

A REVIEW OF THE PREVALENCE AND CAUSES OF MYOPIA

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ABSTRACT

In this study, we reviewed the prevalence of myopia by country. Different types of myopia are elaborated and the causes of myopia are presented. It appears that the origin of myopia is due to both environmental and genetic factors.

Keywords: Prevalence, nature and causes of myopia.

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PREVALENCE

For over a century, many studies of the prevalence of myopia have dealt with select populations making comparisons and generalizations difficult (1,2). Surveys of refractive error have been performed on students, army recruits, eye clinic patients, different age groups, and races. Such data are difficult to analyze due to different methods of refraction, methods of analysis, and definitions of myopia (3).

On the other hand, prevalence data can be useful in searching for etiologic mechanisms especially if the sample characteristics and sample size information are known. Changes with age and variations with race are especially important in this regard, as well as information as to general distribution.

Practitioners are not necessarily enlightened by lengthy lists of studies and statistics such as those provided in standard texts (4-6). The purpose of this review is therefore to present some of the most recent information on this subject and landmark studies rather than to be exhaustive.

Also, there is a general lack of agreement that myopia is a significant abnormality of health even though it was estimated that its annual cost in the mid-1980's in the U.S. was over \$4 billion (4).

In one of the largest early studies, Sheerer and Betsch published data from more than 12,000 clinical patients in 1928 and 1929 in Germany. The patients were over 25 and refractions were performed without cycloplegia (1,4-6). 13% were found to be myopic (4). The distribution curve was published by Duke-Elder and was subsequently analyzed by Stenstrom, who concluded that not only is it more leptokurtic than a

normal distribution but it is also skewed towards myopia. That is, there is a greater incidence of myopia than would be presented if refractive errors were normally distributed (1). Although the Sheerer and Betsch study only included a clinical sample, and we know those with refractive errors (especially myopia) are more likely to present themselves for vision care (3), it is often quoted in the literature as representing general populations.

Emmetropization is the term often employed to explain the finding that the frequency distribution of each of the dioptric components of the eye is substantially normal (6). The refractive curve of infants is almost normal (7) and yet the distribution of refractive powers of adult eyes is sharply peaked at or near emmetropia (8). However, the leptokurtic nature with a peak at emmetropia can be expected even on the basis of chance association (8,9). Some studies seem to indicate that the frequency distribution of human eyes peaks more nearly at about a diopter of hyperopia and that there is a slight favour toward a hyperopic design for the human eye (1,8,10).

Several recent studies of myopia in the U.S. are of interest. Leibowitz and associates found myopia to be present in 17.7% of eyes in the Framingham Eye Study population (11). The Framingham Eye Study of 1973-75 was a study of 2631 adults who were survivors of the Framingham Heart Study cohort in the town of Framingham, Massachusetts, and were therefore an ageing population. These subjects were not selected in terms of refractive status.

In 1971-81 as part of the US National Health and Nutrition Examination Survey (HANES), a national probability sample of 14,147 persons, aged 1 through 74 years, was selected to represent the 192.7 million persons in the civilian US population of that age at the time of the survey. Sperduto analyzed these data to obtain national prevalence estimates of myopia for persons age 12 to 54 years based on the refractions of 5282 persons (2). He found 25% were myopic, with significantly lower prevalence rates for male subjects than for females and for blacks than for whites (2). The incidence of myopia, that is, the number of persons who become myopic in a given period of time, was greatest between the ages of 12 and 17 years (2).

The incidence of myopia was given to Safir in 1979 as being 15-20% of the population (12). Young in 1980 showed that about 5% of the adult population has myopia greater than 4D (13).

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Differences in refraction between the sexes have been reported by a number of authors (1,3,4) indicating that higher degrees of myopia are predominant in females, but this is probably not true at all ages and with all ethnic groups (1,9,14).

Many investigators have noted that certain groups appear to have a high incidence of myopia. Historically, the highest incidence was reported among Chinese, Japanese, Egyptians, Germans, Jews and Middle Eastern peoples, and the lowest incidence was among Negroes, Eskimos and Indians (1,4,10,15). However for native populations, variations with age possibly due to changing lifestyle (15), schooling (16), or diet (17), and an excess of young persons in the sample can affect percentages reported. For example, Woodruff and Samek (17) report a mean refraction of about one-half a diopter of myopia in the Amerind population of Ontario, but they note an excess of myopia among teenagers and young adults, and a relative excess of hyperopia in older persons. 52% of their sample was made up of

children between ages 5 and 20.

