Influences on childhood height: comparing two generations in the 1958 British birth cohort

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Background Genetics and early environment are known to influence height, but evidence is sparse on changes in these influences over time.

Methods The 1958 British birth cohort study includes all children born between March 3, 1958 and March 9, 1958, who were followed to age 41 yr, and one-third of their offspring in 1991. Childhood height in each generation (measured at 7 yr for cohort members and 4–18 yr for offspring) was converted to a standard deviation score based on the 1990 British growth reference. We used multilevel models to analyse influences on height in order to allow for the hierarchical within-family data structure.

Results Childhood height increased by 1 cm between 1958 cohort members and their offspring. Several influences on childhood height in the older generation (maternal smoking, breastfeeding, maternal age, social class, maternal education, and parental divorce) did not affect childhood height in the younger generation. Parental height was most strongly associated with childhood height and effects did not diminish between generations [adjusted increase ~2 cm for 1 maternal or paternal height standard deviation score (SDS)]. Third- or later-borns and those with three or more siblings had deficits of 1–2 cm (adjusted estimates) in both generations. Other factors, particularly indicators of socioeconomic position, showed weaker effects in the younger generation. For example, the growth deficit of 1.1 cm (adjusted estimate) among cohort members from households with >1.5 persons/room had disappeared in the offspring.

Conclusions Within Great Britain, the adverse effects of environmental factors on childhood height have lessened between recent generations.

Keywords Childhood height, early-life influences, cohort study, intergenerational comparison, Britain

Genetic effects on height are well accepted.1,2 Environmental influences have also been identified,3–6 with several factors, especially in early life, acting to delay growth. Depending on the severity and duration of the inhibitory factor, adult height may also be affected.7 However, given the increases in height seen across many populations,8–10 the magnitude of genetic and environmental influences may have changed over time.11,12 Greater increases in height have been found among children from poorer socioeconomic backgrounds, indicating that improvements in environmental factors may be having less effect on groups that already have favourable living conditions.13,14 Some evidence exists to suggest that in wealthy countries environmental factors explain a decreasing proportion of the variance in height,15 but this evidence is scant. The 1958 British birth cohort includes information on height and its determinants for two generations, and hence provides a rare opportunity to evaluate the changing role of genetic and environmental factors.

We investigated influences on childhood height in participants in the 1958 British birth cohort and their offspring. These generations span a period when the standard of living increased, as reflected in the decline of infant mortality in Britain: from ~230 per 100 000 population in 1958 to 100 per 100 000 in 1983.16 We examined potential influences on childhood height, as identified from the literature,3,17–19 including (i) parental height, as an indicator of genetic

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potential; (ii) prenatal factors, including maternal age at childbirth, maternal smoking during pregnancy, and birthweight; and (iii) early-life factors, including infant-feeding method, birth order, number of younger siblings, social class, housing tenure, household crowding, parental divorce, maternal education, and disability in childhood. Our aim was to establish whether genetic and environmental factors have similar effects on height in both generations.

Methods
Study samples
The 1958 cohort includes ~17 000 singletons born in 1 week in March 1958 and followed to age 41 yr. We use data up to the 33-yr survey in 1991, when information was collected on cohort members (n = 11 405) and a random sample of one-third of their offspring (Figure 1). The sample of cohort members responding at 33 yr is generally representative of the original birth cohort.

