PERGAMON

Vision Research 40 (2000) 1019–1026

Vision Research

www.elsevier.com/locate/visres

Astigmatism and the development of myopia in children

Jane Gwiazda *, Kenneth Grice, Richard Held, James McLellan, Frank Thorn

Myopia Research Center, The New England College of Optometry, 424 Beacon Street, Boston, MA 02115, USA

Received 17 May 1999; received in revised form 25 October 1999

Abstract

While it is now established that astigmatism is more prevalent in infants and young children than in the adult population, little is known about the functional significance of this astigmatism, especially its role, if any, in emmetropization and the development of myopia. Manifest refractions (mean of 16 per subject) were obtained from 245 subjects starting in the first year, with 6–23 years of regular follow-up. Results showed that infantile astigmatism is associated with increased astigmatism and myopia during the school years. Two possible mechanisms underlying this association are discussed: (1) infantile astigmatism disrupts focusing mechanisms; and (2) ocular growth induces astigmatism and myopia. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Astigmatism; Myopia; Refraction; Children

1. Introduction

While it is now established that astigmatism is more prevalent in infants and young children than in the adult population, percentages vary according to many factors, including the method of refraction, the criterion for significant astigmatism, and the children's ethnicity (reviewed in Lyle, 1991). Longitudinal studies have shown that the early cylindrical error is greatly reduced or eliminated during the first two years of life (Atkinson, Braddick & French, 1980; Gwiazda, Scheiman, Mohindra & Held, 1984; Gwiazda, Thorn, Bauer & Held, 1993a; Erhlich, Atkinson, Braddick, Bobier & Durden, 1995). Little is known, however, about the functional significance of this astigmatism, especially its role, if any, in emmetropization. Fulton, Hansen and Petersen (1982) have suggested that uncorrected astigmatism early in life could influence the development of myopia.

One possibility is that early cylindrical blur might contribute a signal, which could either disrupt or aid emmetropization of spherical power. An active feedback process of emmetropization has been proposed to operate in animals, with the rate of axial growth modulated to compensate for blur imposed by spherical lenses. However, little is known about the response of the eye to blur imposed by cylindrical lenses. In two recent reports, using monkeys (Smith, Huang & Hung, 1997) and chicks (McLean & Wallman, 1997), astigmatic defocus was reported to cause young eyes to grow slightly toward hyperopia. This suggests that astigmatic defocus could influence the mechanism producing axial growth. However, a recent study of hyperopic children (Erhlich, Braddick, Atkinson, Anker, Weeks, Hartley et al., 1997) found that changes in the cylinder power and spherical equivalent over the period from 9 to 20 months were almost independent of each other, suggesting independent mechanisms for axial elongation and corneal change in the early months of life.

Numerous reports have linked astigmatism to the development and progression of myopia in children (reviewed in Grosvenor & Goss, 1998). An oft-cited connection between spherical equivalent and cylindrical refractive error is the association of juvenile-onset myopia with against-the-rule astigmatism. In a longitudinal study of refraction from our laboratory, infants with against-the-rule astigmatism in conjunction with a negative spherical equivalent had earlier onset of myopia at school-age than infants with either with-the-rule or no astigmatism (Gwiazda et al., 1993a). Against-the-rule astigmatism in 5- and 6-year-old children is predictive of later development of myopia (Hirsch, 1964) and

* Corresponding author. Tel.: +1-617-236-6234; fax: +1-617-369-0188.

E-mail address: gwiazdaj@ne-optometry.edu (J. Gwiazda)

PII: S0042-6989(99)00237-0

faster progression of existing myopia (Grosvenor, Perrigin & Maslovitz, 1987). These data suggest a connection between spherical equivalent and cylindrical refractive error.

In order to uncover the role of early astigmatism in emmetropization and the development of refractive errors, a longitudinal study beginning at birth and carried out for a sufficient number of years is needed. Our longitudinal study of refraction, begun 23 years ago, now contains over 4000 refractions from children with a first refraction in infancy and from 6 to 23 years of regular follow-up. In this paper we confirm and extend previous reports from this laboratory tracking changes in refractive errors in children, with an emphasis on astigmatism.

2. Methods

2.1. Subjects

Refraction data from 245 subjects with a first refraction in the first year and from 6 to 23 years of regular follow-up are included. Mean age at last refraction is 12.7 years. There are 128 females and 117 males. The mean number of refractions per subject is 16. No child had strabismus, cataract, or significant eye disease. Two percent of the refractions showed anisometropia, defined as a difference of one or more diopters between the spherical equivalents in the two eyes.

Informed consent was obtained from the subjects and/or their parents after the nature and possible consequences of the study were explained to them. The research was approved by the institutional review board and followed the tenets of the Declaration of Helsinki.

