Child Development, November/December 2007, Volume 78, Number 6, Pages 1799 – 1817

Family Environments, Adrenarche, and Sexual Maturation: A Longitudinal Test of a Life History Model

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Life history theorists have proposed that humans have evolved to be sensitive to specific features of early childhood environments and that exposure to different environments biases children toward development of different reproductive strategies, including differential pubertal timing. The current research provides a longitudinal test of this theory. Assessments of family environments, based on interviews with mothers and fathers, were conducted in preschool, and children were then followed prospectively through middle childhood. Adrenal hormones were assayed in a selected subsample of 120 children (73 girls) at age 7, and parent and child reports of secondary sexual characteristics were collected in the full female sample of 180 girls at age 11. Higher quality parental investment (from both mothers and fathers) and less father-reported Marital Conflict/Depression forecast later adrenarche. Older age at menarche in mothers, higher socioeconomic status, greater mother-based Parental Supportiveness, and lower third-grade body mass index each uniquely and significantly predicted later sexual development in daughters. Consistent with a life history perspective, quality of parental investment emerged as a central feature of the proximal family environment in relation to pubertal timing.

Perhaps the most enduring mystery surrounding human sexual development is its variation: Some individuals complete pubertal development in elementary school, whereas others are still relatively undeveloped when they graduate from high school. The present study examines potential influences on variation in timing of pubertal development. Our specific focus is on familial and ecological stressors in early childhood and their effects on variation in timing of adrenarche and development of secondary sexual characteristics in early adolescence.

Variation in pubertal timing occurs in the context of a larger developmental continuum. At early points on this continuum are upstream developmental factors and processes (e.g., nutritional history, parasite loads, energetic expenditure, body mass, family composition, psychosocial stress) that influence timing of pubertal maturation (reviewed in Ellis, 2004). At later points on this continuum are downstream developmental consequences of pubertal timing. Along these lines, an extensive body of research in Western societies now indicates that early pubertal maturation in girls is associated with a variety of negative health and psychosocial outcomes, including mood disorders, substance abuse, adolescent pregnancy, and a variety of cancers of the reproductive system (e.g., Caspi & Moffitt, 1991; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Kelsey, Gammon, & John, 1993; see Mendle, Turkheimer, & Emery, 2007, for a review). Timing of pubertal maturation, therefore, not only is important for understanding variable patterns of human development but also has substantial social and biological implications.

An important emerging framework for analyzing the causes and consequences of individual differences in pubertal timing is life history theory (Charnov, 1993; Roff, 1992; Stearns, 1992). The key units of analysis in life history theory are life history traits: the suite of maturational and reproductive characteristics that determine speed of reproduction and population turnover (e.g., age at weaning, age at sexual maturity, adult body size, time to first reproduction, interbirth interval). Life history theory attempts to explain variation in life history traits in terms of trade-offs in distribution of metabolic resources to competing life functions: growth, maintenance, and reproduction. These trade-offs are inevitable because metabolic resources are finite and time and energy

This work was supported by grants from the National Institute of Mental Health (R01-MH44340, P50-MH52354) and the MacArthur Foundation Research Network on Psychopathology and Development. We are indebted to Douglas Granger for assistance with coding the adrenal hormone data and to Jay Belsky, Dejana Braithwaite, Lorah Dorn, Jane Mendle, Gregory Pettit, and Elizabeth Shirtcliff for comments on earlier drafts on this paper.

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used for one purpose cannot be used for another. For example, resources invested in growth and development cannot be spent on current production of offspring; thus, the benefits of a prolonged childhood (e.g., larger adult body size, increased social quality and competitiveness, better health in Western societies) are traded off against the costs of delayed reproduction. Each trade-off constitutes a decision node in allocation of resources, and each decision node influences the next decision node (opening up some options and foreclosing others) in an unending chain over the life course. Life history theory posits the existence of phenotypic mechanisms that actually make these trade-offs by selecting between or "making decisions" about alternative ways of distributing resources (Chisholm, 1999). Natural selection favors mechanisms that, in response to ecological conditions, trade off resources between growth, maintenance, and reproduction in ways that recurrently enhanced inclusive fitness during a species' evolutionary history. These mechanisms should be engineered to monitor evolutionarily relevant features of one's environment as a basis for contingently altering the timing and velocity of pubertal maturation (Ellis, 2004).

A central question in life history theory is: When should individuals reach sexual maturity? That is, when should individuals stop converting surplus energy into growth and begin converting it into reproduction? And most critically, what are the relevant developmental experiences and environmental cues that bias individuals toward relatively early versus late reproductive development? Life history theory provides a set of core starting assumptions (i.e., a metatheory) for addressing these questions. Within this metatheoretical framework, more specific middle-level theories have applied these assumptions to the analysis of psychosocial influences on timing of puberty, as follows.

Psychosocial Acceleration Theory

Belsky, Steinberg, and Draper (1991) were the first to propose a middle-level life history model — psychosocial acceleration theory — of the role of familial and ecological stressors in accelerating pubertal maturation in girls. They posited that "a principal evolutionary function of early experience — the first 5 – 7 years of life — is to induce in the child an understanding of the availability and predictability of resources (broadly defined) in the environment, of the trustworthiness of others, and of the enduringness of close interpersonal relationships, all of which will affect how the developing person apportions reproductive effort" (p. 650). Drawing on the concept of sensitive-period learning of reproductive strategies, Belsky et al. theorized that humans have evolved to be sensitive to specific features of their early childhood environments, and that exposure to different environments biases children toward the development of different reproductive strategies. Ecological stressors in and around the family (e.g., scarcity or instability of resources) create conditions that undermine parental functioning and lower the quality of parental investment (such as by escalating marital conflict, increasing negativity and coercion in parent – child relationships, and reducing positivity and support in parent – child relationships). The theory posits that children respond to these familial and ecological contexts by developing in a manner that speeds rates of pubertal maturation, accelerates sexual activity, and orients the individual toward relatively unstable pair-bonds. In contrast, children whose experiences in and around their families are characterized by relatively high levels of support and stability are hypothesized to develop in the opposite manner (Belsky et al., 1991). Either way, the child adaptively adjusts timing of pubertal development to match local conditions.

In a comprehensive review of the literature on psychosocial acceleration theory, Ellis (2004, pp. 935 – 936) concluded, "Empirical research to date has provided reasonable, though incomplete, support for the theory." Extant research is limited, however, because an adequate test of psychosocial acceleration theory requires assessment of the quality of family environments prior to onset of puberty (in the first 5 – 7 years of life) followed by assessment of pubertal development in adolescence.[1] Only three studies have employed this longitudinal methodology (Moffitt, Caspi, Belsky, and Silva, 1992, $N = 379$ New Zealand girls and their families; Ellis, McFadyen-Ketchum, Dodge, Pettit, and Bates, 1999, $N = 173$ American girls and their families; Belsky et al., 2007, $N = 756$ American children and their families). Moffitt et al. and Belsky et al. (2007) found significant correlations between negative aspects of family environment (e.g., family conflict, maternal harsh control) and subsequent age at menarche. Consistent with Belsky et al. (1991), greater negativity predicted earlier menarche. Ellis et al. (1999), however, failed to replicate this finding: No significant relations were found between either observation- or interview-based measures of family conflict and coercion and age-adjusted pubertal development in the seventh grade. On the other

1. Of course not even this longitudinal design can provide a truly adequate test of the theory because it cannot rule out gene effects.

hand, Ellis et al. documented significant relations between family positivity and subsequent timing of pubertal development. Consistent with Belsky et al. (1991), greater family positivity forecast later pubertal development. By contrast, Belsky et al. (2007) found that negative family experiences rather than positive ones most consistently predicted pubertal timing.

Given the paucity of relevant studies and somewhat inconsistent findings, the first goal of the present study was to test the core hypothesis from psychosocial acceleration theory—that children whose experiences in and around their families of origin are characterized by relatively high levels of psychosocial stress will develop in a manner that speeds pubertal maturation—in a long-term, prospective design in which quality of family environments (economic stress, parental psychopathology, marital relationships, and parent – child relationships) was assessed in the first 5 years of children's lives. Following Ellis et al. (1999) and Belsky et al. (2007), measures of family environment included a broad range of indicators of warmth and support as well as conflict and coercion.

