Sun Protection and ANTI-AGEING

SunActin is a combination of organic sunflower sprouts and natural liposomal alpha-tocopherol. Mibelle Biochemistry designed SunActin to be a complementary suncare product as it can counteract the negative effects of UVA and UVB radiation and protect the skin.

The sun protection mechanism and antiageing properties are evidenced by a reduction in MMP-3 levels, which damage the structure of the skin, preventing any further progression along the collagen degradation pathway. It is well positioned as a naturally derived active ingredient combined with nanotechnology. SunActin enhances a suncare product's ability to boost the protective effect of sunscreens by increasing its SPF; it therefore offers formulation options to reduce sunscreen filter quantities while neutralizing its oxidant effect on skin cells.

UV RADIATION: THE MAIN CAUSE OF PREMATURE AGEING

Photoageing is the main cause of skin ageing. Solar UV radiation is known to be responsible for 80% of skin ageing. The consequences of acute exposure of the skin to UV radiation can range from sunburn to pigmentation modification, inflammation and even immune suppression and tissue damage. Continued exposure to UV radiation for a period of years intensely affects the dermal connective tissue, which leads to premature skin ageing (also called photoageing). Chronically sun-damaged skin is characterized by having a thick and rough appearance, deep wrinkles and discolourations combined with a yellow complexion (hypo- and hyper-pigmentations). It is acknowledged that both UVA and UVB light contribute to photoageing by generating free radicals and reactive oxygen species (ROS). The subsequent oxidative stress affects the structural proteins of the extracellular matrix (ECM) that form the dermal connective tissue.[1] This causes damage and disorganization of the collagen and elastin fibres, as well as an increase in the activity of the matrix metalloproteinases (MMPs). This group of proteolytic enzymes degrade ECM proteins such as collagen.

THE FAILINGS OF SUNSCREENS

Photoageing can be delayed and reversed by reducing sun exposure and by applying sunscreens to the skin. Sunscreens guard the skin from sun damage by reducing the penetration of solar UV rays in the skin via absorption and/or reflection. Nevertheless, sunscreens have a number of disadvantages. First, the use of high sun protection factors (SPFs) does not provide 100% protection to the skin and the development of high SPF products might have driven people to remain in the sun for longer without perceiving any acute warning signs.[2] Secondly, the activity and/or photostability is of limited durability and they have a negative impact on the environment, especially on aquatic organisms.[3] These products are also associated with potential allergy and sensitization effects, as well as reducing the biosynthesis of vitamin D.[4] Finally, the energy absorbed by the UV filters may not be completely converted into heat, forming free radicals that lead to oxidative stress.

SUNFLOWER SPROUTS TO COMPLEMENT THE PROTECTIVE EFFECT OF SUNSCREENS

Mibelle Biochemistry has developed a cosmetic active based on sunflower sprouts (commercial name SunActin) that protects the skin against oxidative stress and complements the protective effect of sunscreens. Sunflower species are native to the Americas. They originate most probably from Mexico where they had been cultivated for centuries for food and medicinal purposes, and also for decorative and ceremonial use. European explorers brought sunflower seeds back to Europe from where they were distributed to

Figure 1: Inhibition of MMP-3 production.

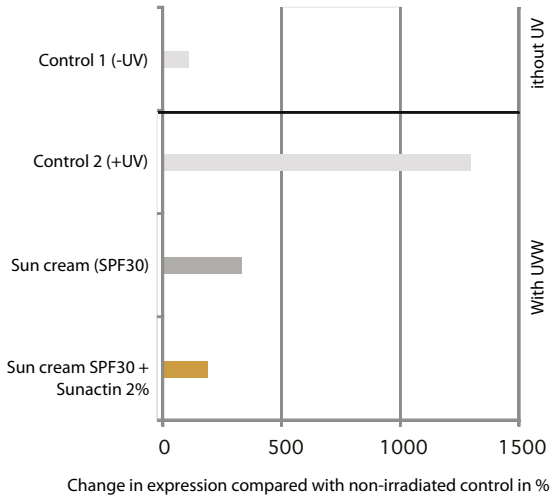
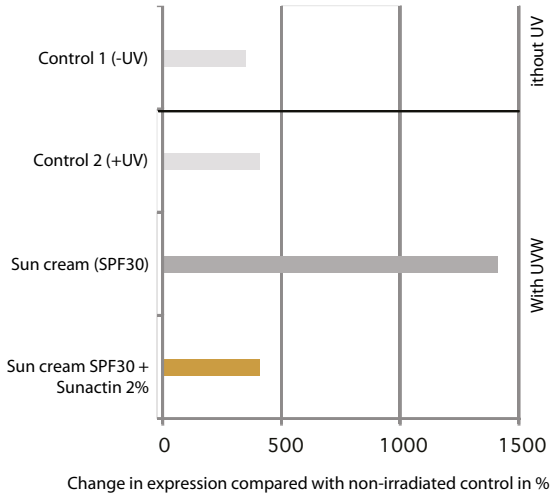


