The Journal of Early Adolescence

http://jea.sagepub.com/

Pubertal Development and Peer Influence on Risky Decision Making

Natalie Kretsch and Kathryn Paige Harden

The Journal of Early Adolescence published online 24 June 2013

DOI: 10.1177/0272431613489373

The online version of this article can be found at:

http://jea.sagepub.com/content/early/2013/06/21/0272431613489373

Published by:

SAGE

http://www.sagepublications.com

Article

Journal of Early Adolescence
XX(X) 1–21

DOI: 10.1177/0272431613489373
jea.sagepub.com

Pubertal Development and Peer Influence on Risky Decision Making

Natalie Kretsch[1] and Kathryn Paige Harden[1]

Abstract

Adolescents engage in more risky behavior when they are with peers and show, on average, heightened susceptibility to peer influence relative to children and adults. However, individual differences in susceptibility to peer influence are not well understood. The current study examined whether the effect of peers on adolescents' risky decision making was moderated by pubertal status. Participants (58 youth, ages 11-16, 50% male, 63.9% African American) completed a computerized measure of risky decision making, once alone and once in the presence of two peers. Pubertal status was assessed using self-report. Adolescents made riskier decisions in the presence of peers, and more advanced pubertal development predicted greater risky decision making, controlling for chronological age. The effect of peer presence on risky decision making was attenuated for adolescents with more advanced pubertal development. These findings suggest that the presence of peers may override biologically based individual differences in propensity for risk taking.

Keywords

decision making, peers, puberty/pubertal development, problem/risky/antisocial behaviors

[1]University of Texas at Austin, Austin, TX, USA

Corresponding Author:
Natalie Kretsch, Department of Psychology, University of Texas at Austin, 108 E. Dean Keeton Stop A8000, Austin, TX 78712-1043, USA.
Email: nkretsch@mail.utexas.edu

Adolescents show elevated involvement in an array of risk-taking behaviors, including criminal behavior, unsafe sexual practices, and initiation of alcohol use (Arnett, 1992; Jessor & Jessor, 1977; Steinberg, 2008). The consequences of adolescent risk-taking behaviors are profound: For example, mortality from unintentional injury (including motor vehicle accidents) peaks in adolescence, accounting for over 40% of deaths among 10- to 24-year-olds (Ozer & Irwin, 2009). Not surprisingly, then, researchers have paid considerable attention to the biological and social changes in adolescence that result in a developmental period of unique psychological vulnerability.

Adolescents are more likely to engage in risky behavior when they are with their peers, and affiliation with risk-taking peers is a robust predictor of individual risk taking. Adolescents report engaging in more substance use (Chassin, Hussong, & Beltran, 2009), criminal activity (Zimring, 1998), and risky driving (Ouimet et al., 2010) when they are with their peers. This trend reflects both peer influence, in which adolescents reinforce each others' risky behavior, and peer selection, in which adolescents with higher propensity for risk taking gravitate toward similarly risk-inclined peers. There are also individual differences in susceptibility to peer influence, and researchers are only beginning to explore the possibility that biological factors moderate peer influence on risk taking. Specifically, peer influence and selection operate in the context of profound biological changes that occur during puberty, and it is unclear how peer influence, selection, and risk taking interact with pubertal development. The current study used an experimental design to test whether adolescents make riskier decisions in the presence of peers and whether this effect was moderated by pubertal development.

Research on the role of peers in shaping adolescent behavior has sought to disentangle peer influence (i.e., socialization) from selection of similar peers (also known as homophily). Regarding socialization processes, several explanations for increased conformity to peers in adolescence have emerged from social psychology research. Adolescence is characterized by a dramatic social reorientation toward valuing peer, romantic, and sexual relationships (Blakemore, 2008; Forbes & Dahl, 2010; Larson & Richards, 1991; Spear, 2009). Social learning (e.g., Bandura, 1986) and deviancy training (e.g., Dishion, Spracklen, Andrews, & Patterson, 1996) may lead adolescents to engage in health risk behavior that they observe or experience to be socially rewarding. Adolescents who associate certain health risk behaviors with high social status are more likely to adopt these behaviors (Cohen & Prinstein, 2006). Adolescents may receive peer reinforcement for talk about antisocial behavior, and this process is associated with increases in risky behavior (Dishion et al., 1996). Evidence for peer selection is as extensive as evidence for peer influence. Behavior genetic studies suggest that at least part of the correlation between an individual's risk taking and his or her best friend's risk taking is due to an individual's genetically influenced characteristics, which underlie both propensity for risk taking and selection of risk-taking peers (Harden, Hill, Turkheimer, & Emery, 2008; Hill et al., 2008). Analysis of longitudinal social network data provides compelling evidence for both selection and socialization in adolescent use of alcohol, tobacco, and other drugs (Cruz, Emery, & Turkheimer, 2012; Ennet & Bauman, 1994; Mercken, Snijders, Steglich, Vartiainen, & de Vries, 2010) and delinquency (Burk, Steglich, & Snijders, 2007; Moody, 2001). In recent years, sophisticated quantitative tools have allowed researchers to model selection and socialization simultaneously, by examining both changes in friendship nominations and changes in individuals' and peers' behavior over time (Snijders, Steglich, & Van de Bunt, 2010).

Other researchers have used experimental approaches to control for selection effects and to examine whether simply being in the presence of peers leads adolescents to make riskier decisions. Using an experimental approach that assessed risk taking with a behavioral task, Gardner and Steinberg (2005) found that adolescents (but not adults) who were randomly assigned to complete the experiment with two same-sex peers observing made riskier decisions than those who completed the study alone. In a follow-up study using fMRI, Chein, Albert, O'Brien, Uckert, and Steinberg (2011) found that adolescents showed increased activity in reward-related brain regions (ventral striatum and orbitofrontal cortex) when making behavioral decisions in the presence of peers, compared to when they were alone. Moreover, individual differences in the "neural peer effect" (i.e., the difference in striatal activity in the peer vs. the alone conditions) were significantly associated with self-reports of resistance to peer influence. These findings suggest a biological mechanism for peer influence on risk taking and, more generally, highlight the importance of considering biological factors that may mediate and moderate the effect of peer presence on decision making.

