Visual performance after correcting higher order aberrations in keratoconic eyes

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Keratoconic eyes are affected by an irregular optical blur induced by significant magnitude of higher order aberrations (HOAs). Although it is expected that correction of ocular aberrations leads to an improvement in visual performance, keratoconic eyes might not achieve the visual benefit predicted by optical theory because of long-term adaptation to poor retinal image quality. To investigate this, an adaptive optics (AO) system equipped with a large-stroke deformable mirror and a Shack–Hartmann wavefront sensor was used to correct the aberrations and measure high contrast tumbling E visual acuity (HCVA) in 8 keratoconic eyes. Eight normal eyes were employed as control. Aberrations were dynamically corrected with closed-loop AO during visual acuity testing, with residual root-mean-square error of around 0.1 μm in both groups over 6-mm pupil (p = 0.11). With AO correction, the HCVA in logMAR was −0.26 ± 0.063 in normal eyes, and in keratoconic eyes, it was −0.07 ± 0.051 (p = 0.0001) for the same pupil size. There was no correlation in the AO-corrected HCVA for normals with the magnitudes of their native HOA. However, within keratoconic eyes, poorer AO-corrected HCVA was observed with an increase of the native magnitudes of HOA ($R^2 = 0.67$). This may indicate that long-term visual experience with poor retinal image quality, induced by HOA, may restrict the visual benefit achievable immediately after correction in keratoconic eyes.

Keywords: physiological optics, spatial vision, visual acuity, adaptive optics, keratoconus


Introduction

It is known that the human eye suffers from lower and higher order aberrations, which degrade retinal image quality (Porter, Guirao, Cox, & Williams, 2001; Smirnov, 1961; Thibos, Hong, Bradley, & Cheng, 2002). Compensation of conventional refractive error is done routinely using spectacles, contact lenses, and refractive surgery. The growth of wavefront sensing and adaptive optics (AO) technology has allowed the measurement and correction of the ocular higher order aberrations in addition to defocus and astigmatism, to provide an image quality ultimately limited by diffraction alone (Liang & Williams, 1997; Liang, Williams, & Miller, 1997). Eyes with abnormal corneal conditions, such as those having keratoconus, are affected by larger magnitudes of higher order aberrations, approximately 5–6 times more in terms of higher order root-mean-square error (RMS), than that typically observed in normal eyes for a 6-mm pupil (Maeda et al., 2002; Pantanelli, MacRae, Jeong, & Yoon, 2007). Effective AO compensation of aberrations requires the maximum correctable magnitude of error (dynamic range) of the wavefront corrector to be at least equal to the peak-to-valley error of the aberrations (Doble, Miller, Yoon, & Williams, 2007). Most conventional wavefront correctors have sufficient stroke to correct higher order aberrations in normal eyes with second order aberrations pre-corrected by other means (such as trial lenses), but they are limited by their inadequate dynamic range to correct the higher order aberrations in keratoconic (KC) eyes. Using a large-stroke deformable mirror incorporated in an AO system, Sabesan, Ahmad, and Yoon (2007) recently demonstrated higher order aberration correction of KC eyes to provide almost perfect retinal image quality.

AO has thus enabled the non-invasive real-time study of the visual system by surpassing the limit imposed by the optics of the eye. In particular, researchers have demonstrated the visual benefit attainable after correcting higher order aberrations (Liang et al., 1997; Yoon & Williams, 2002). The improvement in contrast sensitivity for normal healthy eyes has been shown to be substantial, yielding a sixfold increase at 27.5 cycles per degree and enabling the detection of high spatial frequency gratings at 55 cycles per degree, which were indiscernible while viewing through the eye’s best refraction (Liang et al., 1997). Yoon and Williams (2002) reported a 1.6-fold improvement in visual acuity after correcting for both monochromatic and chromatic aberrations in seven eyes.