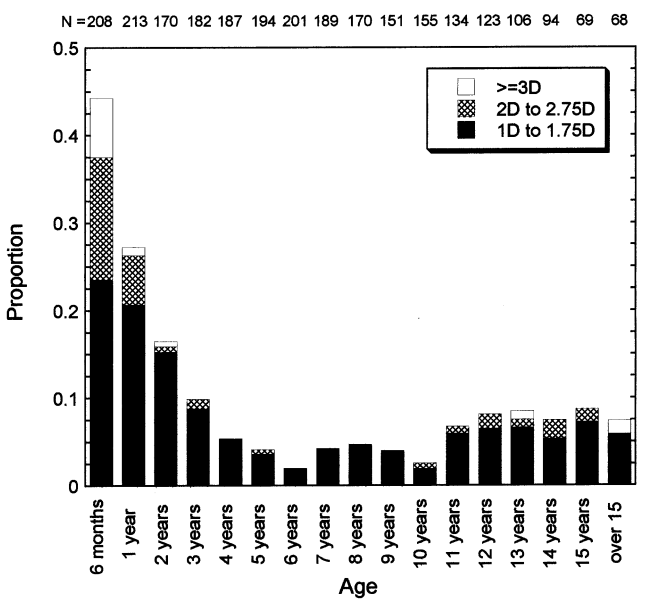

Fig. 1. Proportion of children from the longitudinal group with significant astigmatism at each age from 6 months to older than 15 years. The number of children in each age group is shown at the top.

2.2. Procedure

Refractions in the first 3.5 years were obtained by the near-retinoscopy procedure (Mohindra, 1977). Non-cycloplegic distance retinoscopy was used for children over 3.5 years. Throughout the 23-year period of the study only three optometrists have performed retinoscopy on the study population. Dr Indra Mohindra, developer of the near retinoscopy procedure, refracted children from 1974 to 1980, Dr Mitchell Scheiman refracted children from 1980 to 1982, and Dr Frank Thorn from 1982 to the present. In addition to being experienced and skilled in refracting pediatric patients, all three have published extensively on topics related to visual development. Retinoscopy was performed without prior knowledge of refractions from children in the study.

2.3. Data analysis

The refraction data, clinically written as sphere, negative cylinder power and axis, were analyzed by using a Fourier analysis of the power profile as described by Thibos, Wheeler and Horner (1997). Each refractive error was broken down into three components, the spherical equivalent (M in the Thibos notation), and two crossed cylinders, one with its meridian of maximum converging power set horizontally (J_0), and the other with its meridian of maximum converging power set obliquely (J_{45}). Means were obtained using this method, and subsequently the data were grouped by axis and/or amount for ease of understanding and the ability to compare results to previous reports.

The axis of the cylinder was classified as against-the-rule (ATR) if the minus cylinder axis was at $90 \pm 22.5°$. The axis of the cylinder was classified as with-the-rule (WTR) if the minus cylinder axis was between 0 and 22.5° or between 157.5 and 180°. Everything in between was oblique, with the most frequently occurring axes 45° in one eye and 135° in the other. Since the correlation between the two eyes for spherical equivalent is 0.96 and for cylinder power is 0.88, only the data from the right eye are reported in this paper.

3. Results

Almost half of the 0–6 month old infants have significant astigmatism (one or more diopters), but the prevalence declines sharply thereafter, as shown in Fig. 1. The prevalence reaches a minimum of less than 5%

