



Published in final edited form as:

J Res Adolesc. 2011 December 1; 21(4): 871–880. doi:10.1111/j.1532-7795.2011.00746.x.

Associations Between Early Life Stress, Child Maltreatment, and Pubertal Development Among Girls in Foster Care

Jane Mendle, Leslie D. Leve, Mark Van Ryzin, Misaki N. Natsuaki, and Xiaojia Ge

Jane Mendle is at the University of Oregon. Leslie D. Leve and Mark Van Ryzin are at the Oregon Social Learning Center. Misaki N. Natsuaki is at the University of California, Riverside.

Abstract

The present study investigated pubertal development in girls with maltreatment histories ($N = 100$), assessed at four time points over 2 years beginning in the spring of their final year of elementary school. This sample is unique, in that participants were subject to an unusual level of environmental risk early in life and resided in foster care at the start of the study. Analyses replicated the previously established association between sexual abuse and earlier onset of maturation and earlier age at menarche. Physical abuse was related to a more rapid tempo of pubertal development across the period assessed. These results strengthen previous investigations of childhood maltreatment and puberty, highlighting the complexity and specificity of early life experiences for later development.

Keywords

maltreatment; puberty; girls; adolescent; longitudinal

As a developmental stage, puberty represents a time of personal, biological, social, and emotional transformation. There is considerable individual variability in both the timing and rate of physical development (Marshall & Tanner, 1969), and it is a well-replicated phenomenon that physical maturation is accelerated in girls whose early life experiences are marked by social risks and disadvantages. This has triggered considerable interest in the role of early childhood stress in pubertal development; it is of particular salience as early physical maturation compared to peers serves as a harbinger for a wide variety of psychological and medical difficulties later in life (see Costenbader, Feskanich, Stampfer & Karlson, 2007; Laitinen, Power, & Jarvel, 2001; Mendle, Turkheimer, & Emery, 2007).

The present paper investigates the timing and rate of pubertal maturation in a sample of girls in foster care. Three separate bodies of research literature suggest this sample might provide a unique opportunity to bolster our understanding of the relation of early life stress with physical maturation. The first relevant literature is the research on childhood maltreatment. Girls who experience child sexual abuse (CSA) tend to have earlier ages of menarche than girls who have not experienced sexual abuse (Bergeton, Bukowski, & Karavasilis, 2003; Romans, Martin, Gendall, & Herbison, 2003; Turner, Runz, & Galambos, 1999; Wise, Palmer, Rothman, & Rosenberg, 2009). In a seminal paper, Trickett and Putnam (1993) proposed that the trauma of CSA introduces physiological as well as psychological consequences for children, accelerating maturation by activating the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes prematurely. The resulting

stress-induced alterations in hormones explain not only timing of maturation, but some of the aggressive and sexualized behavior that tends to emerge in CSA victims. In accordance with this, a number of studies have confirmed dysregulation of the HPA axis in women with a history of sexual abuse (e.g., Heim et al., 2000; Heim & Nemeroff, 2001; Putnam & Trickett, 1997). A relation between childhood physical abuse and early maturation has also been observed, though less robustly and less consistently than for sexual abuse (Bergevin et al., 2003; Wise et al., 2009), and studies do not always control for the possibility of concurrent sexual abuse (e.g., Romans et al., 2003).

Second, sociobiological models of maturation seek to explain individual differences in pubertal timing in terms of an evolved, psychophysiological response to stressful family environments (e.g., Belsky, Steinberg & Draper, 1991, Ellis, 2004; Surbey, 1990). Children whose early social environments are characterized by high levels of conflict, absent parents, poverty, harsh or inconsistent discipline, and poor parent-child relationships learn that the world is precarious, resources scarce, and relationships untrustworthy. Under such conditions, an earlier physical development is – evolutionarily speaking – reproductively advantageous. Children therefore mature in such a way that speeds puberty and the onset of child-bearing, tending to be oriented towards mating and erratic romantic partnerships rather than high-quality investments in their own relationships and offspring.

Lastly, the concept of “weathering” has been proposed to understand why some populations age more swiftly and detrimentally than others (Geronimus, 1994; Geronimus et al., 2001). “Weathering” is a type of premature aging believed to be a consequence of racial/ethnic or socioeconomic group disadvantages, which constrain health care and create daily environments that demand considerable physical and emotional resources. Although much of the research on weathering concentrates on health disparities among groups later in life, it has been suggested that early menarche in girls from disadvantaged backgrounds may reflect one of the earliest signals that weathering begins in childhood (Foster, Hagan, & Brooks-Gunn, 2008).

