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Sunburn and what happens at lower UV dosages?

If our skin receives UV light with the first ray damaging effects will happen to various cellular and extracellular constituents. For keratinocytes, after »enough« cells have been »disabled« by a certain UV-induced damage level and undergo the programmed cellular death, a distinct buildup of inflammatory cytokines occurs. The thus initiated inflammation process causes blood vessels to widen, and this turns the skin red. A sunburn becomes manifest macroscopically not only in skin reddening but also in a growing level of pain on the affected spot. The corresponding energy dosage necessary to induce this skin answer is called one minimal erythemal dosage (1 MED). Dosages below this value usually do not induce any visible skin redness, with the exception they are received subsequently for a longer period of days. If a single UV

Introduction

After 3 decades of intense public campaigns sun protection is in the mind of almost every consumer in particular if it concerns holiday or sport activities. Does this mean that industry stakeholders and consumers can lean back? Surely not. There is still a lot more work and education on safe sun exposure to do, in particular because only one country so far, Australia (1), has achieved a beginning decline of the melanoma incidence. Holidays and sport themes are often communicated together with sun protection messages – with a compelling reason: a significant portion of the annual UV exposure dose is collected in these episodes. But to draw the whole picture: more than half of the UV dosage is not linked to vacation times but rather to day by day activities such as gardening, grill parties or lunch breaks. People have significantly lower awareness for sun protection in those situations which might result in occasional localized sunburn, e.g. the facial area. In holiday episodes most consumers have learnt how to avoid sunburn of larger skin areas; however, quite frequently they still have localized weak sunburns on super-exposed skin parts or areas which had been treated only sparsely with sunscreen. Sunburn does cause pain and significant discomfort and therefore represents a strong driver to apply sun protection products. Most people consider the avoidance of sunburn as a success signal for their personal protection strategy and feel on the save side if they succeeded in doing so. But is this really true? Are dosages below the erythemal dose level (1 MED) well compensated in our body? This article looks at such dosages in a few dimensions, e.g. DNA damage as mutagenic marker, oxidative stress as anti-aging initiator and immunosuppression as a cancer enhancement marker. It also gives recommendations based on the findings to protect people of UV radiation beyond sunburn.

dosage does not cause sunburn it is called suberythemal.
The energy or duration of UV radiation necessary to induce sunburn is not constant and can vary a lot from person to person mainly depending on skin type. Sunburn is also wavelength dependent as UVB is more damaging than UVA. Being in a holiday location in the tropics makes our skin burn faster, not only because of a general higher radiation intensity but also because at that latitude sunlight contains more UVB than e.g. in Europe **(Table 1)**. But it is important to keep in mind that sunburn is not 100% UVB based. There is also an energy level or time point at which pure UVA radiation would cause erythema as well if UVB were filtered out **(Table 2)**. This situation may evolve with sunscreens providing no or little UVA protection.
When reporting on human trials involving UV radiation, the wavelength and individual sensitivity dependency of the MED is less suitable and may lead to confusion in data comparisons. Additional parameters would have to be measured and reported to make experiments comparable. The Standard Erythemal Dosage (SED) has therefore been introduced (2) to avoid or define dependencies. For a reference example: 1 MED for a skin type 2 person is about 2 SED. **Table 2** (3-6) lists average energy values to induce 1 MED in a skin type 2 person of some commonly used light sources, note the much higher number for UVA to induce sunburn.