Paternal Investment Theory

Paternal investment theory is a variant of psychosocial acceleration theory and is based, fundamentally, on the theorizing of Draper and Harpending (1982, 1988) and Ellis (2004). These authors hypothesized that the developmental pathways underlying variation in daughters' reproductive strategies are sensitive to the father's role in the family and mother's sexual attitudes and behavior in early childhood. As formulated by Ellis and colleagues. (Ellis, 2004; Ellis & Garber, 2000; Ellis et al., 1999, 2003), paternal investment theory posits a unique role for 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. These unique (separate) effects are hypothesized to occur because of different mediating mechanisms (i.e., pheromonal influences vs. activation of stress response systems; see Ellis, 2004). The theory posits that girls detect and internally encode information about levels of paternal investment during approximately the first 5 years of life as a basis for calibrating the development of (a) neurophysiologic systems involved in timing of pubertal maturation and (b) related motivational systems, which make certain types of sexual behavior more or less likely in adolescence. Specifically, early experiences associated with low paternal investment are hypothesized to entrain development of reproductive strategies that, during human evolution, were statistically linked to increased reproductive success in that social milieu—a milieu in which male parental investment is relatively unreliable and/or not closely linked to variation in reproductive success. Girls in this context are predicted to develop in a manner that accelerates pubertal maturation and onset of sexual activity.

Relevant cues to paternal investment are provided by both fathers and mothers. Perhaps the most important cue is father presence versus absence (i.e., the extent to which women rear their children with or without consistent help from a man who is father to the children). Other important cues may include frequency of father – daughter interactions, levels of cohesion and conflict in father – daughter relationships, the quality and stability of the father – mother relationship, mother's attitudes toward men, mother's sexual and repartnering behavior, and daughter's exposure to mother's boyfriends and stepfathers.

In his review of the literature on paternal investment theory, Ellis (2004) concluded that several key predictions had received provisional empirical support. First, in well-nourished populations, girls from father-absent homes tend to experience earlier pubertal development than do girls from father-present homes, and the earlier the father absence occurs, the greater the effect (e.g., Quinlan, 2003; Surbey, 1990). Second, better quality relationships between mothers and their male partners have been recurrently associated with later pubertal timing in daughters (e.g., Ellis & Garber, 2000; Romans, Martin, Gendall, & Herbison, 2003). Third, there is consistent evidence that father absence and martial quality uniquely predict timing of pubertal development in daughters, independent of other aspects of the family ecology (e.g., Moffitt et al., 1992; Surbey, 1990).

Despite this empirical support, there is very little research examining whether levels of cohesion and conflict in father – daughter relationships in early childhood (prior to onset of puberty) predict subsequent timing of puberty. The two studies that addressed this question (Belsky et al., 2007; Ellis et al., 1999) produced contradictory results. Whereas Belsky et al. did not find any main effects of quality of father – daughter relationships on pubertal timing, Ellis et al. found that (a) greater time spent by the father in childcare during the first 5 years of life (as reported by the mother) predicted later pubertal development in daughters, (b) higher frequencies of observed father – daughter interactions at age 5 (whether those interactions were positive or negative) forecast later pubertal development in daughters, and (c) this observed variation in father – daughter

interactions accounted for more variance in daughters' pubertal timing than did observed variation in mother – daughter interactions.

Given these inconsistent findings, a second goal of the present study was to examine a broad range of indicators of paternal (as well as maternal) investment in the first 5 years of life and then test their relations with pubertal timing. Because 94% of the target children in the current study were living in biologically intact families at the time of the preschool assessment of family environments, we could not examine the effects of family disruption/father absence on pubertal timing. However, within those intact families, both fathers and mothers were intensively interviewed. These interviews provided the basis for testing a key hypothesis of paternal investment theory: that quality of paternal investment within families uniquely predicts timing of pubertal development, separate from the effects of maternal investment.

Mediating Mechanisms

What are the mechanisms through which variations in family environments potentially influence timing of pubertal development? Ellis (2004; see also Cameron et al., 2005) has reviewed some candidate mechanisms, including alterations in sympathetic adrenomedullary reactivity, changes in glucocorticoid profiles, and exposure to pheromones. Another possibility is diet and its effects on growth in weight and fat. There is some evidence that experiences of stress in children precede weight gain (Lissau & Sorensen, 1994; Mellbin & Vuille, 1989a, 1989b). This effect may be mediated by shifts in dietary preferences. Specifically, rats that are experimentally stressed increase consumption of high-fat and high-sugar foods in their diet (i.e., ''comfort foods'') relative to consumption of rat chow, resulting in increases in fat depots and parallel increases in leptin and insulin (Pecoraro, Reyes, Gomez, Bhargava, & Dallman, 2004). Consumption of such comfort foods functions to reduce both autonomic and adrenocortical reactivity to stress (Dallman, Pecoraro, & la Fleur, 2004). As reviewed by Dallman et al., humans, like rats, have also been found to disproportionately consume comfort foods during times of stress. Accordingly, children growing up under conditions of high familial and ecological stress may be disposed to overconsume high-fat and high-sugar foods. Children from low socioeconomic status (SES) families may also tend to consume such foods because they are inexpensive and readily available (inverse relations between childhood SES and body mass index [BMI] are well documented; e.g., Booth, Macaskill, Lazarus, & Baur, 1999; Dekkers et al., 2004; Lamerz et al., 2005).

Beginning at approximately ages 5 – 7 in girls, greater weight and BMI are associated with earlier development of secondary sexual characteristics and attainment of menarcheal status (Cooper, Kuh, Egger, Wadsworth, & Barker, 1996; Davison, Susman, & Birch, 2003; Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001; St. George, Williams, & Silva, 1994; Wang, 2002). Although this association in pubertal age children may be influenced by the effects of gonadal estrogens on both fat mass and sexual maturation (Ellison, 2001), higher percentages of body fat at ages 5 – 7 both precede and predict onset of puberty, even after controlling for height (Davison et al., 2003). This gain in fat may be one of the key signals (possibly through secretion of the fat-derived protein leptin) for stimulating the hypothalamus to increase secretion of gonadotropin-releasing hormone (GnRH) (Wilson et al., 2003), which in turn stimulates the pituitary – ovarian axis and initiates the pubertal cascade.

In total, the accelerating effects of childhood stress and low SES on pubertal development may operate through accumulation of prepubertal fat. In the current research, a crude estimate of fatness—BMI—was taken in the third-grade data collection. We hypothesized that BMI would partially mediate the effects of familial and ecological stressors in early childhood on timing of pubertal development.

Potential Genetic Influences

In stable, well-nourished populations, allelic variations have considerable influence on timing of pubertal maturation (reviewed in Ellis, 2004). It follows, therefore, that observed correlations between family environments and pubertal timing could be genetically confounded. One possibility is that parents who experience early puberty not only pass on genes to their children for early puberty but also pass on dysfunctional family environments that are a downstream consequence of early pubertal and sexual development (e.g., Moffitt et al., 1992; Surbey, 1990). Another possibility is that correlated variation in pubertal timing and family environments is regulated, in part, by the same alleles (Comings, Muhleman, Johnson, & MacMurray, 2002). The correlational methods used in the present study cannot rule out these potential genetic confounds. However, the current analyses do incorporate an important control variable—mother's age at menarche—that should account, at least in part, for possible genetic influences.

Assessment of Pubertal Timing

Timing and tempo of pubertal development are regulated by the functional maturation of the adrenal glands (adrenarche) and the hypothalamic – pituitary – gonadal (HPG) axis (gonadarche). Adrenarche and gonadarche, which are largely independent processes, are responsible for increased secretion of sex steroids during the peripubertal and pubertal periods. Adrenarche has been described as the ''awakening of the adrenal glands'' and occurs at approximately 6 – 8 years of age in both boys and girls (Dorn & Chrousos, 1997; Grumbach & Styne, 2003). Adrenarche represents a distinct time in adrenal development when levels of the adrenal androgen dehydroepiandrosterone (DHEA) and its sulfate begin to rise. The development of pubic hair, increased skeletal maturation, increased oil on the skin, and body odor are all thought to represent physiological manifestations of increased concentrations of adrenal androgens (Dorn & Chrousos, 1997; McClintock & Herdt, 1996). In the current study, adrenarcheal status was assessed through salivary assays of DHEA when children were approximately 7 years old (in the summer after first grade). Girls and boys do not differ in levels of adrenal androgens at this age (Tung, Lee, Tsai, & Hsiao, 2004) and thus were analyzed together. To our knowledge, this is the first study to examine relations between early familial and ecological stressors and subsequent timing of adrenarche.