Collectively, these three bodies of literature provide convergent evidence that children with stressful life experiences are likely to have an earlier timing of maturation than those who do not. While each posits a different pathway by which acceleration is believed to occur, the goal of the present paper is not to examine or contrast these pathways. Rather, we aim to deepen understanding of the relation between early life stress and pubertal development by examining pubertal maturation in an unusual and under-researched sample: girls with histories of child maltreatment and placement in foster care. This sample provides a rich lens for considering stress and puberty, as the girls have been subject to severe and diverse risks during childhood. In fact, the stress history of these girls far exceeds that of participants in all previous studies of timing of puberty and early life experiences. Our work additionally extends prior research by utilizing documented histories of childhood maltreatment drawn from child welfare records; virtually all of the extant literature in this field relies on retrospective self-reports, which have been critiqued for their differing interpretations of abuse (Bergevin et al., 2003). We are therefore able to examine multiple forms of documented maltreatment within a single sample.

Because of the dearth of research examining pubertal maturation in maltreated populations, the present study is primarily exploratory in nature. Analyses are guided by three main aims. First, we aim to establish a basic understanding of the timing and tempo of pubertal development in this population. We utilize a continuous measure of maturation representing changes in skin, body hair, and breast development collected at four time points. Second, we distinguish among a variety of different stressful early life experiences, to clarify whether the magnitude of the stress-puberty association varies according to the type of risk. Third,

we estimate the relation of these different stressors with age at menarche. This allows for a cross-validation of findings with a different measure of pubertal timing.

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. Recruitment occurred on a rolling basis for 4 years, and ceased when enrollment reached 100 participants. 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 ($n = 52$; Chamberlain, Leve, & Smith, 2006). 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.

The girls were, on average, 11.54 years of age ($SD = .48$, range = 10.44 – 12.92) at the start of the study. The ethnic distribution was 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 at 6 months (T2), 12 months (T3), and 24 months (T4) post-baseline. 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 hours and were aimed at measuring child and family characteristics, child behaviors, and parenting practices. Retention rates across the two-year study period were high (range: 86–93%).

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., inadequate food or living conditions). Coders examined child welfare case records¹ 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. 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 were acceptable (physical abuse = 86%, sexual abuse =

¹Case files included all information on incidents of child maltreatment and family history available to child welfare at the time of the study.

86%, neglect = 82%). In addition, coders attained high levels of agreement (81%) about the total number of incidents per case.

For each girl, we calculated 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 0 to 6, 0 to 7, and 0 to 13 times for physical abuse, sexual abuse, and neglect, respectively. Descriptive information and correlations among all study variables are provided in Table 1.

Foster care history—Age at first foster care placement and total number of foster care placements were drawn from child welfare case records. Descriptive statistics for these variables are included in sample demographic information, above.

Pubertal development—At all four time points, girls and their caregivers completed a slightly abbreviated version of the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988), comprising three 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*). In this sample, 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 measures demonstrate significant stability across time (r s ranged .53 – .58, $p < .001$). For the present analyses, a pubertal development score was created by taking the mean of reports of body hair changes, skin changes, and breast growth from both girls and their caregivers at each wave.

In addition, at each assessment, each girl was asked if she had begun menstruating and, if so, her age in years and months at first menstruation. The percentage of girls who had reached menarche was 25% (T1), 44% (T2), 61% (T3), and 74% (T4). A T5 follow-up telephone interview inquired about menarche, among other questions, for the 26 girls who were still premenarcheal at T4. In this assessment, 19 girls reported menarche between T4 and the T5 telephone follow-up and 3 girls reported not reaching menarche by T5. Four additional girls did not report menarche at T1 and did not return for any of the subsequent assessments. Of the 96 girls on whom menarche data were available, 93 had begun to menstruate and their average age of menarche was 11.95 years ($SD = .97$, Range: 9.5 to 14.0).

Other variables—T1 age was included as a covariate to control for age variation and the association between age and more advanced pubertal development. Although it seemed unlikely that intervention condition might influence development given that adrenarche typically begins well before baseline in our study, intervention condition was controlled for in analyses.

