Contribution of the cornea and internal surfaces to the change of ocular aberrations with age

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We studied the age dependence of the relative contributions of the aberrations of the cornea and the internal ocular surfaces to the total aberrations of the eye. We measured the wave-front aberration of the eye with a Hartmann–Shack sensor and the aberrations of the anterior corneal surface from the elevation data provided by a corneal topography system. The aberrations of the internal surfaces were obtained by direct subtraction of the ocular and corneal wave-front data. Measurements were obtained for normal healthy subjects with ages ranging from 20 to 70 years. The magnitude of the RMS wave-front aberration (excluding defocus and astigmatism) of the eye increases more than threefold within the age range considered. However, the aberrations of the anterior corneal surface increase only slightly with age. In most of the younger subjects, total ocular aberrations are lower than corneal aberrations, while in the older subjects the reverse condition occurs. Astigmatism, coma, and spherical aberration of the cornea are larger than in the complete eye in younger subjects, whereas the contrary is true for the older subjects. The internal ocular surfaces compensate, at least in part, for the aberrations associated with the cornea in most younger subjects, but this compensation is not present in the older subjects. These results suggest that the degradation of the ocular optics with age can be explained largely by the loss of the balance between the aberrations of the corneal and the internal surfaces.

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1. INTRODUCTION

Different aspects of visual performance degrade continuously with age, even in normal and healthy subjects.[1] In particular, spatial vision, assessed by either visual acuity or contrast sensitivity,[2] declines throughout the life span. A variety of factors are responsible for this deterioration, ranging from purely optical degradation to retinal and neural losses. Objective measurements of retinal image quality show a nearly linear decline with age.[3,4] This result suggested a significant increase in the optical aberrations of the eye with age, in agreement with studies in which aberrations were measured directly.[5–7] In addition, intraocular scatter increases noticeably in older eyes.[8] The deterioration of the retinal image of older eyes caused by these optical factors plays an important role in limiting spatial vision.

Although it is now widely accepted that the ocular optics deteriorate with age, the underlying causes of this age-related increase in aberrations remain to be precisely determined. Recently Guirao *et al.*[9] studied how the aberrations associated with the anterior surface of the human cornea changed with age in a normal population. The aberrations were computed from the elevation data provided by a videokeratographic (corneal topographer) system. The spherical aberration was found on average to be slightly larger in middle-aged and older corneas. Coma and other higher-order aberrations also tended to increase with age. Oshika *et al.*[10] also found an increase in the overall corneal aberrations with age (although only for a large, 7-mm, pupil diameter), but spherical aberration was invariant with age. In any case, the results of both studies indicate that the increase in corneal aberrations is too small to account for the complete reduction of the retinal image quality with age.

This indicates that mechanisms other than changes in the cornea should be primarily responsible for the increase in the ocular aberrations with age. One possibility is an increase in the aberrations of the crystalline lens caused by the continuous changes in the lens with age. As the lens grows, its dimensions, surface curvatures, and refractive-index change, altering the lens aberrations. Glasser and Campbell[11] found a large change in the spherical aberration of excised older lenses measured *in vitro*.

On the other hand, it was also shown that in young subjects, the lens tends to compensate for part of the corneal aberrations, thus producing an improved retinal image.[12–14] This indicates that there is a fine coupling between corneal and lenticular aberrations in young adults. As the aberrations of the lens change with age, it is possible that this compensation is partially or completely lost. This may explain the overall increase in aberration and the reduction of retinal image quality throughout the life span.

In this paper we address this problem by measuring both the wave aberration (WA) produced by the anterior

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corneal surface (from its shape) and the aberrations of the complete eye [with use of a Hartmann–Shack (HS) wave-front sensor] in a group of normal subjects with different ages. The main objective is to determine which of the previously mentioned factors is primarily responsible for the age-related increase in the ocular aberrations.

