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ABSTRACT

Purpose: To assess the correlation between scleral shape and corneal astigmatism.

Methods: Twenty-two participants (11 non-astigmatic and 11 astigmatic) aged from 19 to 36 years and with no previous ocular surgeries were included in this study. Three-dimensional (3D) corneo-scleral maps from both eyes (44 eyes) were acquired using a corneo-scleral topographer (Eye Surface Profiler). Each 3D map was split up in 13 concentric annuli, each 0.5 mm-wide, staring at 1.0 mm radius from the corneal apex to the scleral periphery at 7.5 mm from the apex. Each ring was fitted to a quadratic function of the radial distance to the apex, to calculate the elevation difference between the raw data and the fitting surface ring. For each ring the resulting elevation difference between original and fit data profile was fit to a sum of sines function. Decentration and astigmatic terms obtained from the sinusoidal fit were analyzed and compared between non-astigmatic and astigmatic groups.

Results: In astigmatic eyes corneal and scleral asymmetry are highly correlated, while both appear independent from each other in non-astigmatic eyes. No significant difference between astigmatic and non-astigmatic eyes was found for decentration term ($p > 0.05/[N$ (Bonferroni)), while for the astigmatic component the differences were statistically significant ($p < 0.05/N$ (Bonferroni)).

Conclusion: Corneal and scleral shape are correlated in astigmatic eyes, suggesting that astigmatism is not restricted to the cornea, but should rather be considered a property of the entire eye globe.

Keywords: astigmatism; cornea; sclera; topography

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INTRODUCTION

Astigmatism is a common refractive error that affects around one third of the population,[1] resulting from an unequal refraction displayed along the different meridians of the eye. If left uncorrected, astigmatism usually leads to worse a visual quality than similar amounts of spherical refraction,[2] which makes it important that the condition is understood and corrected as well as possible. However, despite extensive research, the factors contributing to astigmatism remain elusive,[3,4] with some authors suggesting genetic [5,6] or environmental influences.[7,8] Since astigmatism generally originates at the level of the cornea, eyelid pressure has been proposed as a contributing factor, supported by the observation that eyelid position influences the degree and direction of corneal astigmatism.[3,9-11] The typical shift in astigmatic axis from with-the-rule in young adults to against-the-rule in elderly adults was explained by Grosvenor[3] through a decrease in lid tension with age, leading to a reduction in with-the-rule corneal astigmatism. The unequal tension that the extraocular muscles exert on the cornea has also been proposed as a cause,[12] but the literature on this is rather sparse. Some works have focused on changes in corneal topography following extraocular muscle surgery.[13-15] Meanwhile, in a previous work by our group analyzed the scleral shape from three-dimensional (3D) corneo-scleral maps with a radius of 6 to 8 mm. This previous work concluded that the human sclera is rotationally asymmetric and substantial inter-subject variation in scleral shape was found.[16] Moreover the magnitude of the scleral asymmetry was shown to increase with radial distance, suggesting that the position of the muscle insertion points might affect scleral shape.[16]

As asymmetries in scleral shape may influence the shape of the corneal periphery, current work revisits the earlier obtained cornea-scleral data[16] to ascertain the relationship between the scleral asymmetry and corneal astigmatism in both astigmatic and non-astigmatic eyes.

MATERIALS AND METHODS

Participants

Twenty-two participants (16 females, 6 males; 44 eyes) aged between 19 and 36 years (mean 27.0 ± 4.7 years) were included in this study. The study was approved by University of Manchester Human Research Ethics Committee and adhered to the tenets of the Declaration of Helsinki. All participants gave written informed consent to participate after the nature and possible consequences of the study were explained. All participants were free of ocular disease and current use of topical ocular medications was specified by the participants as part of a background questionnaire. Participants with moderate or high myopia (< −2.00 D) were excluded. It is well-known that the visco-elastic properties of the sclera are markedly altered in myopic eyes[17] and consequently that myopic sclera reduces its rigidity.[18] According to these results we conjecture that myopia alone might have an influence on scleral shape. To avoid this potential influence we decided not to include participants with moderate or high myopia. Since astigmatism is often accompanied by myopia this significantly reduced the number of participants that could be included. Exclusion criteria also included the presence of significant tear film abnormalities or any corneal, conjunctival or scleral pathology, any history of ocular surgery, as well as contact lens wear. The refractive state was measured monocularly using a wide-view open window autorefractometer (Shin Nippon SRW-500, Ajinomoto Trading Inc., Japan). For statistical analysis, participants were

