

Psychosocial Antecedents of Variation in Girls' Pubertal Timing: Maternal Depression, Stepfather Presence, and Marital and Family Stress

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Drawing on Belsky, Steinberg, and Draper's evolutionary theory of the development of reproductive strategies, we tested a model of individual differences in girls' pubertal timing. This model posits that a history of psychopathology in mothers results in earlier pubertal maturation in daughters, and that this effect is mediated by discordant family relationships and father absence/stepfather presence. The model was supported in a short-term longitudinal study of 87 adolescent girls. In the primary test of the model, it was found that a history of mood disorders in mothers predicted earlier pubertal timing in daughters, and this relation was fully mediated by dyadic stress and biological father absence. In families in which the mother's romantic partner was not the biological father, dyadic stress accounted for almost half of the variation in daughters' pubertal timing. Stepfather presence, rather than biological father absence, best accounted for earlier pubertal maturation in girls living apart from their biological fathers. We propose that stepfather presence and stressful family relationships constitute separate paths to early pubertal maturation in girls.

INTRODUCTION

The onset of pubertal development has typically been viewed as an important marker of the transition into adolescence and is accompanied by major social and cognitive changes (Conger, 1984; Feldman & Elliot, 1990). Variations in the timing of pubertal maturation—in levels of physical and sexual development of adolescents in comparison to their same-age peers—has received considerable research attention. The most consistent finding to emerge from the literature is that early timing of puberty in girls is associated with negative health and psychosocial outcomes. In particular, early-maturing girls are at greater risk for breast cancer (e.g., Kampert, Whittemore, & Paffenbarger, 1988; Vihko & APter, 1986), obesity (e.g., Ness, 1991; Wellens et al., 1992), and teenage pregnancy (e.g., Udry, 1979; Udry & Cluquet, 1982), and tend to show more disturbances in body image, report more emotional problems such as depression and anxiety, and engage in more problem behaviors such as alcohol consumption and sexual promiscuity (e.g., Caspi & Moffitt, 1991; Flannery, Rowe, & Gulley, 1993; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Susman, Nottelman, Inoff-Germain, Loriaux & Chrousos, 1985).

Although a good deal is now known about the sequelae of variations in pubertal timing in girls, relatively little is known about the social and psychological antecedents of this variation. Recent theory and data (e.g., Belsky et al., 1991; Graber, Brooks-Gunn, & Warren, 1995) suggest that an individual's experiences during childhood may influence the physiological mechanisms that initiate and control timing of pubertal maturation. The present paper examines an-

tecedents of variation in pubertal timing among adolescent girls. The authors seek to test and extend predictions derived from an evolutionary model of individual differences in pubertal timing. These predictions concern the relation between several forms of familial stress—maternal depression, family conflict, and divorce and remarriage—and the timing of pubertal maturation.

Sources of Variation in Pubertal Timing

Individual differences in the timing of pubertal maturation are influenced by both genes and environment. Behavioral genetic studies using twin and sibling designs have demonstrated that genetic differences account for substantial variation in pubertal timing (Farber, 1981; Kaprio et al., 1995; Treloar & Martin, 1990). At the same time, however, such studies have also documented the importance of shared environmental influences on age of menarche in girls. Farber (1981) reported that the degree of similarity between female twins in menarcheal age was positively related to the degree of consanguinity. Monozygotic twins reared together were most similar in menarcheal age (average difference = 2.8 months), followed by monozygotic twins reared apart (average difference = 9.3 months), followed by dizygotic twins reared together (average difference = 12 months). That monozygotic twins reared apart were similar in menarcheal timing to dizygotic twins reared together

suggests that individual differences in onset of menarche may be influenced by the degree to which girls share common environments (as well as common genes).

Sources of environmental influence on pubertal timing have both physical and psychosocial components. Potential physical influences include factors such as weight, nutrition, and exercise (Brooks-Gunn, 1988). High levels of exercise, for example, have been found to delay pubertal maturation in female dancers (Calabrese et al., 1983; Warren et al., 1991). Potential psychosocial influences include factors such as family environment, child behavioral problems, and stressful life events (Belsky et al., 1991). This paper specifically focuses on psychosocial antecedents of variation in pubertal timing in young adolescent girls.

The Belsky, Steinberg, and Draper Evolutionary Model of Pubertal Timing

Belsky, Steinberg, and Draper (1991) have proposed an evolutionary model of psychosocial influences on the timing of puberty. They posit that "a principal evolutionary function of early experience—the first 5 to 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. suggested that humans have evolved to be sensitive to specific features of their early childhood environments, and that exposure to different environments biases children toward acquisition of different reproductive strategies. Children whose experiences in and around their families of origin are characterized by relatively high levels of stress (e.g., scarcity or instability of resources, father absence, negative and coercive family relationships, lack of positive and supportive family relationships) are hypothesized to develop in a manner that speeds rates of pubertal maturation, accelerates sexual activity, and orients the individual toward relatively unstable pair bonds.

In essence, Belsky et al. (1991) proposed that the context of early rearing "sets" the person's reproductive strategy in a way that was likely to have functioned adaptively in that context in the environments in which humans evolved. Over the course of our natural selective history, ancestral females growing up in adverse family environments may have reliably increased their reproductive success by accelerating

physical maturation and beginning sexual activity and reproduction at a relatively early age, without the expectation that paternal investment in childrearing would be forthcoming, and without the precondition of a close, enduring romantic relationship (Belsky et al., 1991). As Chisholm (1996 p. 21) suggests, "when young mammals encounter conditions that are not favorable for survival—that is, the conditions of environmental risk and uncertainty indexed by emotional stress during development—it will generally be adaptive for them to reproduce early."

Childhood Stressors and the Timing of Pubertal Maturation in Girls

The literature on environmental antecedents of pubertal timing has focused on three general classes of stress: physical stress (e.g., malnutrition, disease, economic hardship), socioemotional stress (e.g., harsh and neglecting family relationships, parental psychopathology), and divorce (presence versus absence of the biological father).

Physical stress. Does physical stress accelerate pubertal maturation in girls? Contrary to the theory of Belsky et al. (1991), current evidence suggests that girls are primed to delay pubertal maturation following episodes of malnutrition, disease, or metabolic imbalance (Ellison, 1990; Surbey, 1998). This delay functions to channel energy toward maintenance (survival) rather than growth and reproduction at a time when the body is not well prepared to undergo the stressful and energetically demanding changes of puberty (see Surbey, 1998). In societies in which there are substantial differences between social classes in access to basic nutritional resources, girls from higher social classes experience earlier pubertal timing than do girls from lower social classes (e.g., early twentieth-century Britain: Frisch, 1983; Singapore: Aw & Tye, 1970; Hong Kong: Lee, Chang, & Chan, 1963; Poland: Bielicki, Waliszko, Hulanicka, & Kotlarz, 1986; Bantu-speaking South Africa: Burrell, Healy, & Tanner, 1961). These data are consistent with the secular trend (beginning at least 150 years ago in England) favoring lower age of menarche in association with general improvements in health care and nutrition accompanying modernization (Tanner, 1990). Effects of social class on girls' pubertal timing are generally absent, however, in countries where lower social classes do not suffer from systematic malnutrition (e.g., Canada: Surbey, 1990; Sweden: Lindgren, 1976; New Zealand: Moffitt, Caspi, Belsky, & Silva, 1992).

Socioemotional stress. Although nutritional and infectious perturbations generally slow down growth and development, socioemotional stressors may have

the opposite effect on developmental tempo (see, especially, Chisholm, 1996). Studies of socioemotional stress are conceptually distinct from studies of physical stress, as an individual in a physically rich environment can still be exposed to high levels of interpersonal stress in and around the family. Hulanicka (1999) has specifically compared the effects of physical and socioemotional stress on maturational tempo in Polish schoolgirls. Although the Polish data show a strong main effect of poverty on pubertal timing (i.e., poorer girls mature later), Hulanicka's research summary demonstrates that various socioemotional stressors in the family (e.g., father absence, parental alcohol abuse, prolonged illness of a parent) can lead to earlier pubertal timing in girls, despite the often lower socioeconomic and nutritional status of girls in stressed families.

Longitudinal research has largely supported the hypotheses that socioemotional stressors are associated with earlier pubertal timing in girls (e.g., Ellis, McFadyen-Ketchum, Dodge, Pettit, Bates, 1999; Graber et al., 1995; Moffitt et al., 1992; Steinberg, 1988). An adequate test of this hypothesis requires assessment of socioemotional stressors prior to adolescence and then assessment of girls' pubertal timing during adolescence. (Of course not even this longitudinal design can provide a truly adequate test of the theory because it cannot rule out gene effects, as discussed below under Environmental and Genetic Influences.

