Adopted children show more problem behaviors than nonadopted children. Given that internationally adopted individuals show earlier puberty than nonadopted individuals, and early puberty is associated with problem behaviors in nonadopted youth, we analyzed data from adopted domestic adoptees to determine whether problem behaviors could be explained by differences in pubertal timing. Relative to nonadopted controls \((n = 153)\), domestically adopted girls \((n = 121)\) had earlier menarche, earlier sexual initiation, and more conduct disorder symptoms. Age at menarche partially mediated the relation of adoptive status to sexual initiation, but not to conduct disorder symptoms. Extending findings from international adoptees, results show that domestic adoption is also linked to earlier puberty, and suggest early puberty as one mechanism linking adoption to problematic outcomes.

Adoption is associated with a greater risk for behavior problems in childhood (Keyes, Sharma, Elkins, Iacono, & McGue, 2008). Adopted children are two to four times more likely to develop behavior problems—including aggressive, defiant, and risky behaviors—than children who are not adopted (Brodzinsky, 1993; Juffer & van Ijzendoorn, 2005; Wierzbicki, 1993). Adopted adolescents are at increased risk for substance use (Fergusson, Lynskey, & Horwood, 1995), juvenile offending (Fergusson et al., 1995), and early sexual activity (Bricker et al., 2006). Explanations proposed to account for the increase in behavior problems in adopted compared to nonadopted individuals include characteristics such as thrill seeking or risk taking (e.g., Markey, Markey, & Tinsley, 2003; Martin et al., 2002), but there have been few direct empirical tests of such mechanisms.

We suggest that pubertal timing is a mechanism linking adoption to the development of behavior problems in adolescence, especially in girls. Like adoptive status, early puberty in girls has been associated with an increased rate of problematic outcomes compared to on-time puberty, including low school achievement (Cavanagh, Riegle-Crumb, & Crosnoe, 2007), increased negative affect (Graber, Brooks-Gunn, & Warren, 2006), more psychological symptoms (Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997), and earlier sexual initiation (Cavanagh, 2004; Moffitt, Caspi, Belsky, & Silva, 1992). A popular explanation for such links focuses on early physical development as a catalyst for early interactions with older boys (Ge, Conger, & Elder, 1996), thus increasing opportunities for sexual activity and other forms of risky behavior.

Adoption status has also been identified as a risk factor for early pubertal timing in international adoptees. Internationally adopted children have been shown to reach puberty earlier than children who remained in their country of origin and than children native to the adoptive country (Krstevska-Konstantinova et al., 2001; Mason & Narad, 2005). Early puberty in internationally adopted children has been suggested to result from gains in height and weight due to improved diet and nutrition in the adoptive country (Mason & Narad, 2005; Virdis et al., 1998) and from exposure, in the country of origin, to environmental toxins that act as endocrine disrupters (Krstevska-Konstantinova et al., 2001). Adoption may also be linked to early puberty through other mechanisms that are applicable to domestic, as well as international, adoptees. These include adverse rearing environments (Bel- sky et al., 2007), being born to a young mother (increasing the likelihood of being small for gestational age, which, in turn, increases risk for obesity.
and early pubertal development; Barker, 1998), and the genetic transmission of pubertal timing (Ge, Natsuaki, Neiderhiser, & Reiss, 2007), given that young mothers may have experienced early puberty themselves (Deardorff, Gonzales, Christopher, Roosa, & Millsap, 2005). The association between adoptive status and early pubertal development is particularly pronounced in girls (Mason & Narad, 2005; Tuvemo & Proos, 1993).

Data from domestic adoptees could help constrain explanations of early puberty in international adoptees, and enhance our understanding of links among adoption, puberty, and problem behavior, given that the adversities associated with the preadoption environment are less severe for domestic adoptees in the United States (Gunnar, Bruce, & Grotevant, 2000; Juffer & van IJzendoorn, 2005). If adverse environmental conditions lead to problematic outcomes (e.g., early puberty, behavior problems) in adoptees, then domestic adoptees should be less affected than international adoptees. In contrast, if the adoption experience itself contributes to problematic outcomes in adoptees, then domestic adoptees should be affected in the same way as international adoptees.

