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ORIGINAL PAPER

LIPOFUSCIN AND CEROID PIGMENTS: CELLULAR HALLMARK OF CEREBRAL SENESCENCE

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Abstract. Lipopigments (LPs) - lipofuscin and ceroid - are the main marker of brain vulnerability, distress, aging and connected pathology. Lipofuscin is the basic feature of cellular senescence, as ceroid is the cummulation product of aggressive external (environmental) and/or intrinsic (mainly genetic) factors. During ontogenesis, neuronal LPs progressively accumulate, as a time dependent phenomenon. In the aged neurons, LPs are present in all cellular compartiments: massively in every perikaryon areas and dendrites, also in axons, and even in terminal buttons. They constantly coexist and are significantly correlate with important changes in nerve cell biochemistry and morphology, such as neuronal loss, decrease in the surface/volume of neurosoma, dendritic aberrations, simplifications and destructions, axonal enlargements to meganeurites, considerably reduction of cortical myelin, and synapses loss. Moreover, neuronal LP accumulations coexist with glial LP storages, in all types of glia (astrocytes, oligodendrocytes, but especially in microglia). Glial systems play an important role in collecting of neuronal LPs. Owing to their transporting properties, and migration capacity of microglia, glial cells deposit the LP clusters in pericapillary areas. Thus, LP conglomerates appear in the whole nervous tissue, from neurons to perineuronal glia, neuropil, pericapillary glia and endothelial cells, realizing specific patterns of LP architectonics. Direct, causal interrelations, critical LP concentrations, which generate cascades of negative subcellular events, and indirect, associative impairment correlations determine characteristic neuropathological aging profiles. These specific and associate negative neuropathologic consequences of LP accumulations have multiple and detrimental impacts on neuron and glia homeostasis, from neurono-glial function to central nervous system physiology.

Key words: accumulations and storages of neuronal and glial lipopigments, causal and associate damages correlated with lipopigments, neuropathological aging profiles.