grouped in two different categories according to their cylinder as low-astigmatic (Cyl < 0.75 D in both eyes; 11 participants), or astigmatic (Cyl > 1.00 D in both eyes; 11 participants). For simplicity, the group of low-astigmatic participants is referred in this work as 'non-astigmatic'. In all participants of the latter group the astigmatism was with-the-rule. This sample size was chosen based on calculations conducted using previous published data on scleral topography,[16] which suggested that a sample size of 10 participants would yield 80% power to detect 40 μm differences in sclera elevation at the 0.05 significant level. This value was chosen according to the inherent noise of the measuring device in the corneo-scleral peripheral area, that was found to amount to below 40 μm error for an extended measurement area of 16 mm diameter in calibrated artificial surfaces.[19]

Data collection

The study was performed in a single visit during which participants were measured with a non-contact corneo-scleral topographer (Eye Surface Profiler (ESP), Eaglet Eye BV, Netherlands), a height profilometer able to measure the corneo-scleral topography several millimeters beyond the limbus. The algorithms used in the ESP achieve similar levels of accuracy for corneal surface heights as Placido disk based videokeratoscopes.[19] Anterior eye surface measurements using ESP require instillation of fluorescein with a solution more viscous than saline. The BioGlo (HUB Pharmaceuticals, United States) ophthalmic strips were used to gently touch the upper temporal ocular surface. They were impregnated with 1 mg of fluorescein sodium ophthalmic moistened with one drop of an eye lubricant (HYLO-Parin, 1mg/ml of sodium hyaluronate). Four measurements were collected from both eyes of each participant, but left and right eyes were always considered separately to account for possible within-subject correlations. Participants were instructed to open their eyes wide, but without using force, prior to the ESP measurements to ensure maximal coverage of the corneo-scleral area. Measurements in which the corneo-scleral area was covered by eyelids were excluded. From the four measurements acquired per eye the one with the largest scleral area coverage was included for data analysis.

Analysis

Following data acquisition the raw 3D anterior eye height data (X, Y, and Z coordinates) were exported from the ESP for further analysis. Each 3D map was split up in 13 concentric annuli, each 0.5 mm-wide, staring at 1.0 mm radius from the corneal apex to the scleral periphery at 7.5 mm from the apex. Each ring was fitted to a quadratic function of the radial distance to the apex, as explained elsewhere,[16] to calculate the elevation difference between the raw data and the fitting surface ring. For each ring the resulting elevation difference between original and fit data profile was fit to a sum of sines function, defined by:

$$y = \sum_{i=1}^{n} a_i \cdot \sin(b_i x + c_i)$$

Where a_i is the amplitude, b_i the frequency, and c_i the phase constant for each sine wave term. The 4th-order (n = 4) sine series was used because preliminary analysis showed that including more terms would not result in significantly better fits (Fisher test; $p > 0.05$). The value of b_i was fixed a priori (b_1=1, b_2=2, b_3=3, b_4=4) to easily decompose each fit into its fundamental modes, decentration and astigmatism. Thus, the first term of the series

(a_1·in(x+c_1)) corresponds to decentration, second term of the series (a_2·sin(2x+c_2)) to astigmatism, while the and third (a_3·sin(3x+c_3)) and fourth (a_4·sin(4x+c_4)) terms correspond to minor, more irregular contributions. This series results in a 8-parameters model (a_1,a_2,a_3,a_4,c_1,c_2,c_3,c_4), of which the amplitudes of the main terms (i.e., a_1,a_2) were include in data analysis.

Furthermore, to assess whether differences in scleral shape exist between non-astigmatic and astigmatic participants the sclera and cornea were automatically separated at the level of the limbus, assuming a mean limbal diameter of 12 mm.[20] This creates a 3D scleral ring of 6.0-7.5 mm radius, that may be fitted to a quadratic function of the radial distance to the apex to calculate the scleral elevation. This ring was divided into four sectors for statistical analysis: superior [60,120]°, inferior [240,300]°, nasal [330,30]° and temporal [150,210]°. Right eyes were corrected for mirror symmetry.

