was discontinued. Among 'non-essential' amino acids, glycine-deficiency resulted in total mortality of larvae, while in other cases fewer larvae became adults in longer time, as compared with normal larvae. Omission of any one 'essential' amino acid made the diet entirely unsuitable for aposymbiotic individuals since, in almost all cases, no or very few larvae became adults. It is therefore evident that the yeast-like micro-organisms supply in varying amounts almost all the ten 'essential' and most of the 'non-essential' amino acids.

The present example of the physiology of symbiotes is probably the first of its type where intracellular micro-organisms have been shown to play an important role in the amino acid requirement of an insect. Aposymbiotic condition is, however, known to cause higher mortality in *Coptosoma*[4], *Triatoma*[5], and *Rhodnius*[6], or to produce additional dietary demands for vitamins of B-complex in *Lasioderma*[1] and *Oryzaephilus*[7]. TÓTH[8] has demonstrated fixation of nitrogen in the host body by microbes from atmosphere, or from nitrogenous waste products such as uric acid.

The full details of the investigation will be published elsewhere in due time.

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Résumé

Les auteurs ont soumis des larves de *Stegobium paniceum* L. à un régime contenant seulement des acides aminés. Ils ont aussi constaté expérimentalement que la levure, de même que les symbiontes intracellulaires fournissent des acides aminés en quantités variables selon l'hôte.

Some Etiological Factors in the Production of Malignant Melanoma

GHADIALLY and BARKER[1] have found that the melanotic tumours produced in hamster skin by repeated applications of 9:10 dimethyl 1:2 benzanthracene arise from a network of melanocytes surrounding some of the pilosebaceous follicles. It is well known that chemical carcinogens penetrate, accumulate, and persist in hair follicles for a considerable period of time and that an early effect of carcinogenic action is disruption of sebaceous glands[2]. It would therefore appear that the peculiar distribution of melanocytes in the hamster skin places these cells in a most vulnerable position for attack by the carcinogen. The fact that similar networks have hitherto not been observed in mouse and rabbit skin explains why hamsters are so prone to the development of melanotic tumours while mice and rabbits are not.

Many large melanotic networks also surround the coarse hair producing giant pilosebaceous follicles situated in the costovertebral spot of the hamster. Repeated painting of the costovertebral spot, however, did not produce melanotic tumours in the spot itself, though many such tumours appeared in the surrounding skin[1]. The most likely explanation of this phenomenon is that the carcinogen was rapidly flushed out by the secretion from these large sebaceous glands.

Whole mount of hamster shin showing network of dendritic melanocytes surrounding pilosebaceous follicles in the region just above and around sebaceous glands. Haematoxylin and eosin × 137

Our experiments on the histogenesis of the hamster melanoma[1] suggest that a close association between melanocytes and the pilosebaceous follicles producing fine hair may be dangerous, while an association between melanocytes and large pilosebaceous follicles producing large coarse hair may not be so dangerous. In man a close association between a large collection of melanocytes and pilosebaceous apparatus, similar to that seen in the hamster, does not occur in normal skin but is sometimes seen in naevi. The hairy mole of man; a well recognised clinical entity, may be considered morphologically similar in certain respects to the costovertebral spot of the hamster for in both these sites we find large coarse hairs, large sebaceous glands, and abundant melanocytes. Since the costovertebral spot is very resistant to the action of chemical carcinogens one might suspect that the same is probably true in the case of the hairy mole of man. Clinical observation supports this idea for malignant changes very rarely occur in the hairy mole[3].

It is now generally accepted that when a malignant melanoma arises in a mole or in normal human skin it is from melanocytes at the dermo-epidermal junction. In this position ultraviolet light in the form of solar radiation

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is more likely to reach and stimulate these cells to melanoma production than chemical carcinogens. Recent studies by LANCASTER[4,5] have drawn attention to the relatively high incidence of malignant melanoma on the exposed part of the body and in geographical locations with more sunlight hours. These studies suggest that solar radiation is a factor in the production of malignant melanomas in man. This however does not exclude the possibility that in some instances chemical carcinogens may be responsible for malignant melanoma production, for there is no reason to suppose that the melanocyte at the dermo-epidermal junction is totally inaccessible to chemical carcinogens, or refractory to such stimulation.

The factors responsible for converting the melanocytes in the skin or the naevus of man into a malignant melanoma are as yet not precisely established but the collective evidence now indicates that endogenous factors such as genetic[6-8] and hormonal[9], and extrinsic factors such as ultraviolet radiations[4,5] and chemical carcinogens may all be involved. It remains for future research to establish the relative importance of these factors in the production of malignant melanomas in man.

