A Life History Theory of Father Absence and Menarche: A Meta-Analysis

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Abstract: Is the absence of biological fathers related to their daughters' earlier age at menarche? Drawing on evolutionary psychology and life history theory, prior research has suggested such a relationship (Belsky, Steinberg, and Draper, 1991; Draper and Harpending, 1982; Ellis, 2004). Although qualitative reviews have shown narrative support for this relationship (Allison and Hyde, 2013; Ellis, 2004; Kim, Smith, and Palermiti, 1997; Susman and Dorn, 2009), no quantitative review exists to provide empirical support for this relationship or to explain mixed results. Thus, we conducted a random-effects meta-analysis of correlations (Card, 2012) on father absence and daughter menarcheal age ($k = 33$; $N = 70,403$). The weighted mean correlation was .14, 95% CI [.09, .19], suggesting that father absence was significantly related to earlier menarche; effect sizes were heterogeneous. Egger's regression (Egger, Smith, Schneider, and Minder, 1997) showed no evidence of publication bias (file-drawer effect; $r = .34$, $p = .052$). Outcome measure differences (menarcheal age vs. menarcheal age embedded in a multi-item pubertal timing scale) did not moderate effect sizes. Study year effects (Schooler, 2011) were also non-significant. Our findings support one aspect of the life history model and provide groundwork for subsequent examination of other pathways in the model.

Introduction

Can growing up in a family without a biological father affect a girl's pubertal timing? More specifically, is father absence related to daughters' earlier age at menarche?

Life history theory (Chisholm, 1993; Ellis, Del Giudice, et al., 2012; Ellis, Figueredo, Brumbach, and Schlomer, 2009; Kaplan and Gangestad, 2005; Surbey, 1998; Vigil, Geary, and Byrd-Craven, 2005) predicts that organisms—including humans—trade-off energy devoted to somatic versus reproductive effort. During puberty, humans shift metabolic energy from the rapid growth of childhood to developing secondary sex characteristics and reaching reproductive capacity (e.g., menarche, spermarche). Menarche can provide a clear demarcation in the more lengthy process of pubertal maturation in girls. Notably, the timing and rate of progression of pubertal development varies widely among girls and boys (Belsky, 2012; Tanner, 1962). In the present meta-analysis, we reviewed empirical studies that accounted for this variance in menarcheal age as a function of father absence/presence. First, however, we present an overview of relevant theoretical perspectives that have framed much of this research.

Theoretical overview: Developmental pathways of divergent reproductive strategies

An evolutionary perspective may be key in helping researchers unlock the contextual contingencies of human development; evolutionary–developmental theories provide a broad framework for understanding the dynamic interplay of genes and environment as they unfold over the lifespan (Ellis and Bjorklund, 2012). Draper and Harpending (1982) suggested that daughters raised in father-absent (vs. -present) homes might view paternal investment as non-essential for reproduction, and thus might have sex earlier, be less discriminating in mate choice, and have unstable pair bonds. Consistent with life history theory, girls' reproductive strategies should be flexible and thus calibrated to be contingent on contextual cues. That is, the optimal reproductive strategy might be to attend carefully to environmental conditions early in life (e.g., investing father vs. absent father) and adapt accordingly—a sort of environmentally informed developmental plasticity based on life history. Although pioneering, Draper and Harpending's (1982) model lacked not only clear predictions, but also a developmental process or mechanism to explain how such contextual effects occur (Belsky, 2012).

Building off of Draper and Harpending's (1982) views, Belsky, Steinberg, and Draper (1991) attempted to integrate biological development—especially pubertal timing—as one such explanatory process. The Belsky et al. (1991) approach—the psychosocial acceleration theory—has been among the most influential theories to integrate both evolutionary and developmental perspectives (Belsky, 2012; Ellis, 2004). Specifically, psychosocial acceleration theory offers a novel and integrative explanation of the developmental timing of puberty and subsequent sexual strategies (Belsky et al., 1991). This theory suggests that a stressful childhood environment, an insecure attachment to parents, or both, can lead to earlier pubertal maturation and, subsequently, to increased adult sexual promiscuity, unstable pair bonds, and decreased investment in offspring (Belsky, 2012). In contrast, children raised in stable environments typically have more secure attachments with their parents, reach puberty relatively later, are less sexually promiscuous as adults, and are often willing to invest more resources in their offspring. For example, highlighting the role of attachment (Bowlby, 1982), insecure infant–mother attachment at 15 months is related to earlier menarche (Belsky et al., 2010).

In terms of process for this and related models, differences in environmental

contexts tend to channel developing children into divergent developmental trajectories regarding pubertal timing. Differential pubertal timing may then lead to reproductive strategies adaptively calibrated to initial childhood conditions (e.g., environmental stressors). Consistent with life history theory, different developmental trajectories during adolescence may steer people toward a quantity or quality sexual strategy by the time they reach adulthood. Evolved sexual strategies of *quantity* involve maximizing reproductive fitness by having as many offspring as possible with relatively low parental investment, whereas *quality* strategies involve maximizing the survival of a few offspring by nurturing them with the resources necessary for survival (Buss and Schmitt, 1993; Hirsch and Paul, 1996). As recent research attests (Vigil et al., 2005), psychosocial acceleration theory is largely consistent with older, broader perspectives on reproductive strategies shaped by life history theory (e.g., Cole, 1954; Low, 1978).

