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Original article

Childhood Sexual Abuse and Early Timing of Puberty

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A B S T R A C T

Purpose: The purpose was to examine whether the timing of puberty, indexed by breast development and pubic hair development, was earlier for sexually abused females compared with a matched comparison group of nonabused females, controlling for key alternative confounds.
Methods: A cohort of sexually abused females and matched comparisons was followed longitudinally at mean ages 11 through 20 years. Sexually abused participants (N = 84) were referred by protective services. Comparison participants (N = 89) were recruited to be comparable in terms of age, ethnicity, income level, family constellation, zip codes, and nonsexual trauma histories. Stage of puberty was indexed at each assessment by nurse and participant ratings of breast and pubic hair development using Tanner staging—the gold standard for assessing pubertal onset and development. Cumulative logit mixed models were used to estimate the association between sexual abuse status and the likelihood of transitioning from earlier to later Tanner stage categories controlling for covariates and potential confounds.
Results: Sexual abuse was associated with earlier pubertal onset: 8 months earlier for breasts (odds ratio: 3.06, 95% CI: 1.11–8.49) and 12 months earlier for pubic hair (odds ratio: 3.49, 95% CI: 1.34–9.12). Alternative explanations including ethnicity, obesity, and biological father absence did not eradicate these findings.
Conclusions: This study confirms an association between exposure to childhood sexual abuse and earlier pubertal onset. Results highlight the possibility that, due to this early onset, sexual abuse survivors may be at increased risk for psychosocial difficulties, menstrual and fertility problems, and even reproductive cancers due to prolonged exposure to sex hormones.

IMPLICATIONS AND CONTRIBUTION

This longitudinal study using gold-standard objective methods for pubertal staging confirmed earlier pubertal onset of both breast and pubic hair development in sexually abused females. Competing alternative models of obesity and biological father absence did not eradicate findings. Early puberty may affect risks of maladjustment and reproductive cancers.

Successfully advancing through developmental milestones involves a synchrony between subjective age and psychosocial maturation such that key transitions occur at normative ages when individuals possess the psychosocial tools necessary to realize mastery at various stages. Maladaptation occurs when transitions are “off-time” or when there is age/maturation developmental discordance. Early pubertal timing exemplifies

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such discordance in that maturation level outpaces age, signaling potential for maladaptive functioning. For over 50 years, early pubertal timing has been recognized as a risk factor for mental health and behavioral problems including early sexual initiation [1], delinquency, and depression [2]. These adverse outcomes have been attributed to discrepancies between physical and cognitive maturity, leaving adolescents who enter puberty earlier than their peers with inadequate psychosocial skills to cope with mature contexts. These adolescents tend to associate with older, more risk-taking peers [1] and face age-inappropriate expectations that they are not prepared to navigate [2].

Early puberty has also been implicated in the development of menstrual and fertility problems (e.g., uterine leiomyomata, endometriosis) and reproductive cancers (e.g., ovarian, breast cancers) due to prolonged exposure to estrogens [3,4]. Stress exposure in early life can affect maturation through processes of biologic embedding [5] and heightened activity of the hypothalamic–pituitary–adrenal (HPA) axis [6]. Circulating gonadal steroids, especially estrogen, are important modulators of the HPA axis, suggesting a potential route for estrogen-mediated increase in glucocorticoid levels that can influence the rate of growth and timing of sexual maturation [7]. Although a growing body of literature has indicated marked changes in HPA activity during and after sexual maturation, especially in girls [8], empirical evidence of stress and adversity leading to earlier pubertal maturation in humans is scarce [9]. Childhood sexual abuse constitutes a powerful, naturalistic laboratory to examine the impact of early life stress on pubertal development.

Herman-Giddens et al. [10] were the first to report that patients with histories of childhood sexual abuse showed pubic/axillary hair or breast development before age of 8 years, leading to theories positing that childhood sexual abuse had an inordinate effect on the stress system, thus setting in motion trajectories of early maturation [11]. Evolutionary theorists suggested that there are environmental conditions in which accelerated maturational development is an adaptive response serving to maximizing reproduction opportunities [12]. Among several forms of stress, the absence of a biological father (as is the case with a large percentage of victims of childhood sexual abuse) signals environmental instability in which early reproductive maturity is adaptive, or once was [13]. Moreover, a recent meta-analysis of over 41 studies [14] demonstrated that childhood maltreatment (including sexual abuse) is consistently associated with the development of obesity—a condition that also confers risk of early pubertal onset due to adiposity-related endocrine mechanisms [15] that stimulate enzymes necessary for the production of adrenal androgens [16].

