

EXTREME PROTECT® SPF 40 AND PREVENTION OF SUNBURN CELL FORMATION

STUDY OBJECTIVE

This study evaluated the ability of Extreme Protect SPF 40 to protect against sunburn cell formation when skin was exposed to full-spectrum UVA and UVB solar radiation.

STUDY DESIGN

A research quality solar simulator was used to expose the in vitro human skin model to measured amounts of UVA and UVB light. Extreme Protect SPF 40 was applied to the skin one hour prior to exposure. Biopsies of the skin were then evaluated for sunburn cells after hematoxylin and eosin (H&E) staining. Photomicrographs were taken to document experimental results. Positive and negative controls were also performed.

SIGNIFICANCE OF STUDY

Solar UV light exposure on skin causes photoaging and DNA damage, associated with an increased incidence of skin malignancy.¹ As packets of free radicals strike skin secondary to full-spectrum solar exposure, sunburn cells are created as a result of DNA damage. Sunburn cells contain the full range of types of DNA damage and are so typical of solar UV damage that they have been termed a “solar signature.”

Clinical evaluation of sunscreen efficacy is measured by SPF ratings in the United States and several other rating types worldwide. Although helpful in establishing regulatory conformity, none of these national or regional rating methods directly measures DNA damage. Evidence that a sunscreen protects against sunburn cell formation would be very valuable in proving that the sunscreen also protects the skin’s DNA. Improved DNA photoprotection indicates decreased risks of malignant transformation and photoaging.

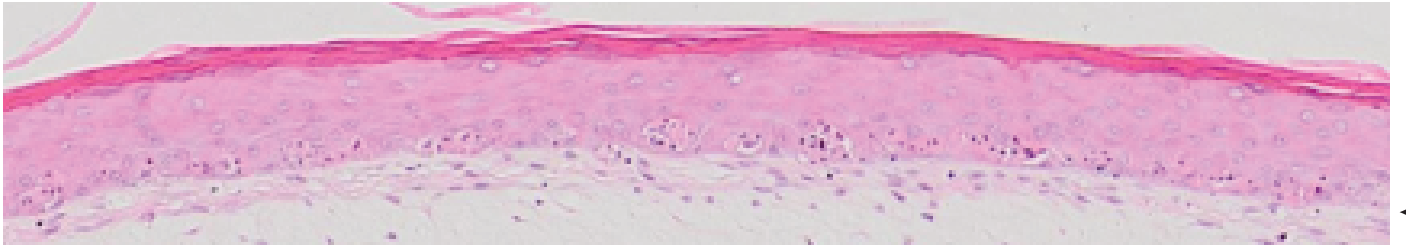
Sunburn cell formation is an apoptotic process that occurs when solar UV damage severely damages DNA.² There is an established relationship between the sunburn cell and skin photocarcinogenesis.³ Some apoptotic cells escape this process of programmed cell death, resulting in a cancer-prone genotype and phenotype. The deregulation of the balance between apoptosis and cells

that escape apoptosis is related to the amount of chromosomal DNA damage they have sustained and the total UV exposure damage profile. Furthermore, solar exposure is responsible for at least 80% of the changes of photoaging.⁴

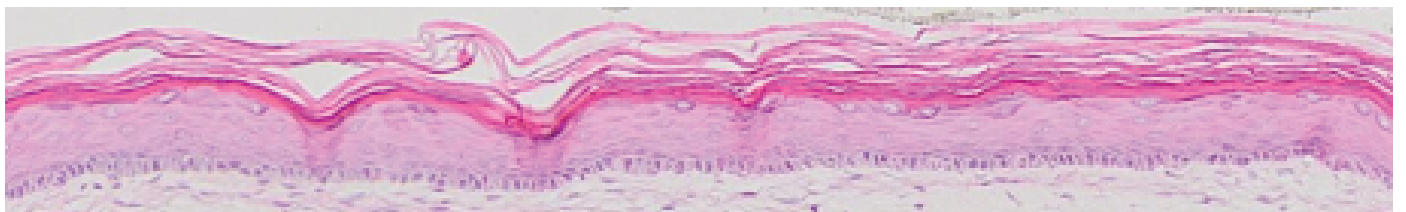
These severely injured sunburn cells, with their damaged DNA and malignant potential, are easily visible microscopically. As a quantitative measure of DNA damage severity, sunburn cells may be counted. Following application of Extreme Protect SPF 40, the number of sunburn cells present, compared to the positive control, measures the amount of free radical protection and DNA damage protection conveyed by Extreme Protect SPF 40 to the skin. This protection would also indicate decreased risks of photoaging and skin cancer – important reasons for consumers to use this product.

