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Photoreceptor pathways differentially contribute to refractive eye growth in mice

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Abstract

The specific retinal pathways that detect visual input and initiate the signaling for refractive development are unknown. We investigated the contributions of the three photoreceptor pathways in refractive development by testing photoreceptor specific mutant models and by selectively stimulating photoreceptor pathways using ambient lighting.

Refractions, corneal curvatures, and ocular parameters were measured in mice with non-functional rods (Gnat1−/−), cones (Gnat2−/−), or intrinsically photosensitive retinal ganglion cells (ipRGCs) (Opn4−/−) and compared to wild-type (WT) age-matched controls. Mice experienced normal laboratory conditions (NLC) or monocular form deprivation (FD) from P28 to P84. Separate cohorts of WT mice were housed under 0.005, 50 or 15,000 lux ambient lighting starting at P28. Half of these mice underwent monocular lens defocus for one week.

Gnat1−/− mice had abnormal refractive development under NLC and no response to FD compared to WT mice ($p<0.001$). In contrast, Gnat2−/− mice had responses similar to WT with NLC (p=n.s.) and FD ($p=0.04$). Opn4−/− mice had abnormal refractive development with NLC and significantly greater FD-induced myopic shifts than WT ($p<0.001$). The different ambient conditions did not alter refractive development with NLC. However, 0.005 lux and 15,000 lux conditions reduced the lens-induced myopic shifts compared to 50 lux lighting ($p<0.05$).

Our findings suggest that photoreceptor pathways differentially influence refractive development in mice. Disruption of rod and ipRGC pathways, but not cone pathways, significantly alters refractive development under NLC and FD conditions, though in different directions. Rod pathway signaling is important for refractive development and may even provide protection from induced myopia.