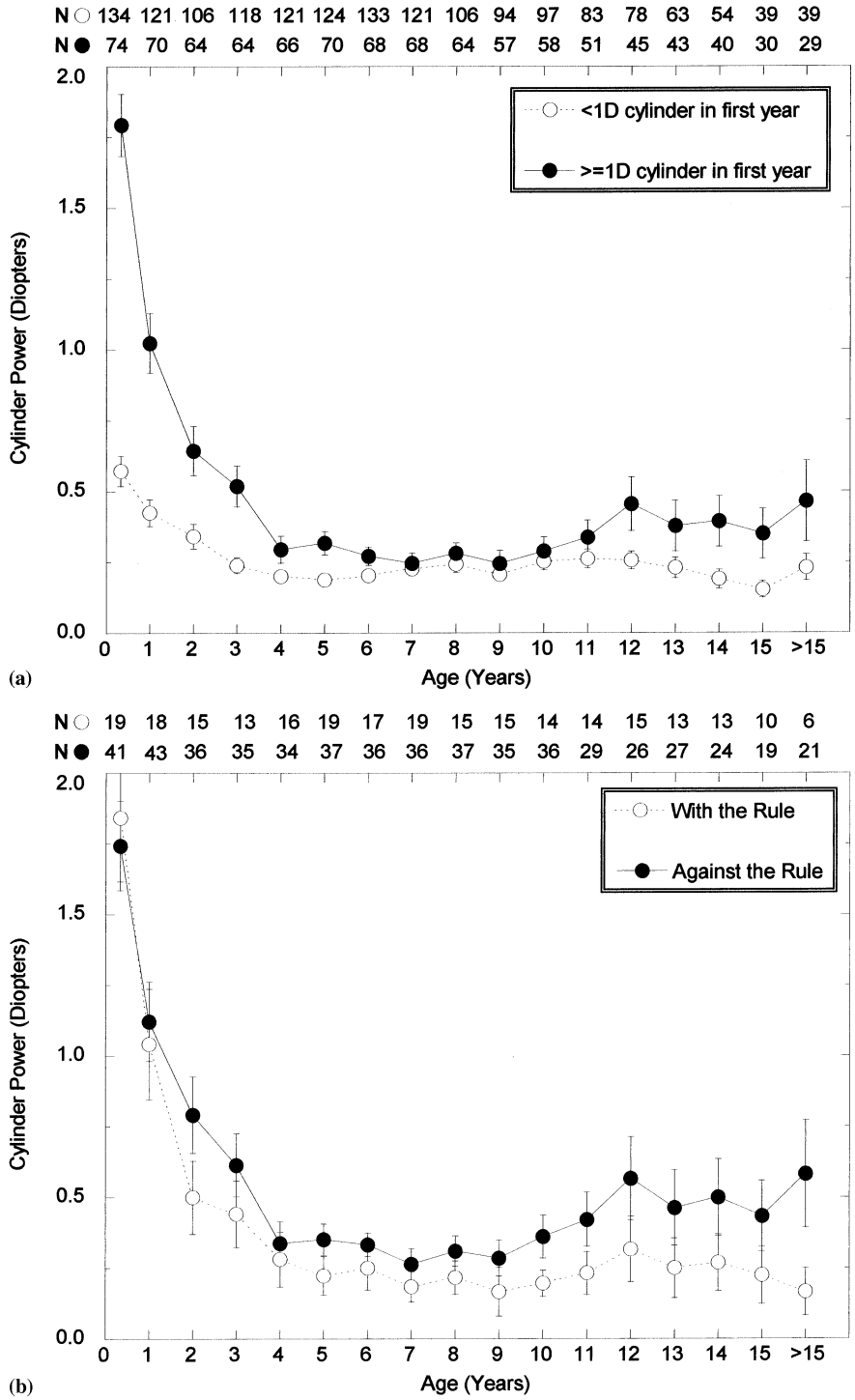

Fig. 2. (a) Cylinder power as a function of age for children with ≥ 1.0D of astigmatism in the first year (●) and for those with < 1.0D in the first year (○). The number of subjects at each age is shown at the top. Error bars represent standard errors. (b) Cylinder power as a function of age for subjects with significant against-the-rule astigmatism in the first year (●) and for those with significant with-the-rule (○). Data from subjects with oblique axis astigmatism are not shown due to the small n. The number of subjects at each age is shown at the top. Error bars represent standard errors.

from 6 to 10 years, and then slowly rises after that. The decrease from 6 months to 6 years is significant ($\chi^2 = 210.1$, $P < 0.0001$), while the later increase is not.

Fig. 2a shows cylinder power as a function of age, plotted on the basis of mean cylinder present in the first year, less than 1.0D versus greater than or equal to 1.0D. Most of the larger amounts of astigmatism found in the first year are greatly reduced or eliminated by age 4 and remain low until at least age 10. After that age, there is an increase in astigmatism for those children

who had at least 1.0D of astigmatism in infancy. A further breakdown of the significant early cylinder by axis shows that the later cylindrical increase occurs only in those children who had infantile ATR astigmatism (Fig. 2b). A repeated measures ANOVA shows a significant effect of age ($F = 3.66$, $P < 0.001$) and axis ($F = 10.34$, $P < 0.0001$), while the interaction is not significant ($F = 1.47$, $P = 0.10$). In addition, the rate of reduction of cylinder over the first 9 years does not differ for ATR and WTR.

