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ORIGINAL ARTICLE

Astigmatism Impact on Visual Performance: Meridional and Adaptational Effects

Maria Vinas*, Pablo de Gracia†, Carlos Dorronsoro‡, Lucie Sawides‡, Gildas Marin‡, Martha Hernández‡, and Susana Marcos‡

ABSTRACT

Purpose. Astigmatic subjects are adapted to their astigmatism and perceptually recalibrate upon its correction. However, the extent to which prior adaptation to astigmatism affects visual performance, whether this effect is axis dependent, and the time scale of potential changes in visual performance after astigmatism correction are not known. Moreover, the effect of possible positive interactions of aberrations (astigmatism and coma) might be altered after recalibration to correction of astigmatism.

Methods. Visual acuity (VA) was measured in 25 subjects (astigmats and non-astigmats, corrected and uncorrected) under induction of astigmatism and combinations of astigmatism and coma while controlling subject aberrations. Astigmatism (1.00 diopter) was induced at three different orientations, the natural axis, the perpendicular orientation, and 45 degrees for astigmats and at 0, 90, and 45 degrees for non-astigmats. Experiments were also performed, adding coma (0.41 μm at a relative angle of 45 degrees) to the same mentioned astigmatism. Fourteen different conditions were measured using an 8-Alternative Forced Choice procedure with Tumbling E letters and a QUEST algorithm. Longitudinal measurements were performed up to 6 months. Uncorrected astigmats were provided with proper astigmatic correction after the first session.

Results. In non-astigmats, inducing astigmatism at 90 degrees, produced a statistically lower reduction in VA than at 0 or 45 degrees, whereas in astigmats, the lower decrease in VA occurred for astigmatism induced at the natural axis. Six months of astigmatic correction did not reduce the insensitivity to astigmatic induction along the natural axis. Differences after orientation of astigmatism were also found when adding coma to astigmatism.

Conclusions. The impact of astigmatism on VA is greatly dependent on the orientation of the induced astigmatism, even in non-astigmats. Previous experience to astigmatism plays a significant role on VA, with a strong bias toward the natural axis. In contrast to perceived isotropy, the correction of astigmatism does not shift the bias in VA from the natural axis of astigmatism. (Optom Vis Sci 2013;90:1430–1442)

Key Words: astigmatism, visual performance, astigmatism correction, adaptive optics, astigmatism and coma, long-term adaptation

Astigmatism is one of the most frequent aberrations in the human eye and has a high impact on vision when uncorrected, even when relatively low amounts of astigmatism are present.[1–4] Astigmatism (as well as other high-order aberrations [HOAs] like coma) is increased in certain pathologies (i.e., keratoconus),[5] induced in several surgical procedures (i.e., keratoplasty, cataract surgery),[6] or with ophthalmic lenses.[7] In clinical practice, astigmatic correction is often provided gradually, as it is assumed that subjects are adapted to the distortion produced by their natural astigmatism. However, the extent to which astigmatic subjects are adapted to their own astigmatism and recalibrate upon correction of their astigmatism has only been recently investigated,[8] and the extent to what these perceptual changes affect visual performance is not well known.[9]

There is increasing evidence that subjects are adapted to the blur produced by their own HOA (magnitude and, to some extent, also orientation). Artal et al.[10] showed that subjects tended to perceive as better quality test images seen through their natural aberrations than those seen through an artificial pattern of aberrations that generates an identical but rotated point spread function. There is also strong evidence that spatial vision is calibrated for the specific

*BOptom, MSc
†BOptom, MSc, FAAO
‡PhD
Instituto de Óptica, Consejo Superior de Investigaciones Científicas, Madrid, Spain (MV, PdG, CD, LS, SM); and Essilor International, Research and Development, Vision Science Department, Saint-Maur, France (GM, MH).

blur levels present in each individual's retinal image.[11] Moreover, the perceived neutral point in habitually non-corrected astigmats is shifted toward oriented images (generally toward their own axis of astigmatism, particularly in myopic astigmats) and, very interestingly, it shifts toward more isotropic images after correction of their astigmatism partly after 2 hours of astigmatic correction wear and fully after 1 week.[8] Strong aftereffects were also found after brief periods of adaptation to simulated images blurred with horizontal/vertical astigmatism (while keeping the blur strength constant), indicating that adaptation can be selective to the orientation of astigmatism.[12] Direct tests of the best-perceived focus therefore indicate that blur judgments are largely influenced by the subject's previous experience and that changes in the environment can rapidly result in a shift of perceived blur (or the orientation of this blur).[8,12]

Furthermore, it is well known that uncorrected astigmatism may limit neural sensitivity.[1,2,13,14] Numerous studies have shown that large amounts of astigmatism left uncorrected in childhood may lead to meridional visual deficits, so-called meridional amblyopia, although those are not found in all visual tasks.[15,16] Also, longer exposures to spherical blur have been reported to induce changes in visual acuity (VA).[17–19] Adaptation to blur has also been suggested to produce improvements in visual performance; however, it is likely that those changes, usually occurring after a longer term exposure to the adapting stimulus, also entail some form of perceptual learning.[20] Perceptual learning is often described as a training for specific visual tasks leading to long-term improvement in performing the task.[21] In that sense, Fogt[22] studied the directional aftereffects associated with the prismatic effects of spectacle lenses after being trained to point accurately through a spectacle lens. Subjects were made myopic using a contact lens and then the myopia was corrected with a spectacle lens, whereas pointing behavior was used to assess directional localization. Fogt[22] found that the ability of the observer to switch between two different optical corrections (spectacle and contact lenses), that induced different visual distortions, was correlated with the presence of aftereffect in localizing objects (a shift in the perceived direction occurring after removal of the optical device used to induce adaptation): those who learned did not show an aftereffect, whereas those who did not learn did show an aftereffect. Yehezkel et al.[20] suggested that, after a long experience, adaptation is transferred to a long-term memory that can be instantly engaged when blur is reapplied or disengaged when blur is removed, thus leaving no aftereffects in shape perception. This pointed out to the possibility of storing multiple transformations of the visual world and applying them when the need arises.

Some studies have reported relatively fast improvements in visual performance on adaptation of blur. Mon-Williams et al.[17] reported an increase in VA in subjects after exposure to spherical blur. Pesudovs[23] observed that patients with increased aberrations after refractive surgery progressively improved VA in the course of 10 weeks after the procedure. In addition, the fact that keratoconic patients show a higher VA than normal subjects with simulated identical aberrations[19] suggests that visual performance is possibly improved after prolonged exposure to optical degradation.[17,19,24] Similar effects have been observed on astigmatism induction. Ohlendorf et al.[25] reported an increase of VA in normal subjects viewing dynamic astigmatic images (either simulated or through + 3.00-diopter [D] cylindrical lenses) after 10 minutes of adaptation, with a significant meridional bias.

A previous study suggested that habitually non-corrected astigmats were adapted to their astigmatism because their measured VA was less impaired by the induction of astigmatism than in nonastigmatic subjects with the same amount of induced astigmatism.[9] The fact that subjects with identical optical properties exhibit very different relative responses is suggestive of adaptation/perceptual learning effects to astigmatic blur in particular. However, in the previous study, astigmatism was systematically induced at 45 degrees (blur in the oblique meridian, OBL), regardless of the orientation of the natural astigmatism, and the sample included hyperopic subjects (who may shift their plane of focus along the Sturm interval by accommodating). The increased performance on induction of astigmatism (with respect to emmetropes or corrected astigmats) could then be the result of adaptation to overall blur. Moreover, the orientation of the astigmatic axis may play an essential role in visual performance in astigmats. Wolffsohn et al.[2] showed that uncorrected astigmatic blur at 45 or at 180 degrees (blur in the horizontal meridian) resulted in worse distance and near VA, as well as worse subjective-rated clarity, than astigmatic blur at 90 degrees (blur in the vertical meridian). Similar trends have been shown in visual performance, where oblique astigmatism has a more deleterious effect on visual performance than with-the-rule (WTR) or against-the-rule (ATR) astigmatism probably because of a higher visual deprivation associated with uncorrected astigmatism.[26]

