JOURNAL OF
Research on Adolescence
Society for Research on Adolescence
www.s-r-a.org

JOURNAL OF RESEARCH ON ADOLESCENCE, 24(4), 689–702

Linking Childhood Maltreatment With Girls' Internalizing Symptoms: Early Puberty as a Tipping Point

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Earlier physical maturation may often be preceded by a range of adversities and life stressors. This study investigates childhood maltreatment, internalizing symptoms, and pubertal timing in girls residing in foster care ($N = 100$, $M = 11.54$ years old at Time 1). Girls were assessed at two time points 2 years apart. There were no direct effects of maltreatment on internalizing symptoms; rather, childhood sexual abuse predicted earlier pubertal development which, in turn, was associated with higher levels of internalizing symptoms concurrently and longitudinally. This distinctive role for early pubertal timing suggests that the heightened sexual circumstances of puberty may be especially disturbing for girls whose lives have already been disrupted by inappropriate and unwanted sexual experiences.

Puberty is a universal experience, but it can also represent a period of risk. A multitude of problem behaviors and clinical disorders first emerge during this transition, often continuing through adolescence. Understanding which children are most vulnerable is critical for prevention and intervention efforts, and the study of individual differences in maturation has provided a key piece of the puzzle. It is well established that earlier pubertal development, particularly in girls, significantly increases risk for an array of psychological sequelae, including depression and internalizing problems (e.g., Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Mendle, Turkheimer, & Emery, 2007; Stice, Presnell, & Bearman, 2001). This clinical correlation is complicated by the simple fact that the pubertal transition does not occur in isolation, but is one segment of a larger developmental continuum. Children enter puberty carrying a complex interplay of biological predispositions, life experiences, individual resiliencies, and environmental risks. These factors play a vital role in children's response, awareness, and capacity for navigating the substantial challenges of maturation.

There is notable overlap between the risks and experiences that predict pubertal timing and those that predict internalizing symptoms and other forms of psychopathology. Among the most striking of these joint risk factors is a history of childhood maltreatment. Childhood maltreatment is among the most consistent predictors of internalizing symptoms (Edwards, Holden, Felitti, & Anda, 2003; Green et al., 2010). Maltreatment—and specifically childhood sexual abuse—is also affiliated with an earlier timing of puberty in girls (Bergevin, Bukowski, & Karavasilis, 2003; Mendle, Leve, Van Ryzin, Natsuaki, & Ge, 2011; Natsuaki, Leve, & Mendle, 2011; Romans, Martin, Gendall, & Herbison, 2003; Wise, Palmer, Rothman, & Rosenberg, 2009). Given the high overlap among these risks, and the putative sequelae attributed to them, developmental psychopathologists have been faced with a long-standing conundrum: How to ascertain which risks are relevant for which outcomes.

In this study, we provide preliminary answers to some of these questions by leveraging an underresearched population: girls in foster care. This sample offers a novel opportunity to disentangle associations of internalizing symptoms with pubertal timing and childhood maltreatment, because few participants in epidemiological samples have

This research was supported by the following grants: R01 MH054257, P30 DA023920, and R21 DA027091, NIDA, U.S. PHS. The authors thank Patricia Chamberlain for her contributions to the larger study, Courtenay Padgett, Priscilla Havlis, and Danielle Guerrero for project coordination, Michelle Baumann and Natalie Finn for editorial assistance, and the children and families who participated in the project.

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DOI: 10.1111/jora.12075

experienced the unfortunate severity and diversity of risks as the girls in this foster care population. We are particularly interested in the ways that childhood maltreatment may intersect with normal developmental pathways in the emergence of internalizing symptoms and therefore consider three foci: (1) internalizing symptoms during early adolescence among all girls in this sample, (2) internalizing symptoms for those girls who exhibit earlier pubertal timing relative to other participants, and (3) the extent to which earlier pubertal timing may mediate or help explain associations of maltreatment with internalizing symptoms.

Childhood Maltreatment

Childhood maltreatment serves as a frequent antecedent for a variety of different forms of psychopathology, associated with over 40% of childhood onset disorders and approximately 30% of later-onset disorders (e.g., Green et al., 2010). Among the most common psychological sequelae are internalizing problems, with rates of depressive disorders and suicide attempts approximately three to five times more prevalent among individuals with histories of childhood maltreatment (Brown, Cohen, Johnson, & Smailes, 1999; Putnam, 2003). The age at which maltreatment occurs may additionally be pertinent, with some studies indicating that individuals who experience maltreatment earlier in life are at greatest risk for subsequent depression and anxiety (Kaplow & Widom, 2007; Maercker, Michael, Fehm, Becker, & Margraf, 2004) and impairments in self-concept (Bolger, Patterson, & Kupersmidt, 1998).

Childhood maltreatment, in and of itself, holds a high degree of multifinality, but a number of different adverse events qualify as maltreatment. Manly and colleagues have advocated the need to consider different subtypes of maltreatment, as each may calibrate developmental trajectories in different ways (Manly, Kim, Rogosch, & Cicchetti, 2001). Although many children experience more than one type of maltreatment, distinctions are commonly made between physical abuse, sexual abuse, and neglect (e.g., inadequate food, shelter, clothing, etc.). Among these subtypes, sexual abuse is the strongest predictor of internalizing symptoms, increasing the risk of dysthymia ninefold, major depressive disorder threefold, and suicide attempts fivefold (Brown et al., 1999). Sexual abuse is also significantly associated with poor self-esteem, whereas other subtypes are not (Bolger et al., 1998).