The statistical analysis was performed using SPSS (v23.0; SPSS Inc., Chicago, Illinois, United States). The Shapiro-Wilk test was used to test the normality of all continuous variables. Wilcoxon sign-rank test was performed to determine whether there were statistically significant differences between non-astigmatic and astigmatic eyes for the decentration and astigmatism amplitudes (i.e. a_1 and a_2). Bonferroni corrections were used to overcome α inflation due to multiple comparisons by dividing the significance level by the number of comparisons being performed. The correlation in amplitude components between right and left eye was assessed using the Spearman correlation coefficient (ρ). Furthermore, two-way ANOVA-repeated-measurements test, adjusted for multiple comparisons, was performed to analyze whether there exists a difference in scleral asymmetry between non-astigmatic and astigmatic eyes. The level of significance was set to 0.05.

Statistical power estimation for a post-hoc analysis with 85% power at the 5% alpha level and a sample size of 11 astigmatic subjects, can demonstrate differences in the astigmatism component (a_2) of about 30 μm. For the sample size of 11 non-astigmatic subjects, differences in astigmatism component (a_2) of less than 15 μm may be differentiated.

RESULTS

The average cylinder for the astigmatic group was −2.00 ± 1.07 D (mean ± SD), range [−1.00, −3.75] D with a mean steepest meridian axis position of 98°, for the right eye and −2.00 ± 1.50 D (mean ± SD), range [−1.00, −4.00] D with a mean steepest meridian axis position of 88°, for the left eye.

In astigmatic eyes corneal and scleral asymmetry are highly correlated (Figure 1), while both appear independent from each other in non-astigmatic eyes (Figure 2). This distinction between groups is especially apparent for the astigmatism term a_2·sin(2x+c_2) (fourth panel in Figures 1 & 2, and Figure 3). Meanwhile the decentration term a_1·sin(x+c_1) forms the dominant contribution in both cases, while higher terms a_3·sin(3x+c_3) and a_4·sin(4x+c_4)) play only a minor role.

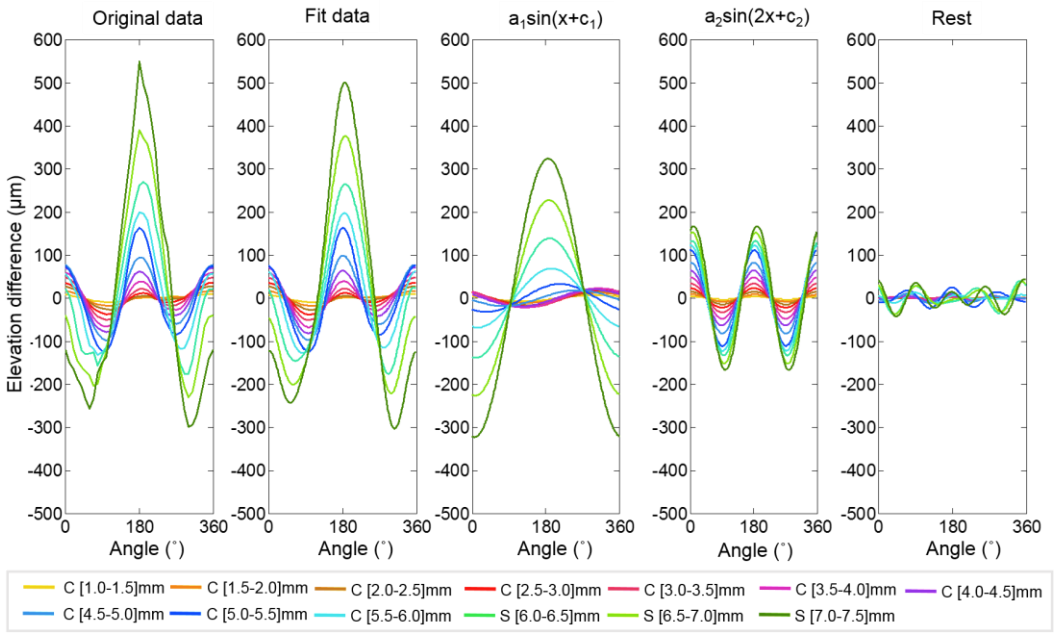

Figure 1. *Elevation profiles of each corneo-scleral ring for an astigmatic participant (male, 33 years-old, left eye, cylinder = –3.5 D), along with a sinusoidal series fit and the decomposition into individual terms. The second term of the series (a2·sin(2x+c2); fourth panel) corresponds to the astigmatism contribution. (C: cornea; S: sclera).*

