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Genetic and Environmental Pathways from Personality Risk to Antisocial Behavior

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Genetic and Environmental Pathways from Personality Risk to Antisocial Behavior

by

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Dissertation
Presented to the Faculty of the Graduate School of
The University of Texas at Austin
in Partial Fulfillment
of the Requirements
for the Degree of

Doctor of Philosophy

The University of Texas at Austin
August 2017

Genetic and Environmental Pathways from Personality Risk to Antisocial Behavior

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The University of Texas at Austin, 2017

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Antisocial behaviors are a constellation of deviant behaviors that are disruptive and harmful to others. Antisocial behavior increases during adolescence and a number of factors are thought to precipitate this rise, including changes in personality, social and familial factors. This dissertation presents three studies that examine how individual differences in sensation seeking contribute to risk for adolescent antisocial behavior. Study 1 finds that the highest rates of delinquency occur from the concurrence of high sensation seeking, high peer deviance, and low parental monitoring. Moreover, peer deviance partially mediates the effects of sensation seeking and parental monitoring on adolescent delinquency. Study 2 finds that affiliation with deviant peers is associated with higher delinquency after controlling for selection effects using a co-twin-control comparison. There is also evidence for person-environment correlation; adolescents with genetic dispositions toward higher sensation seeking are more likely to report having deviant peers. Moreover, the environmentally-mediated effect of peer deviance on delinquency is moderated by individual differences in sensation seeking. Finally, study 3 examines the role of sensation seeking situated within a multivariate array of behavioral and self-report measures that index individual differences in risk-taking propensities.

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INTRODUCTION

Antisocial behavior is commonly defined as aggressive or deviant behavior that is harmful to others and/or violates societal norms. When frequent and severe, antisocial behaviors constitute externalizing psychopathology, such as oppositional defiant disorder or antisocial personality disorder. Antisocial behavior tends to increase during adolescence and a number of factors are thought to precipitate this rise, including personality, social and familial factors. Though, knowledge of how these factors combine and intersect in a multivariate framework remains nascent, and the manner in which researchers and clinicians operationalize antisocial behavior continues to evolve.

A large body of empirical research has identified common personality traits that target individuals at heightened risk for antisocial behavior. With respect to broad dimensions of personality, results of a meta-analysis indicate that low levels of agreeableness and low levels of conscientiousness are indicative of antisocial behavior (Jones, Miller & Lynam, 2011). Furthermore, a more fine-gained focus on associations between personality and antisocial behavior identified impulsiveness and excitement seeking as additional personality traits that index risk for antisocial behavior. Furthermore, cross-disciplinary research in cognitive neuroscience and developmental psychology (Steinberg et al., 2008; Steinberg, 2010) highlights sensation seeking and related traits as salient precursors to risk-taking behavior, including antisocial expressions of risk-taking. Motivated by dimensional perspectives of psychopathology[1], the present dissertation presents three studies that draw on methods from

[1] Dimensional perspectives of psychopathology view clinical symptoms and associated disorders as extreme manifestations of normal-range behaviors, emotions and cognitions. In contrast to taxonomic models, which view psychiatric disorders as constituting discrete classes or "natural kinds" (Meehl, 1992), dimensional models

quantitative genetics, developmental psychology and personality psychology to understand how individual differences in sensation seeking combine and intersect with environmental factors to place certain adolescents at heightened risk for antisocial behavior.

In Study 1, I simultaneously test mediating and moderating relations between sensation seeking, peer deviance and parental monitoring as concurrent predictors of delinquent behavior. Results indicate that high levels of adolescent delinquency resulted from an interaction between (high) sensation seeking, (high) peer deviance and (low) parental monitoring. In addition, peer deviance partially mediates associations between sensation seeking and delinquency, as well as parental monitoring and delinquency. These results suggest that adolescents prone to sensation seeking may be more likely to engage in delinquent behavior because they affiliate with delinquent peers, and may also be more susceptible to the influence of those peers.

In study 2, I test the interactive effects of peer deviance and sensation seeking on delinquency in a genetically informative design. Specifically, I test (1) whether sensation seeking mediates genetic influences on delinquency, (2) whether sensation seeking mediates gene-environmental correlations with peer deviance and (3) whether sensation seeking moderates the environmental effects of peer influence on adolescent delinquency. Results indicate that within-twin-pair differences in affiliation with deviant peers are associated with higher delinquency. This result is consistent with a socialization effect of peers on adolescent delinquency. However, there is also evidence for person-environment correlation; adolescents with genetic dispositions toward higher sensation seeking are more likely to report having deviant peers. Moreover, genetic variance in sensation seeking overlaps substantially with genetic variance in adolescent

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conceptualize psychiatric disorders as occupying extreme ends of personality trait continua spanning the range from normal to abnormal functioning.

delinquency. These results are consistent with both selection and socialization processes in adolescent peer relationships, and highlight the role of sensation seeking as an intermediary phenotype for genetic risk for antisocial behavior.

In addition to measuring individual differences in sensation seeking, researchers have employed a variety of self-report questionnaires and behavioral tasks to measure risk-taking propensities, which concurrently and prospectively predict a wide range of externalizing outcomes, including antisocial behavior. Although researchers commonly interpret these self-report and behavioral measures in terms of the same underlying constructs, results of a recent study (Harden et al., 2016) indicate that four latent constructs (premeditation, fearlessness, cognitive dyscontrol and reward-seeking) are needed to account for patterns of covariation among these self-report and behavioral measures. Moreover, the self-report measure of sensation seeking loaded positively onto the fearlessness *and* reward-seeking factors, as well as negatively onto the premeditation factor, reflecting the multidimensional complexity of this construct. Therefore, in Study 3 I examine how sensation seeking and latent risk-taking constructs relate to different subtypes of antisocial behavior.

Chapter 1: Person x Environment Interaction on Adolescent Delinquency: Sensation Seeking, Peer Deviance and Parental Monitoring

SENSATION SEEKING

Sensation seeking, defined as a disposition to select and prefer novel, stimulating, or exciting experiences, is an intrapersonal risk factor for a number of problematic behaviors (Harden, Quinn & Tucker-Drob, 2012; Popham, Kennison & Bradley, 2011; Sargent, Tanski, Stoolmiller, Hanewinkel, 2010; Zuckerman, 1994). Population-average developmental increases in sensation seeking and delinquent behavior co-occur across adolescence (Moffitt, 1993; Steinberg et al., 2008; Zuckerman, 1994), and individual differences in longitudinal changes in sensation seeking account for much of the adolescent spike in delinquent behavior (Harden et al., 2012). As a personality risk factor for adolescent delinquency, sensation seeking may index behavioral reaction norms (Sih, Bell & Johnson, 2006) or a "reaction range" (Nigg, 2006) for the potential emergence of delinquent behaviors, with environmental contexts possibly mediating and/or moderating this risk. Researchers have therefore begun to examine specific contextual factors that facilitate, exacerbate or attenuate personality risk for delinquent behavior. In the current paper, we consider the relations between sensation seeking and two social contexts: deviant peer groups and parental monitoring.

PEER DEVIANCE

Peer group deviance is one the most robust contextual correlates of adolescents' delinquent behavior (Kandel, 1986), an association that has been found to reflect both social selection and social influence (Burk, van der Vorst, Kerr, & Stattin, 2011; Willis & Cleary, 1999). Social selection is a process by which adolescents with dispositions toward delinquency

select (and are selected into) deviant peer groups (Kandel, 1978; Gottfredson & Hirschi, 1990). Social influence occurs when befriending and socializing with deviant peers increases one's likelihood to engage in delinquent behavior. Sensation seeking may play both mediating and moderating roles in these peer dynamics.

As a mediator, sensation seeking may be a psychological mechanism of social selection processes, shaping who an adolescent's friends are. For instance, affiliating with deviant peers may be one way that adolescents high in sensation seeking find a social-ecological niche that is conducive to their motivational and behavioral dispositions. Hampson, Andrews and Barckley (2008) demonstrated that the link between sensation seeking and marijuana use was mediated by affiliation with deviant peers. Similarly, Yanovitzky (2005) found that sensation seeking predicted peer deviance and pro-drug discussion, which further predicted motivation for marijuana use. These studies underscore the role of social section effects, whereby high sensation seekers befriend deviant peers that, in turn, increase risk for drug use.

As a moderator, sensation seeking may also play a role in social influence processes, affecting how an individual responds to peer influences. High sensation seekers may be more responsive to the immediate rewards of peer interaction and approval – and thus more vulnerable to deviant social influence. Consistent with moderating relations between personality and contextual risk, behavior genetic research has found evidence for gene × peer deviance interaction effects, whereby genetic risks on delinquency and substance use are exacerbated among adolescents with deviant peers (Harden, Hill, Turkheimer, & Emery, 2008; Hicks, South, DiRago, Iacono, & McGue, 2009; Kretschmer, Vitaro, & Barker, 2014). Although the specific genetic vulnerabilities underlying these effects are unknown, other research has shown that

sensation seeking is a heritable personality trait (Koopmans, Boomsma, Heath & van Doornen, 1995) that partly accounts for heritable variation in adolescent delinquency (Harden et al., 2012). These findings suggest that the effects of peer groups on delinquent behavior may be intensified when genetic risk for delinquency—including risk conferred by high sensation-seeking—is present.

PARENTAL MONITORING

Finally, negative effects of sensation seeking on adolescent delinquency may wane in protective environmental contexts. Parental monitoring – including parental knowledge and rules about adolescent's activities and friends – is a protective contextual factor that has been found to mitigate the deleterious effects of various risks on adolescent behavior (Hill, & Tyson, 2009; Lac & Crano, 2009). Parental monitoring may buffer the negative effects of high sensation seeking by preventing adolescents' affiliation with deviant peers *and* by limiting the influence of those peers (Dorius, Bahr, Hoffman & Harmon, 2004; Kiesner, Poulin & Dishion, 2010; Steinberg, Fletcher & Darling, 1994). For instance, a longitudinal study of adolescents found that the protective influence of parental monitoring on later problem behavior was mediated by reduced peer deviance (Ary et al. 1999_b) Likewise, in a large sample of adolescents, peer influence was found to mediate the protective effect of parental monitoring on alcohol-use (Kim & Neff, 2010). Moreover, a study with late adolescents found a moderating effect of parental monitoring on the relation between peer influence and drinking behavior (Wood, Read, Mitchell & Brand, 2004). Finally, molecular genetic research has found evidence of a gene × parental monitoring interaction, whereby genetic risks for externalizing behavior decrease under high levels of parental monitoring (Dick et al. 2007, 2009, 2011).

In sum, deviant peer groups and parental monitoring are contextual factors that likely influence the association between sensation seeking and adolescent delinquency. Deviant peer groups may increase risk for high sensation-seeking adolescents by mediating social selection, and sensation seeking may in turn moderate the socializing effects of peers. High parental monitoring may decrease risk for delinquency by preventing deviant peer affiliation and by limiting the effect of peer influence.

GOALS OF THE PRESENT STUDY

Although multiple personal and contextual factors are known to contribute to delinquent behavior in adolescence, less research has focused on the combined influence of such factors. Accordingly, the current study examines individual differences in adolescences' sensation seeking, peer deviance and parental monitoring as predictors of delinquent behavior. We test five hypotheses. First, high levels of sensation seeking and peer deviance and low levels of parental monitoring will independently predict adolescent delinquency. Second, peer deviance will partially mediate the effect of sensation seeking on delinquent behavior, such that adolescents high in sensation seeking will select deviant peer groups and, in turn, increase risk for delinquency. Third, peer deviance will also moderate the association between sensation seeking and delinquency, such that adolescents high in sensation seeking will be more vulnerable to the influence of deviant peers. Fourth, peer deviance will mediate the protective effect of parental monitoring on delinquent behavior, such that high levels of parental monitoring will prevent adolescents from affiliating with deviant peers and, in turn, prevent exposure to contextual risk for delinquency. Fifth, we expect the protective effects of parental monitoring to be highest for youth high in both intra- and inter-personal risk. Therefore, we hypothesize that high levels of

parental monitoring will moderate the combined influence that sensation seeking and peer deviance has on delinquent behavior, such that a three-way interaction between sensation seeking, peer deviance and parental monitoring will be observed.

METHOD

Participants

Participants were 362 adolescent siblings (identical and fraternal twins), ages 14-21 years (mean age = 15.99 years) from the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013). The sample was 52% male and 48% female. The racial composition of the sample was 60.3% non-Hispanic Caucasian, 21.9% Hispanic/Latino, 9.4% African-American, 1% Native American, 1% East Asian, 1.4% Southeast Asian and 5% mixed-race/other. The highest level of education completed by parents ranged from 7th grade to graduate school. Approximately 5.9% of parents did not complete high school, 26.8% graduated high school, 2.7% completed a vocational or technical degree, 6.7% completed an associate degree, and 57.9% a bachelor degree or higher.

Participants were identified as twins from public school rosters and recruited via invitation by phone call or mailing. Verbal and written consent was obtained from parents and adolescents prior to participation and the study was granted a federal certificate of confidentiality to ensure honest reporting without risk of legal sanction. Parents completed a survey, and adolescents visited the laboratory, during which time they completed a number of computerized tasks and a survey that asked a variety of questions about family, friends, school-related activities, prosocial behavior and antisocial behavior. Trained research assistants administered all tasks; a different research assistant assessed each sibling separately.

Measures

Sensation seeking. Individual differences in sensation seeking were measured using an abbreviated version of Zuckerman's Sensation Seeking Scale (1966). The measure included 8 items, such as 'I would like to explore strange places', 'I like wild parties', 'I like to do frightening things' and 'I prefer friends who are excitingly unpredictable'. Items were rated on a scale ranging from 1 (*Strongly Disagree*) to 5 (*Strongly Agree*). See Table 1 for descriptive statistics and reliabilities for untransformed variables.

Peer deviance. Peer deviance was measured with an 22-item self-report questionnaire adapted from Thornberry, Lizotte, Krohn, Farnworth, & Jang (1994), which asked adolescents how many of their friends engage in various delinquent behaviors, including stealing, destroying property, and selling drugs, and prosocial behaviors, such as participating in school activities, getting along with teachers and staying out of trouble at school. Items were rated on a scale ranging from 1 (*None of them*) to 4 (*All of them*). Prosocial items were reverse scored, and all items were aggregated to form a composite scale.

Parental monitoring. Parental monitoring was measured using a 15-item self-report questionnaire that examined parental knowledge about friends, activities and household rules. Seven items assessed parental knowledge about adolescents' friends and activities. These items were rated on a scale ranging from 1 (*They don't know*) to 3 (*They know a lot*)." Five items assessed parental control over adolescents' friends and activities. For example, participants were asked whether they need permission to go out on weekends and weeknights and whether they have curfews. These items were rated on a 3-point scale (1 = *Never*, 2 = *Sometimes*, 3 = *Always*). Items that indexed parental knowledge and control were aggregated to form a composite scale.

Delinquency. Adolescent delinquent behavior was measured using a 36-item self-report measure adapted from Huizinga, Esbensen and Weiher (1991). Adolescents were asked if they had ever engaged in a number of delinquent behaviors, which ranged in severity from minor delinquent offenses to relatively severe criminal offensives. Minor delinquent offensives included behaviors such as, "driven a car very fast (> 20 MPH over the speed limit)", "purposely damaged or destroyed your own property", "been loud, rowdy, or unruly in a public place" and "been suspended or expelled from school". More serious criminal offenses included behaviors such as, "broken into a building or vehicle (or tried to break in) to steal something or just to look around", "sold marijuana or hashish ('pot', 'weed', 'hash')", "carried a hidden weapon (a knife or a gun)" and "hit or threatened to hit a teacher, parent, or another adult." Items were assessed on a 3-point scale (1 = *Neve*r, 2 = *Once*, 3 = *More than once*).

ANALYTIC PROCEDURES

Data were analyzed using structural equation modeling in the software program M*plus* version 7.1 (Muthén & Muthén, 1998-2010). All standard errors and model statistics were adjusted for nonindependence of data from children living in the same household (i.e. sibling clusters; Asparouhov & Muthen, 2006) using the Complex Survey option. Age trends and gender differences associated with delinquency are well documented (Moffitt, 1993; Simourd & Andrews, 1994; McCabe, Lansing, Garland & Hough, 2002); therefore, age in years and gender were treated as covariates in all analyses. Peer deviance and parental monitoring scales were log-transformed to correct for positive skew and all focal predictors (but not covariates) were standardized prior to computing interaction terms and conducting analyses. Inspection of the distribution of adolescent-report delinquency indicated the presence of a floor effect (i.e., left-

censoring), as is commonly the case with measures of delinquency, which tend not to index minor social offenses. We therefore employed a Tobit model to produce unbiased parameter estimates for censored data (Muthen, 1990; Tobin, 1958).

The full model fit is illustrated in Figure 1. Mean-centered age and sex were included as covariates of sensation seeking, peer deviance, parental monitoring, and delinquency, and sex was regressed onto age to account for potential codependency. To evaluate main effect and mediating hypotheses direct paths from sensation seeking, peer deviance, and parental monitoring to delinquency were estimated, as well as indirect paths from sensation seeking and parental monitoring through peer deviance. To evaluate moderating hypotheses two-way interactions between each combination of focal predictors were estimated (sensation seeking × peer deviance, sensation seeking x parental monitoring, peer deviance × parental monitoring), as well as a three-way interaction (sensation seeking × peer deviance × parental monitoring).

