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CLINICAL SUMMARY

IMI Experimental Models of Emmetropization and Myopia report

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INTRODUCTION

This report describes and reviews the major contributions that experimental models of myopia have made to the current knowledge surrounding emmetropization (the development process that matches the eye's axial length to its optical power so that the unaccommodated eye is focused at distance), and development of myopia. These studies established many important concepts informing our knowledge of the visual regulation of eye growth and refractive development and provide the evidence and scientific foundation for the current treatment strategies for myopia.

KEY FINDINGS

1. Visual signals relating to retinal defocus control eye growth, guiding emmetropization and the refractive development of the eye. Imposing hyperopic or myopic defocus in animal models results in compensatory changes in eye growth that reduces the imposed refractive error. Visually regulated changes in eye growth produce the largest effects in the eyes of younger animals, but can produce compensatory changes in the eyes of older animals as well.

2. The visual signals guiding eye growth are processed locally within the eye. Optic nerve section does not prevent compensation for defocus and restricting defocus to local retinal regions results in local changes in eye growth. Visual signals in large areas of peripheral retina produce growth changes that can affect axial length and central refractive state.

3. The choroid is an active component in the visual control of eye growth and refraction. Choroidal thickness changes are part of the compensatory response to imposed defocus and may act as an accommodative response that modulates emmetropization and eye growth.

4. The eye growth response to visual signals involves changes to sclera extracellular matrix synthesis and biomechanical properties.

5. Light intensity and the spectral composition of light affect eye growth in complex ways that interact with ocular circadian rhythms and the temporal response characteristics of visual signals.

6. Atropine affects eye growth and prevents experimentally imposed myopia through cellular mechanisms that do not involve accommodation or ciliary muscle activity, and may act through muscarinic and non-muscarinic actions.

7. Experimental studies have identified several biochemical compounds, most notably retinal dopamine, retinoic acid, and nitric oxide, that are involved in the modulation of eye growth. Various changes in the retina, the retinal pigment epithelium (RPE), choroid, and sclera suggest the existence of a cascade of cell signals arising from the retina that modulates scleral biochemistry and regulates eye growth.

8. Molecular changes in gene expression in retina, RPE, choroid, and sclera support the signal cascade hypothesis and suggest that the retina signals hyperopic defocus and myopic defocus for eye growth through different pathways. Identifying the components of these pathways offer specific targets for the development of novel drug treatments for controlling eye growth and myopia progression.

CONCLUSION

Results from experimental studies using animal models have provided the rationale and foundation for both commonly used optical and pharmaceutical treatment strategies for myopia management.

Reference: Troilo D, Smith EL, 3rd, Nickla DL, et al. IMI - Report on Experimental Models of Emmetropization and Myopia. Invest Ophthalmol Vis Sci 2019; 60(3): M31-M88.

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A listing of the IMI committee members, in particular the IMI Experimental Models of Emmetropization and Myopia Report, the white paper itself can be found at https://www.myopiainstitute.org/imi-white-papers.html. Thank you to Praveen Bandela for his professional assistance in this summary. The publication cost of the clinical summary was supported by donations from the Brien Holden Vision Institute, ZEISS, EssilorLuxottica, CooperVision, HOYA, Théa, and Oculus.

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