In particular, it is important to consider how the influence of peers may interact with biological changes that adolescents undergo at puberty. The current study examined whether self-reported pubertal timing moderated the effect of peer presence on adolescents' risky decision making. Pubertal timing itself is a robust predictor of risk taking in adolescence. A large body of cross-sectional, longitudinal, and behavior genetic research has found that earlier pubertal timing is associated with increased involvement in delinquency and substance use in both girls (reviewed in Mendle, Turkheimer, & Emery, 2007) and boys (reviewed in Mendle & Ferrero, 2012). Most of the research on pubertal timing has focused on girls' menarcheal age and has emphasized the unique environmental challenges faced by early maturing girls. However, recent genetically informed research has suggested that, for girls, the association between earlier pubertal timing and delinquent behavior, specifically, can be attributed to biological rather than environmental mechanisms (Harden & Mendle, 2012). Moreover, associations between earlier pubertal timing and risk-taking behaviors are also evident in boys (Bratberg, Nilsen, Holmen, & Vatten, 2007; Duncan, Ritter, Dornbusch, Gross, & Carlsmith, 1985; Tschann et al., 1994) although male pubertal development has been the focus of considerably less research.

In both males and females, the biological basis for the relation between pubertal timing and risky behavior may lie in the neurological changes that coincide with pubertal onset. According to the Dual Systems Model (Casey, Getz, & Galvan, 2008; Somerville, Jones, & Casey, 2010; Steinberg et al., 2008), increased propensity for risk taking in adolescence results from the temporal gap in the development of two neurological systems, one of which is more closely linked to pubertal development than to chronological age. The *cognitive control system*, which includes prefrontal cortical regions and their connections to subcortical areas, governs self-regulatory processes such as impulse control and develops gradually throughout adolescence and young adulthood (Casey et al., 2008; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999). In contrast, the *socioemotional system*, which comprises dopaminergic pathways in limbic and paralimbic regions, governs responses to novelty, emotion, and reward and matures relatively rapidly during early adolescence (Nelson, Leibenluft, McClure, & Pine, 2005; Steinberg, 2008). The maturation of the socioemotional system results in heightened sensitivity to the potential rewards of risky activity. The gap between the arousal of the socioemotional system and the relatively slower maturation of the cognitive control system is thought to result in a period of elevated vulnerability to risk taking during middle adolescence (Casey & Jones, 2010; Chambers, Taylor, & Potenza, 2003; Galvan et al., 2006; Spear, 2011; Steinberg, 2008).

Development of the socioemotional system, and the corresponding increases in sensation seeking and reward sensitivity, may be more closely tied to pubertal development than to chronological age (Martin et al., 2002; Nelson et al., 2005; Steinberg, 2008; Steinberg et al., 2008; Zuckerman, Buschbaum, & Murphy, 1980). In addition to causing extensive somatic changes, the hormonal events at puberty also precipitate a cascade of neural changes—"a second period of structural reorganization and plasticity in the brain" (Blakemore, Burnett, & Dahl, 2010, p. 927). Specifically, the release of gonadal hormones and remodeling of gonadal steroid receptors in the limbic system may lead to changes in emotional responses to social stimuli (Nelson et al., 2005; Steinberg, 2008). Gonadal hormones may also exert indirect effects on the socioemotional system through their regulation of other neurotransmitter systems, including the dopamine, oxytocin, and opioid systems (McEwen, 2001; Nelson et al., 2005). Consistent with this hypothesis, pubertal status—controlling for age—is associated with sensation seeking (Martin et al., 2002; Steinberg et al., 2008; Zuckerman et al., 1980). In addition, among early adolescents, more advanced pubertal development is associated with a greater postauricular reflex (a measure of appetitive motivation) in response to pleasurable visual stimuli (Quevedo, Benning, Gunnar, & Dahl, 2009), greater amygdala responding to threatening social stimuli (angry human faces; Forbes, Phillips, Ryan, & Dahl, 2011), and less activity in the striatum and greater reactivity in the medial prefrontal cortex in response to monetary rewards (Forbes & Dahl, 2010). Given the link between pubertal development and activity in the socioemotional system, the "size" of the maturity gap between the socioemotional and cognitive control systems—and the resulting propensity for risk-taking behavior—may be determined by pubertal timing. That is, adolescents who experience the physical changes of puberty at an earlier chronological age may be *more* sensitive to the rewarding aspects of risk-taking behavior, without any corresponding increase in their capacities for impulse control.

How individual differences in pubertal timing intersect with peer influence on risk taking is unclear. On the one hand, puberty may sensitize adolescents to the effects of peers. Forbes and Dahl (2010) suggested that the social reorientation of adolescence was driven, at least in part, by the hormonal events of puberty. Tangential evidence for this hypothesis comes from Gardner and Steinberg (2005) who found the effect of peer presence on risk taking was greater among non-White adolescents—who have, on average, earlier pubertal timing than White adolescents. In addition, Harden and Mendle (2012) found that nonshared environmental influences on delinquency (which included nonfamilial influences such as peers) were more pronounced for earlier maturing girls than later maturing girls. On the other hand, pubertal status may interact with peer presence in the opposite direction. The presence of peers may override individual differences in propensity for risk taking that are due to pubertal timing. An early-maturing adolescent who is high in sensation-seeking and generally inclined to take risks may take fewer risks in the presence of peers if their peers implicitly (i.e., through modeling) or explicitly (i.e., through verbal advice and feedback) discourage risky decision making. The "social push" hypothesis posits that biological influences on delinquent behavior are less apparent in contexts with strong social pressure (Raine, 2002). This hypothesis may also apply to risky decision making in general, such that individual differences in propensity for risk taking due to pubertal development may not be expressed in the presence of peers. This question has not yet been examined empirically.

