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Impact of Fathers on Daughters' Age at Menarche: A Genetically and Environmentally Controlled Sibling Study

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Girls growing up in homes without their biological fathers tend to go through puberty earlier than their peers. Whereas evolutionary theories of socialization propose that this relation is causal, it could arise from environmental or genetic confounds. To distinguish between these competing explanations, the authors used a genetically and environmentally controlled sibling comparison design to examine the effects of differential exposure to family disruption/father absence in a community sample of sister pairs. As specified by evolutionary causal theories, younger sisters had earlier menarche than their older sisters in biologically disrupted families ($n = 68$) but not biologically intact families ($n = 93$). This effect was superseded, however, by a large moderating effect of paternal dysfunction. Younger sisters from disrupted families who were exposed to serious paternal dysfunction in early childhood attained menarche 11 months earlier than either their older sisters or other younger sisters from disrupted families who were not exposed to such dysfunction. These data suggest that early exposure to disordered paternal behavior, followed by family disruption and residential separation from the father, can lead to substantially earlier menarche.

Keywords: evolutionary psychology, behavior genetics, father absence, father–child relations, divorce, puberty.

Age at menarche varies widely across and within populations, and this variation has substantial social and biological implications. An extensive body of research in Western societies now indicates that early pubertal maturation in girls (relative to same-age peers) is associated with a variety of negative health and psychosocial outcomes, including mood disorders, substance abuse, adolescent pregnancy, and a variety of cancers of the reproductive system (e.g., Caspi & Moffitt, 1991; Ellis, 2004; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Kelsey, Gammon, & John, 1993; Mendle, Turkheimer, & Emery, 2007). Given these links, it is critical to understand the life experiences and pathways that place girls at increased risk for early pubertal maturation. This understanding would have great relevance to the long-term goal of informing early intervention–prevention strategies for high-risk youth.

Many correlational studies have identified biological family disruption/father absence (i.e., separation or divorce of the birth parents followed by absence of the birth father from the home) as a risk factor for early pubertal development in daughters (reviewed in Ellis, 2004). Moreover, the earlier that family disruption/father absence occurs, the earlier daughters tend to experience puberty (Ellis & Garber, 2000; Moffit, Caspi, Belsky, & Silva, 1992; Quinlan, 2003; Surbey, 1990). Although this body of research has established a replicable empirical phenomenon, it has not determined causality because extant correlational designs have not been able to rule out selection effects—the possibility that pre-existing differences (e.g., genetic differences, socioeconomic differences) between biologically intact/father-present and biologically disrupted/father-absent families account for the association with pubertal timing.

There are three competing classes of explanation for the observed relations between family disruption/father absence and earlier pubertal development:

1. *Family disruption/father absence and associated factors may actually cause earlier pubertal development in daughters*. One theoretical camp has explicitly advanced this causal argument: Evolutionary-based models of developmental experience, such as psychosocial acceleration theory (Belsky, Steinberg, & Draper, 1991; Chisholm, 1999) and paternal investment theory (Draper & Harpending, 1982, 1988; Ellis, 2004), posit that family disruption/father absence places daughters at risk for precocious sexual development and reproductive behavior. Central to these models is the concept of *conditional adaptation*, described by Boyce and Ellis (2005) in the following passage as:

> . . . evolved mechanisms that detect and respond to specific features of childhood environments—features that have proven reliable over evolutionary time in predicting the nature of the social and physical world into which children will mature—and entrain developmental pathways that reliably matched those features during a species' natural selective history.

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> Conditional adaptations . . . underpin development of contingent survival and reproductive strategies and thus enable individuals to function competently in a variety of different environments. (p. 290)

Paternal investment theory emphasizes that girls detect and internally encode information specifically about the quality of paternal investment in childhood as a basis for calibrating the development of (a) neurophysiologic systems involved in the timing of pubertal maturation and (b) related motivational systems, which make certain types of sexual behavior more or less likely in adolescence. An assumption of the theory is that early experiences provide assays of the quality of male–female relationships and the father's investment in the family, and these assays in turn provide input to the regulatory mechanisms that control sexual development. Girls whose early family experiences are characterized by discordant male–female relationships and relatively low paternal investment (e.g., divorce, unreliable provisioning or childcare by the father, paternal antisocial behavior) register that male parental investment is not crucial to reproduction; these girls are hypothesized to develop in a manner that speeds rates of pubertal maturation, accelerates onset of sexual activity, and orients the individual toward relatively unstable pair bonds. Conversely, girls whose early family experiences are characterized by more harmonious male–female relationships and relatively high paternal investment are hypothesized to develop in the opposite manner. Either way, the girl entrains a developmental trajectory that, in the adult social environment into which she will mature, was likely to have promoted reproductive success during human evolutionary history (see especially Belsky et al., 1991).

2. *The relation between family disruption/father absence and earlier pubertal development in daughters may derive from a family-wide environmental confound*. Family-wide environmental effects are causal factors that differ among families but are shared within families. A family-wide environmental confound could cause both family disruption/father absence and earlier pubertal development. For example, poverty is associated with both elevated rates of family disruption/father absence (e.g. Ellis et al., 2003) and, according to recent studies in the United States, earlier pubertal development in girls (Braithwaite et al., 2007; Davison, Susman, & Birch, 2003; Ellis & Essex, 2007). If poverty (or some other family-wide environmental factor) is the underlying cause of the relation between family disruption/father absence and earlier pubertal development, then the "effect" of family disruption/father absence is in fact spurious (i.e., it arises from a third environmental variable).

3. *The relation between family disruption/father absence and earlier pubertal development in daughters may derive from a shared genetic confound*. Behavior geneticists refer to this type of association as a gene–environment correlation. Specifically, girls who mature earlier tend to exhibit earlier onset of sexual activity and earlier age at first marriage and first birth (reviewed in Ellis, 2004). This covariation may occur because early pubertal timing results in precocious sexual and reproductive behavior or because pubertal, sexual, and reproductive timing are genetically correlated traits (Rowe, 2002). Early reproduction in turn is associated with increased probability of divorce and lower quality paternal investment (e.g., Amato, 1996; Bennett, Bloom, & Miller, 1995). Because mothers who are early maturers tend to have daughters who are early maturers (e.g., Malina, Ryan, & Bonci, 1994; Salces, Rebato, Susanne, San Martin, & Rosique, 2001), the correlation between family disruption and timing of pubertal maturation in girls may be spurious; that is, it may simply be due to genetic transmission of pubertal timing and associated behavioral characteristics (Belsky et al., 1991; Kim & Smith, 1998; Moffit et al., 1992; Rowe, 2000; Surbey, 1990). This noncausal explanation converges with molecular genetic research demonstrating the effects of allelic variations on pubertal timing (e.g., Kadlubar et al., 2003; Stavrou, Zois, Ioannidis, & Tsatsoulis, 2002).