Therefore, we consider links among adoption, puberty, and behavior problems in a sample of domestically adopted individuals. In particular, we integrated findings linking adoption to both early puberty and behavior problems in order to investigate whether pubertal timing is one mechanism by which adoption leads to negative psychological outcomes in adolescent girls. We hypothesized that domestically adopted girls would show early puberty relative to nonadopted girls, and that early puberty would mediate the link between adoptive status (adopted vs. nonadopted) and two problem behaviors: early sexual initiation and symptoms of conduct disorder.

METHOD

Participants

Participants were drawn from the Colorado Adoption Project (CAP; Petrill, Plomin, DeFries, & Hewitt, 2003; Plomin & DeFries, 1985), a longitudinal study designed to explore the influence of genes and the social environment on individual differences in cognitive and socioemotional development. Birth mothers who relinquished their children through local private adoption agencies in Denver, Colorado between 1975 and 1983 were invited to participate in the CAP. Adoptive parents received information about the CAP during adoption orientation procedures and were invited to participate following adoptions. Adopted children were placed in adoptive homes at an average of 29 days of age. Control families with only biological children were recruited through birth records of local hospitals and matched to adoptive families on child sex and number of siblings, and parent age, education, and occupation. All participants were from the United States (i.e., domestic).

Most participants in the CAP were Caucasian (>90%) and adoptive families were largely middle class according to occupational ratings of prestige (National Opinion Research Center; Duncan, 1961). Biological mothers had lower socioeconomic status (SES) than adoptive and control parents. This difference likely reflected the relative youth of biological mothers. SES for biological grandparents of adopted children was comparable to that of adoptive and control families (Plomin & DeFries, 1985). SES did not differ between adoptive families and control families (t(271) = .99, p = .32).

The current study is focused on girls given that early pubertal development is more consistently found in internationally adopted girls than boys (Mason & Narad, 2005; Tuvemo & Proos, 1993) and most links between pubertal timing and behavior problems are seen in girls. Information on puberty was available for 121 adopted girls and 153 nonadopted girls, representing 94% of the girls in the original CAP sample. Reflecting the distribution in the CAP sample as a whole, the majority of girls in the current study were born to Caucasian birth mothers (89.1%; African-American: 0.4%, Mexican-American: 3.3%, Other: 1.5%) and birth fathers (85.8%; African-American: 1.1%, Mexican-American: 4.0%, Other: 1.8%). At the child’s birth, birth mothers averaged 20.11 years of age (range: 13.62–33.40).

Girls included in the current study did not differ from those who were excluded because of missing puberty data on adoptive status ($\chi^2_{11} = .06$, $p = .81$), birth weight ($t(256) = -.59$, $p = .56$), or race of biological mother ($F(1, 310) = 2.61$, $p = .11$) or biological father ($F(1, 310) = .78$, $p = .38$).

Measures

Menarche. Pubertal development was assessed with age at menarche, which is a salient marker for pubertal development in girls and has shown to be more reliable than self-report of other aspects of physical development (Brooks-Gunn, Warren,
Age at menarche was determined from annual assessments of pubertal development conducted from ages 9 through 15, during the summer following the school year in which the majority of children reached the nominal age of assessment (e.g., year 9 corresponds to post 3rd grade, and year 12 to post 6th grade). For girls with puberty data, mean ages (and standard deviation) at each of the yearly assessments were as follows: 9.39 (.33), 10.36 (.34), 11.34 (.34), 12.38 (.36), 13.38 (.36), 14.41 (.36), and 15.35 (.30) years. Age at assessment did not differ significantly between adopted and nonadopted girls at any occasion (t(212) = 1.74, p > .05).

Age at menarche was determined by asking adopted and nonadopted girls the age at which they began to menstruate. Parent report data were used in cases where self-report data were missing (n = 16). This information was elicited at each assessment and inconsistencies were resolved in favor of the report closest in time to the event. The reliability of self-reported age at menarche has been shown to be high; correspondence with physician ratings range from 90% to 100% (Brooks-Gunn et al., 1987).

