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Bidirectional Interaction Between Childhood Astigmatism and Myopia Development

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Abstract: ***Astigmatism and myopia are among the most common refractive errors in childhood and have traditionally been considered distinct manifestations of abnormal refractive development. However, accumulating epidemiological, clinical, and experimental evidence suggests that their relationship may be bidirectional and dynamically influenced by ocular growth, visual experience, and individual susceptibility. Astigmatism may potentially affect myopia development by degrading retinal image quality, altering visual feedback, and modifying the signals involved in emmetropization and axial growth. Conversely, progressive myopia and axial elongation may influence astigmatic characteristics through changes in ocular geometry, corneal biomechanics, anterior–posterior corneal coupling, and internal optical compensation. In addition, genetic susceptibility, environmental exposure, age, and sex may modify the magnitude and timing of these interactions. Evidence from longitudinal and intervention studies further suggests that the association between astigmatism and myopia may vary according to astigmatic magnitude, axis orientation, baseline refractive status, and developmental stage. Nevertheless, substantial heterogeneity in study populations, measurement methods, and follow-up duration, together with the predominance of observational evidence, limits definitive conclusions regarding causality. Overall, current evidence supports substantial interaction rather than a clearly established direct causal relationship between childhood astigmatism and myopia. Concurrent monitoring of spherical and cylindrical refractive changes, together with axial-length assessment when feasible, may improve the identification of children at increased risk of refractive abnormalities. Further prospective longitudinal studies integrating refractive, biometric, corneal tomographic, optical, and biomechanical parameters are warranted to clarify the temporal and causal pathways linking astigmatism and myopia and to facilitate more precise strategies for prevention and individualized management of childhood refractive errors.***

Keywords: Astigmatism, Myopia, Refractive development, Axial elongation, Corneal biomechanics, Bidirectional interaction**.**

1. Introduction

Myopia has become increasingly prevalent worldwide [1], with especially high prevalence rates reported among children and adolescents in East and Southeast Asian countries [2]. Meanwhile, astigmatism is common among infants and preschool children [3], with reported prevalence exceeding 15% in certain populations. Although myopia and astigmatism are often investigated as distinct refractive conditions in clinical and research settings, accumulating evidence suggests that they may be dynamically interrelated during childhood ocular development. Importantly, this relationship may not be unidirectional: astigmatic blur may influence the visual signals involved in ocular growth, whereas progressive myopia and axial elongation may, in turn, alter ocular geometry and astigmatic characteristics. In addition, genetic susceptibility, environmental exposure, age, and sex may modify the magnitude and timing of these interactions. Therefore, understanding the relationship between childhood astigmatism and myopia requires an integrated perspective encompassing epidemiological associations, developmental trajectories, visual and physiological mechanisms, and ocular structural changes. This review systematically examines the current evidence regarding the interaction between childhood astigmatism and myopia, with particular emphasis on epidemiological characteristics, genetic and environmental determinants, potential bidirectional mechanisms, axial and corneal changes, and implications for clinical practice and public health. By integrating evidence from different study designs and highlighting areas of uncertainty, this review aims to provide a framework for understanding the complex interaction between astigmatism and myopia and to inform future research and individualized clinical management strategies.

2. Epidemiological Associations: Comorbidity and Patterns of Progression

2.1 Comorbidity

Epidemiological evidence increasingly suggests that childhood astigmatism may be associated with subsequent myopia development and progression. Preschool-aged children with astigmatism greater than 1.00 D have been reported to have a significantly increased risk of developing myopia before adolescence. Moreover, the prevalence, progression, and patterns of comorbidity between myopia and astigmatism vary across ethnicities and geographic regions [4]. For example, refractive changes during childhood are generally greater among Asian populations than among European and American populations, a difference that may reflect the combined effects of genetic background, educational environment, and time spent outdoors. These population-level differences indicate that the relationship between astigmatism and myopia is unlikely to be explained by a single determinant and may instead arise from interactions among biological susceptibility and environmental exposures.