	UVB : UVA
Natural day light, Europe noon, noon time, June	1 : 25
Natural day light, tropics noon	1 : 18
Solar Simulated Radiation (SSR, artificial light for SPF measurements)	1 : 10

Table 1 Light sources: Proportional energy ratios between UVB and UVA light

Molecular components related to sunburn and evolvement of UV damage until sunburn

It is widely accepted that sunburn is linked with DNA damage in the cellular nucleus, followed by apoptosis of the keratinocytes and in consequence induction of an inflammation, which leads finally to skin redness. However not all connections in this cascade of events are fully understood. The most prominent primary target for UV interaction with skin is DNA. UVB light gets physically absorbed e.g. by the thymine chromophore. The epidermis is densely packed with cells and therefore UVB does not penetrate much deeper than the basal layer due to absorption and some back scattering. The absorbed energy allows the base pairs of the DNA to undergo a variety of reactions which lead to alterations in the structure of DNA. The genetic code at that structurally altered location is not correctly readable anymore and thus could give rise to mutations. Two neighboring thymines in the DNA strand are prone to a particular mutation: they form in a photo-catalyzed reaction a dimer, called cyclobutane pyrimidine dimer (CPD) or more specific, thymine dimer (TT). Fortunately, our cells have evolved several DNA repair mechanisms such as the nucleotide excision repair (NER). While it is well known that UVB is the major source of DNA damage, only recently it has been shown that UVA could also induce direct DNA damage particularly in deeper layers of the skin (7). UVA light interacts also with DNA in more indirect mechanism through formation of excited oxygen species which finally might lead to the formation of CPDs, 8-oxoguanine and strand breaks.
p53, a key protein, orchestrates the repair process. It also initiates apoptosis, the programmed cell death to protect against mutagenesis. Such affected cells are called sunburn cells (SBCs). Damage on p53 itself and persisting mutations of that protein are very critical as they can represent one major route to carcinogenesis. The complex repair process, particularly for the CPDs, takes place within hours and days and is therefore generally much slower than the time to produce the damage under solar radiation.
Different molecular biological markers are known to track the buildup of this damage and repair process, which happens unnoticed by the consumer until the skin shows visible signs of redness due to exposure to erythemal UV dosages (> 1 MED). Indicative damage markers after UV radiation can be: relative number of SBCs and CPDs. Also monitoring of p53 by immunofluorescence can be helpful, as it indicates the cells' begin of repair. In addition to molecular biological methods requiring biopsies, measurement of the erythemal index represents a sensitive technology to follow up the subsequent rise towards sunburn before it is really visible to the human eye.

Damages revealed before onset of sunburn after single and subsequent suberythemal UV Exposure

Fig. 1 shows the slow evolvement of skin redness by a series of subsequent suberythemal dosages (11 x 0.6 MED of SSR)

Type of Light	Energy necessary to induce 1 MED in a skin type II
Monochromatic UVB (300 nm) median	25 mJ/cm²
Monochromatic UVA (360 nm) median	32 J/cm²
Simulated Day Light (SDL)	15.2 J/cm²
Sun (295–400 nm)	~5–12 J/cm²
Solar Simulated Radiation (SSR) as used for SPF measurement	~2 J/cm²

Table 2 Energy values of certain light sources to induce 1 MED in a skin type 2 person

volunteers of skin type I and II were exposed to (8). At a value of about 80 skin redness gets visually perceivable. Over the whole period of 11 days there seems to be a gradually rising level of damage indicated by progressing skin redness **(blue line, Fig. 1)**. During the first 8 – 10 days this damage goes on unnoticed, only at the last day it seemed to have become finally manifest in sunburn appearance. A low level sunscreen (SPF of 7.5) was enough to suppress a major evolvement of skin redness, but - is this really an indication for the complete absence of DNA damage? We therefore investigated the CPD level with biopsies **(Fig. 2)**.

Fig. 1 also suggests that there seems to be not much damage for a single radiation period, e.g. at day 1. This aspect has been addressed by several authors (5, 8-10), who analyzed biopsies of skin type I, II or III volunteers after a single radiation of 0.5 MED with several light sources, including also pure UVA light (11). CPDs could already be detected in keratinocytes and melanocytes, as an indicator of DNA damage. Also p53 seems to be up-regulated, indicating the switch on of the repair signal in skin type II and III.