Puberty, Maltreatment, and Internalizing Symptoms

In general, the pubertal transition is a critical one for internalizing problems. While rates of depression for boys and girls are roughly comparable during childhood, adult women are twice as likely as adult men to be depressed (e.g., Kessler, 2003). This profound disparity first emerges at puberty, between the ages of 11 and 15 (e.g., Cyranowski, Frank, Young, & Shear, 2000; Ge, Lorenz, Conger, Elder, & Simons, 1994; Wade, Cairney, & Pevalin, 2002). The evolving gender discrepancy—and its striking coincidence with the pubertal period—has been ascribed to a variety of factors, including surges in gonadal and adrenal hormones (e.g., Angold, Costello, Erkanli, & Worthman, 1999; Steiner, Dunn, & Born, 2003); greater propensity for rumination in females (Nolen-Hoeksema & Girgus, 1994); pubertal weight gain, which distances girls from the "thin ideal" and results in low self-esteem and body dissatisfaction (Hankin & Abramson, 2001; Wichstrom, 1999); intensification of sex role socialization at puberty and increased pressure for girls to conform to traditional gender roles (Hill & Lynch, 1983; see also Priess, Lindberg, & Hyde, 2009); and girls' higher levels of stress during the transition to adolescence compared with boys (Ge, Conger, & Elder, 2001; Rudolph & Hammen, 1999).

Despite global increases in depressive vulnerability at puberty, risk is particularly high for girls who experience early pubertal timing (e.g., Ge & Natsuaki, 2009). They are more likely to attempt suicide (Graber et al., 1997) and meet DSM-IV diagnostic criteria for an episode of major depressive disorder (Stice et al., 2001). These associations have been obtained across a variety of measurement modalities for both early pubertal timing and internalizing symptomatology and continue to persist into early adulthood (see Mendle et al., 2007 for a review). Such susceptibility can be conceptualized as an interplay between genetic predispositions for internalizing and the distinctive socioenvironmental challenges that accompany early development. Based on a seemingly older appearance, others may ascribe unwarranted perceptions of maturity onto early developing girls. This sets off a cascade of reactions (e.g., reduced parental monitoring, difficulty maintaining friendships with peers, greater affiliations with older boyfriends, and increases in unwanted sexual comments from others; Marin, Coyle, Gomez, Carvajal, & Kirby, 2000; Petersen & Hyde, 2009; Petersen, Sarigiani, & Kennedy, 1991), which can create and

amplify feelings of isolation, loneliness, and helplessness among early developers.

Such situations—often part of the typical early puberty narrative—may be particularly harrowing for girls with histories of childhood sexual abuse. Because of its explicit nature, childhood sexual abuse is believed to produce a characteristic set of sexual distortions and preoccupations in response to sexually suggestive experiences later in life (Noll, Shenk, Barnes, & Putnam, 2009; Noll, Trickett, & Putnam, 2003). Early puberty may qualify as such an experience, and it is logical that the emergence of permanent and unambiguous signs of sexual development would hold unusual resonance for girls with histories of sexual trauma. Girls may be stymied or perplexed by burgeoning feelings of sexual desire (which tend to emerge in conjunction with rises in androgen hormones at puberty), and they may be uncertain how to handle looming expectations of romantic relationships and, potentially, expressions of physical intimacy.

Critically, the emergence of secondary sexual characteristics is not simply a private observation, but visibly evident to others. Early adolescents may be both curious and awkward when they observe physical changes in their peers, but they may also be malicious. Girls who reach puberty ahead of peers are substantially more likely to be targets of peer sexual harassment (McMasters, Connolly, Pepler, & Craig, 2002; Petersen & Hyde, 2009; Stattin & Magnusson, 1990). Early maturing girls also receive unsolicited commentary about their changing bodies from nonpeers (Stattin & Magnusson, 1990) and are more likely to be approached by and to date older boys (Marin et al., 2000).

Considering these social and physiological shifts together means that the heightened sexual circumstances of early pubertal timing may resemble a revictimizing process for girls whose lives have already been traumatically disrupted by inappropriate sexual contact, reactivating distressing memories and increasing likelihood of unwanted sexual attention, comments, and harassment from both peers and adults. Rather than an isolated risk for internalizing symptoms, early maturation becomes akin to a "tipping point" within a larger continuum. As in the field of epidemiology, where a tipping point denotes the onset of marked—and sometimes immutable—change, the profound challenges of early puberty may be particularly evocative for girls who have already powerlessly endured one premature sexual transition. The distinctive inter- and intrapersonal strains presented during this transition coincide with the feelings of shame, humiliation, and powerlessness which already tend to be pronounced in victims of childhood sexual abuse (Coffey, Leitenberg, Henning, Turner, & Bennett, 1996; Feiring & Taska, 2005; Negrao, Bonanno, Noll, Putnam, & Trickett, 2005). The particular social experiences and feedback they encounter during this period may additionally set a critical foundation for future emotions and behavior.

