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Astaxanthine secured apoptotic death of PC12 cells induced by beta-amyloid peptide 25-35: its molecular action targets.

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Abstract

Astaxanthine (ASTx) is a novel carotenoid nutraceutical occurring in many crustaceans and red yeasts. It has potent antioxidant, photoprotective, hepatodetoxicant, and anti-inflammatory activities. Documented effect of ASTx on treatment of neurodegenerative disease is still lacking. We used the beta-amyloid peptide (Abeta) 25-35-treated PC12 model to investigate the neuron-protective effect of ASTx. The parameters examined included cell viability, caspase activation, and various apoptotic biomarkers that play their critical roles in the transduction pathways independently or synergistically. Results indicated that Abeta25-35 at 30 microM suppressed cell viability by 55%, whereas ASTx was totally nontoxic below a dose of 5.00 microM. ASTx at 0.1 microM protected PC12 cells from damaging effects of Abeta25-35 in several ways: (1) by securing the cell viability; (2) by partially down-regulating the activation of caspase 3; (3) by inhibiting the expression of Bax; (4) by completely eliminating the elevation of interleukin-1beta and tumor necrosis factor-alpha; (5) by inhibiting the nuclear translocation of nuclear factor kappaB; (6) by completely suppressing the phosphorylation of p38 mitogen-activated protein kinase; (7) by completely abolishing the calcium ion influx to effectively maintain calcium homeostasis; and (8) by suppressing the majority (about 75%) of reactive oxygen species production. Conclusively, ASTx may have merit to be used as a very potential neuron protectant and an anti-early-stage Alzheimer's disease adjuvant therapy.

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