 2003

Moore 2002 Moore DE. Drug-induced cutaneous photosensitivity: incidence, mechanism, prevention and management. Drug Saf. 2002;25:345-372