**Age at sexual initiation.** During annual assessments from age 17 to age 24, CAP participants (N = 189) were asked about their age at sexual initiation (Bricker et al., 2006), defined as the age of first sexual intercourse. Discrepancies between reported dates were resolved in favor of the report closest in time to the event. For example, if a 17-year old girl reported sexual initiation during the previous year, but 3 years later reported having initiated sex at age 18, age 17 was taken as the age at sexual initiation. A discrepancy of 1 year was resolved for 39 girls. A discrepancy of 2 years was resolved for eight girls. The test-retest reliability for self-reported age at sexual initiation was .95 [95% CI: .93-.96].

**Conduct problems.** Lifetime symptoms of conduct disorder were assessed at the age 17 assessment using the Diagnostic Interview Schedule for Children (DISC-IV; Shaffer et al., 1996), a reliable and valid measure of symptoms of conduct disorder as defined by the DSM-IV (APA, 1994). Similar to previous reports (Young et al., 2006), scores were derived by standardizing the total number of conduct disorder symptoms in a sample of community participants and then regressing out effects of age, gender, and type of assessment. Among individuals with available data on conduct disorder symptoms (N = 230), 12.17% had 3 or more lifetime symptoms, which is the minimum criterion for diagnosis of Conduct Disorder. Over half (58.70%) had at least one lifetime symptom of conduct disorder.

**Plan for analysis.** Hypotheses were tested in two steps. First, the hypothesized difference in age at menarche between adopted and nonadopted girls was tested with a one-tailed t-test. Second, age at menarche was tested as a mediator of the association between adoptive status and behavior problems (Baron & Kenny, 1986) using three criteria: Adoptive status must be significantly linked to problem behaviors; adoption must be significantly linked to the proposed mediator, age at menarche; and the association between adoptive status and problem behaviors must be significantly reduced (as assessed by the Sobel (1982) test) when age at menarche is considered in the regression.

**RESULTS**

**Group Differences in Menarche, Sexual Initiation, and Conduct Problems**

Descriptive data for age at menarche, sexual initiation, and conduct problems in adopted and nonadopted girls are shown in Table 1. Birth weight was not significantly correlated with age at menarche for adopted (r = -.05, p > .05) or nonadopted girls (r = .15, p > .05) and was similar in the two groups (t(228) = -1.08, p > .10).

Adopted girls reported earlier menarche (d = .33, t(272) = -2.69, p < .01) and more conduct problems (d = .45, t(228) = 3.37, p < .001) than did nonadopted girls. As reported previously (Bricker et al., 2006), adopted girls in this sample had an earlier age of sexual initiation (d = .66, t(187) = -4.45, p < .001) than did nonadopted girls. These differences suggest examination of menarche as a mediator of the link between adoption and problem behaviors.

**Age at Menarche as a Mediator of Adoption Effects on Problem Behaviors**

Age at menarche was significantly correlated with both age of sexual initiation (r = .25, p < .01) and conduct disorder symptoms (r = -.13, p < .05). Age at menarche was a significant partial mediator (Sobel test: z = 1.96, p < .05) of the association between adoptive status and sexual initiation (Table 2, Figure 1a). This association was slightly but significantly reduced (from $\beta = .31$, $t = 4.46$, $p < .01$ to $\beta = .27$, $t = 3.86$, $p < .01$) when age at
Menarche was included in the regression. Both adoptive status and age at menarche ($\beta = .20$, $t = 2.84$, $p < .01$) were significantly linked to age at sexual initiation when age at menarche was included in the regression.

In contrast, age at menarche did not mediate the link between adoptive status and symptoms of conduct disorder (Sobel test: $z = -1.46$, $p > .05$). Rather, the relation between age at menarche and conduct problems became nonsignificant ($\beta = -.10$, $t = -1.51$, $p > .10$) when the regression included both adoptive status and age at menarche (Figure 1b).

