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Brain signatures of metabolic syndrome and new treatment options for cognitive decline

Metabolic Syndrome (MetS) epidemic is spreading around the world. MetS components (type 2 diabetes (T2DM), obesity and hypertension) have been shown to alter regional brain perfusion, vascular reactivity and micro- and macroscopic structural integrity. These cumulative effects subsequently result in functional decline of cognition and mobility. T2DM leads to gray matter (GM) atrophy and demyelination of white matter (WM) cognitive pathways in fronto-temporal and parietal networks, thus accelerating brain aging by ~ 5 years. In T2DM, worse performance on verbal fluency, learning and memory correlated with loss of WM microstructural integrity in the angular gyrus. An increasing BMI has been also linked to GM atrophy and an increase in WM fractional anisotropy in corpus callosum and other regions. Hypertension-related micro-infarcts and WM hyperintensities are signatures of slow gait speed and balance impairments. Central insulin plays a key role as neuromodulator of astrocyte-neuron signaling, cognition, homeostasis and food intake etc. In MetS, brain insulin resistance, inflammation and micro-vascular disease share a common pathophysiology of altered metabolism, hypoperfusion and WM degeneration. Intranasal insulin (INI) delivery directly across the blood-brain barrier has shown promise for treatment of cognitive and memory impairment. INI improves perfusion, functional connectivity and cognition in older adults and T2DM. New treatments targeting central effects (demyelination, hypoperfusion and inflammation) with direct delivery into the brain are needed to prevent and treat MetS-related cognitive and functional decline and dementia.

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