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Original Contribution

Childhood Overweight and Obesity and Pubertal Onset Among Mexican-American Boys and Girls in the CHAMACOS Longitudinal Study

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Secular trends in earlier initiation of puberty have been observed in recent decades. One risk factor appears to be increases in adiposity, as measured by body mass index. This trend is particularly notable among Latino populations, who have higher rates of overweight/obesity compared with non-Latino White youth. Previous research has focused primarily on White girls, resulting in data gaps regarding male puberty and among potentially high-risk populations. Using data from the Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS) study, we examined body mass index at age 5 years (2005–2006) and multiple markers of pubertal onset, assessed repeatedly and longitudinally at 7 in-person visits, starting at age 9 and continuing through age 14 (2009–2015), among 336 Mexican Americans in Salinas, California. We observed no associations among boys, but found significantly earlier thelarche in overweight (HR = 1.7, 95% CI: 1.1, 2.7) and obese girls (HR = 1.5, 95% CI: 1.0, 2.4), menarche in overweight girls (HR = 1.6; CI: 1.0, 2.4), and pubarche in obese girls (HR = 1.9; CI: 1.2, 3.0), compared with normal-weight girls. This study examined an understudied population and included key covariates, such as birth weight and early adverse events, which are typically omitted in studies.

adversity; childhood obesity; Mexican American; puberty

Abbreviations: BMI, body mass index; CHAMACOS, Center for the Health Assessment of Mothers and Children of Salinas.

Editor's note: *An invited commentary on this article appears on page 17.*

The initiation of puberty has been occurring earlier in developed countries, with more consistent evidence of earlier pubertal onset among girls than boys (1–8). Earlier puberty has been associated with a host of negative health outcomes, including depression, cardiovascular disease, cancer, and all-cause mortality (9–16), and it is therefore of major public health concern. One of the primary factors that accounts for this secular trend is increases in overweight and obesity (5, 8, 17–19). Previous studies, which have focused primarily on White populations (20), suggest that higher body mass index (BMI) in childhood is associated with earlier puberty among girls (2, 5, 8, 18, 21–28). However, the association between BMI and pubertal onset is more complicated among boys (29), and previous studies with boys have shown mixed results (19, 25–28, 30–33).

The role of BMI in timing of pubertal onset is particularly important to study among Latino youth, given their higher prevalence of obesity compared with non-Latino Whites (34, 35). Studies have also found that lower childhood socioeconomic status can be associated with earlier puberty (36, 37), and Latino populations in the United States are disproportionately affected by poverty (38). Moreover, ethnic minorities such as Blacks and Latinos start puberty earlier than their White counterparts (39–41), which signals that overweight/obesity might be a causal factor driving disparities in timing of pubertal onset.

Several challenges in data acquisition and interpretation limit our understanding of this phenomenon. Many studies are cross-sectional, measure BMI too late in childhood to capture weight status before onset of puberty, or do not

span long enough time periods to disentangle the temporal order of prepubertal weight and puberty onset among both boys and girls, who enter puberty on average at different ages (8, 42). Studies have also varied in their assessment of pubertal stage and often rely on self-report rather than the gold standard, physical exam (43, 44). While several studies find associations of higher BMI and earlier puberty in girls (2, 5, 8, 18, 21–28), few have controlled for early-life adversity, an established antecedent of earlier pubertal timing in girls (45), despite its potential influence on both BMI and puberty. Even fewer studies have examined the association of BMI with timing of puberty in boys (19, 25–28, 30–33), and research among potentially vulnerable populations including Latino youth, those living in nonurban settings, and those experiencing multiple adversities has been extremely limited.

Here, we address these limitations by using data from an ongoing longitudinal study of Mexican Americans growing up in a low-income agricultural region of California. We tested associations between prepubertal BMI at age 5 years and multiple markers of pubertal onset assessed repeatedly from ages 9 to 14 years. We also tested associations between waist circumference, a measure of abdominal fat, at age 5 and at pubertal onset. A number of early-life adversity exposures (assessed prospectively from 0 to 5 years of age) were included as covariates.