Longitudinal studies have further strengthened the evidence for an association among astigmatism, axial length, and myopia development. One study demonstrated that internal astigmatism was closely associated with axial length, suggesting that alterations in internal ocular optics may be involved in refractive development [5]. Similarly, other studies have reported that greater astigmatic magnitude and against-the-rule (ATR) astigmatism are associated with accelerated axial elongation in children [6]. Li X et al. reported that longitudinal changes in the axial length-to-

corneal radius ratio (AL/CR) were associated with corneal astigmatism [7]. In a 2-year follow-up study, Gong W et al. further demonstrated a positive association between astigmatic magnitude and axial elongation, particularly among children with myopia [8]. At both the population and genetic levels, therefore, astigmatism and myopia appear to exhibit substantial co-occurrence. In addition, several studies have shown that, with increasing age, the corneal astigmatic axis in individuals with high myopia tends to shift from with-the-rule (WTR) toward ATR astigmatism, a pattern that may be related to axial elongation and changes in overall ocular shape [9]. Collectively, these findings suggest that changes in axial dimensions and astigmatic characteristics may occur in parallel during childhood refractive development.

Evidence from intervention studies provides additional, although indirect, support for an association between astigmatism and myopia control. In a study evaluating the efficacy of DIMS spectacle lenses, Domsa P et al. reported that the presence of astigmatism influenced the effect of myopia control [10]. Zhong G et al. similarly suggested that baseline astigmatism may act as a potential modulator of axial elongation [11]. Meanwhile, mechanistic and observational studies have provided complementary evidence from different perspectives. Liang Y et al. highlighted the roles of corneal and internal astigmatism in changes in refractive astigmatism and reported associations between changes in corneal and internal astigmatism and near-work behavior [12]. Jiang F et al. identified astigmatism as a potential covariate affecting axial elongation [13]. Taken together, these findings suggest that astigmatism may be associated with the development and progression of myopia, while also indicating that its effects may depend on baseline refractive status, ocular biometric characteristics, and environmental exposure.

Importantly, however, epidemiological associations should not be interpreted as evidence of a direct causal relationship. The consistency of associations among astigmatic magnitude, axial length, and myopia across different populations and study designs supports the hypothesis that these refractive characteristics may interact during ocular development, but residual confounding, differences in measurement methods, and variations in population characteristics remain important considerations. Thus, the epidemiological evidence provides a rationale for investigating the biological mechanisms underlying this potential interaction.

2.2 Genetic and Environmental Factors

Beyond epidemiological associations, the development of myopia and astigmatism is increasingly recognized as a complex process involving interactions between genetic susceptibility and environmental exposure. Twin and family studies have demonstrated substantial heritable components for both myopia and astigmatism [14]. Large-scale genome-wide association studies (GWAS) have further identified multiple genetic loci associated with refractive errors and suggested that genetic risk scores (GRSs) may help predict individual susceptibility to myopia [15]. Nevertheless, genetic factors alone cannot adequately explain the rapid increase in myopia prevalence observed over recent decades, highlighting the important contribution of environmental exposures. Extensive epidemiological and longitudinal cohort studies have shown that increased near-work demands and reduced time spent outdoors are important environmental risk factors for the onset and progression of myopia [16]. More importantly, recent multicenter pediatric cohort studies have suggested interactions between genetic susceptibility and environmental exposures, with excessive near work and insufficient outdoor exposure potentially accelerating myopia onset and progression among children with higher genetic susceptibility [17]. These findings support a multifactorial model in which environmental exposures may modify the phenotypic expression of genetically determined susceptibility.

With respect to astigmatism, genetic contributions should likewise not be overlooked. Significant associations between parental refractive errors and refractive status in offspring have been reported [18,19]. The Hong Kong Children Eye Study found that parental histories of astigmatism and myopia were both associated with an increased risk of astigmatism in children, suggesting that genetic susceptibility and shared family environmental factors may jointly contribute to the development of astigmatism. Furthermore, family-based and twin studies conducted in different populations have demonstrated variation in the estimated genetic contribution to astigmatism, suggesting that the phenotypic expression of astigmatism may be influenced by population-specific genetic backgrounds as well as lifestyle and environmental factors. Thus, although the genetic architecture of astigmatism remains less well characterized than that of myopia, current evidence supports the existence of both hereditary and environmental components.

