

Anthocyanins attenuate lipid peroxidation and mitochondrial dysfunction generated by APP^{swe} mutation or by chemical inhibition of mitochondrial complex I.

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Medox- a mix of antioxidants

Medox is a combination of anthocyanins obtained by purification from black currants (*Ribes nigrum* L.) and from bilberries (*Vaccinium myrtillus*).

By crossing the brain blood barrier, anthocyanins may modulate signalling pathways in the central nervous system in brain regions important for learning and memory. Pre-clinical research has suggested the health benefits of dietary anthocyanins-rich extracts in reducing incidence of Alzheimer disease (AD).

At the mitochondrial level, we demonstrated Medox anthocyanins protect from complex I inhibition and APP^{swe} mutation through modulation of the mitochondrial fission and fusion pathways.

In this study, we found that anthocyanins help reduce cytotoxicity via enhancement of the antioxidant capacity and inhibition of the lipid peroxidation.

Aim

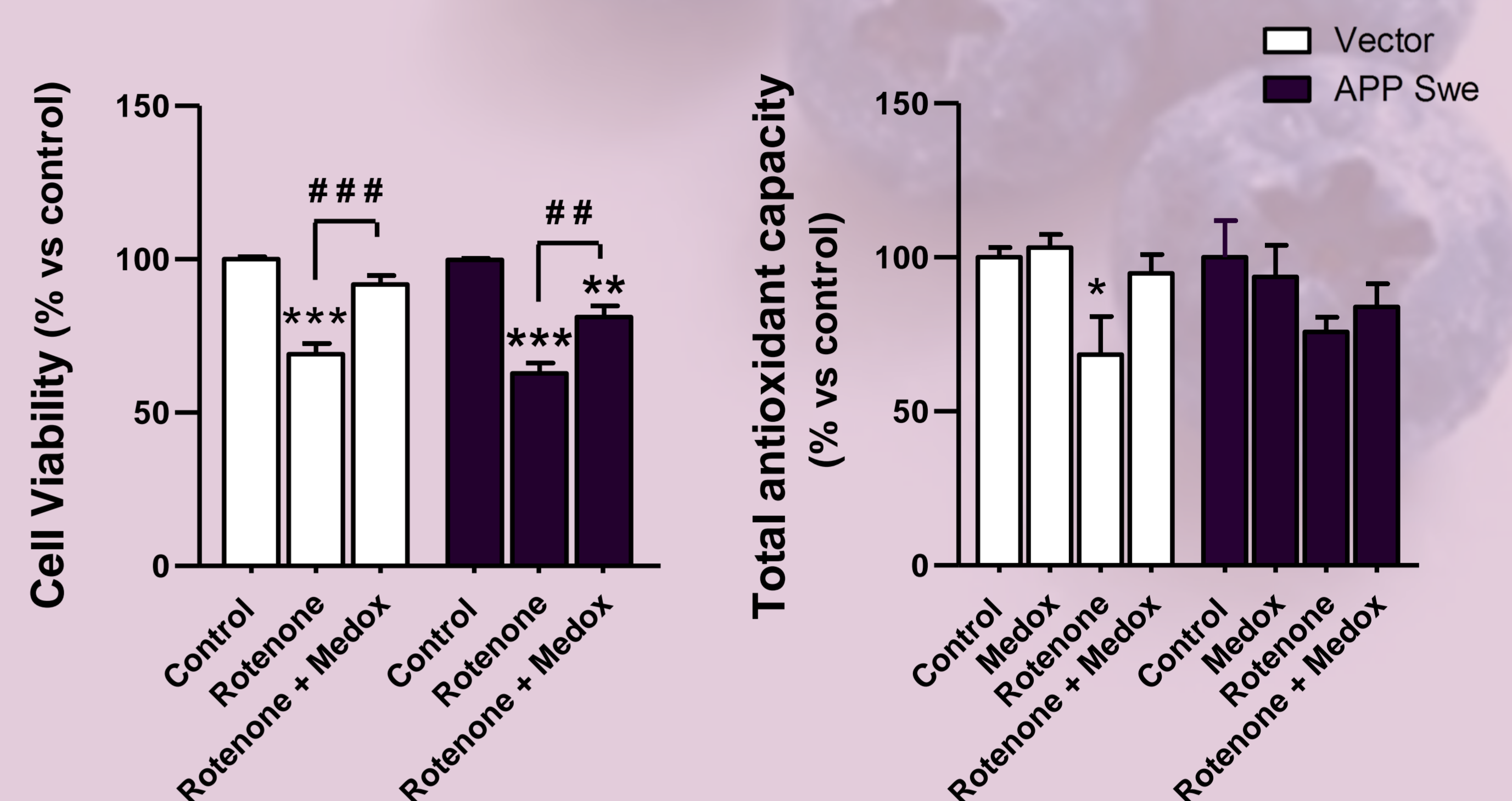
We examined the ability of nutraceutical Medox anthocyanins, rich in cyanidin 3-O-glucoside (C3G) 3-O-b-glucosides and delphinidin 3-O-glucoside (D3G) to counteract the induced toxicity of complex I inhibition and/or amyloid- β peptide (A β) in vitro.