METHODS

Study population

The Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS) is a longitudinal birth cohort study that was designed to examine the effects of environmental exposures on the health of pregnant women and their children living in the agricultural region of Salinas Valley, California. Study recruitment and procedures have been described previously (46). Briefly, pregnant women were recruited from Salinas Valley community health clinics in 1999–2000. Eligibility criteria included age of ≥18 years, being at <20 weeks of gestation, speaking Spanish and/or English, and qualifying for low-income health insurance. Of the 601 women initially enrolled, 527 remained in the study through delivery. We restricted the present analyses to participants who had BMI measured at age 5 and who completed at least 1 pubertal assessment conducted between 9 and 14 years of age ($n = 302$). We omitted participants who were not of Latino origin ($n = 5$), for a total of 298 participants in the present investigation (136 boys, 162 girls). The majority of attrition occurred before age 3, and year-to-year retention has been >95% since age 9. Overall, participants who remained in the study were demographically similar when compared with those who did not, although they were more likely to have mothers who were older, had lived in the United States longer, and were overweight or obese compared with those who were lost to follow-up (Web Table 1, available at https://doi.org/10.1093/aje/kwab100).

Written informed consent was obtained by bilingual English- and Spanish-speaking staff from mothers at all study visits. Youth provided verbal assent up to age 12 years and written assent at all visits following the 12-year assessment. All documents were provided in English and Spanish and read aloud as needed. Study procedures were approved by the University of California Berkeley Committee for Protection of Human Subjects.

Outcome assessment

Tanner staging (47) by physical exam was used to determine pubertal stage. Trained research assistants (under the supervision of pediatric endocrinologists) examined participants at 7 assessment points repeatedly and prospectively, starting at the 9-year visit: 9 years (n = 326), 9.75 years (n = 268), 10.5 years (n = 313), 11.25 years (n = 291), 12 years (n = 330), 12.75 years (n = 298), and 14 years (n = 257). Participants were not evaluated at age 14 if they had already reached Tanner stage 5 at a previous visit. Palpation and visual inspection were used to assess girls' breast development (thelarche), and visual inspection was used to determine pubic hair development (pubarche). Research assistants were trained to distinguish breast from adipose tissue. Female participants were asked if they had reached menarche at each visit, and age at menarche (year/month) was determined at the first visit after menarche. Visual inspection and assessment using orchidometer beads (>3 cm for Tanner stage 2) were used to determine boys' genital development (gonadarche), and visual inspection was used for pubic hair (pubarche) development. In quality control assessments, the research assistants' categorization of whether a child was in stage 2+ versus stage 1 agreed with that of the pediatric endocrinologists 90% and 92% of the time for girls' breast and pubic hair stage, respectively, and 92% and 100% of the time for boys' genital and pubic hair stage, respectively. To assess pubertal onset, we focused on the transition from Tanner stage 1 (no development) to Tanner stage 2 (development) for each pubertal marker. Age at menarche was treated as a continuous variable.

BMI and waist circumference exposure assessment

We measured the child's weight and height in light clothing without shoes or jackets at the 5-year study visit. We measured height (meters) using a fixed stadiometer (Seca 222; Seca, Chino, California) and body weight (kilograms) using a foot-to-foot bioimpedance scale (TBF 300A; Tanita, Arlington Heights, Illinois). Waist circumference was measured by placing a measuring tape around the abdomen at the level of the iliac crest, parallel to the floor. Height and waist circumference measurements were conducted in triplicate and averaged for analysis. BMI was calculated as weight (kg)/height $(m)^2$. Age- and sex-standardized BMI z scores were calculated using Centers for Disease Control and Prevention (CDC) norms (48). BMI was categorized into <85th percentile for normal, 85th–95th percentile for overweight, and ≥95th percentile for obese. Anthropometrics measurements at age 5 were selected to capture early childhood body weight parameters that occurred before the changes in body fat that are part of the pubertal transition.

Covariate data collection

Study staff administered structured questionnaires to mothers at multiple time points from delivery through child's age 14 years to collect demographic data. Here we focus on covariates assessed from 0–5 years. In cases where these were measured more than once, composite measures were created as described below. Covariates were selected a priori based on previous literature that showed associations between these variables and pubertal onset. We included: father absence at any time between birth and 5 years of age (dichotomous: ever absent vs. always present), maternal marital status (dichotomous: always married or living as married from birth to 5 years vs. not married or living as married at any time during that period), maternal depression (dichotomous: a score of $\geq$16 points on the Center for Epidemiologic Studies Depression Scale (CES-D) (49) at either time point: 12 or 42 months), Home Observation for the Measurement of the Environment–Short Form (50) z score (continuous composite index: mean score from 6 months and 1 year), household income (continuous, at baseline), years lived in the United States prior to delivery (continuous, at baseline), child birth weight (continuous, at delivery), and adverse life events assessed using a modified version of the Coddington Life Events Scale (CLES) (51) (dichotomous: 0 vs. $\geq$1 event between birth and 5 years). Child birth weight was obtained from medical records.

