Developmental Psychology
2017, Vol. 53, No. 2, 329–339

0012-1649/17/$12.00 http://dx.doi.org/10.1037/dev0000217

BRIEF REPORT

The Spread of Substance Use and Delinquency Between Adolescent Twins

Brett Laursen and Amy C. Hartl
Florida Atlantic University

Frank Vitaro
Université de Montréal

Mara Brendgen
Université du Québec à Montréal

Ginette Dionne
Université Laval

Michel Boivin
Université Laval and Tomsk State University

This investigation examines the spread of problem behaviors (substance use and delinquency) between twin siblings. A sample of 628 twins (151 male twin pairs and 163 female twin pairs) drawn from the Quebec Newborn Twin Study completed inventories describing delinquency and substance use at ages 13, 14, and 15. A 3-wave longitudinal actor–partner interdependence model (APIM) identified avenues whereby problem behaviors spread from one twin to another. Problems did not spread directly between twins across domains. Instead, 2 indirect pathways were identified: (a) Problems first spread interindividually (between twins) within a behavioral domain, then spread intraindividually (within twins) across behavioral domains (e.g., Twin A delinquency → Twin B delinquency → Twin B substance use); and (b) problems first spread intraindividually (within twins) across behavioral domains, then spread interindividually (between twins) within a behavioral domain (e.g., Twin A delinquency → Twin A substance use → Twin B substance use). Controls for genetic effects, gene–environment correlations, friend substance use and delinquency, and parenting behaviors increase confidence in the conclusion that twin siblings uniquely contribute to the spread of problem behaviors during adolescence. Twin sibling influence is a risk factor for illicit substance use, both because substance use by one twin predicts substance use by the other twin, but also because delinquency in one twin predicts delinquency in the other twin, which then gives rise to greater substance use.

Keywords: substance use, delinquency, sibling influence, problem behavior, twins

Delinquency and substance use are fellow travelers. Odds ratios indicate a three- to fivefold increase in delinquency among adolescents who abuse alcohol (Armstrong & Costello, 2002). Siblings bear some responsibility for the spread of problem behaviors. Twin and adoption studies indicate that siblings are a unique source of social influence, separate from parents and peers (Rende, Slomkowski, Lloyd-Richardson, & Niaura, 2005). Adolescents with a delinquent brother or sister are more likely to misuse alcohol and other substances than those without a delinquent sibling (Stormshak, Comeau, & Shepard, 2004). Although examples of behavioral convergence are compelling, they offer no explanation as to how problems spread from one sibling to another across different domains of misconduct. To better understand the processes responsible for the transmission of substance use and delinquency, we applied an innovative genetically controlled design to a longitudinal sample of monozygotic (MZ) and same-sex dizygotic (DZ) twins, disentangling the spread of problem behaviors between twin siblings from genetic and other environmental factors that underlie some of the overlap in the growth of adjustment difficulties.

One puzzle confronting scholars is how problems spread between siblings (interindividual) across different forms of misconduct (interdomain). Direct mechanisms have been proposed to explain how one sibling's delinquency could promote the other sibling's substance use. Delinquent adolescents affiliate with delinquent friends, many of whom drink heavily, smoke cigarettes,

This article was published Online First November 10, 2016.

Brett Laursen and Amy C. Hartl, Department of Psychology, Florida Atlantic University; Frank Vitaro, Département de Psychoéducation, Université de Montréal; Mara Brendgen, Département de Psychologie, Université du Québec à Montréal; Ginette Dionne, École de psychologie, Université Laval; Michel Boivin, École de psychologie, Université Laval and Institute of Genetic, Neurobiological, and Social Foundations of Child Development, Tomsk State University.

Funding for this study was provided by the Social Sciences and Humanities Research Council of Canada, the Canadian Institutes of Health Research, and the Fonds Québécois de Recherche sur la Société et la Culture. We thank Jocelyn Malo and Marie-Elyse Bertrand for coordinating the data collection and Hélène Paradis for data management and preparation. We also thank the twins and their families as well as their friends for participating in this study. Brett Laursen received support from the U.S. National Institute of Child Health and Human Development (HD068421) and the U.S. National Science Foundation (0909733).

Correspondence concerning this article should be addressed to Brett Laursen, Department of Psychology, Florida Atlantic University, 3200 College Avenue, Fort Lauderdale, FL 33304. E-mail: laursen@fau.edu

and use drugs (Ferguson & Meehan, 2011). Thus, one sibling could expose the other to new models for the consumption of illicit substances and new sources of peer pressure. It may also be the case that exposure to delinquent behavior erodes respect for norms, breaking down taboos against substance use (Kaplan, Martin, & Robbins, 1984).

Indirect mechanisms may also account for the spread of problems between siblings, across different forms of misconduct. Two distinct, sequential processes may explain how one sibling's delinquency could promote increases in the other sibling's substance use. First, problems may spread between siblings (interindividual) within a specific form of misconduct (intradomain). Most theories on the adoption of problem behaviors during adolescence emphasize exposure and conformity pressures (Petraitis, Flay, & Miller, 1995). Deviant siblings may persuade nondeviant siblings to participate in illicit activities and may serve as a model for deviant behavior. Second, problems may spread across different forms of misconduct (interdomain) within each sibling (intraindividual). Delinquency may lead to substance use in the same individual because deviant acts are often conducted in the company of deviant affiliates who also have access to alcohol and drugs (Mason, Hitchings, McMahon, & Spoth, 2007). Increases in one sibling's substance use can spread to the other sibling through modeling and reinforcement. Working together, the two processes (in either order) may explain how problems spread between siblings across different forms of misconduct.

There is scarce evidence that behavior problems spread between partners, across domains. Results from prior longitudinal studies fail to reveal direct associations between one sibling's delinquent behavior and the other sibling's substance use (e.g., Low, Shortt, & Snyder, 2012). The same is true for friends: The only studies that suggest interindividual interdomain influence involve deviance and substance use measures that contain overlapping latent constructs (e.g., Dishion & Owen, 2002), making it impossible to determine whether and how problems spread. Few studies have explored the indirect spread of problems. There is support, however, for each of the separate steps described above (e.g., intraindividual, interdomain processes: Buist, 2010; Low et al., 2012 and interindividual intradomain processes: D'Amico, Edelen, Miles, & Morral, 2008). To date, no studies have included both steps in the same model to establish the temporal order of effects.

In the present study, we adopt a quasicausal approach to identifying environmental risk from the sibling in the spread of problem behaviors. Sibling and especially twin comparison designs represent a form of quasiexperimental research that can be used to test causal environmental hypotheses (Lahey & D'Onofrio, 2010). Their use is particularly important when randomized experiments are not feasible. Genetically informed studies suggest that genetic and shared environmental contributions contribute to twin similarity on antisocial behavior and substance use; environmental factors that are not shared between siblings also account for a large part of the variance (e.g., Carey, 1992; Cleveland, Wiebe, & Rowe, 2005). Genetically informed designs are an important step in isolating potentially causal environmental effects (Turkheimer & Harden, 2014). Twin comparisons, especially of MZ twins who are genetically identical, control for genetic influences and rule out gene–environment correlations, helping to isolate intertwined effects that are otherwise difficult to disentangle. Shared environmental confounds are also eliminated among twins who live in the same family environment. Comparisons between DZ and MZ twins can strengthen confidence in causal conclusions about sibling influences. Yet despite these advantages, few studies have examined influence between cotwins in a longitudinal framework.

