

# **Dermatest GmbH: Sun Protection: Dermatological and Cosmetical Aspects**

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## **Abstract:**

Consumers have become aware of the dangers caused by unprotected sun exposure. There is an increasing risk of skin cancer and premature ageing. Therefore sun protection is widely advised since years. Sunscreen is the largest sector of the sun care market and higher SPF's (20+) are demanded more and more, always referring to the "healthy tanned" body-ideal. - UV-radiation, especially UVB, triggers the vitamin D synthesis and in low doses supports the immune system, but in increasing doses suppresses the immune system by suppressing the antigenic presenting cells in the spleen. The eyes can be injured by the radiation, producing inflammation of the cornea, degeneration of the retina and cataracts which can lead to blindness. The skin diseases caused by UV-radiation are not only skin cancers (malignant melanoma, basaloma and squamous cell carcinoma), but also increasing numbers of polymorphic light eruption and sun allergies or allergies occurring after the use of sun cosmetics. One of the most important cosmetic aspects occurring after years of sun exposition is the presenility of skin. Deep wrinkles and lines occurring before the biological age is reached. Often unprotected parts of the skin are characterised by darker lentigines (lentigines seniles) decreasing elasticity and reduced water-binding capacity. - Two main types of sun-protecting strategies - chemical and physical substances - are used in sun preparations. A lot of chemical effects of these components are yet unknown, esp. the long-term effects on human health. SPF-determinations are done in vivo according to the COLIPA method: the erythema doses of UVB in the skin of test persons is measured. The determination of UVA – according to scientific standards - is yet impossible on human skin. It is possible only as an in vitro reaction and not reproducible. There are several methods to determine the SPF in vitro but none of these can be used as a standard methods till today. - Sun lotions are good for preventing acute sunburns and, were hoped, to reduce melanoma incidence and chronic sun damages. But the misinterpreted fact is that sun factor use or not, not even the most expensive sun lotion can prevent the early ageing of the skin. Still, the cumulative dose of UV-radiation is the reason for chronic skin damages. Use of sun protection lotions leads to prolonged sunexposure and this is followed by an increased amount of UV-radiation which is not filtered by the sun lotion and suppression of the immune system. Therefore the early ageing and other chronic sun damages can be expected in a great extent within the next 20 years.

## **1.0. Introduction**

### **1.1. The psychology of sun protection**

A suntan has been a status symbol among Caucasians since the industrial revolution. Before that time, pale skin was fashionable because it indicated wealth and no need to work outdoors. With industrialization, however, the status of tanned skin reversed. It was now considered a sign of abundance of leisure time to spend outdoors. Suntanning as a fashion statement began in the 1940s promoted by Coco Chanel, the French fashion designer. The suntan's association with health began in the early 1900s when a treatment known as "heliotherapy" came into vogue. Though mostly discredited in the 1940s and 1950s, the belief that sun exposure was an cure-all persisted. The popularity of suntanning as a symbol of health, wealth and fashion has risen almost unabated since the end of World War II. Only recently, spurred by a rapid rise in skin cancers and decline in the ozone layer, there has been an attempt to reverse this popularity. The campaign has been difficult – not so much in spreading information, but in affecting change in beliefs and behaviour. While the level of knowledge concerning skin protection and the dangers of skin cancer is considered high, many continue to believe that the risks are outweighed by the benefits of a suntan. Even for those who have a strong knowledge of the dangers, changes in behaviour are reluctant. A factor is the "optimistic bias" whereby an individual believes that something negative is less likely to happen to them than to their peers. There is a marked difference in health beliefs, behaviour and choice of sun protection among groups segregated by occupation, age and/or gender. For example younger people will more likely rely on a sunscreen for sun protection while older people prefer to cover-up with clothing. Such differences are important to consider when designing education campaigns. Females are more likely to be concerned with health and healthful behaviour and engage in more preventative medical behaviours than males. Females also generally have a higher level of knowledge of effects of sun on the skin. However, this does neither seem to have a marked change in their desire for a suntan nor their belief that a tan is healthy. It can be stated, that although at present the public has been found to have a high level of knowledge on the dangers of over-exposure to the sun many people still desire a suntan. Some of the major barriers in this area are that having a suntan is seen as being both healthy and attractive, and it is not "cool" to cover-up.

### **1.2. UV-radiation**

It is a well known fact that life on earth is impossible without UV-radiation. But what is UV-radiation, how can we explain the controversial effects that sun light can induce?