2. METHODS

For each subject, both ocular and anterior corneal aberrations were measured in order to permit a direct comparison. From these two WA maps, the relative contributions of the anterior corneal surface and the internal optics to the overall ocular WA were evaluated. Assuming a simple model for the eye, the aberrations of the internal ocular media were obtained by direct subtraction of the anterior corneal WA from the ocular WA. Figure 1 shows a schematic diagram for this procedure.

A. Estimation of the Anterior Corneal Surface Wave-Front Aberration from Corneal Elevation Data

The WA of the anterior corneal surface was estimated from its geometry. A Placido-based videokeratographic device (MasterVue corneal topography system; Humphrey Instruments, San Leandro, Calif.) was used to measure the corneal shape. The corresponding discrete set of corneal elevation data over the pupil plane was processed to calculate the WA for the anterior corneal surface, expressed as a Zernike polynomial expansion.[15] The computational procedure, together with an analysis of its main limitations, is extensively described elsewhere.[16] In summary, first the corneal elevation (height) z, provided by the topographer was fitted to a Zernike expansion,

$$z(\rho, \theta) = \sum_{i=1}^{N} a_i Z_i(\rho, \theta), \tag{1}$$

by using a Gram–Schmidt orthogonalization method. The corneal WA was calculated as the difference in optical path between the chief ray and the marginal rays over the pupil. Taking into account the Zernike representation for the corneal surface [Eq. (1)], the WA was also obtained as a Zernike expansion,

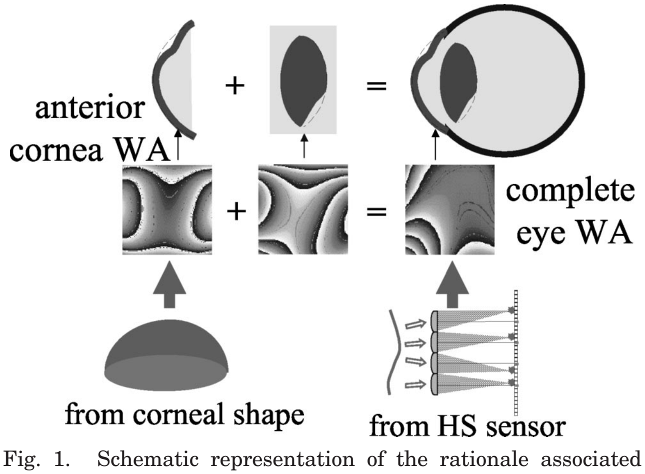

Fig. 1. Schematic representation of the rationale associated with the experiment.

$$W(\rho, \theta) = \sum_{i=1}^{N} A_i Z_i(\rho, \theta), \tag{2}$$

where the coefficients A_i are linear combinations of the coefficients a_i (see Ref. 16 for details). The accuracy of this procedure for estimating the corneal WA was previously evaluated with use of reference surfaces.[16] For central regions of 4 mm and 6 mm diameter, the root-mean-square (RMS) error between the actual and the measured aberrations was near 0.05 and 0.2 μm, respectively, which renders this method appropriate for this type of study.

