

Biochemical basis of lipofuscin, ceroid, and age pigment-like fluorophores

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Abstract

Serious studies of the formation mechanisms of age-related pigments and their possible cellular influence have been hampered for a long time by discrepancies and controversies over the definition, fluorescence emission, origin, and composition of these pigments. This review discusses several critical controversies in this field and lay special emphasis on the cellular and biochemical reactions related to the formation mechanisms of lipofuscin, ceroid, advanced glycation end-products (AGEs), and age pigment like fluorophores (APFs). Various amino compounds and their reaction with secondary aldehydic products of oxygen free radical-induced oxidation, particularly lipid peroxidation, are important sources of the fluorophores of ceroid/lipofuscin, which progressively accumulate as a result of phagocytosis and autophagocytosis of modified biomaterials within secondary lysosomes of postmitotic and other cells. Lipofuscin is the classical age pigment of postmitotic cells, while ceroid accumulates due to pathologic and experimental processes. There are good reasons to consider both ceroid and lipofuscin as materials of the same principal origin. The age-related intracellular fluorophores of retinal pigment epithelium (RPE) seems to represent a special class of lipofuscin, which partly contains derivatives of retinoids and carotenoids. Saccharide-originated fluorophores, principally AGEs formed during glycation/Maillard reactions, may be mainly responsible for the extracellular fluorescence of long-lived proteins, such as collagen, elastin, and lens crystalline. Although lipofuscin, ceroid, AGEs, and APFs can be produced from different types of biological materials due to different side reactions of essential biology, the crosslinking of carbonyl-amino compounds is recognized as a common process during their formation.