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A longitudinal study of accommodative changes in biometry during incipient presbyopia

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Abstract

Purpose: To profile accommodative biometric changes longitudinally and to determine the influence of age-related ocular structural changes on the accommodative response prior to the onset of presbyopia.
Methods: Twenty participants (aged 34–41 years) were reviewed at six-monthly intervals over two and a half years. At each visit, ocular biometry was measured with the LenStar biometer (www.Haag-Streit.com) in response to 0.00, 3.00 and 4.50 D stimuli. Accommodative responses were measured by the WAM 5500 Auto Ref/Keratometer (www.grandseiko.com).
Results: During accommodation, anterior chamber depth reduced ($F = 29$, $p < 0.001$), whereas crystalline lens thickness ($F = 39$, $p < 0.001$) and axial length ($F = 5.4$, $p = 0.009$) increased. The accommodative response ($F = 5.5$, $p = 0.001$) and the change in anterior chamber depth ($F = 3.1$, $p = 0.039$), crystalline lens thickness ($F = 3.0$, $p = 0.042$) and axial length ($F = 2.5$, $p = 0.038$) in response to the 4.50 D accommodative target reduced after 2.5 years. However, the change in anterior chamber depth ($F = 2.2$, $p = 0.097$), crystalline lens thickness ($F = 1.7$, $p = 0.18$) and axial length ($F = 1.0$, $p = 0.40$) per dioptre of accommodation exerted remained invariant after 2.5 years. The increase in disaccommodated crystalline lens thickness with age was not significantly associated with the reduction in accommodative response ($R = 0.32$, $p = 0.17$).
Conclusion: Despite significant age-related structural changes in disaccommodated biometry, the change in biometry per dioptre of accommodation exerted remained invariant with age. The present study supports the Helmholtz theory of accommodation and suggests an increase in lenticular stiffness is primarily responsible for the onset of presbyopia.

Introduction

Current presbyopia theories are derived from our understanding of the mechanism of accommodation in young eyes, based on the Helmholtz theory.[1] However, despite at least a century of investigation, the exact mechanism of accommodation, and the impact of age-related changes in the accommodative apparatus, remains equivocal.[2,3]

Present understanding of the mechanism of accommodation suggests the contractile increase in ciliary muscle thickness[4,5] reduces zonular tension, which instigates a reduction in crystalline lens equatorial diameter,[6,7] a reduction in the radii of curvature of the anterior and posterior crystalline lens surfaces[8,9] and an increase in crystalline lens axial thickness.[6,7] The increase in lenticular thickness during accommodation is produced entirely by an increase in the thickness of the nucleus[10] and the increase in steepness of the crystalline lens surfaces is greater anteriorly than posteriorly.[8,9] Moreover, the posterior axial movement of the posterior crystalline lens surface during accommodation is smaller than the anterior movement of the anterior crystalline lens surface.[9,11]

The force of ciliary muscle contraction is thought to be transmitted along the uveal tract; pulling the equatorial

choroid centripetally and therefore necessitating posterior pole elongation to maintain a constant ocular volume.[12,13] Indeed, a significant increase in ocular axial length has been observed with accommodation.[12–15] The recoil of the choroid, assisted by the posterior zonules,[16] is thought to restore the accommodative apparatus to a disaccommodated state following the cessation of accommodation.[1]

With age, Strenk *et al.*[4] and Sheppard & Davies[17] reported ciliary muscle contractility remains invariant, despite an increase in ciliary muscle mass with age.[18] However, more recent research by Croft *et al.*[19] has suggested the contractile response may attenuate with age. Additionally, the increase in the stiffness of the choroid[20,21] and sclera[22,23] with age may dampen the ability of the choroid to restore the accommodative apparatus to a disaccommodated state following the cessation of accommodation. However, significant age-related change in crystalline lens size and shape,[7, 24, 25] and the increase in lenticular stiffness with age,[26] have led to age-related crystalline lens changes becoming central to several presbyopia theories.[2,3,27]

Incipient presbyopia represents the phase of presbyopia where the decline in the amplitude of accommodation is critical in functional terms,[28] with a loss of approximately 3.00 D between 35 and 45 years of age.[29] Therefore, understanding the influence of age-related structural changes on the mechanism of accommodation during this period will aid the refinement of models for presbyopia development.

The aim of this study was to profile accommodative biometric changes longitudinally and to determine the influence of age-related ocular structural changes on the accommodative response during incipient presbyopia.

Method

The study was approved by the Aston University Audiology and Optometry Research Ethics Committee and was conducted in accordance with the tenets of the Declaration of Helsinki. Informed written consent was obtained from all the participants after an explanation of the nature and possible consequences of the study.

Incipient presbyopic individuals with an amplitude of accommodation >4.50 D (measured by the push-up, pull-down RAF rule method) and astigmatic error of <0.75 D in their right eye were recruited. In order to collect longitudinal data, the following experimental protocol was repeated every 6 months over 2.5 years. To control for diurnal fluctuations in axial length,[30,31] the allotted appointment time for each participant was kept as similar as possible for each review visit. One UK registered optometrist (DL) collected the data at each visit.