Notably, genetic background may influence not only the natural history of refractive errors but also the response to preventive and therapeutic interventions. Systematic reviews have indicated that the effects of certain myopia-prevention and control strategies, including increased outdoor exposure and low-dose atropine, may vary according to genetic background [17–20]. At the same time, environmental factors remain important modifiable determinants. Increased academic demands, prolonged near work—including reading and electronic screen use—and reduced outdoor exposure during school age may accelerate axial elongation and consequently contribute to rapid myopia progression [21–23]. This association became particularly evident during periods of home confinement and online learning associated with the COVID-19 pandemic, when multiple studies reported reductions in outdoor activity and increases in electronic screen exposure accompanied by higher rates and greater degrees of myopia than those observed during corresponding periods in previous years.

Taken together, these observations indicate that genetic susceptibility and environmental exposure should be considered within the same developmental framework. Importantly, however, the available evidence for environmental determinants of astigmatism is less consistent than that for myopia. Therefore, environmental factors should not be assumed to affect both refractive conditions to the same extent. Rather, gene–environment interactions may influence the timing, magnitude, and progression of myopia and astigmatism through partially overlapping but potentially

distinct pathways. This distinction is important when considering individualized strategies for refractive error prevention and control.

2.3 Age and Sex

In addition to genetic and environmental determinants, age and sex represent important modifiers of refractive development. Multiple large-scale cohort studies, cross-sectional investigations, and systematic reviews have demonstrated that both variables are associated with differences in the onset, prevalence, and progression of myopia and astigmatism.

Age appears to be a particularly important determinant of the temporal pattern of myopia development. The incidence of myopia increases substantially with age among school-aged children and adolescents, with adolescence representing a critical period during which myopia progression may accelerate; this pattern has been consistently reported across multiple countries and regions [22–24]. Astigmatism also undergoes age-related changes in magnitude, distribution, and axis. Corneal and refractive astigmatism exhibit distinct developmental patterns among children and adolescents, with changes in astigmatic type and axis occurring with increasing age [21–25]. These findings indicate that both myopia and astigmatism should be regarded as dynamic rather than static refractive conditions during childhood.

Sex may further modify these developmental trajectories. Females generally demonstrate a higher prevalence and faster progression of myopia than males and frequently emerge as an independent risk factor in multivariable analyses [24]. Although the mechanisms underlying these sex-related differences remain incompletely understood, differences in ocular growth, pubertal timing, educational exposure, and behavioral patterns may contribute. Importantly, age- and sex-related differences may also influence the observed association between astigmatism and myopia by modifying the timing and magnitude of refractive changes.

Taken together, these age- and sex-related patterns provide an important developmental context for understanding the interaction between astigmatism and myopia. Because both refractive conditions evolve dynamically during childhood, their relationship is unlikely to remain constant over time. This temporal dependence provides an important rationale for examining the potential bidirectional mechanisms through which astigmatism may influence ocular growth and, conversely, myopia may alter astigmatic characteristics.

3. Potential Mechanistic Interactions

3.1 Effects of Astigmatism on Myopia

Epidemiological studies suggest an association between childhood astigmatism and myopia; however, the biological basis of this relationship remains incompletely understood. Increasing attention has therefore been directed toward the possibility that astigmatism may influence myopia development by altering retinal image quality and the visual signals involved in ocular growth. Importantly, these mechanisms remain largely hypothetical in humans, and available evidence should be interpreted as supporting biological plausibility rather than establishing causality.

One potential mechanism involves persistent degradation of retinal image quality caused by astigmatic blur. Studies analyzing wavefront aberrations after Toric ICL implantation and SMILE have shown that residual preoperative astigmatism is significantly associated with higher-order aberrations (HOAs), suggesting that astigmatism may contribute to degraded retinal image quality [26,27]. Because visual input plays an important role in regulating emmetropization, persistent retinal blur may potentially interfere with the feedback mechanisms governing ocular growth. In myopic eyes, where the focusing mechanism and retinal image quality may already be altered, substantial uncorrected astigmatism could further increase visual blur and thereby compromise the normal regulation of refractive development [28]. However, the extent to which optical blur caused specifically by astigmatism translates into axial elongation in children remains uncertain.

