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A Twin Study of Objective and Subjective Pubertal Timing and Peer Influence on Risk-Taking

Natalie Kretsch
University of Texas at Austin

Jane Mendle
Cornell University

Kathryn Paige Harden
University of Texas at Austin

This study used a behavioral genetic design to test whether three measures of pubertal timing moderated peer influence on risk-taking in a sample of 248 female adolescent twin pairs (M_{age} = 16.0, SD = 1.5) from the National Longitudinal Study of Adolescent Health. Peer influence was operationalized as the quasi-causal association between girls' self-reported risk-taking and the risk-taking reported by their friends. Girls with earlier ages at menarche and who perceived themselves as more developed than peers were more susceptible to peer influence on risk-taking. However, age-standardized ratings of body changes did not moderate peer influence. This study highlights distinctions between multiple measures of pubertal timing, using an innovative synthesis of genetically informative data and peer nomination data.

The increasing salience of peer relationships is a hallmark of adolescent social development. Compared to children and adults, adolescents are more sensitive to the influence of their peers (Chein, Albert, O'Brien, Uckert, & Steinberg, 2010; Gardner & Steinberg, 2005; Steinberg & Monahan, 2007). Although the growing importance of peers reflects a normal developmental process, peer influence is often studied in the context of maladaptive behaviors, such as substance use, delinquency, and other forms of risk-taking. Adolescents are more likely to engage in these behaviors in the presence of peers (Chassin, Hussong, & Beltran, 2004; Ouimet et al., 2010; Zimring, 1998), and longitudinal and experimental studies provide convergent evidence for a causal association between exposure to peer risk-taking and individual risk-taking (Cruz, Emery, & Turkheimer, 2012; Gardner & Steinberg, 2005; Jaccard, Blanton, & Dodge, 2005). Intervention and prevention efforts have attempted to understand peer influence processes both in terms of preventing negative influence (e.g., iatrogenic effects of group treatment for adolescents, reviewed by Dishion & Dodge, 2005) and garnering positive influence (e.g., "Above the Influence" antidrug campaign; Slater, Kelly, Lawrence, Stanley, & Comello, 2011).

Of particular interest are factors that render certain adolescents more vulnerable to negative peer influence than others (for a review see Brechwald & Prinstein, 2011). Pubertal timing may be one factor that contributes to individual differences in susceptibility to peer influence. Among adolescent girls, early pubertal timing predicts an array of negative psychosocial outcomes, including heightened involvement in risky behavior (Buchanan, Eccles, & Becker, 1992; Mendle, Turkheimer, & Emery, 2007; Stattin, Kerr, & Skoog, 2011). Moreover, behavioral genetic research suggests that pubertal timing moderates the relative influences of genes and environment on risk-taking; environmental influences on delinquency are stronger (and genetic influences weaker) for girls with early pubertal timing (Burt, McGue, DeMarte, Krueger,

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Requests for reprints should be sent to Natalie Kretsch, Department of Psychology, University of Texas at Austin, 108 E. Dean Keeton Stop A8000, Austin, TX 78712-0187. E-mail: natalie.kretsch@gmail.com

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& Iacono, 2006; Harden & Mendle, 2012). One of these environmental influences may be the peer group; several previous studies have found interactions between peer risk behavior and pubertal timing in predicting delinquency (Caspi, Lynam, Moffitt, & Silva, 1993; Fergusson, Vitaro, Wanner, & Brendgen, 2007; Stattin et al., 2011) and substance use (Biehl, Natsuaki, & Ge, 2007; Costello, Sung, Worthman, & Angold, 2007).

The importance of the peer group is highlighted in nearly all of the theories proposed to explain the negative correlates of early pubertal timing. For example, the *maturation disparity hypothesis* (reviewed in Ge & Natsuaki, 2009) focuses on the gap between physical and psychological development that is widened for early-maturing adolescents. Pubertal development brings substantial social changes, including upheaval in long-standing friendships and attention from opposite-sex peers. Adolescents who face these transitions at younger ages are less psychologically mature and may lack the cognitive and emotional resources necessary to cope with them (Ge & Natsuaki, 2009). The *contextual amplification hypothesis* (Ge, Brody, Conger, Simons, & McBride-Murray, 2002) considers pubertal development in the context of adolescents' social background. The negative effects of pubertal timing may be intensified in adverse environments, where girls must confront numerous risk factors, including greater exposure to delinquent peers. In support of this, the association between early maturation and deviant behavior is attenuated for girls in same-sex schools, presumably because they are "protected" from the influence of boys (Caspi et al., 1993). Other variants, such as the *peer socialization hypothesis* (Stattin et al., 2011), suggest that early maturation motivates affiliation with deviant peers because early-maturing adolescents select friends who are similar to them; these friends are likely to be older and to engage in more norm-breaking behavior than same-age, typically developing peers. This motivation for an alternate social group may be fueled by difficulty maintaining friendships with same-age peers, as early-maturing adolescents tend to experience rejection and even victimization from other children their age (Haynie & Piquero, 2006; Reynolds & Juvonen, 2011). Finally, affiliation with deviant peer groups may represent a reaction to the fact that adolescents are reproductively mature but are still not considered adults (Moffitt, 1993). This theory of adolescent-limited delinquency may explain why early maturers, who experience an extended period between reproductive and sociocultural maturity, are at heightened risk for antisocial behavior.

