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Review Article

Mechanical Coupling of Extraocular Muscles, Optic Nerve Tension, and Orbital Fat as a Driver of Directional Posterior Ocular Deformation

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Posterior ocular deformation is central to myopia-associated structural change, yet a unified mechanical explanation for directionally biased deformation remains lacking. We propose a biomechanical hypothesis in which extraocular muscles, optic nerve tension, and orbital fat act as a mechanically coupled system that generates directional loading on the posterior globe. In this framework, the medial rectus contributes posteriorly directed force, the superior oblique contributes a rotational component, optic nerve tension transmits and concentrates posterior traction, and orbital fat provides a viscoelastic constraint that redistributes load within the orbit. Their combined action is proposed to bias posterior ocular deformation in specific directions, thereby providing a common mechanical basis for optic disc tilt, optic disc torsion, and related posterior pole shape changes. This article develops the mechanical logic of the model, distinguishes it from scalar growth-centered accounts of myopia progression, and outlines testable predictions for imaging, gaze-dependent biomechanics, and clinical intervention. The model is intended as a hypothesis-generating framework for directional ocular biomechanics rather than as a completed experimental account.

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1. Introduction

Posterior ocular deformation is a defining structural feature of myopia and is closely associated with optic disc tilt, optic disc torsion, and related changes of the posterior pole. While substantial progress has been made in characterizing axial elongation and scleral remodeling, existing frameworks

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predominantly describe deformation in scalar terms and do not account for its directionality. In particular, the mechanisms that generate consistent directional bias in posterior ocular shape remain insufficiently explained within current models.

In this article, we propose a biomechanical hypothesis in which extraocular muscles, optic nerve tension, and orbital fat act as a mechanically coupled system that produces directional loading on the posterior globe. In this framework, muscle-generated forces and rotations, optic nerve traction, and viscoelastic orbital support interact to generate anisotropic deformation of the posterior pole. This coupled system provides a unified mechanical basis for directionally biased phenomena, including optic disc tilt and torsion, and suggests that posterior ocular deformation should be understood as the emergent result of multi-component mechanical interactions rather than isolated tissue-level processes.

Current accounts of myopia have largely emphasized visual regulation, axial elongation, scleral remodeling, and biochemical signaling pathways. These approaches are important for describing growth-related change, but they do not by themselves specify how directional deformation patterns arise at the posterior pole. In particular, they are better suited to explaining the magnitude of elongation than the directional geometry of deformation. A framework centered only on scalar growth therefore remains incomplete when applied to phenomena such as optic disc tilt, optic disc torsion, and asymmetric posterior pole shape change.

The present hypothesis addresses this gap by shifting the primary explanatory focus from tissue growth alone to force transmission within the orbit. Rather than treating the globe as an isolated structure, we consider it as mechanically embedded within a coupled system that includes extraocular muscles, the optic nerve, and orbital fat. Within such a system, directional posterior deformation can emerge from the interaction of posteriorly directed muscle loading, rotational components of force, traction transmitted through the optic nerve, and viscoelastic redistribution of load by surrounding orbital tissues. The proposed model is therefore not intended to replace established growth-related mechanisms, but to complement them by accounting for the directional mechanics that they do not explicitly resolve.

This article is concerned primarily with human ocular biomechanics, especially directionally biased posterior deformation associated with near work, optic disc tilt, and optic disc torsion. It does not attempt to provide a complete account of all forms of myopia, all developmental growth processes, or all experimental animal models. Rather, its purpose is to establish the internal mechanical logic of a coupled-orbit hypothesis, to show why directional deformation requires a vector-based framework, and to derive testable predictions that can be examined in future imaging, biomechanical, and clinical studies.

2. Clinical spectrum: usual myopia, tilt, and torsion

Typically, myopia is often perceived clinically as a relatively symmetrical elongation of the eyeball. On the other hand, tilt indicates a directional deviation of the optic nerve head insertion site and can be considered a phenotype with strong bending or shearing. Furthermore, torsion is a twisting of the optic nerve head structure around its axis and is the phenotype with the strongest directional dependence. Tilt and torsion also have different visual field defect patterns and cannot be considered the same[1][2][3].