Psychosocial acceleration theory can be expressed through five major life history stages. In relative chronological order, these stages or domains include family context, childrearing, psychological and behavioral developmental, somatic development, and reproductive strategies. Family context, childrearing practices, and subsequent child development are clearly inter-related, and empirical evidence has largely supported these associations over the past 20 years (Belsky, 2012). A harsh family environment can include marital discord, parental abuse, divorce, death of a parent, and insufficient or inconsistent economic recourses. These factors, including parental—and especially *paternal*—absence and inconsistent parenting (Draper and Harpending, 1982) often contribute to increased child stress. Children raised in households where fathers are present typically have access to more resources, greater stability over time in resources, and greater marital harmony. Such environments favor normal or delayed timing in sexual maturation and the development of quality sexual strategies in later adulthood (Belsky et al., 1991). In addition, parents who are economically stressed or in conflict often exhibit insensitive, neglectful, rejecting, and/or inconsistent childrearing practices (see Conger and Donnellan, 2007, for a review of family stress models). On the other hand, stable family contexts with adequate resources are associated with supportive and affectionate childrearing practices (Belsky et al., 1991).

Hence, substantial evidence indicates that children reared in harsh environments by insensitive parents are more likely to develop psychological and behavioral traits adapted to a fast life history strategy (Belsky, 2012; Belsky et al., 1991; Vigil, 2005). Focusing on the attachment literature, evidence suggests that insecurely attached children often mistrust their parents (Belsky et al., 1991; Chisholm, Quinlivan, Petersen, and Coall, 2005). Fathers involved in insecure relationships with their children often display aggressive or noncompliant behaviors, whereas mothers of insecurely attached children are often anxious or depressed. In contrast, children reared in nurturing, supportive environments are more likely to develop secure attachments to their parents, and will often develop trusting personalities (Belsky et al., 1991).

According to psychosocial acceleration theory, the previous three factors (family context, childrearing, and psychological development) appear to mediate biological effects (e.g., hormonal changes) of the timing of pubertal maturation (Belsky, 2012; Belsky et al., 1991). Specifically, the theory suggests that children reared in harsh environments,

especially those with insecure attachments to their parents, are more likely to mature earlier and more quickly than their peers reared in less-stressful environments with secure attachments to their parents. This relationship appears to be especially critical between biological fathers' presence (vs. absence) and their daughters' age at menarche.

The resulting post-pubertal outcome of childhood experience and puberty are variations in reproductive strategy. Specifically, adolescents who sexually mature earlier (vs. later) would be more likely to engage in sexual activity earlier, have more sexual partners throughout their lifespan, have a greater number of short-term relationships (e.g., one-night stands), produce more children, and invest relatively less in their children (Belsky et al., 1991). "Late-blooming" adolescents would be more likely to develop sexual strategies and child investment patterns contrary to those of early-maturing adolescents (i.e., later sexual activity, fewer sex partners, lasting pair bonds, fewer children, and greater investment in offspring; Belsky et al., 1991).

Research evidence related to key components of psychosocial acceleration theory

When psychosocial acceleration theory was proposed, animal studies indicated that proximity to related individuals impacted reproductive functioning. For example, the sexual development of males can be disinhibited by the removal of more dominant males in a group (Belsky et al., 1991). Similarly, juveniles separated from their parents as they enter puberty reach reproductive maturity more rapidly. Along with evidence from animal studies, an emergent literature in humans reported a small but significant association between earlier ages at menarche and father absence during childhood (Surbey, 1990).

In humans, puberty is a relatively long process, including initial hormonal changes and subsequent development of secondary sexual characteristics that typically take five or more years from initial physical signs of development through completed development (see Susman and Dorn, 2009). The onset and progression of puberty vary substantially in normatively developing children and adolescents (Belsky, 2012; Tanner, 1962). Although it is well established that heredity, health, nutrition, and behavior influence the onset of puberty, relatively few studies have considered psychosocial influences.

Much of the subsequent literature has reported that, on average, girls who are raised in father-absent family contexts attain menarche earlier than girls raised in intact families (Belsky et al., 1991; Draper and Harpending, 1982, 1988; Ellis, McFadyen-Ketchum, Dodge, Pettit, and Bates, 1999). In a recent study, income and ethnicity moderated the relationship between father absence and pubertal timing in girls, but only after controlling for body mass index (BMI); specifically, father absence was related to earlier breast development in affluent families, and to earlier pubic hair development in affluent African families (age at menarche was not measured; Deardorff et al., 2011). These findings are somewhat inconsistent with psychosocial acceleration theory, which posits that the increased stress of being raised in more difficult or inconsistent (vs. affluent) environments may cause children to become "more biologically reactive to social conditions" as the age of pubertal onset approaches (Belsky et al., 1991, p. 658). Further research will be needed to understand the dynamic interplay among BMI, income, ethnicity, father absence, and pubertal timing.

Research on child abuse, economic poverty, marital discord, and occupational

frustration also shows a consistent relationship between contextual stress and dysfunctional parenting (Belsky et al., 1991), with several of these factors now also linked to pubertal timing and sexual behavior (Vigil et al., 2005). Contextual stress affects the psychological wellbeing of parents, which in turn affects the quality of their childrearing practices. As indicated, harsh childrearing practices have a well-established link to more serious behavioral and social adjustment problems in children (Belsky et al., 1991; Draper and Harpending, 1988). In studies of pubertal timing, maternal harshness when daughters were 4.5 years old predicted earlier menarche, and both maternal harshness and earlier menarche predicted daughters' increased sexual risk-taking (Belsky et al., 2010). In addition, family conflict has been associated with earlier age of menarche in girls (Graber, Brooks-Gunn, and Warren, 1995).