We therefore investigated the CPD level with biopsies. **Fig. 2** shows significantly elevated DNA damage levels after 5, 11 and 12 days of suberythemal radiation for the untreated area. This means the erythema index does not parallel the presence of CPDs. The DNA repair is already leading to a balance of newly incoming and already repaired damage. After the last radiation (day 12) the CPD value is going down, which is indicating successful repair. A broadspectrum sunscreen having only an SPF of 7.5 is able to cause a significant protection against DNA damage. However, the low protection level was not fully sufficient to suppress CPD formation completely compared to the non-radiated site, so a still small level of DNA damage persists.

Similar observations had also been made in a study with 9 times subsequent suberythemal UV radiations at 0.25, 0.5 and 0.75 MED (12), in which also the difference of SSR used for SPF testing and a simulated day light (SDL) had been compared. A radiation of only 0.25 MED of either SSR or SDL was enough to generate p53 upregulated cells. SBCs were present at a significant level already at only 0.25 MED SSR, while SDL needed 0.5 MED to generate a similar number.

UVA radiation is setting an aging process into motion

Visible signs of skin aging become evident as wrinkles, in particular around the so called »crow feet« area, but also an uneven skin tone (13) delivers information for a visional judgment on age. The desire to look attractive is high, being the main driver of the global demand for effective treatment concepts. A second strategy, which actually should be the primary one to fight signs of aging is to protect skin against their origin before aging signs appear or get worse. In particular UVA induces a multitude of effects which lead to visual skin aging phenomena. Most often UVA-induced changes start by UVA light generating oxidative

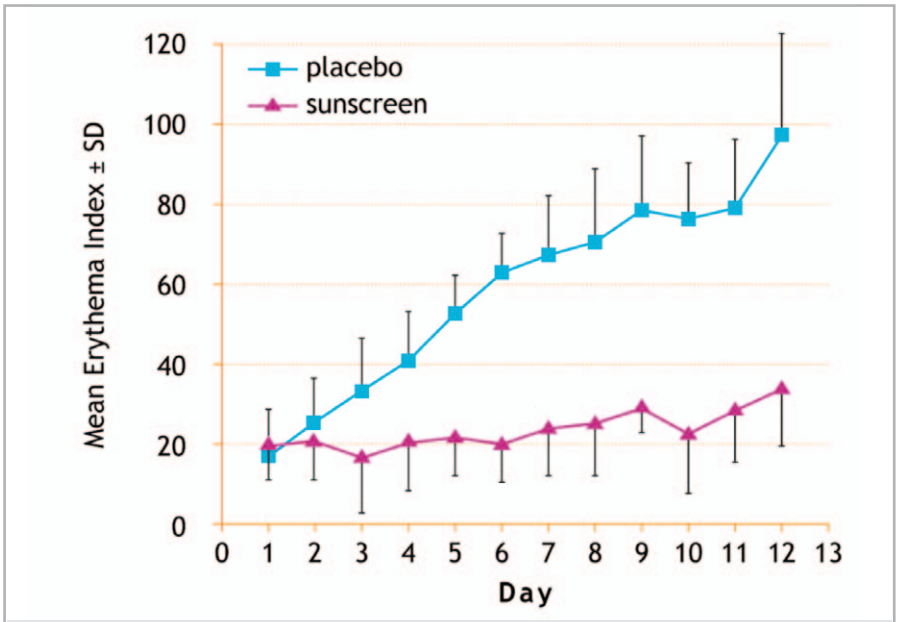

Fig. 1 Erythema index during 11 subsequent suberythemal dosages of 0.6 MED. The sunscreen used was broadspectrum and had an SPF of 7.5

