Leg and trunk length at 43 years in relation to childhood health, diet and family circumstances; evidence from the 1946 national birth cohort

MEJ Wadsworth,* RJ Hardy,* AA Paul,b SF Marshall*a and TJ Colec

Background  This is a study of the associations of adult leg and trunk length with early life height and weight, diet, socioeconomic circumstances, and health, and parental height, divorce and death.

Method  The data used were collected in a longitudinal study of the health, development and ageing of a British national birth cohort (N = 2879 in this analysis) studied since birth in 1946. Multiple regression models were used to investigate the relationships.

Results  Adult leg and trunk length were each positively associated with parental height, birthweight, and weight at 4 years. Leg length was associated positively with breastfeeding and energy intake at 4 years. Trunk length was associated negatively with serious illness in childhood and possibly also parental divorce, but not with the dietary data.

Conclusion  Adult leg length is particularly sensitive to environmental factors and diet in early childhood because that is the period of most rapid leg growth. Trunk growth is faster than leg growth after infancy and before puberty, and may be associated with the effects of serious illness and parental separation because of the child's growing sensitivity to stressful circumstances, as well as the result of the biological effects of illness.

Keywords  Leg length, trunk length, child health, diet

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Short adult height is known to be a risk for cardiovascular and cancer mortality and for poor adult health,1–3 and leg length in childhood is also a marker of risk for cardiovascular disease and cancer.4–6

Greatest leg or lower body length is associated with advantaged socioeconomic circumstances in childhood,7–10 and environmental advantage in the preschool period in particular is associated with growth.11 Leg growth contributes markedly to stature attainment,12 and increased leg length was the major component of the secular trend in height increase in populations in Japan and Norway.13,14

Childhood height growth is associated also with prenatal growth,15 parental height,16 the child's health,16,17 history of emotional disturbance,18,19 and nutrition.10,20

This study asks whether the childhood health and environmental effects known to be associated with growth have different impacts on adult leg and trunk length, in order to further understanding of the observed associations of height with disease. Information used is from a national longitudinal study from birth to adulthood.

Method

Population

The Medical Research Council (MRC) National Survey of Health & Development is a social class stratified sample of 5362 of all the single, legitimate births that occurred in England, Wales and Scotland, 3–9 March 1946. That population has been
studied on 21 occasions between birth and age 53 years.\textsuperscript{21,22} By age 43 years when outcomes reported here were made, permanent losses comprised 365 (6.8\%) deaths, and 540 (10.1\%) refusals, and temporary losses were 607 (11.3\%) emigrations or residence overseas and 370 (6.9\%) failures to contact. The population visited at 43 years (N = 3262) tended to under-represent the never married, the least literate, those always in manual social class circumstances, and the mentally ill.\textsuperscript{22} Those with physical illness were well represented.\textsuperscript{22} Losses through death were greater in the manual social classes than in the non-manual.\textsuperscript{23}

**Measures**

**Anthropometry**
Outcome measures were adult leg and trunk length, derived from standing and sitting heights measured by a research nurse at a home visit when study members were aged 43 years. Leg length was calculated as the difference between standing and sitting heights, and trunk length was represented by sitting height. Standing height was measured to the nearest 0.5 cm using a lockable tape with foot plate, head piece and spirit level, with the head in the Frankfort plane position. This stadiometer was manufactured for the study by CMS Measuring Equipment (London). Sitting height was measured with the subject sitting upright on the base plate, which was on a flat seat, and with the head in the Frankfort plane position, feet on the floor, and the thighs unsupported.\textsuperscript{24}

Birthweight was used to indicate prenatal development, and was recorded by midwives or obstetricians at birth, or taken from records by health visitors. Heights and weights at 4, 7, 11 and 15 years were measured at clinics by health visitors using their clinic’s equipment and the study’s protocol.\textsuperscript{25}

Parental heights were included as markers of genetic influence, mother’s heights were measured by health visitors, at clinics using their own protocols, when their study child was aged 6 years, and mothers reported father’s height.

**Nutrition**
Information on exclusive breastfeeding was obtained from mother’s reports to health visitors when children were aged 2 years, and used here as ever/never breastfed, since breastfeeding was not associated in a dose-related fashion with either outcome. Health visitors asked, when study children were aged 4 years, ‘What did this child have for each meal yesterday?’ This was coded in terms of foods and nutrient content.\textsuperscript{26} Energy was calculated in kilojoules. Some micronutrients have a role in bone matrix and connective tissue formation, and some may have specific effects on long bone growth,\textsuperscript{27,28} whilst others are markers of diet quality. As such, estimated mean daily intakes of energy, protein, calcium, phosphorus, magnesium, zinc, iron, vitamins A, C, and E, thiamin, riboflavin and niacin were included in this analysis.

