Published in final edited form as:

Dev Psychobiol. 2017 July ; 59(5): 561–573. doi:10.1002/dev.21529.

Affiliation with substance-using peers: Examining gene-environment correlations among parent monitoring, polygenic risk, and children's impulsivity

Kit K. Elam¹, Laurie Chassin², Kathryn Lemery-Chalfant², Danielle Pandika², Frances L. Wang³, Kaitlin Bountress⁴, Danielle Dick⁵, and Arpana Agrawal⁶

¹T. Denny Sanford School of Social and Family Dynamics, Arizona State University, Tempe, Arizona

²Department of Psychology, Arizona State University, Tempe, Arizona

³Department of Psychiatry, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania

⁴National Crime Victims Research & Treatment Center, Medical University of South Carolina, Charleston, South Carolina

⁵Department of Psychology, Virginia Commonwealth University, Richmond, Virginia

⁶Department of Psychological & Brain Sciences, Washington University in St. Louis, Saint Louis, Missouri

Abstract

Parental monitoring can buffer the effect of deviant peers on adolescents' substance use by reducing affiliation with substance-using peers. However, children's genetic predispositions may evoke poorer monitoring, contributing to negative child outcomes. We examined evocative genotype-environment correlations underlying children's genetic predisposition for behavioral undercontrol and parental monitoring in early adolescence via children's impulsivity in middle childhood, and the influence of parental monitoring on affiliation with substance-using peers a year and a half later ($n = 359$). Genetic predisposition for behavioral undercontrol was captured using a polygenic risk score, and a portion of passive rGE was controlled by including parents' polygenic risk scores. Children's polygenic risk predicted poorer parental monitoring via greater children's impulsivity, indicating evocative rGE, controlling for a portion of passive rGE. Poorer parental monitoring predicted greater children's affiliation with substance-using peers a year and a half later. Results are discussed with respect to gene-environment correlations within developmental cascades.

Keywords

deviant peers; gene-environment correlation; impulsivity; parent monitoring; polygenic risk score

Correspondence Kit K. Elam, Assistant Professor, T. Denny Sanford School of Social and Family Dynamics, Arizona State University, P.O. BOX 873701, Tempe, AZ 85287-3701. kit.elam@asu.edu.

SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

1 INTRODUCTION

Poor parental monitoring in early adolescence is associated with a range of adverse outcomes including conduct problems, substance use, and affiliation with substance-using peers (Laird, Marrero, & Sentse, 2010). As children enter adolescence they spend larger amounts of time away from their family and with less parental supervision, providing increased opportunities for associating with substance-using peers (Steinberg, Fletcher, & Darling, 1994). Parental monitoring, that is, parents' knowledge or their efforts to obtain knowledge of their children's daily activities (Racz & McMahon, 2011), is thought to buffer the effect of deviant peers on adolescents' substance use by reducing both affiliation with deviant peers and the influence of those peers (Kiesner, Poulin, & Dishion, 2010; Mann, Kretsch, Tackett, Harden, Tucker-Drob, 2015).

In recent years, parental monitoring has been investigated as a moderator of children's genetic predispositions for substance abuse and externalizing behaviors (e.g., Dick et al., 2011). Several studies have found evidence of gene-environment interactions in which high levels of monitoring are protective against the effects of children's genetic predispositions for risky and negative behavior (e.g., Dick et al., 2009; Dmitrieva, Chen, Greenberger, Ogunseitan, & Ding, 2011). However, less is known about gene-environment correlations (rGEs), in which children's genetic predispositions are associated with their environment. It is possible that children's inherited genetic predispositions are related to their rearing environment, both of which are provided by their biological parents, known as a passive rGE (Plomin, Defries, & Loehlin, 1977). That is, parents provide both their child's genetic liability and parental monitoring, which may be related to one another if monitoring is partially driven by this shared genetic liability (each biological parent and his/her offspring share 50% of their genes). Children's genetic predispositions may also evoke the type of parenting that they receive, known as an evocative rGE. A genetically influenced child trait, such as impulsivity, may evoke poorer parental monitoring, which may in turn contribute to greater affiliation with substance-using peers. This study presents a novel longitudinal examination of children's genetic risk for behavioral undercontrol, its association with parental monitoring during early adolescence, and the mediating role of impulsivity in middle childhood. Mediation by impulsivity, a facet of behavioral undercontrol, would indicate an evocative rGE. We examine this relation while controlling for a portion of passive rGE, which may confound evocative rGE, by including the same index of genetic risk for parents. Moreover, we investigate the role of evocative rGE in predicting subsequent substance-using peer affiliation a year and a half later in middle adolescence.

1.1 Parental monitoring

Poor parental monitoring during early adolescence is a known risk factor for later substance use and affiliation with substance-using peers (see Racz & McMahon, 2011 for a review). Parental monitoring encompasses parents' knowledge of their adolescent's whereabouts, peer groups, and the activities their child engages in during free time (Lamborn, Mounts, Steinberg, & Dornbusch, 1991). This knowledge is in large part driven by what their child discloses (Stattin & Kerr, 2000). Through monitoring, or parent solicitation of information,

parents are able to reduce their child's exposure to risky situations where initiation of conduct problems and substance use could occur (Steinberg et al., 1994). Parental monitoring also reduces the influence of peers' substance use on their adolescent's use (Kiesner et al., 2010). Whereas poor parental monitoring is consistently associated with greater affiliation with substance-using peers, the quality of monitoring that children receive is influenced by pre-existing child and familial factors (Kerr, Stattin, & Ozdemir, 2012).

Multiple theoretical models propose that the parenting children receive is influenced by interpersonal and contextual determinants (Belsky & Jaffee, 2006), one primary influence being that of children's own behavior and personality (Bell, 1968; Patterson, 1982; Sameroff, 2010). These models propose bidirectional effects between the parent and child in which children's behavior affects the parenting they receive, which subsequently influences later child outcomes. This is especially salient for disruptive and disinhibited child behavior which evoke poor parenting, subsequently contributing to poor child outcomes later in life (e.g., Elam, Chassin, Eisenberg, & Spinrad, 2017; Latzman, Elkovitch, & Clark, 2009; Prinzie et al., 2004). In a review, Kiff, Lengua, and Zalewski (2011) found such bidirectional transactions to exist between child impulsivity and negative parenting. Within this bidirectional framework a number of difficult child behaviors have been found to contribute to poor parental monitoring, including delinquency (Laird, Pettit, Bates, & Dodge, 2003), general problem behavior (Willoughby & Hamza, 2011), conduct problems (Pardini, Fite, & Burke, 2008), defiance (Kerr & Stattin, 2003), and impulsivity (Neumann, Barker, Koot, & Maughan, 2010). In the face of difficult behavior, parents may feel that they are unable to monitor their child's behavior and they may withdraw from the parenting role (Racz & McMahon, 2011). In particular, greater child impulsivity is concurrently and longitudinally associated with poorer monitoring during pre-adolescence (Flannery, Vazsonyi, Torquati & Fridrich, 1994; Neumann et al., 2010). The effect of child impulsivity on parental monitoring appears more prevalent in the context of at-risk familial factors, such as neighborhood poverty and single parent family structure (Chen & Jacobson, 2013; Neumann et al., 2010). Poorer monitoring is also associated with parental characteristics including parental substance abuse (Chassin, Curran, Hussong, & Colder, 1996). In addition to these child, familial, and parental factors, recent research has begun to examine children's genetic risk in relation to parental monitoring.

1.2 Gene-environment interplay and parental monitoring

Research to date has investigated the moderating effects of parental monitoring in the relation between children's genetic predisposition and problem behaviors, a gene-environment interaction. A handful of studies have found attenuated genetic effects on problem behavior in the presence of high parental monitoring using twin samples (Dick et al., 2007). This pattern is supported with studies measuring single genetic variants. For example, adolescent boys with the 7R allele of the dopamine receptor D4 (DRD4) polymorphism had higher levels of delinquency and thrill seeking, but, in the presence of high levels of parent monitoring, genetic effects were absent (Dmitrieva et al., 2011). More recently studies have examined single nucleotide polymorphisms (SNPs), variations in a single base pair of DNA, as genetic variants. Similar buffering effects have also been found when examining interactions between single SNPs and parental monitoring in predicting

early adolescent externalizing behavior and substance use (Chen et al., 2009; Dick et al., 2009, 2011; Miranda et al., 2013; Trucco, Villafuerte, Heitzeg, Burmeister, & Zucker, 2014). These findings have been attributed to parental monitoring acting as an environmental index of social control, reducing the potency of genetic effects on children's difficult or deviant behavior (Dick & Kendler, 2012). However, studies have not examined passive and evocative rGEs as a mechanism contributing to substance use involvement when considering the interplay between child behavior and parental monitoring.

An evocative rGE exists when a child's genetic predisposition evokes a reaction from the environment, through a genetically influenced behavior. A growing body of literature provides evidence that the genetic components of children's impulsive and disruptive behaviors evokes negative parenting across twin and stepfamily designs (Button, Lau, Maughan, & Eley, 2008; Deater-Deckard, Dunn, O'Connor, Davies & Golding, 2003; Forget-Dubois et al., 2007; Klahr, Klump, & Burt, 2014; Klahr, Thomas, Hopwood, Klump, & Burt, 2013; Narusyte et al., 2011; Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007; Neiderhiser et al., 2004), adoption designs (Elam et al., 2014; Ge et al., 1996; Harold et al., 2013; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998), and molecular genetic designs (Keltikangas-Järvinen et al., 2007; Kryski, Smith, Sheikh, Singh, & Hayden, 2014; Pener-Tessler et al., 2013; Propper, Shanahan, Russo, & Koonce, 2012).

