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PROGRAM ABSTRACTS

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lenium, an essential micronutrient. Among other functions, Selenbps play an important role in modulating oxidative stress and inflammation in the CNS. Recent findings from our laboratory have revealed Selenbp1 as a translationally downregulated gene in retinal ganglion cells (RGCs) after neurotoxic injury. We are, therefore, interested in investigating the role of Selenbp1 in oxidative and metabolic RGC injury. Using a mouse model of retinal ischemia reperfusion (I/R) injury, we examined whether Selenbp1 alters retinal activity, neuroinflammation, and cell survival. Our results indicate significantly increased levels of Selenbp1 expressed in the retina 24h after ischemia when compared with non-induced samples. Immunostaining of retinal cross sections reveals that Selenbp1 predominantly resides in the nerve fiber layer (NFL) and ganglion cell layer (GCL), and that it co-localizes with RBPMS labeling 24h following I/R injury. Notably, Selenbp1 immunoreactivity was reduced in the GCL but remained in the NFL seven days after I/R injury. To study the influence of Selenbp1 on cell survival, we established an overexpression cell line that was exposed to the oxidative mitochondrial stressor 2-Methyl-1,4-napthoquinone (Menadione). Resulting cell viability of Selenbp1 overexpression was reduced compared to wild type control cells following oxidative damage. These changes in Selenbp1 expression and localization *in vivo*, combined with effects on cell viability, demonstrate that Selenbp1 may play an important role in retinal ganglion cell survival and function following injury.

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Is contrast sensitivity suitable for early detection of primary open-angle glaucoma?

Poster number: W-09

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Introduction
Primary open-angle glaucoma (POAG) is an optic neuropathy characterized by progressive damage of the optic nerve and retinal ganglion cells, leading to irreversible visual loss. An early diagnosis, with adequate treatment, can minimize the loss of vision. Patients are generally asymptomatic early in the disease, therefore diagnosing POAG at first stages is still a challenge. Since POAG is a leading worldwide cause of blindness, it is essential to explore potential tools for identifying the changes produced in the very early stage, before a permanent visual loss occurs.
Contrast sensitivity (CS) is a robust tool for evaluating functional vision. It is the inverse of the minimum contrast detected at a stimulus size (spatial frequency). Although there is broad evidence that CS is affected in moderate to advanced POAG, there is not enough information about the first stages which are critical for early diagnosis. Therefore, to investigate if functional changes measured by CS could provide evidence of early deficits caused by POAG is especially important.

Objectives
The main objective was to assess CS for detecting visual changes at early stages of POAG.

Methods
CS was measured using sinusoidal gratings of 4 cycles/degree. First, foveal and peripheral CS were assessed in suspected POAG patients age-matched with healthy control subjects. Second, foveal CS was assessed in early POAG patients age-matched with the suspected POAG group. Analyses were carried out considering two age ranges: Under and Over 50 years of age.

Results
Peripheral CS was decreased in older POAG suspect patients compared to the control group. Foveal CS was reduced in POAG suspect participants and in early POAG patients, both compared to the control group and for the two age ranges. For the older patients, foveal CS in early POAG was lower than for POAG suspects. Foveal CS was correlated with age in suspect POAG, early POAG, and healthy control groups. Also, it was correlated with cup-disc ratio only in early POAG patients. Finally, foveal CS differentiated suspect (under 50) and early POAG (under and over 50) patients from control individuals with fair diagnostic accuracy.

Conclusion
CS is affected in both groups, patients with a high risk of developing POAG and with early POAG diagnosis. We argue that early visual damage may occur before structural changes can be detected. Foveal CS could be a fair discrimination tool for early POAG and young suspect patients.

Abstract ID: 536

Transmembrane immune receptor CD300LF attenuates microglial inflammasome signaling in vitro.

Poster number: W-10

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Introduction
Neuroinflammation contributes critically to glaucoma pathogenesis and is thought to be driven in part by microglia, the resident immune cells of the central nervous system. Although microglia have been previously shown to influence disease progression, the mechanism by which these cells mediate neuroinflammation remains poorly understood. CD300LF is a transmembrane, lipid-sensing immune receptor expressed by microglia and macrophages that modulates immunometabolism, inflammatory phenotype and phagocytosis in these cells. As *CD300lf* is upregulated by microglia in mouse glaucoma models, we hypothesize that it plays a critical role in regulating microglial proinflammatory response in glaucoma.

Objectives
To evaluate the effect of genetic deletion of *CD300lf* on microglial phagocytic ability and response to inflammatory stimuli.

Methods
Primary microglial cultures were obtained by isolating microglia from brains of C57BL/6J (wildtype) and *CD300lf*$^{-/-}$ mice. Inflammasome activation was evaluated *in vitro* following pre-stimulation with lipopolysaccharide (LPS) for either 6 or 24 hours, followed by treatment with either nigericin (2 hours) or ATP (30 min). Changes in gene expression were determined using qPCR. The ability of *CD300lf*$^{-/-}$ microglia to phagocytose fluorescently labeled apoptotic neurons (isolated primary retinal ganglion cells or the neuronal cell line SH-SY5Y) was evaluated using FACS.

Results
While both wildtype and *CD300lf*$^{-/-}$ microglia strongly responded to LPS, *CD300lf*$^{-/-}$ microglia showed a statistically significant increase in *Il1*β mRNA level compared to wildtype in response to LPS and ATP at the 6-hour timepoint in two independent experiments. There was no significant difference in Nlrp*3*, *Il18* or *Aim2* mRNA levels between the two groups. In the phagocytosis assay there was no difference in the phagocytic ability between *CD300lf*$^{-/-}$ and wildtype microglia.

Conclusion
Loss of function in *CD300lf* did not directly affect the phagocytic ability of microglia, but it heightened their proinflammatory response to LPS and ATP in terms of Il1β production. Thus, CD300LF acts as a novel modulator of inflammasome signaling in microglia *in vitro* and its contribution to glaucoma pathogenesis merits further investigation *in vivo*.