Stimulus response

Change in objective refractive error during accommodation was measured by the binocular open-field Grand Seiko WAM-5500 autorefractor (http://www.grandseiko.com/english/WAM-5500e.htm). The right eye of all myopic participants was fitted with a soft daily disposable spherical contact lens (Focus Dailies, nelfilcon A, 69% water content; Ciba Vision, Duluth, GA) for the duration of the study. A bespoke +5.00 D Badal lens system with a 90% high contrast Maltese cross target was mounted on the WAM-5500 autorefractor. The fixation target was placed 20 cm, 8 cm and 2 cm away from the Badal lens in order to stimulate 0.00, 3.00 and 4.50 D of accommodation, respectively. The left eye of each patient was occluded and participants were asked to focus on the centre of the Maltese cross as accurately as possible throughout data collection.[32] Participants were exposed to the stimulus for 20 s prior to the acquisition of data, which is adequate time to achieve the maximal accommodative response.[33] A 1-min distance-viewing break was permitted between the presentation of each stimulus level. Three consecutive measurements of refraction were acquired and the change in mean sphere was calculated.

LenStar biometry

Sequentially, optical biometry was measured by the Haag-Streit LenStar LS-900 (http://www.haag-streit.com/products/biometry/lenstar-ls-900r.html), with the addition of a bespoke Badal lens system incorporating a 92% transmission, 8% reflection pellicle beamsplitter (http://www.edmundoptics.co.uk), as described previously.[34] The LenStar provides accurate measurements of biometry (with a resolution of 0.01 mm) even with a contact lens in place.[34,35]

In order to provide a 0.00, 3.00 and 4.50 D accommodative stimulus, the target was positioned 10, 7 and 5.5 cm away from the +10.00 D Badal lens, respectively. Each participant wore an eye patch over their left eye and was encouraged to maintain clear focus of the fixation target throughout data collection.[32] The presentation order of each accommodative level was randomised and participants were exposed to the stimulus for 20 s prior to acquisition of data and were permitted a 1-min distance-viewing break between stimulus levels. The average of three repeat measures of corneal thickness (CT), anterior chamber depth (ACD), crystalline lens thickness (LT) and axial length (AXL) were recorded at each accommodative level. Anterior segment length (ASL = CT + ACD + LT) and vitreous chamber depth (VCD = AXL − ASL) were calculated from these values.

Visante AS-OCT crystalline lens thickness measurements

Where the LenStar biometer was unable to obtain crystalline lens thickness values, presumably due to high light transmittance of the posterior crystalline lens surface, measures were substituted with data collected from a Zeiss Visante AS-OCT (http://www.zeiss.co.uk/meditec/en_gb/home.html) with version 3.0 software.

Accommodated crystalline lens OCT images were acquired by a Visante AS-OCT (with a resolution of 0.01 mm) by modulating the internal Badal lens optometer to stimulate 0.00, 3.00 and 4.50 D of accommodation. Three crystalline lens images with the vertical fixation line visible[36] were acquired in raw image mode each and were subsequently exported in binary form (512 × 995 pixels) for analysis with custom-designed Matlab R2012b software (http://uk.mathworks.com). A pixel:mm conversion factor of 93 pixels per millimetre was derived by comparing disaccommodated Visante crystalline lens pixel thickness values to disaccommodated LenStar crystalline lens millimetre thickness data obtained on the same day from 46 individuals (mean age 39.1, SD 3.2 years; mean spherical equivalent −1.17, SD 2.09 DS).

LenStar error calculations

The LenStar uses an average refractive index to convert an optical path length into a geometrical AXL. Therefore, to correct for an overestimation of AXL due to the increase in LT with accommodation, the induced error was estimated using *Equations* 1–6.

In line with previous publications,[14,37] the relative proportions of the crystalline lens taken up by the anterior cortex, nucleus and posterior cortex during accommodation were kept constant because, despite the well-established thickening of the nucleus during accommodation,[38] the exact nature of the change in refractive index during accommodation is not fully understood.[9,39,40] In order to compensate for age-related changes in anterior cortex thickness (ACT), nucleus thickness (NT) and posterior cortex thickness (PCT), *Equations* 1–3 were used to modify the segmentation of the crystalline lens according to age (in years).[38] OPL and the average refractive index of the eye (n_{av}) were calculated using the refractive indices specified by Gullstrand's No. 1 (exact) eye with shell lens (*Equations* 4 *and* 5).[41] *Equation* 6 was used to calculate the error (E), which is subtracted from the geometric AXL reported by the LenStar to provide corrected AXL values.

$$\mathrm{ACT} = \mathrm{LT} * \frac{(0.51 + 0.012 * \mathrm{age})}{(0.51 + 0.012 * \mathrm{age}) + (2.11 + 0.003 * \mathrm{age}) + (0.33 + 0.0082 * \mathrm{age})} \quad (1)$$

$$\mathrm{NT} = \mathrm{LT} * \frac{(2.11 + 0.003 * \mathrm{age})}{(0.51 + 0.012 * \mathrm{age}) + (2.11 + 0.003 * \mathrm{age}) + (0.33 + 0.0082 * \mathrm{age})} \quad (2)$$

$$\mathrm{PCT} = \mathrm{LT} * \frac{(0.33 + 0.0082 * \mathrm{age})}{(0.51 + 0.012 * \mathrm{age}) + (2.11 + 0.003 * \mathrm{age}) + (0.33 + 0.0082 * \mathrm{age})} \quad (3)$$

$$\mathrm{OPL} = (\mathrm{CT} * 1.376) + (\mathrm{ACD} * 1.336) + (\mathrm{ACT} * 1.386) + (\mathrm{NT} * 1.406) + (\mathrm{PCT} * 1.386) + (\mathrm{VCD} * 1.336) \quad (4)$$

