

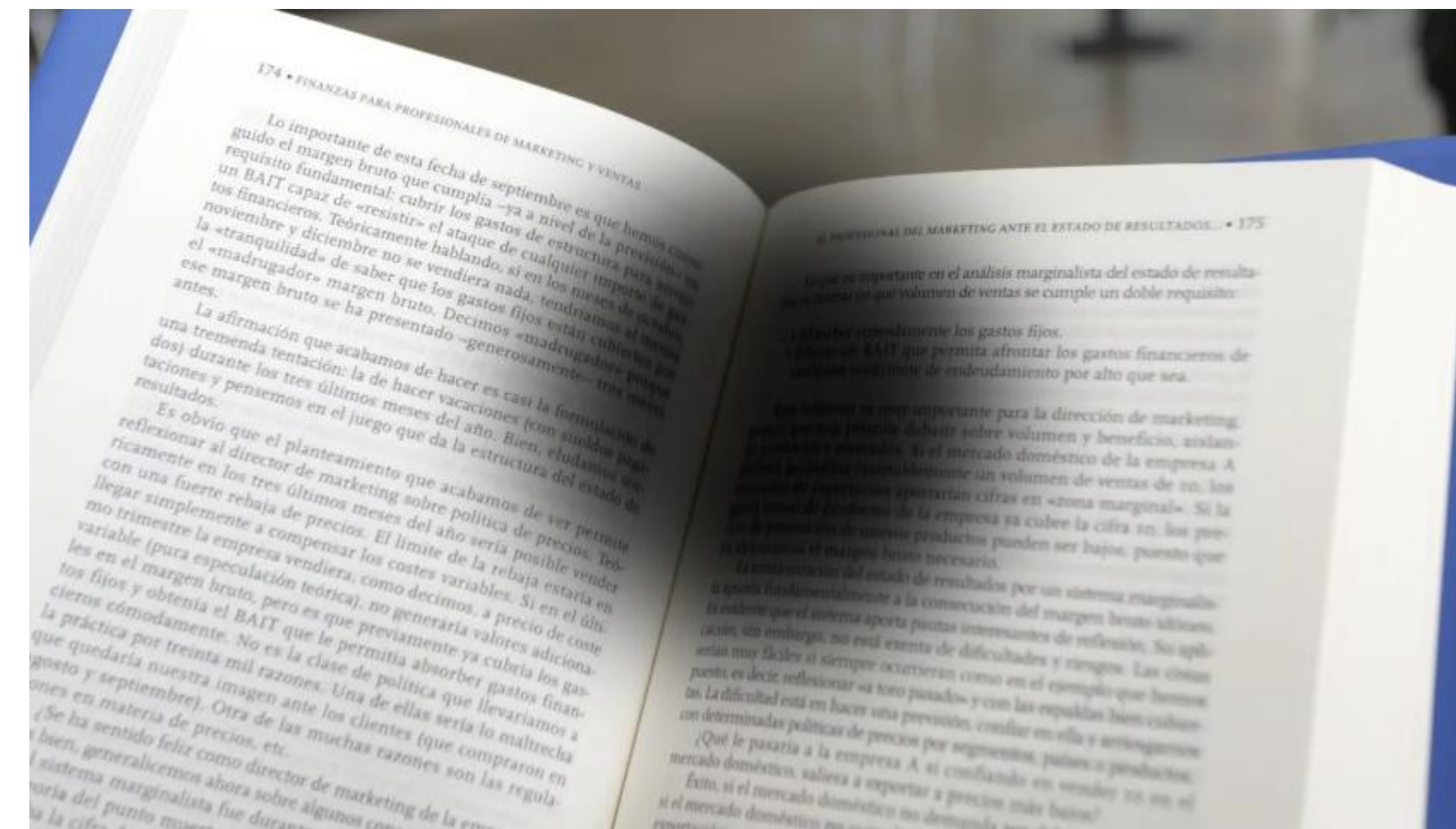
Quantifying macrophage presence of retinal tissue sections in a Stargardt Disease Mouse Model

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Introduction

- **Stargardt disease (STGD1)** is a genetic eye disease that affects 1 in 8,000-10,000 individuals.
- *ABCA4* loss-of-function gene mutation leads to accumulation of N-retinylidene-phosphatidylethanolamine (N-ret-PE), precursor of lipofuscin.
- Lipofuscin accumulation, photooxidative stress, damage of the retinal pigmental epithelium (RPE), and complement system activation all contribute to photoreceptor degeneration.