Comings, Muhleman, Johnson, and MacMurray (2002) have proposed a more specific version of the genetic transmission hypothesis based on a variant of the X-linked androgen receptor gene. According to Comings et al., fathers carry X-linked genes that are associated with aggression and impulsivity, sexual promiscuity, and associated patterns of marital conflict and dissolution. These genes are transmitted to daughters, in whom they are associated with paternal absence, earlier age at menarche, and precocious sexual activity. Comings et al. (2002) found support for this theory in molecular genetic research with two clinical samples (males hospitalized for substance abuse, female outpatient volunteers for a weight control program). Jorm, Christensen, Rodgers, Jacomb, and Easteal (2004), however, found no support for the theory in two epidemiological molecular genetic studies using general population samples. Further research is needed to reconcile these contradictory results.

Past Attempts To Address Potential Environmental and Genetic Confounds

To distinguish between these opposing classes of explanation (causal vs. spurious), various investigators have tested for associations between family disruption/father absence and daughters' pubertal timing while controlling for such potential confounds as children's initial body size or level of pubertal development, mothers' age at menarche, race, and socioeconomic status (reviewed in Ellis, 2004). Although father-absent effects often remain statistically significant after these variables have been controlled, the covariate adjustment method necessarily relies on an arbitrary and incomplete set of control variables that the researcher has measured; it cannot account for unmeasured environmental or genetic factors. This limitation highlights the need for genetically and environmentally controlled research designs that incorporate environmental measures.

One previous research group (Mendle et al., 2006) used a genetically controlled research design in a study that tested for the effects of family composition (family disruption/father absence/stepfather presence) on age at menarche. Mendle et al. (2006) used the children-of-twins (CoT) methodology, which obtains measures of twins' other family members along with specific measures of family environment to test for environmentally mediated effects (see D'Onofrio et al., 2003; Turkheimer, D'Onofrio, Maes, & Eaves, 2005). Although traditional twin models have been used to partition sources of variance in pubertal timing into genetic and environmental components, these models do not enable researchers to test for specific effects of family disruption/father absence on pubertal timing because twins are usually concordant for family dissolution. The CoT design circumvents this problem by comparing female monozygotic twins who have each married and had children, but who are discordant for divorce. The children have

genetically identical mothers (and thus receive the same genetic risk of family disruption and pubertal timing from their mothers) but have different environmental exposure to family disruption. If the daughters of Twin A, who experienced family disruption, attain menarche earlier than the daughters of Twin B, who did not experience family disruption, then a causal (environmentally mediated) influence of family disruption on timing of puberty is provisionally supported. Mendle et al.'s (2006) CoT analyses, however, did not support a causal interpretation of the effect of family composition on age at menarche.

Although the CoT design constitutes a major advance in the study of the effects of family disruption on child development, it has significant limitations. First, the CoT design is only 50% genetically controlled; it does not account for the genetic and environmental influences of the twins' spouses (Eaves, Silberg, & Maes, 2005). Second, the CoT method does not control for environmental risk factors that influence only one of the adult twins and her children (D'Onofrio et al., 2003, 2005). Third, the CoT design cannot feasibly test for moderators of divorce effects; that is, the approach is underpowered to test whether the effects of family disruption/father absence on children are moderated by the behavioral adjustment of the father (which is a major focus of the current study).

The Current Sibling Design

The potential causal influence of amount of exposure to family disruption/father absence can be tested through the use of a differential sibling exposure design. Because full biological siblings have the same mother and father and normally grow up in the same home, the current sibling design does not share the limitations of the CoT approach. Given that more prolonged exposure to (earlier onset of) family disruption is associated with earlier pubertal development in daughters (reviewed in Ellis, 2004), then the following conditions should be met if this association is in fact causal: Within families in which (a) full biological sisters are discrepant in age and (b) younger sisters have more prolonged exposure to family disruption/father absence than do their older sisters, younger sisters should tend to experience earlier puberty. In addition, this should not be explicable as a birth order/birth spacing effect because sisters of different ages from biologically intact families should not systematically differ in timing of puberty. By contrast, according to the genetic transmission model, full biological sisters should not systematically differ in pubertal timing as a function of birth order/birth spacing (even if they have spent different amounts of their childhoods in biologically disrupted families).

Central to this design are comparisons between full biological sisters (a) who are discrepant in age, (b) who experienced the dissolution of their biological parents' union (either marital or cohabitating) while growing up, and (c) who then (at least in the case of the younger sister) lived primarily with their mother following the dissolution. If the siblings are born 7 years apart, for example, then the childhood environment of the older sibling is characterized by 7 more years of residence in a biologically intact, father-present family, whereas the childhood environment of the younger sibling is characterized by 7 more years of residence in a biologically disrupted family without the birth father in the home. The current study examined the effects of these differences between sisters in amount of exposure to family disruption/father absence (a nonshared environmental factor) on age at menarche.

A methodological advance of the current sibling design is that it controls for both family-wide environmental effects and genetic effects. First, family-wide environmental effects are controlled through within-family analyses. Specifically, direct comparisons between biological siblings in the same home obviate confounding effects associated with comparisons between individuals from different homes, such as differences between individuals in race/ethnicity, socioeconomic status, and religion (Rodgers, Cleveland, van den Oord, & Rowe, 2000; Sulloway, 1996). Second, genetic effects are controlled through randomization. The assumption is that genetic differences between sisters are randomly distributed in relation to birth order. That is, there is no reason to expect that either younger sisters (who spend more of their childhood in biologically disrupted families) or older sisters (who spend less of their childhood in biologically disrupted families) have systematically greater genetic liability for anything. This randomization method differs from traditional methods in quantitative genetics (which are geared toward deriving heritability estimates by comparing levels of similarity between individuals who vary in degrees of genetic relatedness).