In the current study, we have tested the effect of prior adaptation to astigmatism in subjects with different refractive (astigmats and non-astigmats) and corrective (habitually corrected and habitually non-corrected astigmats) profile in visual performance. In particular, we have measured the impact of astigmatism induction on VA at different axes of astigmatism, including the natural axis of astigmatism, while controlling the natural aberrations of the eye in each subject. Furthermore, to test the effect of astigmatic correction on visual performance in the presence of astigmatism, measurements were performed in astigmatic patients before correction of their astigmatism and at various times, up to 6 months, after astigmatic correction wear. We also tested the effect of interactions between astigmatism and coma on VA because previous reports showed a lack of agreement between optical predictions and visual performance in the presence of a combination of those aberrations (which is axis dependent)[27] in non-corrected astigmatic patients, which suggest a role of adaptation to prior astigmatism.[9]

METHODS

Subjects

The sample consisted of 25 subjects (ages ranging from 23 to 51 years, 31.96 ± 8.15 years). Subjects were selected *a priori* and followed an exhaustive optometric evaluation at the Faculty of Optometry Clinic of the University Complutense de Madrid, in which they were classified according to their natural astigmatism and whether this was habitually corrected or not. The subjects were classified in three groups: G1 (control group of subjects with no clinical astigmatism; n = 9); G2 (astigmatic subjects, habitually corrected, wearing an astigmatic correction since childhood; n = 7); G3 (astigmatic subjects, habitually non-corrected, n = 9).

The inclusion criterion for the different groups was, for G1, emmetropic subjects with astigmatism lower than 0.25 D, and

for G2 and G3, subjects with myopic astigmatism ≥0.75 D.[28] Only myopic astigmats were included in the study because non-corrected hyperopic astigmats could shift their best focus by means of accommodation and, therefore, may experience images blurred along different orientations throughout the Sturm interval for distance vision,[29] which might interfere in the study of the astigmatism orientation effect on visual performance. Some of the subjects also participated in a previous study in which the perceived neutral point was measured from a series of images degraded with astigmatism and defocus.[8] Table 1 shows the refractive and corrective profile of all subjects of the study, which were measured using standard clinical optometric procedures.

After an initial test, all subjects in G3 were provided with proper astigmatic spectacle correction of their natural astigmatism (in the Faculty of Optometry Clinic of the University Complutense de Madrid) and were asked to wear them continuously for 6 months. Tests were performed only on one (naked) eye per subject (less myopic eye in G1 and less myopic eye with ≥0.75 D of astigmatism in G2 and G3).

All participants were acquainted with the nature and possible consequences of the study and provided written informed consent. All protocols met the tenets of the Declaration of Helsinki and had been previously approved by the Spanish National Research Council Bioethical Committee. Optometric measurements of VA (2000 Series Revised ETDRS Translucent Chart 1, Chart 2; catalogs 2121 and 2122; Precision Vision) were performed in habitually non-corrected astigmats to measure the improvement in VA with astigmatic correction spectacles with respect to noncorrection.

Experimental Setup

Measurements were conducted in a custom-developed Adaptive Optics (AO) system, described in detail in previous publications,[30–32] which was used to measure and correct the aberrations of the subject, as well as to induce the different patterns of aberrations, astigmatism, and coma. The main components of the system are a Hartmann-Shack wavefront sensor (32 × 32 microlenses, 3.6 mm effective diameter; HASO 32 OEM, Imagine Eyes, France), a superluminescent diode (wavelength, 827 nm for wavefront sensing), an electromagnetic deformable mirror (52 actuators, a 15-mm effective diameter and a 50-μm stroke; MIRAO, Imagine Eyes), a motorized Badal system, a natural pupil monitoring system, and a stimulus display. The state of the mirror that compensates for

TABLE 1.
Subjects' profile

ID	Measured eye	Refraction			Type of astigmatism	Blur axis, degrees	Age, y
		Sph	Cyl	Axis			
G1_A	Right	0.50	—	—	—	—	29
G1_B	Right	0.00	—	—	—	—	33
G1_C	Right	0.00	—	—	—	—	31
G1_D	Right	0.00	—	—	—	—	30
G1_E	Right	−0.25	−0.25	80	ATR	170	30
G1_F	Right	0.25	−0.25	90	ATR	90	34
G1_G	Right	0.00	—	—	—	—	23
G1_H	Right	0.00	—	—	—	—	32
G1_I	Right	0.00	—	—	—	—	50
		Average Sph: 0.06 ± 0.10			Average Cyl: −0.06 ± 0.06		
G2_A	Right	−3.50	−1.00	10	WTR	100	33
G2_B	Right	−5.25	−1.25	105	ATR	15	27
G2_C	Right	−4.00	−1.00	75	ATR	165	34
G2_D	Right	−0.75	−1.25	90	ATR	0	30
G2_E	Right	−2.25	−0.75	90	ATR	0	51
G2_F	Left	−1.75	−1.00	170	WTR	80	31
G2_G	Left	0.25	−1.25	175	WTR	85	24
		Average Sph: −2.46 ± 1.92			Average Cyl: −1.07 ± 0.19		
G3_A	Right	−1.50	−0.75	10	WTR	100	27
G3_B	Left	0.00	−1.25	80	ATR	170	29
G3_C	Right	−0.75	−0.75	120	ATR	30	27
G3_D	Right	0.50	−0.75	170	WTR	170	27
G3_E	Left	−0.75	−0.75	175	WTR	85	48
G3_F	Left	−1.00	−0.75	90	ATR	0	45
G3_G	Right	0.00	−1.00	90	ATR	0	26
G3_H	Left	0.00	−1.25	175	WTR	85	23
G3_I	Right	0.00	−1.25	10	WTR	100	33
		Average Sph: −0.39 ± 0.64			Average Cyl: −0.94 ± 0.24		

Optometric subjective refractions (spherical error, cylinder, axis) and orientation of the retinal blur (most myopic meridian) on measured eye and ages. Averaged spherical error and natural astigmatism are shown for every group.

Cyl, cylinder; Sph, spherical error.

the aberrations of the subject was found in a closed-loop operation, and measurements of the subjects' aberrations throughout the test ensured proper correction. The same operation was used to generate and induce the different combinations of astigmatism and coma. Measurements were performed for 6-mm pupils (limited by an artificial pupil of 6 mm placed in a plane conjugate to the natural pupil). Visual stimuli were presented on a CRT monitor (Mitsubishi Diamond Pro 2070) through the Badal system and AO mirror correction. The stimulus display was controlled by the psychophysical platform ViSaGe (Cambridge Research System, UK). The average luminance (after losses in the system) was approximately 50 cd/m^2 in an otherwise dark environment.

Experimental Protocol

After dilation, the eye's pupil was aligned to the optical axis of the instrument, and the subject's head was stabilized using a dental impression on a bite bar. The subject's spherical refractive error was corrected with a Badal system. All the measurements were performed after the pupils of the subjects were dilated (by tropicamide 1%; Alcon Cusi, Barcelona, Spain) to normalize the pupil size with an artificial pupil of 6 mm placed in a plane conjugate to the natural pupil. In addition, measurements were performed with the naked eye (without spectacles). Best subjective focus was selected by the subject him/herself using a remote control to move the motorized stage while viewing a Maltese cross as a fixation target.