<https://miranza.es/en/symptoms/loss-of-central-vision/>

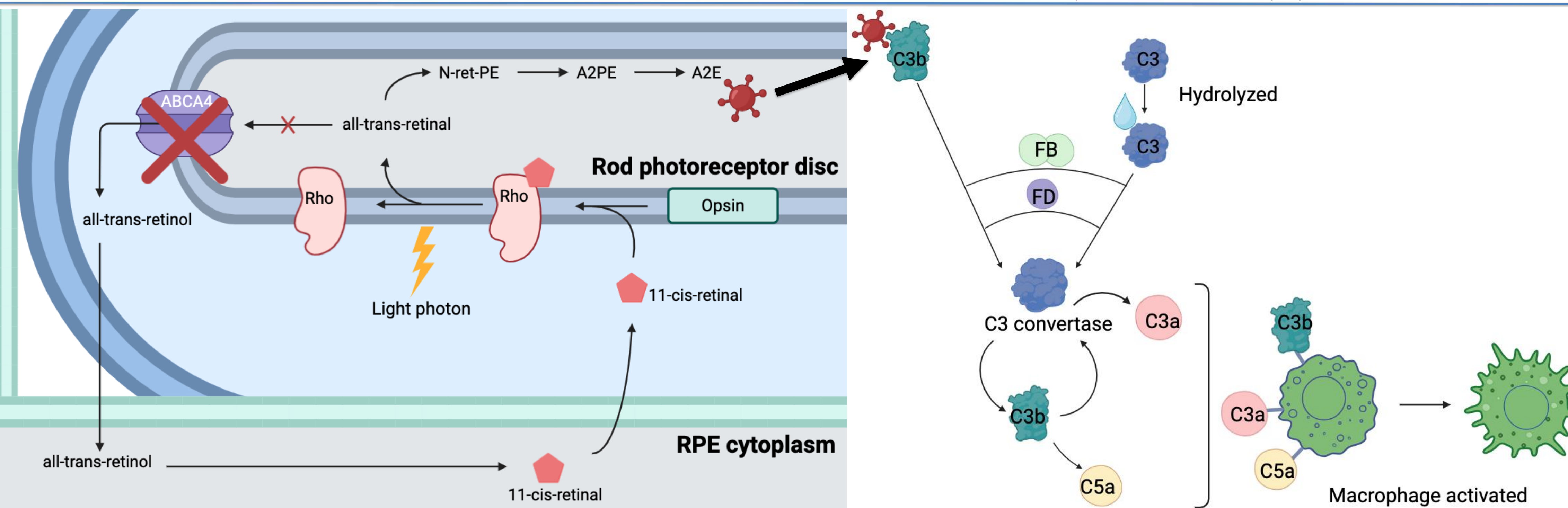


Figure 1. Visual cycle without functioning *ABCA4* leads to A2E that activates the complement system. Complement cleavage products C3a, C3b, and C5a activate macrophages for the phagocytosis of photoreceptors.

We are investigating retinal macrophage presence in a STGD1 mouse model that have photooxidative damage following light stress.