Visual performance however is influenced by retinal and neural factors in addition to optical aberrations...
Neural adaptation to blur is one profound mechanism that influences visual performance (Webster, Georgezon, & Webster, 2002). Adaptation to blur induced by refractive error has been studied in emmetropes and myopes and an improvement in visual acuity was reported after extended periods of viewing through myopia (George & Rosenfield, 2004; Mon-Williams, Tresilian, Strang, Kochhar, & Wann, 1998). Moreover, Rossi, Weiser, Tarrant, and Roorda (2007) found that myopes with low refractive error do not benefit to the same extent after correcting higher order aberrations as emmetropes, attributing the incapacity to neural factors. The effect of adaptation to optical blur induced by their own particular higher order aberrations has also been studied in normal eyes (Artal, Chen, Fernández et al., 2004). Subjects preferred the blur induced by their own higher order aberrations in comparison to a rotated version of the same, indicating that their neural visual system was adapted to their own optical blur caused by modest magnitudes of aberrations. Blurred retinal image quality may be compensated for by such a neural adaptation mechanism thereby reducing its impact on visual performance. This effect of adaptation might be higher in KC eyes, which are affected by an optical blur due to significantly larger magnitude of higher order aberrations. Furthermore, analogous to neural compensation for a blurred retinal image, neural insensitivity to a perfect retinal image might also be plausible. Adaptation to poor optical quality might render their visual system insensitive to diffraction-limited retinal image quality. Accordingly, visual benefit achievable immediately after correction of higher order aberrations in these eyes might be limited. This study examined the role of adaptation in KC eyes to their native higher order aberrations, by investigating the limit of visual performance achievable, after their correction using AO. Henceforth in this article, we will use the following terminology:

a. “neural compensation” to refer to the phenomenon that might lead to improved visual performance by compensating for a blurred retinal image and

b. “neural insensitivity” to refer to the phenomenon that might limit visual performance for a diffraction-limited retinal image.

### Methods

#### Subjects

The University of Rochester Research Review Board approved this research and all patients signed an informed consent form before their participation in this study. Eight normal and eight KC eyes were employed for the study. The mean age in years was 34.1 ± 7.6 and 38.9 ± 10.6 in normal and KC eyes, respectively, and was not statistically different ($p = 0.3$). Among the KC group, 7 were moderate and 1 was advanced. The keratometric readings from corneal topography maps were used to classify the KC patients as moderate or advanced, as suggested in the Collaborative Longitudinal Evaluation of Keratoconus study (Zadnik et al., 1996). Paralysis of accommodation and dilation of the pupil in all patients were achieved with 1% tropicamide ophthalmic solution. Head movements were stabilized in all patients using a bite bar mount. Defocus and astigmatism were partially compensated using a phoropter, which was optically conjugated to the eye’s pupil, and a Badal system prior to closed-loop AO. The magnitude of sphere and cylinder pre-compensated in KC eyes was 4.25 ± 2.20 D and 2.66 ± 1.01 D, respectively. The average higher order RMS wavefront error was 0.44 ± 0.12 μm and 2.29 ± 1.15 μm in normal and KC eyes, respectively, over a 6-mm pupil. Two-tailed, unpaired student's $t$-test of statistical significance was employed to evaluate the difference in correction performance and visual performance between the two groups.

#### Large-stroke AO system

An AO system, described elsewhere (Sabesan, Ahmad et al., 2007), was used to correct the aberrations in all the subjects. It consisted of a large-stroke deformable mirror (Mirao 52D; Imagine Eyes, Orsay, France; Fernandez et al., 2006) and a custom-made, in-house Shack–Hartmann wavefront sensor. The Shack–Hartmann microlens array had a spacing of 300 μm and a focal length of 7.6 mm. Each spot formed by the microlens array was imaged on a charge coupled device camera having a pixel size of 7.4 μm. The wave aberrations, expressed in terms of Zernike polynomials, were calculated from this spot array pattern. Wave aberrations were computed by decomposing into 63 Zernike coefficients corresponding to 10th order Zernike polynomials. The Zernike coefficients were expressed according to ANSI Z80.28-2004 standard (Thibos, Applegate et al., 2002).