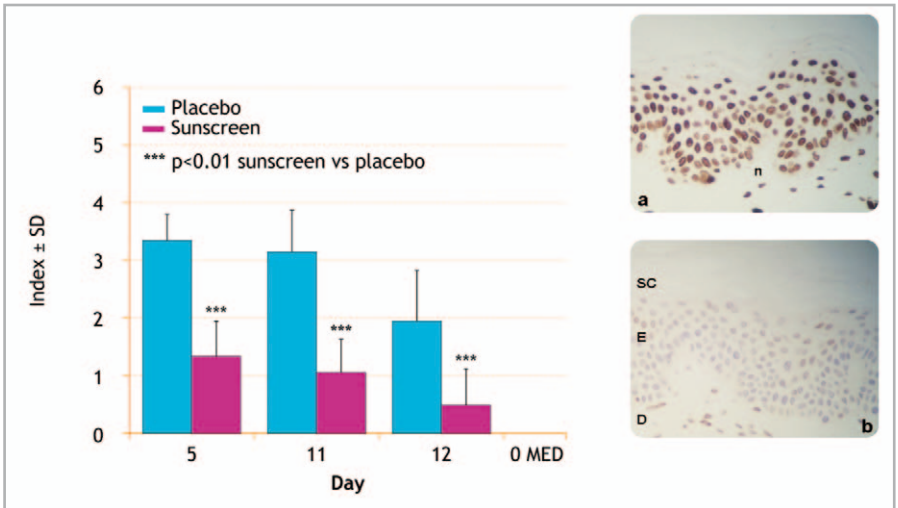

Fig. 2 Evolvement of CPDs with 11 subsequent suberythemal treatments with 0.6 MED. Pictures represent day 12, a (unprotected), b (sunscreen SPF 7.5)

stress in the tissue. An action spectrum has been measured for this. Multiplied with the solar radiation spectrum the resulting solar radical generation spectrum shows the strongest peak around 360 nm (14), which indicates that most oxidative stress caused by UV originates from UVA. Unfortunately UVA penetrates deep into the dermis and can there initiate partly irreversible damage of cell constituents and matrix proteins. Over the years modified proteins could build up as they are hard to metabolize and form e.g. the basis solar elastosis. The inhomogeneity of protein distribution causes also an uneven whiteness distribution of the skin. Considering that UVA is present much longer during the day time than UVB, sunscreens should therefore be all equipped with a sufficiently strong UVA protection. Day care formulas may even go over the recommended minimum guidelines to achieve an extra benefit in fighting the face changing effects of UVA.

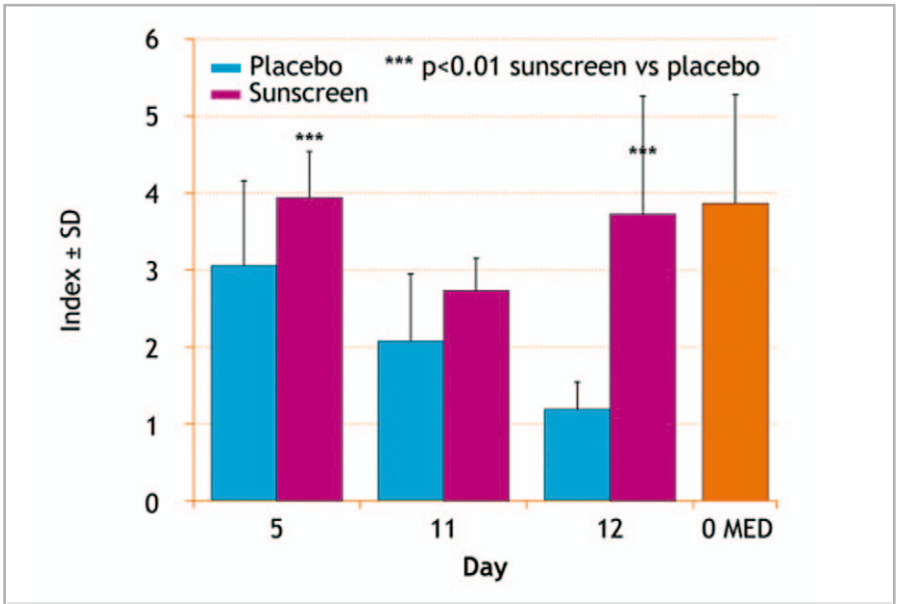

Fig. 4 Influence of 11 subsequent suberythemal radiations with 0.6 MED on the number of Langerhans cells

