

Family Rearing Antecedents of Pubertal Timing

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Two general evolutionary hypotheses were tested on 756 White children (397 girls) studied longitudinally: (1) rearing experiences would predict pubertal timing; and (2) children would prove differentially susceptible to rearing. Analysis of pubertal measurements, including some based on repeated physical assessments, showed that mothering and fathering, earlier and later in childhood, predicted pubertal development, but only for girls, with negative parenting appearing most influential; maternal harsh control predicted earlier menarche. Rearing effects varied by infant negative emotionality, proving stronger (and opposite) for girls who in infancy were lower rather than higher in negativity. Maternal menarche, controlled in all analyses, was a stronger predictor than rearing. Findings are discussed in terms of theory development, genetic and nutritional influences, and sample restrictions.

Although the sequence of pubertal changes in adolescence is predictable, the timing of puberty is variable (Marshall & Tanner, 1969, 1970; Largo & Prader, 1983a, 1983b). Importantly, a substantial body of evidence indicates that such variability is of developmental significance: Girls who mature earlier than their peers are at elevated risk for a wide array of emotional and behavioral problems (Ge, Conger, Simons, & Murry, 2002; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Stattin & Magnusson, 1990), though, for boys, effects of early maturation are more mixed in terms of putatively positive outcomes (e.g., popularity, self confidence) and negative ones (e.g., delinquency; Collins and Steinberg, 2006; Steinberg et al., 2006). These observations highlight the need to understand the determinants of pubertal timing, the focus of the current report.

Pubertal maturation is controlled by a multiplicity of complex interactions between and among biological and environmental factors (Susman, Dorn, &

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Schiefelbein, 2003; Susman & Rogol, 2004). Indeed, the timing of puberty is influenced by genes, socioeconomic status, environmental toxins, diet, exercise, prepubertal fat and body weight, and the presence of chronic illness and stress. The link between stress and pubertal timing is especially intriguing—and complicated. Whereas high levels of *chronic and severe* stress (e.g., nutritional deprivation, extreme exercise regimens) are associated with delayed pubertal onset (e.g., Ghana: Adadevoh, Agble, Hobbs, & Elkins, 1989; Nigeria: Abioye-Kutyei et al., 1997), a recent state-of-the-art review of the determinants of pubertal timing in girls finds that “most human research on the effects of familial environments on pubertal timing suggests that family adversity is associated with earlier, rather than later, pubertal development” (B. J. Ellis, 2004, p. 27).

Because much remains to be learned about which factors and processes play exactly what roles in shaping pubertal timing (Susman, Dorn, & Schiefelbein, 2003), in this report we examine the link between pubertal timing and the kinds of nonextreme family factors and processes typically associated with relatively earlier maturation using prospective, longitudinal data from the NICHD Study of Early Child Care and Youth Development (SECCYD). Moreover, and in contrast to virtually all related studies, we use *repeated physical assessments* of pubertal development conducted by a trained nurse or physician to assess pubertal onset, along with reports by girls of their age of menarche. It seems noteworthy that both outcomes are investigated in this inquiry, given recent longitudinal research showing “only moderate correlation between menarche and onset of puberty (assessed via Tanner growth staging of pubic hair and the Garn-Falkner system for areolar stages)” (Biro, et al., 2006, p. 234; see also Slyper, 2006).

Family Experiences and the Timing of Pubertal Development

Much of the work highlighting linkages between family factors and processes and pubertal timing reported over the past 15 years has sought to test the prediction of Belsky, Steinberg, and Draper’s (BSD) (1991) evolutionary theory of socialization linking early rearing environment with pubertal timing, especially features of the rearing environment reflective of parental investment. Drawing on life-history theory and behavioral ecology within the broad arena of evolutionary biology, BSD recast research findings on the determinants and consequences of family rearing experiences in terms of reproductive strategy. More specifically, they argued that the home environment, shaped as it is by extrafamilial stressors and

supports, influences not just children’s psychological and behavioral development, including sexual, mating, and parenting behavior, but somatic development as well.

Indeed, distinguishing BSD theory from more familiar social-learning, attachment and life-course frameworks was the prediction that stress-inducing family processes accelerate the onset of puberty, whereas more emotionally supportive rearing environments exert the opposite effect. BSD reasoned specifically that experiences, such as harsh parenting, conflicted parent-child relationship, and marital conflict convey to the child that the world is a precarious place, that confidence cannot be placed in the nurturance or support of others, and thus that the wisest strategy for achieving the (unconscious) evolutionary goal of passing one’s genes onto future generations is to mature early, initiate sex early, have multiple and unstable partnerships, bear more rather than fewer offspring, and invest limited time and effort in caring for them. Such a reproductive strategy, it was theorized, would have enhanced reproductive fitness in the ancestral environments—or at least some of them—in which humans developed. Under contrasting ecological and developmental conditions, BSD theorized that it would have been most adaptive—in terms of enhancing the dispersion of genes in future generations (i.e., reproductive fitness)—to defer maturation and sexual activity, establish enduring pair bonds, and invest heavily in terms of time and effort in raising fewer rather than more children. (For further explication of the explanatory evolutionary logic underlying pubertal predictions, see Belsky et al., 1991; B. J. Ellis, 2004.)

A good deal of evidence has emerged consistent with BSD’s theoretically derived prediction that less supportive home environments would be associated with (somewhat) earlier onset of puberty, leading B. J. Ellis (2004, pp. 935-936) to conclude in his comprehensive review that “empirical research has provided reasonable, though incomplete” support for BSD theory. Consider the following findings that must be regarded as striking from the standpoint of traditional theories of familial influence on child and adolescent development, even if not from the standpoint of what is known about social influences on pubertal timing in many mammalian species (Steinberg, 1988) and on other aspects of reproductive functioning in humans (Belsky et al., 1991). First, greater parent-child conflict and coercion predict earlier timing of puberty—in both prospective longitudinal work (B. J. Ellis & Garber, 2000; Moffitt, Caspi, Belsky, & Silva, 1992; Graber, Brooks-Gunn, & Warren, 1995) and in research adopting retrospective

or concurrent-assessment designs (Jorm, Christensen, Rodgers, Jacomb, & Easteal, 2004; Kim & Smith 1998a, 1998b; Kim, Smith, & Palermiti, 1997; Mezzich et al., 1997; Surbey, 1990; Weirson, Long, & Forehand, 1993). Second, it is not just indices of family dysfunction that systematically relate to pubertal timing, as the positive quality of early parent involvement also appears to play a role in regulating the timing of menarche, again in both prospective studies (N. Ellis, 1991; Graber et al., 1995; Steinberg, 1988) and retrospective or concurrent ones (Kim & Smith 1998a; Kim et al., 1997; Miller & Pasta, 2000; Romans, Martin, Gendall, & Herbison, 2003; Rowe, 2000). B. J. Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates (1999) found, for example, that the more affectionate-positive interaction that took place between parents and their 5-year-old daughters, the later the age of menarche.

Evidence also indicates that family structure is predictive of pubertal timing. Girls growing up in father-absent homes mature earlier than do other girls, as revealed by prospective studies (Campbell & Udry, 1995; B. J. Ellis & Garber, 2000; B. J. Ellis et al., 1999; Hetherington & Kelly, 2002; Moffitt et al., 1992; Rowe, 2000; Wierson et al., 1993) and retrospective research on adult samples (Boegert, 2005; Doughty & Rodgers, 2000; Hoier, 2003; Hulanicka, 1999; Hulanicka, Gronkiewicz, & Koniarek, 2001; Jones, Leeton, McCleod, & Wood, 1972; Jorm et al., 2004; Kiernan & Hobcraft, 1997; Quinlan, 2003; Romans et al., 2003; Surbey, 1990), though some investigators have failed to detect such family structure effects (e.g., Boothroyd & Perrett, 2006). Despite the fact that the effect of father absence on girls' pubertal timing has been detected in a variety of Western countries, it does not seem to hold for African American samples (Campbell & Udry, 1995; Rowe, 2000). This could be a function of the "extraordinary secular trend" in the downward age of pubertal onset in this population (B. J. Ellis, 2004, p. 940) or, alternatively, high body fat levels in African American girls. Indeed, the dramatic difference in the timing of pubertal development between African American and White populations (e.g., Harlan, Harlan, & Grillo, 1980; Herman Giddens et al., 1997; Sun et al., 2002; Wu, Mendola, & Buck, 2002), something also detected in the NICHD SECCYD dataset, coupled with the limited number of Black children available for study in this longitudinal investigation, leads to an exclusive focus on (non-Hispanic) White children in this report.