**Health in childhood**
Mothers reported the child’s illnesses to health visitors. This analysis included only serious illness, defined as lasting longer than 3 months in any one year or necessitating a continuous hospitalization of one month or more between birth and age 59 months. Reports of admissions were checked with hospital records.\textsuperscript{23}

**Socioeconomic circumstances in childhood**
Father’s occupation when the child was aged 4 years was coded using the Classification of Occupations\textsuperscript{29} (categorized into manual and non-manual). Crowding was coded in terms of people per room and categorized as overcrowded (≥2 people per room) or not (<2 people per room).

**Emotional upheaval in childhood**
Health visitors recorded custodial parents’ reports of any parental separation as having occurred or not before the child’s sixth birthday, and of parental death by the same age. This age was chosen to be consistent with that available for childhood illness.

**Analysis**
Leg and trunk length were sex standardized, by converting to internally derived standard deviation scores (z scores). A third outcome measure, defined as the difference in z score for leg length and z score for trunk length, was also considered, as a measure of disproportion, in order to test whether there were differential influences on leg and trunk length.

Unadjusted relationships between each of the independent variables and the three outcomes were tested using linear regression models. Where appropriate measures were entered as continuous variables and tested for linearity. Each nutrient was adjusted for energy intake at age 4 years. All nutrients, energy intake and weight at 4 years were logged using natural logarithms to reduce skewness, and multiplied by 100 so that the regression coefficients could be interpreted as the change in outcome per one per cent in the variable.\textsuperscript{30} Since there were no statistically significant sex by risk factor interactions (P > 0.05 in all cases), all models included data from both sexes. These analyses were restricted to those who had been contacted at 4 years of age.

All factors associated in the initial analyses (with a P-value <0.1) with an outcome were considered for inclusion in the multiple regression models for that outcome. Where there was clear confounding only the stronger factor was included in the multiple regression model. These models were then adjusted for both weight and height at 4 years of age. That age was chosen because diet information was collected then, and because it represented a stage of early life growth that is well differentiated from prenatal growth. Height at 4 years was then replaced by height at ages 7, 11 and 15 years in turn in order to investigate the stage of growth during which the early life factors were influential. If any height measure cancelled out the effect of an early life factor then it may be assumed that the effect of that factor is accounted for, because its influence is seen already in terms of height.

Analyses were performed using the computer software Statistical Package for the Social Sciences.\textsuperscript{31}

**Results**
Mean leg length at 43 years was greater in men (83.3 cm, SD 4.99) than in women (75.6 cm, SD 4.72). Description of the explanatory variables is provided in Table 1. The correlation between trunk and leg length z scores was low at 0.1. The difference was strongly correlated with both components of height (0.7 for leg length and –0.7 for trunk length). The unadjusted relationships of leg and trunk length with each of the dependent variables are shown in Table 2. Leg length and trunk length were positively associated with mother’s and father’s height, with the effect being greater for leg length. The
associations between father’s height and leg and trunk length were curvilinear, with the positive effect becoming greater with increasing paternal height \((P < 0.01\) for a quadratic trend in both cases). Birthweight was positively associated with both components of height. Those from a non-manual social class had greater mean leg and trunk length than those from a manual social class, and this association was stronger for leg length. Leg length and trunk length were shorter in those not breastfed and when home circumstances were crowded, with the effects being considerably stronger for leg length. Energy intake was associated with leg and trunk length, the effect being stronger for leg length. After adjustment for energy intake, leg length was associated positively with intakes of all nutrients and particularly with vitamins A and C and with riboflavin and niacin, and trunk length was positively associated with intakes of vitamins C and E. Trunk and leg length were each shorter in those who experienced serious illness with the effect being greater for trunk length. Trunk length but not leg length was also shorter in those who experienced parental divorce.

**Multiple regression analysis of leg length**

After further adjustment of each nutrient that showed a significant relationship with leg length (in Table 2) for parental height, birthweight, father’s social class, breastfeeding and serious childhood illness, none remained significant. All coefficients were reduced (vitamin A and vitamin C to 0.0005, riboflavin to 0.0003 and niacin to 0.09). Energy intake was considered for further analysis rather than the individual nutrients. Father’s social class, crowding and parental heights accounted for this weakening of the effects, and so it is likely that the nutrients were a proxy for socioeconomic circumstances. Crowding and father’s social class were confounded, since the effect of each was considerably weakened when included in a model together with neither remaining significant at the 5% level. Social class was selected for further analysis, as the stronger factor.