The Importance of Studying a Maltreated Sample

Childhood maltreatment is understandably a highly sensitive topic, particularly when coupled with investigations of internalizing symptoms and puberty. Perhaps for this reason, much of the research in this area has employed retrospective reporting of both maltreatment and pubertal development. Correspondingly, studies that target the psychological sequelae of maturational timing in nonmaltreated populations often obtain robust effects. Yet clinical risks are predicated in a complex nexus of individual experiences and predispositions, of which developmental stage is only one component. For example, earlier timing of pubertal development correlates not just with maltreatment, but with a broad variety of other adversities predictive of psychopathology—including insecure attachment during infancy, family structure, low socioeconomic status, harsh parenting, and exposure to intimate partner violence (e.g., Belsky et al., 2007; James-Todd, Tehranifar, Rich-Edwards, Titievsky, & Terry, 2010; Obeidallah, Brennan, Brooks-Gunn, Kindlon, & Earls, 2000). Therefore, it can be difficult to distill the extent to which early pubertal timing represents a distinct risk or is simply one facet of a larger matrix of adversity.

Studying a maltreated sample is advantageous for both research and practical purposes. At a humanitarian level, maltreated children are rarely included in the research literature, even though they experience a disproportionate share of both clinical disorders and environmental risks. The unique nature of the sample holds additional promise for resolving larger questions of developmental science. Understanding the causal role of psychological risks has presented a longstanding challenge to the field of developmental psychopathology, as it is impossible to engage in the sorts of random assignment typically used in psychological research. Therefore, although many risk factors may be both logical and powerful antecedents of psychological distress, conclusions are necessarily limited by the caveats of correlational designs.

When studying maltreated children who have been placed in foster care, it is important to note that *all* of these children have a history of maltreatment and all are therefore at increased likelihood of developing internalizing symptoms. But not all of these children experience earlier puberty or heightened internalizing problems. To the extent that pubertal timing continues to exhibit predictive influence on internalizing symptoms within this highly disadvantaged population, we can verify prior findings regarding early pubertal timing which have been based on results obtained from typical populations. We can moreover take the first step toward considering more intricate hypotheses—such as the notion of the "tipping point" we have described here. Similar special population investigations have been previously used within the puberty literature among girls training to be professional ballerinas (Brooks-Gunn, Attie, Burrow, Rosso, & Warren, 1989; Brooks-Gunn & Warren, 1985) and enrolled in single-sex schools (Caspi, Lynam, Moffitt, & Silva, 1993). Yet these studies—as is the case with the majority of pubertal timing research—have targeted only the sequelae of pubertal timing. Considering puberty in the context of both early life antecedents and adolescent outcomes is a surprisingly rare research endeavor, despite widespread acknowledgement that puberty is one segment of a larger developmental continuum (Graber, Nichols, & Brooks-Gunn, 2010).

The Present Study

The overarching aim of this study is to clarify pathways among childhood maltreatment, pubertal timing, and girls' internalizing symptoms. Using a sample of girls in foster care, we can approach and resolve general questions of development, particularly how multiple risks may intersect over the course of time, and which risks are most critical for adjustment or maladjustment later in life. Specifically, maltreatment may globally increase risk for internalizing symptoms, but those girls who report more advanced physical development at a younger chronological age are subject to a critical additional stressor. Yet girls' likelihood of experiencing early puberty is not fully random, but rather rooted in individual histories, with girls subjected to sexual abuse disproportionately at risk for earlier physical development (e.g., Bergevin et al., 2003; Wise et al., 2009). This fact has muddied previous research and complicated the inferences that may be drawn from results.

Based on previous analyses of this sample (Mendle et al., 2011), we expect that childhood sexual abuse will predict reports of earlier puberty compared with other forms of maltreatment. Given the heightened sexual circumstances and stressors associated with early pubertal timing, our overarching hypothesis is that the pubertal transition represents a particularly distressing time (i.e., a tipping point) for girls with histories of childhood sexual abuse, which will be evident in early maturing girls' levels of internalizing symptoms expressed over the 2-year span of the study. Because we consider earlier pubertal timing a key link between maltreatment and internalizing problems, our analyses therefore examine (1) the direct effect of sexual abuse, physical abuse, and neglect on internalizing symptoms; (2) the association between these forms of maltreatment and earlier perceived pubertal development; (3) whether maltreatment and/or early pubertal timing continue to predict internalizing symptoms longitudinally; and (4) the extent to which early pubertal timing may mediate associations of childhood maltreatment—particularly childhood sexual abuse—with internalizing symptoms. Our proposed model is shown in Figure 1. We initially examined the direct effect of maltreatment on internalizing symptoms at Times 1 and 2, then fit the full model, and evaluated indirect effects.

METHOD

Participants

Eligible study participants comprised girls living in state-supported foster homes in one of two counties containing major metropolitan areas in the Pacific Northwest and who were finishing elementary school between 2004 and 2007. The girls were referred through the local child welfare systems. From the pool of participants who met the above criteria (N = 145), 100 girls and their foster parents were recruited during the spring of their final year of elementary school (typically the 5th or 6th grade, depending on school district). The participants were part of a longitudinal intervention trial in which girls were randomly assigned to a behavioral support intervention condition (n = 48) or to a regular foster care control condition (n = 52; Smith, Leve, & Chamberlain, 2011). Although intervention effects were not a focus of the present study, we controlled for intervention condition in our analyses. Foster parent and caseworker consent and youth assent were obtained for each girl.

