The Antecedents of Menarcheal Age: Heredity, Family Environment, and Stressful Life Events

Julia A. Graber, Jeanne Brooks-Gunn, and Michelle P. Warren

Columbia University

Variations in pubertal timing, specifically age at menarche, have been associated with several antecedents, both genetic and environmental. Recent research has considered a broader range of environmental stressors and their influence on the development of the reproductive system. In this investigation, the following possible antecedents were considered: (a) hereditary transmission, (b) weight and weight for height, (c) stressful life events, (d) family relations, (e) absence or presence of an adult male in the household, and (f) psychological adjustment. Subjects were 75 premenarcheal girls between the ages of 10 and 14 drawn from a larger longitudinal investigation of adolescent development. Girls were from white, well-educated, middle- to upper-middle-class families and attended private schools in a northeastern urban area. While breast development, weight, family relations, and depressive affect were predictive of age at menarche, family relations predicted age at menarche above the influence of breast development or weight. A trend for maternal age at menarche to predict adolescent’s age at menarche was found. Weight for height, presence of an adult male in the household, and stressful events were not predictive of age at menarche. These complex interactions of biological and psychosocial development demonstrated here may account for some extent for the inter- and intradimensional variation observed in pubertal development.

The transition into adolescence is demarcated by the onset of pubertal development and the concomitant changes in psychological development, social interactions, and environmental context (Feldman & Elliott, 1990; Lerner & Foch, 1987). The hormonal and physical changes occurring during puberty have often been linked to perturbations in mood as well as longer-term alterations in psychological development (e.g., changes in cognitive development or self-esteem). The examination of pubertal influences on behavior have most often focused on three areas: (a) hormonal effects on behavior indicated by mood changes, (b) pubertal status effects, that is, behavioral changes that are linked to attaining a particular stage of development, and (c) pubertal timing effects which attribute the influence of pubertal development on behavior to developing within a social context, that is, going through puberty earlier, later, or at about the same time as one’s peers or some referent group. Such models focus on the underlying premise that pubertal development influences behavior with less emphasis on bidirectional or transactional influences between pubertal, behavioral, and environmental processes (Brooks-Gunn, Gruber, & Paikoff, 1994; Buchanan, Eccles, & Becker, 1992).

In the present paper, we examine the antecedents of pubertal development in order to identify biological, psychological, and environmental predictors of pubertal development, specifically timing of menarche in girls. We also examine models which delineate paths for linking antecedents and consequences of menarcheal timing.

The research reported in this paper was funded by the National Institutes of Health (NICHHD) and the W. T. Grant Foundation. Additionally, we wish to thank all of those who contributed their time and energy to the Adolescent Study Program: J. Gargiulo, D. Friedman, L. Ferrington, and M. Samelson for data collection; the schools; and, especially, the adolescent girls and their families for participating in the study. Portions of this paper were presented in a symposium, "Psychosocial Antecedents of the Timing of Puberty," at the biennial meeting of the Society for Research on Adolescence, March 1992, Washington, DC. Reprint requests may be addressed to Julia A. Graber, Ph.D., The Adolescent Study Program, Box 39, Teachers College, Columbia University, 525 W. 120th Street, New York, NY 10027, (212) 678-3337, fax: (212) 678-3676, e-mail: JAG51@Columbia.edu.

[Child Development, 1995, 66, 346–359. ©1995 by the Society for Research in Child Development, Inc. All rights reserved. 0009-3920/95/6602-0011850.00]
Pubertal development is not a singular event but rather a combination of biologically and temporally linked processes such as the growth spurt in height, growth of pubic hair, or breast development. In seminal work charting the progression of key indicators of pubertal development, Marshall and Tanner (1969, 1970) emphasize that the timing of onset and rate of progression of pubertal development varies greatly across adolescents. Furthermore, while the pattern of development among indicators remains fairly stable across individuals (e.g., girls pass peak acceleration in growth in height prior to the onset of menses), asynchronies in pattern and rate do occur within an individual (Brooks-Gunn, Petersen, & Eichorn, 1985; Eichorn, 1975).

Predicting Menarcheal Timing

Menarche is perhaps the most studied pubertal event as it signals not only the advent of reproductive capacity but also has psychological significance within the familial, peer, and societal domains (Brooks-Gunn & Reiter, 1990; Petersen, 1983; Riedman & Koff, 1985). The consequences of menarcheal timing have been noted most frequently for girls who reach menarche earlier than their peers. Most often early maturation has been associated with negative consequences for girls such as depressive affect (Magnusson, Stattin, & Allen, 1985; Petersen, Sarigiani, & Kennedy, 1991; Riedman & Koff, 1991), negative body image (Simmons & Blyth, 1987), problem behavior and long-term consequences demonstrated in lowered career achievement (Stattin & Magnusson, 1990).