Some Issues in Need of Additional Research

Despite the substantial evidence that has accumulated on relations between family factors and processes

and pubertal timing, several issues merit further investigation. One involves the differential impact of positive relationship experiences in the family (e.g., parent-child closeness) versus negative ones (e.g., parent-child conflict, harsh control) (see B. J. Ellis et al., 1999; Graber et al., 1995; Kim & Smith, 1998b; Kim et al., 1997), as few studies have focused upon both aspects of the parent-child relationship. In view of the fact that these do not always predict the same developmental outcomes (Petit & Bates, 1989; Petit, Bates, & Dodge, 1997), both are included in this inquiry. Based on prior work, it is predicted that *positive and negative relationship experiences will contribute to pubertal timing, with pubertal maturation occurring earlier when family relationships are more negative and less positive.*

It is also uncertain whether it is principally the presence/absence of fathers and the quality of father-child relations that predict pubertal timing or whether maternal behavior and the mother-child relationship are also influential (see B. J. Ellis et al., 1999; Kim & Smith, 1998a; Kim et al., 1997; Mekos, Hetherington, & Clingempeel, 1992), again because not all relevant investigations have measured both. B. J. Ellis (2004, p. 938, emphasis added) has highlighted the distinctive role of the father, arguing that "girls detect and internally encode information specifically about the quality of *paternal* investment . . . as a basis of calibrating . . . the timing of pubertal maturation and certain types of sexual behaviour." He has further stipulated that "Paternal investment theory . . . posits a unique and central role for the quality of paternal investment in regulation of daughters' sexual development, separate from the effects of other dimensions of psychosocial stress and support in the child's environment" (B. J. Ellis, 2004, p. 938). Based on this view and prior work, we hypothesize that *father-child relations will, like mother-child relations, predict pubertal timing, especially in girls.*

In view of the fact that not all studies detect father-absence effects and that even among those that do, amount of time spent residing in a father-absent home has not been considered, we test the hypothesis that *greater exposure to father absence will predict earlier timing of puberty.* We purposefully eschew consideration of amount of exposure to stepfather figures in light of (a) recent evidence from two nationally representative studies that fail to detect main effects of stepfather presence on pubertal development (Boegert, 2005; Quinlan, 2003), and (b) B. J. Ellis' (2004, p. 944) contention that stepfather effects emerge principally under particular family conditions that will not be considered in this inquiry. Moreover, because of a limited number of families with absent

fathers in the current study, the reader should be alerted that the prospect of testing father presence/absence effects in this inquiry may be more limited than in others conducted to date (see discussion).

Given the very limited research on the familial determinants of pubertal timing in males, partly stemming from the difficulty in reliably measuring it, there is also the question of whether the effect of family experiences on pubertal timing varies by child gender (N. Ellis, 1991; Kim & Smith, 1998b; Mao & Tremblay, 1997). Some (retrospective) work by Kim and Smith (1998a; Kim et al., 1997) and by Boegert (2005) indicates that many of the same factors found to predict earlier age of menarche in females operate similarly with respect to spermarche and other indicators of male pubertal development (e.g., father absence, stressful family relationships, marital conflict; but see Malo & Tremblay, 1997), but virtually all prospective longitudinal studies of the effects of family experience on pubertal timing have focused solely upon females (e.g., B. J. Ellis & Garber, 2000; B. J. Ellis et al., 1999; Moffitt et al., 1992; Graber et al., 1995). In the current inquiry, both boys and girls are a focus of attention, though we anticipate that *familial forces will prove more predictive of girls' than of boys' pubertal timing*, based on the view that female reproductive development is more sensitive to social experience than is that of males.

Another important issue concerns the developmental timing of familial experience. BSD theorized that it was relatively early family experiences—in the first 5–7 years of life—that shape reproductive strategy; and several datasets indicate that it may be early family experiences that are particularly influential with respect to pubertal timing, a hypothesized component of reproductive strategy (but see B. J. Ellis, 2005). Quinlan's (2003) study of more than 10,000 U.S. women found, for example, that parental divorce/separation between birth and age 5 predicted early menarche—and did so better than such family experience when children were 6–11 years of age. Relatedly, more years of father absence predicted earlier age of menarche in a New Zealand study (Moffitt et al., 1992) and greater pubertal development at 7th grade in a U.S. one (B. J. Ellis & Garber, 2000; see also Surbey, 1990). Such results lead to the prediction that *family experience in the first 5 years of life will prove more predictive of pubertal timing than experiences thereafter*. In recognizing that development remains open to modification as children age, it is further anticipated that *experiences subsequent to the first 5 years of life also will prove influential*.

However much evidence has emerged over the past 15 years consistent with BSD's evolutionary

theory linking family experience, especially the quality of parent-child relations and male presence, with pubertal timing, it needs to be noted that the detected relations have not been large. This may be due to the fact that the acceleration of pubertal onset that has taken place in the Western world in the past 150 years (i.e., the secular trend) has severely truncated its range of reaction (Belsky et al., 1991; B. J. Ellis, 2004). Further, the amount of variance that can be explained by family experience may be limited, because the timing of puberty is largely under the control of specific genes and, thereby, inherited, or influenced by health and nutrition. In view of the fact that no developmental perspective other than an evolutionary biological one anticipated the link between rearing experience and pubertal timing, the very existence of reliable associations between rearing and pubertal timing may be of as much significance, theoretically, as the magnitude of the linkage (Belsky, 2000, 2007; Belsky et al., 1991).

Differential Susceptibility to Rearing

Additional evolutionary thinking raises another possible reason for the modest effects detected to date linking rearing experience to pubertal timing, namely, that children should vary in their susceptibility to rearing influences (Belsky, 1997, 2000, 2005; Boyce & B. J. Ellis, 2005). Were this the case, failure to distinguish more and less susceptible children would result in the underestimation of effects for some (i.e., the more susceptible) and the overestimation of effects in the case of others (i.e., the less susceptible). To the extent that relatively more susceptible individuals are small in number, the average effect size will be very small.

Belsky (1997, 2000, 2005) theorized that, because the future is uncertain, parents in ancestral environments could not have known whether their efforts to direct their child's development would, even if successful relative to their (conscious or unconscious) goals, prove to be in their own and their child's reproductive best interests. Accordingly, natural selection could have shaped human development in a manner consistent with a "bet-hedging" strategy, one in which the degree to which children are susceptible to rearing influence varies, so that any rearing strategy would not exert comparable effects on all children in a household. This way, should the future turn out to be different from what parents "prepared" their children for via the creation of a particular family environment, only some children would end up being poorly served by having been influenced by a type of rearing that was not well suited to the child's future environment.

Intriguingly, a growing body of empirical research consistent with this argument comes from studies of infants and toddlers who vary in their negative emotionality, including work specifically designed to test the differential susceptibility hypothesis. In fact, not only do a surprisingly large number of correlational findings from diverse cross-sectional and longitudinal studies indicate that more negatively emotional infants/toddlers manifest a greater developmentally-facilitative response to supportive parenting *and* a more adverse response to harsh or unsupportive parenting than do children scoring lower on negative emotionality (for review, see Belsky, 2005), but so does some experimental work—on primates (Suomi, 1995, 1997) and humans (Blair, 2002, Velderman, Bakermans-Kranenburg, Juffer, & van IJzendoorn, 2006). In the current work, we test the differential-susceptibility hypothesis via interactions between a heritable index of infant negative emotionality, observed distress in the Strange Situation (Bokhurst, Bakermans-Kranenburg, Fearon, van IJzendoorn, Fonagy, Schuenge, 2003), and various measures of the home rearing environment (e.g., father absence, harsh parental control). In doing so, we are extrapolating from correlational and experimental evidence mostly having to do with rearing effects on the development of self control (e.g., externalizing problems, conscience) in this investigation where the phenomenon to be explained is somatic development.