Natural astigmatism and HOA were fully corrected and/or selectively induced (astigmatism and coma) with the deformable mirror. The mirror states were measured just before and after each VA measurement. The accuracy of the achieved aberrations (combination of mirror and eye) with respect to the attempted pattern (i.e., astigmatism at a given meridian) was tested before and after VA measurements (a maximum discrepancy of 0.10 μm in the astigmatism or coma terms was allowed). Further details on the mirror control and validations of the achieved mirror states can be found in previous publications.[8,9,27] In the current study, we set the orientation of induced astigmatism to the orientation of the retinal blur of the most myopic meridian caused by the native astigmatism of each subject, as obtained from the optometric data. Because all astigmatic subjects were myopic and measurements were performed for distance vision, we replicated the oriented blur of the focal line closer to the retina, the most myopic meridian, by inducing ±0.50 D of defocus. For example, when the most myopic meridian of the subject was at 0 degree, C^2_2 = 0.92 μm and C_2^{-2} = 0.00 μm were induced with the mirror and +0.50 D defocus with the Badal system, so that a horizontally blurred image on the subject's retina was achieved. In other words, the vertical meridian was in focus, and the horizontal meridian was made artificially myopic by 1.00 D. The AO mirror was used so that the subject was exposed to 1.00 D of astigmatism (at different orientations), regardless of the magnitude of the subject's natural astigmatism. The difference between the attempted and achieved astigmatism was small (<2.1% in G1 and <5.5% in astigmatic groups).

Astigmatism and HOA were measured and corrected in a closed-loop AO operation. The subject was then asked to adjust the Badal system position to obtain again the best subjective focus for the AO correction condition. The state of the mirror that achieved the correction was saved and applied during the measurements. Visual acuity measurements were performed under full static AO-corrected aberrations and best spherical refraction error correction. The steps of an experimental session were, sequentially, (1) focus setting, (2) measurement of ocular aberrations with the Hartmann-Shack sensor, (3) closed-loop for natural aberration correction, (4) set of mirror status for the different conditions (aberration correction + specific astigmatism/coma combination), (5) measurement of ocular aberrations, (6) measurement of VA, (7) measurement of eye + mirror aberrations. The sequence was repeated for each condition tested. The order in which the different conditions were tested was randomized. A training trial, under induced astigmatism, was performed in the first session to familiarize the subject with the procedure.

Measurements were performed in four different sessions for all groups: first day (S0A), 1 week (S1), 1 month (S2), and 6 months after (S3). An additional measurement session was performed for the habitually non-corrected astigmats (G3) after 2 hours of spectacle correction wearing, provided right after the initial session.

Tested Conditions

A total of 14 different conditions were tested, summarized in Fig. 1. First, as a baseline, VA measurements with and without AO correction were performed (conditions 1 and 2, respectively). Then VA was measured under induction of 1.00 D (0.92 μm for 6-mm pupil size) of astigmatism at three different orientations with (conditions 3, 4, and 5) and without (conditions 6, 7, and 8) correction of HOAs. For nonastigmatic subjects (G1), the orientations tested were 0 degree (horizontal retinal blur) (no. 3), 90 degrees (vertical retinal blur) (no. 4), and 45 degrees (oblique retinal blur) (no. 5). For astigmatic subjects (G2 and G3), the orientations tested were the natural axis of astigmatism (i.e., axis of retinal blur of the most myopic meridian caused by the native astigmatism, according to the optometric readings) to replicate the astigmatic orientation of retinal blur of the most myopic meridian (no. 3), the perpendicular orientation (no. 4), and at 45 degrees fixed (oblique retinal blur) (no. 5). The oblique astigmatism (45 degrees) was used for comparison across groups and with previous work where only astigmatism induced at 45 degrees was tested.[9]

Furthermore, the influence of prior adaptation to astigmatism on potential interactive effects between astigmatism and coma was tested following previous work by De Gracia et al.[27] Optical simulations had shown that optical interactions between astigmatism and coma could result in an improvement in optical quality: adding amounts of coma between 0.15 and 0.35 μm to 0.5 μm could lead to an increase in peak Strehl ratio values in the absence of other HOAs.[27] However, psychophysical measurements showed that the visual improvement produced by adding coma to astigmatism seem to be highly dependent on the presence of natural astigmatism and whether this was habitually corrected or not.[9] For comparison with the previous study,[9] combinations of 1.00 D (0.92 μm for 6-mm pupil size) of astigmatism (at three orientations) and 0.41 μm of coma at a relative angle of 45 degrees were also tested because this relative angle between astigmatism and coma provided the best results in the previous study (conditions 9, 10, and 11). All tested conditions were also performed in the

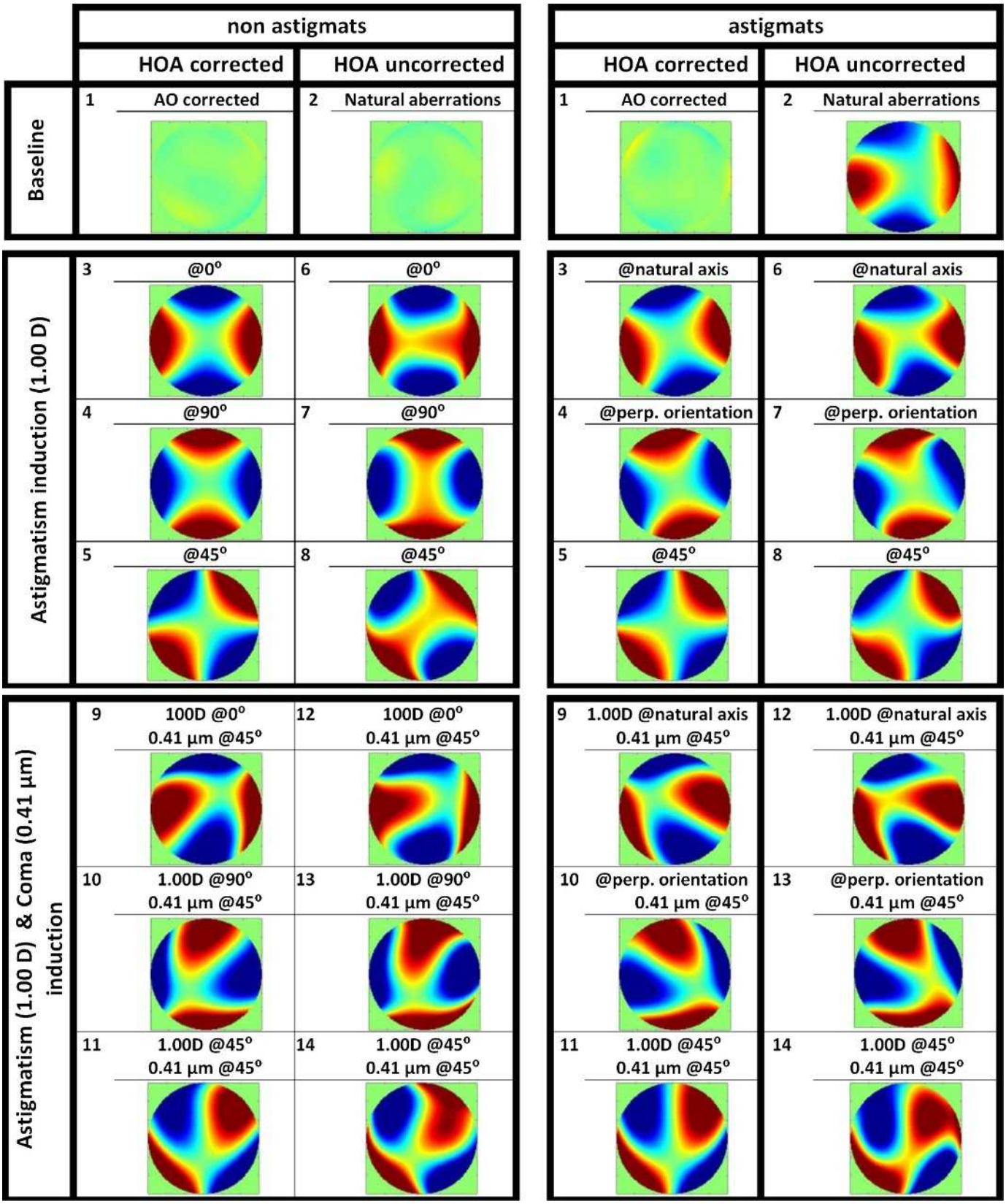

FIGURE 1.