The deformable mirror consisted of a magnetic membrane whose movement could be controlled by applying voltages to 52 independent actuators. The eye’s pupil, the deformable mirror, and the microlens array were conjugated using image relay optics. The pupil of the eye and the microlens array had unity magnification between them and both were magnified 2.33 times on the deformable mirror aperture. The closed-loop control of the deformable mirror was obtained using the direct-slope algorithm (Chen, 2006).

The optical performance of AO correction was evaluated using Shack–Hartmann measurement, which is incapable of detecting high spatial frequencies. Therefore, the RMS error derived from these measurements might be insufficient to assess optical quality. The measurement and correction of normal and KC eyes was further verified.
using phase plates to simulate aberrations in both cases. A phase plate was used to generate an advanced KC eye’s aberration and had a total RMS and higher order RMS of 4.16 μm and 3.42 μm, respectively, over a 6-mm pupil. The most dominant aberrations were astigmatism and coma. The aberration measured with the Shack–Hartmann wavefront sensor was used to compute the point-spread function. The point-spread function was also experimentally imaged and compared with the theoretical point-spread function. To verify correction performance, the point-spread function was imaged through the same KC phase plate after its aberration was corrected with AO. For comparison, the same measurement was also made with a phase plate designed for a normal eye. This phase plate had total RMS and higher order RMS of 1.72 μm and 0.51 μm, respectively. The pupil size was 6 mm in all cases. The peak intensity and the cross-correlation of the point-spread functions were compared to verify AO correction.

Vision testing

Visual acuity (VA) was measured using a tumbling letter ‘E’ test at 100% contrast in white light. The size of the visual field on the retina in this study was 1 deg. A psychometric function based on 30 trials was derived, and visual acuity was determined as the line thickness of the letter for which at least 62.5% of responses were correct. The black letter on a white background was displayed on a computer projector (Sharp Model no. PG-M20X) placed at the retinal plane. The retinal illuminance for a 6-mm pupil was 100 cd/m². Subjects viewed the stimulus through an artificial adjustable pupil placed conjugate to the eye’s pupil. Aberrations were corrected over a 6.5-mm pupil with AO while vision testing was performed over an artificial 6-mm pupil. Aberrations were corrected continuously throughout the visual acuity test in a closed-loop manner to maintain optical quality close to the diffraction limit. The subjects were asked to blink at their discretion during psychophysics, at which point the correction was temporarily suspended. AO closed-loop gain of 90–100% ensured fast convergence of correction within 1–3 iterations after blink. High closed-loop gain also facilitated a stable optical quality throughout the visual acuity test.

Results

Verification of aberration measurement and correction with AO

Figure 1A shows the theoretical and experimental point-spread functions for the KC phase plate. Both the point-spread functions agree qualitatively, establishing the reliability of the Shack–Hartmann measurement. Similar level of residual RMS, lower than 0.1 μm, was obtained for both normal and KC phase plates after AO correction. Figure 1B shows the corresponding point-spread functions obtained after AO correction in both cases. The cross-correlation between the two point-spread functions was 0.98. The ratio of the peak intensity between the normal and KC point-spread functions was 1.03. This difference can be attributed to the different roughness between the two phase plates. The same optical and retinal image quality was thus ensured in both normal and KC eyes after AO correction.