Goals of the Current Study

The current study examined the associations between pubertal development, peer presence, and risky decision making on an experimental task. In particular, this study tested three main hypotheses. First, in accordance with previous experimental findings (Chein et al., 2011; Gardner & Steinberg, 2005), adolescents were expected to make riskier decisions in the presence of same-age peers than when alone. Second, after controlling for chronological age, adolescents with more advanced pubertal development were expected to show more risky decision making than less developed adolescents. This prediction follows from the Dual Systems Model and the association between puberty and activity in the socioemotional system. Third, we hypothesized an interaction between peer presence and pubertal status, in one of two directions: The relationship between pubertal status and risk taking would be either heightened or suppressed in the presence of peers. Because there is theoretical support for both of these interactions, we made no directional hypothesis about this potential moderating effect.

Method

Participants

Participants were 58 youth (50% male), ages 11 to 16 (mean age = 13.6, *SD* = 1.67) who attended after-school programs in a metropolitan area in the southwest United States. The sample was predominantly African American (63.9%) and Hispanic (19.7%). Seventy-six percent of participants reported receiving free or reduced-price lunch at their school, for which children are eligible if their family income is at or below 185% of the federal poverty line.

Recruitment and Procedure

The recruitment procedure used in the current study was similar to that used by Gardner and Steinberg (2005). All recruitment and data collection occurred on-site at two afterschool club locations. During the first week of data collection, investigators distributed parental consent forms to all youth ages 11 to 16 and obtained parental consent from large numbers of adolescents at both sites (Gardner & Steinberg, 2005). As consent forms were returned, a list was compiled of interested participants and their ages and gender. Adolescents who brought back signed parental consent forms were assigned to a group with two peers of the same age (within 1 year) and gender. Because attendance at the clubs was variable and unpredictable, groups were formed on a daily basis, based on which adolescents were at the club at the time of data collection. Each day, the researchers arrived on site and determined which of the children on the list were on site. Groups were formed based on age. For example, if seven 12-year-old children were present that day, the first three 12-year-olds on the list would be grouped together, the second three would be grouped together, and the last one on the list would move ahead on the list. Although the participants in each group were not always close friends (they were not self-selected), all participants knew one another. This method of group assignment was consistent with the procedure used by previous studies (Gardner & Steinberg, 2005) and has the advantage of simulating the context in which adolescent risk taking often occurs—adolescents are often with same-gender peers who are not necessarily close friends.

Participants were told that they were participating in a study of "how kids and teenagers make decisions." Each participant completed a simulated driving task designed to measure risky decision making twice—once individually (Alone condition) and once while being observed by his or her two peers (Peer condition). In the Peer condition, a "round robin" design was used, in which members of the triad took turns completing and observing the task (Participant 1 played the game while Participants 2 and 3 observed, Participant 2 played the game while Participants 1 and 3 observed, etc.). The peer observers were permitted to talk to the player and to each other during the task and could give the player advice on how to proceed. Dialogue during the Peer condition was not recorded or transcribed, but it was noted that for every triad, peers did give explicit advice on how to proceed ("Keep going!" or "Stop!") and expressed disappointment when crashes occurred. The player could choose whether or not to follow the advice of the observing peers. To control for learning effects, groups were counterbalanced for the order in which the behavioral tasks were completed, with half the groups completing the task in the Alone condition first and half completing it in the Peer condition first. Within the Peer condition, the order in which the participants completed the task was also recorded. In addition to the risky driving task, a working memory test was administered by trained research assistants. Sixty adolescents, clustered into 20 triads, participated initially. Two adolescents did not complete the puberty self-report measures and were excluded from subsequent analyses. The experimental protocol took between 60 and 90 minutes. All participants were compensated US$25 for their time.

Measures

Pubertal status. Pubertal status was assessed with a modified version of the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988), a widely used self-report measure of perceived pubertal changes. The PDS includes five items asking about skin, height, underarm and pubic hair, breast development (for females), menarche (for females), voice changes (for males), and facial hair (for males). Participants rated each measure with the following scale: 1 = *Has not yet begun to change*, 2 = *Has barely started*, and 3 = *Is definitely underway*. Participants could also select *I Don't Know*; Items rated *I Don't Know* were treated as missing.

Pubertal development is triggered by two distinct hormonal release signals—the early release of adrenal hormones (adrenarche) and the later release of gonadal hormones (gonadarche). These two release signals are independent (Auchus & Rainey, 2004; Grumbach & Styne, 2003), and researchers have emphasized the importance of distinguishing between these two separate maturational processes (McClintock & Herdt, 1996). The timing and hormonal events that characterize adrenal development are the same for both sexes; in contrast, gonadal development occurs earlier in females and involves different hormones (McClintock & Herdt, 1996). For both sexes in the current study, the mean adrenal score was computed based on skin changes and pubic hair development. The mean adrenal score was 2.35 for females ($SD = .60$) and 2.22 for boys ($SD = .49$). The mean gonadal score was computed based on growth spurt, breast development, and menarche for females and on growth spurt, voice changes, and facial hair for males. The mean gonadal score was 2.45 for females ($SD = .57$) and 2.16 for boys ($SD = .52$, range). Scores on both scales ranged from 1, indicating that pubertal development had not yet started, to 3, indicating that development was underway. Because in the current study, males and females were aggregated in a single group, we used the adrenal score as the primary measure of pubertal development for these analyses. The PDS has been shown to have high reliability ($\alpha = .77$ for boys, $\alpha = .81$ for girls; Shirtcliff, Dahl, & Pollak, 2009). PDS scores have been shown to predict basal hormone levels (estradiol, testosterone, and DHEA) as well as physical examinations (Shirtcliff et al., 2009). Adrenal scores and gonadal scores were highly correlated with each other ($r = .43$, $p < .001$) and with chronological age (adrenal score and age: $r = .29$, $p = .03$; gonadal score and age: $r = .23$, $p = .08$).