During the past decade, affordable genetic/hormonal assaying and access to longitudinal twin/sibling/family data have helped researchers address some of the biological correlates and mediators of psychosocial acceleration theory. Higher quality parental investment predicts later adrenarche—an increase in androgens that is typically one of the first hormonal changes of puberty in children around 6–8 years old (Ellis and Essex, 2007). Quasi-experimental methods using a controlled sibling design in sister pairs indicate that higher quality fathering is related to less-risky sexual behavior (Ellis, Schlomer, Tilley, and Butler, 2012). Not all family designs, however, have supported psychosocial acceleration theory or its broader life history approach. Using a children-of-twins/sisters design, researchers found that, in cousins discordant for father absence or stepfathering (e.g., one cousin has a [step]father, one does not), age of menarche (Mendle et al., 2006) or first sexual intercourse (Mendle et al., 2009) did not differ significantly. Moreover, after adjusting for mother's age at menarche, no reliable relationship remained linking stepfathering to earlier menarche in girls (Mendle et al., 2006).

Sexual behavior. A key aspect of psychosocial acceleration theory is its linking of childhood environmental context to post-pubertal sexual behavior (Belsky, 2012; Belsky et al., 1991; Ellis, 2004). In general, adolescents who mature earlier initiate sexual intercourse at an earlier age than their peers, and have more sexual partners throughout their lifespan (Belsky et al., 1991), although effects are more consistent for early maturing girls and vary by context (Ellis, 2004). Moreover, adolescents from single-parent homes are more likely to be sexually active than their dual-parent peers. Father absence was related to earlier sexual activity and risk for adolescent pregnancy in girls from the U.S. and New Zealand (Ellis et al., 2003). Adults who were raised in divorced families as children typically have more sexual partners than adults raised in families without divorce (Belsky et al., 1991). In addition, children raised without their biological parents were more likely to initiate sex at earlier ages than children raised by both biological parents (Belsky et al., 1991).

To be sure, personality traits such as impulsivity also play a role in predicting sexual behavior. For example, impulsivity was positively related to sociosexuality (Simpson and Gangestad, 1991)—particularly sociosexual attitudes (Webster and Bryan, 2007)—and negatively related to willingness to delay sex (Webster and Crysel, 2012). Moreover, personality factors may be relevant not only to sexual behavior but also relationship formation and maintenance (i.e., father absence). Overall, given patterns of relationship change among parents in our current era (e.g., divorce, remarriage), variation

in effects are likely due to variation in the experience of different family contexts for varying lengths of time during the prepubertal years. Hence, examination of effects across studies using different approaches to defining father absence is warranted.

Notably, the majority of studies on family context and childrearing influences on pubertal timing have either focused solely on women or only found significant effects for women (Belsky, 2012; Draper and Harpending, 1982). This limitation of the literature is likely due in part to the salience of menarche and the relative ease of assessing age at menarche retrospectively (Susman and Dorn, 2009). Age at menarche is a clear indicator of the same level of pubertal development across women. That is, it signals the emergence of the menstrual cycle and reproductive capacity, though full maturation of the reproductive functioning may take another 2 years. In contrast, retrospective reporting of a comparable event for men (i.e., spermarche) is less reliable. At the same time, the literature may also reflect the absence of comparable effects on pubertal timing in men. For example, Belsky and colleagues (2010) reported very small effects for boys in contrast to the findings for girls in the large sample from the NICHD Early Childcare Study. In general, evidence suggests that female reproductive physiology has a more heightened sensitivity to psychosocial and environmental stressors than that of men (Surbey, 1998). Hence, for the present investigation, we have focused on age at menarche in women.

Summary and critique. Psychosocial acceleration theory integrates multiple biological and environmental factors into a novel and useful explanation of how divergent reproductive strategies develop (Belsky, 2012). Environmental stressors in the family context (e.g., economic hardship, marital discord) may lead to harsh parenting practices, which may in turn lead to insecure attachment styles, and ultimately, early pubertal maturation. Early onset of puberty (in addition to the aforementioned factors) may lead to an increased number of sexual partners, unstable pair bonds, and limited parental investment in adulthood (i.e., a quantity-oriented strategy). The opposite circumstance in each of these factors, however, typically leads toward an opposite developmental path and its respective reproductive strategy—one of lasting pair bonds and greater parental investment (i.e., a quality-oriented strategy). From this perspective, psychosocial acceleration theory is clearly consistent with a broader life history theory approach.

Empirical support for some of the links proposed by psychosocial acceleration theory has been largely positive, though not without ambiguity (Belsky, 2012; Ellis, 2004). For example, one study provided support for the direct link between early childhood stress and early menarche, but it found that this relationship was not mediated by a negative or insecure attachment style (Coall and Chisholm, 1999). The present study attempts to resolve (and possibly explain) some of the ambiguity surrounding mixed findings using meta-analytic methods while focusing on one aspect of the theory: father absence and pubertal timing (i.e., menarcheal age).

The present research: Meta-analysis

Over the past 20 years, psychosocial acceleration theory has inspired a great deal of studies; some have provided mixed empirical results (e.g., Kim and Smith, 1998; Mekos, Hetherington, and Clingempeel, 1992), but most have been supportive (Ellis, 2004; Susman and Dorn, 2009). To help resolve this discrepancy quantitatively, we conducted a meta-

analysis. We focused on studies that included measures of father absence/presence and measures of daughters' age at menarche. We chose these variables because (a) they tend to be the most frequently studied variables pertaining to psychosocial acceleration theory, and (b) both represent clear-cut social and biological events versus more subjective self-reports (e.g., perceived closeness to father, daughter's breast or pubic hair development).

Meta-analyses are unique because they allow researchers to examine broad questions at the between-study level, such as the possibilities of publication bias (or file-drawer effect; Rosenthal, 1979) and the decline effect (a decline in effect sizes over time; Jennions and Møller, 2001; Schooler, 2011). Publication bias happens when statistically significant (i.e., $p < .05$) results are published, but non-significant effects are not published (i.e., they are left in the researcher's file drawer). Publication bias can be detected in a number of ways via meta-analysis. For example, a funnel plot (see Figure 2)—which is assumed to produce a roughly symmetric distribution of effect sizes that narrows as sample sizes increase—will appear asymmetric due to a "missing" area of non-significant effect sizes, presumably suppressed by not appearing in the published literature. The decline effect—a decrease in effect sizes over time—can also be tested via meta-analysis. Possible explanations for decline effects include regression to the mean over time, changes in historical context, and academic journal editors' focus on novel findings.

