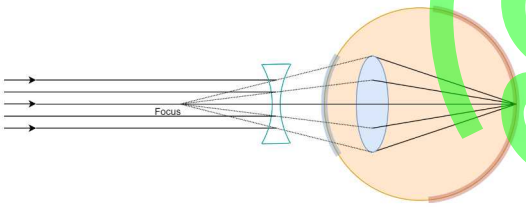

Physiologic Myopia: Breakthrough Adaptive Theory predicting conditions for emmetropization

5 May 2023, 23:50 Draft Revision 0.61

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0. Abstract

Current understanding is incomplete when it comes to explaining observations about physiologic Myopia. We aim to propose an alternative framework for explaining these observations taking into account the accurate interplay of multiple environmental and lifestyle factors for physiologic Myopia.

Careful physical characterization resulted in the two breakthrough refractive state equivalences. These equivalences together form the framework of our Continuous Adaptation Theory (CAT) that treats physiological Myopia as an adaptative response of the eye. By extension, the same adaptive mechanism responsible for onset of Myopia (in emmetropic eyes) is predicted to be behind Myopia progression also and can induce hyperopic adaptation to stabilize Myopia progression and even possible emmetropization.

The breakthrough insights on light therapy when combined with behavioural interventions for myopia management if implemented properly could result in first clinical observations of long-term axial shortening translating into significant visual improvements.

0.1 Keywords

Axial Shortening, Progression, Myopia Management, Emmetropization

0.2 Ethics declarations

The author declares no competing interests.

0.3 Abbreviations

ILS – Ideal Lens System, RDS – Relative Dioptre Scale, AL – Axial Length, CAT – Continuous Adaptation Theory, ADV – Actual Distance Viewing

1. Introduction

Myopia – is a refractive error widely regarded as irreversible with a suspected multifactorial aetiology lacking any viable long-term solution[1]. Extreme axial elongation[2] resulting from severely high Myopia increases risk of vision threatening complications (even after appropriate refractive compensation) including but not limited to Open-angle glaucoma, cataract, retinal tears that can lead to retinal detachment, and macular degeneration (also termed myopic maculopathy)[3]. It is estimated that by 2050, half of the world's population will be myopic if the current trends continue[4].

There is an urgent need for development of interventions that can reverse/stabilize or at least slow down long-term rate of Myopia progression. This article describes breakthrough adaptive characterization (Continuous Adaptation Theory) for commonly observed physiologic Myopia. The governing hypothesis behind the theory is viability of axial shortening and emmetropization for Physiologic Myopia. It proposes that common myopia results from simultaneous 'push-pull' interaction of near-work induced accommodative stress (pull) combined with underutilisation of distance vision (push). The role of lighting levels only serves to direct adaptation from blur thereby providing an answer to this decade long open question to 'how eyes decide direction of growth from blur?'.

2. Background

This article employs prefixes like *compensated* myopic/hyperopic eye or suffixes like a myopic/hyperopic/presbyopic eye *wearing prescription* to denote an eye using refractive interventions different from a naked eye without any intervention. The mention of the word infinity or symbol ∞ should be taken to mean optical infinity at a distance of 10 m or greater (≤0.1 D).

We have strived to provide terminologies and abbreviation whenever applicable at the beginning of each section.

The objective of this article is to answer the following questions.

1. What are the factors/causes behind physiologic Myopia?
2. Why so far there has been still no well established aetiology for physiologic Myopia?
3. Why is Myopia considered irreversible so far?
4. Can a truly viable management protocol/method exist for physiological Myopia? There are mounting evidences that seemingly contradict widely assumed irreversibility of Myopia and hint at the existence of a viable protocol that can result in possible emmetropizationi.

2.1 Methodology

Article selection reporting experimental results/observations used terms associated with primarily physiological Myopia and human eyes on PubMed. Special emphasis was on including articles reporting

unusual or conflicting observations about Myopia. The presentation follows the natural order: physiological myopia (onset and stabilization or progression) → proposed causes of Myopia → effects of myopia → Current Myopia management methods. Characterizing physiological aspects of vision was done using a grounds up mixed mode approach involving ray optics to rule out suspected improper isolation of physiologic variables responsible for Myopia.

2.2 Physiologic Myopia and Pathologic Myopia

Physiologic Myopia is the proper inability to bring distant objects into focus compared to an emmetropic eye (image formation behind the retina even after relaxing accommodation). All observed instances of myopic refractive error can be categorised under physiologic or pathologic/malignant/degenerative Myopia[5]. Even high degree of refractive error from physiologic Myopia (increasing risk of sequelae) must be distinguished from Pathologic Myopia alongside pathological complications that can accompany highly severe forms of physiologic Myopia[6]. Pathologic myopia is often associated with high myopia and complications of the fundus such as degeneration of the macula, optic nerve, or the peripheral retina. Nevertheless complications (for instance, posterior staphyloma) have been observed to occur in eyes without high myopia and even in emmetropic individuals[7].

For the scope of the article, Physiologic Myopia is defined as Myopia without other ocular anomalies except the ones from axial elongation. This is required from the standpoint of consistent classification of Myopia – Myopia can be either termed physiological or pathological but not both in the sense that Physiological Myopia can develop pathological complications later on indicating differences in the underlying mechanism and possible control/management. This article is limited to the scope of Physiological Myopia unless stated otherwise.

2.3 Animal models of Experimental Myopia and Form Deprivation

The exact aetiology of physiological myopia is still elusive so far[8]. However there are a large pool of studies documenting experimental myopia in animals. We expect only a subset of these observations to actually extend to the human eyes.

Both hyperopia and myopia resulted in young chicks when convex and concave lenses respectively were used as defocus indicating the capability of the eye to respond and direct axial changes in the opposite direction to compensate for the induced lens. Also observed was the disproportionately stronger response of myopic defocus compared to same duration of hyperopic defocus as long as the cycle time wasn't too frequent (< 30 minutes)[9]. Once external defocus was removed, induced experimental Myopia slowed and then reversed towards emmetropia suggesting that the built in mechanism is able to detect and swiftly adapt to changes in external stimulus. This was validated for higher primate (rhesus macaques) as well[10].

Efforts to induce Form Deprivation Myopia were successful even after Optic Nerve Section (ONS) indicating that the eye possesses internal mechanisms that can still direct axial changes (this mechanism

may also act as a fall back)[11]. This is extended and further supported from evidences regarding hemi-retinal form deprivation influencing local axial changes in infant monkeys and chicks before eye-growth is completed. The study on infant rhesus monkeys concluded that effects of form deprivation on refractive development in primates are mediated by presumably retinal mechanisms integrating visual signals in a spatially restricted manner[12]. It can be stated that the aforementioned temporally adaptive observations are a consistent feature across multiple species that include higher primates (extending the possibility for humans as well) and that retina acts as the primary initiator for most of these localized refractive changes.

2.3.1 Changes in the eye resulting from Myopia

Hyperopic defocus using minus lenses leads to the observation of choroid thinning (causing the retina to shift backward) increasing AL compensating for the introduced defocus (in this case, resulting in Myopia). Choroidal and scleral thinning was observed to be most prominent at the posterior pole compared to the equator of the eye[13]. Discussing the exact pathways and mechanisms behind these changes is beyond the scope of this article.

Human eyes starts forming around third week of gestation and the process is mostly completed by the tenth week[14]. Eyes continue to grow rapidly after birth to the age of one to two years, and then in a gradual manner until adult eyeball size is usually attained around onset of puberty. The primary goal is to match the eye's axial length with its optical components (emmetropization). Eye size and shape at birth influences subsequent eye growth but is not observed to be associated with refractive error development[15].

The distinct nature of axial elongation in response to external environmental factors separately from eye growth is required to explain observations of focally controlled ocular growth in infant monkeys and chicks. It point towards the possibility that AL changes might result from superposition of growth and adaptive signals after birth. Terming Axial elongation as eye growth conflicts with evidences pointing towards existence of an active emmetropization mechanism. Such a mechanism is possibly present and functional in Human eyes also and influences axial changes apart from eye growth[16]. Axial elongation due to Myopia results in a marked shape change (elongation) distinct from overall (globe) growth phase. For these reasons, we will prefer to use the term axial change/elongation instead of axial growth for this article. This also absorbs the term axial shortening conflicting with the directional connotations of eye growth in its similarity with bodily growth.

2.4 Multi factorial associations of Myopia

2.4.1 Genetics

For decades, investigations were primarily focused on figuring out the suspected genetic causes behind Myopia. There are multiple studies stating Myopia to be somewhat associated with Genetic factors. but evidences are now mounting that at most, there can be genetic susceptibility towards physiological Myopia in the face of mostly missing heritable markers for physiological Myopia unlike the mostly

hereditary/parental nature for cases of pathological Myopia[17]. This argument is further supported from environmental relation of common Myopia including its negative connection with outdoor activity and the recently uncovered protective effect of sunlight[18] from Myopia onset and progression. The environmental associations of physiologic Myopia can't be accounted by genetic considerations alone.

2.4.2 Environmental and behavioural Associations with near work and outdoor exposure

When it comes to physiologic Myopia, its causes are commonly predicted to be a multifactorial interplay of environmental and lifestyle factors. Myopia shows strong correlations with urban lifestyle, higher education, along with intensive visual near-work (defined as eyes working at or near their accommodation limit) that comes with it including but not limited to reading, writing and time spent looking at displays whether on PC/Laptop or smartphones/tablets[19]. Myopia is not significantly associated with time spent doing near work after adjustment for other factors such as age, sex, ethnicity, parental myopia, school type, and outdoor activity. There were independent associations with closer reading distance (< 30 cm) and continuous reading (> 30 minute) leading the authors to speculate intensity rather total duration of near work as influential factor for Myopia. The study additionally reinforces the need for targeted workplace measures to reduce occupational exposure and mitigations such as allowances for regular breaks from near work[20].

2.4.3 Myopia onset and its progression

Myopia onset (in previously emmetropic eyes) is primarily observed in the early childhood stabilizing in the mid twenties. However, Myopia onset can occur at any age. There are observations regarding adult onset and progression of Myopia after enrolling into college[21]. The COVID-19 pandemic also resulted in significant reports of Myopia onset and progression in both adult[22] and school aged children[23] in sync with the lockdown confinement.

The onset of Myopia is usually seen as setting the stage for progression of Myopia. This translates to a disease like presumed aetiology of Myopia where worsening Myopia is viewed as the natural course of its progression. This standpoint is mostly justified in the absence of any viable long-term methods demonstrating the ability to slow or stabilize Myopia progression or possibly reverse it (myopia control[24]). As of now there is still no viable method directly targeting/affecting long-term structural consequences of axial elongation resulting from Myopia.

However, environmental and behavioural factors plays a key role in the development of Myopia via altering the equilibrium of homeostatic control mechanisms present inside the eye has been speculated for decades[[25], [26]]. These observation in combination with the properties of axial changes highlighted in Section 2.3.1 together suggest that an adaptive mechanism might be responsible for human physiological Myopia also. This adaptive standpoint necessitates treating Myopia progression separately from stabilized myopia.

2.5 Prevalent Myopia control and management methods

Myopia control/management refers to interventions[27] concerned with stabilization/reversal of Myopia only and should be distinguished from existing modes of refractive intervention such as lenses, contacts and surgical procedures whose primary goal is to improve vision by compensating for the defocus resulting from Myopia (subjective refraction[28]). The term compensation is more appropriate than using the term 'correction' when it comes to refractive interventions. There can be no fundamental difference between refraction induced by glasses, contacts or refractive surgeries from the standpoint of Ray optics.

The scope of this article is not to review existing myopia control/management methods, only to bring attention to the observation that none of the existing methods can be considered as truly affecting the

2.5.1 The prevalence of Ortho-K and Atropine compared to other approaches

Ortho-K lenses are specially designed rigid gas-permeable contact lenses worn overnight and designed to reshape the corneal refracting surface. They are also designed to temporarily compensate low to moderate myopia demonstrating a clinically acceptable safety profile as a corneal reshaping intervention[29].

