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MENARCHE AND FATHER ABSENCE IN A NATIONAL PROBABILITY SAMPLE

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Summary. The relation between women's timing of menarche and father absence was examined in a national probability sample of Great Britain (NATSAL 2000; *N*>5000). Current body mass index (as a proxy for childhood weight) was examined as a potential mediator of this relationship, along with the potential moderating role that siblings (e.g. number of older brothers) had on this relationship. As in a number of previous studies, an absent father (but not an absent mother) during childhood predicted an earlier age of puberty (i.e. an early menarche). There was no evidence that weight mediated this relationship or that siblings moderated it. Both a lower body mass index and more siblings (e.g. more younger sisters and brothers) were independent predictors of a later timing of puberty. The results confirm that certain psychosocial factors (i.e. father absence; presence of siblings) may affect the timing of sexual maturation in adolescent girls.

Introduction

Puberty is a complex physical process of sexual/reproductive development accompanied by major social and cognitive changes (e.g. Feldman & Elliot, 1990; Underwood & Van Wyk, 1992). There are notable individual differences in pubertal timing, and this variation is interesting from a general developmental perspective. This variation is also important from a clinical/health perspective because early pubertal timing is associated with negative health and psychosocial outcomes. Early maturing girls are at risk for teenage pregnancy (e.g. Udry & Cliquet, 1982), sexual promiscuity (e.g. Caspi & Moffitt, 1991; Flannery *et al.*, 1993), breast cancer (e.g. Vihko & Apter, 1986; Kampert *et al.*, 1988), weight problems (e.g. Ness, 1991; Wellens *et al.*, 1992), low birth weight infants (Scholl *et al.*, 1989) and problem drinking (e.g. Mezzich *et al.*, 1997). There is less research on pubertal timing and health issues in boys, but early maturing boys have been found to be at risk for sexual promiscuity (e.g. Flannery *et al.*, 1993) and delinquency (Flannery *et al.*, 1993; Cota-Robles *et al.*, 2002). Recent evidence also suggests that early maturing boys

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are at risk for testicular (Weir *et al.*, 1998) and prostate cancer (Giles *et al.*, 2003).

There are a number of factors that may influence pubertal timing: weight, physical activity and genetics (see Underwood & Van Wyk, 1992). There is also evidence that psychosocial factors such as familial/parental instability, particularly an absent father, are associated with an earlier menarche in girls (Hetherington, 1972; Jones *et al.*, 1972; Surbey, 1990; Mekos *et al.*, 1992; Moffitt *et al.*, 1992; Wierson *et al.*, 1993; Kim & Smith, 1998, 1999; Ellis *et al.*, 1999; Hulanicka, 1999; Ellis & Garber, 2000; Hoier, 2003; Quinlan, 2003; Romans *et al.*, 2003; Bogaert, 2005). For example, several studies have shown that girls reared in father-present homes have an onset of first menstruation several months later than girls reared in father-absent homes (e.g. Jones *et al.*, 1972; Surbey, 1990; Moffitt *et al.*, 1992; Wierson *et al.*, 1993; Bogaert, 2005). In addition, several studies indicate that the longer the period of father absence, the earlier daughters reach menarche (Surbey, 1990; Moffitt *et al.*, 1992).

The relation between familial/parental instability and puberty is often interpreted as supporting Belsky *et al.*'s (1991a) evolutionary model of psychosocial influences on pubertal timing. Building on an earlier evolutionary theory by Draper & Harpending (1982, 1988), Belsky *et al.* (1991a) argued that the home/parental environment could profoundly affect the cues and decision rules about which reproductive strategy is likely to be the most optimal in adult environments. If, for example, children do not have a stable home life (e.g. an absent father), then this may lead them to an unrestricted or short-term reproductive strategy (early reproductive viability, including early menarche) because it suggests their adult environments are likely to be unstable and not conducive to a stable long-term mating life history. In contrast to Belsky *et al.*'s (1991a) psychosocial model, some researchers have argued that the relationship between parental discord (e.g. father's absence) and early menarche is genetic in nature (e.g. Comings *et al.*, 2002). Comings *et al.* (2002) found evidence that a variant of the X-linked androgen receptor (AR) gene predisposes fathers to various impulsive behaviours (including family abandonment) and their daughters to early menarche. In addition, there is evidence that adolescent girls may have an accelerated reproductive maturity in response to pheromones from a non-related adult male when, for example, a stepfather lives in the household (see Ellis & Garber, 2000). However, Bogaert (2005) recently found no evidence that the presence of a stepfather (independent of an absent natural father) is associated with an early menarche in girls (cf. Mendle *et al.*, 2006).

