ORIGINAL ARTICLE

Effects of Early Life Adversity on Pubertal Timing and Tempo in Black and White Girls: The National Growth and Health Study

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ABSTRACT

Objective: Although exposure to abuse in early life predicts earlier pubertal timing, especially for girls, it is unclear if this association generalizes to nonabuse stressors. In addition, the impact of race on the stress-maturation association remains unknown. To address these issues, we examined whether race moderates the effects of early adversity on pubertal timing and tempo using a longitudinal study design.

Methods: In a cohort of 9- and 10-year-old Black and White girls, pubertal (areolar and pubic hair) maturation was assessed annually for 7 years. In adulthood, 368 participants (186 Black, 182 White) reported on abuse and nonabuse stressors they experienced from ages 0 to 12 years.

Results: Early life abuse was associated with earlier pubertal timing, as indexed by younger age at menarche ($b = -0.22$, $p = .005$, 95% confidence interval [CI] = −0.39 to −0.06) and greater pubic hair development ($b = 0.11$, $p = .003$, 95% CI = 0.04 to 0.18), in addition to slower pubertal tempo, as indexed by slower rate of pubic hair ($b = -0.03$, $p < .001$, 95% CI = −0.05 to −0.01) and areolar ($b = -0.02$, $p = .02$, 95% CI = −0.03 to −0.003) development. These associations were not found for nonabuse adversity. Black girls with early life abuse had greater pubic hair development ($b = 0.23$, $p < .001$, 95% CI = 0.12 to 0.35) and were slower in pubic hair tempo ($b = -0.07$, $p < .001$, 95% CI = −0.09 to −0.04) than their White counterparts.

Conclusions: Screening for early life abuse may help address health disparities related to earlier pubertal timing.

Key words: early life stress, pubertal development, puberty, race, STRAIN.

INTRODUCTION

Early life adversity can have lifelong consequences in the form of poor physical and mental health, including increased risk of cardiovascular disease, obesity, asthma, and depression (1–3). Early adversity has been conceptualized as exposure to adverse acute or chronic stressors that threaten a child's physical or emotional security, including experiences of abuse and neglect, discrimination, material deprivation (e.g., poverty), and neighborhood disorder (1–7). Early adversity increases the risk for 9 of the 10 leading causes of death in the United States and significantly increases the likelihood of problematic substance use and mental illness (4–6). Understanding the mechanisms linking early adversity to health is necessary to elucidate the pathophysiology of stress-related disorders and help inform the development of more effective interventions for reducing the negative effects of early adversity.

One potential mechanism by which early adversity may affect health is through earlier pubertal maturation (7). Particularly for girls, earlier pubertal maturation has serious costs including psychosocial (e.g., depression, teenage pregnancy) and physical health outcomes (e.g., cardiovascular disease, reproductive cancers) (8–12). The term "pubertal timing" refers to a child's physical pubertal development (i.e., secondary sexual characteristics) compared with other children of the same age and same sex; in girls, age at first menstruation (menarche) is the most commonly used assessment of pubertal timing. Early pubertal timing (i.e., maturing early in comparison to same-age same-sex peers) has been associated with higher rates of mortality from all causes, but particularly those related to cardiovascular disease and reproductive cancers (10–17). Early pubertal timing also has been associated with risk factors such as higher fasting insulin, higher diastolic blood pressure, decreased high-density lipoprotein cholesterol, higher adult body mass index (BMI; e.g., (18–22)), higher insulin resistance and fasting glucose (23), and accelerated biological aging (24).

As alluded to the previous discussion, there is evidence that early life adversity may contribute to earlier pubertal maturation

BMI = body mass index, **HPA** = hypothalamic-pituitary-adrenal, **HPG** = hypothalamic-pituitary-gonadal, **NGHS** = National Heart, Lung, and Blood Institute Growth and Health Study, **STRAIN** = Stress and Adversity Inventory

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(25). For example, one study found evidence that childhood maltreatment accelerated pubertal development in girls in the foster care system, and early pubertal development mediated the relation between maltreatment and internalizing symptoms (26). Rooted in the principles of life history theory (27,28), the psychosocial acceleration hypothesis proposes that adverse environments signal to girls that the risks associated with delaying reproduction outweigh the risks of earlier reproductive readiness. Consequently, to enhance reproductive fitness, pubertal maturation may be accelerated. The original formulation of the hypothesis was specific to early adversity characterized by stressful family environments (e.g., family conflict and coercive family relationships), and "resource-poor" environments were theorized to delay puberty (27,28).

Racial Differences in Pubertal Development

The negative sequalae associated with earlier pubertal timing may be at least partly responsible for the subsequent health disparities evident for Black versus White women (29). Black girls have significantly earlier menarche (30) and breast and pubic hair development (31) than White girls. Black girls also have larger prepubertal BMI, which may contribute to earlier pubertal maturation (32); however, even after accounting for BMI, Black girls still show significantly earlier pubertal timing than White girls (30,31). Other factors in addition to BMI (e.g., early adversity) may help to explain the earlier development of Black girls in reference to White girls.