Risky decision making. Risky decision making was assessed using *The Stoplight Game* (Chein et al., 2011; Gardner & Steinberg, 2005; Steinberg et al., 2008), a simulated driving task in which the player "drives" a car along a straight track, trying to reach a specified location in under 5 minutes. The game was played on a laptop computer and was set up from the driver's point of view. The car passed through 20 intersections, each of which had a stoplight that cycled from green to yellow to red as the vehicle approached it. When the light turned yellow, the player decided whether to stop, which resulted in a short delay, or to go through the intersection and risk a "crash" with another vehicle, which resulted in a longer delay. The timing of the traffic signals and the probability of a crash were varied—at some intersections, not braking in time inevitably resulted in a crash, while at others, it was possible to drive through safely. Each intersection had three possible outcomes: (a) the player applied the brakes and safely stopped, (b) the player went through the intersection successfully, or (c) the player crashed into another car, which could result from either failure to brake at all or failure to brake in time to avoid the crash. The computer recorded the total time to complete the task, the outcome at each intersection, and the latency between the appearance of the yellow light and the application of the brakes. The current study used two primary outcome variables: Percentage of Risky Decisions, which was percentage of intersections at which the player did not stop, and Latency to Brake, which was the average time between the appearance of the yellow light and the application of the brakes.[1] The mean Percentage of Risky Decisions was 30% (*SD* = .18); the mean Latency to Brake was 1676.75 ms (*SD* = 465.92).

Working memory. Working memory was assessed using a digit span memory test similar to the Wechsler digit span subtest (forward and backwards digits). Participants were asked to recall digit sequences of increasing length. The total number of sequences that were accurately recalled was used as an index of working memory (maximum possible score 26). The mean number of correctly recalled sequences was 13.11 (*SD* = 2.84). Working memory scores were included as a covariate in all analyses to be consistent with previous published research using the Stoplight Game (e.g., Steinberg et al., 2008). Controlling for working memory allowed us to assess the effects of peers on variance in decision making that was independent of more basic cognitive processes.

Analytic Plan

The current study aimed to assess three main hypotheses: (1) Adolescents would make more risky decisions when they were with peers (main effect of experimental condition); (2) Adolescents with more advanced pubertal development (controlling for chronological age) would make more risky decisions than less developed adolescents (main effect of pubertal status); and (3) Peer presence would moderate the relationship between pubertal status and risky decision making (condition × status interaction).

Table 1. Predictors of Risky Decision Making.

	Outcome	
Predictor	Percent risky decisions	Latency to brake (ms)
Intercept	−15.17 (21.81)	286.52 (579.21)
Condition (0 = *Alone*/1 = *Peer*)	16.20 (10.03)	569.65 (254.82)*
Age	.63 (1.30)	22.94 (34.72)
Gender (0 = *Female*/1 = *Male*)	−3.08 (4.40)	−60.32 (118.13)
African American (0 = *No*/ 1 = *Yes*)	8.51 (5.57)	205.64 (146.05)
Hispanic (0 = *No*/1 = *Yes*)	−6.39 (5.68)	−127.54 (149.52)
Working memory (Digit span score)	.88 (.85)	16.79 (22.39)
First time playing (0 = *No*/ 1 = *Yes*)	8.14 (2.38)**	100.85 (60.50)
Pubertal status[a]	8.47 (4.32)	311.48 (112.44)*
Condition × Pubertal status[a]	−7.33 (4.26)	−256.34 (108.16)*

Note. Values are parameter estimates (standard error).
[a]Pubertal status is based on Mean Adrenal Score.
*$p < .05$. **$p < .01$.

Analyses were performed using R version 2.11.1. The Linear Mixed Model (LMM) procedure was used to account for multiple observations for individuals and for individuals clustered within groups. Separate analyses were conducted for each outcome variable (Percentage of Risky Decisions and Latency to Brake) and for each measure of pubertal development (adrenal and gonadal). In addition, for each analysis, age and working memory were entered as continuous predictor variables, and gender, race, ethnicity, and condition order (whether the participant was completing the task for the first time) were entered as fixed factors.

Results

Results from the LMM analyses, using mean adrenal score as the measure of pubertal development, are shown in Table 1. For Latency to Brake, a main effect of condition was found, $t(49) = 2.24$, $p < .05$; adolescents in the Peer condition had greater latency to brake, indicating riskier decision making among peers, as predicted. A main effect for adrenal pubertal status was also found. More advanced pubertal development predicted riskier decisions, $t(27) = 3.13$, $p < .05$. There was a Status × Condition interaction,

$t(49) = -2.39$, $p < .05$. As shown in Figure 1, the association between pubertal development and risky decision making was only apparent in the Alone Condition.

In contrast, for the Percentage of Risky Decisions, we found no significant main effect for condition, $t(49) = 1.61$, $p = .11$. The main effect for pubertal status approached significance, with more advanced development predicting riskier decision making, $t(27) = 1.96$, $p = .06$. There was no significant Status × Condition interaction, $t(49) = -1.72$, $p = .09$. There was also evidence that adolescents became less risky the second time they attempted the task, as they made more risky decisions when they completed the task for the first time, $t(49) = 3.42$, $p < .01$).

Secondary analyses were conducted using the gonadal measure of pubertal development, yielding slightly different results. For Latency to Brake, there were no main effects of condition, $t(49) = 1.04$, $p = .30$, or status, $t(27) = 1.80$, $p = .08$, and no Status × Condition interaction, $t(49) = -1.17$, $p = .25$. For the Percentage of Risky Decisions, there was a main effect for pubertal status, with more developed adolescents making more risky decisions, $t(27) = 2.06$, $p < .05$. There were no main effects of condition, $t(49) = .45$, $p = .65$, and no Status × Condition interaction, $t(49) = -.54$, $p = .59$. Again, results showed adolescents made more risky decisions the first time they completed the task, $t(49) = 3.27$, $p < .01$.