A Tobit model is important for ensuring unbiased point estimates with censored data; however, current methodological recommendations (Shrout & Bolger, 2002) suggest bootstrapping parameter estimates and confidence intervals when testing mediation. Because a Tobit model is unavailable with bootstrapped parameters, mediating and moderating hypotheses were first tested with a Tobit model using a mean and variance adjusted weighted least squares estimator without bootstrapping. The full model was then refit with bootstrapped parameter estimates and standard errors using maximum likelihood estimation. We report results from the former method (i.e. Tobit model without bootstrapping) but, results from both approaches were similar in terms of effect sizes and confidence intervals

RESULTS

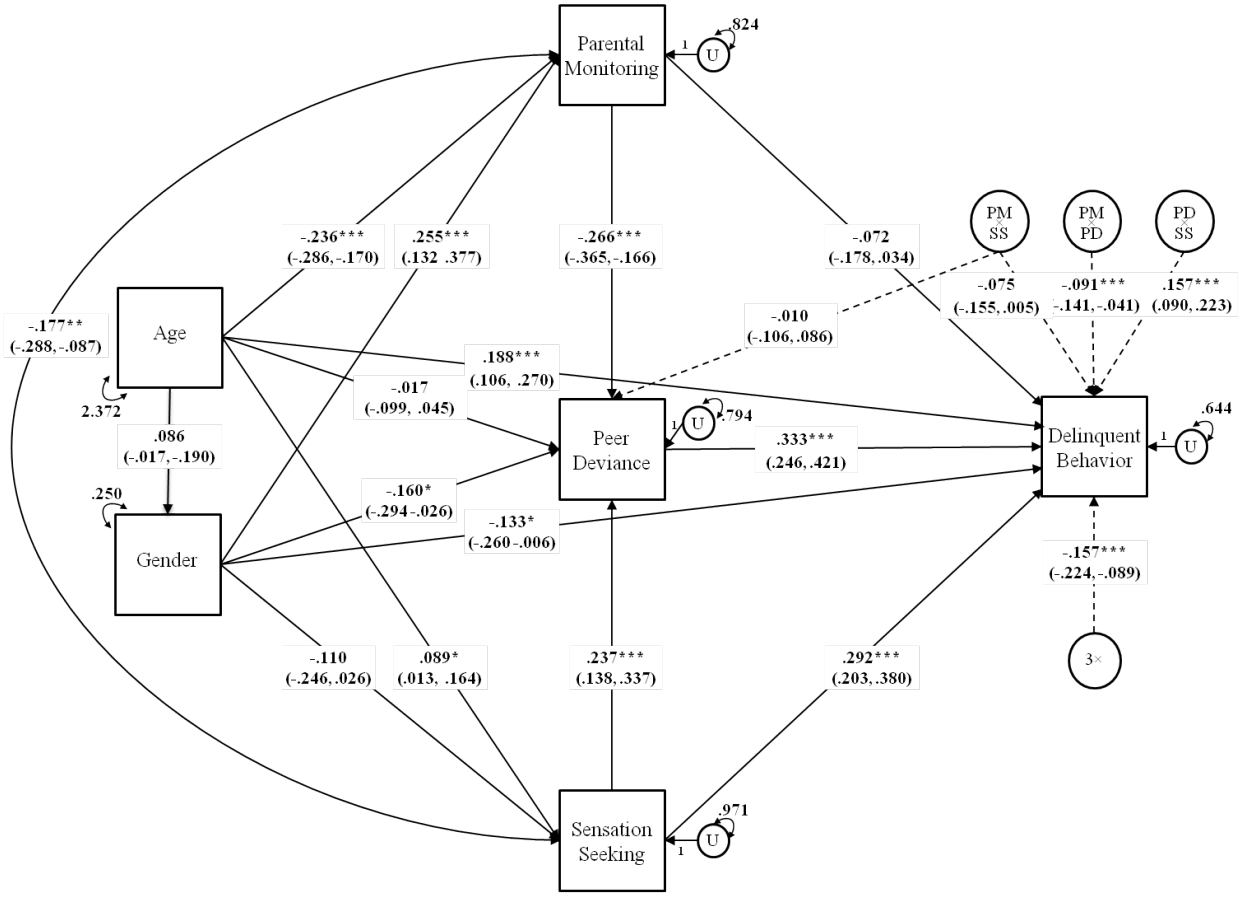
Table 1. Zero-order Correlations, Descriptive & Reliability Statistics

N = 362		M (SD)	Age	SS	Peer	Pmon	Del
Sex		.480 (.500)	.132	-.093	-.242***	.211**	-.199**
Age		15.990 (1.543)		.121*	.067	.330***	.322***
SS	(α = .731)	3.237 (.711)			.313***	-.243***	.449***
PD	(α = .857)	1.756 (.329)				-.348***	.483***
PM	(α = .850)	2.571 (.341)					-.397***
Del	(α = .878)	7.525 (7.785)					

Note. descriptive statistics for untransformed variables & correlations for transformed variables are reported. α = Cronbach's alpha. M = mean. (SD) = standard deviation. For sex, 0 = male, 1 = female. SS = sensation seeking. PD. = peer deviance. PM = parental monitoring. Del = delinquent behavior. * $p_{(two\text{-}tailed)} < .05$. ** $p_{(two\text{-}tailed)} < .01$. *** $p_{(two\text{-}tailed)} < .001$.

Table 1 summarizes the zero-order correlations between transformed study variables. Females reported lower peer deviance ($r = -.242$, $SE = .069$, $p < .001$), higher parental monitoring ($r = .221$, $SE = .067$, $p < .01$), and less delinquent behavior than males ($r = -.199$, $SE = .073$, $p < .01$). Older adolescents reported higher sensation seeking ($r = .121$, $SE = .079$, $p < .05$), lower parental monitoring ($r = -.330$, $SE = .055$, $p < .001$), and more delinquent behavior than younger adolescents ($r = .332$, $SE = .049$, $p < .001$). Higher sensation seeking ($r = .313$, $SE = .047$, $p < .001$) and lower parental monitoring ($r = -.348$, $SE = .047$, $p < .001$) were both correlated with higher peer deviance. Furthermore, all covariates and focal predictors were correlated with delinquency.

Figure 1. Path Diagram of Mediating and Moderating Pathways to Adolescent Delinquency



Note. Unstandardized path coefficients reported for the full model with focal predictors and self-reported delinquency standardized. Product terms computed from standardized predictors. 95% confidence intervals are reported in parentheses. Gender coded as male = 0, female = 1. Circle labeled "3×" represents sensation seeking × peer deviance × parental monitoring interaction. Interaction terms were regressed on age and gender, and all covariances among interaction terms and focal predictors were estimated, but these associations are not illustrated for ease of presentation. Results are therefore estimates from a fully saturated model.

Results from our full model are illustrated in Figure 1 and include several notable findings. Note that because all focal predictors were standardized, the main effects in the model can be interpreted as population-average effects, i.e. effects holding all moderators at their mean levels. In support of our first hypothesis, sensation seeking (b = .292, 95% CI = .203, .380, p < .001) and peer deviance (b = .333, 95% CI = .246, .421, p < .001) had significant main effects on adolescent self-report delinquency, even after controlling for age and gender. However, contrary to our first hypothesis, after taking into account all other model predictors, the population-average direct effect of parental monitoring on delinquency was not significant (b = -.072, 95% CI = -.178, .034, p = .183). Second, high sensation seeking adolescents (b = .237, 95% CI = .138, .337, p < .001) and adolescents with lower levels of parental monitoring (b = -.266, 95% CI = -.365, -.166, p < .001) reported higher levels of peer deviance.

Table 2. Total, Direct & Indirect Effects on Adolescent Self-Report Delinquent Behavior

	Total		*Direct*		*Indirect*	
	B	S.E.	*B*	S.E.	*B*	S.E.
Sensation Seeking	.371***	(.049)	.292***	(.045)	.079***	(.021)
Parental Monitoring	-.161***	(.052)	-.072	(.054)	-.089***	(.021)

Note: mediator variable = peer deviance; *B* = unstandardized path coefficients for standardized variables; standard errors are reported in parentheses; * = $p_{(two\text{-}tailed)}$ < .05; ** = $p_{(two\text{-}tailed)}$ < .01; *** = $p_{(two\text{-}tailed)}$ < .001.

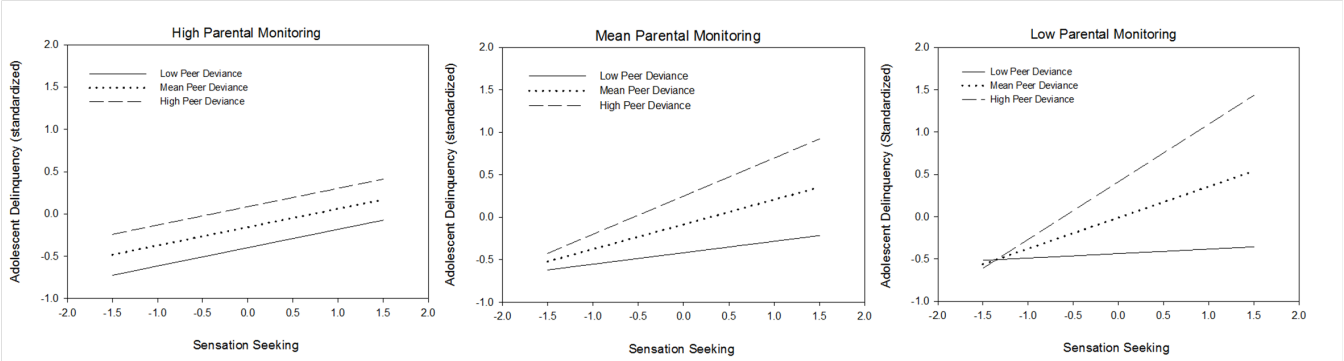
Consistent with our mediation hypotheses, tests of indirect effects (summarized in Table 2) indicated that affiliation with deviant peers partially mediated the association between sensation seeking and delinquency (b = .079 95% CI = .039, .119, p < .001), as well as parental monitoring and delinquency (b = -.089, 95% CI =-.130, -.047, p < .001). That is, these results indicate that delinquency increases by approximately .08 standard deviations for every standard deviation increase in sensation seeking by way of its effect on deviant peer affiliation. Likewise, adolescent delinquency is predicted to decrease by approximately .09 standard deviations for every 1 standard deviation increase in parental monitoring by way of its effect on deviant peer affiliation.

In support of our moderation hypotheses, there were significant two-way interactions between sensation seeking and peer deviance (b = .157, 95% CI = .090, .223, p < .001), peer deviance and parental monitoring (b = -.091, 95% CI = -.141, -.041, p < .001), and a marginally significant interaction between sensation seeking and parental monitoring (b = -.075, 95% CI = -.115, .005, p = .067). There was also a significant three-way interaction between sensation seeking, peer deviance and parental monitoring (b = -.157, 95% CI = -.244, -.089, p < .001), such that the association between sensation seeking and delinquency was magnified among adolescents who socialized with deviant peers *and* who were low in parental monitoring. Thus, the highest rates of individual delinquency appeared in the context of high levels of sensation seeking and peer deviance, as well as low levels of parental monitoring. See Figure 2 for a plot of the simple slopes from the three-way interaction.

Finally, three effects of gender were revealed. Controlling for all other predictors,

adolescent boys were more likely to engage in delinquent behavior than adolescent girls (b = -.133, 95% CI = -.260, -.006, p < .05) and adolescent boys were also more likely to report deviant peer affiliation (b = -.160, 95% CI = -.294, -.026, p < .05). Conversely, adolescent girls were more likely to be monitored by their parents (b = .255, 95% CI = .132, .377, p < .001). In sum, covariates (i.e. age and gender) and mediating and moderating pathways between focal predictors (i.e. sensation seeking, peer deviance & parental monitoring) accounted for more than a third of the variance in adolescents` self-report delinquent behavior (R^2 = .36).

Figure 2. Sensation Seeking × Peer Deviance × Parental Monitoring Interaction on Delinquency



Note. Predicted adolescent delinquent behavior calculated from model parameters shown in Figure 1. Simple slopes are displayed for low (-1σ), average and high (+1σ) peer deviance. Panels present sensation seeking × peer deviance interaction across high (+1σ), average and low (-1σ) levels of parental monitoring.

DISCUSSION

This study documents a nexus of mediating and moderating pathways between adolescent sensation seeking and social contexts underlying individual differences in delinquent behavior. Our findings build on the dual systems model of adolescent risk-

taking (Somerville, Jones, & Casey, 2010; Steinberg, 2010), which suggests that adolescence is generally a developmental period of heightened propensity toward deviant and dangerous behavior, because of asynchrony between the development of sensation and reward seeking (the incentive processing system) and the development of impulse control and inhibition (the cognitive control system). We find that, rather than conferring a uniform level of risk, sensation-seeking may be better conceptualized as a "reaction norm" (Sih, Bell & Johnson, 2004) or providing a "reaction range" (Nigg, 2006) which may result in higher or lower levels of delinquent behavior in the presence of certain contextual factors. Specifically, higher sensation seeking is translated into deviant behavior only when peers provide opportunities for delinquent behavior *and* when they lack an "external prefrontal cortex" – i.e., parents who monitor and regulate their behavior.

Furthermore, results suggest that adolescents prone to personality risk (i.e. high levels of sensation seeking) may be more likely to engage in delinquent behavior because they often choose to befriend delinquent peers (i.e. mediation by social selection). In addition, in the context of deviant peers, these high sensation-seekers may be more susceptible to peer influence, which further exacerbates risk for delinquency (i.e. moderation by socialization). Thus, personality guides selection processes, by which adolescents search for and select social- ecological niches (e.g. deviant peer groups) that are conducive to their personalities and moderates social influence processes, in the form of heightened vulnerability to contextual (peer and parental) influence (Caspi, Roberts & Shiner, 2005). In fact, personality traits such as sensation-seeking may represent risk in one context, but resiliency in another (Nigg, 2006; Tackett, 2006) – thus, clear

delineation of specific contextual factors is essential to offer a better understanding of associations between sensation-seeking and consequential adolescent outcomes.

The three-way interaction documented in the current study suggests that the pathway between personality risk, peer groups and individual delinquency is heightened in unrestrictive and risky social environments, including environments facilitated by low levels of parental monitoring. In other words, delinquency emerges when individuals with certain behavioral dispositions select and are vulnerable to the influence of risky social environments, which is far more likely to occur in family contexts that allow adolescents to affiliate with whomever they choose. Moreover, the moderating effect of parental monitoring suggest that, even if parents fail to prevent adolescents from affiliating with deviant peers, parents may still buffer the negative effects associated with peer deviance by restricting socialization. For example, even if adolescents select (or are selected into) deviant peer groups, parental monitoring may limit social influence to relatively benign settings, like school classrooms, the cafeteria and supervised extracurricular activities; as opposed to risky environments, like unsupervised parties and late-night joy rides.

Sensation seeking, peer deviance and parental monitoring were measured by adolescent-report. The current results would be further substantiated by future efforts to capitalize on measures of parental, peer, and school report, as well as behavioral measures of sensation seeking. Moreover, while the current study builds off previous longitudinal work indicating both selection and socialization processes (Curran, Stice & Chassin, 1997; Gordon et al., 2004; McCabe et al., 2005), the current project made use of cross-sectional data. Results of the current study alone therefore do not allow us to make

causal inferences about the associations uncovered. Future research making use of longitudinal data would allow us to examine whether sensation seeking prospectively predicts deviant peer affiliation or whether such affiliations prospectively predict individual delinquency.

In conclusion, the current study provides evidence for specific contextual factors that exacerbate and mitigate a well-established marker of personality risk: sensation seeking. We found that sensation seeking, deviant peer groups and parental monitoring interact to predict adolescent delinquency: Sensation seeking is most strongly related to delinquency in the context of more deviant peers and lower parental monitoring. These results highlight the importance of considering theoretically grounded, synergistic intersections among intrapersonal and contextual factors when elucidating the complex pathways that underlie adolescent delinquency.

Chapter 2: Sensation Seeking, Peer Deviance, and Genetic Influences on Adolescent Delinquency: Evidence for Person-Environment Correlation and Interaction

Adolescence is a peak period of risk for engaging in antisocial behavior, i.e., acts that violate rules, social norms, and/or the rights of others (Jessor & Jessor, 1977; Moffitt, 1993). Research on antisocial behavior from a developmental psychopathology perspective investigates how normative developmental processes in adolescence intersect with individual vulnerabilities and social contexts to shape why teenagers are generally more prone to antisocial behavior than are children or adults, and why certain teenagers are at particular risk compared to others (Frick & Viding, 2009; Krueger, Markon, Patrick & Iacono, 2005). In the current study, we adopt a developmental psychopathology approach to consider the intersections among genes, sensation seeking, and peer deviance in the etiology of adolescent delinquency. We test for *gene-environment correlation*, in which adolescents with greater genetic liability toward sensation-seeking and antisocial behavior are more likely to affiliate with deviant peers, and *person × environment interaction,* in which adolescents higher in sensation seeking (a genetically influenced trait) are more susceptible to the environmental effects of peer deviance on antisocial behavior.

Peer Selection and Socialization

Teenagers whose friends engage in delinquent behaviors are more likely than teenagers without such friends to engage in delinquency themselves. Some part of peer group similarity for delinquency is due to selection processes, as friends are not chosen at random. Adolescents may select friends partly on the basis of delinquency itself or on the basis of correlated behaviors (e.g., smoking or drinking; Caspi, 1994; Ennett & Bauman,

1994; Burk, Vorst, Kerr & Stattin, 2011) and traits (e.g. sensation seeking, e.g. Mann et al., 2015). Additionally, an adolescent's potential pool of friends (e.g., other teens in the same school or neighborhood) are stratified by demographic variables (e.g., low socioeconomic status) that may be risks for delinquent behavior, inducing similarity among peer groups (social homogamy) in the absence of active and evocative selection processes (McPherson, Smith-Lovin & Cook, 2001). Nevertheless, research using longitudinal and quasi-experimental methods capable of controlling for selection factors, confirms that deviant peer groups do, in fact, exert a causal influence on adolescent delinquency, above and beyond selection processes and social homogamy (Dishion & Tipsord, 2011).

One method for estimating peer influences on delinquency is a co-twin-comparison design. When comparing two identical twins, who have overlapping but not entirely identical peer groups, does the twin with more deviant peers also engage in more delinquent behavior? Although any cross-sectional design is, of course, not able to establish direction of causation definitively, a within-twin-pair comparison controls for selection and social homogamy processes that are driven by genetic and environmental factors that are shared by identical twins raised in the same home. That is, a within-identical-twin-pair association between peer deviance and delinquency cannot be confounded by genetically-based propensities to seek out certain types of friends, because identical twins are genetically identical; nor can this within-twin-pair association be attributed to social homogamy due to school or neighborhood environments, socioeconomic status, or race/ethnicity – all of which are identical within both identical and fraternal twin pairs raised together.

