Purpose and Motivation
Our research goal is to discover the initiators of photoreceptor cell death and retinal remodeling. We model retinal oxidative stress by exposing albino (BALBC/c) mice to bright lights. Exposure to bright light induces rapid cell death of photoreceptors (A-C, TUNEL labeling) and rapid activation of Müller cell (D-F, GFAP upregulation). Our present objective is to define the metabolic signatures that underlie photoreceptor cell death and that underlie Müller cell activation.

Metabolic analyses
Plastic sections are 200 nm and are stained against the following markers:
- Arginine (R): polyamine biosynthesis
- Aspartate (D): core metabolite
- Glutamate (E): core metabolite
- Glutamine (Q): heterocellular group transfer
- Glutathione (J): redox regulation
- Cellular aldehyde binding protein (CRALBP): retinoid binding
- Taurine (τ): osmoregulation
- Terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL): labels the 3-OH ends of fragmented DNA.

Aspartate and glutamate display dynamic levels and spatial distribution in stressed photoreceptors
a) Anomalous elevated aspartate and glutamate levels are early stress markers in photoreceptors (red triangles)

Müller glia are highly sensitive to photoreceptor stress and localize their neuroprotective response
a) Arginine upregulation is an early marker of glial activation

b) Decreased aspartate and glutamate levels correlate with photoreceptor cell death (white triangles)

c) Hypothesis: Oxidative stress converts the mitochondrial aspartate-glutamate carrier into a mitochondrial exporter of α-

Acknowledgements