

MYOPIA

Authored by
mohammad looti

November 1, 2025

RECOMMENDED CITATION

mohammad looti (2025). *MYOPIA*. PSYCHOLOGICAL SCALES. Retrieved from <https://scales.arabpsychology.com/?p=63245>

MYOPIA

Primary Disciplinary Field(s): Ophthalmology, Optometry, Vision Science

1. Core Definition and Manifestation

Myopia, commonly referred to as **near-sightedness** or **short-sightedness**, constitutes a pervasive refractive error of the eye wherein distant objects appear blurred while close objects can be seen clearly. This visual phenomenon arises fundamentally because the eye focuses the image of a distant object not upon the light-sensitive layer of the retina, as is necessary for clear vision, but rather at a point situated in front of the retina. The resulting image projected onto the retinal surface is diffuse and indistinct, leading to the characteristic blurring of faraway scenes. This condition represents a significant deviation from **emmetropia**, which describes the state of perfect vision where the eye is able to accurately focus images onto the retina without requiring optical correction. The severity of myopia is quantified using diopters (D), a unit measuring the optical power of the lens required to correct the error, with negative values indicating myopic correction.

The practical manifestation of myopia involves a noticeable impairment in activities requiring focused distance vision, such as driving, watching a screen across a large room, or identifying faces at a distance. Patients often report having to squint or partially close their eyelids to temporarily improve visual clarity. This action, known as the **pinhole effect**, helps to reduce the size of the blurred circle on the retina by limiting the peripheral light rays entering the eye, thereby enhancing depth of focus. Crucially, the blurring experienced by myopic individuals is not due to a failure of light to enter the eye or a malfunction of the retina itself, but rather an error in the precise alignment between the eye's optical power (determined by the curvature of the cornea and lens) and the axial length (the physical distance from the cornea to the retina). This delicate balance is central to achieving sharp vision, and its disturbance defines the myopic state.

Understanding myopia requires recognizing it as a global public health concern due to its increasing prevalence, particularly in industrialized and highly educated populations. While often perceived as merely an inconvenience correctable by spectacles or contact lenses, myopia, especially in its high-grade forms, is associated with substantial ocular pathology that can lead to permanent vision loss. Therefore, the definition of myopia extends beyond a simple refractive error to encompass a complex condition influenced by genetics, environmental behaviors, and developmental processes impacting the growth and structure of the eyeball, demanding careful epidemiological study and clinical intervention strategies designed to slow its progression.

2. Underlying Pathophysiology and Optical Error

The optical deviation that characterizes myopia originates from an inappropriate relationship

between the refractive power of the eye's structures--primarily the **cornea** and the crystalline lens--and the total axial length of the eyeball. The majority of myopic cases are classified as **axial myopia**, where the primary anatomical defect is an excessive elongation of the eyeball along the anterior-posterior axis. In a typically emmetropic eye, the axial length measures approximately 23.5 to 24 millimeters. When the eye grows excessively, perhaps extending to 26 millimeters or more, the total refractive power, even if normal, is insufficient to pull the focal point back onto the extended retina. The light rays converge at the point where the retina *should* be in a standard eye, leaving the actual, elongated retina behind the focal point, resulting in a blurred image.

A less common form is **refractive myopia**, where the axial length is normal, but the refractive components possess excessive optical power. This usually stems from an abnormally steep curvature of the cornea or increased thickness or refractive index of the lens. For instance, conditions like **keratoconus**, which causes progressive corneal thinning and steepening, can induce significant refractive myopia. Similarly, certain metabolic changes within the crystalline lens, often associated with the early stages of cataract development or uncontrolled diabetes, can increase the lens's refractive power, shifting the focal point forward. Both axial and refractive factors can often coexist, contributing synergistically to the total myopic error, complicating the precise determination of the underlying cause for clinical management.