Gonadarche occurs at approximately 9 or 10 years of age in girls and soon thereafter in boys (Dorn, Dahl, Woodward, & Biro, 2006; Grumbach & Styne, 2003), although actual ages vary widely across and within populations. Gonadarche constitutes a second phase of pubertal development that begins with reactivation of pulsatile secretion of GnRH after a period of relative quiescence during childhood. This reactivation triggers a cascade of pubertal events, including maturation of primary sexual characteristics (ovaries and testes) and full development of secondary sexual characteristics (pubic hair, breasts, and genitalia) (see Dorn et al., 2006; Ellison, 2002; Grumbach & Styne, 2003, for overviews of the neurophysiology of puberty). In the current research, we did not include a direct measure of gonadarche. Instead, we collected parent and child reports of development of secondary sexual characteristics in the fifth grade (ages 10 – 11). Although secondary sexual development has typically already begun for females in this age group, this is not the case for the large majority of boys (e.g., Dick, Rose, Pulkkinen, & Kaprio, 2001; Herman-Giddens, Wang, & Koch, 2001; Papadimitrou, Stephanou, Papantzimas, Glynos, & Philippidis, 2002). Consequently, the current study only reports data on development of secondary sexual characteristics in girls.

In total, guided by psychosocial acceleration theory and paternal investment theory, the current study tests hypotheses concerning the effects of familial and ecological context on timing of pubertal maturation. Assessments of the child's psychosocial environment were conducted in preschool, followed by assessment of adrenarcheal status at age 7 and secondary sexual development in girls at age 11. The following hypotheses were tested that: (a) greater exposure to familial and ecological stressors (more conflict – coercion and less warmth – support) forecasts earlier adrenal and secondary sexual development; (b) quality of paternal investment uniquely predicts adrenal and secondary sexual development, separate from the effects of maternal investment; and (c) BMI mediates the effects of SES and proximal family environment on secondary sexual development. Mother's age at menarche was included in the analyses as a partial control for genetic confounds.

Method

Participants

The children in this study represent a subset of those participating in an ongoing longitudinal study of child development, the Wisconsin Study of Families and Work (WSFW; see Hyde, Klein, Essex, & Clark, 1995). The original sample comprised 570 women and their partners recruited during the second trimester of pregnancy from obstetric/gynecology clinics and a low-income clinic in Milwaukee (80% of sample) and Madison (20%). We attempted to recruit as diverse a sample as possible in regard to ethnic heritage and social class, subject to the constraints that mothers and fathers had to be cohabiting when recruited and obtaining prenatal care. Families were excluded if women were age 18 or younger, unemployed, a student, or disabled. At initial recruitment (pregnancy), 95% of the 570 couples were married, 89% were Caucasian, and mothers' average age was 29.4 years (range = 20 – 43 years) and fathers' was 31.3 years (range = 20 – 55 years). Mothers and fathers each had an average of 15 years of education (range = less than high school to professional degree); annual family income ranged from $7,500 to over $200,000 (median = $45,000).

Eleven waves of data have been collected from pregnancy through the infancy, preschool, and elementary school years. At the child age 4.5-year

assessment (T7), approximately 500 families remained in the study, and at the Grade 5 assessment (T11), approximately 400 families remained in the study. The present study relies primarily on the data from the preschool assessments (T6 and T7) for assessing the family environment. The two primary outcomes are (a) adrenarche at Grade 1 (T9; age range = 6.8 – 7.8 years; mean = 7.25; *SD* = 0.24) on a selected subsample of 120 children (73 girls; all mothers and 111 fathers in this group participated in the preschool family environment assessments) and (b) secondary sexual characteristics at Grade 5 (T11; age range = 10.5 – 11.9 years; mean = 11.18; *SD* = 0.29) for the full sample of 180 girls (and their mothers) who completed puberty ratings in this wave of data collection (all mothers and 157 fathers in this group participated in the preschool family environment assessments).

The subsample of 120 children was selected from the first cohort of the WSFW in the summer following the first grade to participate in an intensive substudy to develop the MacArthur Assessment Battery for Middle Childhood (Boyce et al., 2002). Because we wanted a balance of ''high''- and ''low''-symptom children for the substudy, we employed a broad definition to classify all WSFW kindergarteners as ''high symptoms'' if they were in the upper 20% of either internalizing or externalizing problems as reported by either mothers or teachers and ''low symptoms'' otherwise. This resulted in an approximately equal number of children classified as high symptoms and low symptoms. The subset of 120 children was randomly selected from each of these two symptom groups to be representative of the larger sample. Thus, there were no statistically significant differences between the demographic profiles of these 120 families and the remainder of the 570 original families. In addition, there were no differences in parents' marital status, ethnicity, fathers' age, mothers' or fathers' educational levels, or total annual family income between the 180 girls participating in the Grade 5 assessment and the remainder of girls in the original sample. There was, however, a minor difference in mothers' age: mothers of the 180 girls were slightly older than mothers of the nonparticipating girls (M = 29.9 vs. 28.8 years of age, $t(281) = -2.22$, $p = .027$). This 1-year difference (0.25 *SD*s) is presumably not substantively meaningful in this age range (20 – 43 years).

Measures: Ecological Stress

Socioeconomic status. SES was employed as the measure of ecological stress. A composite measure of SES across the infancy and preschool periods was calculated on the basis of family income (at the 12-month and 4.5-year assessments) and father's and mother's education (in years). The four measures were combined using principal components analysis (PCA) and saving component scores. There was a single component, accounting for 54% of the variance.

Measures: Family Environment (Preschool)

Ten measures of proximal family processes, or adjustment factors that directly impinge on family processes (i.e., parental depression), were assessed in the preschool period. Parental depression, family negativity and positivity, marital compatibility and conflict, and parental security were included in both preschool assessments (T6, child age 3.5 years; T7, child age 4.5 years); because the measures were quite stable across the two assessments (correlations exceeding .50), they were averaged to obtain a more reliable estimate of each proximal family factor during the preschool period. The remaining four measures (i.e., parental negativity and positivity toward child and authoritarian and authoritative parenting styles) were assessed only at T7. For all measures, data were collected from both mothers and fathers.

Parental depression. Parental depression was assessed by the Center for Epidemiological Studies-Depression scale (Radloff, 1977), a well-established self-report measure which asks respondents how frequently they experienced each of 20 symptoms during the past week (e.g., ''could not shake off the blues,'' ''bothered by things that don't usually bother you''), rated from 0 (*rarely or none of the time*) to 3 (*most or all of the time*). Alpha coefficients were above .85 for mothers and fathers at both assessments.

Family negativity and positivity. The Family Expressiveness Questionnaire (FEQ; Halberstadt, 1986, 1991) tapped mothers' and fathers' perceptions of the family affective environment. Questions covered the frequency of both negative and positive examples of family expressiveness rated from 1 (*not at all frequently in my family*) to 9 (*very frequently*). The Family Negativity scale comprises the FEQ subscales for Negative Dominant (e.g., ''Blaming one another for family troubles'') and Negative Submissive (e.g., ''Sulking over unfair treatment by a family member'') behaviors. The Family Positivity scale comprises the FEQ subscales for Positive Dominant (e.g., ''Showing forgiveness to someone who broke a favorite possession'') and Positive Submissive (e.g., ''Expressing gratitude for a favor'') behaviors. Alpha coefficients for each scale were above .80 for mothers and fathers at both assessments.

Marital compatibility and conflict. The Partner Role Quality Scale (Barnett & Marshall, 1989) measures both positive features (rewards) and negative aspects (concerns) of the marital relationship. Both mothers and fathers were asked a series of items, rated on a 4-point scale from 1 (*not at all*) to 4 (*extremely*), to assess how rewarding their marriage was in terms of compatibility (nine items, e.g., ''you have a partner who is easy to get along with,'' ''your partner likes you as a person'') and how concerned they were about marital conflict (three items, e.g., ''arguing or fighting''). Alpha coefficients for each scale were above .80 for mothers and fathers at both assessments.