B. Measurement of the Ocular Wave-Front Aberration with a Hartmann–Shack Wave-Front Sensor

The WA of the complete eye was measured with a HS wave-front sensor.[17–19] A narrow near-infrared beam projected onto the subject's retina acts as a beacon source. On the way out, the light propagates through the eye's optics, suffering local phase shifts in the wave front, before reaching a microlens array that samples the local average of the WA tilt over the eye pupil. This sampling generates a distribution of spots that is captured by a CCD camera in the focal plane of the microlenses. In the HS image, the relative displacement of each sample spot is proportional to the WA slope within the corresponding subaperture. Although a similar version of the HS sensor has previously been presented,[19] a description is included here for the sake of completeness. A schematic diagram of the apparatus is shown in Fig. 2. The system was specially designed to be used in naïve subjects and, in particular, in older subjects. In the first pass, 780-nm light emitted by a laser diode, LD (Spindler and Hoyer DL 25) is linearly polarized (p orientation), spatially filtered (an aspheric lens, AL, focuses the beam on a pinhole of 25 μm, PH), and collimated by an 190-mm achromatic doublet, C; and the beam is limited by a 1.3-mm-diameter aperture, A. In the second pass, the light is reflected by a polarizing cube beam splitter, c-BS and propagates through an array of square microlenses, MMLS, with individual aperture of 0.6 mm and 40-mm focal length. A scientific-grade cooled CCD camera (Photometrics-Sensys KAF0400) of 768 × 512 pixels (9 × 9 μm in size; 12 bits/pixel) is placed at the focal plane of the microlenses to record the HS image. The microlenses are conjugated with the eye pupil through a pair of achromatic doublets, L1 and L2 (148 mm and 190 mm focal length, respectively). These lenses produce a magnification of 0.78 between the MMLS plane and the pupil plane. The focus corrector system, FC, is used to easily modify the optical path between L1 and L2. The position of each subject's head was stabilized during the measurements by a bite bar (for young to middle-aged persons) or a chin rest (for older persons) with *XYZ* positioning stages. In every case, the centering of the pupil as well as eye movements during the measurements were controlled by real-time monitoring of the position of the corneal reflection with a video camera, VCCD. To eliminate the corneal reflection on the HS image, an optical window, W, was placed in the entering light path. When W is rotated, the narrow entrance beam is slightly decentered from the visual axis, removing the corneal reflection from the HS images. A fixation target, FT, illuminated by a red light-emitting diode, LED, is included in the first passage to facilitate the alignment of the subject. The amount of light reaching the cornea during the measurements was below 250 nJ, three orders of magnitude below the American National Standards Institute's maximum permissible exposure.[20]

C. Experimental Procedure and Selection of Subjects

All the calculations and comparisons were done for two pupil diameters: 3.85 mm and 5.9 mm. Both the ocular and the corneal series of Zernike coefficients were estimated up to the first six orders for the 5.9-mm pupil and up to the first five orders for the 3.85-mm pupil. In every case, the WA was calculated with respect to the geometrical center of the eye pupil. The accommodation was paralyzed and the pupil dilated in every subject by instilling two drops of cyclopentholate 1%. To estimate the ocular aberrations of every subject, three HS images of 1-s exposure were recorded during a single experimental session. The average of three series of Zernike coefficients obtained for each pupil, together with the associated standard deviation, was computed. The corneal WA was estimated from one single set of corneal elevations.

We also computed modulation transfer functions (MTF's) from the WA of the eye, the cornea, and the internal ocular optics. From each WA $[W(x, y)]$, the generalized pupil function $[P(x, y)]$ is constructed:

$$P(x, y) = p(x, y)\exp[i(2\pi/\lambda)W(x, y)], \tag{3}$$

where $p(x, y)$ denotes a pupil aperture of radius R. The MTF is the modulus of the optical transfer function (OTF) obtained as the complex autocorrelation of $P(x, y)$:

$$\mathrm{OTF}(x, y) = \int P(x', y')P^*(x' - x, y' - y)\mathrm{d}x'\mathrm{d}y'. \tag{4}$$

The 17 subjects selected to participate in the study represent a normal healthy population covering a range of age from 26 to 69 years (one eye from each subject was studied). The subjects were selected after passing a complete ophthalmologic exam. The following exclusion criteria were used to limit the study population to healthy eyes: refractive error (spherical equivalent) more than 2 D, keratometric astigmatism more than 1.5 D, best corrected visual acuity lower than 1 (0.8 for the group of older subjects), any previous surgery on the eye to be tested, amblyopia, any known ocular or retinal pathology (assessed by slit lamp with and without retroillumination with standard clinical criteria), and any systemic disease affecting refractive error (diabetes or disorders of the nervous system). A signed informed consent was obtained from every subject after the nature and all possible consequences of the study had been explained.