Genetic Influences

Variations in pubertal timing, specifically age at menarche, have been associated with several antecedents, both genetic and environmental. Hereditary transmission has generally been assessed via associations between maternal and daughter age at menarche (Brooks-Gunn & Warren, 1988; Damon, Damon, Reed, & Valadian, 1969; Zacharias & Wurtman, 1969). In these studies, correlations between maternal and daughter age at menarche have ranged from .26 to .45. It has been suggested that maternal/daughter associations, in and of themselves, may include common environmental factors rather than being solely attributed to genetic factors (Steinberg, 1989). However, in a comparison of menarcheal timing between girls who were and were not enrolled in national dance company schools, Brooks-Gunn and Warren (1988) reported an association between maternal and daughter age at menarche for nondancers only, indicating that environmental factors override genetic links for girls who engage in intensive physical training. Hence, in the average sample of girls, links between maternal and daughter age at menarche are likely to be in some part due to genetic factors. The average size of this association (approximately .30) suggests that additional factors contribute to the timing of the onset of menses. While one factor is likely to be paternal hereditary influences, environmental contexts have demonstrated an influence on the timing of the onset of puberty.

Environmental Influences: Physical Stressors

Environmental factors such as exercise, nutrition, and weight have also all been associated with pubertal timing (Brooks-Gunn, 1988). High levels of exercise, observed in competitive athletes, have been found to delay pubertal development (Calabrese et al., 1983; Malina, 1983; Warren et al., 1991; Warren, Brooks-Gunn, Hamilton, Hamilton, & Warren, 1986). For example, adolescent dancers have later ages at menarche than other girls. While it is possible that girls who pursue dance or other athletic training are genetically predisposed to have later menarcheal timing, it is more likely that the physical activity itself delays pubertal development. In an intensive study of physical development and exercise behaviors of adolescent ballet dancers, Warren (1980) found that cessation of exercise and training for as little as 2 months prompted menarche and advances in breast development.

While the associations reported by Warren (1980) indicate that physical activity influences pubertal development independent of changes in weight, the percent of body fat or weight for one’s height has also predicted onset of menarche (Frisch & Revelle, 1971). While weight, height, and body fat have a genetic component, they are subject to environmental influences and may reflect both genetics and environment. Initially, Frisch (1983) proposed that a critical amount or percentage of body fat is required before the onset of menses or mature reproductive functioning will occur. The premise, using animal models, is that the mother must first be capable of sustaining her own health and survival prior to supporting offspring, as demonstrated in some species by the reabsorption or spontaneous abortion of fetuses under conditions of environmental stress such as starvation. (See Wasser & Barash,
Concurrently, Steinberg (1989) suggested that family conflict and/or distance was associated with earlier pubertal maturation in girls. Steinberg and colleagues (Belsky, Steinberg, & Draper, 1991; Steinberg, 1988, 1989) have proposed an evolutionary model of socialization in which stressful family environments lead to behavioral problems and earlier onset of pubertal development. Individuals growing up in conflictual or unstable home environments may experience adjustment difficulties such as behavioral problems or depression as well as an earlier pubertal transition. They go on to suggest that earlier development and adjustment problems lead adolescents to make reproductive choices, such as single parenthood or early parenthood, which will in turn result in unstable environments for their offspring. While these researchers encourage testing their proposals via studies in a behavioral genetics framework, several existing datasets are applicable for testing specific components of their model such as psychosocial influences on pubertal development.

Steinberg’s (1988) initial study of influences of parent-child distance on pubertal development reported accelerating effects of mother-daughter distance on change in pubertal development. No effects were found for boys or for relationships with fathers. In this study, pubertal status was assessed via noninvasive observer ratings of the adolescents during the course of a home visit. Other, more exploratory work focusing on global indicators of development have found similar accelerating effects for girls only (Ellis, 1991).

In a large longitudinal study of girls in New Zealand, Moffitt and her colleagues (Moffitt et al., 1992) considered additive models for predicting age at menarche which included family stressors, behavioral problems, and weight. Family conflict and behavior problems were assessed at age 7 and weight was obtained from self-report at age 9. They found support for family conflict and father absence at age 7 leading to earlier maturation after accounting for the influence of weight. Social class effects were not found in this sample although it spanned a range of socioeconomic levels. In contrast to the prediction of the evolutionary model (Belsky et al., 1991), psychological problems did not mediate the association among family conflict, weight, and age at menarche. Instead, family conflict, father absence, and weight
exhibited additive influence on menarcheal timing.

As these authors acknowledge, the New Zealand data set does not have several possibly critical variables such as maternal timing of puberty which may influence the effects. In addition, they do not have information on the timing of pubertal events other than menarche. It may be that, with the onset of puberty (e.g., hormonal changes), conflict in the family increases so that it is not conflict causing early maturation but early maturation causing conflict. Note that the first hormonal increases of pubertal development begin around age 8 in most girls and would be earlier for early-maturing girls (Reiter & Grumbach, 1982). The New Zealand data set also does not include information on stressful life events. No studies have as yet incorporated multiple measures of pubertal development, maternal factors, stressful life events, and adjustment in the same design. The present study has data on each of these factors which may influence pubertal timing.

