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Father Absence, BMI, and Pubertal Timing in Girls: Differential Effects by Family Income and Ethnicity

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Abstract

Purpose—Numerous studies show associations between father absence and girls' early puberty. However, most research has been retrospective, focused on menarche, and failed to consider BMI, ethnicity and income in the analyses. This study resolves these scientific gaps.

Methods—This was a prospective study of 444 6–8-year-old girls and their caregivers (96% mothers). Data were collected annually in clinic, including weight, height and Tanner stage for breast and pubic hair. Caregivers reported on father absence and demographics. This report focuses on the assessment of father absence at baseline and two years of follow-up for pubertal outcomes. Cox proportional hazards regression models were used to test whether father absence at baseline predicted pubertal onset by follow-up 2. BMI was assumed to be in the causal pathway. Differences by ethnicity and income were examined.

Results—Income and ethnicity moderated associations between father absence and pubertal onset when adjusting for BMI. Father absence predicted earlier onset of breast development only in higher-income families. Father absence predicted earlier onset of pubic hair development only in higher-income African Americans families. BMI was not related to father absence and therefore was not in the causal pathway.

Conclusions—Among girls from higher-income but not lower-income families, father absence is linked to earlier puberty. This was particularly true for African Americans in terms of pubic hair development. These effects are not explained by body weight. Future research is needed to identify social and biophysiological mechanisms through which father absence, ethnicity and income impact pubertal onset.

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Keywords

fathers; puberty; body mass index; ethnicity; income; effect modifiers

Early-maturing girls are at risk for negative health outcomes, including emotional and substance use problems, early sexual debut, and, later in life, breast and other reproductive cancers.^{1–3} The impact of this developmental period on short and long-term health trajectories is substantial and spans a broad range of physical and mental health issues. This is particularly alarming given evidence that girls in the U.S. now enter puberty earlier than in the past.⁴

Environmental conditions, particularly those in the family domain, influence girls' timing of puberty.⁵ Specifically, the absence of a biologically-related father has been shown to accelerate reproductive development. Evolutionary life history theory,^{6–8} which posits that humans have evolved to display adaptive plasticity in response to contextual circumstances, provides a theoretical foundation for how human biology responds to environmental conditions. Two decades ago, Belsky, Steinberg and Draper⁹ extended this theory to familial conditions and sexual maturation. These researchers posited that, during human evolutionary history, when girls encountered familial conditions that were unfavorable for survival (e.g., insecure and unsupportive family relationships), it was adaptive to become reproductively mature earlier. Since then, numerous empirical studies have confirmed that father absence predicts earlier maturation.^{10–13} Girls in father-absent homes are about twice as likely to experience menarche prior to age 12y. Mother absence does not appear to influence pubertal timing.¹⁰

Animal models illustrate biophysiological mechanisms through which parent-child processes affect maturation of the hypothalamic-pituitary-gonadal (HPG) and hypothalamic-pituitary-adrenal axes (HPA). In rodents, physical and social environments produce alterations in parent-offspring interactions, which in turn calibrate the development of stress- and steroid-responsive neural circuits and associated reproductive strategies, including pubertal timing and hormone profiles.^{14 15} Ecological stress causes low-quality parental care, which predicts a faster reproductive strategy in offspring.

A major gap in the epidemiological literature is that most studies focus on menarche, which occurs relatively late in puberty, or overall sexual development without distinguishing between onset of breast (thelarche) and pubic hair (pubarche) development. This is problematic given that these pubertal events represent observable markers of underlying hormones that are dependent on the maturation of unique endocrine axes (HPG, HPA), which may play differential roles in the etiology of health outcomes, such as breast cancer.

Another significant gap is that studies rarely examine mechanisms through which father absence may influence puberty. Although the most commonly suggested determinant of early puberty is overweight,¹⁶ only two known studies have examined weight in the pathway between father absence and puberty.^{10 17} Neither found mediating effects; however, one was retrospective¹⁰ and both focused on menarche. A related prospective study examining family adversity, but not father absence, found associations with BMI and pubertal timing, but BMI was not a mediator.¹⁸ However, elevated cumulative risk in a child's home is strongly associated with heightened cardiovascular and neuroendocrine problems, including increased body fat deposition and allostatic load.¹⁹ A possible mechanism is that father absence signals other stressors in the home that influence BMI and subsequently impact puberty. Given that father absence is not considered modifiable, the study of BMI may yield one potential intervention target.