Parental insecurity. The parenting stress index (PSI) (Abidin, 1986) is a parent self-report instrument designed to yield a measure of the relative magnitude of stress in the parent – child system and to identify various sources of stress. The Attachment subscale was used in the present study to assess parental insecurity. Mothers and fathers were asked seven items, answered on a 1 (*strongly disagree*) to 5 (*strongly agree*) scale, tapping the degree of emotional closeness felt with the child and the ability to understand the child's feelings and/or needs (e.g., ''It takes a long time for parents to develop close, warm feelings for their children,'' ''I expected to have closer and warmer feelings for my child than I do and this bothers me''). Alpha coefficients ranged from .55 to .75 for mothers and fathers at both assessments. Higher scores indicated less attachment security.

Parental negativity. Parental negativity was assessed with a composite (PCA) of three scales. Two subscales of the PSI (see above) assessed mothers' and fathers' Sense of Competence (11 items; e.g., ''I feel capable and on top of things when I am caring for my child,'' ''I have had many more problems raising children than I expected''; alpha coefficients above .79) and Reinforcement received from their child (6 items; e.g., ''My child rarely does things for me that make me feel good''; alpha coefficients above .66). Three items from the *Child-Rearing Practices Report* (CRPR; Block, 1965), answered on a 7-point scale from 1 (*extremely untrue*) to 7 (*extremely true*), were used to assess maternal and paternal negative affect toward their child (i.e., ''There is a great deal of conflict between me and my child,'' ''I often feel angry with my child,'' ''I feel my child is a bit of a disappointment to me''; alpha coefficients above .62).

Parental warmth/positivity. Parental warmth/positivity was assessed with four items (e.g., ''My child and I have warm intimate times together,'' ''I express affection by hugging my child'') from the CRPR (Block, 1965) (see above). Alpha coefficients were above .60 for mothers and fathers.

Authoritative – democratic and Authoritarian – restrictive parenting styles. Two scales were derived from the CRPR (Block, 1965) tapping parenting styles characterized as Authoritative – democratic or Authoritarian – restrictive. The Authoritative scale comprised seven items (e.g., ''I reason with my child when s/he misbehaviors,'' ''I believe that praising gets better results than punishment,'' ''I encourage my child to talk about his/her troubles''). The Authoritarian scale comprised seven items (e.g., ''I teach my child to control his/her feelings,'' ''I do not allow my child to question my decisions,'' ''I believe that scolding and criticism is good for a child''). Alpha coefficients were above .65 for mothers and fathers.

Derivation of family environment measures. In order to reduce the array of family environment measures into a smaller number of components that extracted their shared variance, we subjected the family environment measures to PCA. Past research indicates that two overarching dimensions—familial warmth – closeness – positivity and familial conflict – coercion – negativity—capture the most fundamental ways in which interpersonal family environments differ (e.g., Belsky, Hsieh, & Crnic, 1998; Hetherington & Clingempeel, 1992; Pettit, Bates, & Dodge, 1997). We thus expected these two basic dimensions to emerge in the current analyses. Because extant theory and data suggest that quality of maternal investment and quality of paternal investment have unique effects on timing of pubertal development (Ellis, 2004), mother and father data were factor analyzed separately.

PCA of mother data. PCA was performed on the 10 maternal self-report measures of family environment ($N = 428$). The PCA was followed by oblique rotation to allow the components to covary (if warranted by the data). On the basis of eigenvalue scree, two components were extracted. The two components were easily interpretable and displayed a clean, simple structure. The first component (alpha reliability = .79) clearly indexed parent – child supportiveness and authoritative parenting, with high structure loadings on parental negativity and dissatisfaction (−.76), warm – positive parental behavior (.74), authoritative – democratic parenting style (.74), parental insecurity (−.72), and family positivity (.62). The second component (alpha reliability = .69) clearly tapped marital quality and parental depression, with high loadings on marital conflict (.83), marital compatibility (−.82), parental depression (.58), and family negativity (.57).

PCA of father data. PCA, followed by oblique rotation, was also performed on the 10 paternal self-report measures of family environment ($N = 346$). As

in the analysis of the maternal measures, two components were extracted on the basis of eigenvalue scree. As delineated in the structure matrix, the two components were again clean and readily interpretable, and these components were virtually identical to those in the mother data. The first component (alpha reliability = .86) clearly tapped parent – child supportiveness and authoritative parenting, with high structure loadings on authoritative – democratic parenting style (.84), warm – positive parental behavior (.84), parental negativity and dissatisfaction (−.81), parental insecurity (−.76), and family positivity (.63). The second component (alpha reliability = .73) clearly indexed marital quality and depression, with high loadings on marital conflict (.88), marital compatibility (−.78), depression (.68), and family negativity (.55).

Estimation of component scores. To derive the main family environment measures used in this study, each measure in each of the PCAs was weighted by its component score regression coefficients in a linear composite. This resulted in two mother components and two father components. Thus, the final four components used in this study were maternal and paternal Parental Supportiveness (a direct measure of the quality of parent – child relationships) and maternal and paternal Marital Conflict/Depression (a more general measure of parental relationships and mental health). Component scores are standardized variables with means of 0 and standard deviations of 1.

Because PCA results in listwise deletion, some of the mothers and fathers who participated in the preschool assessment of family environments were excluded from the analysis. To recover these lost cases, missing values were imputed using the expectancy-maximization (EM) algorithm (see Little & Rubin, 2002), which constructed a complete data matrix based on patterns displayed by the nonmissing scores on all family environment measures in the WSFW. The EM algorithm was implemented through the Missing Values Analysis module in SPSS 15.0. (The imputation of missing values did not have an overall directional effect on the size of the relations between the family environment components and the pubertal outcome measures.)

Measures: Physical and Pubertal Development

Mother's age at menarche. At the third-grade data collection, mothers reported on their age at menarche (estimated to the nearest year). The reliability of retrospective reports of age at menarche has been established in several long-term prospective studies in which self-reported age at menarche was first obtained in adolescence and then again 17 – 37 years later. Correlations across these two measurement periods have been consistently high, ranging from .67 to .79 (Casey et al., 1991; Damon, Damon, Reed, & Valadian, 1969; Livson & McNeill, 1962; Must et al., 2002). In the current sample, mothers' mean age of menarche was 12.55 years (SD = 1.32).

Adrenarche. To assess maturation of the adrenal axis (adrenarche), salivary DHEA was collected four times over the course of a 4-hr, in-home visit. To avoid circumstances known to interfere with the assay of DHEA (Granger, Schwartz, Booth, Curran, & Zakaria, 1999; Granger, Shirtcliff, Booth, Kivlighan, & Schwartz, 2004), unstimulated passive drool was collected by expectoration into a plastic cup. Samples were transported on dry ice from the families' homes to the laboratory immediately after the home visit, temporarily stored at −20°C for prelaboratory accounting, and then transported overnight on dry ice to Salimetrics laboratories and stored frozen at −80°C until assayed for DHEA. On the day of testing, all samples were centrifuged at 3,000 rpm for 15 min to remove mucins. All samples were assayed for DHEA using a commercially available enzyme immunoassay specifically designed for use with saliva; the manufacturer's (Salimetrics, State College, PA) recommended protocols were followed without modification. The test used 50 μl of saliva with a range of sensitivity from 10.0 to 1,000 pg/ml. All samples were assayed in duplicate, resulting in a total of eight assays. The average intra- and interassay coefficients of variation for these assays were well within the recommended guidelines of less than 10% and 15%, respectively (Chard, 1990). To minimize variation, all samples from the same participant were tested in the same assay batch.

To maximize the probability of detecting DHEA, which has been shown to increase in response to social challenge (Shirtcliff, Zahn-Waxler, Kllimes-Dougan, Granger, & Slattery, 2006), saliva samples were collected during a time of social challenge. Specifically, a team of research assistants, unknown to the child, arrived at the home to conduct a series of tasks that involved social negotiations with the research assistants and were designed to activate the stress response systems. As described in Ellis, Essex, and Boyce (2005), the protocol constituted a series of mildly stressful, ecologically valid tasks, representing different stressor domains (i.e., social, cognitive, physical, and emotional). Samples were collected first immediately following the arrival of the study team at the child's home, second approximately 90 min later (prior to the stress reactivity protocol), third approximately 1 hr later (following completion of the stress reactivity protocol), and fourth approximately 90 min later at the end of the visit.