3. RESULTS

Figure 3 shows the RMS of the WA's for the complete eye as a function of the age of the subjects. Error bars indicate the standard deviation for the set of three HS measurements. The pupil size is 5.9 mm, and defocus and astigmatism were not included in the RMS calculations. The magnitude of the aberrations is correlated with age ($r = 0.73, p = 0.0007$). However, there is important intersubject variability among the older individuals. Some of the older subjects, compared with the younger subjects, have fewer or a similar amount of aberrations. In the older subjects, even though the error bars are generally larger, they are still much smaller than the differences found in a comparison of the old with the young subjects. In Fig. 3 we also included as an example the WA's and the associated point-spread functions (PSFs) for one young and one old eye. The top panel of Fig. 4 shows the RMS of the WA's for the complete eye and for the anterior sur-

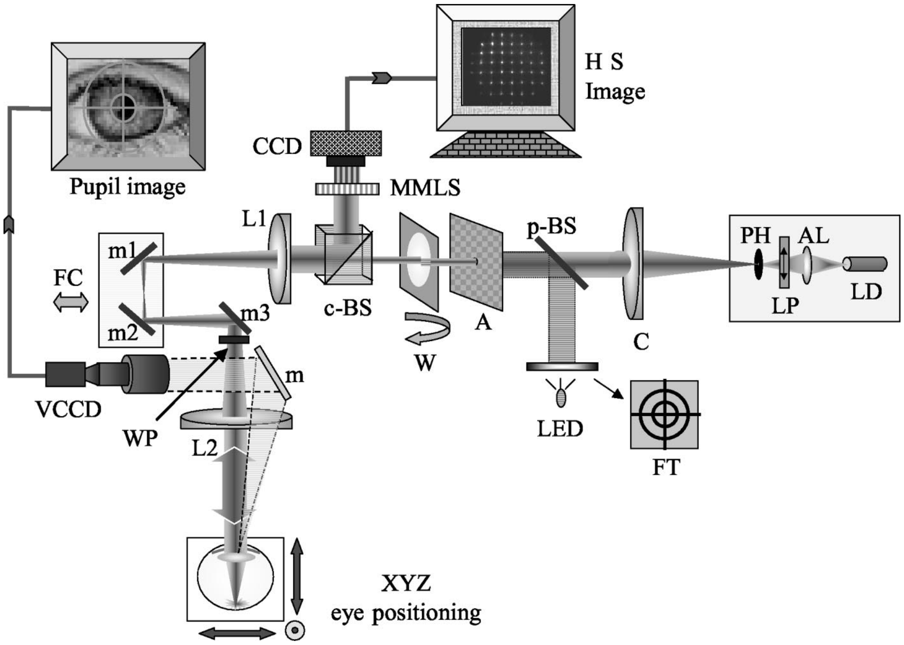

Fig. 2. Schematic diagram of the HS wave-front sensor (details are provided in the text).

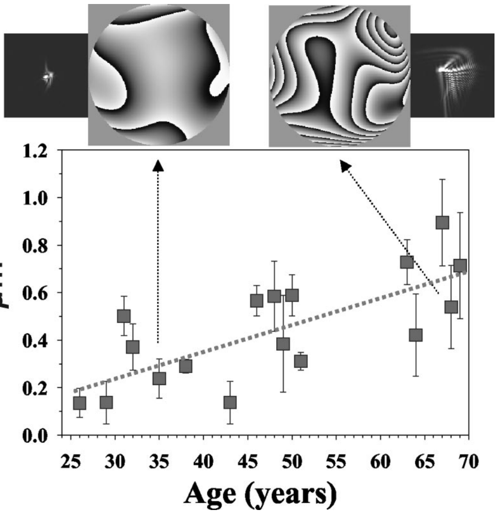

Fig. 3. RMS of the aberrations of the eye expressed in micrometers as a function of age (5.9 mm pupil diameter; defocus and astigmatism not included). The WA and the associated PSF for one young and one older subject are also shown as an illustrative example.