Recent clinical trials demonstrated low-dose atropine eye drops such as 0.01% resulted in retardation of myopia progression, with significantly less side effects compared to higher concentration preparation. However, Atropine treatment suffers with frequently observed rebound effects. Suggested mitigations include stepwise increase of atropine dosing, and combining low-dose atropine treatment with lifestyle interventions such as increasing outdoor exposure[30].

However, it must be emphasised that both Ortho-K and Atropine treatment do not account for or alter the environmental association of Myopia outlined previously. These widely recommended management options also frequently suffer from rebound effects[31]. The frequent observations of rebound myopia after cessation of treatment shows that these management strategies do not address the underlying processes and environmental variables affecting axial elongation and can not be treated as such.

2.5.2 Light Therapies

There is recent promising advancement of management strategies towards achieving axial shortening in the form of light therapies. RLRL[32] (Repeated Low-level Red Light) and HEI[33] (High Environmental Illuminance based on protective effect of outdoor daylight exposure) therapies have shown promise for Myopia management. Light therapies build upon the demonstrated safety and efficacy of bright daytime outdoor exposure demonstrated to have protective effect against Myopia onset/progression[34].

3. Refractive characterization of the eye

Refractive characterization of a myopic/hyperopic/presbyopic eye for developing the theoretical framework utilizes Ray optics. The preliminary included with this article serves as an in-depth refresher for the same. This article builds upon characterization of the Ideal Lens System (ILS from now on) along with the

phenomena of shift in observation range from introduced defocus or screen distance changes using Relative Dioptre Scale for visualisation of observation ranges.

3.1 Representing observation ranges on the Relative Dioptre Scale (RDS)

The Relative Dioptre Scale (RDS from now on) emerges from the need for a tool to visualize optical systems and any subsequent modifications to them whether from defocus or screen distancing.

The RDS is simple Cartesian co-ordinate system modified for power (inverse instead of linear distances) onto which observation ranges for optical systems are represented in this article. The x-axis of the RDS represents distances in (D or m^{-1}) with the origin representing infinity (∞). Thus all real world distance lie to the right quadrant on the RDS. The sign of object distance on the RDS is inverted from the sign used for object distance (u) in ideal lens relation due to co-ordinate conventions. The idea of representing observation range of optical systems on an inverse length scale stems from the inverse nature of the ideal lens relation itself.

The ideal lens relation $\frac{1}{v}-\frac{1}{u}=\frac{1}{f}$ itself can then be written as Inverse of Object distance (in m^{-1}) = Lens Power (in D) – Inverse of Screen distance (in m^{-1}). This has the important implication of making transformations on the RDS linear with respect to changes in Power.

The y-axis was chosen to represent the accommodation ability of the system (also in Dioptre). Systems are vertically ranked/placed higher-up on the Relative Dioptre Scale based on their accommodation ability. On the RDS, the left end of the system's observation range represents its far-point while the right end its near-point. Accommodation ability refers to the power difference between the two extremes (near and far-point) of the observation range.

The gradient from red to green and light to dark was used to indicate reaching the end of the system's observation range due to constraints. The 'I' symbol indicates the impossibility of any physical extension towards the right-hand direction (near-point) for a fixed focal length system.

3.1.1 Shift in observation range after defocus (Accommodation shift)

What happens to the system in Error: Reference source not found when accommodation ability is kept same (+5 D) but accommodation itself is incremented by +1 D on both ends (+5 D to +10 D)? Such a change can

be termed as ‘accommodation shift’ and can be easily achieved by introducing another +1 D defocus such that the power addition law holds (Section Error: Reference source not found).

Just like before, it is sufficient to calculate the shifted far and near-points of the system (which are now 1 m and 16.66 cm respectively). The modified system is represented on the RDS (Figure 3.1). The original system is shown using slightly thicker grey for ease of comparison.

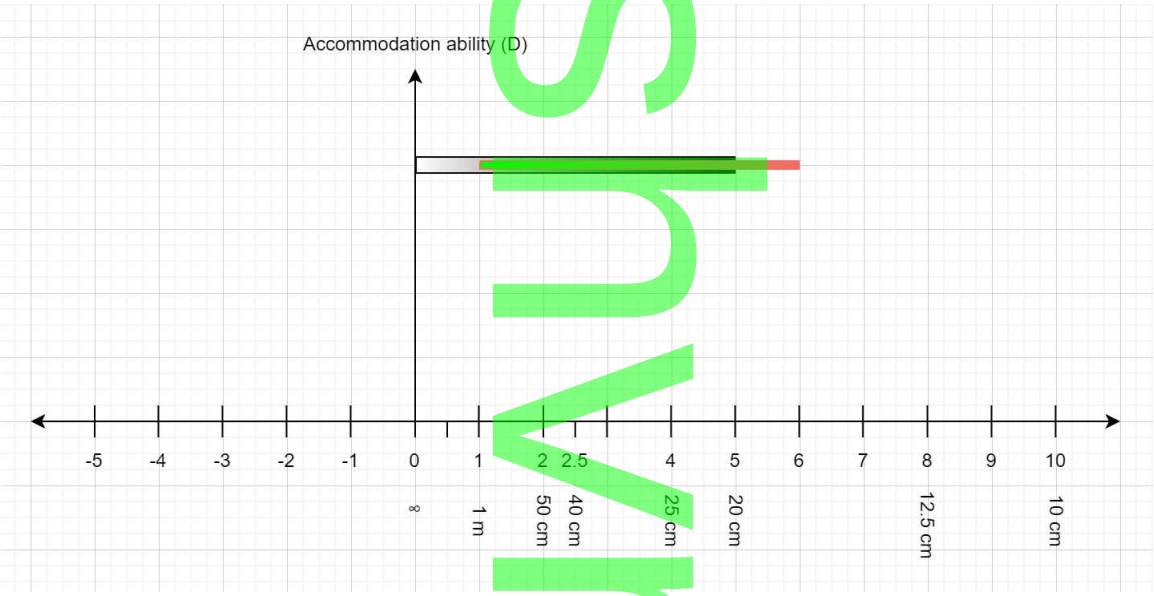

Figure 3.1: Shift in observation range after introduction of +1 D defocus

The modified system’s observation range with Lens Power = +5 D to +10 D gets shifted 1 unit towards the positive/right direction (the sign of introduced lens).

It is evident that an increment in both extremes of the lens power range results in right-shifting of the system by the same amount on RDS. Our modifications to system in Error: Reference source not found has rendered the system Myopic (unable to image objects farther than 1 m). The inverse also holds true. It is trivial to point out that introducing a -1 D accommodation shift in our newly modified system restores the original accommodation range. This is equivalent to introducing a -1 D lens or removing the previously introduced +1 D lens. Which means that the observation range on RDS must also shift one unit towards left. This is what the word ‘Relative’ in the RDS stands for. Relative to our modified system, the system in Error: Reference source not found can be said to be hyperopic (unable to image objects closer than 20 cm).

It should be now easy to understand why *introducing converging (positive) lenses/removing diverging (negative) lenses* to a system is termed as ***Myopic defocus*** – because it renders the system myopic (shifts the observation range towards right).

Similarly, *removing converging (positive) lenses/introducing diverging (negative) lenses* to a system is called ***Hyperopic defocus*** – because it renders the system hyperopic (shifts the observation range toward left).

3.1.2 Shift in observation range after changes to screen distance (Axial changes)

We've previously described shifting of a system's observation range under induced defocus. Calculating shift in Observation range due to screen distance changes is also as easy as recalculating the far and near-points. Changes to the screen distance can also be termed as Axial changes because of changes to the overall size of the system.

For instance, the system in Error: Reference source not found has the screen fixed at 25 cm from the lens. If we position the screen 5 cm closer maintaining the same lens power range, the near-point recedes farther from 20 cm to 25 cm (1 D towards left) signifying that the system has become hyperopic. Similarly, if we move the screen 25cm farther from its initial position – the far-point comes closer from ∞ to 50 cm (2 units towards right) rendering the system Myopic. The near-point also shifts closer (by the same unit amount) from 25 cm to 16.66 cm.

3.2 Lumped Lens consideration of the eye

Our main goal is quantifying observation range changes due to refractive conditions. Instead of taking the traditional approach which has historically been attempts to precisely model complex ocular components inside the eye, we have chosen to study simpler systems like the ILS for the eye.

A labelled diagram[35] of the human eye is given below in Figure 3.2 for reference.

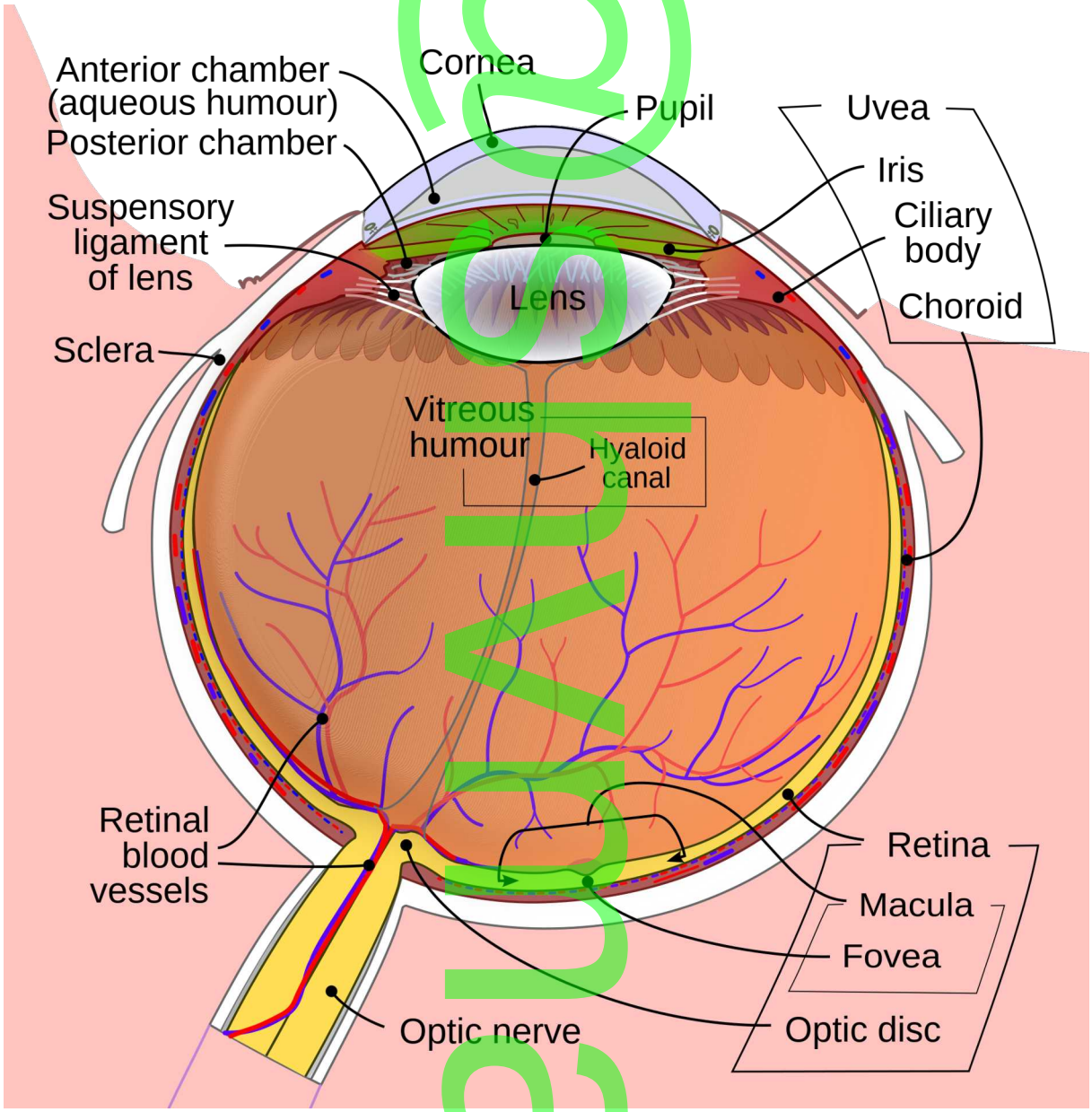

Figure 3.2 Labelled diagram of the human eye. It shows the lower part of the right eye after a central and horizontal section.