Thus, in the present investigation, we made three predictions:
1. Based on psychosocial acceleration theory and prior studies showing positive relationships between father absence and earlier menarche, we predicted that the average weighted effect size (correlation) would be positive and significantly different from zero, but not necessarily large in magnitude (some studies have shown non-significant effects).
2. Because the above relationship (a) is not experimentally manipulated (cf. DelPriore and Hill, 2013) and (b) was not the central focus of some studies, we did not expect—but nevertheless tested for—publication bias in the effect sizes (i.e., a file-drawer effect; Rosenthal, 1979).
3. Given recent research on the decline effect—a decrease in effect sizes over time (Jennions and Møller, 2001; Schooler, 2011)—we tested for change in effect sizes over time as a function of study year; however, we remained agnostic about whether such a decline effect would exist.

Method

Search, sample, and selection

We searched for relevant studies in databases (PsycINFO, Google Scholar) using the search terms *menarche* and combinations of *father*/*paternal* and *present*/*presence* or *absent*/*absence*. In addition, we culled relevant studies cited in comprehensive narrative reviews of the literature (Ellis, 2004; Kim et al., 1997; Susman and Dorn, 2009). This search yielded over 600 possible candidate studies. After reading titles and abstracts, we identified 63 promising candidate studies to read. Of these, we found 37 studies that collected both (a) daughter's age at menarche (either by itself or embedded in a multi-item pubertal-timing measure) and (b) a measure of father presence/absence during childhood

(either by itself or embedded in a multi-item measure).

A few studies were relevant but not included in the meta-analysis. These included three studies in which the effect of interest was not tested or reported (Joinson, Heron, Lewis, Croudace, and Araya, 2011; Kim et al., 1997; Vigil et al., 2005). These also included two more studies that reported non-significant effects but not effect sizes (Kim and Smith, 1998; Mekos et al., 1992); however, because sample sizes were reported (see Ellis, 2004, for a review of Mekos et al., 1992), a range of possible non-significant effect sizes could be assumed, and these were used in the publication bias analyses covered in the Results section.

Because meta-analysis requires independent effect sizes, and because a few studies in our sample produced two or three non-independent effect sizes (e.g., two correlations based on two different measures of menarcheal age or father presence), we averaged the non-independent effect sizes (via *r*-to-*z* transformations; see Card, 2012). These included two instances where Ellis (2004) provided effect sizes from secondary analyses of two studies (i.e., Ellis et al., 1999; Moffitt, Caspi, Belsky, and Silva, 1992; see Table 1).

The final sample had 33 independent effect sizes ($k = 33$) from a total of 70,403 females (range = 58–21,437 per study; *Mdn* = 416; *M* = 2,133.4; *SD* = 4,183.5; see Table 1). We examined a continuous between-study moderator—study year—which spanned over 40 years (Range = 1972–2012, *Mdn* = 2004, *M* = 2000.2, *SD* = 10.5; see Table 1).

Data analysis

In our analyses, we present random (vs. fixed) effects meta-analyses. Random effects results are more conservative because, unlike fixed effects, they do not make strong assumptions regarding homogeneity of effect sizes, and focus on estimating the true population parameter of all studies (or effect sizes), not just those sampled in the meta-analysis. As a result, random effects have wider confidence intervals (CIs) than fixed effects (Card, 2012; Hunter and Schmidt, 2004; Schmidt, 2010). We performed random- and mixed-effects meta-analyses with a structural equation modeling (SEM) approach using the Mplus 6 statistical package (Muthén and Muthén, 2010). This new SEM procedure takes advantage of maximum likelihood estimation, whereas older approaches use weighted ordinary least squares (see Card, 2012, pp. 245–249; see also Hadden, Smith, and Webster, 2014, for an example). Following Card (2012), in all analyses, we treated effect sizes as random effects and moderators (e.g., study year) as fixed effects; thus, analyses involving moderators represent a mixed-effects meta-analysis. We also report variance statistics (i.e., τ^2). If the variance is significant (via *z* test), then it suggests that the effect sizes are heterogeneous—variability remains beyond what the weighted mean effect size (i.e., the model's intercept) can explain. This variability may be due to unexplained sources or systematic between-study differences (e.g., study year, method variance, published vs. unpublished studies). In contrast, if the test is non-significant, then it suggests that the effect sizes are sufficiently homogeneous.

Results

Father absence and earlier menarche

Supporting our first prediction, biological father absence was positively related to daughters' earlier age at menarche across 33 independent effect sizes ($r = .14$, 95% CI [.09, .19]; see Table 1, Figure 1). The average effect size was in the small-to-moderate range and corresponded to a Cohen's *d* of 0.28 [0.19, 0.38] (Cohen, 1988). These effect sizes were significantly heterogeneous ($\tau^2 = 0.013$, *SE* = 0.005, $z = 2.45$, $p = .014$).

