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Psychological Science 2010 21: 1195 originally published online 16 August 2010
DOI: 10.1177/0956797610379867

The online version of this article can be found at:
http://pss.sagepub.com/content/21/9/1195
Infant Attachment Security and the Timing of Puberty: Testing an Evolutionary Hypothesis

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Abstract

Life-history theories of the early programming of human reproductive strategy stipulate that early rearing experience, including that reflected in infant-parent attachment security, regulates psychological, behavioral, and reproductive development. We tested the hypothesis that infant attachment insecurity, compared with infant attachment security, at the age of 15 months predicts earlier pubertal maturation. Focusing on 373 White females enrolled in the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development, we gathered data from annual physical exams from the ages of 9½ years to 15½ years and from self-reported age of menarche. Results revealed that individuals who had been insecure infants initiated and completed pubertal development earlier and had an earlier age of menarche compared with individuals who had been secure infants, even after accounting for age of menarche in the infants’ mothers. These results support a conditional-adaptational view of individual differences in attachment security and raise questions about the biological mechanisms responsible for the attachment effects we discerned.

Keywords

attachment, puberty, reproductive strategy, menarche, evolution

Received 12/21/09; Revision accepted 3/10/10

Puberty defines reproductive maturity and is a central event in human development. The fact that individual differences in pubertal timing are heritable does not preclude the possibility that developmental experiences also influence this life-history characteristic (Ellis, 2004). Indeed, research on rodents shows that the early rearing environment regulates pubertal, sexual, and reproductive development through epigenetic processes (Cameron, Del Corpo, et al., 2008; Cameron et al., 2005; Cameron, Fish, & Meaney, 2008; Champagne et al., 2006). Here, we tested the evolutionary-developmental hypothesis that the security of infant-mother attachment predicts and perhaps programs pubertal development in human females.

Although evolutionary theory figured centrally in the development of attachment theory (Bowlby, 1982), most attachment research over the past 40 years has neglected evolutionary theory and has been guided instead by mental-health thinking. Thus, attachment security is widely considered to reflect optimal development by fostering empathy, prosocial behavior, self-regulation, and the establishment of close, trusting interpersonal relationships. Building on evolutionary critiques of such mental-health thinking about attachment (Hinde & Stevenson-Hinde, 1990; Lamb, Thompson, Gardner, Charnov, & Estes, 1984), Belsky, Steinberg, and Draper (1991; Belsky, 1997) advanced a life-history theory of socialization by recasting traditional developmental thinking, including attachment theory, in conditional-adaptational, reproductive-strategy terms.

Belsky et al. (1991; Belsky, 1997) argued that the early family environment, including the infant-parent attachment relationship, conveys to children the risks and uncertainties they are likely to encounter in their lifetimes. Such information adaptively regulates psychological, behavioral, and reproductive development, either toward a mutually beneficial orientation to interpersonal relations or toward an opportunistic, advantage-taking point of view. These orientations affect mating behavior, pair bonding, and parental investment; they are also responsible for earlier or later sexual debut, unstable or stable relationships with intimate partners, and a quantity or...
quality approach to children and parenting. It is theorized that these divergent developmental trajectories fit the organism to the environment in ways that enhance reproductive success—or at least did so in the environments of evolutionary adaptation.

The fact that many of these reproductive-strategy-oriented predictions could be derived from traditional, nonevolutionary accounts of human development (e.g., attachment, social learning, and life-course theories) made an additional, “uncanny” prediction by Belsky et al. (1991; Belsky, 1997) critically important: that early family experiences, including those reflected in the security of the infant-parent attachment relationship, would influence and thus predict faster or slower development and thus, respectively, earlier or later pubertal onset. This prediction distinguished the account of Belsky et al. from all prevailing theories of development. In essence, Belsky et al. predicted that insecure and unsupportive family relationships would accelerate pubertal development, and, in particular, females with such relationships would be enabled to initiate mating and reproduction earlier than females in secure and supportive family relationships. This effect would be advantageous in environments in which survival and thereby reproduction could be compromised. The opposite effect would occur in secure and supportive family relationships: Pubertal development would decelerate, and females with such relationships would be able to delay reproduction and mating. This effect would be advantageous in environments in which survival is less difficult.

A comprehensive review of older studies on the determinants of female pubertal development provides qualified support for this pubertal-timing prediction (Ellis, 2004); more recent research provides similar support (Belsky et al., 2007; Ellis & Essex, 2007; Tither & Ellis, 2008). Nevertheless, questions remain. Most notably, researchers have asked whether the apparent effects of family relationship processes on pubertal development are genetically mediated (Comings, Muhleman, Johnson, & MacMurray, 2002; Rowe, 2002) and whether early attachment security itself predicts pubertal timing.

