RESEARCH REPORT

Life course body size and lipid levels at 53 years in a British birth cohort

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Objectives: To investigate the association between growth in height and change in body mass index (BMI) during the life course on lipid levels at 53 years.

Methods: 2311 men and women from a British cohort study were included in analyses. Non-fasting total, high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol levels were measured at 53 years. Height and BMI at 2, 4, 7, 11, 15 and 36 years in relation to the lipid outcomes at 53 years were assessed using multiple regression models. The effects of z scores of height and BMI at 2 years and yearly rates of change (velocities) in height and BMI between 2–7, 7–15 and 15–36 years were also considered.

Results: Total cholesterol level decreased by 0.119 mmol/l (95% CI −0.194 to −0.045) per SD increase in height at 2 years and by 0.073 mmol/l (95% CI −0.145 to −0.001) for every SD increase in height velocity between 15 years and adulthood. Similar, but weaker associations were seen for LDL cholesterol. The relationships between leg length and total and LDL cholesterol were stronger than the relationship with trunk length. Higher BMI at 36 and 53 years and greater BMI increases between 15–36 and 36–53 years were associated with higher total and LDL cholesterol and lower HDL cholesterol levels. The effects of growth could not be explained by birth weight or lifetime socioeconomic status.

Conclusions: Early life exposures, which restrict height growth in infancy, resulting in shorter adult leg length, may influence lipid levels in adult life.

Lipid levels during childhood are age-dependent and have been shown to vary by growth rate,[1–3] and lipid levels in individuals track from childhood to adulthood.[4 5] It is unclear whether the effects of growth during childhood have long-term effects on lipid levels in addition to the effects of current height and body mass index (BMI). A few studies have suggested that leg length of adults, considered as a marker of prepubertal growth, has a stronger relationship than total height with risk factors for cardiovascular disease (CVD), including lipids,[6 7] pointing to the importance of childhood growth. Although many studies have investigated the association of birth weight, a marker of prenatal growth, with lipid levels and found the effect size to be small,[8 9] there have been few studies investigating the relationship between body size in childhood and lipid levels in adults.[3 7 10–12] Only two of these studies[11 12] have been conducted on older adults, but neither study has measures of height and weight over the entire life course.

Using heights and weights measured at 2, 4, 7, 11, 15, 36 and 53 years of age in participants from the Medical Research Council National Survey of Health and Development (NSHD), a prospective birth cohort study, we carried out an investigation of the effect of growth in height and change in BMI during childhood and adolescence on lipid levels at 53 years of age. We adjust for the potential confounding variables of social class as both height and BMI are socially distributed and there is a social gradient in cardiovascular disease risk, and birth weight, as associations between postnatal size and lipids may simply reflect birth size. We also investigated the association between adult components of height (leg and trunk length) and cholesterol levels.

METHODS

Subjects and study design

The Medical Research Council's NSHD is a birth cohort study consisting of a sample of 5362 births in 1 week of March 1946 in England, Scotland and Wales stratified by social class. There have been 21 contacts with the whole cohort from birth to the most recent follow-up when survey members were 53 years of age when 3035 cohort members (1472 men, 1563 women), out of a total of 3386 for whom contact was attempted, provided information. The majority (n = 2989) were interviewed and examined in their own homes by trained research nurses, with others completing a postal questionnaire (n = 46). Contact was not attempted for the 1976 (37%) individuals who had previously refused to take part (12%), were living abroad (11%), were untraced since last contact at 43 years (5%) or had already died (9%). Comparison of the responding sample at age 53 years with national population census data showed that it remained, in most respects, nationally representative.[13] Avoidable losses were, however, higher in those from adverse socioeconomic circumstances and in those with low scores on childhood cognitive function measures.[13]

During the home visits at 53 years, non-fasting venous blood samples were taken. Total cholesterol was measured by enzymatic cholesterol oxidase phenol 4-aminoantipyrine peroxidase. Precipitation for measurement of high-density lipoprotein (HDL) cholesterol was carried out using phosphotungstic Mg^{2+}, triglycerides were measured using a glycerol/kinase POD-linked reaction of glycerol liberated enzymatically from triglycerides. All of these measurements were made with a Bayer DAX-72. Low-density lipoprotein (LDL) cholesterol level was calculated using the Friedewald's formula:

LDL cholesterol (mmol/l) = total cholesterol − HDL cholesterol − 0.45 × triglycerides. The interview nurses recorded any information on drugs currently used by participants. This information was coded according to the British National Formulary Number 40 (2000). Cohort members on lipid

Abbreviations: BMI, body mass index; CVD, cardiovascular disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; NSHD, National Survey of Health and Development

regulating drugs, defined as any drug from the British National Formulary section 2.12, were identified.