Current Study

To summarize, the current investigation extends research on family influences on the timing of pubertal development and, thereby, evolutionary thinking about human reproductive strategy (a) by focusing upon boys as well as girls and (b) on mothers as well as fathers, (c) by relying on repeated physical assessments of stage of pubertal development to complement girls' reports of menarcheal age, (d) by investigating the effects of both family structure (i.e., father absence/presence) and family process (i.e., parenting, parent-child relationships), (e) by examining both positive features of the rearing environment (e.g., sensitive parenting) and negative ones (e.g., harsh control), (f) by distinguishing family experiences in the first 4.5 years of life from those early in the primary-school years, and (g) by testing the proposition that children who were emotionally more negative as infants are more susceptible to rearing influence.

The fact that the current work cannot disentangle genetic from more exclusively nongenetic influences must be acknowledged at the outset. This is an

important limiting feature of our investigation, because research indicates that pubertal timing is heritable (Golden, 1981; Kaprio et al., 1995; Rowe, 2002; Treloar & Martin, 1990), with some candidate genes even identified (e.g., Comings, Muhleman, Johnson, & MacMurray, 2002; Kadlubar et al., 2003; Seminara et al., 2003; Stavrou, Zois, Ioannidis, & Tsatsoulis, 2002), although specific genetic determinants remain largely unknown (Sisk & Foster, 2004). Rowe's (2000) inability to discount nongenetic influences on menarcheal age using a genetically informative design, coupled with Jorm et al.'s (2004) failure to replicate Comings et al.'s (2002) findings linking family experiences and pubertal timing to the same gene, along with questions that have been raised about the interpretation of behavior-genetic data on pubertal timing (B. J. Ellis, 2004) suggest that it would be a mistake to conclude that the evidence summarized above reflects nothing more than behaviour-genetic processes "masquerading" as environmental ones (see also Mendle et al., 2006). Given appropriate concerns about the role of heritability in accounting for any linkage between family experience and pubertal timing, as well as about the potential gene-environment correlations and correlated maternal and child rearing environments, we control in all analyses for mother's (retrospectively reported) age of menarche, which research indicates can be accurately reported even three decades after the event (Must et al., 2002).

Method

Participants

Participants were a subset of the families in the NICHD Study of Early Child Care and Youth Development. Families in the study were recruited during the first 11 months of 1991 from 24 hospitals in the vicinity of 10 data collection sites (Charlottesville, VA; Irvine, CA; Lawrence, KS; Little Rock, AR; Madison, WI; Morganton, NC; Philadelphia, PA; Pittsburgh, PA; Seattle, WA; and Wellesley, MA). A total of 8,986 women who gave birth during selected 24-hour periods and their infants were screened in the hospital for participation in the study.

Mother-newborn dyads were excluded from the study if the mother was under 18 years old, did not speak English, had acknowledged substance abuse, was too ill to participate, was placing her infant for adoption or refused the hospital screening interview or a follow-up telephone call two weeks later; if the infant had serious medical complications or was a multiple birth; or if the family lived more than an

hour's drive from the lab site, planned to move from the area within one year, lived in a neighbourhood deemed by police too unsafe for visitation, or was enrolled in another study. A total of 5,416 families met the eligibility criteria. Study participants were selected from among eligible families based on conditionally random sampling to insure that the sample would include at least 10% single-parent households, 10% mothers with less than a high school education, and 10% ethnic minority mothers. Recruitment and selection procedures are described in detail in previous study publications (e.g., NICHD ECCRN, 1997) and on the study Web site (<http://secc.rti.org>).

A total of 1,364 families with healthy newborns were ultimately enrolled in the study, with approximately equal numbers of families at each site. The study sample was demographically similar to the population of families with young infants in the communities from which it was recruited.

Analysis Sample. The analysis sample for the current study consisted of 756 White children ($n = 397$ girls). Children were included in these analyses if they participated in any of the study's repeated physical assessments of pubertal status (see below). Minority children were excluded from the sample because their numbers were too small to allow for analyses that would permit us to test the study hypotheses within minority sub-samples.

In the analysis sample, mothers were 29.36 years of age at enrollment in the study, had completed an average of 14.7 years of education and were living with a spouse or male partner on an average of 90% of measurement occasions from the time the child was 1 month of age until the child was in 3rd grade. Their average family income, assessed in terms of income-to-needs averaged from the time the child was 6-months old to the time the child was in 3rd grade, was higher than the U.S. government-determined poverty line by a factor of 4.11. The participants differed in several ways from the 286 White children who were recruited at birth but not included in this analysis due to total absence of puberty data. Mothers of participants were older ($M = 29.36$ years vs. 27.88 years, $p < .0001$), and had more education ($M = 14.70$ years vs. 14.14, $p < .002$).

Procedures and Measures

The children in the current study were followed from birth through eighth grade. Families were visited at home when the children were 1, 6, 15, 24, 36, and 54 months of age, and when they were in 1st and 3rd grades. At each home visit, mothers responded to a demographic interview and completed question-

naires about themselves, the child, and their family, and children were observed in interactions with one or both parents. Mothers and children also came to university laboratories when they were 15, 24, 36, and 54 months old, and again during 1st and 3rd grades. At these visits the children completed various standardized assessments and developmental tasks and were observed during play and interaction with their mothers. Beginning when they were approximately 9½ years old, children received an annual health and physical development assessment from a nurse practitioner or physician. Between visits, data were obtained using periodic telephone interviews and questionnaires (see below for schedule).

This section describes the specific measures used in this study. Measurements are presented in terms of how they were used in the analyses. Additional details about all data collection procedures and measures are documented in the study's Manuals of Operation, which are available on the study Web site (<http://secc.rti.org>). Descriptive statistics on all study variables are presented in Table 1, except for those that went into calculating pubertal onset, a topic addressed in the Results' section.

Control variable: Maternal age of menarche. Maternal age of menarche was used to (partly) control for genetic effects on children's timing of puberty. Mothers reported their age of menarche in years and months in a questionnaire about their own pubertal history and the pubertal development of their children. We averaged the information on menarcheal ages mothers provided when their children were in 4th, 5th, and 6th grades.

Moderator variable: Infant negative emotionality. Negative emotionality was operationalized as the mean level of observed infant distress during the three separation episodes of the Strange Situation administered at 15 months of age (Ainsworth, Blehar, Waters, & Wall, 1978; NICHD ECCRN, 1997). Distress during each episode was rated on a 5-point scale, with a rating of 1 reflecting no overt distress (and no attenuation of the child's exploration) and a rating of 5 reflecting immediate, high distress resulting in termination of the separation. Cronbach's alpha for the composite score was .83.

Family predictors. Several measures were available reflecting family structure/composition vis-à-vis father presence/absence, observed and reported parenting and the parent-child relationship. (The inter-correlation matrix of the predictor variables is available on request from the first author.)

Family structure measurements reflected the number of measurement occasions in which (a) the biological father was living in the same home as the child.

Table 1
 Descriptive Statistics of Study Variables

Study Variables	Girls					Boys				
	N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
Biological father, 0-54 mos.	397	10.59	3.05	0.00	12.00	359	10.19	3.55	0.00	12.00
Biological father, G1-G3	397	3.75	2.06	0.00	5.00	359	3.62	2.12	0.00	5.00
No father, 0-54 mos.	397	0.82	2.29	0.00	12.00	359	1.18	2.85	0.00	12.00
No father, G1-G3	397	0.60	1.46	0.00	5.00	359	0.57	1.43	0.00	5.00
Mother sensitivity, 6-54 mos.	385	3.32	0.33	1.60	3.93	343	3.27	0.33	2.17	4.00
Father sensitivity, 6-54 mos.	322	3.43	0.35	2.00	4.00	294	3.41	0.34	2.08	4.00
Mother sensitivity, G1-G3	386	3.36	0.38	1.75	4.00	351	3.31	0.38	1.17	4.00
Father sensitivity, G1-G3	379	7.82	4.29	0.86	21.00	348	7.56	3.93	0.69	19.00
Mother harsh control, 54m	376	2.07	0.32	1.20	2.90	335	2.05	0.29	1.30	2.70
Father harsh control, 54m	298	2.13	0.32	1.20	2.90	277	2.13	0.31	1.30	3.00
Mother harsh control, G1-G3	391	2.37	0.35	1.12	3.34	354	2.41	0.35	1.30	3.67
Father harsh control, G1-G3	331	2.43	0.35	1.50	3.67	312	2.45	0.37	1.47	3.67
Mother closeness w/ child, 54m	374	4.61	0.29	3.36	5.00	339	4.56	0.32	3.00	5.00
Father closeness w/ child, 54m	299	4.37	0.37	3.09	5.00	281	4.33	0.40	2.82	5.00
Mother close w/ child, G1-G3	391	4.74	0.27	3.25	5.00	353	4.69	0.28	3.50	5.00
Father close w/ child, G1-G3	332	4.51	0.43	1.63	5.00	315	4.44	0.50	2.00	5.00
Mother conflict w/ child, 54m	380	2.27	0.60	1.00	4.42	340	2.30	0.64	1.00	4.33
Father conflict w/ child, 54m	299	2.15	0.56	1.00	4.33	281	2.27	0.56	1.08	4.50
Mother conflict w/ child, G1-G3	391	2.28	0.80	1.00	4.64	354	2.24	0.78	1.00	4.64
Father conflict w/ child, G1-G3	332	2.09	0.69	1.00	4.00	314	2.16	0.68	1.00	4.29
Emotionality	385	3.21	1.17	1.00	5.00	345	3.41	1.23	1.00	5.00
Mothers' age of menarche	378	12.81	1.43	9.00	18.00	346	12.83	1.36	9.00	17.75
Daughter's age of menarche	369	12.46	1.03	9.75	15.05					