The role ocular components play inside the eye are explained in the order in which a light ray entering the eye traces its path towards photoreceptors on the retina.

Cornea → Aqueous humor → Eye lens → Vitreous humor → Photoreceptors on the Retina

The Pupil is a hole in the Iris acting like aperture from ILS. The aqueous & vitreous humors act as an optical medium for light travel – any non-destructive/physiological changes to the index of the refractive media results in power changes, leaving us with remaining ocular components of the eye as:

Cornea → Eye lens → Photoreceptors on the Retina

The cornea and the eye lens can be viewed as two fixed and variable power optical element with the retina acting as an image sensor. During the process of image observation, the relative positions of these components remains unchanged.

The cornea, the eye lens, and the mediums surrounding them combined serve to converge light rays onto a spot on the retina forming an inverted image just like the ILS[36]. For our considerations, this will be referred to as the 'lumped lens' with refractive power of cornea and eye lens combined together and the retina acting as an image screen. The imaginary distance of retina from the optical centre of lumped lens will be referred throughout this article as Retinal Distance (RD from now on) and the same can be treated as screen distance equivalent of the eye. Introducing external defocus/refractive intervention (in the form of glasses, contacts etc.) must take into account the Vertex Distance.

Long term changes from Myopia[37] involve the cornea (changes to the corneal curvature and ACD[38]) and the posterior scleral shape involving physical distancing of the retina[39]. These changes have been incorporated into the lumped lens consideration as shown in Table 1 below.

Table 1: Lumped lens consideration of the eye

Ocular component	Lumped Lens consideration
Accommodation of the eye lens due to ciliary muscle action	Instantaneous/Short-term Accommodation of the lumped lens
Long term changes to the corneal curvature and Anterior Chamber Depth	Long term accommodative shift of the lumped lens
Long term changes to the posterior Scleral shape	Long term changes to the Retinal Distance

3.3 Observation range of an Emmetropic eye

While the far-point of an emmetropic is ideally fixed at ∞, a representative value needs be taken as the near-point of an emmetropic human eye. For this article, we have taken this to be 25 cm for simplicity without loss of generalisation. It represents the closest distance an adult emmetropic eye is capable of continuous focusing without immediate fatigue/discomfort under ordinary circumstances. This value has been represented with a red dashed vertical line at the +4 D mark on the RDS. An elder person might not be able to observe objects this close due to presbyopia. Throughout this article, the word emmetropic without any prefix will refer to *an emmetropic eye without presbyopia* (Accommodation ability of 4 D or greater).

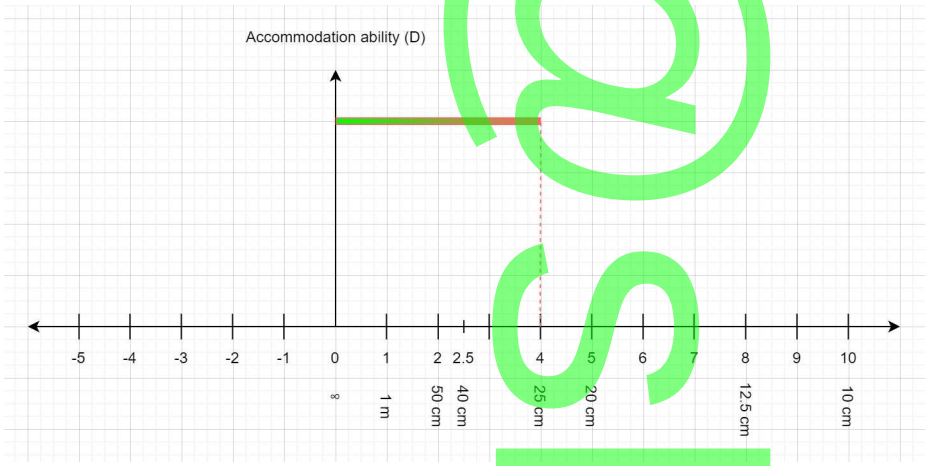

Figure 3.3 The observation range of an emmetropic eye (observation range: 25 cm up to ∞)

Because accommodation is facilitated by the ciliary muscle, the left end of observation range corresponds to relaxation (focusing at far-point) while the right end corresponds to maximum ciliary muscle contraction. We have subdivided the observation range into two distinct regions as shown in Figure 3.4 assuming a neutral point.

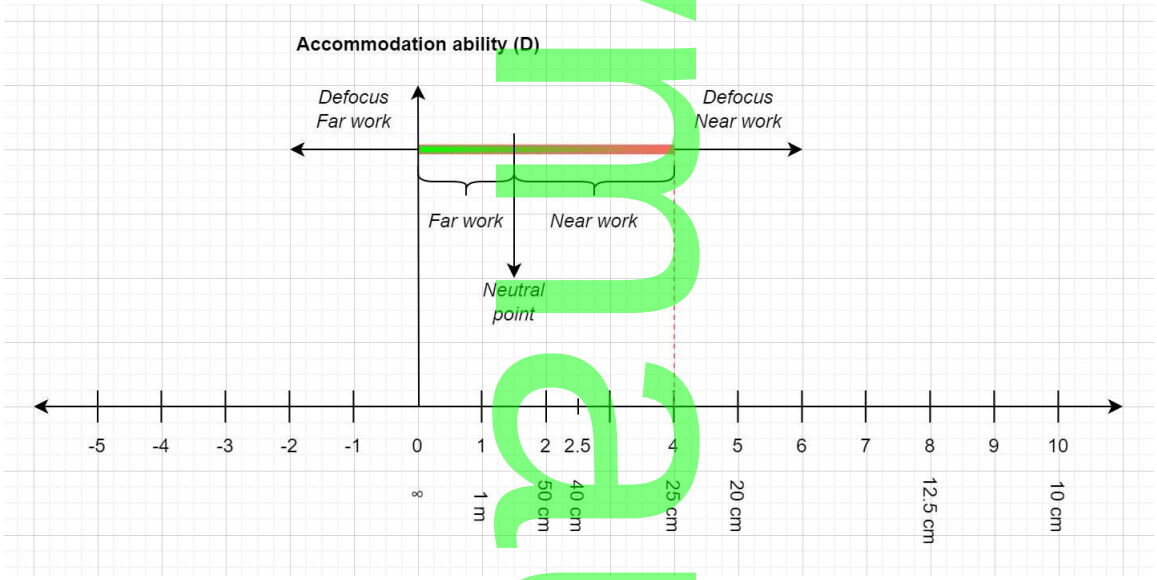

Figure 3.4: Observation range demarcation

This neutral point divides the observation range into two subsets – corresponding to far-work beyond the neutral point [d_{far}, $d_{neutral}$) and Near-work closer than the neutral point ($d_{neutral}$, d_{near}]. Once outside the observation range, defocus far work can be defined as (-∞, d_{far}) and defocus near work as (d_{near}, ∞). It must be noted that these classifications apply regardless of observation range changes.

3.4 Myopia/Hyperopia

For refractive error, a best possible refractive compensation[40] can be determined resulting in an observation range ideally close to an emmetropic eye (Figure 3.3). This best compensated observation range will be referred to as pseudo-emmetropic observation range for the rest of this article.

Assuming the power addition law holds, this refractive compensation can then be 'subtracted' from the pseudo-emmetropic observation range giving the originally uncompensated observation range of the eye. Simply put, by knowing the near and far-points of a myopic/hyperopic eye after proper refractive compensation, its actual observation range can be determined by 'negating' the prescription. Astigmatism, floaters, and aberrations due to retina/refractive media can affect final image quality even with best possible refractive compensation. Visual acuity/resolution is a property primarily associated with the retina that must be differentiated from defocus[41]. It has also implications towards pathological Myopia in that it must be accurately ascertained whether the loss of visual function results from defocus or loss of visual acuity.

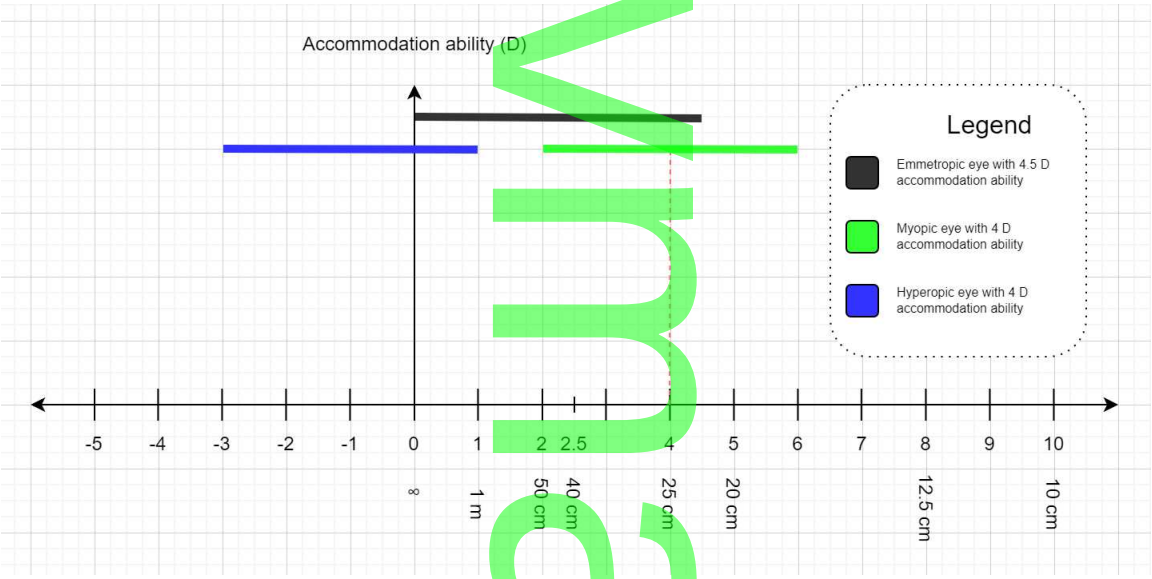

Figure 3.5: Representing Myopic and Hyperopic observation ranges on the RDS

It can be stated that physiologic **Myopia** (green) is a refractive condition in which the eyes' far point (the maximum distance at which the eye can **focus properly**) is closer than/no longer located at optical infinity.

Similarly, physiologic Hyperopia (blue) can be stated as a refractive condition in which the far point is situated well beyond optical infinity resulting in eyes needing to accommodate for focusing at infinity. Hyperopia is much less common than Myopia. Hyperopia/far-sightedness primarily results from axial changes to the eyeball such that images of closer objects are formed behind the retina. Prebyopia unlike Myopia/Hyperopia involves changes to the accommodation ability of the eye. Presbyopia can be also represented on the RDS.

In the lumped lens consideration, both physiologic Myopia/Hyperopia mostly result in screen distance changes which gets compensated by external defocus from refractive intervention. It should be now

intuitive to understand how appropriate refractive compensation restores pseudo-emmetropia. It is also obvious that Myopic eyes need hyperopic shift in observation range for emmetropization and vice-versa.

3.5 Focusing vs. Exposing & Actual vs. Apparent focus

Having already differentiated between image distance and screen distance for ILS, one can extend this distinction in the context of exposing and focusing for the eyes too. This is required in order to be able to describe for instance, the physical possibility in which an eye is **focused** at infinity but **exposed** to a nearby white wall. An eye (optical system) can be exposed to all possible distance but focused only within its observation range. Exposure beyond observation range results in defocus blur from this inability to focus. The characteristic blur due to Myopia/Hyperopia results from *exposing* eyes to distances farther/closer their respective far/near-point. For instance, even if a myopic eye is exposed to and observing infinity, it will remain *focused* at a point closer than infinity. Myopia/Hyperopia is inability to focus distinct from visual acuity.

This focusing and exposing distinction is important to describe what actually happens with refractive interventions. We will refer to focusing after refractive interventions as *apparent* focus to distinguish it from *actual* focusing without refractive intervention. The term **apparent focusing** refers to focusing involving the eye and refractive intervention combination while **actual focusing** refers to unassisted focusing involving only the eyes.

