Investigating a-synuclein dependent neurodegeneration in two novel transgenic mouse lines modeling increased aggregation and lack of age-dependent trophic a-synuclein signalling

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Denmark

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Investigating a-synuclein dependent neurodegeneration in two novel transgenic mouse lines modeling increased aggregation and lack of age-dependent trophic a-synuclein signalling

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3

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Research Abstract

Alpha-synuclein’s (a-syn) native state changes in the course of neurodegenerative synucleinopathies where it ultimately gets deposited in intracellular Lewy-like inclusions as filamentous aggregates. Soluble oligomeric aggregates are formed in the process of aggregation and are considered toxic species that cause a range of cell-autonomous effects and tissue responses thereby being responsible for the spreading degeneration of nerve cells characterizing the synucleinopathies. We have demonstrated that p25a stimulates aggregation of a-syn so we generated a mouse model of enhanced oligomer formation by expressing human p25a in forebrain neurons together with human a-syn from the Thy1 promoter (model I – Rosa26-p25a/Nex-Cre/Thy1-AS).

Further information available at:

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