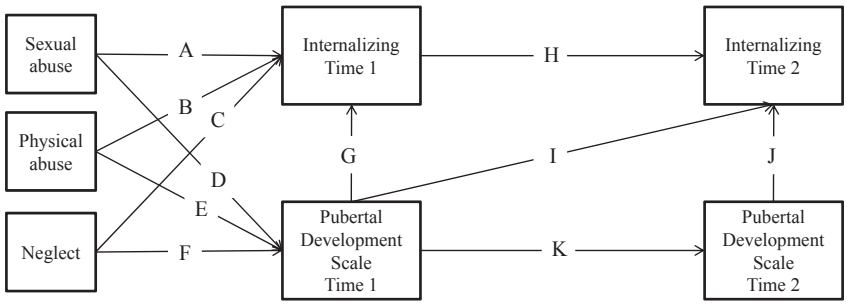

FIGURE 1 Hypothesized model of pubertal timing, childhood maltreatment, and internalizing symptoms. *Note.* Covariates and correlations among maltreatment variables were included in model but are not depicted. Sexual abuse, physical abuse, and neglect represent number of reported events for each type of maltreatment. Scores on Pubertal Development Scale expressed as a deviation from the average score reported for children of the same chronological age, to indicate pubertal timing.

The girls were, on average, 11.54 years of age (*SD* = .48, range = 10.44–12.92) at the start of the study, with an ethnic distribution of 63% European American, 14% multiracial, 10% Latino, 9% African American, and 4% Native American. The mean age of foster care entry was 7.63 years (*SD* = 3.14), and the mean time in foster care was 2.90 years (*SD* = 2.25). Most girls had experienced more than one foster caregiver transition since placement (80.8%; *M* number of foster care placements = 3.90, *SD* = 3.03, Range = 1.0–18.0).

Procedure

Girls and their foster parents were assessed at baseline (T1) and 24 months (T2) postbaseline. The assessment consisted of a standardized interview and questionnaires for each girl and foster parent, an interview with each girl's caseworker, and the collection of child welfare records. The interviews lasted approximately 2 hr and were aimed at measuring child and family characteristics, child behaviors, and parenting practices. Sample retention across the two time points was high (93%).

Measures

Childhood maltreatment. The girls' cumulative maltreatment experiences at T1 were drawn from child welfare case records that were coded using a modified version of the Maltreatment Classification System (MCS; Barnett, Manly, & Cicchetti, 1993). This system allows for the coding of different types of maltreatment, including physical abuse, sexual abuse, and physical neglect (e.g., failure to provide adequate food or living conditions). Coders examined child welfare case records to identify incidents of maltreatment, which (1) had to match the MCS definitions of maltreatment and (2) had to be reported by a mandatory reporter or verified by the child welfare system caseworker. Case files included all information on incidents of childhood maltreatment and family history available to child welfare at the time of the study. Training in the use of the MCS was initially conducted by one of its authors (Manly). Because of the complexity of the coding system, two-thirds of the cases were double-coded and then discussed to attain a final consensus rating. Interrater reliability was computed from the 67% of files that were double-coded (prior to consensus discussions). The average percent agreement for the number of incidents of each maltreatment type was acceptable (physical abuse = 86%, sexual abuse = 86%, neglect = 82%). In addition, coders attained high levels of agreement (81%) about the total number of incidents per case.

For each girl, we used the number of incidences of maltreatment for three subtypes: sexual abuse, physical abuse, and neglect. In this sample, maltreatment rates were as follows: 56% had at least one incident of physical abuse, 67% had at least one incident of sexual abuse, 78% had at least one incident of neglect, 41% had incidents of both physical abuse and sexual abuse, 52% had incidents of both sexual abuse and neglect, 44% had incidents of physical abuse and neglect, and 32% had incidents of all three maltreatment types. Although all girls had experienced at least one type of maltreatment, there was adequate variability in the frequency of maltreatment incidents, ranging from 0 to 6, 0 to 7, and 0 to 13 times for physical abuse, sexual abuse, and neglect, respectively.

Pubertal timing. At both time points, girls and their caregivers completed a slightly abbreviated version of the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988), comprised of items assessing changes in body hair,

skin (e.g., development of pimples and acne), and breast growth. Items on the PDS are measured using a 4-point Likert scale (1 = *no changes yet*, 4 = *seems completed*). For the present analyses, we took the mean of reports of body hair changes, skin changes, and breast growth from both girls and their caregivers at each wave (*r*s between reporters ranged .49–.58, $p < .001$). According to this averaged report, 68% had at least some body hair growth at T1 ($M = 2.90$, $SD = 1.27$), 75% had at least some skin changes at T1 ($M = 2.30$, $SD = .98$), and 89% had at least some breast growth at T1 ($M = 2.33$, $SD = .64$). The items demonstrated significant stability across time (*r*s ranged .53–.58, $p < .001$). Each participant's individual PDS score was then expressed as a standardized ($M = 0$, $SD = 1$) deviation from the mean PDS score reported for adolescents of the same age in this sample. Thus, higher scores reflect a perception of more advanced development relative to other adolescents of the same age and can be considered an indicator of earlier perceived *pubertal timing*. Separate means before standardization were 2.37 ($SD = .69$) and 2.95 ($SD = .59$) for girl reports and 2.28 ($SD = .67$) and 2.86 ($SD = .55$) for caregiver reports for the T1 and T2 assessments, respectively. Cronbach's alpha = .68 for girl reports and .76 for caregiver reports at T1 and .69 for girl reports and .70 for caregiver reports at T2.