Although an association between childhood sexual abuse and early puberty has been loosely established, the majority of studies relied on retrospective reports of both abuse and age at menarche (e.g., Boynton-Jarrett et al. [17]). Age at menarche is a notoriously inexact method for estimating pubertal onset [18] and does little to characterize the amount of time that individuals are actually exposed to pubertal hormones. Length of hormone exposure is an important indicator characterizing incidence of breast cancer [19]. The gold standard for assessing pubertal development is Tanner staging [20], a standardized index of physical ratings on a continuum of 1 (prepubertal or no signs of pubertal growth), 2 (pubertal onset via the presence of breast buds and pubic hair), 3–4 (subsequent stages of maturation), and 5 (postpubertal or full maturity). Of the two extant studies using Tanner staging with samples of sexually abused females, one showed no association [21] and the other showed more advanced breast development for abused females in foster care [22]. Neither of these studies focused on onset transitions or tested plausible alternative explanations.

The present examination is unique in several ways. First, it is a longitudinal study of females with substantiated sexual abuse and a matched comparison group. Second, pubertal stage was assessed via secondary sexual characteristics using Tanner staging [20] of both breast and pubic hair stages as obtained from research nurses and participants after nurse training. Third, alternative explanations for earlier pubertal onset such as race/ethnicity [18], obesity [23], and biological father absence [13] were considered. We hypothesized that after controlling for these key confounds, the onset of puberty would be earlier for sexually abused females relative to matched comparisons.

Methods

Participants

Sexually abused female participants (N = 84) were referred by child protective services (CPS) agencies in the Washington, DC, metropolitan area. Eligibility criteria included (1) age 6–16 years at the time of study entry, (2) participation within 6 months of disclosure of the abuse, (3) substantiated sexual abuse, including genital contact and/or penetration, (4) perpetration by a family member (e.g., parent, grandparent, older sibling, uncle), and (5) participation of a nonabusing caregiver (usually the biological mother). All participants remained in caregiver custody. Comparison participants (N = 102) were recruited via advertisements in newspapers and in welfare, child care, and community facilities. Comparison families contacted study personnel and were screened for eligibility, which included having no previous contact with CPS agencies and being demographically similar to a same-aged abused female. Comparison and abused female participants were similar in terms of residing zip codes, racial/ethnic group, age (6–16 years), predisclosure socioeconomic status (SES), family constellation (one- or two-parent families), and other nonsexual traumatic events including both interpersonal trauma (exposure to family and community violence) and noninterpersonal trauma (accidental injury, natural disasters, witnessing violence/accidents) assessed via a standardized trauma interview (Comprehensive Trauma Interview [24]). At some point after entry into the study, 13 comparison participants revealed some form of sexual abuse via the Comprehensive Trauma Interview and were excluded, resulting in a comparison sample of 89. The study began in 1987 (time 1); Tanner staging was conducted at baseline (mean age = 11 years) and four subsequent follow-up (time 2–5) assessments (mean age = 12, 13, 18, and 20, respectively). Ninety-six percent of the original sample was retained and available for longitudinal analyses (n = 166; Table 1). The study received approval from the affiliated institutional review board and was awarded a federal certificate of confidentiality. At each assessment, caregivers provided consent, and participants provided assent. The sample was 54% white, 43% black, 2% Hispanic, and 1% Asian American. Families ranged from low to middle SES, with mean Hollingshead [25] scores of approximately 36. There were no statistical differences across groups for mean SES or minority status.

