



PHOTOBIOLOGY AND PHOTOPROTECTION

## PHOTOPROTECTIVE EFFICACY OF A NEW MEDICAL DEVICE AGAINST CHRONIC UV-INDUCED PRECANCEROUS CUTANEOUS LESIONS IN AN EX VIVO SKIN MODEL

*S. Julié<sup>(1)</sup> - D. Bacqueville<sup>(1)</sup> - A. Couttet<sup>(2)</sup> - C. Jean Decoster<sup>(2)</sup> - L. Vidal Poulou<sup>(2)</sup> - V. Georgescu<sup>(2)</sup> - S. Bessou-touya<sup>(1)</sup> - H. Duplan<sup>(1)</sup>*

*Pierre Fabre Dermo-cosmétique, Department Of Pharmacology, Toulouse, France<sup>(1)</sup> - Laboratoires Dermatologiques Avène, Medical Department, Lavour, France<sup>(2)</sup>*

**Introduction:** The prevalence of actinic keratosis (AK) has increased in last decade and is a part of skin field of cancerization induced by chronic sunlight exposure. Therefore, it is important to prevent AK by using photoprotection strategies.

**Objective:** To assess the photoprotective efficacy of an innovative medical device containing specific sunfilters combination to protect the skin against UV-induced precancerous lesions.

**Materials and Methods:** An ex vivo human skin model was developed to mimic AK lesion formation in response to chronic exposure of solar-simulated radiation (SSR). The medical device presented a high sun protection factor (SPF50+) and was applied before each SSR. Skin received 3 SSR acute doses for a day. P53 and apoptosis signaling pathways were then analyzed by using immunoassays (Multiplex, Simple western) and fluorescent immunolocalization.

**Results:** The AK skin model showed that chronic SSR induced an increase of the expression of the tumor suppressor p53 in keratinocyte nuclei. This biomarker is commonly mutated in AK where its level has been associated to an enhanced stability. The p53 signaling pathway was also activated since SSR exposure was able to promote p53 phosphorylation on serine 15. Chronic UV treatment also revealed that apoptotic effectors were up-regulated in response to DNA damage in the skin explants (Caspase-3, PARP, sunburn cells). Topical application of medical device afforded an effective photoprotection against SSR-induced skin lesions. The SSR-exposed explants treated with medical device were protected from p53 upregulation (nuclei location) and phosphorylation (-96%). Concomitantly, the activation of apoptosis was almost prevented after sun formulation application.

**Conclusion:** The medical device had a high photoprotective efficacy in AK skin model and





protected the skin from both the expression and activation of the p53 precancerous biomarker. Thus, the sunfilter formulation may help to take care of patients in AK prevention and improve the skin cancerization field.