These findings showed an inconsistent effect of peer presence. Only one of the four models tested (using the adrenal measure and Latency to Brake as the outcome) showed evidence of increased risk taking in the presence of peers. We hypothesized that adolescents may have been conforming to the behavior of their peers that they observed, such that the direction of peer influence depended on the decision making of the other peers in the triads. A post hoc exploratory analysis was conducted to test this possibility. This analysis focused on the Peer condition and behavior of the participant who completed the task third—that is, after observing two peers complete the task. We examined whether this participant's risk taking in the Peer condition was predicted by the risk taking that he or she observed. Table 2 shows results from linear regression analysis. The outcome was the risk taking of the third participant in the group; predictors included observed risk taking—that is, the risk taking of the first and second participants in the Peer condition—and the third participant's risk taking in the Alone condition. As shown in Table 2, Latency to Brake in the Peer condition for the third participant was predicted not only by his or her Latency to Brake in the Alone condition ($\beta = .63$, $p < .01$) but also by the risk taking he or she observed in the first participant ($\beta = .44$, $p < .01$) and second participant ($\beta = .40$, $p < .01$).

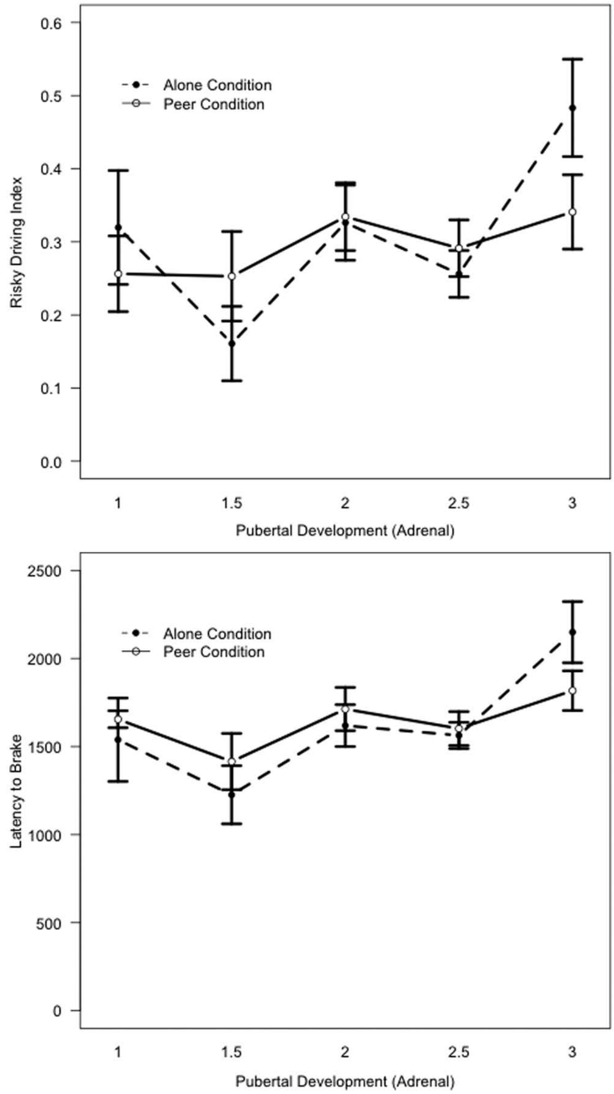

Figure 1. Risky decision making by adrenal pubertal status and experimental condition.

Note. Pubertal Development (Adrenal) reflects mean response to items on the adrenal scale, which range from 1 (*has barely started*) to 3 (*is definitely underway*). Error bars represent 95% confidence intervals.

Table 2. Predictors of Risky Decision Making in the Peer Condition for the Third Participant (*N* = 21).

Predictor	Outcome	
	Percent risky decisions	Latency to brake (ms)
Intercept	–.85 (6.63)	–790.12* (325.36)
Risky decision making in alone condition	60.94 (12.11)**	.63** (.10)
Peer 1's risky decision making	23.96 (12.51)	.44** (.11)
Peer 2's risky decision making	18.41 (11.07)	.40** (.10)

Note. In the Peer condition, the participants took turns playing the Stoplight Game while the two others observed. The third participant was the last participant to play in each group and therefore had observed two peers play the game before attempting it himself or herself.
$^{*}p < .05$. $^{**}p < .01$.

Discussion

The current study examined the influence of pubertal development and peer presence on risky decision making in adolescents using an experimental paradigm. Results partially supported the hypothesis that adolescents took more risks in the presence of peers. In the Peer condition, adolescents took more time to decide whether to apply the brakes than they did when alone. As predicted, results showed that after controlling for chronological age, self-reported pubertal development predicted risky decision making. There was an interaction between pubertal status and peer presence, such that the effect of pubertal status was only apparent in the Alone condition.

This study provides some support for a causal relationship between peer context and increased risk taking, which is consistent with previous experimental, observational, and epidemiological research on peer influence (see Brechwald & Prinstein, 2011, for a review). Peer influence has been investigated using a wide range of methodological approaches, including direct self-report (Steinberg & Monahan, 2007), social network approaches (Snijders et al., 2010), performance-based measures (Allen, Porter, & McFarland, 2006), observed peer interactions (Dishion et al., 1996), and genetically informative studies (Harden et al., 2008), all of which have unique strengths and drawbacks. Experimental designs offer an opportunity to empirically test causal relationships between context and behavior. Using random assignment and direct behavioral measures, researchers can control for selection effects and self-report biases while also approximating the real-world environment in which risk taking occurs.

Notably, the effect of peers and pubertal status on risk taking was only found for one of our outcomes. The percentage of risky decisions was no greater in the Peer condition than in the Alone condition; however, latency between the appearance of a yellow light and the player's application of the brakes was longer. This suggests that peer presence did not significantly influence the ultimate decision that participants made about whether or not to brake; however, in the presence of peers, it took them longer to arrive at these decisions. Previous studies have shown that adolescents and adults make similar judgments about risky driving; however, adolescents take longer than adults to make these judgments (Feenstra, Ruiter, & Kok, 2012). In one recent study, adolescents and adults were asked to judge whether a risky activity (e.g., driving drunk, driving at night without headlights) was a good idea or a bad idea. Results showed that adolescents took longer to judge whether an idea was good or bad (Feenstra et al., 2012). An fMRI study using the same procedure (Baird, Fugelsang, & Bennett, 2005) showed different patterns of neural activity during the decision-making process in adolescents relative to adults, with adolescents employing more effortful reasoning. The current study findings suggest that peer presence influences risk taking in subtle but significant ways. In real world risky situations, particularly those that involve driving, even small differences in decision-making time can have profound consequences. Laboratory studies that attempt to simulate these situations can benefit from using multiple measures of risk taking.

