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Optically induced hyperopic defocus: axial length changes in emmetropes from mobile screen exposure

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HIGHLIGHTS

This study highlights the significance of different light wavelengths interventions with and without optical defocus on axial length, sub-foveal choroidal thickness and ocular biometrics and its role in myopia management.

ABSTRACT

Background: The rapid increase in smartphone screens led to eye problems. The study measured transient axial length variations during mobile screen exposure before and after hyperopic defocus.
Methods: A quasi-experimental study on students utilizing non-probability sampling was conducted at Al-Shifa Trust Eye Hospital. LogMAR measured visual acuity, and retinoscopy measured refractive status of right eye only. Samsung Galaxy A7 provided full-blue screen exposure. -3.00 DS lens in trial frame caused hyperopic defocus. IOL Master Zeiss 700 measured axial length and ocular biometrics. The online web program Data tab was entered, and data was analyzed.
Results: A total of 30 subjects, which includes 6 (20%) males and 24 (80%) females, with a mean age of 20.67 (±0.96) years. Mean visual acuity and Spherical Equivalent Refraction were 0.00 (±0.00) and 0.10 (±0.01). Comparing the median interquartile range (Median-IQR) pre-defocus axial length (PDAXL) and after defocus axial length (ADAXL), pre-defocus lens thickness (PDLT) and after defocus lens thickness (ADLT) following 1 h exposure to mobile screen a statistically significant difference were observed respectively – 23.2 (±0.93), 23.14 (±0.92), p value = 0.003 and 3.61 (±0.28), 3.61 (±0.22), p value = 0.001. Other ocular parameter like pre-defocus anterior chamber depth (PDACD) and after-defocus anterior chamber depth (ADACD), pre-defocus central corneal thickness (PDCCT) and after-defocus central corneal thickness (ADCCT) no statistically significant were observed respectively – 3.45 (±0.29), 3.44 (±0.26), p value = 0.861 and 528 (±31.50), 525.17 (±24.75), p value = 0.139.

Conclusion: Exposure to mobile screens in blue mode, along with hyperopic defocus, was found to cause axial length shortening, offering potential implications for managing myopia progression.

Key words: axial length, myopia, optical defocus, screen exposure

INTRODUCTION

Myopia (shortsightedness) is a global public health issue with an increasing prevalence worldwide. If the current rate of progression continues without any preventive or control strategies, it is projected to affect 5 billion people globally by 2050 [1]. In this digital era, environmental factors seem to have a greater impact than genetic factors on the myopia onset and progression [2]. Myopia and high myopia are associated with irreversible vision loss conditions such as myopic macular degeneration (MMD), glaucoma and retinal detachment (RD) [3]. Axial elongation in myopia progression is the key factor responsible for both progression and complications [4].

Various modalities have been practiced worldwide to halt the progression of myopia and control axial length, which is a key factor. These include multi-focal lenses (spectacles and contact lenses), low-dose atropine, orthokeratology, or a combination of these [5]. Different parts of the electromagnetic spectrum can cause dysregulation of eye growth [6]. According to the International Commission on Illumination (CIE, *Commission Internationale de I'Eclairage*), the lowest wavelength in optical radiation (visible spectrum) is 360–400 nm violet light, which merges with the ultra-violet (UVA) spectrum [7]. Recent findings suggest that shorter wavelengths, especially violet light (360–400 nm), can retard axial elongation and suppress myopia progression. However, this wavelength is rarely present in indoor lighting and predominantly exists in the outdoor electromagnetic spectrum, thereby suppressing myopia progression and controlling axial elongation [8]. Moreover, distinct wavelengths in sunlight, as compared to fluorescent or LED (light-emitting diode) lighting, retard myopia progression [9]. It is also hypothesized that indoor lighting lacks shorter wavelengths (violet and blue spectrum), therefore resulting in the onset and progression of myopia with increasing prevalence [10].