Moffitt et al. (1992) and Ellis et al. (1999) employed this research design in separate 8-year longitudinal studies. In their study of New Zealand girls ($N = 416$), Moffitt et al. found a significant correlation between mothers' reports of conflictual family interactions, assessed when their daughters were age 7, and daughters' reports of menarcheal age, obtained at age 15. Consistent with Belsky et al. (1991), greater family conflict predicted earlier menarche. In their study of American girls ($N = 173$), Ellis et al. assessed quality of family relationships in the summer prior to girls' entry into kindergarten (through direct behavioral observation and interviews with the mothers) and then assessed levels of pubertal development in grade 7 (based on daughters' self-reports). The assessment of quality of family relationships indexed both positive and negative dimensions of both mother-child and father-child relationships. Ellis et al. found that it was the relative absence of positive, harmonious parent-child relationships (rather than the presence of negative, conflictual ones) that forecast early pubertal timing, and that quality of fathers' investment in the family was the most important feature of the early family environment in relation to daughters' subsequent pubertal timing. The results of

these two long-term studies concur with more short-term longitudinal studies, which have generally found that greater parent-child conflict and/or less parent-child closeness predict earlier or faster pubertal maturation in girls (Graber et al., 1995; Steinberg, 1988; cf. Mekos, Hetherington, & Clingempeel, 1992, which did not find an association between quality of parent-child relationships and speed of daughters' pubertal growth).

Divorce. A third type of stressor highlighted by Belsky et al. (1991) is divorce, which is widely viewed as a stressful life event related to higher rates of maladjustment in adolescents (Emery, 1988). Research on the effects of divorce on female pubertal timing has primarily focused on the presence versus absence of the biological father, as emphasized by Belsky et al. and previously by Draper and Harpending (1982) in their work on the role of father absence in shaping reproductive strategies. Most studies suggest that girls reared in father-absent homes reach menarche several months earlier than their peers reared in father-present homes (Jones, Leeton, McLeod, & Wood, 1972; Mekos et al., 1992; Moffitt et al., 1992; Surbey, 1990; Wierson, Long, & Forehand, 1993) and display greater pubertal development in the seventh grade (Ellis et al., 1999). Moreover, several studies have found that the longer the period of father absence, the earlier the onset of menstruation (Mekos et al., 1992; Moffitt et al., 1992; Surbey, 1990; cf. Campbell & Udry, 1995, who did not find evidence that years of father absence had an accelerating effect on menarcheal age).

Separate Mechanisms?

Are father-absent effects on pubertal timing different from more general effects of interpersonal stress on pubertal timing? In their original formulation, Belsky et al. (1991) hypothesized that divorce would accelerate pubertal maturation in girls specifically because family disruption is stressful. However, Steinberg (1992) suggested two reasons why stress is probably not the operating mechanism. First, family conflict and father absence are not closely related. Moffitt et al. (1992) found that family conflict at age 7 and father absence in childhood were uncorrelated, $r(326) = -.01$, and that each contributed unique variance to the prediction of menarcheal age. Second, although mother absence is at least as stressful as father absence, it does not appear to have the same relation to daughters' pubertal timing. Mekos et al. (1992) found that years of father absence but not years of mother absence had an accelerating effect on girls' pubertal maturation. Similarly, Surbey (1990) found that girls who grew up in father-absent homes, but not those from mother-absent

homes, experienced earlier menarche than girls who grew up with both parents present. Taken together, these data suggest that the mechanisms underlying father-absent effects on pubertal timing may be different from the mechanisms underlying the more general effects of family stress on pubertal timing, and that father absence and interpersonal stress in the family may represent separate paths to early pubertal maturation.

Father Absence versus Stepfather Presence

What is the mechanism underlying possible father-absence effects on daughters' pubertal timing? One possibility is that girls reared in homes without their biological fathers present may experience earlier sexual maturation because of increased exposure to unrelated adult males, especially stepfathers and mothers' dating partners. Research on a variety of mammalian species (e.g., mice, cows, pigs, tamarins) indicates that exposure to pheromones produced by unrelated adult male conspecifics accelerates female pubertal development (Izard, 1990; Sanders & Reinisch, 1990; Ziegler, Snowdon, & Uno, 1990). Research on humans has also provided definitive evidence of regulation of women's reproductive functioning by pheromones (Stern & McClintock, 1998). For example, controlled experimental studies have shown that pheromones produced by men's axillary sweat glands reduce variability in women's menstrual cycles (Cutler et al., 1986). If, consistent with the animal literature, human females possess physiological mechanisms that accelerate pubertal maturation in response to pheromonal stimulation by unrelated adult males, then exposure to stepfathers and mothers' dating partners, rather than absence of the biological father per se, should most strongly predict early pubertal timing in girls. This hypothesis is consistent with data reported by Mekos et al. (1992) showing that girls in stepfather-present homes experienced faster pubertal growth than girls in single-mother homes.

Maternal Psychopathology

Major psychopathologies, such as mood disorders and substance abuse, are strongly predictive of marital discord, conflictual relationships with children, and divorce (Beach & Nelson, 1990; Cummings & Davies, 1994; Downey & Coyne, 1990). Maternal psychopathology, therefore, may be a distal cause of the marital and family dysfunction that has been found to predict earlier pubertal timing in girls. A history of psychopathology in mothers should provoke earlier pubertal maturation in daughters through its impact on stressful interpersonal relationships in the family and father absence/stepfather presence.

Hypotheses

The primary goal of this study was to test an evolutionary model of individual differences in the timing of pubertal maturation in girls. We used Belsky et al. (1991) as the guiding framework for this model, but made some revisions and extensions based on the theoretical considerations and empirical findings reviewed above. Specifically, the following set of hypotheses were tested:

1. A history of psychopathology in mothers will predict earlier timing of pubertal maturation in daughters, and this relation will be mediated by stressful family relationships and father absence/stepfather presence. The measure of maternal psychopathology used in the current study was a history of mood disorders.
2. Higher levels of interpersonal stress in the family will be associated with earlier pubertal timing in daughters. The measures of interpersonal stress in the current study were level of stress in the mother's romantic relationship and dysfunctional family relationships.
3. Girls from father-absent homes will experience earlier timing of puberty than girls from father-present homes.
4. The amount of time that girls are exposed to unrelated adult males in the home, rather than the amount of time that girls live without their biological fathers in the home, will best account for the relation between father absence and early pubertal timing.
5. Interpersonal stress and father absence/stepfather presence will constitute separate paths to early pubertal timing. The test of this hypothesis included an analysis of both additive and multiplicative effects. The purpose of the multiplicative analysis was to examine whether stress in the family environment was an especially strong predictor of daughters' pubertal timing in father-absent/stepfather-present homes.

This set of hypothesized relations is diagrammed in Figure 1.

Environmental and Genetic Influences

The Belsky et al. (1991) model of pubertal timing rests on the concept of conditional reproductive strategies; that is, it emphasizes environmentally triggered processes that shunt an individual toward a given reproductive strategy. An alternative explanation, however, is that individual differences in reproductive strategies result from genetic differences. Consider this possibility: Girls who mature earlier ex-

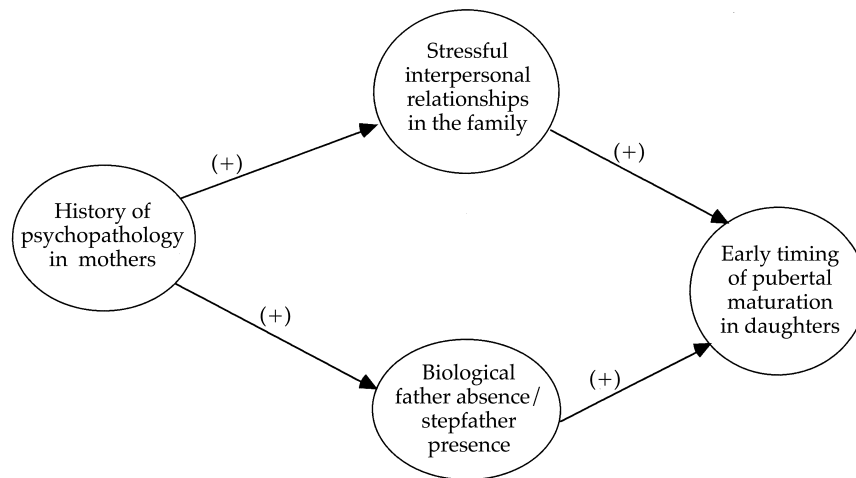


Figure 1 Path diagram of proposed psychosocial model of female pubertal timing.

hibit earlier age of first marriage and first birth (Udry, 1979; Udry & Cliquet, 1982), which in turn are associated with heightened levels of family conflict, increased probability of divorce, and increased exposure of daughters to stepfathers. Because mothers who are early maturers tend to have daughters who are early maturers (Brooks-Gunn & Warren, 1988; Garn, 1980), the correlation of familial stressors and father absence/stepfather presence with early sexual maturation in daughters may be spurious; that is, it may be due simply to genetic transmission of the timing of pubertal maturation (Surbey, 1990).