Measures
Height
Cohort members were measured at 7 yr (to the nearest inch) and values were converted into centimetres. Offspring aged 4 yr or older were measured to the nearest centimetre in 1991.
Explanatory variables
Information for cohort members was recorded in 1958; for offspring it was recorded in 1991, unless otherwise stated.
(i) Parental height. The cohort member’s mother was measured (in inches) in 1958; if information was missing, reported height in 1969 was used. Father’s height (in inches) was reported in 1969. Values were converted into centimetres. The cohort member’s adult height (in centimetres) measured in 1991 was used as a parental measure for the offspring.
(ii) Prenatal factors. Age of the mother at childbirth was reported for both generations. Maternal smoking during pregnancy was coded as ‘non-smoker’ (<1 cigarette/day), ‘1–10’, and ‘≥10 cigarettes/day’, with mothers smoking a ‘variable’ number of cigarettes categorized as smoking ≥10 cigarettes/day because the effect on height was similar in the two groups. Birthweight (in ounces) was measured for cohort members and reported for the offspring, and values were converted into grams. Gestational age, recorded at birth for cohort members and reported for the offspring, was categorized as <38, 38–42, and ≥42 weeks.
(iii) Early-life factors. Infant-feeding method was grouped as ‘never’ or ‘ever’ breastfed, the latter category including those breastfed for a short period (<1 month) or longer, although the exclusivity of breastfeeding is unknown. Birth order of the child and number of younger siblings were recorded for both generations. Social class for the cohort members was based on their father’s occupation when they were aged 7 yr (or at birth if missing). Social class for the offspring was based on the occupation of the male head of the household (cohort member or the male partner) in 1991. Social class was categorized as ‘I&II’ (professional/managerial), ‘IIINM’ (skilled nonmanual), ‘IIIM’ (skilled manual), and ‘IV&V’ (semi-skilled/unskilled manual). Information on housing was collected in 1965 and 1991, respectively, for cohort members and offspring: tenure was classified as ‘owner–occupier’, ‘private-renter’, and ‘council or housing association renter’ (social housing); crowding was grouped as ‘<1’, ‘1–1.5’, and ‘≥1.5’ persons/room. A mother’s education level was indicated by whether she stayed beyond the minimum

Figure 1 The offspring sample.
school-leaving age, which was 15 yr for cohort members’ mothers aged <25 yr and 14 yr for mothers ≥25 yr; for the mothers of the offspring the categories were ‘<A-level’ or ‘A-level/higher’ qualifications by age 33 yr. Parental separation or divorce occurring by age 7 yr was defined for cohort members and offspring from information reported in 1991. Disability was identified from school doctors’ reports of moderate/severe handicap in respect of ordinary schooling, or from mothers’ reports of a physical handicap at 7 yr for cohort members; identification of disability for offspring was based on mothers’ reports of any physical, emotional, or mental difficulties limiting normal school work or usual activities.

Data analysis

All height measures, including childhood height of cohort members (at 7 yr) and offspring (at ages 4–18 yr) and parental heights, were converted to standard deviation scores (SDSs) for the specific age and sex, based on the 1990 British growth reference.23 As expected, heights for the two generations were correlated, both between cohort members and offspring \( (r = 0.43) \) and between offspring from the same family \( (r = 0.39) \). To allow for the hierarchical data structure, effects on childhood height were estimated using multilevel models.24 with cohort members and offspring as ‘level-1’ units and families as ‘level-2’ units (see Appendix). We first modelled height SDS on age, sex, and each potential explanatory factor separately. Because the cohort members’ age at the birth of their child was negatively correlated with the age of their offspring—and also because the effect of environmental factors could vary during the growth period—we tested for interactions with age for each factor of interest. All interactions were nonsignificant, possibly because most (77%) of the offspring sample were aged <10 yr. We therefore included age of the child in models of offspring height to reduce possible confounding effects of maternal age. For the models of childhood height in the cohort, we then added mid-parental height SDS, the average of both parents, and for the models of offspring height we added one parental measure. For both generations we included prenatal and early-life factors separately to estimate their effects; paternal and maternal height SDSs were also analysed separately. The relationship between birthweight and childhood height was estimated before and after adjustment for gestational age. Differences in effects between generations were tested. The percentage of variance explained by each factor was calculated as the change in the variance after adding the factor to the model divided by the total variance of height. The sample size for offspring \( (3077 \text{ 4–18-year-olds}) \) (Figure 1) was reduced to 2462 with complete data on explanatory factors. This analysis sample for the offspring was smaller than that for cohort members \( (n = 7993) \); hence, nonsignificant estimates for offspring were calculated using the larger sample size for cohort members \( (t\text{-tests}, \text{based on the standard errors calculated from standard deviation for the offspring sample, but using the size of the cohort sample}) \). All estimates remained nonsignificant (except for disability), and thus changes in the magnitude of effects could not be attributed to differences in sample size. Differences in height SDS were transformed to centimetres for a 7-year-old.