Figure 1. Meta-analysis effect size plot with 95% confidence intervals ($r \pm$ 95% CI), ordered from smallest to largest sample size (see Table 1)
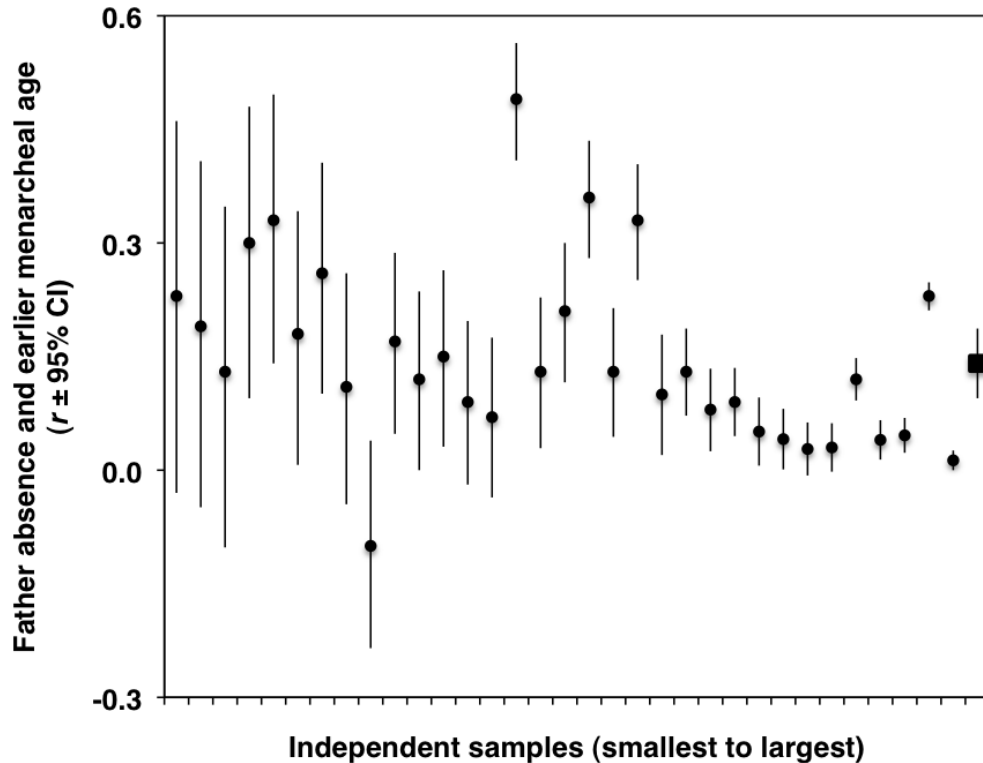
Notes: The square marker (far right) indicates the weighted mean effect size across all 33 studies ($r = .14$, 95% CI [.09, .19]). Effect sizes were heterogeneous ($\tau^2 = 0.013$, *SE* = 0.005, $z = 2.45$, $p = .014$).

Table 1. Meta-analysis of correlations between father absence and menarche/pubertal timing

Study	N	r	95% CI LL	UL
Alvergne, Faurie, and Raymond (2008)[a]	601	.10*	.02	.18
Belsky et al. (2007)	342	.07	-.04	.17
Bogaert (2005)[b]	1,921	.05*	.01	.10
Bogaert (2008)	5,824	.04*	.01	.07
Burkett (2000)[c]	58	.23	-.03	.46
Campbell and Udry (1995)	518	.13*	.04	.21
Chisholm, Quinlivan, Petersen, and Coall (2005)[d]	98	.33*	.14	.50
Doughty and Rodgers (2000)[e]	3,145	.03	-.00	.06
Ellis and Garber (2000)[f]	87	.30*	.10	.48
Ellis, McFadyen-Ketchum, Dodge, Pettit, and Bates (1999)[f,g]	144	.26*	.10	.41
Foster, Hagen, and Brooks-Gunn (2008)	4,812	.12*	.09	.15
Gesselman and Webster (2012)[c]	201	-.10	-.24	.04
Graber, Brooks-Gunn, and Warren (1995)	74	.13	-.10	.35
Hoier (2003)	268	.12*	.00	.24
Hulanicka (1999; data from 1977)[d]	256	.17*	.05	.29
Hulanicka (1999; data from 1981)[d]	21,437	.01	-.00	.03
Hulanicka, Gronkiewicz, and Koniarek (2001; data from 1978)	271	.15*	.03	.26
James, Ellis, Schlomer, and Garber (2012)[f]	129	.18*	.01	.34
Jean et al. (2011)	523	.33*	.25	.40
Jones, Leeton, McLeod, and Wood (1972)	371	.49*	.41	.56
Jorm, Christensen, Rodgers, Jacomb, and Easteal (2004)	3,692	.03	-.00	.06
Kiernan and Hobcraft (2010)[b,d]	2,349	.04*	.00	.08
Mendle et al. (2006)[b]	1,284	.08*	.03	.13
Moffitt, Caspi, Belsky, and Silva (1992)[d]	416	.21*	.12	.30
Mustanski, Viken, Kuprio, Pulkkinen, and Rose (2004)[f]	1,872	.09*	.04	.13
Neberich, Penke, Lehnart, and Asendorpf (2010)	374	.13*	.03	.23
Quinlan (2003)	10,135	.23*	.21	.25
Romans, Martin, Gendall, and Herbison (2003)	488	.36*	.28	.43
Surbey (1990)	1,115	.13*	.07	.19
Tither and Ellis (2008)	161	.11	-.05	.26
Toromanovi and Tahirovi (2004)[d]	7,045	.05*	.02	.07
Wierson, Long, and Forehand (1993)	69	.19	-.05	.41
Zabin, Emerson, and Rowland (2005)	323	.09	-.02	.20
Sum or weighted mean	70,403	.14*	.09	.19

Notes: CI = confidence interval; LL = lower limit; UL = upper limit; *$p < .05$; [a]Average of three effect sizes; [b]Partial correlation; [c]Unpublished study; [d]Secondary analysis of tabular results; [e]Contains some non-independent data due to twin, sibling, or family-level designs; [f]Age at menarche was embedded in the Pubertal Development Scale (Petersen, Crockett, Richards, and Boxer, 1988) and/or Index of Adolescent Development (Morris and Udry, 1980); [g]Average of two effect sizes: one from cited study, one from Ellis' (2004) secondary analysis.

