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EFFECTIVENESS OF SPF50+ SUNSCREEN CONTAINING PHENYLENE BIS-DIPHENYLTRIAZINE IN THE PREVENTION AND THE ACCOMPANYING TREATMENT OF ACTINIC KERATOSIS

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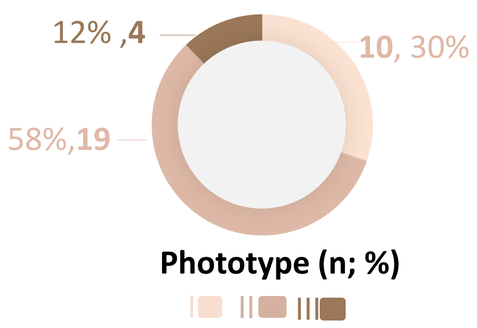
INTRODUCTION

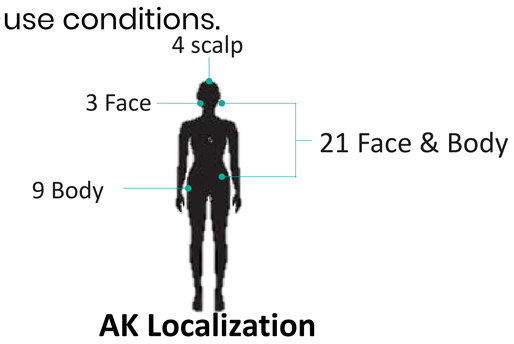
Actinic keratosis (AK) is a common precancerous skin lesion caused by cellular and genomic damage induced by chronic exposure to UV radiation. AK lesions are susceptible to carcinogenesis in the field, and around 0.5 to 10% of them may develop into squamous cell carcinoma (SCC). Photoprotection is therefore essential to prevent AK development and worsening.

We developed a SPF50+ sunscreen containing a very high photoprotective system with TriAsorB™ dedicated to AK patients. SPF50+ sunscreen was evaluated using reconstructed human epidermis (RHE) on genoprotection and skin lipidome protection against sun exposure. Skin lipidome plays a critical role in the development and progression of AK by influencing the skin's barrier function and its ability to respond to UV-induced damage. The compatibility with methyl-5 aminolevulinic acid (MAL) daylight photodynamic therapy (dPDT) was also studied on RHE models. Skin tolerance and acceptability are key parameters for improving compliance and encouraging re-application of sunscreen among AK (actinic keratosis) patients; these two factors were evaluated in a clinical study.

MATERIAL & METHODS

Efficacy in preventing UV-induced DNA damage and lipids modifications was assessed through *in vitro* studies using reconstructed human epidermis and solar simulated irradiation (Suntest, 300 to 800nm) . Dermatological tolerance and acceptability study was performed on 33 adults 100% with grade I AK, according to JAAD 2021 Guidelines KA management e-App, Olsen *et al.* (mean age = 61 years old; from 48 to 79 years old) after 3 weeks of real-use conditions.





Finally, the compatibility with MAL-photodynamic therapy was measured through in vitro study mimicking the dermatologists' protocol for daylight photodynamic therapy.

RESULTS

Lipidome modulation

There were 252 different lipids in the RHE models which were detected and statistically significantly deregulated (up- or down-regulated) by solar irradiation, including multiple phospholipids classes, triacylglycerols, diacylglycerols, sphingolipids and ceramides. The heatmap in Figure 1 represents selected lipids (50 from the 252 lipids) from the different categories that were deregulated by irradiation.
Sphingolipid pathway was upregulated with sun exposure. We observed upregulation of 8 ceramides, 5 glucosylceramides but also 5 sphingosines. Ceramides can be considered as a hub in sphingolipid metabolism and can induce apoptosis by activating p53. Stress conditions such as sun exposure led to increase levels of ceramides in skin cells, which in turn can activate p53, leading to cell cycle arrest or apoptosis. When the SPF50+ sunscreen is used none of these lipids were still significantly modulated.