Analyses are presented for both sexes, combined with an adjustment for sex. This approach was justified because interactions of sex with each factor were mostly nonsignificant \( [P > 0.05], \text{except for the interaction with housing tenure for the offspring} (P = 0.03)] \). The nonsignificant interactions with sex within generations ensure that changes in the effect of each factor between generations do not differ by sex. In respect of sample representativeness, we found that the sample of cohort members available for analysis \( (n = 7993) \) resembled the original birth study for childhood social class (21.3% were from classes IV & V, compared with 20.4% of those with data at age 7 yr) and for childhood height (122.5 cm at 7 yr in the analysis sample vs 122.4 cm in the full sample at 7 yr). Finally, the analyses were repeated separately for each factor using all subjects with information on that factor, and the results were similar to those presented here. Analyses were performed using SAS for UNIX and MLwiN.

Results

The characteristics of the cohort members and their offspring are summarized in Table 1. Offspring were taller on average than their parents by an SDS of 0.19 (1 cm), and the adult height of parents also increased between generations (Table 1).

Living conditions had improved between generations, with a decreased percentage living in overcrowded homes, for example. However, the prevalence of some potential influences on height had remained unchanged.

Estimated effects on childhood height, compared across the two generations, are presented in Table 2. The effect size for most factors was smaller among offspring than among cohort members (Table 2). In both generations, parental height was strongly associated with height in childhood: unadjusted estimates ranged from 0.35 to 0.47 (increases of 1.8–2.4 cm in childhood height for an increase of 1 parental height SDS), with significantly stronger effects in offspring than cohort members. Effects of parental height were almost unaffected by allowing for other factors (Table 2). In terms of the percentage of variance in childhood height explained, parental height was the most powerful factor: mid-parental height SDS accounted for 20.4%, birthweight for 6.6%, and other factors combined for 7.1% of the variance in cohort members’ height; for offspring height, 18.2% of variance was explained by parental height SDS, 3.8% by birthweight, and only 2.2% by other early-life factors.

A weak but significant linear relationship between maternal age and childhood height was seen for cohort members, with older mothers having taller children. This relationship strengthened after allowing for other factors (Table 2) due to negative confounding with birth order; later-borns were more likely to have older mothers and were shorter on average than first- or second-borns. Among offspring, maternal age was not associated with height. Fewer years of maternal education was associated with shorter childhood height among cohort members, although this relationship disappeared after adjusting for other factors. No effect of maternal education was found in offspring (Table 2).

Prenatal factors

The association between birthweight and childhood height was statistically significant in both generations: for every kilogram of...
INFLUENCES ON CHILDHOOD HEIGHT

Birthweight, mean height SDS increased by 0.51 (2.6 cm), compared with 0.32 (1.7 cm) in offspring. The effect of birthweight was similar after controlling for gestational age, with an adjusted estimates of 0.55 (2.8 cm) and 0.39 SDS (2.0 cm), respectively (Table 2), although it weakened with adjustment for parental height and was unaffected by further adjustment for early-life factors. Maternal smoking during pregnancy was significantly associated with childhood height only among cohort members, with a growth deficit for children of heavier smokers (≥10 cigarettes/day) of 0.24 SDS (1.2 cm), reducing to 0.06 SDS (0.3 cm) with adjustment for mid-parental height, birthweight, and other factors (Table 2).

Early-life factors

No effect of breastfeeding on childhood height was found in either generation after adjustment for other factors. Birth order was associated with height in both generations, independently of other factors, with third- or later-borns being shorter on average than first-borns, by 0.32 SDS (1.7 cm) among cohort members and by 0.26 SDS (1.3 cm) among offspring (Table 2). Number of younger siblings was also consistently associated with childhood height, with children who had ≥3 siblings being shorter than those with <2 by 0.17 SDS (0.9 cm) and 0.14 SDS (0.7 cm) among cohort members and offspring, respectively, after adjusting for other factors (Table 2).

Housing tenure was significantly associated with height, with children from social housing being shorter than those from owner–occupier households, by 0.36 SDS (1.9 cm) among cohort members and by 0.15 SDS (0.8 cm) among offspring. After allowing for other factors, the difference reduced but remained significant in both generations. Fewer offspring (4.2%) lived in private-rented housing than cohort members.