Further, to account for possible birth order/birth spacing effects (which may affect pubertal timing; e.g., Matchock & Susman, 2006), we compared the magnitude of sibling differences across a primary sample of sister pairs from biologically disrupted families and a matched control sample of sister pairs who grew up in biologically intact families. The central hypothesis of the current study was that the birth order/birth spacing of the sisters (older vs. younger) would interact with family type to predict sibling differences in age at menarche. Specifically, birth order/birth spacing of sisters should produce larger differences in age at menarche in biologically disrupted than in biologically intact families because in biologically disrupted families (but not in biologically intact families), birth order/birth spacing is a proxy for differences between sisters in amount of exposure to family disruption/father absence.

Using sister pairs from biologically intact families as a comparison group and controlling for environmental and genetic confounds through within-family analyses enabled us to test whether the different (nonshared) experiences of sisters within families cause sibling differences in age at menarche. The measured nonshared environmental influence in this study was differential amounts of exposure to family disruption/father absence. Many factors, however, covary with family disruption/father absence (e.g., residential mobility, stepfather exposure, child abuse and neglect, mother's mental health, economic resources; see Amato, 2000; Daly & Wilson, 1998; McLoyd, 1990). The current sibling design could not isolate which of these related nonshared environmental factors (whether measured or unmeasured) had the most causal traction. Thus, the current study could only test the broader hypothesis that differential amounts of exposure to family disruption/father absence and associated factors within families cause sibling differences in age at menarche. Further, the study specifically tested for unique effects of family disruption/father absence and associated factors on age at menarche; the study design controlled for but did not negate potential family-wide environmental and genetic influences.

Dysfunctional Paternal Behavior as a Moderating Factor

Because all fathers are not equal, it is unlikely that living in a biologically intact, father-present family is always either good or bad for children. Rather, the effects of fathers in families are likely to be moderated by the personal characteristics of the father (see especially Jaffee, Caspi, Moffitt, Taylor, & Dickson, 2001), as well as the larger familial and ecological contexts. For example, a study by Jaffee, Moffitt, Caspi, and Taylor (2003) suggested that the mere presence of the biological father in the home does not produce uniformly positive outcomes for children; rather, the potential benefits of living with two biological parents may be contingent on the mental health of the parents and the quality of care they provide. Specifically, Jaffee et al. (2003) found that among children whose fathers engaged in low levels of antisocial behavior, more prolonged co-residence with the father was associated with *lower* rates of conduct problems. Conversely, among children whose fathers engaged in high levels of antisocial behavior, more prolonged co-residence was associated with *higher* rates of conduct problems. These results suggest that the effects of fathers in families may be moderated by the degree to which fathers engage in antisocial behavior (and perhaps by father psychopathology and functioning more generally). Accordingly, among the group of sister pairs in the current study from biologically disrupted families, we examined the interaction between dysfunctional paternal behavior and the birth order/birth spacing of sisters in prediction of age at menarche. The hypothesis that differential exposure to paternal dysfunction would influence age of menarche is consistent with past research demonstrating that variation in paternal behavior across families forecasts timing of puberty in daughters (Ellis & Essex, 2007; Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999; Steinberg, 1988).

In sum, the proposed sibling design offers a unique method for addressing questions of substantial scientific and social importance: Does greater exposure to family disruption/father absence and associated factors cause earlier onset of puberty? And is this effect moderated by levels of paternal dysfunction?

Method

The current research necessitated a retrospective design. On the one hand, the methodology requires the pairs of sisters to be several years apart in age (so as to ensure large within-pair differences in amount of exposure to family disruption/father presence or absence). On the other hand, the design requires that researchers collect information about the same developmental time span—childhood to early adolescence—so as to obtain equivalent and uncensored information about family environment and age of menarche from each sister. To meet both requirements, all participants had to be at least 16 years old.

The foremost challenge of this research was obtaining a sample of full biological sister pairs who were several years apart in age, whose parents separated or divorced while the younger sister was still prepubertal, and who, at least in the case of the younger sister, had resided primarily with the mother following the separation/divorce. Because such sister pairs are rare, they are difficult to obtain through normal sampling methods and instead must be located through targeted advertising. Although advertising results in a self-selected sample, this was not a major issue in the current research because within-family comparisons control for both family-wide environmental effects and genetic effects that differ between families. Further, as summarized in the following section, the demographics of the sample were representative of the population.

Participants

Recruitment. To obtain the sample, we distributed circulars advertising the study to approximately 65,000 mailboxes in urban areas in New Zealand (primarily Christchurch). Respondents to these circulars were initially screened for family composition, and then sister pairs who met the selection criteria were invited to complete the main questionnaire. We were able to obtain 93 pairs of sisters from biologically intact families and 68 pairs of sisters from biologically disrupted families. In all biologically intact families, the biological parents were married or cohabitating throughout both of the sisters' childhoods (birth to age 16). In all biologically disrupted families, the parental union terminated through divorce or separation when the younger sister was premenarcheal (at the time of the divorce/separation, average age of the younger sister was 5.41 years [$SD = 3.35$], and the average age of the older sister was 11.79 years [$SD = 3.61$]). Following the divorce/separation, the younger sister lived either primarily with her mother or in joint custody between her mother and father. This ensured that differences between sisters in the amount of time that they lived in a biologically disrupted/father-absent home was equal to or greater than their age difference. For younger sisters, age range at participation was 16–44 years old ($M = 27.27$, $SD = 6.6$); for older sisters, age range was 19–52 years old ($M = 33.92$, $SD = 6.86$). The average age difference between sisters from biologically intact families was 6.83 years ($SD = 2.19$) and between sisters from biologically disrupted families was 6.48 years ($SD = 2.06$). All participants in the current sample were fluent speakers of English; information about other language(s) spoken in the home was not collected.

Demographics. Because the current sample was obtained through advertising (self-selection), it is important to show that the demographic characteristics of the sample are not unusual. Accordingly, we report demographic comparisons between the current sample and a general population sample. The average age of the two samples was approximately the same. The general population sample was a birth cohort of women born in Christchurch, New Zealand, in 1977 (the Christchurch Health and Development Study, or CHDS). CHDS data on biologically intact versus biologically disrupted families were drawn from Ellis et al. (2003).