A number of mechanisms may account for the modest effect of peers observed in the current study, and future experimental studies using similar designs could shed further light on these mechanisms. Previous research has distinguished between explicit and implicit peer influence (Harakeh & Vollenbergh, 2012). Explicit influence ("peer pressure"), in which peers express support for behavior, may explain study findings. In the Peer condition in this experiment, participants talked to each other, and observers gave the player advice during the task. This advice, however, did not always support risk taking; sometimes the peer observers would advise the driver to stop at the light. Thus explicit peer influence did not always support increased risk taking. Implicit peer influence, such as observational learning or modeling, is another possible mediator. In the current study two out of the three adolescents in each triad observed the game being played by their peers before playing it themselves. Post hoc analysis suggested that the third participant to complete the task in each group may have modeled the decision making of his or her peers. Our results may reflect social learning; that is, participants observed their peers playing the game and adjusted their own behavior based on these observations, in an effort to maximize chances for winning and/or to conform to the behavior of their peers. Thus the influence of peers may not have necessarily resulted in increased risk taking, but rather in conformity to observed peer behavior. The role of observational learning in adolescent behavior is of considerable interest, given that adolescents frequently observe their peers engage in risky activity and model their own behavior based both on the consequences they observe and their desire to conform regardless of the consequences. The evidence for a homogenizing effect in the current study aligns with how peer influence is conceptualized in longitudinal studies that examine the extent to which an adolescent conforms to the behavior of his or her peers over time. Our post hoc analysis is exploratory, and to better understand the mechanisms underlying peer influence on risky decision making, future experimental studies would benefit from coding adolescent communication during risk analogue tasks.

Results also showed a main effect of pubertal development on risky decision making. Controlling for chronological age, adolescents with more advanced development showed more risk taking than their less developed same-age peers. Adrenal pubertal status predicted increased latency to brake, and gonadal status predicted percentage of risky decisions. The main effect of pubertal status on risky decision making is consistent with the large body of evidence showing higher rates of risk taking in adolescents with more advanced pubertal development (Dick, Rose, Pulkkinen, & Kaprio, 2001; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997). Adolescents—particularly girls—with early pubertal timing show higher rates of delinquency, substance use, and risky sexual activity (Mendle, Turkheimer, & Emery, 2007). Recent research suggests that this may be due to the temporal gap between the development of the socioemotional system, which is linked to pubertal maturation and the cognitive control system (Casey & Jones, 2010; Forbes & Dahl, 2010; Nelson et al., 2005; Spear, 2011; Steinberg et al., 2008). The neurological mechanisms of the behavioral outcomes in this study are unknown; however, the finding that risk taking was associated with pubertal status controlling for chronological age is consistent with this biological model.

This study also found an interaction between the effect of peers on risky decision making and pubertal status, with the effect of pubertal development only apparent when adolescents were alone. These findings are consistent with the social push hypothesis (Raine, 2002). Individual differences in propensity for risk taking, which, in this study, were associated with pubertal timing, may have been suppressed in the presence of peers. Initially developed to explain interactions between socioeconomic status and biological influences on more serious forms of antisocial behavior (Gao, Baker, Raine, Wu, & Bezdjian, 2009; Raine, 2002), this hypothesis may apply to more proximal social influences (such as peer presence) on risky decision making in general. Adolescents who were biologically predisposed to risky behavior may have adjusted their behavior in the presence of peers. Broadly speaking, these findings highlight the importance of examining social context as a moderator of individual differences in decision making.

Findings from this study must be interpreted in light of several limitations. First, as with any laboratory-based behavior analogue measure of risky decision making, the ecological validity of the task used is unclear. Performance on the Stoplight Game was previously found to be correlated with self-reported sensation seeking and risky decision making as well as self-reported resistance to peer influence (Chein et al., 2011; Gardner & Steinberg, 2005; Steinberg et al., 2008), which provides some evidence for convergent validity. However, whether either self-report measures or behavioral analogue measures reflect real-world behavior remains an important question for further inquiry. Second, while the PDS is a well-validated self-report measure, self-report is not the ideal way to assess physical development. More accurate measures include results from physical examinations and hormone samples, neither of which was feasible in the current study. The composition of the sample also presents limitations. The homogenous racial composition of the sample—namely, lack of White participants (5% of participants were White and non Hispanic)—did not allow us to fully examine racial and ethnic differences. It remains uncertain whether these findings would be replicated in a predominantly White sample. The effects of race and ethnicity are of particular interest given racial and ethnic differences in pubertal timing (Sun et al., 2002). This study also did not test whether the familiarity of peers within each triad affected results. Research indicates that friendship quality moderates peer influence, with more positive friendship quality (i.e., support, intimacy, and positive affect) predicting more conformity among peers on a range of behaviors (Brechwald & Prinstein, 2011). To the extent that closer friends wield greater influence, the fact that the peers in the current study were not always close friends provides a more conservative test of peer influence (Gardner & Steinberg, 2005). Understanding how familiarity and general friendship characteristics moderate peer influence is another important objective for future research.

Despite these limitations, this study offers a novel contribution to the scientific investigation of peer influence in adolescence, which has moved well beyond determining whether peers play a role in risk taking and into understanding how, why, and when this socialization process occurs (Brechwald & Prinstein, 2011). These findings suggest that the social context plays an important role in the expression of biologically based individual differences in propensity for risk taking and underscore the importance of considering biological factors—specifically, measures of pubertal timing—in experimental studies of adolescent decision making.

Acknowledgments

The authors acknowledge Dr. Laurence Steinberg at Temple University for providing the computerized tasks used in the current study; the children, parents, and staff at the Boys and Girls Clubs of the Austin Area; and Rachel Polk, Claire Cantu, and Katie Legband for their assistance with data collection.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was supported by Grant R24-HD042849 from the National Institute of Child Health and Human Development to the Population Research Center at the University of Texas at Austin.