Analytical Strategies

Study Aim 1: What is the timing and tempo of pubertal development in the sample?—We examined the trajectory of the timing and tempo of pubertal development using latent growth modeling (LGM; Meredith & Tisak, 1990). Analyzing longitudinal data using LGM allows one to characterize both intra-individual and inter-individual differences in change (McArdle & Nesselroade, 2002). LGM is especially useful when all individuals

are expected to follow the same general path, as would be expected in studies of pubertal development. The unconditional model is represented in the left-hand half of Figure 1, with PDS scores at each time point regressed onto a latent factor representing initial level of development (i.e., the latent intercept) and a latent factor representing change in the level of development over the four time points (i.e., the latent slope). The latent intercept provides a measure of pubertal timing and the slope provides a measure of the rate of pubertal development (aka, pubertal tempo). The uneven spacing of the assessments was controlled by declaring the paths between the latent slope and the developmental measures to be 0 (T1), .5 (T2), 1 (T3), and 2 (T4).

We fit the LGMs in MPlus (Muthén & Muthén, 1998–2009). The models were fit using Maximum Likelihood (ML) estimation, which can provide unbiased estimates in the presence of missing data. For each model, standard measures of fit are reported, including the chi-square value (χ^2), the comparative fit index (CFI), the non-normed or Tucker-Lewis index (TLI), and the root-mean squared error of approximation (RMSEA). Typically, CFI and TLI values greater than .95, RMSEA values less than 0.5, and a non-significant χ^2 indicate adequate fit (Bentler, 1990; Bentler & Bonett, 1980; Bollen, 1989; Hu & Bentler, 1999).

Study Aim 2: How do early childhood stressors predict the timing and tempo of development?—We next expanded the unconditional latent growth model to test the relation between pubertal development and early childhood experiences by incorporating the following variables: age of first foster care placement; number of foster care placements; and number of neglect incidents, physical abuse incidents, and sexual abuse incidents per girl (e.g., the conditional model; see Figure 1). This model allows for (1) the estimation of the effects of each type of early childhood stressor on the initial level of development, and (2) the estimation of the effects of each type of early childhood stressor on the linear rate of change in development across the age range of our study. Predictors were entered into latent growth models individually using a stepwise approach in the following order: age and intervention condition entered together (as both are considered covariates); age of first foster care placement; number of foster care placements; number of incidents of neglect; number of incidents of physical abuse; number of incidents of sexual abuse. At each step, a chi-square difference test was conducted to determine whether the predictor added significantly to the model fit as compared to the previous step. The stepwise approach was implemented by initially including all paths in the model but constraining them to be zero. At each step, one path was freed, and the chi-square difference test corresponded to the freed path.

Study Aim 3: How do early childhood stressors predict age at menarche?—To cross-validate findings with a different measure of pubertal timing, we examined the relation between age of menarche and the same early childhood stressors described above. As not all participants had reached menarche, we employed continuous-time survival analysis using Cox regression. Cox regression is a method for investigating the effect of several variables upon the amount of time before the occurrence of a specific event (here, menarche). Cox regression considers both time-to-event (e.g., number of years of age at menarche) and a dichotomous indicator of whether that event occurred during the duration of the study (if not, the data can be said to be *right censored* in that the actual time of the event is not known). The results of Cox regression can be interpreted in terms of the cumulative hazard function, representing the risk that a particular event will occur at time $t + 1$, assuming that event has not yet occurred by time t . Smaller estimates of the hazard function indicate less risk or likelihood of the event occurring, whereas larger estimates indicate greater risk. We first estimated the baseline hazard function, which is a representation of the likelihood of menarche across a given age range without consideration of any covariates. Then, in the next step, we entered covariates into the model. As in Aim 2, predictors were entered in a

step-wise fashion and difference tests were conducted at each step (in this case, the tests were done using log-likelihood). One participant lacked data on early life experiences and was thus unable to be included in survival analyses (due to different techniques for handling missing data in LGM, this participant was included in those analyses).

Results

Study Aim 1: Latent Growth Models of Pubertal Development

Results from the unconditional latent growth model of pubertal development are shown in Table 2. The mean growth curve contained a significant intercept [$B = 2.45$, $SE(B) = .06$, $p < .001$] and, as would be expected, a significant positive linear slope [$B = .26$, $SE(B) = .03$, $p < .001$], indicating greater pubertal development at subsequent waves. The intercept and slope were inversely correlated with each other ($r = -.43$, $SE = .15$, $p = .004$). This negative correlation likely reflects a ceiling effect: children who were already at an advanced stage of development at the beginning of the study would have both a higher latent intercept and, because they had less pubertal development remaining to complete, a lower latent slope.