Measurement of Puberty

Much of the research reviewed above has used age at menarche as a measure of pubertal timing. Menarche is a discrete and personally salient event that is recalled with moderate-to-high reliability (Casey et al., 1991; Dorn, Sontag-Padilla, Pabst, Tissot, & Susman, 2013). In considering the intersection of puberty and peer environment, different indicators of pubertal timing may be more or less relevant. For example, menarche is a private event, whereas breast development is a visible social signal of reproductive maturity and potential readiness to engage in romantic or sexual relationships (Blyth, Simmons, & Zakin, 1985; Brooks-Gunn & Warren, 1989; Petersen & Taylor, 1980). There is growing recognition that different measures of pubertal development may differentially predict behavior (e.g., Carter, Silverman, & Jaccard, 2013; Michael & Eccles, 2003; Natsuaki, Biehl, & Ge, 2009). In particular, "subjective" measures of pubertal development, which rely on girls' self-reports of how they perceive their own bodies, have been criticized for their poor correspondence with objective benchmarks like age at menarche and with physician ratings of physical development (Dorn, Dahl, Woodward, & Biro, 2006). Yet these subjective self-perceptions may nevertheless be psychologically meaningful. Even if a girl is objectively "on time" (i.e., her development is typical of her age group), if she perceives herself as more developed, and if she judges these physical changes negatively, she may be at risk for psychological problems (Brooks-Gunn, Attie, Burrow, Rosso, & Warren, 1989; Brooks-Gunn & Warren, 1989; Rierdan, Koff, & Stubbs, 1988). In the case of risk-taking, if delinquent peer affiliation and delinquent behavior are attempts to assume adult status before one is granted adult privileges by society (Moffitt, 1993), then whether an adolescent "feels like" an adult may matter more than a physician's assessment of her Tanner stage. In fact, subjective measures of pubertal development are often more potent predictors of health outcomes than more objective measures (e.g., Deppen, Jeannin, Michaud, Alsaker, & Suris, 2012; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Lanza & Collins, 2002; Stice, Presnell, & Bearman, 2001; Wichstrom, 2000).

In addition, subjective perceptions of pubertal timing may be particularly relevant for understanding

effects of puberty that persist into later adolescence. By middle adolescence, when rates of risk-taking increase (CDC, 2009; FBI, 2012), most girls have experienced menarche and have "caught up" to each other in terms of pubertal stage. Yet older adolescents may continue to *perceive* themselves as early maturers or late bloomers. Consistent with this idea, Cance, Ennett, Morgan-Lopez, and Foshee (2012) analyzed longitudinal data on perceived pubertal timing from a large sample of adolescents aged 11–17 and concluded that "while pubertal development is a dynamic process, perceptions of pubertal timing based on early adolescent experiences are stable throughout adolescence and contribute to adolescent identity development" (p. 775). Thus, subjective measures of pubertal timing in older adolescents may be tapping psychologically meaningful differences in identity that are rooted in the pubertal transition.

There are important distinctions between types of self-report measures of pubertal timing. Some measures, which have been referred to as "stage-normative" pubertal timing (Cance et al., 2012), ask adolescents to rate aspects of physical development (e.g., breast growth, body hair, height, voice changes) on a scale with visual or descriptive anchors, and these ratings are normed by age. Several studies have identified interactions between the peer environment and pubertal timing using these stage-normative measures (Biehl et al., 2007; Costello et al., 2007; Fergusson et al., 2007). Other measures assess "peer-normative" pubertal timing and ask adolescents to rate their development compared to their same-age peers. Peer comparison measures, while correlated with stage-normative measures, are also unique predictors of health outcomes (Carter et al., 2013; Harden, Mendle, & Kretsch, 2011; Yuan, 2007). Moreover, because these measures provide insight into an adolescent's self-image in relation to her peer group, they are particularly relevant to understanding peer influence, the focus of this study.

Measuring Peer Influence

Studies of peer influence vary in the way that peer influence is defined and measured, for both empirical and theoretical reasons. Prevailing theories of peer influence all recognize the interplay between selection and socialization. Understanding when and how peers influence individual risk-taking requires controlling for the fact that adolescents tend to affiliate with peers who share behavioral characteristics. A twin design is a "quasi-experimental" research design that is useful in parsing selection versus socialization effects. This approach tests whether twins who are discordant for exposure to an environmental variable, such as peer risk-taking, are also discordant for a behavioral outcome (Heath et al., 1993; Lahey & D'Onofrio, 2010; Silberg et al., 2003). Previous twin and sibling studies of peer influence have generally supported a socialization model of peer influence, while also identifying selection effects. In a study of siblings and their best friends, Harden, Hill, Turkheimer, and Emery (2008) found genetic influences on an individual's substance use also influenced his or her exposure to peer substance use (a gene–environment correlation). At the same time, exposure to peer substance use predicted individual substance use after controlling for gene–environment correlation. Studies that have used the twin design in conjunction with longitudinal and social network analysis (e.g., Cruz et al., 2012) have found further support for a causal model of peer influence on risky behavior. This study employs the twin design to test whether the "quasi-causal" association between peer and individual risk-taking—that is, the within-twin pair association that remains after controlling for between-family differences in genetic and environmental background factors—is moderated by pubertal timing.

"Peer influence" is frequently inferred from the similarity between peer behavior and individual behavior; making this inference often requires that adolescents report on the behavior of their peers (Costello et al., 2007; Dick, Rose, Viken, & Kaprio, 2000). Perception of peer behavior is an important construct in itself, as perceived norms are one of the strongest correlates of adolescents' willingness to engage in risky behavior (Neighbors, Lee, Lewis, Fossos, & Larimer, 2007). However, adolescents also tend to overestimate the similarity to their peer group (Jussim & Osgood, 1989), which may lead researchers to overestimate peer influence (Gottfredson & Hirschi, 1990; Yun, Cheong, & Walsh, 2011). One approach that overcomes this potential limitation is the use of a direct report of peer risk-taking. Such data are available from studies in which adolescents nominate friends from school rosters, and the delinquency reported by one's nominated friends is used as a measure of exposure to risky peers. Following previous research on pubertal timing and peer influence on delinquency (Fergusson et al., 2007), this study used peer-reported behavior as a more conservative measure of peer group similarity.

Goals of the Current Study

We addressed two research questions. First, after controlling for genetic and environmental selection factors, does affiliation with risk-taking peers predict individual risk-taking? Based on previous research, particularly genetically informed studies, we expected that affiliation with risk-taking peers would predict individual risk-taking after controlling for selection factors. Second, is the relationship between peer and individual risk-taking moderated by pubertal timing? We predicted that girls with earlier pubertal timing would be more susceptible to peer influence on risk-taking. That is, the relationship between peer and individual risk-taking that remained after controlling for selection factors would be strongest for early-maturing girls. We used three indicators of pubertal timing: age at menarche, self-rated body changes, and a peer comparison item that asked girls to compare their own physical development to other girls of the same age. As reviewed above, previous studies have found interactions between pubertal timing and peer context using both objective and subjective measures. Thus, we predicted moderation by all of these measures.

