Extreme low temperatures reduced encapsulation in *C. hesperidum* reared on squash; developing parasitoids were detected in 16 to 24% of the parasitized scales. High temperatures had a greater effect on the reduction of encapsulation; thus, in *C. hesperidum* kept on detached oleander leaves, heat reduced the incidence of parasitized scales having encapsulated eggs to less than 50%, consequent upon which adult parasitoids (males and females) emerged. Good results of using high temperatures were attained with *S. coffeae* reared on potato sprouts, and with *C. hesperidum* reared on squash, the overall best treatment being 40 °C for 24 h before exposure to parasitoids. No encapsulated eggs were found in either of the parasitized scale nymphs subjected to this treatment, while only 8.2% of the young *S. coffeae* females and 7.6% of the young *C. hesperidum* females contained encapsulated eggs together with developing parasitoid larvae. Only 28.5% of the 'rubber' stage females of *S. coffeae* and 5.5% of the ovipositing females of *C. hesperidum* escaped the parasitoid's effect by encapsulating all the latter's eggs.

The health and vigour of the host are among several factors which affect the occurrence of an encapsulation response[11,13]. The extreme temperatures, to which the brown soft scale and the hemispherical scale were subjected before exposure to parasitoids, probably reduced encapsulation by weakening the scale hosts by a yet unknown mechanism. We are presently rearing *C. hesperidum* and *S. coffeae* as hosts for various parasitoids employed in current biological control projects, and the findings herein reported are facilitating the mass rearing of these parasitoids. A full account of this and related work will be published elsewhere.

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Stable Lipid Peroxidation Products in Human Skin: Detection, Ultraviolet Light-Induced Increase, Pathogenic Importance

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Summary. Products of lipid peroxidation (malonaldehyde, Schiff-bases) were detected in human skin. These products were increased after UV-light exposition, on chronically sun-exposed areas as well as with advancing age. Malonaldehyde cross linked epidermal glucose-6-phosphate-dehydrogenase and diminished their activity.

It is not clear whether the damage to human skin caused by irradiation with UV-light is effected mainly by changes in the nucleic acids, or via a labilization of the cell membranes, particularly the lysosomal membranes. As far as the latter aspect is concerned, lipid peroxidation provides a suggestive conception by means of which the sunburn, the aging of the skin and probably a number of pathological conditions, such as photosensitization and light provocation of skin diseases, can be explained. After irradiation with UV-light, free radicals appear in the skin[2] and lysosomal hydrolases are released[3]. We were able to demonstrate in human surface lipids and in the skin, the formation of lipid peroxides and also of substances which develop in the course of destructive free radical reactions and are positive in the thiobarbituric acid test (mainly malonaldehyde)[4–6]. Minimal concentrations of these products inhibit cell respiration[7]. But because of their high reactivity, these products are difficult to measure, and it could therefore only indirectly be inferred that physiological doses of UV-light may cause harmful lipid peroxidation in the skin[8].

The stable final products of lipid peroxidation to be found in the internal organs have been thoroughly characterized[9]. These are, above all, 1-amino-3-imino-propenes (Schiff base products) which are formed by a reaction of malonaldehyde with amino groups and can hardly be split by enzymes. These Schiff base products can be identified by means of their typical spectroscopic data.

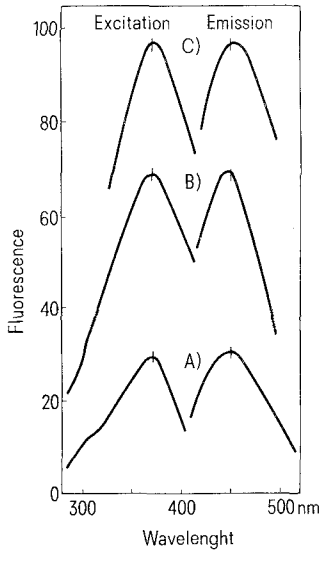

Fig. 1. Fluorescence spectrum of the chloroform/methanol extract of the human epidermis. A) non-irradiated abdominal epidermis; B) after irradiation with 10 MED of UV-light; C) after addition of malonaldehyde. 200 mg of epidermis were homogenized in the presence of 2 *mM* of EDTA by means of the TP 18/2 Ultra Turrax (Jahnke & Kunkel, Stauffen im Breisgau). Fluorescence in arbitrary units.

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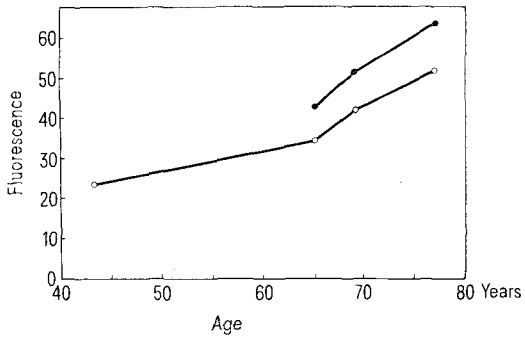

Fig. 2. Maximal values of the fluorescence spectrums from the abdominal (lower curve) or forehead skins (upper curve) of 4 subjects. Mean values from repeated tests.