Current Study

We utilized data from a prospective study of ethnically-diverse girls to test whether father absence predicts pubertal markers that occur prior to menarche (i.e., thelarche, pubarche). The activation of the gonadal axis is linked to fat deposition, therefore, we predicted that BMI would mediate the effect of father absence on thelarche, but not pubarche. We expected father absence to directly influence pubarche, which would lend mechanistic support to the hormonal stress response related to the development of the adrenal axis.

We also examined differences across income and ethnicity. Given that cumulative stress may augment pubertal acceleration, we hypothesized that girls growing up in lower-income families would be at greatest risk for experiencing early puberty as a result of father absence. Conversely, we anticipated that girls in higher-income father-absent families would be buffered from pubertal acceleration given greater access to resources. Potential cultural differences across ethnic groups (e.g., access to kinship networks and alternative caregivers) might also modify the effect of father absence on puberty. African American families historically have stronger extended familial networks to support child rearing compared to Caucasians.²⁰ We predicted that African American girls growing up in father-absent homes may be less prone to accelerated puberty given availability of alternative caregiver support.

Method

This project was carried out as part of the NIEHS/NCI Breast Cancer and the Environment Research Centers, four centers with transdisciplinary research collaborations across biologic, epidemiologic, and community outreach projects.²¹ The present investigation focused on one epidemiologic project, the Cohort Study of Young Girls' Nutrition, Environment, and Transitions (CYGNET). Study approval was obtained by Institutional Review Boards of participating universities and Kaiser Permanente Northern California (KPNC).

Participants and Procedure

This study recruited 444 girls and their caregivers (96% mothers) from the KPNC membership. Girls were 6–8 years old at baseline and ethnically diverse: 96 (22%) African American, 107 (24%) Latina, 52 (12%) Asian American, 187 (42%) non-Hispanic White, and 2 (>1%) Other. Girls were members of the KPNC health care system at birth and at recruitment. A letter was sent to the girl's health care provider indicating that study recruiters would like to approach the family. If the health care provider did not object within 2 weeks, then the family was sent a recruitment letter. Research staff followed up by phone and conducted an eligibility screening. If the family was eligible and agreed to participate, a visit was scheduled at a KPNC clinic. During baseline visit, informed consent and assent were obtained. At each annual visit (baseline, follow-up 1, follow-up 2), girls' anthropometric measurements and Tanner staging for breast and pubic hair development were assessed. Interviews were conducted with caregivers to collect information, including father absence and demographics. Participant attrition over the 2 years of the investigation was minimal (<2%).

Measures

Father absence was assessed at baseline using an in-person interview with the caregiver that asked who was living in the child's home and their relationship to the child. The focus of this investigation was on biologically-related father absence; therefore, any home in which there was not a biologic father in residence was considered a father-absent home. We also examined the presence of non-related adult males, including stepfathers.

Pubertal onset was assessed using clinic-based Tanner staging, an established 5-stage classification scheme,²² conducted by trained research assistants under the supervision of a board certified pediatric endocrinologist. Breast stage was assessed using visual inspection and palpation. Pubic hair was assessed using visual inspection. Because we were interested in onset, Tanner stages were recoded as 0 = no development (stage 1) and 1 = pubertal onset (stages 2–5) for breast and pubic hair, separately. We also examined a combined outcome, which was calculated based on onset of *either* breast *or* pubic hair development.

Girl's body mass index (BMI) was calculated as weight in kg/height in meters squared. Height was measured to the nearest 0.1 cm, using a mounted wall stadiometer, with the participant in stocking feet and head in the neutral position. Weight was measured without shoes and in light clothing and rounded to the nearest 0.5 kg. BMI z-scores were used in analyses.

Girl's ethnicity was reported by the caregiver: non-Hispanic White, African American or Black, Hispanic or Latino, Asian American or Other.

