Published in final edited form as:

Cancer Causes Control. 2018 October ; 29(10): 915–926. doi:10.1007/s10552-018-1068-2.

Childhood Diet And Growth In Boys In Relation To Timing Of Puberty And Adult Height: The Longitudinal Studies Of Child Health And Development

Aliya Alimujiang, MPH^{1,2}, Graham A. Colditz, MD DrPH¹, Jane D. Gardner, ScD³, Yikyung Park, ScD¹, Catherine S. Berkey, PhD⁴, and Siobhan Sutcliffe, PhD¹

¹Division of Public Health Sciences and the Alvin J. Siteman Cancer Center, Department of Surgery, Washington University School of Medicine, St Louis, MO, United States.

²Department of Epidemiology, School of Public Health, University of Michigan, Ann Arbor, MI, United States.

³Consultant, Bedford, MA, United States.

⁴Channing Division of Network Medicine, Department of Medicine, Brigham & Women's Hospital and Harvard Medical School, Boston, MA, United States.

Abstract

Purpose: To begin to explore the possible roles of childhood diet and growth in prostate cancer (PCa) development, we investigated these exposures in relation to two known/suspected PCa risk factors, earlier pubertal timing and greater attained height, in the Longitudinal Studies of Child Health and Development.

Methods: We used biannual/annual height, weight, and dietary history data to investigate childhood diet, body mass index (BMI), birth length, and childhood height in relation to PCa risk factors (age at peak height velocity (APHV), height at age 13, and adult height) for 64 Caucasian-American boys.

Corresponding author information: Siobhan Sutcliffe, Division of Public Health Sciences, Department of Surgery, Washington University School of Medicine, 600 S. Taylor Avenue, 2nd floor, Rm. 208S, Box 8100, St Louis, MO 63110. sutcliffes@wustl.edu, Telephone: +1 314-362-3788.

Authors' contributions

Aliya Alimujiang performed the scientific literature review, and contributed to the design of the present analysis, data analysis, interpretation of the data, and drafting and critical revision of the manuscript. She also reviewed and approved the final submitted version.

Graham A. Colditz contributed to the design of the present analysis, data analysis, interpretation of the data, and critical revision of the manuscript. He also approved the final submitted version.

Jane D. Gardner contributed to the design of the parent study, data collection, and critical revision of the manuscript. She also approved the final submitted version.

Yikyung Park contributed to the interpretation of the data and critical revision of the manuscript. She also approved the final submitted version.

Catherine S. Berkey contributed to the design of the present analysis, data collection, data analysis, interpretation of the data, and critical revision of the manuscript. She also approved the final submitted version.

Siobhan Sutcliffe contributed to the design of the present analysis, data analysis, interpretation of the data, and drafting and critical revision of the manuscript. She also approved the final submitted version.

Financial Disclosure Statement

The authors have no financial relationships relevant to this article to disclose.

Conflict of Interest Statement

The authors have no conflicts of interest relevant to this article to disclose.

Results: In adjusted models, childhood fat and animal protein intake was positively associated with height at age 13 and adult height ($P<0.05$). A childhood diet high in fat and animal protein and low in vegetable protein was also associated with earlier APHV ($P<0.05$), whereas no associations were observed for childhood energy intake or BMI. Birth length and childhood height were positively associated with height at age 13 and adult height, and childhood height was inversely associated with APHV ($P<0.05$).

Conclusion: Our findings suggest that both childhood diet and growth potential/growth contribute to earlier pubertal timing and taller attained height in males, supporting roles these factors in PCa development.

INTRODUCTION

Although prostate cancer (PCa) is the most common cancer and the third leading cause of cancer death in American men, few modifiable risk factors have been identified (1). One possible reason for this dearth of information may be the predominant focus of epidemiologic research on mid- to later-life exposures long after the prostate has developed. However, an accumulating body of evidence from several different disciplines supports an earlier life contribution to PCa risk – for instance, during gestation and adolescence when the prostate grows and develops rapidly (2–5). In mouse models, stronger associations have been observed for *in utero* and adolescent exposures with prostate lesion development than for adult exposures (6–8), and in humans, small foci of high-grade prostatic intraepithelial neoplasia and PCa have been detected in men starting in their 20s (9–15), and differences in the prevalence of these lesions, as well as in PCa incidence and mortality, are already apparent by race in men in their 30s–50s (2, 16, 17). These findings suggest that exposures responsible for these differences must have occurred several decades beforehand.

Despite these compelling observations, few studies have focused on early-life exposures because of inherent difficulties in studying this earlier life stage – most notably, the long span of time between early-life and PCa diagnosis. This long time span makes traditional epidemiologic study designs, such as case-control or cohort studies, challenging and motivates the development of new strategies to avoid the limitations of either decades-long recall or decades-long participant follow-up (2). One such strategy, which has been used successfully for breast cancer, is to study early-life exposures in relation to disease risk factors (e.g., earlier menarche) rather than in relation to the disease itself (e.g., breast cancer) (18). Although conclusive risk factors have not yet been identified for PCa, findings from recent studies of puberty using more accurate measures than in the past – i.e., measured height at age 13 (19, 20) and genetic markers of pubertal timing (21) – suggest that earlier timing of puberty may increase PCa risk/mortality. In addition, greater adult height has been found consistently to be associated with elevated PCa risk/mortality (22, 23), and thus may be a further useful tool for studying early-life exposures, as it can be measured well before PCa diagnosis.

In the present study, we sought to use these two factors – pubertal timing and adult height – to begin to explore the possible influence of childhood diet and body size on PCa development, using existing data from the Longitudinal Studies of Child Health and

Development. We focused on these two exposures because of their strong ecological and biological support (i.e., diet (24–28)), and their preliminary support from previous observational studies (i.e., inverse findings for large childhood body size and PCa, possibly through delayed pubertal timing (29)). We also included birth length and childhood height in our analyses to capture, albeit crudely, the independent influence of genetics (i.e., birth length), and the combined influence of diet and genetics (height) on our outcomes of interest. These analyses were modelled after our previous investigation of girls in the Longitudinal Studies of Child Health and Development (30), in which we observed significant associations for childhood fat and animal protein intake, and body mass index (BMI) with earlier timing of puberty (age at menarche and age at peak height velocity (APHV)) and greater speed of puberty (peak height velocity (PHV)).

MATERIALS AND METHODS

Study population and design

Beginning in 1929, the Longitudinal Studies of Child Health and Development enrolled pregnant women obtaining regular prenatal care at the Boston Lying-in Hospital and intending to deliver and receive their postnatal care at that hospital. Women were enrolled as early as possible during pregnancy (typically at the beginning of the second trimester), and were selected if they were likely to remain in the Boston area and committed to having their child participate in a long-term study. Children born prematurely or with birth defects were excluded, leaving 229 participants in the study. Children were followed by in-person examinations at: birth; 14 days; 3, 6, 9, and 12 months; semi-annually from 1–10 years; annually from 11–18 years; and once during adulthood. Of the 229 children enrolled in the study, 95 were lost to follow-up, leaving 67 boys and 67 girls in the study until age 18 (31). We further excluded three boys with incomplete dietary data for a final analytic sample size of 64 boys for all analyses.