METHOD

Participants

This study used data from the National Longitudinal Study of Adolescent Health (Add Health; Udry, 2003), a longitudinal study of health and risk behaviors among adolescents in the United States. Add Health used a school-based sampling procedure, in which a survey was administered to all students in participating schools. Schools in the United States with at least 30 enrollees were stratified by geographic region, demographic composition, and school type, and a random sample of schools was selected from these strata. Of these schools, 134 (79%) agreed to participate, yielding a sample of 90,118 adolescents who participated in the initial, in-school survey, administered in 1994–95. The school survey included items that queried whether the adolescent had a twin. An in-depth, in-home interview was conducted with a subsample of 20,745 adolescents (10,480 females) who were of ages 12–19 at Wave I (1994–95). Since this initial interview, three follow-up interviews have been conducted, in 1995–96 (Wave II), 2001–02 (Wave III), and 2007–08 (Wave IV).

The Add Health study deliberately oversampled sibling pairs for the in-home interview. A total of 253 female–female twin pairs completed the Wave I home interview. Twin zygosity was assessed using 11 molecular genetic markers (Smolen & Hewitt, 2003), by self-reports, and by responses to four questions concerning similarity of physical appearance. These questionnaire items have been cross-validated by genetic analyses (Spitz et al., 1996). A previous analysis of the Add Health twin sample indicated that demographic characteristics of the twin sample did not differ from the full sample (Jacobson & Rowe, 1999). This study sample included 248 female twin pairs (496 individuals; 137 monozygotic pairs, 111 dizygotic pairs), ages 12–19 (M = 16.0, SD = 1.5) who participated in the in-home interview. Five pairs were excluded because they did not have any data on pubertal timing, individual risk-taking, or peer risk-taking. Race and ethnicity, reported by adolescents, was classified as White (53.2%), African American (24.4%), Hispanic (15.6%), or other, including Asian and Native American (6.8%).

Measures

Risk-taking. Risk-taking was measured using seven items from the in-school survey. Adolescents were asked how often in the past year they had smoked cigarettes, consumed alcohol, gotten drunk, raced on a bike or skateboard or in a car or boat, lied to their parents, skipped school, or done something dangerous because they were dared to (α = .73). Frequency was based on a 6-point scale: *never* (0), *one or two times* (1), *once a month or less* (2), *2–3 days a month* (3), *once a week* (4), *3–5 days a week* (5), and *nearly every day* (6). Scores were averaged to obtain a mean level of risk-taking for each adolescent.

Peer risk-taking. Peer risk-taking was assessed using friendship nominations to identify each adolescent's friends. On the in-school survey, adolescents nominated up to five male and five female friends. From these nominations, we calculated the average risk-taking (based on the same seven items used to calculate individual risk-taking) reported by peers who either nominated or were nominated by the "target" adolescent. Limitations of the peer risk-taking data should be noted. Adolescents were allowed to nominate friends who did not attend the same school, but data were only collected from identifiable in-school nominations. Nominations

were nonidentifiable if the nominee was not on the school roster or not in the study. Of the 496 individuals in the current sample, 139 (28%) had no peer data because they had no identifiable peer nominations. These individuals were more likely to be Hispanic ($\chi^2 = 7.87$, $df = 1$, $p < .01$). There were no other demographic differences and no differences in average risk-taking between adolescents with and without identifiable peer nominations. These individuals were retained in the sample because they were informative regarding covariation between twins' individual risk-taking scores.

Other peer characteristics. Although not all nominations were identifiable, all were classified as either male or female. For adolescents who nominated at least one friend, the proportion of nominated friends who were male and the average age of nominated friends were calculated.

Pubertal timing. Three measures of pubertal timing were used in the current analyses.

Age at menarche. Participants reported at Waves I and II if they had "ever had a menstrual period" and, if so, during which month and year they had experienced their first menstrual cycle. At Wave III, they were asked "how old were you when you got your period for the first time?" This study used the earliest reported age at menarche for each adolescent (i.e., if an adolescent initially reported that she began menstruating at age 12 and subsequently reported that she began menstruating at age 13, age 12 was used as age at menarche). The first report of age at menarche was used to avoid telescoping bias (Pickles et al., 1994), which occurs when individuals remember events as closer to the date of the interview than they actually are. The Pearson correlation between age at menarche reported at Waves I and II ($r = .76$, $p < .001$) was higher than that between Waves II and III ($r = .53$, $p < .001$) and between Waves I and III ($r = .53$, $p < .001$). This may be attributable to telescoping or the fact that menarche at Wave III was reported in years, whereas menarche at Waves I and II was reported in months and years. Ten girls (2% of the sample) were missing data on age at menarche; they were retained in the sample because they provided data on peer and individual risk-taking.

Self-rated body changes. At Wave I, adolescents' ratings of body changes were assessed using two Likert scale items that asked about breast development (1 = *My breasts are about the same size as when I was in grade school* to 5 = *My breasts are a whole lot bigger than when I was in grade school; they are as developed as a grown woman's breasts*) and body curviness (1 = My *body is about as curvy as when I was in grade school* to 5 = *My body is a whole lot more curvy then when I was in grade school*). Both these items were correlated with age (breast development: $r = .18$, $p < .001$; curviness: $r = .21$, $p < .001$). To account for age differences among participants, scores were standardized within year of chronological age ($M = 0$, $SD = 1$), and the standardized scores for the two items were summed. Thus, higher scores reflect that *an adolescent perceived herself to be more physically developed than her same-age peers perceived themselves*. This is a commonly used technique for transforming a measure of pubertal status to one of pubertal timing in an age-heterogeneous sample (Carter et al., 2013; Ge, Natsuaki, Neiderhiser, & Reiss, 2007).

