

Also from a therapeutic view it is important to know to what degree bandaging of an edematous leg decreases blood flow and blood pressure.

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Induction of UVA Pigmentation in Pressure Areas by Hydrogen Peroxide

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Tegner E, Björnberg A. Induction of UVA pigmentation in pressure areas by hydrogen peroxide. *Acta Derm Venereol (Stockh)* 1986; 66: 65-67.

Heavy external pressure, caused by the weight of the body when lying on a hard transparent surface during UVA irradiation, prevents pigmentation in pressure exposed skin areas. After percutaneous H₂O₂ administration a delayed pigmentation appeared on the pressure sites. This finding provides evidence for the role of oxygen in delayed pigmentation by UVA. *Key words: UVA; Delayed pigmentation; Pressure effects; Oxygen.* (Received April 29, 1985.)

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Areas of skin under pronounced external pressure (caused by the individual lying on the hard transparent plate of a UVA sunbed) will not exhibit immediate or delayed pigmentation from UVA (1). This results in "white spots" on the scapular region and on the medial sacro-gluteal region, a phenomenon well-known from commercial UVA tanning booths.

The absent UVA pigmentation is probably due to low concentrations of oxygen in the tissues at the pressure sites (1, 2). The aim of the present study was to investigate if H₂O₂ could replace oxygen in preparing the tissue for UVA pigmentation. Since solutions of hydrogen peroxide are unstable a new stable preparation of hydrogen peroxide, dispersed in the water phase of monoglycerides of laurin and myristin, has been used (3, 4).

MATERIAL AND METHODS

Five healthy men in the age range 22-25 years, who tan easily in the summer sun (skin types III and IV), were selected for the study.

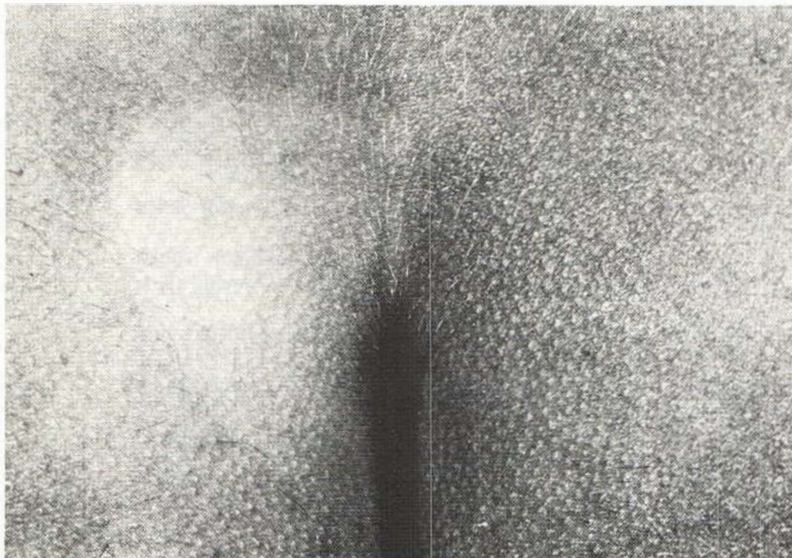


Fig. 1. Pressure areas after 8 days' UVA exposure on a sunbed. Right side pretreated with hydrogen peroxide cream.

Light exposure

A Philips sunbed was used, equipped with 10 UVA tubes (Sylvania F 75/85W/PUVA) with an emission spectrum of 320 to 390 nm and a peak emission of 365 nm. The intensity of the radiation just above the acrylic sheet of the sunbed was about 11 mW/cm² around the 360 nm band (Waldmann UV-meter).

Creams

The active cream contained hydrogen peroxide 2%, monolaurin 7%, monomyristin 21%, water 70%; the placebo cream contained the same ingredients except for the hydrogen peroxide. The two creams, not being identifiable by sight or smell, were supplied in identical tubes with a code sign and marked "left" and "right".

"Treatment"

The subjects were irradiated with UVA from below while reclining fairly still on the hard acrylic surface of the sunbed. The subjects were exposed for 30 min daily, except Saturdays and Sundays, for a total of 8 days. Before each exposure the hydrogen peroxide cream and its placebo were randomly applied according to the double-blind principle, on symmetrical areas known to remain hypopigmented after exposure from UVA sunbeds (scapular areas and sacro-gluteal areas). The creams were applied to the selected areas 5 times (once every 5 min) before the irradiation. The last application was carried out 5 min before commencement of the irradiation, and the creams were washed off with water just before exposure. The originally selected sides were kept strictly during the whole trial period.

RESULTS

After 8 days of "treatment" the delayed tanning reaction could easily be observed. In all the 5 tested subjects the pressure sites treated with the hydrogen peroxide cream were hyperpigmented as was the rest of the exposed body, while the placebo-treated contralateral areas remained unpigmented (Fig. 1).

COMMENTS

Pressure sites on the back (scapular region, medial sacral region) remain unpigmented on the reclining subject upon exposure to UVA from sunbeds, probably because of lack of

oxygen. Application of a stabilized hydrogen peroxide cream induced delayed tanning on the pressure sites, while the placebo-treated contralateral sites remained unpigmented.

The results of this study show that the hydrogen peroxide-containing cream has biological effects on intact normal skin, and that the effects of pressure on delayed pigmentation by UVA will be normalized by an extra percutaneous H₂O₂ supply. This supports the assumption that oxygen is responsible for the induction of delayed pigmentation by UVA (1, 2). This is in contrast to pigmentation caused by UVB and PUVA, which seems to be independent of the presence of oxygen in the tissue at the time of exposure (5).

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Subtotal C4 Deficiency and SLE-like Disease

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Extremely low C4 values were found in a 65-year-old man with relapsing arthritis and skin lesions of many years duration of the scalp, face, hands and feet together with painful ulcerations of the toes and fingers. The discovery was made during an exacerbation, but the deficiency of C4 persisted in repeated controls after remission. The clinical findings in connection with these low C4 values are in congruence with the diagnosis of inherited deficiency of C4. *Key words: Complement; Genetic deficiency; IgM.* (Received June 18, 1985.)

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Hereditary deficiency of all components of the classical pathway of complement activation may occur (1). It has been shown that deficiency of C1, C2 and C4 is related to immune complex diseases similar to SLE (1). Both complete and partial genetic C4 deficiency are rare conditions but have been described in connection with immune complex diseases (3, 4). The present report is of a further case of subtotal C4 deficiency with a SLE-like disease.

CASE HISTORY

A man born in 1919 suffered during his teens from arthralgias. Since about 1970 he has recurrent episodes of angina pectoris and in 1977 a myocardial infarction was diagnosed. For at least 10 years he has had skin lesions. In the scalp there has been scarring alopecia and purple lesions with telangiecta-