Statistical analysis

Survival analysis with interval censoring was used to analyze the data, which allows for estimation of pubertal onset given that the transition from stage 1 (no development) to stage 2 (development) occurs between study visits. We investigated the association between BMI and timing of puberty by modeling time of onset of Tanner stage 2 (for each marker separately) using parametric proportional hazards regressions, assuming a Weibull distribution. BMI was treated categorically (based on Centers for Disease Control and Prevention categories) and BMI z score and waist circumference were treated continuously.

We used the *icenReg* package in R (R Foundation for Statistical Computing, Vienna, Austria) (52) to account for left- and right-censored data and to accommodate for interval censoring for pubertal outcomes occurring between 2 known time points. Participants who had already reached Tanner stage 2 by their first clinic visit (age 9) were considered left-censored, whereas those who never reached Tanner stage 2 by the final visit (age 14) were considered right-censored. The *mice* package was used to impute missing values on covariates using multivariate imputation with chained equations. In sensitivity analyses, we used inverse probability of censoring weights to address selection bias due to loss-to-follow-up.

RESULTS

Table 1 provides descriptive statistics for BMI and covariates for the study sample. Mothers of study participants were predominantly of low income and educational level. Most women had lived in the United States for less than 10 years prior to delivery, and most were married or living as married. We observed relatively high levels of social adversity, with about 40% of children having experienced an adverse life event and over 60% of participants having a mother with depression at 1 or 3.5 years of age. Prevalence of overweight and obesity were high in this sample, with more than half of girls (55%) and boys (53%) being overweight or obese at age 5 years.

In boys, the median age for pubertal onset was 10.8 years (95% CI: 9.7, 11.7) for gonadarche and 12.2 years (95% CI: 11.4, 12.8) for pubarche. In girls, the median age for pubertal onset was 9.4 years (95% CI: 8.6, 10.0) for thelarche, 10.3 years (95% CI: 9.3, 11.0) for pubarche, and 11.7 years (95% CI: 10.9, 12.2) for menarche. Bivariate tests between covariates and BMI (Web Table 2) and covariates and puberty (Web Table 3) showed few significant associations. Web Tables 4a and 4b show the distributions of Tanner stage and obesity status at each study visit for boys and girls, respectively.

Table 2 shows adjusted interval-censored results of the association of BMI at age 5 years and timing of pubertal onset for boys and girls. For boys, there were no significant associations between either overweight or obesity and gonadarche or pubarche. Conversely, we observed associations between earlier puberty in overweight and obese girls compared with normal-weight girls. Both overweight and obesity were associated with earlier thelarche (HR = 1.7, 95% CI: 1.1, 2.7, and HR = 1.5, 95% CI: 1.0, 2.4, respectively) compared with normal weight. Additionally, we observed earlier pubarche in obese but not overweight girls compared with normal-weight girls (HR = 1.9, 95% CI: 1.2, 3.0) and earlier menarche in overweight but not obese girls compared with normal-weight girls (HR = 1.6, 95% CI: 1.0, 2.4). The Kaplan-Meier curves of these associations are shown in Figure 1.

Results were consistent when BMI z score and waist circumference were examined as continuous variables (Table 3). Adjusting for covariates, neither BMI z score nor waist circumference was associated with timing of pubertal onset for boys. Among girls, higher childhood BMI z score was associated with earlier thelarche (HR = 1.3, 95% CI: 1.0, 1.5) and pubarche (HR = 1.3, 95% CI: 1.1, 1.6) but not menarche (HR = 1.1, 95% CI: 0.9, 1.3). Higher waist circumference in girls was similarly associated with earlier thelarche (HR = 1.4, 95% CI: 1.1, 1.7) and pubarche (HR = 1.6, 95% CI: 1.3, 2.0) but not menarche (HR = 1.1, 95% CI: 0.9, 1.4).

The associations presented in Tables 2 and 3 did not change substantively in models using inverse probability weighting (not shown).