Although the present study cannot rule out the possibility that genetic influences account for substantial variation in the system of relations depicted in Figure 1, we did collect data relevant to evaluating the specific genetic pathway outlined above. This pathway conceptualizes early onset of reproduction in the mother (i.e., early age of first marriage and/or first birth) as mediating the relation between early pubertal timing in the mother and later marital and family dysfunction. Early onset of reproduction in the mother is a link in the chain explaining why early-maturing daughters inhabit more stressful family environments. To address this alternative explanation, additional tests of the model were conducted after partialling out mothers' age of first marriage and first birth from daughters' pubertal timing.

METHOD

Participants

Participants in this study constituted a subsample of a larger longitudinal study of maternal depression and adolescent development. The larger study, which

includes three cohorts, consists of 240 children and their mothers. Of the 240 children, 130 are girls. Girls in two cohorts completed a measure of pubertal timing in the seventh grade. The 87 participants in this study included all girls from these two cohorts for whom complete data were available at Time 1 and Time 2 (87 out of 96 girls). Girls in the third cohort did not receive the puberty measure (due to time constraints) and were excluded from the present study.¹ At first assessment, the average age of the participants was 12.26 ($SD = .49$, $range = 11-13$). Eighty-five percent were European American, 12% were African American, and 3% were other (Hispanic, Asian, Native American). The sample was predominantly lower-middle to middle class, with a mean SES (Hollingshead, 1975) of 41.16 ($SD = 13.52$).

¹ Participants ($N = 87$) and nonparticipants ($n = 43$) were compared on demographic and substantive variables (see description of variables in the Measures section). Specifically, t tests were conducted to compare participants and nonparticipants on race (European American versus African American), $t(125) = 1.67$, $p = .10$; SES, $t(129) = -2.02$, $p = .05$; mother's age at first marriage, $t(120) = 1.01$, $p = .38$; mother's age at first birth, $t(129) = -.40$, $p = .77$; presence versus absence of maternal history of depression, $t(130) = .36$, $p = .71$; presence versus absence of the biological father in the home, $t(127) = -1.18$, $p = .24$; mother's dyadic adjustment, $t(100) = .27$, $p = .79$; the Family Relationships Index, $t(123) = -1.27$, $p = .21$; and the Family Assessment Device, $t(121) = .91$, $p = .37$. These t tests indicate that participants and nonparticipants were not significantly different on any of the substantive variables used in this study. There was a significant tendency for participants ($M = 41.26$) to have higher SES than nonparticipants ($M = 36.21$), but it is important to note that variances in SES for participants and nonparticipants were not significantly different—as indicated by Levene's test for heterogeneity of variance, $F(1, 127) = .06$, $p = .81$ —and SES itself was unrelated to pubertal timing, $r(87) = -.11$, $p = .32$.

Procedures

Letters were sent to parents of children in the fifth grade in a metropolitan public school district during 3 consecutive school years. Parents were invited to participate in the study and were asked to complete a brief health history questionnaire about whether they had ever had any of 24 medical conditions such as diabetes, cancer, heart disease, and depression, or if they had ever taken any of 34 medications. Mothers who indicated a history of depression or no psychiatric problems were interviewed further. Sixty-seven mothers in the present sample had a history of mood disorders (e.g., major depression, dysthymia, adjustment disorder with depressed mood) with a broad range of severity and chronicity; the remaining 20 mothers were lifetime free of psychopathology.

The daughters were first assessed in sixth grade (Time 1). An interviewer, blind to the mothers' psychiatric history, administered a battery of questionnaires to the mothers and children. Another evaluation (Time 2) was conducted approximately one year after Time 1. The predictor variables were assessed at Time 1 and pubertal timing was assessed at Time 2. Only those measures relevant to the present study are described here.

Measures

Mothers' history of mood disorders. Mothers' psychiatric histories were assessed with the Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First, 1990), which is a semistructured clinical interview from which DSM-III-R (American Psychiatric Association, 1987) diagnoses can be made. Interrater reliability of the SCID interviews has been reported elsewhere: (see Garber, Robinson, & Valentin, 1997). Because the focus of the larger study was on maternal depression, extensive information was obtained regarding mothers' histories of mood disorders, including the timing of depressive episodes throughout the daughters' lifetimes. For the present analyses, mothers received a score of 0 (not present) or 1 (present) with regard to their history of mood disorders.

Age of mother at first marriage and first birth. During the Time 1 interview, mothers were asked how old they were when they were first married and first gave birth to a child. Mean age of first marriage was 20.43 ($SD = 3.13$) and mean age at first birth was 22.65 ($SD = 3.70$).

Biological father absence. During the Time 1 interview, mothers were asked to describe their marital status and the current composition of their household. Families were classified as father-absent if par-

ticipants reported that the biological father was not living in the home at Time 1. Of the 97 families in the study, 47 were father-absent.

Age of daughter when biological father moved out. For the 47 father-absent families, mothers reported the age of the daughter at the time the biological father moved out of the home.

Age of daughter when first alternative father figure came into her life. During the Time 1 interview, mothers reported whether there had ever been an alternative father figure (not the biological father) in the daughter's life. Father figure was defined as a "significant other" in the mother's life (e.g., stepfather, mother's boyfriend) and excluded male relatives such as grandfathers or uncles. In the 47 father-absent families, 25 daughters currently had an alternative father figure and another 6 had previously had an alternative father figure. For these 31 families, the mother reported the age of the daughter when the first alternative father figure came into her life.

Level of stress in mothers' romantic relationships. Of the 87 mothers, 74 were involved in romantic relationships at Time 1 (with either the child's biological father, stepfather, or a boyfriend). Mothers' reports of the level of stress in their romantic relationships were obtained at Time 1 using the 32-item Dyadic Adjustment Scale (DAS; Spanier, 1976). The scale was reverse-scored so that higher scores represented higher levels of dyadic stress. The DAS has been found to have high internal consistency and to discriminate between maritally distressed and nondistressed couples (Spanier, 1976). Coefficient α in this sample was .96. An inspection of the present DAS scores revealed a bimodal distribution, with clustering of mothers at relatively high and low ends of the scale. Thus, to meet the assumptions of multivariate analysis, we performed a median split and coded the mothers below the median as 0 (low dyadic stress) and above the median as 1 (high dyadic stress).

Dysfunctional family relationships. Mothers' perceptions of the quality of the family environment were assessed at Time 1 with the Family Relationship Index (FRI; Holahan & Moos, 1983) and the Family Assessment Device (FAD; Epstein, Baldwin, & Bishop, 1983). The FRI is a composite of three scales from the Family Environment Scale: Cohesion, Expressiveness, and Conflict. The FRI has been found to correlate with other measures of family functioning (Hoge, Andrews, Faulkner, & Robinson, 1989), and to have a one-year stability coefficient of .61 (Billings & Moos, 1985). Coefficient α in this sample was .83. The FAD is a 60-item questionnaire based on the McMaster Model of Family Functioning (Epstein, Bishop, & Levine, 1978). It contains a subscale measuring General

Family Functioning and six additional subscales. The present study used the General Functioning subscale, which has been found to have good internal consistency and validity (Byles, Byrne, Boyle, & Offord, 1988). Coefficient α in this sample was .96.

The FRI and the FAD General Functioning Scale were highly correlated, $r(87) = -.82, p < .001$; thus, the two scales were combined into a composite measure of “dysfunctional family relationships.” This measure was computed by standardizing the FRI and the FAD General Functioning Scale, reverse coding where necessary so that higher scores indicated greater dysfunction, and then averaging the two scales.

Pubertal timing. Daughters’ self-reported level of pubertal development was assessed at Time 2 with the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988) and with additional items from Morris and Udry’s (1980) index of adolescent physical maturation. Both scales have shown good internal consistency and reliability across raters (Morris & Udry, 1980; Petersen et al., 1988). Girls rated their breast and hip development, growth spurt in height, amount of body hair, skin changes, and menarcheal status. To compute overall pubertal development scores, individual puberty items were standardized and averaged. Coefficient α was .81. Pubertal development scores were then converted into pubertal timing scores by partialling out daughters’ age. The pubertal timing scores were used in all data analyses. Higher scores indicated earlier pubertal timing (i.e., greater pubertal development in seventh grade, controlling for age).

RESULTS

Overview

The primary focus of this study was on psychosocial antecedents of pubertal timing in girls. This presented a challenge because initial data collection occurred when the target children were already in sixth grade and in varying stages of pubertal development. It is important, therefore, to distinguish between predictors that (in all probability) anteceded puberty and those that could have been caused by puberty (e.g., dysfunctional family relationships). We used three independent variables that were plausible antecedents of pubertal development: mothers’ history of mood disorders, absence of the biological father in the home, and level of stress in the mothers’ romantic relationships. We present empirical evidence that mothers’ history of mood disorders and father absence were not caused by variations in daughters’ pubertal timing. In addition, marital quality shows strong stabil-

ity over time (Porter & O’Leary, 1980; Wierson et al., 1993) and may provide an index of family stress that is less directly influenced by child’s pubertal status than are more general measures of family dysfunction. Our data analytic strategy was to focus on these three independent variables in the primary test of the model and then to conduct secondary tests incorporating overall family functioning as an additional marker of stress in the family environment. Means and standard deviations for all variables, as well as the correlations among these variables, are shown in Table 1. The predictor variables were assessed at Time 1 and are based on parents’ reports; pubertal timing was assessed at Time 2 and is based on daughters’ reports. Because correlations across different data sources control for method variance associated with reliance on a single data source (e.g., scale usage, halo effects), the correlations with pubertal timing can be considered conservative estimates of the real relations.