In particular, impulsivity is known to have genetic continuity across childhood, adolescence, and young adulthood (Bezdjian, Baker, & Tuvblad, 2011; Hay, Bennet, McStephen, Rooney, & Levy, 2004; Niv, Tuvblad, Raine, Wang, & Baker, 2012). Previously, children's impulsivity has been implicated in evocative rGE within bidirectional parent-child interactions. Using an adoption study design, Harold et al. (2013) found that a genetic predisposition for impulsivity in children evoked greater levels of hostile parenting in mothers. Pener-Tessler et al. (2013) found evidence of an evocative rGE in relation to the serotonin transporter gene (5-HTTLPR), which was associated with lower levels of positive parenting via children's poor self-control. However, these studies utilized cross-sectional data and only investigated impulsive behaviors in early childhood.

A passive rGE is present if the same genetic influences underlie both children's impulsive behavior and parents' poor monitoring, which make them appear to be associated through an environmental influence. Preliminary supportive evidence is provided by an extended children of twins study finding variance in parental monitoring to be attributable to evocative and passive rGE (Neiderhiser et al., 2004, 2007). However, this design only partitions variance in parenting behavior into evocative and passive rGE influences but is not able to examine the child-specific trait or behavior involved, and so cannot examine the longitudinal interplay between parent and child behaviors in rGEs. Consequently, there is limited investigation of rGE in longitudinal mediation models. By utilizing such a design we investigated whether children's genetic predisposition for behavioral undercontrol may initiate a developmental cascade by evoking poor parental monitoring via impulsivity, which, in turn, contributes to greater affiliation with substance-using peers later in life.

Given evidence that impulsive child behavior longitudinally predicts poor parental monitoring in studies that do not use genetically informed designs (e.g., Neumann et al.,

2010), and that genetically informed studies have found evocative rGE between impulsivity and parenting (Harold et al., 2013; Pender-Tessler et al., 2013), it is important to examine the possibility that children's genetic predispositions are associated with parental monitoring via evocative rGE. It is also important to consider passive rGE in light of past support from twin studies (e.g., Neiderhiser et al., 2004, 2007). An alternative method to estimating passive rGE is to control for such effects across child and parent(s). This can be done by including the same index of genetic risk for the child and parent as covariates for child and parent behavior, respectively. This controls for passive rGE where the index of genetic risk is associated with the parenting behavior. This approach is advantageous for two reasons. First, this approach allows a test of evocative rGE effects while controlling for a portion of the possible underlying effect of passive rGE. Differentiating evocative rGE from passive rGE provides stronger evidence for the effect of evocative rGE. Second, controlling for passive rGE eliminates the possibility of common genetic influences that may underlie similarities in parent and child behavior (Klahr, McGue, Iacono, & Burt, 2011). Such an approach is useful because by covarying out a portion of the same genetic influence for both parenting and child behavior, a small portion of the remaining association between parenting and child behavior is likely due to an environmental influence. This approach is used in the present study in which we test for evocative rGE between children's genetic predisposition for behavioral undercontrol and parental monitoring via children's impulsivity, while controlling for the effects of parent's genetic predisposition for behavior undercontrol on parents' monitoring.

1.2.1 Polygenic risk scores in gene-environment interplay—Polygenic risk scores are composed of multiple SNPs that are combined into a single score or measure of genetic predisposition. Polygenic risk was first employed to examine the additive association between a trait and multiple SNPs derived from Genome Wide Association Studies (GWAS) (Dudbridge, 2013; Purcell et al., 2009). SNPs from a GWAS have previously been used to create polygenic risk scores for behavioral undercontrol and impulsivity (Salvatore et al., 2014; Vrieze, McGue, Miller, Hicks, & Iacono, 2013). Genome wide studies are useful in their ability to identify SNPs without a priori hypotheses, with each SNP proposed to exert a small effect (Agrawal, Edenberg, & Gelertner, 2016). However, significant SNPs found in one GWAS might not replicate in another (Plomin, 2013), especially if the discovery GWAS is modestly sized, as is currently the case for GWAS of behavioral undercontrol. Furthermore, depending on the sample size, loci of modest effect may be missed by the discovery GWAS. Additionally, at times when a SNP is found to be significantly associated across multiple studies the risk allele indicated can be contradictory (Plomin, 2013). Finally, using a GWAS-based approach in which all SNPs are examined, and significant SNPs are formed into a polygenic score, does not provide a conceptual basis for why certain SNPs were chosen, or their possible functional importance.

In the absence of well-powered discovery GWA analyses, replicated candidate loci may serve as an alternative to a GWAS-based polygenic risk score. We conducted a review of the literature for SNPs previously found to be associated with impulsivity and related measures of behavioral undercontrol, which we then combined into a polygenic risk score for children (see Elam et al., 2016 for details). The same SNPs were used to create polygenic risk scores

for parents to partially control for passive rGE. Full details can be found in the following methods section. This approach is useful as it allows only SNPs with replicated associations to be included, making it more likely that our polygenic score is composed of true genetic signals. In the present study only SNPs for which the identical risk allele replicated across two or more studies were included. This represents a more rigorous approach to choosing candidate loci from a literature which has previously been criticized for a high false discovery rate, and increases confidence our polygenic score is representative of genetic effects. Additionally, this approach allows for a preliminary mechanistic pathway for the action of the risk score. The SNPs in the present score each have an identified functional biological mechanisms (see Table 1), primarily affecting the dopaminergic and serotonergic systems, increasing their likely relevance in the etiology of impulsive behavior. Thus, in the absence of well-powered GWAS, the current polygenic score can be considered a conservative genetic estimate composed of meaningful functionally informed markers.

Previously, we found that this polygenic risk score was associated with children's impulsivity, but found no evidence of an evocative rGE between this polygenic risk score and family conflict (Elam et al., 2016). Based on previous research it is known that indices of behavioral undercontrol, such as impulsivity, are associated with poor parental monitoring (Flannery et al., 1994; Neumann et al., 2010), and that evocative and passive rGEs are unique to parental monitoring, rather than broadly influential on all aspects of the family environment (Neiderhiser et al., 2004, 2007). The present study, therefore, examined evocative rGE underlying children's genetic predisposition for behavioral undercontrol within bidirectional transactions among children's impulsivity in middle childhood, parental monitoring in early adolescence and the subsequent influence of parental monitoring on affiliation with substance-using peers a year and a half later. Within this model a portion of passive rGE was controlled between children's impulsivity and parental monitoring. To control for passive rGE mothers' and fathers' monitoring scores were residualized for their individual polygenic risk scores. This was necessary to control for any genetic variance in monitoring due to polygenic risk for behavioral undercontrol individually for mothers and fathers prior to combining scores for mother's and father's monitoring.

Specifically, we tested the following hypotheses. See Figure 1 for the conceptual model. First, given a wealth of evidence that genetic influences exist for impulsivity (e.g., Bezdjian et al., 2011; Niv et al., 2012), and that genetic effects have recently been found for polygenic risk scores indexing impulsivity and undercontrol (Salvatore et al., 2014; Vrieze et al., 2013) we predicted that our polygenic risk score would be associated with children's impulsivity in middle childhood as indicated by path A in the conceptual model (previously reported in Elam et al., 2016).

Second, because research indicates that genetic predispositions for impulsive and disruptive child behavior evoke poor parenting (e.g., Harold et al., 2013; Pender-Tessler et al., 2013) we predicted that polygenic risk would be associated with parental monitoring in early adolescence as indicated by path C in the conceptual model. This would provide preliminary evidence of evocative rGE.

Third, we predicted that children's impulsivity during middle childhood would mediate the relation between the child's polygenic risk and parental monitoring during early adolescence (paths A and B), which would subsequently be associated with affiliation with substance-using peers a year and a half later in middle adolescence (path D), while controlling for previous levels of substance-using peer affiliation. This would provide evidence of an evocative rGE between polygenic risk and parental monitoring and its subsequent effect on affiliation with substance-using peers. This connects previous genetically sensitive adoption studies (e.g., Harold et al., 2013) and family based literature evidencing such bidirectional transactions between child impulsivity and negative parenting behaviors (e.g., Belsky & Jaffee, 2006; Kiff et al., 2011). It also links previous twin studies finding evidence of rGE for parental monitoring (Neiderhiser et al., 2004, 2007) with literature on parental monitoring and affiliation with deviant peers (Kiesner et al., 2010; Steinberg et al., 1994).

Finally, given evidence of evocative and passive rGE in parental monitoring (Neiderhiser et al., 2004, 2007), we hypothesized that these associations indicative of evocative rGE within parent-child bidirectional transactions would be observed while including mothers' and fathers' polygenic risk in relation to mothers' and fathers' monitoring, controlling for a small portion of passive rGE with respect to polygenic risk for behavioral undercontrol.

2 METHODS

2.1 Participants

Participants were from a larger longitudinal three-generation study of risk for alcoholism (Adolescent/Adult Family Development Project (AFDP); Chassin, Rogosch, & Barrera, 1991). Children in the current sample ($n = 359$), were from the third generation of the study who were living with their two biological parents. The current analyses only included families who had a child 11–15 years old at the early adolescent assessment either of non-Hispanic Caucasian or Hispanic ethnicity, who was living in a two-parent household in which both parents were biologically related to the child. Children were 46.4% female and were assessed in middle childhood (M age = 6.35, $SD = 1.55$), early adolescence (M age = 12.16, $SD = 1.40$), and in middle adolescence a year and a half later (M age = 13.37, $SD = 1.56$). The majority of the child sample was Caucasian (65%) and the remaining proportion was Hispanic (35%). At the early adolescent assessment, parents were on average 35.59 years old ($SD = 4.43$) and the majority of the parent sample was Caucasian (66%) and the remaining proportion was Hispanic (34%).