The purpose of the present investigation was to examine antecedents of age at menarche in a sample of premenarchal girls between the ages of 10 and 14 who have been followed longitudinally. The following possible antecedents were considered: (a) hereditary transmission as observed through maternal age at menarche, (b) weight and weight for height or percent body fat, (c) stressful life events occurring within and outside of the family context, (d) family relations including the relationship with parents, parental approval, and conflict in family relations, (e) father absence or the presence of an adult male father figure in the household, and (f) psychological adjustment as indicated by depressive affect, and more general internalizing and externalizing behaviors. By considering differences in the development of secondary sexual characteristics, it can be established that environmental influences on menarcheal timing are not due to responses to external signs of development by factors in the environment.

Method

Participants

The subsample of premenarchal girls was drawn from a larger longitudinal investigation of female adolescent development. Subjects were 75 girls between the ages of 10 and 14 at the first time of testing. While more girls participated in the larger study program, only girls who were premenarchal were used for the present investiga-

Graber, Brooks-Gunn, and Warren 349
tion. Girls were from white, well-educated, middle- to upper-middle-class families and attended private schools in a major northeastern urban area.

Girls completed questionnaires about several aspects of their psychosocial functioning and development annually. In addition, physical examinations were conducted at a medical laboratory in a hospital in the community.

Measures of Pubertal/Physical Development

Girls provided reports of their age at menarche during interviews. As the subsample of girls in this study were premenarchal, age at menarche was obtained from subsequent times of measurement, specifically, from the assessment in which it was first reported. Adolescent girls have been found to be accurate reporters of their age at menarche (Bean, Leeper, Wallace, Sherman, & Jagger, 1979; Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Rierdan & Koff, 1985). When inaccuracies between girls and their mothers have been observed, they were most often associated with an initial denial or hiding of the event by the daughters; in such cases, accuracy improved in subsequent interviews (Petersen, 1983).

Physician ratings of Tanner stages for breast development (Marshall & Tanner, 1969) were collected yearly during visits to the laboratory by one of the coauthors or a nurse practitioner. Tanner ratings are made on a five-point scale ranging from “no development” to “adult development.”

Weight and height were measured annually during the laboratory visit. Assessments of the percentage of body fat based on the height and weight data were calculated via two methods: (a) estimations based on equations developed by Mellits and Cheek (1970), and (b) the ponderal index, weight/height$^2$ (using kilograms and meters). The Mellits and Cheek equations are the same as those used by Frisch in her comparisons of body fat and menarche (e.g., Frisch & Revelle, 1971). However, estimations from regression equations have been criticized as they were not developed on normative samples. Alternately, the ponderal index is a well-established estimate of body mass and is highly associated with measurements of body fat obtained through hydrostatic weighing ($r = .89$; Roche, Siervogel, Chunlea, & Webb, 1981). In comparisons of the two measurement methods in the total sample, estimates were highly corre-
Maternal Correlates

Maternal age at menarche was used as a proxy for hereditary transmission although it includes environmental influences. Mothers completed questionnaires annually. Age at menarche was reported at the first time of measurement and was reported to the nearest year, as this is the increment for which adult women have demonstrated that they can accurately report their age at menarche (Bean et al., 1979).

Stressful Life Events

A measure of life events covering the domains of family, school, and peer events was administered annually. Girls were asked to indicate which life events had happened to them in the past 6 months. The occurrence of negative life events in school, peer, and family domains were summed for each category. Examples of negative events are: called to principal's office for disciplinary action, did worse than expected in school work, broke up with a boyfriend, falling out/broke up with close or best friend, parents got separated or divorced, and someone in your family died. Positive events were summed for school and peer domains only. Examples of positive events are: did better than expected in school work, won an award at school, started dating, found a boyfriend, and made a new best friend. The total number of negative and total number of positive events across domains as well as the total number of any events were also calculated. The life events measure was adapted from the Coddington (1972) to be more specific to early adolescence; as such, it is similar to the life events inventory used by Surbey (1990).

Family Environment and Relations

Determination of exposure to an adult male was made from girls' reports of who lived with them at the first time of measurement. Each girl reported whether there was a father, stepfather, or any other adult male living in the home. Twelve girls reported no adult male in the household. As most girls lived with their biological father and as the number living with an unrelated adult male (i.e., stepfather) was quite small (N = 1), it was not possible to examine differences among girls with no adult male present, stepfathers present, and father present. The one girl who lived with a stepfather was removed from analyses although her presence or absence in analyses did not change any of the results. In addition, family constellation did not change substantially over the first year of the study; that is, very few girls began to live with a stepfather or ceased to live with their father prior to becoming menarcheal.

Girls completed a 12-item version of the Family Relations Scale of the Self-Image Questionnaire for Young Adolescents (SIQYA; Petersen, Schulenberg, Abramowitz, Offer, & Jarcho, 1984). The SIQYA has established reliability and validity with early adolescents. Adolescents rated how much each statement described themselves on a Likert-type scale from 1, "does not describe me at all," to 6, "describes me very well." The standard scoring of the SIQYA reverse codes negative items so that high scores reflect better family relations. The abbreviated version of the SIQYA has been found to be comparable to the longer version (Brooks-Gunn, Rock, & Warren, 1989).