In accordance with the psychosocial acceleration hypothesis, experiencing early life adversity in a sociocultural context of institutional racism may signal an especially risky environment and lead to earlier pubertal maturation in Black girls. The "weathering" hypothesis posits that exposure to race-related disadvantage prematurely ages or "weathers" the bodies of Black women and leads to early health deterioration (e.g., (33,34)). There has been related evidence that the same risk or protective factors may lead to different physiological effects for Black and White individuals. For example, education has been associated with younger biological aging for White adults but not for Black adults; in fact, completing a college degree can forecast worse physical health for Blacks (35,36). Similarly, after accounting for BMI, a lack of neighborhood recreation was associated with earlier pubertal development, but only for Black girls (37).

Timing and Tempo of Pubertal Development

To fully understand the antecedents and consequences of puberty, research needs to move beyond examining pubertal timing alone by also considering other individual factors related to pubertal development (38). "Pubertal tempo" refers to a child's rate of change of secondary sexual characteristics over a period of time (usually the duration of the pubertal transition). Indeed, faster pubertal tempo has also been associated with increases in depressive symptoms, internalizing and externalizing problems, and riskier sexual behavior, and slower pubertal tempo has been associated with higher depressive symptoms, increases in depressive symptoms, and risk for breast cancer (39–44). To date, only a few studies have examined how early adversity relates to pubertal tempo and the resulting findings have been inconsistent. In one study, physical abuse predicted faster pubertal tempo (26), and in another study, maltreatment was unrelated to pubertal tempo (45).

Girls who enter puberty earlier may spend more time in the pubertal transition, which would lead to an overall slower pubertal tempo (46). Black girls demonstrate earlier pubertal timing as compared with White girls; however, this does not necessarily indicate that Black girls have a significantly different pubertal tempo than White girls (29). More research on racial differences in pubertal tempo is needed; however, there is evidence that Black girls may have a slower pubertal tempo than White girls, and earlier pubertal timing and a slower pubertal tempo were associated with experiencing more depressive symptoms, specifically for Black girls (43).

The Present Study

To date, research on the relation between early life adversity and pubertal timing has usually examined one specific form of early life adversity—for example, abuse or maltreatment. Most studies have investigated sexual abuse in early life and found an association between sexual abuse and earlier pubertal timing (e.g., (45,47–51)). Moreover, a meta-analysis has concluded that abuse/threat in early life is related to earlier pubertal development, whereas deprivation and socioeconomic status (SES) are not (52). A few studies have examined pubertal tempo, but these investigations have specifically examined abuse or maltreatment, except for Ellis et al. (53), who found that among children with higher stress reactivity, low-quality parent-child relationships were associated with earlier pubertal timing and a faster pubertal tempo (from approximately ages 9 to 12.5 years).

Extant research has yet to establish that the association between early life abuse and early pubertal timing generalizes to nonabuse adversity in early life. In fact, some evidence suggests that deprivation or neglect in early life may result in delayed pubertal development (54). Using self-report of pubertal development, recent research with Puerto Rican youth found that cumulative early adversity (i.e., parental loss, neglect and abuse, parental maladjustment, and exposure to violence) led to earlier pubertal timing for girls; however, no association was evident for adversity and pubertal tempo (55). In the present study, therefore, we sought to examine if both abuse and nonabuse adversities in early life influence pubertal timing and pubertal tempo. Moreover, most research on pubertal development has been conducted with White samples, and research on the associations between early adversity and pubertal maturation in Black girls is limited. To our knowledge, the present study is the first to examine if both abuse and nonabuse adversities significantly influence pubertal timing and tempo in both Black and White girls.

To address these questions, we used data from a well-characterized longitudinal cohort study, the National Heart, Lung, and Blood Institute Growth and Health Study (NGHS; (56)), which followed young Black and White girls for 10 years. Girls were initially recruited to be age 9 or 10 years at the first assessment. We included NGHS data from seven annual assessments of pubertal development (i.e., age at menarche, areolar and pubic hair evaluations) in Black and White girls between ages 9 and 17 years. Our primary aim was to characterize how abuse and nonabuse stressors occurring in early life were related to pubertal timing and pubertal tempo. Some studies have operationalized early life adversity as low SES. In the present study, SES was treated as a demographic variable and included as a covariate. Pubertal timing was operationalized as pubic hair and areolar development at age 10 years, as well as age at menarche. Pubertal tempo was operationalized as the rate of pubic hair and areolar development from ages 9 to 16 years. Secondarily, we examined if documented associations between early adversity (abuse and nonabuse) and pubertal timing and/or pubertal tempo differed for Black versus White girls. Based on the prior research summarized previously, we

hypothesized that both abuse and nonabuse adversities in early life would predict early pubertal timing for both Black and White girls. Because pubertal tempo is understudied, we had no specific a priori hypotheses about the effects of early life adversity on pubertal tempo.