Nonindependent data pose another methodological and statistical obstacle to sibling research. The use of correlated partner reports violates assumptions of statistical independence and renders traditional parametric statistics inappropriate (Kenny, 1995). To overcome these challenges, the actor–partner interdependence model (APIM; Kenny, Kashy, & Cook, 2006) partitions variance shared across partners on the same variable from variance that uniquely describes associations within partners (intraindividual) and between partners (interindividual). Modifications for longitudinal data address over time influence (Laursen, Popp, Burk, Kerr, & Stattin, 2008). A longitudinal APIM is akin to a residual change model, in that autoregressive effects are included that describe the stability of a variable (Popp, Laursen, Kerr, Stattin, & Burk, 2008). By controlling for stability and within-time correlations, residual change can be predicted. The present study is unique in that it introduces a test for direct and indirect effects within the framework of a three-wave, two-variable indistinguishable dyad longitudinal APIM using sibling twin data in such a way as to isolate sibling effects, net of the genetic and shared environmental influences that make members of a twin pair similar. The use of APIM analyses with a sample of same-sex twins raised together can thus provide a powerful test of the potential environmental effect from one sibling to the other while controlling for genetic and shared environmental contributions in the emergence of behavior problems. Although the APIM strategy does not permit estimation of the extent to which confounding factors are genetic or environmental, it is an excellent strategy for investigating potential causal effects between siblings after controlling for unmeasured familial confounding factors. Moreover, by showing that cross-twin effects vary by zygosity, we can establish that genetic factors (including gene–environment correlations) do not account for the putative sibling influence processes (see Slutske et al., 2008 for a similar approach). We also control for other potential confounding factors, such as characteristics of parents and peers, to isolate the contributions of siblings. Previous studies (Mason & Windle, 2002; Trim, Leuthe, & Chassin, 2006) found that sibling influence on substance use and delinquency varied for boys and girls and for those in households with and without marital troubles, so we will consider the possibility that direct and indirect processes vary by sex and family structure.

The analyses describe sibling transmission of substance use and delinquency. Two competing hypotheses have been proposed to explain how problems spread between twins (interindividual) across different forms of misconduct (interdomain). *Direct mechanism models* posit a single-step transmission process (e.g., Twin A delinquency → Twin B substance use). The single-step process is illustrated in Figure 1 with cross-lagged partner paths (*g* and *h*) that represent interindividual, interdomain transmission. *Indirect mechanism models* posit a two-step transmission process (e.g., Twin A delinquency → Twin A substance use → Twin B substance use). One step in the process, illustrated with cross-lagged actor paths (*e* and *f*), represents the spread of problems within twins (intraindividual), across different forms of misconduct (interdomain). The other step in the process, illustrated with cross lagged partner paths (*c* and *d*), represent the spread of problems

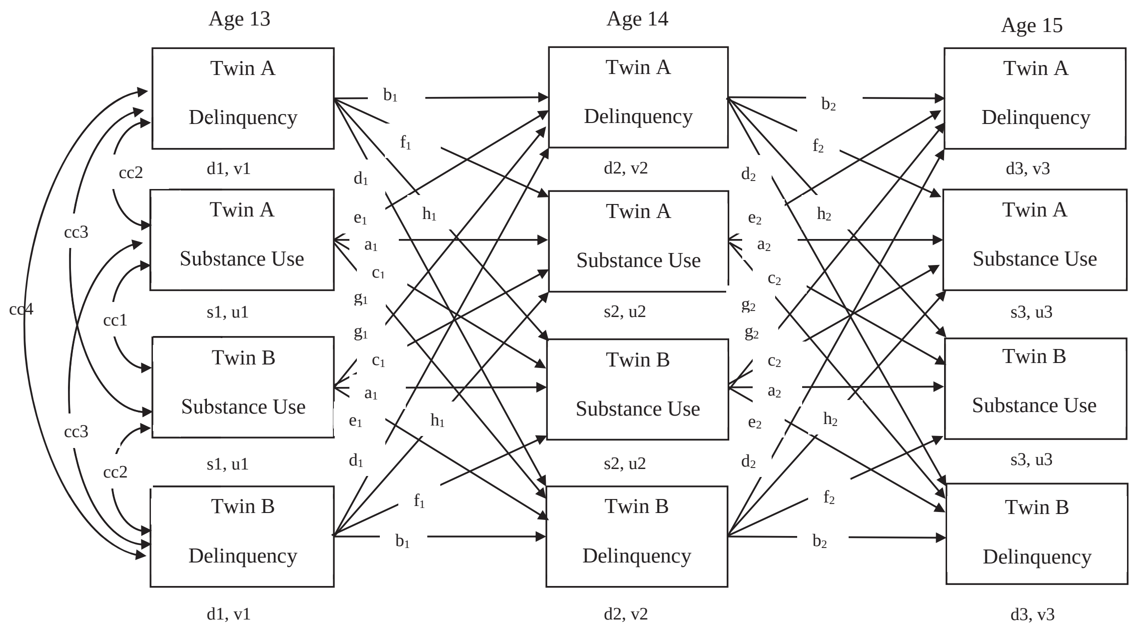

Figure 1. Measurement model of longitudinal actor–partner interdependence model for indistinguishable dyads assessing interindividual and intraindividual associations between delinquency and substance use from age 13 to age 14 and from age 14 to age 15. Concurrent age 14 and age 15 error covariances are not depicted. Identical labels reflect equality constraints.

between siblings (interindividual), within the same form of misconduct (intradomain). The sequence of steps in an indirect model can occur in either order.

Method

Participants

The 628 participants (302 boys, 326 girls) belonged to 179 MZ and 135 same-sex DZ twin pairs, drawn from the Quebec Newborn Twin Study, an ongoing longitudinal study of a population-based sample of twins born between 1995 and 1998 in the greater Montreal area (Boivin et al., 2013). Zygosity was assessed at 18 months through the analysis of genetic markers, supplemented by diagnoses based on physical similarity (Forget-Dubois et al., 2003). Ten highly polymorphous genetic markers were tested. Zygosity comparisons revealed a 96% correspondence rate, which is similar to rates obtained in older twin samples.

Demographic characteristics of the twin families were comparable to those of a sample of single births representative of urban centers in the province of Quebec (Santé Québec, Jetté, Desrosiers, & Tremblay, 1998). At the outset, 95% of parents lived together, 66% of mothers and 60% of fathers were between 25 and 34 years old, 17% of mothers and 14% of fathers had not finished high school, 28% of mothers and 27% of fathers held a university degree, 83% of parents were employed, and 10% of the families received social welfare or unemployment insurance. Eighty-four percent of the families were of European descent, 3% were of African descent, 2% were of Asian descent, 2% were Native North Americans, and 9% did not specify ethnicity.

Data for the present study were collected at ages 13 (Grade 7), 14 (Grade 8), and 15 (Grade 9), via personal interviews in the twins' homes. When the twins were 13 years old, mothers were, on average 43.5 years old ($SD = 4.74$), and fathers were, on average, 45.8 years old ($SD = 5.29$). Active written consent from the children and parents was obtained. Data collection was approved by the Institutional Review Boards of the University of Quebec in Montreal and the Ste.-Justine Hospital Research Center.

Of the 662 twin pairs in the initial sample, 453 participated in data collection at age 13, 14, or 15. There were no statistically significant differences between those who did and did not participate on family income, family structure, or birth weight, nor did they differ on a variety of problem behaviors in kindergarten, Grade 1, and Grade 4.