Summary of tested conditions. Visual acuity was measured under 14 different conditions, numbered in the table for future reference. Baseline conditions (no. 1, 2): VA measurements with and without AO correction. To test whether the effect of astigmatism depends on prior adaptation to astigmatism with and without AO correction (no. 3 and no. 6). To test the axis dependency of astigmatism with and without AO correction (no. 3–8). To test the benefit of adding coma to astigmatism with and without AO correction (no. 9–14). Scale of wavefront maps is ±1.00 µm. A color version of this figure is available online at www.optvissci.com.

presence of the natural aberrations of the subjects (conditions 12, 13, and 14). In addition, measurements of VA with full correction of aberrations and astigmatism and under natural aberrations were also performed as control conditions.

In summary, to further explore the effect of prior adaptation to astigmatism on visual performance in the presence of astigmatism and possible interaction between astigmatism and coma, a total of 14 conditions were tested. All tests were performed monocularly, always in the same eye (less myopic eye in G1 and less myopic eye with ≥0.75 D of astigmatism in G2 and G3).

VA Measurement

Visual acuity was measured using an 8-Alternative Forced Choice[33] (8AFC) procedure with Tumbling E letters and a QUEST (Quick Estimation by Sequential Testing) algorithm programmed with the Psychtoolbox package[34] to calculate the sequence of the presented stimulus (letter size and orientation) in the test following the subject's response. Subjects had to determine the orientation of the letter E (eight orientations: pointing up, down, left, right, oblique up-right, oblique up-left, oblique down-right, oblique down-left) while aberrations were controlled with the deformable mirror: correction of natural aberrations and/or induction of astigmatism and combination of astigmatism and coma with and without natural aberrations correction following the different tested conditions. This eight-orientation test avoided potential convergence problems in the response of the subjects associated with the traditional 0-degree/90-degree preferential orientation test. The QUEST routine for each VA measurement consisted of 50 trials, each one presented for 0.5 seconds, where the threshold criterion was set to 75%. The threshold, VA measurement, was estimated as the average of the 10 last stimulus values. Visual acuity was expressed in terms of decimal acuity (logMAR = $-\log_{10}$ [decimal acuity]).[35]

Data Analysis

Wave aberrations were fitted by seventh-order Zernike polynomials, and OSA convention was used for ordering and normalization of Zernike coefficients. Visual acuity was expressed in decimal units and reported in terms of absolute and relative values. Relative values refer to the AO correction benefit (ratio VA [AO]/VA [no AO]), sensitivity to astigmatism induction (ratio VA [astigmatism + AO]/VA [AO]), and visual benefit of adding coma to induced astigmatism (ratio VA [astigmatism + coma + AO]/VA [astigmatism + AO]). Statistical analysis was performed with SPSS software (IBM) to test differences across groups, sessions, and conditions. More precisely, differences across groups and sessions were analyzed performing one-way analysis of variance (ANOVA) *post hoc* tests, whereas specific differences between relative data (ratios) were established by performing paired sample t-tests.

RESULTS

Subjects' Natural Aberrations

The subject's natural aberrations are shown in Fig. 2 in terms of average ocular root mean square (RMS) wavefront error (in micrometers) for HOAs and astigmatism (black bars), only astigmatism (oblique line bars), only coma (gray bars), and residual aberrations after AO correction of all natural aberrations (white bars) in each group. The RMS for HOA (RMS_{HOA}) ranged between 0.38 and 0.29 μm across subjects, with no statistical significant differences across groups.

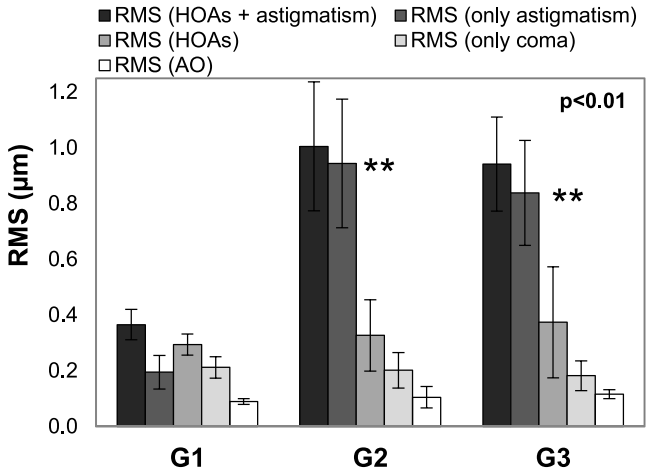

FIGURE 2.

Subject's aberrations. Root mean square wavefront error (excluding defocus) for HOAs and astigmatism (black bars), only astigmatism (oblique line bars), only HOAs (black dots), only coma (gray bars), and residual aberrations after AO correction (white bars), averaged across subjects, groups, and measurement sessions. Error bars indicate intersubject variability.** A significantly larger RMS for HOA + astigmatism for G2 and G3 than for G1.

On the contrary, the astigmatism contribution to the global amount of aberrations of the subjects differed across groups. As expected, $RMS_{HOA+ast}$ was significantly higher for G2 and G3 than for G1 (one-way ANOVA; $p = 0.006$), and astigmatism contribution (RMS_{ast}) to the total amount of aberrations ($RMS_{HOA+ast}$) was 47% for G1, 93% for G2, and 88% for G3. Natural aberrations of the subjects were properly AO corrected, and the achieved optical correction was similar across groups and measurement sessions (one-way ANOVA; $p > 0.05$).

The residual RMS after AO correction of HOA and astigmatism was similar for all groups and lower than 0.11 μm in all cases (mean higher order RMS for 6-mm pupils is ~0.3 μm on average across groups). The AO correction was similar throughout the study (6 months).

Visual Benefit of Adaptive Optics Correction

Visual acuity improved significantly with AO correction for all groups following previous results[30] and along all measurement sessions (paired samples t-test; $p < 0.05$). Fig. 3 shows VA with natural aberrations and after AO correction (HOA + astigmatism) (best subjective focus in each condition) in all individual subjects of the study (each panel showing data for each group) at day 0. As expected, VA under natural aberrations was higher for G1 than for G3 and, especially than G2, as a result of the higher amount of natural astigmatism of G2 versus G3 and G1 (on average, G2 has 0.11 μm of astigmatism more than G3 and 0.65 μm more than G1). Visual acuity on correction of aberrations (HOA and

