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The Myopia Epidemic: Exploring the Pathology and Management of the World's Most Common Eye Disorder

By Gabriella Baldassare, BS | Faculty Reviewer: Kammi Gunton, MD

Myopia is the most common eye disorder in the world and an increasing cause of vision impairment in children.[1] Myopia is an imbalance in the refractive components of the eye and its length, leading to inaccurate focusing of light rays in front of the retina tissue instead of on it. There is a spectrum of severity of myopia, from mild nearsightedness to a more severe form known as high or degenerative myopia, which has the potential to cause vision loss and blindness. There is evidence of a developing myopia epidemic, with an estimated one-third of the world population affected by some degree of myopia as of 2020. This number is projected to reach nearly one-half of the population by the year 2050. Moreover, 10% of the world population is predicted to have high myopia by 2050.[1] High myopia not only leads to worsening uncorrected vision in those affected but can also lead to vision-threatening ophthalmic complications. As the axial length of the eye increases, so does the risk of retinal detachment, myopic macular degeneration, cataracts, and open angle glaucoma, all of which can lead to vision loss or blindness.[2] With its growing prevalence and the risks that accompany high myopia, there is clinical importance to understanding the pathology of myopia, the factors leading to the current epidemic, and the efficacy of current interventions used to manage myopia in pediatric populations.

Myopia may occur by lengthening of the eye as measured from front to back, referred to as axial elongation. It may also occur due to increased curvature in the cornea or lens, referred to as refractive myopia. In either case, compared to a normal eye in which the focal point is directly on the retina, light in an eye with myopia will converge at a point anterior to the retina. This convergence of light in front of the retina that occurs in myopic eyes is the cause of blurred distance vision, which may be corrected with negative power spectacles or contact lenses. The onset of myopia generally occurs between the ages of 8 to 13, and from its onset, individuals will continue to experience axial elongation and/or associated refractory changes until eye growth stabilizes, which occurs on average at 15 years of age.[3,4] An earlier age of myopia onset gives the eye more time to grow, equating to an increased

risk of developing high myopia and subsequent ocular pathology.[5] With this pathology in mind, the goal of myopia treatment is to slow myopic progression during childhood while the eye is still growing. In children with myopia, the decision to treat is based on family history of high myopia, the age of onset, and the rate of progression seen each year. Environmental factors may also be considered in assessing a child's risk for progression.

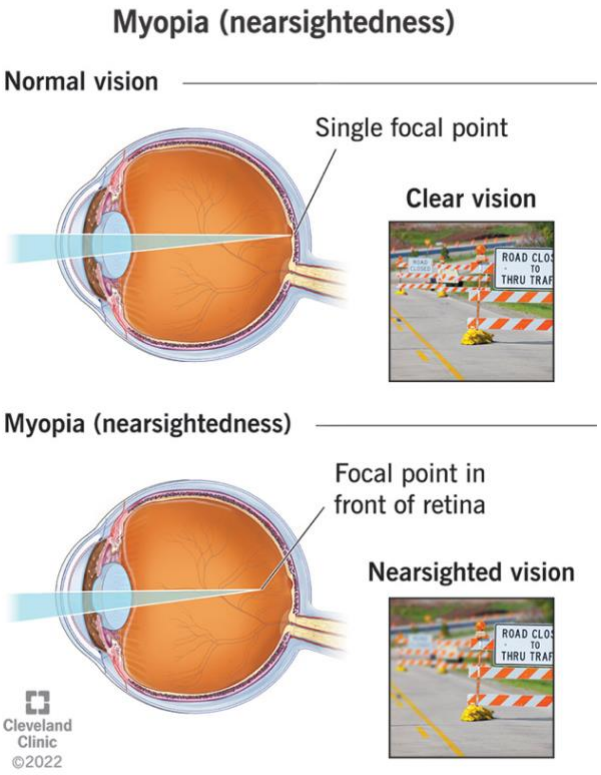

Figure 1: Diagram demonstrating a focal point in front of the retina, causing myopia.[26]