and (b) no father figure was living in the child's home. This information was gathered during interviews with the mother every three months when the child was 6-42 months of age, every four months from the age of 42 months through 54 months and every six months from the age of 54 months through 3rd grade. In order to receive a score, children were required to have data at nine of the 12 possible measurement occasions. Scores for each of the two above-listed variables were generated for two separate measurement periods, 0-54 months and Grade 1 and Grade 3 (see Table 1).

Observed parenting measurements were obtained on multiple occasions. Positive, nonintrusive, responsive and supportive parenting was assessed by observing maternal and paternal behaviors during (a) mother-child semistructured interaction with toys when the children were 6, 15, 24, 36, and 54 months old and when they were in 1st and 3rd grade, and (b) father-child semistructured interactions with toys when the children were 6, 36, and/or 54 months old and when they were in 1st and 3rd grades. Mothers were required to have data at four of the five measurement

points between six and 54 months to receive a score on the composite. Due to the fact that not all 10 participating sites gathered father-child interaction data during the first three measurement occasions, fathering data were available at all three of these ages of measurement at two sites, for two of these measurement occasions at three sites, and at just one of these ages at five sites. Detailed descriptions of the procedures and coding of the mother-child and father-child interactions are provided in publications of the NICHD Early Child Care Research Network (1999, 2000, 2003, and 2004). Videotapes of the parent-child interaction sessions from the 10 data collection sites were later coded centrally at a site not involved in data collection, by coders blind to family circumstances.

Parent behavior was rated on a series of 4- or 7-point rating scales, which were composited to create a summary measure of sensitive parenting reflecting positive, nonintrusive, responsive, and supportive care. When the children were 6, 15, and 24 months of age the composite was constructed from the sum scores of sensitivity to nondistress, intrusiveness (reverse scored), and positive regard. The sensitivity

composites at 36 and 54 months and in 1st and 3rd grade were formed from the sum of supportive presence, hostility (reverse scored), and respect for autonomy. Internal consistency (Cronbach's alpha) for the maternal sensitivity composites ranged from .70 to .84. Cronbach alphas for the paternal sensitivity composite ranged from .71 to .79. For purpose of analysis, scores were rescaled to a 4-point scale and sensitivity scores up to and including 54 months were averaged, as were scores from 1st and 3rd grade, thereby generating earlier and later measures of sensitive mothering and of sensitive fathering.

Reported parenting measurements also were collected repeatedly. When the children were 54 months of age, and when they were in 1st and 3rd grade, mothers and fathers completed a questionnaire assessing parenting strategies along three dimensions: harsh, firm, and lax (Shumow, Vandell, & Posner, 1998). For conceptual and psychometric reasons, only the harsh-parenting score is used in this report. At 54 months and in 1st grade, the scores were the mean of responses to six items on the questionnaire, with higher scores reflecting harsher control. Internal reliability (Cronbach's alpha) ranged from .66 to .71 across mothers and fathers and over time. In 3rd grade, the measure was the mean of responses to nine items, proportionally weighted and somewhat overlapping with items included at earlier ages (Cronbach's alpha = .75). To generate earlier and later measures of harsh control for purposes of analysis, separately for mothers and fathers, the 54-month scores served as the earlier measures of maternal and paternal harsh control, and the means of the 1st and 3rd grade scores served as later measures.

Reported parent-child relationship measurements were collected repeatedly by means of a questionnaire adapted from the Student Teacher Relationship Scale (STRS; Pianta, 1993), measuring the study child's attachment to each parent at 54 months of age and in 1st and 3rd grade. Items queried parents about their feelings and beliefs about their relationship with the study child and about the child's behavior toward the parent. For the purposes of this investigation, we selected two aspects of the parent-child relationships, closeness and conflict. Once again we averaged 1st and 3rd grade measurements and kept the 54-month measurement separate, thereby generating earlier and later measures of mother-child and father-child closeness and conflict. Closeness was based on the mean of 11 items at 54 months and 8 items in 1st and 3rd grade; conflict was based on the mean of 12 items at 54 months and 7 items in 1st and 3rd grade. Cronbach alphas across both scales, parents, and all times of measurement ranged from .65 to .84.

It is important to note, especially with respect to the data obtained on fathering, that it was provided entirely by men who were ready to participate in the study, and these were almost always resident biological fathers rather than stepfathers or nonresident biological fathers. What this means, of course, is that the measurement procedures used in this inquiry, which purposefully did not rely on maternal reports of father involvement, may fail to capture the parenting of the least involved fathers. The reader should be alert to the fact that this could restrict the range of fathering measured and thus this inquiry's ability to detect fathering effects.

Developmental outcome: Pubertal timing. Pubertal development was assessed on the basis of reports of daughter's age of menarche and annual physical exams of pubertal status using Tanner Criteria (Marshall & Tanner, 1969, 1970). Girls were on average 9.56 (SD = 0.13), 10.60 (SD = 0.16), 11.57 (SD = 0.14), 12.57 (SD = 0.14) and 13.55 (SD = 0.12) years of age at the 4th, 5th, 6th, 7th, and 8th grade assessments, respectively; boys were on average 9.58 (SD = 0.14), 10.60 (SD = 0.16), 11.58 (SD = 0.15), 12.57 (SD = 0.14) and 13.55 (SD = 0.13) years of age at the 4th, 5th, 6th, 7th, and 8th grade assessments, respectively. Importantly, then, there was limited variation within measurement occasion regarding when assessments were carried out, with standard deviations within measurement occasions ranging from a low of 1.4 months to a high of 2.0 months.

Age of menarche was determined at the physical exams in 5th through 8th grade (see below) by asking girls whether they had begun to menstruate and, if so, their age at their first menstrual period (in years and months). Mothers were also queried about their daughter's first menstrual period, and these data were used if information from the girls themselves was missing.

In the case of 59 girls seen at 8th grade who reported not yet having had their first period, estimates of age of menarche were randomly assigned as follows, drawing on results of Chumlea's et al. (2003) study of pubertal development: First, random ages based on a normal distribution with mean of 12.55 and SD of 0.70 were generated and, if the generated random value was greater than the girl's age at the 8th grade assessment, the value was kept. This procedure continued until the randomly generated age fit the criteria of being greater than the girls' age at her final assessment. The range of imputed values varied from 13.5 to 15.0. The normal distribution used to generate the random ages reflects the distribution of the ages of menarche reported for White girls in Chumlea et al. (2003)'s cross-sectional,

NHANES III-based study of more than 700 nationally representative non-Hispanic White girls between the ages of 8 and 20 years of age. In a study, such as the current one focused upon pubertal timing, this strategy of randomly assigning menarcheal age to a small subset of indisputably late maturers seemed far more scientifically appropriate than excluding them from the analysis.