Diet assessment

Diet in the past six months or year was assessed by dietary history interviews (32). Interviews were performed by the study nutritionist and completed by participants' mothers while their children underwent study visits. The nutritionist used participants' dietary interview data to estimate their average daily intake of energy and a small number of macronutrients (total fat, animal protein, and vegetable protein) in the past six months or year.

Anthropometric measurements

Height (or length) and weight were measured at each visit by the Principal Investigator/study pediatrician (Dr. Harold Stuart), except during World War II (1942–43) when he was on war assignment. During this time, study visits were interrupted, resulting in one or more missing values for all participants. We imputed these values by taking the average (for one missing value) or the predicted value from a linear regression model (for >1 consecutive missing values), using participants' nearest non-missing measurements. Imputed data were used to derive our anthropometric exposures and outcomes of interest. Exposures included childhood BMI as a measure of childhood body size, and birth length as a *crude* marker of

genetic growth potential (33–35), recognizing that this variable is also influenced by gestational length and maternal nutrition (36). Birth length was included rather than birth weight because birth weight was only available for a subset of participants (n=45). Finally, we examined childhood height as a *crude* marker of the combined influence of genetic growth potential and diet.

Outcomes of interest were: 1) APHV, a marker of pubertal timing that captures a later developmental event; 2) height at age 13, a variable previously found to be associated with later PCa risk and mortality (19–21) and one that we hypothesized captures both the timing of puberty and height potential; and 3) adult height. We also explored PHV, a marker of the speed of pubertal growth, as an outcome because of its previously observed associations with childhood diet and BMI in girls in the Longitudinal Studies of Child Health and Development (30). PHV was estimated by selecting the maximum value of each participant's annual growth rates (cm/year) and APHV was defined as the age at which participants experienced their PHV.

Statistical analysis

Prior to the analysis, we grouped all exposure data into multiple-year age categories, similar to our previous analysis in girls (1–2, 3–5 years, 6–8, and 9–10 years (30)). Age group-specific estimates of dietary and anthropometric measures were calculated by taking the average of each participant's values within that particular age group. Values for dietary measures were adjusted first for year of age and energy intake by the residual method (37), and then averaged to obtain age- and energy-adjusted estimates. We investigated age group-specific associations between dietary and anthropometric measures and each outcome of interest by computing Pearson correlation coefficients. As several of our exposures were highly correlated with each other (e.g., macronutrient intake at successive ages, and total fat and animal protein intake), we developed summary scores to examine the combined, rather than independent, influence of these highly correlated exposures on our outcomes of interest. Specifically, we created summary measures for childhood: 1) fat and animal protein intake; 2) vegetable protein intake; and 3) height (as a *crude* marker of the combined influence of diet and genetic growth potential) from ages 1–10. We did not create summary scores for energy intake or BMI because of their null findings in correlation analyses. Scores were calculated by assigning a value from 1–4 to each exposure quartile within each age group, and then by summing these values to obtain scores ranging from 4–16 (values for the fat-animal protein score were divided by 2). Associations between these scores and each outcome were investigated by linear regression. Finally, to help interpret our findings in the broader context of markers (and possibly mechanisms) of PCa risk, we examined the correlation between outcomes to inform whether these measures reflected common or distinct pathways of risk. Analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC).

RESULTS

All participants were Caucasian of predominantly northern European descent from low- to middle-class families in Boston in the 1930s. As expected, participants' intake of energy and

macronutrients, including total fat, animal protein, and vegetable protein, increased with age from 1 to 10 years, although the percentage contribution of each nutrient to energy intake remained relatively constant with age: ~32–36% for fat, 9–10% for animal protein, and 4% for vegetable protein (Table 1). These values were within recommended ranges for protein, but at the high end of the range for fat from 4 years of age onwards. Energy and nutrient intake demonstrated a high degree of autocorrelation with age.

With respect to anthropometric exposures, the mean birth length was 49.7 cm. Mean BMI was relatively stable with age (15.2–17.9 kg/m²) with a low percentage of overweight and obesity (4–13%) (38). Height increased steadily with age similar to CDC 1977 and 2000 reference standards. All anthropometric exposures demonstrated high degrees of autocorrelation (Table 2). With respect to our outcomes of interest, mean values were 13.6 years for APHV, 8.6 cm/year for PHV, 156.0 cm for height at age 13, and 179.1 cm for adult height. Figure 1 illustrates the median growth trajectory for participants up to age 18, as well as the 10th and 90th percentiles.

In age- and energy-adjusted analyses, greater fat intake at several childhood ages, particularly ages 9–10, was correlated with an earlier APHV, greater PHV, and taller height at age 13 and in adulthood. Generally similar findings were observed for animal protein intake. Greater intake of animal protein at ages 3–5 and 9–10 was correlated with an earlier APHV, and greater animal protein intake from ages 1–10 was correlated with taller height at age 13 and in adulthood. In contrast to these findings, greater vegetable protein intake at ages 9–10 was correlated with later APHV, and greater vegetable protein intake from ages 1 to 10 was correlated with shorter height at age 13. Energy intake was not significantly correlated with any of the outcomes of interest. With respect to our anthropometric exposures, no statistically significant correlations were observed for BMI with any of the outcomes of interest, with the possible exception of suggestive positive correlations for BMI at ages 1–2 with adult height, and BMI at ages 6–8 with height at age 13. Greater birth length was correlated with taller height at age 13 and in adulthood. Finally, greater height from ages 1–10 was correlated with earlier APHV, taller height at age 13, and taller adult height. Correlations tended to be stronger with increasing age (Table 3).

Given the similarity of findings across nutrients, we next investigated the correlation between dietary measures. At most ages, greater energy-adjusted fat intake was correlated with greater animal protein intake, and increased fat and animal protein intakes were correlated with lesser vegetable protein intake (Appendix Table 1). Given this high degree of inter-nutrient correlation, as well as the previously observed high autocorrelation with age, we developed overall dietary scores (childhood fat and animal protein intake, and childhood vegetable protein intake from ages 1–10) to reduce concerns of collinearity and to examine the combined, rather than independent, influence of our exposures. Using these new scores, we observed that boys with greater childhood fat and animal protein intake from ages 1–10 had an earlier APHV, and were taller at age 13 and as adults (Table 4, unadjusted estimates; and Figure 2). In contrast, boys with greater childhood vegetable protein intake had a later APHV and were shorter at age 13. With respect to birth length, boys longer at birth were taller at age 13 and as adults. Finally, children who were taller from ages 1–10 had an earlier

APHV and were taller at age 13 and in adulthood. We did not explore energy intake or BMI in regression analyses because of their non-significant findings in correlation analyses.

