Commentary

A Mechanical Model of Myopia: Implications of Eye Movements and Orbital Fat Redistribution

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Current myopia models cannot explain important clinical findings such as unilateral progression, adult onset, and optic disc tilt. We hypothesize that chronic downgaze induces mechanical traction via the superior oblique muscle, leading to orbital fat redistribution and axial elongation. This model redefines myopia as a potentially reversible mechanical disease. This hypothesis is testable through longitudinal MRI and ultrasound imaging and may lead to novel treatment strategies.

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Introduction

Myopia is a rapidly growing global public health concern, projected to affect nearly 50% of the world's population by 2050[1][2]. Existing myopia models, broadly divided into genetic growth models and tissue remodeling models, fail to adequately explain several phenomena, including the recent surge in incidence, progression into adulthood, and unilateral progression. Furthermore, while the tissue remodeling theory emphasizes biochemical pathways, it rarely takes into account the mechanical influence of eye movement and orbital structures. These points highlight the need for an alternative framework.

The Hypothesis

This hypothesis is based on the following causal chain:

1. During prolonged downward gaze, the eyeball is translated nasally and superiorly (toward the trochlea) by traction from the medial rectus and superior oblique muscles[3].

2. This displacement causes asymmetric redistribution of orbital fat.
3. Asymmetric external pressure generates mechanical stress, inducing axial elongation and scleral remodeling[4].

The direction of movement of the eyeball and orbital fat is shown in Figure 1.

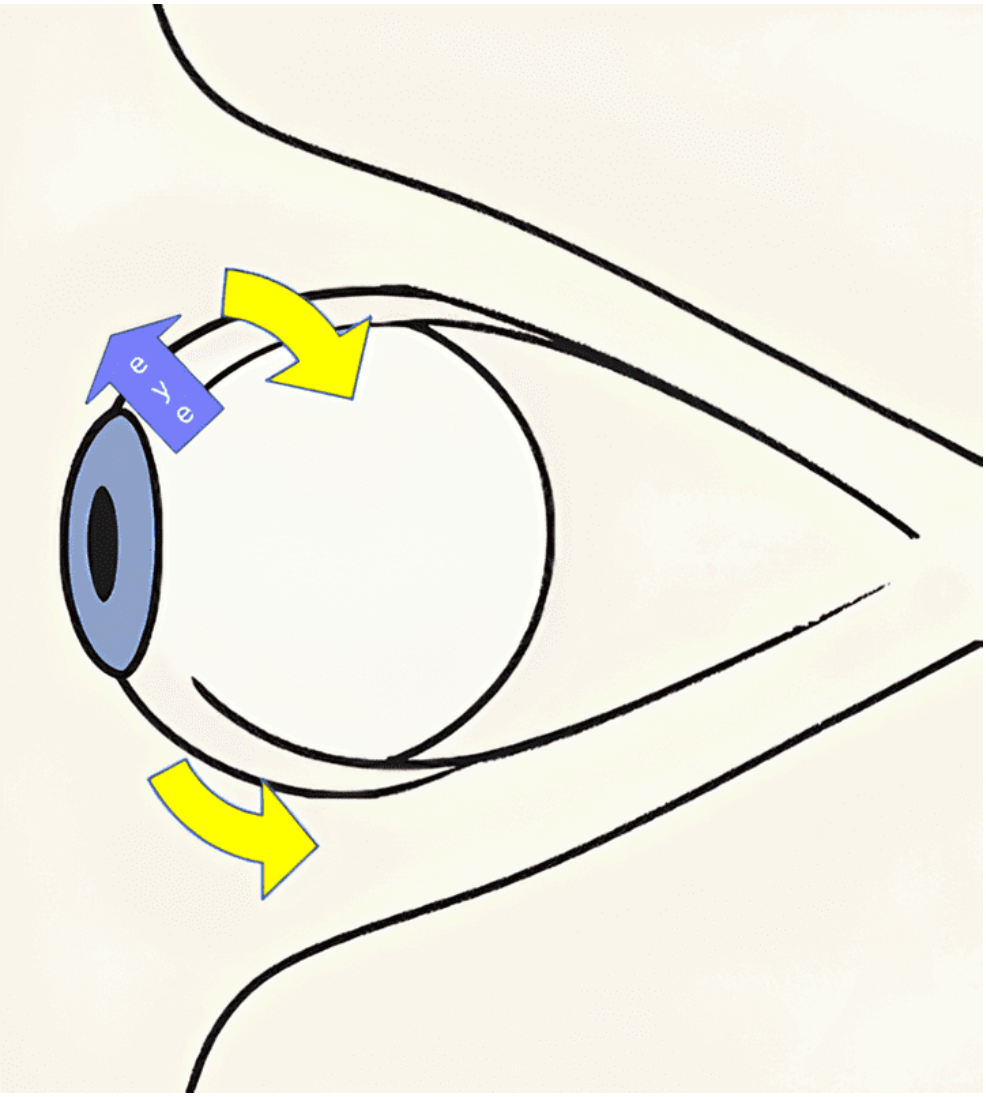

Figure 1. Direction of movement of the eyeball and orbital fat

Theoretical Rationale

Myopia is a condition in which the axial elongation of the eye causes defocusing, resulting in blurred images. This raises a fundamental question: what induces axial elongation? The causes of axial