In identifying the potential causes of the current epidemic, it is important to note that myopia is modulated by genetic as well as a multitude of environmental factors. Myopia is strongly linked to genetics, as having myopic parents increases a child's risk of developing myopia and is associated with more rapid myopic progression.[6] While there is robust evidence of a genetic component associated with myopia, the lack of traditional inheritance patterns indicates there are other key factors that determine its development. One of the most compelling environmental factors in myopia development is education, for which epidemiological evidence suggests a causal relationship.[7] The theory that increased education causes an increase in myopia occurrence is supported by the finding that there is little myopia observed in countries where fewer children attend school, while there is an increased rate of myopia in countries with national education systems.[8-10] This relationship is largely assumed to be mediated by the increased time doing near work, such as reading and writing, that necessarily occurs with education. In further support of the idea that near work impacts myopia, a study including 525 Dutch teenagers revealed a positive correlation between 20-minute sessions of continuous cell phone use and increased axial length and refractive error.[11] Home confinement as a result of COVID-19 provided further insight into the relationship between time spent doing near work and myopia, with a large Chinese study finding increased myopia

among 6-8-year-olds in 2020 compared to 2015-2019.[12]

Just as important as understanding the factors involved in the current rise in myopia is the potentially protective effect of outdoor activity on myopic progression. Population data among several different countries demonstrate increased myopia rates in urban areas as compared to rural areas. It is largely thought that the lower rates of myopia in rural areas is due to increased time outdoors as compared to urban areas, but this has yet to be tested directly.[13] The theory that outdoor activity decreases myopia is given further support by multiple animal studies which have demonstrated that light exposure equivalent to ambient daylight slows the progression of myopia.[14] Given the sum of the data regarding the environmental factors impacting myopia, the recommended behavioral modifications for children with myopia are to increase their time outdoors, limit time spent doing near work, and hold items such as reading material farther away from the face. Even so, key factors implicated in myopia development such as genetics, education, and urban location, are difficult if not impossible to modify. Environmental modification alone is insufficient in preventing pathologic progression of myopia, emphasizing the importance of effective clinical interventions in myopia management.

Optical interventions can either be used to correct nearsightedness, such as with single-vision spectacles or contact lenses, or to treat the underlying pathology of myopia, such as with orthokeratology (ortho-k) or peripheral defocus lenses (PDLs). Of note, there are multiple different models and brands of multifocal and progressive spectacles and contact lenses used to treat myopia. The term PDLs used here refers only to the general concept of these lenses rather than the specifics of each subtype. Ortho-k lenses are rigid, gas permeable contact lenses that are typically worn at night to reshape the cornea during sleep. By flattening the curvature of the cornea, the lens improves the focusing power of the cornea to allow the focal point to fall on the macula, or central retina. The shape of the lens also results in peripheral defocus as discussed below. The goal of ortho-k is to inhibit axial elongation as well as eliminate the need for prescription glasses or contacts during the day. Alternatively, PDLs are worn during the daytime and are used to simultaneously correct distance vision and induce inhibition of axial elongation. Ortho-k and PDLs both inhibit axial elongation by correcting peripheral hyperopic defocus in myopic eyes, a phenomenon in which light converges posterior to the focal point of the peripheral retina, signaling the eye to continue elongating.[15,16] By creating a peripheral myopic defocus, ortho-k and PDLs have shown clinical efficacy in slowing myopia progression.[17] Both ortho-k and PDLs have areas with

negative refractive power to correct distance vision and areas with positive refractive power to correct peripheral hyperopic defocus. Ortho-k is not as commonly utilized in current practice, yet it remains an effective intervention for myopia. While PDLs are generally effective, certain subgroups of myopic children seem to benefit from their use more than others.[3] As far as surgical interventions in the management of myopia, posterior scleral reinforcement, although theoretically understandable, has so many vision-threatening complications that it has been abandoned.[18] An emerging area of research is scleral crosslinking, which has only been studied on human eyes *in vivo* in a very small cohort, but holds the potential to be a novel intervention for inhibiting axial growth and in turn preventing high myopia and related complications.[19,20]