The model fit the data well [$\chi^2(5) = .6830$ *ns*, CFI = .990, TLI = .988, RMSEA = .06(.00|.16)], suggesting that change in the outcome across our four time points tended to proceed in a linear fashion. To confirm this, we tested a quadratic term and it was not significant [$B = -.06$, $SE(B) = .04$ *ns*]. In addition, the variance estimate for the intercept was significant ($Var_{int} = .24$, $SE = .05$, $p < .001$) and the variance estimate for the slope was marginally significant ($Var_{slope} = .03$, $SE = .01$, $p = .08$). This marginal significance for the slope variance may be related to a power issue due to our relatively small sample size. As there has been longstanding interest within the puberty literature for studies which consider aspects of pubertal development other than timing (e.g., Brooks-Gunn, Petersen, and Eichorn, 1985), we opted to investigate predictors of the slope in an exploratory fashion, with the understanding that any findings related to the slope would be provisional and should be interpreted with caution.

Study Aim 2: Latent Growth Models of Pubertal Development and Early Childhood Stressors

We next examined the impact of different early life stressors on pubertal development by regressing the intercept and linear slope onto these experiences (see Table 3). As discussed above, a step-wise approach was used. The chi-square difference test between steps was non-significant until physical abuse was entered; this step was marginally significant ($p < .10$). When sexual abuse was added, this step was significant ($p < .05$). Results from the full series of models are available on request; for clarity, we report only results for the final model (see Table 2).

Model fit for the model with all stressors included was quite good [$\chi^2(19) = 22.263$, *ns*, CFI = .98, TLI = .97, RMSEA = .04(.00|.10)]. Age and sexual abuse predicted a higher intercept (i.e., more advanced pubertal development at T1; $B = .10$; $SE = .04$ for sexual abuse). Physical abuse predicted a steeper linear slope ($B = .06$; $SE = .02$), although these results should be interpreted with caution given the small amount of variance in our slope parameter. Sexual abuse, in contrast, predicted a more gradual slope ($B = -.04$; $SE = .02$). This is likely because girls who experienced sexual abuse were already at a more advanced stage of development at T1 and therefore had less development to complete (see Table 2). Neglect, age of first placement, and number of foster care placements were not significantly related either to level of pubertal development or to the rate of developmental change in puberty across the study period. This model accounted for 27% of the variance in the growth curve intercept and 50% of the variance in the growth curve slope. Figure 2 provides a

prototypical plot depicting the trajectory of development for children who experienced each type of maltreatment.

Study Aim 3: Age at Menarche and Early Childhood Stressors

We next examined the relation of early childhood stressors and age at menarche. As not all participants in our sample reported menarche, we employed continuous-time survival analysis using Cox regression. The baseline hazard function for the sample can be seen in Figure 3a. Next, we entered predictors in the same stepwise progression as described above. No steps were significant until sexual abuse was entered into the model (the final step). In the final model, the number of incidents of sexual abuse experienced by a participant significantly increased the likelihood of menarche. This effect is illustrated in Figure 3a, which presents two cumulative hazard functions. The baseline hazard function represents the likelihood of menarche over time, without the inclusion of predictors. Because no predictors are entered into the model, it can be interpreted as the hazard function for those individuals who experience no incidents of sexual abuse. In contrast, the sexual abuse hazard function depicts the effect for individuals who experienced a number of incidents of abuse 1 or more standard deviations above the mean (e.g., 3 or more incidents). The Y-axis represents the amount of risk (or likelihood) of menarche occurring, with higher numbers representing more risk (or greater likelihood). Given that all girls will eventually experience menarche, the “risk” of menarche unsurprisingly increased for both groups as age increased; however, the cumulative hazard was greater for those girls who experienced sexual abuse compared to those who did not, indicating an increased likelihood of menarche across the age range in our study for those participants.

Figure 3b presents the survival function for the baseline model and for those individuals with a high frequency of sexual abuse. The survival function is based upon the hazard function and represents the estimated proportion of individuals which have not yet reached menarche at a given age. As can be seen in Figure 3b, of those individuals who experienced a high frequency of sexual abuse, 50% are predicted to reach menarche at approximately 11.50 years of age. For individuals with no incidents of sexual abuse, the predicted median is approximately 12.5 years of age. These predicted estimates are similar to the medians in the actual data (11.63 for girls with a high frequency of abuse and 12.42 for girls with no abuse), but the survival function accounts for the small amount of censoring among participants.