Examining publication bias

We addressed possible publication bias in four ways: (a) comparing published and unpublished studies, (b) adding two published studies that reported non-significant findings and sample sizes but no effect sizes, (c) visual inspection of a funnel plot, and (d) Egger's regression.

First, we compared the weighted average effect sizes of published versus unpublished studies (i.e., Burkett, 2000; Gesselman and Webster, 2012). Assuming a positive relationship between father absence and earlier menarche, if publication bias exists, then effect sizes for published studies should be substantially *greater* than those of unpublished studies; however, no significant difference emerged ($b = 0.13$, $SE = 0.11$, $z = 1.13$, $p = .26$, $d = 0.41$), and effect sizes remained heterogeneous ($\tau^2 = 0.012$, $SE = 0.005$, $z = 2.43$, $p = .015$). Nevertheless, the average effect size for published studies was significantly different than zero ($r = .15$ [.10, .19]), whereas the same was *not* true for unpublished studies ($r = .02$ [-.19, .23]).

Second, we added two published studies that reported null findings and sample sizes but not effect sizes (i.e., Kim and Smith, 1998; Mekos et al., 1992). We then re-ran the meta-analysis three ways to test boundary conditions: assuming the correlations were zero, and assuming the lowest or highest possible non-significant correlations for both studies given their sample sizes ($ks = 35$). Biological father absence remained positively related to daughters' earlier age at menarche regardless of assuming the lowest ($r = .13$, [.08, .17]; $\tau^2 = 0.015$, $SE = 0.006$, $z = 2.50$, $p = .012$), null ($r = .14$, [.09, .18]; $\tau^2 = 0.013$, $SE = 0.005$, $z = 2.44$, $p = .015$), or highest ($r = .14$, [.10, .19]; $\tau^2 = 0.012$, $SE = 0.005$, $z = 2.47$, $p = .014$) correlations.

Third, we visually examined a funnel plot (Card, 2012) of raw effect sizes (*r*s) against sample size (*N*). As expected, this plot produced a roughly normal distribution of effect sizes that were generally symmetric around the weighted mean effect size and became closer to the mean as sample size increased (i.e., a narrowing funnel shape from left to right; see Figure 2).

Fourth, we empirically tested publication bias using Egger's regression (Egger et al., 1997), which had adequate power (> 80%) to test for severe publication bias (Card, 2012). Results showed no significant evidence of publication bias (i.e., a linear association between effect size and sample size; this was true regardless of whether the two non-significant studies were included ($k = 35$: $b = 1.80$, $SE = 0.96$, $t_{33} = 1.88$, $p = .069$, $r = .31$) or excluded ($k = 33$: $b = 2.06$, $SE = 1.02$, $t_{31} = 2.02$, $p = .052$, $r = .34$).

Using four methods, and consistent with our second prediction, results suggested that between-study differences in sample size did not systematically bias the results, and that publication bias (or the file-drawer effect) was not a substantial problem. As a result, we excluded the two studies that gave no effect sizes for subsequent moderation analyses ($ks = 33$).

**Figure 2.** Meta-analysis funnel plot of effect sizes (*r*) against sample size (*N*; log scale)
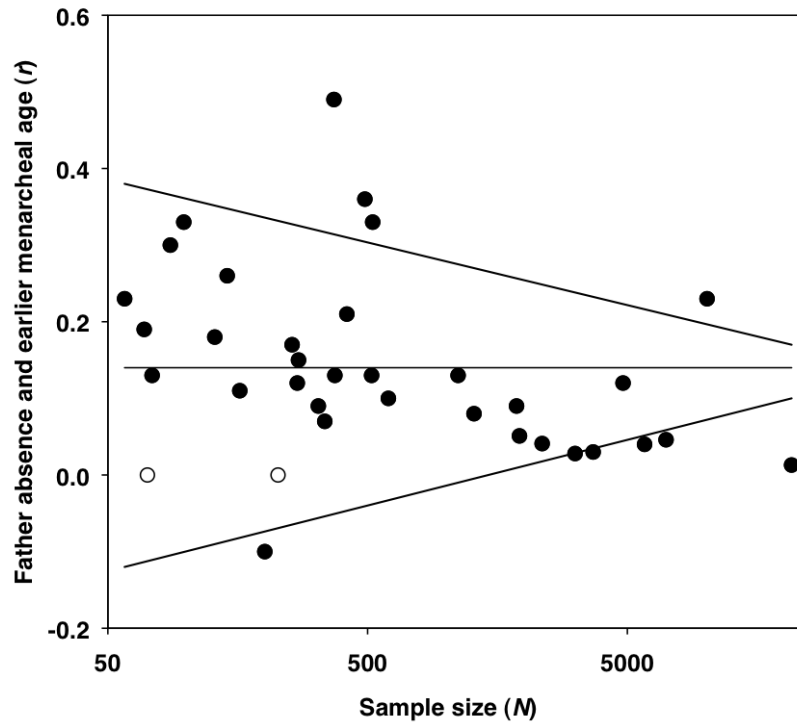
Notes: The horizontal line shows the weighted mean effect size (*r* = .14) along with its 95% CI for various sample sizes (sloping lines). Hollow circles indicate two studies that reported non-significant results and sample size, but no effect size (i.e., Kim and Smith, 1998; Mekos et al., 1992; these were set to *r* = .00). Symmetry around the mean effect size, and decreasing variance in larger samples, suggests a lack of publication bias (Card, 2012).

