

Abstract

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Docosahexaenoic acid supplementation of primary rat hippocampal neurons attenuates the neurotoxicity induced by aggregated amyloid beta protein(42) and up-regulates cytoskeletal protein expression.

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BACKGROUND: Alzheimer's disease (AD) is a progressive neurodegenerative disease characterized by extracellular deposits of fibrillar aggregates of amyloid-beta peptide (Abeta). Levels of docosahexaenoic acid (DHA, 22:6n-3), the major fatty acid component of the neuronal membrane, are reduced in the AD hippocampus.

OBJECTIVE: We hypothesized that hippocampal neurons with reduced DHA levels would be more susceptible to aggregated Abeta-induced death and that this might be overcome by increasing hippocampal neuronal DHA levels.

METHODS: Embryonic Day 18 rat hippocampal cells were cultured in neurobasal medium with B27 supplemented with 0-100 μ M DHA for 8 days, then were treated with 5 μ M aggregated Abeta(42) for 1 day.

RESULTS: We found that supplementation with 5-10 μ M DHA, which resulted in hippocampal neuron DHA levels of 12-16% of total fatty acids, was optimal for primary hippocampal neuronal survival, whereas supplementation with 5 or 25 μ M DHA attenuated aggregated Abeta(42)-induced neurotoxicity and protected hippocampal neurons, with 25 μ M DHA being more effective. DHA supplementation also resulted in significant up-regulation of expression of tyrosine tubulin and acetylated tubulin.

CONCLUSION: We suggest that hippocampal neuronal DHA levels may be critical for AD prevention by attenuating the neurotoxicity induced by Abeta and in maintaining hippocampal neuron survival.

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