The physiological mechanisms driving this optical mismatch are still under intense investigation, but recent research strongly suggests that the environment within the eye, specifically the retina and choroid, plays a critical regulatory role in controlling ocular growth. Local biochemical signals originating from the retina in response to visual input appear to modulate the growth rate of the sclera, the tough outer layer of the eye. When light focuses properly, the signal promotes balanced growth; however, when the image is consistently focused behind the retina (a state known as **hyperopic defocus**), the eye interprets this as a signal to elongate rapidly to reach the focal point. Paradoxically, in early stages of myopia development, the eye may exhibit peripheral hyperopic defocus even when central vision is properly corrected, leading to continued axial elongation. This discovery has been pivotal in designing therapeutic interventions aimed at controlling peripheral focus rather than just correcting central vision.

3. Classification and Severity Levels

Myopia is classified primarily based on its severity, measured in diopters (D), and its underlying pathology. Clinicians typically distinguish between three main levels of severity. **Low myopia** generally refers to refractive errors ranging from -0.50 D to -3.00 D. This level of near-sightedness is highly prevalent and usually presents minimal risk of ocular complications, being easily corrected with standard spectacles or contact lenses. **Moderate myopia** spans errors from -3.25 D up to -6.00 D. Individuals in this range require stronger correction and begin to face a slightly elevated risk for certain retinal issues later in life, necessitating regular comprehensive eye examinations.

The most clinically significant category is **high myopia** (also known as pathological myopia or degenerative myopia), defined as a refractive error of -6.00 D or greater, or an axial length exceeding 26 millimeters. High myopia is not merely a quantitative increase in the refractive error but often indicates structural changes within the posterior segment of the eye due to extreme elongation. The retina, choroid, and sclera are stretched and thinned, leading to distinct pathological changes such as lacquer cracks, Fuchs spots, and chorioretinal atrophy. This stretching predisposes the eye to severe, vision-threatening complications irrespective of optical correction, making timely identification and monitoring crucial.

Furthermore, myopia can be subcategorized based on its onset and progression pattern. **School-age myopia** or **juvenile-onset myopia** is the most common form, typically developing between ages 6 and 14 and progressing until early adulthood. This form is strongly linked to near work and educational pressures. Conversely, **congenital myopia** is present at birth or develops shortly thereafter, often being severe and potentially associated with other systemic disorders. Finally, **late-onset myopia** develops after the age of 20, often attributed to occupational demands involving intensive near tasks or specific medication use. Recognizing these classifications is essential for predicting prognosis, counseling patients on lifestyle modifications, and selecting appropriate myopia control strategies.

4. Etiology and Risk Factors

The etiology of myopia is widely recognized as multifactorial, stemming from a complex interplay between genetic predisposition, environmental pressures, and behavioral patterns. Genetic studies, including twin studies and genome-wide association studies (GWAS), have identified dozens of genetic loci associated with myopia development, demonstrating that heredity plays a critical role, particularly in cases of high myopia. If one or both parents are myopic, the likelihood of a child developing the condition increases significantly, suggesting that the underlying mechanisms governing axial growth rate and scleral rigidity are partially inherited. However, genetics alone cannot account for the rapid, global rise in myopia prevalence observed over the past few decades, which points overwhelmingly to powerful environmental triggers.

Environmental factors center primarily on intensive **near work** activities and a lack of **time spent outdoors**. Intensive near work, defined as prolonged periods spent focusing on tasks within arm's reach (such as reading, writing, or using electronic devices), is hypothesized to stimulate the ocular growth mechanism toward elongation. The continuous accommodation required for close focus, coupled with insufficient breaks, may contribute to tonic ciliary muscle spasm or increase intraocular pressure changes that promote axial growth. Educational systems demanding early literacy and extended hours of study further exacerbate this risk profile, leading to the term "school-age myopia" being highly descriptive of this environmentally induced progression.