It is obvious that for ***apparent*** focus, a myopic eye must ***actually*** focus at a distance closer than the said distance. For a hyperopic eye to achieve apparent focus at the same distance, it must actually focus farther than the distance in question. Refractive interventions in this context act as a mapping between actual and apparent observation ranges of the eye.

A diagram explaining this for Myopia is given in Figure 3.6:

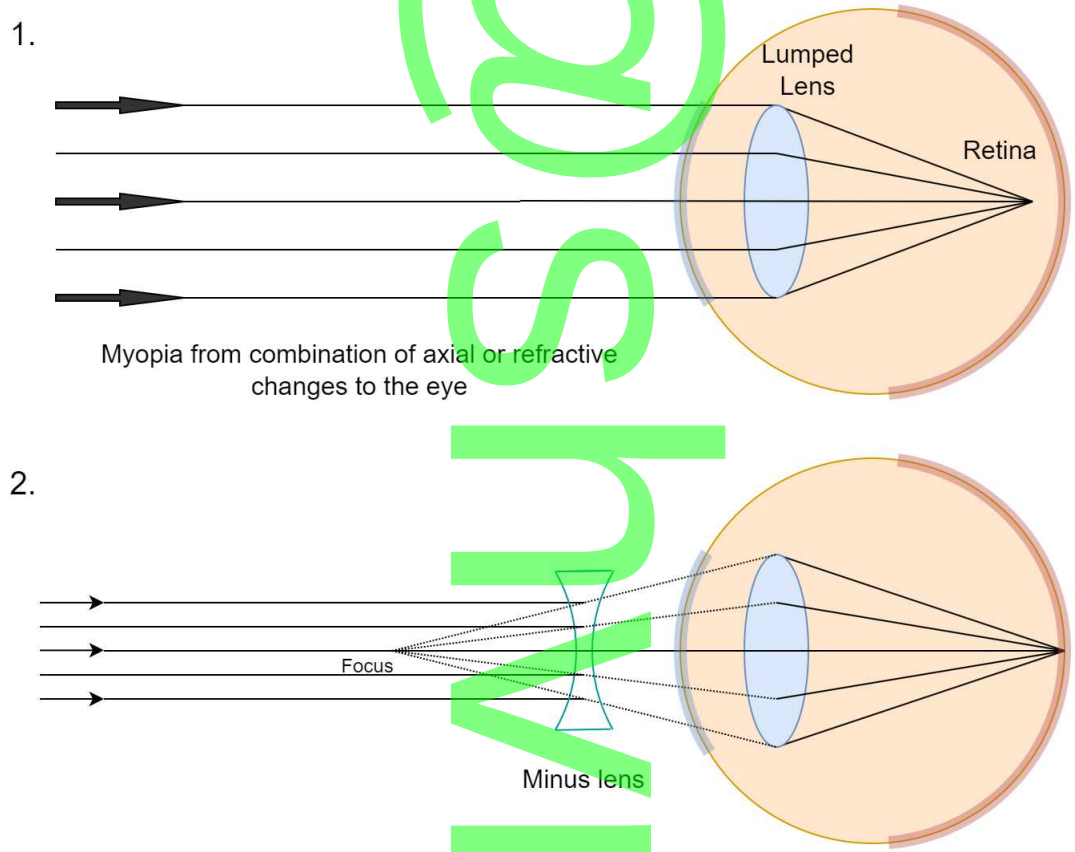

Figure 3.6 A diagram showing how refractive intervention compensate for Myopia.

In Ray optics speak, the introduced prescription introduces additional divergence to the incoming light rays shifting the resulting image plane to coincide with the retinal plane. It is intuitive to visualize how the compensated eye actually focuses at a distance closer than the apparent focusing distance.

3.6 Sign and severity designation of the refractive conditions

From the standpoint of Ray Optics, plus (+) sign for degree of Myopia encodes the true 'compensating' behaviour refractive interventions with opposite (-) sign have on the myopic observation range. It also allows for natural interpretation of under/over compensation without confusion such as "A person with 2.5 D Myopia using -2.0 D refractive compensation."

3.6.1 Severity of Myopia/Hyperopia

According to WHO, High Myopia is defined at 5.0 D spherical equivalent or more. Other literature define High Myopia to start at 6.0 D spherical equivalent or more.

Myopia being mostly axial in nature affects observation range mainly due to SPH nature of defocus. In the interest of rigour, we will treat SPH and CYL components separately. The position of near and far point compared to an emmetropic eye should ideally indicate the severity of myopia/hyperopia. We propose defining refractive state severity on the basis of lack of overlap between the actual observation range and its

pseudo-emmetropic counterpart shown in Figure 3.7. The overlap factor has been taken as the value for threshold Myopia (≥ 0.5 D according to WHO).

Figure 3.7: The 'overlap' criteria for determining severity of Myopia. The coloured observation range represents the pseudo-emmetropic observation range after best possible refractive compensation.

The following arguments favour our overlap criteria for deciding severity of refractive states:

1. Encodes the observation that a person satisfying the criteria is not capable of focusing at a 'reasonable' working distance without refractive intervention. Conversely, subjects with low myopia can focus closer than their far-point comfortably without refractive compensation because of the overlap.
2. Yields equivalent classification as the existing scientific consensus values for Myopia.
3. Predicts an increase in risk with age due to onset of Presbyopia. A borderline severe case of Myopia in adulthood can become severe myopia with age.
4. Severity of Myopia depends on factors affecting observation range which may or may not be affected by refractive errors like astigmatism. This is one of the primary reasons behind avoiding spherical equivalents.

Severe Myopia indicates the difficulty faced by eye's extraocular muscles when focusing close to its near-point (for highly severe myopia, even the far-point may be beyond reach of comfortable convergence without refractive intervention).

3.7 Changes to Accommodation-convergence reflex during Myopia

Human eyes form a stereoscopic pair for depth perception which necessitates the presence of convergence[42]. Convergence is basically simultaneous tilting of the axis of both eyes towards the object

point in focus to form an aligned image. This article is limited to convergence aspects related to Myopia and how accommodation-convergence reflex differs in myopia compared to emmetropia.

For an adult emmetropic eye focusing on an object equidistant from both eyes, the relation between convergence angle θ and the accommodated power of eye is governed by

$\theta = \sin^{-1}\left(\frac{IPD \times Accommodation\ Power}{2}\right)$, where IPD (InterPupillary Distance) has been used as distance between axes of both eyes. The angle between the respective image planes depicted in blue happens to be the sum of convergence angle measured from parallel axes of both eyes (Figure 3.8).

Figure 3.8 Depiction of convergence with observed distance compared to observing at infinity. Note the increasing angle between the perceived image planes shown in blue as focusing distance gets closer.

Using avg. Value of IPD (63 mm) gives 1.8°±0.1° of convergence angle for each eye per Dioptre of Accommodation. For focusing at 25 cm (+4 D accommodation), each eye converges by ~ 7.2°. The increasing angle between the perceived image planes due to stereoscopic fusion as focusing distance comes nearer should also be noted.

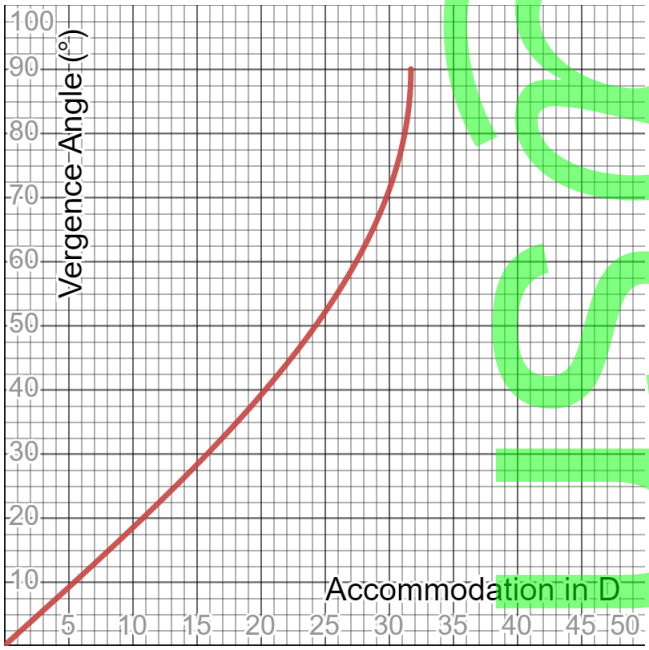

Figure 3.9: A plot of convergence angle with accommodation (IPD: 63 mm)

As can be seen from Figure 3.9, the reflex acts in a linear fashion even beyond the usual accommodation ability for the eye (+4 to +5 D). It is not a mere coincidence that the range of ciliary accommodation lies mostly inside the contraction range of extraocular muscles responsible for convergence. Extraocular muscles responsible for convergence start hitting their limits at close-up distances characteristic of severe myopia and the same is reflected in definitions of severe Myopia.

For an emmetropic eye with its far-point at infinity, the reflex acts for all observable distances. The same applies to a pseudo-emmetropic eye also with best possible refractive compensation. For an uncompensated Myopic eye with its far-point no longer at infinity, accommodation begins only when convergence 'reaches' distances closer than the far-point. For distances beyond the myopic far-point, eyes remain in a relaxed state. This can be termed as introduction of **convergence lag** for Myopia[43]. In both cases, it can be said that the reflex starts acting only when the observed distances reach closer than the far-point of the eye whether myopic/emmetropic.

The roughly cylindrical nature of converged image plane distortion with its axis normal to the line joining both eyes suggests its association with astigmatism. It predicts presence of baseline levels of astigmatism in population primarily involved with significant near-work. It remains to be investigated how this particular form of cylindrical image plane distortion actually affects ocular biometry. It is possible that fully compensating for this form of astigmatism can result in experienced discomfort and further progression of astigmatism. Astigmatism of this form is best left slightly uncompensated unless it starts to perceptibly affect vision.

It is possible that subsequent conflict and recalibration of the accommodation-convergence reflex plays a crucial role influencing reports of initial discomfort associated with major changes in prescriptions. The common observation of discomfort stabilizing in the span of few days suggests that the convergence reflex could also get recalibrated within this time-frame.

4. The Continuous Adaptive Theory

This section describes the theoretical framework of Continuous Adaptation Theory (CAT) characterizing adaptive changes responsible for physiologic Myopia and how they apply to explain observed phenomena.

4.1 Variable time-scale adaptive processes of the eye

A lot has been already described about accommodation in multiple texts. Accommodation happens to be a very short-term (almost instantaneous) response due to ciliary muscle action. The ciliary muscle is relaxed when accommodation is not needed for distance focusing. A high-quality video of accommodation in action[44] can be accessed here: https://youtu.be/1yIpyitm6eE

Axial changes take place on a relatively long-term time frame mostly due to changes involving the cornea (shape curvature and ACD) and the outer non-muscular scleral shape requiring time-scales of months and longer. Commonly encountered Myopia involves changes that are mostly axial in nature.

There are only two ways in which the observation range of the ILS can be shifted: accommodative shift or screen distance changes. The same is applicable to the Lumped Lens consideration for the human eye. We have already reviewed how retinal distancing occurs during Myopia as a component of AL elongation. It is expected that accommodative shift of some sort also happens during the course of Myopia. This accommodative-shift contributing towards the non-axial component myopia has to involve the ciliary body because only the ciliary muscle mechanically attaches to the eye lens. Myopic accommodative shift can thus be termed as state change in ciliary muscle translating into increased relaxed and accommodated power of the eye lens.

Hypothesis H1: We predict that the aforementioned accommodative shift acts as a medium-term (happening on the time-scale of few days to weeks) bridging process between short-term accommodation and long-term axial changes. H1 also implies the presence of a biological mechanism capable of responding to and compensating state shifts in the ciliary muscle with axial changes.

This hypothesis is strengthened from the continuous structures of iris, the ciliary body and the choroid (the layer between the retina and the sclera) comprising the uvea. Its connection with the observation of choroidal thickness exhibiting inverse association with AL changes should also be investigated[45].

