Nanodelivery of cerebrolysin reduces depressive stress induced exacerbation of Alzheimer's disease brain pathology following amyloid-beta peptide infusion

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Previous studies show that stressful situations alone induce breakdown of the blood-brain barrier (BBB) and neuronal damages that is long lasting. Thus, a possibility arises that breakdown of the BBB in stress could play critical roles in development of AD. In present innovation we demonstrated that AD induced brain pathology caused by amyloid beta peptide (A?P) infusion is exacerbated in rats subjected to chronic hypertension either induced by repeated immobilization for 2 h for 1 week or renal hypertension produced by 2 Kidney 1 clip (2K1C) method. Chronic hypertension (CHR) induced marked BBB breakdown to Evans blue albumin and radioiodine tracers in the cerebral cortex, hippocampus, thalamus, hypothalamus, caudate nucleus, cerebellum and brainstem from the naive rats. Infusion of A?P in these CHR rats further enhanced the BBB breakdown to protein tracers by several-folds and aggravation of neuronal damages, astrocytic activation and brain swelling. It appears that TiO2-nanwored delivery of cerebrolysin has superior neuroprotective effects in this AD model as compared to PLGA-delivery of identical doses of cerebrolysin. Taken together our observations are the first to demonstrate that CHR exacerbates AD brain pathology and nanodelivery of cerebrolysin has superior neuroprotective effects, not reported earlier.

Alzheimer's disease

Brain pathology

Cerebrolysin

Chronic hypertension

Immobilization stress

Neuroprotection

PLGA-nanoprticles

TiO2 nanowired delivery

Glycoproteins

Neurodegenerative diseases

Pathology

Peptides

Rats

Alzheimer's disease

Brain pathologies

Cerebrolysin

Chronic hypertension

Neuroprotection

PLGA-nanoprticles

TiO2 nanowired delivery

Brain