Evidence from animal models provides additional support for this hypothesis. Experimental studies in monkeys and chicks have demonstrated that astigmatic visual conditions can alter patterns of axial growth and refractive development [29–31]. Although animal models cannot fully reproduce the complex optical, neural, and environmental conditions of human childhood, these findings provide experimental evidence that directional retinal image degradation can influence ocular growth. Consistent with this concept, cross-sectional studies have reported greater differences in HOAs among individuals with compound myopic astigmatism than among those with simple myopia, potentially indicating differences in retinal image quality between refractive states [32]. Nevertheless, because these studies are primarily observational, they cannot determine whether increased aberrations contribute to myopia progression or instead arise as a consequence of ocular growth.

At the neurovisual level, retinal and central visual pathways may provide an additional link between astigmatic blur and axial growth. Reviews of Müller glial cells have suggested that these cells may respond to visual signals associated with blur or defocus and potentially participate in the regulation of axial growth through metabolic, ionic, and mechanotransductive pathways [33]. Moreover, prolonged exposure to blurred visual input has been associated with neuroplastic changes in the adult visual cortex, raising the possibility that persistent abnormal visual input may induce adaptive changes within visual processing pathways [34]. Although these findings do not directly demonstrate that astigmatism causes myopia, they provide a potential biological framework through which chronic optical degradation could influence refractive development.

The magnitude and orientation of astigmatism may further modify its potential effect on visual development. Clinical and epidemiological studies have shown that high astigmatism is relatively common among individuals with myopia, and uncorrected astigmatism may be associated with axial elongation and changes in spherical-equivalent refraction in myopic eyes [35]. However, substantial heterogeneity among studies has prevented definitive conclusions regarding causality, particularly in children. In

addition, the visual consequences of astigmatism may vary according to axis orientation. Oblique and ATR astigmatism may produce greater degradation of visual quality than WTR astigmatism under certain optical conditions, potentially increasing visual discomfort or accommodative demand [36]. Therefore, rather than assuming that all forms of astigmatism exert equivalent effects, future studies should consider astigmatic magnitude, axis orientation, age, accommodation, and the interaction between anterior and internal astigmatism when evaluating their potential influence on myopia progression.

Taken together, the available evidence supports a biologically plausible pathway linking astigmatism to myopia through altered retinal image quality, visual feedback, and potentially neurovisual signaling. Astigmatism produces directional and asymmetric blur that may persist across different retinal locations and visual conditions. Experimental evidence indicates that abnormal visual input can modify ocular growth, while clinical studies suggest that higher astigmatism and specific astigmatic patterns may be associated with greater axial elongation. Nevertheless, the available evidence does not yet establish that astigmatism is an independent causal driver of myopia progression. Instead, astigmatism may act as one component of a broader visual and biological environment that influences refractive development.

3.2 Effects of Myopia on Astigmatism

The potential interaction between astigmatism and myopia may not be unidirectional. Whereas astigmatic blur may influence the visual signals regulating ocular growth, progressive myopia and axial elongation may subsequently alter ocular geometry and astigmatic characteristics. Evidence supporting this reverse pathway primarily involves longitudinal changes in axial length, corneal shape, and ocular biomechanical properties.

3.2.1 Axial Elongation and the Evolution of Astigmatism

Longitudinal population-based studies provide some of the strongest evidence for an association between myopic ocular growth and changes in astigmatism. Studies following children and adolescents have shown that astigmatic magnitude may increase as axial length progressively elongates, and some individuals also exhibit changes in astigmatic axis, including transitions from WTR toward ATR astigmatism [6]. These observations suggest that axial elongation may be accompanied by systematic changes in the refractive surfaces of the eye. However, because axial elongation and astigmatic changes may be influenced by common developmental factors, the observed temporal association does not by itself establish a direct causal effect of axial elongation on astigmatism.

3.2.2 Corneal and Scleral Biomechanical Mechanisms

Changes in the biomechanical properties of the cornea and sclera may provide a potential structural explanation for the association between myopic axial elongation and astigmatic changes. Multiple cross-sectional studies using corneal biomechanical measurements have reported that myopic eyes exhibit reduced corneal stiffness and increased anisotropy, with these properties being associated with axial length and corneal astigmatic magnitude [37]. Mathematical modeling has further suggested that reduced corneal stiffness may amplify the effects of axial elongation on the corneal refractive surface, thereby potentially contributing to astigmatic changes [38]. Emerging technologies, including optical coherence elastography, have additionally demonstrated associations among corneal biomechanical anisotropy, corneal curvature, and tissue deformation, providing new biophysical evidence for investigating the relationship between ocular biomechanics and refractive development [39].