Consistent with a socialization effect of peers, previous twin research on peer influence has found evidence for a within-twin-pair association between peer deviance and delinquency and related behaviors (Cruz, Emery, & Turkheimer, 2012; Harden, Hill, Turkheimer, & Emery, 2008; Kretsch, Mendle, & Harden, *in press*). At the same time, twin research has also documented the importance of gene-environment correlations (*r*GE): More genetically similar individuals (e.g., identical twins as compared to fraternal twins) experience more similar peer environments, indicating that adolescents are selecting and being selected into peer groups partially on the basis of their genetically-influenced traits (Agrawal et al., 2010; Cleveland, Wiebe, & Rowe, 2005; Fowler, Settle & Christakis, 2011; Harden et al., 2008; reviewed in Brendgen, 2012). Interestingly, *r*GE for peer relationships appears to emerge in adolescence; twin research with pre-pubertal children has failed to find substantial genetic effects on peer characteristics (Brendgen, 2012).

Person × Context Interactions

A twin-comparison-approach can also be used to estimate whether individual characteristics confer greater sensitivity (or resilience) to the environmental effects of peer influence, a person × environment interaction. Identifying individual differences that moderate the strength of peer influence processes is a research goal that is "essential [for] the development of more precise, targeted intervention efforts" (Brechwald & Prinstein, 2011, p. 174). Because characteristics that make adolescents more vulnerable to peer influence may themselves be genetically influenced, such person × environment interactions may serve as mechanisms of gene × environment interaction.

Gene × environment interactions involving the peer environment are among the most consistently replicated effects in behavioral genetics literature. Multiple twin studies have found that genetic influences on delinquency and related behaviors (e.g., drinking) are magnified among teenagers with deviant peers (Boardman, Saint Onge, Haberstick, Timberlake, & Hewitt, 2008; Button et al., 2007, 2009; Fowler et al., 2007; Guo, Elder, Cai, & Hamilton, 2009; Harden et al., 2008; Hicks et al., 2009). Similar results have been obtained in candidate gene studies, with variants in the *BDNF* and *CHRM2* genes found to interact with peer characteristics to predict adolescent antisocial behavior (Kretschmer, Dijkstra, Ormel, Verhulst & Veenstra, 2013; Kretschmer, Vitaro, & Barker, 2014; Latendresse et al., 2011).

Most recently, Salvatore and colleagues (2015) constructed a polygenic risk index based on results from a genome-wide association study of externalizing disorders (i.e., substance use disorders, antisocial personality disorder) in adults, and then examined interactions between peer deviance and this polygenic score in the prediction of antisocial behavior in adolescents. As hypothesized, they found that the polygenetic risk score was more strongly associated with adolescent antisocial behavior among adolescents who reported high levels of peer deviance. A polygenic risk score approach is similar to a twin study, in that it estimates the effects of genetic variants across the entire genome (rather than single variants, as in a candidate gene study), but involves entirely different assumptions, as genetic risk is measured directly from DNA rather than inferred from resemblance between biological relatives. The convergence of results across candidate gene, polygene, and twin methods is a testament to the robustness of gene × peer effects. However, it is not currently clear what specific genetically-influenced psychological

traits interact with peer context to give rise to these gene × environment interactions. The current study focuses on sensation seeking, a personality trait that may act as a mechanism of genetic effects on delinquency.

Sensation Seeking as an Endophenotype

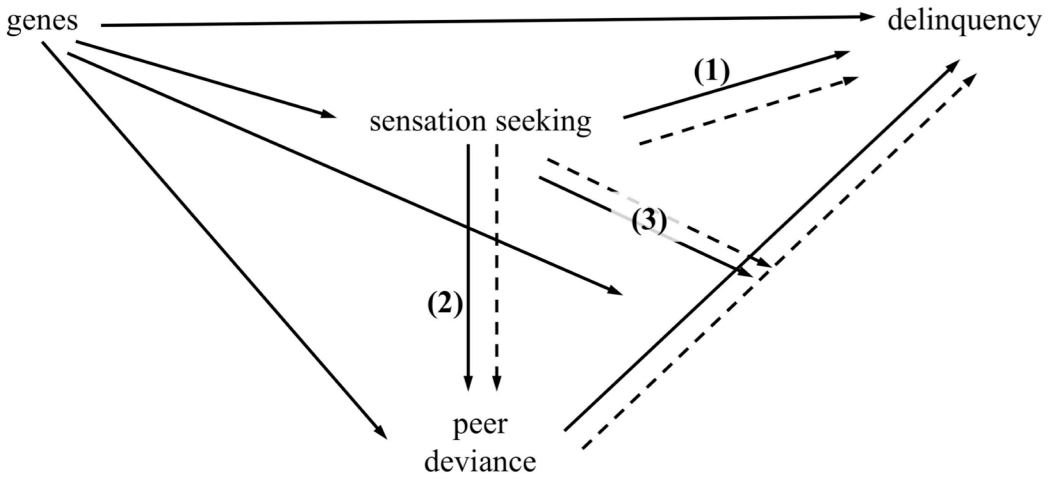
Although previous research has found consistent evidence for both *r*GE and G×E in the association between peer deviance and delinquency, there has been comparatively little research into the specific genetically influenced psychological traits that serve as mechanisms of these effects. A largely separate line of research suggests that sensation seeking may be an *endophenotype* (Gottesman & Gould, 2003) that mediates genetic influences on delinquency. Sensation seeking is a facet of disinhibited personality characterized by a preference for novel, rewarding, and/or thrilling experiences and sensations (Zuckerman, 1971). Phenotypically, average levels of sensation seeking increase dramatically from late childhood (~age 10) to mid-adolescence (~age 16; Harden & Tucker-Drob, 2011), an age-trend that parallels the rise of delinquency (Harden, Quinn, & Tucker-Drob, 2012). Moreover, genetically influenced individual differences in the rapidity of sensation seeking change in adolescence substantially account for individual differences in the escalation of delinquency (Harden et al., 2012).

Convergent evidence for the utility of sensation seeking as an index of genetic risk comes from a genetic association study by Aliev and colleagues (2014). They focused on a candidate set of 215 measured genetic variants, each of which had been previously found to be associated with alcohol dependence or externalizing disorder phenotypes in adults. They tested the associations between each variant and antisocial behavior outcomes, substance use outcomes, and personality traits (including

extraversion, conscientiousness, impulsivity, and sensation seeking). Interestingly, they found the greatest number of significant genetic associations with sensation seeking, with enrichment analyses indicating that sensation seeking was more consistently associated with the candidate genetic variants than any other phenotype.

Sensation seeking is also correlated with affiliation with more deviant peer groups (Hampson, Andrews & Barckley, 2008; Mann et al., 2015; Yanovitzky, 2005) and has been shown to moderate the phenotypic association between peer deviance and adolescent delinquency (Mann et al., 2015). Putting these lines of evidence together, as illustrated in Figure 1, we hypothesize that (1) sensation seeking mediates genetic influences on delinquency; (2) sensation seeking mediates gene-environment correlations with peer deviance; and (3) sensation seeking moderates the effects of peer influence on delinquency. Although individual components of this model have been tested in – and supported by – previous research, the relations between sensation seeking, peer deviance, and antisocial behavior have not yet been comprehensively tested in a genetically informative study that simultaneously considers both person-environment correlations and interactions.

Figure 3. Theoretical Pathways between Genes, Sensation Seeking, Peer Deviance, and Delinquency



Note. Solid lines represent genetic pathways and dashed lines represent environmental pathways. Numbers correspond with hypotheses tested in the current study. (1) sensation seeking mediates genetic influences on delinquency. (2) sensation seeking mediates gene-environment correlations with peer deviance. (3) sensation seeking moderates the environmental effects of peer influence on delinquency.

GOALS OF THE PRESENT STUDY

We test three hypotheses. First, motivated by previous research indicating that sensation seeking is an endophenotype for delinquent behavior (Harden et al., 2012), we examine the extent to which genetic influences on sensation seeking account for genetic influences on delinquent behavior. Second, we examine the extent to which genetic influences on sensation seeking account for genetic influences on affiliation with deviant peers. We hypothesize that the genetic correlations between sensation seeking, peer deviance, and delinquency will be moderate–to-large in magnitude. Third, we examine the extent to which sensation seeking moderates the environmental association between peer deviance and delinquency, using a model that controls for *r*GE while using a co-twin-control to estimate peer influence. We hypothesize the effect of peer deviance on

delinquency will be magnified among adolescents high in sensation seeking. We test our hypotheses using multivariate behavioral genetic twin-comparison models.

METHOD

Participants

Households with identical and fraternal twins were identified using public school rosters, and families were invited by phone-call or mailing to participate in an in-laboratory study as part of the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013). Verbal and written consent was obtained from parents, and assent was obtained from adolescents prior to participation. Participants were adolescents (n = 549), ages 13-20 years (M age = 15.8 years, SD = 1.4 years), who were either enrolled in high school during the previous school year or expected to enroll in high school in the next fall semester. The sample was 52% male and 48% female. The racial composition of the sample was 59.7% non-Hispanic Caucasian, 20.3% Hispanic/Latino, 10.4% African-American, 1.0% Native American, 1.5% East Asian, 2.2% Southeast Asian and 4.8% mixed-race/other. Approximately 7% of parents did not complete high school, 7% completed no more than high school, 3% completed a vocational or technical degree, 19% attended college but did not obtain a degree, 6% completed an associate degree, and 58% a bachelor degree or higher.

Each twin was independently assessed by a different trained research assistant. Honest reporting of sensitive information (e.g. delinquent behavior) was encouraged by allowing participants to enter information directly into the computer and by notifying them that the study data was protected from disclosure by a federal certificate of confidentiality.

Zygosity Classification

Zygosity information was missing for 5 twin pairs; therefore, behavioral genetic analyses were performed on a subsample of 539 adolescent twins. Same-sex pairs were classified on the basis of self, parent and experimenter report of twin pairs` physical similarity and likelihood/frequency of being mistaken for each other. Specifically, items were completed by the twins' parents, two research assistants following the twins' lab visit, and both twins themselves. Responses to items were analyzed using latent class analysis (LCA), which assigns participants to groups (in this case, monozygotic [MZ] and dizygotic [DZ] twins). Compared to determining zygosity using molecular genetics techniques, LCA of questionnaire data has a misclassification rate of less than 1% (Heath et al., 2003). The LCA solution had an entropy statistic of 0.999, indicating very little uncertainty in classifying pairs. (Opposite-sex twin pairs were not included in LCA because they are necessarily DZ.). The sample included 255 sets of twin and 27 sets of triplets from 9 families, with each triplet family contributing three pair-wise combinations; there were 97 MZ pairs (45 female-female, 52 male-male), and 185 DZ pairs (60 female-female, 39 male-male, 86 female-male).

Measures

Sensation seeking. Sensation seeking was measured using the Brief Sensation Seeking Scale (BSSS; Hoyle, Stephenson, Palmgreen, Lorch & Donohew, 2002; Stephenson, Hoyle, Palmgreen & Slater, 2003). Adolescents rated 8 items on a scale ranging from 1 (*Strongly Disagree*) to 5 (*Strongly Agree*). Sample items include, 'I like to do frightening things', 'I prefer friends who are excitingly unpredictable' and 'I would like to explore strange places'. Descriptive statistics (*means*, *standard deviations*, and

internal consistencies) for sensation seeking and other study constructs are summarized in Table 3.

Peer deviance. Peer deviance was assessed with a self-report questionnaire adapted from Thornberry, Lizotte, Krohn, Farnworth, & Jang (1994), which asked how many friends engage in delinquent behaviors, such as skipping school and destroying property, and prosocial behaviors, like participating in school activities and getting along with teachers. All 22 items were rated on a scale ranging from 1 (*None of them*) to 4 (*All of them*). Prosocial items were reverse scored before summing item scores to form a composite scale.

Delinquency. A self-report measure adapted from Huizinga, Esbensen and Weiher (1991) was employed to assess individual differences in adolescents` delinquent behavior. The severity of delinquent behaviors ranged from minor offenses to relatively severe crimes, including "skipped class or school without an excuse" and "made obscene telephone calls (calling someone and saying dirty things)", or "sold drugs such as heroin, cocaine, LSD, ecstasy or prescription pills" and "been involved in a group fight or gang fight." 36 items were assessed on a 3-point scale (1 = *Neve*r, 2 = *Once*, 3 = *More than once*) and summed to form a composite scale.

RESULTS

Table 3. Descriptive Statistics and Zero-Order Correlations

N = 547	α	*M (SD)*	Age	SS	PD	DEL
Age	-	15.82 (1.45)	1.0	.09*	.08	.35**
SS	.72	3.20 (.69)		1.0	.31**	.45**
PD	.86	1.73 (.33)			1.0	.50**
DEL	.87	6.66 (7.26)				1.0

Effects of Biological Sex on Study Variables

	t	*df*	*p*	*d*	Male *M (SD)*	Female *M (SD)*
Age	-0.97	547	.33	-.08	15.76 (1.35)	15.88 (1.54)
SS	1.85	538	.06	.16	3.25 (0.70)	3.14 (0.68)
PD	4.56	521	< .01	.40	1.79 (0.33)	1.67 (0.32)
DEL	3.78	515	< .01	.33	7.80 (7.88)	5.49 (6.38)

Note. Means (M) and standard deviations (SD) reported for untransformed variables. Correlation coefficients and test statistics reported for transformed variables. N = total number of participants. α = Cronbach's alpha. SS = sensation seeking. PD = peer deviance. DEL = delinquent behavior. *t* = two-sample test statistic. *df* = degrees of freedom. *p* = two-tailed probability. *d* = standardized difference between means (Cohen's d). * $p < .05$ ** $p < .01$

Prior to fitting univariate and multivariate twin models, internal consistencies (Cronbach's alpha), descriptive statistics (means and standard deviations) and phenotypic associations (*t*-tests and zero-order Pearson correlations) were calculated using *R* version 3.1.2 (R Core Team, 2015) (see Table 3). Behavioral genetic analyses were conducted within a structural equation modeling (SEM) framework using M*plus* version 7.1 (Muthén & Muthén, 1998-2010). For phenotypic analyses, participants with missing data were excluded pairwise. For behavioral genetic analyses, the full dataset was analyzed with missing data estimated using full information maximum likelihood (Enders & Bandalos, 2001). The delinquency scale was log-transformed to correct for positive skew. Standard errors and fit statistics were adjusted for nonindependence of data (Asparouhov & Muthén, 2006), which was necessary in behavioral genetic models because pair-wise combinations of triplets were nested within the same household. MZ and DZ twin-pair

correlations and SEM parameters control for the main effects of Caucasian, Hispanic and African American race, age, sex, and age × sex interaction (McGue & Bouchard, 1984). All models were estimated using maximum likelihood with robust standard errors, and model fit was evaluated using model χ^2, root mean squared error of approximation (RMSEA) and comparative fit index (CFI). Nested models were compared using Satorra-Bentler scaled chi-squared difference tests (Satorra & Bentler, 2001). Non-nested models were compared using Akaike Information Criteria (AIC).

Univariate Twin Models. Twin models were fit as SEMs that decomposes total observed variance in a construct into three components: additive genetic variance (*A*), shared environmental variance (*C*), and nonshared environmental variance, plus measurement error (*E*). Heritability is the proportion of total variance in a phenotype attributable to additive genetic differences between individuals, i.e., the *A* variance. Heritability is inferred from the extent to which MZ twins correlate higher on that phenotype than do DZ twins. Shared environmental variance includes factors that occur at the family-level that make sibling similar to one another, like socio-economic status, culture and religion. Shared environmental factors are inferred from the extent to which twin correlations are higher than can be explained by genetics alone. Non-shared environmental variance, on the other hand, refers to factors uniquely experienced by siblings that make them different from one another (e.g. peer groups). Non-shared environmental factors are inferred from the extent to which identical twins raised together do not perfectly resemble one another on the phenotype.

With respect to model specification, MZ and DZ cross-twin correlations between *C* factors and between *E* factors are fixed to 1.0 and 0, respectively. For MZ twins, the

cross-twin correlation between *A* factors are fixed to 1.0, reflecting the fact that MZ twins share approximately 100% of their segregating genes. For DZ twins, the cross-twin correlation between *A* factors is fixed to .50 because they share approximately 50% of their segregating genes.

Univariate ACE models were fit to each of the focal study variables (sensation seeking, peer deviance and delinquency) and parameter estimates and model fit statistics can be found in Table 4. The MZ twin-pair correlation (*r*MZ = .449) for sensation

Table 4. Twin-Pair Correlations and Results from Univariate Twin Models.