Although studies on rodents document the programming of reproductive strategy by maternal behavior (e.g., licking and grooming) extremely early in life (Cameron et al., 2005), all work on humans linking rearing experiences to pubertal timing relies on measurements made substantially after infancy. We tested the proposition that females with insecure infant-mother attachment histories mature earlier than females with secure infant-mother attachment histories. We took into account the mother’s age of menarche in an attempt to discount the well-established heritability of pubertal timing as an explanation of this effect (Ellis, 2004). Because attachment security reflects the influence of distal contextual factors (e.g., socioeconomic status) and proximate contextual factors (e.g., quality of maternal care; Belsky & Fearon, 2008), these factors were neither conceptualized as alternative explanations nor statistically controlled in this inquiry. Such influences on attachment and, thereby perhaps, pubertal development should not be regarded as third variables that need to be accounted for before evaluating attachment effects.

Method

Participants

To test the evolutionary hypothesis under consideration, we drew on data from 373 White females enrolled in the multisite National Institute of Child Health and Human Development (NICHD) Study of Early Child and Youth Development (NICHD Early Child Care Research Network, ECCRN, 2005). These women had participated in the study’s various assessments of pubertal status. Minority children were excluded from the current study because the small number of them precluded hypothesis testing within these subsamples; males were excluded from this study because evidence indicates that family experience does not regulate their pubertal development (Belsky et al., 2007).

On average, analysis-sample mothers were 29.6 years of age at study enrollment, had completed 14.8 years of education, and were living with a male partner or male spouse on 90.7% of measurement occasions across the child’s first 8 years of life. Family income-to-needs ratio, averaged across the time period of the study, was higher than the U.S.-government-determined poverty line by a factor of 4.23. The 373 White females included in the analysis and 129 White females who were excluded because they had neither pubertal-onset nor pubertal-completion data did not differ significantly in mother’s age or education, family income-to-needs ratio, or, most critically, proportion with secure (or insecure) attachments.

Procedure and measures

Children were followed from birth to 15 years of age. Complete details about all procedures and measures are documented on the Web site of the NICHD (2009) Study of Early Child Care and Youth Development.

Control variable: maternal age of menarche. Each mother’s report of her own age of menarche was used to partially control for genetic effects on her children’s timing of puberty.

Primary predictor: infant-mother attachment security. Infant-mother attachments in our sample were assessed as secure or insecure using the Strange Situation procedure. At 15 months of age, infants were videotaped in the Strange Situation, a separation-reunion procedure designed to evoke attachment behavior (Ainsworth, Blehar, Waters, & Wall, 1978). When stressed in this procedure, secure infants establish unambiguous psychological contact with their mothers on reunion. This contact occurs either across a distance (e.g., smiling, vocalizing) or physically (e.g., approaching, reaching). Although secure infants find comfort and solace in their mothers’ arms if distressed, insecure infants avoid such
psychological and physical contact, physically resist contact, or combine approach behavior with avoidance behavior. All videotapes were double-scored by highly reliable coders unaware of the children’s rearing experiences (NICHD ECCRN, 1997).

**Primary outcomes: onset and completion of puberty and age of menarche in children.** Pubertal development was assessed via annual physical exams using Tanner criteria (Marshall & Tanner, 1969, 1970) and by following instructions from the American Academy of Pediatrics manual Assessment of Sexual Maturity Stages in Girls (Herman-Giddens & Bourdony, 1995). These measures were augmented with breast-bud palpation when girls averaged the following ages: 9.56 years (SD = 0.13), 10.60 years (SD = 0.16), 11.57 years (SD = 0.14), 12.57 years (SD = 0.14), 13.55 years (SD = 0.12), 14.57 years (SD = 0.15), and 15.55 years (SD = 0.14).

Exams were conducted by trained nurses and physicians blind to attachment history until girls reported their first men- struation and reached Tanner Stage (TS) 5 on the clinical ratings of breast and pubic-hair development (see Belsky et al., 2007, for additional details). These assessments yielded separate TS scores from 1 to 5 each year for breast development and for pubic-hair development. To estimate pubertal onset, we recoded TS scores to indicate, at each age of measurement, whether the child showed any evidence of pubertal breast or pubic-hair development (TS > 1 vs. TS = 1); to estimate pubertal completion, we recoded these factors to indicate whether the child had completed pubertal breast or pubic-hair development (TS = 5 vs. TS < 5).