Pubertal development was assessed by means of a physical examination. Beginning at age 9 ½ years, all study children and their mothers (or alternate primary caregivers) were asked to participate in an annual health and physical development assessment. A primary component of the assessment was a physical examination of the child. Pubertal status was assessed using Tanner staging. For girls, staging criteria were based on instructions from the American Academy of Pediatrics Manual, *Assessment of Sexual Maturity Stages in Girls* (Herman Giddens & Bourdony, 1995), augmented with breast bud palpation. Tanner staging for boys was based on Tanner's original criteria (adapted from Tanner, 1962; Marshall & Tanner, 1970). Most of the exams were conducted by nurse practitioners, but some were administered by pediatric endocrinologists, depending upon staff employed at each data collection site. All clinicians were experienced with Tanner staging of children in the evaluated age groups. If Tanner staging revealed the child to be between two of the five defined stages (e.g., between Stages 3 and 4) for any aspect of pubertal development (e.g., breast development), the child was assigned the lower stage score for that feature of development. The exams were conducted until the youngsters reached Tanner Stage 5 on the clinical ratings of breast/genital and pubic hair and until girls experienced menarche. These assessments yielded Tanner Stage scores (1-5) each year for (a) breast (girls) or genital (boys) development, and (b) pubic hair. For purposes of analysis, each of these scores was recoded to reflect either any evidence of puberty (Tanner Stage >1) vs. no evidence of puberty (Tanner Stage = 1).

To ensure across-site consistency in Tanner staging procedures and to introduce all clinicians to overall Study practices, central training was provided at NICHD before the study children turned 9 ½. The training session was videotaped for the use of personnel who were unable to attend and for those hired at a later date. Certification of basic competency in Tanner staging of girls and boys was done through the use of photos. Examiners qualified as certified when their overall score reached 87.5% or better on the photo tests. Details about the procedures used for Tanner Staging are available at <http://secc.rti.org/>.

Results

Two sets of results are presented, the first and preliminary having to do with the parameterization of the pubertal timing using repeated measurements and the second and primary having to do with the prediction of pubertal timing.

Preliminary Analyses: Timing of Pubertal Onset

In order to estimate timing of pubertal onset, we used latent transition analysis (LTA; Collins & Flaherty, 2002; Muthen & Muthen, 1998-2006). Separate models were run for boys and girls. The data included in these models indicated, at each age of measurement, whether the child showed any evidence of pubertal development (i.e., yes/no) on (a) physical exam genital (for boys) or breast (for girls) development, and (b) physical exam pubic hair development. The LTA model for girls included four measurement points between 9½ years and 12½ years, whereas the model for boys included five measurement points between 9½ and 13½ years. The girls' model did not include the 13½ year assessment, because all girls had started puberty by that time. LTA analyses were run using MPlus (version 4.1: Muthen & Muthen, 1998–2006); it uses maximum likelihood (ML) estimation under the assumption of data missing at random (MAR). Thus, children were included in the LTA if there was any nurse/physician measurement of them at any point.

The LTA models constrained the weights of the measures listed above to be equal at each age and constrained children who had "started puberty" at one age to remain "in puberty" at later ages. Thus, the weighting given to any particular indicator was equal across time points (i.e., measurement invariance) and children, once categorized as having initiated puberty, could not revert to a no-initiation state.

Results of this analysis are presented in Table 2. The table presents the *n*'s and percents of children having started puberty at each age and the mean probability (and standard deviation) of a child being in the group to which he or she has been estimated to belong and thus assigned. The mean probabilities provide some evidence for how accurate the model is in classifying children into each timing group. In general, girls and children with more data were more confidently classified than boys and children with less data. The final timing of pubertal onset variable to be used in subsequent prediction analyses ranged from 1 to 5 ($\leq 9\frac{1}{2}$ to $\geq 12\frac{1}{2}$ years) for girls and from 1 to 6 ($\leq 9\frac{1}{2}$ to $\geq 13\frac{1}{2}$ years) for boys.

The data presented in Table 2 underscore the validity of the pubertal onset measurement in showing

that it is sensitive to gender differences in pubertal maturation: Girls mature faster than boys, with many more girls than boys having initiated puberty by 9.5 years of age (19.7% vs. 3.3%) and many more boys than girls having yet to do so by 12.5 years of age (14.5% vs. 1.1%). Moreover, height measurements taken at the first physical examination in 4th grade correlated significantly (and negatively) with girls' pubertal onset ($r = -.34, p < .0001, n = 342$) and with boys' pubertal onset ($r = -.20, p < .0001, n = 330$), with the same being true of weight in 4th grade in the case of girls ($r = -.29, p < .0001, n = 342$), though only marginally so in the case of boys ($r = -.10, p < .10, n = 330$). Given that a lower pubertal-onset score reflects earlier onset, these data are consistent with the expectation that larger children mature earlier than smaller children. Evidence for the validity of the pubertal onset measure also derives from its positive and significant association with age of menarche ($r = .53, p < .0001, n = 342$).

Primary Analyses: Predicting Pubertal Timing

In order to examine potential effects of rearing experience on pubertal timing and to explore the possibility that children who varied in their negative emotionality as infants might prove differentially susceptible to rearing effects, a series of hierarchical regression analyses was conducted. Each prediction equation for each of three outcomes of interest—age of menarche, girls' pubertal onset, and boys' pubertal onset—included four predictor variables (a) mother's age of menarche (to control for heritability to some extent), (b) a rearing variable of interest, (c) infant negative emotionality, and (d) the rearing-X-emotionality interaction. All variables were centered, and interaction variables were calculated utilizing the

centered variables to avoid nonessential ill-conditioning (Aiken & West, 1991). Whenever the interaction between the rearing variable and negative emotionality proved even marginally significant (i.e., $p < .10$), follow-up testing was conducted to illuminate the nature of the interaction. This approach was judged appropriate given the documented difficulty detecting interactions in nonexperimental designs (McClelland & Judd, 1993). Only one specific rearing variable was included in each prediction equation (as a main effect and in interaction with infant negativity) in order to maximize the sample size used to evaluate the predictive power of each rearing predictor. Inclusion of multiple rearing predictors in the analyses risked reducing sample size due to case-wise deletion as a result of missing data, a problem that would have especially plagued any effort to include maternal and paternal data in the same prediction equation due to the fact that paternal data were far more likely to be missing than maternal data.

Predicting boys' pubertal onset. Data displayed in Table 3 show that boys' pubertal onset was not significantly predicted by maternal age of menarche nor by any of the rearing variables, nor by infant negativity. One marginally significant rearing-X-emotionality interaction emerged, but given the overall number of tests carried out, this single marginal result was considered likely to be a product of chance and was therefore not subjected to follow-up analysis.

Predicting girls' pubertal development. Data displayed in Table 3 show that mothers who reported an earlier age of menarche had daughters who reported earlier age of menarche (range of betas = 0.36-0.38, $p < 0.0001$) and who, according to nurse/physician evaluations, initiated pubertal development earlier than did girls whose mothers reported a later age of menarche (range of betas = 0.16-0.23,

Table 2
N's (%) of Children Starting Puberty at Each Age and the Mean (SD) Probability of Children's Most Likely Group

Age of Pubertal Onset	Girls				Boys			
	N	%	Prob. of Group Membership		N	%	Prob. of Group Membership	
			Mean	SD			Mean	SD
≤ 9.5	73	19.7	0.90	0.09	12	3.3	0.89	0.08
9.5 – 10.5	148	40.0	0.76	0.24	31	8.6	0.89	0.11
10.5 – 11.5	89	24.1	0.88	0.16	96	26.7	0.85	0.13
11.5 – 12.5	56	15.1	0.89	0.17	168	46.8	0.66	0.25
12.5 – 13.5	4	1.1	1.00	0.00	50	13.9	0.77	0.12
> 13.5					2	0.6	1.00	0.00

$p < 0.001$). As a main effect, infant negative emotionality did not significantly predict either pubertal outcome for girls.

With maternal menarcheal age and infant negativity taken into consideration, two main effects of rearing, both of which were unqualified by interactions with negative emotionality and involved maternal harsh control, emerged in the case of girls' age of menarche but none in the case of pubertal onset: Girls who experienced more maternal harsh control at 54 months or in 1st and 3rd grade had their first period at a younger age, findings in line with expectations.