Perhaps the most robust environmental finding relates to the protective effect of **outdoor time**. Numerous longitudinal and interventional studies have demonstrated that children who spend significant time outdoors--generally defined as two hours or more per day--have a dramatically reduced risk of developing or progressing myopia, regardless of the amount of near work they perform. The mechanism is believed to involve high levels of ambient light exposure, which stimulates the release of **dopamine** from the retina. Retinal dopamine is thought to be a potent inhibitor of abnormal ocular elongation, acting as a natural brake on axial growth. This evidence has shifted public health recommendations worldwide, emphasizing the need to balance educational demands with mandatory periods of outdoor activity to mitigate the myopia epidemic.

5. Diagnosis and Clinical Assessment

Diagnosis of myopia begins with a comprehensive eye examination performed by an optometrist or ophthalmologist. The initial step involves measuring **visual acuity**, typically using a Snellen chart, to quantify the degree of uncorrected distant vision impairment. A crucial component of the diagnosis is the objective and subjective determination of the refractive error. Objective measurements are often obtained using an **autorefractometer**, a device that automatically estimates the refractive state of the eye by shining light into the eye and analyzing its reflection. However, the definitive measurement involves **retinoscopy** or subjective refraction, where the clinician manually refines the prescription by placing different lenses in front of the patient's eye until the clearest possible vision is achieved, thereby determining the precise diopter power required for correction.

For children and young adults, **cycloplegic refraction** may be necessary. Cycloplegic drops temporarily paralyze the ciliary muscle, preventing the eye from accommodating or flexing its natural lens to compensate for minor refractive errors (a condition known as latent hyperopia or pseudomyopia). Since accommodation can mask or temporarily reduce the apparent degree of myopia, especially in children, cycloplegia ensures the measurement reflects the true structural refractive state of the eye, which is vital for accurate prescription and effective myopia control management.

Beyond simple refraction, a comprehensive assessment involves measuring the **axial length** of the eye, usually via **biometry** (such as optical coherence biometry). This measurement is increasingly important, particularly in pediatric care and in the management of high myopia, as axial length is a better predictor of future pathological risk than the diopter measurement alone. Furthermore, the clinician must perform a detailed inspection of the ocular structures, including a thorough examination of the anterior segment, crystalline lens, and a dilated fundus examination to assess the peripheral and central retina. This posterior segment check is essential to rule out early signs of myopic pathology, such as peripheral retinal thinning, lattice degeneration, or early myopic maculopathy, which require immediate attention and preventative measures.

6. Management and Treatment Modalities

Management strategies for myopia fall into two broad categories: **correction** of the existing refractive error to restore clear vision, and **control** aimed at slowing or halting the underlying axial elongation, primarily applied in pediatric populations. The standard form of correction involves prescription **spectacles** (glasses) or **contact lenses**, which introduce diverging power to shift the focal point backward onto the retina. While highly effective for correcting vision, standard single-vision lenses do not typically slow the progression of myopia in children.

Myopia control therapies have gained significant traction due to the clinical recognition that slowing progression reduces the lifetime risk of high myopia and associated complications. Pharmacological intervention, primarily low-dose **atropine eye drops** (typically 0.01% to 0.05%), has proven highly effective in slowing axial elongation in children, although the exact mechanism remains debated--it is thought to act on the sclera or choroid rather than solely through cycloplegia. Optical interventions for control include specialized contact lenses, such as **multifocal contact lenses** or lenses designed with **peripheral defocus management** (PDM). These lenses incorporate differing powers across the lens surface to provide clear central vision while simultaneously inducing relative myopic defocus in the peripheral retina, which signals the eye to cease excessive growth.

Another effective optical control method is **Orthokeratology** (Ortho-K). This involves wearing rigid, gas-permeable contact lenses overnight to temporarily reshape the cornea, thereby reducing the refractive error during the day. Beyond temporary vision correction, the corneal reshaping effect also creates the necessary peripheral myopic defocus signal, providing significant myopia control benefits. For adults with stable refractive errors, surgical correction options like **LASIK** (Laser-Assisted in Situ Keratomileusis) or **PRK** (Photorefractive Keratectomy) permanently reshape the cornea to reduce or eliminate the need for glasses. However, it is crucial to understand that surgical correction only changes the focal point; it does not eliminate the underlying risk associated with the eye's pre-existing, elongated axial length, meaning high myopic patients remain susceptible to retinal complications regardless of having 20/20 post-operative vision.

