Alterations in autophagy in frontotemporal lobar degeneration: identifying new targets for therapeutic intervention

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Research Abstract

The term Frontotemporal lobar degeneration (FTLD) encompasses a group of disorders characterised by degeneration of the frontal and temporal lobes of the brain resulting in profound behavioural and personality alterations. FTLD presents with different genetic, pathological and clinical features, butalways with distinct abnormal protein accumulations. Autophagy is the mechanism responsible for clearing old and damaged proteins from cells, and

the system has been reported to be defective in Alzheimer’s disease (AD) and Parkinson’s disease (PD). Therapeutic strategies that restore autophagy in AD and PD have in some disease models demonstrated significant rescue effects, but in others have been shown to worsen symptoms and pathology, suggesting disease and model specific variation in autophagy impairment. Despite many FTLD genetic mutations/risk factors being known to encode autophagy associated proteins, the role of impaired autophagy in FTLD is poorly understood, with no detailed studies in human brain tissue. This study aims to investigate alterations in autophagic pathway recruitment, activity and regulation in human FTLD brain tissue and identify novel therapeutic targets within these pathways. Understanding the subtle, disease specific variations in autophagy deficits will lead to better targeted therapeutic approaches more likely to avoid negative therapeutic effects reported in other investigations.

Further information available at:

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