Although most of the empirical studies are supportive of a relationship between father absence and early puberty, there are notable exceptions (e.g. Graber *et al.*, 1995; cf. Campbell & Udry, 1995; Mendle *et al.*, 2006). Thus, additional research on puberty and parental instability (i.e. father absence) could help establish whether this relationship is a reliable one. Moreover, most studies have used relatively small convenience or other specialized samples, which are not representative of the general population. As such, there are limits to the generalizablity of these findings. Additional research is also needed to investigate whether certain factors mediate or underlie this relationship. For example, a potential mediator is excessive weight gain. There is a small to medium sized relationship (e.g. $r = -0{\cdot}20$ to $-0{\cdot}30$) between weight/body mass index and age at menarche (e.g. Lin-Su & Vogiatzi, 2002; Wang

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et al., 2006; Lee *et al.*, 2007; Sloboda *et al.*, 2007). Excessive weight gain, then, may precipitate an early puberty (e.g. Underwood & Van Wyck, 1992; Wellens *et al.* 1992; but see Wang *et al.*, 2006). If so, it is possible that father-absent children are heavier than children from father-present homes, either because a father-absent home disrupts the diet of the children or because the stress of an absent father can disrupt the metabolism of children and lead to weight gain (see Belsky *et al.*, 1991a, for a discussion of the latter explanation). There is evidence that absent-father households may have fewer monitored meals (e.g. Videon & Manning, 2003). Moffitt *et al.* (1992; cf. Campbell & Udry, 1995) examined whether the father absence/puberty relationship could be accounted for by children's weight. Moffit *et al.* (1992) found no support for such a mechanism, but an additional test of weight issues in children and adolescence is needed, particularly in a large probability sample, to establish the reliability of this finding.

An additional issue worth examining is whether early puberty in girls (and father absence) is related to other family factors. One important family factor is the presence or absence of siblings. Hoier (2003) found that the presence of younger siblings was associated with a later onset of menarche in girls. Hoier (2003) explained these results from an evolutionary/anthropological perspective (see also Surbey, 1990; Crognier *et al.*, 2002), arguing that in ancestral times a delayed sexual maturity in elder girls facilitated support of their mothers, who may have needed help in rearing their younger children. However, the relationship between menarche and the presence of (younger) siblings needs to be replicated in a large probability sample to establish its reliability. In addition, how siblings relate to the father absence/puberty relationship should be examined. In particular, the presence or absence of siblings may interact with an absent father to predict timing of menarche. For example, the presence of an older brother may act as a parental (father) surrogate, and mitigate the effect of an absent father on early pubertal timing in girls. Thus, the moderating role of siblings (e.g. older brothers) on the father-absent relationship in girls was examined in the present study.

In the present study, the relationship between father absence and timing of menarche, along with weight (i.e. body mass) and sibling characteristics, was examined using secondary data analysis of NATSAL 2000, a large national probability sample from Great Britain (National Centre for Social Research *et al.*, 2005). This study is among the best general sexuality/reproductive surveys of recent years, and it contains relevant information on father absence during childhood and pubertal timing, along with sibling and weight information, in women.

Methods

Sample

The National Survey of Sexual Attitudes and Lifestyles 2000 (NATSAL 2000) used a probability sample of households in Great Britain (England, Wales and Scotland; National Centre for Social Research *et al.*, 2005). The age of the participants ranged from 16 to 44. Along with a face-to-face interview, a computerized self-completion questionnaire was administered to most participants. The main

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criterion to determine who would complete this questionnaire was sexual experience (i.e. they had to report some level of sexual experience). The demographics (e.g. father absence) were not affected by this criterion, but menarche was partially affected (see below).

NATSAL 2000 contained two samples (a 'core' and an 'ethnic-boost' sample). Both of these samples were used in the present study (N=12,109). When both samples are used, a specific weighting of the data is recommended (National Centre for Social Research *et al.*, 2005), which adjusts for inequities in sampling (e.g. age, ethnicity and regional disparities). That weight was used in the present study. Eighty-two participants were also eliminated because they exhibited 'severe' language, literacy or other problems during the interview/questionnaire process, as assessed by the interviewers. Of the remaining 12,027 participants, 5913 were women. Men were not asked their age of puberty in NATSAL 2000.