The independent relationship of leg length with nutrition, in the form of breastfeeding and energy intake, was confirmed in a multiple regression model (Table 3, column 2). The breast fed had an estimated leg length 0.13 standard deviations greater than others, while an increase of one per cent in energy intake was associated with an increase of 0.0025 standard deviations in leg length. Parental heights, birthweight and weight at 4 years were also associated independently with leg length (Table 2, column 2). Father’s social class was to some extent confounded with mother’s height and father’s height. After adjustment for height and weight at age 4 years, the effect of father’s social class was weakened and was no longer significant (Table 3, column 3). Height at 4 years accounted for some, but not all, of the effects of breastfeeding and energy intake. The effect of birthweight also became weaker, but remained significant \((P = 0.04)\). The initially weak influence of childhood serious illness got even smaller with each stage of adjustment. Height and weight at 4 years were strongly positively associated with leg length.

All later childhood heights were positively associated with leg length. Height at 7, 11 and 15 years greatly weakened the effects of breastfeeding, energy intake and weight at 4 years. The effect of birthweight was weakened only by the addition of height at 15 years.

**Multiple regression analysis of trunk length**

The dietary effects (both individual nutrients and energy intake) were confounded with parental heights and father’s social class and crowding. The effects of father’s social class and crowding were confounded. Hence, as for the leg length analysis, the individual nutrients and crowding were not considered in further analyses.

In a multiple regression model (Table 4, column 2) those who were ill in childhood had an average trunk length 0.31 standard deviations less than those who were not ill, and those whose parents separated had a trunk length 0.24 standard deviations less than those whose parents did not. The unadjusted effects of these two variables are stronger in this restricted sample than in the sample used in Table 2. Energy intake was not independently associated with trunk length, although the unadjusted effect of energy intake in this reduced sample was slightly weaker.
<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Leg length at age 43 years (z score)</th>
<th>Trunk length at age 43 years (z score)</th>
<th>Difference at age 43 years (leg length z score – trunk length z score)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother's height (cm)</td>
<td>2626</td>
<td>0.048 (0.042–0.053)</td>
<td>0.040 (0.034–0.046)</td>
<td>0.007 (–0.001–0.016)</td>
</tr>
<tr>
<td>Father's height (cm)</td>
<td>2162</td>
<td>0.036 (0.031–0.041)</td>
<td>0.030 (0.025–0.035)</td>
<td>0.006 (–0.001–0.013)</td>
</tr>
<tr>
<td>Birthweight (kg)</td>
<td>2872</td>
<td>0.33 (0.26–0.40)</td>
<td>0.31 (0.24–0.38)</td>
<td>0.003 (–0.008–0.013)</td>
</tr>
<tr>
<td>Non-manual father's social class</td>
<td>2827</td>
<td>0.24 (0.17–0.32)</td>
<td>0.18 (0.11–0.25)</td>
<td>0.06 (–0.04–0.17)</td>
</tr>
<tr>
<td>Not overcrowded at 2 years</td>
<td>2784</td>
<td>0.20 (0.11–0.30)</td>
<td>0.06 (–0.03–0.15)</td>
<td>0.14 (0.02–0.28)</td>
</tr>
<tr>
<td>Breastfed</td>
<td>2782</td>
<td>0.17 (0.08–0.25)</td>
<td>0.04 (–0.05–0.13)</td>
<td>0.13 (0.00–0.25)</td>
</tr>
<tr>
<td>Energy intake at 4 years (%)</td>
<td>2851</td>
<td>0.0027 (0.0013–0.0041)</td>
<td>0.0016 (0.0002–0.0030)</td>
<td>0.0010 (–0.0010–0.0031)</td>
</tr>
<tr>
<td>Protein (%)</td>
<td>2851</td>
<td>0.0003 (–0.0021–0.0027)</td>
<td>0.08 (–0.0038–0.0010)</td>
<td>0.0017 (–0.0017–0.0052)</td>
</tr>
<tr>
<td>Calcium (%)</td>
<td>2851</td>
<td>0.0002 (–0.0013–0.0016)</td>
<td>0.8 (0.00012–0.0026)</td>
<td>0.1 (–0.0001–0.0015)</td>
</tr>
<tr>
<td>Vitamin A (%)</td>
<td>2851</td>
<td>0.0009 (0.0002–0.0016)</td>
<td>0.0016 (0.00052–0.00085)</td>
<td>0.0007 (0.0003–0.0017)</td>
</tr>
<tr>
<td>Vitamin C (%)</td>
<td>2851</td>
<td>0.0012 (0.0007–0.0018)</td>
<td>0.0007 (0.0002–0.0013)</td>
<td>0.0005 (0.0003–0.0013)</td>
</tr>
<tr>
<td>Magnesium (%)</td>
<td>2851</td>
<td>0.0006 (–0.0016–0.0031)</td>
<td>0.0014 (–0.0030–0.0011)</td>
<td>0.0020 (0.0015–0.0055)</td>
</tr>
<tr>
<td>Iron (%)</td>
<td>2851</td>
<td>0.0015 (–0.0003–0.0033)</td>
<td>0.0006 (–0.0024–0.0013)</td>
<td>0.0021 (0.0005–0.0047)</td>
</tr>
<tr>
<td>Vitamin E (%)</td>
<td>2851</td>
<td>0.0009 (–0.0003–0.0021)</td>
<td>0.0016 (0.0004–0.0029)</td>
<td>0.003 (–0.0026–0.0010)</td>
</tr>
<tr>
<td>Zinc (%)</td>
<td>2851</td>
<td>0.0002 (–0.0020–0.0024)</td>
<td>0.9 (0.0003–0.0019)</td>
<td>0.8 (–0.0033–0.0030)</td>
</tr>
<tr>
<td>Phosphorus (%)</td>
<td>2851</td>
<td>0.0007 (–0.0014–0.0029)</td>
<td>0.0041 (–0.0026–0.0018)</td>
<td>0.0012 (–0.0020–0.0043)</td>
</tr>
<tr>
<td>Thiamin (%)</td>
<td>2851</td>
<td>0.0010 (–0.0010–0.0030)</td>
<td>0.3 (–0.0022–0.0018)</td>
<td>0.0012 (–0.0017–0.0040)</td>
</tr>
<tr>
<td>Riboflavin (%)</td>
<td>2851</td>
<td>0.0013 (0.0002–0.0025)</td>
<td>0.0002 (–0.0009–0.0014)</td>
<td>0.0003 (0.0021–0.0026)</td>
</tr>
<tr>
<td>Niacin (%)</td>
<td>2851</td>
<td>0.0013 (0.0001–0.0027)</td>
<td>0.0000 (0.0014–0.0014)</td>
<td>0.0013 (0.0007–0.0033)</td>
</tr>
<tr>
<td>Serious illness before 6 years</td>
<td>2879</td>
<td>–0.14 (–0.30–0.01)</td>
<td>0.07 (–0.22–0.37 to 0–0.36)</td>
<td>0.007 (0.07–0.15)</td>
</tr>
<tr>
<td>Parental divorce before 6 years</td>
<td>2879</td>
<td>0.02 (–0.20–0.23)</td>
<td>0.19 (0.41–0.03)</td>
<td>0.21 (0.10–0.52)</td>
</tr>
<tr>
<td>Parental death before 6 years</td>
<td>2866</td>
<td>–0.13 (–0.40–0.15)</td>
<td>0.08 (–0.08–0.04)</td>
<td>0.04 (–0.04–0.34)</td>
</tr>
</tbody>
</table>