Heights and weights were measured by trained personnel at ages 2, 4, 7, 11 and 15 years, and in adulthood at home visits at ages 36, 43 and 53 years. BMI, defined as weight/height^2, was calculated from these measurements. Sitting height was measured at age 53 years and leg length was obtained by subtracting this from the total height.

Social class was defined using the registrar general's classifications in childhood using father's occupation when the cohort member was aged 4 years, and in adulthood, based on cohort member's occupation at age 53 years. As menopausal status at age 53 years has been shown to be associated with lipid levels and BMI in the NSHD,[14] it is a potential confounder of the relationship between body size and lipid levels in women. Women were divided into five categories according to their menopausal status at 53 years of age: premenopause, perimenopause, postmenopause, hysterectomised and hormone replacement therapy users. Perimenopause and postmenopause were defined on the basis of the criteria used in the Massachusetts Women's Health Study[15] using data on menstrual characteristics collected annually in postal questionnaires to the women in the NSHD.[16]

Statistical methods

Total cholesterol, HDL and LDL cholesterol levels were all approximately normally distributed, and hence multiple linear regression models were used throughout. Men and women were analysed together, with an adjustment for sex. Tests for interaction were carried out to assess whether the effect of each body size measure varied by sex, and, where significant differences were observed, results are reported separately for men and women.

All height and BMI measures were standardised, separately for each sex, giving a mean of zero and a SD of 1. First, the associations between z scores of height and BMI at each age during childhood and in adulthood with each lipid measure were modelled. The associations with childhood measures were adjusted for adult height (36 years) and current BMI (53 years). Height at age 36 years was chosen as adult height as shrinkage might have occurred at older ages. Growth velocity variables (change in body size/change in age) were then calculated for various periods of growth: childhood (2–7 years), pubertal (7–15 years) and post-pubertal (15–36 years). An additional BMI velocity for the period 36–53 years was derived. These velocities were standardised separately for each sex. Multiple linear regression models were fitted, including standardised height and BMI at age 2 years and all the standardised height and BMI velocities. These models were then adjusted for birth weight, and tests for interaction between birth weight and each component of growth were carried out to see whether the association between growth and lipid levels was dependent on birth weight. The potential confounding variables of childhood and adult socioeconomic status were then added to the models. In addition, for women, the potential confounding effect of menopausal status at age 53 years was investigated.

Multiple regression models were also used to assess the relationship between standardised leg length, trunk length and leg:trunk ratio at 53 years and the lipid outcomes.

All analyses were repeated excluding those on current lipid-regulating drugs.

RESULTS

There were 2539 cohort members with a valid measure of total cholesterol level and height and weight measures at 53 years. Of these, 49 (3.9%) men and 21 (1.7%) women were taking cholesterol-lowering drugs. Mean total cholesterol levels were 6.05 mmol/l for males and 6.14 mmol/l for females, mean LDL levels were 3.56 mmol/l for males and 3.51 mmol/l for females, and mean HDL levels were 1.51 mmol/l for males and 1.84 mmol/l for females. These mean lipid levels were similar to those of comparable populations.[17 18]

Men without a lipid measure at age 53 years, and therefore excluded from analyses, were similar in terms of mean height and BMI at all ages to those with a valid measure. The same was true for BMI in women, but women excluded from analyses were significantly shorter than those included at ages 4, 7 and 36 years. Excluding those taking cholesterol-lowering drugs essentially made no difference to any of the analyses, and hence results presented are those from analyses including this group.

Height and BMI (absolute values)

A significant negative association was found between height at every age and total cholesterol ($p<0.01$ in all cases); the strongest associations were found with height at ages 2 and 36 years (table 1). There were negative associations with BMI at ages 2 ($p = 0.007$) and 4 years ($p = 0.003$), but positive associations with BMI in adulthood. The findings for LDL cholesterol followed a similar pattern (table 2). Higher BMI from age 11 years onwards was associated with lower HDL cholesterol ($p = 0.02$ for age 11 years and $p<0.001$ for all other ages, table 2). Tests for interaction indicated that the associations with BMI at 36 and 53 years were stronger in women, although significant in both sexes. A negative association with final stature was seen only among men (tests for interaction $p = 0.03$). Otherwise the effects of height were not significant.