Participants were coded as preadrenarcheal (45%) if at least six of their eight DHEA assays were below the detection threshold (10.0 pg/ml) and all DHEA scores were <16 pg/ml. All other participants were coded as adrenarcheal (55%). Consistent with past research on 6- to 8-year-old children (Tung et al., 2004), boys (53%) and girls (56%) did not significantly differ in rates of adrenarche. Further, within the narrow age range of the current sample, adrenarcheal status was not associated with age, $F(1, 118) = 0.29$. The current rate of adrenarche (55%) based on salivary DHEA assays converges with previous research on rates of adrenarche in 6- to 8-year-olds based on serum assays (Tung et al., 2004).

Because increasing DHEA levels at adrenarche cause subsequent pubic hair growth (Grumbach & Styne, 2003), Tanner staging of pubic hair development in the fifth grade, based on self-rating of gender-appropriate Tanner drawings, was used as a criterion variable to validate the adrenarche coding. Tanner self-ratings of pubic hair among early adolescents have documented validity in relation to physician Tanner ratings (Schmitz et al., 2004). Boys who were coded as preadrenarcheal displayed significantly less pubic hair development (Tanner stage: $M = 1.8$) than boys who were coded as adrenarcheal (Tanner stage: $M = 2.5$), $t(30) = -3.03$, $p < .01$. Likewise, girls who were coded as preadrenarcheal displayed significantly less pubic hair development (Tanner stage: $M = 2.1$) than girls who were coded as adrenarcheal (Tanner stage: $M = 2.7$), $t(55) = -2.11$, $p < .05$.

Body mass index. Height and weight measurements were taken at the third-grade (mean age = 8.79 years, $SD = 0.25$) and fifth-grade (mean age = 10.49 years, $SD = 0.30$) data collections. Shoes were removed before the height and weight measurements. Height was measured against a wall, using a centered level on the child's head. Repeated height measurements were taken until two measurements were within ¼ inch of each other. The two closest readings were averaged to create the measure of children's height. Using a lithium electronic scale, repeated weight measurements were also taken until two measurements were within ½ pound of each other. The two closest readings were averaged to create the measure of children's weight. Average heights and weights were 54 in and 76 lb in the third grade and 58 in and 94 lb in the fifth grade. BMI was calculated as weight (kg)/height (m^2). Mean BMI was 18.14 ($SD = 3.27$) in the third grade and 19.27 ($SD = 3.74$) in the fifth grade. Typical of Wisconsin, the sample exceeded U.S. norms on BMI: On average, girls placed at the 60th and 58th percentiles and boys at the 62nd and 61st percentiles at Grades 3 and 5, respectively, based on sex-specific BMI-for-age growth charts from the Centers for Disease Control and Prevention. The third-grade BMI measure, standardized for age using Center for Disease Control and Prevention 2000 Growth Charts for the United States (Ogden et al., 2002), was used in the primary data analyses. Because the standardized third-grade and fifth-grade BMI-for-age measures were strongly correlated, $r(167) = .90$, missing values at third grade ($n = 5$) were replaced by the fifth-grade values.

Secondary sexual characteristics. Given the young age of the sample, the current research only focused on development of secondary sexual characteristics and not menarche, which is a late pubertal event (only about 10% of girls were menarcheal). At the fifth-grade data collection, 177 mothers of girls in the study and 145 girls answered questions concerning the girl's level of pubertal development. Mothers completed both a questionnaire version (Carskadon & Acebo, 1993) of the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988; 4-point response scales) and ratings of Tanner stages (anatomical sketches; five developmental stages). Girls only completed ratings of Tanner stages. Using the line drawings of Morris and Udry (1980), the Tanner ratings indexed pubic hair and breast stages. As summarized in table 2 of Dorn et al. (2006), past research indicates that both parent ratings and self-ratings of female pubertal development, using either the PDS or the Tanner drawings, are moderately to highly correlated with ratings by health-care professionals based on physical examinations.

The following steps were taken to derive a measure of female pubertal development. Mother's PDS ratings of breast development ($M = 2.34$, $SD = 0.66$) and body hair growth ($M = 2.14$, $SD = 0.87$) correlated .61 ($N = 176$) and were thus standardized and averaged. Similarly, mother's Tanner ratings of breast stage ($M = 2.43$, $SD = 0.84$) and pubic hair ($M = 2.18$, $SD = 1.04$) correlated .70 ($N = 144$) and were thus standardized and averaged. These two composite measures correlated .82 ($N = 171$) and were thus averaged to form an overall mother report measure of daughters' secondary sexual characteristics. The daughter's Tanner ratings of breast stage ($M = 2.60$, $SD = 0.89$) and pubic hair ($M = 2.60$, $SD = 1.10$) correlated .68 ($N = 144$) and were thus standardized and averaged to form an overall self-report measure of secondary sexual characteristics. The mothers' and daughters' overall measures correlated .81 ($N = 141$) and were thus averaged to form the final, composite measure of girls' pubertal development. (At each computational step, if either data source was missing, the derived score was based solely on the other nonmissing

source.) This measure was then converted into a pubertal timing measure—an index of levels of sexual development compared with same-age peers—by partialing out the child's age. This pubertal timing measure ($M = 0.00$, $SD = 0.85$, range = −1.75 – 1.64) was then used in all data analyses. Higher scores indicated earlier pubertal timing (i.e., greater pubertal development in fifth grade, residualized for age).

Two criterion variables were used to validate the female pubertal development measure: height and weight. Greater height and weight, both in the third grade and fifth grade, should be associated with more pubertal development in the fifth grade (given that height and weight both predict and correlate with female pubertal development; e.g., Cooper et al., 1996; Kaplowitz et al., 2001; St. George et al., 1994). Both these validation predictions were supported: third-grade height, $r(177) = .36$, and weight, $r(175) = .38$, and fifth-grade height, $r(173) = .51$, and weight, $r(172) = .49$, were all moderately to strongly positively correlated with the pubertal development measure. These results, together with the strong correlation across mother and daughter data sources, suggest that the current measure constitutes a viable means of assessing variation in female pubertal development.

Results

Intercorrelations Among Family Environment Measures

Intercorrelations among family environment measures are shown in Table 1. As noted above, previous research has documented that interpersonal family environments have largely independent positive and negative dimensions. Consistent with this research, Parental Supportiveness and Marital Conflict/Depression were only weakly ($r = -.15$ in the mother data) to moderately ($r = -.32$ in the father data) negatively correlated. Further, there were moderate to strong positive correlations between mother-based and father-based Parental Supportiveness ($r = .27$) and Marital Conflict/Depression ($r = .51$). These positive correlations indicate only modest convergence between mothers and fathers in specific ratings of parent – child relationships (i.e., in mothers' perceptions of mother – daughter relationships vs. fathers' perceptions of father – daughter relationships) but substantial convergence between mothers and fathers in their perceptions of mother – father relationships and parental functioning more generally.

Effects of Ecological Stress, Parental Functioning, and Parent – Child Relationships on Pubertal Timing

Adrenarche. To test the hypothesis that early experiences in and around the family that are characterized by relatively high levels of ecological stress (lower SES), high levels of parental dysfunction (higher Marital Conflict/Depression), and low-quality parental investment (lower Parental Supportiveness) lead children to develop in a manner that accelerates adrenarche, we conducted a series of one-way analyses of variance. These analyses did not adjust for mother's age at menarche because maturation of the adrenal glands and maturation of the HPG axis (which regulates menarche) constitute separate systems that are largely independent (Apter & Vihko, 1983; Ibanez et al., 1992). As shown in Table 2, in each analysis, children who had and had not experienced adrenarche by the end of first grade were compared on mean levels of the preschool family environment components and SES. As predicted by the theory, attainment of adrenarcheal status was forecast by lower levels of Parental Supportiveness in preschool.