**Results**

To first estimate pubertal onset and pubertal completion, we used latent transition analysis (LTA; Lanza & Collins, 2008; Muthén & Muthén, 2006), a longitudinal extension of latent class analysis, to identify underlying subgroups in the population while allowing individual membership in identified classes (e.g., no pubertal development, some pubertal development) to change over time. LTA handles multiple indicators (e.g., breast development, pubic-hair development) to define class membership (e.g., pubertal onset, no pubertal onset) and can estimate group membership for cases with missing data. LTAs, run using Mplus (Version 5.2: Muthén & Muthén, 2006), use maximum-likelihood estimation under the assumption of data missing at random. Pubertal onset and pubertal completion were estimated for 327 of the girls (87.7%) in the analysis sample, pubertal onset only was estimated for 43 girls (11.5%), and pubertal completion only was estimated for 3 girls (0.8%).

Girls with onset-only data were more likely to experience early puberty (79%) than were girls with both onset data and completion data (55%). Nonetheless, girls missing onset data or completion data did not differ significantly from girls with complete data on mother’s age or education and family income-to-needs ratio, or in proportion of girls with secure (or insecure) attachments. Because analysis of the combination of pubertal onset and pubertal completion required both pieces of information, those results may underrepresent girls who started puberty early. Evidence of the validity of pubertal-onset estimates and pubertal-completion estimates comes from data showing that taller and heavier girls initiated and completed puberty earlier than did smaller children (data available on request); girls who experienced menarche earlier also completed puberty earlier—onset: \( r(349) = .55, p < .0001; \) completion: \( r(320) = .45, p < .0001. \) Estimates indicated that all girls initiated pubertal development by 13½ years and that no girls had completed it by 10½ years. Table 1 shows participants’ ages at pubertal onset and pubertal completion.

### Table 1. Timing of Pubertal Onset and Pubertal Completion

<table>
<thead>
<tr>
<th>Age in years</th>
<th>n</th>
<th>%</th>
<th>Mean</th>
<th>SD</th>
<th>n</th>
<th>%</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 9.5</td>
<td>73</td>
<td>19.7</td>
<td>.91</td>
<td>.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.5–10.5</td>
<td>140</td>
<td>37.8</td>
<td>.78</td>
<td>.24</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10.5–11.5</td>
<td>97</td>
<td>26.2</td>
<td>.88</td>
<td>.15</td>
<td>4</td>
<td>1.2</td>
<td>.97</td>
<td>.07</td>
</tr>
<tr>
<td>11.5–12.5</td>
<td>56</td>
<td>15.1</td>
<td>.88</td>
<td>.18</td>
<td>34</td>
<td>10.3</td>
<td>.83</td>
<td>.23</td>
</tr>
<tr>
<td>12.5–13.5</td>
<td>4</td>
<td>1.1</td>
<td>1.00</td>
<td>.00</td>
<td>70</td>
<td>21.2</td>
<td>.88</td>
<td>.15</td>
</tr>
<tr>
<td>13.5–14.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>131</td>
<td>39.7</td>
<td>.71</td>
<td>.28</td>
</tr>
<tr>
<td>14.5–15.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>72</td>
<td>21.8</td>
<td>.90</td>
<td>.11</td>
</tr>
<tr>
<td>&gt; 15.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>19</td>
<td>5.8</td>
<td>.87</td>
<td>.14</td>
</tr>
</tbody>
</table>

Note: Probability of group membership is the probability of girls being assigned to each age group in the latent transition analysis.
To determine whether pubertal onset or pubertal completion was related to infant attachment security, we dichotomized pubertal onset and pubertal completion so that early onset was defined as occurring prior to 10½ years and early completion was defined as reaching TS 5 before 13½ years. Figure 1 presents the distribution of early and later pubertal onset and completion among girls with secure and insecure attachments. A logistic regression using attachment security (0 = insecure, 1 = secure) as the primary predictor was run without and then with maternal age of menarche controlled (see Table 2). Attachment security significantly predicted both pubertal onset and pubertal completion. Having an insecure attachment at the age of 15 months increased the odds of experiencing pubertal onset before the age of 10½ years by a factor of 1.60 (95% confidence interval, CI: 1.01–2.52), though this finding became marginally significant \( p = .06 \) when maternal age of menarche was controlled. Having an insecure attachment increased the odds of experiencing pubertal completion before the age of 13½ by a factor of 1.98 (95% CI: 1.21–3.23); this result remained virtually unchanged with maternal age of menarche controlled, odds ratio = 1.95 (95% CI: 1.19–3.20).