Menarche and body mass

Women were asked their age at menarche, and this was recorded in full years. As mentioned, some women did not answer the menarche question because they did not have sexual experience with a partner, although it only affected a small percentage of the participants (3·9%). Participants were asked for their height and weight. The NATSAL investigators used these physical measures to calculate the participants' body mass index (BMI). This measure was used as the indicator of excess weight or body fat in the present study. Although the body mass index of the participants in this study is a current measure, the stability of weight and body mass index has been noted across time (Stallone & Stunkard, 1991) and thus it should be a reasonable proxy of weight and excess fat at adolescence. Also, as a test of its utility to indicate body weight at adolescence, one should find high scores on the body mass index to predict earlier puberty in this sample. This prediction is based on the findings that heavier weight in adolescence is associated with an earlier puberty (e.g. Underwood & Van Wyck, 1992). If so, this current measure is probably a reasonably valid indicator of body mass index at adolescence.

Father absence

Participants were asked: 'Did you live more or less continuously with both your natural parents at home until you were 16?' If not, the participants were asked to indicate their living arrangement situation while growing up. The three options available for 'father-absent' situations were: (1) with mother, (2) with other relative, or (3) other (in care, fostered, etc.). Women who responded to one of these three options were combined to form the 'father-absent' group. Women who indicated the remaining option (with father) were combined with those who lived with both natural parents, and these women formed the 'father-present' group.

Sibling variables

The participants were asked several sibling questions: whether they had only sisters, only brothers, or both brothers and sisters (or none); what birth position they

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occupied within their family (first born, last born and in-between); and their total number of siblings. From these variables, specific sibling categories – number of older brothers, number of older sisters, number of younger brothers and number of younger sisters – were constructed using various decision rules (see Bogaert, 2003, for these decision rules from similar surveys). These rules give exact quantities for all sibling categories for those participants with no siblings, only sisters and only brothers. For the remainder of the participants, some of the sibling categories are estimated.

Demographics

The following additional variables were assessed: age (in years); education (1='degree', 2='higher education, but below degree level', 3='O level or equivalent', 4='other/foreign', or 5='none/no exams passed'); social class or SES (1='professional', 2='intermediate', 3='skilled non-manual', 4='skilled manual', 5='part-skilled', 6='unskilled' or 7='other'). Only education was used in the analyses, as SES had a high number of missing cases and education and SES were highly correlated. The items for education were reverse coded so that high scores indicated higher levels. Race–ethnicity (1='White', 2='Black', 3='Asian', or 4='other') was also assessed, and this variable was re-coded so that 0='White' and 1='non-White'. The vast majority of the sample (91·6%) was White.

Results

Most of the women (4602; 77·8%) lived continuously with their natural mother and father until the age of 16; 16·5% (975) lived with their mother but no father; 2·7% (159) lived with their father; 1·1% (63) lived with another relative; 1·2% (70) lived in an 'other' arrangement (including care or fostered); and 0·8% (45) were not classified or listed as 'inapplicable'. This latter group was eliminated from further analysis. As mentioned, women who responded that they lived with their mothers but no father, some other relative, or some 'other' arrangement were combined to form a 'father-absent' group. Women who indicated that they lived with their father were combined with those who lived with both natural parents, and these women formed the 'father-present' group.

The mean age of the women was 30·64 years (SD=8·09), and the mean age at menarche was 12·98 years (SD=1·63). The mean weight of the women was 65·13 kg (SD=13·21), and the mean height was 164·07 cm (SD=7·07). For means and standard deviations for all the variables used in this study, see Table 1.