4.2 Observation range changes due to Myopia

The eye experiences continuous changes to its observation range while undergoing refractive state changes. By comparing the observation range of a Myopic eye with an emmetropic eye and multiple real-world

observations, it can be said that myopia results in the eye gradually gaining additional close-range vision capability while *sacrificing* capability to observe distant objects as shown in Figure 4.1.

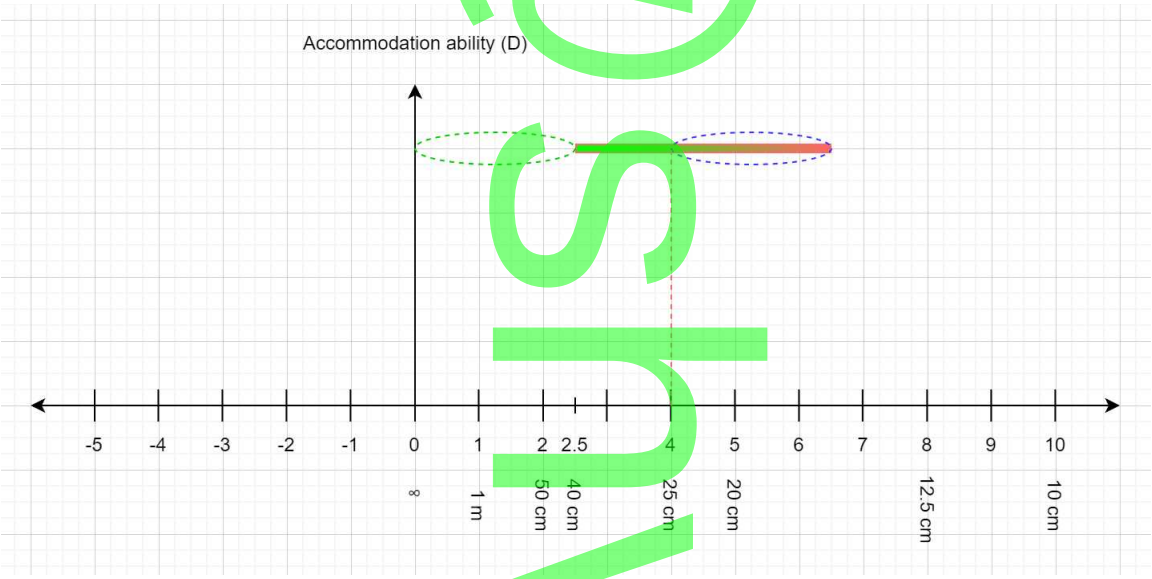

Figure 4.1: Compared to an emmetropic eye, the gained close-range observation capability by a Myopic eye (2.5 D) is shown with an blue oval while its sacrificed observation range is shown with a green oval.

The blue oval denotes gain between myopic near-point and emmetropic near-point while the green oval denotes loss between myopic far-point up to infinity (its emmetropic counterpart).

Hypothesis H2: We propose that the human eyes try to adapt continuously towards the **exposed** visual stimulus in order to alleviate accommodation load on ciliary muscle. It is assumed that eyes respond to stimulus from accommodative load, defocus, Form Deprivation or even pathological causes. The scope of CAT is limited to non-pathological factors only. For physiologic myopia, this basically translates into eyes responding to superposition of physiological stimulus.

The adaptive framework and the eyes as an organ dedicated towards visual perception means environmental factors optically affecting the eye such as object distance (accommodation), contrast, brightness/intensity (affecting the pupil) or their combination are to be termed as physiological factors. Factors not optical, for instance: hereditary/genetic, birth defects/congenital anomalies and environmental factors different from physiological ones mentioned above such as, toxins, trauma, and other unidentified factors internal to the eye are to be clubbed under pathological factors.

It might even be possible to derive the assumption (H2) from other elementary assumptions in the near-future but will not be attempted presently. Due to limits on extent of ciliary accommodation ability, an under-utilisation of observation range extreme (the ability to focus at distant objects in this case) permits an even stronger adaptive response. The aforementioned observation of Myopia initiation/progression resulting from the eye gaining additional close-range capability while sacrificing capability to observe distant objects can be explained as shift in refractive equilibrium resulting from close-range adaptive

stimulus driven by under-utilisation of distant vision capability. The simultaneous 'push-pull' analogy for shifting of observation range makes this clearer. For myopia, accommodative load can be said to 'pull' while under-utilisation of distant vision capability exerts 'push' on the observation range. Push or pull in isolation is insufficient towards shifting the observation range (causing axial changes).

This answers the first among many unexplained mysteries of Myopia: Why it is observed that a population does not experiences Myopia even with significant near-work incidence[46]? We now have an answer in the form of sufficient utilisation of distant vision keeping in check the adaptation from near-work. It also explains why long-duration near-work correlates[47] with Myopia but not sufficient towards causing Myopia with the simple implication being under-utilisation of distant vision capability (not the same as near-work) also contributing equally towards Myopia. This is also corroborated by multiple review studies[48].

Simply put, under-utilisation of distant vision capability is a more important consideration factor for Myopic adaptation resulting in the

4.3 Proposed mechanism behind Myopic adaptation

Section 4.1 describes differing time-scale of the adaptative processes in operation inside the eye. These short-term, medium term and long-term processes together are responsible for maintaining the refractive state in order to best adapt the eye to its environment. We have also described how the observation range of an emmetropic eye continuously changes during Myopia in section 4.2.

The onset of Myopia has been mainly predicted to result from the ciliary muscle 'tiring' out during long-duration near-work and developing some form of (accommodative) spasm in the ciliary (pseudomyopia and NITM[49]). This accommodative load on the ciliary should then translate into an adaptive shift towards Myopia. Doing near-work closer to the focusing limit of the ciliary muscle should theoretically bring faster exhaustion of the ciliary. Defocus near work should predictably result in even faster adaptive stimulus due to combination of defocus on top of extreme accommodative stress.

Without 'suitable' interventions, the ciliary muscle starts developing accommodative-shift (in the medium-term time-span of days to weeks) in order to alleviate this accommodative stress. It can be expected that a person with myopic ciliary shift should possess markedly better capability of doing near work for a longer time period along with suppression of near-work induced eye-stress. Myopic shift in the ciliary could also result in discouragement towards future utilisation of distance vision capability in the form of excessive tear formation, rapid uncontrolled blinking, increased sensitivity towards bright lighting and signalled discomfort (usually clubbed under HARE[50]) establishing a subtle feedback loop that should promote further progression of Myopia.

Hypothesis H1 stated earlier is essential towards explaining this missing link between how accommodative requirements from near-work might translate into eventual long-term axial changes from Myopia. Under-utilisation of distant vision capability in the presence of accommodative shift then allows long-term

adaptive processes to initiate Axial Changes in order to alleviate this myopic shift. The subsidence of external factors forcing myopic adaptation results in equilibrium being established again with the ciliary gradually returning to its previous state with the axial changes in place observed as stabilization of Myopia.

Because accommodative-shift is a novel construct towards understanding Myopia, physical details about shift in the ciliary are hard to come by. We speculate that the reported observation of ciliary body thickening[51, 52] in Myopic subjects can serve as an indicator for myopic ciliary shift.

We've already mentioned that a component of astigmatism should result from increasing angle between image planes resulting from closer observation distances in section 3.7. Another component of astigmatism is expected to result from shifts that the ciliary undergoes and then reverses in the process of developing accommodative shift factored with changes to retina and cornea from long-term AL changes. The 'recalibration' of the accommodation-convergence reflex as introduction of convergence lag mentioned earlier is also predicted to happen alongside myopia. These description are also consistent with the adaptive component of axial changes reinforcing its distinction from axial growth resulting from actual eye-growth.

An important consequence of the CAT is that it does away with the 'eye-growth' dichotomy and simultaneously explains many contradictions in the commonly accepted age-bound theory for Myopia progression and stabilization during mid-twenties. This hints that physiologic Myopia stabilization happening during mid-twenties must be linked to changes brought by environmental and lifestyle factors from attaining adulthood and conscious improvements in viewing habits including eye-strain awareness during long duration near-work. It also makes it trivial to explain why some subjects can still experience continued 'Myopia progression' throughout their adult lives.

4.4 Influence of light levels and the problem of deriving adaptive direction from blur

The pupil of the iris[53] evolved as an aperture control mechanism to regulate the amount of light entering the eye. [Pupil size[54] in adults usually varies from 2 to 4 mm in diameter in bright light to 4 to 8 mm in the dark.] We will refer to environmental lighting conditions in terms of how it relatively affects the pupil size in healthy eyes: pupil constricted (bright) and pupil dilated (dim/dark) lighting conditions.

The lumped lens consideration also naturally dictates that the dilated pupil's shallower Depth of Field should result in additional accommodation demand on the ciliary in dim lighting conditions to maintain same focus distance.

It is well known at this point that near-work under pupil dilated lighting conditions is associated with Myopia[55]. There is also an established body of research on Myopia being the 'default' behaviour of various species of animal eyes when subjected to form deprivation in dim/dark environments[56,57 and 58]. Form deprivation has also been successfully induced in primates (Rhesus Macaques[59]) as well which leads to a simple implication that the same should be applicable for human eyes as well.

Hypothesis H3: It is hypothesised that inadequate levels of light (pupil dilated lighting conditions) during near-work accelerates the adaptation towards Myopia by additionally invoking FDM pathways[60]. It is highly probable that the aforementioned genetic susceptibility for physiologic myopia has something to do with the strength of FDM pathway response.

It explores the possibility that efforts towards inducing hyperopic adaptation could be rendered ineffective by the dominant nature of the FDM pathway under pupil dilated lighting conditions. The activation of FDM pathway might explain why the same blur from myopic defocus results in Myopic adaptation in pupil dilated lighting instead of the expected adaptive emmetropization response according to animal models.

We've previously outlined how adaptive requirements from accommodative-stress and under-utilisation of distant vision capability together shift the refractive equilibrium towards Myopia. In this context, less than adequate lighting levels can be said to speed up the rate of Myopic adaptation by parallel activation of the FDM pathway. Lighting as a factor happens to be the last remaining puzzle piece for our understanding of physiological factors affecting Myopia[61]. This should also explain why highly myopic people experiencing Myopia progression can show sensitivity to bright lighting environment which normally do not affect emmetropic individuals. Lower light levels also result in reduction of information, implying increased susceptibility towards alignment errors and visual aberrations thereby making Myopic adaptation inherently 'inferior' in this regard.

4.5 Equivalences in observation range and shifting behaviour of an emmetropic eye and an eye compensated for Myopia

The similarities between an emmetropic eye and an eye compensated for Myopia can be summed up into two equivalences. These physical equivalences emerges from the optical equivalence between refraction (bending of light rays) introduced by refractive interventions (whether glasses, contacts or surgical).

1. Observational equivalence of far-point:
   The apparent far-point of a pseudo-emmetropic (best compensated myopic/hyperopic) eye is equivalent optically from actual far-point of an emmetropic eye. An emmetropic eye and a pseudo-emmetropic eye should be equivalent when it comes to their actual and apparent far-points respectively. This equivalence can not always be extended to the near-point due to prebyopia.

2. Behavioural Equivalence of shifting:
   A myopic shift in refractive equilibrium affects the observation range in the same manner whether it happens to an emmetropic eye (onset of Myopia) or previously myopic eye (Myopia progression). This equivalence naturally extends for hyperopic adaptive shift also.

For a myopic eye to experience reversal in Myopia – it goes without saying that the same must be physically accompanied by hyperopic shift in its far-point. It is trivial to demonstrate experimentally that a myopic eye apparently focusing at infinity with refractive compensation is actually focused physiologically at its myopic far-point. It basically implies that SVS closely emulates state of emmetropia for a myopic eye. In the earlier sections, we saw how the eye lacks specific mechanisms to deduce this direction of blur. The preliminary material supplied already demonstrates this for a camera. We have termed this as 'clamping' of actual far-point due to refraction intervention shown in Figure 4.2.