Figure 2: Down-regulation of the oxidative stress marker OXSR-1.



the rest of the world. They became a rich supply of seeds for snacking and cooking. During the 18th century, the sunflower (*Helianthus annuus* L.) started being cultivated for its oil. Today, this is its primary use; its oil is considered to be a premium cooking oil because of its high level of monounsaturated fatty acids.

Phytochemicals (water-soluble phenols or terpenes) are highly abundant in the shoots of a plant because, at this stage of growth, they are especially vulnerable. Shoots have higher levels of nutrients than mature plants. Plant seeds contain the embryo and stored food reserves and, under favourable conditions, when the seeds begin to germinate, the food reserves are mobilized. The fats are transformed into free fatty acids, starch into maltose and proteins into free amino acids. At this stage, some other very important nutrients start forming in the growing seed, such as vitamins, enzymes and the previously mentioned phytochemicals. Many of these phytochemicals are known to exert beneficial effects on human health or to play an active role in the amelioration of disease.

STUDY RESULTS

Ex vivo analysis of the inhibition of MMP production: The effect of SunActin on the expression of selected markers was quantified by gene array analysis. A SPF30 sun cream with and without 2% SunActin was applied to the surface of a reconstructed human epidermis (RHE). Following irradiation (0.3J/cm^2) of the RHE, the expression of genes that are known to be important stress markers was analysed by DNA microarray technology. UV irradiation of the RHE led to a strong increase in MMP-3 gene expression. MMP-3 is responsible for the degradation of type III collagen, one of the most abundant collagens in the dermis. Application of the SPF30 sun cream resulted in a strong down-regulation of the MMP-3 gene. Moreover, the addition of 2% SunActin to the SPF30 sun cream significantly improved this effect (Figure 1).

NEUTRALIZING THE OXIDANT EFFECT OF SUNSCREENS

In the same ex vivo study, application of the SPF30 sun cream was shown to strongly increase the expression of oxidative stress responsive 1 (OXSR-1), a marker gene for oxidative stress. Results showed that OXSR-1 was slightly up-regulated in the irradiated control RHE and strongly up-regulated in the

RHE treated with the SPF30 sun cream. This suggests that the energy absorbed by the UV filters is not completely converted into heat … but also into free radicals that can damage the epidermis. Nevertheless, the addition of 2% SunActin to the SPF30 sun cream significantly counteracted this effect. Thus, SunActin can neutralize the oxidative effect of UV filters (Figure 2).

DECREASE IN THE NUMBER OF SUNBURN CELLS

The capability of SunActin to protect the skin against UV-induced stress was assessed by determining its influence on the creation of sunburn cells in the skin. These apoptotic keratinocytes form in the epidermis as a result of excess UV radiation. Their presence in human skin indicates severe UV-induced cell damage. An SPF30 sun cream with and without 2% SunActin was applied to the surface of skin explants. After 24 hours of incubation, the skin explants were irradiated with UVB (1000 mJ/cm^2), then sectioned transversally and stained (Figure 3a). The protective effects of the test products were then quantified by microscopic counting of the sunburn cells. Results showed that the radiation of the skin explants led to the appearance of 10 times more sunburn cells and that the application of the SPF30 sun cream resulted in a much lower number of sunburn cells (as expected). The addition of SunActin to the SPF30 sun cream strengthened its protective effect as the formation of sunburn cells was further inhibited (41% higher protection compared with the sun cream alone) (Figure 3b).

PROTECTION AGAINST UV-INDUCED STRESS

The capacity of SunActin to protect the skin against UV-induced stress was evaluated by determining its influence on the minimal erythemal dose (MED) — a measure of the resistance of the skin against UV. MED was determined by assessing visual skin redness 24 hours after UV irradiation using a sun simulator. At the beginning of the study, MED was determined on the untreated skin of 20 volunteers aged from 19 to 63. Then, the test areas were treated once with an SPF10 cream with and without 2% SunActin. Subsequently, the skin was irradiated with UV doses whose intensities were in the range of the MED multiplied by the SPF. One day (24 h) later, the redness of the skin was evaluated again. Results showed that SunActin significantly increased the MED compared with the placebo; the skin tolerated a 24% higher UV dose, which corresponds to increasing the SPF from 10.3 to 12.8 (Figure 4).