From this perspective, it is more natural to consider typical myopia, tilt, and torsion not as separate disease groups with different etiologies, but rather as phenotypic spectrums that come to the forefront due to differences in loading direction, constraint conditions, and material response within the same mechanical system. The general idea is that typical myopia occurs under relatively symmetrical posterior pole loading, tilt appears when posterior pole displacement and shear are prominent, and torsion appears when the torsional moment is strong.

3. Why intraocular-only explanations are insufficient

When considering the etiology of tilt/torsion, it is first necessary to clearly distinguish between clinically important stages. Posterior staphyloma is a structural change that appears in pathological myopia (generally -8 D or higher, often accompanied by degenerative findings in the fundus), and is characterized by extensive thinning of the sclera and asymmetrical posterior pole bulging. In contrast, tilt and torsion are already clinically present at the stage of moderate myopia (around -3 D to -6 D). That is, at the time tilt/torsion appears, neither posterior staphyloma nor severe scleral degeneration is often present. This fact demonstrates the limitations of explaining tilt/torsion as an associated phenomenon of pathological myopia or as a consequence of extensive scleral material deterioration[2][4][5][6].

Since intraocular pressure acts basically isotropically, it can determine local bulging sensitivity, but it is difficult to naturally induce a torque with a rotational direction by this alone. Here, we consider the position that attempts to explain directional deformation solely by structural asymmetry within the eyeball. It is known that the lamina cribrosa has asymmetry in thickness in the vertical and nasotemporal directions, and that there are local differences in the compliance of the peripapillary sclera.

If isotropic intraocular pressure acts on these local compliance differences, asymmetric deformation can occur in principle. However, this mechanism can only explain preferential bulging in the direction of low compliance, i.e., deformation in the tilt direction. Torsion—that is, rotation around the axis of the papillary structure—by definition requires a torsional moment. Here, it can be argued that since the collagen fibers of the peripapillary sclera have circumferential and radial orientation anisotropy, bending–torsional coupling occurs due to the interaction between isotropic pressure and anisotropic material, and a rotational component can be generated even without external torque. However, even if this endogenous mechanism exists, collagen orientation is anatomically almost fixed within the same individual, so the predicted torsion direction is uniquely determined within that individual. Clinically, there is inter-individual variation in the direction of torsion (medial and lateral rotation), and asymmetry can even be observed between the left and right eyes within the same individual. This inter-individual and interocular variation is difficult to explain with passive coupling from a fixed collagen structure and suggests the involvement of a variable external torque source. Therefore, while differences in local compliance and collagen anisotropy can modify tilt direction preference and torsion sensitivity, explaining the direction of torsion and its inter-individual variation requires the introduction of a separate variable torque source from outside the eyeball[3][6][7][8][9][10][11].

Furthermore, the optic nerve head is a highly constrained area surrounded by supporting structures such as the lamina cribrosa, ring of sclera, and optic nerve sheath. Morphological changes such as rotation and tilt are more mechanically consistent when understood as the result of sustained directional external loading rather than passive yielding of the supporting structures. In particular, the clinical fact that tilt/torsion appears at a stage of moderate myopia when extensive material degradation of the sclera and lamina cribrosa is not yet significant further strengthens the motivation to assume external mechanical factors as the driving force of deformation[2][6][8][9].

In summary, the difficulties faced by explanations based solely on internal ocular factors are twofold. Firstly, the rotational component of torsion cannot be derived from the combination of isotropic pressure and scalar compliance difference. Secondly, the stage in which tilt/torsion appears precedes posterior staphyloma and severe scleral degeneration, which does not align with explanations based on widespread material degradation. For these reasons, it is necessary to actively consider the external mechanical environment within the orbit as a factor that determines the direction of tilt/torsion[3][4][5][6].

4. Core biomechanical chain

Figure 1 shows a conceptual diagram of the mechanical relationship between the medial rectus muscle, superior oblique muscle, and optic nerve as proposed in this paper.