Table 1 Characteristics of two generations: cohort members born in 1958 and their offspring born 1973–87

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Cohort members (n = 7993)</th>
<th>Offspring (n = 2462)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex males n (%)</td>
<td>3912 (48.9)</td>
<td>1210 (49.1)</td>
</tr>
<tr>
<td>Maternal age (yr) mean (range)</td>
<td>27.6 (15–46)</td>
<td>24.5 (15–38)</td>
</tr>
<tr>
<td>Height</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age at height measurement (yr) mean (range)</td>
<td>7.3 (7–8.5)</td>
<td>8.1 (4–18)⁶</td>
</tr>
<tr>
<td>Childhood height SDS mean (s.d.)</td>
<td>−0.19 (1.07)</td>
<td>−0.003 (1.05)</td>
</tr>
<tr>
<td>Maternal height SDS mean (s.d.)</td>
<td>−0.42 (1.04)</td>
<td>−0.25 (1.03)¹</td>
</tr>
<tr>
<td>Paternal height SDS mean (s.d.)</td>
<td>−0.52 (1.07)</td>
<td>−0.22 (0.99)²</td>
</tr>
<tr>
<td>Maternal or paternal height SDS mean (s.d.)</td>
<td>−0.47 (0.86)</td>
<td>−0.24 (1.02)</td>
</tr>
<tr>
<td>Prenatal factors</td>
<td></td>
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<tr>
<td>Maternal education n (%)</td>
<td>Left at minimum age 5899 (73.8)</td>
<td>433 (27.4)⁶</td>
</tr>
<tr>
<td></td>
<td>&gt;Minimum age 2094 (26.2)</td>
<td></td>
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<tr>
<td>Maternal smoking in pregnancy n (%)</td>
<td>None 5461 (68.3)</td>
<td>1744 (70.8)</td>
</tr>
<tr>
<td></td>
<td>1–10 cigarettes/day 1177 (14.7)</td>
<td>215 (8.7)</td>
</tr>
<tr>
<td></td>
<td>&gt;10 cigarettes/day 1355 (17.0)³</td>
<td>503 (20.4)</td>
</tr>
<tr>
<td>Birthweight (g) mean (s.d.)</td>
<td>Boys 3420 (514)</td>
<td>3360 (552)</td>
</tr>
<tr>
<td></td>
<td>Girls 3270 (513)</td>
<td>3260 (523)</td>
</tr>
<tr>
<td>Gestational age n (%)</td>
<td>&lt;38 weeks 954 (9.3)</td>
<td>263 (9.7)</td>
</tr>
<tr>
<td></td>
<td>38–42 weeks 8286 (80.4)</td>
<td>2362 (87.4)</td>
</tr>
<tr>
<td></td>
<td>&gt;42 weeks 1066 (10.3)</td>
<td>77 (2.9)</td>
</tr>
<tr>
<td>Postnatal factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breastfeeding n (%)</td>
<td>5610 (70.2)</td>
<td>1595 (64.8)</td>
</tr>
<tr>
<td>Birth order n (%)</td>
<td>1st 2983 (37.3)</td>
<td>1454 (59.1)</td>
</tr>
<tr>
<td></td>
<td>2nd 2588 (32.4)</td>
<td>767 (31.1)</td>
</tr>
<tr>
<td></td>
<td>3rd or later 2422 (30.3)</td>
<td>241 (9.8)</td>
</tr>
<tr>
<td>Younger siblings n (%)</td>
<td>0 or 1 6193 (77.5)</td>
<td>1992 (80.9)</td>
</tr>
<tr>
<td></td>
<td>2 1235 (15.5)</td>
<td>381 (15.5)</td>
</tr>
<tr>
<td></td>
<td>3 or more 565 (7.1)</td>
<td>89 (3.6)</td>
</tr>
<tr>
<td>Social class n (%)</td>
<td>I&amp;II 1700 (21.3)</td>
<td>785 (31.9)</td>
</tr>
<tr>
<td></td>
<td>IIINM 837 (10.5)</td>
<td>301 (12.2)</td>
</tr>
<tr>
<td></td>
<td>IIIM 3633 (45.4)</td>
<td>835 (33.9)</td>
</tr>
<tr>
<td></td>
<td>IV&amp;V 1823 (22.8)</td>
<td>541 (22.0)</td>
</tr>
<tr>
<td>Housing tenure n (%)</td>
<td>Owner–occupier 3558 (44.5)</td>
<td>1835 (74.5)</td>
</tr>
<tr>
<td></td>
<td>Private rental 1367 (17.1)</td>
<td>104 (4.2)</td>
</tr>
<tr>
<td></td>
<td>Social housing 3068 (38.4)</td>
<td>523 (21.2)</td>
</tr>
<tr>
<td>No. of persons/room mean (range)</td>
<td>&lt;1 person/room 2781 (34.8)</td>
<td>1115 (45.3)</td>
</tr>
<tr>
<td></td>
<td>1–1.5 persons/room 3823 (47.8)</td>
<td>1199 (48.7)</td>
</tr>
<tr>
<td></td>
<td>&gt;1.5 persons/room 1389 (17.4)</td>
<td>148 (6.0)</td>
</tr>
<tr>
<td>Parental divorce n (%)</td>
<td>220 (2.8)</td>
<td>334 (13.6)</td>
</tr>
<tr>
<td>Disability n (%)</td>
<td>348 (4.4)</td>
<td>111 (4.5)</td>
</tr>
</tbody>
</table>