Optical defocus, either myopic or hyperopic, by placing plus and minus lenses, is another factor responsible for axial shortening and elongation, respectively, as evident from most experimental models [11]. However, in myopia, the peripheral hyperopic defocus is thought to be the primary factor for the progression of myopia and axial elongation. All modern myopia management strategies depend on reducing this peripheral hyperopic defocus, thereby controlling the increase in myopia and axial length [12]. The myopiagenic impact of induced hyperopic defocus is remarkably uniform among species [13]. According to longitudinal chromatic aberration (LCA), shorter wavelengths tend to refract more than longer wavelengths. This causes axial shortening, while longer wavelengths cause axial elongation [14]. Exposure to blue light in the evening causes axial elongation in chicks, providing evidence for the development of myopia [15]. Blue light exposure along imposed optical defocus had no significant change in choroidal thickness [16].

This study was conducted to determine the combined effect of hyperopic defocus and screen exposure on the axial length among emmetropes.

METHODS

This quasi-experimental study design employed at Al-Shifa Trust Eye Hospital, focusing on students of optometry selected through a non-probability sampling technique. A sample size of 30 subjects was calculated using G*Power Software version 3.1.9.4, keeping the effect size at a medium-high level (0.7), α error probability at 0.05, and the power of the study at 1-β error probability (0.95). To enhance the power of the study, a sample size of 30 was calculated by adding 25%. The subjects, aged between 19 and 23 years, were considered emmetropes if they had a spherical equivalent refraction ranging from less than +0.50 DS to -0.50 DS and a LogMAR visual acuity of 0.0. Subjects with chronic ocular diseases, a history of trauma or intraocular surgery, and chronic systemic diseases such as hypertension or diabetes mellitus, as well as those with obesity or who were on systemic or topical medications like steroids, sulfa drugs, and prostaglandin analogs, were excluded from the study. The distance from the anterior cornea to the retinal pigment epithelium, known as the axial length, is determined using optical biometry. Hyperopic defocus was induced by placing a concave lens of -3.00 DS in front of one eye. Visual acuity was measured using a standard LogMAR chart, and refractive status was determined with a retinoscope (Heine 200). Exposure to the mobile screen in full blue mode was given using a Samsung Galaxy A7 smartphone for 1 h.

The axial length was measured using optical biometry (IOL Master Zeiss 700). Data were entered and analyzed using the online web software "Data tab.". Fully informed consent was obtained from all subjects, ensuring confidentiality of data as per the tenets of the Helsinki Declaration.

Subjects with normal eye health and emmetropia were selected from the diagnostic division of Al-Shifa Trust Eye Hospital at the Pakistan Institute of Ophthalmology. In order to reduce the influence of daily fluctuations in axial length, measurements were conducted exclusively between 8:30 a.m. and 12:30 p.m., adhering to the time standard of Pakistan. In addition, participants were mandated to refrain from consuming alcohol based medication, caffeine, and nicotine for a duration of 12 h prior to the commencement of the experiment. The baseline assessment of the eye's axial length was conducted on the right eye using the IOL-MASTER 700 device. This was done after a 10-minute cleansing interval, during which the subjects were seated in a dimly lit chamber to remove any residual effects. Following these measurements, the right eye was exposed to the Samsung Galaxy A7 mobile screen for a duration of 1 h. The IOL MASTER 700 was then used again to evaluate the axial lengths after this exposure. The induced defocus level was maintained constant throughout the experiment by avoiding dilation of the right eye.

For an analysis of statistics, axial length measurements were used to calculate the mean and standard deviation (SD) before and after defocus. The data was assessed for normal distribution using the Shapiro–Wilk test. Due to the non-normal distribution of the data, group comparisons were conducted using the non-parametric Wilcoxon rank test. The chosen significance level was 0.05.

RESULTS

Table 1 presents socio-demographic and clinical data for 30 study subjects, with an average age of 20.67 years, ranging from 19 to 23 years. The group consists of 6 males with an average age of 21.17 years (range 20–22 years), and 24 females with an average age of 20.54 years (range 19–23 years). All subjects have a visual acuity right eye (VAOD) measure of 0.00. The spherical equivalent right eye (SEROD) measure averages at 0.09 with a standard deviation of 0.10, indicating some variation, and ranges from 0.00 to 0.25.

Table 2 provides descriptive statistics of axial length and ocular biometrics for 30 subjects. It includes measures such as pre-defocus and after defocus: axial length (PDAXL and ADAXL), anterior chamber depth (PDACD and ADACD), lens thickness (PDLT and ADLT), central corneal thickness (PDCCT and ADCCT). The data presents the mean, standard deviation, standard error of mean, and the 95% confidence interval for each measure, offering insights into the changes in ocular biometrics before and after defocus.