SUMMARY

SunActin offers two appealing benefits, sun protection and antiageing; it can be used in personal care product formulations for both young and mature consumers as the awareness of sun protection and the need for early antiageing prevention in both sexes is rising. The ex vivo evaluations were performed using reconstructed human epidermis, a more efficacious and ethical way to replace animal testing. SunActin showed a reduction in sunburn cells when compared with the control. The DNA microarray screening of MMP-3 and OXSR-1 gene expression enabled the rapid, accurate and quantitative analysis of SunActin's treatment effects according to the skin's genetic profile. A down-regulation of MMP-3 gene expression was shown. Thus, SunActin protects the extracellular matrix proteins that form the skin's connective tissue and whose degradation accelerates photoageing. The sunflower shoot-based ingredient was shown to neutralize the oxidative effect of UV filters and to boost the SPF. **PHM**

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FOR MORE INFORMATION

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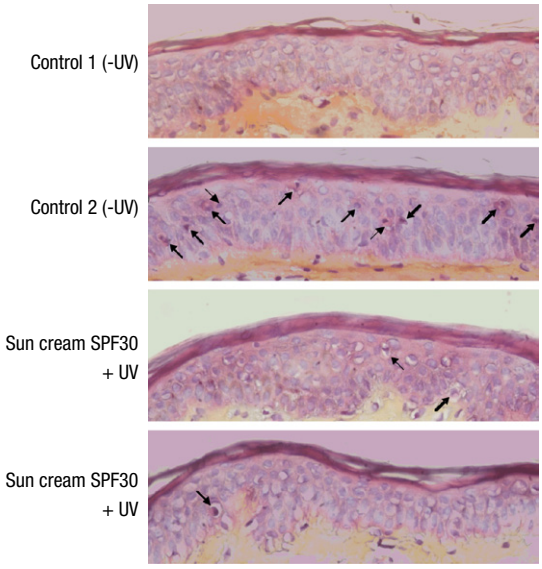
Figure 3: Reduction of sunburn cells in skin explants.

3a



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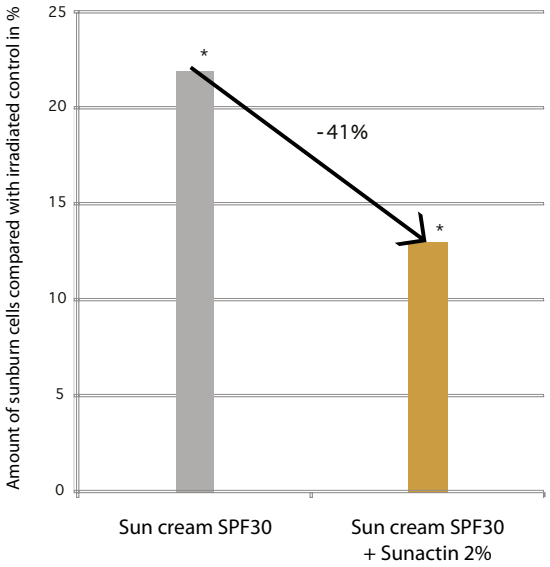
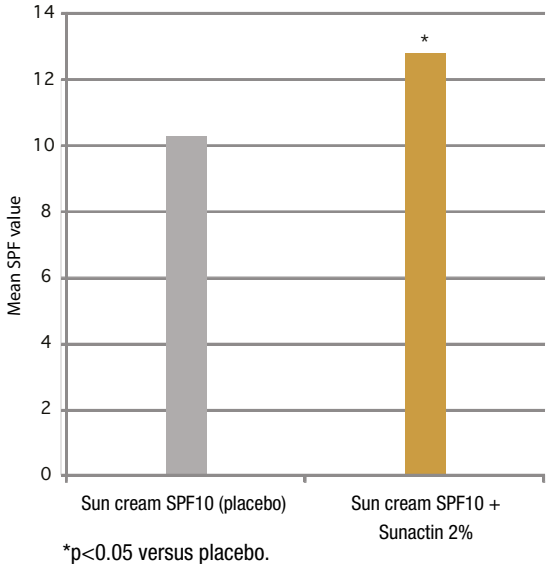


*p<0.01, extremely significant versus irradiated control.

Figure 4: SunActin's SPF boosting effect.



*p<0.05 versus placebo.