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Intranasal insulin attenuate signs of Alzheimer's disease following chronic hypoxia

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Alzheimer's disease (AD) is a metabolic neurodegenerative disease featured by cerebrovascular dysfunction in addition to cognitive decline. Amyloid β ($A\beta$) plaques followed by up-regulation of amyloid precursor protein (APP) and seladin-1 down-regulation, as well as insulin signaling impairment are associated with this disease. This study was designed to evaluate the effect of insulin on Alzheimer's signs induced by chronic hypoxia. 24 male rats were randomly divided into four groups: control (C), sham (Sh), hypoxia (H), hypoxia + insulin (HI) and were exposed to hypoxic chamber (8% O₂, 92% N₂) for 30 days (four hours/day) in H and HI groups. Pro-inflammatory cytokines and insulin receptor substrate (IRS-1) in sera were measured on day 30 after hypoxia period. Intranasal insulin administration was used as a neuroprotective and antidiabetic drug. Spatial learning and memory were analyzed using the Morris water maze task. Amyloid precursor protein gene (APP) and seladin-1 gene expression were studied in the hippocampus by real time-PCR. TNF- α , IL-1 β and IRS-1 had significant magnification in H group compared with C and Sh groups ($p < 0.05$). Insulin improved Alzheimer's signs such as seladin-1 fallen, APP risen gene expression and memory impairment. In conclusion, we indicate that chronic hypoxia mediates AD pathogenesis and using insulin hormone as a neuroprotective and antidiabetic drug could be beneficial in neurodegenerative damage induced by hypoxia.

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