⁶94% of offspring were under 14 yr.
¹Children of female cohort members (n = 1580).
²Children of male cohort members (n = 882).
³Includes 436 mothers reporting ‘variable’ smoking.
(17.1%), and this group was taller than children from owner–occupier households only among offspring (Table 2). Mean height SDS reduced with increasing level of crowding, although the strength of the relationship reduced significantly in the offspring. Cohort members from households with >1.5 persons/room were shorter than those from households with <1 person/room by 0.65 SDS (3.4 cm); a deficit of 0.3 SDS (1.6 cm) in offspring became nonsignificant after allowing for other factors. Social class was significantly associated with the childhood height of cohort members, with children from classes IV and V being shorter by 0.39 SDS (2 cm) than those from classes I and II; however, this became nonsignificant after allowing for other factors (Table 2). No effect of social class was seen in the offspring.

Cohort members whose parents had separated or divorced were shorter on average, by 0.2 SDS (1 cm), than those whose parents had not, but the effect was nonsignificant after allowing for other factors and no effect was found in offspring (Table 2). Finally, there was an association with disability in both generations: a height deficit of 0.14 SDS (0.7 cm) and 0.16 SDS (0.8 cm) remained in cohort members and offspring, respectively, after adjustment for other factors (Table 2).
Discussion

The major finding from this comparison of childhood height in two generations is that a strong effect of early-life conditions, evident for those born in 1958, was less apparent in a later-born generation comprising their offspring. Prenatal and postnatal influences explained a smaller proportion of variance in height in the younger generation. This finding is consistent with other studies suggesting that the influence of the environment on height has weakened over time, at least in prosperous industrialized countries and with indications that social inequalities in height are diminishing in Western countries. The weakening of environmental influences on height seen in our study has occurred while living standards have improved: fewer offspring lived in overcrowded accommodation and more in owner-occupied properties in 1991 than was the case with cohort members in 1965. An additional finding is the absence of differences between males and females in the effects of environmental factors on childhood height. Previous studies, though inconclusive, have suggested that males are more sensitive to the environment than females. Our findings therefore provide no support for the view that environmental influences on height differ between boys and girls.

This is, to our knowledge, the first study to examine the changing influence on childhood height of a wide range of factors starting from before birth, although there is an extensive literature on social inequalities in childhood and adult height in the 1958 cohort and elsewhere. The 1958 cohort is representative of the initial birth sample. However, the offspring are a generation born to parents before their 30th birthday, rather than a randomly selected population sample, and their mothers are therefore younger on average (24.4 yr) than the general population (27.0 yr in 1986). Yet, most importantly, the offspring resemble the general population in many respects, including birthweight, social class, and height. One limitation is that some information was available only for one parent of the offspring (e.g. paternal or maternal height, and maternal education). Mid-parental height was used for cohort members, and height of one parent for the offspring. Thus we are better able to control for genetic influences in cohort members. The diminished effect of environmental influences on height was, however, already evident before allowance was made for parental height. Data available on the two generations in this study are extensive and present a unique opportunity to explore the change or stability of early-life influences on height. The Application of multilevel models was important to incorporate the covariance structure of these data and to permit a comparison of influences across generations. Further analyses of these data (reported elsewhere) based on a comparison of parent-offspring pairs (rather than all cohort members) support the main findings of the present study of weakening environmental influences on height.