Suberythemal UV radiation leads to immunosuppression

Our skin has several lines of defense against deleterious UV effects. The skin's immune system can be seen as a last fortress in the fight against cancer formation. It detects mutated cells and destroys them. However, the immune system reacts very sensitive to UV stress. Recently an action spectrum for immunosuppression has been established (15). It shows two peaks, a smaller one at 300 nm and a huge one in the UVA region peaking at 370 nm. It has been shown that only 0.3 MED is enough to damage the immune response of the skin towards allergens (and in essence also towards mutated cells). The Mantoux reaction against tuberculin has been utilized to measure this effect (16) **(Fig. 3, blue bars)**. We were also able to see disappearance of Langerhans cells upon subsequent radiation of 0.6 MED (8) **(Fig. 4, blue bars)**. *Seité* (4) had observed an significant immunosuppressive effect already at 0.25 MED. A SPF 7.5 broadband sunscreen protected the Langerhans cells significantly against the suberythemal UV radiation **(Fig. 4, pink bars)**. Interestingly, it was found that topical Niacinamide, also known as Vitamin B3, almost completely preserved the immune status of the volunteers exposed to 0.3 to 1 MED SSR **(Fig. 3, pink bars)**.

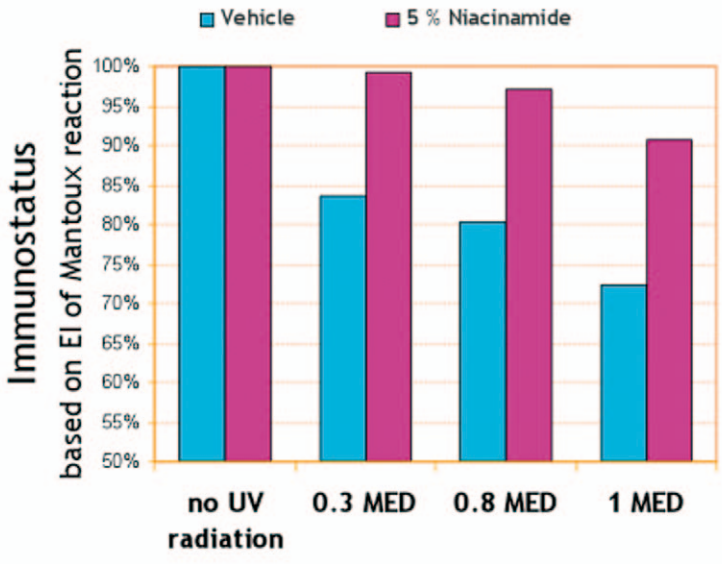

Fig. 3 Immunosuppressive effect of erythemal and suberythemal SSR. Protection can be achieved with Niacinamide (15)

Two typical UV exposure scenarios – is there a need for more protection?

1st Scenario – Shorter periods of exposure in summer

Many leisure activities including lunch breaks happen around and at the zenith of sun, where the UVB intensity is the highest. During lunch people are often facing the same angle towards the sun, so that there is little relief by changing exposure to other body areas. Some parts,

e.g. the nose tip or ears can actually be hit perpendicular by the sun, which means reception of the full UV dosage compared if the sun had a certain angle to the plain of the skin. The risk of getting sunburn varies of course with the skin type. Skin type I and II, both particularly sensitive, would have most likely no longer than 0.5 h under those conditions until they reach 1 MED and exhibited a slight sunburn. Skin Type III would have eventually about 15 - 30 minutes longer and Type IV would most likely have no burn symptoms during a lunch break.