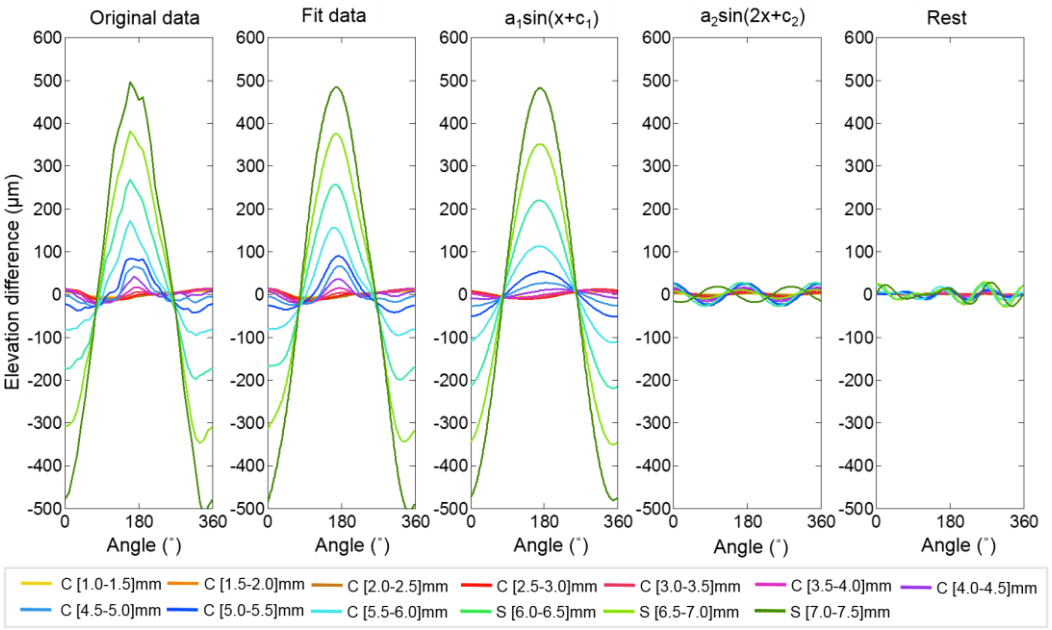

Figure 2. *Elevation profiles of each corneo-scleral ring for a non-astigmatic participant (female, 19 years-old, left eye, cylinder = –0.25 D), along with a sinusoidal series fit and the decomposition into individual terms. The second term of the series (a_2·sin(2x+c_2); fourth panel) corresponds to the astigmatism contribution. (C: cornea; S: sclera).*

The amplitude changes of the astigmatic term (a_2·sin(2x+c_2)) occur differently in non-astigmatic and astigmatic participants, with statistically significant differences between

astigmatic and non-astigmatic eyes found for all annuli under analysis (Mann-Whitney U test, all $p<0.05$). For non-astigmatic eyes the orientation c_2 of the astigmatic term varies between annuli, while it remains fairly constant in astigmatic eyes (Figure 3).

Averaging the amplitude of the decentration and astigmatism components (a_1, a_2) for all participants over each of the 13 annuli, it is clear that overall the decentration a_1 is the most important contribution (Figure 4). In participants with astigmatism, however, this is not the case for the peripheral cornea, where astigmatism a_2 becomes the major contribution.