Income was reported by the caregiver based on annual household income and was assessed using 6 response choices ranging from <\$12K to >\$100K. For interpretation of interactions, income was dichotomized into lower (< \$50K) and higher (> \$50K). Also, a continuous measure of income was created using the midpoint of each of the 6 categories.

Statistical Analyses

Data were analyzed using SAS (SAS Institute, Cary, NC, USA). To estimate the effect of father absence on pubertal onset, while adjusting for ethnicity, income, and BMI, we utilized survival analysis, which measures the time to occurrence of an event of interest,²³ in this case pubertal onset. Survival analysis is useful when data are censored, or there is incomplete information about time to event.²³ These data were right-censored because not all participants exhibited onset by follow-up visit 2.

Breast and pubic hair onset were examined separately. Girl's age at the exam in which she transitioned from Tanner stage 1 to stage 2 or above was used to estimate age at pubertal onset. While this provides a crude estimate, given that exams occurred annually, it also represents a particularly conservative test of our hypotheses because girls may have experienced pubertal onset up to a year earlier than estimated. Multivariable Cox proportional hazards regression models were then used to analyze the effect of father absence at baseline on age at onset of breast and pubic hair. We also investigated BMI in the causal pathway. In addition, we examined the interactive (moderating) effects of ethnicity and income, respectively, on the relationship between father absence and pubertal onset and the 3-way interaction between father absence, ethnicity, and income.

Results

Preliminary Analyses

Median age at baseline was 7.4 years (Table 1). The age distribution was similar across ethnic groups. Of the 444 participants, 80 (18%) reported biological father absence at baseline. Of these 80, 21% (n=17) reported other males in the household, including stepfathers (n=12) and other non-related adult males (n=5). Analyses were run with and without these 17 participants to ensure that findings were due to father absence and not other males. Results were consistent, therefore, findings are presented for the total sample.

Father absence was highest among African Americans and lowest among non-Hispanic Whites. Of the 96 African American girls, fathers were absent for 42 (44%) compared to 11

(6%) of 187 White girls. Seven girls refused pubic hair staging in all years. In unadjusted bivariate analyses (Table 2), ethnicity, income, BMI, and father absence were associated with onset of breast and pubic hair development. Mother's age at menarche was not associated with pubertal onset and was not included in subsequent analyses. During follow up, the average rate of breast onset was 21 in 100 girls per year in father-absent homes compared to 14 in 100 per year in father-present homes. A similar pattern was observed for pubic hair onset.

BMI as a mediator in the causal pathway—Father absence was not related to BMI, therefore, BMI was not in the causal pathway. We included BMI as a covariate given its association with pubertal onset (Table 2).

Primary Analyses

Over the 2 years of investigation, the average rate of pubic hair onset among African Americans was about 27 in 100 girls per year, compared to about 8 in 100 per year for non-African Americans. Given that the rate was much higher among African Americans, and similar across non-African American groups, we regrouped ethnicity into African Americans and non-African Americans. All models were tested with the income variable dichotomized and also treated continuously. Findings were consistent across both. Therefore, results using the dichotomized income variable are presented.

Effect of father absence on breast onset

Results from the multivariable model (Table 3) predicting onset of breast development showed main effects for ethnicity and BMI and an interactive effect for father absence by income. Other interaction terms were not statistically significant, including the 3-way interaction (not shown). We stratified analyses by income level and controlled for ethnicity. Father absence predicted earlier onset of breast development in higher-income families (HR=2.4, 95% CI=1.2–4.9) but not lower-income families (Table 4).

Effect of father absence on pubic hair onset

Results from the multivariable model (Table 3) for onset of pubic hair development showed a main effect for BMI, an interactive effect for father absence and ethnicity and a marginally significant effect for father absence and income. The 3-way interaction was not significant (not shown). We stratified our analyses by ethnicity and income (Table 4). For African Americans, income moderated the association between father absence and pubic hair development, such that among African Americans with higher income, father absence predicted earlier onset (HR=4.6, 95% CI=1.6–12.7). This was not true for lower-income African Americans or non-African Americans.