Table 1
Demographic and study variables

	Total sample	Sexually abused	Comparison
Number	173	84	89
Age, mean ± SD (range)			
Time 1	11 ± 3 (6–16)	11 ± 3 (6–16)	11 ± 3 (6–16)
Time 2	12 ± 3 (7–18)	12 ± 3 (7–17)	12 ± 3 (7–18)
Time 3	13 ± 3 (8–20)	13 ± 3 (8–18)	13 ± 3 (8–20)
Time 4	18 ± 4 (11–25)	19 ± 4 (11–25)	18 ± 3 (11–23)
Time 5	20 ± 3 (13–26)	21 ± 3 (13–26)	20 ± 3 (13–26)
Number of longitudinal analyses (96%)	166	82	84
Minority status, %[a]	46	39	51
SES, mean ± SD (range)[b]	36 ± 12 (11–44)	35 ± 14 (10–47)	37 ± 11 (12–43)
Ever obese, %[c]	20	21	20
Ever overweight, %[d]	27	29	25
Normal weight at all time points, %[e]	53	50	55
Biological father absence, %[f]	53	65	40

SD = standard deviation; SES = socioeconomic status.
[a] Minority status includes black (90%), Hispanic (9%), or Asian American (1%).
[b] SES was defined using Hollingshead [25] ratings.
[c] Ever obese status defined per Centers for Disease Control and Prevention guidelines of body mass index *z* score ≥95th percentile.
[d] Ever overweight status defined per Centers for Disease Control and Prevention guidelines of body mass index *z* score 85th to <95th percentile.
[e] Normal weight at all time points status defined per Centers for Disease Control and Prevention guidelines of body mass index *z* score 5th to <85th percentile.
[f] Biological father absence defined as 1 = absent at all time points; 0 = present at least one time point.

Predictor

Childhood sexual abuse was substantiated via CPS records. The median age at abuse onset was 7.8 years, median duration was 24 months, 70% experienced vaginal and/or anal penetration, and 60% of perpetrators were the primary father figure (biological fathers, stepfathers, or mothers' live-in boyfriends). These abuse characteristics were similar to comparable information reported in national studies of child abuse and neglect from the same time period [26].

Outcome

Pubertal stage was measured using Tanner staging [20]. Assessments took place in a clinic setting, during which research nurses conducted a physical examination and provided detailed training to participants about how to assess their development using a set of standardized pictures depicting the five stages of pubertal development for breast growth and presence of pubic hair (prepubertal stage 1; pubertal stages 2–4; postpubertal stage 5). At time 1–3, both nurse and participant ratings were obtained. At time 4 and 5, only participant ratings were obtained because research nurses were no longer active in the study protocol given that participants had received ample training and correlations between nurse and participant ratings were very high (at various time points, correlations ranged from .83 to .91 for breast ratings and .89 to .93 for pubic hair ratings). For consistency across all five assessments, participant reports were used in analyses. Both nurses and participants were blinded to the study hypothesis throughout the study.

Alternative explanatory variables

Ever obese. Height and weight were obtained at each assessment by trained study personnel using a calibrated upright Health-O-Meter Balance Beam Scale with participants in street clothing without shoes. Body mass index (kg/m^2) was used to categorize if females where ever obese during the course of the study (1 = obese at one or more time points, 0 = never obese) per Centers for Disease Control and Prevention guidelines of body mass index *z* score ≥ 95th percentile.

Minority status. Caregivers reported on participant's race/ethnicity, which was coded as 1 = minority and 0 = Caucasian.

Biological father absence. At each assessment point, caregivers reported whether the biological father was absent or present in the home (1 = absent at all time points; 0 = present at least one time point).

Statistical analyses

A cumulative logit mixed model (CLMM) [27] was used to estimate the probability that a given female in the sexually abused group would transition to a more mature (i.e., later) Tanner stage at a significantly younger age than a given female in the comparison group, controlling for covariates. Suppose that y_{ij} is the Tanner stage score of the i^{th} female at the j^{th} age. The CLMM model is written as $P(y_{ij} > c) = [1 + exp\{\theta_c - X_i\beta - u_i\}]^{-1}$, where $P(y_{ij} > c)$ is the probability that a female's classification falls in a category higher than the c^{th} category; θ_c is a cut point–specific intercept; X_i is the design matrix containing age, a dummy coding for group (sexual abuse = 1, comparison = 0), and the control variables with associated fixed-effects regression vector β; and u_i is the random-effects estimate. CLMM accounts for dependency due to repeated measures with the random effect and accommodates missing data with maximum likelihood estimation under the missing at random (MAR) mechanism [27], that is, inferences regarding category transitions are unbiased to the extent that missingness is unrelated to observed or unobserved variables, or to the extent that observed variables are predictive of missingness. Moreover, Tanner stage at study entry did not correlate with abuse status, minority status, obesity, or biological father absence. Because these data featured some left censoring (i.e., participants entered the study at varying stages; 39% prepubertal, 39% pubertal, and 22% postpubertal) and we did not assess every participant at contiguous ages, we tested this assumption using the graphical methods of Hu and Sale [28] to evaluate if individuals with missing data at a subsequent age had a different current mean Tanner stage value than those who had the subsequent data. Results showed negligible variation in parameter estimates when employing this evaluation method, thus supporting the MAR assumption. Hence, the stringent MAR tests and the parameter estimation procedures of CLMM should alleviate concerns about left censoring. All models were estimated using the mixcat package and the ordinal package in R software for statistical computing [29]. For the current analyses, the sample size was 154 because 12 participants had missing data on one or more of the covariates.