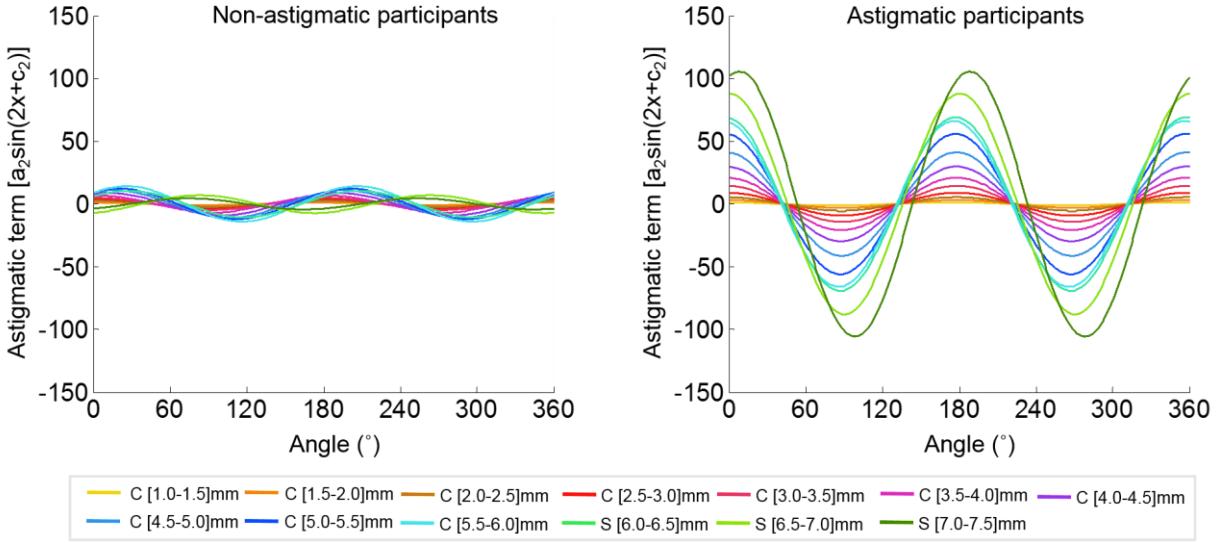

Figure 3. *Average of the second term of the series (a2·sin(2x+c2)) corresponding to the astigmatism contribution, for the participants' left eyes for astigmatic (right; 11 participants) or non-astigmatic (left; 11 participants). (C: cornea; S: sclera)*

No significant difference between astigmatic and non-astigmatic eyes was found for decentration a_1 (Wilcoxon sign rank test plus Bonferroni correction ($\alpha_{\text{per comparison}} = 0.05/13 = 0.0038$), OS: $p = 0.293$; OD: $p = 0.013$). The astigmatic component a_2, however, was significantly different between groups (OS: $p << 0.001$; OD: $p << 0.001$). Astigmatic component a_2 was statistically significant different between non-astigmatic and astigmatic participants, independently if the ocular surface was analyzed as a whole (i.e. cornea and sclera together) as it was already shown, or if corneal region or scleral region are solely compared. In this manner, similar statistical results were obtained when comparing only corneal region, i.e. data corresponding up to 6.0 radius ($\alpha_{\text{per comparison}} = 0.05/9 = 0.0056$)) between astigmatic and non-astigmatic participants, and within scleral region, i.e. data corresponding to 6.0-7.5 mm radius ($\alpha_{\text{per comparison}} = 0.05/4 = 0.0125$)), for decentration a_1 (Cornea, OS: $p = 0.293$; OD: $p = 0.0126$; Sclera, OS: $p = 0.743$; OD: $p = 0.351$) and for astigmatic component a_2 (Cornea, OS: $p = 0.0010$; OD: $p = 0.0046$; Sclera, OS: $p << 0.001$; OD: $p << 0.001$). In addition, statistically significant difference between decentration component a_1 and astigmatic component a_2 of non-astigmatic participants was found in cornea (OS: $p << 0.001$; OD: $p << 0.001$), and also in sclera (OS: $p << 0.001$; OD: $p << 0.001$). The same statistical results were obtained for astigmatic participants. Very strong positive correlations between right and left eye were found ($\rho = 1.000$; $p < 0.001$), independently of the group (astigmatic or non-astigmatic) or the component (a_1,a_2) analyzed.

There was no statistically significant difference in scleral shape (i.e. the data corresponding to the 6.0-7.5 mm radius ring) between astigmatic and non-astigmatic participants (two-way ANOVA-repeated-measurements test, OD: p = 0.363; OS: p = 0.675).