* Adjusted for logarithm of energy intake at 4 years. + not linear (increasing effect with increasing father’s height) but displayed as linear.
Table 3 Regression coefficients for the final model for leg length (unadjusted and adjusted coefficients given for the same sample for comparison)

<table>
<thead>
<tr>
<th>N = 1934</th>
<th>Unadjusted</th>
<th>Adjusted for all variables except weight and height</th>
<th>Adjusted for all variables listed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Reg coeff (95% CI)</td>
<td>P-value</td>
<td>Reg coeff (95% CI)</td>
</tr>
<tr>
<td>Mother's height (cm)</td>
<td>0.048 (0.042–0.055)</td>
<td>&lt;0.001</td>
<td>0.036 (0.030–0.043)</td>
</tr>
<tr>
<td>Father's height (cm)</td>
<td>0.036 (0.031–0.041)</td>
<td>&lt;0.001</td>
<td>0.027 (0.022–0.032)</td>
</tr>
<tr>
<td>Birthweight (kg)</td>
<td>0.31 (0.22–0.39)</td>
<td>&lt;0.001</td>
<td>0.21 (0.13–0.29)</td>
</tr>
<tr>
<td>Non-manual father's social class</td>
<td>0.20 (0.11–0.29)</td>
<td>&lt;0.001</td>
<td>0.08 (0.00–0.16)</td>
</tr>
<tr>
<td>Breastfed</td>
<td>0.19 (0.09–0.30)</td>
<td>&lt;0.001</td>
<td>0.13 (0.03–0.22)</td>
</tr>
<tr>
<td>Energy intake at age 4 years (%)</td>
<td>0.0037 (0.0021–0.0054)</td>
<td>&lt;0.001</td>
<td>0.0025 (0.0010–0.0040)</td>
</tr>
<tr>
<td>Serious illness before 6 years</td>
<td>–0.16 (–0.36–0.04)</td>
<td>0.1</td>
<td>–0.11 (–0.29–0.07)</td>
</tr>
<tr>
<td>Birthweight</td>
<td>0.027 (0.024–0.031)</td>
<td>&lt;0.001</td>
<td>–0.0049 (0.0009–0.0089)</td>
</tr>
<tr>
<td>Height at 4 years (cm)</td>
<td>0.0088 (0.0081–0.0096)</td>
<td>&lt;0.001</td>
<td>–</td>
</tr>
</tbody>
</table>