In both the current study and the CHDS, father's occupational status was classified using the Elley–Irving (1976) scale of occupational status for New Zealand. This scale classifies families into six groups on the basis of paternal occupation. For the present purposes, the Elley–Irving coding was reduced to a three-level classification as follows: Levels 1/2 (professional and managerial); Levels 3/4 (clerical, technical, and skilled); and Levels 5/6 (semiskilled, unskilled, and unemployed). Sisters reported their father's primary occupation for the duration of their teenage years and the highest educational qualification attained by their mother. Mothers' education level was coded into a three-level classification: no formal educational qualifications; high school qualifications; or postsecondary certificate or degree. Sisters also reported their mother's age at first birth.

As shown in Table 1, the demographic profiles of the current sample and CHDS sample were remarkably similar across all

variables and in both biologically disrupted and biologically intact family types. Further, in each study, biologically intact families were more commonly found among European New Zealanders than among ethnic minorities, as well as among families with higher paternal occupational status, higher maternal educational attainment, and later maternal age at first birth (Table 1). In total, the demographic profiles of sister pairs from both biologically disrupted and biologically intact families were representative of their respective family types, as well as differences between those family types, in the general population.

Measures

Participating sisters were mailed questionnaire packets and instructed to complete them independently of each other. Among the scales included in these packets were the following measures:

Age at menarche. Participants were asked, "How old were you when you first menstruated (got your period)?" Responses were scored in years. The reliability of retrospective reports of age at menarche has been established in several long-term prospective studies in which self-reported age at menarche was first obtained in adolescence and then again 17–37 years later. Correlations across these two measurement periods have been consistently high, ranging from .67 to .79 (Casey et al., 1991; Damon, Damon, Reed, & Valadian, 1969; Livson & McNeil, 1962; Must et al., 2002). Correlations between sisters in age at menarche were .36 ($n = 93$, $p < .001$) in biologically intact families and .24 ($n = 68$, $p < .05$) in biologically disrupted families.[1]

Father warmth. For assessment of perceived levels of warmth in father–daughter relationships during childhood, each sister completed the 12-item Care subscale of the Parental Bonding Inventory (PBI; Parker, Tupling, & Brown, 1979). When completing the PBI, participants rate their father as they remember him in the first 16 years of life (sample items: "My father spoke to me with a warm and friendly voice"; "My father did not help me as much as I needed" [reversed]; "My father seemed emotionally cold to me" [reversed]). Responses to these statements are given on a 4-point scale (1 = *very unlike*, 4 = *very like*). Items were appropriately keyed so that higher scores indicated higher levels of father warmth (Cronbach's $\alpha = .93$). The two sisters' ratings of father warmth were strongly correlated, $r(158) = .59$, $p < .001$. The Care subscale of the PBI has demonstrated high test–retest reliability over a 10-year period (Wilhelm & Parker, 1990) and correlates with many forms of adolescent behavioral adjustment and mental health (e.g., Chambers, Power, Loucks, & Swanson, 2001; Gerra et al., 2004; Martin, Bergen, Roeger, & Allison, 2004).

Father psychopathology. An eight-item checklist, completed by both sisters, was used to assess symptoms consistent with father psychopathology. The instructions stated, "The next questions concern your father's mental health. Please think back to your childhood, up to the age of 16 years." Participants responded to items on the checklist on a three-point scale (*yes*, *no*, *don't know*). The first two items assessed relatively mild behavioral problems ("Did your birth father suffer from nervous or emotional problems [such as anxiety or depression]?" "Did your birth father have trouble with drinking or other drug use?"). The next six items assessed more serious behavioral problems (e.g., "Did your birth father have any history of suicide/attempted suicide?" "Did your birth father have any history of offending involving violence?" "Did your birth father have any history of imprisonment?"). The number of items marked *yes* by the two sisters was highly correlated, $r(161) = .75$, $p < .001$, indicating high agreement between sisters in their perceptions of paternal behaviors consistent with psychopathology.

Computation of paternal dysfunction. Given the likely limitations of the sisters' knowledge of symptoms consistent with psychopathology in their fathers, sisters' ratings were used to divide fathers into three broad groups, reflecting different overall levels of paternal dysfunction. This three-level measure was based primarily on sisters' ratings of father psychopathology and secondarily on ratings of father warmth. Because paternal dysfunction was computed as a between-family variable (i.e., levels of paternal dysfunction differed between families but were the same within families), we focused on consensual information provided by sisters in calculation of this variable. Specifically, for each item on the psychopathology checklist, fathers were only given an affirmative score if both sisters answered *yes* or if one sister answered *yes* and the other answered *don't know*. If a father received no affirmative answers on the checklist, then he received a provisional psychopathology rating of 0 (*low*). If a father received an affirmative answer on either of the first two checklist items (indicating moderate behavioral problems) but no affirmative ratings on the subsequent six items (indicating serious behavioral problems), then he received a provisional psychopathology rating of 1 (*moderate*). Finally, if a father received an affirmative answer on any of the final six checklist items (indicating serious behavioral problems), then he received a provisional psychopathology rating of 2 (*serious*). These provisional psychopathology ratings were negatively correlated with composited ratings (averaged across the two sisters) of father warmth, $r(161) = -.46$, $p < .001$.

As in Jaffee et al. (2003), the provisional psychopathology ratings formed the primary basis of the paternal dysfunction measure that was used as the moderating variable in the analyses. However, because it is possible that a father could have a history of symptoms consistent with psychopathology but still be a good father, or vice versa, the provisional psychopathology ratings were adjusted according to father warmth. These adjustments were only made when the provisional ratings were contradicted by clear, consensual evidence of either high or low paternal warmth. Specifically, if a father's provisional psychopathology rating was in the moderate to serious range, but his two daughters consensually rated him as high on warmth (i.e., if he received a father warmth score of 3 or higher, averaged across the two sisters), then his provisional score was reduced by 1 point (from 2 to 1 or from 1 to 0). Conversely, if a father's provisional psychopathology rating was in the low to moderate range, but his two daughters consensually rated him as low on warmth (i.e., if he received a father warmth score of 2 or below, averaged across the two sisters), then his provisional score was increased by 1 point (from 0 to 1 or from 1 to 2). The resulting *paternal dysfunction* scores were distributed as follows: Of the 93 fathers in biologically intact families, 69%

[1] The trend toward a lower correlation in age at menarche between sisters from biologically disrupted families presumably results from these sisters having more disparate rearing experiences (i.e., differential exposure to family disruptions/father absence) than do sisters from biologically intact families.