DISCUSSION

This is the first known study to examine associations between prepubertal BMI and onset of puberty among a cohort of Mexican-American children growing up in a low-income agricultural setting. A marked strength of this study is the inclusion of boys as well as girls. The majority of past research has focused on nonrural settings and has

Table 1. Characteristics[a] of Study Participants According to Sex, Center for the Health Assessment of Mothers and Children of Salinas, California, 2005–2015

Characteristic	Total (*n* = 298)		Boys (*n* = 136)		Girls (*n* = 162)	
	No.	%	No.	%	No.	%
Maternal/household characteristics						
Education, grade[b]						
<6	130	43.6	61	44.9	69	42.6
7–12	108	36.2	49	36.0	59	36.4
Completed high school	60	20.1	26	19.1	34	21.0
Country of birth[b]						
Mexico	265	88.9	122	89.7	143	88.3
Other	33	11.1	14	10.3	19	11.7
Years in US prior to delivery[b]						
≤5	132	44.3	64	47.1	68	42.0
6–10	94	31.5	41	30.1	53	32.7
≥11	72	24.2	31	22.8	41	25.3
Age at delivery, years[b]						
18–24	113	37.9	54	39.7	59	36.4
25–29	105	35.2	48	35.3	57	35.2
30–34	51	17.1	18	13.2	33	20.4
35–45	29	9.7	16	11.8	13	8.0
Marital status[c]						
Married or living as married	221	74.2	100	73.5	121	74.7
Not married or living as married	77	25.8	36	26.5	41	25.3
Maternal depression[d]						
Depressed	183	62.2	77	57.9	106	65.8
Not depressed	111	37.8	56	42.1	55	34.2
Household income[b]						
At or below poverty level	188	63.1	90	66.2	98	60.5
Above poverty level	110	36.9	46	33.8	64	39.5
Child characteristics						
Birth weight, pounds[e]	7.5 (6.9–8.2)		7.5 (6.9–8.4)		7.6 (6.9–8.1)	
Weight status at age 5						
Normal weight[f]	136	45.6	64	47.1	72	44.4
Overweight	65	21.8	23	16.9	42	25.9
Obese	97	32.6	49	36.0	48	29.6
Biological father present/absent in home[g]						
Present	248	83.2	113	83.1	135	83.3
Absent	50	16.8	23	16.9	27	16.7

Table continues

not included Latino populations, who typically have higher rates of overweight/obesity (34, 35) and earlier pubertal onset (39–41) than their White counterparts. This study is also unique in its longitudinal design spanning from birth to age 14 years, repeated assessment of multiple pubertal markers using standardized physical exam, and inclusion of key covariates including birth weight and multiple adverse childhood experiences. Results confirmed that, even when adjusting for a number of adverse events, overweight and obesity in childhood (age 5 years) were associated with earlier pubertal onset for girls. These results held across various measures of puberty, but associations were more consistent with age at breast and pubic hair development than with menarche. Associations with menarche did not show a

Table 1. Continued

Characteristic	Total (n = 298)		Boys (n = 136)		Girls (n = 162)	
	No.	%	No.	%	No.	%
Adverse life events[h]						
No	176	61.3	87	66.9	89	56.7
Yes	111	38.9	43	33.1	68	43.3
HOME score composite index[e,i]	34.0 (32.0–36.0)		33.5 (32.2–35.5)		34.0 (31.9–36.5)	

Abbreviation: Home Observation Measurement of the Environment.
[a] Number (%) for categorical variables; median (interquartile range) for continuous variables.
[b] Measured at baseline.
[c] Refers to whether mother was married or living as married through the entire period between birth and 5 years.
[d] Refers to whether or not the mother was depressed (≥16 on the CES-D) when child was either 12 months or 42 months of age. Maternal depression is missing 4 values (3 boys and 1 girl).
[e] Values are expressed as median (interquartile range). One pound is equal to 454 grams.
[f] Includes 1 underweight participant.
[g] Refers to whether biological father was ever absent at any time between 6 months and 5 years of age.
[h] Refers to presence of any negative life event before age 5, defined as one or more of the following: a close family member died, child or family member hospitalized, parent in jail, family member was a crime victim, parents separated or divorced, parent lost their job, brother or sister left home, unwed teen sister became pregnant, a close family member or friend was deported, parent was in trouble with the law, or their best friend or close relative moved away.
[i] Mean HOME score index at 6 months and 1 year. Adverse life events is missing 11 values (6 boys and 5 girls).

dose-response relationship, with earlier menarche observed in overweight but not obese girls. Among boys, prepubertal BMI was not associated with pubertal onset. Few studies have examined the association of BMI and boys' age at pubertal onset, and they have revealed inconsistent results (26), with some showing associations with later timing to puberty (25, 30, 32) and others showing associations with earlier pubertal onset (19, 27, 28, 33).