Tests of distinguishability (Kenny et al., 2006) indicated that the 139 mixed-sex DZ twin pairs who also participated in the Quebec Newborn Twin Study could be distinguished on the basis of sex for the main variables in the study, $\Delta\chi^2(34) = 173.32$, $p < .05$, so mixed-sex DZ twins were excluded from the indistinguishable dyad APIM analyses. The final sample included 628 participants (302 boys and 226 girls), consisting of 179 MZ twin pairs and 135 same-sex DZ twin pairs.

Measures

Instruments were administered either in English (21%) or in French (79%), depending on the language spoken by the children and their parents. Back-translation procedures were employed and bilingual translators verified the semantic similarity of the questionnaires. Further details on the instruments are given in the Appendix.

Substance use. At each wave, participants completed the Personal Experience Screening Questionnaire (Henly & Winters, 1989; Winters, Stinchfield, Henly, & Schwartz, 1990). *Substance use* was assessed with four items that separately described alcohol use, marijuana use, binge drinking, and other drug use. Participants rated the frequency of each during the past 12 months on a scale ranging from 1 (*never*) to 7 (*daily*). At the outset, approximately 41.4% of participants reported experience with at least one substance at least once and approximately 7.1% of participants used at least one substance regularly (i.e., more than once a month). Item scores were averaged. Internal reliability was adequate ($\alpha = .74$).

Delinquency. At each wave, participants completed the Self-Report Delinquency Scale (LeBlanc & Fréchette, 1989). *Delinquency* was assessed with nine items (e.g., "Have you stolen something from parents or strangers?") that described specific delinquent behaviors. Participants rated the frequency of each during the past 12 months on a scale ranging from 1 (*never*) to 4 (*very often*). At the outset, approximately 68.5% of participants engaged in at least one delinquent behavior at least once and approximately 3.4% of participants engaged in at least one delinquent behavior regularly (i.e., very often). Item scores were averaged. Internal reliability was adequate ($\alpha = .68$–.69).

Confounding variables. In addition to demographic variables, supplemental analyses included nine different confounding variables. When the twins were 13 years old, each was asked to nominate up to five friends. With the permission of the twins, we contacted those nominated to complete the same substance use and delinquency inventory as the twins completed. *Friend substance use* and *friend delinquency* represent scores from each twin's first nominated friend, and *peer group substance use* and *peer group delinquency* represent the average score of all friends nominated by each twin ($\alpha = .53$–.62). When the twins were 13 years old, mothers completed a parenting questionnaire drawn from scales used at earlier time points in the study (Boivin et al., 2005; Strayhorn & Weidman, 1988), adapted for use with adolescents. *Inconsistent parenting* was measured with three items rated on a scale from 1 (*never*) to 5 (*always*). Internal reliability was adequate ($\alpha = .66$). *Punishment* was measured with four items rated on a scale from 1 (*never*) to 5 (*always*). Internal reliability was good ($\alpha = .71$). *Conflict* was measured with 10 items rated on a scale from 1 (*not at all*) to 5 (*almost always*). Internal reliability was good ($\alpha = .75$). *Positive interaction* was measured with five items rated on a scale from 1 (*never*) to 7 (*many times a day*). Internal reliability was good ($\alpha = .79$). *Parental efficacy* was measured with 10 items rated on a scale from 1 (*not at all how I feel or think*) to 10 (*exactly how I feel or think*). Internal reliability was good ($\alpha = .79$).

Plan of Analysis

Preliminary analyses examine the relative strength of genetic and environmental effects on substance use and delinquency to illustrate the degree to which genetic and shared environmental effects may have affected previous studies of sibling influence. The effects of each can be estimated by comparing the within-pair correlations of the MZ twin pairs with those of the DZ twin pairs (Falconer, 1989). The relative strength of genetic effects is approximately twice the MZ and same-sex DZ within pair correlation difference, $g^2 = 2(r_{MZ} - r_{DZ})$. The relative strength of shared environmental effects can be estimated by subtracting the MZ correlation from twice the DZ correlation, $c^2 = (2r_{DZ}) - (r_{MZ})$. Nonshared environmental effects can be approximated by the extent to which the MZ correlation is less than 1, $e^2 = 1 - r_{MZ}$. Comparison of the within-pair, cross-phenotype correlations for the MZ twin pairs with those of the DZ twin pairs indicates whether a similar set of genetic (or shared environmental) influences contribute to sibling substance use and delinquency (Neale & Maes, 2004). If left uncontrolled, these genetic and shared environmental influences could mask possible interindividual influences.

Path analyses were conducted in a structural equation modeling framework using *Mplus* Version 7.12 (Muthén & Muthén, 1998–2014). Figure 1 depicts the three-wave longitudinal APIM measurement model. Identical labels indicate paths that were constrained to be equal to reflect the interchangeable nature of the twin participants (Olsen & Kenny, 2006). Equality constraints include intraindividual (i.e., within-twin) intradomain (within forms of misconduct) stability paths (a_1 and a_2, b_1, and b_2), interindividual (i.e., between-twin) intradomain (within forms of misconduct) influence paths (c_1 and c_2, d_1, and d_2), intraindividual (i.e., within-twin) interdomain (across forms of misconduct) influence paths (e_1 and e_2, f_1, and f_2), and interindividual (i.e., between-twin) interdomain (across forms of misconduct) influence paths (g_1 and g_2, h_1, and h_2). Equality constraints were also applied to means, variances, intercepts, residuals, covariances, and error covariances across twin-siblings.

The APIM analyses account for interindividual correlations at a given time-point (both within and between domains). In the twin sample, these correlations reflect genetic and shared environmental effects on problem behaviors that make members of a twin pair similar. The APIM also accounts for the temporal stability and intraindividual interdomain (within-twin between-domain) associations that reflect genetic and (shared and nonshared) environmental contributions to the stability and intrapersonal spread of problem behaviors. The remaining influence paths reflect the interindividual intradomain spread of behavior problems (between twins within domains) and the interindividual interdomain spread of behavior problems (between twins between domains), over and above the contribution of genetic effects and shared-environmental effects.

A progressive model fitting procedure was employed. First, the measurement model depicted in Figure 1 was estimated. Constraints were then added to analogous influence paths at consecutive time points (e.g., d_1 Twin A age 13 delinquency to Twin B age 14 delinquency and d_2 Twin A age 14 delinquency to Twin B age 15 delinquency). Constraints were removed if model fit significantly worsened ($p < .05$). Fit indices for the indistinguishable dyad APIM were adjusted as recommended (Olsen & Kenny, 2006).

Indirect pathways examined the indirect spread of problems between twins across different forms of misconduct. Two indirect pathways begin with interindividual intradomain processes, followed by intraindividual interdomain processes: (a) One twin's age 13 delinquency predicts the other twins age 14 delinquency, which predicts the same twin's age 15 substance use (e.g., Twin A age 13 delinquency → Twin B age 14 delinquency → Twin B age 15 substance use); (b) one twin's age 13 substance use predicts the other twin's age 14 substance use, which predicts the same twin's

age 15 delinquency (e.g., Twin A age 13 substance use → Twin B age 14 substance use → Twin B age 15 delinquency). Two indirect pathways begin with intraindividual interdomain processes, followed by interindividual intradomain processes: (a) One twin's age 13 delinquency predicts the same twin's age 14 substance use, which predicts the other twin's age 15 substance use (e.g., Twin A age 13 delinquency → Twin A age 14 substance use → Twin B age 15 substance use); (b) one twin's age 13 substance use predicts the same twin's age 14 delinquency, which predicts the other twin's age 15 delinquency (e.g., Twin A age 13 substance use → Twin A age 14 delinquency → Twin B age 15 delinquency).

