

MYOPIA AND ITS COMPLICATIONS

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Annotation. This review explores current views on the causes and development of complications related to myopic refraction. It summarizes domestic and international research using modern diagnostics and highlights that myopia involves not only refractive and anatomical changes, but also pathological processes linked to disrupted ocular hemodynamics and hydrodynamics.

Keywords: Myopia, macular dystrophy, glaucoma, neurotrophins, optical coherence tomography, fluorescein angiography

Approximately 1.6 billion people worldwide have refractive errors. According to J.H. Kempen et al., by 2020, around 2.5 billion people were expected to suffer from myopia. Myopia remains one of the most common eye disorders globally and is the leading cause of visual impairment. According to the World Health Organization, the prevalence of myopia ranges from 10% to 90% in developed countries. In Russia, over 10% of the population is myopic, compared to more than 25% in the USA and Europe, and up to 80% in some Asian countries.[1]

The World Health Organization identified uncorrected refractive errors as a major target in eliminating avoidable blindness by 2020. Uncorrected myopia impairs visual tasks, reduces professional adaptability, and lowers quality of life. It may lead to retinal detachment and myopic maculopathy—serious complications that can cause disability at a young working age. Myopia accounts for 18.0% of visual disability across all age groups, ranking third among the causes. Additionally, myopia imposes a significant economic burden; in the United States, correction-related costs reached approximately \$4.9 billion in 2011.[2]

In 1956, J. Otsuka associated the development of myopic refraction with pathological ciliary muscle tone, leading to weakened accommodation. As a result, the ciliary muscle and subsequently the choroid undergo atrophy, causing scleral elongation. According to the theory proposed by A.I. Dashevsky in 1973, myopic refraction also results from reduced accommodative ability of the ciliary muscle, primarily due to autonomic dysfunction and prolonged near work under unfavorable conditions. This leads to a pre-spasm state and, with progression, to accommodative spasm, accompanied by suppression of the convergence, accommodation, and pupillary reflexes. Persistent strain of the extraocular muscles and increased intraocular pressure during transient convergent elongation of the eye, along with cumulative micro-deformations of the sclera, contribute to the development of axial myopia.[3]

A.P. Nesterov emphasized the role of elevated intraocular pressure in the pathogenesis of myopia. In 1986, Balacco-Gabrielli proposed a hormonal theory, suggesting that endocrine dysfunction disrupts collagen metabolism, leading to scleral overstretching. Currently, the most widely accepted and well-substantiated concept is the three-factor theory of myopia development, proposed by E.S. Avetisov. This theory identifies two key mechanisms: first, the mismatch between the weakened accommodative apparatus and visual load; and second, the reduced biomechanical strength of the sclera, which stretches under intraocular pressure.[4]

Conclusions: Myopia is a widespread visual disorder affecting billions of people globally and is a leading cause of visual impairment. Its prevalence is especially high in developed and Asian countries. Uncorrected myopia not only impacts quality of life and professional functionality but can also lead to severe complications such as retinal detachment and myopic maculopathy. Various theories explain its pathogenesis, including ciliary muscle dysfunction (Otsuka, Dashevsky), elevated intraocular pressure (Nesterov), and hormonal imbalances (Balacco-Gabrielli). The most comprehensive explanation is provided by

Avetisov's three-factor theory, which integrates weakened accommodation, biomechanical scleral weakness, and genetic predisposition. These insights underline the importance of early diagnosis, preventive strategies, and tailored therapeutic approaches in managing myopia.

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