Sun light consists of radiation with differing wavelength: UV-radiation, infra-red radiation and visible radiation.

Concerning skin damages and sun protection, the UV-radiation is important. UV-radiation consists of UVA (320-400 nm), UVB (280-320 nm) and UVC (190-280 nm). UVC is the most dangerous radiation, but it is mostly absorbed by the ozone layer within the stratosphere and does not reach the earth's surface

Three billion years ago, when the ozone layer was not thick enough, there was no life on earth. Only life under water was possible. With the strengthening of the ozone layer, until now, only UVB- and UVA-radiation is observed on earth, and life on the surface became possible.

In the human body high doses of UV-radiation produce vasodilatation and erythema. The minimal dose of radiation causing an erythema is named minimal erythemal dose (MED). The MED is no fixed value, but it depends on the individual, its skin colour, age and the radiated limb and the season. In the skin the melanocytes begin to produce melanin, according to the dose of UV-radiation. UVA-radiation is known to produce weaker erythema and pigmentation than UVB.

Deoxyribonucleic acid is the main content of the genes, the four bases (thymine, guanine, cytosine and adenine) have conjugated double-bondings and can absorb UV-radiation and transform it in a higher energy level.

These double bondings are sensitive to UV-radiation with wavelength under 320 nm and convert to hydrates, dimerisations and additions. We know that it is the thymine dimerisation that produces damages in the DNA, necroses and cell mutation.

## **2.0. Effects of UV-radiation and the prognosis for the skin**

### **2.1. Skin Types**

Since the unintentional experiment of the Britain Empire in 1786, when prisoners were deported to Australia, today's experiences concerning the skin-type and the individual's predisposition to skin cancer has changed. The white Australians with British origins have the highest susceptibility to skin cancer in the world, it is not only much higher than in the British Islands, but also much higher than in the Australian natives. So it became clear, that the colour of skin has a important influence on the development of skin cancer, as has the age of exposition to dangerous UV-radiation.

Individuals are classified with skin-types and so prognosis can be given about the dangerous effects of UV-radiation.

Type 1	always burns, never tans
Type 2	usually burns, tans with difficulty
Type 3	sometimes burns, sometimes tans
Type 4	burns minimally, always tans (Asians, native Americans, Latin Americans)
Type 5	rarely burns, tans profusely (ligh-complexed African-Americans, east Indians) may need protection with intense exposure
Type 6	never burns, deeply tans (dark-skinned African Americans) may need protection with intense exposure

### **2.2. Acute and chronic damages of the skin**

The energy level of UVA is about 100 times greater than that of UVB, but UVB is much more able to cause acute skin damages. On the other side, UVA is not able to provoke pigmentation while short-time radiation, but is known to cause presenile skin-ageing and is suspected to be in direct connection with the formation of melanoma. Because of its wavelength, UVA reaches the corium and the connective tissue and gives rise to denaturation of elastine and affects the skin ageing.

UVB-radiation is rarely able to come through the skin and has only effects on the epidermis. So it is responsible for acute skin damages such as sunburn and polymorphic light eruption.

UVB is known to induce the vitamin D synthesis with a wavelength between 290 and 315 nm, but even smallest amounts of UVB-radiation are sufficient.

But much more important is the fact that UVB-radiation is indirectly inducing the tumorprogression via induction of vascular endothelial growth factor. This means UVB is stimulating new vessels in the skin, and if this happens under "sleeping" tumors, like squamous cell carcinomas these may arise to spreading the tumor-cells, which leads to growth.

Furthermore, there are negative effects on the cornea and retina described, coming up to snowblindness and cataracts.

Today's sun screens should not only protect from sunburn and genetical harm but also from neovascularisation.

It was assumed that wavelength under 320 nm (UVB-radiation), is to blame for DNA damages, which means the provocation of skin cancer.

But the carcinogenic potential of UVA (320-360nm) was previously overlooked. In human, exposure to artificial UVA has been shown to lead to an approximately 2 fold increase in melanoma.

The very high exposure to UVA-rays, either due to long sun exposure in the presence of UVB-sunscreens or due to pure UVA-lamp exposure, is likely to produce, in humans, DNA lesions and mutations in the skin.

### 2.3. Immunosuppression and UV-radiation

Immunocompromised individuals are at increased risk of infectious diseases and malignancies. Renal transplant patients on chronic immunosuppressive therapy for example have an increased frequency of skin cancer. Squamous cell carcinomas directly relate to sun exposure in these patients.