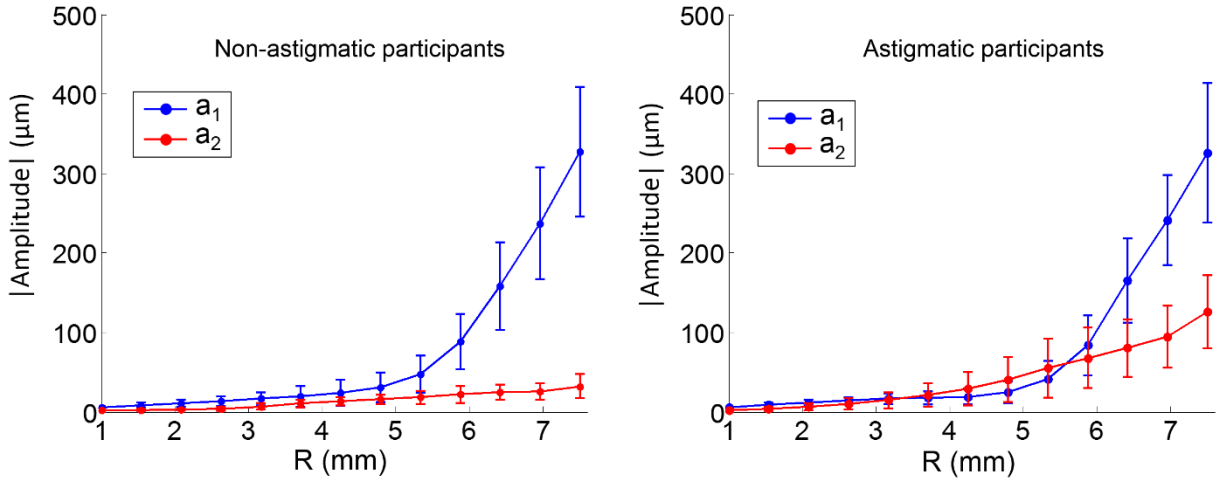

Figure 4*. Decentration component amplitude (a1, blue) and astigmatism component amplitude (a2, red) as a function of radial distance from corneal apex, for left eye of participants separated as astigmatic or non-astigmatic. Error bars indicate +/- one standard deviation from the mean.*

DISCUSSION

In order to better understand the origin of astigmatism, works regarding the influence of genetics,[5-6] eyelid pressure [3,9-11] and extraocular muscle tension[12-15] have been reported. Our work expands on these results by considering the potential effect of the sclera on corneal shape. From the 44 ocular topographic maps of 22 participants we found that, even though scleral shape is not being significantly different between non-astigmatic and astigmatic participants (OD: p = 0.363; OS: p = 0.675), scleral and corneal asymmetry are related in astigmatic eyes (Figure 1). Furthermore, when isolating the astigmatic component a_2 (Figure 3), the corneal astigmatism seems to *'follow'* the scleral astigmatism in astigmatic eyes. This is not seen in non-astigmatic eyes, where corneal and scleral astigmatism seem to be independent from each other (Figure 3). The decentration amplitude a_1 was found to be not statistically significant different between non-astigmatic and astigmatic eyes ($\alpha_{\text{per comparison}}$ = 0.0038; OS: p = 0.293; OD: p = 0.013). This was also seen in Figure 4 where the decentration amplitude (in blue) increases in much the same way in both the non-astigmatic and astigmatic panels. For the astigmatic component a_2, on the other hand between both groups: while in non-astigmatic participants a_2 increases only very slowly, astigmatic participants seen a far more rapid increase in the peripheral cornea and sclera due to the presence of the astigmatism.