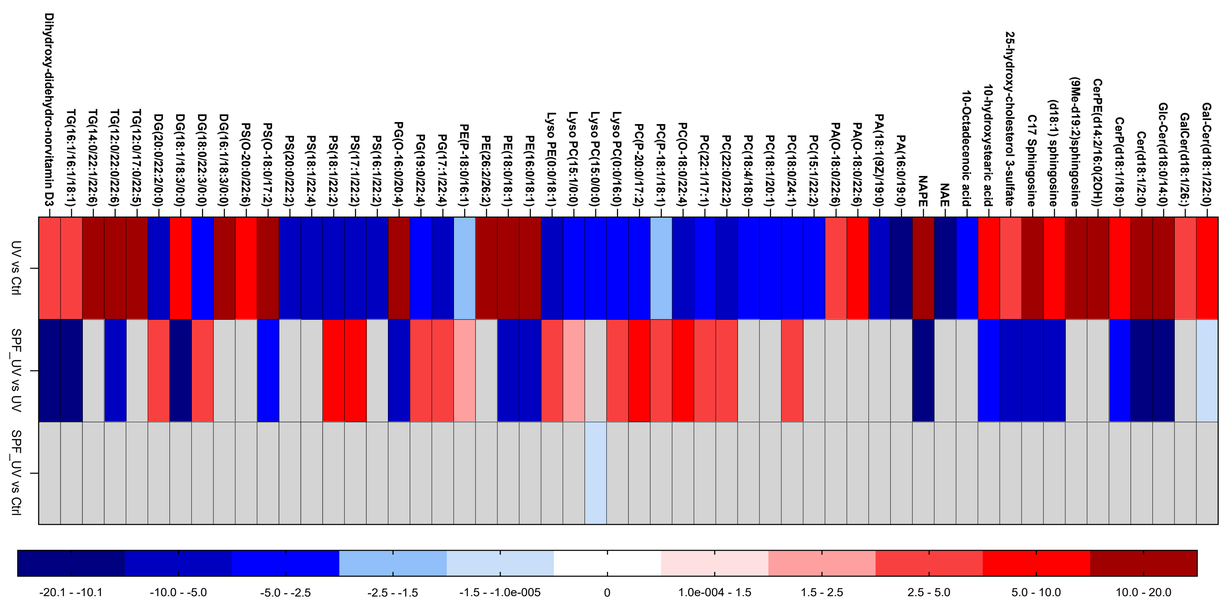
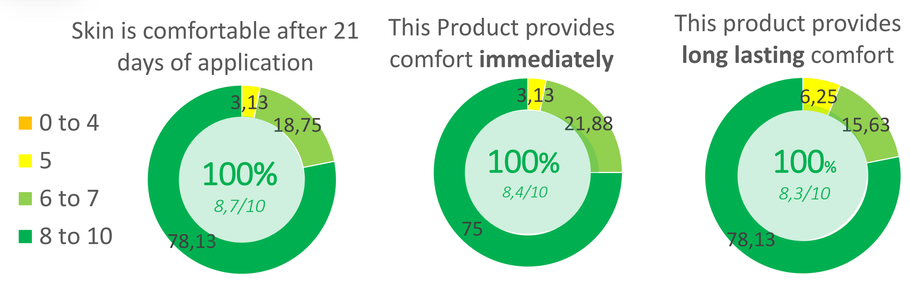


***Fig.1** : Effect of solar irradiation on skin lipidome using LC-MS/MS analyses. Heatmap of 50 selected lipids from the different categories*

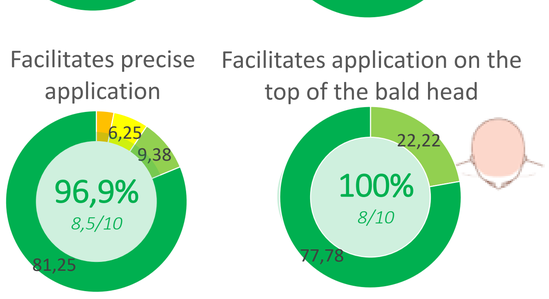
Clinical evaluations

Clinical evaluations concluded that the product showed an **excellent cutaneous tolerance** (0 reaction on AK, 100% sensitive skin) and a very good product acceptability (product providing comfortable, supple, and protected skin with a texture adapted to daily use).