Peer comparison. At the Wave I interview, adolescents were asked "how advanced is your physical development compared to girls your age?" Responses fell on a 5-point scale (1 = "*I look younger than most*" to 5 = "*I look older than most*"). This item was used as a measure of pubertal timing based on peer comparison, with higher scores indicating that *an adolescent perceived herself to be more physically developed than she perceived her peers to be.* This item was correlated with age-standardized self-rated body changes ($r = .36$, $p < .01$). Unlike the items used to assess body changes, this measure was not correlated with age ($r = -.09$, $p = .07$). Moreover, there was homogeneity of variance across ages ($F(6, 476) = .40$, $p = .83$), indicating equivalent variability in the peer comparison measure in older versus younger girls. Additional information about how this measure related to chronological age is provided in Table S1.

Analytic Plan

Initial exploratory analyses were conducted in SAS to assess within- and between-pair correlations between pubertal timing, risk-taking, and peer risk-taking. Subsequent structural equation modeling (SEM) analyses were performed in MPlus (Muthén & Muthén, 1998–2010), using full information maximum likelihood (FIML) to account for missing data. We conducted separate analyses for each measure of pubertal timing (age at menarche, body changes, and peer comparison). Absolute model fit was assessed using root mean square error of approximation (RMSEA) and chi-square values, and nested models were compared using differences in log-likelihood. Age was included as a covariate in predicting individual and peer risk-taking.

First, to assess whether peer risk-taking predicted individual risk-taking, controlling for genetic and environmental selection effects, we fit three multivariate twin models, each of which included peer risk-taking, target risk-taking, and one measure of pubertal timing. This model is illustrated in Figure 1 (for one twin only). The twin model (for a full explanation of the twin model, see Neale & Cardon, 1992) decomposes variation in a measured phenotype into three sources—additive genetic (A) factors, shared environmental factors (factors that make siblings similar, C), and nonshared environmental factors (factors that make siblings different, plus measurement error, E). Based on genetic theory, the correlation between additive genetic factors is fixed at 1.0 for monozygotic (MZ) twins and 0.5 for dizygotic (DZ) twins. The models used in the current analysis are based on assumptions that may be overly simplistic (Charney, 2012), as recent studies suggest that monozygotic twins are not genetically identical, due to mutations, postnatal transpositions, somatic mosaicism, and epigenetic effects. However, the aim of this study was not to estimate heritability but rather to control for genetic and environmental confounds by leveraging the fact that both MZ and DZ twins share an array of genetic and environmental characteristics that are not controlled for in standard epidemiological designs.

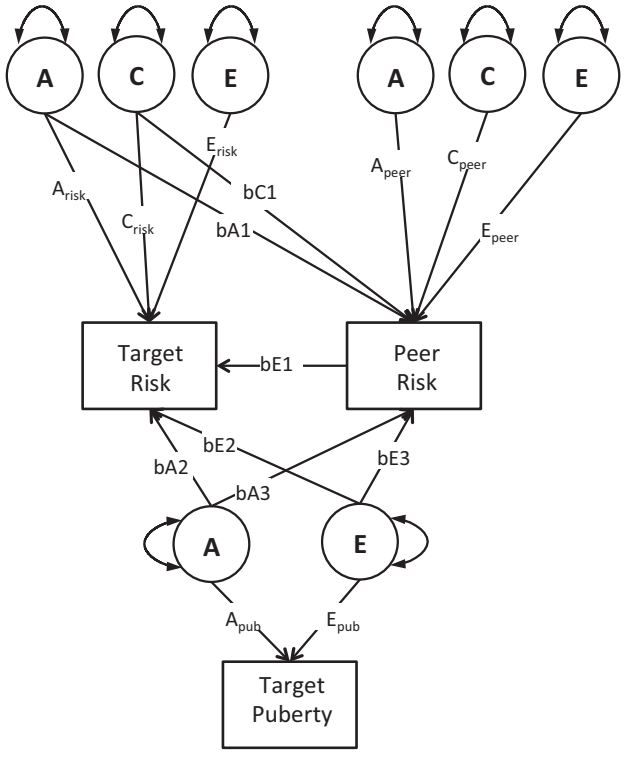

FIGURE 1 Trivariate model of pubertal timing, risk-taking, and peer risk-taking. A = additive genetic; C = shared environmental; E = nonshared environmental. Only one twin per pair shown. Each measure of pubertal timing modeled separately.

Because previous studies with these data have shown minimal effects of shared environment on pubertal timing (Ge et al., 2007; Harden & Mendle, 2012), we fit models in which the shared environmental variance in each measure of pubertal timing was set to 0. Dropping this parameter did not compromise fit for the models of peer comparison ($\Delta\chi^2 = .47$, $\Delta df = 4$, $p = .98$), body changes ($\Delta\chi^2 = .1.25$, $\Delta df = 3$, $p = .74$), or age at menarche ($\Delta\chi^2 = .26$, $\Delta df = 4$, $p = .97$).

The multivariate model also decomposes associations between phenotypes into genetic, shared environmental, and nonshared environmental pathways. In Figure 1, the path labeled bA1 represents the association between peer and individual risk-taking that is due to overlapping genetic factors—the extent to which genes that influence propensity for risk-taking also influence one's tendency to associate with risk-taking peers. The path labeled bC1 represents the association between individual and peer risk-taking due to environmental factors that make siblings similar. Environmental factors such as parents, schools, neighborhoods, cultural values, and socioeconomic status may make siblings similar in both their own risk-taking and their peers' risk-taking. Together, paths bA1 and bC1 measure genetic and environmental "selection effects"—the extent to which genetic and environmental factors shared by twins raised in the same home predict both individual risk-taking and affiliation with risk-taking peers. The path labeled bE1 is the association between peer and individual risk-taking that remains after controlling for these selection effects. This path reflects whether the twin who has a riskier peer group engages in more risk-taking than her co-twin. This pathway, referred to as the "quasi-causal" pathway or the "within-pair effect," is how we operationalize peer influence in this study.