Easily sunburn can be avoided during outdoor lunch or other shorter outdoor activities by utilizing a SPF 15 UVB based sunscreen, a formula type often realized in former generations of day care formulas. However, such a formulation principle would leave the door open for UVA based premature aging effects. In addition there could also be a UVA based sunburn. In solar radiation the UVB contribution is only 80-85 %. UVA light is clearly much less effective in causing sunburns, but on the other side it is 25 times more abundant. At the French Riviera e.g., if somebody were solely protected against UVB it would take about 2-3 h in noon time for a UVA sunburn of a non-tanned person with skin type II (4). UVA is still very much present in morning and afternoon hours, when it is »UVB safe«. Even more concerning are the devastating premature aging effects of UVA light. Therefore day care formulas need to be equipped with a functional UVA protection screen. On top of that should be also an immuno-protective agent, e.g. Niacinamide, because already lower levels of radiation could cause damage here as shown above. People typically apply significantly less than 2 mg/cm^2, which call for additional measures for facial care products. The residual oxidative stress by still transmitted UVA light needs to be taken care of. Therefore a day care formula should also contain an anti-oxidant complex. Combinations of Vitamins and plant extracts offer good solutions here. A signature plant associated with blue sky and high sunshine radiation is Edelweiss (*Leontopodium nivale*, subsp. alpinum). It grows only in high altitudes up to 3000 m with a lot of UV radiation and

has developed powerful anti-oxidant defense systems to survive in this habitat. The constituents are able to protect human skin too (17). It is a protected wild life species; however, due to high altitude alpine cultivations according to Bio Suisse organic standards this material (ALPAFLOR® EDELWEISS) is available to the personal care industry.

The design and realization of facial day care formulas can be particularly challenging to comprise all above-described functionality and together with desirable sensory features. Day creams should neither be too greasy or too glossy nor should the play time too long. After a rather quick dry-out, there should be a nice, silky and matt finish. Extra care claim formulas could purposely leave a perceivable lipid film with a little thicker residue. Usually most UV filters add an oily and greasy feature to the formula, and generate a lot of shine in the residue on the skin after the rub out. For broad UV spectrum functionality UVA and UVB filters are a must but careful selection of the UV filters and building on the SPF contributing synergy between the UV filters will help to tweak the formulas towards a drier, less greasy direction. UV filters occupying the water phase of emulsions, like Phenylbenzimidazole Sulfonic Acid (PARSOL® HS), show very good performance with no oily or greasy skin feel. Polysilicone-15 (PARSOL® SLX) adds not only synergistically UV protection performance to such a system, but also a silky skin feel. The high-spreading silicone feature of this UVB filter implies an auto-rearrange on the skin to correct local film irregularities and weak coverage spots. Formulas with Polysilicone-15 (PARSOL® SLX) therefore perform usually much better on human skin than on in vitro plates. The high SPF boosting synergy between Phenylbenzimidazole Sulfonic Acid (PARSOL® HS) and Polysilicone-15 (PARSOL® SLX) had been clearly demonstrated by *in vivo* SPF determinations (18).

2nd Scenario – Summer holiday exposure

This 2nd scenario includes beach holidays with quite intense and consecutive UV exposure with a typical length of about 1-3 weeks. A skin type II person could theoretically easily gather 10-15 MED on a clear day in Southern Europe in the summer. But as even sun worshipper on a deck chair move and turn their bodies