Because the Family Relations Scale measures multiple aspects of family functioning, the 12 items were factor analyzed using principal factor extraction and oblique rotation, as subscales were expected to be correlated. Factor analysis was done with the complete sample available rather than only the premenarcheal girls whose mothers participated, in order to increase the N and generalizability for the procedure. Items were classified onto two subscales measuring parental approval and warmth, and conflict with parents. Items were classified onto the subscale for which they had the highest loading. Items reflecting approval and warmth in the parent-adolescent relationship include: my parents are ashamed of me (reverse coded), most of the time my parents are satisfied with me, my parents will be disappointed in me in the future (reverse coded), I can count on my parents most of the time, I feel that I have a part in making family decisions, and, usually I feel that I am a bother at home (reverse coded). Items reflecting conflict and negative feelings about the home include: I try to stay away from home most of the time; my parents are almost always on the side of someone else, such as my brother or sister; my parents are difficult to understand; I like one of my parents much better than the other; (all reverse coded); and most of the time my parents get along well with each other. Because high scores reflect positive feelings using the standard scoring of the SIQYA, high scores on the conflict subscale reflect the absence
of conflict, or reflect more positive feelings about the home environment. The reliabilities for these subscales were reasonable as measured by inter-item correlations and coefficient alpha given the reduced number of items for each subscale ($\alpha = .76$ for parental approval and $\alpha = .65$ for conflict with parents). The correlation between the subscales was $r = .52$.

**Depressive Affect**

As pubertal timing has been associated with depression and depressive affect (e.g., Petersen et al., 1991) and as the evolutionary model predicts that emotional distress such as internalizing and externalizing more generally are a path for environmental influences on maturational timing (Belsky et al., 1991; Moffitt et al., 1992), depressive affect, internalizing, and externalizing were examined. These constructs were measured using the Youth Self-Report (YSR; Achenbach & Edelbrock, 1986). The YSR is a measure of self-reported behavioral problems with demonstrated validity in clinical and non-clinical samples. Girls rated how much each item was true of themselves on a 0–2 scale where 0 is “not at all true of me” and 2 is “very true or often true of me.” Items for each scale are summed to form scale scores with high scores reflecting greater behavioral problems. The Depressive Withdrawal scale is an 11-item subscale which included items such as “I am unhappy, sad, or depressed,” and “I like to be alone.” The Internalizing and Externalizing scales are more global scales which are derived from summing subscales. Internalizing is the sum of the Depressive Withdrawal, Anxious Obsessional, Somatic Complaints, and Schizoid subscales. Externalizing is the sum of the Immature Hyperactive, Delinquent, Aggressive, and Cruel subscales. As depressive affect is a component of the Internalizing scale, these variables are linearly dependent and, hence, were not used in the same models. Models were analyzed using depressive affect, the more specific adjustment measure, and then reanalyzed substituting Externalizing and Internalizing, the global adjustment measures.

**Checks on Sample Bias**

As the age range in this group of girls spanned a 3½-year period and as several of the constructs have demonstrated age-related change, analyses were conducted with and without controlling for the age of the girls in order to check for bias due to age differences and control for confounding pubertal timing and age (Steinberg, 1987). In addition, as the sample of premenarcheal girls was selected from a larger study of girls, we are likely to have oversampled for late-maturing girls, especially in grades 7 and 8. However, the overall sample has a mean age at menarche that is somewhat older than average but is comparable to other studies of girls from these social and ethnic backgrounds (Brooks-Gunn et al., 1987). Because girls who are in grades 7 and 8 and are premenarcheal represent a slightly later maturing group of girls, initial analysis of this data examined only girls in the fifth and sixth grades. Although most premenarcheal girls were in the lower grades (69%), using data from all possible girls and controlling for age produced virtually the same results. Hence, results are reported for the larger group of girls.

Further consideration of selection bias in age at menarche was analyzed by comparing means for the constructs under investigation by menarcheal status (i.e., pre- or postmenarcheal). No differences were found on any of the measures (e.g., total number of life events, family relations, depressive affect, maternal age at menarche) between pre- and postmenarcheal girls. Family relations, specifically warmth and approval, was the only measure that approached significance ($p < .07$); means were in the hypothesized direction of the study such that postmenarcheal girls reported lower warmth and approval. Pre- and postmenarcheal girls also did not differ on the distribution of living in a father-absent home versus with biological father.