Note

1. If the participant did not apply the brakes on a round, a maximum value was imputed for Latency to Brake to reflect the time that the player had the opportunity to brake, but did not. In the case of a crash, this was the time between the yellow light and the crash. Otherwise, this was the time between the yellow light and the beginning of the next round.

References

Allen, J. P., Porter, M. R., & McFarland, C. F. (2006). Leaders and followers in adolescent close friendships: Susceptibility to peer influence as a predictor of risky behavior, friendship instability, and depression. *Developmental Psychopathology*, *18*, 155-172.

Arnett, J. (1992). Reckless behavior in adolescence: A developmental perspective. *Developmental Review*, *12*, 339-373.

Auchus, R. J., & Rainey, W. E. (2004). Adrenarche–physiology, biochemistry and human disease. *Clinical endocrinology*, *60*, 288-296.

Baird, A., Fugelsang, J., & Bennett, C. (2005, April). *What were you thinking? An fMRI study of adolescent decision-making*. Poster presented at the 12th Annual Cognitive Neuroscience Society (CNS) Meeting, New York.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. Englewood Cliffs, NJ: Prentice Hall.

Blakemore, S. J. (2008). The social brain in adolescence. *Nature Reviews, Neuroscience*, *9*, 267-277.

Blakemore, S. J., Burnett, S., & Dahl, R. E. (2010). The role of puberty in the developing adolescent brain. *Human Brain Mapping*, *31*, 926-933.

Bratberg, G. H., Nilsen, T. I., Holmen, T. L., & Vatten, L. J. (2007). Perceived pubertal timing, pubertal status and the prevalence of alcohol drinking and cigarette smoking in early and late adolescence: A population based study of 8950 Norwegian boys and girls. *Acta Paediatrica*, *96*, 292-295.

Brechwald, W. A., & Prinstein, M. J. (2011). Beyond homophily: A decade of advances in understanding peer influence processes. *Journal of Research on Adolescence*, *21*, 166-179.

Burk, W. J., Steglich, C. E., & Snijders, T. A. (2007). Beyond dyadic interdependence: Actor-oriented models for co-evolving social networks and individual behaviors. *International Journal of Behavioral Development*, *31*, 397-404.

Casey, B. J., Getz, S., & Galvan, A. (2008). The adolescent brain. *Developmental Review*, *28*, 62-77.

Casey, B. J., & Jones, R. M. (2010). Neurobiology of the adolescent brain and behavior: Implications for substance use disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, *49*, 1189-1201.

Chambers, R. A., Taylor, J. R., & Potenza, M. N. (2003). Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *American Journal of Psychiatry*, *160*, 1041-1052.

Chassin, L., Hussong, A., & Beltran, I. (2009). Adolescent substance use. In R. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology*, (3rd ed., pp. 723-763). New York, NY: Wiley.

Chein, J., Albert, D., O'Brien, L., Uckert, K., & Steinberg, L. (2011). Peers increase adolescent risk taking by enhancing activity in the brain's reward circuitry. *Developmental Science*, *14*(2), F1-F10.

Cohen, G. L., & Prinstein, M. J. (2006). Peer contagion of aggression and health-risk behavior among adolescent males: An experimental investigation of effects on public conduct and private attitudes. *Child Development*, *77*, 967-983.

Cruz, J. E., Emery, R. E., & Turkheimer, E. (2012). Peer network drinking predicts increased alcohol use from adolescence to early adulthood after controlling for genetic and shared environmental selection. *Developmental Psychology*, *48*, 1390.

Dick, D. M., Rose, R. J., Pulkkinen, L., & Kaprio, J. (2001). Measuring puberty and understanding its impact: A longitudinal study of adolescent twins. *Journal of Youth and Adolescence*, *30*, 385-399.

Dishion, T. J., Spracklen, K. M., Andrews, D. W., & Patterson, G. R. (1996). Deviancy training in male adolescent friendships. *Behavior Therapy*, *27*, 373-390.

Duncan, P. D., Ritter, P. L., Dornbusch, S. M., Gross, R. T., & Merrill Carlsmith, J. (1985). The effects of pubertal timing on body image, school behavior, and deviance. *Journal of Youth and Adolescence*, *14*, 227-235.

Ennett, S. T., & Bauman, K. E. (1994). The contribution of influence and selection to adolescent peer group homogeneity: The case of adolescent cigarette smoking. *Journal of Personality and Social Psychology*, *67*, 653-663.

Feenstra, H., Ruiter, R. A., & Kok, G. (2012). Go fast! Reaction time differences between adults and adolescents in evaluating risky traffic situations. *Journal of Health Psychology*, *17*, 343-349.

Forbes, E. E., & Dahl, R. E. (2010). Pubertal development and behavior: Hormonal activation of social and motivational tendencies. *Brain and Cognition*, *72*, 66-72.

Forbes, E. E., Phillips, M. L., Ryan, N. D., & Dahl, R. E. (2011). Neural systems of threat processing in adolescents: Role of pubertal maturation and relation to measures of negative affect. *Developmental Neuropsychology*, *36*, 429-452.

Galvan, A., Hare, T., Parra, C. E., Penn, J., Voss, H., Glover, G., & Casey, B. J. (2006). Early development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. *Journal of Neuroscience*, *26*, 6885-6892.

Gao, Y., Baker, L. A., Raine, A., Wu, H., & Bezdjian, S. (2009). Brief report: Interaction between social class and risky decision-making in children with psychopathic tendencies. *Journal of Adolescence*, *32*, 409-414.

Gardner, M., & Steinberg, L. (2005). Peer influence on risk taking, risk preference, and risky decision making in adolescence and adulthood: An experimental study. *Developmental Psychology*, *41*, 625-635.

Graber, J. A., Lewinsohn, P. M., Seeley, J. R., & Brooks-Gunn, J. (1997). Is psychopathology associated with the timing of pubertal development? *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 1768-1776.