In the second step, to assess whether pubertal timing moderated the association between individual and peer risk-taking, we allowed the cross-paths between these phenotypes to be moderated by each measure of pubertal timing. The moderation model is shown in Figure 2. The terms bA1′, bE1′, and bC1′ represent interactions between pubertal timing and the genetic, nonshared environmental, and shared environmental associations between individual and peer risk-taking. Of central interest in this second step is the term labeled bE1′, which is the interaction between pubertal timing

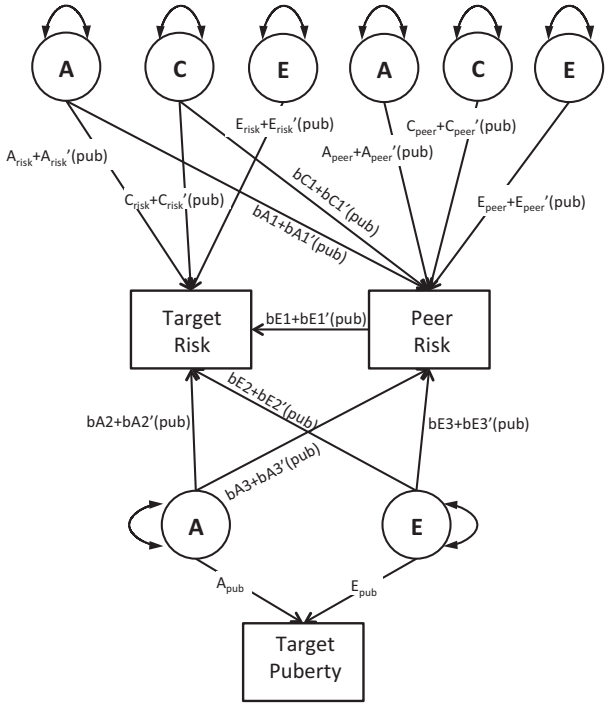

FIGURE 2 Interaction model of pubertal timing, risk-taking, and peer risk-taking. A = additive genetic; C = shared environmental; E = nonshared environmental; pub = pubertal timing. Interaction terms denoted by apostrophe. Only one twin per pair shown. Each measure of pubertal timing modeled separately.

and peer influence on individual risk-taking. A significant interaction term would indicate that pubertal timing moderated the influence of risky peer affiliation on individual risk-taking. Analyses were conducted separately for each measure of pubertal timing. For peer comparison and self-rated body changes, higher values reflect earlier pubertal timing; a *positive* interaction term bE1′ would therefore indicate that girls with earlier pubertal timing are more susceptible to peer influence than later maturing girls. For age at menarche, because higher values reflect later pubertal timing, a *negative* interaction term bE1′ would indicate increased susceptibility for early-maturing girls.

RESULTS

Descriptive Statistics

Descriptive statistics and correlations between study variables are shown in Table 1. Because target and peer risk-taking scores were positively skewed, analyses were also performed using log-transformed scores. This transformation did not affect results, and we report results that use the nontransformed scores for both target and peer risk-taking. Peer risk-taking (M = 1.05, SD = .57) was correlated with individual risk-taking (M = .89, SD = .95), r = .24 ($p < .001$). The twin pair correlation for risk-taking was r = .40 ($p < .01$) for DZ twins and r = .51 ($p < .01$) for monozygotic MZ twins. The twin pair correlations for peer risk-taking were similar for MZ twins (r = .64, $p < .01$) and for DZ twins (r = .62, $p < .01$), indicating that shared environment accounted for much of the variance in peer risk-taking; this is likely due to the fact that both MZ and DZ twins attended the same school as their co-twins and were therefore selecting friends from the same pool. As shown in Table 1, the three measures of pubertal timing—age at menarche, body changes, and peer comparison—were correlated with each other in the expected directions. In addition, all three measures were more highly correlated in MZ twins than in DZ twins, indicating significant genetic variance across all measures of pubertal timing. None of the measures of pubertal timing significantly correlated with individual or peer risk-taking.

In addition to risk-taking, other basic characteristics of the peer groups were examined. The mean

TABLE 1
Correlations and Descriptive Statistics for Key Study Variables

	Risk-Taking	*Peer Risk-Taking*	*Age at Menarche*	*Peer Comparison*	*Body Changes*
Risk-taking		.24**	.05	−.07	.05
Peer risk-taking			−.06	.02	.09
Age at menarche				−.33**	−.20**
Peer comparison					.36**
Twin pair correlations	.47**	.62**	.46**	.43**	.30**
MZ twins	.51**	.62**	.59**	.54**	.35**
DZ twins	.40**	.64**	.30**	.28**	.24*
Mean (*SD*)	.89 (.95)	1.05 (.57)	12.31 (1.44)	3.02 (1.01)	0 (1.00)

Note. N = 248 twin pairs. Self-rated body changes were standardized by age.
*$p < .05$; **$p < .01$.

TABLE 2
Unstandardized Parameter Estimates From Models of Pubertal Timing, Peer Risk-Taking, and Target Risk-Taking

	Age at Menarche	*Peer Comparison*	*Body Changes*
Model fit indices			
χ^2 (*df*, *p*)	72.77 (42, .005)	52.94 (43, .14)	63.11 (43, .04)
RMSEA	.07	.04	.06
Variance in pubertal timing			
A_{pub}	1.11**	.75**	.64**
E_{pub}	.92**	.69**	.77**
Variance in target risk-taking			
A_{risk}	.61**	.76**	.59**
C_{risk}	.42	.49*	.43
E_{risk}	.69**	.68**	.69**
Variance in peer risk-taking			
A_{peer}	0	0	0
C_{peer}	.34**	.33**	.34**
E_{peer}	.33**	.25**	.33**
Regression parameters			
Peer risk-taking → target risk-taking			
Genetic path (bA1)	.23*	.32**	.21**
Shared environmental path (bC1)	.21	.32**	.21**
Nonshared environmental path (bE1)	−.29	−.34	−.30
Pubertal timing → target risk-taking			
Genetic path (bA2)	0	−.07	.15
Nonshared environmental path (bE2)	.03	0	−.04
Pubertal timing → peer risk-taking			
Genetic path (bA3)	−.01	.05	.11
Nonshared environmental path (bE3)	−.04	−.03	−.03

Note. $N = 248$ twin pairs. Variance in target risk-taking indicates variance that is independent from pubertal timing. Variance in peer risk-taking indicates variance that is independent from pubertal timing and individual risk-taking.
*$p < .05$, **$p < .01$.

proportion of males in the peer group was .41 (*SD* = .22). This was weakly correlated with peer risk-taking ($r = .11$, $p < .05$) but not significantly correlated with any other measured variables (age, age at menarche, body changes, peer comparison, or individual risk-taking). The average age of the peer group was correlated with age ($r = .88$, $p < .01$) and with peer risk-taking ($r = .20$, $p < .01$) but not significantly correlated with any other measured variables.