The Mediational Model

We hypothesized that a history of mood disorders in mothers would predict earlier pubertal timing in daughters, and that this relation would be mediated by stressful family relationships and father absence/stepfather presence (see Figure 1). Further, we hypothesized that stressful family relationships and father absence/stepfather presence would constitute separate paths to pubertal timing. We tested these hypotheses in a primary and secondary set of path analyses. In the primary path analyses ($n = 74$), we used dyadic stress as the marker of stressful family relationships. This 74-family subsample was necessitated by the use of the DAS, which was only completed by mothers who were currently involved in romantic relationships. In the secondary path analyses ($n = 87$), we substituted dysfunctional family relationships for dyadic stress to provide a broader measure of stressful family relationships. Both the primary and secondary analyses used maternal history of mood disorders (as the marker of maternal psychopathology) and biological father absence (as the marker of father absence/stepfather presence). All path analyses were performed using the EQS program (Bentler, 1993). Parameter estimates were based on maximum likelihood estimation.

Primary path analyses. The initial set of path analyses were referred to as “primary” because they employed the three primary independent variables used in this research. In this initial set, the first path analysis tested a direct effects model. Mothers’ histories of mood disorders were treated as the independent variable and there were direct paths leading from this

Table 1 Descriptive Statistics and Zero-Order Correlations among Variables

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7	8	9	10
1. Socioeconomic status	41.16	13.52										
2. Race (European American = 0; African American = 1)	.11	.32	-.32** (87)									
3. Maternal history of depression	.78	.41	-.15 (87)	-.07 (87)								
4. Mothers' age at first birth	22.63	3.74	.44** (87)	-.25* (87)	-.16 (87)							
5. Mothers' age at first marriage	20.43	3.13	.35** (84)	-.04 (84)	-.11 (84)	.58** (84)						
6. Biological father absence (present = 0; absent = 1)	.52	.50	-.21 (87)	.20 (87)	.44** (87)	-.32** (87)	-.07 (84)					
7. Daughters' age when biological father moved out	4.53	3.40	.35* (46)	-.43** (46)	.26 (46)	.34* (46)	.16 (42)	^b				
8. Daughters' age at arrival of first alternative father figure	6.47	3.07	.22 (30)	-.06 (30)	^a (30)	.12 (30)	.15 (28)	^b	.46 (30)			
9. Dyadic stress	.49	.50	-.21 (74)	.01 (74)	.30** (74)	-.17 (74)	-.10 (70)	.16 (74)	.06 (33)	-.53** (26)		
10. Dysfunctional family relationships	-.16	.94	-.17 (87)	.13 (87)	.29** (87)	-.23* (87)	-.17 (84)	.12 (87)	.03 (45)	-.25 (30)	.56** (74)	
11. Pubertal timing	.05	.66	-.10 (87)	.25* (87)	.30** (87)	-.20 (87)	-.08 (84)	.30** (87)	-.10 (45)	-.41* (29)	.37** (74)	.30** (87)

Note: Decimal points omitted from correlations. Pairwise *n* in parentheses below correlations.

^aAll daughters with alternative father figures also had mothers with a history of depression.

^bAll daughters whose biological fathers had moved out and/or who had alternative father figures were classified as father-absent.

* $p < .05$; ** $p < .01$.

variable to dyadic stress, father absence, and daughters' pubertal timing. Although each of the specified paths was statistically significant, the overall model did not adequately account for the covariation in the data, $\chi^2(3, N = 74) = 9.65, p = .02$; Comparative Fit Index (CFI) = .81. As predicted, a history of mood disorders in mothers was associated with earlier pubertal timing in daughters ($\beta = .34, p < .01$), more father absence ($\beta = .45, p < .001$), and more dyadic stress ($\beta = .30, p < .01$).

The second path analysis tested a partially mediated model. It retained the paths from the direct effects model, but added paths from dyadic stress and father absence to pubertal timing. The partially mediated model fit the data very well, $\chi^2(1, N = 87) = .07, p = .79$; CFI = 1.00. Comparison of the two path analyses indicated that the partially mediated model provided a significantly better fit than did the direct effects model, $\chi^2_{\text{diff}}(2) = 9.58, p < .01$. With the inclusion of the mediators in the second path analysis, the direct path from maternal history of depression to daughters' pubertal timing became statistically non-significant (dropping from .34 to .17).

The third path analysis tested for full mediation and is shown in Figure 2. The fully mediated model dropped the direct path from maternal history of de-

pression to daughters' pubertal timing while retaining the other paths from the partially mediated model. The fully mediated model fit the data very well, $\chi^2(2) = 2.04, p = .36$; CFI = 1.00. Comparison of the second and third path analyses indicated that dropping the direct path from maternal mood disorders to daughters' puberty did not adversely affect the fit of the model, $\chi^2_{\text{diff}}(1) = 1.97, p = ns$. Thus, on the basis of parsimony (Loehlin, 1992), the third path analysis provided the best fit to the data and indicates that the relation between maternal history of mood disorders and daughters' pubertal timing was fully mediated by father absence and dyadic stress. The total amount of variance explained in daughters' pubertal timing was 20% in the fully mediated model.

We also tested for the presence of an interaction between father absence and dyadic stress in predicting puberty. A regression analysis was conducted in which daughters' pubertal timing was the dependent variable, dyadic stress and father absence were entered on the first step, and the product term (dyadic stress*father absence) was entered on the second step. The product term was computed from centered variables (as recommended by Jaccard, Turrissi, & Wan, 1990). The interaction was marginally significant ($\beta = -.20, p < .07$), accounting for an additional 4% of

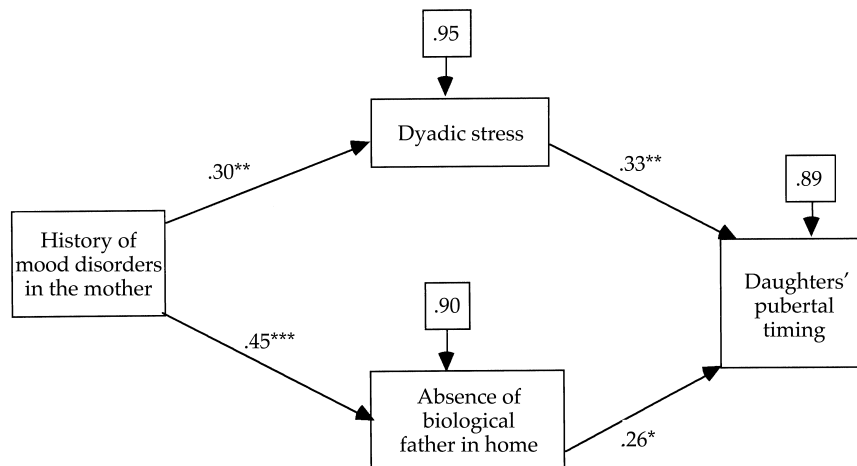


Figure 2 Path analysis of daughters' pubertal timing: * $p < .05$, ** $p < .01$, *** $p < .001$.

the variance in daughters' pubertal timing. To interpret this interaction, we calculated the correlation between dyadic stress and pubertal timing in both father-absent and father-present homes. The interaction was driven by an especially strong relation between dyadic stress and pubertal timing in father-absent homes, $r(33) = .67, p < .001$. Because the interaction was not statistically significant, it was not included in the path analyses. However the meaning and possible implications of this interaction are examined in later analyses (see Relation between Dyadic Stress and Pubertal Timing in Father-Absent/Stepfather-Present Homes).

Secondary path analysis. The secondary path analysis substituted dysfunctional family relationships for dyadic stress. This change enabled us to use the full sample in the secondary analysis. The secondary path analysis attempted to replicate the fully mediated model that is shown in Figure 2. This secondary path analysis fit very well, $\chi^2(2) = 1.59, p = ns$; CFI = 1.00. All paths were significant, $p < .01$, and the modification indices did not suggest any additions or subtractions from the model. For the two paths leading to daughters' pubertal timing—dysfunctional family relationships and father absence—the β s were both .27. The total amount of variance explained in daughters' pubertal timing was 16% in the secondary analysis.

In sum, the mediational model was supported by the data: A history of mood disorders in the mother predicted earlier pubertal timing in the daughter, and this relation was fully mediated by stressful interpersonal relationships in the family and father absence. In addition, the hypothesis that stressful interpersonal relationships in the family and father absence would constitute separate paths to pubertal timing

was supported: These two mediators were independent and each accounted for unique variance in daughters' pubertal timing. The correlations between father absence and dyadic stress, $r(74) = .16, p = ns$, and between father absence and dysfunctional family relationships, $r(87) = .11, p = ns$, were nonsignificant. Finally, all aspects of the model (including full mediation, independence of mediators, and unique prediction of pubertal timing) maintained whether dyadic stress or dysfunctional family relationships was used as the marker of stressful interpersonal relationships in the family.