Pyrimidine dimer repair mechanisms play a central role in preventing carcinogenesis. Xeroderma pigmentosum (XP) is a rare, genetic condition involving a defect in this nucleotide excisional DNA repair system. A reduced capacity to excise UVB-induced pyrimidine dimers leaves these patients at a 2000-fold risk of developing melanoma, and a 4800-fold chance of forming squamous and basal cell carcinomas by the age of 20.

A study by Ley et al. supports the evidence for an inverse correlation between UVB-repair mechanisms and development of cutaneous melanoma. The authors conclude that UVB can function as a complete carcinogen for the initiation and promotion of a malignant melanoma, specifically via DNA pyrimidine dimer formation. (Inference from this photoreactive repair model might help explain why higher melanoma risk is related more to episodic compared to prolonged exposures.)

UVB has been shown to inhibit cell-mediated immunity (delayed-type hypersensitivity) and immunosurveillance of transformed epidermal cells. Several health consequences of this immunosuppression are recognized. HSV-I and -II are reactivated by ultraviolet exposure, in a dose-response fashion. UVB can affect the course of infectious diseases of the skin such as onchocerciasis and dermatophytosis, and diseases where the skin is a portal of entry such as Leishmaniasis. In sandfly-borne Leishmaniasis, parasite replication occurs in the dermis or epidermis, and is followed by hematologic dissemination and diffuse incurable disease. In a murine model of cutaneous Leishmaniasis, Giannini found that low doses of UVB-irradiation increases the likelihood of systemic spread or reactivation of the protozoa. Additionally, UVB does not diminish the viability of parasites in the skin and immunity to subsequent inoculations absent among animals irradiated with UVB during the first infection. These findings are consistent with diminished epidermal defense mechanisms and suggestive of reduced systemic immunity.

Suppression of tumor cell rejection has been specifically linked to UVB, while assays using UVA show no such suppression. Moreover, in animal transplant studies, Daynes et al. found that UVB-induced tumors fail to grow in normal recipient mice, but continue to progress when transplanted into irradiated mice.

### 2.4. Skin cancer

Nonmelanomatous skin cancer and superficial spreading melanoma are correlated with sunlight exposure. In the U.S., malignant melanoma incidence among the white male population shows the steepest rise of all neoplasms, and from 1973 to 1988 increased by 86.3%.

While there is no direct relationship between cumulative ultraviolet exposure and melanoma, excessive UV-exposure during childhood is associated with incidence. For every 1% decline in the stratospheric ozone layer, a 3-5% rise in squamous cell carcinoma, 2-3% rise in basal cell carcinoma, and 1-2% increase in melanoma are expected. In 1998 one million Americans developed skin cancer. About 9,200 people died of skin cancer this year, 7,300 from melanoma alone.

As shown above, skin cancer is caused by UV-radiation. There is no doubt, that the amount of UV-radiation is closely connected to the appearance and frequency of skin cancer.

Reasons for this are:

- People with dark skin are rarely troubled by skin cancer, additionally, not even areas exposed to light are more frequent infested.
- People from northern races more often get skin cancers.
- The white-skinned citizens of Australia show skin cancer much more often than white-skinned people e.g. in western Europe. Australia shows 50% of all skin cancers in the world.
- 95% of all skin cancers are found at light-exposed skin areas (face, neck)
- White-skinned people spending much time outside show skin cancer much more often than e.g. office worker
- In Asia the white skin is still desired, therefore the sunbath is not really practised and skin cancer is scarcely appearing.

As known, there are not only very malignant skin cancers such as melanoma, but also local growing cancers of the skin, growing slowly, like the basaloma and the squamous cell carcinoma.

- Basalomas develop from the basal layer of the epidermis and grow very slowly. Metastases never occur. Basalomas grow only primary on light exposed skin, they do not develop on precancerous skin. A healing of 97% can be expected. Basalomas often occur with elderly patients.