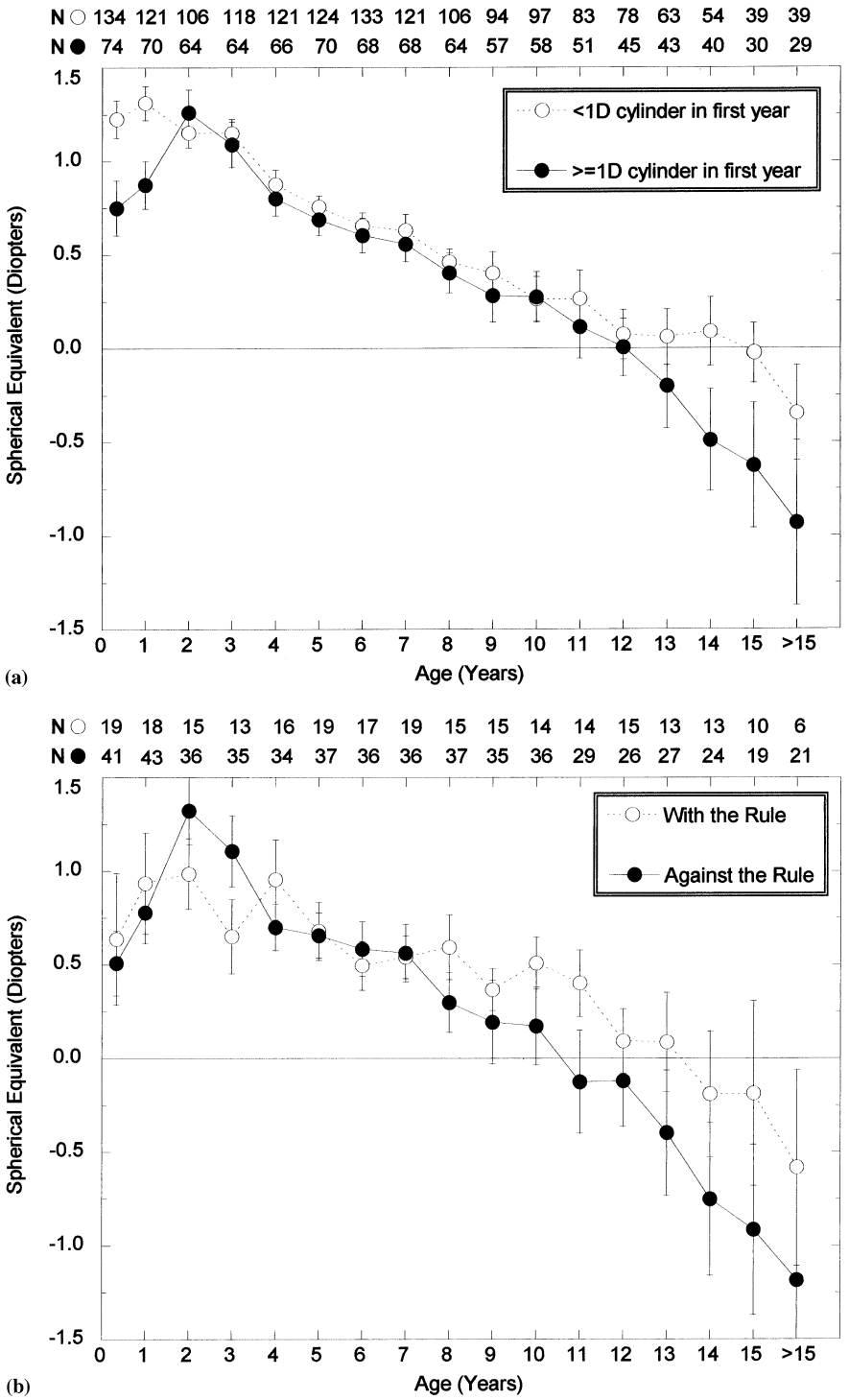

Fig. 3. (a) Spherical equivalent refractive error as a function of age for subjects with ≥ 1.0D of astigmatism in the first year (●) and for those with < 1.0D in the first year (○). The number of subjects at each age is shown at the top. Error bars represent standard errors. (b) Spherical equivalent refractive error as a function of age for subjects with significant against-the-rule astigmatism in the first year (●) and for those with significant with-the-rule (○). Data from subjects with oblique axis astigmatism are not shown due to the small n. The number of subjects at each age is shown at the top. Error bars represent standard errors.

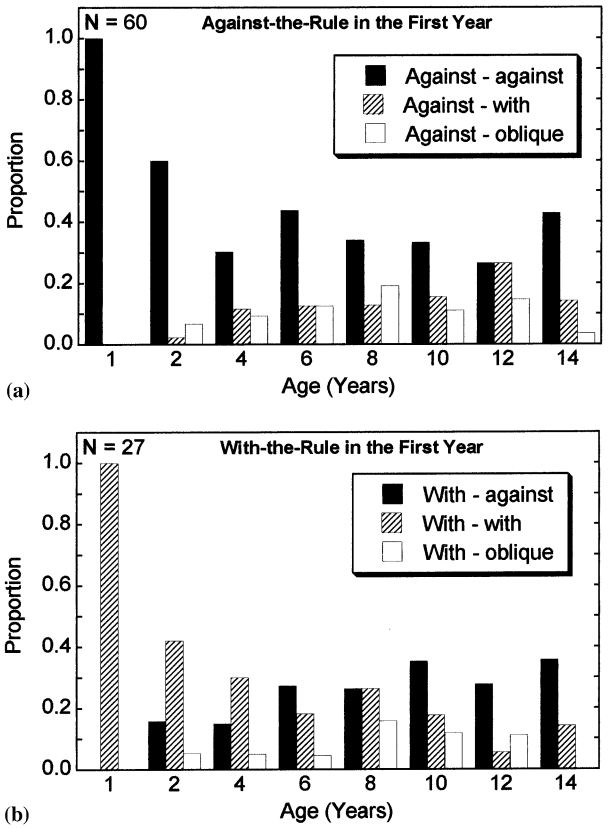

Fig. 4. (a) Change in axis of astigmatism at subsequent ages for subjects with at least 0.5D of against-the-rule astigmatism in the first year. (b) Change in axis of astigmatism at subsequent ages for subjects with at least 0.5D of with-the-rule astigmatism in the first year. Note that at ages after the first year many of the children had no astigmatism.

As shown in Fig. 3a, the spherical equivalent is less positive in the first year for children with higher amounts of cylinder in infancy. It is also less positive after age 10.

In addition, ATR astigmatism is associated with less hyperopia or more myopia after age 7 years than WTR, as shown in Fig. 3b. A repeated measures ANOVA on mean spherical equivalent refractive error (MSE) from 7 to 15 years shows a significant effect of age ($F = 20.53$, $P < 0.0001$), a marginal effect of axis ($F = 2.53$, $P = 0.08$), and a significant age by axis interaction ($F = 1.86$, $P = 0.02$).