Table 4 Regression coefficients for the final model for trunk length (unadjusted and adjusted coefficients given for the same sample for comparison)

<table>
<thead>
<tr>
<th>N = 2012</th>
<th>Unadjusted</th>
<th>Adjusted for all variables except weight and height</th>
<th>Adjusted for all variables listed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Reg coeff (95% CI)</td>
<td>P-value</td>
<td>Reg coeff (95% CI)</td>
</tr>
<tr>
<td>Mother's height (cm)</td>
<td>0.041 (0.035–0.048)</td>
<td>&lt;0.001</td>
<td>0.030 (0.023–0.037)</td>
</tr>
<tr>
<td>Father's height (cm)</td>
<td>0.031 (0.026–0.037)</td>
<td>&lt;0.001</td>
<td>0.024 (0.019–0.029)</td>
</tr>
<tr>
<td>Birthweight (kg)</td>
<td>0.32 (0.24–0.41)</td>
<td>&lt;0.001</td>
<td>0.24 (0.16–0.33)</td>
</tr>
<tr>
<td>Non-manual father's social class</td>
<td>0.18 (0.09–0.26)</td>
<td>&lt;0.001</td>
<td>0.08 (0.00–0.17)</td>
</tr>
<tr>
<td>Energy intake at age 4 years (%)</td>
<td>0.0012 (0.0005–0.0028)</td>
<td>0.2</td>
<td>0.0001 (0.0004–0.0007)</td>
</tr>
<tr>
<td>Serious illness before 6 years</td>
<td>–0.35 (–0.55 to –0.15)</td>
<td>0.001</td>
<td>–0.31 (–0.50 to –0.13)</td>
</tr>
<tr>
<td>Parental divorce before 6 years</td>
<td>–0.39 (–0.71 to –0.07)</td>
<td>0.02</td>
<td>–0.33 (–0.63 to –0.03)</td>
</tr>
<tr>
<td>Weight at 4 years (%)</td>
<td>0.029 (0.025–0.032)</td>
<td>&lt;0.001</td>
<td>0.012 (0.008–0.016)</td>
</tr>
<tr>
<td>Height at 4 years (cm)</td>
<td>0.0078 (0.0070–0.0087)</td>
<td>&lt;0.001</td>
<td>–</td>
</tr>
</tbody>
</table>

Discussion

Summary

In this analysis adult leg and trunk length were associated with the partial genetic and environmental effects of parental height and the developmental effect of birthweight and weight at 4 years. They differed in their apparent sensitivity to childhood environmental factors. Leg length was associated with breastfeeding and energy intake at 4 years. Trunk length was associated with childhood serious illness and less strongly with parental separation. Leg length is likely to have greater error than trunk length as it is calculated as the difference between standing and sitting heights, each with its associated error. The effects of likely errors in leg length measurement may have attenuated reported associations.

Diet

Validity of the dietary data is discussed elsewhere. Although error in this measure is certain, we argue that micronutrient rich foods, such as fish and fruit provide a sufficient indicator of diet to categorize children broadly on the range of nutrient intake. Dietary effects were, in this analysis, most evident on leg length, as found in another study. Nutrients associated with stature independently of energy were confounded with parental height and socioeconomic indicators such that no differentiation of effect of individual nutrients over that of energy intake was apparent.

Difference between leg and trunk length

No further modelling was carried out for this outcome as all associations became insignificant, and the effects were small, making interpretation difficult.
Serious illness and parental separation

The association of shorter adult trunk length with childhood serious illness, and more weakly with parental separation, may be because these chronic effects continue into the period of more rapid trunk growth later in childhood. The change in unadjusted estimates for these variables on restriction of the sample size, and the small numbers experiencing these events means that the findings, particularly in relation to parental separation, should be interpreted with some caution. Childhood serious illness had long-term psychosocial and health effects in this and other studies. Long-term effects of parental separation have been associated with disturbances of behaviour, health and educational attainment; parental death did not have these long-term effects, implying that chronic parental discord is the explanation for this association. The proposed glucocorticoid-hippocampal hypothalamic pathway may account for this finding.

Parental height

Mother’s measured height was more strongly associated with offspring’s leg and trunk length than was father’s reported height, which was not linearly associated with leg length. This may be due to differences in measurement error since there were peaks of measures at even inches for father’s but not mother’s height. Children of manual social class fathers were shorter in both components of height, although this effect was confounded with parental height and child’s weight at 4 years. It is likely that parental height is a marker of socioeconomic position.