To explore the relative contributions of lifestyle and genetics to our outcomes of interest, we next included both childhood diet and birth length in the same model, and found that the point estimates attenuated slightly but remained statistically significant (Table 4, columns 2, 5, and 8). Further adjustment of the childhood fat and animal protein intake findings for vegetable protein intake resulted in a small degree of attenuation for the point estimates for height at age 13 or adult height (Table 4, column 3), and attenuation to a non-significant value for APHV. Results for childhood vegetable protein intake with APHV and height at age 13 attenuated to the null after adjustment for childhood fat and animal protein intake (column 6). We did not adjust the findings for childhood height for birth length and childhood diet because we believe these findings reflect the combined influence of genetics and diet. Finally, strong correlations were observed for height at age 13 with both APHV ($r=-0.70$, $p<0.0001$) and adult height ($r=0.51$, $p<0.0001$), but not for APHV with adult height ($r=-0.03$, $p=0.79$).

DISCUSSION

In this longitudinal study of American boys, we observed that a childhood diet high in fat and animal protein and low in vegetable protein (i.e., a “Western-style” diet) was associated with an earlier APHV, and that both childhood diet and birth length were associated with taller stature at age 13 and in adulthood. Consistent with these findings, greater childhood height (from ages 1–10), which we included as a combined measure of childhood diet and genetic growth potential, was also associated with earlier APHV, taller stature at age 13, and taller adult height. Finally, strong correlations were observed between height at age 13 and both APHV and adult height, but not between APHV and adult height, suggesting distinct pathways of action for APHV and adult height, but possibly overlapping pathways for height at age 13 with APHV and adult height – i.e., height at age 13 may serve as a marker of both earlier timing of puberty and greater height potential.

Our findings for childhood diet and body size are consistent with those from the small number of studies conducted to date. Specifically, our findings for childhood diet and APHV are in line with those from our previous study of girls in the Longitudinal Studies of Childhood Growth and Development (30), as well as those from several other, but not all (39–43), prospective studies of adequately-nourished boys (44, 45) and girls (44–50) of primarily European ancestry. In addition, although few cohort studies have investigated the influence of childhood diet on adult height, our findings are consistent with those from studies that examined the correlation between individual and per-capita intake of animal protein and adult height (51, 52). For energy intake, our null findings are consistent with most of the available literature, suggesting no association between childhood energy intake and measures of puberty, at least in studies predominantly of girls (18, 53). In contrast, our null findings for childhood BMI differ from most previous studies that observed positive associations for pre-pubertal BMI/adiposity with earlier onset or speed of puberty in boys (53–62), and inverse associations with adult height (58, 59). Reasons for these differences are not immediately clear, as positive/inverse findings were observed across studies from a

range of different countries and calendar periods. Our findings for birth length and childhood height are consistent with those from most previous studies of boys, supporting positive associations for birth length and adult height (33–35), inverse associations for childhood height and timing of puberty (53, 55, 56, 63), and positive associations for childhood and adult height (59, 64). Finally, our null findings for timing of puberty and attained height differ from most previous studies that observed that boys who entered puberty earlier were shorter as adults (58, 59, 65). However, reasons for these discrepancies are unclear.

While the relations for childhood diet and genetic growth potential with pubertal timing and attained height are important in and of themselves, our primary goal in examining these associations was to inform the contribution of these factors to PCa risk. By interpreting pubertal timing (19–21) and adult height (22, 23) as markers of PCa risk, our findings suggest that both childhood consumption of a “Western-style” diet and genetic growth potential may contribute to risk, and that they may do so through two separate mechanisms related to earlier puberty and greater attained height. Elements of a Western diet may contribute to earlier puberty by several possible mechanisms, including: 1) increased leptin secretion, which may be permissive for puberty initiation (44); 2) increased insulin-like growth factor 1 (IGF-1) secretion, which regulates growth and has been related to earlier puberty in some studies (45, 52, 66); 3) increased insulin secretion, which suppresses IGF binding protein 1 secretion, and thus may allow greater circulating IGF-1 levels (45); and 4) increased androgen and other steroid hormone levels (67). Genetically determined levels of each of these factors could also conceivably contribute to earlier puberty initiation. Once initiated, puberty has been proposed to contribute to later PCa risk largely by extending the time during which the prostate is exposed to androgens (2). With respect to adult height, the most commonly proposed mechanism by which elements of a Western diet may contribute to taller height is by increased IGF-I secretion. In addition to promoting height, elevated levels of this growth factor may influence cancer risk by stimulating epithelial cell proliferation, preventing apoptosis, and amplifying the effects of DNA-damaging agents. It is also possible that taller height may serve as a marker of greater organ size and/or cellularity, thereby increasing the pool of cells at risk for transformation (68). Finally, as earlier pubertal timing was uncorrelated with adult height in our analysis and has frequently been found to be inversely correlated with shorter adult height in previous analyses (58, 59, 65), it is possible that additional pathways besides IGF-1 may contribute to either adult height or pubertal timing.

Our study has several limitations and strengths that merit discussion. Besides our use of markers of PCa risk, we were also limited, to some extent, by our exposure and outcome assessments. With respect to diet, we were limited by the small number of macronutrients estimated from dietary interviews, which precluded a comprehensive analysis of diet and dietary patterns. Similarly, for genetic growth potential, we were limited by lack of access to biologic specimens (e.g., blood, saliva) and information on parental characteristics (e.g., timing of puberty and attained height) from which to infer genetic potential. Furthermore, although birth length informs growth potential, it is also determined by other factors, such as maternal nutrition (a possible correlate of offspring childhood nutrition) and gestational length (36). Finally, for pubertal timing, we were limited to two possible markers of this

process: APHV, an event that occurs later during puberty and is difficult to estimate even with serial height measurements; and height at age 13, which likely captures both pubertal timing as well as participants' height potential. Therefore, even though we performed many analyses and adjustments, it was not possible for us to discern the relative contributions of childhood diet and genetics to our outcomes of interest. Additional limitations include our small sample size, which may have hindered our ability to detect weaker associations; lack of access to other possible determinants of pubertal timing and adult height (e.g., having been breastfed, passive smoking exposure, socioeconomic status, and physical activity) (18); and restriction to American boys of European descent born in the 1930s. Therefore, whether our findings apply to boys of other race/ethnicities or boys born more recently in the current obesity epidemic is unclear.

Offsetting the above-described limitations are several unique strengths. These include our prospective study design; repeated measurement of anthropometric variables from birth through to early-adulthood by the same study investigator; and repeated collection of diet by a well-validated method of dietary assessment (dietary history interviews by a trained nutritionist). These strengths increase the validity of our exposure and outcome measurements, particularly compared to values recalled later in life. Finally, use of APHV as a marker of timing of puberty aligns well with the onset of pubertal prostate growth, as APHV has been shown to correlate with genital Tanner stages, which in turn are correlated with levels of prostate-specific antigen, a marker of prostate growth during adolescence (69, 70). Therefore, although our outcomes were derived from height, they should still capture growth and development of our target organ of interest.