The change in axis with age is plotted for those children who had at least 0.5D of either against-the-rule or with-the-rule astigmatism over the first year. As shown in Fig. 4a, those children who start out with ATR astigmatism tend to remain ATR. The pattern is different for those who start out with WTR, as shown in Fig. 4b. The axis of astigmatism for most of these children changes to ATR at school-age.

Fig. 5a shows the rate of change in cylinder power between the ages of 4 months (mean of 0–6 months) and 2 years plotted against the rate of change of MSE over the same period. Cylinder power is declining for most subjects, with changes in MSE occurring in both directions toward emmetropia. Negative spherical equivalents are becoming more positive and positive spherical equivalents are becoming more negative. While the correlation is only -0.20, it is statistically significant ($P = 0.017$). The rate of change of MSE and cylinder power between 2 and 6 years is not significant (Fig. 5b), with much smaller changes occurring over this age range compared to the first 2 years. Between 6 and 12 years the changes are still small (Fig. 5c), but the correlation between the changes in cylinder and spherical error is highly significant ($r = -0.43$, $P < 0.0001$). The larger changes in spherical equivalent power reflect the onset of myopic progression in some subjects, with these subjects also having an increase in cylindrical power, as shown in Fig. 5d, an enlarged version of Fig. 5c.

4. Discussion

In this paper we confirm the association between the development of school-age myopia and astigmatism, especially against-the-rule, including that found in infancy and that which increases during the school years. We have previously reported an association between infantile against-the-rule astigmatism and the development of school-age myopia with a smaller longitudinal sample (Gwiazda et al., 1993a). Parssinen (1991) has reported an association between astigmatism, especially against-the-rule, and progressing myopia in children. In a 3-year study of myopic children starting at a mean age of 11 years, the prevalence of against-the-rule astigmatism increased from 45 to 56%, while with-the-rule astigmatism increased from 10 to 18% (Parssinen, 1991).

Possible mechanisms linking astigmatism to the development of myopia are speculative at this point. Astigmatism results from asymmetries in the anterior segment. These asymmetries may involve corneal curvature or decentration; lenticular curvature, decentration, or tilt; and/or pupillary position. Spherical equivalent power involves the relationship between overall optical power in the anterior segment and axial length, but numerous human and animal studies have demonstrated that the structural change underlying most of the spherical equivalent power in progressive myopia is elongation of the vitreous chamber (Wildsoet, 1997). How might the optical asymmetries in the anterior segment underlying astigmatism be related to the vitreous chamber elongation underlying progressive myopia?

Extensions of two previously proposed hypotheses, summarized as the blur hypothesis and the ocular growth hypothesis, may help to answer this question. In

the first, astigmatism joins others factors that degrade or bias optical blur cues for emmetropization. Such cues are necessary for improving image clarity, and their absence may drive the progression of axial myopia. In the second, astigmatism is simply an offshoot of the hypothesized structural growth of the eye during axial myopia progression. These two mechanisms, discussed below, are not mutually exclusive. It is possible that either or both mechanisms could operate in the same eye.

4.1. Mechanism 1: infantile astigmatism disrupts focusing mechanisms

We and others have suggested that blur or the inability to use blur cues appropriately, combined with extensive near work, can initiate myopic progression (Gwiazda, Thorn, Bauer & Held, 1993b; Flitcroft, 1998). This blur hypothesis can involve any one of numerous factors. Spherical aberration and astigmatism are two such factors that may make the blur cue ambiguous or ineffective. Recently, Paquin, Hamam and Simonet (1998) reported that spherical aberrations are more prevalent in adult myopes than in adult emmetropes.

Infantile astigmatism may disrupt emmetropization and lead to myopia in school-age children through several possible routes. First, the chronically ambiguous blur signal of the astigmatic infantile eye may permanently reduce the sensitivity of the control mechanisms used to focus the eye. In addition, this reduction may underlie the poor accommodation to negative lens defo-