METHODS

Participants and Procedure

The initial aims of NGHS were to track cardiovascular risk factors and other health-related variables annually from childhood through young adulthood in 1209 Black and 1166 White girls. In 1987 to 1988, the NGHS Contra Costa County cohort (887 girls) was initially recruited at ages 9 and 10 years from public and parochial schools in the Richmond Unified School District area. The original investigators chose Richmond, California, based on census data that showed approximately equal percentages of Black and White children with the smallest income and occupational disparity between the races. More details about the initial study sample recruitment are available (56). Retention across the initial 10-year study period was 89% (57).

In 2016, we began a follow-up of the NGHS Contra Costa County cohort to assess adult health and well-being in midlife (ages 38–43 years). Eligibility criteria for this follow-up study included the following: a) being an original NGHS participant; b) not pregnant at the time of recruitment, and had not experienced a pregnancy, miscarriage, or abortion within the last 3 months; and c) not living abroad or incarcerated or otherwise institutionalized. Institutional review board approval for the study was obtained from the University of California, and all participants provided informed consent. As a result of extensive recruitment efforts, more than 73% of eligible women (307 Black and 317 White) were enrolled in the follow-up study. Of these women, 396 completed an interview-based assessment of early life adversity. The study sample consisted of 368 women (186 Black and 182 White, mean {standard deviation, or M [SD]} age = 39.1–1.1 years) who had at least one assessment of pubertal maturation from the first 7 years of the initial NGHS study and who had completed the assessment of early life adversity.

Measures

Early Life Adversity

The Stress and Adversity Inventory (STRAIN) (58) was used to retrospectively assess the number of early life stressors experienced (see https://www.strainsetup.com). The STRAIN is a National Institute of Mental Health/Research Domain Criteria–recommended system that reliably assesses a person's cumulative exposure to stress over the life course by systematically inquiring about a diverse array of acute life events and chronic difficulties. Stressors occurring in early life (e.g., childhood maltreatment, separation from a parent/caregiver, serious illness/death of family member or friend, living in an unsafe neighborhood) are queried in detail. Among other questions, respondents are asked at what age the stressor occurred. We subdivided stressors that were endorsed as happening before age 13 years into the categories abuse and nonabuse adversities. Examples of abuse-related adversity included stressors involving emotional abuse, sexual abuse, physical abuse, and prolonged harsh parental discipline. Examples of non–abuse-related adversity included stressors involving education (e.g., failing out of school), housing (e.g., not having a stable place to live), and crime (e.g., house broken into, held-up, or robbed). The "abuse" and "nonabuse adversity" variables represent the cumulative endorsed items of abuse or non–abuse-related adversity, respectively, before the age of 13 years.

Existing literature has shown that cumulative early life adversity predicts health across the life span (59). The STRAIN has demonstrated good concurrent and discriminant validity, strong predictive and incremental validity across a variety of clinical and behavioral outcomes, and outstanding test-retest reliability over time (e.g., $r_{icc} > 0.9$; (60,61)). It has also been shown to detect race-based differences in lifetime stress exposure (62).

Pubertal Assessment

Physical evaluations of pubertal maturation were made by trained registered nurses, and all evaluations were reviewed by a specialist in adolescent medicine (63). Age at menarche (first menstruation) is the most common operationalization of pubertal timing. Girls were asked to self-report age at menarche (if menstruating) at each physical evaluation. Pubic hair and areolar maturation evaluations were conducted based on Tanner staging principles (64), which are the criterion standard of pubertal assessment (65). Reference stages illustrated the criteria for Black girls and White girls. Pubic hair development was rated from stages 1 to 5, and areolar development was rated from stages 1 to 4, ranging from 1 ("prepubertal/no development") to 4/5 ("mature stage"). Areolar staging is highly correlated with Tanner breast staging (e.g., areolar stage 2 = Tanner breast stage 2) and considered less subjective (66), as adipose tissue may be mistaken for breast tissue in young girls. To assess pubertal timing, age was centered (the intercept) at the average age at time 1 (10 years old). For pubertal tempo, a linear slope represented pubertal development from ages 9 to 16 years.

Socioeconomic Status

Highest level of education for each parent (both mother and father, if known) and annual household income were self-reported at baseline (ages 9–10 years). Parental education was coded as 0 (both parents have high school diploma or less) and 1 (at least one parent has had some college education). Annual household income was coded 0 (<$20,000) and 1 (≥$20,000). Maximum level of parental education and annual household income were used as fixed covariates in all models.

Body Mass Index

Height and weight were measured at each of the seven time points. All measurements were taken twice and repeated a third time if the first two measurements differed by more than 0.5 cm (height) or 0.3 kg (weight). During the measurements, the girls wore either paper hospital gowns or large T-shirts of known weight. BMI (weight in kilograms/height in meters squared) was calculated for each time point and was subsequently used as a time-varying covariate in all analysis.