Table 1
Intercorrelations Among Predictor Variables

Measure	1	2	3	4	5	6
1. SES	—					
Components derived from mother data						
2. Parental Supportiveness	.13 (180)	—				
3. Marital Conflict/Depression	−.04 (180)	−.15* (180)	—			
Components derived from father data						
4. Parental Supportiveness	.16* (157)	.27** (157)	−.24** (157)	—		
5. Marital Conflict/Depression	−.05* (157)	−.03 (157)	.51*** (157)	−.32*** (157)	—	
Biological measures						—
6. Mother's age at menarche	−.06 (156)	.14 (156)	.08 (156)	.00 (137)	.21* (137)	
7. Third-grade BMI	−.14 (180)	−.12 (180)	.18* (180)	−.14 (157)	.07 (157)	−.07 (156)

Note. Number of participants for each analysis is given in parentheses. All significance tests are two tailed.
*$p < .05$. **$p < .01$. ***$p < .001$.

Table 2
Effects of SES and Family Environment Components on Adrenarcheal Status and Secondary Sexual Development

Preschool family environment measures	Adrenarcheal status: age 7 (girls and boys)			Age-adjusted secondary sexual development: fifth-grade girls
	Preadrenarcheal group *M*	Adrenarcheal group *M*	*F*	*r*
1. SES	−.02	−.11	.31 (1, 118)	−.25** (178)
Components derived from mother data				
2. Parental Supportiveness	.30	−.09	4.39* (1, 118)	−.29*** (178)
3. Marital Conflict/Depression	−.09	.18	2.02 (1, 118)	.07 (178)
Components derived from father data				
4. Parental Supportiveness	.29	−.15	5.41* (1, 109)	−.15† (155)
5. Marital Conflict/Depression	−.22	.23	6.70* (1, 109)	.13 (155)

Note. Degrees of freedom for each analysis are given in parentheses.
†$p = .06$. *$p < .05$. **$p < .01$. ***$p < .001$.

Specifically, children who were adrenarcheal in the first grade were approximately 0.5 standard deviation lower in Parental Supportiveness. This finding replicated across both mother and father data sources. Also consistent with the theory, higher levels of father-reported Marital Conflict/Depression in preschool predicted higher rates of adrenarche. Specifically, children who were adrenarcheal in the first grade were approximately 0.5 standard deviation higher in father-reported Marital Conflict/Depression. However, rates of adrenarche were not significantly associated with variation in either mother-reported Marital Conflict/Depression or SES.

To test the hypothesis that quality of paternal investment within families uniquely predicts timing of pubertal development, separate from the effects of maternal investment, a multiple logistic regression analysis was conducted. Adrenarcheal status was the dependent variable, and the three family environment components with significant zero-order relations with adrenarche (maternal and paternal Parental Supportiveness and paternal Marital Conflict/Depression) were simultaneously entered into the equation as predictor variables. Only paternal Marital Conflict/Depression emerged as a statistically significant predictor of adrenarche. Specifically, when controlling for maternal and paternal Parental Supportiveness, a 1 standard deviation increase in paternal Marital Conflict/Depression increased the odds of experiencing adrenarche by approximately 1.7 times: $N = 111$, $B(SE = 0.25) = .52$, $\chi^2 = 4.34$, $p = .037$, OR = 1.68. In addition, when the foregoing logistic regression analysis was run as a hierarchical regression, with maternal Parental Supportiveness entered on the first step and paternal Parental Supportiveness and Marital Conflict/Depression entered on the second step, the second step was statistically significant ($\chi^2 = 8.04$, $p = .018$) and resulted in a 9% increment in Nagelkerke R^2 (from .05 to .14). In sum, consistent with paternal investment theory, the paternally based measures of family environment accounted for unique variance in adrenarcheal timing, above and beyond the effects of the maternally based measures.

Development of secondary sexual characteristics. To initially examine the hypothesis that early experiences in and around the family that are characterized by relatively high levels of ecological stress (lower SES), high levels of parental dysfunction (higher Marital Conflict/Depression), and low-quality parental investment (lower Parental Supportiveness) lead girls to develop in a manner that accelerates development of secondary sexual characteristics, we calculated a series of correlation coefficients. As predicted by psychosocial acceleration theory, higher levels of both SES and Parental Supportiveness in preschool were associated with significantly less development of secondary sexual characteristics in the fifth grade (Table 2). The effects of Parental Supportiveness largely replicated across both mother and father data sources (though the effect was only marginally significant in the father data). Levels of Marital Conflict/Depression, however, were not significantly correlated with development of secondary sexual characteristics.

The data on development of secondary sexual characteristics provided little support for paternal investment theory. Neither of the two paternal family environment measures alone nor the two measures entered together accounted for unique, significant variance in secondary sexual development, above and beyond the effects of maternal Parental Supportiveness.

Path Analysis of Development of Secondary Sexual Characteristics

In order to examine the larger set of relations among variables and incorporate appropriate controls and mediators into the analysis of secondary sexual characteristics, path analyses were conducted. Using SAS Version 9.1 (PROC CALIS procedure), analyses were performed on the variance – covariance matrix and employed the maximum likelihood parameter estimation method. To ensure adequate power (sample size), adrenarche was not included in this analysis. In addition, because neither of the paternally based measures of family environment were statistically significant predictors of secondary sexual development, these measures were also excluded from the analyses. A total of 156 girls and their families had data on all the necessary variables and were thus included in the path analysis. (The reduction from 180 to 156 subjects was due to missing data on mother's age at menarche; unfortunately, there were not sufficient covariates in the data set to impute missing values on this variable.) The analysis incorporated indicators of ecological stress (SES), parental functioning (maternal Marital Conflict/Depression), and parent – child relationship quality (maternal Parental Supportiveness). In addition, mother's age at menarche was included as a control variable and third-grade BMI was included as a potential mediator. Paths were estimated from each of these variables (except Marital Conflict/Depression, which was uncorrelated with pubertal timing; see Table 2) to daughter's development of secondary sexual characteristics in the fifth grade (Figure 1). In addition, based on past research (e.g., Ellis et al., 1999), we expected maternally based measures of Parental Supportiveness and Marital Conflict/Depression to be negatively correlated and thus estimated this path in the model (see two-headed arrow in Figure 1). Finally, to test mediational hypotheses, we considered estimating paths from Parental Supportiveness, Marital Conflict/Depression, and SES to BMI. However, as shown in Table 1, neither maternal Parental Supportiveness nor SES correlated with BMI; thus, the effects of these variables could not be mediated by BMI. Only the path from Marital Conflict/Depression to BMI, therefore, was estimated in the model.

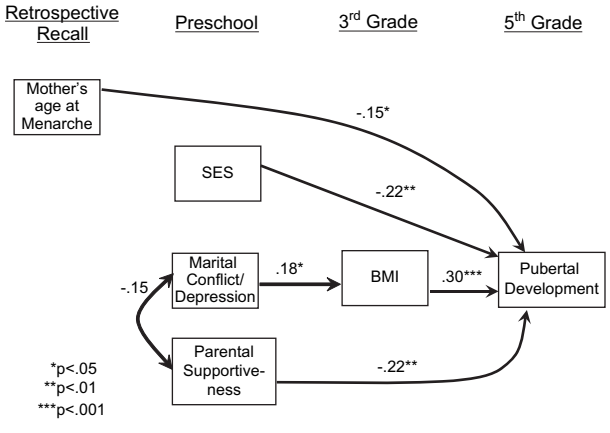

Figure 1. Path analysis of development of secondary sexual characteristics in daughters ($N = 139$). Preschool family environment measures based on maternal report. All path coefficients are standardized (beta-weights).

In the initial run of the path analysis, the expected negative correlation between maternal Parental Supportiveness and Marital Conflict/Depression was not statistically significant. However, in the full sample of girls, these variables were significantly negatively correlated (see Table 1). Because this correlation was theoretically relevant and present in the full sample, it was retained in the path analysis.