Results

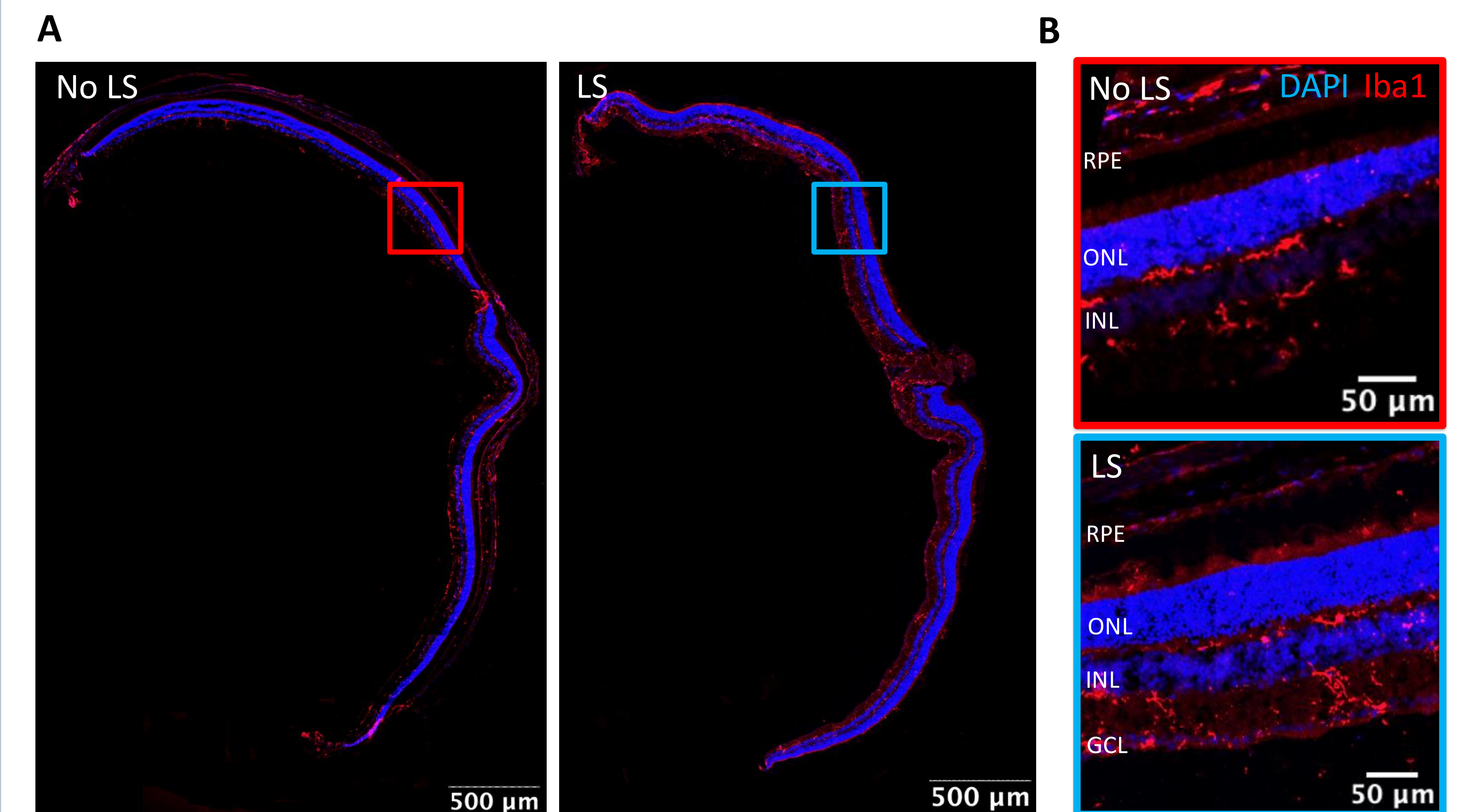
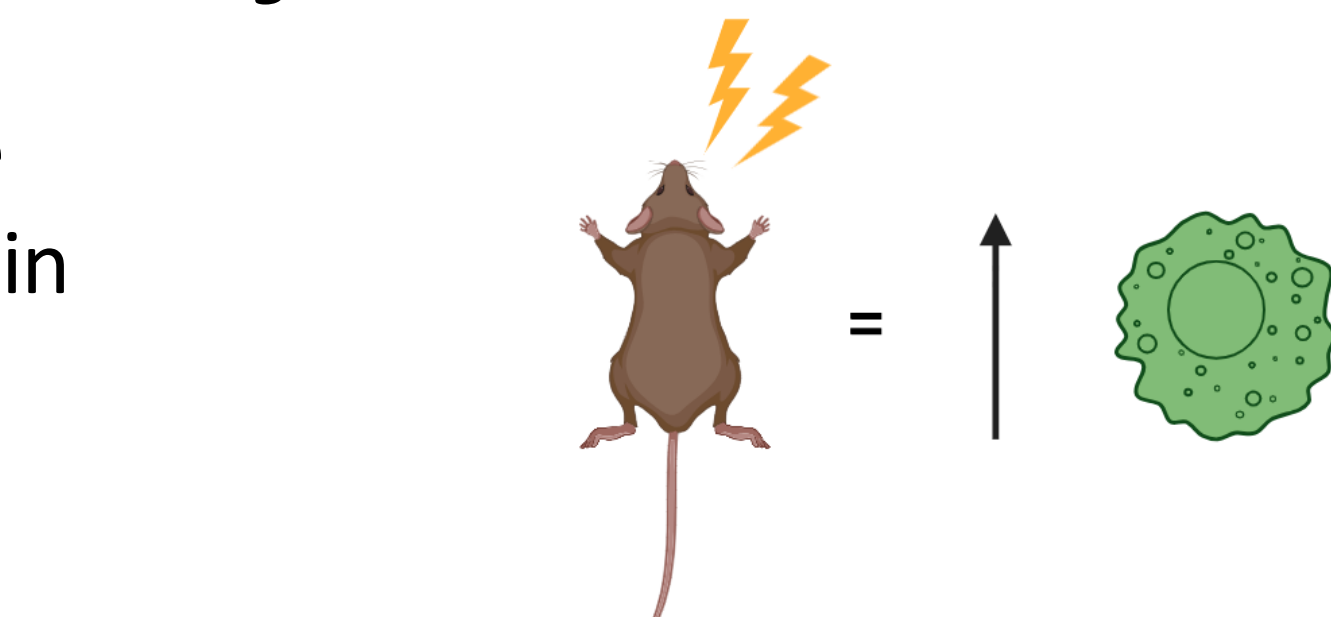


