C-Abl Tyrosine Kinase Signaling: A New Player in AD Tau Pathology

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Abstract

Hyperphosphorylated tau is a cardinal feature of Alzheimer's disease (AD) pathology. The deregulation of kinases that phosphorylate tau can alter normal tau-related processes, including microtubule dynamics, growth cones, and axonal transport, and induce tau aggregation in paired helical filaments. Here we discuss the possible roles of the Abl family of tyrosine kinases, which are essential regulators of cytoskeleton cellular signaling cascades, in AD tau pathology and how the physiological roles of Abl kinases could be connected with the cytoskeletal alterations induced by Aβ aggregates and AD progression.

Keywords: Aging; Alzheimer; BMI; MET; TELE; TICS; c-Abl; dementia; genetic allele; genetic epidemiology; memory impairment; metabolism; obesity; phosphorylation; predisposing effect; tau; twins.