Families were only included in the current analyses if genetic data were available for the child, with missing mother ($n = 14$) or father ($n = 53$) genetic data permitted. We selected families for the current analyses in which both parents were biologically related to the child because this was necessary to account for genetic correlations among mothers', fathers', and children's polygenic risk scores. We examined all study variables for selection bias in this subsample versus the larger child (third generation) sample. The only study variable significantly different in the subsample was mothers' lifetime substance disorder diagnosis, with lower diagnosis rates present in the current subsample (only biologically related mothers and fathers) as compared to the larger sample which included non-biologically related mothers and fathers ($X^2(2) = 14.84, p = .001$).

Biological samples (buccal and saliva) containing DNA were collected from children and parents. Genotyping was completed at the Washington University Genome Sequencing Center. The Illumina Golden Gate technology was used to genotype 1536 SNPs from a previous array designed for the candidate gene study of addictions (Hodgkinson et al., 2008) with substitutions reflecting advances in the literature and to include ancestry informative markers that distinguished Hispanic and Mexican Americans from other populations. Quality controls included cluster plots to exclude ambiguous genotype calls, examining for Mendelian inconsistencies, improper gender assignments and sample swaps, cryptic relatedness, and flagging SNPs with low call rates ($p < 10^{-6}$).

The larger study focused on the intergenerational transmission of familial alcoholism resulting in elevated rates of parental lifetime alcohol and drug disorder diagnoses in the present sample. In the current sample, 29% of G2 fathers had either a lifetime alcohol or drug disorder diagnosis, and 20% of fathers had both alcohol and drug disorder diagnoses; 18% of G2 mothers had either a lifetime alcohol or drug disorder diagnosis, and 13% of mothers had both alcohol and drug disorder diagnoses. Lifetime substance use disorder diagnoses were controlled for in the present analyses.

2.2 Measures

2.2.1 Polygenic risk—To create the literature-based polygenic risk score a systematic review of the literature was completed in HUGO Navigator Database and Google Scholar to identify studies in which SNPs were significantly associated ($p < .05$) with indices of behavioral undercontrol (see Table 1 for the search terms). We matched significantly associated SNPs within the resulting studies to available SNPs within the AFDP sample or a SNP in high linkage disequilibrium at $r^2 \geq 0.80$. This resulted in 48 SNPs. Additional SNP inclusion criteria were replication in at least one other study and partial or full sample composition of European descent. Adding these criteria resulted in 26 SNPs. SNPs were subsequently pruned for linkage disequilibrium at $r^2 \geq 0.80$ using the pruning option within PLINK, resulting in 25 SNPs. As there is much dissension in the literature regarding the identification of the risk alleles of individual SNPs (e.g., Gizer, Ficks, & Waldman, 2009), we required that a SNP's risk allele be replicated in at least two published studies. To fulfill this requirement we completed a second, targeted literature search to investigate if the refined list of 25 SNPs were associated with broader measures of behavioral undercontrol including substance use. When contradictory risk alleles were reported across multiple studies a risk allele had to be indicated by a margin of at least two studies. Applying this additional criteria resulted in six final SNPs (see Table 1).

Minor allele frequencies of all SNPs were checked in Broad Institute's SNAP database and were below 38%. We combined these SNPs into the present literature-based polygenic risks score using the —score procedure in PLINK (Purcell et al., 2007) and created polygenic risk scores for children, and their biologically related mothers and biologically related fathers. We utilized an additive coding approach, such that for each of the six SNPs, participants could obtain scores between 0 and 2 (i.e., 0, 1, or 2 “risk” alleles; children's scores ranged: 0.17–1.50; skew: 0.26; kurtosis: 0.11; mothers' scores ranged: 0.17–1.33; skew: 0.33;

kurtosis: -0.47 ; fathers' scores ranged: $.00$ – 1.33 ; skew: $-.09$; kurtosis: -0.26 ; means and standard deviations can be found in Table 3).

2.2.2 Ancestry informative markers—The larger dataset of all participants who provided genetic data included 32 ancestry marker SNPs which—in previous literature (Tian et al., 2007)—have differentiated Hispanics from non-Hispanic Caucasians (see Supplemental Table S1 for a list of SNPs). We included these 32 SNPs in a factor analysis in Mplus, using Maximum Likelihood estimation. These factor scores significantly correlated with self-reported ethnicity, both in the larger dataset ($r = 0.86$, $p < .001$) and in the current sample ($r = 0.83$, $p < .001$), suggesting that this ancestry gene score significantly differentiated between non-Hispanic Caucasians and Hispanics. Higher scores indicated lower levels of Hispanic ancestry (Caucasian $M = 0.53$, $SD = 0.34$, range: -1.00 – 1.32 ; skew: -0.76 ; kurtosis: 1.45 ; Hispanic $M = -1.12$, $SD = 0.75$, range: -2.87 – 0.76 ; skew: 0.22 ; kurtosis: -0.53).

2.2.3 Child impulsivity—Child impulsivity was assessed in middle childhood using items from the hyperactive-impulsive subscale of the Disruptive Behavior Rating Scale (DBRS; Barkley & Murphy, 1998). Mothers and fathers rated three items ($0 = \text{never or rarely}$ to $3 = \text{very often}$) regarding levels of impulsivity of the child (e.g., “Has difficulty awaiting turn,” “Blurts out answers before questions have been completed”; $\alpha_s = 0.64$, and 0.74 for mothers and fathers). Combined parent report of children's impulsivity in middle childhood was used to capture an average of parent's perceptions of impulsivity during an age when parents are likely the most accurate reporter of child behavior (middle childhood). Mother and father items were examined using confirmatory factor analysis. Comparing a model with two separate but correlated latent factors, one for mothers' items and one for fathers' items, to a model with one latent factor for both mothers' and fathers' items showed no significant differences (χ^2 difference (1) = 0.70). Factor scores for the single latent variable for both mothers' and fathers' items were extracted into a single measure of child impulsivity.

2.2.4 Parental monitoring—Mothers' and fathers' monitoring was assessed in early adolescence using the five-item monitoring scale developed by Lamborn et al. (1991). Children rated items separately for mothers' and fathers' monitoring on a five-point scale ($1 = \text{Didn't know at all}$ to $5 = \text{Knew all the time}$), which assessed the degree to which mothers' and fathers' were aware of their child's activities and interests (e.g., “What you did with your free time,” “Where you spent most afternoons after school”). Child report of parental monitoring in early adolescence was used to capture children's perceptions of their parent's monitoring and to reduce method variance. Children's report of both mothers' and fathers' monitoring report was found to be reliable ($\alpha = 0.78$, $\alpha = 0.88$), and moderately correlated ($r = 0.50$, $p < .001$).

To control for passive rGE the following steps were taken in SPSS. Mothers' and fathers' polygenic risk scores were residualized from their respective monitoring scores. Unstandardized residuals for mothers and fathers, which represent monitoring scores controlled for polygenic risk, were subsequently averaged together to form a composite of parental monitoring with greater scores indicating greater monitoring. This was necessary to

control for any individual genetic variance in mother's and father's monitoring due to polygenic risk prior to creating a composite measure of parental monitoring.

2.2.5 Affiliation with substance-using peers—Affiliation with substance-using peers was assessed in early adolescence and middle adolescence using an eight-item scale adapted from the Monitoring the Future Study (Osgood, Johnston, O'Malley, & Bachman, 1988). Children rated items on a 6-point scale (1 = *None* to 6 = *All*), which assessed the number of peers who used substances occasionally and regularly (e.g., “How many of your peers drank alcohol occasionally?,” “How many of your peers drank alcohol regularly?”). This was assessed for cigarette, marijuana or hashish, and other drug usage. Scores were averaged across peer substance usage, within each time, to form scores indicating high levels of peer use in early adolescence and middle adolescence. Report of peer use was found to be reliable (early adolescence $\alpha = 0.93$, adolescence $\alpha = 0.92$).

2.2.6 Parent substance abuse—Parents' alcohol and drug abuse and dependence symptoms were assessed using the Substance Abuse Module of the CIDI (World Health Organization, 1990). Diagnoses for abuse and dependence were based on DSM-IV criteria. In the present study, parents were separately coded as having no previous lifetime diagnosis (0), having had either an alcohol or substance disorder diagnosis (1) or having had both alcohol and substance disorder diagnoses (2). This approach was used to capture any additive effects of comorbid alcohol and drug diagnoses (e.g., McGue, Slutske, Iacono, 1999).

2.2.7 Covariates—Covariates were child age, gender, and ancestry. As described earlier, ancestry informative genetic markers were used to control for genetic variation across ethnicity. Two-way interactions among covariates were included to control for their confounding effects, a best practice proposed by Keller (2014) but were not a focus of the present study.

2.3 Statistical analyses

Structural equation modeling (SEM) with full information maximum likelihood for missing data was used to conduct all primary statistical analyses. All relevant statistical assumptions inherent to the application of SEM (e.g., multivariate normality) were examined and affirmed a priori. All models were tested using Mplus 7.11 (Muthén & Muthén, 1998–2016).