RESULTS AND CONCLUSIONS

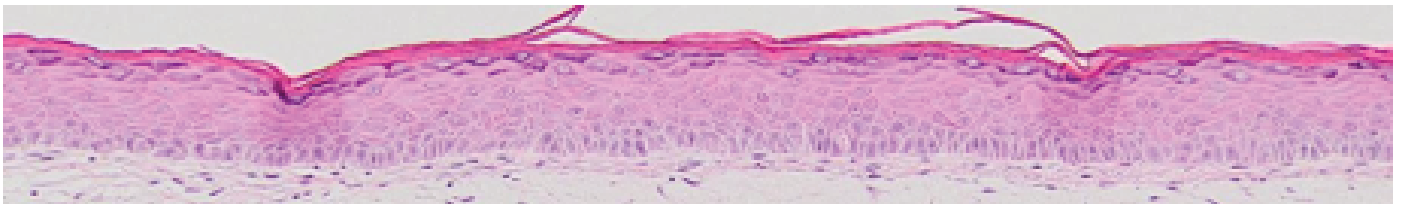
Many sunburn cells were formed in the basal epidermis of irradiated skin without application of Extreme Protect SPF 40. These visualized sunburn cells are pyknotic, i.e. they exhibit the dark purple, shrunken nuclei and foamy, pink, swollen cytoplasm consistent with severe UV photodamage. When skin was protected by applying Extreme Protect SPF 40 prior to full-spectrum solar exposure, no sunburn cells were visibly seen, and when counted, were nearly equivalent to the negative control NC (skin without UV exposure/skin in the dark). This indicates dramatic protection conveyed by Extreme Protect SPF 40 to the genetic material of the cell with near-baseline numbers of sunburn cells seen in the negative control NC (skin in the dark without solar exposure) and the skin exposed to UVA+UVB with application of Extreme Protect SPF 40. In contrast, irradiated skin without application of Extreme Protect SPF 40, the positive control PC, contained very high numbers of sunburn cells. These findings are consistent with DNA photoprotection by Extreme Protect SPF 40 from the effects of UV damage that would otherwise encourage the development of photoaging and skin cancer.



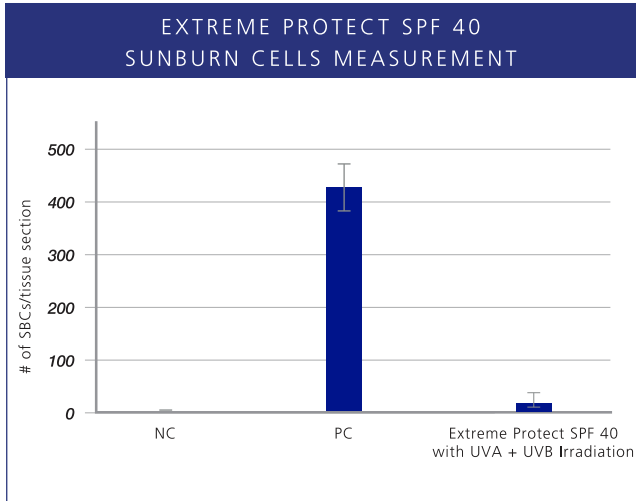
PC Positive Control shown above. Full spectrum UVA + UVB irradiation. No application of Extreme Protect SPF 40. Arrow indicates the basal epidermis with many apoptotic sunburn cells seen as large, foamy, pink cells with dark, shrunken, pyknotic nuclear material. Consistent with large amounts of DNA damage and increased risks of photoaging and skin cancer.



No visible sunburn cells in photomicrograph above. Full spectrum solar irradiation. Extreme Protect SPF 40 applied one hour prior to UVA + UVB irradiation. Appearance of this skin is identical to that in NC Negative Control, indicating approximately the same amount of DNA damage, risk of photoaging, and risk of skin cancer as skin in the dark (no solar irradiation) without product application.



NC Negative Control shown above. No solar irradiation. No application of Extreme Protect SPF 40. Basal layer of epidermis shows no visible sunburn cells.



The bar graph above shows counted sunburn cells for NC Negative Control (skin without sun exposure in the dark and no application of Extreme Protect SPF 40), PC Positive Control (solar irradiated skin without application of Extreme Protect SPF 40), and irradiated skin (UVA+UVB) with application of Extreme Protect SPF 40. $P < 0.001$

Application of Extreme Protect SPF 40 prior to broad spectrum solar UV exposure protects DNA from photodamage as measured by sunburn cell formation. These results were highly statistically significant. Numbers of sunburn cells for sun-exposed skin protected with Extreme Protect SPF 40 were equivalent to skin without sun exposure. Furthermore, these results are consistent with decreased risks of photoaging and malignant transformation to skin cancer.

REFERENCES

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- ⁴Flament F, Bazin R, Laquieze S, Rubert V, Simonpietri E, Piot B. Effect of the sun on visible clinical signs of aging in Caucasian skin. *Clin Cosmetic Inv Dermatol.* 2013. 6: 221-232.

DISCLOSURES

Study performed at MatTek Inc.

Study type: Skin tissue study

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