An average of 11.6% (range: 0.0%-23.8%) of study variable data were missing. Little's test indicated that data were missing completely at random, $\chi^2(27) = 28.79$, $p = .37$. Missing data were handled with full information maximum-likelihood estimation, which allowed participants with incomplete data to be included in the models.

Multiple group analyses were conducted separately with sex, zygosity, and family structure as moderators. There were no statistically significant χ^2 differences in the pattern of associations. Supplemental analyses included the following confounding variables, entered into the model as correlated paths at each age: family income; maternal reports of punishment, conflict, inconsistent parenting, positive interaction, and perceptions of parental efficacy; friend reports of substance use and delinquency; peer group substance use, and peer group delinquency. Confounding variables were included in the analyses to control for characteristics of parents and peers, to isolate the unique contributions of sibling twins. The same pattern of statistically significant results as in the final model emerged.

Results

Preliminary Analyses

Separate 2 (Sex) × 2 (Zygosity) × 3 (Age) repeated measures analyses of variance were conducted with substance use and delinquency as the dependent variables. To avoid statistical bias arising from nonindependence, one member of each twin pair was randomly selected for inclusion in these analyses. There were statistically significant main effects of age on substance use, $F(1, 202) = 82.26$, $p < .001$, $\eta^2 = 0.29$ (95% confidence interval [CI] [0.19, 0.38]), and delinquency, $F(1, 202) = 9.93$, $p = .002$, $\eta^2 = 0.05$ (95% CI [0.01, 0.11]). Substance use increased over time (age 13 $M = 1.14$, $SD = 0.30$; age 14 $M = 1.28$, $SD = 0.49$; age 15 $M = 1.67$, $SD = 0.85$), as did delinquency (age 13 $M = 1.10$, $SD = 0.16$; age 14 $M = 1.11$, $SD = 0.16$; age 15 $M = 1.15$, $SD = 0.21$). There were no other statistically significant main effects nor were there any interactions.

Bivariate correlations revealed statistically significant positive concurrent ($r = .30$ to $.57$, $p < .001$) and over time ($r = .27$ to $.47$, $p < .001$) associations between substance use and delinquency. Over time autocorrelations were also statistically significant for substance use ($r = .38$ to $.63$, $p < .001$) and delinquency ($r = .52$ to $.68$, $p < .001$).

Table 1 presents within-pair correlations for study variables, separately for MZ twins and same-sex DZ twins. MZ twin within-pair correlations were larger than same-sex DZ twin within-pair correlations at each time point, indicating a genetic component in substance use, (range: 24–50%) and delinquency (range: 22–50%). The results indicate a substantial shared environmental component in substance use (range: 16–30%); in contrast, the shared environmental component in delinquency was modest (range: 0–16%). MZ twin within-pair correlations were considerably less than 1.0 for all study variables, indicating the presence of a nonshared environmental component in substance use (range: 34–47%) and delinquency (range: 46–62%). MZ twin within-pair, cross-phenotype correlations were higher than same-sex DZ twin within-pair, cross-phenotype correlations at each time point, suggesting some overlap in genetic contributions to substance use and delinquency (Neale & Maes, 2004).

Table 1
Within-Pair Correlations for Monozygotic (MZ) and Same-Sex Dizygotic (DZ) Twins

Variable	MZ	DZ
Age 13 delinquency	$.38^{**}$ [.25, .51]	$.27^{*}$ [.10, .43]
Age 14 delinquency	$.54^{**}$ [.42, .65]	$.29^{*}$ [.12, .45]
Age 15 delinquency	$.47^{**}$ [.34, .60]	$.22^{*}$ [.04, .30]
Age 13 substance use	$.53^{**}$ [.41, .64]	$.38^{**}$ [.23, .54]
Age 14 substance use	$.54^{**}$ [.43, .65]	$.42^{**}$ [.27, .57]
Age 15 substance use	$.66^{**}$ [.56, .75]	$.41^{**}$ [.25, .57]
Age 13 delinquency – substance use	$.28^{**}$ [.18, .37]	$.13^{**}$ [.08, .18]
Age 14 delinquency – substance use	$.25^{**}$ [.16, .34]	$.21^{**}$ [.13, .28]
Age 15 delinquency – substance use	$.41^{**}$ [.31, .50]	$.27^{**}$ [.20, .35]

Note. $N = 628$ participants in 314 twin pairs (179 MZ twin pairs and 135 same-sex DZ twin pairs). 95% confidence intervals given in brackets.
* $p < .05$. ** $p < .001$, two-tailed.

Longitudinal APIM Analyses Describing the Spread of Substance Use and Delinquency

Table 2 describes the results of the final model. The model fit the data, $\chi^2(2, N = 314 \text{ twin pairs}) = 0.38$, $p > .05$, Tucker–Lewis index = 1.02, root mean square error of approximation = .00. Table 1 describes results for cross-lagged paths describing interindividual and intraindividual influence across two consecutive time points.

The spread of problem behaviors from age 13 to age 14 and from age 14 to age 15: Interindividual processes. The paths for interindividual influence indicated that problem behaviors spread between twins within each form of misconduct (intradomain) but not between twins across different forms of misconduct (interdomain). There were statistically significant paths from one twin's substance use to the other twin's subsequent substance use (c_1 and c_2) and from one twin's delinquency to the other twin's subsequent delinquency (d_1 and d_2). In each case, higher initial levels of one twin's problem behavior predicted greater increases in the other twin's levels of the same problem behavior (e.g., Twin A delinquency → Twin B delinquency and Twin A substance use → Twin B substance use), from age 13 to 14 and from age 14 to 15. There were neither sex (c_1 and c_2 boys/girls 95% CI [.03, .20]/[.08, .24]; d_1 and d_2 boys/girls 95% CI [.04, .19]/[.04, .20]) nor zygosity (c_1 and c_2 MZ/DZ 95% CI [.06, .22]/[.09, .26]; d_1 and d_2 95% CI [.05, .20]/[.03, .18]) differences in either set of interindividual, intradomain paths. There were no statistically significant paths from one twin's substance use to the other twin's

Table 2
Longitudinal Actor–Partner Interdependence Model Results Describing the Direct Spread of Problem Behaviors

Path (Figure 1 path label)	Age 13 to 14		Age 14 to 15	
	β (*SE*)	[95% CI]	β (*SE*)	[95% CI]
Interindividual processes across domains				
Substance use → Delinquency (g_1 and g_2)	.08 (.05)	[−.01, .17]	−.02 (.04)	[−.09, .05]
Delinquency → Substance use (h_1 and h_2)	.00 (.04)	[−.08, .08]	.02 (.04)	[−.05, .10]
Interindividual processes within domains				
Substance use → Substance use (c_1 and c_2)	.14** (.03)	[.08, .20]	.14** (.03)	[.08, .19]
Delinquency → Delinquency (d_1 and d_2)	.12** (.03)	[.06, .18]	.09** (.03)	[.08, .19]
Intraindividual processes across domains				
Substance use → Delinquency (e_1 and e_2)	.06 (.05)	[−.02, .15]	.13* (.04)	[.06, .21]
Delinquency → Substance use (f_1 and f_2)	.23** (.04)	[.16, .31]	.25** (.04)	[.17, .33]
Intraindividual processes within domains (stability)				
Substance use → Substance use (a_1 and a_2)	.28** (.04)	[.20, .39]	.43** (.04)	[.36, .51]
Delinquency → Delinquency (b_1 and b_2)	.47** (.03)	[.40, .54]	.62** (.03)	[.56, .68]