	Twin Correlations	
Variable	***r*MZ**	***r*DZ**
SS	.45 (.32, .58)	.18 (.03, .33)
PD	.56 (.42, .71)	.33 (.19, .47)
DEL	.59 (.43, .74)	.37 (.19, .47)

	Standardized Parameter Estimates from ACE Models		
Variable	**A**	**C**	**E**
SS	.43 (.31, .55)	.00 (.00, .00)	.56 (.45, .69)
PD	.30 (.01, .60)	.00 (.00, .00)	.70 (.40, .99)
DEL	.42 (.03, .81)	.14 (.00, 44),	.44 (.29, .59)

	Standardized Parameter Estimates from ADE Models		
Variable	**A**	**D**	**E**
SS	.22 (.00, .82)	.24 (.00, .88)	.54 (.42, .66)
PD	.30 (.01, .60)	-	.70 (.40, .99)
DEL	.58 (.45, .70)	-	.42 (.30, .55)

Model Comparison Results

Variable	**Model**	**AIC**	**RMSEA**	**CFI**	χ^2	***df***	***c***	**Nested Comparison**	$\Delta\chi^2$	***p***	**Preferred Model**
SS	ACE	1538.9	.00	1.00	4.27	6	1.20				
	ADE	1538.3	.00	1.00	4.42	6	1.03				
	AE	1536.9	.00	1.00	4.99	7	1.03	ADE vs. AE	.57	.44	AE
PD	ACE	1480.1	.05	.938	7.47	5	1.39				
	AE	1478.1	.05	.926	8.96	6	1.16	ACE vs. AE	.00	.96	AE
DEL	ACE	1457.2	.00	1.00	4.21	6	0.88				
	AE	1455.9	.00	1.00	5.01	7	0.89	ACE vs. AE	.78	.37	AE

Note. Sibling contrast effects were modeled for peer deviance. 95% confidence intervals are reported in parentheses. SS = Sensation Seeking. PD = Peer Deviance. DEL = Delinquency. *r*MZ = monozygotic twin-pair correlation. *r*DZ = dizygotic twin-pair correlation. A = Additive genetics. C = Shared environment. E = Non-shared environment. D = Dominance genetics. AIC = Akaike Information Criteria. *df* = degrees of freedom. *c* = scaling correction factor for model chi-squared. Differences between χ^2values obtained using maximum likelihood estimation with robust standard errors are not themselves χ^2 distributed. Therefore, model χ^2values with scaling correction factors were used to compute Satorra-Bentler χ^2 tests for nested model comparisons.

seeking was more than double the DZ twin-pair correlation (*r*DZ = .176), indicating possible dominance genetic effects[2]. However, although a model that included dominance genetic effects in place of shared environmental effects for sensation seeking showed better fit (AIC = 1538.295) than an ACE model (AIC = 1538.873), an ADE model did not fit significantly better than a more parsimonious AE model that only included additive genetic and non-shared environmental effects (χ^2 diff. = .569, p = .440 for nested model comparison). The preferred model (AE), which showed good fit to the data (model $\chi^2 = 4.896$, $df = 7$, p = .662, RMSEA = .000, CFI = 1.00), was therefore selected and carried forward to multivariate analyses.

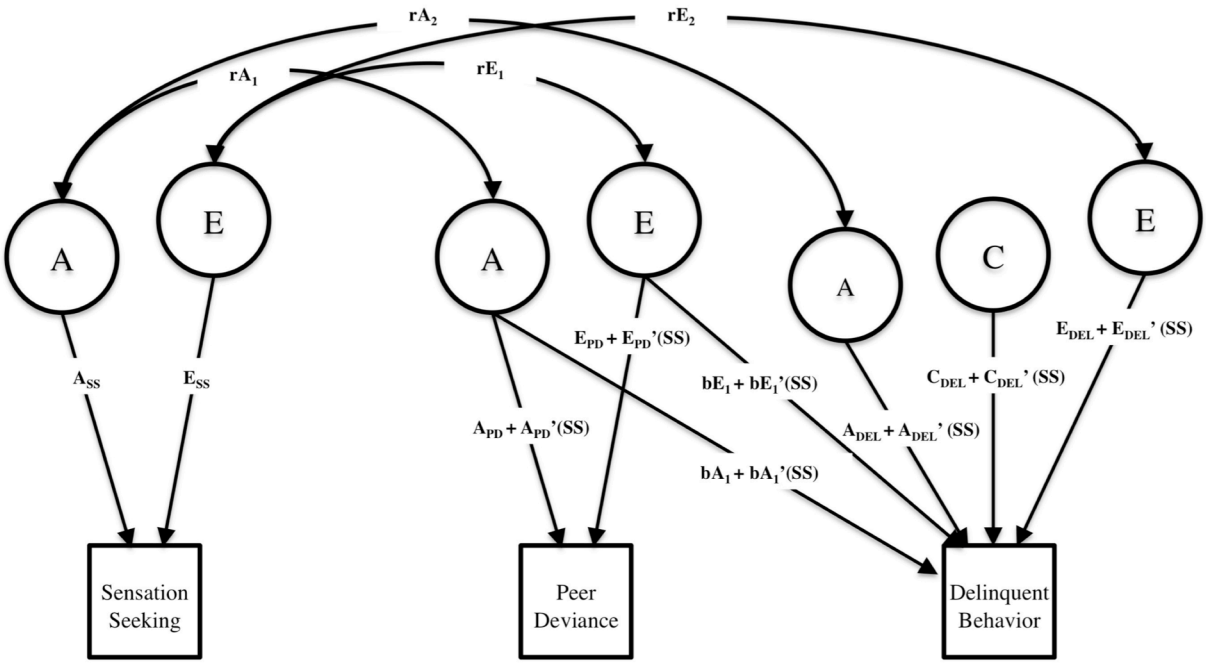
A similar procedure was used for selecting univariate twin models of peer deviance and delinquency (see Table 4). The ACE model of adolescent delinquency showed good fit to the data (model $\chi^2 = 4.213$, df = 6, p = .647, RMSEA = .000, CFI = 1.000). Although nested model comparisons indicate that omitting estimates of shared environmental influences did not result in significant misfit to the data, past behavior genetic research has emphasized the importance of shared environmental influences on a range of externalizing and antisocial behaviors, particularly rule-breaking behaviors (Burt, Krueger, McGue & Iacono, 2001; Burt, Drueger, McGue & Iacono 2006; Burt,

[2] At any particular locus (i.e. a specific location on a chromosome) there are two copies of a particular version of a gene, called alleles. One allele is inherited from one's mother and the other from one's father. If an individual inherits different alleles from each parent, then they are *heterozygous* (Aa) on that particular gene. If an individual inherits the same alleles from each parent, then they are either *homozygous recessive* (aa) or *homozygous dominant* (AA) on that gene. For phenotypes influenced by *additive genes*, the presence of one allele does *not* alter the effects of the other allele at the same locus. For phenotypes influenced by dominant genes, one allele– i.e. the dominant one –masks the effects of the second allele– i.e. the recessive one. Thus, for additive genes, individuals who are heterozygous (Aa) express a phenotype that is intermediary between individuals who are homozygous recessive (aa) and homozygous dominant (AA). For dominant genes, however, individuals who are heterozygous (Aa) express the same phenotype as those who are homozygous dominant (AA); the alternate phenotype is only expressed when two recessive alleles are inherited– i.e. homozygous recessive (aa). In terms of twin-pair correlations, compared to what is predicted under an exclusively additive model, dominance genetic effects *decrease* the phenotypic similarity of dizygotic twins (who *are not* always matched on the other allele), but not monozygotic twins (who are guaranteed to be matched on the other allele).

McGue & Krueger, 2007; Burt, McGue, Iacono, 2010). Therefore, estimates of shared environmental influences on delinquency were included in multivariate analyses. An initial ACE model of peer deviance showed relatively poor fit to the data (model $\chi^2 = 10.939$, $df = 6$, $p = .093$, RMSEA = .076, CFI = .876). Therefore, we fit a second ACE model that also estimated sibling effects (Carey, 1986), which improved model fit (model $\chi^2 = 7.465$, $df = 5$, $p = .188$, RMSEA = .059, CFI = .938). Sibling effects are parameterized in terms of two regression coefficients (Twin 1's phenotype on Twin 2's phenotype, and Twin 2's phenotype on Twin 1's phenotype) that are constrained to be equal in magnitude. Conceptually, these regressions posit that twins are similar to one another, not just because of shared genes and shared environmental influences, but also because they directly influence each other. Given that teenage twins likely share friends, this twin-to-twin influence is particularly plausible for peer deviance. Mathematically, sibling effects imply that the variances of the phenotypes will differ in MZ versus DZ twins, as was observed in this sample (MZ $s^2 = 1.260$, DZ $s^2 = 0.797$). Similar to sensation seeking, predictive fit indices and nested model comparisons indicated that AE models were preferred over ACE models. Consequently, for peer deviance, an AE model with sibling effects was carried forward to multivariate analyses.

Multivariate Twin Model: Gene-Environment Correlation and Interaction. First, to provide a baseline model for comparison and to assess whether peer deviance has a main environmental effect on delinquent behavior after genetic and shared environmental factors are controlled (i.e., a within-MZ twin-pair association), we fit a multivariate twin model.

F*igure 4*. Twin Model of Sensation Seeking, Peer Deviance and Delinquent Behavior



Note. Person-environment correlations and person-environment correlations are depicted for only one twin per pair. *A* = additive genetics. *C* = shared environment. *E* = non-shared environment. SS = sensation seeking. Sibling effects were modeled for peer deviance, but omitted from presentation.

This model showed adequate fit to the data (model $\chi^2 = 56.182$, $df = 37$, $p = .023$, RMSEA = .061, CFI = .935, AIC = 4290.001, BIC = 4351.913). The association between peer deviance and delinquency is divided into two parts. The genetic cross-path (bA_1) between peer deviance and delinquency tests whether adolescents who are genetically predisposed to select more deviant peers also show higher delinquency. In contrast, the environmental cross-path between peer deviance and delinquency (bE_1) tests whether identical twins who differ in peer deviance also differ in their levels of delinquency. This within-twin comparison controls for potential genetic and environmental confounds that are shared by twins raised in the same family. This model also simultaneously accounts for genetic and environmental correlations with sensation seeking. The genetic (bA_1 =

.533, CI.95 = .306, .761, $p < .001$) *and* the environmental (bE_1 = .144, CI.95 = .054, .234, p = .002) cross-paths from peer deviance to delinquency were positive and significant. This suggests that adolescents who are genetically predisposed to select more deviant peers also show higher levels of delinquency. Moreover, after controlling for these genetic selection effects, the environmental effect of peer deviance on adolescent delinquency remains.

The multivariate twin model described above was then expanded to include interaction terms, such that the components of variance in peer deviance and adolescent delinquency, as well as the genetic and environmental cross-paths between peer deviance and delinquency (bA_1 and bE_1), were allowed to interact with individual differences in sensation seeking. This model is designed to test for G×E interaction in the presence of rGE (Rathouz, Van Hulle, Rodgers, Waldman & Lahey, 2008) and is depicted in Figure 4. Note that the primary pathways of interest (i.e., genetic and environmental cross paths between peer deviance and adolescent delinquency) now include two freely estimated parameters: a main effect (bA_1 and bE_1) and an interaction with sensation seeking (bA_1 'and bE_1').

This interaction model fit significantly better than the previous model (χ^2 difference = 41.473, df = 7, $p < .001$, AIC = 4246.050, BIC = 4332.323), which did not allow genetic and environmental variance in peer deviance and adolescent delinquency to interact with individual differences in sensation seeking. Parameter estimates and confidence intervals from the interaction model are reported in Table 5.

The genetic (A_{PD}' = -.182, p = .002) and environmental (E_{PD}' = .562, $p < .001$) components of peer deviance were moderated by individual differences in sensation

Table 5. **Unstandardized Parameter Estimates from Model of Person-Environment Interaction and Correlation**

Regression Parameters	***b***	***(CI.95%)***
Variance in Sensation Seeking		
Main genetic effect (A_{SS})	.632	(.522, .742)
Main nonshared environmental effect (E_{SS})	.769	(.683, .855)
Variance in Peer Deviance		
Main genetic effect (A_{PD})	.794	(.695, .894)
Gene × Sensation Seeking interaction (A_{PD}')	-.182	(-.299, -.064)
Main nonshared environmental effect (E_{PD})	.582	(.489, .675)
Gene × nonshared environmental interaction (E_{PD}')	.562	(.468, .656)
Peer Deviance → Delinquency		
Main genetic path (bA_1)	.357	(.206, .509)
Gene × sensation seeking interaction (bA_1')	-.098	(-.194, -.002)
Main nonshared environmental path (bE_1)	.191	(.083, .299)
Nonshared environment × sensation seeking interaction (bE_1')	.214	(.120, .308)
Variance in Delinquency Unique of Peer Deviance		
Main genetic effect (A_{DEL})	.642	(.506, .779)
Gene × Sensation Seeking interaction (A_{DEL}')	-.020	(-.126, .087)
Main shared environmental effect (C_{DEL})	.000	(-.001, .001)
Shared Environmental × Sensation Seeking interaction (C_{DEL}')	.000	(.000, .000)
Main nonshared environmental effect (E_{DEL})	.623	(.532, .714)
Gene × nonshared environmental interaction (E_{DEL}')	.033	(-.069, .135)

Correlation Coefficients	***r***	***(CI.95%)***
Sensation Seeking & Peer Deviance		
Additive genetic (rA_1)	.415	(.256, .575)
Non-shared environmental (rE_1)	.068	(-.084, .219)
Sensation Seeking & Delinquency		
Additive genetic (rA_2)	.534	(.286, .783)
Non-shared environmental (rE_2)	.181	(-.003, .366)

Note. *b* = unstandardized parameter estimates. r = correlation coefficients. CI.95% = 95% confidence intervals.

seeking. The *E* component of peer deviance represents the extent to which identical twins differ in their experience of peer deviance, whereas the *A* component represents the extent to which identical twins experience more similar peer environments than fraternal twins. Therefore, the positive moderation of the *E* path by sensation seeking, in conjunction with the negative moderation of the *A* path, indicates that highly sensation seeking twins experience more differentiated and idiosyncratic peer environments.

Additionally, individual differences in sensation seeking significantly moderated the environmental (bE_1' = .214, $p < .001$) and genetic (bA1' = -.098, $p = .046$) paths

between peer deviance and adolescent delinquency, such that the environmental and genetic effects of peer deviance on delinquency increased and decreased, respectively, among high sensation-seeking adolescents. Moderation of the environmental (*E*) path between peer deviance and adolescent delinquency is consistent with our main hypothesis that peer influence will be magnified among adolescents high in sensation seeking.

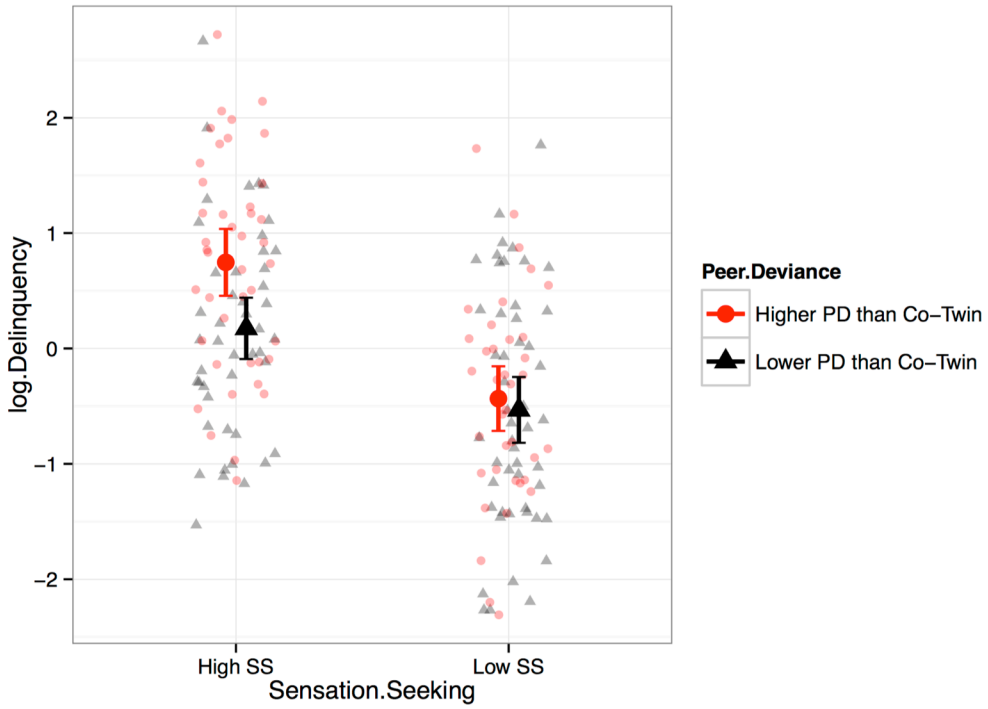
This *E* moderation effect is illustrated in Figure 5, which shows individual-level data on delinquency from MZ twins. Each twin was classified according to whether he or she reported higher or lower peer deviance *than his or her identical twin* ("Higher PD than Co-Twin" [plotted using black triangles] or "Lower PD than Co-Twin" [plotted using red circles]). In addition, each twin was classified as either "High Sensation Seeking" or "Low Sensation Seeking" based on whether he or she was above or below the sample mean for sensation seeking. As shown in Figure 5, regardless of peer deviance, adolescents who reported "Low Sensation Seeking" were clearly less delinquent than adolescents who reported "High Sensation Seeking." Moreover, the *difference* between the "Higher PD than Co-Twin" and "Lower PD than Co-Twin" groups is the difference in delinquency between identical twins who are discordant for peer deviance. This within-MZ-twin-pair difference represents the *E* pathway from peer deviance to delinquency in the structural equation model. Consistent with the results from the interaction model reported in Table 5, identical twins who experience differing levels of peer deviance show corresponding differences in delinquent behavior only at high levels of sensation seeking and not at low levels of sensation seeking.

We are hesitant to interpret the interaction between the *A* path from peer deviance of delinquency, because we did not have an a priori hypothesis regarding this moderation

effect and because the significance of the parameter was very close to $p = .05$. Nevertheless, we can consider the implications of the negative genetic interaction in conjunction with the positive (and larger) non-shared environmental interaction. Compared to adolescents who are low in sensation seeking, the total association between peer deviance and sensation seeking is stronger in adolescents who are high in sensation seeking. Moreover, the relative balance of genetic versus non-shared environmental processes shifts. Among adolescents who are low in sensation seeking, the relationship is almost entirely accounted for by genetic selection, whereas among adolescents who are high in sensation seeking the association between peer deviance and delinquency is less reflective of genetic selection and more reflective of an environmental pathway.

To summarize results, we found that the genes that influence sensation seeking overlap with genetic influences on affiliation with deviant peers (*r*GE). In a co-twin control design, identical twins who differ in peer deviance also differ in delinquency, indicating an environmental effect of peers on delinquency. Finally, as hypothesized, this environmentally-mediated peer effect was moderated by individual differences in sensation seeking, a person × environment interaction.