Does Affiliation With Risk-Taking Peers Predict Individual Risk-Taking After Controlling for Genetic and Environmental Selection Factors?

Parameter estimates for the multivariate models that were used to address this initial question are shown in Table 2. Separate models were estimated for each of the three measures of pubertal timing: age at menarche, peer comparison, and self-rated body changes.

Age at menarche. The total variance in age at menarche (shown in the first column of Table 2) is estimated by summing the squared paths from the A and E components (labeled A_{pub} and E_{pub}) to age at menarche. The proportion of variance due to genetic and environmental factors can be calculated by dividing each squared path to age at menarche by the total variance in age at menarche. Following this formula, we found variance in age at menarche was due primarily to genetic factors (59%), with remaining variance due to nonshared environmental factors (41%). The regression paths between peer risk-taking and individual risk-taking are labeled bA1, bC1, and bE1. We found that the association between peer and target risk-taking was due to common genetic effects (bA1 = .23, $p < .05$). There were no significant shared or nonshared environmental pathways between peer and individual risk-taking (bC1 = .21, $p = .20$; bE1 = −.29, $p = .28$). Residual variance in peer risk-taking (independent of target risk-taking) was due to shared environmental (52%), and nonshared environmental (48%) factors.

Peer comparison. Variance in the peer comparison measure of pubertal timing was due to genetic

factors (54%) and nonshared environmental factors (46%). The first trivariate model found that the genetic, shared environmental, and nonshared environmental paths between peer and individual risk-taking were not significant (bA1 = .20, $p = .07$; bC1 = .23, $p = .16$; bE1 = −.25, $p = .36$). However, none of the paths was reliably different than zero. Given our relatively small sample size, we suspected we did not have sufficient power to differentiate between genetic (bA1) and shared environmental (bC1) selection effects. Consequently, we fit an additional model that set the genetic and shared environmental paths to be equal to each other (following Harden et al., 2008) to reduce the number of estimated parameters in our model. We found these paths to be significant and positive (bA1 = bC1 = .32, $p < .01$). Setting these paths to be equal did not compromise model fit ($\Delta\chi^2 = .21$, $\Delta df = 1$, $p = .65$). This suggests that there was a positive association between affiliation with risk-taking peers and individual risk-taking and that this association was due to unmeasured genetic and/or environmental confounds shared by twins raised in the same family. The nonshared environmental pathway, bE1, was not significant in any of the models (bE1 = −.34, $p = .18$), indicating that siblings who differed in exposure to risky peer groups did not differ in their own risk-taking.

Self-rated body changes. Variance in self-rated body changes was due to genetic (41%) and nonshared environmental effects (59%). Again, the genetic and shared environmental pathways between peer and individual risk-taking were set to equality and were positive significant (bA1 = bC1 = .21, $p < .01$), whereas the nonshared environmental pathway was not significant (bE1 = −.30, $p = .26$). Residual variance in peer risk-taking was again due to shared environmental (52%) and nonshared environmental (48%) effects.

Is the Relationship Between Peer and Individual Risk-Taking Moderated by Pubertal Timing?

Age at menarche. Results of moderation models are shown in Table 3. Again, the primary paths of interest are the regressions between peer and individual risk-taking. Each path had an intercept, and an interaction term that reflected moderation by age at menarche. Age at menarche moderated the nonshared environmental path between peer and individual risk-taking ($bE1' = -.48$, $p < .01$). The negative interaction term indicated that "peer influence"—the within-twin pair association between peer risk-taking and a girl's own risk-taking—was stronger for girls with earlier age at menarche. In addition, later age at menarche was associated with decreased shared environmental influences ($C_{risk}' = .40$, $p < .01$) on risk-taking. In sum, results support the hypothesis that girls with earlier pubertal timing, as defined by age at menarche, are more susceptible to peer influence on risk-taking.

Peer comparison. The nonshared environmental pathway between peer and individual risk-taking was moderated by the peer comparison measure of pubertal timing ($bE1' = .46$, $p < .05$). The within-twin pair association between peer and individual risk-taking was stronger for girls who perceived themselves as more developed than their peers. The genetic and shared environmental pathways, which represented selection effects, were also moderated by peer comparison, in the opposite direction; this was indicated by negative interaction terms on these paths ($bA1' = bC1' = -.09$, $p < .01$). Thus, it appeared that for girls who perceived themselves as more mature than their peers, the association between peer and individual risk-taking was due less to selection and more to socialization. The peer comparison measure of timing also moderated the unique genetic and environmental variance in risk-taking (i.e., residual variance in risk-taking that was not shared with either pubertal timing or peer risk-taking). Genetic influence on risk-taking was suppressed among girls with earlier pubertal timing ($A_{risk}' = -.30$, $p < .05$), as was nonshared environmental influence ($E_{risk}' = -.12$, $p < .05$). Both of these interaction effects were in the same direction, indicating that there is less overall residual variance in risk-taking (independent of puberty and peers) among early-maturing girls.

Self-rated body changes. Unlike age at menarche and peer comparison, self-rated body changes did not appear to moderate the quasi-causal path between peer and individual risk-taking ($bE1' = .04$, $p = .79$). Thus, there was no evidence that associations between peer and individual risk-taking were moderated by age-standardized ratings of body changes.