Indirect effects were estimated using RMediation (Tofighi & MacKinnon, 2011). As part of the AFDP sample, multiple members were assessed from the same family at both the parent level (siblings) and at the child level (siblings, cousins). To account for this interdependence, clustering at the family level was included in Mplus, which adjusts standard errors for multilevel data. To assess statistical power Monte Carlo simulations were run in Mplus for the proposed mediational model ($n = 360$) with simulations indicating the current analyses were sufficiently powered for the pathways of interest (polygenic score to impulsivity: 0.79; polygenic score to monitoring: 0.78, impulsivity to monitoring: 0.98; monitoring to peer affiliation: 0.97).

Additionally, affiliation with substance-using peers in early adolescence was included as a control, to estimate the effect of parental monitoring on change in affiliation with substance-using peers in middle adolescence. Analyses also controlled for child age and sex, as well as the interactions between ancestry, age, and sex.

3 RESULTS

As an initial test of our selection of SNPs, we tested the relations between a PRS composed of randomly selected SNPs to parents' report of children's impulsivity and children's report of parental monitoring. The children's random PRS was not significantly associated with either children's impulsivity, or children's report of parental monitoring (r 's = .07, and 0.11, respectively, $ps > 0.10$). Mothers' and fathers' random PRS were also unrelated to children's impulsivity and to children's report of parental monitoring (r 's ranged from .03 to .09, all $ps > 0.31$).

Means, standard deviations, and correlations among study variables are presented in Table 2. The general pattern of correlations broadly supports our hypotheses. Children's polygenic risk scores were associated with children's report of parental monitoring. Children's polygenic risk scores were associated with parents' report of children's impulsivity, which was, in turn, associated with parental monitoring.

Next, we used path analysis to examine the hypothesized relations when controlling for covariates. A significant association between children's polygenic risk scores and parent's report of children's impulsivity in middle childhood was evidence of a genetic effect ($B = 0.35$, $SE B = 0.15$, $\beta = 0.15$, $p = .02$; $R^2 = .02$). Following this, the association between children's PRS and children's report of parental monitoring in early adolescence was examined and found to be significant ($B = -0.35$, $SE B = 0.17$, $\beta = -0.11$, $p = .04$), providing preliminary evidence for an evocative rGE.

Finally, a mediated model tested the effect of children's polygenic risk scores on children's report of parental monitoring via parent's report of impulsivity, and the effect of parental monitoring on affiliation with substance-using peers. This model had adequate fit ($n = 359$), $\chi^2(16) = 27.44$, CFI = 0.98, TLI = 0.94, SRMR = .03, RMSEA = .045, $p = .037$. Results are presented within Figure 2, full results can be found in Table 3. Children's PRS was found to significantly predict parents' report of children's impulsivity in middle childhood ($B = 0.34$, $SE B = 0.15$, $\beta = 0.14$, $p = .02$), which in turn predicted children's report of parental monitoring in early adolescence ($B = -0.20$, $SE B = .09$, $\beta = -0.16$, $p = .032$). Parental monitoring predicted affiliation with substance-using peers in middle adolescence while controlling for previous levels of affiliation with substance-using peers in early adolescence ($B = -0.11$, $SE B = .04$, $\beta = -0.10$, $p = .045$). An indirect effect was found from children's PRS to children's report of parental monitoring via children's impulsivity ($\beta = .022$, 95%CI [.001–.056], $p < .05$), indicating evidence of an evocative rGE. This evocative rGE was present while controlling for a portion of passive rGE. An indirect effect was also found from children's impulsivity to affiliation with substance-using peers in middle adolescence via parental monitoring ($\beta = .02$, 95%CI [.001–.049], $p < .05$).

4 DISCUSSION

The present study examined evocative rGE underlying children's genetic predisposition for behavioral undercontrol, children's impulsivity, and parental monitoring in predicting later affiliation with substance-using peers, while controlling for a small portion of passive rGE. A genetic predisposition for behavioral undercontrol was captured using previous literature to select SNPs and determine their risk alleles. Preliminary analyses found children's PRS to be related to parental monitoring, providing preliminary evidence of an evocative rGE. Within the final mediated model the PRS was found to have an indirect effect on children's report of parental monitoring via parent's report of children's impulsivity, supporting the presence of an evocative rGE. This evocative rGE contributed to poorer parental monitoring which in turn predicted greater affiliation with substance-using peers in middle adolescence, while controlling for previous levels of affiliation with substance-using peers. This evocative rGE was found while controlling for a portion of passive rGE between parental polygenic risk for behavioral undercontrol and parental monitoring. These results indicate that when children have a genetic predisposition for behavioral undercontrol (as indexed by the current PRS), that this may evoke poorer monitoring via children's impulsivity, contributing to affiliation with substance-using peers later in life.

The present results support family based theories proposing bidirectional relations between disruptive child characteristics and parenting behavior (e.g., Bell, 1968; Patterson, 1982; Sameroff, 2010). Patterson's coercion theory (1982) posits that negative parent-child exchanges in early to middle childhood can initiate a bidirectional cycle of coercive interaction into adolescence. In the current study, greater impulsivity in middle childhood was found to predict poorer parental monitoring in early adolescence, which in turn contributed to greater affiliation with substance-using peers in middle adolescence. It may be that impulsive children are more difficult to parent, contributing to less engaged parenting (Kiff et al., 2011). When this occurs over the period of adolescence, parents may feel unable or ineffective in monitoring their child's behavior, resulting in poorer monitoring (Racz & McMahon, 2011). This poorer monitoring, in turn, provides adolescents with greater opportunity to affiliate with deviant peers and to experiment in substance use (Racz & McMahon, 2011). This replicates previous literature finding an indirect effect of impulsivity on adolescent's antisocial behavior via monitoring (Neumann et al., 2010), and extends it to affiliation with deviant peers. This study also extends knowledge regarding the genetic underpinnings of these bidirectional transactions.

The present study is the first to examine whether children's genetic predispositions for behavioral undercontrol and resulting impulsivity are implicated in evocative rGE with parental monitoring from middle childhood to early adolescence. Our measure of one aspect of the genetic component of impulsivity was associated with poor parental monitoring. Therefore, children's genetic predisposition for behavioral undercontrol, via children's impulsivity in middle childhood, evoked poor parental monitoring. This is indicative of an evocative rGE. This is consistent with previous findings from twin studies that evocative rGE underlies parental monitoring (Neiderhiser et al., 2004, 2007), and extends findings by identifying a longitudinal relation in which a specific child genetic predisposition (behavioral undercontrol) and child behavior (impulsivity) evoke parental monitoring.

Although previous findings with this PRS did not find evocative rGE with family conflict (Elam et al., 2016), previous literature indicates that evocative rGEs vary across distinct aspects of parenting (Neiderhiser et al., 2004, 2007). Children's genetic predisposition may therefore only evoke certain parenting factors, such as poor monitoring. This indicates that children's genetic predisposition may serve as a latent risk factor for poorer parental monitoring in early adolescence when expressed as impulsivity. This may occur as impulsive children are more disruptive and harder to parent, resulting in poorer monitoring (Neumann et al., 2010). Alternatively, impulsive children may act more erratically and therefore be harder to actively monitor (Waizenhofer, Buchanan, & Jackson-Newsom, 2004), or may disclose less to their parents (Racz & McMahon, 2011).

In addition, these analyses included both mothers' and fathers' PRSs. This approach is useful as it controls for a small portion of passive rGE with parental monitoring, given the association between parents' PRS and monitoring. The presence of passive rGE may be indicated by the correlations in which mothers' and fathers' PRSs were both associated with parental monitoring. The evocative rGE was present while controlling for mothers' and fathers' PRS, suggesting that passive rGE did not spuriously cause the relation between child PRS and parent monitoring. By controlling for a very small portion of passive rGE within this relation, the association between children's impulsivity and parental monitoring is likely due to an environmental effect. This reinforces previous research in non-genetically sensitive samples that have found associations between impulsive child characteristics and parental monitoring (Flannery et al., 1994; Neumann et al., 2010).

Second, this study found evidence that genetically evoked poor parental monitoring is associated with later affiliation with substance-using peers. Few studies have examined child outcomes resulting from genetically evoked parenting (e.g., Elam et al., 2014; Harold et al., 2013). Within a developmental cascades framework (Masten & Cicchetti, 2010), the present genetic predisposition for behavioral undercontrol can be viewed as initiating a cascade within the family that leads to poor parental monitoring, which ultimately contributes to later affiliation with substance-using peers. This approach highlights evocative rGE as a mechanism within the family that contributes to poor monitoring and ultimately later affiliation with substance-using peers.

One alternative explanation for these findings may be that more impulsive children *perceive* poorer parent monitoring, regardless of actual monitoring. We investigated this in post-hoc analyses and found that the correlation between child and parent ratings of monitoring did not differ by levels of children's impulsivity (see Supplemental Table S2). Thus, it is likely that greater impulsivity actually predicted poorer parental monitoring, rather than reflecting children's biased perceptions of parental monitoring based on their impulsivity.

The current study has several important strengths. The use of child report of parental monitoring was used to capture children's perceptions of their parent's monitoring and when combined with parent report of children's impulsivity reduced method variance. Additionally, this study examined evocative rGE longitudinally from middle childhood to early adolescence, while controlling for a small portion of passive rGE, giving greater confidence in the direction of child-to-parent effects. This study also controlled for

affiliation with substance-using peers in early adolescence in predicting affiliation with substance-using peers in middle adolescence from parental monitoring. Finally this study used a polygenic risk score as a measure of children's genetic predisposition, and examined this relative to evocative rGE.