- Squamous cell carcinoma develop from the spinous layer of the epidermis. They grow faster than basaloma, generalisation is possible, but with timely excision, complete recovering quotes of 95% are reached. They often develop on precancerous skin areas on the elderly, often exposed skin.
- Melanoma may occur without the influence of sun light. But often they occur after skin damage caused by sunlight. Mostly, melanoma spread horizontally, while no generalisation occurs, but afterwards rapid deep-growths develops and metastases appear. The melanoma is the most malicious cancer known. Excessive UV-exposition in the age up to 15 years, which goes along with one or more severe sunburns, is the most often cause for SSM (superficially spreading melanoma) and NM (nodular melanoma) between the 20<sup>th</sup> to 40<sup>th</sup> year of live. Another important factor is the number of pigmented lesions and nevi. People with a great number of nevi have a great risk of developing melanomas. A special place have the congenital naves, which have, growing over the size of 1.5 centimetres, a 6% chance to become malignant after the 18<sup>th</sup> year of life.

## **3.0. Conclusions**

### **3.1 Sunscreens and UV-radiation**

There are two basic types of sunscreen options available: Chemical sunscreens act by absorbing ultraviolet (UV) light, and physical sunscreens reflect or scatter light in both the visible and UV-spectrum. Effectiveness of sunscreens depends upon their UV-absorption, their concentration, formulation, and ability to withstand swimming or sweating.

Within the last years, the body has just been protected from the UVB-radiation, which is responsible for the sunburns and acute sun damages. The sun protection factors were determined by the protection against UVB-radiation, UVA-protection was not estimated.

Sunscreens are available for 60 years now. Safety and effectiveness are dependent on quantity and kind of the formulation. Physical sunscreen is made from nonorganic pigments like zinc oxide or titanium dioxide, which reflects or diffuses the radiation. In micronized form (size: 10-100nm) the particles absorb the UV-radiation. The concentration of physical filters is not limited in Europe, as are the chemical filters. The extinction and effectiveness of titanium dioxide microparticles sized under 100 nm reaches in the UVB-wavelength, both substances mixed have a perfect broad spectrum effect. A good effect can be reached with different particle sizes of either substance.

The substances even in smallest particles do not penetrate into the skin and are chemically inert, so do not produce allergies, and work immediately after application.

Though, there are some negative aspects concerning physical filters. In pigmented form it whitens the skin and is not very effective against UV-radiation. In micro particles it tends to agglomerate and aggregate due to electrostatic effects, which means a enormous loss in efficacy. Therefore the micropigments have to be coated and kept in dispersion, which is still a great challenge for the cosmetic industry.

In chemical UV-filters the UV-radiation activates the electrons from the passive in an short-time active condition. With returning into the stable condition, the energy is emitted as warmth or fluorescent irradiation.

If the sun protecting factors are not photo-stable, secondary photo-reaction like photolysis, photo-isomerisation and photodimerisation take place. UV-filters were developed to form harmful UV-radiation in harmless long-wave radiation like warmth.

Typical chemical UVB-filters are octyl triazone, urocanic acid, octyl methoxycinnamate, methylbenzylidene camphor, 3-benzylidene sulfonic acid and PABA.

Solar radiation consists of 5% UVB- and 95% UVA-radiation, UVB-radiation directly harms the DNA, UVA-radiation just operates indirectly with producing free radicals and reactive oxygen. Because of this, newer sun protection contains UVA-filters, such as benzophenone-3, benzophenone-4 butyl methoxydibenzoylmethane and terephthalylidene sulfonic acid. They all have deficiencies, e.g. work in UVB and UVA, but in UVA just in restricted borders, others are not photostable or likely to induce allergies.

Additionally, to diminish the destructive effects of UV-induced oxygen radicals, antioxidants are added to sunscreens, but these substances are mostly used to protect the substances from oxidation. These effects can be observed in vitro using vitamins (A, E, C), minerals (selenium, zinc) or reduced glutathion. It was tried to use the antioxidants as sunscreens, but a SPF over 5 can not be reached. Additionally, there are problems with the solubility and photostability of the above mentioned antioxidants.

Increasingly, sun protection becomes more and more used to the sunbathing population.

### **3.2. SPF and UVB**

Since now, the determination of the SFP and so the protection of UVB-radiation, is done by radiating the backs of probands and estimating the minimal erythral dose by producing a sunburn. In 1956 R. Schulze defined the sun protection factor as the ratio of the Minimal Erythral Dose (MED) of protected versus unprotected skin visually assessed about 24 hours after irradiation. This definition of the SPF has gained a world-wide acceptance over a period of 25 years now.

Based on today's knowledge, instead of the term "Sun Protection Factor" scientists prefer the term "Erythema Protection Factor" or "Sunburn Protection Factor" because only the 24 hour response is considered in the SPF determination and calculation. Suberythemally sunlight induced effects and chronic effects are probably not adequately considered (e.g. immunosuppression, skin ageing, skin cancer).