Combined pubertal onset variable—In addition to examining thelarche and pubarche separately, we conducted analyses using the combined outcome. Results from these models were equivalent to those reported for pubic hair.

Discussion

This is the first known study to prospectively examine the effects of father absence on breast and pubic hair development, while taking into account BMI, ethnicity and income. Findings indicated that girls with absent fathers from higher-income, but not lower-income, families exhibited earlier pubertal onset than those from father-present homes. Despite evidence that the secular trend in pubertal timing among U.S. girls is largely influenced by body weight, our results show that BMI neither accounted for all of the variance in pubertal onset nor operated as a mechanism in the causal path between father absence and puberty.

Contrary to our expectations, higher-income girls were at greater risk for earlier maturation. For breast development, only in the higher-income group was father absence predictive of early onset. For pubic hair development, there was a combined interactive pattern of father absence, ethnicity and income. Although the 3-way interaction term did not reach significance in the total sample, in stratified analyses, father absence predicted earlier pubic hair onset for higher-income African American girls only. These findings are consistent with recent research showing that higher-income African American girls are at greater risk for early menarche compared to their lower-income counterparts.²⁴ As girls in the current study get older and experience more pubertal events, these preliminary findings will be examined further.

Importance of findings

Father absence research has largely focused on menarche or self-reports of pubertal development. This study measured outcomes that occur prior to menarche and were assessed using “gold standard” techniques, including in-clinic breast palpation. Palpation allows for identification of breast buds and avoids false positives due to overweight.

Pubarche and thelarche were treated separately and revealed slightly different findings, which might be expected based on neuroendocrine pathways that influence development.²⁵ Father absence interacted with both ethnicity and income to predict pubarche. Pubarche, but not thelarche, would presumably be accelerated in response to cortisol release (a measure of stress response) given the developmental biology of pubic hair maturation, which is advanced by sex hormones from the adrenal gland often in conjunction with cortisol.²⁶ Our findings may indicate differential exposure to stressors experienced by African American girls growing up in higher-income families. For example, exposure to discrimination among higher-income African American girls or negative self-perceptions regarding social status may influence biophysiological stress responses that initiate pubic hair development.

Given documented effects of body fat on gonadal development, we predicted that BMI would operate in the causal path between father absence and thelarche. This hypothesis was not supported. Our research fell short of identifying a potentially modifiable mechanism that might explain the relationship between father absence and puberty. Future research that continues to differentiate between pubertal outcomes, while taking contextual and psychosocial circumstances into consideration, is called for to delineate the neuroendocrine and biophysiological pathways through which father absence may accelerate puberty.

Other potential mechanisms

Other unmeasured factors in a girl’s environment may influence the effect of father absence on pubertal development. It is conceivable that among higher-income families in the U.S., there are weaker social support networks compared to lower-income communities, for whom reliance on alternative caregivers may be more normative.^{20 27} An absence of social support may be particularly deleterious when mothers are the sole income earners and are working long hours, as may be the case in higher-income, father-absent families. Maternal workload may result in familial stress and maternal emotional distance, which have been linked in previous studies to girls’ early puberty.⁵ While this may appear to contradict the literature on poverty, stress, and poor health outcomes,²⁸ past studies examining the relationship between socio-economic status and pubertal timing have yielded equivocal findings, suggesting that other familial factors, such as maternal warmth and parent-child emotional closeness, may be proximal contributors to pubertal timing.^{29 30} Alternatively, higher-income families tend to experience less father absence, therefore, early departure of the father from higher-income homes may signal a history of early childhood exposure to high marital conflict and related familial stress, which may have influenced puberty. We did

not have information regarding parent-child relationships or father involvement prior to baseline, which precluded examination of these possibilities.