In summary, our study provides evidence that both greater childhood intake of fat and animal protein, and greater genetic growth potential contribute to earlier pubertal timing and taller attained height in males. As both of these outcomes have been found to be associated with PCa risk, our findings support roles for childhood diet and growth in PCa development, and suggest that early-life may be a promising life stage to study for PCa etiology. Future studies should explore these findings further for their insight into PCa mechanisms and primary PCa prevention. In addition, although not the focus of our study, our findings also offer insight into the etiology of other cancers or chronic conditions associated with pubertal timing or adult height, and could be used to further prevention in those fields as well.

Acknowledgments

Funding source

AA, GAC and CSB were supported by the Breast Cancer Research Foundation. YP and SS were supported by the Foundation for Barnes-Jewish Hospital and the Alvin J. Siteman Cancer Center (P30 CA091842). The funders had no role in the study design, data collection, analysis, interpretation of data, preparation of the report, or decision to publish. All authors had full access to the data and analyses, and had final responsibility for the decision to submit for publication.

APPENDIX

TABLE 1a.

Correlations between intake of macronutrients^a at ages 1–2 years in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

Macronutrient	Fat	Animal protein	Vegetable protein
Fat	1.00	0.21	−0.54 **
Animal protein	0.21	1.00	−0.53 **
Vegetable protein	−0.54 **	−0.53 **	1.00

**
p<0.05.

^aIntakes of fat, animal protein, and vegetable protein were adjusted for age and energy.

TABLE 1b.

Correlations between intake of macronutrients^a at ages 3–5 years in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

Macronutrient	Fat	Animal protein	Vegetable protein
Fat	1.00	0.30 **	−0.58 **
Animal protein	0.30 **	1.00	−0.48 **
Vegetable protein	−0.58 **	−0.48 **	1.00

**
p<0.05.

^aIntakes of fat, animal protein, and vegetable protein were adjusted for age and energy.

TABLE 1c.

Correlations between intake of macronutrients^a at ages 6–8 years in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

Macronutrient	Fat	Animal protein	Vegetable protein
Fat	1.00	0.52 **	−0.55 **
Animal protein	0.52 **	1.00	−0.47 **
Vegetable protein	−0.55 **	−0.47 **	1.00

**
p<0.05.

^aIntakes of fat, animal protein, and vegetable protein were adjusted for age and energy.

TABLE 1d.

Correlations between intake of macronutrients^a at ages 9–10 years in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

Macronutrient	Fat	Animal protein	Vegetable protein
Fat	1.00	0.53 **	−0.39 **
Animal protein	0.53 **	1.00	−0.28 **
Vegetable protein	−0.39 **	−0.28 **	1.00

**
p<0.05.

^aIntakes of fat, animal protein, and vegetable protein were adjusted for age and energy.

REFERENCES

1. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2018. *CA Cancer J Clin* 2018;68(1):7–30. [PubMed: 29313949]
2. Sutcliffe S, Colditz GA. Prostate cancer: is it time to expand the research focus to early-life exposures? *Nature reviews Cancer* 2013;13(3):208–518. [PubMed: 23363989]
3. Rotkin ID. Studies in the epidemiology of prostatic cancer: expanded sampling. *Cancer Treat Rep* 1977;61(2):173–80. [PubMed: 68821]
4. Ross R, Bernstein L, Judd H, Hanisch R, Pike M, Henderson B. Serum testosterone levels in healthy young black and white men. *Journal of the National Cancer Institute* 1986;76(1):45–8. [PubMed: 3455741]
5. Andersson SO, Baron J, Wolk A, Lindgren C, Bergstrom R, Adami HO. Early life risk factors for prostate cancer: a population-based case-control study in Sweden. *Cancer Epidemiol Biomarkers Prev* 1995;4(3):187–92. [PubMed: 7606192]
6. Suttie AW, Dinse GE, Nyska A, Moser GJ, Goldsworthy TL, Maronpot RR. An investigation of the effects of late-onset dietary restriction on prostate cancer development in the TRAMP mouse. *Toxicologic pathology* 2005;33(3):386–97. [PubMed: 15805078]
7. Suttie A, Nyska A, Haseman JK, Moser GJ, Hackett TR, Goldsworthy TL. A grading scheme for the assessment of proliferative lesions of the mouse prostate in the TRAMP model. *Toxicologic pathology* 2003;31(1):31–8. [PubMed: 12597447]
8. Angelsen A, Falkmer S, Sandvik AK, Waldum HL. Pre- and postnatal testosterone administration induces proliferative epithelial lesions with neuroendocrine differentiation in the dorsal lobe of the rat prostate. *Prostate* 1999;40(2):65–75. [PubMed: 10386466]
9. Hansen JL, McMeekin RR. Unsuspected tumors in aircraft accident fatalities as a guide to evaluation of physical-examination standards. *Aerosp Med* 1974;45(8):959–62. [PubMed: 4367456]
10. Sakr WA, Grignon DJ, Crissman JD, Heilbrun LK, Cassin BJ, Pontes JJ, et al. High grade prostatic intraepithelial neoplasia (HGPIN) and prostatic adenocarcinoma between the ages of 20–69: an autopsy study of 249 cases. *In Vivo* 1994;8(3):439–43. [PubMed: 7803731]
11. Elliott GB, Silverberg DS, Dossetor JB, Muir CS. Latent carcinoma of the prostate in a 24-year-old man receiving cyclophosphamide and azathioprine. *Can Med Assoc J* 1977;116(6):651–2. [PubMed: 612349]
12. Davis BE, Weigel JW. Adenocarcinoma of the prostate discovered in 2 young patients following total prostatesctomy for refractory prostatitis. *J Urol* 1990;144(3):744–5. [PubMed: 2388343]
13. Gu FL, Xia TL, Kong XT. Preliminary study of the frequency of benign prostatic hyperplasia and prostatic cancer in China. *Urology* 1994;44(5):688–691. [PubMed: 7526525]
14. Sanchez-Chapado M, Olmedilla G, Cabeza M, Donat E, Ruiz A. Prevalence of prostate cancer and prostatic intraepithelial neoplasia in Caucasian Mediterranean males: an autopsy study. *Prostate* 2003;54(3):238–47. [PubMed: 12518329]
15. Yin M, Bastacky S, Chandran U, Becich MJ, Dhir R. Prevalence of incidental prostate cancer in the general population: a study of healthy organ donors. *J Urol* 2008;179(3):892–5; discussion 5. [PubMed: 18207193]
16. Jahn JL, Giovannucci EL, Stampfer MJ. The high prevalence of undiagnosed prostate cancer at autopsy: implications for epidemiology and treatment of prostate cancer in the Prostate-specific Antigen-era. *Int J Cancer* 2015;137(12):2795–802. [PubMed: 25557753]
17. Howlander N, Noone AM, Krapcho M, Neyman N, Aminou R, Waldron W, et al. SEER Cancer Statistics Review, 1975–2009 (Vintage 2009 Populations) National Cancer Institute Bethesda, MD, http://seer.cancer.gov/csr/1975_2009_pops09/, based on November 2011 SEER data submission, posted to the SEER web site, 4 2012.