Method

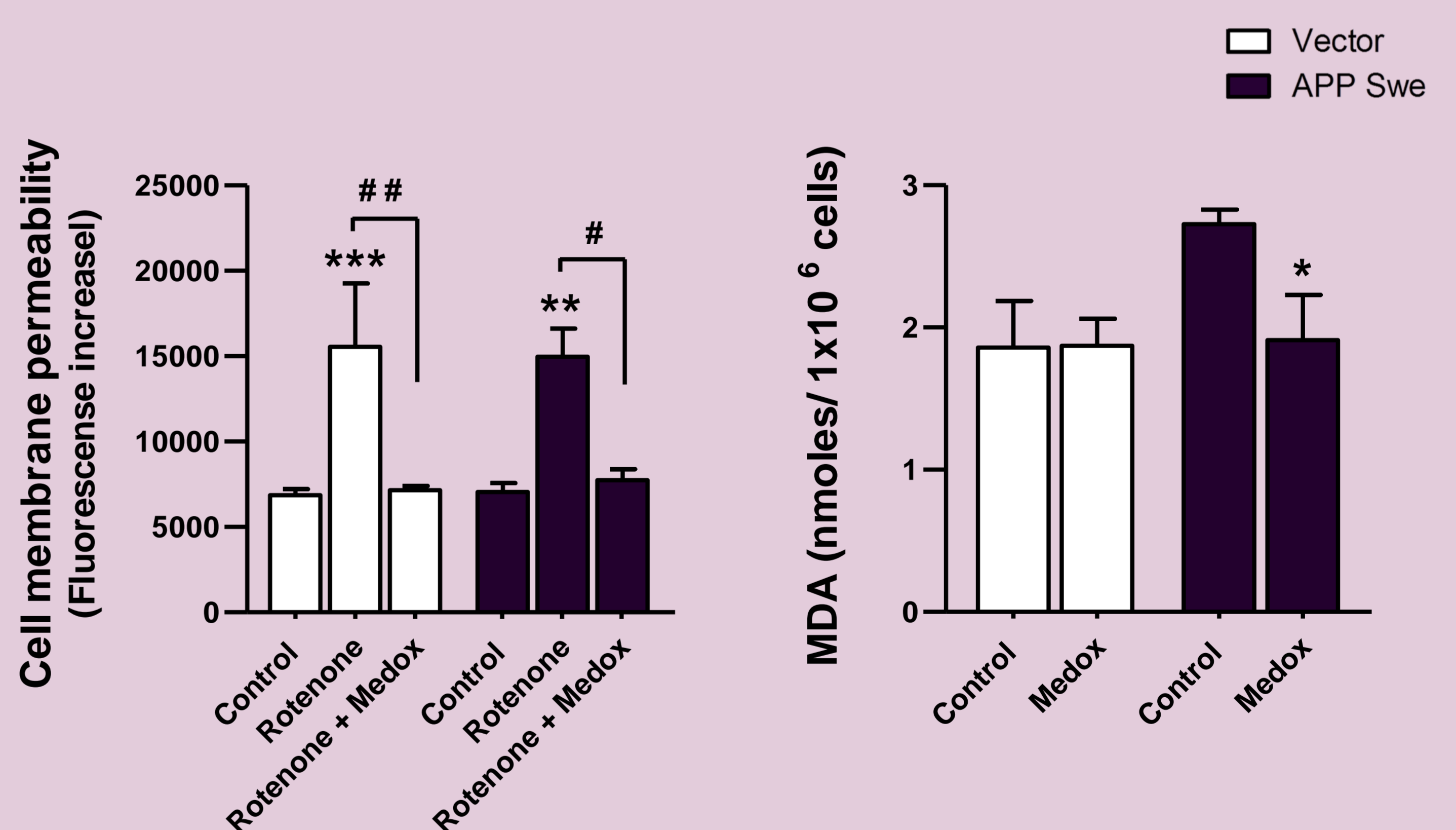
We used SH-SY5Y neuroblastoma cells stably transfected with APP Swedish K670N/M671L double mutation (APP^{swe}) or with the empty vector and treated with rotenone (100 nM) in presence or absence of Medox anthocyanins (0.05 μ g/ml; containing 20 nM C3G and 30 nM D3G) for 18h. In this study, we evaluated cell viability, lipid peroxidation (MDA assay) and membrane permeability of cell membranes, and total antioxidant capacity (TAC) status.

Results

Medox anthocyanins counteract the rotenone-induced cytotoxicity and recover the antioxidant capacity.



Medox anthocyanins prevent cell membranes from the rotenone-induced effects and amyloid beta-peptide-induced lipid peroxidation.



Future perspectives

Study whether Medox anthocyanins may slow down the cognitive decline in middle-aged and older individuals with pre-dementia AD or mild AD dementia, and in coronary heart disease.

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