**DISCUSSION**

Extending findings from international adoptees, we showed that domestically adopted girls had earlier menarche than girls who were not adopted, and that early menarche partially accounted for their early age at sexual initiation but not their conduct disorder symptoms. The findings suggest that the adverse outcomes associated with adoption arise in different ways, with pubertal timing linking adoption to some risky behaviors (i.e., sexual initiation) but not to others (i.e., conduct disorder symptoms). It is logical that pubertal development is more closely linked to the initiation of sexual activity than to conduct disorder symptoms given that reproductive maturity has direct implications for sexual activity. Indeed, early physical development in girls is associated with involvement with older boys (Caspí, Lynam, Moffitt, & Silva, 1993; Cavanagh, 2004; Kim & Smith, 1998), with whom girls are more likely to initiate sexual activity (Bozon &

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**TABLE 1**

Means and Standard Deviations for Study Variables

<table>
<thead>
<tr>
<th></th>
<th>Adopted Girls</th>
<th>Nonadopted Girls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight (kg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$N$</td>
<td>107</td>
<td>125</td>
</tr>
<tr>
<td>$M$</td>
<td>3.16</td>
<td>3.23</td>
</tr>
<tr>
<td>$SD$</td>
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<td>.52</td>
</tr>
<tr>
<td>$t$</td>
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<td>-2.69</td>
</tr>
<tr>
<td>$d$</td>
<td>.15</td>
<td>.33</td>
</tr>
<tr>
<td>Age at menarche (years)</td>
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<td>153</td>
</tr>
<tr>
<td>$M$</td>
<td>12.72</td>
<td>13.06</td>
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<tr>
<td>$SD$</td>
<td>1.03</td>
<td>1.03</td>
</tr>
<tr>
<td>$t$</td>
<td>-2.69</td>
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<tr>
<td>$d$</td>
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<td>.66</td>
</tr>
<tr>
<td>Age at sexual initiation (years)</td>
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<td>98</td>
</tr>
<tr>
<td>$M$</td>
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<tr>
<td>$SD$</td>
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<td>-4.45</td>
</tr>
<tr>
<td>$d$</td>
<td>.66</td>
<td>.66</td>
</tr>
<tr>
<td>Conduct problems (z-score)</td>
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<td>128</td>
</tr>
<tr>
<td>$M$</td>
<td>.42</td>
<td>-.10</td>
</tr>
<tr>
<td>$SD$</td>
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<td>$t$</td>
<td>3.37</td>
<td>-2.69</td>
</tr>
<tr>
<td>$d$</td>
<td>.45</td>
<td>.33</td>
</tr>
</tbody>
</table>

*Group differences tested by two-tailed t-test, significant at $p < .01$.

---

**TABLE 2**

Descriptive Statistics and Results From Sobel Tests of Mediation

<table>
<thead>
<tr>
<th></th>
<th>$n$</th>
<th>$B$</th>
<th>$SE (B)$</th>
<th>$R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at sexual initiation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Adoptive status</td>
<td>189</td>
<td>1.52</td>
<td>0.34</td>
<td>0.10</td>
</tr>
<tr>
<td>2. Adoptive status</td>
<td>189</td>
<td>1.32</td>
<td>0.34</td>
<td>0.13</td>
</tr>
<tr>
<td>Age at menarche</td>
<td></td>
<td></td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td>Conduct disorder symptoms</td>
<td></td>
<td></td>
<td></td>
<td>0.05</td>
</tr>
<tr>
<td>1. Adoptive status</td>
<td>230</td>
<td>-0.51</td>
<td>0.15</td>
<td></td>
</tr>
<tr>
<td>2. Adoptive status</td>
<td>230</td>
<td>-0.47</td>
<td>0.15</td>
<td>0.06</td>
</tr>
<tr>
<td>Age at menarche</td>
<td></td>
<td></td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.07</td>
<td></td>
</tr>
</tbody>
</table>

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Kontula, 1998). Furthermore, increases in testosterone levels in girls during puberty are related to increases in sexual interest and activity, although this effect is moderated by the family environment (Halpern, Udry, & Suchindran, 1997).