TABLE 1

Descriptive socio-demographic and clinical characteristics of study subjects.

	N	Mean (±SD)	Range
Age	30	20.67 (±0.96)	19–23
Male	6	21.17 (±0.00)	20–22
Female	24	20.54 (±0.00)	19–23
VAOD	30	0.00 (±0.00)	0.00–0.00
SEROD	30	0.09 (±0.10)	0.00–0.25

SD – standard deviation; SEROD – spherical equivalent right eye; VAOD – visual acuity right eye.

TABLE 2

Descriptive statistics of axial length and ocular biometrics.

			95% Confidence interval (mean)	
N = 30	**Mean (±SD)**	**Standard error of mean**	**Upper**	**Lower**
PDAXL	23.20 (±0.69)	0.13	23.46	22.95
ADAXL	23.19 (±0.68)	0.13	23.45	22.94
PDACD	3.48 (±0.23)	0.04	3.57	3.39
ADACD	3.49 (±0.23)	0.04	3.58	3.41
PDLT	3.61 (±0.19)	0.03	3.68	3.54
ADLT	3.58 (±0.19)	0.03	3.65	3.51
PDCCT	527.06 (±22.74)	4.15	535.55	518.58
ADCCT	526.17 (±21.38)	3.90	534.15	518.18

ADACD – after defocus anterior chamber depth; ADAXL – after defocus axial length; ADCCT – after defocus central corneal thickness; ADLT – after defocus lens thickness; PDACD – pre-defocus anterior chamber depth; PDAXL - pre-defocus axial length; PDCCT – pre-defocus central corneal thickness; PDLT – pre-defocus lens thickness; SD – standard deviation.

Table 3 presents the results of a Wilcoxon signed-rank test, which is used to compare paired measurements of axial length and ocular biometrics for 30 subjects. The test results include the median and interquartile range (IQR) for each pair, along with the test statistic (W), the z-score, and the p-value. Significant differences ($p < 0.05$) were found between pre-defocus and after defocus axial length (PDAXL–ADAXL) and lens thickness (PDLT–ADLT). However, no significant differences were found for anterior chamber depth (PDACD–ADACD) and central corneal thickness (PDCCT–ADCCT).

DISCUSSION

The increasing number of mobile phones anticipates over the potential health effects of extended screen exposure, specifically on visual health. This study explored the rela-

TABLE 3

Wilcoxon signed rank test results for the paired measurement of axial length and ocular biometrics.

N = 30	Median (±IQR)	W	z	p
PDAXL–ADAXL	23.15 (±0.93) 23.12 (±0.92)	281	2.68	0.003
PDACD–ADACD	3.45 (±0.29) 3.44 (±0.26)	168	-1.07	0.861
PDLT–ADLT	3.66 (±0.28) 3.61 (±0.22)	399	3.43	< .001
PDCCT–ADCCT	528 (±31.50) 525 (±24.75)	251	1.09	0.139

ADACD – after defocus anterior chamber depth; ADAXL – after defocus axial length; ADCCT – after defocus central corneal thickness; ADLT – after defocus lens thickness; IQR – interquartile range; PDACD – pre-defocus anterior chamber depth; PDAXL – pre-defocus axial length; PDCCT – pre-defocus central corneal thickness; PDLT – pre-defocus lens thickness.

tionship between mobile screen use and axial length elongation in individuals with emmetropia experiencing a shift toward hyperopic defocus. The research, focused on short-term focus shifts, sheds light on the interplay between digital screens and ocular function. Conducted on young adults with healthy eyes, the study involved exposing the right eye to 1 h of full blue mode on a Samsung Galaxy A7 smartphone. Measurements of axial length, anterior chamber depth, lens thickness, and central corneal thickness were taken before and after the screen exposure.