Note. CI = confidence interval. $N = 628$ participants in 314 twin pairs.
* $p < .05$. ** $p < .001$, two-tailed.

delinquency (g_1 and g_2) and from one twin's delinquency to the other twin's substance use (h_1 and h_2). There were neither sex (g_1 and g_2 boys/girls 95% CI [−.12, 0.18]/[−.12, .22]; h_1 and h_2 boys/girls 95% CI [−.15, .09]/[−.11, 0.18]) nor zygosity (g_1 and g_2 MZ/DZ 95% CI [−.07, .22]/[−.19, .18]; h_1 and h_2 MZ/DZ 95% CI [−.03, .17]/[−.18, .07]) differences in either set of interindividual, interdomain paths.

The spread of problems from age 13 to age 14 and from age 14 to age 15: Intraindividual processes. The paths for intraindividual influence indicated that problems spread within twins across behavioral domains. There were statistically significant paths from one twin's delinquency to his or her subsequent substance use (f_1 and f_2) and from one twin's substance use to his or her subsequent delinquency (e_2). With one exception, higher initial levels of one twin's problem behavior predicted greater increases in his or her own levels of the other problem behavior (e.g., Twin A delinquency → Twin A substance use and Twin A substance use → Twin A delinquency), from age 13 to 14 and from age 14 to 15. The path from substance use at age 13 to delinquency at age 14 (e_1) failed to reach conventional levels of statistical significance ($p = .15$). There were neither sex (e_1 and e_2; boys/girls 95% CI [−.07, .22]/[−.05, .23]; f_1 and f_2 boys/girls 95% CI [.16, .42]/[.09, .34]) nor zygosity (e_1 and e_2 MZ/DZ 95% CI [−.07, .15]/[−.03, .32]; f_1 and f_2 MZ/DZ 95% CI [.08, .30]/[.18, .40]) differences in either set of intraindividual, interdomain paths.

The spread of problem behaviors from age 13 to age 15: Interindividual processes, followed by intraindividual processes. Indirect effects tested the hypothesis that one twin's age 13 delinquency influenced the other twin's age 14 delinquency, which, in turn, influenced his or her own age 15 substance use. A Sobel test of the indirect effect was statistically significant, $z = 3.18$, $p = .001$. One twin's delinquency was linked to increases the other twin's substance use via interindividual changes in delinquency (e.g., Twin A age 13 delinquency → Twin B age 14 delinquency → Twin B age 15 substance use).

Indirect effects tested the hypothesis that one twin's age 13 substance use influenced the other twin's age 14 substance use, which, in turn influenced his or her own age 15 delinquency. A Sobel test of the indirect effect was statistically significant, $z = 2.71$, $p = .007$. One twin's substance use was linked to increases in the other twin's delinquency via interindividual changes in substance use (e.g., Twin A age 13 substance use → Twin B age 14 substance use → Twin B age 15 delinquency).

The spread of problem behaviors from age 13 to age 15: Intraindividual processes, followed by interindividual processes. Indirect effects tested the hypothesis that one twin's age 13 delinquency influenced his or her own age 14 substance use, which, in turn, influenced the other twin's age 15 substance use. A Sobel test of the indirect effect was statistically significant, $z = 3.59$, $p < .001$. One twin's delinquency was linked to increases in the other twin's substance use via intraindividual changes in substance use (e.g., Twin A age 13 delinquency → Twin A age 14 substance use → Twin B age 15 substance use). Indirect effects tested the hypothesis that one twin's age 13 substance use influenced his or her own age 14 delinquency, which in turn influenced the other twin's age 15 delinquency. A Sobel test of the indirect effect was not statistically significant, $z = 1.31$, $p = .19$.

Comparing the indirect spread of problem behaviors. Indirect effects were contrasted to determine whether interindividual processes from delinquency to substance use were stronger than the interindividual processes from substance use to delinquency. In both cases, the indirect effects from delinquency to substance use were stronger than the indirect effects from substance use to delinquency, Wald's $\chi^2(1) = 8.07–11.63$, $p < .05$. Problems are more likely to spread from delinquency to substance use than from substance use to delinquency.

Discussion

Our study was designed to examine how problem behaviors spread between adolescent twin siblings. Two competing models were contrasted: (a) a single step, direct transmission model and (b) a two-step, indirect transmission model. As expected, there was evidence that problems spread between twins. However, problems did not spread directly between twins across domains of miscon-

duct. Instead, problems spread indirectly via two two-step processes: (a) interindividually (between twins) within a behavioral domain, then intraindividually (within twins) across behavioral domains (e.g., Twin A delinquency → Twin B delinquency → Twin B substance use) or (b) intraindividually (within twins) across behavioral domains, then interindividually (between twins) within a behavioral domain (e.g., Twin A delinquency → Twin A substance use → Twin B substance use).

Independent of the processes involved, delinquency was more apt to predict later substance use than substance use was to predict later delinquency. Delinquency provides a gateway to substance use, primarily through selection and affiliation with delinquent peers who offer access to illicit substances and who endorse, model, and reward their consumption (Mason et al., 2007). Less is known about the mechanisms whereby substance use begets delinquency. One possibility is that substance use produces disinhibition and cognitive distortions, which can set the stage for antisocial acts (White, Tice, Loeber, & Stouthamer-Loeber, 2002). Whatever the mechanism, evidence from the present study suggests that the contribution of substance use to later delinquency is relatively modest when compared with the reverse.

Like others (Buist, 2010; D'Amico et al., 2008; Low et al., 2012), we found no evidence that problems spread directly between twins across domains. In theory, one sibling could expose the other to new models for the consumption of illicit substances and new sources of peer pressure to engage in delinquent acts, but in practice the peer networks of twins are fairly redundant (Samek, McGue, Keyes, & Iacono, 2015), so opportunities for meeting and befriending new peers with bad habits are quite limited. The same reasoning may explain why most studies do not find evidence for the interdomain spread of problems between friends (e.g., Brechwald & Prinstein, 2011). Siblings who differ in age have different peer networks, yet they too tend not to evince a direct spread of problems across domains (e.g., Low et al., 2012). Of course, null findings must be interpreted with caution, but the preponderance of evidence seems to argue against the proposition that deviance training is a global process that motivates generic misconduct. When problems spread from one twin to another, they spread within the same behavioral domain, suggesting that modeling and reinforcement are specifically targeted influence processes whose effects neither generalize to nor encourage different forms of misbehavior.