To assess the relationship between menarche and father absence, a linear regression was conducted, with menarche as the criterion, and father absence (0=absent; 1=present) and the demographics as predictors. As shown in Table 2, women who were older had a later puberty, and those women with a higher education had an earlier puberty. Both of these findings are consistent with some previous research (e.g. Jacobson & Lund, 1990; Herman-Giddens *et al.*, 1997). In addition, an early age of puberty was predicted by father absence, controlling for education, ethnicity/race and age of the participant. A 'mother-absent' group was also constructed (lived with father, some other relative or some 'other' arrangement) and

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Table 1. Means and standard deviations for the measures

Measures	Mean	SD
Age in years	30·65	8·09
Level of education	3·34	1·18
Age at first period	12·98	1·63
Height (cm)	164·07	7·07
Weight (kg)	65·13	13·21
Body mass index	24·23	4·76
No. older brothers	0·59	0·79
No. older sisters	0·56	0·79
No. younger brothers	0·57	0·74
No. younger sisters	0·58	0·76

Note: Sample sizes differ for some variables because of missing cases.
Education is coded so that 1=low and 5=high.

Table 2. Linear regressions predicting pubertal timing (menarche) in women, with father absence and demographics entered simultaneously

Predictor	β	t	p
Age	0·05	3·58	<0·001
Education	− 0·04	− 2·93	=0·003
Ethnicity/race	0·02	1·27	=0·205
Father absence	0·03	3·01	=0·020

Note: Education coded 1=low and 5=high. Ethnicity is coded 0=White and 1=non-White. Father absence is coded 'absence'=0 and 'presence'=1·

compared with the 'mother-present' group (lived with father, or lived with both natural parents). A linear regression controlling for education, ethnicity/race and age of the participant revealed no evidence that a mother's absence was associated with an earlier puberty in women (p>0·30).

To assess body mass index as a mediator of the relationship between father absence and menarche in women, Baron & Kenny's (1986) rules for testing mediation were used. The first step was the first analysis above, showing that father absence predicted age of menarche. The second step attempted to demonstrate that a relationship exists between father absence and the proposed mediator (body mass index). However, a linear regression with father absence (controlling for demographics) predicting body mass index indicated no significant relationship (p=0·25). This indicates that body mass index is not a mediator of the relationship between father absence and early puberty in girls. However, the final steps testing mediation were also conducted, assessing whether the relation between father absence and age of menarche still exists controlling for body mass index (and other demographics). As

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Table 3. Linear regressions predicting pubertal timing (menarche) in women, with father absence, body mass index and demographics entered simultaneously

Predictor	*β*	*t*	*p*
Age	0·08	5·81	<0·001
Education	− 0·06	− 4·58	<0·001
Ethnicity/race	0·02	1·20	=0·230
Body mass index	− 0·16	− 11·53	<0·001
Father absence	0·03	2·04	=0·041

Note: Education is coded 1=low and 5=high. Ethnicity is coded 0=White and 1=non-White. Father absence is coded 'absence'=0 and 'presence'=1·

expected, father absence still predicted an early menarche controlling for body mass index, which independently predicted an early puberty in women (see Table 3). Thus, no evidence existed that body mass index mediates the relationship between father absence and early puberty in women. This analysis does suggest, however, that current body mass index is probably a reasonable proxy of adolescent body mass index because a heavier weight was, as expected, related to an earlier onset of menarche.

To assess the role of siblings in their relation to puberty onset, a new regression analysis was conducted, with number of older brothers, number of older sisters, number of younger brothers and number of younger sisters entered simultaneously, along with allowing interaction terms of each sibling category by father absence (e.g. Older brothers × Father absence, Older sisters × Father absence, etc.) to enter in a stepwise fashion ($p<0·05$). As shown in Table 4, no significant interactions occurred, but number of older sisters, number of younger sisters and number of younger brothers significantly predicted a later puberty in women. Thus, no evidence existed that siblings (e.g. older brothers) moderated the relationship between father absence and menarche. However, there was evidence that number of younger siblings (i.e. younger sisters and brothers), along with number of older sisters, independently predicted a later menarche in women.

Discussion

The present study demonstrated that an absent father in childhood predicts an early menarche in a British national probability sample of women. This finding supports previous studies on this issue (e.g. Hetherington, 1972; Jones *et al.*, 1972; Surbey, 1990; Mekos *et al.*, 1992; Moffitt *et al.*, 1992; Wierson *et al.*, 1993; Graber *et al.*, 1995; Kim & Smith, 1998, 1999; Ellis *et al.*, 1999; Hulanicka, 1999; Ellis & Garber, 2000; Quinlan, 2003; Bogaert, 2005). The present study also advances this research programme in a number of ways. First, most prior work in this area has not used large national probability samples, and, as such, the present study adds confidence that the relationship between father absence and early puberty in women is