Valid application of CLMM rests on the proportional odds assumption that the difference between groups is constant for each cut point indexed by *c* [30]. A preliminary analysis was performed to determine the sensitivity/specificity of each Tanner

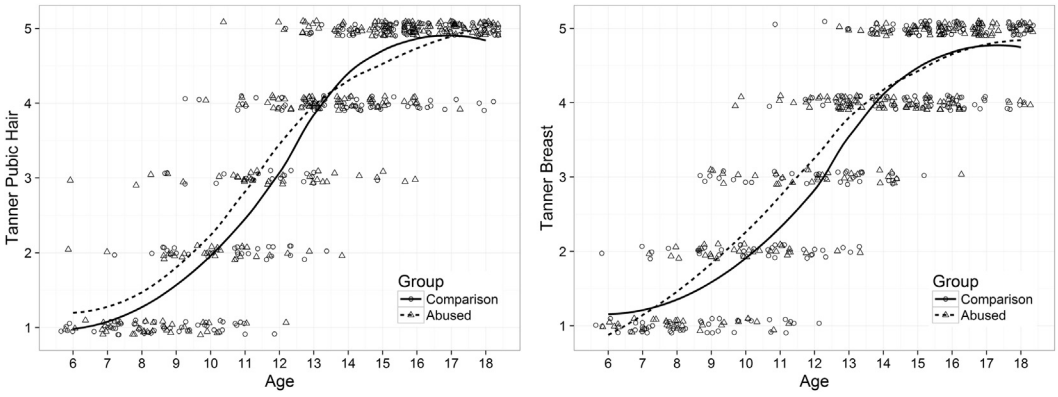

Figure 1. Observed Tanner stage scores (circles) and locally weighted regression curves (lowess) by age and sexually abused versus comparison groups.

stage for discriminating among groups. This preliminary test revealed that the optimal transition cut point was at Tanner stage 3. This can be seen in Figure 1, which shows observed data (filled circles) and locally weighted regression (lowess) curves. The curves intersect at Tanner stage 4, indicating a lack of discrimination among groups after stage 3. Therefore, to satisfy the proportional odds assumption and to be consistent with the sensitivity test, stages 3, 4, and 5 were combined into one category, yielding three ordered categories for transition analysis: Tanner 1, Tanner 2, and Tanner 3 or higher.

Results

Table 2 shows the CLMM results for Tanner pubic hair (upper half) and Tanner breast stage (lower half). There was a significant age effect for both pubic hair and breast stage, indicating that the probability of transitioning to a higher category of pubertal stage increased over time as a function of age. The absence of a biological father in the home was not a significant predictor of either Tanner pubic hair or breast stage transitions. Obesity was a significant predictor of breast stage but not pubic hair stage transitions. The odds ratio (OR) for breast stage was 6.80 (95% confidence interval [CI]: 2.06–22.55), indicating that a given obese female was almost seven times more likely transition to a higher breast category than a given nonobese female at any age. There was also a statistically significant effect of race/ethnicity in the pubic hair model and in the breast stage model, indicating that a given minority female was almost six times more likely to transition to a higher pubic hair category than a given Caucasian female at any age (OR: 5.69, 95% CI: 2.10–15.48) and almost three times more likely to transition to a higher breast stage category (OR: 2.95, 95% CI: 1.04–8.32).