The current study also has limitations to consider. Only 6 SNPs were included in the current polygenic risk score. Traditional GWAS-derived PRS can include thousands of loci, and thus, our approach significantly limits the genetic variance captured within the polygenic risk score, and when controlling for passive rGE limits conclusions regarding environmental transmission between children's impulsivity and parental monitoring. In addition, our reliance on the current literature in the absence of a well-powered GWAS of behavioral undercontrol increases the likelihood that true genetic loci were missed (i.e., false negatives). However, by imposing a more stringent requirement for replication, we attempted to reduce our false discover rate. One potential advantage of our conceptualization of the present PRS is that these SNPs may have functional significance as a number of the SNPs in our polygenic risk score are from the dopamine and serotonin systems. These SNPs may have an effect on the influx of dopamine in the midbrain and prefrontal cortex, affecting planning and inhibition and contributing to impulsive behavior (Jentsch et al., 2014; Koeppe et al., 1998; Thut et al., 1997). However, that hypothesized relationships between neurotransmitter pathways and behavioral outcomes are not definite and we cannot exclude the possibility that some or all of the loci that comprise our PRS might be false positives. Also, passive rGE only exists and is therefore only controlled when child and parent genetic variants are associated with both the child and parenting behaviors, respectively. However, the children's polygenic risk score for impulsivity was significantly associated with impulsivity, and correlations indicated that parents' polygenic risk scores were associated with parental monitoring, both indicative of genetic effects. These associations are noteworthy given that this score is conservative due to multiple inclusion criteria in our literature search, such as requiring SNP effects to be replicated. The polygenic risk score also explained a small amount of variance in children's impulsivity ($R^2 = .02$). This estimate is consistent with current literature and the state of genetic findings using polygenic risk scores (e.g., Salvatore et al., 2014). Power analyses indicate the current sample was sufficiently powered to detect such effects, however, we do note that these estimates are specific to the current sample and proposed population values drawn from the wider literature and indicate that this sample is underpowered to detect effect sizes smaller than 0.10, so false negatives are a possibility. Nonetheless, larger GWAS ascribe much smaller effect sizes to polygenic risk scores and therefore, it will be important in future studies to attempt replication with our polygenic risk score in additional samples. Additionally, the results pertain only to children living in two-biological parent families, which had lower rates of mothers' substance disorder diagnosis than the full sample. Results may not reflect parent-child transactions in single parent households, or to non-biologically related parents. Despite this, mothers' substance disorder diagnoses did predict impulsivity in middle childhood.

The present study extends the current research on gene-environment interplay with parental monitoring in predicting affiliation with substance-using peers. This has important implications for the prevention of youth substance use and conduct problems given the strong role that monitoring plays during adolescence (Dodge & McCourt, 2010).

Preliminary evidence has shown that prevention efforts which incorporate aspects of parental monitoring can dampen genetic influences on youths' risky behavior (Brody, Beach, Philibert, Chen, & Murry, 2009). Typically prevention and intervention programs provide a range of parent training thought to broadly promote positive parenting and improve family climate. Recently, prevention programs have focused on targeting parent's *reactions* to difficult child behavior (e.g., Leve, Harold, Ge, Neiderhiser, & Patterson, 2010). It may be advantageous to specifically promote monitoring in parents' with impulsive children as a way of disrupting later affiliation with substance-using peers. Emphasizing monitoring skills for such parents may better support impulsive children who have underlying risk factors, including genetic predisposition.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding information

National Institute on Alcohol Abuse and Alcoholism, Grant numbers: 2P60AA011998, AA016213, AA021612, AA022097, AA023128; National Institute of Mental Health, Grant number: T32MH018387

References

- Agrawal A, Edenberg HJ, Gelernter J. Meta-analyses of genome-wide association data hold new promise for addiction genetics. *Journal of Studies on Alcohol and Drugs*. 2016; 77:676–680. [PubMed: 27588522]
- Baehne CG, Ehliis AC, Plichta MM, Conzelmann A, Pauli P, Jacob C, Fallgatter AJ. Tph2 gene variants modulate response control processes in adult ADHD patients and healthy individuals. *Molecular Psychiatry*. 2009; 14:1032–1039. [PubMed: 18427560]
- Barkley, RA., Murphy, KR. *Attention-deficit hyperactivity disorder: A clinical workbook*. New York, NY: Guilford Press; 1998.
- Batel P, Houchi H, Daoust M, Ramoz N, Naassila M, Gorwood P. A haplotype of the DRD1 gene is associated with alcohol dependence. *Alcoholism: Clinical and Experimental Research*. 2008; 32:567–572.
- Bell RQ. A reinterpretation of the direction of effects in studies of socialization. *Psychological Review*. 1968; 75:81–95. [PubMed: 4870552]
- Belsky, J., Jaffee, SR. The multiple determinants of parenting. In: Cicchetti, D., Cohen, DJ., editors. *Developmental psychopathology: Risk, disorder, and adaptation*. Hoboken, NH: Wiley; 2006. p. 38–85.
- Bezdjian S, Baker LA, Tuvblad C. Genetic and environmental influences on impulsivity: A meta-analysis of twin, family and adoption studies. *Clinical Psychology Review*. 2011; 31:1209–1223. [PubMed: 21889436]
- Brody GH, Beach SR, Philibert RA, Chen YF, Murry VM. Prevention effects moderate the association of 5-HTTLPR and youth risk behavior initiation: Gene× environment hypotheses tested via a randomized prevention design. *Child Development*. 2009; 80:645–661. [PubMed: 19489894]
- Button TM, Lau JY, Maughan B, Eley TC. Parental punitive discipline, negative life events and gene–environment interplay in the development of externalizing behavior. *Psychological Medicine*. 2008; 38(01):29–39. [PubMed: 17803832]
- Chan TWS, Bates JE, Lansford JE, Dodge KA, Pettit GS, Dick DM, Latendresse SJ. Impulsivity and genetic variants in DRD2 and ANKK1 moderate longitudinal associations between sleep problems

- and overweight from ages 5 to 11. *International Journal of Obesity*. 2014; 38:404–410. [PubMed: 23828101]
- Chassin L, Curran PJ, Hussong AM, Colder CR. The relation of parent alcoholism to adolescent substance use: A longitudinal followup study. *Journal of Abnormal Psychology*. 1996; 105:70–80. [PubMed: 8666713]
- Chassin L, Rogosch F, Barrera M. Substance use and symptomatology among adolescent children of alcoholics. *Journal of Abnormal Psychology*. 1991; 100:449. [PubMed: 1757658]
- Chen LS, Johnson EO, Breslau N, Hatsukami D, Saccone NL, Grucza RA, Rice JP. Interplay of genetic risk factors and parent monitoring in risk for nicotine dependence. *Addiction*. 2009; 104:1731–1740. [PubMed: 20871796]
- Chen P, Jacobson KC. Impulsivity moderates promotive environmental influences on adolescent delinquency: A comparison across family, school, and neighborhood contexts. *Journal of Abnormal Child Psychology*. 2013; 41:1133–1143. [PubMed: 23673971]
- Chen GL, Vallender EJ, Miller GM. Functional characterization of the human TPH2 5' regulatory region: Untranslated region and polymorphisms modulate gene expression in vitro. *Human Genetics*. 2008; 122:645–657. [PubMed: 17972101]
- Deater-Deckard K, Dunn J, O'Connor TG, Davies L, Golding J. Using the stepfamily genetic design to examine gene-environment processes in child and family functioning. *Marriage & Family Review*. 2003; 33(2–3):131–155.
- Derringer J, Krueger RF, Dick DM, Saccone S, Grucza RA, Agrawal A, Gene Environment Association Studies (GENEVA) Consortium. Predicting sensation seeking from dopamine genes a candidate-system approach. *Psychological Science*. 2010; 21:1282–1290. [PubMed: 20732903]
- Dick DM, Aliev F, Wang JC, Grucza RA, Schuckit M, Kuperman S, Goate A. Using dimensional models of externalizing psychopathology to aid in gene identification. *Archives of General Psychiatry*. 2008; 65:310–318. [PubMed: 18316677]
- Dick DM, Meyers JL, Latendresse SJ, Creemers HE, Lansford JE, Pettit GS, Buitelaar JK. CHRM2, parental monitoring, and adolescent externalizing behavior evidence for gene-environment interaction. *Psychological Science*. 2011; 22:481–489. [PubMed: 21441226]
- Dick DM, Kendler KS. The impact of gene-environment interaction on alcohol use disorders. *Alcohol Research: Current Reviews*. 2012; 34:318–324. [PubMed: 23134047]
- Dick DM, Latendresse SJ, Lansford JE, Budde JP, Goate A, Dodge KA, Bates JE. Role of GABRA2 in trajectories of externalizing behavior across development and evidence of moderation by parental monitoring. *Archives of General Psychiatry*. 2009; 66:649–657. [PubMed: 19487630]
- Dick DM, Viken R, Purcell S, Kaprio J, Pulkkinen L, Rose RJ. Parental monitoring moderates the importance of genetic and environmental influences on adolescent smoking. *Journal of Abnormal Psychology*. 2007; 116:213–218. [PubMed: 17324032]
- Dmitrieva J, Chen C, Greenberger E, Ogunseitan O, Ding YC. Gender-specific expression of the DRD4 gene on adolescent delinquency, anger and thrill seeking. *Social Cognitive and Affective Neuroscience*. 2011; 6:82–89. [PubMed: 20203140]
- Dodge KA, McCourt SN. Translating models of antisocial behavioral development into efficacious intervention policy to prevent adolescent violence. *Developmental Psychobiology*. 2010; 52:277–285. [PubMed: 20175096]
- Doehring A, von Hentig N, Graff J, Salamat S, Schmidt M, Geisslinger G, Lötsch J. Genetic variants altering dopamine D2 receptor expression or function modulate the risk of opiate addiction and the dosage requirements of methadone substitution. *Pharmacogenetics and Genomics*. 2009; 19:407–414. [PubMed: 19373123]
- Dudbridge F. Power and predictive accuracy of polygenic risk scores. *PLoS Genetics*. 2013; 9(3):e1003348. [PubMed: 23555274]
- Eisenberg DP, Kohn PD, Hegarty CE, Ianni AM, Kolachana B, Gregory MD, Berman KF. Common variation in the DOPA Decarboxylase (DDC) gene and human striatal DDC activity in vivo. *Neuropsychopharmacology*. 2016; 41(9):2303–2308. [PubMed: 26924680]
- Elam, KK., Chassin, L., Eisenberg, N., Spinrad, TL. Marital stress and children's externalizing behavior as predictors of mothers' and fathers' parenting; *Development and Psychopathology*. 2017. p. 1-14.