The final path analysis is shown in Figure 1, $\chi^2(4, N = 156) = 6.08$, $p = .19$; comparative fit index = .96; root mean square error of approximation = .06. This final model accounted for about one fourth of the variance ($R^2 = .26$) in age-adjusted development of secondary sexual characteristics in daughters. As can be seen in Figure 1, mother's age at menarche, SES, maternal Parental Supportiveness, and BMI each significantly and uniquely predicted pubertal timing. Older age at menarche in the mother, higher SES, and greater Parental Supportiveness were all associated with less pubertal development in the fifth grade, whereas higher BMI was associated with more fifth-grade pubertal development. The inclusion of mother's age at menarche in the model is important because it partially controls for genetic influences. Although BMI had the largest effect on pubertal timing, it did not act as a mediating variable, as had been hypothesized, because none of the variables that predicted pubertal timing also predicted BMI. Conversely, although higher levels of maternal Marital Conflict/Depression were associated with greater BMI, Marital Conflict/Depression did not directly predict pubertal timing, and thus, there was no relation to mediate. Nonetheless, there was a statistically significant indirect effect of maternal Marital Conflict/Depression on daughter's fifth-grade pubertal development through BMI (Sobel test: $z = 2.14$, $p < .05$).

Exploratory Analyses

Exploratory receiver operator characteristics (ROC) analyses (Hoblyn et al., 2006; Kiernan, Kraemer, Winkleby, King, & Taylor, 2001) were conducted to

identify those dimensions of family environment that, in combination with each other, were mostly closely associated with adrenarcheal or pubertal status. When conducting ROC analyses, it is necessary to identify a "gold standard" or binary outcome criterion of interest. For our two ROC analyses, the gold standards were attainment of adrenarcheal status in the first grade (55% of boys and girls) and mid-level secondary sexual development in the fifth grade (37% of girls; defined as Tanner Stage 3 or above [composited across pubic hair and breast development], based on the average of mother and daughter ratings of Tanner diagrams). Using a signal detection algorithm, the ROC software (ROC Version 4, publicly available at http://mirecc.stanford.edu) searches a given set of predictor variables to determine which predictors, and which cut points within those predictors' range of values, best discriminate between children who do and do not meet the gold standard. Compared with regression interaction analyses, signal detection methods provide a more interpretable and useful result when frequent or higher order interaction effects are anticipated or encountered. In the present analyses, five predictor variables reflecting children's experiences of stress and support in and around their families of origin (SES, mother- and father-based Marital Conflict/Depression, and mother- and father-based Parental Supportiveness) were entered into the signal detection algorithm. This exploratory mathematical algorithm then selects the variable and cut point that maximized the efficiency with which children at risk for adrenarcheal status or mid-level secondary sexual development could be identified. The algorithm calculates a chi-square statistic that estimates the likelihood of a Type I error at an alpha probability level of .01. After choosing the first optimally efficient variable and cut point, the program then searches the two subgroups defined by the branch point for the next most efficient predictor variable and cut point. This procedure continues until there are too few subjects in a subgroup to continue analysis or until no other significantly discriminating variable is identified at $p < .01$.

All the predictor variables used in the signal detection analyses were standardized. The first analysis was conducted on adrenarcheal status. For prediction of this outcome variable, however, no significant interactions emerged between any of the five family environment measures. The second analysis was conducted on secondary sexual development (attainment of Tanner Stage 3). The results of this analysis are shown in Figure 2. The first shaded box in Figure 2 displays the variable—mother-based Parental Supportiveness—that was most efficient in distinguishing between girls who had and had not reached Tanner Stage 3 by the fifth grade ($N = 180$; $\chi^2 = 8.13$; $p < .01$). Forty-five percent of children who scored below .60 on this variable met the gold standard (Tanner Stage 3), compared with only 24% of children who had scores greater than or equal to .60. No other predictor offered further significant discrimination among the group of children with low mother-based Parental Supportiveness scores. Among the subgroup with high mother-based Parental Supportiveness scores, however, SES further discriminated participants ($n = 67$; $\chi^2 = 7.77$; $p < .01$), as indicated by the second shaded box in Figure 2. Only 11% of children with the combination of high SES scores ($\geq -.14$) and high mother-based Parental Supportiveness scores ($\geq .60$) had reached Tanner Stage 3. On the other hand, among children who were from lower SES families ($< -.14$) but had high levels of mother-based Parental Supportiveness scores ($\geq .60$), 40% had attained Tanner 3. In sum, among the subgroup of children whose early childhood experiences were characterized by a combination of high maternal investment and high SES, the vast majority (89%) were still largely prepubertal in the fifth grade. Thus, consistent with psychosocial acceleration theory, girls from the most protected environments displayed the least pubertal development.

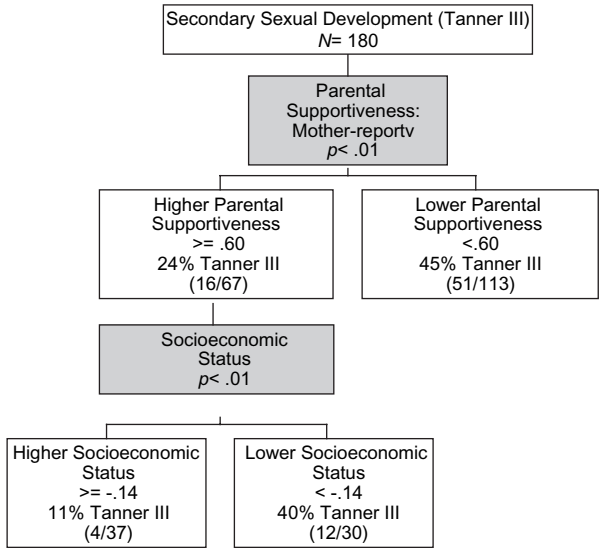

Figure 2. Signal detection analysis of secondary sexual development.

Discussion

In their now classic article on childhood experience and development of reproductive strategies, Belsky

et al. (1991) proposed that individuals have evolved to be sensitive to specific features of their childhood environments, and that children whose early experiences are characterized by high levels of support and stability in and around their families will develop in a manner that slows rates of pubertal maturation, delays onset of reproduction, and orients the individual toward relatively stable and enduring pair-bonds. In an extension of this original theory, Ellis (2004) posited a unique and central role for fathers in regulation of daughters' sexual development, separate from the effects of other dimensions of psychosocial stress and support in the child's environment.

As predicted by psychosocial acceleration theory (Belsky et al., 1991), higher quality parental investment in the preschool years—greater Parental Supportiveness—predicted lower rates of adrenarche in boys and girls in the first grade and less development of secondary sexual characteristics in girls in the fifth grade. (We were not yet able to test the theory on boys' secondary sexual development.) Importantly, this finding generally replicated across both mother and father data sources. These results replicate and extend previous longitudinal research, indicating that higher levels of positive investment and support in family relationships in preschool predict lower levels of pubertal maturation in daughters in the seventh grade (Ellis et al., 1999; cf. Belsky et al., 2007, which found that negative family experiences were most influential on pubertal timing). In addition to these linear effects, exploratory analyses indicated that the combination of high mother-based Parental Supportiveness and high SES reliably forecast low levels of secondary sexual development in the fifth grade (only 11% of girls in this family context had reached Tanner Stage 3 compared with 44% of the rest of the sample).

As further predicted by psychosocial acceleration theory, lower levels of ecological stress in early childhood, as indexed by higher SES, predicted *later* development of secondary sexual characteristics in daughters. In societies in which there are substantial differences between social classes in nutritional and health status, girls from higher social classes actually experience *earlier* pubertal development than do girls from lower social classes; by contrast, effects of SES on girls' pubertal timing are generally absent in countries where lower SES groups do not suffer from systematic malnutrition and disease (reviewed in Ellis, 2004). However, consistent with recent analyses of National Growth and Health Study data (Braithwaite et al., 2007; see also Davison et al., 2003), the current findings suggest that a change may be underway in the United States, with higher SES becoming linked to later pubertal timing in girls. Although lower levels of weight and fat in high-SES girls (e.g., Booth et al., 1999; Dekkers et al., 2004; Lamerz et al., 2005;) may be the causal pathway to later puberty, the current research did not support BMI as a mediating mechanism. The relatively weak negative correlation between SES and BMI in the current sample of 180 girls may have resulted from a restriction of range on the low end of BMI (relative to national norms). In addition, the current sample may have lacked power to detect this relation. For the larger sample of girls in which SES and BMI data were available (including 24 additional girls who did not participate in the fifth-grade puberty assessment), the correlation between these two variables was statistically significant, $r(204) = -.16$, $p < .05$.