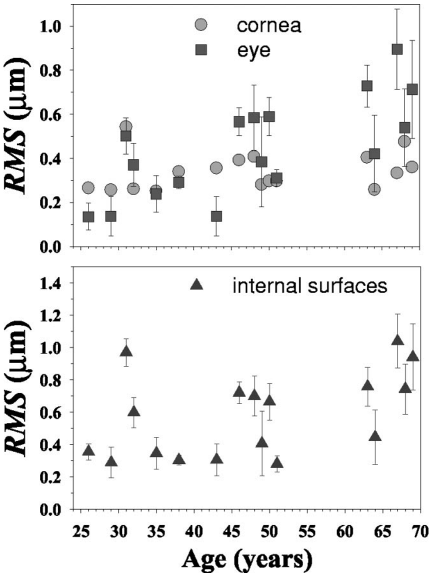

Fig. 4. RMS of the aberrations of the eye (squares) and the cornea (circles) as a function of age (top panel) and for the internal surfaces (bottom panel). In both cases, 5.9 mm pupil diameter, with defocus and astigmatism removed.

face of the cornea. In general, the corneal aberrations of the younger subjects are larger than the total ocular aberrations, indicating that the internal optics compensates for the corneal aberrations, in agreement with previous findings.[13,14] However, in general, the contrary occurs in older subjects: The cornea has lower aberrations than the complete eye: The slopes of the linear regressions to the eye and corneal aberrations with age are 0.011 and 0.0013 μm/year, respectively. On average, the ocular aberrations increase at a rate nearly ten times greater than that of the corneal aberrations with age. In the bottom panel of Fig. 4, the RMS of the aberrations of the internal surfaces is also presented.

To better illustrate how the internal optics fails in partially compensating for the corneal aberrations as age increases, we calculated for each eye a parameter, called compensation factor (cf), given by

$$\mathrm{cf} = 1 - [\mathrm{RMS(eye)}/\mathrm{RMS(corneal)}]. \tag{5}$$

Figure 5 shows this parameter versus the age of each subject. In most of the young subjects cf is positive, indicating the role of the internal optics to compensate for the aberrations of the cornea. However, in middle-aged and older eyes this parameter reaches negative values, which shows that the compensation of the aberrations fails, and in fact the internal optics adds aberrations to that of the cornea. This factor is significantly negatively correlated with age ($r = 0.72$, $p = 0.001$). Figure 6 shows two examples of WAs for the cornea, internal surfaces, and the eye; one is for a younger eye and the other is for an older eye (different from the eyes selected as example in Fig. 3). In the younger eye (upper maps) the cornea and the internal-optics aberrations have a similar magnitude and shape but are opposite in sign, producing an eye with overall lower aberrations. However, in the older eye this finely tuned compensation is not present.

Figure 7 shows the averaged MTF's for the younger (25–45 years old) and the older (45–75 years old) eyes for the anterior corneal surface, the internal surfaces, and

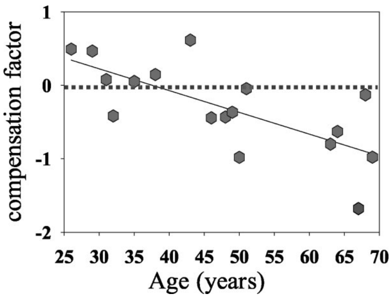

Fig. 5. Compensation factor defined by expression (5) as a function of age. Positive values denote compensation of corneal aberrations by the optics of the internal surfaces, zero represents no compensation, and negative values represent the situation in which the internal surfaces add aberrations to that of the cornea. The solid line is a linear fit to the data, and the dotted line marks the zero value of the compensation factor.