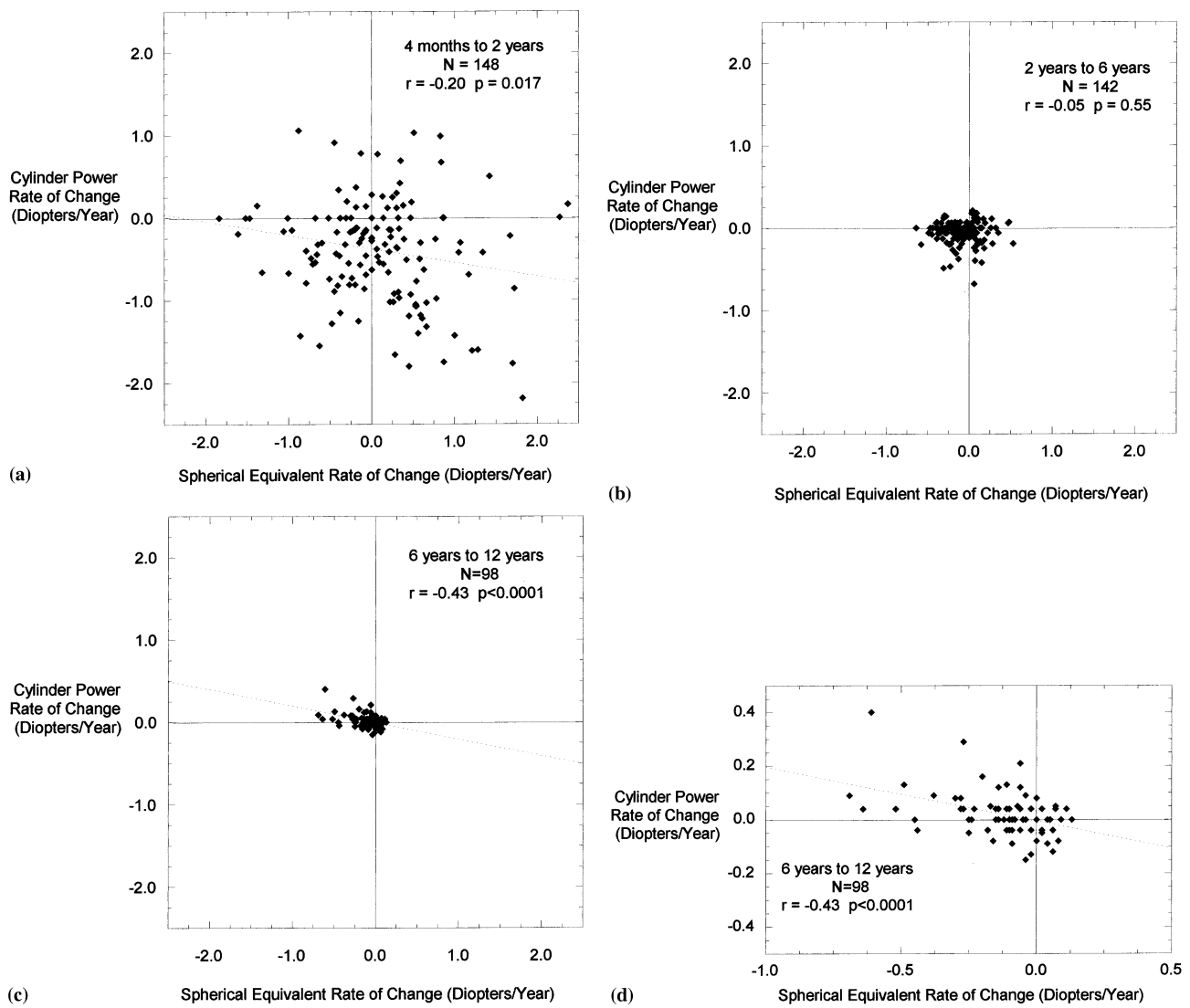

Fig. 5. (a) Rate of change (diopters per year) in cylinder power and spherical equivalent from 4 months to 2 years. (b) Rate of change (diopters per year) in cylinder power and spherical equivalent from 2 to 6 years. (c) Rate of change (diopters per year) in cylinder power and spherical equivalent from 6 to 12 years. The scale is the same for all three graphs. (d) Rate of change (diopters per year) in cylinder power and spherical equivalent from 6 to 12 years plotted on expanded axes.

cus that is seen in school-age children prior to and during myopic progression, (Gwiazda et al., 1993b; Gwiazda, Bauer, Thorn & Held, 1995) which may also underlie the disruption of emmetropization that leads to myopia.

Another possible factor that could reduce the precision of focusing ability is meridional amblyopia induced by early astigmatism. Gwiazda, Mohindra, Brill and Held (1985) have shown that astigmatism during the first year of life has little or no effect on acuity for gratings in the astigmatically blurred meridian. However, they also have shown that astigmatism occurring within the first year has a significant effect on vernier acuity at school-age for easily seen abutting gratings with small positional offsets (Gwiazda, Bauer, Thorn & Held, 1986). The detection of positional offset for two contiguous targets is very sensitive to both defocus and Gaussian blur (Westheimer, 1979; Williams, Enoch & Essock, 1984). If these blur sensitive juxtapositions are used as cues to differentiate between defocus and clarity, meridional amblyopia resulting from early astigmatism could weaken the fine control of the eye's focusing systems (either accommodation or emmetropization).

The infants who are most likely to become myopic during their school years have against-the-rule astigmatism and myopic manifest refractions in infancy. These infants tend to lose vernier sensitivity for vertically oriented gratings. Reduced stereoacuity has also been reported in the presence of meridional amblyopia (Ukwade & Bedell, 1999). The development of stereoacuity using vertical gratings is associated with the development of vernier acuity using vertical gratings (Held, Birch & Gwiazda, 1980; Shimojo, Birch, Gwiazda & Held, 1984). Held (1985) has hypothesized that the common development or loss of these cortical functions in infancy is related to the developmental course of cortical circuits that among other things are associated with the segregation of ocular dominance columns. An early weakening of stereopsis and binocular fusion is often associated with a misalignment of the eyes such as an esophoria (or even esotropia) or exophoria.