Testing the decline effect: Moderation by study year

Addressing our third prediction, between-study differences in study year did not significantly moderate the father-absence–menarcheal-age relationship (*b* = -0.004, *SE* = 0.003, *z* = -1.30, *p* = .19, *r* = -.23; see Figure 3).[1] Simple effects tests (Aiken and West, 1991) showed that this relationship remained significant—but was decreasingly positive—across study years, including 1990 (*r* = .18 [.10, .26]), 2000 (*r* = .14 [.10, .19]), and 2010 (*r* = .10 [.05, .16]). Effect sizes remained heterogeneous after controlling for study year (τ^2 = 0.011, *SE* = 0.004, *z* = 2.95, *p* = .003).

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[1] The unstandardized coefficient, *b*, can be interpreted like this: for every unit increase in study year, the *r*-to-*z*-transformed correlations decreased by 0.004; alternatively, for each ensuing decade, *z*-transformed correlations decreased by 0.04.

Figure 3. Testing the decline effect (Schooler, 2011) in meta-analysis: Effect sizes (*r*) as a function of study year (time) for 33 effect sizes
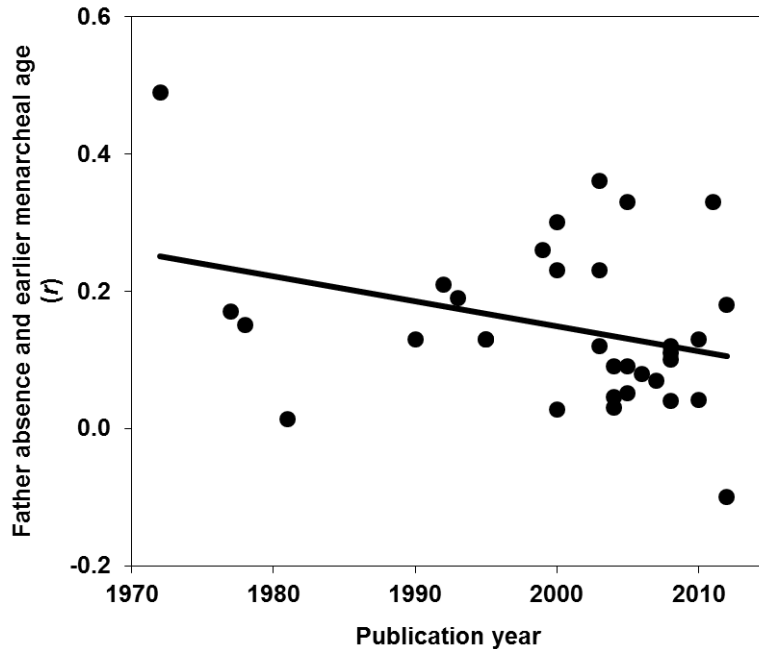
Notes: The solid line indicates the regression slope *unweighted* by sample size. We report *weighted* regression analyses in the Results section.

Menarcheal age versus menarcheal age embedded in a multi-item scale

Given the data we obtained (see Table 1), on a post-hoc basis we also tested possible between-study differences in outcome measures—menarcheal age versus menarcheal age embedded as an item in a pubertal timing survey (i.e., Pubertal Development Scale [Petersen, Crockett, Richards, and Boxer, 1988] and/or Index of Adolescent Development [Morris and Udry, 1980]). This difference did not significantly moderate the father-absence–menarcheal-age relationship ($b = 0.057$, $SE = 0.051$, $z = 1.14$, $p = .26$, $d = 0.41$). Simple effects tests (Aiken and West, 1991) confirmed a trivial difference between the four studies that asked about menarcheal age in pubertal timing surveys ($r = .19$ [.10, .28]) versus the 29 studies that simply reported menarcheal age ($r = .14$ [.09, .18]). Effect sizes remained heterogeneous after controlling for these outcome differences ($\tau^2 = 0.013$, $SE = 0.005$, $z = 2.40$, $p = .016$).

Discussion

The present meta-analysis is the first review to quantify the relationship between fathers' absence and earlier menarche in their daughters across studies. This meta-analysis is important because it provides evidence of a small-to-moderate effect size. In term of Cohen's *d*, girls raised in homes in which their biological father was absent reached menarche earlier than girls raised in intact homes by between one- and two-fifths standard deviations on average. We also showed that publication bias, outcome measure, and study

year did not moderate effect sizes. In practical terms, these small effects are far from trivial. According to a review by Ellis (2004), women from father-absent homes showed earlier menarcheal onset by 2–8 months (Belsky, 2012). Although these differences may seem small, they can have substantial cascading effects on reproductive ability: "early menarche was associated with early onset of ovulatory menstrual cycles; for example, the time from menarche until 50% of cycles were ovulatory was approximately 1.0 year if menarche occurred before age 12, 3.0 years if menarche occurred during age 12, and 4.5 years if menarcheal age was 13 or older" (Ellis, 2004, pp. 927–928).

Although our meta-analytic results can speak to the relation between father absence and menarche in humans, it remains unclear whether these findings generalize to other mammals. For example, in many non-human primates (e.g., female chimps and baboons that lack status or resources), social and environmental stressors *delay* rather than *accelerate* pubertal timing (Hrdy, 1999). In contrast, supporting psychosocial acceleration theory, differential parental care in rats—maternal licking and grooming—predicts earlier sexual maturation, sexual behavior, and parenting style (see Cameron et al., 2005, for a review). Moreover, within and across species, different mechanisms may underlie effects. The presence of related males suppresses pubertal development in some species, hypothetically via pheromones or other mechanisms (Susman and Dorn, 2009). The absence of related males constitutes a stressful environment in humans due to a range of socio-contextual factors (e.g., SES, parenting behaviors). Often, environmental stress is a catchall that includes stressors or stressful conditions that have different physiological impacts on organisms. Further comparative studies will be needed to test psychosocial acceleration theory's generalizability to other animals.