Internalizing symptoms. Internalizing symptoms were assessed using the Internalizing subscale of the Child Behavior Checklist from the Achenbach System of Empirically Based Assessment (CBCL; Achenbach & Rescorla, 2001). The CBCL is a widely used caregiver-report measure of symptoms of depression, anxiety, and social withdrawal and has demonstrated extensive construct and content validity within the research literature. On the CBCL, raw scores of caregiver endorsements are typically summed and converted to *T*-scores, and these *T*-scores were employed in the present analyses. A *T*-score of 63 (90th percentile) is typically used to demarcate a clinically significant level of internalizing symptoms. In the present sample, CBCL scores at Time 1 ranged from 33 to 88 ($M = 58.2$; $SD = 12.2$) and scores at Time 2 ranged from 33 to 74 ($M = 55.5$; $SD = 10.02$). At both time points, 41% of the sample had a *T*-score over the clinical cutoff of 63. Internal reliability was good ($\alpha = .90$ at Time 1 and $\alpha = .86$ at Time 2).

Control variables. Although it seemed unlikely that intervention condition might influence timing of puberty (given that the earliest adrenal signals of maturation typically begin well before baseline in our study), we were concerned that it might be relevant for internalizing symptoms (e.g., Kim & Leve, 2011) and we therefore controlled for intervention condition in the analyses. We also included ethnicity (White vs. non-White) as a covariate, because some research suggests racial/ethnic differences in timing of pubertal maturation (e.g., Biro et al., 2010). Lastly, we also included age at first foster care placement as a covariate, because earlier maltreatment has been shown to predict more severe symptoms of depression and anxiety (Kaplow & Widom, 2007) and because some scholars suggest that biological father absence early in childhood correlates with earlier pubertal onset (see Ellis, 2004). Descriptive information and correlations among all study variables are provided in Table 1.

Analyses

We utilized path analysis to estimate associations of childhood maltreatment, pubertal timing, and internalizing symptoms at T1 and pubertal timing and internalizing symptoms at T2 (see Figure 1). In our analyses, we utilized the number of instances of each type of maltreatment experienced by a girl, which had the advantage of allowing us to estimate the possibility that a girl experienced multiple types of maltreatment. We initially fit a model that examined the direct effect of all three types of maltreatment on internalizing symptoms at T1 and T2. We then fit the full model depicted in Figure 1 and evaluated indirect effects. Control variables (i.e., intervention condition, ethnicity, and age at first placement) were included as predictors of all outcomes in the model. Correlations between the maltreatment subtypes were also included. Descriptive information and correlation data are presented in Table 1.

According to the traditional "causal steps" approach, requirements for mediation include (1) a significant direct effect of a predictor (here, maltreatment) on a distal outcome (i.e., internalizing symptoms; Path A in Figure 1) and on a presumed mediator (i.e., pubertal timing; Path D in Figure 1), (2) a significant direct effect of the mediator on the outcome (Path G in Figure 1), and (3) a significant indirect effect of the predictor on the outcome by means of the mediator (Path D*G in Figure 1; Judd, Kenny, & McClelland, 2001; MacKinnon & Dwyer, 1993). However, such an approach may be too restrictive, and MacKinnon, Lockwood,

TABLE 1
Correlations and Sample Descriptives for All Study Variables

Variable	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*
1. Intervention condition	—									
2. Pubertal Development Scale (T1)	–.04	—								
3. Pubertal Development Scale (T2)	.14	.56***	—							
4. CBCL Internalizing (T1)	.02	.24*	.15	—						
5. CBCL Internalizing (T2)	–.08	.29**	.08	.49***	—					
6. Childhood sexual abuse	.04	.26*	.13	.05	.14	—				
7. Childhood physical abuse	–.03	–.06	.07	.08	–.11	.35***	—			
8. Childhood neglect	–.34***	–.11	–.20	–.10	.00	.05	.13	—		
9. Ethnicity	.06	–.10	.01	.00	–.23*	–.03	.17	.04	—	
10. Age at first foster care placement	–.12	–.11	–.01	–.07	–.11	–.27**	–.28**	–.03	.06	—
N	100	100	89	93	89	100	100	100	100	99
M	—	2.40	2.96	58.25	55.49	1.42	1.06	2.15	.63	7.63
SD	—	.62	.50	12.18	10.02	1.48	1.32	2.24	—	3.14

Note. PDS = Pubertal Development Scale. PDS is scaled using a 4-point Likert scale (1 = *no changes yet,* 4 = *seems completed*) for indicators of breast development, hair growth, and skin changes. Mean and correlations reported here represent the average of these three indicators for combined caregiver–child report. Sexual abuse, physical abuse, and neglect represent number of reported events of each type of maltreatment. CBCL = Child Behavior Checklist. Internalizing symptoms on the CBCL are scaled as *T*-scores.
*$p < .05$; **$p < .01$; ***$p < .001$.

Hoffman, West, and Sheets (2002) have advocated for the consideration of other approaches that focus solely on the joint significance of the mediational paths between the predictor and the outcome (i.e., from childhood maltreatment to pubertal timing to internalizing symptoms). In the absence of a direct effect of the predictor on the outcome, we would not be able to follow the traditional approach to evaluating mediation; in that case, we would follow the broader approach discussed by MacKinnon et al. (2002), which refers to the purported mediator as an "intervening variable" and reserves the term "mediator" for those situations where a direct effect exists and the causal steps approached can be followed. Thus, in this study, if we were to find direct effects of maltreatment on internalizing symptoms, we would evaluate pubertal timing as a mediator; if direct effects were not present, we would evaluate pubertal timing as an intervening variable, which remains both theoretically and clinically meaningful (Sandler, Schoenfelder, Wolchik, & MacKinnon, 2011). We would consider evidence of either mediation or an intervening variable supportive of the idea of earlier pubertal timing as a "tipping point," as both describe a mechanism by which associations between childhood sexual abuse and internalizing symptoms are accounted for by individual differences in pubertal timing.