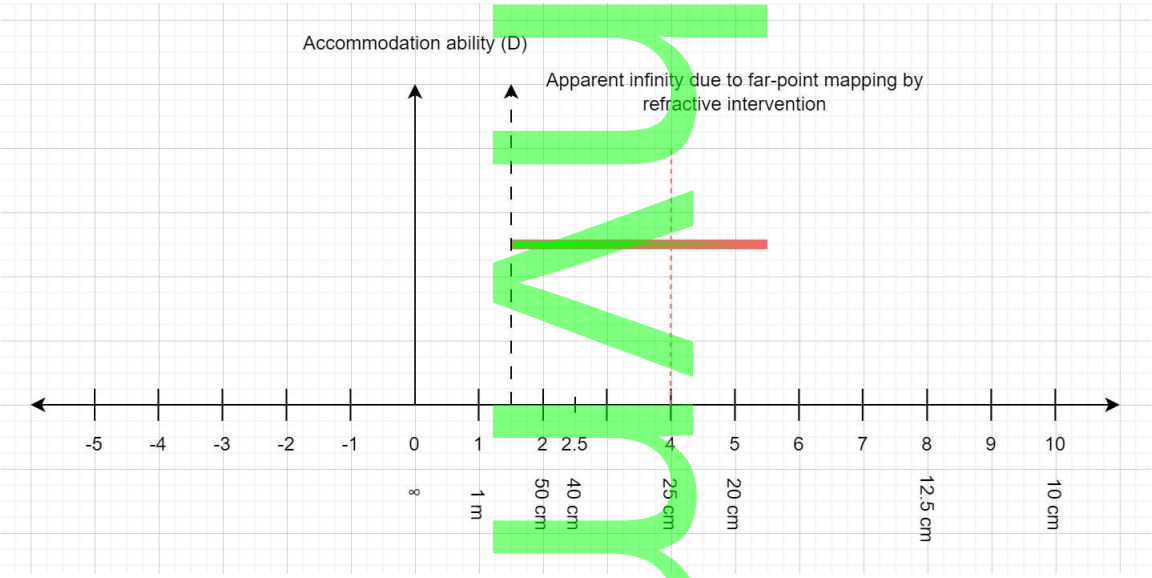

Figure 4.2: Clamped far-point of a Myopic eye (1.5 D) by refractive compensation

It can even be stated the other way around: expecting Myopia reversal for a pseudo-emmetropic eye whose far-point is clamped by refractive intervention is equivalent to expecting the actual far-point of an emmetropic eye to shift farther away (become hyperopic) from its clamped position at infinity. Nothing happens in both cases simply because relaxation of the ciliary muscle does not constitutes adaptive requirement combined with lack of adaptive scope with far-point at infinity. It should also offer an explanation so as to why truly physiologic hyperopic progression unlike Myopia progression is a rare observation[62]. For adaptive equilibrium, this has a simple physical implication that myopic defocus should be the physiologic cause for inducing hyperopic adaptation in the absence of accommodative stress. Myopic defocus happens to be a necessary but not sufficient[63] requirement for Myopia reversal.

The second equivalence predicts that factors resulting in onset of physiologic Myopic shift in an emmetropic eye should be responsible for Myopia progression in a compensated Myopic eye as well. Myopia progression is equivalent to 'onset of Myopia' in an eye already compensated for Myopia. It has a parallel implication that an eye with stabilized Myopia has more in common with an emmetropic eye compared to an eye experiencing active Myopia progression. This equivalence alone outlines the urgent

need to revisit and fully isolate behaviour shown by stabilized Myopia from progressive Myopia in pertinent literature and explains why efforts to gather insights about Myopia from studies on stabilized Myopic population should fail due to emulation of emmetropia by SVS.

The predictions from these two equivalences are given below (Table 2):

Table 2: Equivalences between a pseudo-emmetropic eye and an emmetropic eye

Myopic eye wearing best possible refractive compensation (pseudo-emmetropic eye)	**Emmetropic eye**
Failure to observe Myopia reversal with best possible refractive compensation even after distance work	No observed hyperopia (shifting of far-point beyond infinity) even after (in-focus) distance work
Myopic shift observed as Myopia progression	Myopic shift observed as Onset of Myopia
Some population reports stabilized myopia even with significant near-work habits.	Some population reports no myopia even with significant near-work habits.
Apparent Far-point is at infinity	*Actual* far-point is at infinity

The refractive state of a physiological myopic/hyperopic eye is as '*valid*' as an emmetropic eye from the standpoint of CAT. These equivalences and the lack of specific anomalies inside the eye should serve to distinguish physiological Myopia from pathological Myopia. Physiological myopia is not some disorder or disease. This is necessary from the self-consistency of our theoretical framework and the resulting behavioural equivalences.

The hereditary nature of myopia is frequently associated with myopic individuals having more chances of a myopic parent. It still remains unclear how much of a role genetics actually play towards progression of physiologic Myopia after accounting for the possibility that myopic children can share most of the same physiological environment with their parents. It must also be noted that most studies on parental Myopia additionally don't strictly differentiate between progressive and stabilized myopia[64, 65, 66]. In most of these reports, we expect definitive trends to emerge when progressive Myopia is properly isolated from stabilized

Myopia. These equivalences also outline the urgent need to fully isolate physiological variables from pathological ones in studies like the ones investigating parental origins of Myopia.

5. Discussions

5.1 CAT

5.1.1 Strength and Limitations

The strength of our theory lies in proposing an aetiology, onset/progression and even the 'to date' 'irreversibility' of Myopia in a manner consistent with existing body of observations. The resulting behavioural and observation range equivalences explain why studies on stabilized Myopes have so far been an exercise in futility. CAT is also the only theory describing myopia so far consistent with the theory of evolution of eyes as a visual organ due to its framework treating axial changes as superposition of growth and environmental adaptive signals.

Because of the way CAT re-defines pathological Myopia from physiological Myopia in terms of reversibility, the theory predictably breaks down for pathological Myopia due to unpredictable alterations to the underlying adaptive mechanism responsible for maintaining adaptive equilibrium of the eye.

5.2 Summing up the Continuous Adaptive Theory (CAT) for physiological Myopia

In the framework of CAT, physiological Myopia is viewed as a 'valid' refractive state of the eye brought on by temporal integration of environmental/lifestyle (physiological) factors predicted as part of the eye's diurnal rhythm. A tabulation of the predicted differences between myopic adaptation vs hyperopic adaptation is being provided below:

Table 3: Physiological factors required to induce adaptation

Function	Onset/Progression of Myopia	Hyperopic adaptation (Duals)
Visual stimulus 'pulling' the adaptive equilibrium	Near work induced accommodative stress	Myopic defocus under pupil constricted lighting conditions so as to overcome FDM pathway
Visual stimulus 'pushing' the adaptive equilibrium	under-utilisation of distance vision capability	Near-work management and periodic breaks as signalled by the eye to alleviate accommodative stress.

Accelerator Lighting Conditions	Pupil dilated utilising FDM pathways	Pupil constricted so as to overcome the FDM pathways
Accommodative shift in the ciliary/feedback loop causing shift in Observation Range (far-point and near-point)	Myopic	Hyperopic
Secondary refractive errors	Results in aberrations and progression of refractive errors like astigmatism	Corrects aberrations and refractive errors
Field of View	Shrinks	Expands
Time Interval	Existing Myopia progression rate	faster than Myopia progression due to extra light information

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5.3 Predicting time taken for long-term axial changes

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There are two approaches for predicting the time taken for long term axial changes. Both approaches should converge at similar estimates.

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The first approach emerges from the consideration that both myopic and hyperopic adaptation are the outcome of same adaptive process. This means myopic and hyperopic adaptations should bear rough similarity on the order of time-scales on which they take place. By our prediction, time required for Hyperopic adaptation can be estimated from the data on Myopia progression[67]. The observed rate of hyperopic adaptation should only be slightly faster than the progression rate of Myopia because it requires pupil constricted lighting conditions with more available information.

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The second approach involves figuring out the relation between axial length of the eye with the retinal distance (RD) from the optical centre of the eye. The second approach is described in detail below.

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In the emmetropic lumped lens consideration, the RD should also be equal to the focal length of the lumped lens when the eye is focused at infinity. The lens formula then gives the required shift in RD for focusing an object at the Myopic eye's far-point. To determine changes to retinal distance from AL measurements, we have assumed RD to be proportional to AL denoting the ratio RD/AL as β.

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$$\text{Lens relation}: \quad \frac{1}{s} - \frac{1}{u} = \frac{1}{f}$$

$$\text{Here, elongated (myopic) RD serves as screen distance } (s) \text{ and emmetropic eRD serves as focal length } (f)$$

$$\frac{1}{RD} + \frac{1}{-u} = \frac{1}{f}$$

$$\text{emmetropic RD } (f) = \frac{-u \times RD}{-u + RD}$$

$$\text{if we assume } RD / AL \text{ as } \beta \text{ then}$$

$$\text{Elongation in Axial Length } \Delta AL \text{ due to Myopia} = AL - \frac{f}{\beta} = \frac{\beta \times (AL^2)}{-u + \beta \times AL}$$

$$\text{For instance, a myopic eye wearing prescription of } -4\ D \text{ corresponding to a far-point of roughly } 25\ cm \text{ will give } u \text{ as } -25\ cm \text{ for the formula.}$$

If AL and degree of Myopia for a non-severe Myopic eye is known, this formula gives change in AL that must be necessary for myopia progression/emmetropization provided β remains constant. Measuring the long-term rate of AL changes then gives a rough estimate of the time period required for axial changes.

5.4 Changes to Field of View due to Axial Changes

FoV depends on the screen distance which in the case of Lumped Lens consideration is taken as the distance of the central part of the retina (RD) from the lumped lens. Axial elongation due to Myopia results in physical distancing of the posterior part of retina while the central region responsible for vision remains mostly unchanged at least for cases of non-severe Myopia.

The resulting FoV reduction can be estimated from *emmetropic AL ÷ myopic AL* assuming the proportionality of AL with Retinal Distance (RD). This works in a manner similar to how focal length changes affect FoV of the image in a camera. For instance, emmetropic AL of 23 mm and Myopic AL of 25 mm result in myopic FoV being roughly 0.92× (times) that of the emmetropic FoV.

Any mainstream investigation into Field-of-View characterization regarding Myopia is virtually non-existent. The closest study that considers FoV changes in highly myopic subject is the one done by {Yanming Chen; Ji Liu; Yining Shi} on visual field features venturiig in the pathological domain[68]. It is reasonable on the basis of optics alone that effects of FoV reduction start resembling 'tunnel vision effect' with highly severe Myopia. This also implies that people managing significant Myopia reversal should experience widening of their FoV.

6. Conclusion

The term ‘Continous’ in CAT basically refers to Continuos integration of stimulus from both near and distance work by eyes. This continuous integration of visual stimulus in accordance with the proper physiologic definition of actual distance work and refractive clamping with the prediction of FDM pathway activation during pupil dilated lighting conditions should be sufficient to explain all the peculiar behaviour exhibited by physiologic Myopia.

Not only outdoor sunlight exposure features naturally in the CAT, it emerges as the crucial factor behind reversing Myopia as well.

It predicts that stabilizing/reversing Myopia might require imposing Myopic defocus in pupil constricted lighting so as to overcome FDM pathways activation in dim/dark lighting conditions. The theory naturally hints at pupil constricted Light Therapy (PCLT) as the only possible and physically viable long-term protocol for Myopia. We expect the sheer predictive prowess of CAT to continue with rapid light therapy optimisations coupled with near-work interventions towards effective Myopia management.

The framework of CAT put forward in this article might very well be the first such explanation put forward characterizing physiological Myopia in a way that manages to consistently and convincingly explain nearly all clinically observed aspects of physiological Myopia including but not limited to its onset, progression and even its apparent irreversibility so far in clinical trials so far. Building on the suspected multi-factorial aetiology of physiological Myopia, the theory with its two equivalences successfully explains failing to understand Myopia on the inability to properly isolate the underlying physiological factors.

