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Lipid metabolites derived from ω-3 long chain polyunsaturated fatty acids promote choroidal neovessel regression

Kip M. Connor

Harvard Medical School, USA

Abstract

Age-related macular degeneration (AMD) is the primary cause of blindness in elderly individuals of industrialized countries, and has a projected 50% increase by the year 2020. There is an urgent need for new nutritional or pharmacological interventions that are safe over the long term for the treatment or prevention of AMD. Prospective clinical studies have suggested that dietary intake of ω-3 long-chain polyunsaturated fatty acids (LCPUFAs) may have a protective effect against AMD. Abnormal blood vessel growth (choroidal neovascularization (CNV)) is a hallmark of wet-AMD and leads to significant vision loss. This study sought to characterize the effects of dietary intake of ω-3 and ω-6 LCPUFAs in a mouse model of AMD. C57BL/6 mice were fed a diet enriched with either ω-3 or ω-6 LCPUFAs for 2 weeks. CNV was induced by photocoagulation using a 532-nm laser; and was evaluated by fluorescein angiography, optical coherence tomography, and choroidal flatmounts. Here we demonstrate that dietary supplementation of ω-3 LCPUFAs mediate choroidal neovessel regression. The serum lipid profiles in these mice promote the formation of anti-inflammatory eicosanoids and are enriched in DHA and EPA, the primary ω-3 LCPUFAs. Bioactive lipid metabolites, derived from ω-3 LCPUFAs, were identified as key mediators of disease resolution. Omega-3 LCPUFAs were also effective in dampening systemic inflammation and suppressed leukocyte recruitment to the disease site. A clear understanding of the mechanisms of these molecules in AMD could bring a major shift in our approach to disease treatment and prevention, since nutritional interventions are safe, inexpensive, and readily put into practice.

Biography

Kip Connor is an Assistant Professor of Ophthalmology at Massachusetts Eye and Ear Infirmary, Harvard Medical School. He received his Ph.D. degree from Albany Medical College in 2005. Dr. Connor did his post-graduate work at Children's Hospital Boston and Harvard Medical School, where he also obtained his first faculty position from 2005 to 2010. In 2010 Dr. Connor joined MEEI's Howe and Angiogenesis Laboratories at MEEI. Dr. Connor's current research focuses on the role of immunity and inflammation using animal models of ocular diseases such as age-related macular degeneration, retinopathies (diabetic retinopathy and retinopathy of prematurity), auto-immune uveitis, retinal detachment and retinal degenerations.