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Relation of Childhood Diet and Body Size to Menarche and Adolescent Growth in Girls

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Adolescent growth and development may be affected by factors such as dietary intake and body size from much earlier in childhood. In a longitudinal study of 67 Caucasian girls in Boston, Massachusetts, data were collected prospectively from birth during the 1930s and 1940s. Heights and weights were measured semiannually, and dietary history interviews were conducted with mothers. Stepwise linear regression methods were used to seek factors which best predicted age at menarche, adolescent peak height growth velocity, and the age at which peak growth velocity occurred. Girls who consumed more (energy-adjusted) animal protein and less vegetable protein at ages 3–5 years had earlier menarche, and girls aged 1–2 years with higher dietary fat intakes and girls aged 6–8 years with higher animal protein intakes became adolescents with earlier peak growth. Controlling for body size, girls who consumed more calories and animal protein 2 years before peak growth had higher peak growth velocity. These findings may have implications regarding adult diseases whose risks are associated with adolescent growth and development factors. *Am J Epidemiol* 2000;152:446–52.

adolescence; body mass index; child nutrition; diet; growth; menarche

There is substantial evidence that earlier menarche increases a female's lifetime risk of breast cancer (1–4), possibly because of longer exposure to estrogens, and recent reports suggest that more rapid adolescent physical growth (4) and earlier completion of adolescent growth (5) may also increase lifetime risk of breast cancer. However, earlier menarche is also associated with higher bone mineral density and lower risk of osteoporosis (6–9), possibly through longer reproductive years. The body of evidence regarding an association between earlier menarche and increased risk of ovarian cancer is mixed (10, 11). Over the past 100 years, age at menarche has declined, the adolescent growth spurt has occurred at younger ages, and peak height growth velocity has increased (12–15). Secular trends in these factors may be reflected in secular trends in related diseases. If adolescent growth and development factors are somehow causally associated with risks of adult diseases, then their associations with earlier, potentially modifiable childhood factors are relevant to disease prevention. In this paper, we consider whether early childhood and preadolescent factors (diet and body size) are associated with age at menarche, peak height growth velocity, and age at peak growth velocity.

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MATERIALS AND METHODS

Data

Dr. Harold Stuart of the Harvard School of Public Health initiated the Harvard Longitudinal Studies of Child Health and Development (16) in 1929. The Harvard Longitudinal Studies include males and females born in the 1930s and 1940s to women who were enrolled during their first trimester of pregnancy while obtaining regular prenatal care at the Boston Lying-In Hospital (Boston, Massachusetts). Women selected were considered likely to maintain residence near Boston and were committed to having their child in a long term study. Newborns with gross defects and premature births were excluded. Sixty-seven Caucasian girls were subsequently followed to age 18 years. Fifty girls who were lost to follow-up were similar in terms of birth weight, length, and head and chest circumferences (17).

For each girl, age at menarche was recorded to the exact month. Dr. Stuart personally measured the children's heights and weights from birth to adulthood (semiannually up to age 11, then annually). From the annual height measurements, we could estimate the year in which each girl experienced her most rapid adolescent height growth (peak height growth velocity (cm/year)), defined as the most growth attained during any single year in adolescence. Figure 1 shows the growth velocity (annual height increment) curve for one girl in the study, and pinpoints the age at which her height growth velocity peaked.

In the Harvard Longitudinal Studies, trained nutritional interviewers conducted a dietary history interview (lasting ~1 hour) regarding intake throughout the past 6 months with each mother while the child was undergoing the semiannual