elongation can be broadly divided into endogenous and exogenous factors. Endogenous factors can be divided into growth (cell proliferation) and tissue remodeling models. In other words, if there is a discrepancy between the growth and tissue remodeling models, it suggests that exogenous mechanical factors may play a more central role than previously recognized.

Endogenous Growth Model

The growth model has traditionally been recognized as a genetic factor and posits that axial elongation occurs due to growth (cell proliferation). However, the growth model has several significant limitations that limit its explanatory power. For example, it cannot explain the progression of myopia that occurs after adulthood, a time when growth ceases for most organs, including the eyes. Furthermore, thinning of the sclera, choroid, and retina is observed as myopia progresses. However, if growth were the cause, these thicknesses would remain constant, which is a contradiction. Furthermore, this model cannot explain the recent rapid increase in the global incidence of myopia, which is increasing at a rate far exceeding genetic transmission[1][2]. Moreover, it has difficulty explaining unilateral myopia, visual acuity improvement from axial myopia, and optic disc tilt and rotation observed in high myopia.

Endogenous Tissue Remodeling Model

The tissue remodeling model posits that scleral thinning leads to axial elongation. In the tissue remodeling model, visual input, i.e., retinal defocus, is thought to trigger a physiological response. First, the retina senses this defocus and generates a physiological signal. This signal is transmitted to the sclera via the choroid. In the sclera, fibroblasts respond to this external signal, triggering extracellular matrix (ECM) remodeling. This process involves molecules such as matrix metalloproteinases (MMPs), their inhibitors (TIMPs), transforming growth factor-β (TGF-β), which promotes fibroblast differentiation and induces collagen synthesis, and reactive oxygen species (ROS), which are involved in stress responses[5][6][7][8]. As a result, the collagen structure of the sclera changes, causing the eye's axis to elongate. This change in axial length alters the retinal image, which in turn alters the signal from the retina, prompting further responses. This series of mechanisms is known as "open-loop control," and it is believed that control continues until the retinal image defocus is resolved.

Contradictions in the tissue remodeling model

The only cells within the sclera are fibroblasts (stromal cells), accounting for 1–4% of the total. While fibroblasts are capable of receiving and responding to external signals, they lack neural input or sensory receptors and lack the ability to provide feedback to the retina or neural pathways. They also lack the ability to autonomously "judge" or "adjust" anything. They also lack the ability to accumulate a history of physiological signals and reflect them throughout the tissue. Because myopia rarely heals naturally, the visual image remains unchanged and defocus signals continue to occur frequently. In this model, continued defocus signals would result in the sclera softening indefinitely. Furthermore, the sclera is an extremely dense connective tissue in which triple-helical type I collagen fibers are highly cross-linked, forming a continuous lamellar structure around the entire circumference. The essence of a lamellar structure is its continuity, which does not allow for partial modification. Local modification would mean a disruption of continuity and lead to structural breakdown. It is structurally contradictory to say that the sclera can be locally modified while maintaining its continuity. Furthermore, it is biologically unlikely that genetically identical tissue would become locally thin and elongate in one direction. Moreover, the sclera has few blood vessels and an extremely slow metabolic rate (turnover is measured in years), making it impossible to explain the rapid progression of myopia. The tilt and rotation of the optic disc that occurs in severe myopia is also difficult to explain[9]. In conclusion, the more natural explanation is that the sclera thinned as a result of mechanical stretching. The idea that the sclera is "making itself fragile and stretching itself" is an extremely unstable control method for a living organism.

Exogenous Mechanical Deformation Model

Biomechanical Basis

It is known that gaze movement involves not only rotation but also translation (positional change)[3].

- Horizontal gaze movement involves a translational distance of approximately 0.7 mm.
- Vertical gaze movement involves not only rotation but also translation in the opposite direction, approximately 0.45 mm.
- In particular, the reverse translation during vertical movement suggests an antagonistic action between the rectus and oblique muscles, with the oblique muscles being stronger.
- Orbital fat is highly plastic, and its shape and position change with sustained pressure[10].