Growth

In this analysis as in others the effect of birthweight was similar for leg and trunk length, even after adjustment for weight at 4 years which was positively associated with both components of height. Thus being heavy at birth or at 4 years, or both, had a positive effect on growth. The effect of birthweight on both components of adult height was independent of prepubertal childhood height. This suggests that prenatal growth does not have a differential influence on the components of height, but rather an influence on absolute height, as Gunnell et al. observed. There was also a positive effect of weight at 4 years on adult trunk length, which was stronger for women than men, after adjustment for height at 15 years. This was accounted for by later childhood height. Weight at 7 years is higher in girls who reach menarche early, and here early maturing boys and girls were also heavier at 4 years. Hence weight at 4 years may be a proxy for age at puberty, suggesting that early matures have longer trunk length as a ratio of height than later matures, as Gunnell et al. suggest. It is not clear, however, why it is more significant in girls.

Sensitivity of leg length to early environment

Leg length was associated with breastfeeding and energy intake, but not with serious illness and parental separation, showing the sensitivity of leg length as a marker of early life environment. This is supported by the argument that the national secular increase in adult height is reflected in height at 2 years, because of the reduction of stunting. The observed association may be stronger than reported because of the probable greater error in measurement of leg length.

Our interpretation that influences on early growth impact primarily on leg length while there are longer term effects of early factors on trunk length, is supported by regression models that included more than one measure of childhood height. The effects of energy intake and breastfeeding on leg length were weakened by the addition of height at 4 years, and weakened further by the addition of later heights. Dietary factors may influence growth before age 7 years. In contrast the effect of childhood serious illness was only decreased after adding height at 15 years, and that of parental separation remained weakly associated after adjustment for all heights. Given that height at 15 years is a measure of post pubertal height and is closely correlated to adult height, this effect may be accounted for by the effect of serious childhood illness on prepubertal height.

Period effect

Later born cohorts are likely to have increased exposure to some of the risks identified here. For example, the proportion of children living in relative poverty increased during the 1980s and early 1990s, breastfeeding is less prevalent but shows signs of increase, and survival of low birthweight is more prevalent, and adult separation has risen greatly, with some maintenance of its adverse effects on growth and education. Studies of later born cohorts, without the constraints of food rationing, may find more variation in childhood nutrient intake, and more power to detect the effects of individual nutrients.

Conclusions

We found that leg length was sensitive to infant (under 5 years) socioeconomic circumstances and diet, whilst trunk length was sensitive to serious illness and possibly to chronic emotional disturbance. The explanation may be that leg growth is stronger than trunk growth during infancy, and trunk growth is stronger thereafter. Trunk length is apparently more rapid at later prepubertal ages, although in puberty trunk growth is not faster than leg growth. Trunk growth appeared more perturbed by factors that affected the child over longer periods between infancy and puberty. These two components of growth were independent of prenatal effects, as marked by weight at birth.

Acknowledgements

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KEY MESSAGES

- Adult leg and trunk length were positively associated with parental height, and own birthweight and weight at 4 years.
- Leg length was positively associated with breastfeeding and with energy intake at 4 years.
- Trunk length was associated negatively with serious illness experienced by the end of the fifth year, and possibly also with parental separation by the same age, but not with the dietary data.
- Leg length may be sensitive to markers of early childhood environment because of a period of rapid leg growth.
- Trunk length may be sensitive to factors that have chronic effects on the child's life because of more rapid trunk growth occurring at a later period, after infancy and before puberty.

References


Commentary: Can adult anthropometry be used as a ‘biomarker’ for prenatal and childhood exposures?

David Gunnell

Life Course Epidemiology

Life course epidemiology is concerned with investigating the effect on health of accumulating and interacting biological, social, and psychosocial processes. Parental health and genetic endowment together with intrauterine, childhood and early adult exposures may all influence an individual’s health, but investigating their joint effects presents two important challenges. The first is that many of the exposures studied are socially patterned, therefore identifying the relevant factors using observational studies, which are prone to confounding, is problematic. Randomized controlled trials with long follow-up are the best means of identifying the long-term effects of possible interventions on health. The second challenge is that there are few cohort studies with exposure and health information from before birth until old age. Most of the studies where data have been prospectively recorded at different stages of the life course are either of relatively young individuals, or currently limited by insufficient power to examine major clinical end-points.

Indirect Measures of Fetal, Infant and Childhood Exposures

When prospectively recorded data on a person’s health, diet, health-related behaviours and living conditions at particular ages do not exist, indirect or proxy measures may be used (Table 1). To interpret the association of these measures with later disease requires a fuller understanding of their meaning and shortcomings. The investigation by Wadsworth and colleagues provides information concerning two possible anthropometric ‘biomarkers’—leg length and trunk length. Their analysis may provide clues concerning the possible exposures underlying stature-disease associations. Furthermore it suggests that leg length and trunk length may act as markers for exposures operating at different stages during childhood.