Our data do not allow us to address important questions about (i) whether adoption itself causes early puberty, or rather marks risk, and (ii) whether or not earlier pubertal timing and risk for externalizing psychopathology are manifestations of the same underlying genetic risk. Previous research has shown that age at menarche is highly heritable (Towne et al., 2005), and that covariation between menarche and age at first intercourse can be accounted for by genetic variation (Rowe, 2002). Thus, it is plausible that age at menarche is linked to sexual initiation via hormonal fluctuations based on changes in gene expression across development. In this way, early puberty serves as a biological marker of a risk for early sexual behavior. Furthermore, early puberty in adoptees may reflect genetic effects on pubertal timing that, to an extent, are mediated behaviorally. Adopted girls might have early puberty because their mothers had early puberty, which resulted in early sexual activity and pregnancy (and thus adoption). In this regard, it is important to measure age at menarche in biological mothers and its link to problem behaviors in adopted girls.

Several limitations should be considered when interpreting the results of the current study. First, not all participants reported on their pubertal development at every assessment. Nevertheless, analyses on individuals with complete data across assessments produced similar results. Second, both direct and mediation effects may have been attenuated as a result of imperfect measurement. It is possible that disparate findings for sexual initiation and conduct disorder symptoms result from differences in measurement or in effect sizes. In particular, the measure of conduct disorder may not have been sufficiently sensitive or focused; we used a composite measure of symptoms of conduct disorder, rather than measures of specific behaviors used in other adoption studies, for example, self- or parent-reported delinquency, police contact, cigarette smoking, or cannabis and alcohol use. Nevertheless, the measure was sensitive enough to show differences between adopted and control girls, consistent with other studies (e.g., Juffer & van IJzendoorn, 2005). Third, although we consider our results in relation to studies in international adoptees, we did not directly compare domestic and international adoptees. Our goal was to use our data to constrain the explanatory mechanisms by which adoption is linked to early puberty and problem behaviors. Fourth, adoption practices have changed since the children in our sample were adopted, with increases in openness and in the diversity of both adopted children and adoptive parents (Zamostny, O’Brien, Baden, & Wiley, 2003). It is not obvious that these changes would affect the generalizability of our results. Fifth, our study is limited to girls given previous work (early puberty more consistently found in internationally adopted girls than boys, and clearer links between early puberty and behavior problems in girls than boys), although data from boys would be helpful in understanding mechanisms linking adoption to behavior problems. Although CAP includes boys, pubertal assessments were restricted in age (stopping before many boys completed puberty) and in quality (relying on a self-report measure of questionable validity; Dorn & Biro, 2011; Dorn, Dahl, Woodward, & Biro, 2006), making it difficult to study the issue in this sample.

Our finding of early puberty in domestic adoptees helps to constrain explanations of early puberty in internationally adopted girls. Given that adoptees in the current sample have not experienced the same extent of deprivation and adversity reported for international adoptees, it is unlikely that early deprivation fully accounts for early pubertal timing in international adoptees. Similarly, while adopted girls in the current sample had an earlier age of menarche than nonadopted girls, their average age of menarche was very close to the national average reported for similar birth cohorts (Mason & Narad, 2005; McDowell, Brody, & Hughes, 2007; Sun et al., 2005). That is, domestically adopted girls showed earlier puberty than nonadopted girls, but did not show menarche as early as has been seen in international adoptees. Thus, it is possible that being reared in the environment provided by the type of adoptive family involved in the CAP study—that is, an intact family with moderate to high levels of income and education and healthy environments—is protective for both adopted and biological daughters (Belsky et al., 2007). These familial effects appear to be stronger for domestic than for international adoptees. Thus, there appear to be some factors that differ between international and domestic adoptees, but other factors that are shared by adopted children, differentiating them from nonadopted children, likely including genetic influences, prenatal care, and the age at which the child entered the family.

In sum, it appears that being adopted is a risk factor for early pubertal development, and that...
early pubertal development partly mediates the link between adoption and early sexual activity. Additional study of these links should help to understand both the origins of behavior problems in adopted children, and the causes and consequences of variations in pubertal timing in all children.

REFERENCES