**Results**

As the goal of this research was to predict maturation, only time 1 measures of psychosocial functioning are used. The primary outcome measure, age at menarche, was drawn from the subsequent year or years of testing. No concurrent analyses are reported here. Descriptive information for the main constructs examined are shown in Table 1.1

---

1 As not all mothers were able to participate in the study, the sample size is reduced to 60 girls for analyses which include maternal age at menarche. All analyses which included this construct were also conducted with the variable removed. No differences were found between analyses using the entire sample of 75 girls versus the subsample of 60 girls whose mothers provided this information.
Correlations with Age at Menarche

Table 1 also shows the zero-order and the partial correlations between each measure and age at menarche with and without controlling for adolescent's age. As can be seen, zero-order and partial correlations were very similar except for a few measures. As expected, maternal age at menarche was positively correlated with adolescent's age at menarche, although this correlation is only significantly different from zero after controlling for adolescent's age. The size of the association also suggests that other factors are clearly influencing adolescent's age at menarche. Also as expected, breast development and weight were strongly and negatively associated with age at menarche (e.g., more advanced breast development and greater weights linked to younger ages at menarche), although both correlations are reduced after controlling for adolescent's age. These differences in correlation size would not be significant but do account for different amounts of variance. Body fat showed little association with age at menarche although it was significantly correlated with breast development ($r_{age} = .34, p < .01$). Interestingly, both the family variables and the affective adjustment variables demonstrated an association with subsequent age at menarche such that better family relations, lower depressive affect, and lower internalizing and externalizing were all associated with later ages of menarche. Correlations of age at menarche with stressful life events were low (absolute value ranging from .01 to .17) with a median value of -0.10; as none were significant or of a size considered to be meaningful (Cohen & Cohen, 1983), only the correlation with the total events score was reported in Table 1. It should be noted, though, that these correlations were similar in size to those reported by Surbey (1990).

Hierarchical Models

In order to test additive models, hierarchical regressions were conducted using two general sets of models; the first examined family structure and relational influences whereas the second examined psychological adjustment influences on age at menarche while accounting for other factors. Analyses were conducted with and without controlling for adolescent age; the change in $R^2$ and significance of change of the other constructs were not affected by this factor. However, as age is a significant predictor of age at menarche, results are presented with age in the model and discussed only if excluding age influences results.

Familial influences.—Predictors were ordered in the following steps for the analyses conducted on family influences: (a) the
TABLE 2
PREDICTION OF AGE AT MENARCHE: FAMILIAL FACTORS

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$R^2$</th>
<th>Change in $R^2$</th>
<th>$p$ Value</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>.07</td>
<td>.05</td>
<td>.05</td>
<td>.26</td>
</tr>
<tr>
<td>2. Maternal age at menarche</td>
<td>.12</td>
<td>.05</td>
<td>(.07)</td>
<td>.23</td>
</tr>
<tr>
<td>3. Male presence</td>
<td>.16</td>
<td>.04</td>
<td>.01</td>
<td>.19</td>
</tr>
<tr>
<td>4. Family relations</td>
<td>.31</td>
<td>.15</td>
<td>.01</td>
<td>.25</td>
</tr>
<tr>
<td>5. Life events</td>
<td>.31</td>
<td>.00</td>
<td>.22</td>
<td>-.03</td>
</tr>
</tbody>
</table>

control for age differences among the girls, 
(b) maternal age at menarche, (c) presence
of an adult male in the household, the family
structure component, (d) family warmth and
family conflict entered at the same time to
reflect family affective environment, and (e)
total number of life events. The order of en-
try was determined for conceptual reasons
drawing upon the existing literature. Family
relations were considered after controlling
for family structure to determine if this factor
predicted above the influence of either the
disruption of divorce and/or proximity to a
related male. As life events had only a small
and nonsignificant association with age at
menarche, it was entered in the model as the
final step to ascertain if controlling for other
factors would illuminate a possible associa-
tion.

Results for this model are shown in Ta-
ble 2. As can be seen, family relationship
variables added significantly to the variance
explained by the model and accounted for
most of the variance of the model, $F(6, 53) =$
4.02, $p < .01$, for the final model. This
finding is comparable to the results reported
by other investigators (Moffitt et al., 1992;
Steinberg, 1988) in that earlier maturation is
associated with less positive family rela-
tions.

In order to determine if the effects for
family relations were, in fact, due to parental
responses to physical signs of puberty oc-
curring prior to menarche, the family influ-
ences model was rerun adding breast de-
velopment after the covariate for age. This
was repeated, first, substituting weight, and sec-
ond, substituting body fat as estimated by
the ponderal index of body mass, for breast
development. Weight and body fat were in-
cluded in this set of analyses as they may act
like breast development as biological corre-
lates of menarche and as visible indicators of
development to which others may respond.
While each of these factors may include a
genetic component, they do not substanz-
tially overlap with maternal age at menarche
which itself includes hereditary and envi-
ronmental factors. We felt that controlling
for external, visible indicators of puberty
first was the strongest test of the models.