Figure 5. Individual-Level Data on Delinquency by Sensation Seeking and Within-MZ-Twin-Pair Differences in Peer Deviance.



Note. Small dots and triangles represent individual-level data. Large dots and triangles represent means. Error bars represent ±2 SEs around the mean. Only data from MZ twins are presented. "Higher PD than Co-Twin" (plotted using red circles) or "Lower PD than Co-Twin" (plotted using black triangles) were classified based on whether the individual reported higher or lower peer deviance than his/her identical co-twin. "High Sensation Seeking" or "Low Sensation Seeking" was classified based on whether the individual reported sensation seeking above or below the sample mean.

DISCUSSION

This article reports results of the first genetically informative study of sensation seeking, peer deviance, and antisocial behavior to consider simultaneously both person-environment correlations and interactions. Our findings build on previous behavioral genetic research on peers and externalizing behaviors, which has found evidence for both selection and socialization processes (Boardman, Saint Onge, Haberstick, Timberlake, & Hewitt, 2008; Button et al., 2007, 2009; Cruz, Emery, & Turkheimer, 2012; Fowler et al., 2007; Harden et al., 2008). More genetically similar individuals (e.g. identical twins) tend to experience more similar peer environments than do less similar individuals (e.g.

fraternal twins), indicating that genetically-influenced traits lead adolescents to systematically select and attract peers that behave similar to themselves. We find that, even after controlling for this gene-environment correlation, even identical twins who differ in their peer environments show differing levels of involvement in delinquent behavior, a result that is consistent with a socializing effect of peers. Our findings also build on past behavior genetic and molecular genetics research indicating gene × peer interactions on delinquency and closely related behaviors, such as substance use (Guo, Elder, Cai, & Hamilton, 2009; Harden et al., 2008; Hicks et al., 2009). Our study implicates sensation seeking as a genetically-influenced trait that underlies individual differences in vulnerability to peer influence. Previous work in both quantitative genetics and molecular genetics has found evidence that genetic variants influencing antisocial behavior are mediated by sensation seeking (Aliev et al., 2014; Harden et al., 2012; Salvatore et al., 2015), but no previous study that we are aware of has examined sensation seeking as a mechanism of both gene-environment correlation and interaction with respect to peer deviance and delinquency. Thus, the current study synthesizes multiple sets of findings from previous work to establish that (a) sensation seeking is an endophenotype that shares a considerable proportion of the genetic influence on delinquent behavior, (b) peer deviance is associated with delinquent behavior even when common genetic and shared environmental influences are controlled, and (c) the environmentally-mediated effect of peer deviance on adolescent delinquency is moderated by sensation seeking.

Avenues for Future Research

Two critical questions for ongoing research on adolescent delinquency (and for all complex, heritable phenotypes) are (1) what are the specific genetic variants that constitute the heritable variation, and (2) what are the intermediary biological pathways that translate genetic differences into phenotypic differences. On the one hand, discovering a specific genetic variant yields new insights regarding the underlying biology of a phenotype. For example, the discovery that a polymorphism in *CACNA1C* is reliably associated with schizophrenia in genome-wide association studies has focused attention on understanding the role of calcium channel signaling in the pathophysiology of serious psychiatric diseases (Curtis et al., 2011; Nyegaard et al., 2010). On the other hand, understanding the underlying neurobiology of a phenotype can point to candidate gene sets. For example, on the basis of research showing that long-term learning depends on proteins in excitatory neuronal synapses, Hill et al. (2014) tested associations between general intelligence and a candidate set of genes coding for these proteins. Discovering specific genes and delineating the pathways from genotype to adolescent delinquency and its precursor traits, such as sensation seeking, are mutually informative enterprises that will ultimately help to propel our understanding of how biological factors intersect with social contexts to shape adolescent delinquency and related externalizing behaviors.

Our results suggest new avenues for research in humans and in animal models to address these questions. For example, Roman high- and low-avoidance (RHA-I vs. RLA-I) rat lines are inbred animals that differ in sensation-seeking and novelty-seeking behavior (Giorgi, Pirsa, & Corda, 2007). In a study using brain tissue to examine differences in gene expression, 38 genes were found to be either up-regulated or down-

regulated in the RLA-I rats versus RHA-I rats, including *PRL* (which codes for prolactin) and *CRHBP* (which codes for corticotrophin releasing hormone binding protein; Sabariego et al., 2011). The finding that much of the genetic influence on delinquency is mediated via genetic influences on sensation seeking may indicate that these genes and the hormonal systems that they regulate may be promising targets for genetic association studies of delinquent behavior in humans.

Additionally, future research using cognitive neuroscience might test the hypothesis that the neural bases of sensation seeking mediate genetic influences on delinquency. Research in developmental cognitive neuroscience has begun to illuminate the neurobiological underpinnings of developmental changes in sensation seeking. For example, adolescents show stronger activation of the ventral striatum in response to rewards, such as money, than do either children or adults (Galvan, 2010). Ventral striatum activity, in turn, is positively correlated with risk-taking on laboratory tasks, preference for immediate rewards, and self-reported delinquency and substance use (Galvan, 2010; Geier, 2013). In addition to rewards, the ventral striatum (along with the amygdala) has also been shown to respond to threatening cues in some adolescents, a response that is associated with increasing testosterone levels (Spielberg, Olino, Forbes, & Dahl, 2014). This pattern of neural activation might underlie thrill-seeking tendencies, in which physically dangerous activities are experienced as rewarding. Synthesizing these findings from developmental cognitive neuroscience with the behavioral genetic findings reported here suggest the interesting hypothesis that ventral striatal function may be a key neurobiological mediator between genotype and delinquency phenotype (Harden & Mann, 2015).

Measuring Peer Influence

Peer influence can be operationalized in a variety of ways. One limitation of the current study is that we rely on adolescents' reports of their peers' behaviors. Previous research has shown that teenagers tend to both overestimate their peers' involvement in socially deviant activities (Prinstein & Wang, 2005) and to overestimate their peers' similarity to themselves (Buaman & Ennett, 1996). Additionally, participants may have differed in how broadly or narrowly they construed the prompt about their "friends," either considering only intimate relationships or counting even casual acquaintances. Nevertheless, the current results converge with results from previous twin research on peer influence that used sociometric nominations to define peer groups and that used peers' reports on their own behavior (Harden et al., 2008; Cruz et al., 2012). In addition, our measure of peer deviance includes items asking about peer involvement in deviant behaviors (e.g., "used force to get money or things", "sold drugs") and (reverse-coded) items asking about prosocial behaviors (e.g., "has been involved in school activities/athletics", "gets along well with teachers at school"). Thus very low scores on the peer deviance measure represents affiliation with peers who are engaged in a variety of normative and constructive activities.

No cross-sectional study can definitively ascertain direction of causation. We have interpreted the correlation between peer deviance and delinquency in terms of the socializing effects of peers on the (twin) target, but socializing influences are reciprocal and dynamic. An additional (and not mutually exclusive) interpretation of our data is that highly sensation seeking adolescents are particularly influential in shaping the behaviors of their friends. It is true that adolescents who engage in minor delinquency and

substance use are more popular (Allen, Porter, McFarland, Marsh, & McElhaney, 2005), consistent with the idea that pseudo-adult behavior is a "coveted social asset" (Moffitt, 1993). However, in laboratory studies where susceptibility to peer influence in operationalized in terms of changing one's opinion in negotiation with a close friend, socially influential teenagers actually showed less engagement in delinquent behavior than did teenagers who were highly susceptible to peer influence (Allen, Porter, & McFarland, 2006). Ultimately, disentangling the impact of sensation seeking on one's susceptibility to peer influence versus the strength of one's influence on others will require longitudinal research that pays close attention to the developmental contexts, in addition to the broader social contexts, in which individuals are embedded.

Considering Other Sub-Types of Antisocial Behavior

Our measure of delinquent behavior inquired about involvement in mostly non-aggressive, rule-breaking acts (e.g., "purposely damaged or destroyed property that did not belong to you", "painted graffiti or signs on someone else's property or in a public place", etc.). Moreover, in our school-based, community sample of teenagers, we expect that most individuals who are currently engaging in delinquent acts will desist from antisocial behavior as they transition to adulthood. Importantly, the etiology of adolescent-limited, rule-breaking antisocial behavior has been found to differ from other, more serious and persistent, subtypes of ASB defined by childhood-onset behavior problems, aggression, and callous-unemotionality (Burt, 2012; Harden et al., *in press*; Viding, Jones, Paul, Moffitt, & Plomin, 2008). It is unclear whether the results observed in this study (high genetic correlations with sensation seeking and environmentally-

mediated effects of peer deviance) will generalize to other subtypes of antisocial behavior. This remains an interesting question for future research.

Conclusions

In sum, the current study combines insights from two separate lines of research, the first of which has examined sensation seeking as psychological mediator of genetic influences on delinquent adolescent behavior, and the second of which has examined gene × peer context interactions on delinquency. We find that that genetic influences on sensation seeking predispose adolescents to affiliate with deviant peers and engage in delinquent behavior themselves. But, even after controlling for this gene-environment correlation using a co-twin-comparison, peer deviance remains associated with adolescent delinquency. Further, we find that sensation seeking moderates the environmental effect of peer deviance on delinquency. Compared with low sensation-seeking adolescents, high sensation-seekers are more vulnerable to the social influence of their peers. These results are consistent with both selection and socialization processes contributing to delinquent behavior in adolescence, particularly mediated through individual differences in sensation seeking.

Chapter 3. Delineating Heterogeneity in Antisocial Behavior: Sensation Seeking as an Index of Multi-Dimensional Risk

HETEROGENEITY IN ANTISOCIAL BEHAVIOR

Antisocial behaviors are socially deviant behaviors that are outwardly expressed and disruptive to others. When persistent and severe, antisocial behaviors are considered symptoms of externalizing disorders, including substance-use disorder and conduct disorder. Lying to parents and skipping class, on the other hand, are relatively normal expressions of antisocial behavior that, on their own accord, do not warrant clinical attention. Antisocial behaviors also vary according to whether they victimize others. For example, substance-use may be considered antisocial because it is illegal but, it doesn't necessarily cause direct harm to others. On the other hand, physical assault is antisocial because it victimizes others. Other antisocial behaviors, such as reckless driving, may or may not victimize others depending on happenstance (e.g. whether a reckless driver is carrying a passenger or collides with another vehicle). Nevertheless, antisocial behaviors "involve the violation of the rights of others (e.g., stealing, physical aggression, destruction of property) or the violation of major societal norms (e.g., lying, running away from home)" (Frick et al., 2014, p.532). Reflecting this broad conceptualization, it is not uncommon for researchers to operationalize antisocial behavior using self-report measures that include heterogeneous items, capturing a diverse array of individual differences (e.g. Huizinga et al., 1991). In efforts to delineate heterogeneity in antisocial behavior, a number of useful distinctions have been proposed. Prominent examples

include time of onset and desistance (Moffitt, 1993), as well as antisocial behavior in the presence of callous-unemotional traits (Frick, Ray, Thorton & Kahn, 2014).

AGGRESSION VS. RULE-BREAKING DISTINCTION

Another useful method for parsing heterogeneity in antisocial behavior centers upon the distinction between aggressive behavior and non-aggressive rule-breaking (Burt 2009; 2013). Aggressive behavior either directly or indirectly causes harm to others (e.g. physical assault or threatening others). Conversely, non-aggressive rule-breaking behaviors do not victimize others but, involve the violation of parental expectations or societal norms (e.g. disobeying household rules, truancy or under-age drinking). Despite being moderately correlated (Burt, 2013), there is considerable evidence that rule-breaking and aggression are independent constructs. For example, the rank-order stability and mean prevalence rates of aggression and rule-breaking vary across development. The stability of rule-breaking is low in childhood and then increases in adolescence. In contrast, aggression is more stable throughout childhood, adolescence and adulthood (Burt, 2012). Furthermore, average levels of rule-breaking increase in adolescence (e.g. teenagers are more often delinquent than toddlers or adults), whereas average levels of aggression typically peak in early childhood (e.g. toddlers pull hair and bite more often than teenagers and adults).

In addition to evincing differential phenotypic expression across development, there is also substantial evidence that the etiology of aggression is different from non-aggressive rule-breaking. For example, aggression tends to be more heritable than rule-breaking, and rule-breaking is more susceptible to shared environmental influences (Burt, 2009). Moreover, the heritability of aggression remains stable from childhood to

adolescence, whereas the heritability of rule-breaking increases with adolescent age and pubertal development (Burt & Klump, 2009; Burt & Neiderhiser, 2009; Harden et al., 2015). In addition to differential patterns of etiology and phenotypic expression, discriminate validity is further supported by differentiated social and personality correlates between these subtypes of antisocial behavior (Tackett, Daoud, De Bolle & Burt 2013). For example, a recent study (Mann, Tackett, Tucker-Drob & Harden, 2017) found that high levels of callous-unemotional traits magnify genetic risk only for aggressive antisocial behavior. In contrast, environmental influences on rule-breaking behavior were magnified among adolescents with high levels of callous-unemotional traits. Moreover, in contrast to rule-breaking, there is evidence that aggression is uniquely associated with high levels of disagreeableness, internalizing symptoms and social problems (Tackett, Daoud, De Bolle & Burt 2013).

SENSATION SEEKING: MULTIPLE DIMENSIONS OF RISK

Sensation seeking has traditionally been defined as the willingness to take risks (physical, social or financial) to pursue novel and intense experiences (Zuckerman, 1994, p. 27). An established body of research indicates that sensation seeking predicts, not only antisocial behavior but, a wide range of deviant behaviors that constitute the externalizing spectrum (Krueger et al., 2002). For example, there is evidence that sensation seeking predicts alcohol-use (Hittner & Swickert, 2006; MacPherson et al., 2010), substance-use (Kaynak et al., 2013; Quinn & Harden, 2013), risky sexual behavior (Hoyle, Fejfar & Miller, 2000; Roberti, 2003; Dir, Costunpinar & Cyders, 2014), reckless driving (Jonah, 1997), aggression (Wilson & Scarpa, 2011) and criminal behavior (Newcomb & McGee, 1991). A common theme that unites these related, yet distinct, behaviors is risk-taking.

Put differently, despite considerable heterogeneity, all antisocial behaviors require that an individual put her or himself at risk, whether it is risk of physical harm, social condemnation or legal sanction.

Consistent with this notation, the dual-systems model (Steinberg, 2008; Shulman et al., 2016) posits developmental changes in sensation seeking as a major contributor to the spike in risk-taking behavior that is typical of adolescence. This model highlights age-groups differences in the neurobiological maturity of cognitive control and incentive processing constructs, which explain why risk-taking is more prevalent during adolescence than any other point in the lifespan. In support of the dual systems model, cross-sectional and longitudinal studies have demonstrated that, on average, sensation seeking increases across adolescence (Steinberg et al., 2008; Harden & Tucker-Drob, 2011), and adolescents who show rapid increases in delinquency and substance-use are those who show increases in tandem with sensation seeking (Quinn & Harden, 2012; Harden, Quinn, & Tucker-Drob, 2012). Thus, the dual systems model has the potential to provide a theoretical and empirical framework for understanding the omnibus effect of sensation seeking across multiple dimensions of antisocial behavior.

Although the dual systems model has focused primarily on the neurobiological correlates of age-group differences in cognitive control and incentive processing, a number of measures have been used to operationalize these constructs, including self-report measures of sensation seeking and impulsivity (Whiteside & Lynam, 2001), as well as a number of computerized tasks including a delayed discounting task (Steinberg et al., 2009), the Balloon Analog Risk Task (BART; Lejuez, Aklin, Daughters, Zvolensky, & Kahler, 2007) the Iowa Gambling Task (IGT; Bechara, 2007; Reynolds,

Ortengren, Richards, & de Wit, 2006), and the Tower of London (Shalice, 1982). Although these measures have been commonly interpreted as indicators of cognitive control or incentive processing, a recent study (Harden et al., 2016) found evidence that a large battery of such measures index four latent constructs: *premeditation, fearlessness, reward seeking, and cognitive dyscontrol*. Adolescents high on *premeditation* consider the outcomes of future actions, plan for future events, and make decisions carefully. In Harden et al., (2016), this construct was defined predominately by self-report measures of impulsivity and future orientation. Defined by self-report measures of risk perception, urgency and sensation seeking, adolescents high on *fearlessness* report not being frightened by negative consequences, believe that advantages of risks outweigh costs, and tend to prefer exciting and potentially dangerous experiences. Adolescents high on *reward seeking* are motivated by the possibility of rewards and are willing to take risks in order to gain rewards. This construct was defined by a profile of "risky play" across behavioral measures (sans the Tower of London). For example, high reward-seekers play more cards from both "good decks" and "bad decks" in the IGT and tend to "drive" through risky intersections in the Stoplight Task. Finally, the cognitive dyscontrol construct is closely related to I.Q. and captures tendencies to act rashly, think little about the future, discount delayed rewards, and perform poorly on objective task-based measures (e.g. excess moves- i.e. errors -in the Tower of London).

Of particular interest to the present study, self-reports of sensation seeking loaded positively onto fearlessness ($\lambda = .21$, $p < .001$) and reward seeking constructs ($\lambda = .16$, $p < .001$), as well as negatively onto the premeditation construct ($\lambda = -.22$, $p < .001$; Harden et al., 2016). This pattern of factor loadings suggests that self-reports of sensation seeking

capture variance in multiple dimensions of personality risk, which may be relevant to understanding the omnibus effects of sensation seeking across multiple dimensions of antisocial behavior. For instance, sensation seeking may be associated with aggression because individuals high on sensation seeking tend to be fearlessness. In contrast, the association between rule-breaking and sensation seeking may be due to the fact that high sensation-seekers tend to have difficulties with premeditation. Then again, sensation seeking may be uniquely associated with different dimensions of antisocial behavior, above and beyond indirect effects that may be channeled through related dimensions of risk.