Data Analysis

To determine if there was a significant association between early life adversity and age at menarche, we conducted hierarchical linear regressions. After covariates of parents' education, household income, BMI at time 1 (ages 9–10 years), and age at time 1 (to two decimal places) were entered, the variable representing abuse-related adversity was entered into the model. In secondary analyses, a term representing abuse moderated by race (0, White; 1, Black) was entered into the model. In a separate model with the same covariates, the variable representing nonabuse adversity was entered into the model. In secondary analyses, a term representing nonabuse adversity moderated by race was entered into the model.

To assess pubertal tempo, we constructed growth curve models separately for pubic hair (Figure 1) and areolar (Figure 2) development. Age (to two decimal places) at the assessments was included as a level 1, time-varying predictor so that the exact age at assessment and the exact time duration between assessments was allowed to vary from child to child and from assessment to assessment. Multilevel modeling (lme4; (67)) was used with restricted-maximum likelihood estimation, with incomplete data treated as missing at random. To assess pubertal timing, age was centered (the intercept) at the average age at time 1 (10 years old). For pubertal tempo, a linear slope represented pubertal development from ages 9 to 16 years. For both areolar and pubic development, there was significant variance in the intercept (Tables 1, 2) but not in the slope (<.01, "singular fit"); thus, the intercept remained random in both models and the slope was fixed (67). We included maximum level of parental education and annual household income at time 1 as fixed covariates in all models. Because BMI was measured at every time point, mean-centered BMI was included as a time-varying covariate. Race, early adversity, and the interaction of

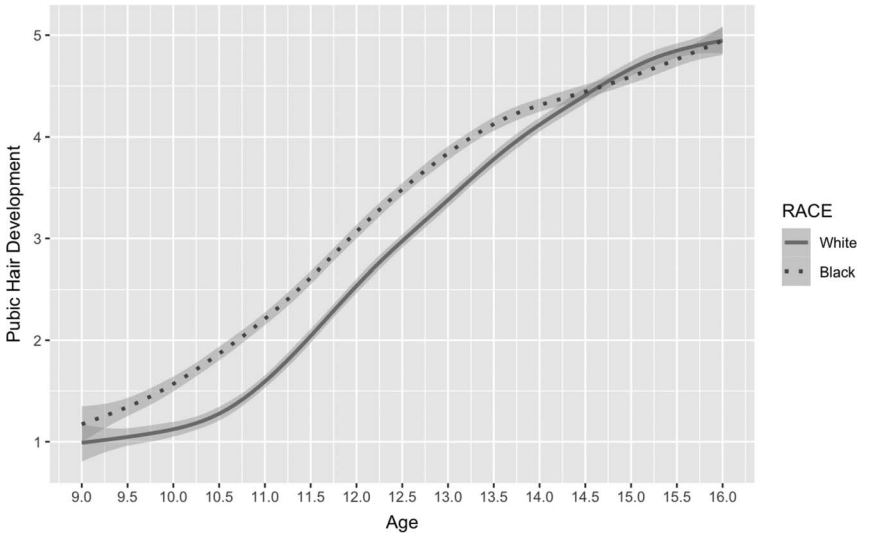

FIGURE 1. Pubic hair development from ages 9 to 16 years in Black and White girls.

race and early life adversity were included as moderators of the intercept and slope. Separate models were conducted for abuse and nonabuse adversities.

RESULTS

Descriptive statistics are reported in Table 1. On average, participants reported 0.61 (SD = 0.83; range, 0–5) stressors involving abuse before age 13 years, with 55.7% reporting no abuse. There was no significant difference in the number of abuse-related stressors reported by Black participants (M [SD] = 0.54 [0.73]) versus White participants (M [SD] = 0.69 [0.92]; $t(366) = 1.79$, $p = .08$). On average, participants experienced 0.74 (SD = 0.92; range, 0–5) early life non–abuse-related stressors, with 50.5% reporting no nonabuse adversity. There was no significant difference in the number of nonabuse stressors experienced by Black (M [SD] = 0.66 [0.86]) versus White participants (M [SD] = 0.82 [0.98]; $t(366) = 1.75$, $p = .08$).

At baseline, which occurred when girls had an average age of 10 years, the majority of participants were Tanner stage 1 (prepubertal/no development) in pubic hair development (77.7%) and areolar development (71.1%). The remainder of participants were Tanner stage 2 (pubic hair, 8.6%; areolar, 25.3%) or 3 (pubic hair, 12.8%; areolar, 3.5%). By approximately age 16 years, all girls had completed pubic hair (stage 5) and areolar development (stage 4). Black girls achieved menarche at approximately 12 years old, which was significantly earlier (approximately 6 months) than White girls. From approximately ages 10 to 13 years, Black girls were significantly more advanced in terms of pubic hair development as compared with White girls; however, this difference disappeared by age 14 years. At all assessments, Black girls were significantly more advanced in areolar development and had higher BMI as compared with White girls.