- Elam KK, Harold GT, Neiderhiser JM, Reiss D, Shaw DS, Natsuaki MN, Leve LD. Adoptive parent hostility and children's peer behavior problems: Examining the role of genetically informed child attributes on adoptive parent behavior. *Developmental Psychology*. 2014; 50:1543–1552. [PubMed: 24364829]
- Elam KK, Wang F, Bountress K, Chassin L, Pandika D, Lemery-Chalfant K. Predicting substance use in emerging adulthood: A genetically informed Study of developmental transactions between impulsivity and family conflict. *Development and Psychopathology*. 2016; 28:673–688. [PubMed: 27427799]
- Esposito-Smythers C, Spirito A, Rizzo C, McGeary JE, Knopik VS. Associations of the DRD2 TaqIA polymorphism with impulsivity and substance use: Preliminary results from a clinical sample of adolescents. *Pharmacology Biochemistry and Behavior*. 2009; 93:306–312.
- Flannery DJ, Vazsonyi AT, Torquati J, Fridrich A. Ethnic and gender differences in risk for early adolescent substance use. *Journal of Youth and Adolescence*. 1994; 23:195–213.
- Forget-Dubois N, Boivin M, Dionne G, Pierce T, Tremblay RE, Pélusse D. A longitudinal twin study of the genetic and environmental etiology of maternal hostile-reactive behavior during infancy and toddlerhood. *Infant Behavior and Development*. 2007; 30(3):453–465. [PubMed: 17683754]
- Fukui N, Suzuki Y, Sugai T, Watanabe J, Ono S, Tsuneyama N, Someya T. Exploring functional polymorphisms in the dopamine receptor D2 gene using prolactin concentration in healthy subjects. *Molecular Psychiatry*. 2011; 16(4):356–358. [PubMed: 20308994]
- Ge X, Conger RD, Cadoret RJ, Neiderhiser JM, Yates W, Troughton E, Stewart MA. The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology*. 1996; 32(4):574.
- Gizer IR, Ficks C, Waldman ID. Candidate gene studies of ADHD: a meta-analytic review. *Human Genetics*. 2009; 126:51–90. [PubMed: 19506906]
- Hack LM, Kalsi G, Aliev F, Kuo PH, Prescott CA, Patterson DG, Kendler KS. Limited associations of dopamine system genes with alcohol dependence and related traits in the Irish Affected Sib Pair Study of Alcohol Dependence (IASPSAD). *Alcoholism: Clinical and Experimental Research*. 2011; 35:376–385.
- Hamidovic A, Dlugos A, Skol A, Palmer AA, de Wit H. Evaluation of genetic variability in the dopamine receptor D2 in relation to behavioral inhibition and impulsivity/sensation seeking: An exploratory study with d-amphetamine in healthy participants. *Experimental and Clinical Psychopharmacology*. 2009; 17:374–383. [PubMed: 19968402]
- Harold GT, Leve LD, Barrett D, Elam K, Neiderhiser JM, Natsuaki MN, Thapar A. Biological and rearing mother influences on child ADHD symptoms: Revisiting the developmental interface between nature and nurture. *Journal of Child Psychology and Psychiatry*. 2013; 54:1038–1046. [PubMed: 24007415]
- Hay DA, Bennett KS, McStephen M, Rooney R, Levy F. Attention deficit–hyperactivity disorder in twins: A developmental genetic analysis. *Australian Journal of Psychology*. 2004; 56:99–107.
- Hendershot CS, Bryan AD, Ewing SWF, Claus ED, Hutchison KE. Preliminary evidence for associations of CHRM2 with substance use and disinhibition in adolescence. *Journal of Abnormal Child Psychology*. 2011; 39:671–681. [PubMed: 21494862]
- Huang W, Li MD. Differential allelic expression of dopamine D1 receptor gene (DRD1) is modulated by microRNA miR-504. *Biological Psychiatry*. 2009; 65(8):702–705. [PubMed: 19135651]
- Huang W, Ma JZ, Payne TJ, Beuten J, Dupont RT, Li MD. Significant association of DRD1 with nicotine dependence. *Human Genetics*. 2008; 123:133–140. [PubMed: 18092181]
- Hodgkinson CA, Yuan Q, Xu K, Shen PH, Heinz E, Lobos EA, Mann J. Addictions biology: Haplotype-based analysis for 130 candidate genes on a single array. *Alcohol and Alcoholism*. 2008; 43:505–515. [PubMed: 18477577]
- Jentsch JD, Ashenhurst JR, Cervantes MC, Groman SM, James AS, Pennington ZT. Dissecting impulsivity and its relationships to drug addictions. *Annals of the New York Academy of Sciences*. 2014; 1327:1–26. [PubMed: 24654857]
- Kazantseva A, Gaysina D, Malykh S, Khusnutdinova E. The role of dopamine transporter (SLC6A3) and dopamine D2 receptor/ ankyrin repeat and kinase domain containing 1 (DRD2/ANKK1) gene

- polymorphisms in personality traits. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*. 2011; 35:1033–1040. [PubMed: 21354244]
- Keller MC. Gene×environment interaction studies have not properly controlled for potential confounders: The problem and the (simple) solution. *Biological Psychiatry*. 2014; 75:18–24. [PubMed: 24135711]
- Keltikangas-Järvinen L, Puttonen S, Kivimäki M, Elovainio M, Rontu R, Lehtimäki T. Tryptophan hydroxylase 1 gene haplotypes modify the effect of a hostile childhood environment on adulthood harm avoidance. *Genes, Brain and Behavior*. 2007; 6(4):305–313.
- Kerr, M., Stattin, H. Parenting of adolescents: Action or reaction. In: Crouter, AC., Booth, A., editors. *Children's influence on family dynamics: The neglected side of family relationships*. Mahwah, NJ: Lawrence Erlbaum; 2003. p. 121–151.
- Kerr M, Stattin H, Özdemir M. Perceived parenting style and adolescent adjustment: Revisiting directions of effects and the role of parental knowledge. *Developmental Psychology*. 2012; 48:1540–1553. [PubMed: 22448987]
- Kiesner J, Poulin F, Dishion TJ. Adolescent substance use with friends: Moderating and mediating effects of parental monitoring and peer activity contexts. *Merrill-Palmer Quarterly*. 2010; 56:529–556. [PubMed: 21165170]
- Kiff CJ, Lengua LJ, Salewski M. Nature and nurturing: Parenting in the context of child temperament. *Clinical Child and Family Psychology Review*. 2011; 14:251–301. [PubMed: 21461681]
- Klahr AM, Klump KL, Burt SA. The etiology of the association between child antisocial behavior and maternal negativity varies across aggressive and non-aggressive rule-breaking forms of antisocial behavior. *Journal of Abnormal Child Psychology*. 2014; 42(8):1299–1311. [PubMed: 24906982]
- Klahr AM, McGue M, Iacono WG, Burt SA. The association between parent-child conflict and adolescent conduct problems over time: Results from a longitudinal adoption study. *Journal of Abnormal Psychology*. 2011; 120:46–56. [PubMed: 21038930]
- Klahr AM, Thomas KM, Hopwood CJ, Klump KL, Burt SA. Evocative gene-environment correlation in the mother-child relationship: A twin study of interpersonal processes. *Development and Psychopathology*. 2013; 25(01):105–118. [PubMed: 23398756]
- Koepp MJ, Gunn RN, Lawrence AD, Cunningham VJ, Dagher A, Jones T, Grasby PM. Evidence for striatal dopamine release during a video game. *Nature*. 1998; 393:266–268. [PubMed: 9607763]
- Kryski KR, Smith HJ, Sheikh HI, Singh SM, Hayden EP. Evidence for evocative gene-environment correlation between child oxytocin receptor (OXTR) genotype and caregiver behavior. *Personality and Individual Differences*. 2014; 64:107–110.
- Laird RD, Marrero MD, Sentse M. Revisiting parental monitoring: Evidence that parental solicitation can be effective when needed most. *Journal of Youth and Adolescence*. 2010; 39(12):1431–1441. [PubMed: 19763801]
- Laird RD, Pettit GS, Bates JE, Dodge KA. Parents' monitoring-relevant knowledge and adolescents' delinquent behavior: Evidence of correlated developmental changes and reciprocal influences. *Child Development*. 2003; 74:752–768. [PubMed: 12795388]
- Lamborn SD, Mounts NS, Steinberg L, Dornbusch SM. Patterns of competence and adjustment among adolescents from authoritative, authoritarian, indulgent, and neglectful families. *Child Development*. 1991; 62:1049–1065. [PubMed: 1756655]
- Latzman RD, Elkovitch N, Clark LA. Predicting parenting practices from maternal and adolescent sons' personality. *Journal of Research in Personality*. 2009; 43(5):847–855.
- Laucht M, Becker K, Frank J, Schmidt MH, Esser G, Treutlein J, Schumann G. Genetic variation in dopamine pathways differentially associated with smoking progression in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2008; 47:673–681. [PubMed: 18434921]
- Leve LD, Harold GT, Ge X, Neiderhiser JM, Patterson G. Refining intervention targets in family-based research lessons from quantitative behavioral genetics. *Perspectives on Psychological Science*. 2010; 5:516–526. [PubMed: 21188273]
- Mann FD, Kretsch N, Tackett JL, Harden KP, Tucker-Drob EM. Person× environment interactions on adolescent delinquency: Sensation seeking, peer deviance and parental monitoring. *Personality and Individual Differences*. 2015; 76:129–134. [PubMed: 25908885]