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Zusammenfassung

Die Bedeutung von Erb- und Hormonalfaktoren bei der Entwicklung des malignen Melanomes wurde vor allem früher hervorgehoben. Es scheint, dass auch karzinogene Stoffe (Chemikalien) und ultraviolette Bestrahlung von wesentlicher Bedeutung sind.

Change of Threshold During Dark Adaptation Measured with Orange and Blue Light in Cats and Rabbits

Recently the change of threshold during dark adaptation has been measured in cats and rabbits by determining the light energy necessary for a constant electroretinographic response after light adaptation with strong and moderate lights (DODT and ELENIUS[1]). Two phases were found in the dark adaptation curves obtained after strong light adaptation, the break occurring on an average after 15 min in the dark in cats and 40 min in rabbits. The electroretinographic dark adaptation curves of cats and rabbits show two phases of change of threshold, and resemble the course of sensory dark adaptation measured in the human peripheral retina, where the initial part of the curve is usually ascribed to the cones and the second part to the rods. In agreement with this theory, KOHLRAUSCH[2] showed that the break in the adaptation curve of the human peripheral retina occurred later and the total change of threshold was smaller if measurements were made with lights of long wavelengths than if they were made with white light or lights of short wavelengths. This effect of the test wavelength on the shape of dark adaptation curve has since been confirmed by using the microelectrode technique in the frog eye (GRANIT[3]) and in the cat (GRANIT[4]; BARLOW, FITZHUGH, and KUFFLER[5]).

The aim of the present experiments was to see if there is a similar change of spectral sensitivity during dark adaptation in the electroretinographically measured dark adaptation curves of cats and rabbits. This problem has become particularly interesting as it has been shown that in cats the maximum of spectral sensitivity (measured electroretinographically) is at 556 mμ when flicker stimulation at high rates (above 30/s) and strong stimuli are used; whereas in rabbits, under the same conditions, the spectral sensitivity curve closely follows the rhodopsin absorption curve (DODT and WALTHER[6]).

In 3 cats and 4 pigmented rabbits the change of threshold during dark adaptation has been measured with white light (Xenon arc) and with orange and blue light (narrow band double interference filters, λ_{max} 605 and 462 mμ). The cats were decerebrated or anesthetized with Evipan. The rabbits were anesthetized with Urethane. Flaxedil was given to all anesthetized animals and they were artificially respirated. The light used for light adaptation and the test light both illuminated the same area (3 cm in diameter) of an opal glass disc placed 15 mm in front of the eye, except in one cat and one rabbit where both beams were focused in the dilated pupil in order to make light adaptation as effective as possible and to facilitate the measurement with colored test lights in the beginning of dark adaptation. The duration of light adaptation was 15 min in all experiments.

Typical results are illustrated in Figure 1 (cat) and Figure 2 (rabbit). Figure 1 shows that in the cat the 'cone phases' of the dark adaptation curves measured with orange light (open triangles) and blue light (open squares) are clearly separated (the curves are made to coincide in full dark adaptation = zero ordinate). This

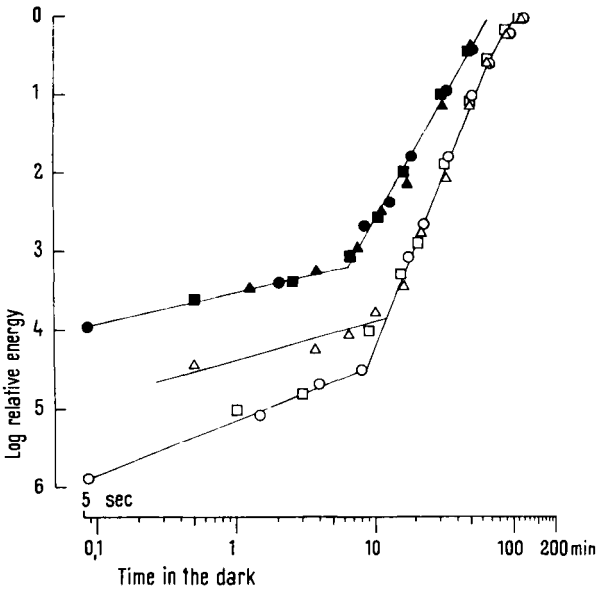

Fig. 1. Cat, Evipan anesthesia, Flaxedil and artificial respiration, pupil dilated with Homatropine and Veritol (Knoll) drops. Dark adaptation curves plotted in log relative energy at threshold (energy necessary for an electroretinogram of constant size, 10 μV b-wave) against log time in the dark. The curves are made to coincide at the threshold preliminary measured in fully (3 h) dark adapted eye. Test light: white (circles), 462 mμ (squares) and 605 mμ (triangles). Light adaptation: white light, duration 15 min, retinal illumination in first light adaptation about 1.05×10^6 Trolands (open symbols), in second light adaptation 0·6 log units weaker light (filled symbols)

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