$$n_{av} = \left[\left(\frac{\mathrm{CT}}{\mathrm{AXL}}\right) * 1.376\right] + \left[\left(\frac{\mathrm{ACD}}{\mathrm{AXL}}\right) * 1.336\right] + \left[\left(\frac{\mathrm{ACT}}{\mathrm{AXL}}\right) * 1.386\right] + \left[\left(\frac{\mathrm{NT}}{\mathrm{AXL}}\right) * 1.406\right] + \left[\left(\frac{\mathrm{PCT}}{\mathrm{AXL}}\right) * 1.386\right] + \left[\left(\frac{\mathrm{VCD}}{\mathrm{AXL}}\right) * 1.336\right] \quad (5)$$

$$E = \frac{OPL}{n_{av}} - AXL_{unaccommodated} \quad (6)$$

Statistical analysis

Repeated measures ANOVA testing was used to determine whether the changes in biometry during accommodation (0.00, 3.00 and 4.50 D) were significant at visit 1, and whether any dependency on ametropia classification existed (SPSS, http://www-01.ibm.com/software/uk/analytics/spss/). Repeated measures ANOVAs were also used to investigate whether the change in axial biometry at each accommodative level was degraded over the course of the study and whether the response per dioptre of accommodation exerted changed. The association between changes in disaccommodated LT and changes in the accommodative response were investigated using linear regression analysis.

The target sample size for repeated measures ANOVA testing (within and between interaction), including an effect size (f) of 0.25, an error probability (α) of 0.05 and required power (1-β) of 0.80 for six repeat measurements amongst two groups, was 20 participants (G * Power, http://www.gpower.hhu.de/en.html).

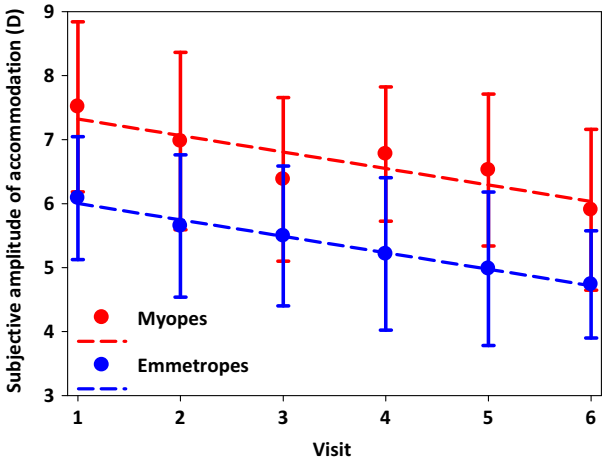

Figure 1. Right eye amplitude of accommodation (with ±1 SD error bars) measured in myopic [red; n = 10; mean age 37.2 (SD 2.1 years)] and emmetropic (blue; n = 10; mean age 38.2, SD 2.0 years) participants at each visit.

Results

Participants

Twenty individuals aged 34–41 years were recruited and completed all study visits. Ten participants were emmetropic (mean MSE −0.25, SD 0.24 D; range −0.62 to +0.17 D) and 10 were myopic (mean MSE −3.18, SD 1.27 D; range −1.29 to −6.06 D). The change in refractive error during the course of the study was not statistically significantly (F = 1.3, p = 0.28). All of the myopic participants had previous contact lens wear experience. The baseline average ages of the myopic (37.2, SD 2.1 years) and emmetropic (38.2, SD 2.0 years) groups were not statistically significantly different (t = 1.2; p = 0.26).

The reduction in subjective amplitude of accommodation after 2.5 years was statistically significant (F = 20, p < 0.001; *Figure* 1), however was not dependent on refractive error classification (F = 1.4, p = 0.25).

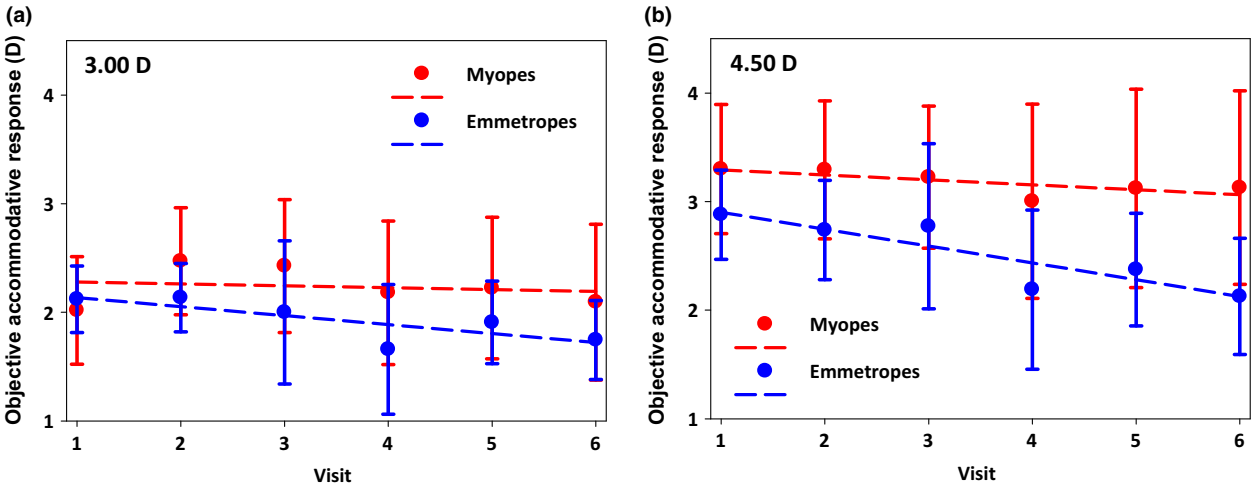

Figure 2. Mean objective accommodative response (with ±1 SD error bars) measured in myopic (red) and emmetropic (blue) participants whilst viewing a 3.00 D (a) and 4.50 D (b) accommodative target at each visit.