AO correction performance

Figures 2A and 2B show the time course of total RMS and higher order RMS, respectively, for one normal and one moderate KC eye during the visual acuity test. With a high closed-loop gain of 100%, the total RMS is reduced to a considerably low value of 0.1 μm in both cases within 1–3 iterations. The time courses of convolved images of the letter “E” based on the measured aberrations are also shown in Movie 1 for (A) the normal and (B) the moderate KC eye. The convolved images are calculated under normal viewing condition by including chromatic aberration to
simulate the theoretical retinal image quality. The letter size used in this simulation corresponded to a 20/20 Snellen letter. Figure 3 shows the total RMS before and after AO correction for the normal and KC eyes. Mean ± standard deviation of the pre-compensated total RMS and higher order RMS across the 8 normal eyes was 0.78 ± 0.22 μm and 0.44 ± 0.12 μm, respectively, for a 6-mm pupil. After AO correction in normal eyes, the total RMS and higher order RMS were reduced to 0.08 ± 0.02 μm and 0.05 ± 0.01 μm, respectively over a 6-mm pupil. In 8 KC eyes, the pre-compensated total RMS and higher order RMS were 2.87 ± 1.22 μm and 2.29 ± 1.15 μm, respectively, for a 6-mm pupil. After AO compensation in the 8 KC eyes, the mean ± standard deviation of the total RMS and higher order RMS was reduced to 0.11 ± 0.02 μm and 0.08 ± 0.03 μm, respectively, over a 6-mm pupil. The residual RMS with AO was not statistically different (p = 0.11) between the two groups as shown in Figure 3. Averaged variability of correction performance, defined as the standard deviation of total residual RMS during acuity test, was 0.027 ± 0.013 μm across both groups of eyes. The correction performance indicates the ability of large-stroke AO to provide almost perfect optical quality in both cases, irrespective of the different magnitudes of native aberrations in the two groups of eyes. In addition, the ability to maintain perfect optical quality for a relatively long period of time with closed-loop correction renders it appropriate for vision testing.

**Visual performance with AO correction of aberrations**

Figure 4 shows the visual performance with AO correction of aberrations in both normal and KC eyes. The high contrast visual acuity (HCVA) measured over a
6-mm pupil was $0.26 \pm 0.063$ logMAR and $0.07 \pm 0.051$ logMAR for normal and KC eyes, respectively, with AO compensation. The improvement in retinal image quality is more pronounced in the KC eyes than in normal eyes with AO, given the relatively larger magnitudes of native higher order aberrations in the KC eyes. However, given the same near-diffraction-limited optical quality, visual performance was significantly worse in the KC eyes compared to normal eyes.

Figure 5 illustrates the correlation between the AO-corrected visual performance and native higher order aberrations. The $x$-axis denotes the retinal image quality with uncorrected higher order aberrations. The $x$-axis represents the area under the modulation transfer function up to 60 cycles per degree in Figure 5, calculated from the native higher order aberrations in both groups of eyes. The $y$-axis shows the AO-corrected HCVA. In normal eyes, there was no correlation between higher order aberrations and visual acuity achieved after their correction. However, KC eyes with larger magnitudes of native higher order aberrations achieved poorer HCVA after AO correction. While estimating native optical quality in terms of the area under the modulation transfer function, a well-correlated trend of decreasing AO-corrected visual acuity was observed with increasing magnitude of native higher order aberrations. A similar well-correlated trend ($R^2 = 0.75$) of decreasing AO-corrected visual acuity in KC eyes was also observed when the magnitude of native higher order aberrations was estimated in terms of polychromatic Strehl ratio. The limit on visual acuity obtained after correction depends on the severity of native higher order aberrations: correction of higher magnitudes of native higher order aberrations leads to poorer visual acuity than that achieved after correcting lower magnitudes of higher
order aberrations. The magnitude of native aberrations among both groups is thus indicative of the visual benefit attainable on their correction.

Discussion

The visual performance, after correcting aberrations to nearly diffraction-limited optical quality, was measured for the first time in KC eyes. Until recently, the stroke of deformable mirror in AO systems inhibited the correction of large magnitudes of aberrations found in KC eyes. Large actuator stroke and high linearity of the actuator response for the wavefront corrector used in this study facilitated a near-perfect correction of aberrations in normal and KC eyes. The high closed-loop gain of 90–100% also assisted in maintaining the optical quality over the duration of the visual acuity test.