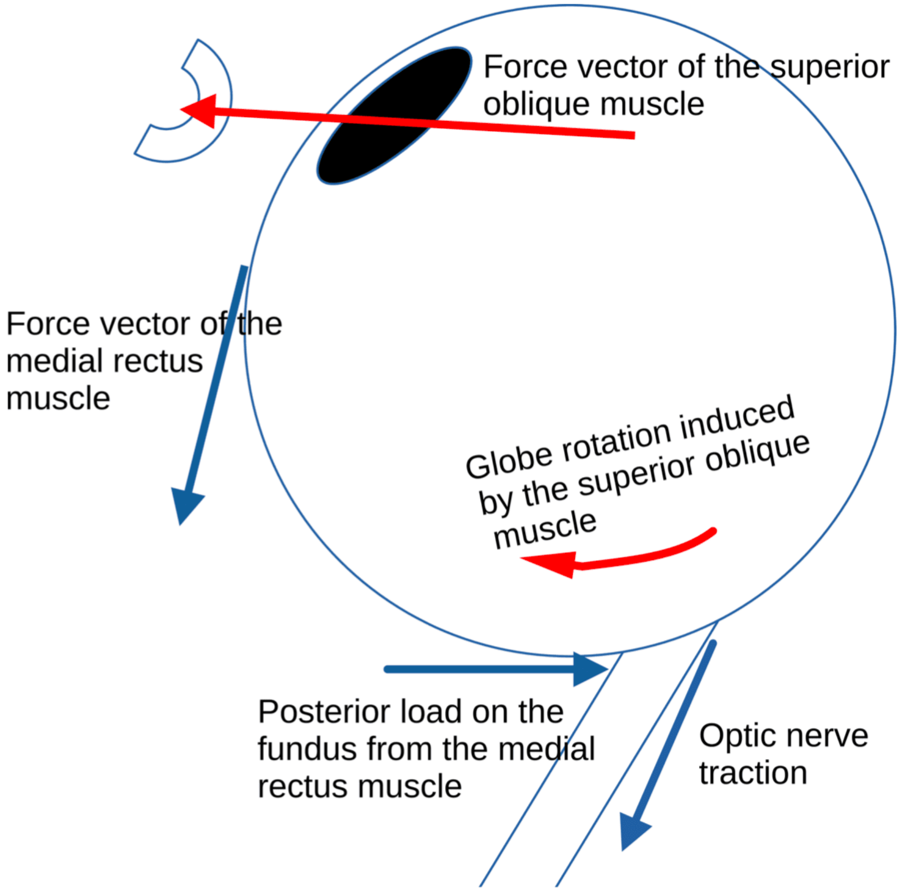

Figure 1. Proposed mechanical coupling among the medial rectus, superior oblique, and optic nerve. The schematic illustrates a hypothesized load path in which the medial rectus generates posterior loading on the posterior globe/fundus, while the superior oblique contributes a rotational component of the globe. Their combined action is proposed to increase optic nerve traction and thereby bias posterior ocular deformation. Arrows indicate the assumed directions of force, rotation, and traction. The figure is conceptual and not to scale.

4.1. Extraocular muscle loading during near work

During close work, not only accommodation but also convergence is sustained, increasing the load on the medial rectus muscles of both eyes. During prolonged close work, it is thought that medial rectus muscle contraction due to convergence, leverage action at the back of the eyeball, loss of optic nerve perforation, and downward rotation by the superior oblique muscle occur simultaneously, resulting in the simultaneous generation of tensile, shear, and torsional stresses near the optic disc[12][13][14][15].

Here, let us clarify why the superior oblique muscle is identified as the torque source. The only extraocular muscles capable of generating rotational moment in the eyeball are the superior oblique, which is responsible for inward rotation, and the inferior oblique, which is responsible for outward rotation. The inferior oblique is primarily active during elevation, but since close work is essentially performed by looking straight ahead or downward, the postural conditions for sustained load on the inferior oblique do not exist during close work. On the other hand, the superior oblique is the main contributor to downward rotation in adduction and is continuously active under the typical postural conditions of convergence and downward looking during close work. Therefore, by process of elimination, the only extraocular muscle capable of repeatedly supplying rotational torque during close work is the superior oblique[13][15].

In this framework, a load predominantly on the medial rectus muscle primarily generates posterior pole displacement and shear, while the component originating from the superior oblique muscle contributes to rotational moment. Therefore, close work can be understood not merely as “the act of looking at things up close,” but as a mode of behavior that repeatedly generates a directional stress field within the orbit[12][13].