Table 1. Mean ocular biometric parameters and the mean disaccommodated change in the myopic and emmetropic groups after 2.5 years with the level of statistical significance

Parameter	Mean baseline value (mm ± SD)		Mean change after 2.5 years (mm ± SD)		Statistical significance (*p*) of changes after 2.5 years
	Myopes	Emmetropes	Myopes	Emmetropes	
CT	0.53 ± 0.04	0.54 ± 0.04	+0.01 ± 0.04	+0.03 ± 0.08	0.34
ACD	3.08 ± 0.29	2.96 ± 0.41	−0.08 ± 0.05	−0.18 ± 0.05	**0.003**
LT	3.84 ± 0.20	3.76 ± 0.42	+0.09 ± 0.10	+0.21 ± 0.16	**<0.001**
ASL	7.45 ± 0.30	7.25 ± 0.36	+0.01 ± 0.11	+0.04 ± 0.18	**0.045**
VCD	17.41 ± 1.15	16.42 ± 0.55	+0.02 ± 0.12	−0.03 ± 0.18	0.067
AXL	24.86 ± 1.25	23.67 ± 0.55	+0.03 ± 0.04	+0.01 ± 0.05	0.30

Bold *p* values denote statistically significant changes with time.

Changes in refractive response

Repeated measures ANOVA testing revealed that the objective accommodative response elicited by the 3.00 D accommodative target decreased significantly over the 2.5 year study (F = 3.9, $p = 0.003$; *Figure* 2*a*) and was not dependent on refractive error classification ($F = 1.7$, $p = 0.13$). Similarly, the accommodative response produced by the 4.50 D accommodative target reduced significantly over the course of the study ($F = 5.5$, $p = 0.001$; Figure 2*b*) and was not dependent on refractive error classification (F = 1.5, $p = 0.23$). The magnitude of the accommodative response exerted by the myopic cohort was significantly greater than the emmetropic response at the 4.50 D level ($F = 5.8$, $p = 0.028$).

Changes in ocular biometry

At 0.00 D accommodative stimulus, a statistically significant reduction in ACD and an increase in LT and ASL were observed after 2.5 years (*Table* 1). The change in disaccommodated LT was significantly larger amongst the emmetropic participants (+0.208, SD 0.157 mm) when compared to the myopic participants (+0.088, SD 0.119 mm; $F = 3.6$, $p = 0.023$), however changes in CT ($F = 0.90$, $p = 0.49$), ACD ($F = 2.3$, $p = 0.056$), ASL ($F = 0.63$, $p = 0.68$), VCD ($F = 0.82$, $p = 0.51$) and AXL ($F = 0.43$, $p = 0.74$) were not dependent on refractive error classification.

The changes in ACD ($F = 29$, $p < 0.001$), LT ($F = 39$, $p < 0.001$) and AXL ($F = 5.4$, $p = 0.009$) stimulated by the 3.00 and 4.50 D accommodative targets were statistically significant at visit 1, however changes in ASL ($F = 1.1$, $p = 0.35$) and VCD ($F = 1.1$, $p = 0.34$) were not. No differences in accommodative response emerged according to refractive error classification (ACD $F = 0.86$, $p = 0.43$; LT $F = 1.1$, $p = 0.34$; ASL $F = 0.001$, $p = 0.99$; VCD $F = 0.016$, $p = 0.98$; AXL $F = 0.76$, $p = 0.48$), however VCD ($F = 6.6$, $p = 0.019$) and AXL ($F = 5.4$, $p = 0.009$) values were significantly longer in myopic eyes (ACD $F = 1.5$, $p = 0.23$; LT $F = 0.057$, $p = 0.81$; ASL $F = 0.19$, $p = 0.19$).

Whilst viewing the 3.00 D accommodative target, the changes in ACD ($F = 2.0$, $p = 0.14$), LT ($F = 1.9$, $p = 0.15$) and AXL ($F = 0.45$, $p = 0.81$) were not significantly attenuated over the course of the 2.5 year study (*Figure* 2) and were not dependent on refractive error classification (ACD $F = 2.8$, $p = 0.055$; LT $F = 1.7$, $p = 0.15$; AXL $F = 1.5$, $p = 0.21$).

The change in ACD ($F = 3.1$, $p = 0.039$), LT ($F = 3.0$, $p = 0.042$) and AXL ($F = 2.5$, $p = 0.038$) whilst viewing the 4.50 D accommodative target significantly reduced over the 2.5 year study (*Figure* 3). The changes in biometry at 4.50 D were not dependent on refractive error classification (ACD $F = 1.7$, $p = 0.18$; LT $F = 1.7$, $p = 0.15$; AXL $F = 1.5$, $p = 0.21$).

The biometric response per dioptre of accommodation exerted to the 4.50 D accommodative target was not significantly attenuated over the 2.5 year study (ACD $F = 2.2$, $p = 0.097$; LT $F = 1.7$, $p = 0.18$; $p = 0.46$; AXL $F = 1.0$, $p = 0.40$; *Table* 2) and did not depend on refractive grouping (ACD $F = 1.2$, $p = 0.33$; LT $F = 1.5$, $p = 0.23$; AXL $F = 0.97$, $p = 0.44$). The magnitude of the change per dioptre of accommodation was not statistically dependent on whether the participant was classified as myopic or emmetropic (ACD $F = 0.72$, $p = 0.72$; LT $F = 3.5$, $p = 0.079$; AXL $F = 0.049$, $p = 0.83$). The magnitude of the change in AXL per dioptre of accommodation was not dependent on baseline AXL at visit 1 ($r = 0.084$, $p = 0.73$).