Consideration of Alternative Explanations

Genetic transmission of pubertal timing through the mother. Although the mediational model was supported by the data, the preceding analyses do not address the possibility that gene effects account for the pattern of covariation with pubertal timing. One possible pathway is that genetic transmission of pubertal timing from mother to daughter accounts for the covariation in the data. According to this scenario, early maturing mothers not only tend to have early maturing daughters but also tend to get married and begin having children at a relatively early age, resulting in more family and marital dysfunction. In the present study, earlier age of first birth in the mother was associated with earlier pubertal timing in daughters as well as with lower SES, more father absence, and more dysfunctional family relationships (see Table 1).² To exam-

² The correlation between mothers' age at first birth and daughters' pubertal timing was marginally significant, $r(87) = -.20, p < .06$.

ine whether early onset of reproduction in mothers accounted for the pattern of covariation with daughters' pubertal timing, we reran both the primary and secondary path analyses after partialling out mothers' age at first marriage and at first birth from daughters' pubertal timing.

In the primary set of three path analyses ($n = 74$), the pattern of results did not change after controlling for mothers' age at both first marriage and first birth. Specifically, in the first path analysis mothers' history of mood disorders still had bivariate relations with daughters' pubertal timing ($\beta = .31, p < .01$), dyadic stress ($\beta = .30, p < .01$), and father absence ($\beta = .45, p < .001$). The addition of paths from dyadic stress and father absence to pubertal timing in the second path analysis significantly enhanced the fit of the model, $\chi^2_{\text{diff}}(2) = 7.31, p < .05$. Finally, dropping the direct path from mothers' history of mood disorders to daughters' pubertal timing in the third path analysis did not adversely affect the fit of the model, $\chi^2_{\text{diff}}(1) = 1.93, p = ns$. Thus, even after controlling for mothers' age at first marriage and first birth, the relation between mothers' history of mood disorders and daughters' pubertal timing remained fully mediated by father absence and dyadic stress. The fit of the third path analysis was excellent, $\chi^2(2) = 2.0, p = .37$; CFI = 1.00.

In the secondary path analysis, using the measure of dysfunctional family relationships ($N = 87$), the pattern of results also remained unchanged. That is, after controlling for mothers' age at first marriage and first birth, the secondary path analysis still fit the data very well, $\chi^2(2) = 1.73, p = .42$; CFI = 1.00. All paths were significant ($p < .05$), and the modification indices did not suggest any additions or subtractions from the model.

Race. African Americans not only tend to experience earlier pubertal maturation than do European Americans (Campbell & Udry, 1995; NHLBI Growth and Health Study Research Group, 1992), but they also tend to experience more father absence and familial stressors (e.g., Dodge, Pettit, & Bates, 1994; Harrison, Wilson, Pine, Chan, & Buriel, 1990). Consistent with past research, African Americans in the present study experienced earlier pubertal timing than did European Americans, as well as lower SES, earlier age of the mother at first birth, and father absence at an earlier age for daughters (see Table 1). Thus, the pattern of covariation with pubertal timing could potentially be accounted for by race. To address this issue, we reran both the primary and secondary path analyses using the European American subsample only. There were not enough African Americans in the study ($n = 10$) to also rerun the analyses on this group.

In the primary set of three path analyses, the pattern of results did not change when using the European American subsample ($n = 66$). Specifically, in the first path analysis mothers' history of mood disorders still had bivariate relations with daughters' pubertal timing ($\beta = .37, p < .001$), dyadic stress ($\beta = .27, p < .05$), and father absence ($\beta = .47, p < .001$). The addition of paths from dyadic stress and father absence to pubertal timing in the second path analysis significantly enhanced the fit of the model, $\chi^2_{\text{diff}}(2) = 6.50, p < .05$. Finally, dropping the direct path from mothers' history of mood disorders to daughters' pubertal timing in the third path analysis did not adversely affect the fit of the model, $\chi^2_{\text{diff}}(1) = 2.89, p = ns$. Thus, in the European American subsample, the relation between mothers' history of mood disorders and daughters' pubertal timing remained fully mediated by father absence and dyadic stress. The fit of the third path analysis was excellent, $\chi^2(2) = 2.89, p = .24$; CFI = .97.

In the secondary path analysis, use of the European American subsample ($n = 77$) also did not change the pattern of results. Specifically, the secondary path analysis still fit the data very well, $\chi^2(2) = 3.36, p = .19$. All paths were significant ($p < .05$), and the modification indices did not suggest any additions or subtractions from the model.

Daughters' pubertal timing as a cause of maternal depression. The theory we have examined suggests that maternal depression is an antecedent stressor that provokes earlier pubertal maturation in daughters (through its effects on marital and family dysfunction). However, the opposite could be true: Early pubertal timing in daughters could be causing maternal mood disorders. Specifically, puberty is associated with an array of behavioral problems and increased distancing of the parent-child relationship (e.g., Steinberg, 1988; Surbey, 1998), which may in turn cause the mothers of pubescent girls to become depressed. To examine this possibility, we looked at daughters' ages at the time of mothers' first episodes of depression. There were 5 mothers in the sample whose first episode occurred after their daughters had turned 10 years old. We recalculated the correlation between maternal history of mood disorders and pubertal timing in daughters excluding these five cases. The correlation in the full sample, $r(87) = .30, p < .01$, remained essentially unchanged when these cases were excluded, $r(82) = .28, p < .01$. Thus, the correlation does not appear to be caused by a small number of mothers whose onset of depression may have coincided with the onset of puberty in their daughters.

Daughters' pubertal timing as a cause of father absence. The theory discussed herein also suggests that

father absence provokes earlier pubertal maturation in daughters. However, the opposite could again be true: Early pubertal timing in daughters could be causing divorce. According to this scenario, the behavioral problems of puberty cause increased family tension and marital disruption and consequently result in more fathers leaving home. To examine this possibility, we looked at the daughter's age at the time the biological father moved out of the home. There were 5 fathers in the sample who moved out after their daughters turned 10 years old. We recalculated the correlation between father absence and pubertal timing in daughters excluding these five cases. The correlation in the full sample, $r(87) = .30, p < .01$, remained essentially unchanged when these cases were excluded, $r(82) = .29, p < .01$. Thus, the correlation does not appear to be driven by a small number of fathers whose departure from the home may have coincided with the onset of puberty in their daughters.

Father Absence versus Stepfather Presence

What is the mechanism underlying earlier pubertal timing in girls in father-absent homes? We hypothesized that exposure to unrelated adult males, especially stepfathers or mothers' boyfriends, would be associated with accelerated sexual maturation in girls. One apparent way to test this hypothesis (see Mekos et al., 1992; Surbey, 1990) is to divide girls in father-absent homes into two groups—those living in single-mother homes versus those living with a mother and stepfather—and then compare them on timing of pubertal maturation. This approach is confounded, however, by the possibility that girls living in single-mother households may still have extensive exposure to mothers' dating partners. Another way to test this hypothesis is to identify girls in father-absent homes who have had a significant alternative father figure in their lives, and then to examine the relation between length of exposure to the alternative father figure and pubertal timing. If exposure to unrelated adult males causes accelerated pubertal development in girls, then greater duration of exposure to unrelated father figures (rather than duration of father absence per se) should be associated with earlier pubertal timing. Consistent with this prediction, there was a significant correlation between age of daughter when an unrelated father figure first came into her life and timing of pubertal maturation, $r(31) = -.37, p < .05$. The younger the daughter at the time of the father figure's arrival, the earlier her pubertal timing. In contrast, there was not a relation between age of daughter when the biological father moved out and timing of pubertal maturation, $r(47) = -.13, p = ns$.

Relation between Dyadic Stress and Pubertal Timing in Father-Absent/Stepfather-Present Homes

As noted earlier (under The Mediational Model), there was a marginally significant interaction between father absence and dyadic stress in predicting daughters' pubertal timing. To examine this interaction further, the mothers were split into two groups: (1) those who rated levels of dyadic stress regarding an unrelated adult male (i.e., stepfather or boyfriend) and (2) those who rated levels of dyadic stress regarding the biological father. In the stepfather/boyfriend families, there was a relation between dyadic stress and pubertal timing, such that greater dyadic stress predicted earlier pubertal timing, $r(33) = .67, p < .001$ (amount of variance accounted for = 45%). As shown in Figure 3, within the stepfather/boyfriend families there was, on average, more than a full standard deviation difference ($SD = .65$) in pubertal timing between girls in the high dyadic stress and low dyadic stress groups. In contrast, in the biologically intact families, there was not a relation between dyadic stress and pubertal timing, $r(41) = .15, p = ns$ (amount of variance accounted for = 2%).