Harakeh, Z., & Vollebergh, W. A. (2012). The impact of active and passive peer influence on young adult smoking: An experimental study. *Drug and Alcohol Dependence*, *121*, 220-223.

Harden, K. P., Hill, J. E., Turkheimer, E., & Emery, R. E. (2008). Gene–environment correlation and interaction in peer effects on adolescent alcohol and tobacco use. *Behavior Genetics*, *38*, 339-347.

Harden, K. P., & Mendle, J. (2012). Gene-environment interplay in the association between early pubertal timing and delinquency in adolescent girls. *Journal of Abnormal Psychology*, *121*, 73-87.

Jessor, R., & Jessor, S. L. (1977). *Problem behavior and psychosocial development: A longitudinal study of youth*. San Diego, CA: Academic Press.

Larson, R., & Richards, M. H. (1991). Daily companionship in late childhood and early adolescence: Changing developmental contexts. *Child Development*, *62*, 284-300.

Martin, C. A., Kelly, T. H., Rayens, M. K., Brogli, B. R., Brenzel, A., Smith, J., & Omar, H. A. (2002). Sensation seeking, puberty, and nicotine, alcohol and marijuana use in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry*, *41*, 1495-1502.

McClintock, M. K., & Herdt, G. (1996). Rethinking puberty: The development of sexual attraction. *Current Directions in Psychological Science*, *5*, 178-183.

McEwen, B. S. (2001). Invited review: Estrogens effects on the brain: Multiple sites and molecular mechanisms. *Journal of Applied Physiology*, *91*, 2785-2801.

Mendle, J., & Ferrero, J. (2012). Detrimental psychological outcomes associated with early pubertal timing in adolescent boys. *Developmental Review*, *32*, 49-66.

Mendle, J., Turkheimer, E., & Emery, R. E. (2007). Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review*, *27*, 151-171.

Mercken, L., Snijders, T. A., Steglich, C., Vartiainen, E., & De Vries, H. (2010). Dynamics of adolescent friendship networks and smoking behavior. *Social Networks*, *32*(1), 72-81.

Moody, J. (2001). Peer influence groups: Identifying dense clusters in large networks. *Social Networks*, *23*, 261-283.

Nelson, E. E., Leibenluft, E., McClure, E. B., & Pine, D. S. (2005). The social reorientation of adolescence: A neuroscience perspective on the process and its relation to psychopathology. *Psychological Medicine*, *35*, 163-174.

Ouimet, M. C., Simons-Morton, B. G., Zador, P. L., Lerner, N. D., Freedman, M., Duncan, G., & Wang, J. (2010). Using the U.S. National Household Travel Survey to estimate the impact of passenger characteristics on young drivers' relative risk of fatal crash involvement. *Accident Analysis and Prevention*, *42*, 689-694.

Ozer, E., & Irwin, C. (2009). Adolescent and young adult health: From basic health status to clinical interventions. In R. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology* (3rd ed., pp. 618-641). New York, NY: Wiley.

Petersen, A. C., Crockett, L. J., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity and initial norms. *Journal of Youth and Adolescence*, *17*, 117-133.

Quevedo, K. A., Benning, S. D., Gunnar, M. R., & Dahl, R. E. (2009). The onset of puberty: Effects on the psychophysiology of defensive and appetitive motivation. *Development and Psychopathology*, *21*, 27-45.

Raine, A. (2002). Biosocial studies of antisocial and violent behavior in children and adults: A review. *Journal of Abnormal Child Psychology*, *30*, 311-326.

Shirtcliff, E. A., Dahl, R. E., & Pollak, S. D. (2009). Pubertal development: Correspondence between hormonal and physical development. *Child Development*, *80*, 327-337.

Snijders, T. A. B., Steglich, C. E. G., Van de & Bunt, G. G. (2010). Introduction to actor-based models for network dynamics. *Social Networks*, *32*, 44-60.

Somerville, L. H., Jones, R. M., & Casey, B. J. (2010). A time of change: Behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain and Cognition*, *72*, 124-133.

Sowell, E. R., Thompson, P. M., Holmes, C. J., Jernigan, T. L., & Toga, A. W. (1999). In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. *Nature Neuroscience*, *2*, 859-861.

Spear, L. (2009). *The behavioral neuroscience of adolescence*. New York, NY: Norton.

Spear, L. P. (2011). Rewards, aversions and affect in adolescence: Emerging convergences across laboratory animal and human data. *Developmental Cognitive Neuroscience*, *1*, 390-403.

Steinberg, L. (2008). A social neuroscience perspective on adolescent risk-taking. *Developmental Review*, *28*, 78-106.

Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology*, *44*, 1764-1778.

Sun, S. S., Schubert, C. M., Chumlea, W. C., Roche, A. F., Kulin, H. E., Lee, P. A., Ryan, A. S. (2002). National estimates of the timing of sexual maturation and racial differences among US children. *Pediatrics*, *110*, 911-919.

Tschann, J. M., Adler, N. E., Irwin, C. E., Millstein, S. G., Turner, R. A., & Kegeles, S. M. (1994). Initiation of substance use in early adolescence: The roles of pubertal timing and emotional distress. *Health Psychology*, *13*, 326-333.

Zimring, F. E. (1998). *American youth violence*. Oxford, UK: Oxford University Press.

Zuckerman, M., Buchsbaum, M. S., & Murphy, D. L. (1980). Sensation seeking and its biological correlates. *Psychological Bulletin*, *88*, 187-214.

Author Biographies

Natalie Kretsch, BA, is a doctoral student in clinical psychology at the University of Texas at Austin. She received her BA from Brown University. Her research focuses on the role of peer relationships in the development of externalizing behaviors during adolescence.

Kathryn Paige Harden, PhD, is an assistant professor in the Department of Psychology and a faculty research associate of the Population Research Center at the University of Texas at Austin. She received her PhD in clinical psychology from the University of Virginia. Her research focuses on the interplay of genes and environment in the development of adolescent psychopathology.