We are not the first to note that twin siblings may be important sources of influence over substance use and delinquency. Our findings are consistent with results indicating that sibling resemblance in problem behaviors cannot be entirely explained by genetics or shared environments (Fagan & Najman, 2003). In the present study, sibling effects emerged in APIM analyses, over and above the modest to moderate genetic and shared environmental contributions to substance use and delinquency that were identified in the quasi-ACE model. Resemblances on misconduct are tied to the sibling relationship itself; others have found that siblings with better quality relationships tend to be the most similar (Rowe & Gulley, 1992). Although parents are often the target of interventions, practitioners would be well advised to focus their efforts on siblings, who are more influential than parents with regard to substance use and delinquency (Fagan & Najman, 2005) and whose influence rivals that of friends (e.g., Scholte, Poelen, Willemsen, Boomsma, & Engels, 2008). By distinguishing between intra- and interdomains of transmission, our longitudinal findings offer important new insights into the focus of interventions with siblings.

Why does it matter that problems spread indirectly between twins across domains and not directly? The distinction is important because indirect and direct models are predicated on different contagion mechanisms, which have different implications for intervention. Direct effects models assume that misconduct transforms as it is transmitted between siblings. One form of misbehavior by one sibling gives rise to a different form of misbehavior in the other sibling. Short of quarantining, it is difficult to imagine an intervention that would protect against effects that snowballed across domains between siblings. In contrast, indirect transmission assumes that siblings adopt the same form of misconduct, which means that interventions can be targeted at a specific behavior. It follows that interventions designed to arrest the development of problem drinking are more apt to be successful than interventions designed to inoculate against all manner of deviance. Results from the present study strongly suggest indirect sibling transmission of behavior problems; problems spread between siblings within domains, which implies that problems can be arrested with interventions that disrupt the behavior not the relationship. The analyses accounted for genetic and shared environmental influences that make the two twins similar to each other, suggesting that factors outside of the home that are specific to each twin may be a source of these behaviors. Friend influence is rarely apportioned equally. Behavior can be encouraged or discouraged depending largely on the characteristics of the more influential friend (Laursen, Hafen, Kerr, & Stattin, 2012). It follows that if nonshared friend experiences can induce one twin to adopt a behavior that then spreads to the other twin, then nonshared friend experiences can also influence problem behavior desistence or resistance.

We did not find sex differences in the strength of interpersonal intradomain influence. Boys may present higher levels of substance use and antisocial behavior than girls, but our findings confirm that sex differences in mean levels of behavior are neither a product of sex differences in genetic effects nor sex differences in sibling influence (e.g., Carey, 1992). Put simply, siblings may be responsible for some of the acquisition of problem behaviors during adolescence, but siblings are not responsible for the fact that boys acquire more of these problems than girls. Here, as above, experiences with friends may be an important avenue for intervention.

There was a substantial genetic component to substance use and delinquency, raising the prospect that previous studies examining the spread of behavior problems may have overestimated the influence that one sibling exerts over another. The present study is unique in that it is one of the first to apply an APIM design to an MZ/same-sex DZ twin sample, thus removing the possibility that effects are a product of emerging biological predispositions linked to substance use and delinquency. Effects did not differ for MZ and same-sex DZ twins, underscoring the environmental nature of sibling influence. The study design also precludes shared environmental effects that reflect common sibling environments (Carey, 1992), ruling out the possibility that the intradomain spread of problem behaviors between twins is a product of joint exposure to shared household risks (e.g., parents who model and provide access to illicit substances).

The present study is also unique in that it is the first to examine mediated effects with a full longitudinal design in an APIM framework. The three-wave longitudinal design is a necessary precondition for causal conclusions about the degree to which an independent variable predicts changes in an intervening variable, which in turn, predicts changes in a dependent variable (Fritz & MacKinnon, 2012). We use the term *causal* advisedly. Our data are quasi-experimental (Lahey & D'Onofrio, 2010), so we are limited to conclusions about temporal predictive causality of sibling effects. The autoregressive design measures change and lends itself to conclusions about variables that predict change. The analyses were designed for dyads, so as to remove bias arising from nonindependent data, which can inflate estimates of influence (Kenny, 1995). Controls for friend substance use and delinquency help to isolate the contributions of siblings by removing concurrent peer similarity and selection effects. Some (but not all) concurrent variance linked to parenting was also removed, helping to eliminate the possibility that the effects were driven by siblings from atypical households. In conclusion, few studies of sibling influence can claim the methodological rigor of this study.

Nevertheless, our study is not without limitations. We could not identify all possible environmental factors potentially correlated with misconduct (e.g., peer status, romantic partner behavior problems, differential exposure to parent substance use or criminality) that may be driving sibling associations. Our analyses assume that dyads were indistinguishable, but uniform influence is unlikely. Individual characteristics linked to elevated interpersonal influence have been identified in friend dyads, such as relative peer acceptance (Laursen et al., 2012) and relative relationship satisfaction (Hiatt, Laursen, Stattin, & Kerr, 2015). Such analyses may help to improve the generalizability of the findings to friends and nontwin siblings, where greater differences in partner characteristics increase the likelihood that behavior problems are primarily spread from the more influential partner to the more susceptible partner. We note our exclusive reliance on self-reports. Survey data cannot do justice to complex family interaction processes; observational data are essential to elaborating the transmission processes identified herein. Our study assesses the spread of problem behaviors during the midadolescent years. Rates of delinquency and substance abuse peak in late adolescence (Elliott, Huizinga, & Menard, 2012), raising the possibility that transmission mechanisms may operate differently when baseline rates of behavior are higher. Our findings indicate sibling influence in the interpersonal intradomain spread of problems. Caution is warranted in interpreting findings concerning the intrapersonal interdomain spread of behavior problems, because the absence of MZ and same-sex DZ differences does not preclude an underlying genetic vulnerability to an expansion of externalizing problems. Further, our model does not fully account for gene–environment correlations, although controlling for friend reports of behavior problems does address the concerns that the genetically influenced friend selection effects are responsible for the spread of behavior problems (Cleveland et al., 2005). Finally, some may question the degree to which findings from twins generalize to other sibling relationships. Twin and nontwin siblings have both been found to be influential figures whose influence rivals that of friends, but we would be remiss if we did not note that twins tend to exert greater influence over one another than nontwin siblings (Fraley & Tancredy, 2012).

Accumulating evidence suggests that problems do not spread across domains between partners in friend and sibling relationships. And yet problems seem to snowball over time between partners within relationships. What accounts for this paradox? Findings from the present study point to partner influence as a risk factor for illicit substance use, both because substance use by one twin predicts substance use by the other twin, but also because delinquency in one twin predicts delinquency in the other twin, which then gives rise to escalating substance use. The indirect transition of behavior problems is critical during the early adolescent years, a period when most youth are initially exposed to illicit substances and when parent supervision tends to decline. There is no reason to assume that the indirect spread of problems is a phenomenon unique to twins. The interpersonal dynamics of adolescent twins resemble those of adolescent friends (Scholte et al., 2008) and adolescent siblings (Fraley & Tancredy, 2012), especially those who are emotionally close, near to one another in age, and the same sex. Unpackaging these indirect influence mechanisms may well offer important insight into why delinquency exacerbates the growth of substance misuse during the first half of the second decade of life.