Results for the model including breast
development are shown in Table 3. As can be
seen, even though breast development
was a predictor of age at menarche as would
be expected, it did not decrease the effect of
family relations as a predictor, $F(7, 52) =$
7.39, $p < .0001$, for the final model. Despite
suggestions in the literature that body fat
would be predictive of age at menarche, this

TABLE 3
PREDICTION OF AGE AT MENARCHE: EXTERNAL CUES AND FAMILIAL FACTORS

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$R^2$</th>
<th>Change in $R^2$</th>
<th>$p$ Value</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>.07</td>
<td>.33</td>
<td>.0001</td>
<td>-.67</td>
</tr>
<tr>
<td>2. Breast development</td>
<td>.40</td>
<td>.02</td>
<td>.00</td>
<td>.14</td>
</tr>
<tr>
<td>3. Maternal age at menarche</td>
<td>.42</td>
<td>.00</td>
<td>.06</td>
<td>.06</td>
</tr>
<tr>
<td>4. Male presence</td>
<td>.50</td>
<td>.08</td>
<td>.22</td>
<td>.22</td>
</tr>
<tr>
<td>5. Life events</td>
<td>.50</td>
<td>.00</td>
<td>.11</td>
<td>-.06</td>
</tr>
</tbody>
</table>
table 4

<table>
<thead>
<tr>
<th>Predictor</th>
<th>$R^2$</th>
<th>Change in $R^2$</th>
<th>$p$ Value</th>
<th>$\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Age</td>
<td>.07</td>
<td>. . .</td>
<td>.05</td>
<td>.26</td>
</tr>
<tr>
<td>2. Maternal age at menarche</td>
<td>.12</td>
<td>.05</td>
<td>(.07)</td>
<td>.23</td>
</tr>
<tr>
<td>3. Depressive affect</td>
<td>.21</td>
<td>.09</td>
<td>.01</td>
<td>−.31</td>
</tr>
<tr>
<td>4. Life events</td>
<td>.22</td>
<td>.01</td>
<td>. . .</td>
<td>−.10</td>
</tr>
</tbody>
</table>

was not a significant predictor in this sample. We found no association between body fat and age at menarche either in zero-order correlations ($r = -.08$) or controlling for age ($r_{age} = -.10$), as shown in Table 1. However, absolute weight rather than body fat was a significant predictor of age at menarche, but, as with breast development, controlling for weight did not decrease the effect of family relations as a predictor, $F(7, 52) = 4.52, p < .001$, for the final model. The results for this hierarchical model are not shown as they are virtually identical in pattern to those for breast development. In fact, absolute weight appears to be acting as an indicator of physical growth in these analyses as breast development and weight were highly correlated ($r_{age} = .63, p < .0001$), although breast development accounts for more of the variance of age at menarche than does weight (change in $R^2 = .33$ for breast development and change in $R^2 = .18$ for weight).

In summary, breast development and family relations were predictive of age at menarche, and family relations predicted age at menarche above the influence of breast development or weight. A trend for maternal age at menarche to predict adolescent's age at menarche was found. Weight for height, presence of an adult male in the household, and stressful events were not predictive of age at menarche. Because accounting for age differences increases the amount of variance accounted for by breast development but does not affect the amount of variance accounted for by the family relations variables after controlling for breast development, this suggests that the association with family relations is stable and robust against possible sample bias.

Affective influences. — The same procedures and models were analyzed examining the influence of either depressive affect or both internalizing and externalizing behaviors on age at menarche. Internalizing and externalizing were grouped as indicators of global adjustment whereas analysis of depressive affect targets a specific affective behavior. Affective predictors replaced the family structure and relationship measures, such that predictors were ordered in the following steps for these analyses: (a) the control for age differences among the girls, (b) maternal age at menarche, (c) either depressive affect or internalizing and externalizing, and (d) total number of life events. Table 4 shows the hierarchical model for depressive affect influences on age at menarche. As can be seen, depressive affect adds significantly to the prediction of age at menarche, with earlier maturation associated with higher reports of depressive affect, $F(4, 55) = 3.96, p < .01$, for the final model. However, the more global adjustment measures, Internalizing and Externalizing, did not significantly predict age at menarche above the control for age differences and maternal age at menarche.

Again, affective influences were examined above the influence of the visible indicators of puberty (e.g., breast development, weight, and body fat). In this case, depressive affect continued to predict age at menarche when body fat was added to the model; however, the addition of breast development or absolute weight to the model reduced the influence of depressive affect to a trend ($p < .07$ for the change in $R^2$ with breast development and, $p < .11$ for the change in $R^2$ with weight in the model).

Interactions between Family and Affective Constructs

In a further attempt to explain the effects for family relations and possible links to depressive affect, interaction terms were tested. Three interactions were considered given the findings from the hierarchical regressions: (a) the interaction between family approval and breast development, (b) the interaction between family approval and depressive affect, and (c) the interaction between family approval and stressful life events. Despite prior null results for life events, the interaction was considered for conceptual reasons. For these analyses, fam-
ily approval was recoded such that higher scores were associated with poorer relations resulting in the expected direction of effects remaining consistent across variables.

Correlations between age at menarche (controlling for age) and each of the interactions with absence of family approval were $r_{age} = -.46, p < .0001$, $r_{age} = -.24, p < .05$, and $r_{age} = -.18, p < .05$, for the interaction with breast development, depressive affect, and stressful life events, respectively.