A similar difference of refraction between age groups was reported in other native populations. Young et al report 30% myopia in Alaskan Eskimos (16) but 56% of their sample was 6 to 25 years, a group with much higher amounts of myopia than Eskimos of other ages. For example, there was virtually no myopia among the grandparents or parents, but approximately 58% of the offspring were myopic (16). Young also found a correlation between age and hypermetropia for the fathers but not for the mothers. In West Greenland where a written Eskimo language has been taught for 100 years, Alsbirk found the refraction of Eskimos over 15 years showed an emmetropic excess and a myopic skewness (15). Myopia greater than 1 diopter was present in 14.1%. In these Eskimos, Alsbirk observed that only females showed a trend to hyperopia with age (15).

Table I shows a few representative studies of the prevalence of myopia in various countries.

Table I
PREVALENCE OF MYOPIA BY COUNTRY

COUNTRY	% MYOPIA	AUTHOR	REFERENCE
China, students	53	Li (1920)	18
China, Peking	52	Dzen (1921)	19
Germany	13.8	Witte (1923)	20
US	19.6	Jackson (1932)	21
UK	27	Harman (1936)	22
China	70	Rasmussen (1936)	23
Sweden	33	Nordgren (1936)	24
US, army >-2.5 D	3.16	Downing (1945)	25
UK	24.2	Giles (1950)	26
UK, army	11	Sorsby (1960)	27
US	25-35	Hirsch (1964)	28
Fiji	0	Rose (1964)	29
India	22	Kuriakose (1967)	30
Sweden, army	14.5	Goldschmidt (1968)	31
Israel, communes	18.4	Hyams (1977)	32
US, Mass, age 52-85	17.7	Leibowitz (1980)	11
US, age 12-54	25	Sperduto (1983)	2

Crawford and Hammar conducted a screening of more than 50,000 school children in Hawaii (33). They reported myopia rates as follows: Chinese 17%, Koreans 13%, Japanese 12%, Caucasians 12%, Spanish 9%, Portuguese 7%, Filipinos 6%, Puerto Ricans 4%, partial Hawaiians 4%, Hawaiians 3% and others 3% (33).

Studies of refractive changes with age characteristically indicate a relative excess of hyperopia in infants, an increase in myopia between birth and adulthood, a stability of mean refractive status through early adulthood, then a change in mean refractive status towards less myopia or more hyperopia after age 40 (1,2,4,7,10,28,34-36).

The problem of determining refractive status of infants is complicated in that there can be wide variations in accommodative ability in infants. Also birth weight and time elapsed since birth has an effect on refraction (4). Thus the literature has reports both of high incidence of hyperopia and myopia, with and without cycloplegia. (1,7),

Gasson's 1932-33 study of changes of refractive state with age was divided into those showing hyperopia and those showing myopia (37). A marked difference between males and females appeared at 45-50 years, where male hyperopes showed a sharp trend toward a decrease in hyperopia and male myopes showed a decrease in myopia, while females showed

little mean change. At 60-65 years, male myopes showed a trend toward greater myopia (1). More recently, Grosvenor in 1977 published the data from a longitudinal study of refractive changes between ages 20 and 40 for 111 subjects. Grosvenor concluded that hyperopes tend to become more hyperopic and myopes tend to become more myopic during these years, also that the more myopic a subject was at age 20, the greater the increase in myopia which might occur by age 40. (34).

There appears to be a tendency to higher myopia in city as opposed to rural populations, and higher socioeconomic groups as opposed to lower (1,4). The association of myopia with both higher income and educational level has been noted in many reports, and is thought by some investigators to be closely related to nearwork (2,4,38,39). Further analyses are needed to explain how the various factors of age, race, sex, education and income related to one another and to myopia (2).

NATURE AND CAUSES

A study of refraction makes it clear that myopia has received the most attention (9,12). There are serious conditions associated with myopia, but it is not the only refractive condition associated with tissue destruction

or loss of function (12). It is however a symptom easily recognized, in contrast to hyperopia which is often hidden (12,40). In addition, myopia often makes its appearance during adolescence when so many things are changing rapidly. During this time, a great emphasis is placed on learning social functions, athletic skills and relating to members of the opposite sex. Also there is increasing demand for performance at school. The need to wear corrective lenses can create more difficulties for parents and children (12).

TYPES OF MYOPIA

Pathological myopia, which has been defined as progressive elongation of the globe with degeneration, is frequently associated with destructive choroidal and retinal lesions, as well as retinal detachment, and higher incidence of glaucoma and cataract (4,12,39,41). The axial elongation often causes peripheral fundus changes and posterior staphyloma because the elongation affects two areas – the ora-equatorial area and the posterior pole (42). There is a general agreement that high myopia is essentially a hereditary disease. (4).

