Adiponectin is protective against oxidative stress–induced cytotoxicity in amyloid-beta neurotoxicity

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Beta-amyloid (Aβ) neurotoxicity is important in Alzheimer's disease (AD) pathogenesis. Aβ neurotoxicity causes oxidative stress, inflammation, and mitochondrial damage resulting in neuronal degeneration and death. Oxidative stress, inflammation, and mitochondrial failure are also pathophysiological mechanisms of type 2 diabetes mellitus (T_2DM) which is characterised by insulin resistance. Interestingly, T_2DM increases risk to develop AD which is associated with reduced neuronal insulin sensitivity (central insulin resistance). We studied the potential protective effect of adiponectin (an adipokine with insulin-sensitising, anti-inflammatory, and anti-oxidant properties) against Aβ neurotoxicity in human neuroblastoma cells (SH-SY5Y) transfected with the Swedish amyloid precursor protein (Sw-APP) mutant, which overproduced Aβ with abnormal intracellular Aβ accumulation. Cytotoxicity was measured by assay for lactate dehydrogenase released upon cell death and lysis. Our results revealed that Sw-APP–transfected SH-SY5Y cells expressed both adiponectin receptor 1 and 2, and had increased AMP-activated protein kinase (AMPK) activation and enhanced nuclear factor–kappa B (NF-κB) activation compared with control empty-vector–transfected SH-SY5Y cells. Importantly, adiponectin at physiological concentration of 10 µg/mL protected Sw-APP–transfected SH-SY5Y cells against cytotoxicity under oxidative stress induced by hydrogen peroxide. This neuroprotective action of adiponectin against Aβ neurotoxicity-induced cytotoxicity under oxidative stress involved (1) AMPK activation mediated via the endosomal adaptor protein APPL1, and possibly (2) suppression of NF-κB activation.

Association between age and rehabilitation outcome in frail older adults

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Background: The association between age and rehabilitation outcome in frail older adults is controversial.

Methods: Patients from 2004 to 2012 of the geriatric day hospital (GDH) of Fung Yiu King Hospital were reviewed in this retrospective cohort study. Age, gender, place of residence, co-morbidities, blood test results (serum creatinine, albumin, haemoglobin), functional status using functional independence measure (FIM), cognitive status, body mass index, and referred diagnosis were collected. Age was stratified into groups (<70, 71-75, 76-80, 81-85, 86-90, and >90 years). Outcome measurement was change of FIM after receiving rehabilitation in GDH.

Results: A total of 833 GDH patients (503 women, 330 men; mean age 80.0±7.1 years) of age 65 years and above were included. Median change of FIM was 4, interquartile range (IQR) was 0-9. There was no significant difference in FIM across different age-groups (age <70: median 5, IQR 2-11; age 71-75: median 5, IQR 0-10; age 76-80: median 4, IQR 0-9; age 81-85: median 3, IQR 0-9; age 86-90: median 3, IQR 0-8; age >90: median 4, IQR 0-8.5; P=0.42).

Conclusion: Age has no influence on rehabilitation outcome in frail Chinese older adults. Older patients should not be excluded from rehabilitation programmes because of increasing age.