Additional Analyses

Lastly, given the prevalence of multiple types of maltreatment in our sample, we examined our data for effects of comorbidity by inserting interaction terms into all of the above analyses. In each 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 kinds 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

Researchers for some time have speculated on puberty’s malleability in response to particular life experiences and social environments. The present study represents a novel addition to the literature on childhood stress and maturation, as it is the first report on the timing and antecedents of pubertal development among girls in foster care. This sample is notable for its particularly high levels of child maltreatment and subsequent home disruptions. It is further advantageous in that the data are prospective, utilizing confirmed

reports of maltreatment from case files provided by the child welfare system, rather than retrospective self-reports. This allowed us to distinguish among the multiple types of maltreatment, and to distill both unique and cumulative effects.

Collectively, our results confirm previously-reported associations between maltreatment early in life and accelerated pubertal maturation. Physical and sexual abuse each significantly predicted development, with those who experienced childhood sexual abuse exhibiting the earliest timing of development and those who experienced physical abuse exhibiting a more rapid developmental tempo across the duration of the study. The sexual abuse effect robustly replicated across both the PDS measure and age of menarche. Although the majority of our sample experienced multiple types of maltreatment, we obtained no noticeable interactive effects when we attempted to investigate the co-occurrence of maltreatment types. In cases of multiple forms of maltreatment, this might manifest as the powerful effects of sexual abuse “trumping” the particular trajectories related to other subtypes. It may also be the case that only sexual abuse precipitates the sorts of HPA and HPG activation hypothesized by Trickett and Putnam (1993), which consequently facilitates premature adrenarche and gonadarche.

In addition to maltreatment type, we included age of first foster care placement and the number of foster care placements in our analyses. Previous research on early childhood stress has shown that age at certain stressful life transitions represents a dose-response relationship with maturation, with earlier ages at these events associated with earlier development (e.g., Ellis & Garber, 2000). It also seemed possible that more frequent foster home transitions might represent greater instability in daily living and thus a more stressful childhood experience. Neither of these factors reached significance in the present study. This might be attributable to the fact that *all* the girls in our sample experienced unusually high levels of adversity; such factors may hold more resonance within normative populations with fewer risk factors. Quinlan (2003), for example, found that the number of childhood caretaking transitions was associated with earlier menarche. Yet only 8% of Quinlan’s sample had three primary caregivers, whereas 80% of our sample had three or more caregivers. Alternatively, these variables may exert different stress loads over different individuals (i.e., entry into the foster care system may represent an alleviation of stress for some but require considerable adaptation for others). Similarly, an earlier *discovery* of maltreatment (preceding entry into foster care) does not necessarily indicate earlier *onset* of maltreatment.

Limitations

Our findings are limited in several respects. First, the small sample size constrained the number of theoretical questions we were able to investigate. In addition, the variance in our slope term, perhaps due to a lack of power, was small and only marginally significant. Second, while early life stress has a robust and well-documented literature as an antecedent of pubertal development, the timing of maturation is also strongly heritable. For example, Mustanski et al. (2004) estimated that an astounding 88% of the variation in the characteristics measured by the PDS (the same measure employed here) was accounted for by genetic factors. In addition, genetically informed analyses of the timing of maturation and more benign measures of early life stress have suggested that this association might be accounted for by passive gene–environment correlation (Mendle et al., 2009). Due to the nature of our sample (girls in foster care), we lacked familial information that might have enabled us to control for genetic predispositions for pubertal timing and tempo, and therefore present a more multi-faceted set of interpretations. Nevertheless, though not a perfect proxy, 33% of the girls resided in kinship foster care placements, and 67% in nonrelative foster care placements. No significant differences were noted in the outcomes examined as a function of kinship status.

Third, as our data are drawn from a behavioral intervention trial targeted at the foster care population, we lack the traditional control of girls not residing in foster care. There is, however, an expansive literature on menarche in the general population. This enables a speculative comparison of the timing of puberty in our sample to prior results. Overall, the mean age of menarche of the girls in our study ($M=11.95$ years, with 93% reporting), is significantly earlier ($t = -4.37, p < .001$) than recent U.S. population estimates of approximately 12.4 years (Chumlea et al., 2003), though this should be interpreted with caution given that not everyone in our sample reported menarche and the magnitude of this effect will be somewhat attenuated when full menarche data are available. Although this time difference -- approximately 6 months -- may superficially seem small, it is worth remembering that pubertal maturation occurs during a strikingly concentrated and emotionally tumultuous period; differences of this magnitude hold considerable social salience during adolescence and also differentiate among fertility and health outcomes later in life (Apter & Vihko, 1983).

