



A Metabolomics Profiling of Glaucoma Points to Mitochondrial Dysfunction, Senescence, and Polyamines Deficiency

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Résumé en anglais	<p>Purpose: To determine the plasma metabolomic signature of primary open-angle glaucoma (POAG).</p> <p>Methods: We compared the metabolomic profiles of plasma from individuals with POAG ($n = 36$) with age- and sex-matched controls with cataract ($n = 27$). A targeted metabolomics study was performed using the standardized p180 Biocrates Absolute IDQ p180 kit with a QTRAP 5500 mass spectrometer. Multivariate analyses were performed using principal component analysis (PCA) and the least absolute shrinkage and selection operator (LASSO) method.</p> <p>Results: Among the 151 metabolites accurately measured, combined univariate and multivariate analyses revealed 18 discriminant metabolites belonging to the carbohydrate, acyl-carnitine, phosphatidylcholine, amino acids, and polyamine families. The metabolomic signature of POAG points to three closely interdependent pathophysiologic conditions; that is, defective mitochondrial oxidation of energetic substrates, altered metabolism resembling that observed in senescence, and a deficiency in spermidine and spermine, both polyamines being involved in the protection of retinal ganglion cells.</p> <p>Conclusions: Our results highlight a systemic and age-related mitochondrial defect in the pathogenesis of POAG.</p>
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Liens

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