From an evolutionary biology perspective, the link between increased adiposity and timing of puberty in girls has long been suggested as necessary to support and protect reproduction (53). Adipokines that are produced by fat tissue, including leptin and adiponectin, appear to play an important role in regulating pubertal onset in girls (54). Leptin is a necessary but not sufficient factor in pubertal onset, stimulating the secretion of kisspeptin in the arcuate nucleus of the hypothalamus, which in turn triggers the activation of the hypothalamic-pituitary-gonadal (HPG) axis beginning the cascade of pubertal onset (55). Conversely, adiponectin inhibits kisspeptin, playing an opposite role.

Table 2. Adjusted[a] Associations of Body Mass Index Category From Survival Analysis Models (n = 298) at Age 5 Years (2005–2006) and Timing to Puberty Assessed at Ages 9–14 (2009–2015), Center for the Health Assessment of Mothers and Children of Salinas, California, 2005–2015

BMI Category	Boys				Girls					
	Gonadarche		Pubarche		Thelarche		Pubarche		Menarche	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
Normal weight[b]	1.0	Referent	1.0	Referent	1.0	Referent	1.0	Referent	1.0	Referent
Overweight	1.3	0.8, 2.3	1.1	0.6, 2.0	1.7	1.1, 2.7[c]	1.3	0.8, 2.0	1.6	1.0, 2.4[c]
Obese	1.0	(0.6, 1.6)	1.1	0.6, 1.8	1.5	1.0, 2.4[d]	1.9	1.2, 3.0[c]	1.3	0.8, 2.0

Abbreviations: BMI body mass index; CI, confidence interval; HOME, Home Observation Measurement of the Environment; HR, hazard ratio.
[a] Adjusted for biological father absence, maternal marital status, maternal years lived in the United States prior to delivery, maternal depression, HOME composite score, average household income per person, child birth weight (pounds), and stressful life events.
[b] Includes 1 underweight child.
[c] $P < 0.05$.
[d] $P < 0.1$.