With maternal menarcheal age, infant negativity and main effects of rearing taken into consideration, seven significant or marginally significant rearing- X -negativity interactions emerged. In one case this interaction involved the prediction of menarche, with the pertinent rearing variable being father harsh control in 1st and 3rd grade. In the six other instances, it was girls' pubertal onset that was predicted by the interaction of infant negativity and (a) 6–54 month maternal sensitivity, (b) 54 month maternal harsh control (marginal), (c) 54 month father harsh control, (d) 1st and 3rd grade maternal harsh control (marginal), (e) 1st and 3rd grade father harsh control, and (f) 1st and 3rd grade father-child closeness.

In order to illuminate the nature of these interactions and thereby determine whether, as hypothesized, relations between rearing and pubertal timing were more pronounced in the case of girls who scored high in negative emotionality as infants, the simple slopes for the rearing variables were reestimated at one standard deviation above and below the mean on infant negative emotionality (Aiken & West, 1991); the resultant standardized parameter estimates for these groups are presented in Table 4. Inspection of the data reveals two striking patterns. First, contrary to (*empirically* derived) expectations that rearing effects would be more pronounced in the case of high-negativity, the opposite proved true, as revealed by comparison of paired parameter estimates. More specifically, in five of seven instances, the absolute value of the estimate for girls with low negativity proved larger than that of girls with high negativity, suggesting that, if anything, it is girls who were the least negative as infants who were most susceptible to rearing effects, at least with respect to pubertal development.

The second striking pattern, this one partially in line with (*theoretically* derived) expectations, indicates that girls with high versus low negativity varied greatly in *how* rearing appeared to influence their pubertal development (i.e., not just the strength of the

effect), with similar rearing experiences seeming to exert opposite effects at different ends of the negativity spectrum. Irrespective of whether the estimates in Table 4 for more and less negative infants proved significant, it seems notable that they were always in line with expectations in the case of those with high-negativity—and opposite (in direction) for those with low-negativity. Consider in this regard the following illustrative findings, the first two sets of which are graphically illustrated in Figures 1 and 2: Whereas *greater* maternal sensitivity at 6–54 months (insignificantly) predicted *later* pubertal onset in the case of girls highly negative as infants (i.e., in the expected direction), for girls who were low in negativity as infants, it (significantly) predicted *earlier* pubertal onset (i.e., opposite the anticipated direction of effect). Whereas *greater* maternal harsh control at 54 months (insignificantly) predicted *earlier* pubertal onset in the case of girls highly negative as infants, for girls who were low in negativity as infants, it (significantly) predicted *later* pubertal onset. And, as a final (ungraphed) example, whereas *greater paternal* harsh control in 1st and 3rd grades (significantly) predicted *earlier* pubertal onset and menarcheal age in the case of girls highly negative as infants, for girls who were low in negativity as infants, it (significantly) predicted *later* pubertal onset and (insignificantly) predicted later age of menarche. In sum, more negative rearing conditions were related to *earlier* pubertal onset (or age of menarche) and more positive rearing conditions were related to *later* pubertal onset, just as anticipated by BSD theorizing, in the case of girls who scored higher in negativity as infants, but in the case of girls who scored lower in negativity as infants, more negative rearing conditions predicted *later* pubertal onset (or age of menarche), and more positive rearing conditions predicted *earlier* pubertal onset.

Discussion

The research presented sought to extend prior work linking family rearing experiences with the timing of puberty, most of it stimulated by Belsky et al.'s (1991) prediction that negative rearing experiences (e.g., father absence, harsh parenting) would accelerate the timing of puberty, with the converse being true of positive-supportive rearing experiences. In addition, we theorized that children would prove differentially susceptible to rearing and, based on work examining rearing effects on self-control-related outcomes, that those who were highly negative as infants would be most likely to manifest rearing effects. Methodologically, we extended prior work

Table 3
Predicting Girls'/Boys' Pubertal Timing

	Girls						Boys		
	Age of Menarche			Pubertal Timing			Pubertal Timing		
	Beta	R ² -Change	(n)	Beta	R ² -Change	(n)	Beta	R ² -Change	(n)
Time with Father Biological Father	Emotionality	.08	(n = 342)	.00	.00	(n = 342)	-.03	.00	(n = 342)
	Bio F	.02		-.11 [†]	.01		.04	.00	
	Bio F*Emotionality	-.03		.01	.00		.02	.00	
	Emotionality	.08		-.01	.00		-.03	.00	
	Bio F	.05		-.06	.00		.02	.00	
No Father at All	Bio F*Emotionality	-.05		-.02	.00		.02	.00	
	Emotionality	.07		-.01	.00		-.03	.00	
	No F	-.07		.05	.00		-.04	.00	
	No F*Emotionality	.02		-.01	.00		-.01	.00	
	Emotionality	.07		-.02	.00		-.03	.00	
Observed Parenting M. Sensitivity	No F	-.07		.00	.00		.06	.01	
	No F*Emotionality	.04		.05	.00		-.06	.00	
	Emotionality	.08	(n = 337)	-.02	.00	(n = 340)	-.04	.00	(n = 325)
	M Sens	.05		-.07	.01		.00	.00	
	M Sens*Emotionality	.01		.14*	.02*		.03	.00	
F. Sensitivity	Emotionality	.07	(n = 333)	.00	.00	(n = 336)	-.02	.00	(n = 331)
	M Sens	.02		-.09 [†]	.01		-.06	.00	
	M Sens*Emotionality	.04		.08	.01		.01	.00	
	Emotionality	.06	(n = 282)	-.03	.00	(n = 286)	-.04	.00	(n = 283)
	F Sens	-.05		.11 [†]	.01		.02	.00	
Reported Parenting M. Harsh Control	F Sens*Emotionality	.02	(n = 328)	-.08	.01	(n = 332)	-.03	.00	(n = 328)
	Emotionality	.08		-.01	.00		-.03	.00	
	F Sens	-.06		-.05	.00		-.02	.00	
	F Sens*Emotionality	-.05		.06	.00		-.05	.00	
	Emotionality	.06	(n = 327)	-.03	.00	(n = 332)	-.03	.00	(n = 316)
F. Harsh Control	M Harsh	-.11*		.01	.00		.01	.00	
	M Harsh*Emotionality	-.07		-.11 [†]	.01 [†]		-.05	.00	
	Emotionality	.07*	(n = 337)	-.02	.00	(n = 338)	-.03	.00	(n = 332)
	M Harsh	-.15**		-.07	.01		-.01	.00	
	M Harsh*Emotionality	-.07		-.10 [†]	.01 [†]		-.02	.00	
F. Harsh Control	Emotionality	.07	(n = 263)	-.03	.00	(n = 266)	-.01	.00	(n = 266)
	F Harsh	-.07		.03	.00		-.03	.00	
	F Harsh*Emotionality	-.09		-.17**	.03**		-.05	.00	
	Emotionality	.07	(n = 284)	-.03	.00	(n = 287)	-.01	.00	(n = 296)
	F Harsh	-.07		.03	.00		-.03	.00	

Table 3. (Cont'd)

	Girls						Boys		
	Age of Menarche			Pubertal Timing			Pubertal Timing		
	Beta	R ² -Change		Beta	R ² -Change		Beta	R ² -Change	
M. P-C Closeness	G1-G3	Emotionality	.06	-.01	-.06		-.06		
		F Harsh	-.07*	.07	-.07	.00	-.07	.01	
		F Harsh*Emotionality	-.16*	.02**	-.27***	.07***	.01	.00	(n = 320)
M. P-C Closeness	54 mos.	Emotionality	.08	-.01	-.03	.00	-.03	.00	
		M Close	-.02	-.04	-.04	.00	-.04	.00	
		M Close*Emotionality	.02	-.06	.00	.00	.00	.00	(n = 331)
F. P-C Closeness	G1-G3	Emotionality	.08	-.02	-.03	.01	-.03	.00	
		M Close	-.08	.01†	-.09	.01	.01	.00	
		M Close*Emotionality	.01	.00	-.02	.00	.01	.00	(n = 270)
F. P-C Closeness	54 mos.	Emotionality	.06	-.02	-.02	.00	-.02	.00	
		F Close	.04	-.04	-.04	.00	.01	.00	
		F Close*Emotionality	-.04	.00	.02	.00	-.07	.01	(n = 298)
M. P-C Conflict	G1-G3	Emotionality	.07	.00	-.05	.01	-.05	.00	
		F Close	-.05	-.10†	-.10†	.01	.00	.00	
		F Close*Emotionality	.06	.00	.12*	.02*	.03	.00	(n = 321)
M. P-C Conflict	54 mos.	Emotionality	.08	-.02	-.03	.00	-.03	.00	
		M Conflict	.02	-.03	.02	.00	.02	.00	
		M Conflict*Emotionality	-.04	.00	-.06	.00	-.10†	.01†	(n = 332)
F. P-C Conflict	G1-G3	Emotionality	.08	-.02	-.03	.00	-.03	.00	
		M Conflict	.00	.01	.01	.00	.01	.00	
		M Conflict*Emotionality	.01	.00	-.03	.00	-.10†	.01†	(n = 270)
F. P-C Conflict	54 mos.	Emotionality	.06	-.03	-.02	.00	-.02	.00	
		F Conflict	.03	.05	-.02	.00	-.02	.00	
		F Conflict*Emotionality	.00	.00	-.07	.01	-.04	.00	(n = 298)
F. P-C Conflict	G1-G3	Emotionality	.06	-.02	-.06	.00	-.06	.00	
		F Conflict	.02	.01	-.02	.00	-.02	.00	
		F Conflict*Emotionality	-.02	.00	-.07	.01	-.08	.01	