The current research provided little support for the prediction that Marital Conflict/Depression would accelerate pubertal development. The only statistically significant finding was that greater father-based Marital Conflict/Depression predicted higher rates of adrenarche. These mostly null findings run counter to psychosocial acceleration theory and further cloud the already contradictory literature (reviewed in Ellis, 2004) on the potential accelerating effects of family conflict and coercion on pubertal development. A possible interpretation of the current data is that the measures of Marital Conflict/Depression were less successful at predicting pubertal timing than were the measures of Parental Supportiveness because the former assessed more family-level stress while the latter directly indexed parent – child processes.

The present results provided some support for paternal investment theory. On the one hand, the father-based measures of family environment accounted for unique variances in adrenarcheal timing, above and beyond the effects of the equivalent mother-based measures. Consistent with paternal investment theory (Ellis, 2004), these results suggest a unique role for fathers in families in regulating maturation of the adrenal axis. On the other hand, the mother-based measures of family environment clearly outperformed the equivalent father-based measures in prediction of secondary sexual development in daughters. The relative weakness of the father-based measures in this context may derive from restriction of range. At the time of the preschool assessments of family environment, 94% of families in our study were biologically intact, and thus, the extreme end of low paternal investment that characterizes many noncustodial fathers was not adequately represented. Further, recent analyses of the National Institute of Child Health and Human Development (NICHD) Study of Early Child Care and Youth Development found that the effects of paternal investment

on pubertal timing were moderated by infant negative emotionality (Belsky et al., 2007). Inclusion of this moderating variable in future research should enhance the ability to detect father effects.

To examine multivariate relations among variables, a path analysis was conducted. The results indicated that later age at menarche in mothers, higher SES, greater mother-based Parental Supportiveness, and higher third-grade BMI each uniquely and significantly predicted later sexual development in daughters. Maternal age at menarche, SES, and BMI are all potential confounds in interpreting the effects of parent – child relationships on pubertal timing. That the relation between Parental Supportiveness and pubertal timing (a) remained statistically significant after controlling for these variables and (b) has now replicated across independent longitudinal investigations (see Ellis et al., 1999) indicates that it is a reliable empirical phenomenon.

The hypothesis that BMI would mediate relations between family environment measures and pubertal timing received limited support. Consistent with past research documenting links between psychosocial stress and weight gain in children (Lissau & Sorensen, 1994; Mellbin & Vuille, 1989a, 1989b), higher levels of mother-based Marital Conflict/Depression were associated with greater BMI. However, Marital Conflict/Depression did not directly predict sexual development. Instead, there was a statistically significant indirect effect of Marital Conflict/Depression on secondary sexual development through BMI. Conversely, higher levels of mother-based Parental Supportiveness predicted later puberty but were not associated with BMI. A possible explanation for this null finding is that BMI was an inadequate measure of body fat. Past research has demonstrated that psychosocial stress is most closely associated with gains in abdominal fat in women (Bjorntorp, 2001; Epel et al., 2000). Further, it is abdominal fat and lower body fat that are most closely associated with earlier pubertal development in girls (de Ridder et al., 1990, 1992; Lassek & Gaulin, 2007), whereas upper body fat has been linked to later pubertal development (Lassek & Gaulin, 2007). Thus, measures of BMI, which do not distinguish between upper and lower body fat or central and peripheral body fat, may be too crude to capture the mediating effects of fat distribution on relations between family environments and timing or tempo of pubertal development.

Life History Theory and Timing of Pubertal Development

Psychosocial acceleration theory posits that children's experiences in and around the family provide them with reliable information about the reproductive opportunities and constraints that they are likely to encounter at adolescence and beyond, and that the developing person uses this information to match reproductive development to those opportunities and constraints. Not clearly addressed in the theory, however, are questions about what types of childhood experiences will have the most influence on reproductive strategies. Life history theory posits that, given adequate bioenergetic resources to support growth and reproduction, relatively high levels of exposure to psychosocial stress (harshness) and stochastic variation in such environmental exposures (unpredictability) each accelerates reproductive development (Ellis, Figueredo, Brumbach, & Schlomer, 2007). This highlights the importance in future research of separately measuring and assessing the impact of environmental harshness and unpredictability on pubertal timing. Further, both experimental data from bonnet macaques (Rosenblum & Andrews, 1994; Rosenblum & Paully, 1984) and cross-cultural human data (Quinlan, 2007) suggest that low – parental investment strategies may be driven primarily by environmental unpredictability, occurring under conditions in which parents have limited ability to affect the survival and long-term reproductive outcomes of their offspring (reviewed in Ellis et al., 2007). Thus, predictability – unpredictability may be an especially important dimension of the larger ecological context of the developing child, whereby variation on this dimension supports or undermines the quality of parental investment (e.g., Parental Supportiveness), which in turn regulates pubertal timing.

Future research could also benefit from further examining the functional nature of the relations between family environments and pubertal timing. Psychosocial acceleration theory conceptualizes variation in pubertal timing as functioning to guide development of alternative reproductive strategies in adulthood. However, an alternative middle-level life history model—child development theory (CDT; Ellis, 2004)—conceptualizes this variation as functioning to calibrate the length of childhood. According to CDT, the benefits of a longer childhood (later sexual maturation and onset of reproduction) are increased in high-investing family contexts that foster development of sociocompetitive competencies, whereas the costs of ''cutting short'' childhood are reduced in low-investing family contexts that do not meaningfully facilitate these competencies. CDT posits that children have been selected to capitalize on the benefits of high-quality parental investment and reduce the costs of low-quality parental investment

by contingently altering the length of the developmental period prior to reproductive maturation. Whereas psychosocial acceleration theory conceptualizes pubertal timing as mediating relations between family environments and qualitative variation in expression of adult reproductive strategies (e.g., pair-bond stability, parental investment strategies), CDT conceptualizes pubertal timing as only mediating relations between family environments and timing of reproductive milestones (e.g., sexual debut, first birth) (see Ellis, 2004, for discussion and evidence). The current results linking Parental Supportiveness to pubertal timing is consistent with both psychosocial acceleration theory and CDT. Further research is needed to determine whether this link functions specifically to calibrate timing of offset of childhood/onset of reproductive status or more generally to guide qualitative variation in reproductive strategies.

Limitations and Further Research Directions

Certain limitations of the current research suggest important avenues for future research. First, and foremost, the research design was not genetically informative. The evolutionary models of pubertal timing presented in this paper rest on the concept of conditional reproductive strategies; that is, they emphasize environmentally triggered processes that shunt individuals toward given reproductive strategies. An alternative explanation, however, is that covariation between pubertal timing and family environmental factors derives from genetic sources (as noted in the introduction). Although controlling for mother's age at menarche partially addresses this potential confound, we cannot be certain by any means that the effects of biological inheritance have been fully accounted for. Genetically informative research designs incorporating environmental measures (e.g., Mendle et al., 2006; Tither & Ellis, 2006) and experimental research designs (e.g., randomized, longitudinally designed prevention trials) are needed to test for the causal influence of family environments on pubertal timing. Second, the current research only tested BMI as a potential mediator of the relations between family environments and pubertal timing. Further research is needed to identify neuroendocrine mechanisms underlying the effects of psychosocial stress and support on maturation of the adrenal and gonadal axes. Third, the current study employed a predominantly White sample. Given that both the major predictor variables (indices of family environment) and the outcome variables (pubertal timing) are known to vary by race/ethnicity (e.g., Ellis et al., 2003; Herman-Giddens et al., 1997), future research could benefit from examining more diverse populations. Finally, the current study only focused on timing of pubertal development. The predictions from life history theory, however, are equally applicable to timing and tempo of pubertal development. Future studies would do well to examine whether children who experience different levels and types of psychosocial stress differ in the speed at which they progress through puberty.

In conclusion, the current research highlights the multiplicity of factors that predict (and potentially determine) pubertal timing: genetic inheritance through the mother, childhood growth in height and weight, socioeconomic factors, and quality of parental investment. Consistent with the theorizing of Belsky et al. (1991) and Ellis (2004), we propose that quality of parental investment, as indexed by measures such as Parental Supportiveness, is the most important mechanism through which young children receive information about levels of stress and support in their local environments, and that this information provides a basis for adaptively adjusting pubertal timing. The present results suggest that these adjustments encompass maturation of both the adrenal and the gonadal axes.

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