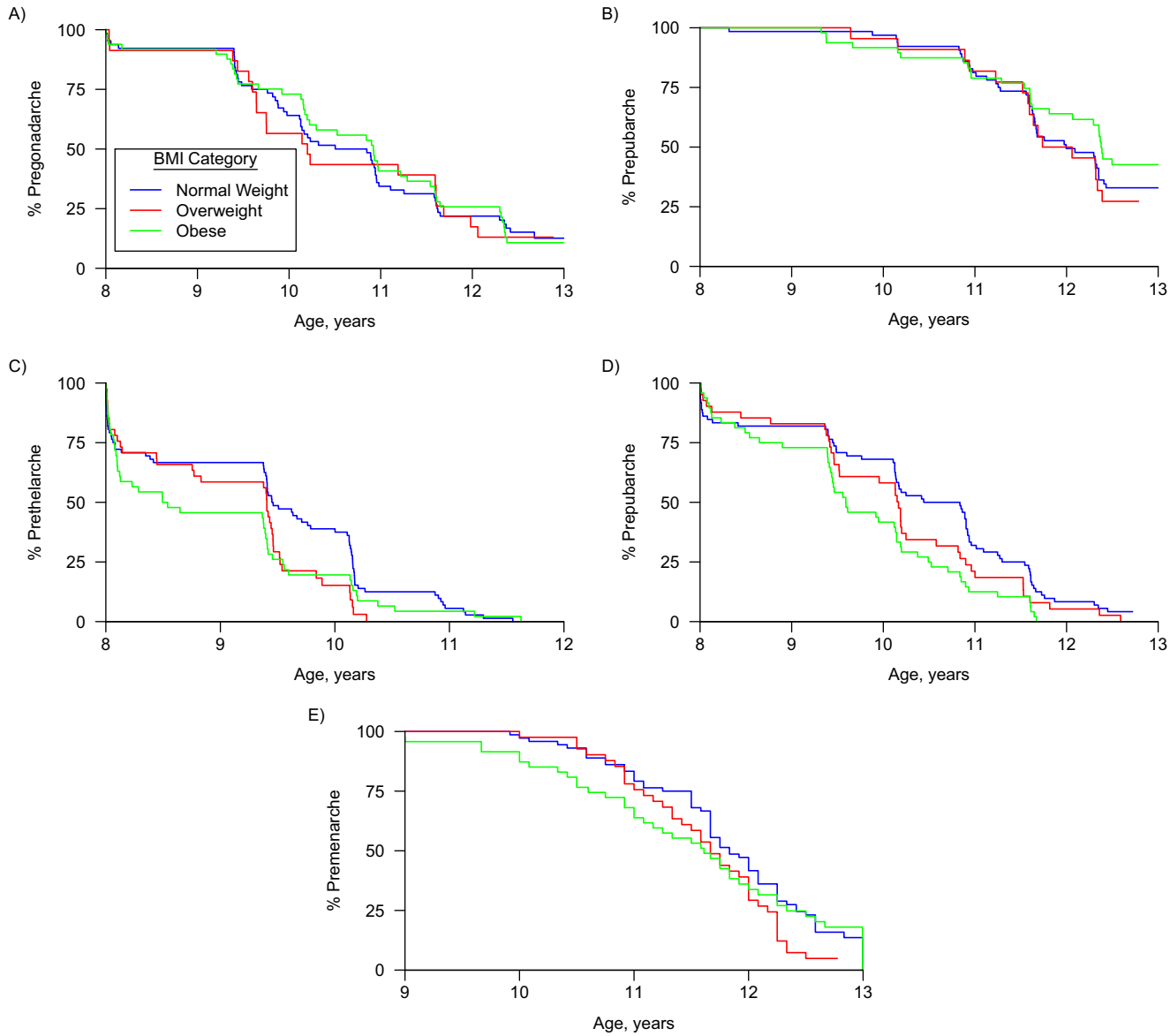

Figure 1. Kaplan-Meier survival curves for onset of puberty in boys and girls, stratified by body mass index (BMI) category, Center for the Health Assessment of Mothers and Children of Salinas, California, 2005–2015. A) Onset of gonadarche in boys; B) onset of pubarche in boys; C) onset of thelarche in girls; D) onset of pubarche in girls; E) menarche in girls.

Although there is evidence of similar processes in males, the role of leptin and adiponectin in pubertal onset in boys is less clear (56). In girls, circulating leptin levels increase during puberty, peaking just before peaks in gonadotropin concentrations. This pattern is not seen in boys, suggesting that leptin might not have the same permissive role in pubertal onset in males (56).

Many previous studies of BMI and pubertal onset have been hampered by their cross-sectional nature. Estradiol promotes energy storage as body fat, while androgens encourage the development of muscle mass (57). Thus, onset of puberty is often accompanied by increased fat deposition in girls, while boys can become leaner and taller. Cross-sectional studies that measure pubertal stage and BMI concurrently are unable to distinguish whether current BMI is a cause or consequence of pubertal development (25). Very few studies in boys have examined BMI and pubertal onset longitudinally. Lee et al. (30) reported that overweight boys (i.e., those in the highest BMI trajectory between age 2 and 11 years), were more likely to be prepubertal at age 11. Two additional longitudinal studies in Italy (28) and China (27) also found associations of higher prepubertal BMI and earlier timing to puberty among boys and girls.

The present study extends previous literature in a number of ways. We assessed puberty at numerous time points with

Table 3. Adjusted[a] Associations of Adiposity Measures From Survival Analysis Models (n = 298) at Age 5 Years (2005–2006) and Timing to Puberty Assessed at Ages 9–14 (2009–2015), Center for the Health Assessment of Mothers and Children of Salinas, California, 2005–2015

Adiposity Measure	Boys				Girls					
	Gonadarche		Pubarche		Thelarche		Pubarche		Menarche	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
BMI z score	1.0	0.8, 1.1	1.0	0.8, 1.3	1.3	1.0, 1.5[b]	1.3	1.1, 1.6[b]	1.1	0.9, 1.3
Waist circumference z score	1.0	0.9, 1.3	1.1	0.8, 1.4	1.4	1.1, 1.7[b]	1.6	1.3, 2.0[b]	1.1	0.9, 1.4

Abbreviations: BMI body mass index; CI, confidence interval; HOME, Home Observation Measurement of the Environment; HR, hazard ratio.
[a] Adjusted for biological father absence, maternal marital status, maternal years lived in the United States prior to delivery, maternal depression, HOME composite score, average household income per person, child birth weight (pounds), and stressful life events.
[b] $P < 0.05$.