These findings raise the possibility that myopia-related changes in the biomechanical environment of the ocular wall may influence the shape and stability of the cornea. Nevertheless, the cornea and sclera are anatomically and mechanically distinct structures, and the precise pathways linking scleral remodeling to corneal astigmatism remain incompletely understood. Accordingly, biomechanical abnormalities should currently be considered a potential intermediary mechanism rather than an established causal pathway.

3.2.3 Anterior–Posterior Corneal Coupling

In addition to global biomechanical changes, alterations in the geometric relationship between the anterior and posterior corneal surfaces may contribute to astigmatic changes in myopic eyes. Multicenter studies have demonstrated significant positive or negative correlations among anterior and posterior corneal curvature, astigmatic magnitude, and asphericity across different levels of myopia [40]. These findings suggest that progressive myopia may be accompanied by coordinated changes in anterior and posterior corneal geometry, potentially modifying the overall corneal refractive contribution.

Furthermore, Mendelian randomization studies have reported associations between corneal biomechanical parameters, including corneal hysteresis and the corneal resistance factor, and the development of myopia and astigmatism [41]. Such findings raise the possibility that biomechanical properties may contribute to both refractive conditions. However, genetic instrumental-variable approaches are dependent on the validity of their underlying assumptions and should therefore be interpreted as complementary evidence rather than definitive proof of a direct biological pathway.

Overall, the available evidence supports a multilayered model in which myopia-related axial elongation may be accompanied by changes in ocular biomechanics and corneal geometry, which in turn may influence astigmatic magnitude and axis. In this framework, changes in the anterior and posterior corneal surfaces may interact with the biomechanical properties of the cornea and sclera, while lenticular and internal astigmatic compensation may further modify the final refractive astigmatism. Thus, changes in astigmatism during myopia progression may reflect the combined effects of ocular growth, corneal remodeling, internal optical compensation, and biomechanical adaptation rather than a single mechanism.

Despite these advances, current research remains limited by insufficient population diversity, inadequate long-term follow-up, and limited longitudinal application of integrated biomechanical parameters such as the stress–strain index (SSI) and stiffness parameter at first applanation (SP-A1). Future studies should therefore combine longitudinal biometric measurements with multimodal corneal imaging, wavefront analysis, and biomechanical assessment. Such an integrated approach may help distinguish whether changes in astigmatism precede, accompany, or follow axial elongation and thereby clarify the directionality and causal pathways of the interaction between myopia and astigmatism.

4. Clinical and Public Health Implications

The evidence reviewed above suggests that childhood astigmatism and myopia should not be regarded as completely independent refractive abnormalities. Instead, their relationship may reflect a dynamic interaction involving retinal image quality, ocular growth, corneal geometry, biomechanical properties, and genetic and environmental determinants. This perspective has important implications for both early clinical identification and public health strategies.

From a clinical perspective, children with higher levels of astigmatism in early childhood may warrant closer surveillance for subsequent myopic development, particularly when astigmatism is accompanied by other established risk factors for myopia. Epidemiological and longitudinal studies indicate that higher astigmatic magnitude may be associated with subsequent refractive abnormalities and axial elongation, although the evidence is insufficient to establish astigmatism as an independent causal risk factor. Therefore, astigmatism may be better considered a potential marker of increased refractive risk rather than a definitive predictor of myopia progression.

At the same time, the parallel changes observed between axial elongation and astigmatic magnitude suggest that monitoring only spherical-equivalent refraction may overlook clinically relevant changes in the cylindrical component of refraction. Comprehensive childhood vision screening should therefore include spherical and cylindrical power, astigmatic axis, visual acuity, and, where feasible, axial length. Serial assessment of these parameters may provide a more complete picture of individual refractive development and help distinguish transient developmental astigmatism from persistent or progressive astigmatic changes associated with ocular growth.