An eye doing long-duration near-work but also sufficiently utilising distance vision capability should only be signalling eye-strain from long-duration near-work without becoming Myopic. It also hints that emphasis towards proper utilisation of distance vision capability forms an essential requirement for management of Myopia.

6.1 Key takeaways

We expect that long-term commitment along with multiple habitual and lifestyle interventions are required from candidates willing to reverse physiological myopic state near emmetropic levels. From modifying their lifestyle to reducing the impact of near-work stimulus to a minimum to not letting go of any chance of viewing distant objects during breaks – it is theoretically very simple but practically very difficult.

The author recognise the tediously slow and time intensive nature (taking months and years) for the Myopia reversal process outlined in this article. Still, we are firmly convinced that the results outlined should be of enormous utility towards maintaining post-refractive surgery outcomes and preventing Myopia progression in subjects.

At the same time we want to stress that our simple lens model can not differentiate between peripheral and central components of vision or predict myriad of other factors that can play a role towards towards promoting Myopia in the human eye.

Although this article provides a method for satisfying necessary conditions for observing stabilization and/or reversal of Myopia, the article should not be taken as against wearing glasses/contacts or refractive interventions in general. The goal of this article is to reduce subject's dependence on glasses and promote safe and minimally invasive management of Myopia.

6.2 Further research

Proposing precise set of conditions that must be satisified for reversing physiological Myopia presents a massive unexplored opportunity in front of the research community. We attempt to highlight a select few of them.

1. Optimal myopic defocus under pupil constricted lighting conditions that promotes fastest rate of hyperopic adaptation.
2. Whether severity criteria mentioned in this article can actually be extended for Hyperopia and investigating implementation of Myopic adaptation for Hyperopic subjects.
3. Further optimisation of lighting environment and distant calibration target requirement for ADV.
4. Experimental studies trying to observe form-deprivation hyperopia under pupil constricted lighting conditions.
5. Investigation into the role played by mechanistic interaction of the iris with ciliary.
6. Corporate, ethical, and lawmaker's role towards prevention of physiologic Myopia.

7. Appendix: Myopia Management Implementation acc. to CAT

Current myopia management[69] interventions in practice including but not limited to atropine and Ortho-K carry reports of rebound Myopia upon cessation – a clear indication that the underlying cause of Myopia is still unaffected.

The naturally obvious end-goal for any theory capable of consistently and convincingly explaining various counter-intuitive observations about Myopia should be its ability to arrive at a physically viable method towards reversal of Myopia. The following sections outline the implementation of a Myopia management regime on the basis of CAT.

Most if not all of the implementation details regarding Myopia management in this section are the product of limited scale experimentation spanning more than a year including the entire time spent documenting and writing this article. It would have been near-impossible otherwise to gather important insights from empirical deliberations alone.

7.1 Predicted requirements for Reversing Myopia

If Myopia is indeed a valid refractive state of the eye as a consequence of adaptation, shift in adaptive equilibrium needs to be induced in the opposite direction for reversing Myopia. To achieve this shift, we need to determine 'duals' or principles opposite to the ones responsible for Myopic shift.

To summarise our findings, section 4.2 covers why accommodative stress from long-duration near-work coupled with under-utilisation of distant vision capability should result in Myopic shift. Section 4.4 predicts why low-light should accelerate Myopia by utilising FDM pathways. Section 4.5 points out how observing no reversal of Myopia even while viewing distant objects with best possible refractive compensation is not sufficient to decide irreversibility of Myopia.

It follows that requirements towards inducing hyperopic shift come out to be Myopic defocus in pupil constricted lighting environment and reduction of existing accommodative stress from near work causing myopia in the first place. This emerges from the idea that a pseudo-emmetropic eye and an eye with very low levels of Myopia differ only in their ability to observe distant objects and the observation that relaxation of ciliary muscle does not results in any accommodative stress.

These summarised findings and their corresponding duals are given below:

Table 4: Duals of physiological factors required for myopic adaptation

Observed requirements that result in Myopic adaptation	**'Duals' for inducing Hyperopic adaptation**
Accommodative stress from near-work resulting in adaptive requirement.	Saturation levels of Myopic defocus (exposure beyond far-point)
Under-utilisation of the distance vision capability	Near-work management to reduce accommodative stress causing Myopic adaptation in the first place.
Myopic adaptation is accelerated under pupil dilated lighting conditions due to FDM pathway	Hyperopic adaptation requires pupil constricted lighting conditions to overcome FDM pathway

These duals form the breakthrough physical requirements for inducing hyperopic adaptation: exposure towards myopic defocus under pupil constricted lighting conditions such as those that occur on a clear sunny day with management of near work to reduce accommodative stress causing myopic adaptation in the first place. These 'duals' expand our theoretical framework that is now able to explain why earlier attempts[70] failed to observe reduction in Myopia utilising reduced prescription.

The distances beyond far-point (myopic defocus) that need to be exposed to under pupil constricted lighting conditions lie inside the green oval for a myopic eye shown in Figure 4.1. Throughout this article, we will refer to this as ADV (short for Actual Distance Viewing). Because hyperopic adaptation requires pupil constricted lighting conditions, it should also be slightly faster (Superior) due to the availability of more information from bright lighting conditions.

The framework of CAT naturally places no restriction on the eyes trying to adapt continuously towards various visual stimulus that can be encountered during the day – which implies that hyperopic adaptation from distance vision should get somewhat 'compensated' by accommodative stress due to 'ordinary work' during the rest of the day. The temporal nature of this integration hints at less than ideal rate of recovery.

The adaptive nature of the process also implies that the requirements for Myopia reversal should be far stricter than that required for Myopia stabilization. This is the 'dual' for observing a subset of population with significant near-work habits not becoming Myopic. It implies that a subset of population should experience stabilization of Myopia (and not reversal).

7.2 Saturation time for hyperopic stimulus and 'relative' lack of near work

The question of what should be the recommended time interval of exposure to myopic defocus emerges from the observation of peaking of maximum axial length reduction (referred from now on as saturation) around 50 minutes as described in [Delshad, S., Collins, M.J., Read, S.A. et al. The time course of the onset and recovery of axial length changes in response to imposed defocus[71]]

"The first statistically significant reduction in axial length occurred after 40 minutes of exposure to myopic defocus, with a mean reduction of −8 ± 9 μm ($p = 0.017$). This change peaked shortly after, reaching a maximum axial length reduction of −10 ± 8 μm at 50 minutes ($p = 0.001$)."

We recognise that the article in question is not a long-term study on axial changes. But even then, the observation of a saturation time and its duration being somewhere around the ballpark of an hour should hold. The presence of a saturation time in itself is an obvious indicator that the eye requires periodic breaks. The time to achieve saturation naturally increases if distributed in small chunks instead of one go. A person wanting to ensure maximum hyperopic adaptation towards reversing myopia should aim for observation time ≥ saturation time. For preliminary trials, the initial duration of observation time to maximize saturation in the absence of further experimental data can be taken close to an hour (more than 50 min). Subjects should be encouraged to determine long-term saturation time on their own from the signals given by their eyes.

Promoting hyperopic adaptation for reversal of Myopia demands under-utilisation of near-focusing capability of the ciliary mentioned in previous section to reduce accommodative stress due to near-work as much as possible. This constitutes the 'extra requirement' part required to reverse Myopia after stabilising

it. This load reduction of near-work on the ciliary muscle can be achieved in two ways – reducing the time or increasing the distance at which near-work is done.

The former can be achieved by reducing the duration of near-work and is the obvious, most effective but largely impractical approach. This also cements the importance of taking regular breaks as signalled/indicated by the eye and looking at far-away objects during breaks. The already established recommendation[72] in the form of 20-20-20 rule is handy in this regard. The second approach refers to mitigations that reduce load on the ciliary due to near-work. This can be done by physically increasing the distance at which near-work is done or by utilising refractive interventions.

7.3 Implementation of ADV

Having elaborated the saturation and near-work management aspect for Myopia reversal in the previous section, we will now describe in detail the physical requirements for ADV. ADV is a targeted method for High Environmental Illuminance viewing utilizing high-intensity outdoor sunlight for imposing calibration myopic defocus conducive towards hyperopic adaptation.

7.3.1 Duration and regime requirements

Due to the lack of any previous experimental data regarding the time interval between ADV sessions, we suggest that subjects should attempt ADV sessions until saturation described in section 7.2 consistently on a daily basis. Effective Myopia management requires augmenting daily ADV sessions with refractive intervention guidelines detailed in the next section for rest of the day.

7.3.2 Prescriptive compensation for Myopic defocus requirements

The lack of any previous large-scale experimental trial also means that the ideal value of myopic defocus for ADV is in dire need for determination. It is possible that a ‘saturation’ defocus could exist resulting in the eyes hitting an adaptive rate limit once defocus is greater than a certain threshold for ADV. This considers the possibility of pupil constricted lighting overpowers FDM pathways for emmetropization.

This leads us to suggest that subjects with non-severe Myopia should attempt ADV with no prescriptive compensation at all. This leads us to recommend doing ADV without wearing any glasses or contacts for subjects with non-severe Myopia resulting in a greatly simplified implementation because practising ADV without refractive interventions directly stimulates emmetropy (ensuring exposure to maximum possible stimulus towards emmetropization). Subjects experiencing difficulties should gradually taper their prescriptive compensation utilising their former prescriptions to view distant targets during the initial days of their ADV sessions.

7.3.3 Exposure requirements under pupil constricted lighting conditions

ADV demands daily exposing entire visual field to distant ‘targets objects’ with good contrast involving blur from myopic defocus (observation beyond far-point by reduction or elimination of prescription power)

in bright daylight until saturation. The idea behind exposing entire visual field to distant 'target objects' having good contrast under very bright lighting emerges from the concept of conveying maximum light information to the Retinal Ganglion Cells (RGCs) of the retina. When it comes to pupil constricted lighting conditions, sunlight on a clear sunny day is the benchmark. One must exclude viewing the Sun directly because the Sun is a point object in the visual field even if one ignores the harms[73] directly viewing the Sun has on the eyes. It is imperative that the subjects keep the Sun behind them at all times to minimize risk of exposure to harmful UV radiation.

Regarding observation of distant sunlit objects, subjects should place no demands or special emphasis in the way distant objects are being observed. They should strive to observe naturally in a relaxed manner without squinting, forcing or stressing their eyes in any manner. Subjects can attempt slow walking, standing, or sitting still while performing ADV according to convenience. This should not differ from the way an emmetropic or a refractively compensated (pseudo-emmetropic) person normally observes objects at a distance. The best analogy we can give is similar to trying to read a distant signboard or resolving fine details presented by a distant structure. We expect contrasting patterns in both vertical and horizontal meridians to be useful and implore researches to optimise target object requirements and explore such synthetic target patterns for ADV.

Figure 7.1 Image demonstrating close to ideal calibration target for ADV

An image showing ideal ADV environment satisfying our criteria is shown in Figure 7.1. The Sun in clear sky is behind the observer in this image and most of the objects are more than 20 m away with the farthest being more than 100 m away resulting in a good calibration target. The difference between an object at 4 m compared to an object located at 20 m is 0.2 D, a distinction important for fairly low myopia.

Because the lighting requirement is for the eyes only, being under direct sunlight or doing it from a shaded place doesn't matter. The outcomes are independent from whether the body is exposed to Sunlight or not. People residing in hot climatic conditions should attempt ADV from a cool and shaded place to combat the sweltering heat outside. Swamp (desert) coolers are effective in this regard.

It remains to be seen how the strategic requirements posed by ADV pans out for myopic subjects living in inclement climatic conditions receiving little sunlight during the year. Artificial lighting that recreate the bright daylight environment might prove useful according to recent suggestions. [NBK470669: "In countries where the intensity of outdoor light is generally lower, because of air pollution or short duration of natural daylight – such as Canada or Scandinavia in the winter, or Beijing year-around – sunlight therapy could be supplemented in the form of SAD lights (approved and used for Seasonal Affective Disorder)"]

7.4 Refractive intervention guidelines for Myopia management.

We have previously established that observing objects closer than the myopic far-point while wearing prescription results in more physical contraction of the ciliary muscles compared to viewing directly without wearing anything. This should also mean that glasses for non-severe Myopia should be worn on a need basis – only for vision requirements farther than the myopic near-point. As myopia reversal progresses, this far-point should get closer to ∞ reducing dependence.