The data presented in Figure 3 indicate that in stepfather/boyfriend homes, earlier pubertal timing in daughters was associated with more dating and marital dysfunction in mothers. It is possible that timing of daughters' pubertal maturation is causing this dysfunction. Specifically, pubertal development in daughters may generate sexual tension between daughters and stepfathers/boyfriends, which may in turn result in conflict between mothers and stepfathers/boyfriends. This sequence of events, however, begs the more interesting question: Why do certain girls in stepfather/boyfriend families experience earlier pubertal maturation than do other girls in stepfather/boyfriend families in the first place? If exposure to unrelated adult males causes earlier pubertal timing in girls, then the 19 girls in the high dyadic stress group (Figure 3, first bar) should have had more exposure to stepfathers/boyfriends than the 14 girls in the low dyadic stress group (Figure 3, second bar). To examine this, we performed a t test comparing the average age of the girls in these two groups when the stepfather/boyfriend first came into their lives. As expected, the girls in the high dyadic stress group were significantly younger ($M = 4.36$ years, $SD = 2.84$) when the stepfather/boyfriend first entered their lives than were the girls in the low dyadic stress group ($M = 7.58$ years, $SD = 2.7$), $t(33) = 2.78, p = .01$. Thus, the girls in the high dyadic stress group not only tended to experience earlier puberty than did the girls in the low dyadic stress group, but they also had longer exposure to stepfathers/boyfriends.

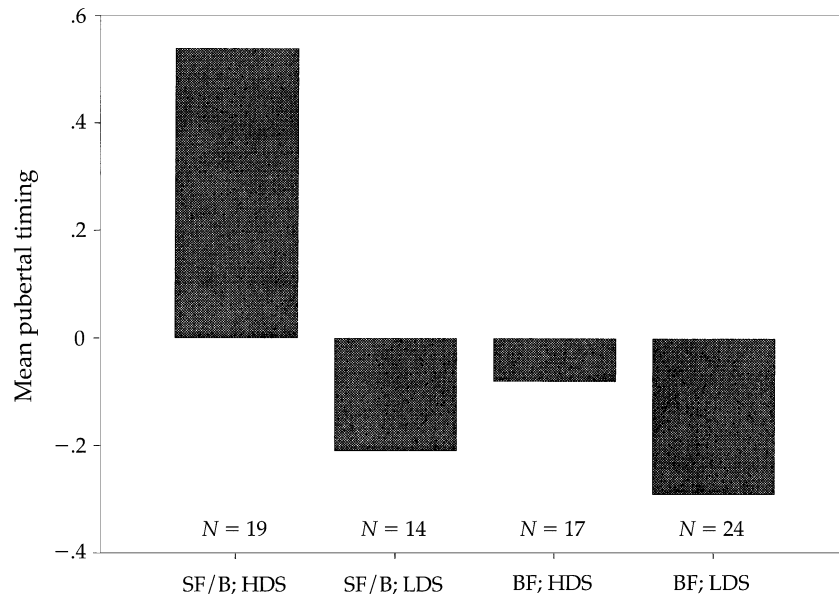


Figure 3 Daughter's pubertal timing as a function of the status of mother's romantic partner (SF/B versus BF) and level of dyadic stress. SF/B = stepfather/boyfriend; BF = biological father; HDS = high dyadic stress; LDS = low dyadic stress.

DISCUSSION

The primary goal of this short-term longitudinal study was to test an evolutionary model of individual differences in girls' pubertal timing. This model posited that a history of psychopathology in mothers would predict earlier pubertal maturation in daughters, and that this relation would be mediated by discordant family relationships and father absence/stepfather presence. The model was supported. In the main test of the model, it was found that a history of mood disorders in mothers significantly predicted earlier pubertal timing in daughters, and this relation was fully mediated by mothers' dyadic stress and absence of the biological father. Early onset of reproduction in the mother also predicted earlier pubertal timing in daughters, as well as more father absence and family dysfunction. Importantly, though, partialling mothers' age at first marriage and first birth out of daughters' pubertal timing did not hurt the model. In addition, the model was upheld when tested in the European American subsample. There were not enough African Americans to evaluate the model in that group.

Does the current study support Belsky et al.'s (1991) evolutionary model of pubertal timing? The most fundamental precept of the model—that childhood stress or conflict in the family environment is associated with early pubertal timing—was supported. All of the indices of stressful family environment employed in this study (i.e., dysfunctional family rela-

tionships, dyadic stress, father absence, maternal psychopathology) predicted earlier pubertal maturation. These data are consistent with past research showing that greater marital and family conflict and/or less marital and family warmth are associated with earlier pubertal timing in girls (Ellis et al., 1999; Graber et al., 1995; Moffitt et al., 1992; Steinberg, 1988; Wiersma et al., 1993). Variation in some family stressors (e.g., dysfunctional family relationships) may have been caused, in part, by onset of puberty in daughters, however, other indices of family stress such as maternal depression and father absence did not prove to be caused by daughters' pubertal timing.

While the present study provides support for the Belsky et al. model, it also suggests new directions for expanding the model. First, maternal depression may be a distal cause of the marital and family dysfunction that has been found to predict earlier pubertal timing in girls. The present study is among the first to examine the relation between parental psychopathology and children's physical maturation (see also Malo & Tremblay, 1997). Second, exposure to unrelated adult males (especially stepfathers and mothers' boyfriends) may provide a second, independent path to early pubertal timing in girls. The present study is the first to show a relation between length of exposure to alternative father figures and daughters' pubertal timing. Thus, this study contributed to the literature on both maternal depression and child development and father absence and child development.

Female Pubertal Timing and Exposure to Unrelated Father Figures

One of the more intriguing findings in this study was the moderating effect of father absence on the relation between dyadic stress and daughters' pubertal timing. In those families in which the mother's primary romantic involvement was with a stepfather or boyfriend, there was a significant relation between dyadic stress and pubertal timing, such that greater dyadic stress predicted earlier pubertal maturation. In these families, mother's dyadic stress accounted for almost half of the variance in daughter's pubertal timing. In contrast, in those families in which the mother's primary romantic involvement was with the biological father, there was not a significant relation between dyadic stress and pubertal timing. Related to this interaction, we found that daughter's age when an alternative father figure moved into her home, rather than daughter's age when the biological father moved out, best accounted for earlier pubertal maturation in daughters. These results highlight a potentially important role for unrelated adult males in regulating timing of pubertal maturation in girls. Females whose childhood experiences include both (1) substantial exposure to unrelated father figures and (2) relatively high levels of discord between their mothers and these father figures may be at considerable risk for early pubertal maturation.

These results are consistent with research on a variety of other mammalian species documenting that pheromones produced by unrelated adult male conspecifics accelerate female pubertal maturation (Sanders & Reinisch, 1990; Vandenberg, 1983). In nonhuman animals, accelerated female pubertal development in the presence of unrelated males presumably functions to facilitate reproduction with those males (analogous to the introduction of unfamiliar males inducing ovulation and estrous behavior in female primates; Hrdy, 1981). If the presence of an alternative father figure causes earlier pubertal timing in girls, and this in turn creates subtle pressure toward sexuality with the father figure, the combination of circumstances could result in substantial conflict between the mother and the father figure (as well as between the daughter and the father figure). This sequence of events may contribute to the strong positive correlation between daughters' pubertal timing and mothers' dyadic stress in stepfather/boyfriend households. Importantly, though, the present data suggest that this positive correlation is an outcome of other processes that anteceded daughters' pubertal development. In the 33 stepfather/boyfriend households, there were 19 girls whose mothers reported relatively high dy-

adic stress and 14 girls whose mothers reported relatively low dyadic stress. The 19 girls in the high dyadic stress group not only tended to experience much earlier pubertal timing than did the 14 girls in the low dyadic stress group (mean difference between the groups exceeded a full standard deviation), but they also had on average more than 3 years of additional exposure to stepfathers/boyfriends. These data are consistent with the hypothesis that greater pheromonal exposure to stepfathers/boyfriends provokes earlier pubertal maturation in daughters, which in turn may generate dyadic stress. In the high dyadic stress group, most girls were still in early childhood (*mean* age = 4.36 years; minimum value = .00, 1st quartile = 2.50, median = 5.00, 3rd quartile = 7.00, max value = 8.00) when the stepfather/boyfriend first entered their lives. One might question whether exposure to unrelated adult males at such an early age could influence timing of pubertal maturation years later. It is interesting to note that the animal literature suggests that even early experience of pheromonal cues can affect reproductive development.³

In total, the stepfather/boyfriend data suggest an important extension of the Belsky et al. (1991) model. We propose that there are two separate classes of psychosocial accelerators acting on female pubertal maturation: interpersonal family stressors and exposure to unrelated adult males. The original formulation of the model conflated these two paths. Specifically, Belsky et al. hypothesized that divorce would accelerate pubertal maturation specifically because family disruption is stressful. The current data are more consistent with the view that divorce accelerates pubertal maturation in girls because of its association with increased exposure to unrelated father figures. This interpretation can account for the stepfather/boyfriend effects reported in the current study, and it is supported by the finding that father absence and family stress were uncorrelated and each contributed unique variance to the prediction of pubertal timing. These unique relations maintained whether dyadic stress or dysfunctional family relationships were used as the measure of family stress. These data are

³ Experimental work on mice has shown that exposure to male pheromones very early in life, long before the onset of puberty, results in earlier pubertal maturation in females (Bronson, 1975; Drickamer, 1988). This early exposure to puberty-accelerating pheromones apparently has a priming effect on the animal's reproductive physiology: female mice that are exposed to the pheromones of adult male mice during the first 3 weeks of life (compared to female mice that are not exposed during this time period) tend to respond to subsequent exposure to male pheromones with significantly greater pubertal acceleration (Caretta, Caretta, & Cavaggioni, 1995).

consistent with past research showing that family conflict and father absence are unrelated and that each contributes unique variance to the prediction of menarcheal age (Moffitt et al., 1992).