Sensitivity analyses. We conducted three sets of sensitivity analyses to determine whether the broad age range of our sample impacted our main findings for moderation. First, to address the possibility that the peer comparison measure confounded pubertal status and timing, particularly

TABLE 3
Unstandardized Parameter Estimates From Best-Fitting Interaction Models of Pubertal Timing, Peer Risk-Taking, and Target Risk-Taking

	Age at Menarche	*Peer Comparison*	*Body Changes*
Regression parameters			
Peer risk-taking and target risk-taking			
Main genetic path (bA1)	.25**	.49**	.19**
Genetic path × Puberty interaction (bA1′)	.03	−.09**	.01
Main shared environmental path (bC1)	.33**	.49**	.19**
Shared environment × Puberty interaction (bC1′)	.03	−.09**	.01
Main nonshared environmental path (bE1)	−.24	−1.61*	−.25
Nonshared environment × Puberty interaction (bE1′)	−.48**	.46*	.04
Pubertal timing and target risk-taking			
Main genetic path (bA2)	.59*	−.64*	.10
Gene × Puberty interaction (bA2′)	−.01	.07	−.04
Main nonshared environmental path (bE2)	.52**	−.10	−.06
Nonshared environment × Puberty interaction (bE2′)	.01	−.09	.04
Pubertal timing and peer risk-taking			
Main genetic path (bA3)	−.01	.11	.11
Gene × Puberty interaction (bA3′)	0	−.02	−.03
Main nonshared environmental path (bE3)	−.05	−.15	−.02
Nonshared environment × Puberty interaction (bE3′)	.01	.04	.06
Variance in pubertal timing			
Main effect of genes (A_{pub})	1.11**	.75*	.64
Main effect of nonshared environment (E_{pub})	.92**	.69*	.77
Variance in target risk-taking			
Main effect of genes (A_{risk})	−.66**	1.07	.50
Gene × Puberty interaction (A_{risk}')	.21	−.30*	.01
Main effect of shared environment (C_{risk})	−.06	1.45**	.52*
Shared environment × Puberty interaction (C_{risk}')	.40**	−.26	.32
Main effect of nonshared environment (E_{risk})	.62*	1.03**	.64**
Nonshared environment × Puberty interaction (E_{risk}')	−.02	−.12*	−.14**
Variance in peer risk-taking			
Main effect of genes (A_{peer})	0	0	.23
Gene x puberty interaction (A_{peer}')	0	0	−.02
Main effect of shared environment (C_{peer})	.17	0	0
Shared environment × Puberty interaction (C_{peer}')	−.029	0	0
Main effect of nonshared environment (E_{peer})	.34**	.16*	.32**
Nonshared environment × Puberty interaction (E_{peer}')	−.04	.05*	.09**

Note. N = 248 twin pairs.
*$p < .05$; **$p < .01$.

for younger girls, we standardized this measure by age in years (as we had done with self-rated body changes). Findings did not change using this age-standardized measure (bE1′ = .72, $p < .001$). Next, following previous studies using Add Health puberty data (Ge et al., 2007; Halpern, King, Oslak, & Udry, 2005), we limited our sample to adolescent ages 12–17 (174 twin pairs) and reran analyses of peer comparison and self-rated body changes. For peer comparison, the moderating effect was of similar magnitude, but the standard errors were larger and the coefficients were at the threshold of conventional statistical significance (bE1′ = .55, $p = .05$). Self-rated body changes did not moderate peer influence in this younger sample (bE1′ = −.08, $p = .72$). Next, we restricted the age range to adolescent ages 14–19 (212 twin pairs). Again we found a significant moderating effect for peer comparison (bE1′ = .93, $p < .001$) and not for self-rated body changes (bE1′ = .18, $p = .34$). Finally, we reran moderation analysis for age at menarche, limiting the sample to girls who had started menstruating by Wave I and found a moderating effect in this restricted sample (bE1′ = −.53, $p < .01$).

In a final set of analyses, we examined whether the moderation effect was driven by racial or ethnic differences in pubertal timing. We ran two additional models, one including dummy-coded race (African American vs. nonAfrican American) as a moderator and one including ethnicity (Hispanic vs. non-Hispanic) as a moderator. There were no moderating effects of race (bE1′ = −.14,

$p = .79$) or ethnicity ($bE1' = -.35$, $p = .79$) on the quasi-causal association between peer and individual risk-taking. Thus, the moderating effects of pubertal timing could not be explained by racial or ethnic differences in pubertal timing.

DISCUSSION

The current study used a quantitative behavior genetic design to examine whether girls' pubertal timing moderated the quasi-causal association between peer risk-taking and individual risk-taking. Results suggested girls with earlier ages at menarche and girls who perceived themselves to be more physically developed than their peers were more susceptible to peer influence on risk-taking. These findings align with previous studies showing that pubertal timing moderates the adverse effects of deviant peer affiliation (e.g., Costello et al., 2007; Fergusson et al., 2007). This pattern is consistent with the "contextual amplification hypothesis" (Ge et al., 2002; Stattin et al., 2011), which proposes that earlier pubertal maturation may magnify the effects of contextual factors such as peers, neighborhoods, schools, and families. These findings are also congruent with recent behavior genetic research that has found that more of the variation in delinquency can be attributed to environmental differences among girls with early pubertal timing (Burt et al., 2006; Harden & Mendle, 2012). To our knowledge, no other studies have used quantitative genetic methods to examine pubertal timing as a moderator of the relation between peer-reported and self-reported delinquency.

The age range of this sample (mean age = 16 years) has important implications for interpreting results. A sample of middle adolescents is ideal for studying risk-taking, which increases markedly during this period and is a leading cause of morbidity and mortality for this age group (CDC, 2009). Yet by middle adolescence, most girls are "postpubertal" (or at least postmenarcheal). Thus, we are not necessarily examining the effects of earlier pubertal timing during the period when most of the physical changes of puberty are occurring. Rather, these effects represent the persisting effects of individual differences in pubertal timing into later adolescence. We are not the first to find that measures of pubertal timing have predictive power beyond the age at which girls are initiating puberty (Ge et al., 2007; Graber et al., 1997; Haynie, 2003). Indeed, some have suggested that studies of older adolescents are necessary to clarify the effects of pubertal timing; as Angold and Costello (2006) noted, "it is not until after puberty, when everyone is fully mature, that the timing of any pubertal event is unambiguously unconfounded" with pubertal status (p. 924). These associations nevertheless pose a developmental enigma: Why does early age at menarche still matter if everyone is postmenarcheal? What does it mean for a girl to "look older" (or think she looks older) than her peers if she and they would likely all be considered reproductively mature on a Tanner stage scale? Our conjecture is that pubertal events occurring in late childhood and early adolescence have relatively enduring effects on girls' self-perceptions and that these differences in self-perception represent an important mechanism for how effects of pubertal timing persist into later adolescence.