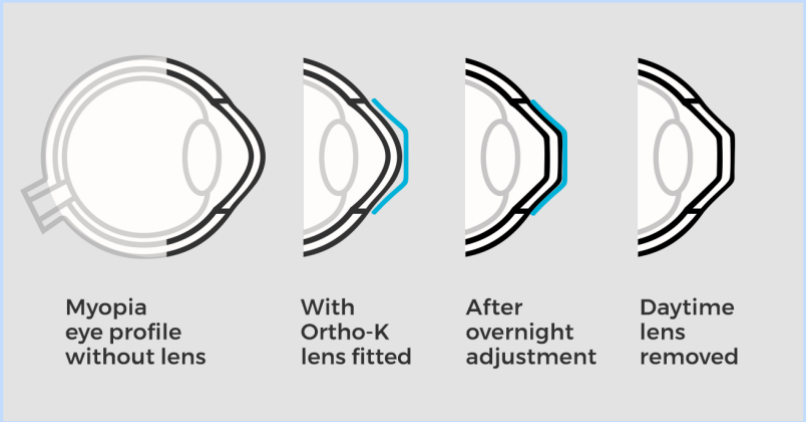

Figure 2: Orthokeratology contact lens treatment.[27]

Furthermore, the pharmacologic arm of myopia management is more controversial, specifically regarding treatment with a daily drop of atropine, a nonspecific muscarinic antagonist that causes dilation of the pupil. Several hypotheses exist to explain atropine's effects on myopia, but the exact mechanism of action is still uncertain. Nevertheless, results of the Atropine for the Treatment of Myopia (ATOM1) trial proved the efficacy of 1% atropine in slowing myopia progression and found that the effect of atropine on myopia is dose dependent.[21] Surprisingly, the results of a follow up trial, ATOM2, concluded that 0.01% atropine was also effective at reducing myopic progression. Additionally, this lower dose did not cause the common side effects of light sensitivity and blurred near vision associated with stronger concentrations.[22] Subsequently, the Low-Concentration Atropine for Myopia Progression (LAMP) trial demonstrated the efficacy of 0.05%, 0.025%, and 0.01% atropine over a one-year period. The reduction in myopic progression in the LAMP trial was found to be dose-dependent, with 0.05% atropine being the most effective.[23] Given its proven efficacy with minimal side effects compared to stronger concentrations, low-concentration atropine (LCA)–ranging from 0.01% to 0.05%–has since become the generally accepted medical management of childhood myopia. While the ATOM and LAMP trials were all conducted in Asian countries, the Childhood Atropine for

Myopia Progression (CHAMP) study evaluated the treatment effect of LCA in North American and European children. The CHAMP study demonstrated that 0.01% atropine was effective in slowing both axial progression and refractive worsening, while 0.02% atropine slowed refractive worsening but showed no change in axial progression.[24] Finally, a recent study published in July 2023 showed that 0.01% atropine did not effectively reduce refractive progression or axial elongation in an ethnically diverse cohort of 187 children in the United States.[25] Despite the relatively small sample size, it is representative of the United States population, indicating that North American children with myopia might require different treatment concentrations of atropine compared to Asian children to achieve similar results. This has sparked controversy regarding whether LCA is beneficial in managing myopia. Adding to the complexity is the risk of rebound myopic progression upon discontinuation of atropine, though this risk is less pronounced with lower doses.[22] Despite the minimal risk of adverse effects and the potential for worsening myopia upon discontinuation, there are compelling reasons to continue using LCA for myopia management, particularly in individuals already benefiting from it. These conflicting findings underscore the complexity of myopia as a disorder, emphasizing the absence of a universally effective method for preventing its progression across diverse populations.

Given the multifactorial nature of myopic progression along with recent controversies in treatment, pediatric ophthalmologists must balance emerging information with historical evidence to effectively combat the myopia epidemic. For now, LCA remains the first-line treatment for myopia along with behavioral recommendations to increase time outdoors and limit near work. As formal education and other forms of near work such as smartphone use continue to be integral parts of many modern societies, the myopia epidemic is unlikely to be halted. Thus, early detection and effective treatment in pediatric populations is crucial to prevent ocular complications and minimize the public health burden associated with the myopia epidemic.

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