References

Armstrong, T. D., & Costello, E. J. (2002). Community studies on adolescent substance use, abuse, or dependence and psychiatric comorbidity. *Journal of Consulting and Clinical Psychology, 70,* 1224–1239. http://dx.doi.org/10.1037/0022-006X.70.6.1224

Boivin, M., Brendgen, M., Dionne, G., Dubois, L., Pérusse, D., Robaey, P., . . . Vitaro, F. (2013). The Quebec Newborn Twin Study into adolescence: 15 years later. *Twin Research and Human Genetics, 16,* 64–69. http://dx.doi.org/10.1017/thg.2012.129

Boivin, M., Pérusse, D., Dionne, G., Saysset, V., Zoccolillo, M., Tarabulsy, G. M., . . . Tremblay, R. E. (2005). The genetic-environmental etiology of parents' perceptions and self-assessed behaviours toward their 5-month-old infants in a large twin and singleton sample. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 46,* 612–630. http://dx.doi.org/10.1111/j.1469-7610.2004.00375.x

Brechwald, W. A., & Prinstein, M. J. (2011). Beyond homophily: A decade of advances in understanding peer influence processes. *Journal of Research on Adolescence, 21,* 166–179. http://dx.doi.org/10.1111/j.1532-7795.2010.00721.x

Buist, K. L. (2010). Sibling relationship quality and adolescent delinquency: A latent growth curve approach. *Journal of Family Psychology, 24,* 400–410. http://dx.doi.org/10.1037/a0020351

Carey, G. (1992). Twin imitation for antisocial behavior: Implications for genetic and family environment research. *Journal of Abnormal Psychology, 101,* 18–25. http://dx.doi.org/10.1037/0021-843X.101.1.18

Cleveland, H. H., Wiebe, R. P., & Rowe, D. C. (2005). Sources of exposure to smoking and drinking friends among adolescents: A behavioral-genetic evaluation. *The Journal of Genetic Psychology, 166,* 153–169.

D'Amico, E. J., Edelen, M. O., Miles, J. N., & Morral, A. R. (2008). The longitudinal association between substance use and delinquency among high-risk youth. *Drug and Alcohol Dependence, 93,* 85–92. http://dx.doi.org/10.1016/j.drugalcdep.2007.09.006

Dishion, T. J., & Owen, L. D. (2002). A longitudinal analysis of friendships and substance use: Bidirectional influence from adolescence to adulthood. *Developmental Psychology, 38,* 480–491. http://dx.doi.org/10.1037/0012-1649.38.4.480

Elliott, D. S., Huizinga, D., & Menard, S. (2012). *Multiple problem youth: Delinquency, substance use, and mental health problems*. New York, NY: Springer.

Fagan, A. A., & Najman, J. M. (2003). Sibling influences on adolescent delinquent behaviour: An Australian longitudinal study. *Journal of Adolescence, 26,* 547–559. http://dx.doi.org/10.1016/S0140-1971(03)00055-1

Fagan, A. A., & Najman, J. M. (2005). The relative contributions of parental and sibling substance use to adolescent tobacco, alcohol, and other drug use. *Journal of Drug Issues, 35,* 869–883. http://dx.doi.org/10.1177/002204260503500410

Falconer, D. S. (1989). *Introduction to quantitative genetics*. Essex, United Kingdom: Longman.

Ferguson, C. J., & Meehan, D. C. (2011). With friends like these . . .: Peer delinquency influences across age cohorts on smoking, alcohol and illegal substance use. *European Psychiatry, 26,* 6–12. http://dx.doi.org/10.1016/j.eurpsy.2010.09.002

Forget-Dubois, N., Pérusse, D., Turecki, G., Girard, A., Billette, J.-M., Rouleau, G., . . . Tremblay, R. E. (2003). Diagnosing zygosity in infant twins: Physical similarity, genotyping, and chorionicity. *Twin Research, 6,* 479–485. http://dx.doi.org/10.1375/136905203322686464

Fraley, R. C., & Tancredy, C. M. (2012). Twin and sibling attachment in a nationally representative sample. *Personality and Social Psychology Bulletin, 38,* 308–316. http://dx.doi.org/10.1177/0146167211432936

Fritz, M. S., & MacKinnon, D. P. (2012). Mediation models for developmental data. In B. Laursen, T. D. Little, & N. A. Card (Eds.), *Handbook of developmental research methods* (pp. 291–310). New York, NY: Guilford Press.

Henly, G. A., & Winters, K. C. (1989). Development of psychosocial scales for the assessment of adolescents involved with alcohol and drugs. *Substance Use & Misuse, 24,* 973–1001. http://dx.doi.org/10.3109/10826088909047324

Hiatt, C., Laursen, B., Stattin, H., & Kerr, M. (2015). Best friend influence over adolescent problem behaviors: Socialized by the satisfied. *Journal of Clinical Child and Adolescent Psychology*. Advance online publication. http://dx.doi.org/10.1080/15374416.2015.1050723

Kaplan, H. B., Martin, S. S., & Robbins, C. (1984). Pathways to adolescent drug use: Self-derogation, peer influence, weakening of social controls, and early substance use. *Journal of Health and Social Behavior, 25,* 270–289. http://dx.doi.org/10.2307/2136425

Kenny, D. A. (1995). The effect of non-independence on significance testing in dyadic research. *Personal Relationships, 2,* 67–75. http://dx.doi.org/10.1111/j.1475-6811.1995.tb00078.x

Kenny, D. A., Kashy, D. A., & Cook, W. L. (2006). *Dyadic data analysis*. New York, NY: Cambridge University Press.

Lahey, B. B., & D'Onofrio, B. M. (2010). All in the family: Comparing siblings to test causal hypotheses regarding environmental influences on behavior. *Current Directions in Psychological Science, 19,* 319–323. http://dx.doi.org/10.1177/0963721410383977

Laursen, B., Hafen, C. A., Kerr, M., & Stattin, H. (2012). Friend influence over adolescent problem behaviors as a function of relative peer acceptance: To be liked is to be emulated. *Journal of Abnormal Psychology, 121,* 88–94. http://dx.doi.org/10.1037/a0024707

Laursen, B., Popp, D., Burk, W. J., Kerr, M., & Stattin, H. (2008). Incorporating interdependence into developmental research: Examples from the study of homophily and homogeneity. In N. A. Card, J. P. Selig, & T. D. Little (Eds.), *Dyadic and interdependent data in developmental research* (pp. 11–37). Mahwah, NJ: Erlbaum.

LeBlanc, M., & Fréchette, M. (1989). *Male criminal activity from childhood through youth: Multilevel and developmental perspective*. New York, NY: Springer-Verlag. http://dx.doi.org/10.1007/978-1-4612-3570-5

Low, S., Shortt, J. W., & Snyder, J. (2012). Sibling influences on adolescent substance use: The role of modeling, collusion, and conflict. *Development and Psychopathology, 24,* 287–300. http://dx.doi.org/10.1017/S0954579411000836

Mason, W. A., Hitchings, J. E., McMahon, R. J., & Spoth, R. L. (2007). A test of three alternative hypotheses regarding the effects of early delinquency on adolescent psychosocial functioning and substance involvement. *Journal of Abnormal Child Psychology, 35,* 831–843. http://dx.doi.org/10.1007/s10802-007-9130-7

Mason, W. A., & Windle, M. (2002). Reciprocal relations between adolescent substance use and delinquency: A longitudinal latent variable analysis. *Journal of Abnormal Psychology, 111,* 63–76. http://dx.doi.org/10.1037/0021-843X.111.1.63

Muthén, L. K., & Muthén, B. O. (1998–2014). *MPlus user's guide* (7th ed.). Los Angeles, CA: Author.

Neale, M. C., & Maes, H. H. M. (2004). *Methodology for genetic studies of twins and families*. Dordrecht, the Netherlands: Kluwer.