We fit the model to the data using Mplus (Muthén & Muthén, 2006) with robust maximum likelihood analysis, which can provide unbiased estimates in the presence of missing data and/or nonnormal distributions. Using Little's test (Little, 1998), we found that the data were missing completely at random [χ^2 (43) = 56.26, ns], suggesting that the missingness did not introduce bias into the results. Standard measures of fit are reported, including the chi-square value (χ^2), the comparative fit index (CFI), the Tucker–Lewis index (TLI), and the root-mean squared error of approximation (RMSEA). Typically, CFI values >.95, TLI values >.90, RMSEA values <.05, and a nonsignificant chi-square (or a ratio of $\chi^2/df < 3.0$) indicate adequate fit (Bentler, 1990; Bentler & Bonett, 1980; Hu & Bentler, 1999).

Standard techniques for assessing the significance of indirect effects assume a normal distribution, so we used an analytic technique that is based upon the *actual* distribution of the indirect effect (PRODCLIN; MacKinnon, Fritz, Williams, & Lockwood, 2007). This technique can provide an unbiased assessment of statistical significance even in situations where the indirect effect is not normally distributed. PRODCLIN provides a 95% confidence interval for the indirect effect. If this confidence interval does not contain zero, the effect is considered to be statistically significant. Missing data were accounted for using full information maximum likelihood, a model-based technique, in which parameters of a model are estimated taking into account the relations among variables that predict which values are missing (Schafer & Graham, 2002).

RESULTS

Our first step was to evaluate the possibility of a direct effect of childhood maltreatment on internalizing symptoms at baseline and at 24 months (controlling for baseline). The model fit the data well, $\chi^2(3) = 2.05$, *ns*, CFI = 1.00, TLI = 1.00, RMSEA = .00(.00|.15), but there were no direct effects of any form of childhood maltreatment on internalizing symptoms at either Time 1 or Time 2 (controlling for Time 1).

We next fit the full model displayed in Figure 1. The model again fit the data well, $\chi^2(7) = 8.15$, *ns*, CFI = .98, TLI = .93, RMSEA = .04(.00|.13). Analyses yielded five key results (see Table 2 for model parameters). First, we replicated the previously established association between the number of sexual abuse instances and earlier pubertal timing (β = .29), but neither frequency of physical abuse instances nor neglect instances were significantly associated with pubertal timing at Time 1. Second, early maturing girls displayed higher levels of internalizing symptoms at Time 1 (β = .23) and continued to increase in symptoms longitudinally (β = .29 at Time 2), even after controlling for initial levels. Third, change in pubertal development scores from Time 1 to Time 2 (β = .56) did not predict change in internalizing symptoms, indicating that once later-developing girls reached puberty, they did not display similarly heightened symptoms. This implicates a distinct effect of *early* development, rather than risk associated with more advanced pubertal status in general. Fourth, and most intriguingly, although there were no direct effects of childhood maltreatment on internalizing symptoms, there were significant *indirect* effects. The number of instances of childhood sexual abuse predicted early pubertal timing, which in turn predicted internalizing symptoms at T1 [unstandardized beta (B) = .54; 95% CI = .03–1.31; standardized beta (β) = .07). The indirect effect of sexual abuse on internalizing symptoms at Time 2 by means of early pubertal timing was also significant [unstandardized beta (B) = .57, CI = .08–1.28; standardized beta (β) = .08)]. Age at first placement, intervention condition, and ethnicity all failed to predict internalizing symptoms or pubertal timing at Time 1 or Time 2 significantly.

TABLE 2
Parameter Estimates for Hypothesized Model of Pubertal Timing, Childhood Maltreatment, and Internalizing Symptoms in 100 Girls With Histories of Child Maltreatment

Model Path	*Estimate (β)*
A: Sexual Abuse → Internalizing symptoms (T1)	−.07
B: Physical Abuse → Internalizing symptoms (T1)	.13
C: Neglect → Internalizing symptoms (T1)	−.03
D: Sexual Abuse → Pubertal Development Scale (T1)	.29*
E: Physical Abuse → Pubertal Development Scale (T1)	−.20
F: Neglect → Pubertal Development Scale (T1)	−.12
G: Pubertal Development Scale (T1) → Internalizing symptoms (T1)	.23*
H: Internalizing symptoms (T1) → Internalizing symptoms (T2)	.46***
I: Pubertal Development Scale (T1) → Internalizing symptoms (T2)	.29**
J: Pubertal Development Scale (T2) → Internalizing symptoms (T2)	−.12
K: Pubertal Development Scale (T1) → Pubertal Development Scale (T2)	.56***

Note. Letters in table above correspond to letters in hypothesized model depicted in Figure 1. Scores on Pubertal Development Scale expressed as a deviation from average PDS score of children of same chronological age to indicate pubertal timing.
*$p < .05$; **$p < .01$; ***$p < .001$.

Additional analyses

Given that many girls in our sample experienced more than one type of maltreatment, we examined the effects of comorbidity by inserting interaction terms into the above analysis. We examined two- and three-way interactions to determine whether the effect of sexual abuse was different when alone versus in combination with other types of maltreatment. The results of the analysis showed no significant interactions, suggesting that the effects of sexual abuse did not vary in the presence of the other maltreatment types.

