

Impact of melanin on vitamin D and DNA photodamage

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Abstract. We report on the impact of melanin on vitamin D production in the skin, compared with its protective effect on photodamage to DNA.

Introduction

Exposure to solar ultraviolet radiation (UVR) has beneficial and detrimental effects on human health. The most established beneficial effect is the cutaneous synthesis of vitamin D, the status of which is assessed by serum 25(OH)D. The most serious detrimental effect is skin cancer, which is especially important in fair-skinned populations. The main trigger for skin cancer is UVR-induced DNA damage in the epidermis. The most common and biologically significant type of DNA photodamage is the cyclobutane pyrimidine dimer (CPD) that can result in highly specific mutations that initiate skin cancers.

Most photobiological research has been done on white-skinned people because of their higher risk of skin cancer than those with pigmented skins. There are relatively few photobiological studies in the latter group and the data are often contradictory (Fajuyigbe and Young, 2016).

Epidemiological studies show that people with pigmented skins tend to have poorer vitamin D status than those with fair skins, and this is usually attributed to attenuation of UVR by melanin. However, laboratory studies to test this attribution have yielded contradictory results (Xiang et al., 2015).

Studies

We have determined the effect of melanin on vitamin D photosynthesis (manuscript in preparation). Dose response studies for 25(OH)D were done on healthy volunteers with different skin phototypes using fluorescent solar simulating radiation (SSR). Each volunteer received serial whole body sub-erythral exposures of a fixed UVR dose. Blood draws were taken before each exposure to generate linear dose-response curves. Comparisons of dose-response slopes of white and black skins show that the protection factor (PF) of melanin against the formation of 25(OH)D is <1.5. However, this modest level of protection may be sufficient to explain the differences that have been reported in the epidemiological studies.

In contrast, acute SSR dose response studies in black and white skins showed that melanin offers much higher protection against CPD formation in black skin, especially in the basal layer (that contains the stem cells). We estimate that the PF against such damage is about 60 (Fajuyigbe et al., 2018).

Conclusions

We suggest that the reason for the large difference in PF for CPD and 25(OH)D is the location of the melanin relative to the chromophore. Melanin is concentrated in the epidermal basal layer, especially in black skin. DNA is the

chromophore for CPD and its greatest protective effect is in the basal layer. 7-dehydrocholesterol is the chromophore for vitamin D production, of which there is sufficient above the basal layer to largely bypass the UVR filtering effects of melanin.

References

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