Body Shape and Disease

Interest in the association between body shape and health dates back over a century (see Burchard 1936). Early studies of physical illness characterized human body forms into three main classes—endomorphs, ectomorphs and mesomorphs. A different form of classification was used in the psychiatric literature—classes include aesthetics, athletics and pyknics. Categorization was based on a number of features including...
height, limb length, weight and fat distribution. More recent investigations have focussed separately on overall stature and adiposity/fat distribution. Adult height is a measure both of genetic endowment and of health and nutrition throughout the growing years. Unlike adiposity measures, height changes little during adulthood. For this reason the association of greater stature with an increased risk of cancer and a decreased risk of cardiovascular disease may reflect the long-term consequences of pre-adult exposures.

Neither the relevant period of growth nor the exposures for which stature may be acting as a ‘biomarker’ are well characterized. Growth disturbances at several stages of development may contribute to short adult stature. It has been suggested that one way of further investigating height-disease associations may be to study associations between the two components of height—leg length and trunk length—and disease risk. The rationale for such an approach lies in the observation that post-natal linear growth is in greater part due to an increase in leg length than trunk growth and that adversity at this time causes impaired lower limb development.

### Table 1 Proxy measures of fetal, infant and childhood exposures

<table>
<thead>
<tr>
<th>Period of Life course</th>
<th>Measure</th>
<th>Factor(s) the measure may act as a marker for</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prenatal</td>
<td>Birthweight/birth length/birthweight/placental weight</td>
<td>Nutrition in utero</td>
<td>Also influenced by maternal size, smoking and ill-health (including pre-eclampsia) as well as gestational age and birth order</td>
</tr>
<tr>
<td>Conception and in utero development during period of famine</td>
<td>Undernutrition in utero</td>
<td>Ecologic marker for famine exposure, mother may not have been affected by famine</td>
<td></td>
</tr>
<tr>
<td>Lung function</td>
<td>Intra-uterine growth retardation/maternal smoking in pregnancy</td>
<td>Also influenced by adult smoking and other exposures in childhood and adult life. Influences on lung growth not fully understood</td>
<td></td>
</tr>
<tr>
<td>Parental social class</td>
<td>A range of socially patterned exposures</td>
<td>Non-specific</td>
<td></td>
</tr>
<tr>
<td>Season of birth</td>
<td>Exposure in utero to infection, food scarcity or particular climatic conditions</td>
<td>Non-specific marker of a range of exposures which show seasonal variation</td>
<td></td>
</tr>
<tr>
<td>Obstetric complications</td>
<td>Anoxia and trauma during delivery</td>
<td>No clear definition of key exposures</td>
<td></td>
</tr>
<tr>
<td>Infancy and childhood</td>
<td>Weight at one year</td>
<td>Nutrition and health in the first year of life</td>
<td>Also influenced by birthweight, genetic determinants of growth</td>
</tr>
<tr>
<td>Urban versus rural residence</td>
<td>Exposure to particular infections, diet differences and differences in social conditions</td>
<td>Non-specific marker of a range of possible exposures</td>
<td></td>
</tr>
<tr>
<td>Season of birth</td>
<td>Exposure after birth to seasonal infection, food scarcity or particular climatic conditions</td>
<td>Non-specific marker of a range of possible exposures which show seasonal variation</td>
<td></td>
</tr>
<tr>
<td>Recalled childhood diet</td>
<td>Aspects of childhood diet</td>
<td>Recall bias</td>
<td></td>
</tr>
<tr>
<td>Parental social class</td>
<td>A range of socially patterned exposures</td>
<td>Non-specific</td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>Socially patterned exposures, later life employment prospects, intellect</td>
<td>Non-specific</td>
<td></td>
</tr>
<tr>
<td>Household crowding</td>
<td>Infection exposure, sleep deprivation and stress</td>
<td>Strongly socially patterned</td>
<td></td>
</tr>
<tr>
<td>Adult height</td>
<td>Diet, health and psychological stress throughout the growing years</td>
<td>Also influenced by birthweight, genetic determinants of growth</td>
<td></td>
</tr>
<tr>
<td>Adult leg length</td>
<td>Pre-pubertal diet, health and psychological stress throughout the growing years</td>
<td>Also influenced by birthweight, genetic determinants of growth (but see text for its advantage over height)</td>
<td></td>
</tr>
<tr>
<td>Body mass index</td>
<td>Balance between energy intake and energy expenditure around the time of measurement</td>
<td>Also influenced by birthweight, genetic determinants of growth</td>
<td></td>
</tr>
<tr>
<td>Lung function</td>
<td>Intra-uterine growth retardation/maternal smoking in pregnancy</td>
<td>Also influenced by adult smoking and other exposures in childhood and adult life. Influences on lung growth not fully understood</td>
<td></td>
</tr>
<tr>
<td>Catch-up growth</td>
<td>Earlier in utero or childhood exposure to factors influencing growth</td>
<td>Nature of factor causing earlier growth impairment may be poorly characterized</td>
<td></td>
</tr>
<tr>
<td>Birthorder/sibship size</td>
<td>Exposure to infection</td>
<td>Non-specific marker for exposure to a range on infections and other exposures</td>
<td></td>
</tr>
</tbody>
</table>
It is of interest, therefore, that studies to date indicate that the component of height generating height-cancer and height-cardiovascular disease associations is the leg.\textsuperscript{12,16,17} This indicates that the relevant exposures underlying these associations operate pre-pubertally. A better understanding of the nature of these exposures may contribute to understanding the biological mechanisms underlying what at first sight appear to be somewhat peripheral epidemiological observations.