The increase in disaccommodated LT with age was not statistically significantly related to the reduction in accommodative lenticular response ($R = 0.43$, $p = 0.059$) or the refractive response at 4.50 D ($R = 0.32$, $p = 0.17$).

Discussion

The present investigation is the first to document an age-related attenuation of changes in ACD, LT and AXL with

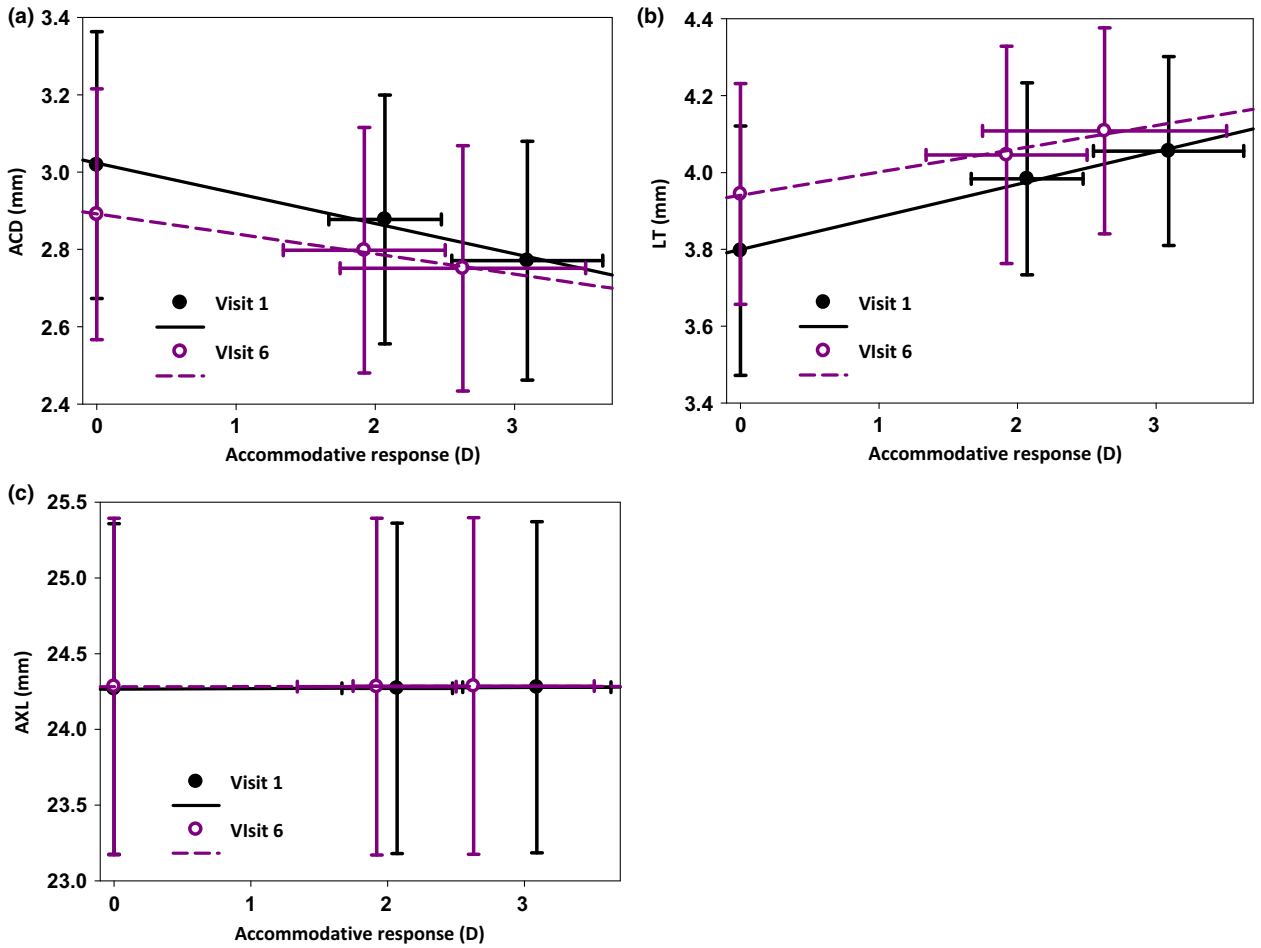

Figure 3. Mean ACD (a), LT (b) and AXL (c) (with ±1 SD error bars) at visit 1 (black filled circles and solid regression line) and after 2.5 years at visit 6 (purple open circles and dashed regression line) according to the accommodative response exerted at visit 1 and 6, respectively, to the 0.00, 3.00 and 4.50 D targets.

Table 2. Mean changes in axial biometry per dioptre of accommodation exerted (whilst viewing the 4.50 D accommodative target) at visit 1 (baseline) and visit 6 (after 2.5 years) for the myopic and emmetropic groups individually

	Mean change per dioptre of accommodation exerted at Visit 1 (mm ± SD)		Mean change per dioptre of accommodation exerted at Visit 6 (mm ± SD)		
Biometry	**Myopes**	**Emmetropes**	**Myopes**	**Emmetropes**	**Statistical significance (*p*) of changes over 2.5 years**
ACD	−0.063 ± 0.020	−0.101 ± 0.078	−0.051 ± 0.025	−0.065 ± 0.026	0.097
LT	+0.066 ± 0.023	+0.104 ± 0.079	+0.054 ± 0.023	+0.073 ± 0.020	0.18
AXL	+0.005 ± 0.004	+0.002 ± 0.006	+0.000 ± 0.005	+0.004 ± 0.008	0.40