Figure 2. **A)** Representative images of a stitched mouse eyecup expressing macrophage marker Iba1 (red) and DAPI (blue) with no LS on the left and LS on the right. **B)** Respective zoomed in regions for no LS and LS conditions.

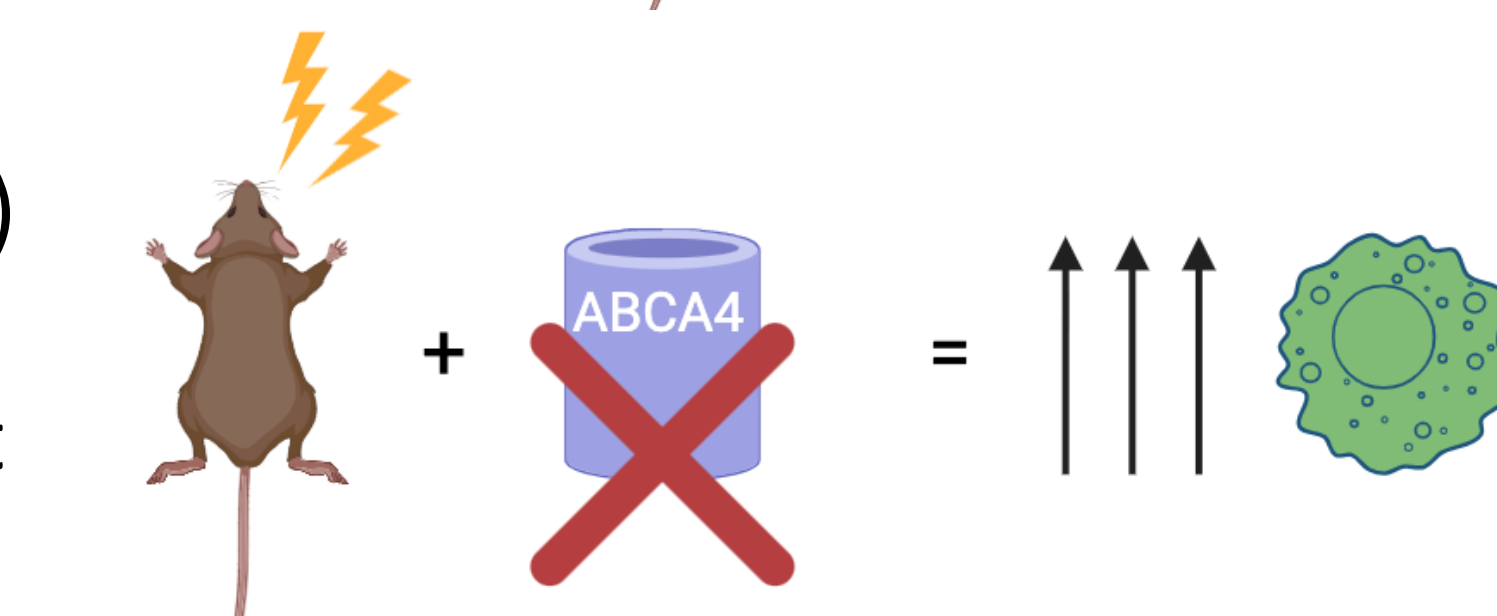
- Iba1 quantification will be performed using two parameters; quantification of total Iba1 labelling and then stringent quantification with more size constraints on particle size.
- Quantification will be done separately for retinal and subretinal layers.