An ordinary least squares regression analysis confirmed the prediction that girls with insecure attachments (compared with girls with secure attachments) would experience an earlier age of menarche: Such girls reported having their first menstruation 3.4 months (95% CI: 0.8–5.9 months) earlier without maternal age of menarche controlled, \( t(474, 1) = 2.60, p = .0095 \) (secure attachments: \( M = 12.49 \) years, \( SD = 1.14 \); insecure attachments: \( M = 12.21 \) years, \( SD = 1.14 \)). With maternal age of menarche controlled, they reported having their first menstruation 2.6 months (95% CI: 0.2–5.0 months) earlier than girls with secure attachments, \( t(458, 1) = 2.10, p = .0366 \) (secure attachments: 12.48 years, 95% CI: 12.35–12.60; insecure attachments: 12.26 years, 95% CI: 12.10–12.42).

Finally, we examined the effect of attachment insecurity on the combination of early pubertal onset and early pubertal completion using multinomial logistic regression. We created a categorical variable indicating the four possible combinations of pubertal onset (early or late) and pubertal completion (early or late). This nominal variable was the dependent variable, and attachment insecurity was the independent variable; girls with late onset and late completion served as the comparison group. The effect of attachment insecurity proved significant with and without maternal age of menarche controlled (see Table 3 and Fig. 2). Having an insecure attachment at the age of 15 months increased the odds of being in the early-onset/early-completion group, relative to the late-onset/late-completion group, by a factor of 2.36 (95% CI: 1.31–4.28) without maternal age of menarche controlled and by a factor of 2.33 (95% CI: 1.27–4.28) with maternal age of menarche controlled.

**Discussion**

In this study, we tested and found support for the theory-distinguishing, developmental-evolutionary hypothesis that the quality of the early rearing environment, as reflected in the security of the infant-mother attachment relationship, predicts and perhaps programs the timing of pubertal development in humans. More specifically, we found that females with insecure attachments in infancy mature earlier than females with secure attachments; these findings emerged from both verbal

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**Fig. 1.** Percentage of children classified as secure and insecure at age 15 months whose pubertal onset and completion occurred early or later.
reports of age of menarche and annual physical assessments of breast and pubic-hair development. Experimental efforts to promote security, perhaps by enhancing maternal sensitivity, are needed to determine whether the documented attachment effects are truly causal. We did not evaluate whether attachment mediated the effect of maternal sensitivity on pubertal timing because prior NICHD studies indicated that although early sensitivity predicted attachment security (NICHD ECCRN, 1997), it did not predict pubertal timing (Belsky et al., 2007).

The apparent accelerating effect of attachment insecurity that we discerned confirms a unique prediction of an evolutionary theory of socialization (Belsky, 1997; Belsky et al., 1991) and thus necessitates a rethinking of the functional significance of the secure and insecure attachments that figure so prominently in the study of human development (Cassidy & Shaver, 2008). Rather than suggesting security as optimal and insecurity as a form of compromised development, theory and now evidence support the view that both outlooks should be considered part and parcel of nascent reproductive strategies that encompass a suite of correlated physical, psychological, and behavioral characteristics. These characteristics include social orientation, pubertal timing, sexual behavior, pair bonding, and

### Table 2. Effects of Attachment Security on Pubertal Development

<table>
<thead>
<tr>
<th>Model and predictor variable</th>
<th>Pubertal onset &lt; 10.5 years</th>
<th>Pubertal completion &lt; 13.5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age of menarche not controlled</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.16</td>
<td>-0.97***</td>
</tr>
<tr>
<td>Insecure attachment</td>
<td>0.47*</td>
<td>0.68**</td>
</tr>
<tr>
<td>Maternal age of menarche controlled</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>3.57***</td>
<td>1.96*</td>
</tr>
<tr>
<td>Maternal age of menarche</td>
<td>-0.26**</td>
<td>-0.23*</td>
</tr>
<tr>
<td>Insecure attachment</td>
<td>0.45†</td>
<td>0.67***</td>
</tr>
</tbody>
</table>

Note: Omnibus chi-square tests of the models were significant—pubertal onset with maternal age of menarche not controlled: $\chi^2(1, N = 344) = 4.15, p = .04$; pubertal completion with maternal age of menarche not controlled: $\chi^2(1, N = 307) = 7.39, p = .007$; pubertal onset with maternal age of menarche controlled: $\chi^2(2, N = 344) = 15.57, p = .0004$; pubertal completion with maternal age of menarche controlled: $\chi^2(2, N = 307) = 14.00, p = .0009$. CI = confidence interval; OR = odds ratio.