4.2. Optic nerve tethering as load amplification

The effect of optic nerve tethering is a continuous phenomenon that depends on the adduction angle. [16] confirmed clear optic nerve tension and eyeball retraction by MRI at a large angle of adduction of approximately 26°, but this is the condition under which tension becomes apparent, and does not mean that the mechanical contribution is zero at angles below this. The convergence angle during near work (usually 5–10°) does not produce significant optic nerve tension on its own, but when it is sustained for several hours, combined with convergence and downward gaze, and superimposed with shortening of the optic nerve excess in myopic eyes [17], stress transmission around the optic disc gradually increases. In other words, optic nerve tethering is not a binary “on/off” phenomenon, but acts as a load amplification factor that is continuously amplified as a function of angle, time, and axial length[18][19][20][21][22][23].

4.3. Orbital fat viscoelasticity as the fixation mechanism

The asymmetrical redistribution of orbital fat is thought to act as a “morphological memory anchor,” making it difficult for the eyeball to return to its original shape even after the load is removed. In other words, muscles provide direction, the optic nerve amplifies the load, and orbital fat preserves the history. This allows transient deformations to become semi-fixed under repeated load[12][24][25][26].

This hypothesis is consistent with recent rheological data on the material properties of orbital fat. [26] systematically measured the shear rheology of human orbital fat and reported $\tan\delta \approx 0.27$ (storage modulus $G' \approx 737$ Pa, loss modulus $G'' \approx 197$ Pa). A $\tan\delta$ of 0.27 means that approximately 27% of the elastic response is coexisting with viscous dissipation, indicating incomplete immediate recovery after load removal. Furthermore, the extremely large fracture strain of $200 \pm 70\%$ and the low fracture shear stress of 617 ± 366 Pa indicate that orbital fat is a material that tolerates large deformations with relatively small stresses. This characteristic means that it has a high structural tolerance to the accumulation of repeated small deformations. Importantly, the semi-fixation timescale assumed by this model is not a linear viscoelastic response on a second-to-minute basis, but rather the accumulation of plastic deformation and adipose tissue remodeling in the nonlinear region during a process in which several hours of close work are repeated on a daily and monthly basis. As is generally observed in soft tissues, repeated loading exceeding the linear viscoelastic limit (0.5% strain according to [26]) can induce irreversible rearrangement of microscopic structures. Direct measurement of this long-term remodeling process in orbital fat has not yet been performed, and this is one of the important validation challenges for this model.

By clearly defining this division of roles, the causal chain in this model becomes considerably simpler:

- Extraocular muscles = determine direction
- Optic nerve = amplify stress
- Orbital fat = make it difficult to return to normal

This is a three-tiered structure. In this paper, these three will be presented as the main series.

5. Mechanical differentiation of phenotypes

Typical myopia can be understood as a phenotype where relatively symmetrical posterior pole loading is dominant. In this case, the axial component is dominant, and the posterior extension of the entire eyeball is brought to the forefront, but clear optic disc rotation is not essential. In contrast, in the tilt-dominant type, posterior pole deviation and peripapillary shear are strong, and the optic nerve entry angle and optic disc tilt are prominent. Furthermore, in the torsion-dominant type, the contribution of extraocular muscle moments with a rotational component is relatively large, and twisting around the axis is emphasized[1][2][3][6].

In actual clinical practice, these are thought to appear as mixed forms rather than pure forms. Therefore, it is more natural to treat myopia, tilt, and torsion as a continuous distribution arising from a common dynamical system, rather than as independent categories. This framework also explains the clinical fact that tilt/torsion appears prominently in only some individuals, even though close work load is almost universal. In other words, inter-individual variability can be understood as a result of individual differences in anatomical parameters such as orbital shape (depth, medial wall angle), extraocular muscle course and trochlear geometry, optic nerve length, orbital fat volume and distribution, and scleral compliance, causing the same close work load to manifest as different phenotypes[10][12][13][17][24].

6. Simplified formalization

The focus of this paper is on a conceptual hypothesis, but in order to demonstrate the order validity of the proposed dynamical system, a minimal formulation is provided. The moment due to the medial rectus muscle is

$$M_m = T_m R \sin\theta_m \tag{1}$$

where T_m is the medial rectus muscle tension, R is the ocular radius (sphere approximation), and θ_m is the angle between the muscle direction and the anterior–posterior axis of the eyeball. Using literature values of $T_m = 0.5\text{–}1.0$ N (based on clinical measurements in [14]; [15], under continuous convergence conditions during close work), $R = 11.75$ mm (emmetropia) to 14.0 mm (high myopia), and $\theta_m \approx 30^\circ$, $M_m \approx 3\text{–}7 \times 10^{-3}$ N·m.

