

Dolley, David H., and Frances V. Guthrie. (i) PIGMENTATION OF NERVE CELLS. (ii) LIPOCHROME A PLANT CAROTINOID PIGMENT. [Journ. Med. Research, 1919, Vol. XL, 295.]

The prevailing opinions of the origin of the fat-holding or lipochrome pigment of the nerve cell have been either that it is a by-product of cell metabolism, an "Abnutzung" or "wear-and-tear" pigment, as designated by Lubarsch, or a more specific product of fat or fatty acid metabolism, the lipofuscin of Borst and Hueck. While its possible identity with the lipochrome of plants has been surmised, the microchemistry alone, in its state of progress, has been insufficient to demonstrate it.

However, recent biochemical and physiological studies have demonstrated that the plant pigments, now generally known as the carotinoids (carotin and xanthophyll), are intimately associated with animal metabolism. Palmer and Eckles and, later, Palmer alone, have shown that the natural yellow pigments of the milk fat, body fat, corpus luteum, blood serum or skin of such animals as the cow, horse, and hen, are identical with carotin, or xanthophyll, or both, as the case may be. Palmer has proved experimentally that chickens, deprived from birth of carotinoid pigments, show absence of yellow pigment in their skin, fat, egg yolk, and blood serum. If they are given the pigment in their food, the color is restored, only to disappear if they are deprived of it. Further, Palmer, has shown a remarkable species difference: species with colored fat, such as the cow, horse and hen, carry the pigments in the blood serum; species with colorless fat, such as sheep, swine and goats, do not carry the pigments in the blood serum.

The working hypothesis for the nerve cell was based on these propositions, and verification was first sought in the chicken. Two series were run, the one lacking carotinoid-containing food from birth, the other carotinoid fed. The latter series consisted both of the experimental introduction of carotinoids in previously carotinoid-free chickens and of other carotinoid-fed from birth. In one half of the chickens of both series, the factor of depression by heat, phosphorous, morphine, or a rice flour diet was introduced to cover the side of disease. That is, the vast majority of abnormal and morbid stimuli are essentially depressant.

The results were uncomplicated. Both normal and depressed chickens on any carotinoid diet showed the presence of the characteristic yellow pigment in all types of nerve cells. The carotinoid-free chickens, both normal and depressed, lacked such a pigment in demonstrable amount. The species difference of Palmer was also shown to hold for nerve cells in the constant occurrence in bovines (association with colored fat) as opposed to the constant absence in swine (associated with colorless fat). Man, who is best known to exhibit lipochrome, is also known to carry carotinoids in his blood serum and has colored fat. Finally, the microchemical reactions of the experimentally introduced lipochrome are throughout identical with those of the lipofuscin, fat-

holding, "Abnutzung" or "wear-and-tear" pigment described by others. The microchemistry, while too superficial for independent analysis, is adequate for the demonstration of an identity. The deposition of the lipochrome is exaggerated by depression. This gives a common basis for its well-known increase under various abnormal conditions. The conception of Lubarsch that the fat-holding pigment is a "wear-and-tear" or metabolic pigment therefore falls to the ground with its demonstration as an exogenous and fortuitous pigment. From previous experimental studies on nerve cell pigmentation, it is the melanin which is the true metabolic pigment of the nerve cell. However, it is not a product of normal or hypernormal activity, but its genesis under all conditions, physiological, morbid, or senile, is referable solely to chronic depression. The exploding of the tradition that melanin is a natural constituent of certain nerve cells, such as those of the *substantia nigra* and *locus caeruleus*, further corroborates its normal absence. The nerve cell is not hampered in its normal process by the accumulation of pigment. The melanin is consequently not a "wear-and-tear" pigment in the normal sense. In so far, however, as depression is a component of old age, disease, and abnormal physiological conditions, the conception of Lubarsch may be transferred to its resulting melanin pigment. [Author's abstract.]

Rasmussen, A. T. THE MITOCHONDRIA IN NERVE CELLS DURING HIBERNATION AND INANITION IN THE WOODCHUCK (*Marmota monax*). [Journal of Comparative Neurology, Vol. 31, pp. 37-49, Oct., 1919.]

The marked functional changes taking place during the onset and again at the termination of hibernation in mammals would seem to offer an excellent opportunity for the study of the relationship of various structural elements to cellular activity. Upon this assumption there was made a quantitative determination of the number of mitochondria (chondriosomes) in the principal nerve cells of the woodchuck at three different periods—(1) just before the onset of hibernation, (2) during the latter part of dormancy, and (3) after waking up and becoming active in the spring. No food or water was available either during or subsequent to winter-sleep. In obtaining the tissue, the blood was first washed out by gradual profusion with oxygenated Locke's fluid and numerous short hemorrhages while the animal was still alive. Regaud's neutral formalin and potassium bichromate solution was then allowed to perfuse the entire animal for an hour. After further chromation the tissue was carried into hard paraffin, sections cut 2 and 3 μ thick, and stained in a 20 per cent. solution of acid fuchsin in aniline water and differentiated with a 1 per cent. aq. sol. of methylgreen, as carried out by E. V. Cowdry. The following cells were studied: spinal ganglion cells, motor cells of ventral and lateral horns of spinal cord, cells of nucleus gracilis and of nucleus cuneatus, Purkinje cells of cerebellum,