There was a statistically significant group difference in pubic hair stage and breast stage, indicating that sexually abused females were almost 3.5 times more likely than comparison females to transition to a higher pubic hair category (OR: 3.49, 95% CI: 1.34–9.12) and about three times more likely to transition to a higher breast category (OR: 3.06, 95% CI: 1.11–8.49). As Figure 2 depicts, sexually abused females had a 50/50 chance of transitioning to pubic hair stage 2 by age 9 years (vs. age 10 years for comparison females) and pubic hair stage 3 by age 10 years 8 months (vs. 11 years 8 months for comparison females). For breast development, sexually abused females (on average) transitioned to stage 2 by age 9 years (vs. 9 years 8 months for comparison females) and stage 3 by age 11 years (vs. 11 years 8 months for comparison females).

Discussion

Using gold-standard assessments in a longitudinal design, controlling for several potential alternative explanations, this study revealed novel findings indicating that sexually abused females entered puberty approximately 1 year earlier for pubic hair development and 8 months earlier for breast development than their nonabused peers. This degree of difference is far from trivial. A recent review of pubertal maturation in the United States revealed that a 7-month decrease is pubertal onset during the previous 50 years (from age 10.6 years in 1940 to 9.9 years in 1993) that could signal a subsequent trend of increased reproductive cancers [31]. A definitive meta-analysis of 117 epidemiological studies of breast cancer spanning 35 countries and over

Table 2
Cumulative logit mixed model results for Tanner pubic hair and Tanner breast

Scale	Effect	Est.	LRT	OR 95% CI
Pubic hair	θ_1	14.18	71.72	
	θ_2	16.58	77.96	
	Age	1.43	415.32	
	Sexual abuse group	1.25	6.75	3.49 (1.34–9.12)
	Ever obese[a]	−.23	.22	.79 (.30–2.08)
	Minority status[b]	1.74	12.99	5.69 (2.10–15.48)
	Biological father absence[c]	.35	.59	.70 (.58–3.50)
	σ^2_u	1.87		
Breast	θ_1	15.44	60.79	
	θ_2	18.55	66.62	
	Age	1.59	424.46	
	Sexual abuse group	1.12	4.62	3.06 (1.11–8.49)
	Ever obese[a]	1.92	11.18	6.80 (2.06–22.55)
	Minority status[b]	1.08	4.35	2.95 (1.04–8.32)
	Biological father absence[c]	.49	.98	.61 (.62–4.26)
	σ^2_u	2.42		

θ_1 = cut point–specific intercept for Tanner stage 1 or higher; θ_2 = cut point–specific intercept for Tanner stage 2 or higher; σ^2_u = random-effects estimate; 95% CI = 95% confidence interval; Est. = estimate; LRT = likelihood ratio test statistic; OR = odds ratio.

[a] Ever obese status defined per Centers for Disease Control and Prevention guidelines of body mass index z score $\geq$ 95th percentile.

[b] Minority status defined as 1 = minority, including black (90%), Hispanic (9%), or Asian American (1%); and 0 = Caucasian.

[c] Biological father absence defined as 1 = absent at all time points; 0 = present at least one time point.