Conclusion

This work proffers fresh evidence for the ways particular social contexts may relate to the trajectory of pubertal development. Puberty is, of course, one segment of a larger developmental continuum. The fluctuations in pubertal development observed in our sample are not only sequelae of early maltreatment, but likely precede (and perhaps moderate) many of the behavioral and emotional difficulties common in individuals with histories of maltreatment and trauma. As such, upon replication, our findings might suggest critical opportunities for calibrating interventions to the unique developmental challenges of the foster care population.

Acknowledgments

Support for this research was provided by grant R01 MH054257, NIMH, U.S. PHS. Additional support for the writing of this report was provided by the following grants: R01 DA024672 and P30 DA023920, NIDA, U.S. PHS. We are grateful to the foster families who generously volunteered to participate in this study and to the Oregon Department of Human Services for their ongoing assistance with this work. We thank the late Xiaojia Ge, formerly of the Institute of Child Development, University of Minnesota, for his contributions to this manuscript. In addition, we thank Heather Wilber, Courtenay Paulic, and Michelle Baumann for assistance with technical aspects of this project.

References

- Apter D, Vihko R. Early menarche, a risk factor for breast cancer, indicates early onset of ovulatory cycles. *Journal of Clinical Endocrinology and Metabolism*. 1983; 57:82–86. [PubMed: 6222061]
- Barnett, D.; Manly, JT.; Cicchetti, D. Defining child maltreatment: The interface between policy and research. In: Cicchetti, D.; Toth, SL., editors. *Child abuse, child development, and social policy*. Vol. 8. Norwood, NJ: Ablex; 1993. p. 7-73.
- Belsky J, Steinberg L, Draper P. Childhood experience, interpersonal development and reproductive strategy: An evolutionary theory of socialization. *Child Development*. 1991; 62:647–670. [PubMed: 1935336]
- Bentler PM. Comparative fit indexes in structural models. *Psychological Bulletin*. 1990; 107:238–246. [PubMed: 2320703]
- Bentler PM, Bonett DG. Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin*. 1980; 88:588–606.
- Bollen, KA. *Structural equations with latent variables*. New York: Wiley; 1989.
- Bergevin, TA.; Bukowski, WM.; Karavasilis, L. Child sexual abuse and pubertal timing: implications for long-term psychosocial adjustment. In: Hayward, C., editor. *Gender Differences at Puberty*. Cambridge, England: Cambridge University Press; 2003. p. 187-216.

- Chamberlain P, Leve LD, Smith DK. Preventing behavior problems and health-risking behaviors in girls in foster care. *International Journal of Behavioral and Consultation Therapy*. 2006; 4:518–530. [PubMed: 18176629]
- Chumlea WC, Schubert CM, Roche AF, Kulin HE, Lee PA, Himes JH, Sun SS. Age at menarche and racial comparisons in the U.S. *Pediatrics*. 2003; 111:110–113. [PubMed: 12509562]
- Costenbader KH, Feskanich D, Stampfer MJ, Karlson EW. Reproductive and menopausal factors and risk of systemic lupus erythematosus in women. *Arthritis & Rheumatism*. 2007; 56:1251–1262. [PubMed: 17393454]
- Ellis BJ. Timing of pubertal maturation in girls: an integrated life history approach. *Psychological Bulletin*. 2004; 130:920–958. [PubMed: 15535743]
- Ellis BJ, Garber J. Psychosocial antecedents of variation in girls' pubertal timing: maternal depression, stepfather presence, and marital and family stress. *Child Development*. 2000; 71:485–501. [PubMed: 10834479]
- Foster H, Hagan J, Brooks-Gunn J. Growing up fast: stress exposure and subjective weathering in emerging adulthood. *Journal of Health and Social Behavior*. 2008; 49:162–177. [PubMed: 18649500]
- Geronimus, AT. The weathering hypothesis and the health of African American women and infants: Implications for reproductive strategies and policy analysis. In: Sen, G.; Snow, RC., editors. *Power and decision: The social control of reproduction*. Cambridge, MA: Harvard University Press; 1994. p. 77-100.
- Heim C, Nemeroff CB. The role of childhood trauma in the neurobiology of mood and anxiety disorders: Preclinical and clinical studies. *Biological Psychiatry*. 2001; 49:1023–1039. [PubMed: 11430844]
- Heim C, Newport DJ, Heit S, Graham YP, Wilcox M, Bonsall R, et al. Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association*. 2000; 284:592–597. [PubMed: 10918705]
- Hu L, Bentler PM. Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*. 1999; 6:1–55.
- Laitinen J, Power C, Jarvelin MR. Family social class, maternal body mass index, childhood body mass index, and age at menarche as predictors of adult obesity. *American Journal of Clinical Nutrition*. 2001; 74:287–294. [PubMed: 11522550]
- Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in girls. *Archives of the Diseases in Childhood*. 1969; 44:291–303.
- McArdle, JJ.; Nesselrode, JR. Growth curve analysis in contemporary psychological research. In: Schinka, J.; Velicer, W., editors. *Comprehensive handbook of psychology, Volume two: Research methods in psychology*. New York: Wiley; 2002. p. 447-480.
- Mendle J, Harden KP, Turkheimer E, van Hulle C, D'Onofrio BM, Brooks-Gunn J, et al. Associations between father absence and age of first sexual intercourse. *Child Development*. 2009; 80:1463–1480. [PubMed: 19765012]
- Mendle J, Turkheimer E, Emery RE. Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review*. 2007; 27:151–171. [PubMed: 20740062]
- Meredith W, Tisak J. Latent curve analysis. *Psychometrika*. 1990; 55:107–122.
- Mustanski B, Viken RJ, Kaprio J, Pulkkinen L, Rose RJ. Genetic and environmental influences on pubertal development: Longitudinal data from 12–14 year-old twins. *Developmental Psychology*. 2004; 40:1188–1198. [PubMed: 15535766]
- Muthén, LK.; Muthén, BO. *Mplus user's guide*. Los Angeles, CA: Muthén & Muthén; 1998–2009.
- Olsson IAS, deJonge FH, Schuurman T, Helmond FA. Poor rearing conditions and social stress in pigs: repeated social challenges and the effect on behavioral and physiological responses to stressors. *Behavioural Processes*. 1999; 46:201–215.
- Petersen AC, Crockett LJ, Richards M, Boxer A. A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*. 1988; 17:117–133.
- Putnam FW, Trickett PK. The psychobiological effects of sexual abuse: A longitudinal study. *Annals of the New York Academy Science*. 1997; 821:150–159.