18. Cheng G, Buyken AE, Shi L, Karaolis-Danckert N, Kroke A, Wudy SA, et al. Beyond overweight: nutrition as an important lifestyle factor influencing timing of puberty. *Nutr Rev* 2012;70(3):133–52. [PubMed: 22364156]
19. Cook MB, Gamborg M, Aarestrup J, Sorensen TI, Baker JL. Childhood height and birth weight in relation to future prostate cancer risk: a cohort study based on the copenhagen school health records register. *Cancer Epidemiol Biomarkers Prev* 2013;22(12):2232–40. [PubMed: 24089459]
20. Aarestrup J, Gamborg M, Cook MB, Baker JL. Childhood height increases the risk of prostate cancer mortality. *Eur J Cancer* 2015;51(10):1340–5. [PubMed: 25899985]
21. Bonilla C, Lewis SJ, Martin RM, Donovan JL, Hamdy FC, Neal DE, et al. Pubertal development and prostate cancer risk: Mendelian randomization study in a population-based cohort. *BMC Med* 2016;14:66. [PubMed: 27044414]
22. Zuccolo L, Harris R, Gunnell D, Oliver S, Lane JA, Davis M, et al. Height and prostate cancer risk: a large nested case-control study (ProtecT) and meta-analysis. *Cancer Epidemiol Biomarkers Prev* 2008;17(9):2325–36. [PubMed: 18768501]
23. Emerging Risk Factors C Adult height and the risk of cause-specific death and vascular morbidity in 1 million people: individual participant meta-analysis. *Int J Epidemiol* 2012;41(5):1419–33. [PubMed: 22825588]
24. Gronberg H Prostate cancer epidemiology. *Lancet* 2003;361(9360):859–64. [PubMed: 12642065]
25. Armstrong B, Doll R. Environmental factors and cancer incidence and mortality in different countries, with special reference to dietary practices. *Int J Cancer* 1975;15(4):617–31. [PubMed: 1140864]
26. Hsing AW, Tsao L, Devesa SS. International trends and patterns of prostate cancer incidence and mortality. *Int J Cancer* 2000;85(1):60–7. [PubMed: 10585584]
27. Chan JM, Gann PH, Giovannucci EL. Role of diet in prostate cancer development and progression. *Journal of clinical oncology : official journal of the American Society of Clinical Oncology* 2005;23(32):8152–60. [PubMed: 16278466]
28. McCullough ML, Giovannucci EL. Diet and cancer prevention. *Oncogene* 2004;23(38):6349–64. [PubMed: 15322510]
29. Robinson WR, Poole C, Godley PA. Systematic review of prostate cancer's association with body size in childhood and young adulthood. *Cancer causes & control : CCC* 2008;19(8):793–803. [PubMed: 18347923]
30. Berkey CS, Gardner JD, Frazier AL, Colditz GA. Relation of childhood diet and body size to menarche and adolescent growth in girls. *American journal of epidemiology* 2000;152(5):446–52. [PubMed: 10981459]
31. Stuart HC, Reed RB. Longitudinal studies of child health and development--Series II. Description of project. *Pediatrics* 1959;24:875–85. [PubMed: 13835357]
32. Reed RB, Burke BS. Collection and analysis of dietary intake data. *American journal of public health and the nation's health* 1954;44(8):1015–26.
33. Sorensen HT, Sabroe S, Rothman KJ, Gillman M, Steffensen FH, Fischer P, et al. Birth weight and length as predictors for adult height. *American journal of epidemiology* 1999;149(8):726–9. [PubMed: 10206622]
34. Tuvemo T, Cnattingius S, Jonsson B. Prediction of male adult stature using anthropometric data at birth: a nationwide population-based study. *Pediatr Res* 1999;46(5):491–5. [PubMed: 10541308]
35. Eide MG, Oyen N, Skjaerven R, Nilsen ST, Bjerkedal T, Tell GS. Size at birth and gestational age as predictors of adult height and weight. *Epidemiology* 2005;16(2):175–81. [PubMed: 15703531]
36. Burke BS, Stevenson SS, et al. Nutrition studies during pregnancy; relation of maternal nutrition to condition of infant at birth; study of siblings. *J Nutr* 1949;38(4):453–67. [PubMed: 18140049]
37. Willett W *Nutritional Epidemiology* 3rd ed. Hofman A, Marmot M, Samet J, Savitz DZ, editors. New York NY: Oxford University Press; 2013.
38. Centers for Disease Control and Prevention, *Defining Childhood Obesity* 2015.
39. Meyer F, Moisan J, Marcoux D, Bouchard C. Dietary and physical determinants of menarche. *Epidemiology* 1990;1(5):377–81. [PubMed: 2078613]