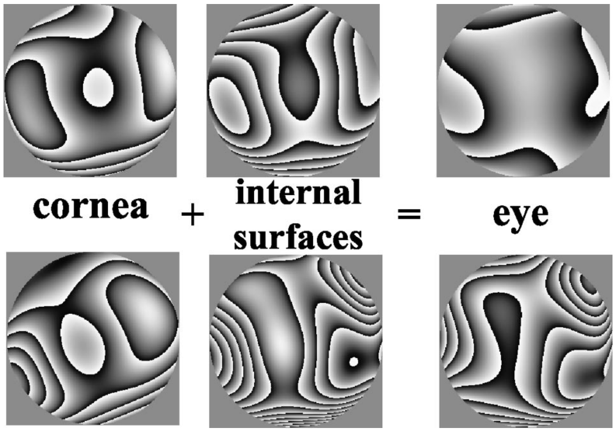

Fig. 6. Two examples of wave aberrations (in a modulus π) representation for the anterior corneal surface, the internal surface, and the eye. Maps at the top are for a young subject and at the bottom for an older subject. (5.9 mm pupil diameter).

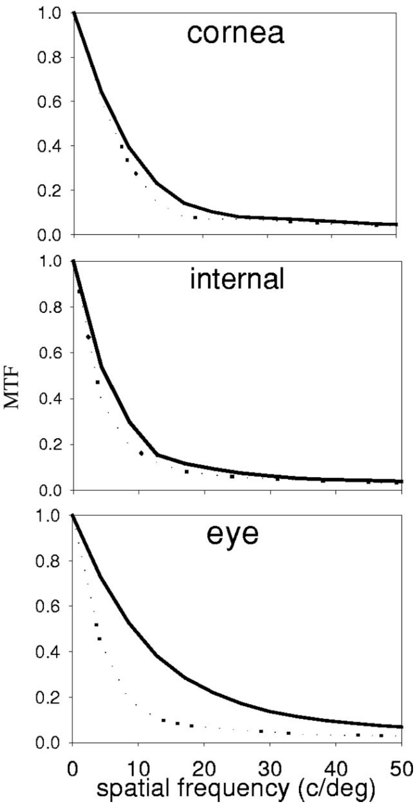

Fig. 7. Averaged MTFs for the cornea, internal surfaces, and the eye (5.9 mm pupil diameter and 555-nm light). Solid curve, younger subjects (25–45 years); dashed curve, older subjects (45–70 years).

the complete eye. These MTF's were computed from the WAs without defocus and astigmatism for 555-nm light as described in Section 2 (Methods). Because the MTF provides a global information on image quality of each component, the results are quite illustrative. In the complete eye, the MTF for the older eye is much lower than that of the younger eye. This is in perfect agreement with direct estimates of the MTF's from double-pass images[3] and simply reflects the overall increase in the aberrations with age. However, the average MTF's for both the cornea and the internal surfaces, when considered separately, decline minimally with age. This again indicates that a major cause for the overall increase in ocular aberrations is the decoupling of the ocular aberration components with age. In younger subjects the MTF for the eye is better than the MTF's of the isolated components (anterior cornea and internal surfaces). A similar result was previously obtained by using the double-pass technique[13] and a HS sensor[14] in subjects in whom the cornea was canceled by use of swimming goggles filled with water. In the older subjects, the MTF in the eye is similar or even lower than the components' MTF's.

We also performed an analysis of individual aberration terms. Figure 8 shows the results for astigmatism. In Fig. 8(a) the RMS of the astigmatism for the cornea and the eye is presented. In most younger eyes, ocular astigmatism is lower than corneal astigmatism what indicates the compensatory role of the internal surfaces. The data presented in Fig. 8(b) further clarify this finding. The plot represents the axis of astigmatism for the anterior corneal surface (circles) and the internal surfaces (triangles) as a function of age (radius in the polar plot represents subject's age). The axis of the internal surfaces remains, in general, constant with age: ~90 deg. However, the axis for the cornea is predominantly 0 deg in the younger eyes and then compensated by the internal surfaces, while in older eyes the axis becomes oblique or the same as in the internal surfaces (90 deg). In consequence, most older eyes have corneal astigmatism not compensated by the internal optics. This behavior of astigmatism with age is well reported in the clinical literature,[21] but we found that for higher-order aberrations something similar happens. In Fig. 9 the RMS for the aberrations of the anterior corneal surface and complete eye of third order (coma-like) and fourth order (spherical-like) is presented. In both cases the internal optics compensates part of the corneal aberrations in younger and some middle-aged subjects but not in general in the older eyes. This behavior is more evident for third-order aberration, where in every older eye, corneal aberrations are significantly lower than total ocular aberrations.