Table 1
Demographic Comparisons in Biologically Disrupted and Biologically Intact Families: The Current Sibling Study Versus the Christchurch Health and Development Study

Demographic variables	Current sibling study		CHDS	
	Biologically disrupted families	Biologically intact families	Biologically disrupted families	Biologically intact families
Race/ethnicity (%)				
European New Zealander	79	87	76	92
Maori/Polynesian	16	11	24	8
Other	5	2		
Fathers' occupation (%)				
Professional and managerial	13	30	13	26
Clerical, technical, and skilled	52	54	46	57
Semiskilled, unskilled, and unemployed	35	16	41	17
Mothers' education (%)				
Postsecondary certificate or degree	16	25	10	26
High school qualifications	32	31	20	31
No formal educational qualifications	52	44	70	43
Mean age (yrs) of mothers at first birth (*SD*)	21.3 (3.4)	23.6 (3.8)	21.8 (4.3)	24.3 (3.9)

Note. CHDS = Christchurch Health and Development Study.

received paternal dysfunction scores of 0, 17% received scores of 1, and 13% received scores of 2; of the 68 fathers in biologically disrupted families, 34% received paternal dysfunction scores of 0, 21% received scores of 1, and 46% received scores of 2. The higher levels of paternal dysfunction in fathers of disrupted families was consistent with past research in New Zealand indicating that such fathers are disproportionately poor, unemployed, involved in abusive and illegal activities, and affected by serious mental health problems (Jaffee et al., 2001).

Results

Tests of Major Hypotheses

To test our central hypothesis—that differences between sisters in age at menarche would be greater in biologically disrupted than in biologically intact families—we conducted a 2 × 2 mixed analysis of variance (ANOVA). Because the theory clearly generated a directional prediction, we employed a one-tailed test of the statistical significance of the hypothesized interaction. The analysis included one within-subjects factor (sister; older vs. younger) and one between-subjects factor (family type; biologically disrupted vs. biologically intact). The dependent variable was age at menarche. The results of this analysis are shown in Figure 1. There were no main effects of either sister or family type. As predicted, however, there was a statistically significant Sister × Family Type interaction, $F(1, 159) = 2.81$, $p < .05$, one-tailed. As shown in Figure 1, the direction of the slopes differed in sisters from biologically intact versus biologically disrupted families. Specifically, an examination of the means for age at menarche revealed that in biologically intact families, there was a slight tendency for

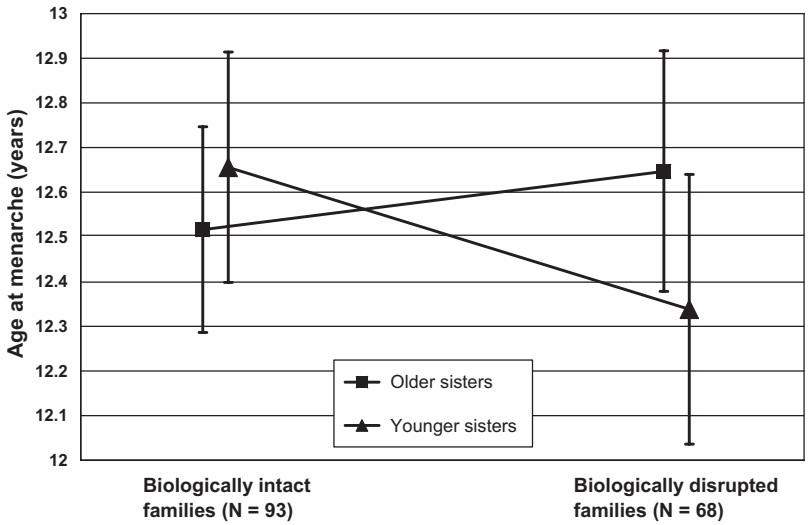

Figure 1. Differences between sisters in age at menarche in biologically intact versus biologically disrupted families. Error bars indicate 95% confidence intervals.

older sisters (M = 12.52 years, SD = 1.29) to attain menarche earlier than their younger sisters (M = 12.66 years, SD = 1.49). By contrast, in biologically disrupted families, the opposite occurred: Younger sisters (M = 12.34 years, SD = 1.52) tended to attain menarche at earlier ages than did their older sisters (M = 12.65 years, SD = 1.41). Thus, consistent with the causal hypothesis, greater exposure to family disruption/father absence was associated with earlier menarche. Nonetheless, the amount of variance accounted for by this interaction was small (partial η^2 = .02).

Next, in the 68 biologically disrupted families, we examined whether the effect of differences between sisters in exposure to family disruption/father absence was moderated by the functioning of the father (paternal dysfunction). Because we did not have strong a priori predictions about the precise nature of this interaction, two-tailed tests of statistical significance were employed. A 2 × 3 mixed ANOVA was conducted, with one within-subjects factor (sister; older vs. younger) and one between-subjects factor (paternal dysfunction; none, moderate, serious). We did not include a family type factor (intact vs. disrupted) in this analysis because the cell sizes for moderate and serious paternal dysfunction in intact families were too small to provide adequate statistical power to detect interactions in a three-way ANOVA. The dependent variable was age at menarche. There were no main effects of either sister or paternal dysfunction. However, as shown in Figure 2, there was a statistically significant interaction between sister and paternal dysfunction, $F(2, 65) = 3.75$, $p < .05$ (two-tailed). This interaction, which accounted for 10% of the variance in age at menarche (partial η^2 = .10), indicates that differences between sisters in age at menarche differed across levels of paternal dysfunction. Figure 2 suggests that this interaction was driven by early menarche in younger sisters from families with serious paternal dysfunction.

To break down this interaction, we performed two contrasts. To keep the overall Type 1 error rate across comparisons at $\alpha = .05$, we divided α by 2 (Bonferroni correction) and set it at .025. The first contrast compared age at menarche in the group of younger sisters from families with either no paternal dysfunction (M = 12.70 years, SD = 1.25) or moderate paternal dysfunction (M = 12.86 years, SD = 1.46) with the group of younger sisters from families with serious paternal dysfunction (M = 11.84 years, SD = 1.61), independent-samples $t(65) = 2.58$, $p < .025$ (two-tailed), partial η^2 = .09. The second contrast compared age at menarche in older sisters (M = 12.74 years, SD = 1.41) versus younger sisters (M = 11.84 years, SD = 1.61) in families with serious paternal dysfunction, paired-samples $t(30) = 2.69$, $p < .025$ (two-tailed), partial η^2 = .20. Thus, in biologically disrupted families, younger sisters exposed to serious paternal dysfunction attained significantly earlier menarche than either (a) younger sisters from other families who were not exposed to serious paternal dysfunction or (b) their own older sisters (who were also exposed to serious paternal dysfunction but for a longer time period). The within-family effect size was more than twice as big as the between-family effect size.

