Such findings so aptly provided for an intracellular occurrence of lipochrome that the working hypothesis for the nerve cell was based on them. The point that first focused our attention on the probable carotinoid identity of nerve cell lipochrome was its absence in the rabbit and dog. The rabbit and dog have colorless fat. Man and cattle, known to show intracellular lipochrome, have colored fat.

Verification was first sought in the chicken. With the use for the most part of Palmer's chickens above described, two series were run, the one lacking carotinoid containing food from birth, the other carotinoid fed. The carotinoid feeding ranged from a one week's introduction in a bird hitherto carotinoid free to a lifelong natural pigment food in others. In one half of the chickens of both series the factor of depression by heat, phosphorus, morphine or a rice flour diet was introduced to cover the side of disease.

The results were uncomplicated. Both normal and depressed chickens on any carotinoid diet showed the presence of the characteristic yellow pigment in all nerve cells. The carotinoid-free chickens lacked such a pigment in demonstrable amount.

However, this physiological demonstration of the introduction of carotinoid pigment demands for completeness the support of microchemistry. The question at once arises if the pigment introduced in nervous and other body tissues is identical with the lipofuscin, "wear-and-tear," fat-holding pigment described for the nerve and other somatic cells as specific. While it is true that the micro-chemistry of the lipochrome pigments is superficial, which is the reason that the analysis by that means has hitherto failed, yet it must be emphasized that it has become quite sufficient to demonstrate this identity. The application of this chemistry was more simple in our problem when following a means of providing or withdrawing the pigment at will. The yellow pigment introduced in nerve cells and the chicken skin, and the pigment of the carrot in frozen sections give the fat stains, the oxidation and decolorization by hydrogen peroxid and ferric chlorid, the fat stains after oxidation, and the rapid solubilities in fat solvents in common with a supposed lipofuscin; while the most characteristic test for lipofuscin, the Nile blue stain of Hueck, equally applies to known lipochrome before and after its oxidation. This supposed metabolic pigment of the nerve cell is then identical with a true lipochrome.

Finally in corroboration of the species difference in the transferrence of the carotinoid pigment from plants, the cow as well as the chicken exhibits it in nerve cells, while swine with their colorless fat line up with the rabbit and dog in a complete absence. Man, who is best known to exhibit lipochrome, is also known to carry carotinoids in his blood serum, and has colored fat. The consistency is complete.

The lipochrome pigment of the nerve cell is therefore a plant carotinoid, derived from the food, but limited to such species as carry the carotinoids in the blood serum. The conception of it as a "wear-and-tear" pigment falls to the ground with its demonstration as an exogenous and fortuitous pigment. The melanin of the nerve cell is a true metabolic pigment, derived from nuclear materials and produced by chronic depression. Because of this, the conception of a "wear-and-tear" pigment is to be transferred to the melanin, as conditioned by agencies without the cell, with a restriction to the abnormal.

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