Growth of Areolar and Pubic Hair Development

Adjusting for mothers' education, household income at time 1, and BMI across the study, when compared with White girls, Black

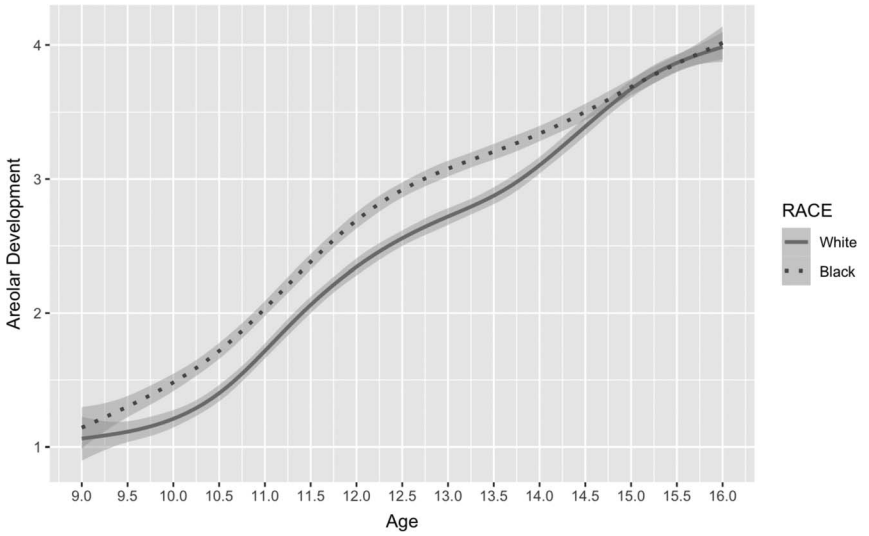

FIGURE 2. Areolar development from ages 9 to 16 years in Black and White girls.

TABLE 1. Descriptive Statistics for Black (n = 186) and White (n = 182) Girls

		Time 1	Time 2	Time 3	Time 4	Time 5	Time 6	Time 7
Age, M (SD), y	Total	9.94 (0.55)	10.88 (0.55)	11.89 (0.58)	12.88 (0.56)	13.87 (0.58)	14.89 (0.57)	15.94 (0.57)
	Black	9.95 (0.57)	10.90 (0.58)	11.93 (0.60)	12.90 (0.57)	13.90 (0.60)	14.89 (0.58)	15.94 (0.58)
	White	9.93 (0.54)	10.86 (0.53)	11.85 (0.57)	12.85 (0.55)	13.84 (0.56)	14.89 (0.55)	15.93 (0.56)
	t	0.42	0.72	1.33	0.80	0.98	0.08	0.18
Areolar Tanner stage, M (SD)	Total	1.32 (0.54)	1.81 (0.69)	2.57 (0.75)	2.79 (0.79)	3.07 (0.81)	3.71 (0.50)	4.00 (0)
	Black	1.48 (0.62)	1.94 (0.71)	2.77 (0.76)	2.93 (0.80)	3.22 (0.72)	3.79 (0.41)	4.00 (0)
	White	1.17 (0.39)	1.68 (0.64)	2.36 (0.69)	2.64 (0.75)	2.92 (0.87)	3.61 (0.57)	4.00 (0)
	t	5.57**	3.52**	5.14**	3.37**	3.40**	3.27*	—
Pubic hair Tanner stage, M (SD)	Total	1.36 (0.71)	1.80 (0.85)	2.70 (0.88)	3.55 (0.95)	4.17 (0.75)	4.55 (0.58)	5.00 (0)
	Black	1.54 (0.81)	2.12 (0.77)	2.98 (0.82)	3.80 (0.92)	4.24 (0.69)	4.58 (0.53)	5.00 (0)
	White	1.16 (0.52)	1.50 (0.77)	2.39 (0.84)	3.28 (0.90)	4.09 (0.81)	4.51 (0.64)	5.00 (0)
	t	5.33**	7.08**	6.47**	5.10**	1.89	1.08	—
Body mass index, M (SD), kg/^{m2}	Total	18.69 (3.78)	19.85 (4.21)	20.67 (4.45)	22.06 (4.75)	23.14 (5.10)	23.66 (5.32)	24.12 (5.44)
	Black	19.21 (4.07)	20.60 (4.61)	21.60 (4.86)	22.93 (5.07)	24.00 (5.57)	24.53 (5.84)	25.10 (5.85)
	White	18.17 (3.40)	19.08 (3.61)	19.70 (3.75)	21.14 (4.21)	22.19 (4.34)	22.72 (4.53)	23.17 (4.85)
	t	2.68*	3.46**	4.09**	3.58**	3.33*	3.13*	3.23*

Age at Menarche (Range. 8.48–17.20 y), M (SD), y		Parents' Education: Some College or More, %Yes (n)		Household Annual Income, $20,000+, %Yes ($n$)		Early Adversity (Abuse; Range, 0–5), M (SD)		Early Adversity (Nonabuse; Range, 0–5), M (SD)	
Total	12.25 (1.21)	Total	78.7 (295)	Total	62.7 (235)	Total	0.61 (0.83)	Total	0.74 (0.92)
Black	11.94 (1.20)	Black	78.2 (147)	Black	44.1 (83)	Black	0.54 (0.73)	Black	0.66 (0.86)
White	12.49 (1.20)	White	79.1 (148)	White	81.3 (152	White	0.69 (0.92)	White	0.82 (0.98)
χ^2	4.31**	χ^2	0.05	χ^2	61.56**	t	1.79	t	1.75

M (SD) = mean (standard deviation).