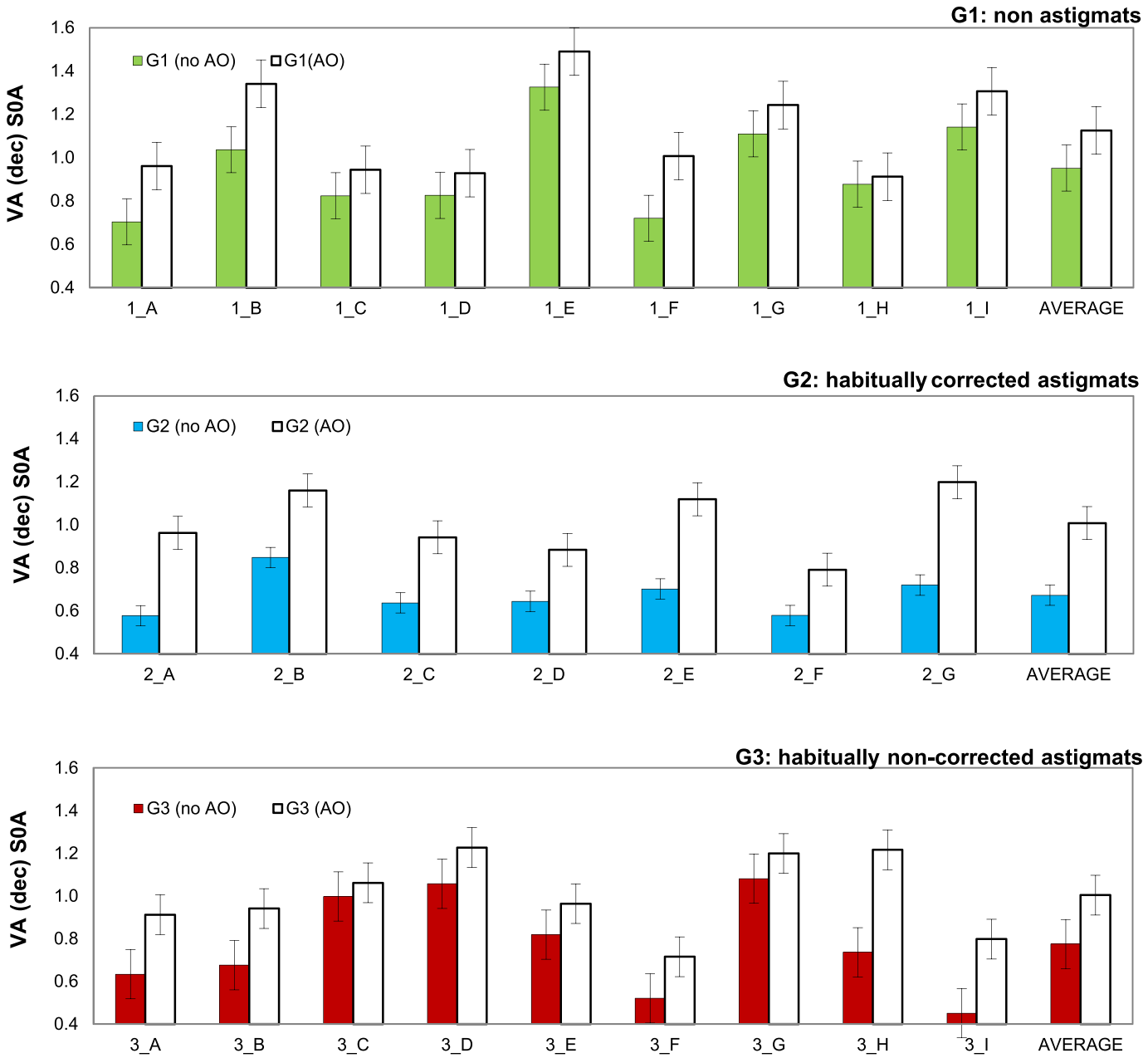

FIGURE 3.

Baseline VA measurements. Visual acuity for natural aberrations (colored bars) and AO-corrected aberrations (white bars) for the first session (S0A) for the three groups for all individual subjects (divided by group) and average. Error bars represent intrasubject measurement variability (SD). A color version of this figure is available online at www.optvissci.com.

astigmatism) was not statistically significantly different across groups (one-way ANOVA; $p = 0.395$).

Fig. 4 shows the visual benefit (ratio VA [AO]/VA [no AO]) for the three groups for the different sessions (first session, up to 6 months). The larger benefit of the AO correction in G2 (1.47, as opposed to 1.16 and 1.26 in G1 and G3, respectively, on average across subjects and sessions) is caused by the larger amount of astigmatism under natural conditions in this group (shown in Fig. 2). We found a slight but consistent trend toward VA improvement with time both for natural aberration and AO-corrected conditions in all groups. However, the AO correction benefit did not change significantly across sessions (one-way ANOVA; $p > 0.05$).

Visual Performance Under Astigmatism Induction at Different Angles

For G1, VA was measured after induction of 1.00 D (0.92 μm) of astigmatism at three different angles: 0 degree (horizontal retinal blur), 90 degrees (vertical retinal blur), and 45 degrees (oblique retinal blur. For G2 and G3, VA was measured after induction of 1.00 D of astigmatism at three different angles; axis of natural astigmatism, 90 degrees from the natural axis of astigmatism and 45 degrees. Fig. 5 shows VA averaged across subjects, tested at the different angles, as a function of session (left panels, A, C, and E, under full AO correction of aberrations,

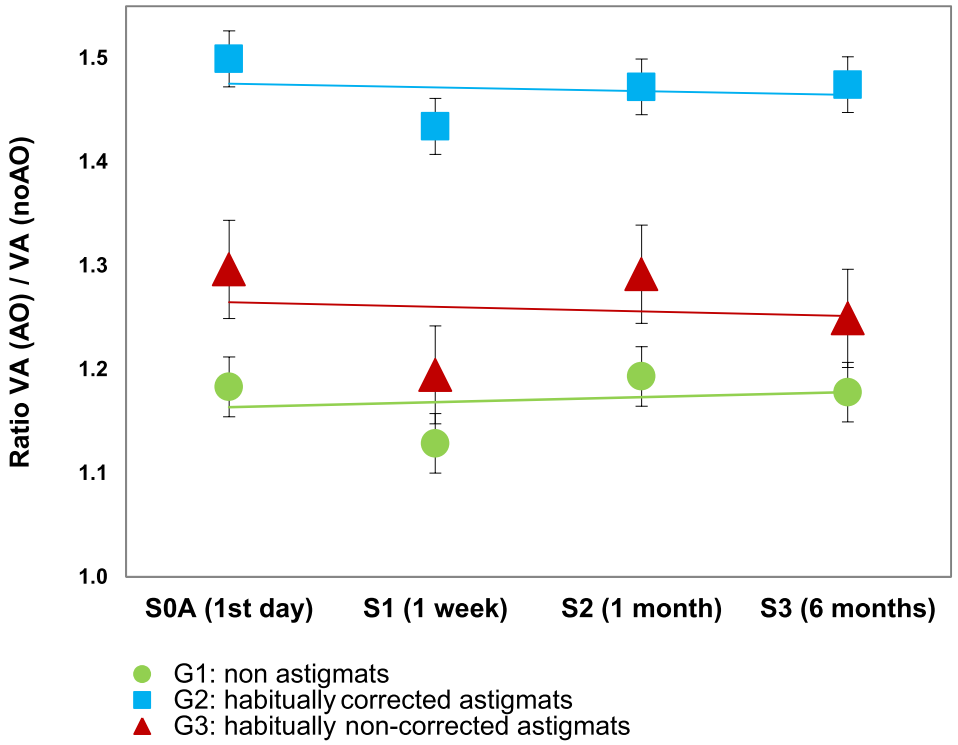

FIGURE 4.

Adaptive Optics correction benefit. Ratio VA (AO)/VA (no AO) as a function of session, averaged across subjects, for the three groups. Error bars represent intersubject variability (SD). G1 (non-astigmats) is represented with circles, G2 (habitually corrected astigmats) with squares, and G3 (habitually non-corrected astigmats) with triangles. Lines represent linear regression. A color version of this figure is available online at www.optvissci.com.

except for the induced astigmatism; and right panel, B, D, and F, under natural aberrations). The corresponding control conditions are shown for reference in black line: VA under full correction of aberrations and no astigmatism and VA under natural correction of aberrations and natural astigmatism.

Visual acuity becomes worse in the presence of induced astigmatism in all groups, conditions and sessions, although the magnitude of that decrease depended on the orientation of the induced astigmatism. For G1, inducing astigmatic blur at 90 degrees produced a statistically lower reduction in VA (29%) than when astigmatic blur was induced at 0 degree (40%) (paired samples t-test; $p = 0.004$) or 45 degrees (41%) (paired samples t-test; $t_8 = 3.465$; $p = 0.009$) (Fig. 5A). The same effect was found in the presence of natural HOA (Fig. 5B).

For G2, VA decreased significantly less when astigmatism was induced at its axis of natural astigmatism (with AO correction) than for other angles, that is, at a perpendicular axis (paired samples t-test; $t_6 = 2.896$; $p = 0.027$), and at 45 degrees (paired samples t-test; $p = 0.003$) (Fig. 5C). Visual acuity was only reduced by 23% when astigmatism was induced at the axis of natural astigmatism, in contrast to 36% for a perpendicular axis and 38% for 45 degrees. Without AO correction, differences across angles were not significant (Fig. 5D).

For G3, VA also decreased significantly less (by 28% in the first session and 16% in the last session) when astigmatism was induced at the axis of natural astigmatism (with AO correction; Fig. 5E) than for other angles, that is, 36% for the perpendicular axis (paired samples t-test; $p = 0.010$), and 31% for 45 degrees (paired samples t-test; $p = 0.034$). In fact, VA did not experience any reduction when astigmatism was induced at the axis of natural astigmatism in the presence of natural aberrations (Fig. 5F).