After adjustment for adult height, only height at 2 years remained independently and negatively associated with total cholesterol ($p = 0.04$) and marginally associated with LDL cholesterol ($p = 0.1$). After adjustment for BMI at 53 years, the regression coefficients for BMI from 7 years onwards for both total and LDL cholesterol levels became more strongly negative, suggesting that greater BMI increases from age 7 years were associated with higher total and LDL cholesterol levels.

Changes in height and BMI

In regression models, adjusted for sex and including height and BMI at 2 years and all the growth velocities, taller height at 2 years and greater height growth after 15 years of age were associated with lower total cholesterol (table 1). Greater increases in BMI between 15 and 36 years and between 36 and 53 years were associated with higher total cholesterol levels. Results were similar for LDL cholesterol level (table 1).

There were significant differences between men and women in the effect of all height velocities and of the 7–15-year BMI velocity on HDL cholesterol level ($p \leqslant 0.05$ for each test for interaction). Hence men and women were analysed separately for this outcome (table 3). The influence of BMI change between 7 and 15 years on HDL cholesterol, although significant and negative for both sexes, was greater in women (–0.098 mmol/l per SD) than in men (–0.044 mmol/l per SD). The conditional effect of the height velocity from 7 to 15 years was negative and significant among men only (–0.061 mmol/l per SD, $p = 0.01$). A similar sex difference was seen for height velocity between 15 and 36 years.

The findings for all lipid outcomes were hardly changed after adjustment for birth weight. Birth weight was not associated with any of the lipid outcomes in these adjusted models. The only possible interaction with birth weight identified was with BMI change from 36 to 53 years for total cholesterol ($p = 0.07$), where the detrimental effect of BMI change was greater in those of low birth weight. Adjusting for social class in

Table 1 Regression coefficients (difference in mean lipid level in mmol/l) for a 1 SD increase in height and body mass index (BMI) at 2 years and subsequent yearly changes in height and BMI*

	Total (n = 1294)		LDL (n = 1206)	
	Reg coeff (95% CI)	p Value	Reg coeff (95% CI)	p Value
Sex (female)	0.098 (–0.014 to 0.211)	0.09	–0.029 (–0.136 to 0.077)	0.6
Height				
2 years	–0.119 (–0.194 to –0.045)	0.002	–0.098 (–0.169 to –0.027)	0.007
2–7 years	–0.052 (–0.122 to 0.018)	0.2	–0.050 (–0.117 to 0.017	0.1
7–15 years	–0.063 (–0.132 to 0.006)	0.07	–0.045 (–0.092 to 0.037)	0.4
15–36 years	–0.073 (–0.145 to –0.001)	0.05	–0.043 (–0.112 to 0.023)	0.2
BMI				
2 years	–0.084 (–0.197 to 0.029)	0.2	–0.078 (–0.184 to 0.028)	0.2
2–7 years	–0.041 (–0.154 to 0.072)	0.5	–0.043 (–0.150 to 0.064)	0.4
7–15 years	–0.011 (–0.073 to 0.051)	0.7	–0.013 (–0.074 to 0.048)	0.7
15–36 years	0.097 (0.037 to 0.156)	0.001	0.067 (0.009 to 0.124)	0.02
36–53 years	0.156 (0.099 to 0.214)	<0.001	0.095 (0.039 to 0.150)	0.001

BMI, body mass index; LDL, low-density lipoprotein; reg coeff, regression coefficient.
*All models include all components of the growth trajectories for height and BMI, adjusted for sex, but not adjusted for birth weight, social class or menopausal status.

childhood and adulthood, or for menopausal status among women, had little impact on the findings.

Adult leg length

Greater leg length was associated with lower total and LDL cholesterol and higher HDL cholesterol levels (table 4). Trunk length was more weakly negatively associated with lower total cholesterol and was not associated with LDL cholesterol level. The effect of trunk length on HDL cholesterol level varied by sex (test for interaction $p = 0.005$), longer trunk length being associated with lower HDL cholesterol in men only. Increases in leg:trunk ratio were thus related to lower total and LDL cholesterol levels for both sexes and higher HDL cholesterol level, particularly in men (test for interaction $p = 0.07$).