- Masten AS, Cicchetti D. Developmental cascades. *Development and Psychopathology*. 2010; 22:491–495. [PubMed: 20576173]
- McGue M, Slutske W, Iacono WG. Personality and substance use disorders: II. Alcoholism versus drug use disorders. *Journal of Consulting and Clinical Psychology*. 1999; 67:394. [PubMed: 10369060]
- Miranda R, Reynolds E, Ray L, Justus A, Knopik VS, McGeary J, Meyerson LA. Preliminary evidence for a gene-environment interaction in predicting alcohol use disorders in adolescents. *Alcoholism: Clinical and Experimental Research*. 2013; 37:325–331.
- Munafo MR, Matheson IJ, Flint J. Association of the DRD2 gene Taq1A polymorphism and alcoholism: A meta-analysis of case-control studies and evidence of publication bias. *Molecular Psychiatry*. 2007; 12:454–461. [PubMed: 17453061]
- Muthén, L.K., Muthén, B.O. *Mplus User's Guide*. Sixth. Los Angeles, CA: Muthén & Muthén; 1998-2016.
- Narusyte J, Neiderhiser JM, Andershed AK, D'Onofrio BM, Reiss D, Spotts E, Lichtenstein P. Parental criticism and externalizing behavior problems in adolescents: The role of environment and genotype-environment correlation. *Journal of Abnormal Psychology*. 2011; 120(2):365. [PubMed: 21280930]
- Neiderhiser JM, Reiss D, Lichtenstein P, Spotts EL, Ganiban J. Father-adolescent relationships and the role of genotype-environment correlation. *Journal of Family Psychology*. 2007; 21:560. [PubMed: 18179328]
- Neiderhiser JM, Reiss D, Pedersen NL, Lichtenstein P, Spotts EL, Hansson K, Elthammer O. Genetic and environmental influences on mothering of adolescents: A comparison of two samples. *Developmental Psychology*. 2004; 40:335–351. [PubMed: 15122961]
- Neumann A, Barker ED, Koot HM, Maughan B. The role of contextual risk, impulsivity, and parental knowledge in the development of adolescent antisocial behavior. *Journal of Abnormal Psychology*. 2010; 119:534–545. [PubMed: 20677842]
- Niv S, Tuvblad C, Raine A, Wang P, Baker LA. Heritability and longitudinal stability of impulsivity in adolescence. *Behavior Genetics*. 2012; 42(3):378–392. [PubMed: 22089391]
- Novak G, LeBlanc M, Zai C, Shaikh S, Renou J, DeLuca V, Le Foll B. Association of polymorphisms in the BDNF, DRD1 and DRD3 genes with tobacco smoking in schizophrenia. *Annals of Human Genetics*. 2010; 74:291–298. [PubMed: 20456319]
- Nyman ES, Loukola A, Varilo T, Taanila A, Hurtig T, Moilanen I, Peltonen L. Sex-specific influence of DRD2 on ADHD-type temperament in a large population-based birth cohort. *Psychiatric Genetics*. 2012; 22:197–201. [PubMed: 22531292]
- Nyman ES, Ogdie MN, Loukola A, Varilo T, Taanila A, Hurtig T, Peltonen L. ADHD candidate gene study in a population-based birth cohort: Association with DBH and DRD2. *Journal of the American Academy of Child & Adolescent Psychiatry*. 2007; 46:1614–1621. [PubMed: 18030083]
- Oades RD, Lasky-Su J, Christiansen H, Faraone SV, Sonuga-Barke EJ, Banaschewski T, Asherson P. The influence of serotonin and other genes on impulsive behavioral aggression and cognitive impulsivity in children with attention-deficit/hyperactivity disorder (ADHD): Findings from a family-based association test (FBAT) analysis. *Behavioral and Brain Functions*. 2008; 4:48–62. [PubMed: 18937842]
- O'Connor TG, Deater-Deckard K, Fulker D, Rutter M, Plomin R. Genotype-environment correlations in late childhood and early adolescence: Antisocial behavioral problems and coercive parenting. *Developmental Psychology*. 1998; 34(5):970. [PubMed: 9779743]
- Osgood DW, Johnston LD, O'Malley PM, Bachman JG. The generality of deviance in late adolescence and early adulthood. *American Sociological Review*. 1988:81–93.
- Pan Y, Luo X, Liu X, Wu LY, Zhang Q, Wang L, Wang KS. Genome-wide association studies of maximum number of drinks. *Journal of Psychiatric Research*. 2013; 47(11):1717–1724. [PubMed: 23953852]
- Pardini DA, Fite PJ, Burke JD. Bidirectional associations between parenting practices and conduct problems in boys from childhood to adolescence: The moderating effect of age and African-American ethnicity. *Journal of Abnormal Child Psychology*. 2008; 36:647–662. [PubMed: 17899362]

- Patterson, GR. A social learning approach. Vol. 3. Eugene, OR: Castalia; 1982. Coercive family process
- Pener-Tessler R, Avinun F, Uzefovsky F, Edelman S, Ebstein RP, Knafo A. Boys' serotonin transporter genotype affects maternal behavior through self control: A case of evocative gene-environment correlation. *Development and Psychopathology*. 2013; 25:151–162. [PubMed: 23398759]
- Plomin R. Child development and molecular genetics: 14 years later. *Child Development*. 2013; 84:104–120. [PubMed: 22469254]
- Plomin R, DeFries JC, Loehlin JC. Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin*. 1977; 84:309–322. [PubMed: 557211]
- Prinzle P, Onghena P, Hellinckx W, Grietens H, Ghesquière P, Colpin H. Parent and child personality characteristics as predictors of negative discipline and externalizing problem behaviour in children. *European Journal of Personality*. 2004; 18(2):73–102.
- Propper CB, Shanahan MJ, Russo R, Mills-Koonce WR. Evocative gene-parenting correlations and academic performance at first grade: An exploratory study. *Development and Psychopathology*. 2012; 24(04):1265–1282. [PubMed: 23062296]
- Purcell SM, Wray NR, Stone JL, Visscher PM, O'Donovan MC, Sullivan PF, O'Dushlaine CT. Common polygenic variation contributes to risk of schizophrenia and bipolar disorder. *Nature*. 2009; 460:748–752. [PubMed: 19571811]
- Purcell S, Neale B, Todd-Brown K, Thomas L, Ferreira MA, Bender D, Sham PC. PLINK: a tool set for whole-genome association and population-based linkage analyses. *The American Journal of Human Genetics*. 2007; 81:559–575. [PubMed: 17701901]
- Racz SJ, McMahon RJ. The relationship between parental knowledge and monitoring and child and adolescent conduct problems: A 10-year update. *Clinical Child and Family Psychology Review*. 2011; 14:377–398. [PubMed: 22086648]
- Reuter M, Hennig J, Amelang M, Montag C, Korkut T, Hueweler A, Stürmer T. The role of the TPH1 and TPH2 genes for nicotine dependence: A genetic association study in two different age cohorts. *Neuropsychobiology*. 2007; 56:47–54. [PubMed: 17986837]
- Salvatore JE, Aliev F, Bucholz K, Agrawal A, Hesselbrock V, Hesselbrock M, Edenberg HJ. Polygenic risk for externalizing disorders gene-by-development and gene-by-environment effects in adolescents and young adults. *Clinical Psychological Science*. 2014; 3:189–201.
- Sameroff A. A unified theory of development: A dialectic integration of nature and nurture. *Child Development*. 2010; 81(1):6–22. [PubMed: 20331651]
- Stattin H, Kerr M. Parental monitoring: A reinterpretation. *Child Development*. 2000; 71:1072–1085. [PubMed: 11016567]
- Steinberg L, Fletcher A, Darling N. Parental monitoring and peer influences on adolescent substance use. *Pediatrics*. 1994; 93:1060–1064. [PubMed: 8197008]
- Thut G, Schultz W, Roelcke U, Nienhusmeier M, Missimer J, Maguire RP, Leenders KL. Activation of the human brain by monetary reward. *Neuroreport*. 1997; 8:1225–1228. [PubMed: 9175118]
- Tian C, Hinds DA, Shigeta R, Adler SG, Lee A, Pahl MV, Gregersen PK. A genomewide single-nucleotide-polymorphism panel for Mexican American admixture mapping. *The American Journal of Human Genetics*. 2007; 80:1014–1023. [PubMed: 17557415]
- Tofighi D, MacKinnon DP. RMediation: An R package for mediation analysis confidence intervals. *Behavior Research Methods*. 2011; 43:692–700. [PubMed: 21487904]
- Trucco EM, Villafuerte S, Heitzeg MM, Burmeister M, Zucker RA. Rule breaking mediates the developmental association between GABRA2 and adolescent substance abuse. *Journal of Child Psychology and Psychiatry*. 2014; 55:1372–1379. [PubMed: 24811113]
- Vrieze SI, McGue M, Miller MB, Hicks BM, Iacono WG. Three mutually informative ways to understand the genetic relationships among behavioral disinhibition, alcohol use, drug use, nicotine use/dependence, and their co-occurrence: Twin biometry, GCTA, and genome-wide scoring. *Behavior Genetics*. 2013; 43(2):97–107. [PubMed: 23362009]
- Waizenhofer RN, Buchanan CM, Jackson-Newsom J. Mothers' and fathers' knowledge of adolescents' daily activities: Its sources and its links with adolescent adjustment. *Journal of Family Psychology*. 2004; 18:348–360. [PubMed: 15222842]