In 1948 Stenstrom showed that the total refracting power of the eye had little or very slight correlation to the various dioptric elements of the eye, but axial length was different and showed a pronounced correlation (0.76 ± 0.014) with refractive error (43). He considered myopia of more than -8.00 diopters to be pathological, and believed that the skewness and frequency of extreme variables in the distribution of refractive error could be wholly explained by increased axial length, and many subsequent authors have agreed (39,40,44). The axial development of the vertebrate eye is the result of the interplay of intraocular pressure and scleral resistance; also included in the process of development is an element of stretch (45,46). The eye develops rapidly postnatally and this elongation would result in many eyes becoming myopic if it were not for compensating reduction in lens and corneal power (34,36,40,44,45,47), referred to as emmetropization. Pathologic myopia is readily distinguished from physiologic myopia for it is more likely to be a congenital condition in which excessive axial elongation can be detected at all stages of development (39). Nearwork is not thought to play a role in pathogenesis of high myopia (4,39)

Physiologic, or non-pathological myopia is thought to occur as the result of correlation failure of the refractive components of the normal eyes (4,9,12,39,44). The fundus appears normal in physiologic myopia (39). Every conceivable cause has been advanced to account for this condition (1,3,9,10). Various investigators have implicated convergence (4,39), head position (48), squeezing by the extraocular muscles (49,50), posture (4,48,51,52), illumination (4,48,51,53,54), higher levels of intraocular pressure (4,39,45), increased vitreous pressure (4,47,55), excessive pressure on the eye by eyelids or eye rubbing (4,40,49,56), scleral weakness (50), or congestion (4), motility imbalances (4), uncorrected corneal astigmatism or exophoria (4), contact lens over-correction of the non-dominant eye (57), dietary deficiencies (1,4,9,17), lack of calcium (1,4,58,59), eating excessive amounts of sugar and overcooked proteins (60), high urine concentrations of acid mucopoly saccharides (1), infectious disease (1,4,17), high incidence of dental caries (1,3,4,17) and "stretching of the posterior segment" (34). Morgan suggests a number of other possible causes of myopia including; endocrine imbalance, hypofunction of the thyroid, ratio of height to weight, protective mechanisms of introverted personality, and hybridization of genes of different races (9). Curtin in 1970 proposed three possible causative mechanisms; a

mesodermal mechanism, an ectodermal mechanism, and disparity in growth of ectoderm and mesoderm (61). More recently Curtin has argued for the division of the lower grades of myopia into "physiologic (low, simple)" and "intermediate", with temporal crescent formation as a sign of intermediate myopia (4).

In addition, myopia can be considered to be divided into functional or organic types. Organic changes can cause myopia of a transient or permanent nature at any age, for example; keratoconus, lens hydration, diabetes, dysentery, lens subluxation, nuclear cataract, drugs such as acetazolamide and oxytetracycline, as well as hyperbaric oxygen treatment and malnutrition (39). Some authors also divide functional into "pseudomyopia" which they consider to be reversible, and "simple", "true" or "school myopia" which is considered to be irreversible (4,47,62). Pseudomyopia has been attributed to accommodative spasm, edema of the ciliary body, swelling of the lens and transient increase of refractive index of the lens, vitreous or aqueous (4).

HEREDITY AND ENVIRONMENT

The inheritance of refractive error is generally agreed to be multifactorial (15,39) and polygenetic (4). That one basic determinant of refraction is heredity is borne out by a concordance of 70.6% to 90% between monozygous twins (4,44). There appear to be racial differences in the distribution of myopia. Unfortunately the results of studies of racial differences cannot be interpreted to attribute myopia to genetic causes (9,63).

Although heredity may be the primary cause for myopia, environment can have substantial effects, as is shown by the effect of maternal rubella, drugs and prematurity in causing myopia in the newborn (4,39,64,65). Young native people in North America who have attended school appear to be much more myopic than their parents (16,17); a trend which has been referred to as an epidemic of myopia in the youth (17). This evidence suggests a strong environmental influence (16,56).

NEARWORK AND ACCOMMODATION

Nearwork has been thought to be a cause of functional myopia since Cohn in 1867 presented the evidence that the percentage of German school children with myopia was directly related to the number of years in school (9,40,66). Tscherning, Witte and Tiffin among many others have espoused this nearwork theory (4,9,15,56). Recent studies have found an unusually high incidence of nearsightedness among those whose occupations require them to do nearwork (40,58,63). For example, in 1979 Richler and Bear found myopia significantly correlated with nearwork for people ages 5 to 60 in three Newfoundland communities (36). However there is no indication whether the nearwork induced the myopia or the myopic individuals choose to do nearwork (9,40). Myopia does appear to develop during school years but it has not been conclusively shown whether nearwork influences its onset or development (9).

Many investigators have implicated accommodation as the cause of myopia (4,9,47,49,55,67). One theory is that accommodation leads to a permanent increase in the convexity of the lens surface since the ciliary muscle holds the lens in position as new lens fibres are laid down (55). There is also some evidence of increased vitreous pressure during accommodation which could cause an increase in axial length (4,47,68). Ciliary muscles spasm is said to be a common effect of nearwork in the adolescent and young adult (10,39).