7. Associated Complications and Long-Term Risks

While low and moderate myopia often remain benign, high myopia is strongly associated with an increased risk of severe, vision-threatening ocular complications, stemming directly from the mechanical stretching and thinning of the posterior ocular layers. The increased axial length places stress on the retinal and choroidal vasculature, leading to conditions collectively known as **pathological myopia**. One of the most serious risks is **retinal detachment**, where the stretching leads to breaks or tears in the retina. Fluid then seeps under the retina, separating it from the underlying choroid. Even after successful surgical repair, retinal detachment can cause significant,

permanent loss of vision. The risk of detachment increases exponentially with the degree of myopia.

High myopia also significantly elevates the risk of developing secondary sight-threatening conditions. The mechanical stress causes characteristic changes, including **myopic maculopathy**, which involves degenerative atrophy of the central retinal tissue (the macula) necessary for fine detail vision. This can manifest as lacquer cracks (breaks in the Bruch's membrane), choroidal neovascularization (abnormal blood vessel growth under the retina), and chorioretinal atrophy, all of which compromise central vision irrevocably. Furthermore, high myopia is an independent risk factor for both **open-angle glaucoma** and **cataracts**. Myopic eyes often exhibit structural changes in the optic nerve head that make them more vulnerable to glaucomatous damage, and the increased size of the eyeball is linked to earlier onset of posterior subcapsular cataracts.

Given these significant long-term risks, the focus of modern ophthalmology has decisively shifted from mere optical correction to proactive **myopia control**. The goal is to minimize the final axial length achieved by the time a child reaches maturity. If control strategies can prevent an eye from progressing from moderate (-4.00 D) to high (-7.00 D) myopia, the lifetime risk of conditions like myopic maculopathy can be substantially reduced, underscoring the vital public health imperative behind early diagnosis and aggressive intervention in pediatric populations globally.

8. Global Prevalence and Public Health Significance

The prevalence of myopia has reached epidemic proportions globally, particularly in East and Southeast Asia, leading experts to define it as a major public health crisis of the 21st century. Epidemiological data indicates that in certain urban Asian populations, rates of myopia among young adults now exceed 80% to 90%, with high myopia rates reaching 10% to 20%. While rates in Western countries are generally lower, they are also rising significantly. Predictions suggest that by the year 2050, half of the world's population (approximately 5 billion people) will be myopic, and one billion individuals will have high myopia, placing an unprecedented burden on healthcare systems due to the complexity and cost of managing associated pathologies like retinal detachment and myopic maculopathy.

The public health significance of this trend is multifaceted. Economically, the cost of managing myopia encompasses not only the lifelong need for corrective lenses but also the expense of treating preventable blindness resulting from high myopia complications. This burden includes specialized surgical procedures, ongoing monitoring, and the indirect costs of lost productivity and diminished quality of life for those suffering from irreversible vision impairment. Socially, the epidemic highlights a tension between modern educational demands and biological ocular development, forcing policymakers to reconsider school structures and mandated outdoor time to mitigate the environmental drivers of the condition.

Effective management requires integrated public health strategies that emphasize prevention, early detection, and accessibility to proven control methods. Prevention focuses on promoting evidence-based lifestyle changes, chiefly increasing outdoor exposure for children. Screening programs are essential for early detection of juvenile-onset myopia, enabling timely intervention with atropine or specialized optical devices before the axial length progresses dangerously. Furthermore, ongoing research into pharmacological agents and novel optical designs continues to seek more effective and universally accessible methods for controlling axial elongation, aiming to curb the global trajectory of this pervasive condition and safeguard the future visual health of populations worldwide.

Further Reading

[Myopia \(Near-sightedness\)](#)

[Genetics of Myopia and Risk Factors](#)

[Vision Science](#)

[Retina](#)

[Ophthalmology](#)

ARABPSYCHOLOGY.COM