In the first session, the highest decrease in VA under induced astigmatism (using the best condition in each group, for comparison) was experienced by G3 subjects, followed by G2 and G1 (Fig. 5A, C, E). Visual acuity tended to become slightly better, but not significantly, across sessions for all groups, consistent with some training effect.[36] However, only G3 changed significantly after the 6 months of astigmatic correction wearing (paired samples t-test; $p = 0.001$). After 6 months of astigmatic correction wearing, G3 subjects were significantly less sensitive to the induction of astigmatism and reached VA under astigmatism induction values similar to those of non-astigmats (in fact, higher VA values) (Fig. 5, left panels).

We further analyzed this orientation dependence effect in terms of the relative decrease in VA on induction of astigmatism. Fig. 6 shows the relative sensitivity to induction of astigmatism, as the ratio VA (astigmatism + AO)/VA (AO), for the different orientations of astigmatism. Non-astigmats showed a significantly lower degradation in VA when astigmatism was induced at 90 degrees (vertical retinal blur) than at the other orientations (one-way ANOVA; $p = 0.024$). Conversely, subjects from either astigmatic group appeared significantly less sensitive to the induction of astigmatism at the subject's natural axis of astigmatism (Fig. 6A). For G2, the relative sensitivity to induction of astigmatism was significantly lower (one-way ANOVA; $p = 0.011$) when astigmatism was induced at the subject's natural axis of astigmatism than at the other orientations (Fig. 6B). G3 subjects showed, on the very first session, also a slightly lower visual degradation when

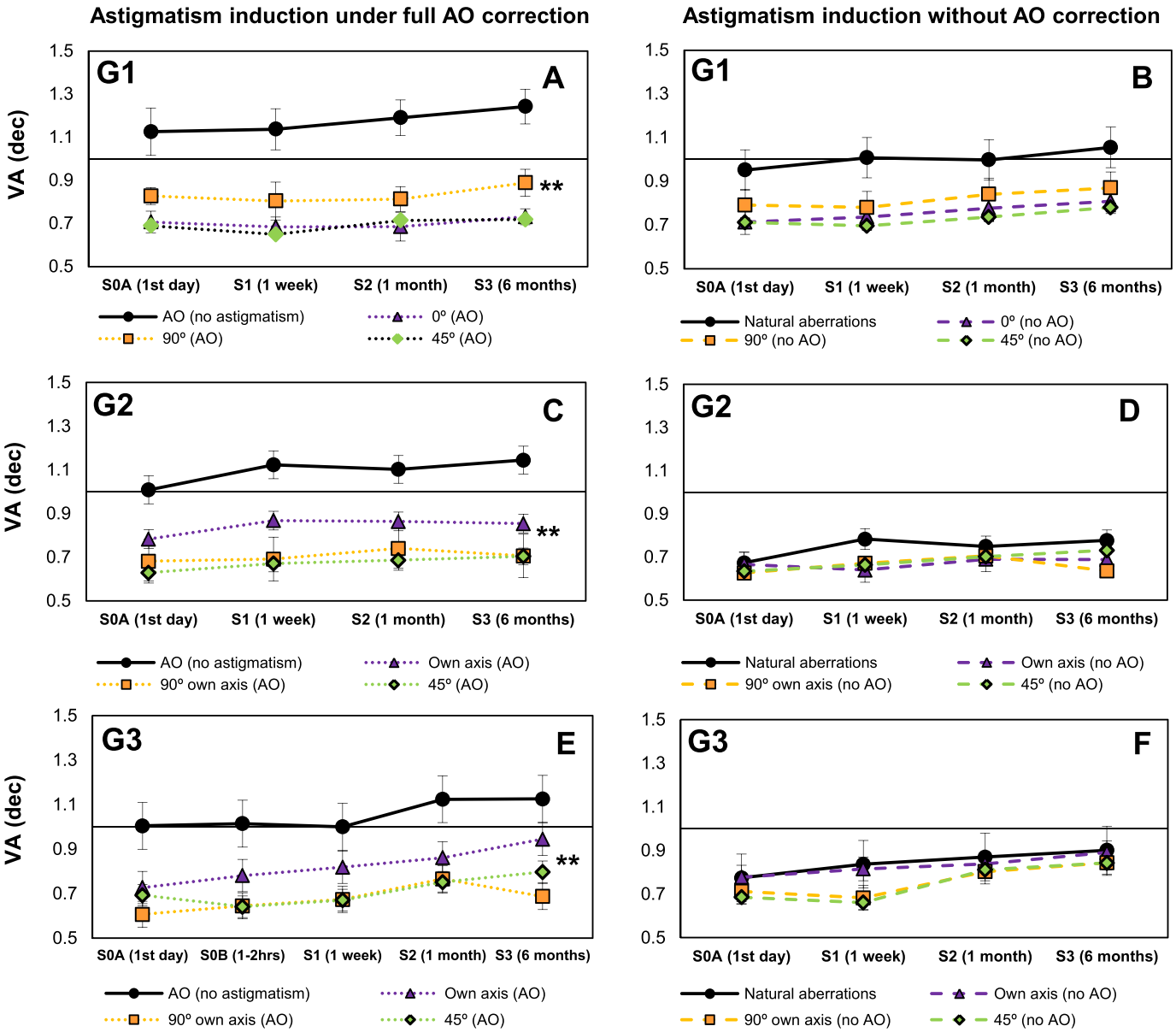

FIGURE 5.

Induction of astigmatism. Decimal VA under induced astigmatism at different angles, averaged across subjects in each group, for different sessions. Left panels (A, C, E) show data under full correction of HOA; right panels (B, D, F), data under natural aberrations. Top panels (A, B) are data for G1 (non-astigmats), middle panels (C, D) are data for G2, and lower panels (E, F) are data for G3. For G1, 1.00 D (0.92 μm) of astigmatism was induced at 0 degree (triangles), 90 degrees (squares), and 45 degrees (diamonds). For G2 and G3, 1.00 D of astigmatism was induced at their own axes of natural astigmatism (triangles), at perpendicular axes (squares), and at 45 degrees fixed (diamonds). Decimal VAs under full AO correction and under natural aberrations (including astigmatism) are shown for reference in the left and right panels, respectively (black line, circles).**Highly significant differences in G1 between VA at 90 degrees versus the others and in G2 between VA at the natural axis and the others. In G3,** indicates highly significant differences for data of S3 with respect to S0A for G3 when astigmatism is induced at the axis of natural astigmatism. In S3 indicates highly significant differences between VA at the natural axis and the others. Error bars represent intersubject variability (SD). A color version of this figure is available online at www.optvissci.com.

astigmatism was induced at its own axis of astigmatism than at the other orientations (Fig. 6C).

After 6 months of astigmatic correction wear of subjects in G3, the better performance under induction of astigmatism at its own axis in comparison with the other orientations (perpendicular orientation and 45 degrees) was statistically significant (one-way ANOVA; $p = 0.04$). In addition, VA changed significantly from the first day to 6 months after correction (paired samples t-test; $p < 0.01$) (Fig. 6D). Clinical measurements of VA, performed on G3 following standard clinical optometric procedures in the first and the last sessions showed that all G3 subjects (except for G3_B) improved VA after wearing astigmatic correction for 6 months (averaged 19.3% improvement in clinical decimal VA).