After aberration correction using AO, visual performance was significantly worse in KC eyes compared to normal eyes. In the absence of monochromatic optical imperfections, the poorer visual performance in KC eyes might be attributed to several factors. These include:

a. ocular dimensions: axial length of the eye and retinal sampling,
b. optical factors: chromatic aberration, off-axis aberration,
c. neural factors: neural insensitivity arising from chronic exposure to asymmetrically aberrated retinal image quality.

Retinal sampling is an important factor affecting the visual acuity achieved on correction of ocular aberrations. However, there is no published data on the photoreceptor mosaic in KC eyes and hence the difference in visual acuity cannot be attributed to the retinal sampling. Axial length of the eye is a potential factor that can alter HCVA estimates due to a difference in magnification on the retina. KC eyes are reported to have longer axial lengths than emmetropes (Lanier, Bullington, & Prager, 1992), which would translate to a higher magnification of the visual stimulus on the retina. Accordingly, the difference in HCVA between KC and normal eyes, by accounting for this magnification, will be further increased. The average axial lengths of 4 KC and 4 normal subjects in our study was $23.81 \pm 0.55$ mm and $24.42 \pm 0.55$ mm, respectively, and were not statistically different ($p = 0.3$). Accordingly, the disparity in visual acuity cannot be attributed to the difference in axial lengths.

The size of the visual field on the retina in this study was 1 deg. It might be reasonable to expect that the size of the isoplanatic patch could be smaller in KC eyes owing to the higher magnitudes of aberrations, especially as compared with the normal eyes. The lens design software CODEV (Optical Research Associates, version 9.82) was used to verify if aberrations change significantly within 1 deg field in KC eyes. To simulate the KC aberrations, measured anterior and posterior corneal surface profiles of KC eyes were incorporated into the Gullstrand’s eye model. This was under the assumption that the majority of the aberrations in KC eyes are contributed by the cornea. The maximum change in aberrations within the 1 degree field was 0.009 μm in RMS wavefront error, which is 4 times smaller than the diffraction-limited RMS as estimated from the Marechal criterion (Born & Wolf, 1980) at 555-nm wavelength. It is important to note that the actual letter for visual acuity subtends a smaller angle on the retina than 1 deg, for instance, letter of 20/20 subtends 5 arcmin. Accordingly, the visual field lay within the isoplanatic patch in KC eyes. Thus retinal image quality within the 1 deg field was unaffected by off-axis aberrations.

The visual acuity test was performed under white light after the subjects had chosen their best focus using a Badal optometer. In this way only longitudinal chromatic aberration was optimized, while lateral chromatic aberration was uncorrected. The lateral chromatic aberration has not yet been measured in KC eyes. If the presence of larger magnitudes of higher order aberrations in KC eyes leads to a larger lateral chromatic blur than normal eyes, part of the difference in visual performance might be attributed to this disparity. To investigate this further, the same Gullstrand’s eye model described above was used to estimate the lateral chromatic aberration in KC eyes. The lateral chromatic blur is quantified by the separation of images for hydrogen blue (0.486 μm) and red (0.656 μm) lines. The difference in chief ray angles at 1 deg field between the two wavelengths was 0.2 arcm. This value of lateral chromatic aberration is comparable to 0.28 arcmin reported by Thibos (1987) who used a model eye at wavelengths of 0.43 μm and 0.77 μm to compute the
same. Therefore, lateral chromatic aberration is unlikely to be the cause for the difference in visual acuity between the normal and KC eyes.