Hypotheses & Objectives

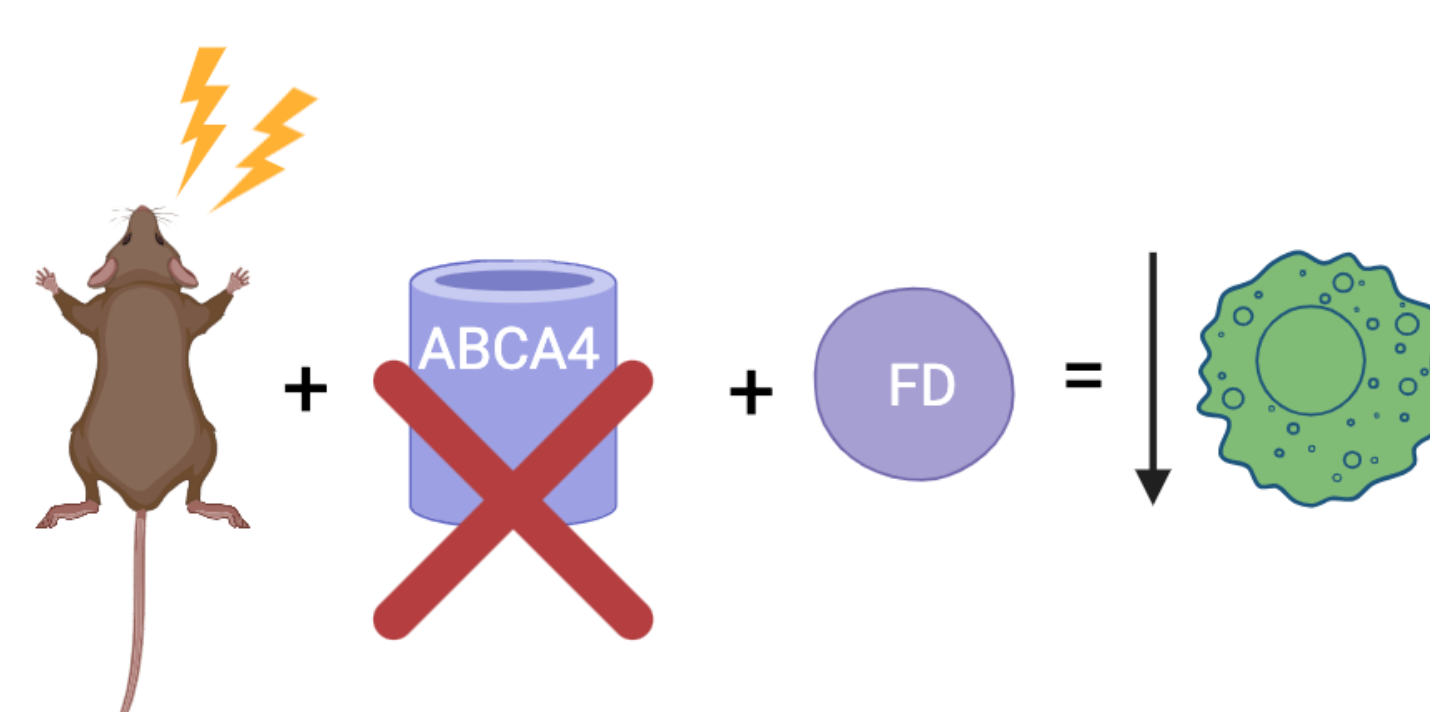
1. Light stress (LS) will cause macrophage recruitment in STGD1 mouse model.



