SCIENTIFIC REPORT

Inferring myopia over the lifecourse from uncorrected distance visual acuity in childhood

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Aim: To report the usefulness of uncorrected distance visual acuity (DVA) at 16 years to ''screen'' for myopia status and to assess the lifetime risk of myopia, based on a national birth cohort.

Methods: 1867 members of the 1958 British birth cohort for whom there were data on acuity at 16 years had autorefraction, as part of a biomedical survey, at 45 years. Reduced uncorrected DVA at age 16 years (6/12 or worse in both eyes) was compared with adult refraction (spherical equivalent).

Results: Only a quarter of individuals in the population studied who had developed myopia by 45 years of age had reduced acuity at 16 years of age. Notably, half of all adults with moderate myopia (−2.99 to −5.99) and 31% (11/35) with severe myopia (⩾−6) had good uncorrected DVA in both eyes at 16 years of age. Thus, sensitivities were low, ranging from 16% for all myopia (cut-off point spherical equivalent −0.5) to 69% for severe myopia (cut-off point spherical equivalent −6). However, a high (91%) lifetime probability of primary myopia (spherical equivalent ⩾−0.5) given a reduced uncorrected DVA at 16 years was found.

Conclusion: In this population, reduced uncorrected DVA in childhood is an inaccurate and inappropriate intermediate ''phenotype'' for capturing adult myopia status. However, our findings support assessment of DVA in secondary school children as an effective method of identifying refractive error (both myopia and hypermetropia).

Myopia is an increasingly important global public health problem. In the past two decades, the prevalence among young adolescents has increased from 5–10% to 10–25% in industrialised societies in Europe and North America, and by over 25% to 60–80% in East Asia.[1–3] The role of genetic factors in the aetiology of myopia is well established,[4] particularly in relation to familial or syndromic high myopia,[5 6] but environmental factors must account for the marked secular change in frequency.[7] The way in which environmental and genetic influences combine to affect the development and progression of myopia over the lifetime remains a research priority. There has been recent interest in assessing uncorrected visual acuity as a proxy or intermediate phenotype for myopia: an uncorrected visual acuity of 6/12 Snellen equivalent acuity has been reported to be the optimal threshold for diagnosing myopia.[8–10]

On the basis of a longitudinal national birth cohort study, we report on the usefulness of uncorrected visual acuity, by age 16 years, to ''screen'' for myopia status and as a means to assess the lifetime risk of myopia.

METHODS

The 1958 British birth cohort comprises everyone born in Britain in one particular week in 1958.[11] Members have been followed since birth using clinical examination or face-to-face interview. Data have been collected on educational, social and lifestyle factors. The current study was part of our programme of work on vision and ophthalmic disorders in the 1958 British birth cohort (the other findings of which will be reported elsewhere). It is based on cohort members who had uncorrected visual acuity measured at 16 years of age and subsequently had autorefraction, as part of a broader biomedical assessment at 44/45 years.[12]

At 16 years, distance vision in each eye was assessed using a standard Snellen chart, both with and without any prescribed optical correction. Children with eye diseases that would have accounted for reduced acuity, such as, congenital cataract, were excluded. In the remaining children, an uncorrected visual acuity of 6/12 or worse in both eyes was assessed as the proxy/intermediate phenotype for myopia.

At 44/45 years, a random sample of members of the 1958 British birth cohort had autorefraction (Nikon Retinomax 2, without cycloplegia). Spherical equivalent values were calculated using the standard formula of the algebraic sum of the dioptric powers of the sphere and half of the cylinder (sphere +0.5 cylinder).

To examine the distribution, refraction in adulthood was categorised into six mutually exclusive categories, based on spherical equivalent of the less extreme eye (ie, the smaller absolute spherical equivalent difference from zero): ⩾−6.00, −5.99 to −3.00, −2.99 to −1.00, −0.99 to −0.50, −0.49 to +0.99, and ⩾+1.00.

Statistical methods

Categorisation of myopia status on the basis of uncorrected distance vision in childhood (ie, ''present'' if 6/12 or worse, or ''not present'' if 6/9 or better) was compared with adult refraction.

Spherical equivalent measurements were summarised using median, interquartile range (IQR 25%, 75%) and range. For investigation of sensitivity, specificity and predictive value, the following ''cutpoints'' for myopia were examined: ⩽−6.00, ⩽−3.00, ⩽−1.00, ⩽−0.75 and ⩽−0.50.

Sensitivity (proportion of adults with myopia correctly identified by reduced uncorrected vision in childhood) and specificity (proportion of adults without myopia correctly identified by good uncorrected vision in childhood) were calculated separately for each defined cutpoint for myopia described above. The positive predictive value (PPV; probability of being myopic in mid-adult life given reduced vision in childhood) was used to estimate the correlation of childhood visual acuity with lifetime myopia status.

This study is part of a broader programme of work approved by the Institute of Child Health's Research Ethics Committee and the South East Multi Centre Research Ethics Committee (ref: 01/1/44).

Abbreviations: PPV, positive predictive value

Table 1 Distribution of spherical equivalent at age 45 years by uncorrected distance acuity at 16 years

	Uncorrected distance visual acuity at 16 years	
SE at 44 years	6/5–6/9 n (%)	6/12 or worse n (%)
⩾−6	11 (0.6)	24 (13.9)
−5.99 to −3	83 (4.9)	84 (4.9)
−2.99 to −1	399 (23.6)	45 (26)
−0.99 to −0.5	339 (20)	6 (3.5)
−0.49 to +0.99	753 (44.4)	10 (5.8)
⩾+1	109 (6.4)	4 (2.3)
Total	1694	173

SE, spherical equivalent.