Another explanation is that girls from higher-income father-absent families may be exposed to higher levels of certain environmental toxins, such as endocrine disruptors (e.g., placenta-containing hair-straighteners, beauty products), which may accelerate puberty.³¹ These girls may also have more exposure to technology (e.g., computers, video games) that increases exposure to artificial light and disturbs sleep patterns, which have been linked to puberty.³²

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Limitations

This study could not rule out genetic confounds. We examined maternal age at menarche and found no association with pubertal onset, however, this provides a weak proxy for heritability of pubertal onset. Only 3 years of data for an ongoing prospective study were included. Given that African American girls enter puberty earlier and are more likely to come from father-absent homes, results may represent an early snapshot that may be modified by future findings, particularly for pubarche. As girls age up and experience more pubertal events, it is plausible that different trends will be seen in ethnic or income subgroups. However, it is noteworthy that the effect of father absence on breast development was modified by income, but not ethnicity, suggesting that racial differences did not account for these results.

We acknowledge that, while statistically significant, our effect sizes were small, which raises the question of clinical or practical significance. Convincing evidence suggests that even a small change in pubertal timing is associated with a relatively large change in onset of fertile menstrual cycles,^{34–36} which increases breast cancer risk. Therefore, small effect sizes may result in clinically important downstream effects. We also caution the reader against interpreting any results as support for null findings. Because pubertal onset was assessed annually, it is conceivable that some girls matured up to a year earlier than estimated, yielding a particularly conservative test of our hypotheses.

Certain pubertal markers were not attained by any girl in the sample, including menarche, which precluded its examination and also the examination of tempo (rate of progression from onset to menarche). Father presence (82%) was higher than the national average for this age, thus limiting generalizability; 2009 U.S. Census data showed 74% of 6–8-year-old children live in a home with a biological father. This study also had a lower percentage of non-related adult males living in the home compared to national data (4% versus about 11% in the National Growth and Health study). However, girls in NGHS were slightly older than our sample, therefore prevalence estimates may be more similar if age-adjusted. Also, we did not have information about father absence or father involvement prior to baseline nor were we able to examine new instances of father absence after baseline; however, existing evidence indicates that early (prepubertal) father absence, particularly within the first 5–7 years of life, is the strongest predictor of puberty.¹⁷

A small number of girls had experienced pubertal onset before baseline. Idiopathic precocious puberty and other related medical conditions were exclusion criteria, and girls in this cohort were quite young, therefore onset likely occurred for the majority within the year preceding baseline. Analyses were based on this assumption and provide a conservative test of the hypotheses given that onset may have occurred earlier than estimated. As the first prospective study to examine these questions using gold standard measures in an ethnically diverse sample, study strengths considerably outweigh limitations.

Future Research

Factors present in the familial and social environment of young girls may explain the relationship between father absence and onset of puberty. By including ethnicity and income, we have uncovered a complex pattern of relationships that warrants further investigation. Negative familial experiences and girls' emotional characteristics may be more important in predicting pubertal timing than father absence.^{30 37} Girls with fathers who exhibit social deviance appear at particularly high risk for early puberty, especially when exposed to socially deviant fathers prepubertally.¹³ Furthermore, recent prospective research indicates that reproductive strategies may be programmed very early in life.³⁸ Findings show that infant attachment insecurity predicted earlier thelarche and pubarche (as measured in annual examinations), earlier pubertal completion, and earlier menarche.³⁸ Thus, family conditions and child characteristics across the life course should be considered to more fully explain the relationship between father absence and pubertal timing within varying contexts.

Our study did not investigate the role of genetics. Some researchers argue that the contribution of father absence to pubertal timing has been over-estimated and that genetics are largely responsible for these associations.³⁹ However, this does not adequately explain the variation across contextual circumstances that were documented in our findings. Research examining the intersection of genetics and environmental predictors of pubertal timing is in its infancy. Emerging studies of genes, stress physiology, and pubertal response to environmental circumstances are just beginning to illuminate the potentially complex interplay between genetics, psychobiological processes, and contextual factors that may influence human reproductive capabilities.^{14 25 40}

Clinical implications

Given confirmation of the direct influence of BMI on both breast and pubic hair onset, body weight remains a primary target to prevent early puberty. However, our results indicate that important pubertal determinants are not limited to those related to overweight. While energetic conditions, such as caloric intake and energy expenditures, set a baseline for pubertal timing, psychosocial factors move individuals around that baseline.⁵ This is a key message to clinical researchers and practitioners, who may assume that the variance in pubertal timing is largely due to hereditary factors and body composition. Future research that focuses more explicitly on family characteristics over the life course and their intersection with income and ethnicity may illuminate other targets for prevention. This is particularly important information for pediatricians, pediatric nurse practitioners, family medicine physicians, and child psychologists, who are often the first to learn of difficult family issues. To the extent that intermediary family factors (such as parenting behaviors and parent-child attachment) are modifiable, intervention in the family domain may foster greater overall well-being for children as well as better pubertal outcomes.