\textbf{The 1946 Cohort}

Previous analyses of the 1946 cohort, the UK’s first national birth cohort study, have made important contributions to understanding early life influences on adult health.\textsuperscript{4,18–21} Indeed an earlier investigation identified a range of social and pre-natal factors associated with the adult height of cohort members.\textsuperscript{22} Relevant factors were: parental height, birthweight, childhood social class, birth order, number of younger siblings, parental education and household crowding.

In this latest analysis, pre-natal, infant and childhood correlates of adult leg length and trunk length are assessed. Most of the factors investigated in the earlier analysis are also examined here. Recent coding of the childhood diet data has also enabled an investigation of diet-stature associations and the influence of infant feeding, parental divorce and death are also assessed. The availability of height measurements at ages 4, 7, 11 and 15 allows the authors to gain insights into the timing of the effects of the exposures, and availability of parental height allows for some control of genetic or inter-generational influences. However, the use of overall parental stature in this way is limited by the possibility that the two components of height may be under separate genetic influence. Information on parental leg length and trunk length was not available.
The low correlation (0.1) between leg length and trunk length underlines the possibility that the two height components provide relatively independent information on exposures influencing growth. The principle findings are that birthweight and parental height were associated with both components of height, energy intake and breastfeeding were independently associated with leg length, whereas serious illness in childhood and parental divorce were associated with trunk length.

A methodological challenge when examining associations with the components of stature is how to take account of overall body size and whether the biologically relevant measurement is proportionate or absolute length of the leg or trunk. Various approaches have been used previously. Analyses of mortality patterns in the Boyd Orr cohort models examining leg length-mortality associations included a term for trunk length and vice versa. In assessing the association of leg length and trunk length with cardiovascular disease in Caerphilly men the ratio of trunk length to leg length was used.  In Wadsworth's analysis the difference between z-scores for leg and trunk length was used. The factors most strongly related to this measure were overcrowding and breastfeeding—overcrowding leading to disproportionately short legs, and breastfeeding to long legs. The authors however make little of this analysis focussing mainly on the multivariable models for leg length and trunk length separately.

How should we interpret these findings? Many factors were examined in the models and the fact that different exposures were associated with each component of height may be a chance finding. The lack of association with most of the nutrients is not surprising in view of the limited nature of the dietary data (mothers recall of the child's diet in the previous 24 hours). This area requires consideration in birth cohorts with more detailed diet information. Whilst the association between leg length and energy intake replicates a finding in univariable analyses of the Boyd Orr cohort, in multivariable analyses in that study energy intake was associated with trunk but not leg length. The association of breastfeeding with leg length replicates findings in the Boyd Orr cohort and suggests this is an area worthy of further study. There is debate concerning the long-term impact of patterns of infant feeding on adult health, but the associations between breastfeeding and leg length suggest this may be a biologically relevant exposure underlying leg length-mortality associations.

Leg Length, Trunk Length and Chronic Disease Risk
This analysis of the 1946 birth cohort provides further evidence of the potential use of leg length as a measure of pre-pubertal exposures. Its independence from birthweight is suggested by the similarity of trunk-birthweight and leg-birthweight associations. Other analyses of the exposures influencing leg and trunk length in childhood and adulthood are now needed to confirm these findings. Similarly further analyses of risk factor and mortality associations with the components of stature are necessary to replicate the findings to date in a limited number of cohorts—Boyd Orr, Caerphilly, NHANES and the Honolulu Heart Programme. Importantly consensus regarding the relevant 'biomarker' is required—is somatic disproportion or absolute leg the more important. In the Caerphilly study, findings were similar for both measures.

References