Myopia Progression Mechanism

External eye muscles and orbital fat are the only structures outside the eye that can physically affect the eye. In addition, numerous existing studies have demonstrated a strong relationship between close work and myopia[11][12]. The superior oblique muscle is an extraocular muscle that rotates the eyeball inward and downward via the trochlea. During close work, the eye turns inward and downward. This is the role of the superior oblique muscle. In other words, excessive use of the superior oblique muscle is thought to be the origin of myopia. During prolonged close work, the eyeball is pulled by the superior oblique muscle, rotating downward toward the nose, and then translating inward and upward (toward the trochlea). As a result, the orbital fat near the trochlea disperses to the depths of the inferior nasal and temporal regions. The direction of movement of the eyeball and orbital fat is shown in Figure 1. At the same time, nearby orbital fat moves into the low-pressure area created on the opposite side of the eyeball's translation. As this process repeats, the translational state becomes fixed, the eye axis elongates, and myopia progresses.

Mechanical Deformation Model and Genetics

It is known that the rate of myopia in East Asia is higher than in other regions, and genetics is thought to be involved[13][14]. However, as mentioned above, there is no direct gene that elongates the eyeball along its axis. In other words, there are likely structural genetic characteristics that make the eyeball more susceptible to misalignment. Bone structure is a likely candidate. Asians have higher eye positions, wider openings, and more forward-facing eyes, which may reduce their tolerance for misalignment[15].

This hypothesis can explain all of the following phenomena:

- Rapid increase in global myopia prevalence: Increase in near work due to the spread of smartphones
- Consistent explanation from mild to pathological myopia: Progression in proportion to the increase in near work
- Consistent explanation from young to old: Progression in proportion to the increase in near work
- Association between near work and myopia: Prolonged traction by the superior oblique muscle
- Association between genetics and myopia: Orbital structure
- Progressive unilateral myopia: Asymmetric posture during near work
- Improvement in visual acuity in axial myopia: Due to coincidental shifts in orbital fat
- Optic disc tilt in severe myopia: Due to the overweight of orbital fat

Supporting Evidence

Biomechanical Basis

- When moving the gaze, the horizontal translation distance is approximately 0.7 mm, and the vertical translation distance is approximately 0.45 mm[3].

Biochemical Basis

- Mechanical stress promotes the TGF-β pathway and MMP expression in scleral fibroblasts, leading to ECM remodeling[4].

Integrated Model

Long-term unidirectional traction of the eyeball → physical factors (eyeball translation and orbital fat redistribution) → local scleral strain → activation of the MMP/TGF-β pathway → ECM structural changes → axial elongation[4].

This process more easily explains many clinical observations (e.g., unilateral progression, adult onset, optic nerve head tilt) than purely biochemically controlled models.

Recent longitudinal observations suggest that optic disc tilt may progress over time as axial length increases, indicating that optic disc tilt may be acquired rather than congenital[16][17][18]. Optic disc tilt and rotation have been reported to occur frequently, particularly in myopic eyes[19]. Optic disc tilt results in a physical structural deformation in which the nasal side protrudes forward and the temporal side sinks backward. The angle is smaller on the nasal side (steep tilt, suggesting the possibility of localized mechanical stress) and larger on the temporal side (gentle tilt). In addition, the structure of the neuroretinal ring is shifted toward the temporal side. In the mechanical deformation model, the rotation and translation of the eyeball due to traction by the superior oblique muscle cause the nasal side of the posterior pole of the eyeball to fold and be pulled forward, while the temporal side sinks backward. This is consistent with the symptoms of optic disc tilt.

Predictions and Testability

- Longitudinal MRI could reveal changes in the posterior pole of the eyeball in the prolonged near-work group, as well as a correlation between orbital fat movement and axial elongation.

- Changes in the posterior pole of the eyeball and axial length can be confirmed after interventions that physically alter the orbital fat position.
- Detailed MRI data on the distribution of orbital fat and extraocular muscles in healthy individuals have been reported, and comparison with data from myopic individuals provides valuable differential data[20][10][4].

Implications and Predictions

- Treatment: If this mechanical deformation model is valid, myopia becomes a potentially treatable disease.
- Treatment: Orbital fat induction therapy is an option for use in combination with or as an alternative to drug therapy or optical therapy.
- Diagnosis: MRI and ultrasound images allow simultaneous assessment of fat distribution and axial length.
- Prevention: Improving posture during close work and reducing overuse of the superior oblique muscle are new preventive strategies.

Limitations

- Details of orbital fat progression remain unclear, particularly on the temporal side.
- Orbital fat induction can potentially improve visual acuity to some extent, but significant visual recovery is unlikely.

Conclusion

We present a new hypothesis that translational displacement and orbital fat redistribution associated with close work are the mechanical drivers of axial elongation.

This hypothesis provides a framework for integrating mechanical factors and biochemical pathways and points to new directions for myopia research and clinical intervention.

We hope that future longitudinal image analysis and numerical modeling will verify the validity of this hypothesis.

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