DISCUSSION

Understanding the ways in which childhood life experiences calibrate later development is an ongoing challenge for psychologists, particularly given the vast web of potential risks and their interrelations with each other. In the present study, we examined associations of childhood maltreatment and internalizing symptoms among a sample of foster care girls at puberty. This population is distinctive, in the severity of both maltreatment experienced and in clinical symptoms exhibited. Of special concern are the paths between these two, with the hope that prevention and intervention efforts incorporate and reflect the daily challenges experienced by these children.

Within this sample, there were no direct associations of the frequency of childhood sexual abuse,

physical abuse, or neglect incidents with higher levels of internalizing symptoms. Although initially surprising—given the high association of depression and anxiety with maltreatment in epidemiological samples—this finding is likely due to the fact that *all* girls in our sample experienced at least one form of maltreatment and many experienced multiple forms. In fact, the mean internalizing score of children in our sample was 58 at study inception, indicating an average level of symptoms 80% higher than typical for children of this age. More than 40% of girls experienced clinically severe levels of internalizing symptoms at each time points. Despite the lack of direct effects, there were significant indirect effects of one particular form of maltreatment: Childhood sexual abuse predicted earlier pubertal timing relative to peers which, in turn, predicted concurrent and longitudinal internalizing symptoms. Thus, even within a population globally at risk for increased depression, girls with early pubertal timing exhibited more severe levels of internalizing symptoms and continued to display significantly increased symptoms 2 years later.

These findings are congruent with an extensive body of previous research that implicates pubertal timing as a risk for depression. Yet perhaps even more salient are the implications these findings have for understanding the role of early pubertal timing in a broader, birth-to-adolescence continuum. Given that early puberty often co-occurs with a wide variety of childhood risks for internalizing problems—ranging from harsh punishment to family structure to low economic status (e.g., Belsky et al., 2007; James-Todd et al., 2010)—it is sometimes difficult to distill or evaluate the unique effects of pubertal timing. Some scholars have therefore emphasized the need for research that considers both the antecedents and the outcomes correlated with early pubertal timing within a single study (e.g., Graber et al., 2010). In the current sample, composed of participants whose histories of adversity exceeded the norm, those girls who experienced childhood sexual abuse in particular were at heightened risk for internalizing symptoms, and this greater severity of emotional distress was attributable to the greater likelihood of earlier pubertal timing within this group rather than the direct effects of childhood maltreatment.

The physiological mechanisms underlying associations of early maltreatment, pubertal timing, and subsequent outcomes are still being discovered. Of particular interest have been insights into the way childhood sexual abuse may shape responses to stress via the hypothalamic–pituitary–adrenal (HPA) axis. When functioning normally, the pituitary gland secretes adrenocorticotropic hormone (ACTH) in response to stressful events; ACTH, in turn, stimulates the adrenal glands to release cortisol and other glucocorticoids (e.g., Tarullo & Gunnar, 2006). A number of studies have demonstrated that both adults (Heim & Nemeroff, 2001; Heim et al., 2000) and children (De Bellis et al., 1999; King, Mandansky, King, Fletcher, & Brewer, 2001) with histories of sexual abuse show excessive levels of ACTH secretion. Because hyperactivity of the HPA axis is among the most stable physiological correlates of major depression (e.g., Pariante & Lightman, 2008), it may be that childhood sexual abuse increases vulnerability to depression by sensitizing the HPA axis's response to stress (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008). That is, for individuals with a high genetic propensity for internalizing problems, elevations in cortisol and other hormonal indices of stress may determine a characteristic neuroendocrine response: The initial *hyper*activity of the HPA axis may eventually down-regulate in response to chronic stress, resulting in hypo-activity (or "blunted" cortisol; Miller, Chen, & Zhou, 2007).

Trickett and Putnam (1993) have proposed that the earlier timing of pubertal maturation among girls with histories of sexual abuse is attributable to this physiological stress response. Specifically, they suggested that childhood sexual abuse prematurely activates both HPA and HPG axes, hastening the onset of physical maturation. Yet while the Trickett and Putnam hypothesis effectively explains one half of the developmental equation—the antecedents of early puberty—it does not capture the other side: the emotional and mood sequelae of early puberty and how these relate to childhood maltreatment. It also does not explain why sexual abuse seems to differ from other forms of maltreatment in predicting pubertal timing (and correlated psychological distress), because early life stress, in general, can be associated with HPA dysregulation.

The correspondence between the sexual developments of puberty and the explicit nature of sexual trauma is difficult to overlook. It seems logical that visible signs of puberty would provoke particular worries, memories, and resonance in girls with a history of childhood sexual abuse, as they have already endured one premature and unwanted sexual transition. Moreover, the earlier puberty occurs, the less likely it may be that girls have developed resources to make sense of the experience, or the more isolated or different they may feel from peers

who have not developed at a similar rate. Yet puberty is experienced at both an individual level and a social level, and early pubertal timing is disturbingly associated with higher incidence of both same and cross-gender peer sexual harassment (McMasters et al., 2002; Petersen & Hyde, 2009; Stattin & Magnusson, 1990). While this sort of harassment would be disturbing for any child, it may be perceived as a revictimizing experience for girls with histories of childhood sexual abuse and even set the stage for future sexual preoccupation, ambivalence, or aversion, or for continued victimization in adulthood, all of which tend to occur in women who have experienced childhood sexual abuse (Barnes, Noll, Putnam, & Trickett, 2009; Noll et al., 2003). Thus, although puberty is a universal transition, this period may take on a heightened salience for girls with histories of sexual abuse and is further complicated by the early life experiences and strains which predispose these girls to earlier pubertal timing, with its related risks and adversities.