In sum, sensation seeking captures multiple dimensions of risk for antisocial behavior in two respects. Self-report measures of sensation seeking predict multiple dimensions of antisocial behavior *and* capture variation across multiple dimensions of risk for antisocial behavior.

GOALS OF THE PRESENT STUDY

The overarching goal of the present study is to examine associations between measures of risk-taking and individual differences in aggression and rule-breaking behavior. Specifically, we aim to answer the following questions: (1) Are self-report and behavioral measures of risk-taking associated with antisocial behavior (i.e. aggression and rule-breaking)? (2) Are individual measures of risk-taking, or the latent constructs they index, more strongly associated with antisocial behavior? (3) Are associations between sensation seeking and antisocial behavior mediated by latent risk-taking constructs? (4) Are associations between latent risk-taking constructs and antisocial behavior mediated through heritable or non-heritable pathways? To answer these

questions, we analyze data from a large, population-representative sample of adolescent twins.

METHOD

Participants

The present study uses data from self-report and task-based measures that were administered to twins and triplets (N = 810) who participated in a component of the Texas Twin Project (Harden, Tackett & Tucker-Drob, 2013). Participants, ages 13-20 years (mean = 15.9 years, SD = 1.4 years), were administered task-based measures by trained research assistants. Self-report measures were administered to participants on a computer in a private room. Details regarding participant recruitment, data collection, and sample demographics are reported elsewhere (see Harden et al., 2016). Note, the socioeconomic and racial composition of the sample is largely representative of the greater Austin and Houston metropolitan areas; approximately 40% of participants reported a race/ethnicity other than Caucasian, and approximately 33% of families reported having received a needs-based form of public assistance since the twins or triplets were born.

Measures

A large battery of measures was administered to participants. Additional information, including the source of measures, a description of task paradigms and dependent variables is reported by Harden et al. (2016).

Self-Report Questionnaires

A total of twelve self-report measures were used.

UPPS Impulsivity Subscales. Impulsive personality was measured using the UPPS Impulsivity Scale (Whiteside & Lynam, 2001). Items were rated on a 4-point scale (1-Disagree Strongly, 4 = Agree Strongly), and three subscale scores were computed: Urgency (e.g. "When I feel bad, I will often do things I later regret in order to make myself feel better now"), Premeditation (e.g. "My thinking is usually careful and purposeful") and sensation seeking (e.g. "I would enjoy parachute jumping").

Future Orientation Subscales. Individual differences in future-oriented mindsets were measured using three subscales from the Future Orientation scale (Steinberg et al., 2009). For each item, there were two statements that describe how people think about the future, and participants must decide which statement best describes them. All items were rated on a 4-point scale. Subscales include planning ahead (e.g. "I like to plan things out one step at a time"), time perspective (e.g. "I often think about what my life will be like 10 years from now"), and anticipation of future consequences (e.g. "I usually think about the consequences before I do something").

Risk Perception Subscales. Two subscales that represent perceived harms, and perceived benefits vs. harms were computed using participants' responses to the Risk Perception Scale (Benthin et al., 1993). This measure has participants imagine 7 risky scenarios and rate whether the potential benefits in the scenario outweigh the risks (1 – Benefits Much Greater Than Risk, 4 – Risks Much Greater than Benefits), whether the scenarios are frightening (1 – Not at all Frightening, 4 – Very Frightening), personally risky (1- Not at all Risky, 4 – Very Much risky), or likely to result in serious harm (1 – Very Mild Harm, 4 – Very Serious Harm).

Pubertal Development Scale. We also measured individual differences in pubertal development using the Pubertal Developmental Scale (Petersen, Crockett, Richards & Boxer, 1998). Males and females reported on both general and sex-specific questions about the growth and maturity of secondary sex characteristics, compared to their peers.

Aggression and Rule-Breaking. Aggression and rule-breaking were measured using adolescents' responses to externalizing items from the Child Behavior Checklist (Lizotte, Chard-Wierschem, Loeber, & Stern, 1992). Based on previously published factor analytic work (Harden et al., 2015), 13 items were used to measure aggression (e.g. "I physically attack people" and "I am mean to others"), and 12 items were used to measure rule-breaking (e.g. "I disobey my parents" and "I lie or cheat"). All items were rated on a 3-point scale (0 = Not True; 2 = Very True or Often True).

Behavioral Tasks

A total of 8 behavioral measures were used.

Iowa Gambling Task (IGT). In this task participants were shown four decks of cards and had to decide whether to play or pass on each card. Playing a card is "risky" because it may result in either winning or losing pretend money, while the amount of pretend money doesn't change by passing on a card. Playing cards from certain decks yielded net gains, while playing cards from other decks yielded net loses. Throughout successive trails, participants tried to "win the most money by learning to avoid the bad decks and play more cards from the good decks." Two scales were computed: play on good decks and play on bad decks.

Delay Discounting Task. Participants were asked a series of response-dependent questions about whether they would prefer to receive a large delayed reward or a small immediate reward (e.g. "Would you prefer $1000 in 1 year or $200 now?"). The length of delay varied across six timeframes (1 week, 1 month, 6 month, 1 year, 5 years, and 15 years), and the starting value of the immediate reward was randomly determined for each of the timeframes. If the participant choice a delayed reward, then the immediate reward would increase on the next trial. On the other hand, if an immediate reward was chosen, then the immediate reward would decrease on the next trial. This processes was repeated until responses converged on an indifference point- i.e. when the small, immediate reward was perceived to be of equivalent value to the larger delayed reward. An indifference scale was computed by averaging the indifferent points across the six timeframes.

Balloon Analog Risk Task. In this task participants were shown 30 balloons, one after another. For each balloon participants were instructed to click a button to "pump up the balloon" in order to "build up points". However, if the balloon popped before the participant clicked "save points", then the points build up on that balloon were lost. A measure of average adjusted pumps was computed as the average number of pumps across trails during which the balloon did not explode.

Stoplight. In this task participants decided whether to "drive" through a series of yellow lights at intersections, or choose to place it safe and stop. Scores were computed as the percent of intersections at which the participant did not stop.

Tower of London. In this task participants were instructed to move colored beads onto pegs in order to match a pre-specified "goal arrangement", as quickly as possible

and using as few "moves" as possible. Two scales were computed: average time to first move and the total excess number of moves (relative to the number of moves in a perfect solution).

Wechsler Abbreviated Scale of Intelligence. Participants were also administered four subtests to measure verbal and spatial ability: Vocabulary, Similarities, Matrix Reasoning and Block design. Performance on all four subtests was used to estimate full-scale I.Q.

Data Analytic Procedures

Descriptive statistics for study variables (means, standard deviants, minimum and maximum observed scores) are reported elsewhere (see Harden et al., 2016). All inferential analyses were conducted within a structural equation modeling framework using M*plus* version 7.1 (Muthén & Muthén, 2010). For phenotypic associations, standard errors were adjusted for the nonindependence of data that results from siblings being clustered within the same family. Mean and variance adjusted weighted least squares estimation was used and the absolute fit of models was evaluated using root mean squared error of approximation (RMSEA) and Tucker-Lewis Index (TLI); RMSEA values $< .05$ and TLI values $> .80$ indicate acceptable fit to the data.

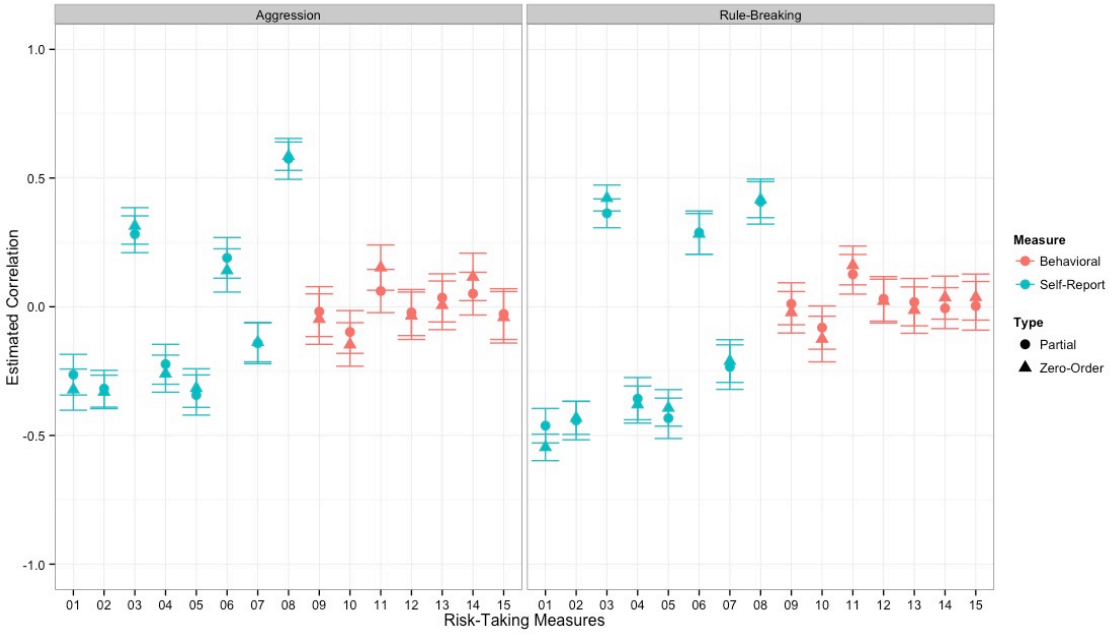
Inferential analyses were conducted in four steps. First, zero-order and partial phenotypic correlations between study variables were estimated. Second, a structural equation model was used to estimate the cross-sectional effects of latent premeditation, fearlessness, reward seeking and cognitive dyscontrol constructs on rule-breaking and aggressive behavior. Third, indirect effects were estimated to test whether premeditation, fearlessness and reward seeking constructs statistically mediate the cross-sectional effects

of sensation seeking on rule-breaking and aggression. Fourth, a multivariate behavior genetic model was used to estimate genetic and environmental correlations between latent risk-taking constructs and measures of antisocial behavior.

RESULTS

Are self-report and behavioral measures of risk-taking associated with aggression and/or rule-breaking? Depicted in Figure 6, self-report measures of risk-taking (colored red) were significantly associated with self-reports of aggression and rule-breaking (*p*s < .001). The zero-order correlations (triangles) between self-report measures of risk-taking and aggression were small-to-moderate in magnitude (range of *r*s = .14 - .59), similar to the zero-order correlations with rule-breaking (range of *r*s = .21 - .55). Partial phenotypic correlations (circles) indicate that this pattern of associations remains largely unchanged after accounting for the cross-sectional effects of chronological age, pubertal development, biological sex and full-scale I.Q.

***Figure 6*. Correlations between Measures of Risk-Taking and Antisocial Behavior**



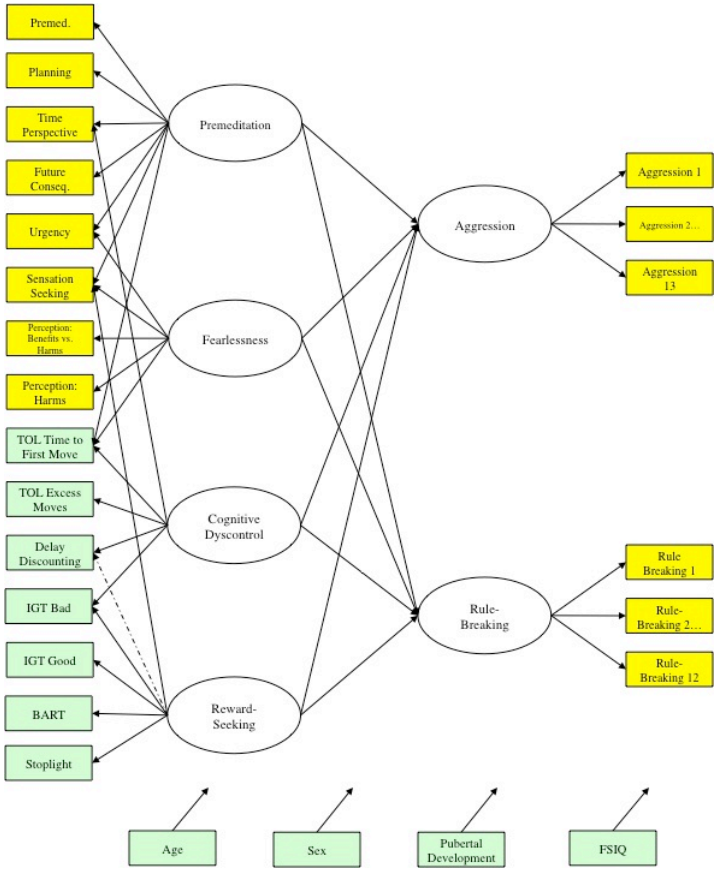
Note. 01 = Risk Perception: Benefit vs. Harm Scale. 02 = Future Consequences Scale. 03 = Risk Perception: Harm Scale. 04 = Planning Ahead Scale. 05 = Premeditation Scale. 06 = Sensation Seeking Scale. 07 = Time Perspective Scale. 08 = Urgency Scale. 09 = Balloon Analog Risk Task. 10 = Delayed Discounting. 11 = Iowa Gambling Task: Bad Desks. 12 = Iowa Gambling Task: Good Desks. 13 = Stop Light Task. 14 = Tower of London: Excess Moves. 15 = Tower of London: Time to First Move.

In contrast to self-report measures, the majority of behavioral measures were not significantly associated with aggression or rule-breaking (*p*s > .05), with three noteworthy exceptions. First, play on bad decks in the Iowa Gambling task (i.e. #11 in Figure 6) was positively correlated with aggression ($r = .15$, $SE = .04$, $p = .001$) and rule-breaking ($r = .16$, $SE = .04$, $p < .001$); however, only the correlation with rule-breaking (partial $r = .13$, $SE = .04$, $p = .001$) remained significant after accounting for the cross-sectional effects of study covariates (i.e. chronological age, pubertal development, biological sex and full-scale I.Q.). Second, performance on the delayed discounting task (i.e. #10 in Figure 6) was negatively associated with aggression ($r = -.15$, $SE = .04$, $p =$

.001) and rule-breaking (r = .13, SE = .04, p = .006), even after accounting for the cross-sectional effects of study covariates. Third, excess moves on the Tower of London was positively associated with aggression (r = .12, SE = .05, p = .013), but this association was not significant after accounting for the cross-sectional effects of study covariates (partial r = .05, SE = .04, p = .23).

Are individual measures of risk-taking, or the latent constructs they index, more strongly associated with antisocial behavior? A structural equation model was specified to estimate the cross-sectional effects of latent premeditation, fearlessness, reward-seeking and cognitive dyscontrol constructs on aggression and rule-breaking (see Figure 7). In this model, premeditation and fearlessness constructs were defined by self-report measures (colored yellow), with the exception of "time to first move" on the Tower of London, which was specified to load onto both premeditation and fearlessness constructs. Cognitive dyscontrol and reward-seeking constructs, on the other hand, were defined by performance on behavioral tasks (colored turquoise), with the exception of sensation seeking and time perspective scales, which were specified to load onto reward-seeking and cognitive dyscontrol constructs, respectively. Latent aggression and rule-breaking constructs were defined by adolescents' responses to thirteen and twelve self-report items, respectively. All latent factors were scaled on a standardized metric (mean = 0, variance = 1) using unit variance identification. Intercepts, thresholds, factor loadings, regression coefficients, covariances and residual variances were freely estimated from the data.

***Figure 7*. Path Diagram of Structural Equation Model**



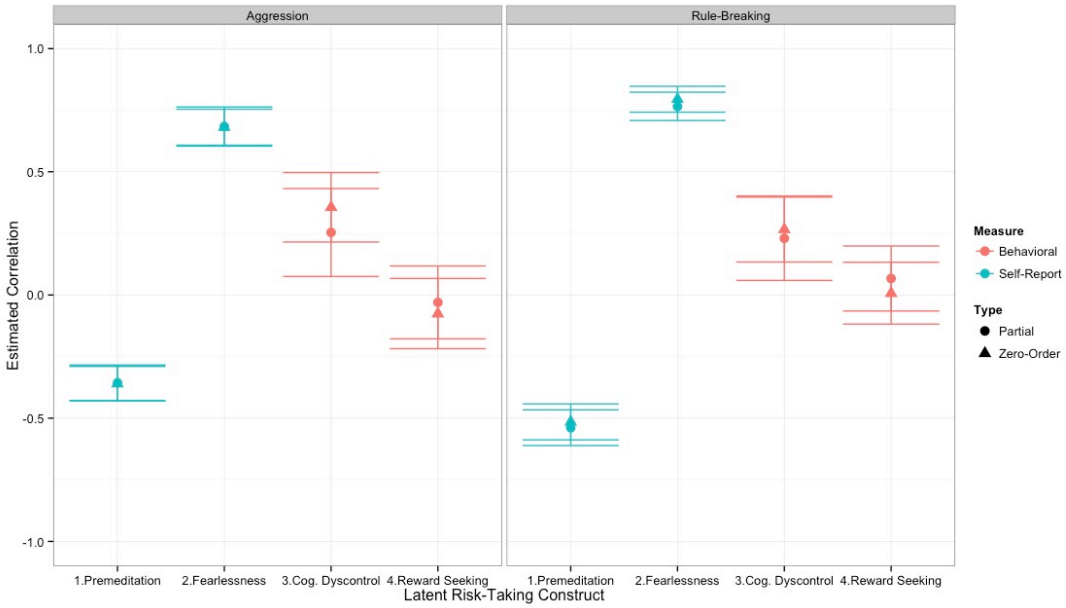
Note. All latent factors were regressed on age, sex, pubertal development and full-scale I.Q. Only three indicators for aggression and rule-breaking factors are shown. Residual variances of latent factors and observed indicators were estimated but omitted from the figure to ease visualization.

The structural equation model depicted in Figure 7 showed good fit to the data (c^2 = 1689.03, df = 859, p < .001, $RMSEA$ = .037, TLI = .85). All aggression and rule-breaking items loaded significantly onto their respective constructs (ps < .001) and, the pattern of factor loadings onto premeditation, fearlessness, cognitive dyscontrol and reward-seeking constructs mirrored those previously reported by Harden et al., (2016).

