Multifarious roles of mTOR signaling in cognitive aging and cerebrovascular dysfunction of Alzheimer's disease

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Age-related cognitive failure is a main devastating incident affecting even healthy people. Alzheimer's disease (AD) is the utmost common form of dementia among the geriatric community. In the pathogenesis of AD, cerebrovascular dysfunction is revealed before the beginning of the cognitive decline. Mounting proof shows a precarious impact of cerebrovascular dysregulation in the development of AD pathology. Recent studies document that the mammalian target of rapamycin (mTOR) acts as a crucial effector of cerebrovascular dysregulation in AD. The mTOR contributes to brain vascular dysfunction and subsequence cerebral blood flow deficits as well as cognitive impairment. Furthermore, mTOR causes the blood?brain barrier (BBB) breakdown in AD models. Inhibition of mTOR hyperactivity protects the BBB integrity in AD. Furthermore, mTOR drives cognitive defect and cerebrovascular dysfunction, which are greatly prevalent in AD, but the central molecular mechanisms underlying these alterations are obscure. This review represents the crucial and current research findings regarding the role of mTOR signaling in cognitive aging and cerebrovascular dysfunction in the pathogenesis of AD.

Alzheimer's disease

blood?brain barrier

cerebrovascular dysfunction

cognitive aging

mTOR

mammalian target of rapamycin

Alzheimer disease

blood brain barrier

brain blood flow

cerebrovascular accident

cerebrovascular disease

cognitive aging

enzyme activity

human

molecular pathology

mTOR signaling

nerve cell plasticity

neuropathology

neurovascular coupling

nonhuman

Review