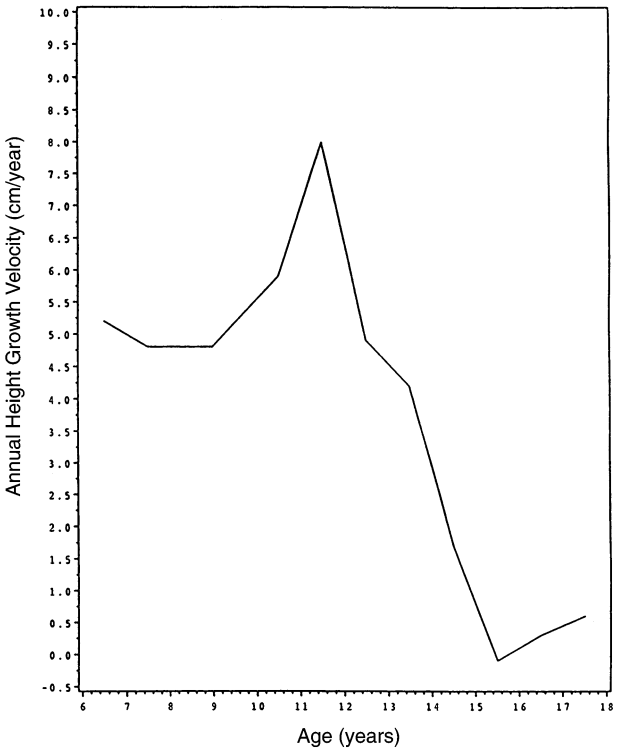

FIGURE 1. The growth velocity (annual height increment) curve for one girl in the Harvard Longitudinal Studies of Child Health and Development who was measured from age 6 years to age 18 years. Her peak height growth velocity was 8 cm/year and was reached when she was 11 years old. Her age at menarche was 12 years and 10 months.

physical examination with Dr. Stuart (18). This interview method produced estimates of consumption which were considered highly reproducible and reliable (19). For example, the reliability of total protein intake was estimated to be 71 percent, and evidence of validity was provided by the strong correlations between daily protein intake and the child's rate of growth of muscle in the lower leg ($r = 0.46$ for girls; $r = 0.68$ for boys) (19).

Total caloric intakes were age-adjusted for these analyses. Age- and calorie-adjusted dietary intakes (animal protein, vegetable protein, and total fat) took into account differences in consumption due to current body size, age, metabolic efficiency, and physical activity levels (20). (Adjusted intakes are expressed in log-scale residuals, with mean values near zero and standard deviations between 0.07 and 0.17.) This adjustment was particularly important in this analysis, because childhood height is positively correlated with total intakes of nutrients, and it is also highly correlated with the adolescent outcomes that we explored. The adjustment thus helped us to separate the effects of childhood body size and dietary intakes. Prior to analysis, these adjusted dietary intakes were averaged over multiyear periods (ages 1–2, 3–5, and 6–8 years and 1 and 2 years before peak growth). For a girl who experienced peak height growth at age 11 years, the period "1 and 2 years before peak growth" included ages 9 and 10. Diet during the years preceding the most rapid growth is more likely to be causally associated with peak growth velocity and menarche than diet observed later, which may be a result of adolescent growth needs.

Body mass indices (weight (kg)/height $(m)^2$) were computed from the child's weight and height at each examination and were then combined within the multiyear periods described above. We computed change in body mass index from age 3 years to age 8 years to evaluate whether 5-year changes in body fatness were associated with later adolescent factors. For height, age-specific Z scores were combined within each age period.

Statistical analyses

Our hypothesis was that modern dietary patterns (more calories, more fat and animal intake, less vegetable intake) are associated with earlier maturation (earlier age at peak height growth and onset of menses) and higher peak growth velocities. We had no prior hypothesis regarding the relevance of the timing of the dietary intakes (whether intakes at very young ages or intakes closer to adolescence were more influential).

Pearson correlations between the adolescent factors and the dietary intakes and body size measures (height and body mass index) from each earlier age period were computed. We then used stepwise linear regression methods (with $p < 0.05$) to search for the strongest set of predictors for each of the adolescent outcomes. We allowed childhood body size (heights and body mass indices) and dietary variables (calories, animal protein, vegetable protein, and fat) from age 1 year to age 5 years to enter each model.

In a separate analysis focusing on the older school-age period (age 6 years to preadolescence), we allowed the stepwise algorithm to select the dietary intakes, heights, and body mass indices from the 6- to 8-year age period and from the 2 years before age at peak growth velocity. Change in body mass index from age 3 to age 8, with a positive change implying that the girl became more fat, was also available for inclusion in these models. From final models, we estimated the impact that certain modifications in childhood diet might have upon adolescent growth and development.