There were two subjective measures used in this study: perceived body changes standardized by age, and a peer comparison measure. Perceived development compared to one's peers moderated the association between peer and individual risk-taking, whereas perceived body changes did not. There are several possible explanations for these discordant findings. These subjective measures (body changes and peer comparisons) reflect different aspects of pubertal timing and probably other constructs as well. The peer comparison measure ("how advanced is your physical development compared to other girls your age?") likely reflects characteristics of an adolescent's reference group—the group of "other girls your age" that forms the basis for self-comparison. In addition, the response options to this item are somewhat ambiguous, ranging from "I look younger than most" to "I look about average" to "I look older than most." Many other factors besides physical development contribute to one's apparent age, including attempts to manipulate apparent age with clothing, makeup, or tattoos. Thus, this measure of peer-normative pubertal timing, while correlated with age at menarche and breast development, also taps constructs such as perceptions of peers and impression management, which may be closely linked with susceptibility to peer influence.

The lack of moderation by self-rated body changes suggests that a girl's perception of how her body has changed may be less influential—at least on susceptibility to peer influence on risk-taking—than her perception of how her body differs from her peers. This pattern of results—in which significant effects were observed for age at menarche and peer comparison but not body changes—was also observed in a study of dieting in adolescence (Harden, Mendle, & Kretsch, 2011). It is also possible that ratings of body and breast development are confounded by body type and weight.

This may be particularly true of the Add Health participants, many of whom were in mid-adolescence and postmenarcheal at the start of the study. Therefore, a response of "my body is a lot more curvy than when I was in grade school" may be more indicative of one's body type than of one's pubertal stage.

A commonly proposed explanation for the link between pubertal timing and risk-taking—whether it is conceptualized in terms of mediation or moderation—is that girls who experience early pubertal timing and who appear older than their same-age peers affiliate with older, more male-dominated peer groups. Because they are younger in chronological age, they may rely more on their older peers for opportunities to engage in risky behavior. However, current findings are unlikely driven solely by the age and gender of one's friends, because phenotypic correlations between pubertal timing and peers' age and gender showed no significant associations. In addition, the moderating effects of pubertal timing found in the current study were not driven by racial or ethnic differences in pubertal timing. However, given the comparatively small number of racial and ethnic minority twin pairs in Add Health, and the disproportionate number of Hispanic girls with no identifiable peer nominations, we did not have sufficient power to test a three way interaction (Peers × Race and ethnicity × Pubertal timing). The fact that Hispanic girls had fewer identifiable peer nominations suggests there may be qualitative and quantitative ethnic differences in peer group composition. There may be important racial and ethnic differences in the relation between pubertal timing and peer influence that are not captured in our study. Cavanagh (2004) found an interaction between peer academic achievement and pubertal timing that predicted age at first sex in Hispanic girls only and found no significant associations between friendship group characteristics, pubertal timing, and age at first sex for African American girls. Understanding the extent to which race and ethnicity may impact susceptibility to peer influence via differences in pubertal timing remains an important question of future study.

LIMITATIONS

Our findings should be interpreted in light of several limitations. First, the sample size is small compared to a typical twin sample, which is important given the complexity of the models tested in this study. Power was also limited due to the substantial portion of missing data on peer risk-taking. Second, other characteristics of the study sample limit the extent to which we can generalize these findings. It is possible that different associations would be found in a higher risk sample or in a sample of males, who engage in more risk-taking on average. Research on pubertal timing in males is limited, likely due to the lack of a "male equivalent" to females' age at menarche, but recent studies suggest pubertal timing has important effects on psychosocial outcomes in males (reviewed in Mendle & Ferrero, 2012) and that peer relationships are key mechanisms for the psychosocial impact of pubertal development in males (Mendle, Harden, Brooks-Gunn, & Graber, 2012). In particular, a recent longitudinal study using data from Add Health examined pubertal timing (measured by age-standardized ratings of body changes), alcohol use, and peer alcohol use and found an interaction between pubertal timing and peer alcohol use that was *only* present among males (Biehl et al., 2007). Further exploration of these gender differences, using longitudinal quasi-experimental designs and peer-reported risk-taking, is warranted. Indeed, given current evidence that the subjective peer comparison measure taps a meaningful psychological construct, the absence of a more objective pubertal milestone should not hold us back from exploring similar questions about puberty, peers, and risk-taking in males.

Third, because the current study used cross-sectional data, the direction of peer influence is not clear. We arbitrarily designated the twin girls in this sample as targets of influence, but it is also likely that they are sources of influence. Thus, an additional explanation of our findings is that girls with earlier pubertal timing exert more influence on their peers. These dual interpretations are not necessarily mutually exclusive. Research on peer influence susceptibility relies on a distinction between "leaders" and "followers" (Allen, Porter, & McFarland, 2006), but it is plausible that individuals oscillate between these roles as they navigate the social transition of adolescence.

Finally, the current study used social network data but did not involve formal social network analysis or statistically control for peer groups nested within schools. Social network analytic methods offer a chance to model selection and influence simultaneously (Snijders, Steglich, & Van de Bunt, 2010), and, when applied to genetically informative longitudinal data, these tools can answer important questions about susceptibility to peer influence. The fact that all twins (both MZ and DZ) were selecting peers from the same pool inflates

shared environmental selection effects over genetic selection effects. It also provides a rigorous quasi-experimental test of peer influence: twins are matched on age and grade and have equal opportunity to select friends from the same pool of grademates.

CONCLUSION

The preponderance of research on peer influence suggests that both selection and socialization shape adolescent behavior. Using a behavioral genetic model of peer influence, this study suggests both processes are moderated by pubertal timing. In recent years, early pubertal timing has been the focus of both scientific study and popular media (e.g., Weil, 2012), particularly given observed trends in earlier pubertal development. These findings highlight the importance of considering the peer context in which biological changes occur and of using multiple measures of pubertal timing. Key areas for research include exploration of gender, race, and ethnicity as additional moderators of peer influence and incorporating longitudinal social network methods.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Table S1. Additional descriptive statistics for peer comparison measure of pubertal timing ($N = 496$ individuals).