The *p* values denote the significance of the change in accommodative response at each parameter over 2.5 years for myopic and emmetropic individuals.

accommodation in an incipient presbyopic population longitudinally. A significant decrease in ACD and an increase in LT and AXL accompanied accommodation. Despite significant age-related structural changes in disaccommodated biometry, the change in biometry per dioptre of accommodation exerted remained invariant with age, as reported by Koretz *et al.*[42]

The magnitude of the change in anterior biometry per dioptre of accommodation exerted was similar to the results from earlier studies utilising a variety of imaging

Table 3. Summary of previous research investigating changes in anterior biometry per dioptre of accommodation exerted ± standard deviation (where possible)

			Change per dioptre of accommodation exerted ± SD (mm D^{-1})		
Study	**Age range (years)**	**Technique**	**ACD**	**LT**	**ASL**
Current study Visit 1 *n* = 20	34–41	LCR, AS-OCT	−0.082 ± 0.061	+0.085 ± 0.060	+0.003 ± 0.050
Richdale *et al.*[7] *n* = 26 Emmetropes	30–50	AS-OCT MRI	–	+0.064 +0.065	–
Sheppard *et al.*[6] *n* = 19	19–30	MRI	–	+0.08 ± 0.05	–
Richdale *et al.*[36] *n* = 22	36–50	AS-OCT	–	+0.051 ± 0.019	–
Bolz *et al.*[43] *n* = 10 Myopes	19–31	PCI	−0.057	+0.072	+0.013
Bolz *et al.*[43] *n* = 10 Emmetropes	19–31	PCI	−0.047	+0.063	+0.009
Ostrin *et al.*[44] *n* = 22	21–30	A-scan US	−0.051 ± 0.008	+0.067 ± 0.008	+0.017 ± 0.005
Garner and Yap[54] *n* = 11	18–28	A-scan US	−0.054	+0.054	–
Koretz *et al.*[42] *n* = 42 Emmetropes	18–40	Scheimpflug	−0.038 ± 0.139	+0.043 ± 0.145	+0.003 ± 0.174

techniques (*Table* 3). The majority of the increase in LT was compensated for by the reduction in ACD, which supports previous findings that the movement of the crystalline lens surface and reduction in radii of curvature is significantly greater anteriorly than posteriorly,[8,9] and the Helmholtz theory of accommodation.[1]

The increase in disaccommodated LT with age was not statistically significantly related to the reduction in accommodative response at 4.50 D. Therefore, it is unlikely age-related changes in lenticular geometry alone are responsible for the reduction in accommodative ability. Consequently, the results of the current study suggest the increase in lenticular stiffness with age is primarily responsible for reduction in lenticular accommodative response.

The change in ASL and VCD with accommodation was not significant. However, previous studies have reported a significant increase in ASL occurs with accommodation in younger individuals.[14,43–45] The negligible change in ASL observed during incipient presbyopia may suggest the mobility of the posterior crystalline lens surface diminishes with age.[19] Indeed, the accommodative movement of the posterior lens surface in individuals aged between 18 and 36 years has been reported to be bi-phasic[46]; that is to say the posterior lenticular movement is negligible until the accommodative demand reaches approximately 2.00 D (eliciting an accommodative response of approximately 1.50 D). The initial static phase is thought to originate from vitreous humour resistance.[46] It is feasible age-related anterior migration of the anterior zonules[47] and decreased flexibility of the posterior crystalline lens[26] and capsule[48] may perpetuate the initial static phase of accommodative posterior crystalline lens surface movement in older individuals. Nevertheless, as demonstrated by the large ASL standard deviation values in *Table* 3, significant intersubject variability in the response of the posterior crystalline lens surface occurs. Further research investigating the link between crystalline lens placement, zonular architecture and accommodative changes in ASL is indicated.

Similarly to research in young adults,[12–14, 37] a statistically significant elongation of AXL during accommodation was observed within the current cohort. No differences in AXL response arose according to refractive error grouping, as reported previously.[14] The AXL elongation per dioptre of accommodation exerted remained invariant after 2.5 years, thus suggesting the susceptibility of the choroid to the force of ciliary muscle contraction did not decrease. It would be of interest to investigate whether AXL change during accommodation is attenuated over the lifespan due

to increases in choroidal[20,21] and scleral stiffness[22,23] and also to investigate longitudinally whether the ciliary muscle contractile response is attenuated with age.

The magnitude of the accommodative response exerted by the myopic group was significantly larger than exerted by the emmetropic group. Indeed, anecdotal and published evidence[49] suggests hypermetropic and emmetropic patients manifest presbyopia before myopic patients. The origin of this phenomenon has been traditionally thought to arise from near vision effectivity of myopic spectacle lenses (reducing the accommodative demand for myopic spectacle wearers)[50] or the increased vitreous chamber depth associated with myopia (requiring a smaller change in axial ocular distances to produce accommodation due to the relatively more distant retinal plane).[51] However, it is feasible myopic structural changes occurring during adolescence, perhaps lenticular thinning[52,53] and lenticular equatorial expansion,[53] may preserve the accommodative ability and delay the onset of presbyopia. The biomechanical reason for this putative association requires further investigation. It would also be of interest to document the reduction in the accommodative response longitudinally over the lifespan to investigate the influence of ametropia on accommodation further.

A possible limitation of this study is the inclusion of LT measurements derived from both LenStar and Visante instruments. However, the pixel thickness measurements of the Visante were calibrated against LenStar measurements, therefore minimising any potential disparity. Nonetheless, the LT for each participant was either wholly measured by the LenStar (11 participants) or by the Visante (nine participants) to minimise any potential inaccuracies.