RESULTS

These 67 girls began menarche at a mean age of 12.83 years (earliest age = 10.25 years, latest age = 15.67 years; three girls at age 10, one girl at age 15) after experiencing their peak height growth at a mean age of 11.12 years (earliest age = 9 years, latest age = 13 years), with a mean peak growth velocity of 7.99 cm/year (minimum = 5.6 cm/year, maximum = 12.1 cm/year) (table 1). Later age at menarche was associated with later age at peak growth ($r = 0.81$, $p < 0.05$) and with lower peak growth velocity ($r = -0.41$, $p < 0.05$). The girl whose growth velocity curve is shown in figure 1 is typical of this cohort regarding these characteristics. For comparison, 70 girls in the Berkeley Study, who grew up in California around the same time as our cohort, experienced peak height growth at a mean age of 11.64 years, with

TABLE 1. Mean values for three adolescent outcome variables (age at menarche, age at peak height growth velocity, and peak height growth velocity) and age-specific dietary intakes and body mass indices in 67 White girls, Harvard Longitudinal Studies of Child Health and Development

Variable	Mean	Standard deviation
Age at menarche (years)	12.83	1.09
Age at peak height growth (years)	11.12	1.15
Peak height growth velocity (cm/year)	7.99	1.29
Caloric intake (kcal/day)		
Age 1 year	1,273	173
Age 2 years	1,377	186
Age 3 years	1,483	182
Age 4 years	1,605	251
Age 5 years	1,704	246
Age 6 years	1,845	276
Age 7 years	1,930	297
Age 8 years	2,025	292
Age 9 years	2,125	378
Animal protein intake (g/day)		
Age 1 year	32.1	6.4
Age 2 years	33.9	7.4
Age 3 years	35.4	7.4
Age 4 years	38.9	7.7
Age 5 years	41.0	9.3
Age 6 years	43.0	10.0
Age 7 years	44.6	10.8
Age 8 years	46.0	9.6
Age 9 years	48.2	10.3
Vegetable protein intake (g/day)		
Age 1 year	12.2	2.4
Age 2 years	13.1	2.8
Age 3 years	13.7	2.8
Age 4 years	14.8	3.4
Age 5 years	15.7	3.1
Age 6 years	17.3	3.5
Age 7 years	18.8	4.4
Age 8 years	19.3	4.4
Age 9 years	21.2	6.0
Total fat intake (g/day)		
Age 1 year	46.8	8.2
Age 2 years	51.3	7.7
Age 3 years	57.0	9.3
Age 4 years	62.4	11.5
Age 5 years	66.2	12.6
Age 6 years	73.3	16.4
Age 7 years	77.6	17.7
Age 8 years	82.2	17.4
Age 9 years	85.5	19.4
Body mass index*		
Age 1 year	17.6	1.6
Age 2 years	16.3	1.4
Age 3 years	16.2	1.3
Age 4 years	15.9	1.4
Age 5 years	15.6	1.5
Age 6 years	15.6	1.4
Age 7 years	15.7	1.6
Age 8 years	16.1	1.9
Age 9 years	16.4	2.1

* Weight (kg)/height (m)^2.

a mean peak velocity of 8.0 cm/year (21). The Nurses' Health Study participants, who resided all over the United States during approximately the same time period, had a mean age at menarche of 12.51 years (4).

Table 1 shows age-specific (ages 1–9 years) mean values for body mass index and the unadjusted dietary intakes. The correlations among dietary intakes from different periods of childhood demonstrated relative stability over time, especially for calories and animal protein. Fat intake over time appeared less stable. As expected, heights at different ages were highly correlated, as were body mass indices (table available from the authors upon request). Table 2 presents correlations of the adolescent factors with the dietary intakes, heights, and body mass indices from each of the younger age periods. Age at menarche was associated with earlier childhood heights and animal and vegetable protein intakes; age at peak growth velocity was associated with childhood heights, caloric intake, and animal protein intake. Earlier menarche was also associated with larger relative increases in body mass index from age 3 years to age 8 years ($r = -0.28$; $p < 0.05$). Peak height growth velocity was most strongly correlated with earlier calories, animal protein, and body mass index.