There are different definitions in the different countries to estimate the erythema threshold radiation time. The countries define the SPF according to the regulations of the appropriate office. In Europe, the COLIPA recommendation determines the following: To determine the SPF, 10-20 probands, skin types I-III mixed, have to be tested at the back in an area bigger than 35 cm<sup>2</sup>. The product applied has to be a quantity of 2mg  $\pm$  0,04 mg/cm<sup>2</sup> and has to be on the skin for 15 minutes before radiation. The reading of the MED has to be done after 20  $\pm$  4 hours visually and with colorimeter and the erythema threshold radiation time is 20-180 seconds.

There are a few methods available to determine the SPF "in vitro", but these are difficult to use and the results are not widely recognized.

### 3.3. SPF and UVA

The definition of UVA-protection is far from harmonisation. It is not only the testing, but also the labelling, that is not standardized.

There are still unanswered questions concerning UVA-protection, e.g.:

- What level of UVA-protection is adequate?
- Which is the most relevant and measurable skin response to indicate UVA-induced skin damages?
- Which absorption profile should an ideal sun care product have?
- Which UVB/UVA-ratio should be considered to meet different irradiation situations?

In 1994 the German health authorities invited experts to discuss UVA-protection issues. It has been concluded that all sun-protection products should have "adequate" UVA-protection, but a clear definition of what adequate means, is not given by the lack of scientific background.

Test methods used until now are the *in-vitro*

- Australian Standard AS,
- the Boots Star Rating system,
- the Broad Spectrum Rating
- and the APP-Method / UVA-Protection Percentage

These methods are all based on transmission/absorption measurements. They differ in details and the methods of calculation. The results are usually used as indicator for UVA-protection or broadness of the absorption characteristic.

*In-vivo* methods are:

- IDP method (Immediate Pigment Darkening)
- PPD method (Persistent Pigment Darkening)
- APF method (Erythema UVA- Protection Factor)
- PPF method (Phototoxic Protection Factor)

The *in-vivo* methods are based on the determination of UVA-induced skin responses (e.g. pigmentation or erythema) followed by a calculation of UVA-protection factors analogue to the SPF. The results obtained diverge significantly between methods, but may also lead to different efficacy rankings.

As the *in-vivo* method cannot measure directly the target skin responses (ageing, immunosuppression, cancer) the results can only be seen as indicative.

Therefore, the value and necessity of *in-vivo* UVA-testing has been questioned by several scientists, especially the additional benefit of the *in-vitro* measurements.

For the customer, having just become familiar with the SPF system, the existing UVA-labelling ("contains UVA-protection", "Broad-Spectrum protection" "PA+PA+++" and "B20A6" or "SPF60-IPD 55-PPD 12") is more confusing than informative.

A simple and easy-to-understand system is to be demanded, like the *in-vitro* determination of "Broad-Spectrum" criterion. An *in-vivo* Broad-Spectrum is reached by Octyl Methoxycinnamate and Zinc Oxide, and an even greater "Full-Spectrum" protection is reached *in-vivo* with the use of Octocrylene and Parsol1789.

### 3.4 Deficiencies and pitfalls in using sunscreens

Erythema is a measurable biological endpoint of UV-exposure and is used to determine sunscreen effectiveness. The "Sun Protection Factor"(SPF) is rated by the length of time required to elicit a "Minimal Erythematous Dose" (MED). Sunscreens have also been shown to partially block the immunosuppressive response, however, in no correlation with their SPF designations. While histologic signs of erythema are blocked by sunscreen application, the protective agents may fail to prevent UVB-induced tumor susceptibility. One study suggests that the dose of UVB necessary to increase animal susceptibility to cancer by immunosuppression is 10-fold less than the amount needed for overt carcinogenesis by molecular damage.

Exposure to UVB in tanning salons can cause subsequent failure to respond to antigen challenge, and suppress enzyme repair of DNA lesions. A major public health concern is that by reducing the erythematous reaction, sunscreens may deceive people into remaining exposed to solar rays and their immunosuppressive effect for longer periods. Measuring DNA nucleotide excisional repair may be capable of predicting subpopulations at risk of developing skin cancer. An assay has been developed to measure this mechanism, which is found to be significantly reduced in skin cancer cases. Xeroderma pigmentosum patients have provided a model for testing this assay, for the defect in their repair mechanism phenotypically varies considerably within this subpopulation.