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Table 4. Linear regressions predicting pubertal timing (menarche) in women, with father absence, body mass index, sibling characteristics and demographics entered simultaneously, and significant interactions entered stepwise

Predictor	*β*	*t*	*p*
Age	0·07	4·96	<0·001
Education	− 0·05	− 3·74	<0·000
Ethnicity/race	− 0·00	− 0·10	=0·912
Body mass	− 0·16	− 11·57	<0·001
No. older brothers	− 0·02	− 1·07	=0·283
No. older sisters	0·05	3·44	=0·001
No. younger brothers	0·06	3·67	<0·001
No. younger sisters	0·03	2·04	=0·042
Father absence	0·03	2·51	=0·012

Note: Education is coded 1=low and 5=high. Ethnicity is coded 0=White and 1=non-White. Father absence is coded 'absence'=0 and 'presence'=1. No interaction terms between father absence and sibling characteristics (e.g. older brothers) were significant and thus were not entered at the second step.

generalizable. Second, this study tested for a mediator (body mass) and a moderator (presence of siblings) affecting the father absence/puberty relationship. It did not find evidence for either. Given that the study used a large national sample, confidence is gained that these variables are not likely to be related to the father absence/puberty relationship. Body mass index and the presence of siblings (e.g. number of younger siblings) were both found to have an independent relationship to the timing of menarche, with a higher body mass index and fewer younger siblings (along with fewer older sisters) predicting early menarche in girls (see also Moffit *et al.*, 1992; Campbell & Udry, 1995; Hoier, 2003, respectively).

Belsky *et al.*'s (1991a) original theory (cf. Belsky *et al.*, 1991b) posited that some girls may adopt an unrestricted or short-term reproductive strategy (early reproductive viability, including menarche) because certain environmental cues – i.e. an absent father – in childhood suggests their adult environments are likely to be unstable and not conducive to a stable long-term mating life history. Belsky *et al.* (1991a) also argued that the mechanism underlying this change in sexual maturation is stress, both physical and psychological. Such stress can lead to, among other things, changes in the metabolism of the body, leading to weight gain, and ultimately shifting it toward an early developmental pattern. However, Moffit *et al.* (1992) and the present study found no evidence for such a weight-gain mechanism underlying the father absence/puberty relationship (cf. Campbell & Udry, 1995). If stress (both physical and psychological) is the underlying mechanism, it is also unclear why an absent mother does not have a similar impact on the timing of her children's puberty, given a mother's presence or absence should have had a profound effect on the life course of a child throughout human history. Perhaps humans never developed specialized psychological mechanisms to deal with an absent mother because in ancestral times

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the likelihood of survival of a child was probably extremely low if a mother was absent.

Other potential mechanisms of the father absence/puberty relationship should be considered. Other mechanisms may include genetic factors (e.g. Surbey, 1990; Comings *et al.*, 2002; Mendle *et al.*, 2006). For example, Comings *et al.* (2002) found evidence that the androgen receptor gene (or AR gene) may predispose a father to impulsive and externalizing behaviours (e.g. family abandonment) and his offspring to early puberty. In addition, genes may predispose a mother to an earlier menarche, which may relate to an early first intercourse and single motherhood, and her offspring to an early puberty (Surbey, 1990; Campbell & Udry, 1995; Mendle *et al.*, 2006). For example, Campbell & Udry (1995) found that childhood stressors and home-life variables (e.g. father absence) accounted for less variation in a girl's age of menarche than her mother's age of menarche. Similarly, Mendle *et al.* (2006) found evidence that a mother's age of menarche (and the genes underlying it) may account for an early age of menarche associated with her female children who were reared with a stepfather. The present study did not directly address these types of genetic mechanisms. Interestingly, though, recent evidence suggests that the relationship between body mass index and menarche onset may result from shared genetic factors rather than environmental factors (Wang *et al.*, 2006). Thus common genes may underlie an early menarche and a higher body mass in girls. However, given that the present study and Moffit *et al.* (1992; cf. Campbell & Udry, 1995) did not find evidence that body mass acts as mediator between father absence and early menarche, these genes are not likely to underlie the relationship.

The present study also did not evaluate whether the presence of stepfathers (via, for example, pheromones or other mechanisms) accelerates pubertal timing in girls. For example, some research has suggested that the presence of a stepfather is a better predictor of early menarche in girls than the absence of a biological father (Ellis & Garber, 2000; Mendle *et al.*, 2006). However, Bogaert (2005) recently found no evidence of a stepfather effect independent of the effect of a biological father's absence from the home.