Preschool predictors model

The most important childhood (ages 1–5 years) predictors of the adolescent factors are summarized in the upper portion of table 3 and were obtained using stepwise linear regression methods. Those girls who, during the age period of 3–5 years, were taller and ate less (age- and calorie-adjusted) vegetable protein and more animal protein tended to have earlier menarche. Animal protein and vegetable protein were each highly significant ($p < 0.02$) in separate models (each including height at ages 3–5 years); we show both models in the upper half of table 3. (In a model with animal and vegetable protein included together, the model R^2 was 0.28, but neither was significant because of their correlation.) Girls who were taller (at ages 3–5 years) and who ate more (age- and calorie-adjusted) fat (at ages 1–2 years) experienced adolescent peak growth at younger ages (table 3, top). Higher adolescent peak growth velocities were found in girls who consumed more (age- and calorie-adjusted) animal protein at ages 3–5 years and who consumed more calories at ages 1–2 years but had lower body mass indices between ages 3 and 5 years.

School-age predictors model

Because girls who mature early are already tall relative to their age-peers in the year or two before peak growth (22), we did not include in these models height 1 and 2 years before age at peak growth velocity; but we did permit height at ages 6–8 years to be included in the models. Girls who were taller at ages 6–8 years and who consumed more (age- and calorie-adjusted) animal protein at the same ages had earlier menarche and earlier age at peak growth velocity (lower portion of table 3). However, peak growth velocity was better predicted by variables recorded closer to adolescence: Girls who, in the 2 years preceding peak height growth, consumed more calories and animal protein but were thinner subsequently had higher growth velocities. (Not shown are results of a model in which we predicted peak growth velocity from only the age 6- to 8-year variables. The same three factors (calories, animal protein, and body mass index at ages 6–8) were significant (model R^2 =

TABLE 2. Correlations between adolescent outcome variables and age-specific dietary intakes, height, and body mass index, Harvard Longitudinal Studies of Child Health and Development†

Variable	Covariate	Age (years)			
		1–2	3–5	6–8	AGEPGV-2‡
Age at menarche (years)	Calories	–0.19	–0.14	–0.20	–0.22
	Fat	–0.14	–0.15	–0.15	–0.15
	Animal protein	–0.18	–0.41*	–0.37*	–0.36*
	Vegetable protein	+0.22	+0.35*	+0.24	+0.24
	Height	–0.35*	–0.42*	–0.43*	–0.52*
	Body mass index§	–0.08	–0.03	–0.18	+0.06
Age at peak growth velocity (years)	Calories	–0.29*	–0.27*	–0.24	–0.28*
	Fat	–0.24	–0.07	–0.17	–0.04
	Animal protein	–0.12	–0.25*	–0.37*	–0.34*
	Vegetable protein	+0.15	+0.23	+0.19	+0.18
	Height	–0.36*	–0.45*	–0.47*	–0.57*
	Body mass index	–0.12	–0.13	–0.19	+0.08
Peak height growth velocity (cm/year)	Calories	+0.42*	+0.31*	+0.35*	+0.38*
	Fat	+0.09	+0.08	+0.18	–0.21
	Animal protein	–0.01	+0.29*	+0.31*	+0.36*
	Vegetable protein	–0.09	–0.07	+0.18	–0.03
	Height	+0.26*	+0.24	+0.20	+0.21
	Body mass index	–0.24	–0.32*	–0.31*	–0.42*

* $p < 0.05$.
† Calories were age-adjusted, while intakes of fat, animal protein, and vegetable protein were both age- and calorie-adjusted.
‡ Age 2 years prior to peak growth velocity.
§ Weight (kg)/height (m)^2.

0.37).) In the peak growth velocity model, the joint appearance of low body mass index with high caloric intake seemed odd; physical activity was undoubtedly involved here (girls with high caloric intakes and high physical activity levels may have low body mass indices), but we did not have physical activity data on these girls and could not explore this further.

Implications

Table 4 provides some insight as to the magnitude of the regression model-estimated dietary effects upon adolescent factors. For example (top of table 4), a girl whose animal protein intake at ages 3–5 years was 1 standard deviation (~8 g/day (table 1)) above the mean would be expected to begin her menstrual cycles 0.63 years earlier (i.e., 13.12 – 12.49) than a girl whose animal protein intake was correspondingly low. The girl with the higher intake would also be expected to have a higher peak growth velocity (8.31 cm/year vs. 7.71 cm/year).

