PHOTOGRAPHIC NATURE OF TANNING OF THE HUMAN SKIN AS SHOWN BY STUDIES OF MALE HORMONE THERAPY[1]

THE condition of the skin is known to be influenced by the endocrine system, as for examples, the bronzing in Addison's disease of the cortex of the adrenal gland, the acne commonly associated with puberty and the pigmentation changes during pregnancy. Changes in the skin of a hypogonadal patient following the use of synthetic male sex substance testosterone propionate[2] have been reported.[3] A further series of three surgically castrated[4] and four hypogonadal males has been studied. Before treatment the skin in all but two cases was of a characteristic pasty, sallow color, gray and lacking in pink tinge. This was most pronounced in the castrated men. After treatment with testosterone propionate there was a rapid flushing, followed by increased oiliness of the skin and growth of hair on the face, chest, abdomen, arms and legs. All patients presented a more tanned appearance, particularly of the face, neck, hands and exposed parts of the skin. Part of this increased pigmentation is due to "developing" of pigment from previous exposure as indicated by the following history:

Case 1. Orchidectomy was performed in May of 1937, after which time the patient tanned but poorly and burned easily upon exposure to the sun. In August of 1937 he spent part of every morning and afternoon for a week lying on the beach clad in an abbreviated bathing suit of a peculiar cut. Only slight coloration resulted. The patient was examined by the authors several times during December and January, at which time the body skin was of a pasty sallow color. Treatment with male hormone substance was begun in the dead of winter, January 17, 1938. Within three weeks there appeared, along with the bronzing of the face, a tanning of the body save where it had been protected by a bathing suit. The patient had not worn the bathing suit, whose peculiar pattern the tan fitted, or any other bathing suit for five months. Neither had he sunned himself or used a sun-lamp in similar fashion.

Upon withdrawal of the hormone treatment in February of 1938 the flush disappeared from the skin and the tanned areas gradually faded. Subsequent injection and withdrawal periods induced, respectively, coloration and a fading of these areas which had not been exposed to sunlight since the previous August. These phenomena might be accounted for in part by assuming either that tanning consists of a continued production of pigment over a long period of time or that the melanin becomes colorless unless an adequate hormone supply be present.

Graded exposures to a sun-lamp have been given to this and to other patients and a similar series of pigmentation changes have been observed during periods of treatment and withdrawal.

From studies of men with low amounts of testicular secretion it appears that male hormone substance exerts a "developing" action upon the rather colorless material which is laid down in the skin following exposure to the sun or sun-lamp. This "developing" action may be exerted as late as five months after exposure. This indicates that tanning may be a "photographic-like process" of "exposure" and "development," with the sex hormone acting to "develop" color-lacking material laid down in the skin by exposure. Further, the pigmentation is not established continuously but will fade upon cessation of hormone treatment and reappear upon later resumption of treatment.

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[1] Supported in part by the International Cancer Research Foundation.

[2] Furnished by the Ciba Company under the trade-name Perandren.

[3] James B. Hamilton, *Endocrinology*, 21: 649, 1937.

[4] Neal E. Miller, Gilbert Hubert and J. B. Hamilton, *Proc. Soc. Exp. Biol. and Med.*, 38: 538, 1938.

ACTION OF VANADIUM ON TISSUE OXIDATIONS

IF 10–20γ of vanadium in the form of sodium meta vanadate is added to rat or guinea pig liver suspension at pH 6.7 a large extra oxygen uptake occurs. This effect is much less in kidney and absent in brain. Concentration curves indicated that some substance in the liver was oxidized in the presence of vanadium. To prove this the following experiment was done. Rat liver was ground with buffer pH 6.7, squeezed through muslin and centrifuged. The solid was resuspended in 40 cc of water to which 10 cc buffer pH 6.7 was added and centrifuged again. This process was repeated four times and the resultant solid material which was free of hemoglobin and light yellow in color was finally suspended in 15 cc buffer pH 6.7. This constituted the enzyme preparation. The substrate was prepared as follows. A guinea pig liver was chopped and ground in buffer pH 6.7. Enough alcohol was added to make the final concentration 70 per cent. The precipitate was filtered and the alcohol evaporated off in vacuo at 40° C. The resulting suspension was extracted with ether three times, leaving a clear light yellow solution. This solution was then treated with a small amount of Lloyd's reagent and filtered. The filtrate was now almost colorless. It could now be evaporated down to dryness and extracted with boiling 95 per cent. alcohol. The alcohol