Retinal-image mediated ocular growth as a mechanism for juvenile onset myopia and for emmetropization

A literature review

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Abstract. The very common ocular clinical condition in children juvenile onset myopia results from axial elongation of the eye. In humans, some studies have found an association of myopia with greater levels of nearpoint activity and with differences in accommodation and convergence function. This paper reviews a variety of laboratory and clinical studies which are consistent with the hypothesis that retinal image defocus is biochemically transformed into an axial elongation expressed through increased posterior segment growth, and thus myopia. This paper also reviews theories of emmetropization, and classifies them as correlational, feedback, and combination. Evidence is presented to suggest that a combination theory, which combines both correlation of the ocular dioptric components and some feedback mechanism for growth of the eye, is the most correct. Current laboratory research suggests that quality and/or focus (defocus) of retinal imagery is involved in this feedback mechanism and that experimentally induced myopia might be enhanced, reduced or eliminated by pharmaceutical application. Direction of defocus may affect the rate of posterior segment growth, and thus the rate of ocular axial elongation.

Introduction

Myopia is the most common ocular clinical condition in children. Juvenile onset myopia is reported to occur at a rate of about 25 percent in Caucasian-Americans and 12 percent in African-Americans [1]. Since it has been a known condition for hundreds of years and is so obvious in many patients, its causes have been a topic of repeated speculations, clinical investigations and basic sciences research works. Researchers and clinicians often assume that emmetropia is the normal refractive condition, and myopia is an anomaly of refractive development. It is possible, however, that both emmetropization and the development of myopia occur as a result of the same feedback mechanism fine tuning along a continuum stretching from hyperopia to myopia. Thus, emmetropia would be just one of many points along the continuum. In this review, we present the hypothesis that retinal image clarity or defocus at the neuroretina leads to cellular and extracellular physiological changes that
directly affect the rate of posterior segment growth and axial elongation in adjacent sclera. These changes impact the OFF and ON channel systems and may be mediated by an excess of extracellular glutamate.

A refraction-driven process would slow axial elongation of the globe, and thus adjust the positioning of the retina, if the ocular refractive apparatus focuses images in front of the retina. If objects in the visual field are conjugate with points behind the retina, then the rate of axial elongation would increase. This hypothesis is consistent with various laboratory and clinical research study findings discussed in this review.

**Classification of myopia**

A logical approach to myopia etiology is a reasonable descriptive classification system of types of myopia. Since the study and treatment of myopia is confounded by possible changes in several refractive components and is clouded by seeming differences in interpretation of data from clinical studies, the classification scheme should not include a priori assumptions about myopia etiology. Grosvenor [2] has proposed such a system and it is consistent with readily recognizable biological stages in the human life span. This classification system is based on age-related myopia prevalence trends and age of myopia onset. The categories of myopia are congenital, youth-onset, early adult-onset, and late adult-onset. Congenital myopia is the type which persists from infancy throughout life. Youth-onset or juvenile-onset myopia begins between about six years of age and the middle teenage years. Early adult-onset myopia appears between the late teenage years and about 40 years of age. Late adult-onset myopia has its onset after about 40 years of age.

**Natural history of juvenile onset myopia**

Once myopia appears in childhood, the condition tends to increase steadily in amount. This phenomenon is known as myopia progression. Myopia progression usually stops or slows in the middle to late teen-age years. The amount of myopia developed is usually between about one and five diopters, although amounts greater than five diopters are not uncommon. Juvenile-onset myopia is associated with normal corrected visual acuity and normal clinical ocular appearance, although a pigment crescent at the temporal margin of the optic disc is common.

An analysis of studies of myopia prevalence in childhood [3–6] suggests that females tend to have earlier onset of myopia than males, with the greatest increases in prevalence occurring at about 10 to 11 years of age in girls and