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Table 1

Sample descriptives and baseline characteristics.

	ALL N=444		African American 96 (22%)		Hispanic 107 (24%)		Asian 52 (12%)		White 187 (42%)		Other 2 (<1%)	
	n	%	n	%	n	%	n	%	n	%	n	%
Age at Baseline												
6 – 6.99	91	20.5	16	16.7	25	23.4	8	15.4	42	22.5	0	0
7 – 7.99	328	73.9	73	76	74	69.2	41	78.8	139	74.3	1	50
8 – 8.99	25	5.6	7	7.3	8	7.5	3	5.8	6	3.2	1	50
Family income												
< \$50,000	162	37	52	55.3	71	67	13	26	24	12.9	2	100
≥ \$50,000	276	63	42	44.7	35	33	37	74	162	87.1	0	0
Biological father at baseline												
Absent	80	18	42	43.8	21	19.6	5	9.6	11	5.9	1	50
Present	364	82	54	56.3	86	80.4	47	90.4	176	94.1	1	50
Breast stage at baseline												
1	417	94.3	85	88.5	98	91.6	52	100	180	97.3	2	100
2	25	5.7	11	11.5	9	8.4	0	0	5	2.7	0	0
Pubic hair stage at baseline												
1	390	91.3	74	78.7	99	97.1	49	98	167	92.8	1	100
2	32	7.5	15	16	3	2.9	1	2	13	7.2	0	0
3	5	1.2	5	5.3	0	0	0	0	0	0	0	0
BMI at baseline												
<18.5	338	76.1	67	69.8	70	65.4	47	90.4	154	82.4	0	0
18.5 – 24.99	90	20.3	23	24	28	26.2	5	9.6	32	17.1	2	100
25+	16	3.6	6	6.3	9	8.4	0	0	1	0.5	0	0
Mother's age at menarche												
<12	99	24.4	27	30.7	24	23.5	10	22.2	38	22.6	0	0
12	101	24.9	19	21.6	24	23.5	7	15.6	50	29.8	1	50
13	110	27.2	24	27.3	23	22.5	13	28.9	49	29.2	1	50
14+	95	23.5	18	20.5	31	30.4	15	33.3	31	18.5	0	0
Pubic hair onset by follow-up 2												
No	306	70	37	38.9	81	77.1	46	88.5	141	76.6	1	100

	ALL N=444		African American 96 (22%)		Hispanic 107 (24%)		Asian 52 (12%)		White 187 (42%)		Other 2 (<1%)	
	n	%	n	%	n	%	n	%	n	%	n	%
Yes	131	30	58	61.1	24	22.9	6	11.5	43	23.4	0	0
Breast onset by follow-up 2												
No	271	61	40	41.7	59	55.1	34	65.4	137	73.3	1	50
Yes	173	39	56	58.3	48	44.9	18	34.6	50	26.7	1	50

Table 2

Bivariate analyses for factors associated with breast and pubic hair onset.