In fact, our myopic children tend to have a much wider distribution of phorias than our emmetropic children, and a significantly greater number are high esophores (Gwiazda, Grice & Thorn, 1999). In addition, the coordination of accommodation and vergence is far more variable in juvenile myopes, who tend to have high response AC/A ratios (Gwiazda et al., 1999; Mutti, Jones, Moeschberger & Zadnik, 1999). Through the cross-links in the dual interactive servosystem model (Hung & Semmlow, 1980; Schor & Kotulak, 1986), excessive esophoria or high AC/A ratios could inhibit accommodation, thereby causing the eye to be in a relatively hyperopic state during near work. One way to adapt to this hyperopic defocus is for the eye to become myopic.

In summary, infantile astigmatism may disrupt emmetropization by reducing a child's sensitivity to focusing cues. This disruption of sensitivity may lead directly to underaccommodation, which in turn induces myopia, or it may cause eye alignment problems that inhibit accommodation, thereby creating a hyperopic blur signal that can only be corrected through the induction of myopia.

4.2. Mechanism 2: ocular growth induces astigmatism and myopia

A proposed structural link between axial myopia progression and accommodation can be extended to include astigmatism (Mutti, Zadnik, Fusaro, Friedman, Sholtz & Adams, 1998; Mutti et al., 1999). It is known that corneal curvature is the major source of infantile astigmatism (Howland & Sayles, 1985). It has recently been reported that in young adults corneal astigmatism is not correlated with myopia, while total astigmatism is (Kaye & Patterson, 1997). This suggests that the astigmatic component associated specifically with myopia in older children and adults may not be corneal shape.

The crystalline lens then becomes the most likely source of myopia-associated astigmatism. The key question is how can lenticular astigmatism be associated with vitreous chamber elongation. Recent research on ocular components in juvenile eyes (Mutti et al., 1998, 1999) may provide a structurally based mechanism for the simultaneous increase in astigmatism and spherical equivalent power during the school years. The crystalline lens thins slightly during this time. Mutti et al. (1998) have suggested that as the vitreous chamber grows, its equatorial expansion pulls the ciliary body away from the lens, tightening the zonules and thus flattening the lens. Mutti et al. (1999) further suggest that the tension on the lens as it reaches its stretching limit induces a pseudo-cycloplegia in the myopic eye that results in the underaccommodation and high AC/A ratios reported for young myopes by our laboratory and others (Gwiazda et al., 1993b, 1999; Mutti et al., 1999). In the framework of this hypothesis we note that spherically asymmetric forces in the tightened ciliary muscles or zonules could induce astigmatism associated with the development of myopia. This structural link provides a plausible connection between astigmatism and myopia at school-age. This hypothesis, however, cannot explain the connection between infantile astigmatism and the later development of myopia or answer the basic question of what drives eye growth.

5. Summary

In summary, infantile astigmatism, especially against-the-rule, is associated with increased astigmatism and

myopia during the school years. The mechanisms underlying this association are not well understood at present, but their clarification should provide insight into the development of juvenile-onset myopia. Two possible mechanisms, which are not mutually exclusive, have been proposed. Further investigations of their implications will determine their contribution to our understanding of myopia.

Acknowledgements

This work was supported by NEI R01 EY01191. We would like to thank the families for their ongoing participation in these studies and to thank Sarah Boocock for editorial assistance.

References

Atkinson, J., Braddick, O., & French, J. (1980). Infant astigmatism: its disappearance with age. *Vision Research*, *20*, 891–893.

Erhlich, D. L., Atkinson, J., Braddick, O., Bobier, W., & Durden, K. (1995). Reduction of infant myopia: a longitudinal cycloplegic study. *Vision Research*, *35*, 1313–1324.

Erhlich, D. L., Braddick, O. J., Atkinson, J., Anker, S., Weeks, F., Hartley, T., Wade, J., & Rudenski, A. (1997). Infant emmetropization: longitudinal changes in refraction components from nine to twenty months of age. *Optometry and Vision Science*, *74*, 822—843.

Flitcroft, D. I. (1998). A model of the contribution of oculomotor and optical factors to emmetropization and myopia. *Vision Research*, *38*, 2869–2879.

Fulton, A., Hansen, R., & Petersen, R (1982). The relation of myopia and astigmatism in developing eyes. *Ophthalmology*, *89*, 298–302.

Grosvenor, T., & Goss, D. A.. (1998). Role of the cornea in emmetropia and myopia. *Optometry and Vision Science*, *75*, 132–145.

Grosvenor, T., Perrigin, D., & Maslovitz, B. (1987). Houston myopia control study: a randomized clinical trial. Part II. Final report by the patient care team. *American Journal of Optometry and Physiolocial Optics*, *64*, 482–498.

Gwiazda, J., Scheiman, M., Mohindra, I., & Held, R. (1984). Astigmatism in children: changes in axis and amount from birth to six years. *Investigative Ophthalmology and Visual Science*, *25*, 88–92.

Gwiazda, J., Mohindra, I., Brill, S., & Held, R. (1985). Infant astigmatism and meridional amblyopia. *Vision Research*, *25*, 1269–1276.

