



# **Diabetes: Chapter 13. The Anti-Oxidative Component of Docosahexaenoic Acid (DHA) in the Brain in Diabetes**

*Emma Arnal, María Miranda, Siv Johnsen-Soriano, Francisco J. Romero*

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The mechanisms underlying diabetic encephalopathy are only partially understood. This review tries to address the mechanisms of diabetes-induced cell and tissue damage in the brain, and discusses whether docosahexaenoic acid (DHA) could attenuate the degenerative changes observed in the diabetic brain. DHA, the main omega-3 fatty acid, is concentrated and avidly retained in membrane phospholipids of the nervous system. DHA is involved in brain and retina physiological functions, aging, and neurological and behavioral illnesses. Neuroprotectin D1 (NPD1), the first identified stereoselective bioactive product of DHA, exerts neuroprotection in models of experimental diabetes. Photoreceptor membranes display the highest content of DHA of any cell. Retinal pigment epithelial cells participate in the phagocytosis of the tips of photoreceptor cells (photoreceptor outer segment renewal). There is a DHA retrieval-intercellular mechanism between both types of cells that conserves this fatty acid during this process. NPD1 promotes the homeostatic regulation of the integrity of these two cells, particularly during oxidative stress, and this protective signaling may be relevant in retinal degenerative diseases. Moreover, neurotrophins are NPD1-synthesis agonists, and NPD1 content is decreased in the CA1 region of the hippocampus of Alzheimer's patients. Overall, NPD1 promotes brain cell survival via the induction of anti-apoptotic and neuroprotective gene expression programs that suppress neurotoxicity. Thus, NPD1 elicits potent cell-protective, anti-inflammatory, prosurvival and repair signaling.

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