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Kaushik A.

LIPOFUSCIN: A PIGMENT OF AGEING AND AGE-RELATED DISEASES

Tutor: PhD, associate professor Prinkova T.Yu.
Department of Biological Chemistry
Belarusian State Medical University, Minsk

This paper provides an overview of lipofuscin, a yellow-brown pigment that accumulates in cells with age and has been linked to age-related decline in cellular functions. The study covers the chemical composition and structure of lipofuscin, as well as the mechanisms of its formation and accumulation. The role of lipofuscin in age-related diseases such as Alzheimer's disease is also discussed.

The aim of the study is to study the structure, properties, and mechanisms of formation, accumulation, and degradation of lipofuscin, to identify the role of lipofuscin in several pathological conditions and its effects on different tissues, to study role of macromolecules in lipofuscin formation, possible approaches to treatment by understanding the biology of lipofuscinogenesis with ageing.

Lipofuscin is a yellowish brown, pigmented, insoluble granule that contains protein and lipid and accumulates in cells as part of the normal aging process or sometimes in association with diseased states. A chemical analysis of lipofuscin granules revealed the presence of protein, lipid 30–70%, carbohydrates account for 4–7%. Metals such as iron, copper, and zinc have been found to increase the formation and accumulation of lipofuscin.

Oxidized proteins may not undergo appropriate proteolytic digestion but instead, cross-link with one another or form extensive hydrophobic bonds. It is believed that the cross-linked proteins react further with other cellular components, forms lipofuscin. It is not an inert waste product, but rather an active component influencing the cellular metabolism, which is especially relevant in senescent cells. Lipofuscin is cytotoxic because of its ability to incorporate redox-active transition metals. This also correlates between lipofuscin acculumalation and ageing as proteins are the main target of free radicals. This type of oxidation increases with age. Oxidized proteins are not functional and they are repaired during digestion to peptides or free amino acids in the proteasome or are not removed in the process of exocytosis. With age, these mechanisms weaken. Hence, the oxidized proteins accumulate in cells and cause tissue damage and deposition of lipofuscin.

Lipofuscin plays a significant role in various pathological conditions, some of which are explained like in neurodegeneration, diabetic encephalopathy, macular degeneration, Parkinson`s and Alzeheimer`s disease. Antioxidants such as vitamin E, vitamin C, and coezyme Q can help to reduce oxidative stress and prevent or slow the accumulation of lipofuscin in cells. These compounds work by neutralizing free radicals and reactive oxygen species, which can damage cellular components and contribute to the formation of lipofuscin. Beside there are some certain drugs that have been studied for their potential to decrease lipofuscin accumulation in the body.

Our study sheds light on the structure, properties, and mechanisms of formation and accumulation of lipofuscin, a pigment associated with aging and several pathological conditions. The complex composition of lipofuscin and the diverse pathways leading to its formation make it a challenging target for therapeutic intervention. However, the growing evidence of its role in cellular dysfunction and disease progression underscores the importance of further research aimed at elucidating the molecular mechanisms of lipofuscinogenesis and identifying potential strategies for its clearance or prevention. By deepening our understanding of the biology of lipofuscin, we may ultimately pave the way for novel therapies and interventions to combat age-related disorders.