gold standard measurement, including testicular volume using orchidometer for boys, which is rare in community-based epidemiologic studies. Studies of hard-to-reach populations rarely utilize Tanner staging by physical exam, given the cost and challenges in collecting these data and the difficulty in retaining participants to track puberty over time. Rigorous training and quality control measures were implemented to ensure accurate pubertal staging, including the close supervision of research assistants by pediatric endocrinologists to distinguish breast tissue from adipose tissue among girls and obtain accurate assessment of testicular volume. Research assistants were also carefully trained to distinguish onset of pubic hair from other hair (i.e., hypertrichosis), which can be misclassified in Latino samples. Unlike previous studies, we included a number of early-life adversity factors that have been implicated in earlier puberty as covariates in our analyses. Adversity was assessed prospectively from birth and we were able to characterize an important window of potential vulnerability (0–5 years) that has been hypothesized to influence pubertal timing in girls (58). Interestingly, in this sample, adversity was not associated with pubertal outcomes. Future research should focus on more socioeconomically diverse samples of Latino youth to investigate the potential influence of adverse events on puberty.

Despite the many strengths, findings should be considered in light of limitations. Although early exposure to a number of adverse life events was included, we did not have data on sexual or physical abuse, which has been linked to early puberty among girls (59, 60). For some adversity measures, such as poverty and maternal educational level, there was low variability in our sample due to our inclusion criteria. Thus, our findings in this largely low-income and overweight/obese sample of Mexican Americans might have limited generalizability to other, more diverse Latino samples. However, our median ages of onset for gonadarche, thelarche, and pubarche were very similar to the most recent National Health and Nutrition Examination Survey (NHANES) data for Mexican-American boys and girls from 1988–1994 (61). Only median age at menarche was markedly earlier in our sample (11.7 years) compared with Mexican Americans in NHANES (12.25 years) (62). Future research should examine age at menarche among Mexican Americans in the United States to determine whether this finding represents a secular trend in menarcheal timing or is unique to our sample.

Results support the critical need for preventive intervention efforts to reduce obesity among Mexican-American children in the United States, particularly among those growing up in poverty. Targeting girls' overweight is particularly important in light of their heightened risk for earlier puberty and subsequent negative health outcomes. Obesity in childhood has been linked to increased risk for obesity across the life course, which puts individuals at risk for a number of serious health consequences, including diabetes, fatty liver disease, cancer, and cardiovascular problems (63–65). It is important to note that intervening in childhood might be too late based on research supporting an intergenerational transmission of risk for obesity (66). Maternal obesity (before and during pregnancy), gestational weight gain, and gestational diabetes have been shown to have negative consequences for offspring, including augmented risk for earlier puberty and obesity in the next generation (67–72). Latina mothers are at particular risk given the high prevalence of obesity before and during pregnancy in this population. Therefore, early intervention with women of reproductive age to help them attain and maintain healthy weights, prior to and during pregnancy, is critical.

Future research should extend this work to include samples of Mexican Americans who exhibit more socioeconomic variability to explore how income and wealth might affect associations between BMI and puberty. It is plausible that early adverse life events influence higher BMI, which in turn, affects pubertal timing. However, given that our sample was uniformly low income and had high exposure to negative circumstances in early life, some of these potential associations might not have been observed in this sample. Moreover, additional work with other populations (e.g., Asian/Pacific Islanders in the United States) is warranted to determine how BMI affects associations in other understudied ethnic groups. Finally, immigration has been found to increase the risk for early puberty (73). It is important to

understand how much of this is due to changes in diet and physical activity and subsequent BMI increases, as opposed to other factors that are related to transitions from home to host countries.

In sum, our results are largely consistent with past research examining BMI and puberty among girls, and our finding of no association of early-life BMI with pubertal onset in boys is an important contribution to the limited and conflicting research on boys. We extended this research by examining associations among a vastly understudied population, Mexican-American youth growing up in a nonurban environment. We also included key covariates, including birth weight and early adverse events, which often are omitted from studies. Given our rich puberty data, we were able to confirm that inverse associations between BMI and earlier pubertal timing generally held across multiple pubertal markers for girls, including breast and pubic hair development and menarche. This research has implications for future intervention efforts targeting low-income populations, and in particular Mexican-American girls growing up in nonurban agricultural settings, where early intervention to prevent obesity and access to a healthful foods and physical activity is often lacking.

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