It has recently been reported that the anterior sclera, unlike a standard cornea, is radially asymmetric.[16] It is well-known that the sclera is a tough, fibrous tissue made of collagen fibers. The collagen of the sclera is continuous with the cornea, however scleral collagen fibers present a irregularity of the Type I collagen fibers,[21] in opposition to the near-uniform

thickness and parallel arrangement of the corneal collagen, which makes the sclera more opaque, stiffer and tougher as a tissue than the cornea. One could therefore hypothesize that the scleral shape somehow *pushes* the cornea, affecting corneal shape and consequently inducing astigmatism. Our results appear to be in line with this hypothesis. Even though there is no clear indication in the data presented of the corneal shape being influenced by the sclera rather than the other way around, there are however arguments outside the current dataset that prompted this assumption, most important of which is the observation that the stiffness of the sclera is higher than that of the cornea. As such it would be more difficult for the cornea to deform the sclera than for the sclera to deform the cornea. Moreover, there are no external forces applied directly to the cornea, other than those exerted by the sclera. The direct influence of the eyelids on the cornea shape is mostly likely limited, and most of the influence is probably transferred through the sclera. Muscle insertion points and muscle tonus, on the other hand, are unable to directly influence the cornea, so their influence can only be transferred by the sclera.

To the best of our knowledge this work is the first evidence to show that scleral astigmatism '*follows*' corneal astigmatism, suggesting that astigmatism is not restricted to the cornea, but should rather be considered a property of the entire eye globe. Consequently the causes for astigmatism should also be sought at the eye globe level. One such cause may be the muscles insertion, based on repeated observations of astigmatism changes induced by extraocular muscle surgery.[13-15] However there is no evidence to date that muscle insertion is different in astigmatic and non-astigmatic eyes.

Ocular surgery can produce significant changes in astigmatism.[22-30] Regarding cataract surgery it has been reported that the location of the incision[22,23] as well as the size of the incision[24,25] are associated with the induced astigmatism. It has been suggested that changes in corneal astigmatism as a consequence of cataract surgery are related to the anatomical and biomechanical properties of the cornea[26] and the type of incision.[27] Similarly, induced irregular astigmatism has been reported as a consequence of trabeculectomy for glaucoma.[28] Furthermore, retinal detachment surgery involving scleral buckling has been also related to significant changes in astigmatism,[29-30] which appear to be related to indentation of the sclera by the buckle resulting in either regular or irregular astigmatism.[29,30] Summing up, extensive information on surgery-induced corneal changes and how these alter astigmatism is already available in the literature.[22-30] However, the potential effect of surgery-induced changes in the sclera on surgery-induced astigmatism is yet to be determined. The presented methodology, that considers the cornea and the sclera as a whole, might help to better understand the ocular surgery included astigmatism and help to prevent it.

Note that the results presented need to be put in perspective by considering the instrument's measurement noise. The ESP corneo-scleral topographer used for data acquisition has been demonstrated to provide an RMS error of < 10 μm for the central 8 mm area of a calibrated artificial surface and < 40 μm for an extended measurement area of 16 mm.[19] Our analysis was performed for an area with a diameter of 0 – 15 mm. It is worth noting that the internal measurement error of the device is minor in comparison to the values reported.

The small sample size could be seen as a limitation of the study, besides the prior power analysis, confirmed by the post hoc test. It is important to clarify that we decided not to include participants with myopia since it has been previously suggested that there exist differences in scleral properties between myopes and other refractive groups,[17,18,31] which limited the number of participants that could be included. Moreover, all astigmatic participants were young with-the-rule astigmatism. Even though there is no evidence to expect a different pattern than the results here shown, it would be of interest to expand this work including against-the-rule participants.

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REFERENCES

1. Wolfram C, Hohn R, Kottler U, et al. Prevalence of refractive errors in the european adult population: The gutenberg health study (GHS). Br J Ophthalmol 2014;98:857-61.
2. Applegate RA, Sarver EJ, Khemsara V. Are all aberrations equal? J Refract Surg 2002;18:S556-62.
3. Grosvenor T. Etiology of astigmatism. Optometry Vision Sci 1978;55:214-8.
4. Read SA, Collins MJ, Carney LG. A review of astigmatism and its possible genesis. Clin Exp Optom 2007;90:5-19.
5. Clementi M, Angi M, Forabosco P et al.. Inheritance of astigmatism: Evidence for a major autosomal dominant locus. Am J Hum Genet 1998;63:825-30.
6. Hammond CJ, Snieder H, Gilbert CE et al.. Genes and environment in refractive error: The twin eye study. Invest Ophthalmol Vis Sci 2001;42:1232-6.
7. Lee KE, Klein BE, Klein R et al. Aggregation of refractive error and 5-year changes in refractive error among families in the beaver dam eye study. Arch Ophthalmol 2001;119:1679-85.
8. Teikari JM, O'donnell JJ. Astigmatism in 72 twin pairs. Cornea 1989;8:263-6.
9. Wilson G, Bell C, Chotai S. The effect of lifting the lids on corneal astigmatism. Am J Optom Physiol Opt 1982;59:670-4.
10. Vihlen F, Wilson G. The relation between eyelid tension, corneal toricity, and age. Invest Ophthalmol Vis Sci 1983;24:1367-73.
11. Read SA, Collins MJ, Carney LG. The influence of eyelid morphology on normal corneal shape. Invest Ophthalmol Vis Sci 2007;48:112-9.