Van Alphen felt that ciliary muscle tone was responsible for the emmetropization process and reduction of hyperopia postnatally, this process being controlled by a feedback loop through the Edinger Westphal nucleus (45,46). Dramatic effects have been reported with cycloplegia; there is a shift toward hyperopia and a reduction in the excess at emmetropia (47,69). Transient myopia after visual work has been shown to occur after as little as three hours of editing text at a video display terminal, which has been interpreted as a work-induced shift of accommodation toward the resting focus (70,71).

ANIMAL MODELS

Animal studies support the view the environment has an influence on myopia (4,40,41,49,54,72). There is evidence that myopia can be caused in laboratory animals by suturing eyelids shortly after birth (41,56) by elevated intraocular pressure with increased body temperature (73,74), by restricted visual space (54,56,75,76), and by gravitational effects caused by restriction of body position with the eyes facing downward (49,52). Young found that myopia did not develop in confined primates if that illumination was high or low, and attributed myopic changes to moderate levels of illumination (53).

Based on animal models it has been proposed that development of emmetropic refractive status is dependent on normal visual experience and early visual deprivation results in a shift toward myopia mediated by the nervous systems in a young animal, but can only be induced before eye growth is completed (4,40,41,48,54,56,77). Such visual deprivation could be caused in human infants by ptosis, hemangiomas of the eyelid, corneal opacities, congenital cataract, or retrolental fibroplasia (4,40,56,78). In different experiments employing lid suturing and injecting polystyrene beads into the corneal stroma, Raviola and Wiesel have found that some types of monkeys who were visually deprived developed myopia in the deprived eye, but myopia could be prevented with a daily application of atropine, or by rearing the animals in the dark (40). In other types of monkeys, myopia developed despite atropine and even if the optic nerve was cut or the visual cortex was removed (41,56). In these experiments one type of monkey who was visually deprived developed myopia and another type did not, under the same conditions. Raviola and Goss believe that the mechanisms leading to myopia in one type of animal may not be the same as that in another animal (41,54).

Raviola and Wiesel suggest that emmetropization is largely programmed on a genetic basis and that some sort of regulatory molecule may be released by the retina to fine-tune the eye growth, but that an abnormal visual experience can disrupt the process of postnatal eye growth and induce axial myopia (40,41). They believe that accommodation has only a small effect in determining the focal length of the eye at rest, and they further suggest that some children may be destined, like some types of monkeys, to develop myopia through excessive accommodation (40,41). Such myopia may be triggered environmentally or

genetically. These children may benefit from the use of atropine. But for children whose myopia is unrelated to accommodation, like the other type of monkeys studies, atropine would be useless (40).

OTHER CAUSES

Goss found myopia stops increasing earlier in teenage girls, which he attributes to the fact that general body growth tends to cease earlier in girls. His findings suggest that hormonal or growth related changes of adolescence are causative (35).

There are conflicting reports of the refractive status of amblyopic eyes and non-amblyopic "fellow" eyes. Lepard reported his findings of an unusual course of refraction in a longitudinal study of children with unilateral amblyopia (79). With normal use, the fixating non-amblyopic eye has found to undergo a steady drift toward myopia, whereas the refraction of the amblyopic fellow eye remained stable. This would seem to contradict other studies showing that the eye which is visually deprived becomes myopic (78,80,81). On the other hand, Woo in 1970 studied the refractive error distributions of monocularly amblyopic eyes, the non-amblyopic "fellow" eyes, and an equal number of amblyopia-free eyes in a cross sectional study of Ontario grade-school children. The refractive error distributions obtained for the three types of eyes were differently distributed. Also his results indicate the refractive error distribution of amblyopic eyes has a greater frequency of hyperopes than the distribution of non-amblyopic eyes, which in turn has more hyperopic eyes than the distribution of normal eyes (82). Woo and Irving repeated the study ten years later with subjects aged 5-55 and obtained essentially the same results. That is, the amblyopic eyes tend to have a greater frequency of hyperopia than normal eyes (83).

The trend toward progressively more low myopes and corresponding fewer moderate high myopes with advancing age has not been adequately explained. Possible explanations include a decrease in power of the lens, or a cohort effect in which more recent birth cohorts are at a greater risk of the development of more severe myopia (2). In addition, Morgan examined the possibility that earlier mortality for myopes might account for the reduced frequency of hyperopes in older population (9).

The evidence seems to be that both the environmental and the genetic schools are right (40). Environmental factors can and do affect refraction and if nearwork can cause myopia, it is only the physiologic variety (7,39).

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