Figure 10 shows the values of every Zernike term (up to fourth order) for all younger (triangles) and older (circles) subjects for the anterior cornea versus the internal optics. In the younger subjects there is a significant negative correlation between corneal and internal optics aberrations ($r = 0.86$), which is clearly reduced for the older eyes

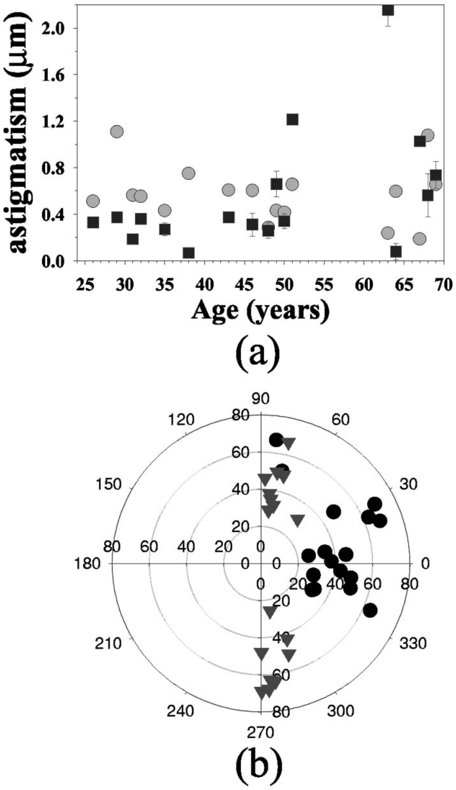

Fig. 8. (a) Astigmatism (in micrometers) for the anterior corneal surface (circles) and the eye (squares) as a function of age. (b) Polar diagram of the axis of astigmatism of the cornea (circles) and the internal surfaces (triangles) as a function of age (an unconventional notation for axis angles were used for the sake of clarity in the figure).

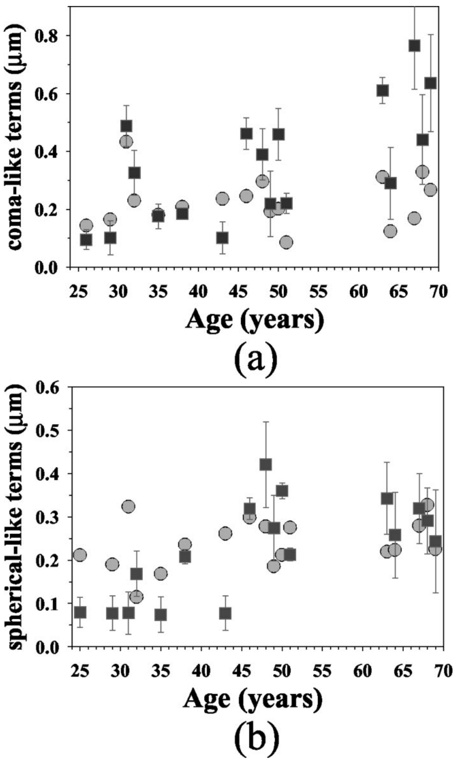

Fig. 9. (a) RMS of the third order (coma-like) terms for the cornea (circles) and the eye (squares) as a function of age. (b) RMS of the fourth order (spherical-like) terms for the cornea (circles) and the eye (squares) as a function of age (5.9 mm pupil diameter).

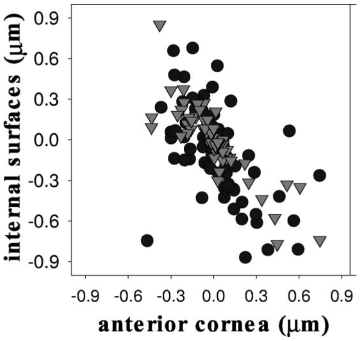

Fig. 10. Values of Zernikes terms up to fourth order for the aberrations of the cornea versus the internal surfaces, for all young (triangles) and older (circles) subjects. (Measured in micrometers; 5.9 mm pupil diameter).