Potential Confounding Effects of Between-Family Differences in Birth Order and Family Size

In the current study, we examined the influence of a nonshared environmental variable—differential exposure within families to family disruption/father absence and associated factors—on age at menarche while controlling for potential family-wide environmental and genetic confounds (through within-family analyses) and main effects of birth order/birth spacing (through inclusion of a biologically intact control group). However, it is still possible that the present results could have been affected by differences between intact and disrupted families in the number or positioning of other siblings in the family. Specifically, past research has suggested that family size and birth order may influence pubertal timing, though findings have been inconsistent (reviewed in Ma-

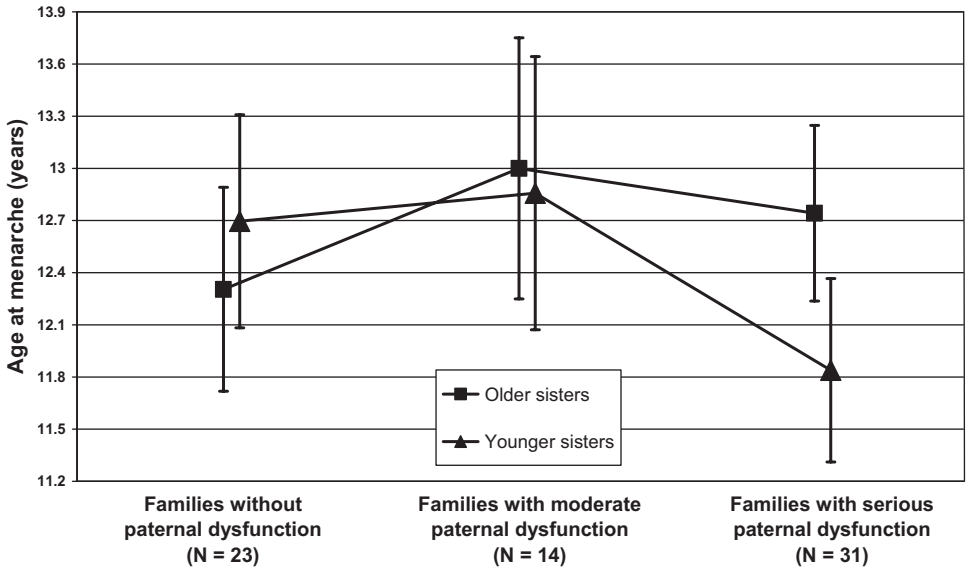

Figure 2. Differences between sisters in age at menarche as a function of paternal dysfunction in biologically disrupted families. Error bars indicate 95% confidence intervals.

lina, Katzmarzyk, Bonci, Ryan, & Wellens, 1997; Matchock & Susman, 2006). Accordingly, we compared (a) the birth order positions of the older and younger sisters and (b) overall family size across the two family types.

Based on full biological siblings only, the average birth order of older sisters was 1.69 ($SD = 1.26$) and 1.43 ($SD = 0.74$) in intact and disrupted families, respectively, $t(159) = -1.64$, $p = .10$ (equal variances not assumed), whereas the average birth order of younger sisters was 3.66 ($SD = 1.46$) and 3.32 ($SD = 0.98$) in intact and disrupted families, respectively, $t(159) = -1.72$, $p = .09$ (equal variances not assumed). Thus, the relative birth order positions of older and younger sisters from biologically intact and disrupted families were very similar, with a small trend toward higher birth order positions in the sisters from disrupted families. This trend essentially disappeared, however, if we included half-siblings in the calculations. (With few exceptions, only sisters from disrupted families had half siblings.) Counting both full and half siblings, the average birth order positions of the older and younger sisters from disrupted families increased to 1.62 ($SD = 0.86$) and 3.51 ($SD = 1.07$), respectively.

Based on full biological siblings only, the average family size was 3.96 ($SD = 1.60$) and 3.56 ($SD = 1.18$) in intact and disrupted families, respectively. Although this difference was not statistically significant, $t(159) = -1.81$, $p = .07$ (equal variances not assumed), there was a clear trend toward larger family size in intact families (as would be expected, given that parents who do not separate/divorce have more time together to reproduce). This trend once again disappeared, however, if half-siblings were included in the calculations. Counting both full and half siblings, the average family size was 3.96 ($SD = 1.42$) in disrupted families. Matchock and Susman (2006) found that the most salient aspect of family size in relation to pubertal timing was number of older sisters; thus, we analyzed the distribution of this variable. For the older participating sisters in our study, the average number of older full biological sisters was 0.28 ($SD = 0.63$) and 0.16 ($SD = 0.44$) in intact and disrupted families, respectively, $t(159) = -1.39$, $p = .17$ (equal variances not assumed). Conversely, for the younger participating sisters in our study, the average number of older full biological sisters was 1.76 ($SD = 0.96$) and 1.62 ($SD = 0.71$) in intact and disrupted families, respectively, $t(159) = -1.10$, $p = .27$ (equal variances not assumed). Counting both full and half siblings in disrupted families, the average number of older sisters increased to 0.22 ($SD = 0.48$) and 1.68 ($SD = 0.76$) for the older and younger participants, respectively. In total, there were not meaningful differences between sisters from biologically intact versus disrupted families in relative birth order positions, family size, or number of older sisters, regardless of whether the calculation of these variables included half siblings.

Discussion

The current study converges with past research that has shown an association between greater exposure to family disruption/father absence and earlier age at menarche. Specifically, in biologically disrupted families, younger sisters (who had greater exposure to father absence) experienced menarche an average of 3–4 months earlier than their older sisters (who had less exposure to father absence). By contrast, in biologically intact families, younger sisters experienced menarche an average of about 1–2 months later than their older sisters. Although the difference between these two regression slopes was statistically significant, the effect size was small, accounting for about 2% of the variance in age at menarche. This is comparable to the effect sizes documented in past studies that have examined the association between amount of exposure to family disruption/father absence and pubertal timing (Ellis & Garber, 2000; Moffit et al., 1992; Quinlan, 2003; Surbey, 1990).