Limitations and implications

Although meta-analyses offer advantages over narrative literature reviews (Card, 2012; Hunter and Schmidt, 2004; Schmidt, 2010), they are not without limitations, and the present study is no exception. First, although the effect sizes did not vary as a function of the between-study variables, they consistently showed residual heterogeneity. This lack of homogeneity in the effect sizes leaves open the possibility that one or more unmeasured study-level variables might explain this variance. One possible moderator may be the way in which father absence was defined across studies. Unfortunately, studies defined this variable in a number of ways (e.g., years father absence, father absence before or after one or more arbitrary age cut-offs, father absence due to death vs. divorce, father absence with or without stepfather)—so many, in fact, that developing a meaningful coding scheme to test these differences in an unbiased way would have proven difficult. Nevertheless, we encourage future researchers to consider measuring and examining variables that might explain the remaining heterogeneity in these effect sizes.

Second, although we found no substantial evidence of publication bias using four methods, absence of evidence is not evidence of absence. A larger sample with more unpublished or "file-drawer" studies might provide an even more convincing test.

Third, given the correlational nature of this meta-analysis, our results cannot speak to causal, mediating, or "third variable" processes. For example, recent research has suggested that the links between family structure and pubertal timing may be mediated or

moderated by lower SES or inconsistent resources (e.g., Belsky et al., 2012; Deardorff et al., 2011; Ellis et al., 2009; James-Todd, Tehranifar, Rich-Edwards, Titievsky, and Terry, 2010; Simpson, Griskevicius, Kuo, Sung, and Collins, 2012). New experimental studies have even found that simply reminding women about paternal absence or disengagement (via priming) can alter their behavior (sexual thought/permissiveness, condom use) in ways consistent with psychosocial acceleration theory (DelPriore and Hill, 2013). Our meta-analysis also does not address recent work that has examined genetic, hormonal, or shared-environment correlates relevant to psychosocial acceleration theory (e.g., Ellis and Essex, 2007; Ellis et al., 2012; Mendle et al., 2006). Future research may wish to consider using both experimental designs and biological measures to test and advance theory.

Fourth, because we focused this meta-analysis on examining the link between father absence and earlier menarche, it is limited in scope. It cannot speak to broader questions such as how fathers' absences affect daughters' stress and SES, how these factors may in turn affect pubertal timing and sexual debut, and the extent to which both may be influenced by individual differences in genetics or personality. For example, our findings do not address the crucial role insecure attachment styles or other environmental stressors may play (Chisholm, Quinlivan, Petersen, and Coall, 2005). Specifically, recent research has shown that socioeconomic harshness (income-to-needs ratio) and unpredictability (moving, parents changing jobs) both predicted accelerated sexual behavior at age 15 (Belsky, Schlomer, and Ellis, 2012). Harsh and unpredictable environments in general, such as those that include a lack of or inconsistent resources, tend to guide development toward a faster life history strategy (Ellis et al., 2009). Unpredictable environments at ages 0–5 predicted a fast life strategy at age 23, including increased risk and sexual behavior; however, neither harsh nor unpredictable environments at ages 6–16 predicted the same, suggesting early childhood instability may be a crucial developmental antecedent of adult risk behaviors (Simpson et al., 2012). Recent research also suggests that family-level socioeconomic disadvantage is related to deviant peer relationships in adolescence, which in turn contributes to sexual promiscuity (Dishion, Ha, and Véronneau, 2012). In at least one study, SES was positively associated with age at menarche; poorer girls reached menarche at earlier ages (James-Todd et al., 2010). Our research also cannot speak to other aspects of individual differences in women's pubertal timing beyond menarche (e.g., waist-to-hip ratio; Perilloux, Webster, and Gaulin, 2010).

Fifth, a related issue is that the life history model focuses on predicting reproductive strategies and sexual behavior; we have discussed the model in the context of that literature. Pubertal timing in girls, however, is associated with both elevated internalizing and externalizing symptoms as well as clinically relevant increased rates of mental disorders during adolescence and young adulthood (Susman and Dorn, 2009). In contrast, later maturation in females is associated with a host of positive outcomes by young adulthood (Graber, Seeley, Brooks-Gunn, and Lewinsohn, 2004). Sexual experiences have been linked, often negatively, to psychopathology, especially in girls (see Graber and Sontag, 2009, for a review of this literature). In particular, sexual behavior poses a new challenge for emotion regulation, especially for younger adolescents and those who experience more frequent relationship changes (i.e., those youth on the pathway for a particular reproductive strategy as per psychosocial acceleration theory). Studies that

integrate childhood experience with the burgeoning literature on pubertal timing and adolescent sexual behavior, adjustment, and risk behaviors would inform the life history model.

Finally, most of the studies in our meta-analysis defined father absence/presence categorically; however, recent research suggests that it may be the time and quality of paternal investment that more directly affects daughters' pubertal timing and sexual behavior (Ellis et al., 2012; Tither and Ellis, 2008). In addition, boys as well as girls are affected by father absence; for example, father absence before age 7 was related to earlier reproduction in men (having at least one child by age 23; Sheppard and Sear, 2011). At the same time, pubertal timing does not appear to be a pathway for such effects in boys. Hence, future studies should also examine psychosocial acceleration theory and potential mechanisms in adolescent boys.

Conclusion

The present research is the first meta-analysis linking father absence to earlier menarche in young women. Effect sizes were heterogeneous, but the weighted average correlation was significantly greater than zero and small-to-moderate in magnitude. We found no significant evidence of publication bias or effect size decline over time. Regarding theoretical advances, the present meta-analysis supports at least one aspect of psychosocial acceleration theory (Belsky et al., 1991), and consequently also supports a life history theory of human pubertal development, specifically for females. We hope our meta-analytic findings will serve as a touchstone for inspiring future research, not only on the relationship between father absence and earlier menarche, but also on broader life history questions, and on the ongoing theoretical integration of evolutionary and developmental psychology (Ellis and Bjorklund, 2012).

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