The current study provides the first prospective, longitudinal insight into how accommodative changes in axial ocular biometry attenuate during incipient presbyopia. In conclusion, the accommodative decrease in ACD and increase in LT and AXL are significantly attenuated with age, however the response per dioptre of accommodation exerted remains constant with age. The mobility of the posterior crystalline lens surface also appears to reduce with age. Further longitudinal research is required to investigate how accommodative changes in ocular biometry change over the lifespan. The present study supports the Helmholtz theory of accommodation and suggests the increase in lenticular stiffness is likely to be primarily responsible for the onset of presbyopia.

Disclosure

The authors report no conflicts of interest and have no proprietary interest in any of the materials mentioned in this article.

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References

1. vonHelmholtz H. Mechanism of accommodation. In: *Helmholtz's Treatise on Physiological Optics, Vol. 1* (Southall JP, editor), Optical Society of America: New York, 1924; pp. 382–415.
2. Atchison DA. Accommodation and presbyopia. *Ophthalmic Physiol Opt* 1995; 15: 255–272.
3. Gilmartin B. The aetiology of presbyopia: a summary of the role of lenticular and extralenticular structures. *Ophthalmic Physiol Opt* 1995; 15: 431–437.
4. Strenk SA, Strenk LM & Guo S. Magnetic resonance imaging of aging, accommodating, phakic, and pseudophakic ciliary muscle diameters. *J Cataract Refract Surg* 2006; 32: 1792–1798.
5. Sheppard AL & Davies LN. *In vivo* analysis of ciliary muscle morphologic changes with accommodation and axial ametropia. *Invest Ophthalmol Vis Sci* 2010; 51: 6882–6889.
6. Sheppard AL, Evans CJ, Singh KD, Wolffsohn JS, Dunne MC & Davies LN. Three-dimensional magnetic resonance imaging of the phakic crystalline lens during accommodation. *Invest Ophthalmol Vis Sci* 2011; 52: 3689–3697.
7. Richdale K, Sinnott LT, Bullimore MA *et al.* Quantification of age-related and per diopter accommodative changes of the lens and ciliary muscle in the emmetropic human eye. *Invest Ophthalmol Vis Sci* 2013; 54: 1095–1105.
8. Brown N. The change in shape and internal form of the lens of the eye on accommodation. *Exp Eye Res* 1973; 15: 441–459.
9. Dubbelman M, Van der Heijde G & Weeber H. Change in shape of the aging human crystalline lens with accommodation. *Vision Res* 2005; 45: 117–132.
10. Hermans E, Dubbelman M, van der Heijde R & Heethaar R. The shape of the human lens nucleus with accommodation. *J Vis* 2007; 7: 16.
11. Beauchamp R & Mitchell B. Ultrasound measures of vitreous chamber depth during ocular accommodation. *Am J Optom Physiol Opt* 1985; 62: 523–532.
12. Drexler W, Findl O, Schmetterer L, Hitzenberger CK & Fercher AF. Eye elongation during accommodation in humans: differences between emmetropes and myopes. *Invest Ophthalmol Vis Sci* 1998; 39: 2140–2147.
13. Mallen EA, Kashyap P & Hampson KM. Transient axial length change during the accommodation response in young adults. *Invest Ophthalmol Vis Sci* 2006; 47: 1251–1254.
14. Read SA, Collins MJ, Woodman EC & Cheong S-H. Axial length changes during accommodation in myopes and emmetropes. *Optom Vis Sci* 2010; 87: 656–662.