Extending past research, however, the within-family analyses in the current study plausibly support a causal interpretation of the effect of family disruption/father absence and associated factors on age at menarche. That is, the current results suggest that the nonshared experiences of sisters associated with differential exposure to family disruption/father absence cause differences in age at menarche, above and beyond any effects of family-wide environmental and genetic factors. Moreover, the between-family analyses indicate that this effect was not an artifact of differences between sisters from biologically intact versus biologically disrupted families in birth spacing (age differences between the sisters), birth order relative to other siblings, or number of older sisters. These results are consistent with evolutionary–developmental models that posit a causal effect of family environments.

Why are the effect sizes in both past research and the present study relatively small? One possibility, supported in the current research, is that the main effect of family disruption/father absence on age at menarche is moderated by the characteristics of the father. In the current study, this small main effect was superseded by a large moderating effect of paternal dysfunction. This moderating effect revealed a specific context in which family disruption/father absence and associated factors appeared to markedly accelerate menarche. Specifically, in disrupted families in which sisters were exposed to serious paternal dysfunction (e.g., substance abuse, criminal offending, violence), the younger sisters had substantially advanced menarche. Indeed, these younger sisters attained menarche almost a year earlier (i.e., 11 months) than did either (a) younger sisters from other families who experienced comparable amounts (high dosage) of family disruption/father absence but were not exposed to serious paternal dysfunction or (b) their older sisters who experienced less family disruption/father absence (low dosage) but had more prolonged exposure to serious paternal dysfunction. Thus, early exposure to serious paternal dysfunction, followed by family disruption and departure of the biological father from the home, contributed to earlier attainment of menarche. This conclusion is based on genetically and environmentally controlled within-family sibling data, as well as between-family comparisons. It should be noted that the effect size for the within-family analysis was about twice as large as that for the between-family analysis, indicating that the effect was primarily driven by direct comparisons between older and younger sisters.

Another possibility, which was not tested in the current research, is that the main effect of family disruption/father absence on age at menarche is moderated by the characteristics of the child. The current study tested only whether this effect occurs independently of—but not through interactions with—genetic factors. Due to genetic and developmental variations, however, some girls may be more susceptible than others to family-rearing influences on pubertal timing (see Belsky et al., 2007). Accordingly, the small main effect of family disruption/father absence on age at menarche found in the current study may underestimate (occlude) the actual

effect in more susceptible populations of adolescents and overestimate it in less susceptible populations.

A Note on Causal Inference

Causation cannot be definitively established without a randomized experimental design. Sibling-difference methodologies are quasi-experimental designs; they can only support soft rather than hard causal inferences (see Björklund & Sundström, 2006; Ermisch & Francesconi, 2001). For example, imagine an idiosyncratic trait, perhaps arising from a set of allelic variations, that causes both early menarche and associated behavioral problems that bring about parental separation/divorce. Although such a trait should be randomly distributed across older and younger sisters, if an older sister has the trait, it reduces the probability that the parents will stay together long enough to produce a younger sister, whereas possession of the trait by the younger sisters is independent of the probability of there being an older sister. This type of trait, therefore, could be more common in younger than older sisters in the current study, which would bias estimates of the causal effect of differential exposure to family disruption/father absence on age at menarche. In total, without random assignment, the current research design enables us to make plausible, but not definitive, causal inferences.

An International Adoption Analogy

Research on the timing of puberty in internationally adopted children may shed light on the current findings. Poor children adopted from third-world countries into affluent Western families experience significantly earlier puberty than do children from either their countries of origin or their host countries, despite histories of chronic early neglect/abuse, infection, and malnutrition prior to adoption (Domine, Parent, Rasier, Lebrethon, & Bourguignon, 2006; Mul, Oostdijk, & Drop, 2002; Teilmann, Pedersen, Skakkebæk, & Jensen, 2006). Moreover, girls adopted into Western families experience earlier menarche if they are adopted at later ages, even though the older adoptees experienced more sustained deprivation prior to adoption. In a study of girls adopted into Swedish families, for example, Indian and Bangladeshi girls who were adopted at 3 years of age or older versus those who were adopted at younger than 3 years of age had averages ages of menarche of 11.1 years vs. 11.9 years, respectively (Proos, Hofvander, & Tuvemo, 1991). Likewise, in a large cohort study of girls growing up in Denmark (including both native Danes and foreign adoptees), girls from developing countries who were adopted when older than 2 years of age versus those who were adopted when younger than 2 years of age had rates of precocious puberty that were approximately 35 times higher vs. 5 times higher, respectively, than the Danish reference group (Teilmann et al., 2006). These data indicate that girls who experience persistent deprivation (later adoptees) respond to a dramatic improvement in environmental quality by accelerating sexual maturation. Sexual precocity may function in this context to exploit a potentially narrow window of reproductive opportunity (see Worthman, 1999).

Although malnutrition has been reported in many children adopted from foreign countries, weight is generally less compromised than height, and many foreign adoptees at risk for early puberty have normal body mass index at arrival (Parent, Teilmann, Juul, & Skakkebaek, 2003), suggesting a prominent role for factors other than just nutrition (see especially Johnson, 2000). Indeed, Domine et al. (2006) contended that alleviation of psychosocial stressors can be crucial in fostering early growth and pubertal development in internationally adopted children.

In terms of the present study, growing up with a resident father who has serious psychopathology is one of the most severe forms of psychosocial stress experienced by children in Western societies and is associated with many negative child outcomes (e.g., Jaffee et al., 2001, 2003). For younger sisters in biologically disrupted families, primary residence with their biological father ended at an average of 5.4 years of age. In the 31 biologically disrupted families in which younger sisters were exposed to serious paternal dysfunction, departure of the father from the home surely translated into alleviation of a major source of psychosocial stress (possibly during a sensitive age window for programming of maturation of the reproductive axis). It was in these girls that age at menarche was markedly accelerated. This finding is consistent with the data on internationally adopted children showing especially high rates of early puberty in children adopted after approximately 3 years of age. The older sisters presumably did not experience accelerated puberty because the major stressor was not removed in time.