Of the factors that we examined, three remained important predictors of childhood height across the two generations, namely, parental height, birthweight, and birth order. Parental height explained most of the variation in height in both generations. However, the effect of parental height was stronger in the second generation. This finding is consistent with a study in Finland in which heritability of height increased from 1325

Heritability is likely to be greater in affluent societies because there will be less impact of environmental factors that interfere with the achievement of individuals’ genetic height potential than in societies with a low standard of living.

Second, for birthweight, there was a strong association in both generations, with a 2.6 cm and a 1.7 cm increase for each kilogram of birthweight in cohort members and offspring, respectively. The slightly weaker relationship among offspring may be partly due to measurement bias, as their reported birthweights may be less accurate than the measurements obtained for cohort members. Also, there was a tendency for a weaker linear relationship in the offspring, although a quadratic relationship was nonsignificant. This might be expected because of improved survival and postnatal growth among low-weight births.

Third, for birth order, later-born children were shorter in both generations examined in this study, as seen elsewhere, though not in all studies. We found an adjusted deficit of 1.7 cm and 1.3 cm for third- or later-borns for the older and younger generation, respectively. Mechanisms for the association are not well established, although some suggest that it is due to an effect of family size on postnatal nutrition. However, the birth-order effect in our study was independent of other factors, and it is therefore possible that it is a reflection of maternal uterine factors during fetal development. The deficit for third- or later-borns was smaller (though not significantly so) in the offspring than in the cohort members. However, the contribution of this factor to the percentage of height variance explained will have reduced over time because there are fewer higher-order births among offspring than among the earlier generation. Our study will underestimate the percentage of third- and later-born offspring (9.8%), since mothers were aged 30 yr or less. Nevertheless, there is evidence from elsewhere that the prevalence of higher-order births has been decreasing in Great Britain.

Similar proportions of children with a disabling condition were identified in both generations, and it is notable that their deficit in growth had not improved over time.

Diminishing influence of environmental factors

Most of the effect of environmental factors on childhood height had diminished between generations in our study. This was notable for prenatal and postnatal influences, including maternal smoking, number of younger siblings, and measures of socioeconomic status. We showed, for example, height deficits of 1.9 cm and 0.8 cm, respectively, for cohort members and offspring living in social housing, and deficits of 3.4 cm and 1.6 cm, respectively, for those in overcrowded homes. Thus the risk associated with childhood disadvantage appears to have reduced over time, as suggested elsewhere by ourselves and others. However, some effects on childhood height have remained in the second generation, notably number of younger siblings and household crowding. But fewer offspring have >3 younger siblings and fewer live in households with >1.5 persons/room, and so proportionally fewer children are exposed to the risks associated with these factors.

The influence of maternal smoking during pregnancy on childhood height was evident only in cohort members, acting...
through its contribution to fetal growth. It is unlikely that the weaker effect of maternal smoking on offspring height is due to a lower mean consumption among those smoking >10 cigarettes/day: 35% of mothers of the offspring sample smoked ≥20 cigarettes/day, compared with 16% of mothers of the cohort generation. Also, the lack of an effect in the offspring is consistent with a study of a more recent generation in which children of smoking mothers had complete catchup in the first few years of life. It therefore appears that impaired growth in height due to adverse fetal conditions can be overcome by improved conditions and nutrition in early life.

With respect to infant-feeding method, studies of early British cohorts found that those who were breastfed during the 1920s–1940s were taller in childhood and adulthood. But in the 1958 cohort the benefit of breastfeeding for height was attributable to other early-life factors, consistent with findings from the 1970 cohort, while the absence of an association in the offspring agrees with results from another recent cohort, born in 1992–93. Thus, the relationship between breastfeeding and growth may have changed over time, possibly due to improvements in the nutritional adequacy of infant formula or to better postnatal diet more generally.

Finally, it is recognized that parental divorce can cause distress and be a major disruption in children’s lives. We and others have previously shown a deficit in height associated with parental divorce or family conflict in boys, but not in girls, although the deficit did not persist into adulthood. In the present study, we found no evidence that the growth of a recent generation of children has been affected by parental divorce or separation.