Benefit of Adding Coma to Induced Astigmatism

Optical simulations showed that certain combinations of astigmatism and coma improve optical performance with respect to astigmatism alone.[27] However, in a previous study, we had

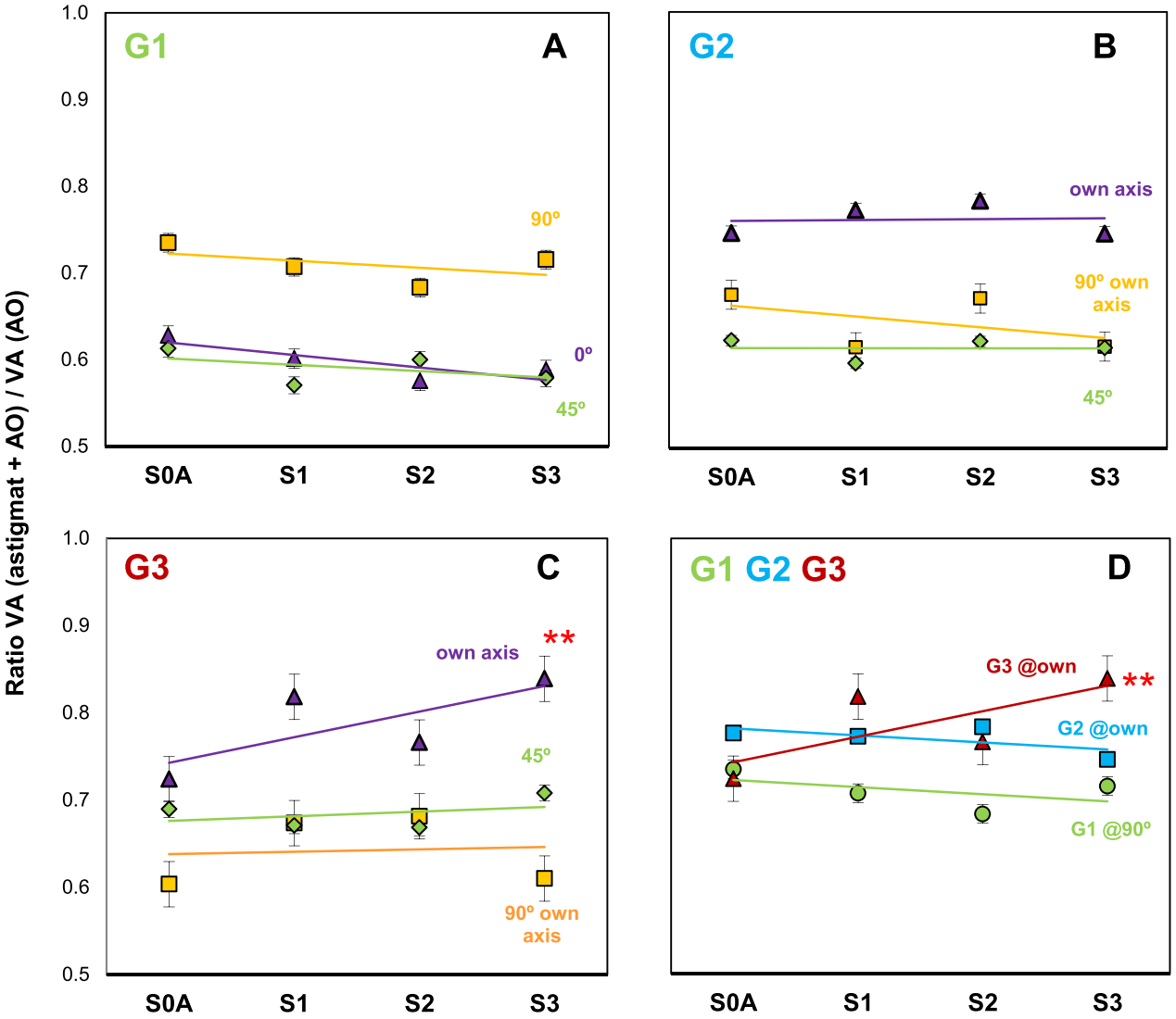

FIGURE 6.

Sensitivity to astigmatism induction. Ratio VA (Astigmatism AO)/VA (AO), averaged across subjects in each group, for the different sessions for G1 (non-astigmats, A), G2 (habitually corrected astigmats, B), and G3 (habitually non-corrected astigmatism, C). For G1, 1.00 D (0.92 μm) of astigmatism was induced at 0 degree (triangles), 90 degrees (squares), and 45 degrees (diamonds). For G2 and G3, 1.00 D of astigmatism was induced at their own axes of natural astigmatism (triangles), at perpendicular axes (squares), and at 45 degrees fixed (diamonds). Panel D shows a summary of the best condition for each group (G1 at 90 degrees, circles; G2, squares; and G3, triangles, at own axis). Data are for full AO correction of natural aberrations.**Highly significant differences for data of S3 with respect to S0A for G3 when astigmatism is induced at the axis of natural astigmatism. Error bars represent intersubject variability (SD). A color version of this figure is available online at www.optvissci.com.

shown that the predicted improvement occurred in non-astigmats but failed in habitually non-corrected astigmats, likely as a result of the subject's adaptation to astigmatism.[9] In the referred study, astigmatism was induced systematically at 45 degrees, and the optimal relative angle referred to this orientation.[9] Fig. 7 shows the results from the current study of the relative change in VA when adding coma to astigmatism with respect to VA with astigmatism alone for different orientations of induced astigmatism (coma at a fixed relative angle of 45 degrees with respect to astigmatism).

For G1, VA increased significantly when coma was added to astigmatism in comparison with induced astigmatism alone. However, the orientation of the induced astigmatism played an important role. Visual acuity increased significantly (paired samples t-test; $p < 0.01$) for combined coma and astigmatism when astigmatism was induced at 0 degree (horizontal retinal blur) and increased slightly at 45 degrees. However, VA decreased significantly (paired samples t-test; $p = 0.02$) when astigmatism was induced at 90 degrees (vertical retinal blur). As shown in Fig. 7 (left panel), in G1, the visual benefit of adding coma to astigmatism over astigmatism alone was therefore statistically different (one-way ANOVA; $p < 0.01$) when coma was added to astigmatism at 0 degree (horizontal retinal blur) or at 45 degrees (oblique retinal blur) than when added at 90 degrees (vertical retinal blur).

For astigmatic groups, as found for VA in the presence of astigmatism alone, the effect of combined astigmatism and coma in VA was greatly influenced by the prior astigmatism and its orientation. For G2, VA improved slightly when coma was added to astigmatism at 45 degrees or at the perpendicular orientation

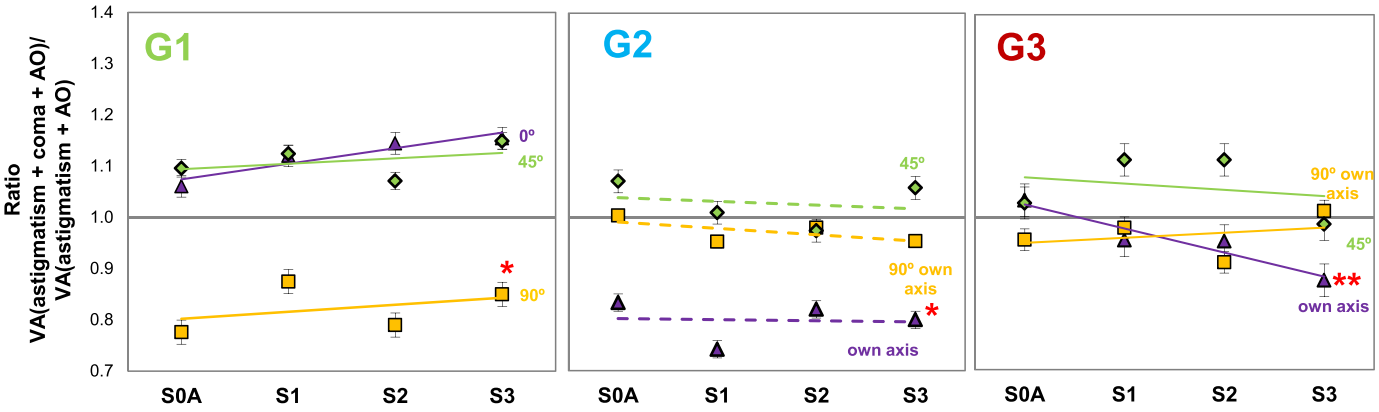

FIGURE 7.

