Revealing the complex nature of amyloid beta and its relation to dementia

Alzheimer disease (AD) is the most common type of dementia and characterized by the accumulation of amyloid plaques in the extracellular space of the brain parenchyma. Amyloid plaques consist of amyloid beta peptides (Aβ). Amyloid pathology can also be involved in other types of dementia, either as a driving force or as a coexisting pathology. In this thesis was investigated the Aβ peptide content in different amyloid deposits, types of dementia and regions, with the goal to improve our understanding of amyloid pathology in dementia. The studies presented here reveal a different Aβ peptide pattern in individuals with amyloid pathology, but cognitively unaffected, compared with AD patients, who suffer from cognitive decline. Moreover, vascular Aβ contribution, due to cerebral amyloid angiopathy, differs from amyloid plaque Aβ contribution. For other groups with plaque pathology, such as Down syndrome, dementia with Lewy bodies, and Parkinson's disease dementia, there are minor differences in the Aβ peptide pattern compared with AD. In this work, the Aβ content of the protofibril/oligomeric forms, a major anti-amyloid therapeutical target, is also revealed. This thesis can be the beginning of a deeper understanding of the complex nature of amyloid pathology and its contribution to dementia.
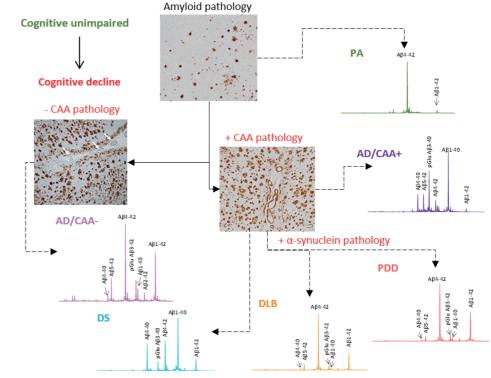
Eleni holds a BSc in Biochemistry and Biotechnology from the University of Thessaly, Greece. Driven by her passion for protein science, she moved to Sweden to obtain an MSc in Applied Biotechnology at Uppsala University. During her MSc, she fell in love w

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Eleni Gkanatsiou

**SAHLGRENSKA ACADEMY
INSTITUTE OF NEUROSCIENCE AND PHYSIOLOGY**

UNIVERSITY OF
GOTHENBURG