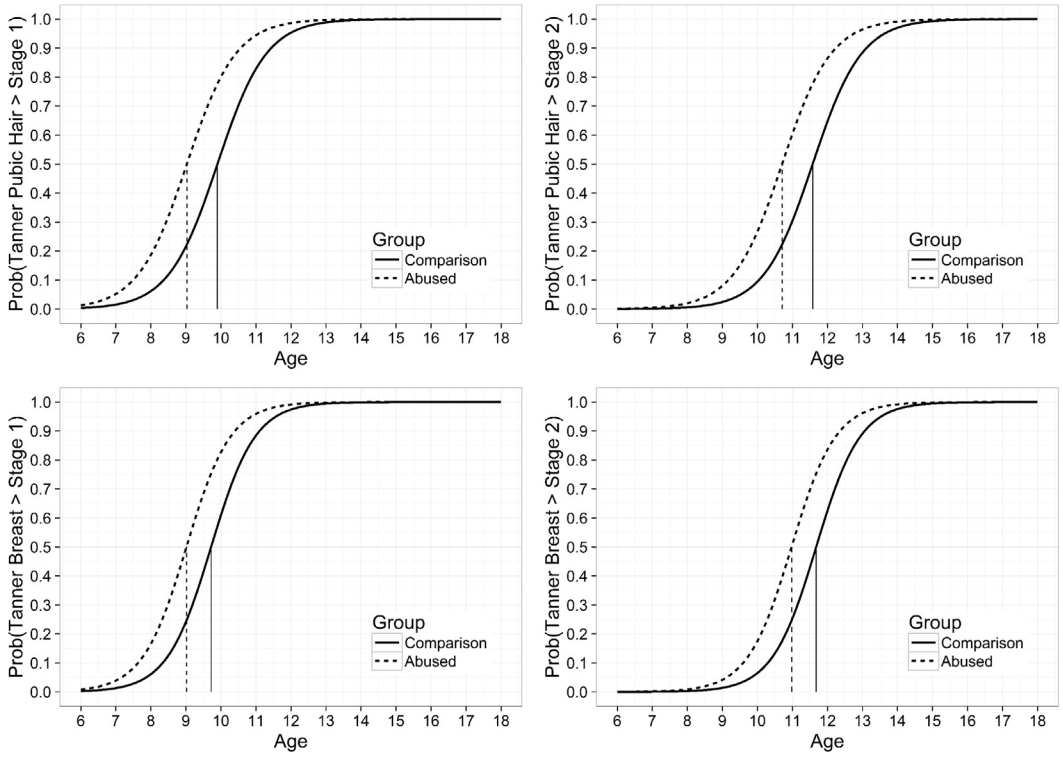

Figure 2. Probability (Prob) of transitioning to a higher Tanner stage as a function of age and abused versus comparison groups. The top row depicts pubic hair development, and the bottom row depicts breast development. In each panel, the curve for the sexual abuse group is shifted to the left, indicating a greater probability of transitioning to a higher category at an earlier age. The horizontal dashed line at the .50 probability and the drop-down vertical dashed lines indicate the estimated ages for a 50/50 chance of transitioning to that higher category. The upper panels show an approximate 1-year shift in these curves, with a 50/50 chance of reaching pubic hair stage 2 or higher by age 9 years for a given sexually abused versus almost age 10 years for a comparison. A given abused had a 50/50 chance of transitioning to pubic hair stage 3 by age 10 years 8 months versus 11 years 8 months for a comparison. The separation was approximately 8 months for breast development (lower panels), with sexually abused having a 50/50 chance of transitioning to breast stage 2 by age 9 years versus 9 years 8 months for comparison. A sexually abused had a 50/50 chance of transitioning to breast stage 3 by age 11 years versus 11 years 8 months for a comparison. All probabilities were adjusted for covariates.

420,000 women reported a relative risk of 1.05 (or 5%) for each year of younger menarche onset [19], with prolonged exposure to estrogens cited as the chief mechanism. Moreover, animal studies have shown that the biggest concern is when estrogen exposure occurs during the initial stages of breast development, a time when estrogen responsiveness is heightened [32]. Sexually abused females exhibit high rates of depression and substance abuse, risky sexual behaviors, teenage pregnancy [33], and early puberty may be a potential contributor to this elevated risk.

Prior research demonstrating a connection between sexual abuse and early pubertal maturation has lacked the ability to test alternative explanations including obesity or the absence of biological fathers. Although we controlled for these theoretically based confounds statistically, we do not purport to have fully tested the mechanistic properties of each, including the sole reliance on biological father absence as a proxy measure of more dysfunctional family environment that extends well beyond father absence [12]. Furthermore, the evolutionary perspective has been considerably extended by Del Giudice et al. [34] via the adaptive calibration model, which posits that individual responses to stress can result in adaptations that enable the organism to calibrate its response to current and future environmental conditions to survive and ultimately reproduce. Thus, adversity-related programming of the HPA axis can affect the organism's behavior and physiological systems to prioritize energy allocation for growth, reproduction, and survival, rather than repair. The brain is highly plastic during early development, and thus exposure to adversity can program or calibrate the activity of the HPA axis to match the energetic demands of the internal and external milieu, setting a trajectory of early sexual maturation.

Nonetheless, our findings point to the possibility that there is something unique about the experience of sexual abuse that influences pubertal maturation. Childhood sexual abuse has been described by many victims as a highly stressful condition that is endured for many years, often during very sensitive periods of development. There is consistent evidence that childhood abuse alters the HPA axis and other centers in the brain and consequently the hormonal correlates of puberty [35]. Such alterations have been predictive of an accelerated pubertal tempo in adolescent females [36]. Consistent findings from this line of inquiry, coupled with findings presented here, contribute to the logical conclusion that stress is likely a major culprit in explaining early puberty.