Although we lacked the traditional control of girls without a history of maltreatment, the rich literature on girls' puberty offers some insight into timing of puberty in our sample relative to other populations. The mean age of participants in a study conducted by Knowles, Niven, Fawkner, and Henretty (2009) most closely matches the mean age of participants in this sample (11.83 years in Knowles et al. vs. 11.54 years in our sample). The participants in the study by Knowles et al. were a nonclinical, nonmaltreated sample recruited through public schools, with a mean PDS score of 1.96 (SD = .69) compared with 2.4 in our sample (SD = .62). This speculatively suggests the possibility of earlier development (perceived or actual) in our participants compared with girls from more secure environments.

Lastly, it is worth noting that our study—like any nonempirical design—can only suggest pathways and mechanisms and remains formally inconclusive regarding cause and effect. We cannot separate out the adversity *specific to abuse*, from the collective adversities experienced by children with histories of abuse. However, we suspect that if findings were attributable to shared risk factors, we would see earlier puberty linked with physical abuse and neglect as well as sexual abuse, because all forms of maltreatment overlap with these shared risks. In addition, the research literature has occasionally raised the question of whether early developers are more likely targets for sexual abuse (e.g., Zabin, Emerson, & Rowland, 2005). In the case of our sample, we find it unlikely that such an explanation would account for our effects, as the average age of entry into the foster care system in our study was 7.63 years. Even very early maturing girls would be unlikely to evidence a high level of visible physical maturity prior to 7.63 years of age.

Limitations and Future Directions

Our study holds a number of strengths, most notably the use of this unique sample. By considering the experiences of maltreated children residing in foster care, we were able to gain insight into the experiences of girls with unusually high levels of adversity. In addition, we confirmed maltreatment history through case files provided by the child welfare system. Although official records may underestimate the true prevalence of child maltreatment (as many cases of maltreatment go unreported), these records are also free of memory biases or inaccuracies in reporting, which are of particular concern when dealing with self-reports of abuse. Yet our data are not fully informative about what we consider some of the most pivotal questions regarding childhood sexual abuse, internalizing symptoms, and puberty. Specifically, while the current analyses are helpful in ascertaining effects, they do not tell us what produces these effects. Possibilities include biomarkers (such as cortisol), which might confirm hypotheses regarding the HPA axis; social information (such as peer relations or harassment), which would shed light on cross-sectional environmental contributors; and cognitive measures of girls' thoughts and attitudes regarding development.

It is also worth commenting on our use of the PDS (Petersen et al., 1988). The PDS was developed, in part, to offer a pubertal assessment that would be both more easily administered and, importantly, less invasive than a physical examination or Tanner Stage images (which might be perceived as sexually explicit). Given the high rates of sexual trauma in our sample, the PDS was advantageous for the girls in our study. Nevertheless, the PDS has a number of limitations. Common concerns associated with the PDS include the measure's conflation of adrenal and gonadal signals of puberty, limited ability to capture changes that occur very early in the process of puberty, and the usual limitations of self-report measures (Dorn, Dahl, Woodward, & Biro, 2006). As any measure of pubertal development that does not involve physical examination is likely to be of variable accuracy,

we believe our findings should be interpreted as a reflection of *perceived* pubertal development.

The question of perceived development is a salient one for intervention. One of the perplexing questions in pubertal timing research is how to utilize findings to inform the development of new interventions. Early puberty is certainly a risk, but its foundations lie in genes and sociobiological pathways canalized many years prior (e.g., Ellis, 2004). It would be unlikely—and perhaps unreasonable—for the goal of prevention and intervention efforts to attempt to shift the actual timing of maturation. Yet girls' interpretations, response, and reactions to puberty are malleable, and addressing these directly may help girls come to terms with the changes that occur during this critical developmental window. Even more importantly, we view the collective socioenvironmental response to early puberty among the most strikingly undertargeted foci for intervention. Given the high frequency of unwanted sexual commentary that girls experience about their rapidly shifting bodies, increases in internalizing symptoms such as social withdrawal, poor self-esteem, or negative affect seem both reasonable and understandable. Such symptoms could certainly be targeted in an individual therapeutic context. Yet perhaps a more effective option might be psychosocial interventions aimed at reducing peer-to-peer harassment in schools and better educating all children how to accommodate the changes that can occur during the early adolescent period. Improved caregiver awareness and understanding of pubertal timing may be an additional target, as caregivers (like peers) have a tendency to react to children based on their observable—rather than chronological—age.

Conclusion

Early pubertal timing is a well-documented risk for psychological distress, but it is hardly an idiosyncratic event. Rather, a particular child's likelihood of early pubertal timing is rooted in a multitude of predisposing factors, including childhood life adversities. In our sample of maltreated girls, pubertal timing served as a pivotal link between childhood sexual abuse and later internalizing symptoms. Because there were no direct effects of childhood sexual abuse on internalizing symptoms, it seems that earlier pubertal timing may be akin to a "tipping point" for emotional difficulties for individuals already at risk, triggering extra challenges and pressures to be navigated, and intensifying negative affect and psychological distress.

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