Visual degradation under astigmatism and coma induction versus astigmatism alone. Ratio VA (astigmatism + coma + AO)/VA (astigmatism + AO) for the three groups (G1, left panel; G2, middle panel; G3, right panel) at the three tested conditions (G1, 0 degree/G2, G3 own axis: triangles; G1, 90 degrees/G2, G3, 90 degrees own axis: squares; G1, G2, G3, 45 degrees: diamonds) and 0.41 μm of coma at a relative angle of 45 degrees with full AO correction. *Significant differences for data of 90 degrees with respect to 0 and 45 degrees for G1, and data of axis of natural astigmatism with respect to perpendicular orientation and 45 degrees for G2.**Highly significant differences for data of S3 with respect to S0A for G3 when astigmatism is induced at the axis of natural astigmatism. Error bars represent intersubject variability (SD). A color version of this figure is available online at www.optvissci.com.

but decreased significantly when coma was combined with astigmatism induced at the natural axis (one-way ANOVA; $p = 0.02$) (Fig. 7, central panel). For G3, before the correction of astigmatism, adding coma to astigmatism did not result in an improvement of VA regardless of the axis of the induced astigmatism. However, astigmatic correction wearing produced a statistically significant progressive decrease in performance when coma was combined with astigmatism at the natural axis of astigmatism. After 6 months of astigmatism correction wear, VA under a combination of astigmatism (at the natural axis) and coma was statistically worse than astigmatism alone (paired samples t-test; $p = 0.012$), similarly to what occurs in G2.

DISCUSSION

We studied the impact of astigmatism induction (at different axes) in non-astigmats and astigmats and found that the visual degradation produced by astigmatism was greatly dependent on the axis of the induced astigmatism.

As expected from previous studies,[9] induction of astigmatism was more deleterious to vision in non-astigmats (compared with non-corrected astigmats and even habitually corrected astigmats) (Fig. 4, right panel). Furthermore, even in non-astigmats (and for full correction of HOA), significant differences were found in VA for astigmatism induced at different orientations but otherwise similar optical degradation (with astigmatism induced at 90 degrees degrading vision less than at other orientations), indicating a neural basis for the differences. Previous studies differ in their conclusions on the impact of the angle of induced astigmatism on vision, although most reports show that letter target acuity varies with the angle of induced astigmatism.[37] Miller et al.[4] found that subjects tended to be less dissatisfied with induced astigmatism of +0.50 D × 180 degrees (vertical retinal blur, following their notation) than with the same astigmatism induced at 90 degrees (horizontal retinal blur) or 45 degrees (oblique retinal blur), in agreement with our results. Moreover, Atchison et al.[38] showed, for high-contrast letter acuity charts, that the blur limits for induced crossed-cylinder astigmatism were 10% lower than for induced defocus, with considerable meridional influences, with astigmatism at 0 degree (vertical retinal blur in their notation), showing approximately 30% larger limits than those at 90 degrees (horizontal retinal blur). In subsequent work, they observed that the larger spreading in the horizontal direction than in the vertical spacing produced by horizontal retinal blur had a greater impact on text legibility than other orientations.[39] Also, Schwendeman et al.[40] found that added positive cylinders reduced VA with increasing effect for the cylinder axes 180, 90, and 45 degrees.[41] In contrast, Remon et al.[42] concluded little effect of the axis of a given astigmatism on VA, although they actually found that that, for some eye charts, VA was best for cylinder axis induced at 90 degrees than at other axes).

For astigmatic subjects, prior experience to astigmatism definitely has an impact on visual performance in the presence of astigmatism. Our results are consistent to a previous study[9] of the effect of induction of astigmatism (and combined astigmatism and coma) on visual performance, in a different population of non-astigmats, habitually corrected astigmats and habitually non-corrected astigmats (which included hyperopic astigmats). Although the previous study only considered induction of astigmatism at 45 degrees, we have investigated and found important meridional differences. In the current study, for both astigmatic groups, G2 and G3, the reduction of VA under induced astigmatism was lower than for non-astigmats (G1), very significantly when astigmatism was induced along the axis of their natural astigmatism. This is indicative of a persistent adaptation to astigmatism, even after its correction (in subjects of G2 and after correction in subjects of G3), which allows subjects who had a prior exposure to astigmatism to function superiorly with astigmatism induced at their natural axis of astigmatism, even if their astigmatism is normally corrected. This orientation preference tended to disappear (for G2 and G3) when HOAs were uncorrected (Fig. 5B, D, F).

A previous study on the influence of astigmatism (and its correction) on perceptual judgment of oriented blur showed that habitually corrected astigmats still tended to identify as isotropic astigmatic images along their axis of astigmatism.[8] Non-corrected astigmats also showed significant shifts of the perceived neutral point away from isotropy before correction, which shifted toward isotropy immediately after correction of astigmatism. Those rapid aftereffects are not paralleled by a change in the sensitivity to astigmatism on visual performance[18] likely because changes in visual performance require forms of learning and a prolonged exposure to the adapting stimulus. In fact, our results seem to be consistent with the suggested capability of the subject of storing multiple stages of adaptation[20] because corrected astigmats (G2) still appear quite insensitive to astigmatism induction[43,44] (along their axis of astigmatism) and so do as well previously uncorrected astigmats (G3) after correction of astigmatism. Also, the fact that simulated astigmatic defocus may degrade VA more than real astigmatic defocus[25] and that myopic observers may not benefit to the same extent as emmetropes from AO correction in a VA task[45] could have biased the response of habitually corrected astigmats.

Furthermore, highly statistically significant longitudinal changes were found in G3, who experienced a change in retinal image (from astigmatic to corrected images) during the study after correction of astigmatism, although the exposure to the new correction made the subjects more insensitive to astigmatism (at their natural axis) rather than more susceptible to VA degradation by astigmatism. However, the mechanism and time course for adaptation to induced astigmatism and its impact on visual performance may differ from that associated with the adaptation to an astigmatic correction in astigmatic subjects and their visual response to astigmatism after correction. The astigmatic subjects of our study reached VAs after correction of their astigmatism similar to those of the non-astigmats (G1). Similarly, their clinical visual function was significantly improved with astigmatic correction. However, we found that, despite correction of astigmatism, astigmatic subjects appear to keep a certain degree of adaptation (or perhaps learned features) to/of their natural astigmatism, which makes them relatively more immune to the induction of astigmatism along their natural axis, and astigmatism correction wear does not eliminate but, rather, reinforces this effect.

The same trends were reflected in the effect of adding astigmatism to coma. Beneficial interactions for coma and astigmatism, as predicted from optical theory occurred in non-astigmats (G1, at 0 and at 45 degrees) and to some extent in astigmats (G2 and G3, for astigmatism perpendicular to the natural axis and at 45 degrees). However (and despite its optical equivalence), combined astigmatism and coma lowered visual performance at 90 degrees for G1 and at the natural axis of astigmatism for G2 and G3, that is, the same orientations for which astigmatism was less deleterious to vision. Again, the longitudinal measurements in G3 showed a decreasing performance with time under this combination, rather than an improvement, consistent with the decreased sensitivity to the induced astigmatism throughout the same period. The fact that astigmatism affects visual performance differently in the presence or absence of aberrations suggests that aberrations may dilute the measurable adaptational effects to astigmatism. On the other hand, the fact that the effects of combined coma and astigmatism differ across groups suggests that mechanisms do not operate independently, but rather combined effects of aberrations on vision are not only driven by the optics but also are affected by prior adaptation to astigmatism.

In summary, although astigmatism lowers visual performance, its impact seems to be dependent on the angle of induced astigmatism both for non-astigmats (for whom inducing astigmatism at 90 degrees produced significantly less degradation than at other axes) and astigmats (who experienced less visual degradation when astigmatism was induced at their angle of astigmatism). Both habitually corrected and initially non-corrected astigmats after correction of astigmatism showed a bias toward better performance with astigmatism induced at their natural axis, which persisted (and actually increased) even after astigmatism correction wear for an extended period, suggesting that astigmats may store adaptation states or cues related to their natural astigmatism.

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María Viñas
Instituto de Óptica
Consejo Superior de Investigaciones Científicas (CSIC)
C/Serrano, 121
28015 Madrid
Spain
e-mail: maria.vinas@io.cfmac.csic.es