The present study found evidence that siblings may affect pubertal timing in women. Specifically, a higher number of younger siblings, along with older sisters, were associated with a later timing of menarche. This finding partly replicated the results by Hoier (2003), who found number of younger siblings predicts a later menarche. What mechanism operating in families with larger sib compositions (e.g. more younger siblings) might trigger a delayed menarche in girls? One possibility is that an increased family size (e.g. more younger siblings) limits the parent's economic and social resources devoted to each child, which tends to delay maturation generally (e.g. Tanner, 1987). Another possibility is that younger siblings alter neuroendocrine mechanisms in elder girls and hence delay their development. Interestingly, there is evidence that men's testosterone levels are lowered once they have children (e.g. Storey *et al.*, 2000), so such altering of neuroendocrine processes in the presence of young children may not be without precedent. Perhaps a mother's pheromones also play a role. There is evidence that women's menstrual cycles synchronize when they live together, and that this effect is pheromonal in nature (Stern & McClintock, 1998; but see Ziomkiewicz, 2006). Perhaps a mother's cycles (or lack of them) can speed up

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or delay a daughter's cycles. If a mother has a number of children, she will have an absence of regular cycles while pregnant; thus, a mother's pheromones (or perhaps the lack of them when pregnant) may alter the neurochemistry of her children's brain affecting their growth and development. A finding in the present study that complicates this pattern (and interpretation) of results is that a greater number of older sisters also predicted a later menarche. There may be a separate mechanism operating here as well, but what that is, is unknown. However, given that a pubertal effect for older sisters may not be a reliable finding (see Hoier, 2003), and that number of older sisters is highly correlated with number of younger siblings (and thus may result as a spurious correlation), this result should be viewed with some caution.

Limitations and future research

Although this study had a number of strengths (e.g. national probability sample; examining a mediator and a moderator), it also has a number of limitations. First, the measure of parental instability did not pinpoint an exact time or the duration in childhood when father absence occurred ('Did you live more or less continuously with both your natural parents at home until you were 16?'). Thus, this absence could have occurred at any point before age 16, and it may not have been for an extended period of time. On the other hand, 'more or less continuously' implies some extended duration, and it is likely that the vast majority of women responded to this question with this in mind. In addition, it is important to note that most separations and divorces are long-lived or permanent, so an extended period of absence in childhood is likely to have occurred for those women indicating that they did not live continuously with the natural father. In addition, if a woman indicated that she did have an absent father prior to 16, most of those year(s) where a father could be absent would have occurred prior to adolescence or during early adolescence (e.g. 0–12 years). For example, marriages that end in divorce last 7 to 8 years on average (US Census Bureau, 1996), and most of the children whose parents divorce are under the age of 6 (Wallerstein *et al.*, 2000). Thus, the measure is probably a reasonable indicator of a father's absence prior to and during early adolescence. It is also important to note that the significant relationship between father absence and puberty occurred *despite* the possible error involved with the measure of father absence. This suggests that this relationship may be even stronger than these data suggest; this is because, of course, random or unsystematic error in measures is *less* likely (and not more likely) to lead to reliable/significant differences.

Second, this sample did not measure pubertal onset in boys. Recent work by Bogaert (2005) has suggested that father absence also predicts an early puberty in boys. Thus, perhaps the same mechanism is occurring in girls and boys to accelerate adolescent sexual maturation. This may limit the number of plausible mechanisms underlying the father absence/puberty relationship. It would be interesting to see if siblings (e.g. presence of younger sisters and brothers) also predict the timing of sexual maturation in boys.

Third, this study was correlational and did not include a longitudinal design; thus, the temporal/causal ordering of variables is unclear. As Ellis & Graber (2000) point out, however, most longitudinal research also has limitations, including not being able

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to determine whether the relationship between parental instability and pubertal timing is genetic in nature (cf. Mendle *et al.*, 2006). Moreover, existing longitudinal work has not concentrated on boys, and the samples of girls have not been representative ones. Thus, this programme of research would advance if new research addressed these limitations, along with including measures that assess genetic influences (e.g. assessment of pubertal timing in parents and/or genetic markers).

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