Conclusion

The general conclusion from this comparison of influences on childhood height in two generations is that the impact of environmental factors on height has reduced over time. This finding has been reported in other populations and has been attributed to improvements in socioeconomic conditions. Our results on the weakening association between social class and childhood height suggest that inequalities in height have diminished, while simultaneously childhood height has increased by 1 cm between cohort members and their offspring, continuing the secular trend of earlier generations.

Future studies on trends in childhood height and its determinants are likely to be relevant to adult health outcomes. Findings from our study suggest that improvements in the growth of British children have been achieved and that this may have favourable long-term consequences for their health as adults.

Acknowledgements

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KEY MESSAGES

- Height in childhood increased by 1 cm between two generations: members of the 1958 British birth cohort and their offspring.
- Parental height and birthweight were strongly associated with childhood height and effects were undiminished across generations.
- Several environmental influences on childhood height in the older generation (maternal smoking, breastfeeding, maternal age, number of younger siblings, parental divorce, and measures of socioeconomic status) had diminished between generations.
- Within Great Britain, adverse effects of environmental factors on childhood height have lessened between recent generations.

References

INFLUENCES ON CHILDHOOD HEIGHT


Appendix: A model for comparing height across two generations

We use a two-level model to analyse early influences on childhood height for two generations. Individuals are treated as level-1 units, clustered within families (level-2 units). Within level 1, indexed by i, the parent is i = 1 and the offspring are i = 2, 3, ..., (k+1) for k children.

Let \( y_{ij} \) be the height SDS of individual i in family \( j \) \( (i = 1, 2, ..., n_j \) and \( j = 1, 2, ..., m) \) and \( x_{ij} \) a level-1 explanatory variable; then height SDS can be modelled as a function of an explanatory variable (at either level). Here k is dependent on \( j \) \( (k = n_j - 1) \). A single two-level model with one level-1 explanatory variable \( X \) can be formulated as:

\[
\begin{align*}
    y_{ij} &= (\beta_0 + \beta_1 x_{ij}) + (\gamma_0 + \gamma_1 x_{ij}) + d_{1ij} + e_{ij} \\
    \beta_0 &= \beta_0 + \mu_0 \\
    \gamma_0 &= \gamma_0 + \mu_1 \\
    d_{1ij} &= 1 \text{ if } i = 1 \text{ (cohort member)} \text{ and } 0 \text{ otherwise} \\
    d_{2ij} &= 1 \text{ if } i > 1 \text{ (offspring)} \text{ and } 0 \text{ otherwise} \\
\end{align*}
\]

where \( d_{1ij} \) equals 1 if \( i = 1 \) (cohort member) and 0 otherwise and \( d_{2ij} \) equals 1 if \( i > 1 \) (offspring) and 0 otherwise. Thus \( d \) indicates the two components of the model: one for cohort members and one for offspring.

The fixed effects \( \beta_0 \) and \( \gamma_0 \) represent the mean intercepts, \( \beta_1 \) and \( \gamma_1 \) represent the slope associated with the explanatory variable for cohort members and offspring, respectively. \( \mu_0 \) and \( \mu_1 \) are random effects (between families) for cohort members.
and offspring with mean 0. The parameter $\varepsilon_{ij}$ is the level-1 residual for the offspring with $\varepsilon_{ij} \sim N(0, \sigma^2_i)$.

This model assumes that there is level-2 variance [between-family variance $\text{Var} (\mu_{ij}) = \sigma_j^2$] for cohort members, but no level-1 variance (within family) can be specified, as there is only one cohort member per family. For offspring, the level-1 and level-2 variances are $\text{Var} (\varepsilon_{ij}) = \sigma^2_i$ and $\text{Var} (\mu_{ij}) = \sigma_j^2$, respectively (total variance $= \sigma_i^2 + \sigma_j^2$). Assuming that the covariance between cohort members and their offspring is $\text{Cov} (\mu_{1j}, \varepsilon_{ij}) = \sigma_{12}$ and also $\text{Cov} (\mu_{2j}, \varepsilon_{ij}) = 0$, the correlation between cohort members and any of their offspring is therefore

$$
\rho_{12} = \frac{\sigma_{12}}{\sqrt{\sigma_i^2 (\sigma_i^2 + \sigma_j^2)}}
$$

and the correlation between children from the same family is

$$
\rho_{22} = \frac{\sigma_i^2}{\sigma_i^2 + \sigma_j^2}.
$$