12 . Howland HC, Sayles N. Photokeratometric and photorefractive measurements of astigmatism in infants and young children. Vision Res 1985;25:73-81.

13. Kwitko S, Sawusch MR, McDonnell PJ et al. Effect of extraocular muscle surgery on corneal topography. Arch Ophthalmol 1991;109:873-8.

14. Bagheri A, Farahi A, Guyton DL. Astigmatism induced by simultaneous recession of both horizontal rectus muscles. J AAPOS 2003;7:42-6.

15. LaMattina KC, DeBenedictis CN. Refractive changes after strabismus surgery. Curr Opin Ophthalmol 2016;27:393-7.

16. Consejo A, Iskander DR, Rozema JJ. Mean shape of the human sclera. Acta Ophthalmol 2017;95(S259).

17. McBrien NA, Gentle A. Role of the sclera in the development and pathological complications of myopia. Prog Retin Eye Res 2003;22:307-38.

18. McMonnies CW. An examination of the relation between intraocular pressure, fundal stretching and myopic pathology. Clin Exp Optom 2016;99:113-9.

19. Iskander DR, Wachel P, Simpson PN. Principles of operation, accuracy and precision of an eye surface profiler. Ophthal Physiol Opt 2016;36:266-78.

20. Consejo A, Llorens-Quintana C, Radhakrishnan H et al. Mean shape of the human limbus. J Cataract Refr Surg 2017;43:667-72.

21. Keeley F, Morin J, Vesely S. Characterization of collagen from normal human sclera. Exp Eye Res 1984;39:533-42.

22. Olsen T, Dam-Johansen M, Bek T et al. Corneal versus scleral tunnel incision in cataract surgery: A randomized study. J Cataract Refr Surg 1997;23:337-41.

23. Tejedor J, Murube J. Choosing the location of corneal incision based on preexisting astigmatism in phacoemulsification. Am J Ophthalmol 2005;139:767-76.

24. Hayashi K, Hayashi H, Nakao F et al. The correlation between incision size and corneal shape changes in sutureless cataract surgery. Ophthalmology 1995;102:550-6.

25. Kohnen T, Dick B, Jacobi KW. Comparison of the induced astigmatism after temporal clear corneal tunnel incisions of different sizes. J Cataract Refr Surg 1995;21:417-24.

26. Meek KM, Newton RH. Organization of collagen fibrils in the corneal stroma in relation to mechanical properties and surgical practice. J Refract Surg 1999;15:695-9.

27. Kohnen S, Neuber R, Kohnen T. Effect of temporal and nasal unsutured limbal tunnel incisions on induced astigmatism after phacoemulsification. J Cataract Refr Surg 2002;28:821-5.

28. Hayashi K, Hayashi H, Oshika T et al. Fourier analysis of irregular astigmatism after trabeculectomy. Ophthalmic Surg Lasers 2000;31:94-9.

29. Tomidokoro A, Oshika T, Kojima T. Corneal astigmatism after scleral buckling surgery assessed by fourier analysis of videokeratography data. Cornea 1998;17:517-21.

30. Örnek K, Yalçindag FN, Kanpolat A et al. Corneal topographic changes after retinal detachment surgery. Cornea 2002;21:803-6.

31. Consejo A, Radhakrishnan H, Iskander DR. Scleral changes with accommodation. Ophthal Physiol Opt 2017;37:263-74.