The main aim of the study was to examine, if brief exposure to optically induced myopic defocus, similar to interactions with screens, may result in substantial alterations in axial length. The results correspond with prior investigations by Thakur et al., Jiang et al., and Rucker et al. [17–19]. The study discovered that the simultaneous exposure to shorter wavelengths, in addition to hyperopic defocus, neutralized the impact of defocus and led to a reduction in axial length. The biometric measures, specifically the anterior chamber depth and central corneal thickness, did not exhibit any notable alterations. This suggests that the combined effect of hyperopic defocus and exposure to shorter wavelengths did not have any discernible impact on these metrics. The shortening of axial length, which is usually accompanied by narrowing of anterior chamber depth, indicates the potential involvement of causes other than defocus and longitudinal chromatic aberration. This suggests the need for additional investigation. Curiously, central corneal thickness stayed unaltered. Significantly, the lens thickness experienced a notable reduction, indicating that the crystalline lens underwent relaxation as a result of the combined influence of hyperopic defocus and exposure to shorter wavelengths. This is in contrast to the separate impacts of hyperopic defocus (which raises lens thickness) and exposure to shorter wavelengths (which also raises lens thickness). The results suggest that the shorter wavelength has the ability to counteract the effects of hyperopic defocus on lens thickness.

The provided findings enhance our comprehension of how the spectrum makeup of light governs the formation of the eye in humans. The notable decrease in the length of the eye, even when exposed to hyperopic defocus under blue light, provides support for Rucker and Wallman's hypothesis [19] that chromatic signals associated with longitudinal chromatic aberration may not be essential for the short-term or temporary regulation of eye growth. It is worth mentioning that the use of negative lenses to create hyperopic defocus did not result in an increase in the length of the eye, even when combined with hyperopic defocus under blue light. This is interesting because blue light exposure usually leads to elongation of the eye in humans. According to the study conducted by Jiang et al. in 2014, it was found that brief exposure to blue light hinders the growth of the eyes by affecting the blue cone-mediated ON-pathway [20], rather than through longitudinal chromatic aberration processes. The text discusses the decreasing amounts of retinoic acid [21], the involvement of intrinsically photosensitive retinal ganglion cells (ipRGCs), the impact of in-focus/out-of-focus images [22], and the greater depth of focus resulting from a reduction in pupil size [23]. Notwithstanding these observations, the precise process remains ambiguous in humans. Considering the established positive correlation between pupil diameter and the size of blur circle on the retina [24], it is necessary to consider the potential impact of blur circle size on ocular size under varying light conditions.

The lighting conditions between indoor and outdoor environments differ due to the combination of different wavelengths [25]. Outdoor lighting predominantly consisted of shorter wavelengths and contained fewer longer wavelengths, which is why it had anti-myopia properties [26]. Current research revealed that exposing the human eye to shorter wavelengths of artificial light also causes axial shortening. These findings align with those of Czepita et al. [27]. According to their research, hyperopia was more prevalent among children exposed to lighting conditions containing shorter wavelengths (fluorescent light) as compared to those with longer wavelengths (tungsten light). Additionally, some evidence shows that reading from printed papers, which absorb longer wavelengths, or using blue-tinted lenses, can have an axial lengthening effect and therefore an anti-myopia effect [28]. Along similar lines, recently developed eyeglasses that transmit shorter wavelength light are designed for myopia control [29].

The findings of this study should be considered in light of several limitations. Firstly, the investigation focused solely on inducing hyperopic defocus by placing a -3.00 DS lens. Further studies are needed to explore the effects of inducing myopic defocus by placing a +3.00 DS lens. As monoocular hyperopic defocus was induced and similar changes in axial length, along with associated biometric parameter changes, were observed, further investigation is needed. This future research should involve inducing hyperopic defocus in both eyes simultaneously and should also consider the impact of exposure to shorter wavelengths. In addition, it's important to note that the change in axial length was not adjusted for choroidal thickness.

Acknowledging certain study limitations is crucial. While the findings are interesting, there is a need for more exploration into the precise mechanisms behind alterations in axial length and lens thickness. Moreover, the extended impacts of these changes on visual health warrant further investigation in future research. A more comprehensive understanding of how digital screen exposure influences eye dynamics could be attained through additional studies involving larger and more diverse samples, along with prolonged exposure times.

CONCLUSION

It was concluded that the human eye adapts to alterations in response to induced hyperopic defocus and shorter wavelengths. These interventions have opposite effects on axial length. When combined, blue light (representing shorter wavelengths) not only counteracts the effect of hyperopic defocus but also dominates by shortening the axial length. Thus, blue and shorter wavelengths could potentially be used as an anti-myopia approach.

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