What are the implications of the stepfather/boyfriend data for research on father absence? Previous studies have found a small (though consistent) relation between age of child when the biological father moved out and timing of female pubertal maturation, such that younger age predicts earlier puberty. Moffitt and colleagues (1992) reported the correlation as $-.12$, Surbey (1990) reported a correlation of $-.13$, and the current study found the correlation to be $-.13$. Why are these relations so small, accounting for less than 2% of the variance? We propose that the small effect sizes result from conceptualizing and operationalizing the construct as father absence rather than stepfather/boyfriend presence. The current study found a significant $-.37$ correlation between age of daughter when the first alternative father figure came into her life and timing of female pubertal maturation. Other researchers may benefit from analyzing father absence data to look for effects of stepfather/boyfriend presence (in conjunction with marital and family stress).

Limitations and Future Directions

Limitations of the present study should be noted because they provide important directions for future research. First, although the findings of the present study are quite provocative, they need to be replicated with a larger sample that includes approximately equal numbers of disordered and nondisordered mothers. One strength of the current study was that mothers had a range of severity of psychopathology, which provided for substantial variability in the maternal psychopathology measure and associated predictors such as marital and family dysfunction. The generalizability of the results to a more normative sample needs to be examined, however.

Second, our attempt to examine the hypothesis that genetic transmission of pubertal timing accounts for the relations between marital and family dysfunction and daughters' puberty was limited by unavailability of data on timing of mothers' pubertal maturation. The alternative behavior genetic model posits that early maturing mothers not only have early maturing daughters (through genetic transmission), but also tend to raise their children in relatively stressful family environments as a consequence of early age at first marriage and at first birth. We addressed this alternative model by controlling for mothers' age at both first marriage and first birth in the path analyses. A better

way to address this alternative, however, would be to actually control for mothers' pubertal timing.

Finally, further research needs to be conducted that examines the mechanisms linking maternal psychopathology, family dysfunction, and father absence/stepfather presence to timing of puberty. What are the processes through which environmental factors influence hormonal changes in girls? In animal research, pheromones produced by unrelated adult male conspecifics have been shown to accelerate female pubertal development (Sanders & Reinisch, 1990; Vandenberg, 1983). In humans, pheromones produced by men's axillary sweat glands have been shown to reduce variability in women's menstrual cycles (Cutler et al., 1986). It seems plausible to suggest that exposure to pheromones of unrelated adult males could also influence the timing of pubertal maturation in human females. The current data on alternative father figures are at least consistent with this hypothesis. If such a pheromonal mechanism does exist, then many questions arise concerning its operation: How much pheromonal exposure is needed to produce the accelerating effect on girls' pubertal maturation? Do girls have "critical periods" in which they are most sensitive to pheromonal exposure? Can male relatives, such as uncles and cousins, produce the effect, or is it only males who are genetically unrelated? How do pheromonal mechanisms interact with stress-related hormonal processes to regulate pubertal timing?

In summary, the present study found support for the evolutionary model of pubertal timing proposed by Belsky et al. (1991) linking stressful family environments to earlier puberty in girls and extended the model to include (1) maternal psychopathology as a more distal predictor of marital and family dysfunction and pubertal timing, and (2) stepfather/boyfriend presence as a second psychosocial path to early pubertal maturation.

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REFERENCES

- American Psychiatric Association. (1987). *Diagnostic and statistical manual for mental disorders* (3rd ed., Rev.). Washington, DC: Author.
- Aw, E., & Tye, C. Y. (1970). Age of menarche of a group of Singapore girls. *Human Biology*, 42, 329–336.
- Beach, S. R. H., & Nelson, G. M. (1990). Pursuing research on major psychopathology from a contextual perspective: The example of depression and marital discord. In G. H. Brody & I. E. Sigel (Eds.), *Methods of family research: Biographies of research projects: Vol. 2. Clinical populations* (pp. 227–259). Hillsdale, NJ: Erlbaum.
- Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, 62, 647–670.
- Bentler, P. M. (1993). *EQS: Structural Equations Program Manual*. Los Angeles: BMPD Statistical Software.
- Bielicki, T., Waliszko, A., Hulanicka, B., & Kotlarz, K. (1986). Social class gradients in menarcheal age in Poland. *Annals of Human Biology*, 13, 1–11.
- Billings, A. G., & Moos, R. H. (1985). Life stressors and social resources affect posttreatment outcomes among depressed patients. *Journal of Abnormal Psychology*, 94, 140–153.
- Bronson, F. H. (1975). A developmental comparison of steroid-induced and male-induced ovulation in young mice. *Biology of Reproduction*, 12, 431–437.
- Brooks-Gunn, J. (1988). Antecedents and consequences of variations in girls' maturational timing. *Journal of Adolescent Health Care*, 9, 365–373.
- Burrell, R. J. W., Healy, M. J. R., & Tanner, J. M. (1961). Age at menarche in South African Bantu schoolgirls living in the Transkei Reserve. *Human Biology*, 33, 250–261.
- Byles, J. A., Byrne, C., Boyle, M. H., & Offord, D. R. (1988). Ontario Child Health Study: Reliability and validity of the General Functioning Scale of the Family Assessment Device. *Family Process*, 27, 97–104.
- Calabrese, L. H., Kirkendall, D. T., Floyd, M., Rapoport, S., Williams, G. W., Weiker, G. F., & Bergfeld, J. A. (1983). Menstrual abnormalities, nutritional patterns, and body composition in female classical ballet dancers. *Physician and Sports Medicine*, 11, 86–98.
- Campbell, B. C., & Udry, J. R. (1995). Stress and age at menarche of mothers and daughters. *Journal of Biosocial Science*, 27, 127–134.
- Caretta, C. M., Caretta, A., & Cavaggioni, A. (1995). Pheromonally accelerated puberty is enhanced by previous experience of the same stimulus. *Physiology and Behavior*, 57, 901–903.
- Caspi, A., & Moffitt, T. E. (1991). Individual differences are accentuated during periods of social change: The sample case of girls at puberty. *Journal of Personality and Social Psychology*, 61, 157–168.
- Chisholm, J. S. (1996). The evolutionary ecology of attachment organization. *Human Nature*, 7, 1–38.
- Conger, J. J. (1984). *Adolescence and youth*. New York: Harper & Row.
- Cummings, E., & Davies, P. (1994). Maternal depression and child development. *Journal of Child Psychology and Psychiatry*, 35, 73–112.
- Cutler, W. B., Preti, G., Krieger, A., Huggins, G. R., Garcia, C. R., & Lawley, H. J. (1986). Human axillary secretions influence women's menstrual cycles: The role of donor extracts from men. *Hormones and Behavior*, 20, 463–473.
- Dodge, K. A., Pettit, G. S., & Bates, J. E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development*, 65, 649–665.
- Downey, G., & Coyne, J. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin*, 108, 50–76.
- Draper, P., & Harpending, H. (1982). Father absence and reproductive strategy: An evolutionary perspective. *Journal of Anthropological Research*, 38, 255–273.
- Drickamer, L. C. (1988). Prewaning stimulation with urinary chemosignals and age of puberty in female mice. *Developmental Psychobiology*, 21, 77–87.
- Ellis, B. J., McFadyen-Ketchum, S., Dodge, K. A., Pettit, G. S., & Bates, J. E. (1999). Quality of early family relationships and individual differences in the timing of pubertal maturation in girls: A longitudinal test of an evolutionary model. *Journal of Personality and Social Psychology*, 77, 387–401.
- Ellison, P. T. (1990). Human ovarian functioning and reproductive ecology: New hypotheses. *American Anthropologist*, 92, 933–952.
- Emery, R. E. (1988). *Marriage, divorce, and children's adjustment*. Newbury Park, CA: Sage.
- Epstein, N. B., Baldwin, L. N., & Bishop, L. N. (1983). The McMaster Family Assessment Device. *Journal of Marital and Family Therapy*, 9, 171–180.
- Epstein, N. B., Bishop, L. N., & Levine, S. (1978). The McMaster Model of Family Functioning. *Journal of Marriage and Family Counseling*, 4, 19–31.
- Farber, S. L. (1981). *Identical twins reared apart: A reanalysis*. New York: Basic.
- Feldman, S., & Elliot, G. (Eds.). (1990). *At the threshold: The developing adolescent*. Cambridge, MA: Harvard University Press.
- Flannery, D. J., Rowe, D. C., & Gulley, B. J. (1993). Impact of pubertal status, timing, and age on adolescent sexual experience and delinquency. *Journal of Adolescent Research*, 8, 21–40.
- Frisch, R. E. (1983). Fatness, menarche and fertility. In S. Golub (Ed.), *Menarche: The transition from girl to woman* (pp. 5–20). Toronto, Canada: D. C. Heath.
- Garber, J., Robinson, N. S., & Valentiner, D. (1997). The relation between parenting and adolescent depression: Self-