### **3.5 Sun protection factor, the higher, the better?**

Since the 50s and 60s, sun protecting products are available, first with a SPF of 4 and 5 considered to be a high protection, then, beginning in the 80s SPF's up to 20 were introduced. Now, all major producers offer SPF's up to 30, since 1998 the Japanese market has been offering a product with a SPF of 123, an end of this SPF-inflation is not in sight.

But what is the sense of this SPF, reaching so high? The maximum necessary SPF results first of all from the real outdoor situation, how much erythematous radiation can reach the skin during one day of sunshine? The available data shows up to 32 MED/day (maximum 6 MED/h at high noon) in case of sensitive skin. In order to become fully effective, this would require a continuous exposure to the sun for the whole day in an unchanged position. In practice, the realistic number of MEDs would be clearly lower. Considering this, an SPF, of 15 to 20 would be sufficient erythematous protection as long as the product is being used under same conditions as in the Sun Protection Factor test. And there is the problem, the test-conditions are normally not the ones outdoors, and so the labelled SPF is higher than the real outdoor one.

The SPF represents the mean of a biological test with typically large deviations. Therefore it can be expected, that about 50% of all consumers are overprotected and the other about 50% are underprotected. Individual protection might deviate considerably from the mean value. A product with a mean SPF of 30 can, even under standardised test conditions, show individual protection factors between 15 and 45.

Another considerable point is the thickness of the applied amount of sunscreen. In tests 2 mg/cm<sup>2</sup> are applied, under realistic conditions it varies between 0,5 mg/cm<sup>2</sup> and 3 mg/cm<sup>2</sup>, depending on the users habit. Consequently, the SPF varies in connection to the applied amount.

Considering the points discussed above, it is clear that the realistic outdoor SPF is often much lower than declared on the label. So, high SPF values between 20 and 30 are well justified, but require a careful interpretation by the consumer. The real exposure time has to be multiplied with the SPF, which bears mistakes for the consumer, because higher SPF has more deviation than lower SPF. Furthermore, the self-protection time has to be estimated correctly by the consumer, otherwise, multiplication by the user increase the mistake.

Another important fact is, that the function estimating the percentage of UV-reduction according to the SPF runs asymptotic, which means only minor increases in reduction with high SPF's. Due to the characteristics of the function, very high protection factors are subject to strong variations even in the case of only slightly different test conditions. So a product with an SPF of 30 (97,6 % elimination of erythematous active radiation) can have a mean value of 20 (95%) or even 40 (97,5%).

Because of these facts, a labelling with 4 SPF groups, defining low (2-5), moderate (6-11), high (12-19) and very high (20 and higher) protection makes sense.

### **3.5 Sun protection - practical guidelines**

No matter what the weather is like, or what you do outside, protect your skin from ultraviolet radiation. Make sure your children are protected as well: would you let a child go out in the rain without a raincoat or an umbrella? Would you let a child out to play in the snow without gloves or boots? Would you let your child go out in the sun without sunscreens and a hat?

1. Wear closely-woven covering clothing (long sleeves, long trousers or a long skirt).
2. Look out for clothing with a label stating its UPF (protection factor) is greater than 40. Tests have shown sun clothing blocks ultraviolet radiation very effectively, much better than sunscreens.
3. Put on a broad-brimmed hat.
4. Try to keep in the shade.
5. Apply a sunblock lightly to all uncovered skin before you go out.
6. Apply sunscreens 30 minutes before sun exposure, and reapply after swimming or sweating.
7. Do not use chemical sunscreen products on children less than 6 months of age. Physical protection from the sun is the best recommendation for this age group.
8. Discontinue use of the product, if a rash appears.
9. Products with PABA (see ingredient classification) may permanently stain clothing yellow; avoid PABA products if sensitive to benzocaine, procaine, sulfonamides, thiazides or PABA.
10. Always wear eye protection (sun glasses) as UV-light can damage your cornea.
11. Remember certain maintenance medications can cause photosensitivity.

12. be aware of ground surfaces. Ground surfaces such as sand, cement and white painted surfaces reflect the UV-radiation. In the water, ultraviolet light penetrates three feet deep.

13. Are you at risk: fair skin? light hair? tendency to freckle? tendency to burn easily? a family history of the disease? chronic sun exposure throughout life? blistering sunburns as a child or adolescent?

There is really no safe way to tan, the best skin tone is our natural skin tone!

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