DISCUSSION

In this analysis, age at menarche, age at peak height growth velocity, and peak height growth velocity were all associated with diet and body size much earlier in childhood. Menarche occurred earlier in girls who were taller and who consumed more (age- and calorie-adjusted) animal protein and less vegetable protein as early as ages 3–5 years. Age at peak growth velocity was also earlier for girls who were taller before age 6 years and for those who consumed more fat (at ages 1–2 years) and animal protein (at ages 6–8 years). Regarding timing of puberty (age at menarche and age at peak growth velocity), diet and body size recorded before age 6 years predicted timing as well as those factors recorded much closer to adolescence. However, our best predictive models for peak growth velocity suggested that factors closer to puberty are more important, although the same three factors emerged from all four age periods: More calories, more animal protein, and lower body mass indices were consistently associated with higher peak growth velocity. Interestingly, childhood height did not predict peak growth velocity. Note that height growth velocities at younger ages were uncorrelated with adolescent peak growth velocity ($r = 0.02$ ($p = 0.84$) for height velocity at ages 3–5 years and $r = –0.01$ ($p = 0.90$) for height velocity at ages 6–8 years), so our findings were not confounded by earlier growth rates.

Because these dietary factors, including fat intakes, are needed for normal early childhood growth and development, the findings of our study do not support radical childhood dietary changes for the sake of disease risk modification later in life.

Other studies have examined body size and nutrition at ages closer to menarche, typically beginning at age 9, while we focused on younger childhood (ages 1–8 years). Koprowski et al.'s (23) and Moisan et al.'s (24) premenarche dietary assessments did not support an effect of any dietary factors (energy or nutrient composition) on age at menarche. Petridou et al. (25) reported that total energy intake was cor-

TABLE 3. Multiple regression models (selected using a stepwise algorithm and $p < 0.05$) for predicting adolescent outcome variables, Harvard Longitudinal Studies of Child Health and Development*

Outcome	Predictors	Model R^2
	Models with predictors measured from birth through age 5 years (sample sizes ranged from 56 to 67)	
Age at menarche (years) =	12.80 (0.12)† – 0.38 (0.12) height at ages 3–5 years + 2.19 (0.91) vegetable protein intake at ages 3–5 years	0.25
Age at menarche (years) =	12.80 (0.12) – 0.33 (0.13) height at ages 3–5 years – 2.23 (0.90) animal protein intake at ages 3–5 years	0.25
Age at peak growth velocity (years) =	11.10 (0.14) – 0.53 (0.14) height at ages 3–5 years – 3.54 (1.54) fat intake at ages 1–2 years	0.27
Peak height growth velocity (cm/year) =	14.21 (1.79) + 4.25 (1.07) calories at ages 1–2 years – 0.39 (0.11) body mass index‡ at ages 3–5 years + 2.08 (0.95) animal protein intake at ages 3–5 years	0.37
	Models with predictors measured from age 6 years to the year before age at peak growth velocity (sample size for each model was 61)§	
Age at menarche (years) =	12.83 (0.12) – 0.36 (0.13) height at ages 6–8 years – 1.69 (0.76) animal protein intake at ages 6–8 years	0.24
Age at peak growth velocity (years) =	11.12 (0.13) – 0.46 (0.13) height at ages 6–8 years – 1.70 (0.81) animal protein intake at ages 6–8 years	0.28
Peak height growth velocity (cm/year) =	12.64 (1.13) + 3.50 (0.87) calories before peak¶ – 0.28 (0.07) body mass index before peak + 3.05 (0.90) animal protein intake before peak	0.43

* Calories were age-adjusted (natural log scale); intakes of fat, animal protein, and vegetable protein were age- and calorie-adjusted (natural log scale). Heights were age-specific Z scores ((height – mean))/standard deviation).
† Numbers in parentheses, standard error of regression coefficient.
‡ Weight (kg)/height $(m)^2$.
§ These models excluded height 1 and 2 years before age at peak growth velocity.
¶ During the 2 years before peak growth velocity was attained.

related with later menarche, a finding not observed in our cohort, and they found no associations with energy-adjusted macronutrients; but their data were collected much closer to menarche (the youngest age was 9 years), when energy intake might represent adolescent physical activity levels. However, Meyer et al. (26) found that higher dietary energy intake was associated with earlier menarche and dietary composition was not. In another study by Merzenich et al. (27), higher energy-adjusted fat intake was associated with earlier menarche.