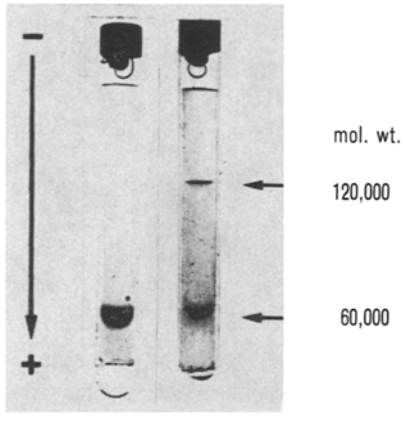

Fig. 3. Sodium dodecylsulfate gel electrophoresis of epidermal glucose-6-phosphate dehydrogenase. A) native enzyme; B) after 12 h of previous incubation with malonaldehyde in 0.1 *M* of phosphate buffer, pH 7.5. Conditions of electrophoresis: 8 mA/tubes; 6 h at 25 °C. Reference proteins for the determination of the molecular weight: trypsin (23,000), aldolase (40,000), catalase (60,000), β-galactosidase (135,000). Protein determination was carried out using the microbiuret method[14].

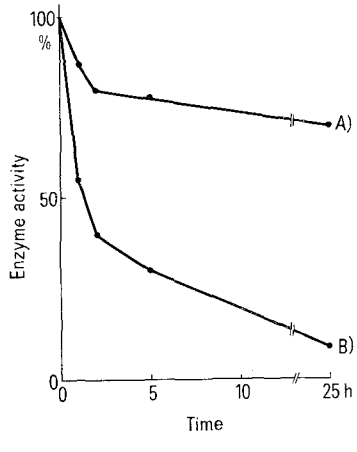

Fig. 4. Inhibition of the glucose-6-phosphate dehydrogenase in dependence on malondialdehyde concentration and incubation time. Enzymatic activity expressed as percentage of the respective controls without malondialdehyde[15].

First, 1- to 3-day-old Wistar rat babies were irradiated by a mercury hot quartz lamp with 8.2×10^7 ergs cm^{-2}, which corresponds to 10 MED (minimum erythema-doses) in man. Thereafter, the animals were decapitated, their skin homogenized, the Schiff base products extracted and measured with an MPF-2A Hitachi fluorescence spectrophotometer[9]. The typical maxima between 360 and 390 nm (excitation) and between 440 and 470 nm (emission) did occur; in non-irradiated animals they were absent. Human epidermis stemming from the abdomen was separated from the dermis and measured immediately after excision[10]. As can be seen in Figure 1A, Schiff base products are to be found already in the non-irradiated epidermis; their concentration increases after irradiation with 10 MED ($p = 0.001$; Figure 1B). To demonstrate directly that malonaldehyde generates the typical fluorescent products, we added 0.01 mg of malonaldehyde per mg of protein to the epidermal homogenate (Figure 1C). In 4 other subjects a comparative study of the abdominal and forehead skins was carried out (Figure 2). In the forehead epidermis which is chronically exposed to solar radiation, the content of fluorescent Schiff base products was higher in all cases and their concentration seems to increase with advancing age.

It was natural to assume a direct correlation between the formation of malonaldehyde, the development of covalent bindings (Schiff base products) and the damage to biological structures. To verify this assumption epidermal glucose-6-phosphate-dehydrogenase (EC 1.1.1.49) was isolated[11] and incubated with 1 mg of malonaldehyde per mg of protein. There appeared, after incubation, in the sodium dodecylsulfate gel electrophoresis[12] a second pattern with a molecular weight of about 120,000 Dalton (Figure 3). Hence, subunits of the enzyme (molecular weight about 30,000 Dalton, Figure 3) were covalently crosslinked. To find out whether or not the biological function of the enzyme is affected by the bindings, the enzymatic activity was determined following addition of 0.01 or 1 mg of malonaldehyde per mg of protein (Figure 4). As compared with the controls, the enzymatic activity is diminished. In all probability, the additional covalent bindings effect a change in the structure of the active centre of the enzyme.

The present findings prove that, after irradiation with UV-light, lipid peroxidation reactions take place in human skin. There are closed relations between lipid peroxidation and aging[13] as evidenced by the findings presented in Figure 2. Usually, certain regions of human skin are exposed to UV-irradiation more than others. By simple ointment therapy, high concentrations of effective substances may be achieved in the skin. Hence, it should be possible to reduce or prevent skin damage resulting from acute or chronic solar irradiation by the application of substances inhibiting lipid peroxidation.

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