($r = 0.45$). This indicates a nearly term-to-term compensation in most younger eyes that is lost with age.

4. DISCUSSION

The amount of monochromatic aberrations in the eye increases approximately linearly with age. This result has been reported in previous studies with the double-pass method,[3,4] the aberroscope,[6] and the spatially resolved refractometer.[7] Larger aberrations in the older eye produce a more degraded retinal image than in the younger counterparts. However, at least two factors reduce the impact of the increment of aberration with age on vision: senile miosis and a larger tolerance to small defocus in systems with larger aberrations.[3] In addition, it should be noted that we found some individual variability in these results. Some particularly good older eyes were similar in their aberration values to younger eyes with particularly bad optical quality. Furthermore, it is possible that there might be a larger variability in the aberrations in populations of any age with a larger range of refractive errors, but in this study we restricted ourselves to near-emmetropic eyes. This optical deterioration accounts for a significant part of the declining in contrast sensitivity with age. We measured only monochromatic aberrations, but chromatic aberrations are believed to be rather stable with age.[22]

We analyzed the aberrations of the anterior cornea and the internal optics considered as isolated systems. On average, the aberrations of the cornea increase moderately with age, although, as is clearly shown in Fig. 4, this increment is too small to account for the increase in aberrations in the complete eye. The aberrations of the internal surfaces showed a larger variability with a tendency to increase in middle-aged and older subjects. Neither ocular component itself appears to explain the change in aberrations in the entire eye. A different coupling between corneal and internal aberrations in younger and older eyes could explain the optical deterioration of the eye with age. As a simple example, one can imagine a crystalline lens with only spherical aberration similar in magnitude in young and older subjects but with opposite sign (indeed measurements *in vitro* showed this behavior[11]). Therefore the lens in younger and older eyes would be similarly aberrated, but the coupling with the cornea would produce an eye either with very little or a larger spherical aberration, respectively.

The balance of the aberrations of the anterior corneal and internal surfaces that we found in many younger and in some of the middle-aged subjects is an example of coupling of two optical systems, each with a relative poor optical quality when considered in isolation, into a system with a better optical performance. In designing optical instruments, a series of lenses is used to optimize the overall quality of the system.[23] How this retinal image-quality optimization occurs in the young eye is not yet clear, since unlike in artificial systems, there is no direct access to the isolated components. Either an active process or a simply passive consequence of some random positioning of the ocular elements could account for this phenomenon (see Ref. 14 for additional discussion of this aspect).

Whatever the reason for this compensating mechanism, we found that this mechanism is disrupted in the older eye as a consequence of normal aging. Although in this study we analyzed a relatively small number of subjects, this trend with age is rather consistent. A turning point for the aberration compensation seems to appear around 45 years of age. We should mention that what we referred to as internal ocular surfaces also includes the posterior surface of the cornea[16]: therefore it is possible that changes in the posterior corneal surface with age were responsible, at least in part, for the change of the aberration balance. However, the difference in refractive index between the cornea and the humor is approximately 10% of the difference of indices between air and cornea. Therefore the contribution of the posterior corneal surface to the aberrations will probably be modest.

Other anatomical changes in the eye with age could explain the aberration decoupling: a decentration of the eye's pupil or misalignments between the optical elements.

The results presented here also provide some explanations for well established, but not so well understood, facts. For instance, that cataract patients experience relatively low image quality after the implantation of intraocular lenses. These lenses have an extremely good image quality when measured on an optical bench, but the final optical performance in the implanted eye is lower than expected with respect to the subject's age.[24] One reason for this is that the ideal substitute for the natural lens is not a lens with the best optical performance when isolated, but one designed to compensate for the aberrations associated with the aging cornea.

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