Gwiazda, J., Bauer, J., Thorn, F., & Held, R. (1986). Meridional amblyopia does result from astigmatism in early childhood. *Clinical Vision Sciences*, *1*, 145–152.

Gwiazda, J., Thorn, F., Bauer, J., & Held, R. (1993a). Emmetropization and the progression of manifest refraction in children followed from infancy to puberty. *Clinical Vision Sciences*, *8*, 337–344.

Gwiazda, J., Thorn, F., Bauer, J., & Held, R. (1993b). Myopic children show insufficient accommodative response to blur. *Investigative Ophthalmology and Visual Sciences*, *34*, 690–694.

Gwiazda, J., Bauer, J., Thorn, F., & Held, R. (1995). A dynamic relationship between myopia and blur-driven accommodation in school-aged children. *Vision Research*, *35*, 1299–1304.

Gwiazda, J., Grice, K., & Thorn, F. (1999). Response AC/A ratios are elevated in myopic children. *Ophthalmic Physiological Optics*, *19*, 173–179.

Held, R., Birch, E., & Gwiazda, J. (1980). Stereoacuity of human infants. *Proceedings of the National Academy of Science USA*, *77*, 5572–5574.

Held, R. (1985). Binocular vision — behavioral and neuronal development. In J. Mehler, & R. Fox, *Neonate congnition: beyond the blooming, buzzing confusion* (pp. 37–44). Hillsdale, NJ: Lawrence Erlbaum Associates.

Hirsch, M. (1964). Predictability of refraction at age 14 on the basis of testing at age 6 — Interim report from the Ojai longitudinal study of refraction. *American Journal of Optometry Archives and American Academy of Optometry*, *41*, 567–573.

Howland, H., & Sayles, N. S. (1985). Photokeratometric and photorefractive measurements of astigmatism in infants and young children. *Vision Research*, *25*, 73–81.

Hung, G. K., & Semmlow, J. L. (1980). Static behavior of accommodation and vergence: computer simulation of an interactive dual-feedback system. *IEEE Transactions in Biomedical Engineering*, *27*, 439–447.

Kaye, S., & Patterson, A. (1997). Association between total astigmatism and myopia. *Journal of Cataract and Refractive Surgery*, *23*, 1496–1502.

Lyle, W. (1991). Astigmatism. In T. Grosvenor, & M. Flom, *Refractive anomalies: research and clinical applications* (pp. 146–173). Boston: Butterworth-Heinemann.

McLean, R. C., & Wallman, J. (1997). Despite severe imposed astigmatic blur, chicks compensate for spectacle lenses. *Investigative Ophthalmology and Visual Science*, *38*, S542.

Mohindra, I. (1977). A non-cycloplegic refraction technique for infants and young children. *Journal of the American Optometric Association*, *48*, 518–523.

Mutti, D. O., Zadnik, K., Fusaro, R. E., Friedman, N. E., Sholtz, R. I., & Adams, A. J. (1998). Optical and structural development of the crystalline lens in childhood. *Investigative Ophthalmology and Visual Science*, *39*, 20–33.

Mutti, D. O., Jones, L., Moeschberger, M., & Zadnik, K. (1999). AC/A ratio, age, and refractive error in children. In *Vision science and its applications*, *OSA technical digest* (pp. 193–196). Washington, DC: Optical Society of America.

Paquin, M., Hamam, H., & Simonet, P. (1998). Objective measurement of optical aberrations for myopic eyes. *Optometry and Vision Science Supplement*, *75*, 234.

Parssinen, O. (1991). Astigmatism and school myopia. *Acta Ophthalmologica*, *69*, 786–790.

Schor, C., & Kotulak, J. C. (1986). Dynamic interactions between accommodation and convergence are velocity sensitive. *Vision Research*, *26*, 927–942.

Shimojo, S., Birch, E., Gwiazda, J., & Held, R. (1984). Development of vernier acuity in infants. *Vision Research*, *25*, 88–92.

Smith, E. L. III, Huang, R., & Hung, L. (1997). Cylindrical spectacle lenses alter emmetropization and produce astigmatism in young monkeys. In T. Tokoro, *Myopia updates* (pp. 336–343). Tokyo: Springer.

Thibos, L. N., Wheeler, W., & Horner, D. (1997). Power vectors: an application of Fourier analysis to the description and statistical analysis of refractive error. *Optometry and Vision Science*, *74*, 367–375.

Ukwade, M. T., & Bedell, H. E. (1999). Stereothresholds in persons with congenital nystagmus and in normal observers during comparable retinal image motion. *Vision Research*, *39*, 2963–2973.

Westheimer, G. (1979). The spatial sense of the eye. *Investigative Ophthalmology and Visual Science*, *18*, 893–912.

Wildsoet, C. (1997). Active emmetropization: evidence for its existence and ramifications for clinical practice. *Ophthalmic and Physiological Optics*, *17*, 279–290.

Williams, R. A., Enoch, J. M., & Essock, E. A. (1984). The resistance of selected hyperacuity configurations to retinal image degradation. *Investigative Ophthalmology and Visual Science*, *25*, 389–399.