- Walitza S, Renner TJ, Dempfle A, Konrad K, Wewetzer C, Halbach A, Lesch KP. Transmission disequilibrium of polymorphic variants in the tryptophan hydroxylase-2 gene in attention-deficit/hyperactivity disorder. *Molecular Psychiatry*. 2005; 10:1126–1132. [PubMed: 16116490]
- Willoughby T, Hamza CA. A longitudinal examination of the bidirectional associations among perceived parenting behaviors, adolescent disclosure and problem behavior across the high school years. *Journal of Youth and Adolescence*. 2011; 40(4):463–478. [PubMed: 20602155]
- World Health Organization. Composite International Diagnostic Interview (CIDI) (Version 1.0). World Health Organization; Geneva, Switzerland: 1990.
- Zai CC, Muir KE, Nowrouzi B, Shaikh SA, Choi E, Berall L, Kennedy JL. Possible genetic association between vasopressin receptor 1B and child aggression. *Psychiatry Research*. 2012; 200:784–788. [PubMed: 22910476]
- Zhou C, Fryer AD, Jacoby DB. Structure of the human M(2) muscarinic acetylcholine receptor gene and its promotor. *Gene*. 2001; 271:87–92. [PubMed: 11410369]

FIGURE 1.

Conceptual model of evocative rGE among child genetic predisposition, impulsivity, and parent monitoring, and relation to affiliation with substance-using peers. a = direct genetic effect on child impulsivity, b = effect of impulsivity on parent monitoring, c = direct genetic effect on parent monitoring, d = effect of parent monitoring on affiliation with substance-using peers. C path indicates preliminary evidence of evocative rGE with parent monitoring. Mediation indicated by A and B paths provides evidence of evocative rGE through child impulsivity

FIGURE 2.

Mediated model results for children's polygenic risk score, parent rated impulsivity, and children's report of parental monitoring in predicting affiliation with substance-using peers. Analyses control for mother and father lifetime substance disorder diagnoses and polygenic risk, affiliation with substance-using peers in early adolescence, as well as child age, sex, ancestry score, and their interaction but are not shown for readability

TABLE 1

Single nucleotide polymorphisms included in the present literature-based gene score

SNP	Risk allele	Gene	Gene-system function	Phenotypes and studies
rs686	G	DRD1	Modulates density of D1 dopamine receptors via miRNA enzyme regulation (Huang & Li, 2009)	Cognitive impulsivity (Oades et al., 2008); nicotine dependence (Huang et al., 2008); alcohol dependence (Batel et al., 2008); Smoking in Sx (Novak et al., 2010)
rs4648317	T	DRD2	Dopamine regulation of hormone prolactin expression (Fukui et al., 2011).	Impulsivity (Hamidovic, Dlugos, Skol, Palmer, & de Wit, 2009); smoking and nicotine dependence (Laucht et al., 2008); opiate addiction (Doehring et al., 2009)
rs1800497	T	ANKK1	Reduces DA binding sites (Laucht et al., 2008).	Impulsivity (Chan et al., 2014), reward dependence (Kazantseva, Gaysina, Malykh, & Khusnutdinova, 2011); aggression (Zai et al., 2012); opiate addiction (Doehring et al., 2009); impulsivity, conduct problems (Esposito-Smythers, Spirito, Rizzo, McGeary, & Knopik, 2009); ADHD, low persistence (Nyman et al., 2007, 2012); ADHD (Gizer et al., 2009); alcoholism (Munafò, Matheson, & Flint, 2007)
rs11575542	A	DDC	Enzyme production facilitating dopamine and serotonin creation (Eisenberg et al., 2016)	Sensation seeking (Derringer et al., 2010); binge drinking (Pan et al., 2013); drug dependence (Hack et al., 2011)
rs4570625	G	TPH2	Alters gene expression for neuronal serotonin synthesis (Chen, Vallender, & Miller, 2008)	Reduced prefrontal function during inhibition task (Baehne et al., 2009); ADHD (Walitza et al., 2005); smoking (Reuter et al., 2007)
rs1455858	A	CHRM2	Regulates acetylcholine release via feedback at cholinergic terminals (Zhou, Fryer, & Jacoby, 2001).	Impulsivity and sensation seeking (Hendershot, Bryan, Ewing, Claus, & Hutchinson, 2011); alcohol use disorder (Dick et al., 2007); sensation/novelty seeking (Dick et al., 2008)

Note. The phenotypes and studies column refers to the studies from which SNPs in the current polygenic risk score were drawn, and the behavioral undercontrol phenotype with which the referenced SNP was found to be associated. Initial search terms for studies were “SNP” and/or “Gene” plus each of the following: Impulsivity, Novelty seeking, Externalizing, Undercontrol, Delay Discounting, P300, Disinhibition, Stop Signal, Gambling Task, Sensation Seeking, Go-no-go, RiskTaking, Hyperactivity-Impulsivity, Conduct Disorder, Self-Regulation, Reward Seeking, Self-Control. Targeted search terms included substance use, alcohol use, drug use, smoking.

TABLE 2

Means, standard deviations, and correlations between primary constructs

	Mean (SD)	Child polygenic risk score	Mother polygenic risk score	Father polygenic risk score	Child impulsivity (mother-father rated)	Mother substance disorder diagnosis	Father substance disorder diagnosis	Parent monitoring (child rated)	SU peer affiliation early adol. (child rated)
Child polygenic risk score	.63 (.24)	1							
Mother polygenic risk score	.66 (.24)	.52 ****	1						
Father polygenic risk score	.61 (.24)	.52 ****	.20 ***	1					
Child impulsivity (mother-father rated)	.00 (.52)	.10 *	-.05	.007	1				
Mother substance disorder diagnosis	.41 (.69)	-.07	-.12 **	.02	.08	1			
Father substance disorder diagnosis	.64 (.78)	.06	.06	.07	.14 **	.33 ****	1		
Parent monitoring (child rated)	.00 (.73)	-.10 *	-.10 **	-.17 **	-.10 **	-.20 ****	-.23 ****	1	
SU peer affiliation—early adol. (child rated)	1.30 (.62)	.08	.10	.07	-.02	.05	.14 ****	.41 ****	1
SU peer affiliation—middle adol. (child rated)	1.44 (.74)	.07	.02	.10	-.002	.11 ***	.21 ****	-.42 ****	.72 ****

Note. SU = substance using; Adol. = adolescence.

*
p < .10,**
p < .05,***
p < .01,****
p < .001.

TABLE 3

Associations among primary constructs and covariates in the full mediated model

	Child impulsivity (mother-father rated)	Parental monitoring (child rated)	SU peer affiliation— early adolescence (child rated)	SU peer affiliation— middle adolescence (child rated)
	β (SE)	β (SE)	β (SE)	β (SE)
Child polygenic risk score	.14 [*] (.06)	-.05 (.02)	.07 (.04)	-.02 (.04)
Child impulsivity	-	-.16 [*] (.07)	.17 ^{**} (.07)	
Parental monitoring	-	-	-.29 ^{**a} (.06)	-.10 [*] (.04)
Mother substance disorder diagnosis	.09 (.08)	-.18 ^{**} (.07)	.07 (.06)	.03 (.05)
Father substance disorder diagnosis	.05 (.07)	-.02 (.06)	.05 (.06)	.09 [*] (.04)
SU peer affiliation—early adolescence	-	-	-	.63 ^{***} (.06)
Covariates				
Child sex	.26 ^{**} (.08)	.01 (.07)	-.07 (.04)	-.05 (.03)
Child age	-.35 ^{***} (.08)	-.40 ^{***} (.08)	.59 ^{***} (.05)	.16 ^{**} (.05)
Ancestry	.11 (.09)	-.02 (.12)	-.07 (.04)	-.05 (.04)
Age × sex	-.09 (.10)	-.01 (.06)	-.05 (.07)	-.04 (.04)
Ancestry × sex	-.05 (.06)	-.01 (.08)	.03 (.04)	-.04 (.04)
Ancestry × age	.02 (.08)	.25 ^{**} (.09)	-.18 [*] (.07)	-.01 (.06)

Note. SU = substance using.

^aThe estimate between parental monitoring and affiliation with substance-using peers in early adolescence is a correlation.

*
p < .05,

**
p < .01,

p < .001.