

Mitochondrial damage in the trabecular meshwork of patients with glaucoma

Izzotti A, Saccà SC, Longobardi M, Cartiglia C.

Department of Health Sciences, University of Genoa, Genoa, Italy. izzotti@unige.it

OBJECTIVES: To analyze the frequency of mitochondrial DNA (mtDNA) damage in patients with primary open-angle glaucoma. Oxidative damage plays a major role in glaucoma pathogenesis. Since no environmental risk factor for glaucoma is recognized, we focused our attention on mitochondria, the main endogenous source of reactive oxygen species.

METHODS: Mitochondrial damage was evaluated analyzing a common mtDNA deletion by real-time polymerase chain reaction in trabecular meshwork collected at surgery from 79 patients with primary open-angle glaucoma and 156 unaffected matched controls. In the same samples, polymorphisms of genes encoding for antioxidant defenses (GSTM1), repair of oxidative DNA damage (OGG1), and apoptosis (FAS) were tested.

RESULTS: Mitochondrial DNA deletion was dramatically increased (5.32-fold; $P = .01$) in trabecular meshwork of patients with glaucoma vs controls. This finding was paralleled by a decrease in the number of mitochondria per cell (4.83-fold; $P < .001$).
CONCLUSIONS: Obtained results indicate that mitochondrion is targeted by the glaucomatous pathogenic processes. Some subjects bearing adverse genetic assets are more susceptible to this event. Clinical Relevance Oxidative damage to the trabecular meshwork exerts a pathogenic role in glaucoma inducing mitochondrial damage and triggering apoptosis and cell loss. This issue may be useful to develop new glaucoma molecular biomarkers and to identify high-risk subjects.

<http://www.ncbi.nlm.nih.gov/pubmed/20547950>