40. Maclure M, Travis LB, Willett W, MacMahon B. A prospective cohort study of nutrient intake and age at menarche. *The American journal of clinical nutrition* 1991;54(4):649–56. [PubMed: 1897472]
41. Moisan J, Meyer F, Gingras S. Diet and age at menarche. *Cancer causes & control : CCC* 1990;1(2):149–54. [PubMed: 2102285]
42. Koprowski C, Ross RK, Mack WJ, Henderson BE, Bernstein L. Diet, body size and menarche in a multiethnic cohort. *Br J Cancer* 1999;79(11–12):1907–11. [PubMed: 10206312]
43. Carwile JL, Willett WC, Wang M, Rich-Edwards J, Frazier AL, Michels KB. Milk Consumption after Age 9 Years Does Not Predict Age at Menarche. *J Nutr* 2015;145(8):1900–8. [PubMed: 26136590]
44. Cheng G, Gerlach S, Libuda L, Kranz S, Gunther AL, Karaolis-Danckert N, et al. Diet quality in childhood is prospectively associated with the timing of puberty but not with body composition at puberty onset. *J Nutr* 2010;140(1):95–102. [PubMed: 19923386]
45. Gunther AL, Karaolis-Danckert N, Kroke A, Remer T, Buyken AE. Dietary protein intake throughout childhood is associated with the timing of puberty. *J Nutr* 2010;140(3):565–71. [PubMed: 20042466]
46. de Ridder CM, Thijssen JH, Van 't Veer P, van Duuren R, Bruning PF, Zonderland ML, et al. Dietary habits, sexual maturation, and plasma hormones in pubertal girls: a longitudinal study. *The American journal of clinical nutrition* 1991;54(5):805–13. [PubMed: 1951150]
47. Merzenich H, Boeing H, Wahrendorf J. Dietary fat and sports activity as determinants for age at menarche. *American journal of epidemiology* 1993;138(4):217–24. [PubMed: 8356963]
48. Koo MM, Rohan TE, Jain M, McLaughlin JR, Corey PN. A cohort study of dietary fibre intake and menarche. *Public Health Nutr* 2002;5(2):353–60. [PubMed: 12020388]
49. Rogers IS, Northstone K, Dunger DB, Cooper AR, Ness AR, Emmett PM. Diet throughout childhood and age at menarche in a contemporary cohort of British girls. *Public Health Nutr* 2010;13(12):2052–63. [PubMed: 20529402]
50. Kissinger DG, Sanchez A. The association of dietary factors with the age of menarche. *Nutrition Research* 1987;7:471–9.
51. Grasgruber P, Cacek J, Kalina T, Sebera M. The role of nutrition and genetics as key determinants of the positive height trend. *Economics and human biology* 2014;15:81–100. [PubMed: 25190282]
52. Wiley AS. Does milk make children grow? Relationships between milk consumption and height in NHANES 1999–2002. *Am J Hum Biol* 2005;17(4):425–41. [PubMed: 15981182]
53. Mills JL, Shiono PH, Shapiro LR, Crawford PB, Rhoads GG. Early growth predicts timing of puberty in boys: results of a 14-year nutrition and growth study. *J Pediatr* 1986;109(3):543–7. [PubMed: 3746550]
54. Amador M, Bacallao J, Hermelo M. Body mass index at different ages and its association with height at age 14 and with the whole growing process. *Nutrition* 1996;12(6):416–22. [PubMed: 8875536]
55. Monteilh C, Kieszak S, Flanders WD, Maisonet M, Rubin C, Holmes AK, et al. Timing of maturation and predictors of Tanner stage transitions in boys enrolled in a contemporary British cohort. *Paediatr Perinat Epidemiol* 2011;25(1):75–87. [PubMed: 21133972]
56. Juul A, Magnusdottir S, Scheike T, Prytz S, Skakkebaek NE. Age at voice break in Danish boys: effects of pre-pubertal body mass index and secular trend. *Int J Androl* 2007;30(6):537–42. [PubMed: 17459124]
57. Silventoinen K, Haukka J, Dunkel L, Tynelius P, Rasmussen F. Genetics of pubertal timing and its associations with relative weight in childhood and adult height: the Swedish Young Male Twins Study. *Pediatrics* 2008;121(4):e885–91. [PubMed: 18381517]
58. Sandhu J, Ben-Shlomo Y, Cole TJ, Holly J, Davey Smith G. The impact of childhood body mass index on timing of puberty, adult stature and obesity: a follow-up study based on adolescent anthropometry recorded at Christ's Hospital (1936–1964). *Int J Obes (Lond)* 2006;30(1):14–22. [PubMed: 16344844]
59. He Q, Karlberg J. BMI in childhood and its association with height gain, timing of puberty, and final height. *Pediatr Res* 2001;49(2):244–51. [PubMed: 11158521]

60. Buyken AE, Karaolis-Danckert N, Remer T. Association of prepubertal body composition in healthy girls and boys with the timing of early and late pubertal markers. *The American journal of clinical nutrition* 2009;89(1):221–30. [PubMed: 19056586]
61. Garn SM, Greaney GR, Young RW. Fat thickness and growth progress during infancy. *Hum Biol* 1956;28(2):232–52. [PubMed: 13345362]
62. De Leonibus C, Marcovecchio ML, Chiavaroli V, de Giorgis T, Chiarelli F, Mohn A. Timing of puberty and physical growth in obese children: a longitudinal study in boys and girls. *Pediatr Obes* 2014;9(4):292–9. [PubMed: 23713062]
63. Boyne MS, Thame M, Osmond C, Fraser RA, Gabay L, Reid M, et al. Growth, body composition, and the onset of puberty: longitudinal observations in Afro-Caribbean children. *The Journal of clinical endocrinology and metabolism* 2010;95(7):3194–200. [PubMed: 20427487]
64. Tanner JM, Landt KW, Cameron N, Carter BS, Patel J. Prediction of adult height from height and bone age in childhood. A new system of equations (TW Mark II) based on a sample including very tall and very short children. *Arch Dis Child* 1983;58(10):767–76. [PubMed: 6639123]
65. Nierop AF, Niklasson A, Holmgren A, Gelander L, Rosberg S, Albertsson-Wikland K. Modelling individual longitudinal human growth from fetal to adult life - QEPS I. *J Theor Biol* 2016;406:143–65. [PubMed: 27297288]
66. Villamor E, Jansen EC. Nutritional Determinants of the Timing of Puberty. *Annu Rev Public Health* 2016;37:33–46. [PubMed: 26789387]
67. Fleshner N, Zlotta AR. Prostate cancer prevention: past, present, and future. *Cancer* 2007;110(9):1889–99. [PubMed: 17893870]
68. Gunnell D, Okasha M, Smith GD, Oliver SE, Sandhu J, Holly JM. Height, leg length, and cancer risk: a systematic review. *Epidemiol Rev* 2001;23(2):313–42. [PubMed: 12192740]
69. Vieira JG, Nishida SK, Pereira AB, Arraes RF, Verreschi IT. Serum levels of prostate-specific antigen in normal boys throughout puberty. *The Journal of clinical endocrinology and metabolism* 1994;78(5):1185–7. [PubMed: 7513717]
70. Tanner JM, Whitehouse RH. Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. *Archives of disease in childhood* 1976;51(3):170–9. [PubMed: 952550]

FIGURE 1.
Median growth velocity (annual height increment) of 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development*.

*The median growth velocity curve was estimated by linking consecutive age-specific median annual height increment values from 1 to 18 years of age for all boys in the Longitudinal Studies of Child Health and Development. The pinpoint at the right side of the figure is the age at which participants reached their median peak height velocity (PHV, 7.10cm/year at 13.5 years of age).

FIGURE 2. Group-specific median growth velocity (annual height increment) for 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development*.

*The median growth velocity curves were estimated by linking consecutive group-specific median annual height increment values from 1 to 18 years of age for boys in the highest and lowest quartiles of each exposure of interest in the Longitudinal Studies of Child Health and Development.

TABLE 1.