*p < .01.

**p < .001.

girls had more advanced pubic hair development than at age 10 years, and a slower pubic hair tempo than from ages 9 to 16 years. On average, Black girls were Tanner stage 1.74 and White girls were Tanner stage 1.20 in pubic hair development at age 10 years. Black girls increased 0.59 Tanner stages per year and White girls increased 0.68 Tanner stages per year in pubic hair development from ages 9 to 16 years.

Adjusting for mothers' education, household income at time 1, and BMI across the study, when compared with White girls, Black girls had more advanced areolar development at age 10 years and a slower areolar tempo than White girls from ages 9 to 16 years. On average, Black girls were Tanner stage 1.71 and White girls were Tanner stage 1.38 in areolar development at age 10 years. Black girls increased 0.40 Tanner stages per year and White girls increased 0.44 Tanner stages per year in areolar development from ages 9 to 16 years.

Effects of Early Life Adversity on Age at Menarche

Adjusting for race, parents' education, household income, BMI at time 1 (age 9/10 years) and age at time 1, greater abuse was associated with younger age at menarche (indicating earlier pubertal timing; b = −0.22, p = .005, 95% CI = −0.39 to −0.06; Table 2). Nonabuse adversity was not significantly associated with age of

TABLE 2. Effects of Early Life Abuse on Age at Menarche

Predictor of Age of Menarche	b	SE	β	t	p
Household annual income (at least $20,000)	−0.23	0.14	−0.09	−1.63	.11
Maximum parental education (at least some college)	0.19	0.15	0.06	1.21	.23
Race (0, White; 1, Black)	−0.60	0.14	−0.25	−4.44	<.001
Body mass index at time 1 (age 9/10 y)	−0.07	0.02	−0.23	−4.44	<.001
Age at time 1	0.24	0.11	0.11	2.14	.03
Early life abuse	−0.22	0.08	−0.14	−2.84	.005

SE = standard error.

menarche ($b = -0.04$, $p = .56$, 95% CI = −0.17 to 0.09). Age at menarche was not related to abuse exposure as moderated by race ($b = -0.15$, $p = .34$, 95% CI = −0.45 to 0.16) or nonabuse stressor exposure as moderated by race ($b = 0.08$, $p = .56$, 95% CI = −0.18 to 0.35).

Effects of Early Life Adversity on Areolar and Pubic Hair Development

Abuse

Adjusting for race, parents' education, household income, and BMI, early life abuse was significantly associated with more advanced pubic hair development at age 10 years (indicating earlier pubertal timing; $b = 0.11$, $p = .003$, 95% CI = 0.04 to 0.18), and also a slower tempo of pubic hair development from 9 to 16 years ($b = -0.03$, $p < .001$, 95% CI = −0.05 to −0.01; Table 3). Adjusting for race, parents' education, household income, and BMI, early life abuse was not significantly associated with areolar development at age 10 years ($b = 0.06$, $p = .07$, 95% CI = −0.004 to 0.12); however, early life abuse was significantly associated with a slower tempo of areolar development from 9 to 16 years ($b = -0.02$, $p = .02$, 95% CI = −0.03 to −0.003).

Moderation by Race

Race moderated the association between early life abuse and pubic hair development ($b = -0.06$, $p = .001$, 95% CI = −0.09 to 0.02). Namely, for Black girls, early abuse was significantly associated with more advanced pubic hair development at age 10 years (indicating earlier pubertal timing; $b = 0.23$, $p < .001$, 95% CI = 0.12 to 0.35) and a slower tempo of pubic hair development from 9 to 16 years ($b = -0.07$, $p < .001$, 95% CI = −0.09, to −0.04). In contrast, race did not moderate the association between abuse and areolar development ($b = -0.02$, $p = .12$, 95% CI = −0.05 to 0.01).

Non–Abuse-Related Adversity

Adjusting for race, parents' education, household income, and BMI, non–abuse-related stressors occurring in early life were not associated with pubic hair development at age 10 years ($b = 0.01$, $p = .67$, 95% CI = −0.05 to 0.08) or with pubic hair tempo from ages 9 to 16 years ($b = -0.01$, $p = .37$, 95% CI = −0.02 to 0.01). Similarly, adjusting for race, parents' education, household income, and BMI, non–abuse-related stressors were not related to areolar development at age 10 years ($b = -0.01$,

TABLE 3. Effects of Early Life Abuse on Linear Growth of Pubic Hair and Areolar Development