DISCUSSION

We found that taller adult height was associated with lower adult total and LDL cholesterol levels, and that tall height at 2 years and rapid growth in height after the age of 15 years were particularly protective. Higher BMI at ages 36 and 53 years and equivalently greater BMI increases between 15–36 and 36–53 years were associated with higher total and LDL cholesterol levels and lower HDL cholesterol level. In addition, fast BMI increases during the pubertal period were related to

Table 2 Regression coefficients (difference in mean lipid level in mmol/l) for a 1 SD increase in height and body mass index

		Height		BMI	
Total	Number	Reg coeff (95% CI)	p Value	Reg coeff (95% CI)	p Value
2 years†	1999	–0.111 (–0.170 to –0.031)	<0.001	–0.081 (–0.140 to –0.023)	0.007
4 years†	2201	–0.082 (–0.130 to –0.034)	0.001	–0.073 (–0.121 to –0.025)	0.003
7 years†	2122	–0.059 (–0.105 to –0.013)	0.01	–0.018 (–0.064 to 0.028)	0.4
11 years†	2128	–0.068 (–0.116 to –0.020)	0.006	–0.010 (–0.058 to 0.038)	0.7
15 years†	1965	–0.073 (–0.122 to –0.024)	0.003	–0.025 (–0.075 to 0.026)	0.3
36 years	2311	–0.104 (–0.149 to –0.059)	<0.001	0.059 (0.013 to 0.105)	0.01
53 years	2539			0.165 (0.121 to 0.208)	<0.001
LDL					
2 years†	1861	–0.071 (–0.126 to –0.016)	0.01	–0.064 (–0.118 to –0.009	0.02
4 years†	2033	–0.055 (–0.099 to –0.011)	0.01	–0.052 (–0.096 to –0.008)	0.02
7 years†	1966	–0.051 (–0.094 to –0.007)	0.02	–0.006 (–0.050 to 0.036)	0.8
11 years†	1974	–0.036 (–0.080 to 0.009)	0.1	–0.005 (–0.050 to 0.040)	0.8
15 years†	1820	–0.058 (–0.103 to –0.012)	0.01	–0.017 (–0.064 to 0.031)	0.5
36 years	2147	–0.060 (–0.101 to –0.019)	0.004	0.058 (0.014 to 0.101)	0.009
53 years	2348			0.137 (0.096 to 0.179)	<0.001
HDL					
2 years†	1871	–0.002 (–0.029 to 0.024)	0.9	–0.009 (–0.036 to 0.017)	0.5
4 years†	2044	0.014 (–0.007 to 0.034)	0.2	–0.012 (–0.032 to 0.009)	0.3
7 years†	1976	0.010 (–0.010 to 0.030)	0.4	–0.012 (–0.032 to 0.009)	0.3
11 years†	1985	–0.024 (–0.023 to 0.018)	0.8	–0.026 (–0.047 to –0.005)	0.02
15 years†	1829	0.006 (–0.015 to 0.027)	0.6	–0.039 (–0.061 to –0.018)	<0.001
36 years	2157	–0.009 (–0.028 to 0.009)	0.3	–0.120 (–0.140 to –0.100)	<0.001
53 years	2359			–0.151 (–0.169 to –0.132)	<0.001

BMI, body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein; reg coeff, regression coefficient.
All models are adjusted for sex.
*Separate models for each age, which include each pair of height and BMI for that particular age.
†Adjusted for adult height and current BMI, but not adjusted for birth weight, social class or menopausal status.

Table 3 Regression coefficients (difference in mean high-density lipoprotein cholesterol level in mmol/l) for a 1 SD increase in height and body mass index (BMI) at 2 years and subsequent yearly changes in SD scores of height and BMI separately for men and women*

	Men (n = 595)		Women (n = 618)	
	Reg coeff (95% CI)	p Value	Reg coeff (95% CI)	p Value
Height				
2 years	0.008 (–0.036 to 0.051)	0.7	0.013 (–0.033 to 0.055)	0.6
2–7 years	–0.031 (–0.073 to 0.010)	0.1	0.016 (–0.025 to 0.057)	0.4
7–15 years	–0.061 (–0.110 to –0.013)	0.01	–0.006 (–0.044 to 0.032)	0.8
15–36 years	–0.054 (–0.105 to –0.003)	0.04	–0.007 (–0.044 to 0.031)	0.7
BMI				
2 years	–0.041 (–0.107 to 0.026)	0.2	0.029 (–0.035 to 0.093)	0.4
2–7 years	–0.020 (–0.088 to 0.048)	0.6	–0.001 (–0.064 to 0.062)	0.97
7–15 years	–0.044 (–0.079 to –0.010)	0.01	–0.098 (–0.139 to –0.057)	<0.001
15–36 years	–0.094 (–0.127 to –0.060)	<0.001	–0.118 (–0.156 to –0.080)	<0.001
36–53 years	–0.072 (–0.105 to –0.039)	<0.001	–0.068 (–0.140 to –0.033)	<0.001

*All models include all components of the growth trajectories for height and BMI, but not adjusted for birth weight, social class or menopausal status.

lower HDL cholesterol. The effects of growth were not explained by birth weight, lifetime socioeconomic status or, in women, menopausal status.