Regression models were also tested in order to determine whether the interaction added to the prediction of age at menarche above and beyond the main effects. In this case, age was entered first, then the main effects, and finally the interaction. As with previous analyses, models were also analyzed which included breast development, entered after age. The interaction of absence of family approval with depressive affect and the interaction of absence of family approval with stressful life events were both significant beyond the influence of their individual contributions with changes in $R^2$ of .11 and .05, respectively. Examining mean differences for girls above and below the median on each construct further explored the nature of these interactions. Girls with positive family relations and low depressive affect had the latest age of menarche ($M = 13.72$); girls with positive family relations and high depressive affect, and girls with poor family relations and low depressive affect had intermediate ages of menarche ($M = 13.56$ and $M = 13.36$, respectively); and girls with poor family relations and high depressive affect had the earliest ages of menarche ($M = 12.66$). The interaction of family approval and life events followed a similar pattern.

When regression analyses controlled for breast development, only the interaction of depressive affect and family approval was significant with a change in $R^2$ of .04. This suggests that the variance accounted for by the interaction of stressful life events and family relations may be attributable to responsiveness to changes in the external cues of pubertal development whereas the interaction of depressive affect with family approval, while only accounting for a small amount of variance in the model, may be associated with intensification of effects resulting from internal responsiveness to multiple affective stressors.

The interaction of family approval with breast development did not add significantly to the model after accounting for the main effects. It is noteworthy, though, that the crosslag correlations among breast development and family approval at the first year of testing and breast development and family approval assessed 1 year later indicated that the strongest cross-association was from initial reports of family approval to subsequent breast development ($r_{age} = -.27, p < .01$). Correlations between family approval and breast development within time period were $r_{age} = -.11$ for the first year, and $r_{age} = -.14$ 1 year later. The strongest correlations were within construct (i.e., breast development at time 1 to breast development at time 2) as expected. There was no association between prior breast development and subsequent family approval. This evidence is consistent with the findings for age at menarche.

**Discussion**

The goals of this research were to identify predictors of age at menarche, in a sample of premenarcheal girls using biological and psychosocial constructs. The results of this paper suggest that psychosocial factors, particularly family relations, were associated with subsequent age at menarche.

Our results are similar to those reported by Moffitt and her colleagues (Moffitt et al., 1992) and Steinberg (1988). The similarity to the Steinberg (1988) study is striking as he reports that, among the dimensions of family relationships investigated, accelerating effects on pubertal development were found predominantly for cohesion and conflict in the maternal relationship; these are essentially the same domains (i.e., warmth and approval, conflict) in which we find associations with age at menarche. In the New Zealand study, Moffitt and colleagues found that family conflict was associated with an earlier age of menarche. This effect was in addition to the association of age at menarche with weight as was also demonstrated in our analyses. In our own sample, weight for height was not predictive of age at menarche despite indications that body fat might be predictive of the onset of menses. As we have measured weight and height concurrently with our affective measures, this may at least partially explain why body fat is not predictive especially given that weight alone appears to act as a proxy for physical development with heavier girls having more advanced breast development. In addition, the body fat of the girls is within the normal distribution for body fat, although this sample was somewhat restricted in range and may not exhibit enough variability to dem-
356 Child Development

onstrate effects. Certainly, the variation is not sufficient to reflect decrements in reproductive fitness.

How family relations affect age at menarche is at this time unclear. Analyses including breast development did not explain the family relations effect. The model delineated by Belsky et al. (1991) proposed that family stress and problems led to psychological and behavioral problems which subsequently led to earlier pubertal maturation in girls. These girls would be more likely to reproduce at an earlier age and have poorer parenting skills. Moffitt and her colleagues suggest that their findings support an inheritance model rather than a "conditional adaptation model derived from sociobiology" (Moffitt et al., 1992, p. 47).

In our sample, we, unlike Moffitt and her colleagues, found evidence of psychological adjustment problems leading to earlier maturation, although this effect was diminished by accounting for the development of secondary sexual characteristics. Parental approval and depressive affect were related constructs in this investigation and were related to age at menarche. While both parental conflict and approval were associated with earlier maturation and are highly correlated, approval was the stronger effect in our analyses. Parental approval may tap the effects of family relations on self-esteem. Related work by Caspi and Moffitt (1991) found that early maturation coupled with prepubertal psychological adjustment problems led to greater pathology postpubertally than either early maturation or adjustment problems considered individually.