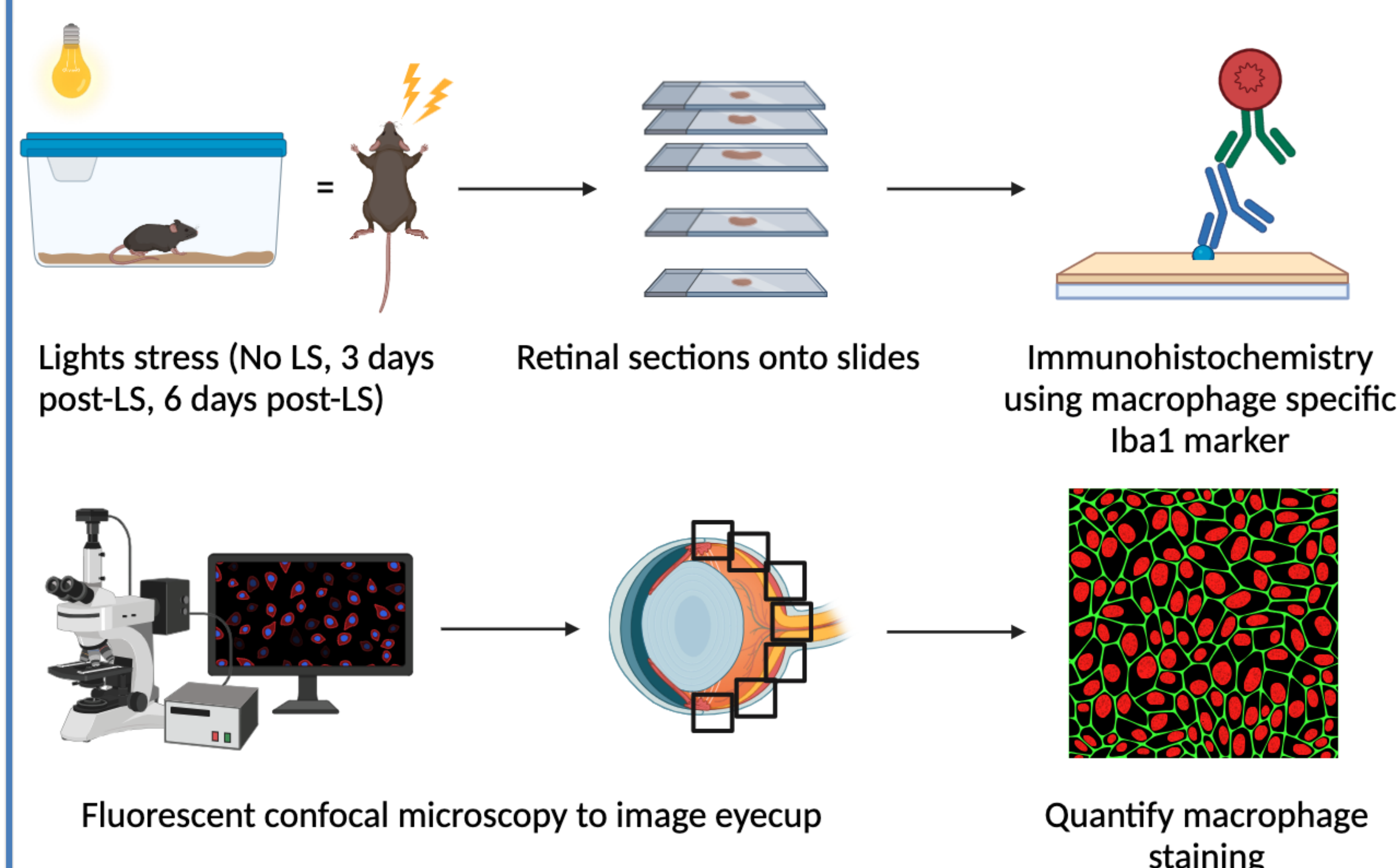
2. STGD1 mutant (*Abca4*^{-/-}) will experience more macrophage recruitment relative to the Wt.



3. *Cfd*^{-/-} mutant will be neuroprotective.



Methods



Next steps...

- Define retinal and subretinal layers for separate quantification of Iba1 labelling
- Run them through the established macro code for the loose and stringent parameter settings

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