Olsen, J. A., & Kenny, D. A. (2006). Structural equation modeling with interchangeable dyads. *Psychological Methods, 11,* 127–141. http://dx.doi.org/10.1037/1082-989X.11.2.127

Petraitis, J., Flay, B. R., & Miller, T. Q. (1995). Reviewing theories of adolescent substance use: Organizing pieces in the puzzle. *Psychological Bulletin, 117,* 67–86. http://dx.doi.org/10.1037/0033-2909.117.1.67

Popp, D., Laursen, B., Kerr, M., Stattin, H., & Burk, W. K. (2008). Modeling homophily over time with an actor-partner interdependence model. *Developmental Psychology, 44,* 1028–1039. http://dx.doi.org/10.1037/0012-1649.44.4.1028

Rende, R., Slomkowski, C., Lloyd-Richardson, E., & Niaura, R. (2005). Sibling effects on substance use in adolescence: Social contagion and genetic relatedness. *Journal of Family Psychology, 19,* 611–618. http://dx.doi.org/10.1037/0893-3200.19.4.611

Rowe, D. C., & Gulley, B. L. (1992). Sibling effects on substance use and delinquency. *Criminology, 30,* 217–234. http://dx.doi.org/10.1111/j.1745-9125.1992.tb01103.x

Samek, D. R., McGue, M., Keyes, M., & Iacono, W. G. (2015). Sibling facilitation mediates the association between older and younger sibling alcohol use in late adolescence. *Journal of Research on Adolescence, 25,* 638–651. http://dx.doi.org/10.1111/jora.12154

Santé Québec,Jetté, M., Desrosiers, H., & Tremblay, R. E. (1998). *"In 2001 . . . I'll be 5 years old!" Survey of 5-month old infants: Preliminary report of the Longitudinal Study of Child Development in Québec*. Québec, Canada: Bibliothèque Nationale du Québec.

Scholte, R. H., Poelen, E. A., Willemsen, G., Boomsma, D. I., & Engels, R. C. (2008). Relative risks of adolescent and young adult alcohol use: The role of drinking fathers, mothers, siblings, and friends. *Addictive Behaviors, 33,* 1–14. http://dx.doi.org/10.1016/j.addbeh.2007.04.015

Slutske, W. S., D'Onofrio, B. M., Turkheimer, E., Emery, R. E., Harden, K. P., Heath, A. C., & Martin, N. G. (2008). Searching for an environmental effect of parental alcoholism on offspring alcohol use disorder: A genetically informed study of children of alcoholics. *Journal of Abnormal Psychology, 117,* 534–551. http://dx.doi.org/10.1037/a0012907

Stormshak, E. A., Comeau, C. A., & Shepard, S. A. (2004). The relative contribution of sibling deviance and peer deviance in the prediction of substance use across middle childhood. *Journal of Abnormal Child Psychology, 32,* 635–649. http://dx.doi.org/10.1023/B:JACP.0000047212.49463.c7

Strayhorn, J. M., & Weidman, C. S. (1988). A Parent Practices Scale and its relation to parent and child mental health. *Journal of the American Academy of Child and Adolescent Psychiatry, 27,* 613–618. http://dx.doi.org/10.1097/00004583-198809000-00016

Trim, R. S., Leuthe, E., & Chassin, L. (2006). Sibling influence on alcohol use in a young adult, high-risk sample. *Journal of Studies on Alcohol, 67,* 391–398. http://dx.doi.org/10.15288/jsa.2006.67.391

Turkheimer, E., & Harden, K. P. (2014). Behavior genetic research methods. In H. T. Reis & C. M. Judd (Eds.), *Handbook of research methods in social and personality psychology* (pp. 159–187). New York, NY: Cambridge University Press.

White, H. R., Tice, P. C., Loeber, R., & Stouthamer-Loeber, M. (2002). Illegal acts committed by adolescents under the influence of alcohol and drugs. *Journal of Research in Crime and Delinquency, 39,* 131–152. http://dx.doi.org/10.1177/002242780203900201

Winters, K. C., Stinchfield, R. D., Henly, G. A., & Schwartz, R. H. (1990). Validity of adolescent self-report of alcohol and other drug involvement. *Substance Use & Misuse, 25,* 1379–1395. http://dx.doi.org/10.3109/10826089009068469

Appendix

Questionnaire Items

Delinquency

1. Did you use hidden notes to cheat during an exam?
2. Have you skipped school, without telling your parents?
3. Have you ever entered a place without paying (when you were supposed to pay)?
4. Have you ever stolen something from a store?
5. Have you taken objects worth $10 or more that do not belong to you?
6. Have you set fire to a store or another place?
7. Have you smashed a door/window to break in and take something?
8. Have you threatened, bullied others to get what you wanted?
9. Have you lied to your parents/teacher to get what you wanted?

Substance Use

1. During the past 12 months, how often did the child drink alcohol?
2. During the past 12 months, how often did you use cannabis (marijuana, pot, hashish)?
3. During the past 12 months, how often did you use cocaine (coke, snow, crack, freebase, powder), glue or paint remover, hallucinogens (LSD, PCP, mescaline, mushrooms, acid, ecstasy), heroin, amphetamines, or other hard drugs without prescriptions?
4. During the past 12 months, how often did you have five or more alcoholic drinks?

Inconsistent Parenting

1. During the past 12 months, how often did you not pay attention to what the child was doing/did nothing?
2. During the past 12 months, how often did you let something pass that should have been punished?
3. During the past 12 months, how often did the child manage to avoid punishment?

Punishment

1. During the past 12 months, how often did you firmly grab/shake this child when they were being difficult?
2. During the past 12 months, how often did you hit the child when they were being difficult?
3. During the past 12 months, how often did you inflict corporeal punishment on the child?
4. During the past 12 months, how often did you raise your voice, scold, or yell at the child?

Conflict

1. Over the past 12 months, another person intervened to settle a dispute.
2. Over the past 12 months, I have withdrawn/given up to end a dispute when in disagreement.
3. Over the past 12 months, we make up easily when we have had a quarrel.
4. Over the past 12 months, I refused to talk to the child when we were in disagreement.
5. Over the past 12 months, I remained very angry for a long time when we have had a dispute.

(*Appendix continues*)

6. Over the past 12 months, we annoyed each other/got on each other's nerves.
7. Over the past 12 months, we have been in disagreement/we have quarreled.
8. Over the past 12 months, we have solved the problem when in disagreement.
9. Over the past 12 months, we shouted at each other.
10. Over the past 12 months, when we were in disagreement, the angry child leaves the house or yard.

Positive Interaction

1. During the past 12 months, how often did you do a special activity with the child?
2. During the past 12 months, how often did you partake in sports activities/hobbies/games with the child?
3. During the past 12 months, how often did you talk and play with this child?
4. During the past 12 months, how often did you tell the child that you were proud or pleased with him/her?
5. During the past 12 months, how often did you play fight with him/her just for fun?

Parental Efficacy

1. I am very good at reassuring/giving security to the upset child.
2. I feel very good at communicating my expectations.
3. I feel very good at disciplining the child.
4. I feel very good at supervising school activities.
5. I feel very good at talking about what is important with the child.
6. I have little effect on the academic success of the child.
7. I have little effect on the development of the personality of the child.
8. I have little effect on the intellectual development of this child.
9. I have little effect on how he will behave later.
10. No matter what, this child will develop in his or her own way.

Received January 28, 2016
Revision received August 25, 2016
Accepted August 29, 2016 ■