- Quinlan RJ. Father absence, parental care, and female reproductive development. *Evolution and Human Behavior*. 2003; 24:376–390.
- Romans SE, Martin M, Gendall K, Herbison GP. Age of menarche: The role of some psychosocial factors. *Psychological Medicine*. 2003; 33:933–939. [PubMed: 12877408]
- Surbey, MK. Family composition, stress, and the timing of human menarche. In: Bercovitch, FB.; Ziegler, TE., editors. *Socioendocrinology of primate reproduction*. New York: Wiley-Liss; 1990. p. 11-32.
- Surbey MK. Parent and offspring strategies in the transition at adolescence. *Human Nature*. 1998; 9:67–94.
- Trickett PK, Putnam FW. Impact of child sexual abuse on females: Toward a developmental, psychobiological integration. *Psychological Science*. 1993; 4:81–87.
- Turner PK, Runtz MG, Galambos NL. Sexual abuse, pubertal timing, and subjective age in adolescent girls : a research note. *Journal of Reproductive and Infant Psychology*. 1999; 17:111–118.
- Wise LA, Palmer JR, Rothman EF, Rosenberg L. Childhood abuse and early menarche: findings from the Black Women’s Health Study. *American Journal of Public Health*. 2009; 99:1–7.

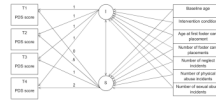


Figure 1. Latent growth curve model of pubertal development and early life stressors. *Note.* PDS = Pubertal Development Scale; T1 = baseline; T2 = 6 months postbaseline; T3 = 12 months postbaseline; T4 = 24 months postbaseline; I = intercept; S = slope

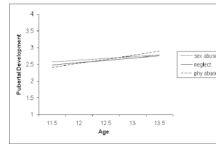


Figure 2.
Prototypical plot of pubertal development by maltreatment category.

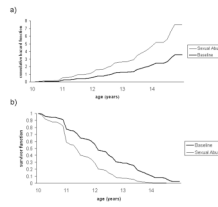


Figure 3. Cumulative hazard and survival functions of age of menarche for individuals who experience either no sexual abuse (e.g., baseline model) or frequency incidents of sexual abuse 1 or more standard deviations above the mean.

