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Ginkgo biloba delays light-induced photoreceptor degeneration through antioxidant and antiapoptotic properties.

Chudhary M(1), Zhang C(2), Song S(1), Ren X(1), Kong L(1).

Author information: (1)Department of Histology and Embryology, College of Basic Medicine, Dalian Medical University, Dalian, Liaoning 116044, P.R. China. (2)Teaching Laboratory of Morphology, College of Basic Medicine, Dalian Medical University, Dalian, Liaoning 116044, P.R. China.

Intense exposure to artificial bright light increases the risk of retinal damage resulting in blurred vision and blindness. Long-term exposure to bright light elevates oxidative stress-induced apoptosis, which results in photoreceptor cell degeneration. However, to the best of our knowledge, the molecular mechanism associated with light-induced retinopathy remains unclear. In the present study, the mechanisms involved in light-induced oxidative stress and apoptosis were investigated along with the protective effects of Ginkgo biloba (EGb 761) in photoreceptor cell degeneration. EGb 761 was administered to mice at a dose of 50 or 100 mg/kg for 7 days prior to exposure to bright light (5,000 lux for 24 h). Furthermore, photoreceptor cell disorders were evaluated using electroretinogram (ERG) and H&E staining analyses. The expression levels of antioxidant genes and proteins ERK, thioredoxin (Trx) and nuclear factor erythroid 2-related factor 2 (Nrf-2) and the induction of apoptosis cytochrome c (Cyc), cleaved caspase-3 and Bax, were determined by reverse transcription-quantitative PCR and western blotting. ERG and histological analysis revealed that exposure to bright light induced functional and morphological changes to the photoreceptor cells. Exposure to bright light increased the levels of Cyc, cleaved caspase-3 and Bax, and decreased the levels of phosphorylated (p-) Erk, Nrf-2 and thioredoxin (Trx). However, treatment of mice with EGb 761 increased the expression levels of antiapoptotic (Bcl-2) and antioxidant (p-Erk, Trx and Nrf-2) proteins and decreased the expression levels of the apoptotic genes (Cyc, cleaved caspase-3 and Bax). Based on these findings, the present study suggested that prolonged exposure to light induces photoreceptor cell degeneration, where EGb 761 treatment may serve a therapeutic effect on the development of photoreceptor cell degeneration.

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