The rotational shear stress due to the superior oblique reinforcement can be estimated as

$$\tau_z \approx \frac{T_{so} \cdot b_z}{2\pi R^2 t} \tag{2}$$

by analogy with the torsion of a thin-walled closed-section tube (Bredt–Batho equation). Here, T_{so} is the tension of the superior oblique reinforcement ($0.3-0.8$ N), b_z is the moment arm, and t is the rigidity of the wall thickness. In an emmetropic eye ($R = 11.75$ mm, $t = 0.8$ mm), $\tau_z \approx 1.4$ kPa, whereas in a highly myopic eye ($R = 14.0$ mm, $t = 0.4$ mm), τ_z increases to 3.9 kPa under the same muscle tension. That is, the covariance of the increase in R and decrease in t associated with myopia progression amplifies the shear stress for the same muscle tension to approximately 2.8 times that of emmetropic eyes. In pathological myopia ($R = 16$ mm, $t = 0.3$ mm), this amplification reaches approximately 4.8 times. This positive feedback structure explains the accelerated appearance of tilt/torsion as myopia progresses.

The above estimation represents the shear stress (scleral wall stress) directly applied to the sclera by the extraocular muscles through their tendon attachments. Even under the most pessimistic parameter setting ($T_{so} = 0.3$ N, emmetropic eye condition), $\tau_z \approx 1.4$ kPa, indicating that the extraocular muscle load is at a biologically significant level for scleral deformation. As myopia progresses from emmetropia to high myopia, the increase in R ($11.75 \to 14.0$ mm) and the decrease in t ($0.8 \to 0.4$ mm) co-variate, amplifying the scleral wall shear stress under the same muscle tension by approximately 2.8 times, and reaching approximately 4.8 times in pathological myopia ($R = 16$ mm, $t = 0.3$ mm). This amplification structure is consistent with the clinical impression that even if the extraocular muscle load is latent in the early stages of myopia progression, it rapidly becomes apparent beyond a certain stage.

On the other hand, the semi-fixation of orbital fat is evaluated from a mechanical pathway independent of scleral wall stress. The extraocular muscles attach directly to the sclera via tendons, and orbital fat is not the primary medium for transmitting muscle force. However, the rotation and displacement of the eyeball associated with convergence and downward gaze directly shear the fat at the ocular–orbital interface. According to [26], the linear viscoelastic limit of orbital fat is a strain of 0.5%, and the linear stress limit is only about 3.7 Pa, given $G' \approx 737$ Pa. The shear strain that eyeball rotation during convergence ($5-10^\circ$, i.e., $0.09-0.17$ rad) imparts to the fat layer between the eyeball surface and the orbital wall geometrically far exceeds this limit, and the fat easily enters the nonlinear region. The low fracture shear stress of 617 ± 366 Pa and the large fracture strain of $200 \pm 70\%$ indicate that orbital fat is a material that tolerates large deformations with small stresses but does not fully recover. Therefore, in this model, scleral deformation (direct loading via tendons) and the nonlinear response of orbital fat (interfacial shear associated with eye movement) are distinguished as two complementary mechanical processes driven by the same extraocular muscle activity. The former determines the direction and degree of deformation, while the latter contributes to the semi-fixation of the deformation history.

It should be noted that the above estimates are based on the thin-walled shell approximation and do not take into account stress concentration near the papillary opening or the curvature effect of the spherical shell. Since the papillary opening is a point of structural discontinuity, it is structurally expected that stress concentration factors of 2–4 will occur around the opening. Therefore, the above estimates are rather conservative (underestimated), and the effective stress near the papilla is likely to be even higher than these estimates. Finite element analysis is necessary to predict the precise stress distribution, but the purpose of this formulation is to show that the proposed mechanical chain can generate biologically meaningful stresses even within a conservative parameter range.[8][9][18][22]

7. Discussion

This section summarizes the verifiable predictions, clinical implications, and limitations of this model derived from this hypothesis. This will position the central argument of this paper from both the perspective of verifiability and scope.

7.1. Testable predictions

This model provides at least three clear predictions. Firstly, immediate changes in the shape of the papilla periphery and the position of the posterior pole should be observed before and after close work. Secondly, the recovery after load removal should not be instantaneous, but rather involve a viscoelastic time constant. Thirdly, reducing convergence load and managing working distance should slow down the rate at which tilt/torsion progresses[16][19][20][21][23][26][27].