The current findings, together with the international adoption data, suggest that timing of onset and offset of exposure to major psychosocial stressors/disruptive events have important effects on pubertal timing. This raises an important interpretive issue: The current sibling design conflates timing of onset of and amount of exposure to family disruption/father absence. That is, younger sisters both have earlier onset of and longer exposure to family disruption/father absence than do their older sisters. In designing and carrying out the current study, we adopted a dose–response metaphor, assuming that length of exposure to family disruption/father absence had cumulative developmental effects (analogous to public health concepts of exposure to environmental toxins that accumulate over time). However, the current data, in conjunction with international adoption studies, may be more parsimoniously explained as a sensitive period than dose-response effect. In the original articulation of paternal investment theory, Draper and Harpending (1982) posited an early sensitive period (approximately the first 5 years of life) for the effects of father absence on daughters' sexual development. This sensitive period approach concurs with contemporary developmental theories emphasizing disruption of developmental trajectories during important transition points in the life course (e.g., see Glenn Elder's [1998] life course theory). In the wake of family disruption, changes in exposure to serious paternal dysfunction in early to middle childhood may be a key life transition that substantially alters trajectories of pubertal development.

Consideration of Competing Explanations

As reviewed above, a widely held view in the literature is that relations between family disruption and timing of pubertal maturation in girls are the spurious results of genetic transmission of pubertal timing and associated characteristics. The present data

suggest that genetic (and family-wide environmental) confounds do not tell the whole story. Specifically, by controlling for both family-wide environmental and genetic confounds, the current sibling design enabled us to test for the unique effects of a measured nonshared environmental variable—differential exposure within families to family disruption/father absence (and associated factors)—on menarcheal timing. The current results suggest that the small but reliable effect of family disruption/father absence on pubertal timing, as documented in past research, may be driven by a relatively small number of girls who are exposed to disordered fathers in early childhood and then experience family disruption and cease living with those fathers.

Consistent with evolutionary-based theories of developmental experience, these data plausibly support a causal role for fathers in families and, more specifically, an interaction between father functioning and timing of family disruption/father absence in regulation of daughters' sexual development. Nonetheless, consistent with Scarr's (1992) concept of "good enough" parenting, amounts of exposure to fathers who were functioning within the ordinary, normal range seemed to have little effect on daughters' pubertal timing; rather, it was levels and timing of exposure to fathers who expressed symptoms consistent with psychopathology that appeared to have the most causal traction. These data challenge the assumption that female sexual maturation is sensitive to the continuous quality of paternal investment (see Ellis et al., 1999). Moreover, the findings highlight the need for revision of evolutionary–developmental models to address the importance of changes in paternal conditions (i.e., responsiveness to windows of opportunity during sensitive age periods).

Limitations and Implications

Limitations of the present study should be noted because they provide important directions for future research. First, and foremost, the current research was based on a relatively small sample and could thus have generated unreliable parameter estimates. It will be especially important in future research to study larger numbers of biologically disrupted families in which sisters are differentially exposed to serious paternal dysfunction.

Second, in the current research, we used extensive screening procedures to find families with the qualities required to conduct relevant sibling comparisons. We do not know the extent to which the results from these screened-in families generalize to screened-out families. This is probably not a major issue in the current research, however, because the demographics of our sample closely paralleled a same-aged birth cohort from Christchurch.

Third, the assessment of paternal dysfunction relied on retrospective reports by siblings. This is potentially problematic because such reports may be affected by time and life experience (e.g., perceptions of paternal substance abuse or criminal behavior may differ when daughters are 16 years old vs. 36 years old). Moreover, sisters may have limited knowledge of paternal behaviors, and thus the paternal dysfunction ratings may reflect more closely sisters' feelings and beliefs about their fathers than veridical assessments of psychopathology. Nonetheless, high levels of agreement between sisters in ratings of paternal symptoms consistent with psychopathology support the validity of the current assessment method.

Fourth, age of menarche was assessed retrospectively. Despite high correlations between reports of age at menarche collected over time (e.g., Must et al., 2002), there is substantial within-person variability in repeated assessments of menarche (Dorn et al., 1999), and accuracy of recall decreases as the time period between menarche and recall increases (Koo & Rohan, 1997). In spite of these limitations, Casey et al. (1991) found that 84% of women (mean age = 50 years) were able to recall their age of menarche to within 1 year. Given the age of our sample, participants should have been reasonably accurate in reporting age at menarche to the nearest year.

Fifth, the method used in the current study to control for genetic confounds—randomization of genetic effects across birth order—presumes that the sisters are full biological siblings; however, extrapair paternity rates have been estimated at approximately 2% (Simmons, Firman, Rhodes, & Peters, 2004) and are probably higher in children of divorce. If (a) there are substantively meaningful rates of extrapair paternity in our sample, (b) the distribution of extrapair paternity is nonrandom with respect to the birth order of the sisters, and (c) instances of inpair versus extrapair paternity have resulted in systematically (directionally) different genetic effects on age at menarche, then it would bias estimates of the causal effect of differential exposure to family disruption/father absence on age at menarche. The likelihood that all of these conditions were met, however, is slim. Nonetheless, future studies could benefit from DNA testing of the sisters.

Finally, we did not test for mediating mechanisms. Future research is needed to identify intervening childhood experiences and associated neuroendocrine processes that account for divergent patterns of sexual maturation in older and younger sisters (e.g., differences between sisters in early experiences of neglect or abuse, in exposure to stepfathers and other males in the household, in production of growth hormones, in peripubertal patterns of fat deposition). Along these lines, Matchock and Susman (2006) found that the presence of half- and step-brothers was associated with earlier menarche and have proposed a pheromonal mechanism (see also Ellis, 2004).

In closing, we would like to comment on possible clinical implications of this work. As stated in the Introduction, early pubertal maturation in girls is associated with an array of negative mental and physical health outcomes. For example, a 1-year advancement in age at menarche, which approximates the within- and between-family effects documented in the current study, increases breast cancer risk by an estimated 4.7% (Decarli, La Vecchia, Negri, & Franceschi, 1996). The radical implication of the current study is that common processes operating in families (e.g., early exposure to serious paternal dysfunction followed by divorce and departure of the father from the home) can substantially alter timing of menarche. These results highlight modifiable determinants of early menarche that could be targeted for intervention.

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