100% of subjects find their **skin supple,** protected, not tight



+90% of subjects find the product **easy to apply** even on the **scalp**



100% of subjects find the product leaves a dry touch and is **suitable for daily use.**

81% of subjects consider this product **better** than their usual photoprotection

In-vitro UV-induced DNA lesions protection

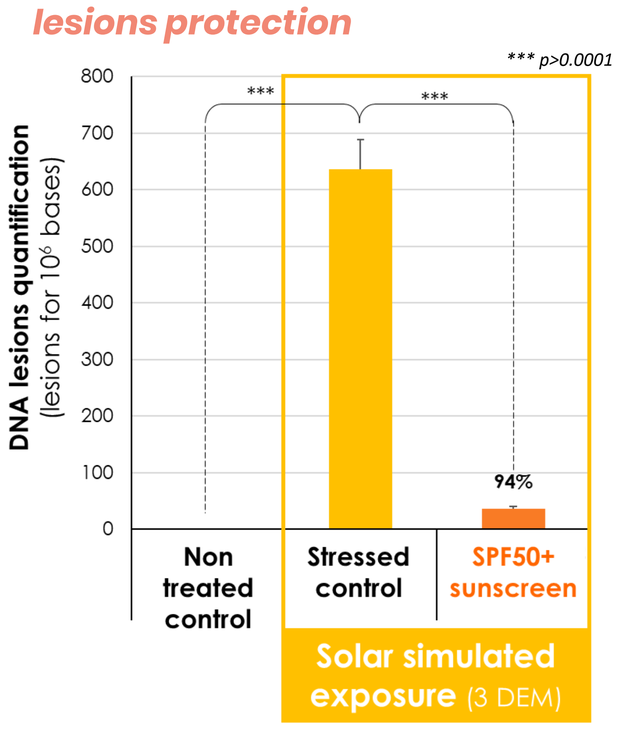


***Fig.3** : UV-induced DNA lesions (CPD + 64PPs + Dewar) protection of SPF50+ sunscreen after solar simulated exposure*

Preventing treatment with the sunscreen showed a high and significant protection of 94.4% ($p<0.0001$) of UV-induced DNA lesions (CPDs, 64PPs and Dewar), and so an UV-induced AK lesions protection.

In-vitro MAL daylight photodynamic therapy (dPDT) compatibility

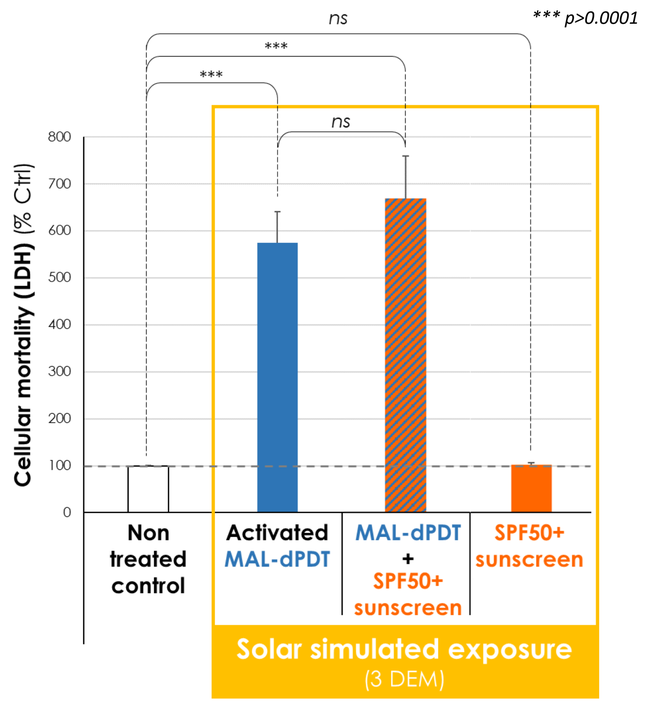


***Fig.4** : Compatibility of the use of SPF50+ sunscreen containing TriAsorB with MAL daylight photodynamic therapy*

Using the dermatologists' protocol for MAL (methyl-5 aminolevulinic acid)-dPDT, we showed that pre-treatment with SPF50+ sunscreen with TriAsorBTM did not interfere with MAL-induced cell death, and therefore MAL activation.

CONCLUSION

This work demonstrates the benefit of the application of SPF50+ sunscreen in preventing AK limiting its worsening. Indeed, SPF50+ sunscreen avoid 94% of the DNA lesions formation in reconstructed human epidermis but also the dysregulation of lipidome (252 lipdis significantly modulated with SSR). Moreover, SPF50+ sunscreen is compatible with its MAL-treatment and showing an excellent skin tolerance and acceptability.