Factor	# of girls	Total years of follow up (person years)	# with pubic hair/breast onset	Rate per 100 girls per year	Hazard ratio (95% CI)	p- value
BREAST ONSET						
Ethnicity						
Non Hispanic White	187	517.3	50	9.7	0.3 (0.2 – 0.5)	<.001
Asian American	52	144.3	18	12.5	0.5 (0.3 – 0.8)	0.004
Hispanic (Latina)	107	273.9	48	17.5	0.6 (0.4 – 1.0)	0.029
Other	2	4	1	25.3	1.1 (0.2 – 8.1)	0.915
African American	96	226.4	56	24.7	ref	
Income						
<\$50,000/annually	162	400.3	85	21.2	1.9 (1.4 – 2.6)	<.001
≥\$50,000/annually	276	749.7	85	11.3	ref	
BMI Z-score at baseline						
BMI						
25+	16	37.7	13	34.5	3.6 (2.0 – 6.5)	<.001
18.5 – 24.99	90	219.7	45	20.5	1.8 (1.3 – 2.5)	0.001
<18.5	338	908.5	115	12.7	ref	
Mother's age at menarche						
Biological Father						
Absent	80	198.5	42	21.2	1.5 (1.1 – 2.2)	0.02
Present	364	967.3	131	13.5	ref	
PUBIC HAIR ONSET						
Ethnicity						
Non Hispanic White	184	491.3	43	8.8	0.3 (0.2 – 0.5)	<.001
Asian American	52	146.1	6	4.1	0.1 (0.1 – 0.3)	<.001
Hispanic (Latina)	105	281.7	24	8.5	0.3 (0.2 – 0.5)	<.001
Other	1	3	0	0	0.0 (0.0 – 0.0)	0.981
African American	95	213	58	27.2	ref	
Income						
<\$50,000/annually	158	398.4	61	15.3	1.6 (1.1 – 2.2)	0.01
≥\$50,000/annually	273	720.9	67	9.3	ref	

Factor	# of girls	Total years of follow up (person years)	# with pubic hair/breast onset	Rate per 100 girls per year	Hazard ratio (95% CI)	p-value
BMI Z-score at baseline						
BMI						
25+	16	38	8	21	2.0 (1.0 – 4.2)	0.05
18.5 – 24.99	88	224.9	32	14.2	1.3 (0.8 – 1.9)	0.27
<18.5	333	872.1	91	10.4	ref	
Mother's age at menarche						
Biological Father						
Absent	78	190	35	18.4	1.9 (1.3 – 2.8)	0.001
Present	359	945	96	10.2	ref	

Table 3

Results from multivariable Cox proportional hazards regression models examining the effects of father absence on onset of breast and pubic hair development.

Factor	Coefficient	HR (95%CI)	p-value
Breast Onset			
Father absence	0.68	2.0 (0.9 – 4.2)	0.08
African American	0.58	1.8 (1.2 – 2.7)	0.01
Low income(<=50K)	0.67	1.9 (1.4 – 2.8)	0.00
BMI Z-score	0.36	1.4 (1.2 – 1.7)	0.00
Father absence * African American	0.23	1.3 (0.6 – 2.7)	0.55
Father absence * Low income	-1.17	0.3 (0.1 – 0.7)	0.00
Pubic Hair Onset			
Father absence	-0.02	1.0 (0.3 – 2.9)	0.97
African American	1.03	2.8 (1.8 – 4.4)	0.00
Low income (<=50K)	0.24	1.3 (0.8 – 2.0)	0.27
BMI Z-score	0.27	1.3 (1.1 – 1.6)	0.00
Father absence * African American	1.08	2.9 (1.0 – 8.5)	0.047
Father absence * Low income	-0.92	0.4 (0.2 – 1.0)	0.06

Effects of father absence on onset of breast development (stratified by income) and pubic hair development (stratified by income and ethnicity).

Table 4

	Before adjusting for BMI		After adjusting for BMI	
	n	Hazard ratio (95% CI)	p-value	Hazard ratio (95% CI)
BREAST ONSET				
Higher Income ($\geq \$50,000$) ^a	276	2.0(1.0 – 3.9)	0.05	2.4(1.2 – 4.9)
Lower income ($< \$50,000$) ^a	162	0.8(0.5 – 1.2)	0.27	0.8(0.5 – 1.2)
PUBIC HAIR ONSET				
Higher Income ($\geq \$50,000$)				
African American	42	3.3(1.2 – 8.6)	0.02	4.6(1.6 – 12.7)
Non-African American ^b	231	--	--	--
Lower income ($< \$50,000$)				
African American	51	1.3(0.6 – 2.8)	0.47	1.1(0.5 – 2.6)
Non-African American ^a	107	0.6(0.2 – 1.7)	0.39	0.7(0.2 – 1.7)

^a Adjusted for ethnicity.

^b Insufficient # of pubertal events in this category.