Albeit arguably on the extreme end of the stress continuum, the question still remains as to whether childhood sexual abuse is somehow qualitatively different from other types of abuse or childhood adversities. Theory and evidence suggest that early maturation due to altered stress physiology may be

operationalized through reciprocal interactions of the central stress centers with other biological systems such as the gonadal system via hypothalamic–pituitary–gonadal axis. Some have proposed that the impact of early sexual contact or intercourse in the context of childhood sexual abuse may stimulate the hypothalamic–pituitary–gonadal axis, which in turn stimulates growth of secondary sexual characteristics [11]. Circulating gonadal steroids, especially estrogen, exert modulating effects on HPA axis functioning, including responsivity and sensitivity to the negative feedback inhibition by cortisol [7]. Animal studies consistently have revealed a strong stimulatory influence of estrogen on HPA axis functioning (for a recent review see Handa and Weisner [37]), with modulatory effects on mineralocorticoid and glucocorticoid receptors [38]. Moreover, estrogen may directly enhance corticotropin-releasing hormone gene transcription in the hypothalamus through binding to estrogen-responsive elements on the corticotropin-releasing hormone gene [39]. Thus, gonadal steroids are important modulators of the HPA axis, which suggests a potential route for estrogen-mediated increase in glucocorticoid levels and enhance resistance to glucocorticoid-mediated negative inhibition of the HPA axis that can influence growth and reproduction. Because breast and ovary tissues are responsive to estrogen, prolonged exposure to estrogen among early maturing females may underlie the increased risk of developing ovarian and breast cancer in adulthood [3].

The epigenetic modification of genes related to physiological plasticity can also provide mechanistic insight into influences on reproductive strategies. Groundbreaking work has shown that differential maternal care in rat pups modified the methylation pattern of the NR3C1 exon 1_7, thereby accelerating pubertal timing. In translational work from rats to humans, a similar pattern of epigenetic modification was found in response to childhood abuse [5]. The proposed mechanism is thought to operate via a methylation-mediated decrease in glucocorticoid receptor gene expression, most notably in the hippocampus, which reduces hippocampal sensitivity to suppress the HPA axis through negative feedback [40], thus increasing HPA responses to stress.

There are several caveats to consider when interpreting these data. This sample included only intrafamilial sexual abuse, and it is not clear how the findings may generalize to other experiences of sexual abuse (e.g., abuse by a nonfamilial perpetrator or abuse without genital contact or penetration). This sample was restricted to females, and these findings cannot be extrapolated to males. Measurement of pubertal development employed self-assessment of Tanner stages in later years and may introduce bias in reporting. Although the analyses did not fully test the effects of adiposity status, the effect for obesity was large in magnitude for breast development only (OR: 11.18), and the effect of abuse remained robust even after controlling obesity. The design also includes some females who had already begun puberty before study entry. However, we were able to confirm negligible variation in parameter estimates that supports the MAR assumption that might be attributable to this left censoring.

Clearly, more research is needed before ruling out an evolutionary perspective and to articulate plausible stress physiology, biologic embedding, and epigenetic mechanistic pathways. Such research will be vital to the formulation of translational models designed to mitigate the impact of childhood sexual abuse—or the stress of this and other types of severe childhood adversities—on reproductive strategies that can ultimately increase the risk of breast and ovarian cancers.

The definitive nature of this research suggests that the field might work toward an agenda that can raise awareness regarding the potential risks for reproductive cancers that early maturing adolescents may face. For example, early maturing adolescents could be encouraged to attain early cancer screening, seek advice from endocrinologists, and practice preventative lifestyle behaviors. Furthermore, practitioners who work with adolescents and who are aware of earlier maturation need to understand the associated psychosocial risks. Referrals to behavioral counselors could help to prevent some of the deleterious outcomes for early maturing adolescents. Practitioners who are aware of a history of sexual abuse and other significant trauma in the patient should be aware of the potential for early maturation. Such awareness can alert practitioners to the possibility that evidence-based trauma-focused behavioral therapies need to be firmly in place. These trauma treatments are designed to enable victims to navigate psychosocial difficulties that may be associated with their abuse. In turn, these therapies can be instrumental in helping abuse victims who are also early maturing adolescents to navigate developmental discordance.

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