RESULTS

The participants in the biomedical survey at 45 years were representative of the whole cohort with regard to visual acuity at 16 years.[13] At 45 years, autorefraction data were available for 2499 individuals (27% random subsample of cohort members surveyed). The present analysis is based on 1867 (74.7%) of these individuals for whom uncorrected visual acuity data were available for both eyes at 16 years. These individuals did not differ significantly by sex, social class or distance acuity at 11 years compared with those who had autorefraction at 44 years but did not have acuity measured at 16 years. Three children with eye disease were excluded.

In all, 173 of 1867 (9.3%) individuals in the present study had uncorrected visual acuity of 6/12 or worse in both eyes at 16 years. The median spherical equivalent in adult life was −3.75 (IQR −5.38, −2.13), and the whole range was −10.25 to +3.88. Specifically, 153 of 173 (88.4%) individuals of this group had a spherical equivalent of −1⩾, but 9.3% were emmetropic and 2.3% hypermetropic.

In those with uncorrected visual acuity of ⩾ 6/9 at 16 years, the median spherical equivalent in adult life was −0.375 (IQR −1, 0), and the full range was −9.25 to +7.00. Specifically, as adults, 493 (29%) individuals of this group were myopic and 109 (6.4%) were hypermetropic.

Table 1 shows that, overall, 646 of 1867 (34.6%) adults had a spherical equivalent of −1⩾ but only 153/647 (23.7%) of these individuals had an uncorrected visual acuity of ⩽6/12 in both eyes at 16 years. Notably, only 108 of 202 (53.5%) adults with spherical equivalent −3⩾ in the present study (10.8% overall) had reduced uncorrected visual acuity at 16 years.

Using different cut-off points for defining myopia at 45 years yielded sensitivities ranging from 69% for severe myopia (spherical equivalent cut-off point −6) to 16% for all, including very mild, myopia (spherical equivalent cut-off point of −0.5; table 2). Specificities were high at all cut-off points. The PPV increased as the spherical equivalent cut-off point decreased, reflecting the generic relationship between PPV and prevalence.

Table 2 Comparison of the accuracy of categorisation of myopia (using different cutpoints of spherical equivalent) based on uncorrected visual acuity of 6/12 or worse at 16 years

SE	Sensitivity	Specificity	PPV (95% CI)
⩽−6	68.6	91.9	13.9 (9.3 to 20.1)
⩽−3	53.5	96.1	62.4 (54.7 to 69.6)
⩽−1	23.7	98.4	88.4 (82.5 to 92.6)
⩽−0.75	19.4	98.4	90.2 (84.5 to 94)
⩽−0.5	16	98.4	91.9 (86.5 to 95.3)

CI, confidence interval; PPV, positive predictive value; SE, spherical equivalent.

DISCUSSION

Only a quarter of individuals in the population studied who had developed myopia by 45 years of age had reduced acuity at 16 years. Notably, half of all adults with moderate myopia (−2.99 to −5.99) and 31% (11/35) with severe myopia (⩾−6) had good uncorrected visual acuity in both eyes by 16 years. However, we found that there was a high (91%) lifetime probability of primary myopia (SE ⩾−0.5) given an uncorrected visual acuity of 6/12 Snellen equivalent or worse by the age of 16 years.

We report that, in this population, reduced uncorrected visual acuity at age 16 years is not an accurate predictor of lifetime risk of myopia. These findings are consistent with either the onset or progression of myopia in many individuals occurring after the age of 16 years, which is not the pattern that is reported in younger cohorts of similar populations.[14] For example, contrasting recent data from the US Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error study[15] and work by Sorsby *et al*[16] 30 years ago in a population of school children in the UK directly comparable with the 1958 British cohort show that, at the age of 14 years, those in the older cohort were on average 1 dioptre (D) more hypermetropic (girls 1.02 D and boys 0.93 D). Thus, our findings add strength to the notion of changes in the natural history of myopia in populations in western Europe and North America, which mirror, although they are less marked than, the increasingly earlier age at onset that has been reported in countries in Asia with the highest prevalences of myopia. However, although current myopia research tends to the view that most cases of myopia have onset at school age, these findings serve as a reminder that late-onset myopia makes an important contribution to the overall incidence of myopia in some populations. The implication of the poor predictive value of uncorrected visual acuity at 16 years in the present study is that it is therefore an inaccurate and inappropriate intermediate (proxy) ''phenotype'' for genetic epidemiological work on myopia.

Previous studies on the correlation between acuity and myopia have been carried out using cross-sectional (contemporaneous) visual acuity and refraction measurements from school children, in order to address questions about the value of measures of uncorrected visual acuity as screening tests for refractive error in population-based screening programmes, or to understand the functional correlates of different levels of refractive error during childhood.[17 18] Our findings support the assessment of visual acuity in children of secondary school age as an effective method of identifying undiagnosed refractive error (both myopia and hypermetropia). We suggest that testing using a threshold approach (set at 6/12 Snellen equivalent) for ''failing'' screening and thus indicating referral would be appropriate. Potentially this could be easily and quickly performed in schools by trained non-ophthalmic professionals such as school nurses or teachers. New methods of acuity testing, using the logarithm of the minimum angle of resolution method, allow the acuity measurement to be scaled up to adjust for testing over a shorter distance, which avoids the problems of finding suitable accommodation for testing in schools.[19]

We suggest that uncorrected visual acuity in childhood can only be used as an intermediate phenotype for myopia in populations for which knowledge of the natural history over the whole life course supports onset of myopia occurring early in the vast majority of affected individuals. This is unlikely to be the case in ''older'' cohorts, particularly in those that form the basis of the large-scale population-based ''biobank'' studies that are being undertaken currently or are planned throughout the world, in which alternative ways of capturing refractive error phenotype are required.[20]

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