Aberrations in both normal and KC eyes were measured and corrected reliably as indicated in Figure 1. In the absence of aberrations, the photoreceptor sampling defines the maximum achievable visual acuity. The photoreceptor spacing at the fovea ranges from 0.42 to 0.54 arcmin (Curcio, Sloan, Kalina, & Hendrickson, 1990; Williams, 1988). Adopting the simplistic assumption that letter acuity of 20/20 corresponds to 30 cycles/degree, letter acuity limits based on cone spacing would range from 20/8.4 to 20/10.8. In this study, the mean ± standard deviation of Snellen acuity obtained after correcting higher order aberrations in normal eyes was 11.1 ± 1.7, which was slightly greater than the limit imposed by cone sampling. The uncorrected residual ocular chromatic aberration was an important contributing factor limiting the HCVA achieved after AO correction in normal eyes (Thibos, Bradley, & Zhang, 1991). In addition, the residual wavefront error, though as low as 0.08 ± 0.05 μm in normal eyes, was still above the diffraction limit estimated from the wavefront error according to Marechal criterion at 830 nm (0.06 μm). In general, the encircled energy within the point-spread function will be a better indicator of system performance, in the KC eyes especially, which if affected by corneal scars can cause scattering artifacts in the wavefront sensor spot pattern.

The average higher order RMS wavefront error before correction was 2.29 ± 1.15 μm in KC eyes compared to 0.44 ± 0.12 μm in normal eyes, over a 6-mm pupil. Higher order aberrations accounted for only 2% of the variance of total aberrations on average in normal eyes compared to 14% in KC eyes on average. Accordingly, the normals, having relatively smaller magnitudes of higher order aberrations, did not undergo a substantial change in their adaptation state, upon their correction. Therefore, they achieved acuity close to the limit imposed by the photoreceptor sampling. However in KC eyes, chronic exposure to asymmetrically blurred retinal image quality, induced by higher order aberrations, may be restricting the visual acuity achievable immediately after correction. Moreover, neural insensitivity to corrected retinal image quality depended on the severity of native higher order aberrations as shown in Figure 5. The findings of Rossi et al. (2007) suggested a similar neural factor limiting visual performance in low myopes after correcting all ocular aberrations as compared to emmetropes. Low myopes experience a refractive blur degrading their retinal image quality analogous to higher order aberrations in the case of KC eyes. Limited visual performance as demonstrated in this study and by Rossi et al. (2007) might be identified as a neural insensitivity to near-diffraction-limited optical quality. However, further experiments are necessary to ascertain that this neural insensitivity to perfect retinal image quality leading to reduced visual performance is in fact a direct consequence of the adaptation state. In contrast to neural insensitivity for a perfect retinal image, adaptation to blurred retinal image quality might lead to neural compensation, alleviating its impact on visual performance. In particular, researchers have demonstrated improved visual acuity after adapting to a retinal image blurred by refractive error. Mon-Williams et al. (1998) noted an improvement in monocular VA 0.12 logMAR after 30 minutes of viewing through a +1.00-D lens without any significant change in the refractive state of the eye. George and Rosenfield (2004) further demonstrated that the mean change in grating visual acuity was significantly greater for myopic subjects than emmetropic subjects, while viewing through +2.50-D lens for 120 minutes concluding that the neural compensation was dependent on the refractive and thus the adaptation state of the eye. Mon-Williams et al. (1998) suggested that a possible reason for improvement in visual resolution was the unmasking of high spatial frequency selective channels while also balancing the channel gains in response to the spatial frequency distribution of the retinal image. Webster et al. (2002) further showed that adaptation to low-pass (blurred) filtered images resulted in the original image appearing “too sharp” and the best subjective focus being shifted toward an image with lower spatial frequencies. The measurement of contrast sensitivity in KC eyes after correcting all aberrations might be able to shed light on how adaptation to large magnitudes of ocular aberrations altered the sensitivity of the visual system to different spatial frequencies.