15. Woodman EC, Read SA, Collins MJ *et al.* Axial elongation following prolonged near work in myopes and emmetropes. *Br J Ophthalmol* 2011; 95: 652–656.
16. Lütjen-Drecoll E, Kaufman PL, Wasielewski R, Ting-Li L & Croft MA. Morphology and accommodative function of the vitreous zonule in human and monkey eyes. *Invest Ophthalmol Vis Sci* 2010; 51: 1554–1564.
17. Sheppard AL & Davies LN. The effect of ageing on *in vivo* human ciliary muscle morphology and contractility. *Invest Ophthalmol Vis Sci* 2011; 52: 1809–1816.
18. Pardue MT & Sivak JG. Age-related changes in human ciliary muscle. *Optom Vis Sci* 2000; 77: 204–210.
19. Croft MA, McDonald JP, Katz A, Lin T-L, Lütjen-Drecoll E & Kaufman PL. Extralenticular and lenticular aspects of accommodation and presbyopia in human versus monkey eyes. *Invest Ophthalmol Vis Sci* 2013; 54: 5035–5048.
20. Van Alphen G & Graebel WP. Elasticity of tissues involved in accommodation. *Vision Res* 1991; 31: 1417–1438.
21. Ugarte M, Hussain A & Marshall J. An experimental study of the elastic properties of the human Bruch's membrane-choroid complex: relevance to ageing. *Br J Ophthalmol* 2006; 90: 621–626.
22. Friberg TR & Lace JW. A comparison of the elastic properties of human choroid and sclera. *Exp Eye Res* 1988; 47: 429–436.
23. Pallikaris IG, Kymionis GD, Ginis HS, Kounis GA & Tsilimbaris MK. Ocular rigidity in living human eyes. *Invest Ophthalmol Vis Sci* 2005; 46: 409–414.
24. Koretz JF, Kaufman PL, Neider MW & Goeckner PA. Accommodation and presbyopia in the human eye—aging of the anterior segment. *Vision Res* 1989; 29: 1685–1692.
25. Atchison DA, Markwell EL, Kasthurirangan S, Pope JM, Smith G & Swann PG. Age-related changes in optical and biometric characteristics of emmetropic eyes. *J Vis* 2008; 8: 29.
26. Heys KR, Cram SL & Truscott RJ. Massive increase in the stiffness of the human lens nucleus with age: the basis for presbyopia? *Mol Vis* 2004; 16: 956–953.
27. Strenk SA, Strenk LM & Koretz JF. The mechanism of presbyopia. *Prog Retin Eye Res* 2005; 24: 379–393.
28. Baker FJ & Gilmartin B. A longitudinal study of vergence adaptation in incipient presbyopia. *Ophthalmic Physiol Opt* 2003; 23: 507–511.
29. Duane A. Normal values of the accommodation at all ages. *JAMA* 1912; 59: 1010–1013.
30. Stone RA, Quinn GE, Francis EL *et al.* Diurnal axial length fluctuations in human eyes. *Invest Ophthalmol Vis Sci* 2004; 45: 63–70.
31. Read SA, Collins MJ & Iskander DR. Diurnal variation of axial length, intraocular pressure, and anterior eye biometrics. *Invest Ophthalmol Vis Sci* 2008; 49: 2911–2918.
32. Stark LR & Atchison DA. Subject instructions and methods of target presentation in accommodation research. *Invest Ophthalmol Vis Sci* 1994; 35: 528–537.
33. Heron G & Winn B. Binocular accommodation reaction and response times for normal observers. *Ophthalmic Physiol Opt* 1989; 9: 176–183.
34. Alderson A, Davies LN, Mallen EA & Sheppard AL. A method for profiling biometric changes during disaccommodation. *Optom Vis Sci* 2012; 89: E738–E748.
35. Buckhurst PJ, Wolffsohn JS, Shah S, Naroo SA, Davies LN & Berrow EJ. A new optical low coherence reflectometry device for ocular biometry in cataract patients. *Br J Ophthalmol* 2009; 93: 949–953.
36. Richdale K, Bullimore MA & Zadnik K. Lens thickness with age and accommodation by optical coherence tomography. *Ophthalmic Physiol Opt* 2008; 28: 441–447.
37. Woodman EC, Read SA & Collins MJ. Axial length and choroidal thickness changes accompanying prolonged accommodation in myopes and emmetropes. *Vision Res* 2012; 72: 34–41.
38. Dubbelman M, Van der Heijde G, Weeber H & Vrensen G. Changes in the internal structure of the human crystalline lens with age and accommodation. *Vision Res* 2003; 43: 2363–2375.
39. Hermans EA, Dubbelman M, Van der Heijde R & Heethaar RM. Equivalent refractive index of the human lens upon accommodative response. *Optom Vis Sci* 2008; 85: 1179–1184.
40. Jones CE, Atchison DA & Pope JM. Changes in lens dimensions and refractive index with age and accommodation. *Optom Vis Sci* 2007; 84: 990–995.
41. Atchison DA & Smith G. Possible errors in determining axial length changes during accommodation with the IOL-Master. *Optom Vis Sci* 2004; 81: 283–286.
42. Koretz JF, Cook CA & Kaufman PL. Accommodation and presbyopia in the human eye. Changes in the anterior segment and crystalline lens with focus. *Invest Ophthalmol Vis Sci* 1997; 38: 569–578.
43. Bolz M, Prinz A, Drexler W & Findl O. Linear relationship of refractive and biometric lenticular changes during accommodation in emmetropic and myopic eyes. *Br J Ophthalmol* 2007; 91: 360–365.
44. Ostrin L, Kasthurirangan S, Win-Hall D & Glasser A. Simultaneous measurements of refraction and A-scan biometry during accommodation in humans. *Optom Vis Sci* 2006; 83: 657–665.
45. Tsorbatzoglou A, Németh G, Széll N, Biró Z & Berta A. Anterior segment changes with age and during accommodation measured with partial coherence interferometry. *J Cataract Refract Surg* 2007; 33: 1597–1601.
46. Gibson GA. *Theoretical and empirical evaluation of phakic and pseudophakic accommodation*. PhD thesis, Aston University, 2008; pp. 116–149.
47. Farnsworth PN & Shyne SE. Anterior zonular shifts with age. *Exp Eye Res* 1979; 28: 291–297.
48. Fisher R. Elastic constants of the human lens capsule. *J Physiol* 1969; 201: 1–19.

49. Rabbetts RB & Mallen EAH. Accommodation and near vision. The inadequate stimulus myopias. In: *Bennett & Rabbett's Clinical Visual Optics* (Rabbetts RB, editor), 4th edition, Elsevier: Oxford, 2007; pp. 125–153.
50. Hunt OA, Wolffsohn JS & García-Resúa C. Ocular motor triad with single vision contact lenses compared to spectacle lenses. *Cont Lens Anterior Eye* 2006; 29: 239–245.
51. Davies LN, Dunne M, Gibson GA & Wolffsohn JS. Vergence analysis reveals the influence of axial distances on accommodation with age and axial ametropia. *Ophthalmic Physiol Opt* 2010; 30: 371–378.
52. McBrien NA & Millodot M. A biometric investigation of late onset myopic eyes. *Acta Ophthalmol* 1987; 65: 461–468.
53. Zadnik K, Mutti DO, Fusaro RE & Adams AJ. Longitudinal evidence of crystalline lens thinning in children. *Invest Ophthalmol Vis Sci* 1995; 36: 1581–1587.
54. Garner LF & Yap MK. Changes in ocular dimensions and refraction with accommodation. *Ophthalmic Physiol Opt* 1997; 17: 12–17.