Premeditation had a significant cross-sectional effect on aggression (b = -.56, SE = .06, p < .001) and on rule-breaking (b = -.924, SE = .06, p < .001) and, the effect of premeditation on aggression was significantly greater than the effect of premeditation on rule-breaking (Δb = .36, SE = .13, p = .007). Fearlessness had a significant cross-sectional effect on aggression (b = .65, SE = .08, p < .001) and on rule-breaking (b = 1.046, SE = .14, p < .001). In contrast, the effect of fearlessness on rule-breaking was significantly greater than the effect of fearlessness on aggression (Δb = .40, SE = .14, p = .005). Cognitive dyscontrol had a moderate cross-sectional effect on aggression (b = .27, SE = .12, p = .029) and on rule-breaking (b = .36, SE = .16, p = .027); the difference between these estimates was not significantly different from zero (Δb = .16, SE = .12, p = .160). Finally, after accounting for the effects of the other latent risk-taking constructs, reward-seeking was not significantly associated with aggression (b = -.06, SE = .10, p = .564) or rule-breaking (b = .40, SE = .14, p = .005).

To compare these associations with those observed between antisocial behavior and individual measures of risk-taking, parameters of interest were recast and plotted as latent zero-order and partial phenotypic correlations (see Figure 8). Note, associations between latent risk-taking construct and antisocial behavior, specifically premeditation and fearlessness are of greater magnitude than that observed between individual measures of risk-taking and antisocial behavior

Figure 8. **Correlations between Latent Risk-Taking Constructs and Antisocial Behavior**

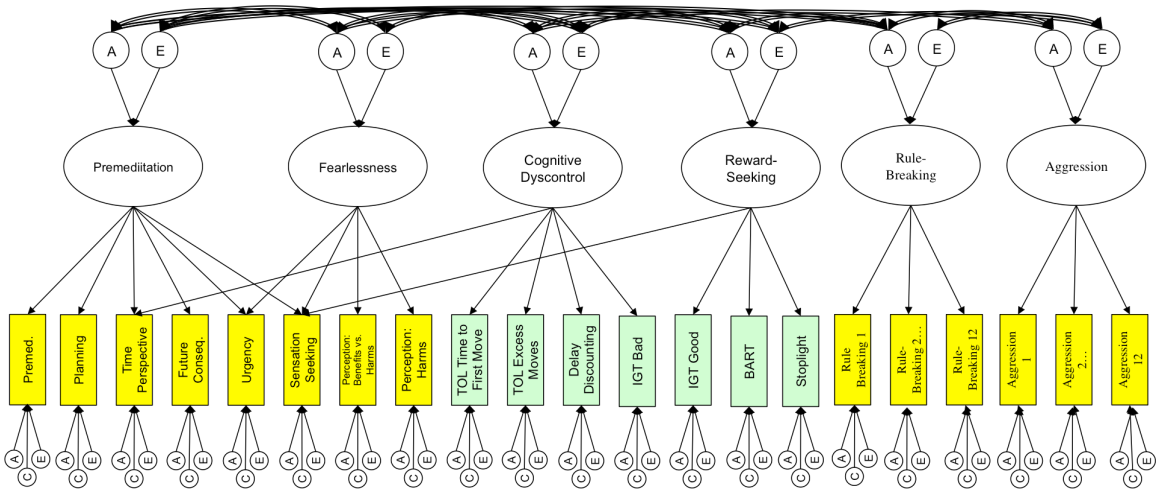


Are associations between sensation seeking and antisocial behavior mediated by latent risk-taking constructs? The same structural equation model was used to estimate the indirect effects of sensation seeking on aggression and rule-breaking via concurrent effects on premeditation, fearlessness and reward seeking constructs. The cross-sectional effect of sensation seeking on aggression was significantly mediated by both premeditation (indirect effect = .157, SE = .030, $p < .001$) and fearlessness (indirect effect = .144, SE = .031, $p < .001$). Similarly, the cross-sectional effect of sensation seeking on rule-breaking was significantly mediated by premeditation (indirect effect = .257, SE = .056, $p < .001$) and fearlessness (indirect effect = .231, SE = .052, $p < .001$). Conversely, the cross-sectional effects of sensation seeking on aggression and rule-

breaking were not significantly mediated by reward sensitivity (indirect effects ~ 0 and *p*s > .10).

Are associations between latent risk-taking constructs and antisocial behavior mediated through heritable or non-heritable pathways? A multivariate behavior genetic model was used to estimate genetic and environmental correlations between latent risk-taking constructs, aggression and rule-breaking. Depicted in Figure 9, this model was parameterized as a two-group, multi-level model. Monozygotic and dizygotic twins defined the two groups and, clustering within vs. between twin-pairs defined the levels. The same phenotypic factor structure (depicted in Figure 7) was specified at the within- and between-levels and, factor loadings were freely estimated but fixed to equality across levels. Sex was specified as a within-level covariate and, age was specified as a between-level covariate. With respect to biometric components of variance,

Figure 9. **Multivariate Behavior Genetic Model**

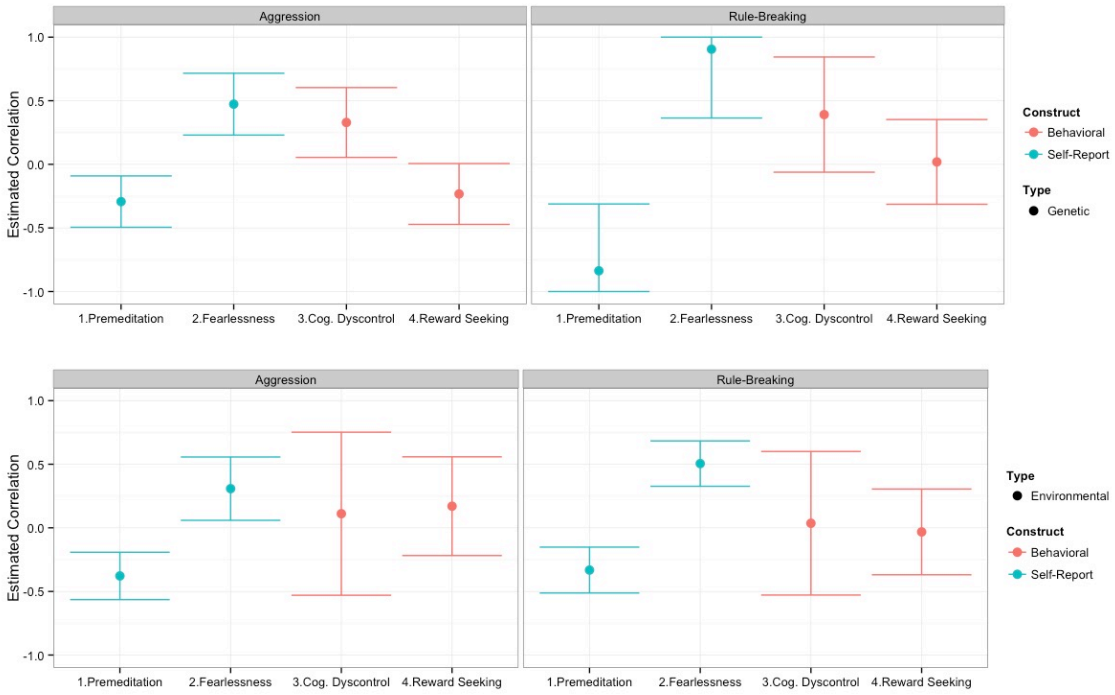


Note. To ease visualization only three indicators are shown for rule-breaking and aggression constructs.

non-shared environmental factors (E) were specified at the within-level, shared environmental factors (C) were specified at the between-level and, additive genetic factors were specified at both the within- and between-levels. To reflect the known degrees of genetic relatedness between monozygotic and dizygotic twins, additive genetic parameters (i.e. variances and covariances) at the within- and between-levels for dizygotic twins were constrained to be equal to half that of monozygotic twins and, genetic variation at the within-levels for monozygotic twins was fixed to equal zero.

The multivariate behavior genetic model showed good fit to the data (χ^2 = 907.786, df = 514, p < .001, $RMSEA$ = .044, TLI = .84). Genetic and environmental correlations are reported below in Figure 10. Note, there were significant genetic correlations between premeditation and rule-breaking (rA = -.83, SE = .26, p = .002), as well as between premeditation and aggression (rA = -.29, SE = .10, p = .005). There were also significant genetic correlations between fearlessness and rule-breaking

***Figure 10*. Genetic and Environmental Correlations Between Focal Constructs**



Note. Additive genetic correlations reported top panel and non-shared environmental correlations reported bottom panel. Correlations with aggression and rule-breaking and plotted on the left and right, respectively. Error bars denote 95% confidence intervals.

(rA = .90, SE = .27, p = .001), as well as between fearlessness and aggression (rA = .48, SE = .12, p < .001). Finally, there was a significant genetic correlation between cognitive dyscontrol and aggression (rA = .33, SE = .14, p = .019).

With respect to non-shared environmental correlations, a similar pattern of results emerged. There were significant non-shared environmental correlations between premeditation and rule-breaking (rE = -.34, SE = .09, p < .001), as well as between premeditation and aggression (rE = -.39, SE = .09, p < .001). There were also significant non-shared environmental correlations

between fearlessness and rule-breaking (rE = .50, SE = .09, p < .001), as well as between fearlessness and aggression (rE = .31, SE = .13, p = .015).

DISCUSSION

The current study examined data from a large, population-representative sample of adolescent twins who completed an in-laboratory battery of self-report and behavioral measures of risk-taking. We examined (1) whether self-report or behavioral measures of risk-taking were more strongly associated with antisocial behavior; (2) whether individual measures of risk-taking, or latent constructs identified by measures of risk-taking, are more strongly associated with antisocial behavior; (3) whether latent risk-taking constructs mediate the association between sensation seeking and antisocial behavior and (4) whether latent risk-taking constructs are associated with antisocial behavior via heritable or non-heritable pathways. On the whole, compared to behavioral measures, we found that self-report measures of risk-taking were more strongly tied to self-report measures of antisocial behavior. We also found that latent risk-taking constructs, specifically premeditation and fearlessness, were more strongly associated with aggression and rule-breaking than individual measures of risk-taking, whether they be behavioral or self-report measures. Moreover, latent premeditation and fearlessness constructs statistically mediated associations between sensation seeking and antisocial behavior.

The multivariate behavior genetic model indicated that there were significant genetic correlations between latent risk-taking constructs and antisocial behavior. That is, genes that contribute to individual differences in premeditation, fearlessness and

cognitive dyscontrol also contribute to individual differences in antisocial behavior. Put differently, latent risk-taking constructs index genetic liability for antisocial behavior. These results are consistent with conceptualizing risk-taking propensities as personality endophenotypes for antisocial behavior (Mann et al., 2017) - i.e. as intermediary constructs that help bridge the explanatory gap between genotype and antisocial phenotype. However, given the use of cross-sectional data, the current study was unable to distinguish between causal and non-causal models of endophenotypes, often referred to as "causal mediation" and "liability index" models. The corollary is that we are unable to determine whether latent risk-taking constructs instantiate causal mechanisms that link polygenic risk to antisocial behavior. It remains an open question whether genetic variants that undergird latent risk-taking constructs cause antisocial behavior or, alternatively, constitute a non-pathological, socially sanctioned expression of polygenic risk for antisocial behavior.

Additionally, to the extent that MZ twins differed in their propensities toward risk-taking (as measured by our multivariate battery of tasks), these within-MZ-twin pair differences were reliably associated with differences in antisocial behavior, as indicated by the significant non-shared environmental correlations between premeditation and antisocial behavior and between fearlessness and antisocial behavior. Put differently, these results indicate that the associations between premeditation and fearlessness with aggression and rule-breaking are due only party to genetic overlap. In fact, results of the current study are also consistent with an environmentally-mediated causal effect of risk-taking propensities on antisocial behavior, and/or vice versa. With that said, results

indicate that the phenotypic associations between risk-taking constructs and antisocial behavior were primarily genetic in origin.

The youth self-report (YSR) version of the Child Behavior Checklist (Achenbach, 2009) was used to measure rule-breaking and aggressive behavior. This measure was designed for use with 8-18 year olds and is not specifically tailored for use in clinical or incarcerated populations. Although the psychometric properties of the YSR are well-established, given the measure was not developed for use in incarcerated or institutionalized populations, it provides little coverage at extreme levels of antisocial behavior. For example, no items measure cruelty toward animals and very few items measure physically violent behavior. Moreover, the YSR is focused developmentally downwards; We assume that many of the teens who participated in the current study will soon desist from antisocial behavior, as "adolescent-limited" trajectories of antisocial behavior are considered common and non-pathological (Moffitt, 1993). Therefore, it remains an open question whether the associations between risk-taking constructs and antisocial behavior documented in the current study extend to other populations of interest, specifically populations with high, severe and persistent levels of antisocial behavior.

There are a number of additional limitations to the current study. First, results of the behavior genetic model depend on certain assumptions, including no assortative mating among parents on focal study constructs, as well as limited power to simultaneously detect shared environmental and non-additive genetic effects. Furthermore, survey-based measures of risk-taking propensities and antisocial behavior

were answered only by adolescent twins, which has the potential to produce biased results due to socially-desirable responding. Future research may stand to benefit from administering survey-based measured to multiple informants and testing the degree of correspondence across different informants. Finally, the current study analyzed a cross-sectional sample of adolescents. Therefore, we are unable to determine the direction of effects between risk-taking propensities and antisocial behavior. Future research would benefit from analyzing repeated-measures of study constructs in a genetically-informative sample to test the direction of effects that were documented in the current study.

Results of the current study builds on an established body of work that emphasizes the distinction between aggressive antisocial behavior and non-aggressive rule-breaking behavior (Burt, 2009, 2012). Results indicate that individual differences in premeditation were more strongly associated with aggression than rule-breaking. Conversely, individual differences in fearlessness were more strongly associated with rule-breaking than aggression. Thus, to the extent the non-shared environmental correlations reflect genuine casual pathways, results suggest that individual differences in premeditation and fearlessness help facilitate the differentiation of aggression and rule-breaking behavior. It remains an open question whether these environmental sources of variation in risk-taking propensities can be identified and leveraged by prevention or intervention programs to help deter different types of antisocial behavior.

The current study also extends research on sensation seeking, risk-taking and antisocial behavior. By considering relations between sensation seeking and antisocial behavior within the context of a large, multivariate battery of risk-taking measures, we

found evidence that the relation between sensation seeking and aggression, as well as sensation seeking and rule-breaking, is mediated entirely by variance shared with latent premeditation and fearlessness constructs. Thus, results of the current study suggest that sensation seeking may be conceptualized as a multi-dimensional risk factor for antisocial behavior; not only does sensation seeking relate to aggressive and non-aggressive dimensions of antisocial behavior but, that relation is mediated by concurrent associations with multiple dimensions of risk, mainly individual differences in premeditation and fearlessness.

Chapter 4. Perspectives on Sensation Seeking and Antisocial Behavior

Sensation seeking and antisocial behavior may be viewed through different theoretical lenses. Clinical psychology research has traditionally emphasized behaviors that are symptoms of *externalizing disorders*. Consequently, antisocial behavior and associated risk factors (including high levels of sensation seeking) are typically viewed as pathological and maladaptive. Developmental neuroscientists and epidemiologists often view sensation seeking and antisocial behavior as *health-risk behaviors*, which may or may not constitute symptoms of a psychiatric disorder. In contrast to clinicians and epidemiologist, evolutionary psychologists often conceptualize sensation seeking and antisocial behavior as the functional output of psychological adaptations. As a result, evolutionary perspectives do not necessarily view sensation seeking and antisocial behavior as pathological or maladaptive. On the contrary, these constructs are viewed as *adaptive responses* to socio-ecological pressures, including those pressures characteristic of the transition from adolescence to adulthood. The overarching goal of this final chapter is to explore these theoretical perspectives on sensation seeking and antisocial behavior and discuss how results of the current dissertation relate to these different perspectives.

CLINICAL PERSPECTIVES

In efforts to develop more effective treatment strategies, research in the field of clinical psychology tends to focus on delineating the cause, course and correlates of psychopathology, including those characterized by antisocial behavior. Simply put, from a clinical perspective, antisocial behaviors are problematic because they are symptoms of externalizing disorders, which cause distress to the self and others and impair functioning

across major role domains. In turn, high levels of sensation seeking are problematic because they increase risk for externalizing disorders. However, as previously discussed (see Chapter 1), instead of conferring a uniform level of risk, sensation seeking is often conceptualized within the context of person-environment interactions, resulting in high levels of antisocial behavior when certain contextual factors are present (such as high levels of peer deviance). In fact, research in clinical psychology has shifted focus from simply identifying the constituent components of person-environment interactions to clarifying the nature of those interactions.

The *diathesis-stress* model posits that individuals with high levels of intra-personal risk express symptoms of psychopathology only when in the presence of adverse contextual factors (Belsky et al., 2009). For example, according to the diathesis-stress model, individuals high on sensation seeking should exhibit high levels of antisocial behavior when in the presence of socio-ecological factors that exacerbate endogenous risk. The *differential susceptibility* model also posits that individuals with high levels of intra-personal risk exhibit symptoms in the presence of adverse contextual factors. However, this model also posits that the same individuals stand to benefit the most from supportive environments, exhibiting exceptionally beneficial outcomes when in the presence of positive contextual factors (Belsky & Pluess, 2009; Pluess & Belsky, 2013). With respect to the nature of person-environment interactions, a fan-shaped interaction is consistent with diathesis-stress and, a crossover interaction is consistent with differential susceptibility (Rioux et al., 2016).