### Table 3. Effects of Attachment Insecurity on Pubertal-Onset-and-Completion Profile

<table>
<thead>
<tr>
<th>Group and predictor variable</th>
<th>Maternal age of menarche not controlled</th>
<th>Maternal age of menarche controlled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early-onset/early-completion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>-0.69***</td>
<td>4.73**</td>
</tr>
<tr>
<td>Maternal age of menarche</td>
<td></td>
<td>-0.43***</td>
</tr>
<tr>
<td>Insecure attachment</td>
<td>0.86**</td>
<td>0.85**</td>
</tr>
<tr>
<td>Early-onset/late-completion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>-0.37†</td>
<td>2.00</td>
</tr>
<tr>
<td>Maternal age of menarche</td>
<td></td>
<td>-0.18†</td>
</tr>
<tr>
<td>Insecure attachment</td>
<td>0.24</td>
<td>0.22</td>
</tr>
<tr>
<td>Late-onset/early-completion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>-2.03***</td>
<td>-3.25</td>
</tr>
<tr>
<td>Maternal age of menarche</td>
<td></td>
<td>0.09</td>
</tr>
<tr>
<td>Insecure attachment</td>
<td>0.62</td>
<td>0.63</td>
</tr>
</tbody>
</table>

Note: The table reports regression analyses comparing the three groups listed with the late-onset/late-completion group. Omnibus chi-square tests of the models were significant—with maternal age of menarche not controlled: $\chi^2(3, N = 304) = 8.77, p = .03$; with maternal age of menarche controlled: $\chi^2(6, N = 304) = 26.38, p = .0002$. CI = confidence interval; RRR = relative risk ratio.
Parenting (Simpson & Belsky, 2008). Certainly consistent with this interpretation is evidence that earlier pubertal development, found here to be related to insecure attachment in infancy, contributes to earlier sexual debut and younger age at first pregnancy (Ellis, 2004).

A developmental profile involving attachment security, prosocial orientation, delayed maturation, deferred sexual debut, stable pair bonds, and high levels of parental investment should not be considered inherently better or more natural than a contrasting profile, even if the former remains more valued in the Western world. As Cameron et al. (2005) observed, “The idea that any form of phenotypic variation in and of itself is necessarily positive or negative is an anathema to biology” (p. 846). In contexts in which risk and uncertainty are high—or are perceived to be so—early maturation, apparently stimulated by early insecurity, reduces the risk of the individual dying before procreating. In more supportive contexts, security and deferred maturation provide the individual with more time to benefit from available physical and psychological resources before reproducing; these extra benefits enhance offspring viability.

The data linking early insecurity with accelerated pubertal development raise questions about mechanisms: Through what biological processes might early attachment regulate pubertal development? Given extensive evidence that the quality of rearing influences attachment security (Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2003; De Wolff & van IJzendoorn, 1997; van IJzendoorn, Juffer, & Duyvesteyn, 1995), recent studies on rodents would seem informative (Cameron, Del Corpo, et al., 2008; Cameron, Fish, & Meaney, 2008; Champagne et al., 2006). Cross-fostering studies show that parental investment in the female rat affects offspring development through epigenetic processes involving the methylation of genes regulating the hypothalamic-pituitary-adrenal and hypothalamic-pituitary-ovarian systems. Critically, maternal licking and grooming of the newborn pup affects estrogen receptor alpha gene expression, which regulates neuroendocrine function and sexual behavior, including pubertal timing (Cameron, Fish, & Meaney 2008). With so many biological systems conserved across mammalian species, there would seem good reason to suspect that related processes operate in humans, thereby accounting for the observed effects of attachment on pubertal timing; however, this possibility remains to be tested.

**Declaration of Conflicting Interests**

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.
Funding
This article is based on a larger study, the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development, which was directed by a steering committee and supported by the Eunice Kennedy Shriver National Institute of Child Health and Human Development, the National Institutes of Health, through a set of cooperative agreements (5U10 HD027040, 5U10 HD025460, 5U10 HD025447, 5U10 HD025420, 5U10 HD025456, 5U01 HD033343, 5U10 HD025445, 5U10 HD025451, 5U10 HD025430, 5U10 HD025449, 5U10 HD027040, and 5U10 HD025455).

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