Note. All models include maternal age of menarche (range of betas = 0.36-0.38, $p < 0.0001$ for Girls' Menarche, 0.16-0.23, $p < 0.0001$ for Girls' Pubertal Onset, and 0.05-0.10, ns, for Boys' Pubertal Onset).

R²: Variance accounted for; n: Number; m: Months; G1-G3: 1st-3rd grade; Bio: Biological; F: Father; M: Mother; Sens: Sensitivity; Harsh: Harsh control; Close: Closeness; P-C: Parent-child. R²-change values are reported for (1) adding main effects beyond maternal menarche, and (2) for adding interactions beyond maternal menarche and main effects. Overall R² ranged from 0.37 to 0.42 for Girls' Menarche, from 0.21 to 0.36 for Girls' Pubertal Onset and from 0.08 to 0.14 for Boys' Pubertal Onset.

M: Mother; F: Father; P-C: parent-child; G1-G3: Grades 1-3.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$, **** $p < .0001$.

Table 4

Predicting Girls Pubertal Development as a Function of Infant Negative Emotionality (Standardized Regression Coefficients)

Rearing Predictor	Puberty Outcome	Parameter Estimates	
		-1 SD Negativity	+1 SD Negativity
M Sensitivity, 6-54 mos.	Pubertal Onset	-.225**	.065
M Harsh Control, 54 mos.	Pubertal Onset	.113	-.091
F Harsh Control, 54 mos.	Pubertal Onset	.188*	-.135 [†]
M Harsh Control, G1-G3	Pubertal Onset	.024	-.165*
F Harsh Control, G1-G3	Pubertal Onset	.325***	-.185*
F Harsh Control, G1-G3	Age of Menarche	.079	-.231**
F P-C Closeness, G1-G3	Pubertal Onset	-.213**	.012

M: Mother; F: Father; P-C: parent-child; G1-G3: Grades 1-3.

[†] $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$.

by following a fairly large sample from early in life through 8th grade while applying a robust methodology for tracking pubertal onset, namely, repeated assessments of children's physical development by a trained nurse/physician. Moreover, by adopting this approach to measurement, greater confidence could be placed in assessments of boys' pubertal status in particular than has been the case in the few previous studies of rearing influence on male pubertal development.

Results indicated (a) that rearing experiences predicted pubertal timing among girls, but not boys; (b) that maternal age of menarche was a far stronger predictor of daughters' pubertal development than were rearing experiences; (c) that greater maternal harsh control (at 54 months and in 1st and 3rd grade) predicted earlier age of menarche; (d) that it was principally negative experiences rather than positive ones that seemed influential; (e) that this was so in the case of both mothering and fathering; and (f) that both

early and later experiences in the family appeared influential. In addition, and contrary to expectation, most detected effects of rearing proved stronger among lower-negative rather than higher-negative girls and were opposite for the two extremes, running in the expected direction only for the girls who were more negatively emotional as infants.

Because most of the variation in pubertal onset is attributable to genetics, nutrition, and maternal health during pregnancy, the fact that any interpersonal factors influence pubertal timing is noteworthy, regardless of the magnitude of detected effects. This would seem especially so in light of the fact that small changes in timing of puberty are associated with relatively large changes in the timing of onset of ovulatory menstrual cycles (for review of relevant literature, see B. J. Ellis, 2004). More specifically, the time from menarche until half of menstrual cycles are ovulatory is about one year when menarche occurs before age 12; but it is 4.5 years if menarche occurs at 13 or older (Apter & Vihko, 1983). Thus, even small

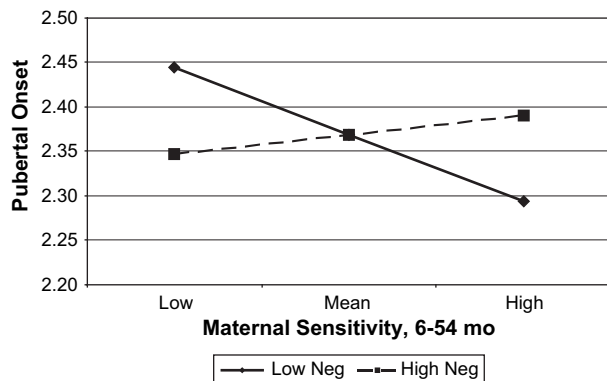


Figure 1. Relation of mother sensitivity at 54 months and girl's pubertal onset as a function of infant negative emotionality. Pubertal onset scored 1-5 reflecting annual increments from $<9\frac{1}{2}$ to $>12\frac{1}{2}$ years.

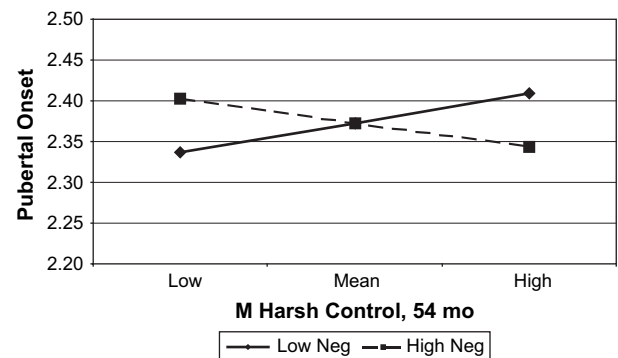


Figure 2. Relation of mother harsh control at 54 months and girl's pubertal onset as a function of infant negative emotionality. Pubertal onset scored 1-5 reflecting annual increments $<9\frac{1}{2}$ to $>12\frac{1}{2}$ years.

changes in the timing of puberty, like the small effects detected in this inquiry, are of importance in the real world (i.e., risk of teenage pregnancy), as well as being theoretically significant.

Also meriting consideration before discussing specific findings in more detail is the fact that the design of this study, involving *annual* assessments of pubertal development, was sensitive only to the detection of a full year's difference in the onset of pubertal development. Given that prior studies suggest that, in the industrialized world, rearing influences are likely to affect pubertal development on the order of months rather than years (B. J. Ellis, 2004), the weak associations detected in the present study may reflect the limited variation in pubertal timing that we were able to measure. It is possible, therefore, that the current study has underestimated the magnitude of some of the discerned effects.

Predicting Boys' vs. Girls' Pubertal Timing

The fact that when it came to predicting pubertal timing, significant associations emerged only in the case of girls addresses one of the important questions raised in the introduction, namely, whether evolutionary analyses of experiential influence on pubertal timing apply equally to boys and girls. The fact that we failed to detect any significant main effects of rearing on boys' pubertal onset as measured via nurse/physician examinations and only one marginally significant emotionality-X-rearing interaction argues against the possibility that the general absence of findings for boys in the literature is due to imprecision in the assessment of their physical development.