Mean age-specific energy and macronutrient intakes in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

	Mean $\pm$ SD	Min-Max	En% ^a	Reference intake
Energy intake (kcal/day)				
Age 1 year	1,287 $\pm$ 198	800–1,800		1,200 ^b
Age 2 years	1,403 $\pm$ 204	800–1,900		1,410 ^b
Age 3 years	1,544 $\pm$ 211	1,050–2,000		1,560 ^b
Age 4 years	1,629 $\pm$ 207	1,200–2,200		1,690 ^b
Age 5 years	1,792 $\pm$ 253	1,200–2,400		1,810 ^b
Age 6 years	1,968 $\pm$ 298	1,450–2,800		1,900 ^b
Age 7 years	2,013 $\pm$ 299	1,450–2,650		1,990 ^b
Age 8 years	2,159 $\pm$ 392	1,400–3,925		2,070 ^b
Age 9 years	2,235 $\pm$ 387	1,500–3,225		2,150 ^b
Age 10 years	2,403 $\pm$ 425	1,700–3,800		2,150 ^b
<i>Within-person correlation over time^c</i>	0.73	0.65–0.81		
Total fat intake (g/day)				
Age 1 year	45.7 $\pm$ 9.3	22.0–68.0	32.0%	30%–40% ^d
Age 2 years	53.4 $\pm$ 9.6	29.0–86.0	34.7%	30%–40% ^d
Age 3 years	59.4 $\pm$ 10.3	36.0–86.0	34.9%	30%–40% ^d
Age 4 years	63.0 $\pm$ 9.4	44.0–92.0	35.1%	25%–35% ^d
Age 5 years	69.3 $\pm$ 11.9	43.0–96.0	35.2%	25%–35% ^d
Age 6 years	75.7 $\pm$ 14.6	51.0–117.0	35.0%	25%–35% ^d
Age 7 years	77.2 $\pm$ 14.7	54.0–118.0	34.8%	25%–35% ^d
Age 8 years	82.0 $\pm$ 18.7	50.0–170.0	34.6%	25%–35% ^d
Age 9 years	87.3 $\pm$ 18.2	52.0–146.0	35.6%	25%–35% ^d
Age 10 years	94.3 $\pm$ 20.4	64.0–144.0	35.7%	25%–35% ^d
<i>Within-person correlation over time^c</i>	0.63	0.46–0.82		
Animal protein intake (g/day)				
Age 1 year	30.6 $\pm$ 6.5	15.0–43.8	9.6%	
Age 2 years	32.5 $\pm$ 7.0	17.5–50.0	9.4%	
Age 3 years	35.7 $\pm$ 7.3	17.5–55.0	9.3%	
Age 4 years	37.7 $\pm$ 7.0	25.0–57.5	9.3%	5%–20% ^d
Age 5 years	39.8 $\pm$ 7.4	25.0–60.0	9.0%	
Age 6 years	44.0 $\pm$ 9.1	25.0–67.5	9.1%	

	Mean $\pm$ SD	Min-Max	En% ^a	Reference intake
Age 7 years	44.9 $\pm$ 9.1	27.5–75.0	9.1%	
Age 8 years	46.8 $\pm$ 9.9	25.0–80.0	8.9%	
Age 9 years	49.9 $\pm$ 10.4	27.5–50.0	9.1%	
Age 10 years	53.4 $\pm$ 11.5	35.5–85.0	9.1%	
<i>Within-person correlation over time^c</i>	<i>0.69</i>	<i>0.64–0.81</i>		
Vegetable protein intake (g/day)				
Age 1 year	13.0 $\pm$ 3.0	7.5–19.4	4.1%	
Age 2 years	13.6 $\pm$ 30.7	7.5–22.5	3.9%	
Age 3 years	14.4 $\pm$ 3.5	10.0–25.0	3.8%	
Age 4 years	15.5 $\pm$ 3.5	7.5–22.5	3.8%	
Age 5 years	17.9 $\pm$ 5.4	8.8–37.5	4.0%	
Age 6 years	19.6 $\pm$ 5.1	10.0–35.0	4.0%	5%–20% ^d
Age 7 years	20.5 $\pm$ 5.7	10.0–37.5	4.1%	
Age 8 years	22.6 $\pm$ 6.6	10.0–40.0	4.2%	
Age 9 years	22.6 $\pm$ 6.5	2.5–38.8	4.0%	
Age 10 years	24.6 $\pm$ 7.2	12.5–45.0	4.1%	
<i>Within-person correlation over time^c</i>	<i>0.63</i>	<i>0.46–0.72</i>		

^a EN% represents the percentage of calories from a certain macronutrient per day. It was calculated by dividing the calories for each nutrient by the total energy consumed by each boy.

^b World Health Organization. (1991). Energy and protein requirements: report of a joint FAO/WHO/UNU expert consultation.

^c Calculated as the average of sequential pairwise Pearson correlation coefficients.

^d Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. (2005), Journal of the American Dietetic Association, 102(11), 1621–1630.

TABLE 2.

Mean anthropometric and pubertal measures in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

	Mean $\pm$ SD	Min-Max	Percentage of overweight and obese children ^a	1977 reference	2000 reference
Anthropometric exposures					
Birth length (cm)	49.7 $\pm$ 2.3	43.2–54.4	-----	49.99 ^b	49.99 ^c
BMI (kg/m ²)					
Age at 1 year	17.9 $\pm$ 1.5	14.2–22.2	Not available	Not available	Not available
Age at 2 years	16.6 $\pm$ 1.2	14.6–20.5	8.96%	16.58 ^b	16.58 ^c
Age at 3 years	16.1 $\pm$ 1.1	13.2–19.0	13.43%	16.02 ^b	16.00 ^c
Age at 4 years	15.6 $\pm$ 1.0	13.4–18.6	7.46%	15.64 ^b	15.63 ^c
Age at 5 years	15.4 $\pm$ 1.0	13.5–18.0	8.96%	15.42 ^b	15.42 ^c
Age at 6 years	15.2 $\pm$ 1.0	13.2–17.8	4.48%	15.38 ^b	15.38 ^c
Age at 7 years	15.4 $\pm$ 1.2	13.3–18.5	7.46%	15.50 ^b	15.51 ^c
Age at 8 years	15.6 $\pm$ 1.4	13.0–19.6	7.46%	15.77 ^b	15.78 ^c
Age at 9 years	16.0 $\pm$ 1.6	13.5–20.7	5.97%	16.15 ^b	16.17 ^c
Age at 10 years	16.5 $\pm$ 2.0	13.5–22.8	8.96%	16.62 ^b	16.65 ^c
<i>Within-person correlation over time^d</i>	<i>0.86</i>	<i>0.74–0.93</i>			
Height (cm)					
Age at 1 year	75.5 $\pm$ 2.6	70.0–84.0	-----	77.52 ^b	76.12 ^c
Age at 2 years	87.2 $\pm$ 3.1	81.8–98.6	-----	87.25 ^b	87.66 ^c
Age at 3 years	95.2 $\pm$ 3.5	89.0–106.0	-----	94.95 ^b	95.45 ^c
Age at 4 years	102.3 $\pm$ 3.9	94.8–113.0	-----	102.22 ^b	102.51 ^c
Age at 5 years	109.1 $\pm$ 4.4	100.2–120.0	-----	108.90 ^b	109.18 ^c
Age at 6 years	115.5 $\pm$ 4.9	106.4–127.2	-----	115.39 ^b	115.66 ^c
Age at 7 years	121.6 $\pm$ 5.2	111.2–134.6	-----	121.77 ^b	122.03 ^c
Age at 8 years	127.5 $\pm$ 5.6	115.8–141.0	-----	127.88 ^b	128.12 ^c
Age at 9 years	133.1 $\pm$ 6.1	120.3–149.2	-----	133.51 ^b	133.73 ^c
Age at 10 years	138.3 $\pm$ 6.6	124.2–152.7	-----	138.62 ^b	138.82 ^c
<i>Within-person correlation over time^d</i>	<i>0.96</i>	<i>0.89–0.99</i>			
Anthropometric/pubertal outcomes					
Age at peak height velocity (years)	13.6 $\pm$ 1.3	10.6–19.1	-----	13.9 ^e	13.7 ^f
Peak height velocity (cm/year)	8.6 $\pm$ 2.0	5.2–15.2	-----	9.49 ^e	9.51 ^f