and the angle of the plain of the skin is often not 90° towards the sun the actual daily UV dosage received is rather the half of the theoretical maximum. However, certain skin areas, let us name them super-exposed areas, e.g. head, top of the ears or shoulders and the nose tip could receive a significant higher UV dosage due to a preferred orientation. It is strongly recommended to use additional sun protection for these super-exposed skin parts. Very recently a sun exposure investigation concluded on Tenerife (19). The participants wore wrist dosimeters, and in only 6 days each of the 25 subjects picked up 57 SED in total on average, which is about 43% of the annual dosage of a Danish indoor worker. This quite intense and highly cumulated UV dose calls for good sun protections measures. A SPF 15 product, as recommended by authorities e.g. in UK or US would protect against sunburn in those circumstances if applied according to guideline, with 2 mg/cm^2 and with re-applications. But there is more to consider than only sunburn. The fully correct usage of such a SPF 15 sunscreen would still result in 0.3-0.5 MED of suberythemal UV radiation on average and super-exposed sites would receive even a higher dosage with all the consequences and risks described above. Products with much higher SPF are therefore recommended for beach holidays or other full-day outdoor activities.

It is a well-known fact that people apply a smaller amount of sunscreen than recommended and required to achieve the labeled SPF factor (20). The SPF that materializes then for the consumer is therefore often only a fraction of the labeled SPF (21, 22). Typical application amounts vary between 0.4 to 0.8 mg/cm^2, which is reducing the (labeled) SPF by about 5 to 2.5 times. To nevertheless cope with the significant UV radiation dosages on the beach, the call is for highest protection products (21) and additional shading strategies to avoid localized sunburns and significant suberythemal damage. In addition, skin care active such as niacinamide might help to reduce the harmful damage by protecting or by stimulating intrinsic cellular repair mechanisms.

A key driver for applying higher amounts of those high performance sunscreens are pleasing sensory features. Most likely it is a sensory endpoint which tells consumers to stop applying more sunscreen at a certain optical or touch sensation (23).

What improved sensory features can do for the application amount shows a study, in which we investigated the right balance of inorganic pigments and Polysilicone-15 (PARSOL® SLX) by comparing the voluntary use level in a consumer study. Participants received two sunscreens; one contained next to other UV filters 6% Titanium Dioxide, the other a mix of 3% Titanium Dioxide and 3% Polysilicone-15 (PARSOL® SLX). Both had similar SPFs, 26 and 27 respectively. The participants had to apply the test creams to a defined skin area for 14 days and thereafter the weight of the used sunscreen was measured. It turned out that the sunscreen with a mix of Titanium Dioxide and Polysilicone-15 (PARSOL® SLX) was unconsciously used at a 29% higher level. This trial can be considered as principal to demonstrate that appealing sensory features can enhance the total amount applied on the skin leading to a better ultimate protection of sunburn and of damages due to suberythemal UV exposure.

Creating high broad spectrum performance and well accepted textures alluring for more product usage represents a tough challenge for the formulator. All options of the sunscreen formulation tool box need to be considered. It starts with the right UV filter combinations for a maximized performance, an optimal use level of non-oily, skin pleasing UV filters like Phenylbenzimidazole Sulfonic Acid (PARSOL® HS), Polysilicone-15 (PARSOL® SLX) and Titanium Dioxide (PARSOL® TX) and in addition the right choice of texture builders. The use of the emulsifier Potassium Cetyl Phosphate (AMPHISOL® K) especially ensures the stability of these complex emulsion systems with relatively high oil loading and high content of pigments and UV filters.

Conclusions

Sunlight is a factor of setting good mood to people. It doesn't wonder that this creates a sun seeking behavior, not just only in holiday situations but also in our daily life, whenever the sun is shining. Unfortunately sunlight comes also with a downside, that can create significant aging and health effects, which call for protective strategies. Avoidance of sunburn turns out to be not enough to ensure successful and sufficient protection. Dosages below sunburn can still affect health and beauty. For shorter exposures a minimum of SPF 15 with an excellent UVA coverage and further protective ingredients is recommended. Longer and subsequent exposures, e.g. beach holidays call for very high SPF numbers with

broadspectrum coverage and additional shade seeking strategies. The sensory profiles of those high performance formulas should be adapted to the consumer's expectations driving the sun seekers to use plenty amount for a better ultimate UV protection. A reduced health risk and a long time young looking facial appearance are waiting as the reward.

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