Furthermore, the following predictions specific to this model can be derived. Fourth, the direction of torsion should be consistent with the direction of torque generated by the superior oblique muscle (inward rotation), and statistically, in groups with a strong habit of close work, inward rotation-dominant torsion should be more common. Fifth, in eyes with congenital superior oblique muscle palsy, ipsilateral torsion should be suppressed or the direction of torsion should be different compared to healthy eyes. Sixth, the acceleration of tilt/torsion associated with myopia progression reflects stress amplification due to an increase in R and a decrease in t (positive feedback), so the relationship between axial length and tilt/torsion should not be linear, but rather show a nonlinear relationship that accelerates beyond a certain threshold[3][5][13][17].

In addition, the direction of optic disc deformation is predicted to be consistent with the direction of the dominant extraocular muscle torque, and asymmetry in sleeping and working postures is predicted to exacerbate the left–right difference. If these predictions are supported, it strongly suggests that direction-dependent optic disc deformation is not a random morphological difference but a reflection of the external mechanical field. In particular, the fifth prediction (torsion suppression in superior oblique muscle-palsy eyes) is specific to this model and cannot be derived from an explanation based solely on endogenous mechanisms[3][13].

7.2. Clinical implications

If this model is correct, myopia management is insufficient with only monitoring refractive error and axial length; managing intraorbital mechanical load becomes crucial. Factors such as convergence intensity, working distance, asymmetrical posture, and rest frequency, in addition to close work time, should be re-evaluated not merely as lifestyle guidance items, but as risk factors for morphological progression. Interventions including reducing close work load and convergence should also be reconsidered in this context.

Furthermore, myopic eyes with tilt/torsion should be understood not simply as having “longer eyeballs,” but as “eyes that have been subjected to long-term directional stress.” This may lead to advancements in imaging diagnosis to include not only static fundus images, but also integrated evaluations such as dynamic OCT, dynamic MRI, optic nerve geometry, and orbital fat distribution[16][19][20][21][26].

7.3. Limitations

This paper presents a conceptual and mechanically integrated model, and at present, it is not based on direct dynamic image evidence or simultaneous measurements of individual material constants. The necessity of this model is most clearly demonstrated in the case of torsion. Since torsion requires an external torque source, tilt, which is the shear component of the same mechanical system, also comes into consideration, and conventional myopia is further positioned on the continuum as its symmetric limit. This paper does not negate the known factors of conventional myopia (genetics, ambient light, retinal signals, etc.), but the extent to which these factors cause morphological changes and in which direction can be modified by the intraorbital mechanical environment[28][29]. Arbitrarily cutting off one end of the continuum would only create boundary problems that need to be explained. It should be noted that the main subject of this paper is human near-work myopia, and it does not claim mechanistic agreement with developmental animal models.

In this paper, the choroid and RPE are positioned as susceptible and modifying factors, while the extraocular muscles, optic nerve, and orbital fat are placed in the main sequence as directional determinants. Furthermore, this model does not completely reject known factors such as the sclera and choroid, but rather takes the position of rearranging them as materials and support conditions that determine "where deformation is likely to occur."

In addition, animal models of lens-induced myopia and form-deprivation myopia have established that axial elongation occurs without extraocular muscle loading. However, these models demonstrate isotropic or quasi-isotropic control of axial growth by the retina–RPE–scleral signaling pathway during development, which is a different phenomenon from the directional deformation (tilt/torsion) that occurs under repetitive close work in adult humans[28][29]. Animal models answer "why the axial lengthens," but they do not answer "why the optic disc tilts and rotates in a specific direction." This model attempts to explain precisely the latter, and the two are consistently integrated as a hierarchical structure. That is, the axial elongation mechanism established in animal models dictates the material conditions (increased scleral compliance, increased R, decreased t), and the orbital mechanics of this model then imparts directionality. The aforementioned shear stress amplification structure (2.8–4.8 times that of emmetropia) reflects this hierarchical causal chain.

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Potential Competing Interests

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Data Availability

No datasets were generated or analyzed for this study. This paper presents a theoretical/conceptual model based on published literature.

Author Contributions

MN: Conceptualization, formal analysis, writing—original draft, writing—review and editing.

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