Pubic Hair Development				
Random Effects	Variance	SD		95% CI
Intercept: centered at age 10 y (pubertal timing)	0.13	0.36		0.32 to 0.40
Fixed Effects	**Estimate**	**SE**	***t***	**95% CI**
Intercept: centered at age 10 y (pubertal timing)	1.24	0.07	17.02***	1.10 to 1.38
Slope (pubertal tempo)	0.66	<0.01	66.72***	0.64 to 0.68
Maximum parental education: some college or more	0.04	0.06	0.66	−0.07 to 0.15
Household annual income: $20,000+	−0.01	0.05	−0.13	−0.11 to 0.10
BMI: centered, time-varying	0.02	<0.01	4.45***	0.01 to 0.03
Race: 0, White; 1, Black	0.32	0.05	6.35***	0.22 to 0.42
Abuse count	0.11	0.04	2.98**	0.04 to 0.18
Abuse by slope interaction	−0.03	<0.01	−3.32***	−0.05 to −0.01
Areolar Development				
Random Effects	Variance	SD		95% CI
Intercept: centered at age 10 y (pubertal timing)	0.09	0.30		0.26 to 0.33
Fixed Effects	**Estimate**	**SE**	***t***	**95% CI**
Intercept: centered at age 10 y (pubertal timing)	1.39	0.06	22.53***	1.27 to 1.51
Slope (pubertal tempo)	0.44	<0.01	51.16***	0.42 to 0.45
Maximum parental education: some college or more	−0.03	0.05	−0.66	−0.13 to 0.06
Household annual income: $20,000+	0.05	0.04	1.20	−0.03 to 0.14
BMI: centered, time-varying	0.03	<0.01	6.94***	0.02 to 0.04
Race: 0, White; 1, Black	0.24	0.04	5.52***	0.15 to 0.32
Abuse count	0.06	0.03	1.84†	−0.004 to 0.12
Abuse by slope interaction	−0.02	<0.01	−2.41*	−0.03 to −0.003

SD = standard deviation; CI = confidence interval; BMI = body mass index.

*$p < .05$.

**$p < .01$.

***$p < .001$.

†$p < .10$.

$p = .77$, 95% CI = −0.06 to 0.05) or with areolar tempo from 9 to 16 years ($b = -0.001$, $p = .84$, 95% CI = −0.01 to 0.01).

Moderation by Race

Race did not moderate the association between non–abuse-related adversity and pubic hair development ($b = 0.01$, $p = .74$, 95% CI = −0.03 to 0.04) or between non–abuse-related adversity and areolar development ($b = -0.02$, $p = .24$, 95% CI = −0.04 to 0.01).

DISCUSSION

To date, few studies have investigated pubertal tempo as an outcome of interest, and to our knowledge, the present study is the first to examine racial differences in the influence of early life adversity on pubertal tempo. In a longitudinal cohort of Black and White girls assessed annually from childhood through adolescence, we found that early life abuse was associated with earlier pubertal timing (i.e., younger age at menarche, more pubic hair development at age 10 years) and slower pubertal tempo (i.e., slower rate of pubic hair and areolar maturation from ages 9–16 years). In contrast, non–abuse-related stressors were not associated with pubertal timing or tempo. Early life abuse seemed to amplify the already-significant race differences in pubertal timing and tempo, in that Black girls with early life abuse were earlier in their pubertal timing and slower in their pubertal tempo than White girls with early life abuse. These results held while adjusting for BMI at all time points, parental education, and household income at baseline.

Consistent with existing meta-analytic data (52), in the present study, only early life abuse was associated with earlier pubertal timing; in addition, nonabuse adversity before age 13 years was not associated with pubertal timing. Conflictual family relationships in childhood have been reliably associated with earlier pubertal timing in exclusively non-Hispanic White samples (e.g., (68)). In the one study that examined race as a moderator of the association between adversity and pubertal timing, poverty in early life was associated with earlier age at menarche for White girls, but there was no association between poverty and menarche for Black girls (69). Only one other study has examined race as a moderator of pubertal tempo, and consistent with Keenan et al. (43), Black girls exhibited earlier pubertal timing and slower pubertal tempo than White girls in the present study. To our knowledge, given that prior research has not included race as a moderator of the early adversity–pubertal maturation relationship, it is difficult to directly compare the present findings with those of previous studies. Studies of predominantly White children that include non-White girls may not have the number of Black participants needed to be sufficiently powered to show significant differences.

Because the amount of early life abuse experienced did not significantly differ between Black and White girls, experiencing more abuse did not seem to explain the earlier pubertal timing and slower tempo of Black versus white girls. Experiencing early life adversity as a Black child in the context of institutional racism (i.e., the United States) may confer an additional level of risk that contributes to the influence on pubertal development. Race-related risk factors (not measured in this study) may interact with early life abuse and result in an adverse environment that fosters earlier reproductive readiness. If Black girls start showing more accelerated biological aging in childhood than White girls (e.g., in the form of earlier pubertal maturation), this may suggest that the weathering process (33,34) begins in childhood. Moreover, the stronger association between early abuse and pubertal maturation in Black girls may result in a higher likelihood of experiencing the negative consequences associated with earlier pubertal timing and slower tempo. For example, earlier pubertal timing and a slower pubertal tempo have been found to be associated with experiencing more depressive symptoms, specifically for Black girls (43).

