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Protein accumulation in neurodegenerative diseases – A role of ubiquilin-1

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Different neurodegenerative diseases, such as Alzheimer's (AD), Parkinson's, and Huntington's disease, share several common features in their pathogenesis. One of these is the abnormal accumulation and aggregation of disease-specific proteins in intracellular or extracellular lesions in specific brain areas. It has been suggested that deficiencies in protein quality control and clearance mechanisms may lead to the abnormal accumulation of proteins in these diseases. Several proteins that are known to associate with neurodegenerative diseases have been identified as central regulators of protein quality control and clearance. We have characterized the role of one of these proteins, the AD-associated protein ubiquilin-1, in the regulation of protein levels and degradation. Our results implicate that specific ubiquilin-1 transcript variants regulate the levels and proteasomal targeting of presenilin-1 (PS1), a key protein in AD pathogenesis. Alternatively, in the case of excessive PS1 accumulation, ubiquilin-1 targets PS1 to the aggresome-autophagosome pathway for disposal. The mechanisms of ubiquilin-1-mediated aggresomal targeting of PS1 and potential effects of this on PS1 function will be discussed.

Biography

Annakaisa Haapasalo completed her PhD degree at the University of Kuopio, Finland and conducted her postdoctoral training at MGH/Harvard Medical School, Boston, MA USA. Currently she holds a position as an Adjunct Professor at the University of Eastern Finland. She studies the molecular and cellular pathways that are involved in the pathogenesis of Alzheimer's disease in different in vitro and in vivo models. Her research is funded by the Health Research Council of the Academy of Finland. She serves as an Associate Editor in the Journal of Alzheimer's Disease and American Journal of Neurodegenerative Disease.

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