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.

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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 maximize 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.
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