



<b>Title</b>	<b>Adiponectin is protective against oxidative stress-induced cytotoxicity in amyloid-beta neurotoxicity</b>
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## Adiponectin is protective against oxidative stress-induced cytotoxicity in amyloid-beta neurotoxicity

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Beta-amyloid (A $\beta$ ) neurotoxicity is important in Alzheimer's disease (AD) pathogenesis. A $\beta$  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<sub>2</sub>DM) which is characterised by insulin resistance. Interestingly, T<sub>2</sub>DM 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 A $\beta$  neurotoxicity in human neuroblastoma cells (SH-SY5Y) transfected with the Swedish amyloid precursor protein (Sw-APP) mutant, which overproduced A $\beta$  with abnormal intracellular A $\beta$  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- $\kappa$ B) activation compared with control empty-vector-transfected SH-SY5Y cells. Importantly, adiponectin at physiological concentration of 10  $\mu$ g/mL protected Sw-APP-transfected SH-SY5Y cells against cytotoxicity under oxidative stress induced by hydrogen peroxide. This neuroprotective action of adiponectin against A $\beta$  neurotoxicity-induced cytotoxicity under oxidative stress involved (1) AMPK activation mediated via the endosomal adaptor protein APPL1, and possibly (2) suppression of NF- $\kappa$ B 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 $\pm$ 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.