This study has the advantage of having height and weight measures at various times across the whole life course as well as adult leg and trunk length. The analysis is however restricted by the ages at which the measures of height and weight were recorded. In particular, no measures were recorded between 11 and 15 years of age, meaning that parameters of potential importance, such as peak height growth velocity, could not be derived. Dealing with multiple measures of body size in relation to an outcome in statistical models is not straightforward owing to the relatively high correlations between measures.[19] The growth velocities approach followed here has been used in previous analyses of NSHD data and has been shown to produce valid conclusions.[19–21] Sensitivity analyses where models were fitted using measures of growth which were conditional on previous body size, an approach similar to that used by Law *et al*[22] to derive measures which were uncorrelated, produced very similar results to those presented in the main analyses. Non-fasting lipid measures were used owing to the large number of subjects and the time constraints, which meant that blood samples were taken at varying times of the day. Non-fasting levels of triglycerides, but not total cholesterol or HDL cholesterol levels, have been found to differ from fasting levels.[23] Triglyceride measurements were used in the calculation of LDL cholesterol level in the NSHD, possibly introducing additional variation which could explain the fact that results for LDL cholesterol followed a very similar pattern to total cholesterol but were weaker. In any long-running study such as the NSHD, missing data are unavoidable, particularly when using variables from many different contacts as with the growth data. There is no reason to suspect that the differences in those with missing data compared with the subjects analysed should have an impact on our findings. The associations between height and lipids in women may have been weakened as those excluded were shorter and may have dropped out owing to the higher risk of CVD morbidity and mortality.

In agreement with other studies,[6 7] we show that adult height was negatively associated with total and LDL cholesterol levels, but also that rapid height growth before 2 years and after 15 years may be particularly protective. No other studies have reported the effects of several different periods of height growth on lipids. A Swedish study[12] found no relationship between height gain from birth to 18 years and total cholesterol level at 58 years, but had no intermediate measures of height. For HDL cholesterol, the only evidence in the present study of an effect of height or height growth was among men, in whom taller height and greater height growth during the pubertal period (7–15 years) were associated with lower HDL cholesterol level. In agreement, the Helsinki cohort study of participants aged around 69 years found that greater increases in height between 7 and 15 years were related to lower HDL cholesterol; no association was observed with LDL cholesterol.[11] These findings may represent the long-term impact of the decreases in HDL cholesterol level with rapid height velocity during puberty observed in longitudinal studies of children.[24 25] As it is unclear as to why such a relationship is observed only in men, this finding should be treated with some caution.

Our results relating childhood height trajectories with total and LDL cholesterol levels are consistent with our findings for components of adult height if leg length, as has been suggested, is a marker of prepubertal growth and particularly growth in the first 2 years of life. For HDL cholesterol, we did not identify a period of early height growth that reflected the beneficial effect of longer legs, but the detrimental impact of fast height

Table 4 Regression coefficients (difference in mean lipid level in mmol/l) for the effect of a 1 SD increase in leg length

	Leg length at 53 years		Trunk length at 53 years		Leg:trunk ratio at 53 years	
	Reg coeff (95% CI)	p Value	Reg coeff (95% CI)	p Value	Reg coeff (95% CI)	p Value
Total (n = 2530)	–0.093 (–0.137 to –0.055)	<0.001	–0.037 (–0.080 to 0.006)	0.09	–0.057 (–0.101 to –0.013)	0.01
LDL (n = 2340)	–0.077 (–0.117 to –0.037)	<0.001	0.001 (–0.039 to 0.041)	0.96	–0.067 (–0.108 to –0.027)	0.001
HDL (n = 2351)	0.023 (0.004 to 0.041)	0.02	–0.026 (–0.044 to –0.007)	0.007	0.034 (0.015 to 0.053)	<0.001
Men	–	–	–0.052 (–0.077 to –0.028)	<0.001	0.052 (0.027 to 0.077)	<0.001
Women	–	–	0.001 (–0.027 to 0.029)	0.94	0.017 (–0.011 to 0.046)	0.2