In the present study, early life abuse seemed to have a stronger association with pubic hair development than areolar development. Early abuse was related to early pubertal timing as indexed by age of menarche and pubic hair development at age 10 years, and with slower pubic hair and areolar tempo; however, the relation between early abuse and areolar development at age 10 years was not significant ($p = .07$). Black girls demonstrated a stronger association between abuse and pubic hair development than White girls, and this moderated effect by race was not evident for the association between early life abuse and areolar development. These discrepancies may be due to lack of power; therefore, research should continue to examine the relation between early life abuse and both pubic hair and areolar development. Replicating the present findings is necessary before drawing strong conclusions from these results.

Pubertal maturation consists of two processes—adrenarche and gonadarche—and each involves a different endocrine axis: the hypothalamic-pituitary-adrenal (HPA) axis and hypothalamic-pituitary-gonadal (HPG) axis, respectively (70). The development of pubic hair is understood to represent adrenarche and breast development (and menarche) to represent gonadarche (71). The duration between pubertal onset and menarche varies across individuals (72), and early breast or pubic hair development is not necessarily followed by early menarche (53,73). Most studies that have examined associations between early life adversity and pubertal timing have used self-reported age of menarche (a relatively late event in the pubertal transition) to represent pubertal timing (52). Girls who have an earlier age of menarche tend to have a much shorter time to full reproductive competence (when cycles are consistently ovulatory and, consequently, a greater likelihood of pregnancy), onset of sexual activity, and earlier age at first birth, although these traits are also genetically correlated (28). Associations found with younger age at menarche would not necessarily hold for earlier pubic hair or breast development, which have been understudied in comparison to menarche.

The specific mechanisms by which early adversity confers risk for earlier pubertal timing are unknown. The signal from abusive environments that results in the acceleration of puberty does not necessarily mean that girls have a conscious assessment that delaying reproduction is risky (74). One possibility is that exposure to highly stressful environments in early life upregulates stress reactivity systems (75), which leads to a recalibration of the HPA axis that accelerates adrenarche (28,76) and results in a more "adult-like" inverse coupling of the HPA and HPG axes (77). This inverse coupling of the HPA and HPG axes may trigger the release of hormones via the HPG axis and so result in earlier maturation (78,79). Early pubertal timing may be accompanied by a compensatory process that extends maturation (80). Girls who enter puberty earlier seem to spend more time in the pubertal transition, which would lead to an overall slower pubertal tempo (46,81).

The study has several limitations that should be considered. First, early life adversity was retrospectively assessed and could have been influenced by cognitive and/or self-report biases that affect accuracy (82,83). Although retrospective report of maltreatment is commonly used, feasible, and useful with an adult sample

(84,85), future research would benefit from including both prospective and retrospective reports of early life adversity, which can differ. Second, some participants had already begun pubertal development at the time of the first assessment. Specifically, at the first examination, 29% of the sample had begun areolar development and 22% had begun pubic hair development (Tanner stage 2 or higher). Ideally, we would have had an earlier assessment where all of the participants were stage 1 in both areolar and pubic hair development. Consequently, we may have missed the onset of maturation for those girls who experienced the earliest onset of pubertal development. Third, the interview we used for assessing early life stress is well validated and assesses stressors associated with race and discrimination, but it is not designed to directly assess these stressors, which is a limitation that could be remedied in future work. Finally, although the present study included both self-report and physical examination of pubertal development, future research should consider also incorporating other metrics of pubertal development, such as hormonal measures.

Despite these limitations, the study has significant strengths. First, the study design was ideal for investigating the influence of early life adversity on pubertal timing at age 10 years, when there was significant variability in both areolar and pubic hair development, as well as pubertal tempo over 7 years. Second, pubertal assessments were conducted annually using the criterion standard of Tanner staging via physical examination by a medical professional. As a result, in contrast to most work, the study was able to examine pubertal timing and tempo using both areolar and pubic hair development. Many studies of pubertal development have been conducted with self-report measures and cannot provide separate assessments of areolar and pubic development. Finally, pubertal development was assessed prospectively and repeatedly (i.e., seven times) from preadolescence to late adolescence, in a moderately large sample of equal numbers of Black and White girls. In contrast to many prior studies of early life adversity and pubertal maturation, our cell sizes were large enough to permit the examination of racial differences with limited SES confounding.

In sum, in the context of a comprehensive longitudinal study, Black girls experienced pubertal development earlier than White girls and over a longer time period, thus indicating slower pubertal tempo. Moreover, abuse (but not nonabuse) stressors occurring in early life were associated with earlier pubertal timing at age 10 years and slower pubertal tempo from ages 9 to 16 years, and this association was stronger for Black versus White girls. Early life abuse therefore seems to result in earlier pubertal maturation, which has serious psychosocial and health-related consequences. Screening for early life abuse could help to identify youth at risk of earlier puberty who would benefit from intervention during this sensitive developmental period to head-off increased risk for health problems later in life. Any modifiable factors that influence puberty, such as exposure to abuse in early life, may be important targets to aid in the amelioration of race-related health disparities.

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