Table 1

Correlations and Sample Descriptives

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|---------------------------------------|--------|---------|---------|--------|-------|--------|-------|---------|---------|--------|--------|-------|
| 1. T1 age | - | | | | | | | | | | | |
| 2. Intervention condition | -.11 | - | | | | | | | | | | |
| 3. Age at first foster care placement | .11 | -.12 | - | | | | | | | | | |
| 4. Number of foster care placements | -.09 | .02 | -.49*** | - | | | | | | | | |
| 5. Number of neglect incidents | -.05 | -.34*** | -.03 | .07 | - | | | | | | | |
| 6. Number of physical abuse incidents | -.01 | -.03 | -.28** | .21* | .13 | - | | | | | | |
| 7. Number of sexual abuse incidents | .03 | .04 | -.27** | .34*** | .05 | .35*** | - | | | | | |
| 8. T1 PDS | .22* | -.08 | -.06 | .09 | -.12 | -.08 | .20* | - | | | | |
| 9. T2 PDS | .23* | -.02 | -.09 | .10 | .01 | -.07 | .12 | .55*** | - | | | |
| 10. T3 PDS | .36*** | .01 | -.05 | .01 | -.04 | .17 | .16 | .54*** | .58*** | - | | |
| 11. T4 PDS | .30** | .09 | .06 | -.01 | -.24* | .05 | .04 | .53*** | .58*** | .57*** | - | |
| 12. Age of menarche | .04 | .03 | .00 | -.05 | .12 | .04 | -.24* | -.37*** | -.45*** | .33** | -.29** | - |
| <i>N</i> | 100 | 100 | 99 | 99 | 100 | 100 | 100 | 97 | 93 | 96 | 86 | 90 |
| <i>M</i> | 11.54 | - | 7.63 | 3.90 | 2.15 | 1.06 | 1.42 | 2.37 | 2.48 | 2.71 | 2.95 | 11.95 |
| <i>SD</i> | 0.48 | - | 3.14 | 3.03 | 2.24 | 1.32 | 1.48 | 0.69 | 0.64 | 0.69 | 0.59 | 0.97 |

Note. T1 = baseline; T2 = 6 months postbaseline; T3 = 12 months postbaseline; T4 = 24 months postbaseline; PDS = Pubertal Development Scale.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 2

Latent Growth Models

| Estimates | Unconditional Model | Conditional Model |
|------------------------------------|---------------------|-------------------|
| <i>Growth factors</i> | | |
| Mean Intercept | 2.45 (.06) | - |
| Mean Slope | .26 (.03) | - |
| Var Intercept | .24 (.05) | .17 (.04) |
| Var Slope | .03 (.01) | .01 (.01) |
| R ² intercept | | .27 |
| R ² slope | | .50 |
| <i>Regression effects</i> | | |
| Age | | |
| Intercept | | .38 (.11)* |
| Slope | | .00 (.05) |
| Intervention condition | | |
| Intercept | | -.07 (.11) |
| Slope | | .07 (.05) |
| Age at first placement | | |
| Intercept | | -.03 (.02) |
| Slope | | .01 (.01) |
| Number of foster placements | | |
| Intercept | | .01 (.02) |
| Slope | | -.01 (.01) |
| Number of neglect incidents | | |
| Intercept | | -.02 (.03) |
| Slope | | .00 (.01) |
| Number of physical abuse incidents | | |
| Intercept | | -.07 (.04) |
| Slope | | .06 (.02)* |
| Number of sexual abuse incidents | | |
| Intercept | | .10 (.04)* |
| Slope | | -.04 (.02)* |
| <i>Model Fit Indices</i> | | |
| Chi-squared (df) | 6.83 (5) | 22.26 (19) |
| CFI | .99 | .98 |
| TLI | .99 | .97 |
| RMSEA | .06 (.00 .16) | .04 (.00 .10) |

Note: Standard errors are included parenthetically

* Regression parameters are significant at $p < .05$

Table 3Fit of Survival Models using Cox Regression ($N = 99$)

| Model | AIC | Deviance | DF | Δ deviance (df) |
|------------------------------------|--------|----------|----|------------------------|
| Baseline model | 207.94 | -103.97 | | |
| Baseline age | 208.70 | -103.35 | 1 | .62 |
| Intervention condition | 210.66 | -103.33 | 2 | .02 |
| Age at first placement | 212.38 | -103.19 | 3 | .14 |
| Number of placements | 214.27 | -103.14 | 4 | .05 |
| Number of neglect incidents | 215.95 | -102.98 | 5 | .16 |
| Number of physical abuse incidents | 217.95 | -102.97 | 6 | .01 |
| Number of sexual abuse incidents | 212.11 | -99.06 | 7 | 3.91* |

Note: Predictor significant at $p < .05$