The mechanisms that link family relationships to pubertal development have yet to be identified but are presumably through the hormonal pathways controlling puberty. Pubertal development is controlled primarily through two hormonal axes: the hypothalamic-pituitary-adrenal (HPA) axis, and the hypothalamic-pituitary-gonadal (HPG) axis. The gonads produce androgens, and adrenal glands produce both androgens and cortisol. As cortisol secretion has been associated with stressful conditions, and as poorer family relations may produce physiological stress responses, the HPA axis is hypothetically the path linking family stress and pubertal maturation. However, research on hormonal activity has found that elevated secretion of cortisol from the adrenals suppresses androgen secretion in the HPG system; development should be slowed rather than accelerated. Unfortunately, hormonal regulation and interaction between the HPA and HPG axes are not well understood at this time. Even if gonadal secretion of steroids (i.e., androgens) is suppressed, the adrenal glands may increase both androgen and cortisol secretion with stress. Most of the studies that reported social influences on puberty did not collect hormonal data (Moffitt et al., 1992; Surbey, 1990); as indicated, these studies and our own find evidence suggesting that stressful or negative affective situations are accelerating development.

It is also important to reiterate that the timing of menarche is only one of several processes that comprise pubertal development. Other processes may be receptive to different environmental influences. In addition, even a single outcome, such as menarche, occurs as the result of several complex hormonal interactions. Environmental influences could be exerted along numerous paths with very different effects depending upon the level of development of the system at the time. For example, the absence of the father living with the adolescent, negative family interactions, or negative affect may all stress the individual, resulting in changes in hormonal functioning and subsequent pubertal development via cortisol secretion and the HPA axis. Alternatively, father absence may not be associated with stress or cortisol/androgen secretion at all. Instead, extrapolating from the work of McClintock (1971), living with a related adult male may affect pheromone secretion or reception which will influence the hypothalamus and the HPG axis directly. Whereas, family interactions and affective state influence maturation through alternate paths.

While father absence was not a significant predictor of age at menarche in the present study, this may have been due to the nature of the sample as these families were predominantly intact. Family structure effects require further investigation in terms of both existence and possible mechanisms.

At this time, the literature on this topic has focused on the pubertal maturation of girls; whether boys' development is also responsive to contextual and affective stress or whether menstrual functioning is particularly vulnerable to these influences remains to be tested. If the adrenal or HPA axis is a causal component in the reported effects for girls, boys' development would presumably be affected similarly. Of course, if stress or
affective state influences age at menarche or the menstrual cycle through mechanisms related to cyclic gonadotropin release or some other system unique to female physiology, boys would not be affected.

Social/environmental influences on and interactions with hormones and pubertal development have only recently been considered despite an existing body of research that has demonstrated that several hormonal systems are responsive to environmental influence and stress (Frankenhaeuser, 1982; Lundberg & Frankenhaeuser, 1980) both immediately after the induction of stress and over longer periods of time (e.g., the association between stress and heart attacks). Future consideration should be given to identifying how social factors affect the developing hormonal system—the time when the system may be most vulnerable to permanent alterations.

It should also be emphasized that these processes are interactions between environmental situations and hormonal changes. Early hormonal activity which occurs prior to external signs of development has been associated with increases in depressive affect (Warren & Brooks-Gunn, 1989) and other alterations in negative mood (Buchanan et al., 1992). While the present study moves beyond previous investigations by controlling for responses to physical signs of development (e.g., breast development), families may already be responding to subtle affective changes which they do not identify as being associated with the hormonal changes of puberty.

The results of this and related studies, in combination, suggest that multiple hormonal pathways may be influenced by internal and external environments. More definitively, this growing body of literature demonstrates the complexity of the associations among biological and psychosocial aspects of development and that the physiological system is responsive to a broader range of influences. These complex interactions may at least partially account for the inter- and intraindividual variation in pubertal development previously noted. Additionally, the association of family affective environment with timing of maturation along with the existing literature on maturational timing effects on subsequent psychological adjustment is the beginning for demonstrating the interactive nature of early adolescent development and for teasing apart developmental processes, in general.

References


Steinberg, L. (1988). Reciprocal relation between parent-child distance and pubertal matura-

Steinberg, L. (1989). Pubertal maturation and parent-adolescent distance: An evolutionary per-
spective. In G. R. Adams, R. Montemayor, & T. P. Gullotta (Eds.), Biology of adolescent 
behavior and development (pp. 71–97). New-

Ziegler & F. B. Bercovitch (Eds.), Socioendo-
ocrinology of primate reproduction (pp. 11– 

of a male on the sexual maturation of female 
mice. Endocrinology, 81, 345–356.

in girls. Journal of Clinical Endocrinology 
Metabolism, 51, 1150–1157.

and behavior at adolescence: Evidence for 
hormonal factors. Journal of Clinical Endocri-

nology and Metabolism, 69, 77–83.

Warren, M. P., Brooks-Gunn, J., Fox, R., Lancelot, 
Lack of bone accretion and amenorrhea in young dancers: Evidence for a relative os-
teopenia in weight bearing bones. Journal of 
Clinical Endocrinology and Metabolism, 72, 
847–853.

Warren, M. P., Brooks-Gunn, J., Hamilton, L. H., 
liosis and fractures in young ballet dancers: 


Zacharias, L., & Wurtman, R. J. (1969). Age at menarche: Genetic and environmental influ-
ences. New England Journal of Medicine, 
280, 868–875.