AB006. Elucidating multiple retinal mechanisms controlling mouse refractive development

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Abstract: Dopamine is known as a key molecule in retinal signaling pathways regulating visually guided eye growth, as evidenced by reduced retinal dopamine levels in various species when experimental myopia is generated. However, in C57BL/6 mice our recent work demonstrated that neither retinal dopamine levels, retinal tyrosine hydroxylase (rate-limiting enzyme in dopamine synthesis) levels, nor dopaminergic amacrine cell density/morphology, were altered during the development of form-deprivation myopia (FDM). These results suggest that retinal dopamine is unlikely associated with FDM development in this mouse strain. The role of dopamine in refractive development was further explored in this mouse strain when retinal dopamine levels were reduced by intravitreal injections of 6-OHDA, a neurotoxin that specifically destroys dopaminergic neurons. The dose was so chosen that retinal dopamine levels were reduced, but no significant changes in electroretinographic responses were detected. 6-OHDA induced significant myopic shifts in refraction in a dose-dependent manner, suggesting the involvement of dopamine in normal refractive development. Biometric measurements of ocular dimensions revealed that 6-OHDA resulted in a shorter axial length and a steeper cornea, while form-deprivation led to a longer axial length without changing the corneal radius of curvature. These results strongly suggest that in addition to the dopamine-independent mechanism, a dopamine-dependent mechanism works for refractive development. We have obtained evidence, suggesting that the dopamine-independent mechanism might be related to intrinsically photosensitive retinal ganglion cells (ipRGCs). Firstly, selective ablation of ipRGCs with an immunotoxin resulted in myopic shifts in refraction. Secondly, form-deprivation induced less myopic shifts in animals with ipRGC ablation.

Keywords: Mouse; retina; myopia; dopamine; refractive development

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