Hydrogen Peroxide Cream for the Prevention of White Pressure Areas in UVA Sunbeds

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A single application of 1% hydrogen peroxide in a stabilized lipid cream during 2 min before UVA exposure prevents white spots in anoxic pressure areas in sunbed use, causing an almost normal pigmentation. During maintenance exposure with UVA once a week this pigmentation will remain unchanged if the pressure areas are pretreated with hydrogen peroxide before each irradiation. White spots will appear 3–4 weeks after finishing hydrogen peroxide pretreatment in the pressure areas thus exposed to UVA alone.

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Pressure areas on the back (scapular region and medial sacral region) remain unpigmented on the reclining subject upon exposure to UVA from sunbeds, probably due to a lack of oxygen (1, 2). It has earlier been demonstrated that the application of a stabilized hydrogen peroxide cream prior to UVA exposure could induce delayed tanning on the pressure areas, while the placebo-treated contralateral sites remained unpigmented (3). This finding provides evidence for the role of oxygen in delayed pigmentation by UVA (4). The hydrogen peroxide cream was used ad libitum with repeated applications during a half hour period before exposure to UVA (3).

The aim of the present study was to evaluate the pigment promoting effect of a single application of the cream a short time before exposure to UV light and to study the pigmentation during maintenance treatment with UVA with and without hydrogen peroxide.

MATERIAL AND METHODS

Twenty-one healthy individuals, 18 men and 3 women, in the age range 23–35 years, who tan easily in the summer sun (skin types III and IV), were selected for the study.

Light exposure

A Philips sunbed was used (Philips TL 85 W/09 T) with an emission spectrum of 310–420 nm and a peak emission at 355 nm. The skin was exposed for 30 min per day, 4–5 times per week (6–12 exposures in all during the initial treatment phase) and then during the maintenance treatment phase once a week.

Creams

The two active creams contained 2% and 1% hydrogen peroxide in a stabilized lipid cream base of 21% monomyristin, 7% monolaurin and 70% water. The two creams used in series II were not identifiable by appearance or smell and were supplied in identical tubes with a code sign and marked "left" and "right".

"Treatment"

Series I: Comparison 2 and 5 min application of 2% H_2O_2 cream. The subjects were irradiated with UVA from below while reclining fairly still on the hard acrylic surface of the sunbed. Before each sunbed session the 2% H_2O_2 cream was applied to symmetrical areas known to remain hypopigmented after exposure to UVA on sunbeds (scapular areas and sacro-gluteal areas) during periods of 5 min and 2 min before irradiation, randomly on the left and right sides. The cream was washed off with lukewarm water just before light exposure.

Series II: Comparison 2% and 1% H_2O_2 cream. In this study 2% and 1% hydrogen peroxide creams were applied with a double-blind technique on the selected symmetrical areas (scapular and sacro-gluteal areas) 2 min before irradiation. The cream was washed off just before light exposure.

Series III: Maintenance treatment. After the provocation of delayed tanning with H_2O_2 and UVA on the pressure sites, maintenance treatment followed once a week. Before each

irradiation one side was pretreated with 2% H_2O_2 cream 2 min before exposure while the contralateral side was not treated. The cream was washed off with water just before light exposure.

RESULTS

After 6–12 initial treatment sessions (H_2O_2 cream + UVA) delayed tanning was observed on the pressure areas. However, compared with the "normal" delayed UVA tanning on non-pressure areas, the pigmentation of the pressure sites was delayed by a few days and was also somewhat uneven, with a slight variation in pigmentation, often with a semilunar hyperpigmentation at the periphery of the pressure sites. It was also noted that it was more difficult to obtain even pigmentation on the scapular region than on the medical sacral region.

In *series I* there was no difference in the pigmentation on the sides pretreated for 5 and 2 min. In *series II* there was no difference in the pigmentation between the sides pretreated with 2% and 1% H_2O_2 cream. *Series III.* When pretreatment with H_2O_2 cream was stopped, the white spots appeared after 3–4 weeks of UVA light alone (maintenance treatment once a week). When, however, one side was pretreated 2 min before irradiation during the maintenance treatment the pigmentation remained unchanged during the whole observation period (3–6 weeks).

Side-effects

During the initial pigmentation phase with 4–5 exposures/week, one patient experienced soreness on the side treated with 2% but not with 1% H_2O_2 cream after 7 exposures when the pressure areas were almost "normally" pigmented. An acute skin reaction with redness and later hyperpigmentation was observed in this area, being more pronounced at the periphery. The condition was clinically similar to a toxic or phototoxic response. Biopsy was not permitted. The UV exposure was stopped and the inflammatory reaction slowly subsided during the next few days, leaving, however, a marginal hyperpigmentation for several days.

DISCUSSION

It has earlier been shown that repeated applications of 2% hydrogen peroxide in a stabilized lipid cream base can restore the UVA pigmentation in oxygen-deficient pressure areas of the skin associated with the use of sunbeds (3). The present study shows that a single application of 1% hydrogen peroxide cream applied for a period of 2 min before each UV exposure is sufficient to prevent the white pressure areas associated with commercial UVA sunbeds. During a maintenance period with UVA-treatment once a week the pigmentation remains unchanged in the pressure areas pretreated with hydrogen peroxide before each irradiation. On the other hand, white spots will appear 3–4 weeks after finishing hydrogen peroxide pretreatment in the pressure areas thus exposed to UVA alone.

One patient suffered a local inflammatory skin reaction in the pressure area treated with the 2% but not the 1% hydrogen peroxide cream. The reaction was similar to a toxic or phototoxic skin response.

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REFERENCES

1. Tegner E, Rorsman H, Rosengren E. 5-S-Cysteinyldopa and pigment response to UVA light. Acta Derm Venereol (Stockh) 1983; 63: 21–25.
2. Tegner E. Tissue anoxia prevents inflammation and pigmentation caused by UVA but not by UVB and PUVA. Photodermatology 1984; 1: 311–312.
3. Tegner E, Björnberg A. Induction of UVA pigmentation in pressure areas by hydrogen peroxide. Acta Derm Venereol (Stockh) 1986; 66: 65–67.
4. Auletta M, Gange W, Tan OT, Matzinger E. Effect of cutaneous hypoxia upon erythema and pigment responses to UVA, UVB, and PUVA (8-MOP + UVA) in human skin. J Invest Dermatol 1986; 86: 649–652.