The interaction between refractive abnormalities and environmental exposures also has implications for preventive strategies. Increased screen-based near work and reduced outdoor exposure are well-established risk factors for myopia, whereas their independent contribution to astigmatism remains less clearly established. Accordingly, environmental interventions should primarily be directed toward established modifiable risk factors for myopia, particularly maintaining adequate outdoor exposure and managing excessive near-work demands. At the same time, appropriate correction of clinically significant astigmatism may help optimize retinal image quality and visual function, although whether astigmatic correction directly modifies axial growth requires further prospective evidence.

At the public health level, the Guidelines for Appropriate Techniques for the Prevention and Control of Myopia in Children and Adolescents and related vision-screening standards emphasize routine monitoring of refractive status, establishment of vision health records, and comprehensive risk assessment. These measures provide a practical framework for population-level primary prevention and early identification of children at increased risk of refractive abnormalities. Integrating longitudinal monitoring of spherical and cylindrical refraction with axial-length assessment, where resources permit, may further improve risk stratification. Coordinated interventions involving schools, families, and communities—including promotion of outdoor activity and reduction of excessive near-work exposure—may therefore contribute to the prevention and control of childhood myopia while supporting overall visual health.

Importantly, the clinical implications of the astigmatism–myopia relationship should be interpreted cautiously. Current evidence supports concurrent monitoring of both refractive components but does not justify treating astigmatism alone as a sufficient indication for myopia-control therapy. Future prospective studies should determine whether specific characteristics of astigmatism — including magnitude, axis, persistence, and changes over time — can improve prediction models for myopia onset and progression and whether targeted correction of astigmatic blur can modify axial growth.

5. Conclusion

The relationship between childhood astigmatism and myopia is complex and appears to involve multiple interacting biological, environmental, and developmental factors. Epidemiological studies consistently demonstrate substantial co-occurrence between the two refractive conditions, with higher astigmatic magnitude in some populations being associated with increased risk of myopia and greater axial elongation. At the same time, longitudinal evidence indicates that myopic ocular growth may be accompanied by changes in astigmatic magnitude and axis. These findings collectively suggest that the relationship between astigmatism and myopia may be at least partially bidirectional.

From one perspective, astigmatism may influence myopia development by altering retinal image quality and visual feedback. Persistent directional blur, increased optical aberrations, and potentially altered neurovisual signaling may affect the regulatory mechanisms governing ocular growth. Experimental animal studies provide biological support for this hypothesis, whereas human epidemiological evidence remains predominantly associative. Thus, although astigmatism may represent a potential visual-signal modifier of myopia development, current evidence is insufficient to establish it as an independent causal determinant of myopia progression.

Conversely, progressive myopia and axial elongation may influence astigmatic characteristics through changes in ocular geometry and biomechanical properties. Alterations in corneal curvature, anterior–posterior corneal coupling, and corneal biomechanical behavior may contribute to changes in

astigmatic magnitude and axis, while internal lenticular compensation may further modify the final refractive phenotype. However, these mechanisms remain incompletely characterized, and the precise causal relationships among axial elongation, corneal biomechanics, and astigmatic progression require further longitudinal investigation.

Importantly, the interaction between astigmatism and myopia does not occur in isolation. Genetic susceptibility, environmental exposure, age, and sex may modify both the timing and magnitude of refractive development and may partly explain the heterogeneity observed across populations. Consequently, differences in study populations, measurement methods, follow-up duration, and environmental exposure should be carefully considered when interpreting apparently inconsistent findings.

Overall, current evidence supports substantial interaction rather than definitive causality between childhood astigmatism and myopia. Astigmatism may function both as an accompanying refractive characteristic and as a potential marker of altered visual input during ocular development, whereas myopia-related axial elongation may subsequently contribute to changes in corneal geometry and astigmatic characteristics. Future prospective longitudinal studies incorporating serial refraction, axial-length measurements, corneal tomography, wavefront aberration analysis, and biomechanical assessment are needed to clarify the temporal and causal relationships between these two refractive conditions. Such evidence may ultimately support more accurate risk stratification, individualized refractive correction, and integrated strategies for the prevention and control of childhood myopia.

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