The Intelligent Cell Protecting Factor Based On Sunflower Sprouts

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Abstract

Mibelle Biochemistry developed SunActin, a cosmetic active based on an organic sunflower sprout extract combined with natural vitamin E in a nano-emulsion. SunActin protects the skin against oxidative stress and complements the protective effect of sunscreens. Sprouts (syn. shoots) have naturally occurring levels of nutrients (vitamins, antioxidants and phyto-nutrients) which are higher than in the corresponding mature plant. Photo-aging is the main aging process as solar UV radiation is known to be responsible for 80% of skin aging. It affects especially the extracellular matrix (ECM) proteins which form the skin's connective tissue and whose degradation accelerates due to the increasing level of matrix metalloproteinases (MMPs). Several studies including gene array analysis showed the capacity of SunActin to inhibit the production of MMPs responsible for the degradation of major skin components and to boost the protective effect of sunscreens while neutralizing their oxidant effect.

Introduction

Excessive UV exposure leads to premature aging Acute exposure of the skin to UV radiation causes sunburn, modification of the pigmentation, inflammation, immune suppression and tissue damage. Chronic exposure to UV radiation over many years strongly affects the dermal connective tissue leading to premature skin aging also called photo-aging. Photo-aging is the main aging process as solar UV radiation is known to be responsible for 80% of skin aging. Chronically sun-damaged skin has the following characteristics:

- a thick and rough aspect with deep wrinkles
- a yellow complexion combined with discolorations (hypo and hyper-pigmentations)

UV radiation mainly affects the dermis

Both UVA and UVB light, which generate free radicals and reactive oxygen species (ROS), contribute to photo-aging. The resulting oxidative stress affects the extracellular matrix (ECM) proteins, which form the skin dermal connective tissue by:

- damaging and disorganizing the collagen fibers and elastin,
- increasing the activity of the matrix metalloproteinases (MMPs), a group of proteolytic enzymes that degrade ECM proteins as collagen.

Sunscreens have drawbacks

Photo-aging can be delayed and reversed by minimizing sun exposure and by applying sunscreens to the skin. Sunscreens protect the skin against sun damage by reducing the penetration of solar UV rays in the skin via absorption and/or reflection. However, sunscreens have several drawbacks:

- even high SPFs (sun protection factors) don't provide a 100% protection
- development of high SPF products might have driven people to stay much longer in the sun without perceiving any acute warning sign
- the activity/photostability is of limited durability
- they have a negative impact on the environment especially on aquatic organisms
- they are associated with potential allergy and sensitization as well as reducing the biosynthesis of vitamin D
- the energy absorbed by the UV filters may not be completely turned into heat but can also form free radicals leading to oxidative stress.

Mibelle Biochemistry developed a cosmetic active (SunActin) that protects the skin against oxidative stress and complements the protective effect of sunscreens.

Results and Discussion

Reduction of sunburn cells

The capacity of SunActin to protect the skin against UV-induced stress was evaluated by determining its influence on the formation 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 UVinduced cell damage.

A sun cream SPF 30 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/cm2), then sectioned transversally and stained. The protective effects of the test products were then quantified by microscopic counting of the sunburn cells. (Fig. 1)



Fig 1: Reduction of Sunburn Cells Formation in Skin Explants

Results:

- The radiation of the skin explants led to the appearance of 10 times more sunburn cells.
- The application of the sun cream SPF 30 resulted in a much lower number of sunburn cells, as expected.
- The addition of SunActin to the sun cream SPF 30 reinforced its protective effect as the formation of sunburn cells was further inhibited (41% higher protection compared to the sun cream SPF30 alone). (Fig. 2)



Fig 2: Reduction of Sunburn Cells Formation in Skin Explants

SunActin inhibits the formation of sunburn cells and improves the protective effect of UV filters.

Inhibition of MMPs production (ex vivo)

The effect of SunActin on the expression of selected markers was quantified using gene array analysis. A sun cream SPF 30 with and without 2% SunActin was applied to the surface of reconstructed human epidermises (RHE). After radiation (0.3J/cm²) of the RHE, the expression of genes which are known to be important stress markers was analyzed by DNA microarray technology. UV radiation of the RHE led to a strong increase of MMP-3 gene expression. (Fig. 3)



Fig 3: Down-Regulation of MMP-3

MMP-3 is responsible for the degradation of collagen type III, one of the most abundant collagens in the dermis. Application of the sun cream SPF30 resulted in a strong down-regulation of the MMP-3 gene. Moreover, the addition of 2% SunActin to the sun cream SPF30 significantly improved this effect.

Neutralization of the oxidant effect of sunscreens (ex vivo)

In the same study, application of the sun cream SPF30 was shown to strongly increase the expression of oxidative stress responsive 1 (OXSR-1), a marker gene for oxidative stress. (Fig. 4)

Results showed that OXSR-1 was slightly upregulated in the irradiated control RHE and strongly up-regulated in the RHE treated with the sun cream SPF30.

This suggests that the energy absorbed by the UV filters is not completely turned into heat but also into free radicals which can damage the epidermis. However, the addition of 2% SunActin to the sun cream SPF30 significantly counteracted this effect. Thus, SunActin can neutralize the oxidative effect of UV filters.

Protection of the skin against UV-induced stress (in vivo)

The capacity of SunActin to protect the skin against UV-induced stress was evaluated by determining its influence on the minimal erythemal dose (MED). MED is the lowest amount of UV that produces skin redness 24 hours after exposure. It is therefore a measure of the resistance of the skin against UV. MED was determined by assessing visually skin redness 24 hours after UV radiation 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 SPF 10 cream with and without 2% SunActin. Afterwards, the skin was irradiated with UV doses whose intensities were in the range of the MED multiplied by the SPF. 24 hours later, the redness of the skin was evaluated again.

Results showed that SunActin significantly increased the MED compared to the placebo; (Fig. 5) the skin tolerated a 24% higher UV dose, which corresponds to increasing the SPF from 10.3 to 12.8.

SunActin can thus protect the skin against UV-induced stress and boosts the SPF.





Fig 4: Down-Regulation of Oxidative Stress Marker

Fig 5: SPF Boosting Effect

Conclusion

SunActin, based on a nanoemulsion of an organic sunflower sprout extract combined with natural vitamin E, showed significant bioactivities in different in vivo and ex vivo tests.

SunActin showed in comparison with the control a reduction of sunburn cells in skin explants, also when protected by a sun cream SPF30. SunActin showed in skin explants the down regulation of MMP-3 gene expression, also when the explant was treated with a sun cream SPF30.

SunActin protects the extracellular matrix (ECM) proteins which form the skin's connective tissue and whose degradation accelerates photo-aging. Sunactin was shown to neutralize the oxidative effect of UV filters. SunActin protects the skin against UV-induced stress and boosts the SPF.

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