Neurobiol Aging. 2013 Feb;34(2):602-9. doi: 10.1016/j.neurobiolaging.2012.04.014. Epub 2012 May 16.

Age-related retinal inflammation is reduced by 670 nm light via increased mitochondrial membrane potential.

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Abstract

The mitochondrial theory of aging argues that oxidative stress, caused by mitochondrial DNA mutations, is associated with decreased adenosine triphosphate (ATP) production leading to cellular degeneration. The rate of this degradation is linked to metabolic demand, with the outer retina having the greatest in the body, showing progressive inflammation, macrophage invasion, and cell loss, resulting in visual decline. Mitochondrial function shifts in vitro after 670-nm light exposure, reducing oxidative stress and increasing ATP production. In vivo, it ameliorates induced pathology. Here, we ask whether 670 nm light shifts mitochondrial function and reduces age-related retinal inflammation. Aged mice were exposed to only five 90-second exposures over 35 hours. This significantly increased mitochondrial membrane polarization and significantly reduced macrophage numbers and tumor necrosis factor (TNF)-alpha levels, a key proinflammatory cytokine. Three additional inflammatory markers were assessed; complement component 3d (C3d), a marker of chronic inflammation and calcitonin, and a systemic inflammatory biomarker were significantly reduced. Complement component 3b (C3b), a marker of acute inflammation, was not significantly altered. These results provide a simple route to combating inflammation in an aging population with declining visual function and may be applicable to clinical conditions where retinal inflammation is a key feature.

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PMID:
22595370
DOI:
10.1016/j.neurobiolaging.2012.04.014
[PubMed - indexed for MEDLINE]