In accordance with our duals established in section 7.5, refractive interventions can ensure that the subject's quality of life remains relatively unaffected from the process of managing Myopia while simultaneously ensuring that near-work incidence on ciliary muscle is minimized.

For the purpose of Myopia management as outlined in this section, conventional glasses emerge as the best piece of equipment because they are easy to wear and remove in accordance with varying refractive demands during the day, cheaper, reliable, safer for the eyes, and allow easier to manage powers in the long run compared to contacts.

Within few weeks of daily ADV sessions, it should be expected for an eye to gradually start experiencing difficulties both while wearing their normal prescription during the day (over-prescription resulting from reduction in Myopia) and in the form of subtle aversion from near-work. Such developments should result from the gradual disruption of myopic feedback loop in the ensuing weeks of daily ADV sessions. The natural course of action suggested is that refractive interventions should be implemented in the order in which they become apparent during Myopia management. Subjects should change things one at a time and that too only when indicated by the eye.

The disruption of myopic feedback loop from ADV sessions means that refractive demands of the eye become somewhat complicated and slightly reduced prescription should be needed than the regular prescription (for near-work beyond the myopic far-point) so as to lessen the feeling of eye-strain during

near-work. As such situations arise, the signalled comfort of the eyes towards worn prescription should be prioritized. The eyes should signal immediate discomfort for both over and too much under-correction and the same should be avoided. As long as the worn prescription is kept within this narrow range of comfort according to the signals given by the eyes, we expect ADV to continue resulting in effective hyperopic adaptation for the eyes.

This concept of refractive intervention is an important aspect of myopia management and requires strictly individual implementation because of the varying near-work requirements of subjects, their present refractive state and preferences/tolerance to defocus. The adaptive nature of the eye makes it obvious that the refractive compensation requirements from the standpoint of reducing accommodative load for a system as dynamic as the human eye can not be accomplished using one refraction value[74]. For the ease of understanding, we have tabulated these guidelines in the table below.

Refractive compensation guidelines for Myopic candidates with non-severe Myopia.

Table 5: Tentative Refractive compensation guidelines for near-work management

Working distance	Daylight (outdoors on a sunny day)	Evening/Night
For ADV until saturation	Without wearing any prescription	NA
Long distance work	Reduced Prescription with sunglasses according to the need	Regular Prescription
Near-work at a distance just beyond the myopic far-point	Reduced Prescription	Reduced Prescription
Near-work done at a distance closer than the myopic far-point	Non-severe Myopics should be able to observe objects closer than their far-point comfortably without needing prescription	
Life-critical task such as driving, operating heavy industrial machinery and other dangerous work	Best Possible Refractive compensation ensuring full visual capability to prevent incidents and to comply with applicable local laws	

These guidelines result in a slightly more complicated answer for the most commonly asked question in myopia – “When should I wear glasses?”

These guidelines are only meant as a starting point for the trial implementation for our Myopia management method. It is expected that the eye's refractive need will change during the course of the trial as Myopia reversal progresses. The evaluation criteria always remains the fastest and safest reversal of Myopia and we fully expect some of these tentative guidelines to fail or get superseded by further understanding gained from future trials.

7.4.1 Blue Light Blocking lenses

Coming to the actual lenses used, we suggest 'normal' clear lenses without any special coating such as blue light blocking filters because of the reasons given below:

1. Increased costs of lenses that will need replacment eventually in the near future as Myopia reversal progresses.
2. Provides no clinically substantiated shielding against Myopia as evident from multiple research attempts into investigating their benefits[75].
3. Nearly every recent computing device with a display already has an inbuilt blue-light reduction/night-comfort feature. [Windows Night Light[76] and Apple Night Shift[77]]. The intended physical purpose of blue-light glasses is already achieved at the source level for modern computing devices.
4. We suspect that blue-light glasses can suppress the ability of the eye to signal eye-stress due to extended near-work (DES) which deters from our guidelines of regular breaks as essential in order to reduce near-work load on the ciliary muscle.
5. We also suspect that Blue-light glasses are less effective as refractive compensation than regular clear-classes due to decreased availability of light in dark conditions that can result in delayed improvement towards Myopia. For candidates wanting fast management of their Myopia, this is not something they might want.
6. Blue light lenses can not replace the utility and eye-protection offered by outdoor sunglasses under direct sunlight.

7.5 The proposed mechanism for Hyperopic adaptation towards emmetropization

Section 4.3 already covers our attempts at characterizing how myopic adaptation takes place.

Onset/progression of physiological Myopia should result from accommodative stress due to near-work in pupil-dilated lighting conditions combined with significant under-utilisation of distance vision capability. This should then result in development of myopic shift in the ciliary muscle. Long term axial elongation is

proposed to result from the eye's eventual attempts towards alleviating this accommodative shift in the ciliary muscle.

This section attempts to describe the mechanism of Myopia reversal based on our observations upon year long implementation of the method described in the preceding sections. For lower degree of Myopia, subject's experience should reduce to only a subset of what has been described in this section.

The initial days of ADV sessions should be relatively uneventful with slow spontaneous onset of brief moments of visual clarity (referred to as clear flashes from now on) vanishing immediately after blinking with long reset time (minutes to hours) between each consecutive clear flash. This suggests that just like Myopia, the ciliary is the first to respond to hyperopic adaptation.

Within weeks of regular ADV sessions, besides a perceptible increase in clarity, clear flashes become more frequent and longer in duration while gaining the capability to 'survive' between blinks. Subjects should be advised not to prolong blinking in order to sustain clear flashes.

This development alone with the occurrence of significant clear flashes coincident with outside exposure to daylight should be enough to conclusively debunk the prevailing hypothesis that these clear flashes result from formation of tear film[78] on the cornea or due to the diurnal variation in vision[79]. ADV can also result in transient intervals of blurrier than usual vision on some days signifying that the structures inside the eye are being repaired/rebuilt.

It can be naturally deduced from the anatomy of the eye that clear flashes due to their instantaneous nature must arise due to increase in focal length of the eye lens. In the case of Myopia, adaptive requirements for closer distances result from accommodative stress on the ciliary muscle. However, the opposite of this is not correct because the ciliary muscle can not get 'tired or exhausted' in its relaxed state – hence the overshoot in 'negative accommodation[80]'. It also explains the commonly reported instances of 'ciliary spasms' in myopic people observing spontaneous brief flashes of clear vision under pupil constricted viewing conditions when not wearing their glasses. We will prefer to use the more descriptive term 'clear flashes' instead.

Within weeks, ADV sessions should force the establishment of a feedback loop with behaviour opposite to the previously described myopic feedback loop – resulting in perceived eye-strain/aversion towards near-work. Experiencing discouragement from near work serves as a reliable indicator that hyperopic adaptation is taking place. For a person experiencing active myopia progression, this requires disruption of existing myopic shift and establishment of hyperopic ciliary shift.

After regular ADV sessions spanning over longer time-frames (months), subjects should start experiencing a perceptible level of visual clarity brought automatically when venturing outside on a sunny day – a telltale marker of hyperopic ciliary shift. Long-term axial changes should soon follow to compensate this hyperopic response of the ciliary as long as under-utilisation of near-work is maintained. Measurable

reduction in retinal distance starts happening once this stage is reached. This stage should continue translating into improvements to baseline vision with time as long as emmetropization is not reached.

Successful observation of hyperopic ciliary shift would cement the bi-directional bridging nature of the accommodative shift as the precursor of long-term axial changes for both myopic and hyperopic adaptations (Hypothesis H1). This alone should be sufficient to confirm that the predicted[81] *active emmetropization mechanism* is not different from the process described in this article inducing Myopia or Hyperopia in accordance with the imposed visual stimulus. The process of Emmetropization naturally involves ocular re-calibration for distance vision and translates into eventual changes to the eyeball shape in the long-term (months). Some subjects can also report feeling changes to the extraocular eyeball muscles both during and after ADV sessions.

It is equally important to mention that most of the aforementioned clarity gains during ADV sessions vanishes swiftly under pupil dilated lighting conditions. This behaviour from the eyes could be looked as a source of frustration upon witnessing the clarity gained during the day vanishing in the night. This observation is so important from the standpoint of light's role in influencing adaptive behaviour of the eye that we predict that it should result from the mechanistic interaction of the iris restricting 'headroom' for negative accommodation (responsible for clear flashes), explaining the loss of clarity under pupil dilated lighting conditions. This can be termed as restrictive effect of the dilated pupil for ciliary relaxation under dim lighting conditions. This mechanistic interaction of the iris with the ciliary should be also associated with why observation of FDM as the default behaviour of the eye[82].

7.5.1 The informal Law of physiological Myopia

Given time, a person with physiological Myopia should be able to focus at infinity without refractive interventions at par or better than their current vision levels with best possible refractive compensation.

This law encapsulates our bold prediction from CAT signifying that best possible visual acuity should be improved or maintained due to the error correcting, superior nature of hyperopic adaptation.

7.6 Precautions, Safety and candidate selection requirements

7.6.1 Candidate Selection Criteria

Managing Myopia according to our method requires active integration into daily lifestyle and trial candidates should be willing to devote the (an hour or two it takes) of their daily time towards achieving saturation requirements for ADV besides implementing near-work management aspects.

The novel nature of the idea and the lack of any widespread experimental trial means heavy emphasis on precautions as a first line of safety. We insist on limiting the first run of trials to **Non-severe** young adults only having good ocular health history without adverse event, Presbyopia, and screened for any presence of musculoskeletal disorders. Further information from the outcome of the preliminary trials on non-severe

myopics will be used towards narrowing down additional requirements for candidates with severe physiological myopia and their eventual transition into non-severe Myopia if possible.

It is recommended that the candidates exercise caution during the initial transition period of the trial, starting with 'less brighter' objects first and take regular breaks until acclimatisation is completed within the first week. We expect the majority of candidates to experience excessive-tearing and strong aversion signals from the eyes in the form of eye-strain and minor headache during the beginning of the trial due to the aforementioned sensitivity of a myopic eye towards bright light. It is also recommended that observation duration too should be gradually ramped up towards saturation over the course of multiple days under constant monitoring so as to prevent the possibility of any adverse complications from over exertion.

7.6.2 Safety of ADV:

The only part of our method resulting in any significant stress on the eye results from ADV. Regular ADV sessions towards inducing hyperopic adaptation should have a risk profile similar to the risks associated with the onset/progression of Myopia because the same adaptive process responsible for Myopia should result in hyperopic adaptation towards emmetropization also.

We would also like to point out positive sentiments of experts about outdoor therapies like ours that involve High Environmental Illuminance trials: ["Outdoor-light therapy may offer the ideal treatment for myopia. Not only does encouraging children to play outside combat other major health concerns – such as childhood obesity, juvenile diabetes, and depression – but also, light therapy presents little to no serious health concerns or side-effects compared to those of other available myopia-treatments."1]

There are multiple studies demonstrating safety profile and tolerance of eyes towards light therapies with light levels far higher than that of indirect sunlight for Myopia management[83]. No serious complications have been reported in RLRL's (Repeated Low-Level Red Light) studies with periods from 6 months up to 2 years. There is even a 12-month study reporting improved accommodative function after RLRL treatment[84].

7.6.3 Important Warning for personal safety and compliance with applicable laws/regulations:

The candidates should be strictly made aware of the potential life-threatening dangers of doing critically important work involving life at risk without wearing best possible refractive correction. Put simply, safety of personal and other's lives while driving during low-light conditions such as night-time or working in dangerous conditions including but not limited to operating construction, industrial, or heavy-machinery/equipments should always be prioritized and best possible refractive compensation must always be worn under such conditions.

Candidates are expected to use fair judgement and not jeopardise their own and other's lives for Myopia management. Because ADV involves observing blur from Myopic defocus and significant reduction in

visual acuity is involved, it is only imperative that personal safety must be prioritised and ADV should always be attempted in a safe environment.

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