	Mean $\pm$ SD	Min-Max	Percentage of overweight and obese children ^a	1977 reference	2000 reference
Height at age 13 (cm)	156.0 $\pm$ 9.6	137.2–177.0	-----	152.25 ^e	156.41 ^c
Adult height (cm)	179.1 $\pm$ 7.0	161.0–196.5	-----	176.16 ^e	176.19 ^c

^aCalculated using the Centers for Disease Control and Prevention Recommended BMI-for-age cutoffs for overweight and obesity. Centers for Disease Control and Prevention. National Center for Health Statistics. 1977 Individual Growth Charts. <https://www.cdc.gov/nccdphp/dnpao/growthcharts/training/bmiage/page4.html>

^bCenters for Disease Control and Prevention. National Center for Health Statistics. 1977 Individual Growth Charts.<http://www.cdc.gov/growthcharts/charts.htm>. Reference data for BMI were available for ages 2–20 years only.

^cCenters for Disease Control and Prevention. National Center for Health Statistics. 2000 Individual Growth Charts. <https://www.cdc.gov/growthcharts/charts.htm>. Reference data for BMI were available for ages 2–20 years only.

^dCalculated as the average of sequential pairwise Pearson correlation coefficients.

^eTanner, J. M., & Whitehouse, R. H. (1976). Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. Archives of disease in childhood, 51(3), 170–179.

^fKelly A, Winer KK, Kalkwarf H, Oberfield SE, Lappe J, Gilsanz V, Zemel BS. Age-based reference ranges for annual height velocity in US children. The Journal of Clinical Endocrinology & Metabolism. 2014 Jun 1;99(6):2104–12.

TABLE 3.

Correlations for childhood diet^a, body size, and growth potential/growth with adolescent growth and attained height in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

Measures of adolescent growth and attained height	Energy intake (cal) at age (years):				
	0	1–2	3–5	6–8	9–10
Age at peak height velocity (years)		0.1	–0.06	–0.08	–0.07
Peak height velocity (cm/year)		–0.15	0.06	0.12	–0.06
Height at 13 years of age (cm)		0.00004	0.14	0.12	0.12
Adult height (cm)		0.13	0.13	0.08	0.10
	Fat intake (g) at age (years):				
	0	1–2	3–5	6–8	9–10
Age at peak height velocity (years)		–0.04	–0.23 *	–0.23 *	–0.25 **
Peak height velocity (cm/year)		0.09	0.09	0.17	0.26 **
Height at 13 years of age (cm)		0.17	0.32 **	0.34 **	0.36 **
Adult height (cm)		0.01	0.13	0.26 *	0.45 **
	Animal protein (g) at age (years):				
	0	1–2	3–5	6–8	9–10
Age at peak height velocity (years)		–0.21 *	–0.27 **	–0.12	–0.27 **
Peak height velocity (cm/year)		0.16	0.03	0.08	0.17
Height at 13 years of age (cm)		0.37 **	0.42 **	0.36 **	0.48 **
Adult height (cm)		0.26 **	0.35 **	0.33 **	0.33 **
	Vegetable protein (g) at age (years):				
	0	1–2	3–5	6–8	9–10
Age at peak height velocity (years)		0.16	0.19	0.2	0.41 **
Peak height velocity (cm/year)		–0.1	–0.13	–0.01	0.06
Height at 13 years of age (cm)		–0.25 **	–0.31 **	–0.33 **	–0.36 **
Adult height (cm)		–0.02	–0.12	–0.1	–0.02
	BMI (kg/m ²)				
	0	1–2	3–5	6–8	9–10
Age at peak height velocity (years)		–0.07	0.06	–0.02	–0.05
Peak height velocity (cm/year)		–0.03	–0.05	–0.15	–0.22
Height at 13 years of age (cm)		0.07	0.09	0.27 *	0.24
Adult height (cm)		0.24 *	0.19	0.18	0.12
	Birth length/height (cm)				
	0	1–2	3–5	6–8	9–10

Measures of adolescent growth and attained height	Energy intake (cal) at age (years):				
	0	1–2	3–5	6–8	9–10
Age at peak height velocity (years)	−0.17 ^b	−0.35 ^{**}	−0.40 ^{**}	−0.44 ^{**}	−0.46 ^{**}
Peak height velocity (cm/year)	0.18 ^b	0.18	0.07	0.08	0.12
Height at 13 years of age (cm)	0.33 ^{**b}	0.74 ^{**}	0.85 ^{**}	0.89 ^{**}	0.91 ^{**}
Adult height (cm)	0.37 ^{**b}	0.64 ^{**}	0.65 ^{**}	0.62 ^{**}	0.64 ^{**}

* 0.05 p<0.1

** p<0.05

^a Energy intake was adjusted for age. Intakes of fat, animal protein, and vegetable protein were adjusted for age and energy.

^b Birth length (cm)

Associations^a for childhood dietary scores, birth length, and childhood height (from 1 to 10 years of age) with adolescent growth and attained height in 64 Caucasian boys born in the 1930s, Longitudinal Studies of Child Health and Development.

TABLE 4.

Measures of adolescent growth and attained height	Childhood fat and animal protein score (Score range from 4–16)			Childhood vegetable protein score (Score range from 4–16)			Birth Length (cm)		Childhood Height Score (Score range from 4–16)	
	β	β^b	β^c	β	β^b	β^c	β	β^c	β	β
Age at peak height velocity (years)	-0.149**	-0.139**	-0.109	0.094**	0.091**	0.034	-0.094	-0.068	-0.155**	
Peak height velocity (cm/year)	0.105	0.085	0.156	-0.008	-0.003	0.079	0.155	0.127	0.023	
Height at 13 years of age (cm)	1.789**	1.637**	1.400**	-1.043**	-1.002**	-0.265	1.393**	1.070**	2.150**	
Adult height (cm)	0.884**	0.741**	1.178**	-0.168	-0.133	-0.487	1.141**	0.923**	1.143**	

* 0.05 p<0.1
** p<0.05

^a Beta coefficient (β) from a linear regression model.

^b Adjusted for birth length.

^c Adjusted for the fat and animal protein score, vegetable protein score, and birth length, as appropriate.