Differential Susceptibility: The Moderating Role of Infant Negative Emotionality

Evolutionary thinking, which called attention to the fundamentally uncertain nature of the future, gave rise to the prediction that children should vary in their susceptibility to rearing influence, with some proving more subject to influence than others (Belsky, 1997, 2000, 2005). Empirical evidence, most of it having to do with parenting effects on self-control related outcomes (e.g., externalizing problems, conscience), gave rise to the specific prediction that it would be children who were more negative as infants who would prove more susceptible to rearing influence (see Belsky, 2005, for review). Although the evidence from this inquiry clearly indicates that girls who were more versus less negative as infants were differentially susceptible to many of the rearing influences investigated, the findings were

inconsistent with expectations in two fundamental respects.

First, of the seven rearing-X-emotionality interactions that were followed up, five indicated more pronounced rearing effects in the case of the girls who scored lower—not higher—in negativity as infants. These unanticipated results raise the prospect that for different developmental outcomes—pubertal development versus self-control—different children may prove more and less susceptible to rearing. In other words, it may be important to think about differential susceptibility in domain-specific rather than domain-general terms (see Belsky, 2005).

The second way in which results proved surprising was in the nature of the differential susceptibility detected. We hypothesized that more adverse experiences would predict accelerated pubertal development (i.e., earlier age of menarche and/or pubertal onset), whereas more supportive rearing experiences would predict delayed development. This, of course, is what the findings involving the main effects of harsh maternal control indicated, as did the associations derived from the follow-up analyses of girls who were highly negative as infants. But in the case of girls who were low in negativity as infants, developmental processes appeared to operate in a reverse (and even stronger) fashion, with more adverse experiences predicting delayed development and more supportive rearing experiences predicting more accelerated development. The surprising nature of the rearing effects detected in the case of girls who scored low in negative emotionality warrants further study.

The fact that the unexpected results emerged only when infant negativity was considered as a moderating factor suggests that future investigations should take seriously not only the notion that children will vary in the *extent* to which they are affected by rearing but also the possibility that they will vary in *how* they are influenced. The fact that the measure of negative emotionality used in this inquiry has been shown to be heritable suggests that some of the differences between children in the degree—and manner—in which they are affected by rearing may be genetically influenced. This is not to say, however, that infant negativity is not itself susceptible to rearing influence (see Belsky, Fish, & Isabella, 1991) and thus could not have been influenced by parenting in the current study. In point of fact, simple correlational analysis showed that the 6-54 month maternal sensitivity composite correlated positively and significantly with girls' 15-month negative emotionality ($r = .11$, $p < .05$, $n = 377$), though the same association in the case of fathers was only marginally significant ($r = .11$,

$p < .10$, $n = 315$). These data and the preceding discussion remind us that variation in susceptibility to rearing may itself be experientially induced (Boyce & B. J. Ellis, 2005).

Positive vs. Negative Rearing Experiences

Despite the fact that Belsky et al.'s (1991) evolutionary theory of socialization called attention to the developmentally accelerating effect of adverse, negative or stressful rearing experiences *and* to the delaying impact of warm, supportive rearing experiences, few investigators have differentiated between the two (but see B. J. Ellis et al. 1999; B. J. Ellis & Essex, 2006). In the present study, it proved to be negative aspects of the parent-child relationship and parenting—specifically, harsh control—that seemed to matter most and not positive or supportive features of the rearing milieu. Consistent with prior research (for review, see B. J. Ellis, 2004), higher levels of harsh control by mothers at 54 months and in 1st-3rd grade forecast earlier age of menarche, irrespective of infant negativity. These main effects of rearing, coupled with related findings involving effects of maternal and paternal harsh control moderated by infant negative emotionality, suggests that it is principally aversive experiences in the home, rather than pleasurable ones, that influence pubertal timing. This may explain why observational measures of parenting figured in such a limited way in this inquiry, as the composite measure of sensitivity principally reflected the degree to which warm, supportive care (rather than hostile treatment) was observed during parent-child interactions.

The fact, however, that two of the seven significant or marginally significant interactions involved maternal sensitivity (from 6-54 months) and father-child closeness (1st and 3rd grade) suggests that it would be a mistake to discount positive rearing influences in their entirety. Not only have others detected such influences (B. J. Ellis et al. 1999; B. J. Ellis & Essex, 2006), but they have done so when compositing multiple aspects of supportive rearing in ways that could not be empirically justified in the current inquiry.

Earlier vs. Later Experiences

Because the NICHD SECCYD included some repeated measurements of similar and even identical rearing constructs, we were positioned to examine whether experiences measured prior to the start of schooling or thereafter differentially predicted pubertal timing. In the case of the main effects of rearing on

age of menarche, as well as all six of the rearing-X-emotionality interactions pertaining to pubertal onset, both early and later measurements of rearing proved predictive in this inquiry. Such results indicate, consistent with much contemporary developmental thinking, that both earlier and later experiences are of developmental significance (e.g., Sroufe, Egeland Carlson, & Collins, 2005). At the same time, it must be acknowledged that by third grade, adrenarche, which influences several aspects of pubertal maturation, is already underway in many, if not all children (McClintock & Herdt, 1966). This raises the possibility that some of the effects of later experiences detected could reflect effects of pubertal timing on parenting and parent-child relations rather than the reverse.

Experiences with Mother vs. Father

With regard to the relative importance of experiences with father and mother in predicting pubertal development, recall that we detected effects of both parents. Even though only harsh control by mother—at 54 months and in 1st and 3rd grade—proved significant as main effects in predicting age of menarche, both maternal and paternal harsh control, as well as maternal sensitivity and the closeness of the father-child relationship, interacted with infant negativity in predicting pubertal onset. Such results are consistent with both Belsky et al.'s (1991) evolutionary theory of socialization, which did not postulate any differential maternal-paternal influence and with B. J. Ellis's (2004; Ellis & Garber, 2000; B. J. Ellis et al., 1999; B. J. Ellis, 2003) paternal investment theory, which highlighted the unique role of father.

When considering why main effects of fathering did not prove significant, it needs to be appreciated that this inquiry differed from many others in that fathering measures were not based on maternal reports, including mothers' reports of nonresident fathers' involvement with the child, but rather on observations of father-child interaction and reports by fathers about their own parenting. As such, data on nonresident fathers were disproportionately absent, as were data from men who perhaps refused to participate in the study due to their low levels of active father involvement. This lacunae likely affected the range of variation in father involvement and behavior measured in this study, something that could well have impaired our ability to detect father effects, especially if it is the lack of high-quality father involvement that influences pubertal development (B. J. Ellis, 2004).

The fact that no evidence indicated that family structure, as operationalized in this inquiry (i.e.,

months child residing with and without biological father in home), predicted girls' pubertal development was somewhat unexpected. Indeed, the failure to detect, in particular, an accelerating effect of father absence on pubertal development is somewhat difficult to explain (but see Boothroyd & Perret, 2006, for similar results) but could well stem from the fact that father-absent families were more subject to attrition from the study than father-present ones.

A related and important limitation of the present work is that our sample of non-Whites was small, which meant that we restricted our analysis to a single racial-ethnic group. Future studies would do well to gather data, especially physical assessments of pubertal development, from a more diverse sample. This would seem especially important in light of the fact that effects detected in this inquiry (or not detected) could have been attenuated due to the restricted range of some predictor variables, itself a function of the characteristics of the sample available for statistical analysis. Certainly consistent with this line of argument is the fact that data were most likely to be missing for more disadvantaged families, including father-absent ones, which are just the kind of households that research shows are at greater risk of engaging in more negative and less supportive parenting (e.g., McLloyd, 1988).

Conclusion

Results of the current inquiry were somewhat consistent with expectations, especially those pertaining to the main effects of maternal harsh control in seemingly accelerating pubertal development vis-à-vis age of menarche, but also in terms of the nature of the rearing effects that emerged for the modest sample of girls who were highly negative as infants. Recall that for these girls, negative rearing experiences were related consistently to earlier pubertal onset and supportive rearing experiences were related consistently to later pubertal onset. Also in line with theoretical expectations were the findings indicating that children varied in the degree to which they seemed affected by rearing.

Entirely inconsistent with empirically derived expectations, however, were the results showing that girls who were low in negativity as infants appeared more susceptible to rearing effects and, moreover, that among these girls it was adverse experiences that seemed to delay pubertal development and supportive ones that seemed to accelerate it. Considered in their entirety, the results raise the prospect that some children may not only be more susceptible than others

to rearing experiences but that the actual manner in which children respond to similar experiences may vary substantially.

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