The table shows the trunk length and leg:trunk ratio at age 53 years on lipid levels at 53 years. Models are adjusted for sex but not adjusted for birth weight, social class or menopausal status.

growth between ages 7 and 15 years seen in men is likely to reflect increases in trunk rather than leg length. As in our findings, leg length has been shown to be more strongly related to cardiovascular risk factors than trunk length.[6 26] It has therefore been hypothesised that environmental exposures, particularly in the prepubertal period, which influence leg growth, such as nutrition, infection or stress, are potential mechanisms relating early development to cardiovascular disease. The lack of confounding of the effects of height growth in our study by birth weight or socioeconomic status in childhood and adulthood suggests that neither fetal programming nor factors which are socially distributed explain all of the relationship. The effect of height growth after age 15 years on total and LDL cholesterol levels may reflect an effect of age at puberty as a previous analysis of this cohort found that those who grew fastest after the age of 15 years were those who reached puberty late.[27] The effect may be explained by subsequent differences in lifestyle because those who reach puberty earlier have been found to have poorer diets[28 29] and exercise less[28] and drink more alcohol.[30] Alternatively, it may reflect the importance of final attained height on lipid levels.

Our finding that higher adult BMI is related to higher levels of adult total and LDL cholesterol levels is in agreement with previous studies,[1 2 12] but the inverse relationship with HDL cholesterol has been less well documented.[12] We also showed that greater increases in BMI from age 15 years were associated with poorer lipid profiles. This partially represents the impact of current BMI, but may also suggest that early adult increases in BMI have a long-term impact, possibly because of the tracking of both BMI and lipid levels[31] through young adult life. Intervening factors including life course patterns of health behaviours such as diet and physical activity levels may also contribute as they influence both BMI[32 33] and lipid levels.[34 35] The interaction between birth weight and BMI change between 36 and 53 years, suggesting that the positive association of BMI change with total cholesterol level was stronger in those of low birth weight, is consistent with our previous finding that the effect of BMI at 53 years was strongest in those of low birth weight.[36] It is consistent with a programming effect where those who are born small and become overweight are at particular risk of CVD.[37]

The additional detrimental impact of a large increase in BMI on HDL cholesterol levels between 7 and 15 years is consistent with the Swedish study which found larger increases in weight between birth and 18 years to be associated with lower HDL cholesterol level at 58 years,[12] and with a study from Japan which observed a significant positive relationship with change in weight between 3 and 20 years and total cholesterol level at age 20 years.[3] Neither of these studies was able to separate childhood increases from adolescent increases. No association was found between change in weight between 7 and 15 years in the Helsinki cohort and HDL cholesterol. It is unclear why the effects of pubertal change in BMI may remain through to adulthood for HDL cholesterol level but not for total or LDL cholesterol level. However, this specific effect on HDL cholesterol level was similar to that seen for height growth between 7 and 15 years and adult trunk length in men.

CONCLUSIONS

Our findings suggest that early life exposures, such as stress, infection and poor nutrition, which restrict height growth in the first 2 years of life and subsequently result in shorter adult leg length, may influence total and LDL cholesterol levels in adult life. Fast pubertal growth in height, possibly reflecting growth in trunk length, and change in BMI may have a detrimental long-term impact on HDL cholesterol level. In those with poor early height growth who have higher total and LDL cholesterol levels, it may be particularly important to prevent fast BMI increases during adulthood which have a detrimental impact on HDL as well as total and LDL cholesterol levels.

What is already known

Size at birth is related to lipid levels in adulthood and there is limited evidence to suggest that lifecourse body size is also related to lipid outcomes, although previous studies only had limited measures available.

What this paper adds

This is the first paper to include multiple measures of body size over lifecourse of older adults and suggests that early life exposures, which restrict growth in infancy, may influence lipid levels in adult life.

Policy implications

Prevention of early life exposures, such as stress, infection and poor nutrition, are vital to ensure that infants reach their optimal height potential. Therefore it is vital that parents are aware of the importance of nutrition in babies and children.

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APHORISM OF THE MONTH

Caveat emptor – warning to consultants: the problem presented is not the real problem

Buyer beware: in dependency situations, the colonially-dominated group will tell the more powerful what they want to hear. The art of real empowerment is to get beyond the submission.

Lowell Levin and JRA