Maximum visual performance, as predicted by ocular optics, might be expected in KC eyes after long-term adaptation of the visual system to near-diffraction-limited ocular optics. Provision of superior optical quality for a longer duration with AO is not practical as it necessitates the subject being fixed on the dental impression. However, efforts to practically correct the higher order aberrations in KC eyes to improve visual acuity have been demonstrated using customized soft contact lenses (Sabesan, Jeong et al., 2007). The rate at which the visual system can adapt to changes in its monochromatic aberrations is still unknown. Adaptation to the spatial frequency content of the stimulus to provide perceptual constancy, as demonstrated by Webster et al. (2002), occurred on the timescale of a few minutes. In normal eyes, Artal, Chen, Fernández et al. (2004) demonstrated an adaptation to their own aberrations, which was an example of the other extreme, where adaptation occurs through everyday viewing over a longer time course to maintain image quality despite of persistent optical aberrations. Rosenfield, Portello, Hong, Ren, and Ciuffreda (2003) showed that significant and large improvements in visual resolution produced by blur adaptation were robust and extended for at least 48 hours following the adapting period and were not attenuated by brief intervening periods of clear vision. Conversely, Artal, Chen, Manzanera, and Williams (2004) found that the adaptation to a rotated version of the individual point-spread functions was partially reversed in 15 minutes for normal eyes. The timescale of decay and perhaps also the
onset of the effects of adaptation might be different for blur induced by either refractive error or higher order aberrations. Normal and KC eyes adapting to their own aberrations through normal everyday viewing, unlike an induced adaptation to either refractive error or spatially filtered images, might lend more robustness to the phenomenon. In KC eyes especially, adaptation to the blur induced by relatively larger magnitudes of aberrations might be even more robust. Although it will be reasonable to expect that adaptation to near-diffraction-limited ocular optics might improve vision in KC eyes, the timescale will depend on the magnitude of the native higher order aberrations; much in a similar manner visual acuity was limited after aberration correction (Figure 5).

Aberrations in the eye are dynamic in nature (Hofer et al., 2001) and change with pupil diameter, accommodation, and age. In both high and low light conditions or when accommodating, KC eyes experience a significant blur due to the magnitude of higher order aberrations, though the shape of the point-spread functions might be different only to within a scaling factor. Figure 6 shows the point-spread function for an advanced KC eye for different pupil sizes. In addition, the visual system is known to get accustomed to more than one point-spread function given sufficient experience to each (Peterson & Peterson, 1938). Also, as mentioned earlier, the mean age in years was $34.1 \pm 7.6$ and $38.9 \pm 10.6$ in normal and KC eyes, respectively, and was not statistically different ($p = 0.3$). Therefore, age-related increase in amount of scatter was unlikely to be responsible for the reduced visual performance in KC eyes compared to normals.

Reduced visual performance on correction of aberrations in KC eyes has important clinical implications in customized correction methods such as customized soft contact lenses, refractive surgery, and intraocular lenses. AO is a powerful tool to assess visual quality after correcting higher order aberrations and thus facilitating an effective screening process before customized corrective procedures. However, optimal visual performance with a corrective option can be provided only by accounting for the neural insensitivity to such customized correction for highly aberrated eyes. Prior to this study, Sabesan, Jeong et al. (2007) have measured visual acuity improvement with customized soft contact lenses designed to correct higher order aberrations in KC eyes. Due to the relatively high residual aberrations, it was difficult to isolate the neural factors from optical factors for restricted visual performance. Nevertheless, with customized laser refractive surgery, contact lenses, and intraocular lenses gaining importance for correction of higher order aberrations and presbyopia (Piers, Weeber, Artal, & Norrby, 2007), the importance of neural factors in limiting visual performance cannot be neglected.

**Conclusion**

With the same perfect optical quality, visual performance was significantly worse in KC eyes compared to normal eyes. In addition, the magnitude of native higher order aberrations directly influenced the extent of this limitation of visual performance in these eyes in the short term. Neural insensitivity to a near-diffraction-limited retinal image might be caused by long-term visual experience with poor retinal image quality, induced by higher order aberrations in KC eyes. Accordingly, maximum predicted visual performance might be expected in these eyes after long-term adaptation of the visual system to near-diffraction-limited ocular optics.

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