Many studies have previously reported that taller girls (23, 25, 28, 29) and girls with more body fat (23, 25, 27–31) have earlier menarche. Other studies have similarly reported that boys and girls with earlier adolescent growth spurts were taller in preadolescent periods and that children who matured earlier had stronger peak growth velocities (13, 22, 32, 33), though not all have noted the latter association (34).

These adolescent growth and development variables, which potentially affect adult disease risks, appear to be responsive to environmental influences. Higher levels of physical activity in girls have been found to be associated with delayed menarche (27). Hughes and Jones (35) reported that menarcheal age was later in countries where dietary fiber intake was higher. Eiben (36) presented data showing later onset of menarche among girls of parents with a lower educational level, which may be a marker for lower socioeconomic status. Onset of menarche and age at peak height growth velocity tend to be earlier with urbanization (15). Age at menarche is also affected by ethnicity, social class differences, number of siblings, and secular trends (13). The importance of the potential link between some of these adolescent factors and risk of adult diseases is that they may be modifiable, as suggested by our analyses, whereas other risk factors such as family history of a disease are not amenable to intervention.

The small size of our longitudinal sample is obviously a limitation of this study, but the meticulous data collection by the same individuals over many years of follow-up should have partially compensated for it. This cohort of Boston children, which was followed for 18 years, requiring very cooperative mothers, is probably not representative of US children at that time, and their participation in this cohort may have resulted in their having better diets or health care. Because many of these children were born during the Depression (1930s) and matured during World War II (1940s), our findings also may not be relevant to present-day girls growing up amid more affluence. Studies in other US populations have illustrated substantial differences in

TABLE 4. Effects of childhood diet on adolescent outcome variables, Harvard Longitudinal Studies of Child Health and Development*

	Age at menarche (years)	Age at peak growth velocity (years)	Peak height growth velocity (cm/year)
Animal protein intake at ages 3–5 years			
1 SD† above mean value	12.49		8.31
1 SD below mean value	13.12		7.71
Replacement of animal protein intake with vegetable protein intake at ages 3–5 years‡			
Animal protein 1 SD above mean value and vegetable protein 1 SD below mean value	12.37		
Animal protein 1 SD below mean value and vegetable protein 1 SD above mean value	13.24		
Dietary fat intake at ages 1–2 years			
1 SD above mean value		10.80	
1 SD below mean value		11.43	
Total calories at ages 1–2 years			
1 SD above mean value			8.54
1 SD below mean value			7.43
Diet 1 and 2 years before age at peak growth velocity			
Calories and animal protein 1 SD above mean value			8.95
Calories and animal protein 1 SD below mean value			7.03

* Estimates are predicted values from the table 3 regression models, with other predictors in the model held constant at their mean values. Calories were age-adjusted; fat and protein intakes were age- and calorie-adjusted.

† SD, standard deviation.

‡ A model including both animal protein and vegetable protein was used here.

the adolescent growth patterns of Black and White children (22, 33), which suggests further difficulties in generalizing these results to Black girls. Another limitation is the lack of physical activity data on these children, since physical activity is related to both adiposity and energy intake. The effect of random measurement errors in dietary intake on our results should have been that our correlations and regression model estimates were biased toward the null, causing us to overlook or underestimate the magnitude and significance of any real associations.

Several lines of evidence suggest that adult disease risks may be influenced by adolescent factors, and this paper suggests that several adolescent factors might be modifiable by dietary changes in young children. Current public health efforts designed to reduce intakes of saturated fat and red meat and excess calories and to increase fruit/vegetable intakes in children could potentially result in lower breast cancer rates, along with other health benefits; but the risk of osteoporosis, and perhaps of other diseases, might increase.

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