- worth as a mediator. *Journal of Adolescent Research*, 12, 12–33.
- Garn, S. M. (1980). Continuities and change in maturational timing. In O. G. Brim, Jr., & J. Kagan (Eds.), *Constancy and change in human development* (pp. 113–162). Cambridge, MA: Harvard University Press.
- Graber, J. A., Brooks-Gunn, J., & Warren, M. P. (1995). The antecedents of menarcheal age: Heredity, family environment, and stressful life events. *Child Development*, 66, 346–359.
- Graber, J. A., Lewinsohn, P. M., Seeley, J. R., & Brooks-Gunn, J. (1997). Is psychopathology associated with the timing of pubertal development? *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 1768–1776.
- Harrison, A. O., Wilson, N. N., Pine, C. H., Chan, S. Q., & Buriel, R. (1990). Family ecologies of ethnic minority children. *Child Development*, 61, 347–362.
- Hoge, R. D., Andrews, D. A., Faulkner, P., & Robinson, D. (1989). The Family Relationship Index: Validity data. *Journal of Clinical Psychology*, 45, 897–903.
- Holahan, C. J., & Moos, R. H. (1983). The quality of social support: Measures of family and work relationships. *British Journal of Clinical Psychology*, 22, 157–162.
- Hollingshead, A. B. (1975). *Four Factor Index of Social Status*. New Haven, CT: Yale University, Department of Sociology.
- Hrdy, S. B. (1981). *The woman that never evolved*. Cambridge, MA: Harvard University Press.
- Hulanicka, B. (1999). Acceleration of menarcheal age of girls from dysfunctional families. *Journal of Reproductive and Infant Psychology*, 17, 119–132.
- Izard, M. K. (1990). Social influences on the reproductive success and reproductive endocrinology of prosimian primates. In T. E. Ziegler & F. B. Bercovitch (Eds.), *Socioendocrinology of primate reproduction* (pp. 159–186). New York: Wiley-Liss.
- Jaccard, J., Turrissi, R., & Wan, C. K. (1990). *Interaction effects in multiple regression*. Newbury Park, CA: Sage.
- Jones, B., Leeton, J., McLeod, I., & Wood, C. (1972). Factors influencing the age of menarche in a lower socioeconomic group in Melbourne. *The Medical Journal of Australia*, 2, 533–535.
- Kampert, J. B., Whittemore, A. S., & Paffenbarger, R. S. (1988). Combined effects of childbearing, menstrual events, and body size on age-specific breast cancer risk. *American Journal of Epidemiology*, 128, 962–979.
- Kaprio, J., Rimpela, A., Winter, T., Viken, R. J., Rimpela, M., & Rose, R. J. (1995). Common genetic influence on BMI and age at menarche. *Human Biology*, 67, 739–753.
- Lee, M. M. C., Chang, K. S. F., & Chan, M. M. C. (1963). Sexual maturation of Chinese girls in Hong Kong. *Pediatrics*, 32, 389–398.
- Lindgren, G. (1976). Height, weight, and menarche in Swedish urban school children in relation to socioeconomic status and regional factors. *Annals of Human Biology*, 3, 510–528.
- Loehlin, J. C. (1992). *Latent variable models: An introduction to factor, path, and structural analysis*. Hillsdale, NJ: Erlbaum.
- Malo, J., & Tremblay, R. E. (1997). The impact of parental alcoholism and maternal social position on boys' school adjustment, pubertal maturation, and sexual behavior: A test of two competing hypotheses. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 38, 187–197.
- Mekos, D., Hetherington, E. M., & Clingempeel, W. G. (1992, March). Psychosocial influences on the rate and timing of pubertal development. In L. Steinberg (Chair), *Psychosocial antecedents of the timing of puberty*. Symposium conducted at the biennial meetings of the Society for Research on Adolescence, Washington, DC.
- Moffitt, T. E., Caspi, A., Belsky, J., & Silva, P. A. (1992). Childhood experience and onset of menarche: A test of a sociobiological model. *Child Development*, 63, 47–58.
- Morris, N., & Udry, J. R. (1980). Validation of a self-administered instrument to assess stage of adolescent development. *Journal of Youth and Adolescence*, 9, 271–280.
- Ness, R. (1991). Adiposity and age of menarche in Hispanic women. *American Journal of Human Biology*, 3, 41–48.
- NHLBI Growth and Health Study Research Group. (1992). Obesity and cardiovascular disease risk factors in black and white girls: the NHLBI Growth and Health Study. *American Journal of Public Health*, 82, 1613–1620.
- Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, 17, 117–133.
- Porter, B., & O'Leary, K. D. (1980). Marital discord and childhood behavior problems. *Journal of Abnormal Child Psychology*, 8, 287–295.
- Sanders, S. A., & Reinisch, J. M. (1990). *Biological and social influences on the endocrinology of puberty: Some additional considerations*. In J. Bancroft & J. M. Reinisch (Eds.), *Adolescence and puberty* (pp. 50–62). New York: Oxford.
- Spanier, G. B. (1976). Measuring dyadic adjustment: New scales for assessing the quality of marriage and similar dyads. *Journal of Marriage and the Family*, 38, 15–28.
- Spitzer, R. L., Williams, J. B. W., Gibbon, M., & First, M. B. (1990). *User's guide for the Structured Clinical Interview for DSM-III-R*. Washington, DC: American Psychiatric Association.
- Steinberg, L. (1988). Reciprocal relation between parent-child distance and pubertal maturation. *Developmental Psychology*, 24, 122–128.
- Steinberg, L. (1992, March). Discussant's comments. In L. Steinberg (Chair), *Psychosocial antecedents of the timing of puberty*. Symposium conducted at the biennial meetings of the Society for Research on Adolescence, Washington, DC.
- Stern, K., & McClintock, M. K. (1998). Regulation of ovulation by human pheromones. *Nature*, 392, 177–179.
- Surbey, M. (1990). Family composition, stress, and human menarche. In F. Bercovitch & T. Zeigler (Eds.), *Socioendocrinology of primate reproduction* (pp. 71–97). New York: Alan R. Liss.
- Surbey, M. (1998). Parent and offspring strategies in the transition at adolescence. *Human Nature*, 9, 67–94.
- Susman, E. J., Nottelman, E. D., Inoff-Germain, G. E., Loriaux, D. L., & Chrousos, G. P. (1985). The relation of relative hormonal levels and physical development and

- social-emotional behavior in young adolescents. *Journal of Youth and Adolescence*, 14, 245–264.
- Tanner, J. M. (1990). *Foetus into man: Physical growth from conception to maturity*. Cambridge, MA: Harvard University Press.
- Treloar, S. A., & Martin, N. G. (1990). Age at menarche as a fitness trait: Nonadditive genetic variance detected in a large twin sample. *American Journal of Human Genetics*, 47, 137–148.
- Udry, J. R. (1979). Age at menarche, at first intercourse and at first pregnancy. *Journal of Biosocial Science*, 11, 433–441.
- Udry, J. R., & Cliquet, R. L. (1982). A cross-cultural examination of the relationship between ages at menarche, marriage, and first birth. *Demography*, 19, 53–63.
- Vandenbergh, J. G. (1983). Pheromonal regulation of puberty. In J. G. Vandenbergh (Ed.), *Pheromones and reproduction in mammals* (pp. 95–112). New York: Academic Press.
- Vihko, R. K., & Apter, D. L. (1986). The epidemiology and endocrinology of the menarche in relation to breast cancer. *Cancer Survey*, 5, 561–571.
- Warren, M. P., Brooks-Gunn, J., Fox, R., Lancelot, C., Newman, D., & Hamilton, W. G. (1991). Lack of bone accretion and amenorrhea in young dancers: Evidence for a relative osteopenia in weight bearing bones. *Journal of Clinical Endocrinology and Metabolism*, 72, 847–853.
- Wellens, R., Malina, R., Roche, A., Chumlea, W., Guo, S., & Siervogel, R. (1992). Body size and fatness in young adults in relation to age at menarche. *American Journal of Human Biology*, 4, 783–787.
- Wierson, M., Long, P. J., & Forehand, R. L. (1993). Toward a new understanding of early menarche: The role of environmental stress in pubertal timing. *Adolescence*, 28, 913–924.
- Ziegler, T. E., Snowdon, C. T., & Uno, H. (1990). Social interactions and determinations of ovulation in tamarins (*Saguinus*). In T. E. Ziegler & F. B. Bercovitch (Eds.), *Socioendocrinology of primate reproduction* (pp. 159–186). New York: Wiley-Liss.