The pattern of person-environment interaction documented in the present dissertation is consistent with a diathesis-stress model of sensation seeking and peer deviance interacting to predict antisocial behavior. However, potential prosocial manifestations of sensation seeking were not measured, which is necessary to differentiate between diathesis stress and differential susceptibility. As we observed, a socially problematic expression of sensation seeking is to engage in antisocial behavior. However, prosocial expressions of sensation seeking are also possible. For example, highly sensation seeking teens might also be more likely to become socially dominant leaders, as has previously been found for high-testosterone males with prosocial friends (Rowe, Maughan, Worthman, Costello, & Angold, 2004). Among adults who were all employed full-time, high sensation seeking was associated with better supervisor ratings of job performance, higher entrepreneurship, and higher involvement with and enjoyment of work (Jackson, 2011). However, whether these results generalize to teenagers, for whom many avenues of prosocial risk-taking (e.g., entrepreneurship, military service) are proscribed, is unclear. How to best conceptualize and measure potentially prosocial manifestations of sensation seeking in adolescents is, therefore, an interesting avenue for future research.

EPIDEMIOLOGY AND NEUROSCIENCE

Perspectives from epidemiology and developmental neuroscience share much in common with clinical psychological perspectives. Both tend to view sensation seeking and antisocial behavior as potentially problematic and, both acknowledge person-environment interactions and interpret these interactions in terms of diathesis-stress.

However, epidemiologists tend to focus on specific health-risk behaviors, like substance-use, which are not necessarily uncommon or antisocial. In fact, some health-risk behaviors are relatively common. Moreover, research in epidemiology and neuroscience has focused on the rewarding properties of risk-taking. Indeed, understanding the rewarding properties of risk-taking is particularly important for understanding the escalation in sensation seeking that is typical of adolescence.

Similar to crime, myriad health-risk behaviors, including drunk driving and accidental injury, increase nearly ten-fold during adolescence (Moffitt 1993; Loeber & Farrington, 2014). Research in developmental neuroscience has found that brain regions involved in reward processing (e.g. the amygdala and ventral striatum) reach neurobiological maturity in adolescence (Shulman et al., 2016). Thus, adolescents are equipped to enjoy the rewarding properties of novel, stimulating and exciting experiences, which may be attained by engaging in risky behavior. Brain regions related to impulse control (e.g. the prefrontal cortex), on the other hand, do not reach maturity until adulthood (Steinberg, 2008). Consequently, adolescents are motivated to engage in risky behavior for the sake of rewards but are not equipped to inhibit such behavior. This model of neuro-developmental asymmetry, often referred to as the *dual systems model* (Steinberg, 2010), is also supported by research in developmental psychology (Harden & Tucker-Drob, 2011; Steinberg et al., 2008).

The large battery of self-report questionnaires and behavioral tasks administered to participants in the present dissertation were those that are commonly used in neuroscience and epidemiological research to measure constructs relevant to the *dual*

systems model. "This model has largely focused on normative adolescent development, in that it seeks to describe personality and behavioral changes that are (a) typical of most adolescents and (b) developmentally unique to adolescence" (p.2; Mann, Paul, Tackett, Tucker-Drob & Harden, 2017). As a result, one may predict that constructs related to normative adolescent development will be more strongly associated with relatively normative expressions of antisocial behavior, compared to more severe expressions. Non-aggressive rule-breaking increases during adolescence, while aggressive antisocial behavior is more rare, peaking in childhood before declining into adulthood (Burt, 2012). Thus, rule-breaking behavior in adolescence may be considered more normative than aggressive antisocial behavior. Consistent with this notion, the battery of self-report questionnaires and behavioral tasks accounted for 70% of the variance in non-aggressive rule-breaking, compared to 48% of the variance in aggressive antisocial behavior.

Based on the dual systems model, one may also expect reward-related processes to be integral to understanding the etiology of antisocial behavior. However, the current dissertation suggests that reward seeking may play only a peripheral role in antisocial behavior. Using computer-based tasks that are commonly employed in neuroimaging studies, we found evidence that performance on three tasks (Tower of London, Iowa Gambling Task and the Balloon Analog Risk Task) may be captured by a latent construct, which we labeled "reward-seeking". Individuals scoring high on this construct engaged in "risky play" on behavioral tasks, seeking out potential rewards irrespective of loss (e.g. playing more cards from both "good decks" and "bad decks" in the Iowa Gambling Task). To our surprise, after accounting for the effects of premeditation,

fearlessness and (lack of) cognitive control, individual differences in reward seeking were not significantly associated with antisocial behavior, neither with aggressive behavior or non-aggressive rule-breaking. Although reward-related processes are important for understanding why adolescents engage in risky behavior more than children or adults, results of the current dissertation suggests that, among adolescents, individual differences in reward-related processes are not strongly related to individual differences in antisocial behavior. Therefore, epidemiological perspectives may benefit by shifting focus away from the rewarding properties of health-risk behavior to other properties that may account for individual variability in adolescent antisocial behavior. For example, in the present dissertation, fearlessness was more strongly related to antisocial behavior than reward seeking. Consistent with this finding, an established corpus of studies indicates that callous-unemotional traits, including shallow affect and dampened reactivity, are central to understanding severe expression of antisocial behavior (Frick, Ray, Thorton & Kahn, 2014).

EVOLUTIONARY PERSPECTIVES

The terms "adaptive" and "maladaptive" are often used by clinicians and epidemiologists to denote behaviors that inflict costs to mental health and society as a whole. Within the nomenclature of evolutionary biology, however, adaptations are inherited characteristics of an organism that were favored by selection due to increased effects on reproduction (or, more precisely, inclusive fitness). Thus, despite frequent associations with socially problematic and dangerous outcomes, antisocial and sensation seeking behavior is not, from an evolutionary perspective, necessarily dysfunctional or

maladaptive. On the contrary, adolescence – for many species – is a time when securing mates, facing adversaries, and staking out new territory are crucial to survival and reproduction, and increases in sensation seeking may drive bold, exploratory behaviors that are necessary to meet these demands. Consistent with this broad evolutionary perspective, sensation seeking also increases, on average, during adolescence in non-human animals (Macri, Adriani, Chiarotti & Laviola, 2002). Consequently, the typical increase in sensation seeking that occurs from late childhood to adolescence, as detailed by the *dual systems model*, may be an example of *adaptive developmental plasticity*- i.e. a developmentally-specific fluctuation in a phenotype that has evolved in response to selection pressures that occurred during a specific point in ontogeny. Sensation seeking and antisocial behavior may therefore be part of a suite of potential behaviors and tendencies that have evolved to meet the new functional demands of reproductive maturity (Ellis et al., 2012).

According to life history theory, sensation seeking and antisocial behaviors are the output of conditional adaptations that coordinate survival and reproductive efforts. Life history theory is a middle-level evolutionary theory that explains individual differences in behavior in terms of adaptive trade-offs that optimize reproductive fitness over the life course, as well as across environmental heterogeneity (Del Giudice & Belsky, 2011; Hill & Kaplan, 1999). Throughout an organism's lifespan, finite resources are divided between maintenance, growth, and reproduction (Gadgil & Bossert, 1970), which may be described in terms of trade-offs between current and future reproduction, as well as quantity of offspring and investment in offspring (Kaplan & Gangestad, 2005;

Stearns, 1989). Varying along a slow-to-fast continuum, the timing and outcome of these resource allocation decisions constitutes an organism's life history strategy.

In humans, insecure attachments, increased somatic development (i.e. early pubertal development), social deviance and high levels of risk-taking behavior (Belsky, Steinberg, Draper, 1991; Belsky, 2000) characterize fast life history strategies, or accelerated developmental trajectories. Conversely, slow life history strategies, or delayed developmental trajectories, are characterized by secure attachments, decelerated somatic development, prosociality and sexual restrictedness. These divergent developmental pathways are thought to reflect adaptive calibration to variations in environmental adversity (MacDonald, 1997).

Interestingly, there was no evidence that risk-taking propensities and antisocial behavior co-occurred because they were influenced by sources of variation shared by siblings living in the same home, including adverse family environments. This finding is at odds with life history perspectives on risk-taking and related constructs (Zietsch, 2016), which view risk-taking and antisocial behavior in terms of adaptive calibration to harsh and unpredictable environments (Ellis et al., 2012). Indeed, previous studies have found associations between family socioeconomic status and antisocial behavior (Pratt & Cullen, 2005). However, the majority of such studies suffer from a relative inattention to passive gene-environment correlation, i.e. when individuals are non-randomly exposed to environments, such as socioeconomic deprivation, based on their genotypes. It remains an open question to what degree such family-level factors exert causal influence on risk-taking propensities and antisocial behavior or reflect passive gene-environment

correlation; given the relative absence of shared environmental influences documented on risk-taking constructs in the current dissertation, results are consistent with the latter.

Note, however, the presence of non-shared environmental variance in focal study constructs may also include family-level factors that affect siblings differently, e.g. paternal incarceration may cause internalizing symptoms in one sibling and externalizing symptoms in another. Future research may benefit from examining similar constructs in a study in which unrelated siblings reside in the same home, i.e. an adoption study. When adopted siblings are measured on the same constructs, this provides a more precise means of probing for the influence of shared environmental factors; in the absence of genetic relatedness, similarities between siblings are necessarily due to shared environmental factors. In the event that shared environmental influences are minimal, even after probing for such influences using an adoption study, life history theorists will need to revise their theory to explain why relevant socio-ecological factors are not stratified between families.

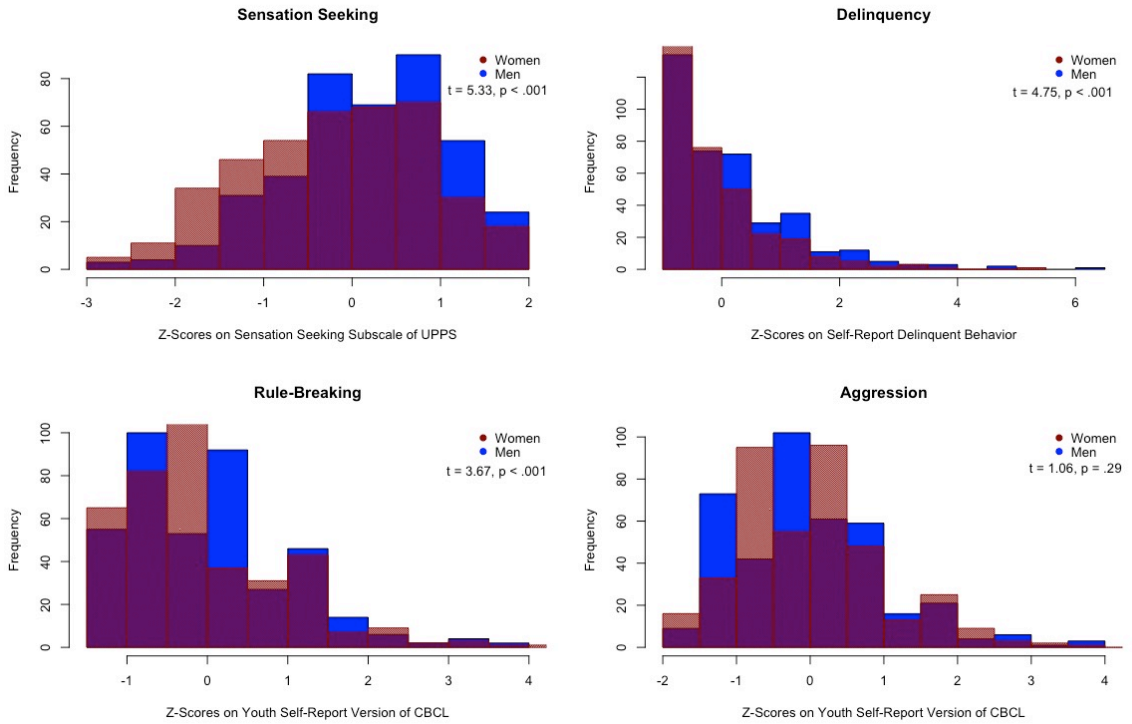
BIOLOGICAL SEX

One of the most reliable risk factors for antisocial behavior is being male. With respect to both prevalence and severity, on average, men report higher levels of antisocial behavior than women (Moffitt, 2001). Men are also more likely than women to be arrested, particularly for "crimes against persons" (p. 462, Steffensmeier and Allan, 1999). Similarly, men report higher levels of sensation seeking than women (Cross, Cyrenne and Brown, 2013). Indeed, in the present dissertation male adolescents reported higher levels of sensation seeking and antisocial behavior (see Figure 11). Although men

and women exhibit a similar window of vulnerability in adolescence (i.e., the ages when sensation seeking is high and impulse control is low), there is also evidence that men begin adolescence with higher levels of sensation seeking, which, in turn, decline more gradually across adulthood (Shulman, Harden, Chein & Steinberg, 2015). These sex-specific developmental trajectories of sensation seeking may be one source of sex differences in antisocial behavior.

On the other hand, the correlates of antisocial behavior tend to be similar for both

Figure 11. Sex Differences in Sensation Seeking and Antisocial Behavior



men and women, including "history of antisocial behavior, antisocial attitudes, antisocial peers and antisocial personality" (p. 1, Hubbard & Pratt, 2002). Moreover, using a

nationally-representative sample of youth, a recent study found that associations between *changes* in sensation seeking, impulsivity and antisocial behavior were similar for men and women (Mann, Paul, Tackett, Tucker-Drob & Harden, 2017). Future research stands to benefit from identifying the mechanisms that may otherwise account for the sex differences in antisocial behavior that are so frequently observed across criminological and psychological studies.

The *recalibration theory* of anger, developed by Aaron Sell and colleagues (Sell, Cosmides & Tooby, 2009; Sell, Cosmides, Tooby, Sznycer, von Rueden & Gurven, 2009), highlights cognitive mechanism that assess physical formidability as potentially integral to understanding sex differences in antisocial behavior. This model predicts that "individuals with enhanced abilities to inflict costs or to confer benefits will anger more easily for two related reasons. First… anger is more likely to be successful for them than for others with less leverage. Second, their greater leverage leads them to expect that others will place greater weight on their welfare" (p. 15074, Sell, Tooby & Cosmides, 2009). Physical strength confers formidability and, therefore, the ability to inflict costs on others. Physical attraction, on the other hand, confers health and fertility and, therefore, the ability to confer benefits on others. Consistent with the recalibration theory of anger, men's physical strength, as measured by their ability to lift weights on four machines at the gym, was positively correlated with how frequently and easily they angered. On the other hand, women's physical attractiveness was positively correlated with proneness to anger, feelings of entitlement and the successful resolution of conflict. These results indicate that physical phenotypes, such as strength and attractiveness, may be important

for understanding sex differences in antisocial behavior. It remains an open question whether the results documented by Sell and colleagues (2009) extend to different expressions of antisocial tendencies, other than anger, such as aggressive behavior and non-aggressive rule-breaking.

Research on the "dark triad" personality traits also indicate that men tend to be more narcissistic, psychopathic and Machiavellian than women, (Furnham, Richards & Paulhus, 2013; Jonason & Webster, 2010; Jones & Paulhus, 2014), which may be partially explained by sex-specific deficits in empathy (Jonason, Lyons, Bethell & Ross, 2013). In concert with this findings, research on callous-unemotional traits indicates that men, over average, suffer more than woman from low levels of empathy, lack of remorse and dampened affect (Fontaine, Rijsdijk, McCrory & Viding, 2010). Future research stands to benefit from expanding the multivariate battery of risk-related task reported in the present dissertation to include more variables that are related to severe expressions of antisocial behavior, including callous-unemotional traits, the dark triad personality traits and physical attributes, like physical strength.

AGGRESSION VS. RULE-BREAKING

In the first two chapters antisocial behavior was measured using a broad self-report questionnaire that captured a variety of delinquent behaviors (e.g. "lied to your parents about where you had been or whom you were with?", "been involved in a group or gang fight", "sold drugs such as heroin, cocaine, LSD, ecstasy or prescription pills?", etc.). In the third chapter, antisocial was divided according to rule-breaking and aggressive behavior. Although this decision was evidence-based (Burt, 2012), there are

other equally valid ways of dissecting heterogeneity in antisocial behavior. For example, aggression is often further divided into more specific components; researchers have made distinctions between proactive or hostile vs. reactive or instrumental aggression (Card & Little, 2006; Crick & Dodge, 1998; Raine et al., 2006), as well as physical aggression, verbal aggression and relational aggression (Scheithauer, Hayer, Petermann & Jugert, 2006; Tackett, Daoud, De Bolle & Burt, 2013). Furthermore, criminologists often make a distinction between violent and non-violent offending (Farrington, 1997). Few twin studies have examined the common and specific etiological factors that underlie the concurrence of these related, yet distinct, expressions of aggressive antisocial behavior. The measure of aggression used in the current dissertation collapsed across physical aggression (e.g. "I destroy things belonging to others" and "I physically attack people") and verbal aggression (e.g. "I argue a lot" and "I tease others"), which may explain the absence of significant sex differences for aggression documented in the current dissertation (see Figure 11). Studies with more finely-nuanced measurement of aggression may help further an understanding of how men and women differ with respect to antisocial behavior and, furthermore, may illuminate intermediary phenotypes that are responsible for sex differences in antisocial behavior.

CONCLUDUING REMARKS

On the one hand, clinical and epidemiological perspectives view sensation seeking and antisocial behavior as pathological and/or problematic. On the other hand, evolutionary perspectives view sensation seeking and antisocial behavior as functional and adaptive. Despite this glaring juxtaposition, there are common themes that unite these

seemingly disparate perspectives. Clinical and epidemiological perspectives acknowledge the importance of person-environment interactions and often interpret those interactions in terms of diathesis-stress or differential susceptibility. With respect to evolutionary perspectives, the notion of adaptive calibration necessarily entails person-environment interaction and, both diathesis-stress and differential susceptibility models of person-environment interaction have origin in evolutionary theory. Moreover, research in neuroscience that is relevant to epidemiology has expounded the complex biological processes that underlie an instance of developmental phenotypic plasticity- a concept that, again, stems directly from evolutionary theory. Thus, despite seemingly incompatible views on whether sensation seeking and antisocial behaviors are pathological and/or problematic, the theoretical perspectives outlined above may be viewed as compatible and complimentary approaches to understanding the etiology of antisocial behavior.

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