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INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin condition that compromises the skin barrier, causing intense itching, redness, and dryness. It significantly impacts quality of life and makes the skin particularly sensitive to environmental irritants like chlorine, salt, sand, and sweat, which can exacerbate discomfort. Sunscreens adapted for AD are crucial, especially during summer exposure. The skin's defense relies on the stratum corneum, which contains natural moisturizing factors (NMFs) and lipids like ceramides, crucial for maintaining skin integrity and hydration. In AD, increased skin permeability heightens sensibility to irritants and allergens, with sun exposure further exacerbating these issues. The effectiveness of sunscreens in preventing these changes is not well understood, requiring more research. This study evaluates the protective effects of a broad-spectrum SPF50+ sunscreen on AD subjects, both *in vivo* and *in vitro*, to assess its impact on skin barrier function and lipids composition.

RESULTS

Clinical results

After 21 days, the sunscreen reduced discomfort from sand (100%), salt (98%), chlorine (97%), and sweat (95%), allowing over 85% of subjects to space out their usual emollient application (Fig.1). A significant decrease in discomfort sensations ($p < 0.0001$) and TEWL ($p < 0.0001$) was observed, with improvements in numerous subjects, showing a protective effect on the skin barrier (Fig.2). 100% of adults appreciated the product.

Fig. 1: a) Baseline discomfort levels b) Discomfort alleviated by the product after 21 days

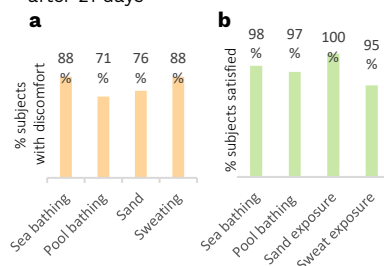
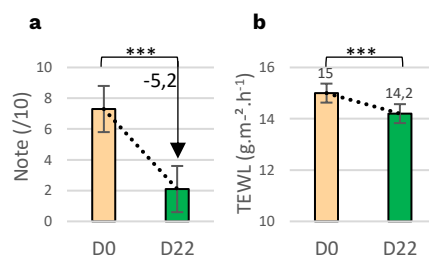


Fig.2: Mean a/Discomfort to external irritative factors and b/TEWL



Metabolites and Lipids modulations

Among the significantly altered metabolites (log2-fold changes with $p \leq 0.05$), we found several natural moisturizing factors (NMFs): amino acids, lactate, glycerol, urocanic acid, pyrrolidone carboxylic acid and derivatives. The observed changes in the quantities of several filaggrin byproducts, such as UCA and PCA, may result from enhanced proteolysis of filaggrin in response to SSR radiation (Fig.3).

Analyses of the lipids also showed that SSR induced lower levels of free fatty acids and higher levels of ceramides, cholesterol and its derivatives. Glycerophospholipid pathway was impacted with higher mean levels of glycerol and lower mean levels of choline, glycerophosphocholine and phosphorylcholine after SSR radiation.

An imbalance in NMFs and ceramides combined to an increase of pro-inflammatory lipids may participate in skin permeability barrier impairment, dehydration and inflammatory reaction to the sun. The use of SPF50+ sunscreen prevented SSR-induced alterations in NMFs and lipids.

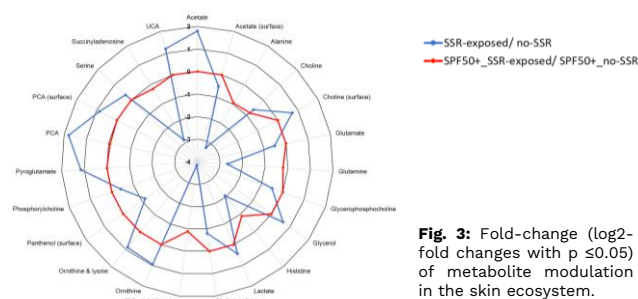


Fig. 3: Fold-change (log2-fold changes with $p \leq 0.05$) of metabolite modulation in the skin ecosystem.

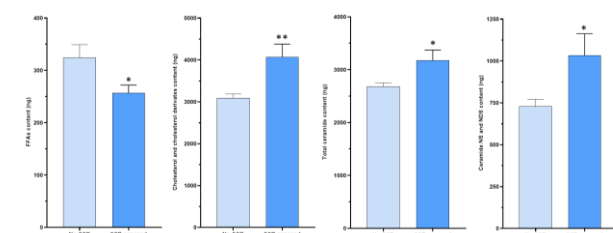


Fig. 4: Lipid levels (qty in ng, $p \leq 0.05$) in the skin ecosystem w/wo SSR exposure.

CONCLUSION

This broad-spectrum SPF50+ sunscreen effectively protects AD skin from environmental irritants and sun exposure. It reduces discomfort and TEWL, maintaining skin barrier integrity by preventing NMFs and lipids alterations. These findings highlight the importance of effective sunscreens for AD-sensitive skin and the potential of dedicated formulations to enhance skin barrier resilience.