**Peers and Delinquency: A Genetically-informed, Developmentally-Sensitive Perspective**

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**Abstract**

Deviant peer affiliation (DPA) has been shown to be one of the strongest predictors of delinquent behavior in children and adolescents. However, the role of DPA may differ depending on the type of delinquent behavior considered (i.e., overt vs. covert delinquency) and participants’ developmental stage (i.e., childhood vs. adolescence). The role of DPA may also vary depending on individuals’ genetic disposition for delinquent behavior. This chapter reviews the literature from quantitative (i.e., behavioral) genetic studies and from molecular genetic studies (1) to determine whether DPA is genetically influenced and (2) to examine its contribution to overt or covert delinquency according to a Selection, a Socialization or an Interactional perspective. The conclusion raises a number of methodological and conceptual issues that need to be addressed in future research.

**Keywords:** Deviant peers, delinquency, antisociality, longitudinal research, quantitative genetics, molecular genetics, children, adolescents, development

In addition to the family, peers play a major role in youngsters’ development. They provide a crucial context for the acquisition of new social skills, the validation of the self-concept, and the learning of social roles and norms ([Vitaro, Boivin, & Bukowski, 2009](#_ENREF_95)). Not all peer relationships are positive, however, and they can sometimes be a significant source of concern for caregivers such as parents and educators. This is notably the case when youngsters affiliate with deviant peers. Deviant peer affiliation (DPA) has sometimes been referred to as the proportion of friends or peers involved in disruptive or delinquent activities such as aggression and violence, theft or substance use. Most often, however, DPA refers to friends’ or close peers’ involvement in disruptive or delinquent activities, as assessed by the participants themselves, the friends/close peers, or a third source (e.g., parents). Apart from youngsters’ own early disruptive behaviors such as aggressiveness and rule breaking, DPA has been shown to be one of the strongest predictors of delinquent behavior in children and adolescents ([Boivin, Vitaro, & Poulin, 2005](#_ENREF_14); [Dishion & Patterson, 2006](#_ENREF_38); [Lacourse, Nagin, Tremblay, Vitaro, & Claes, 2003](#_ENREF_64)).

Three explanations for the association between DPA and children’s or adolescents’ delinquent behavior have been debated for the past three decades ([see Vitaro, Tremblay, & Bukowski, 2001 for a detailed overview](#_ENREF_100)). According to one perspective, the (predictive) association between DPA and delinquent behavior does not necessarily indicate a causal influence of one on the other, but is instead explained by one or more other underlying factors. That is, the same (genetic or environmental) factors that lead to a child’s or an adolescent’s delinquent behavior also contribute to DPA ([Gottfredson & Hirschi, 1990](#_ENREF_48)). This viewpoint is compatible with a *Selection model* whereby disruptive children or delinquent adolescents affiliate with each other by virtue of the similarities in their behavioral dispositions ([Beaver, Ratchford, & Ferguson, 2009](#_ENREF_6); [Kendler, Schmitt, Aggen, & Prescott, 2008](#_ENREF_57); [Lacourse et al., 2006](#_ENREF_65)). An alternative perspective proposes that DPA truly contributes to the development of delinquent behavior in youth even when possible selection processes and other risk factors are controlled. This explanation is compatible with a *Social influence* (i.e., socialization) model ([Elliott, Huizinga, & Ageton, 1985](#_ENREF_44); [Thornberry, Krohn, Lizotte, & Chard-Wierschem, 1993](#_ENREF_89)). Finally, the *Social interactional* perspective (also referred to as the Social enhancement model) views DPA not so much as an independent contributor to delinquent behavior, but rather as influencing delinquent behavior in interaction with personal characteristics ([Lacourse et al., 2003](#_ENREF_64); [Vitaro, Tremblay, Kerr, Pagani, & Bukowski, 1997](#_ENREF_101)). According to this perspective, deviant friends are not necessary for disruptive children to become delinquent but early disruptiveness is even more likely to develop into delinquency later on for those who do affiliate with delinquent peers.

The goal of this chapter is to contribute to the ongoing debate between the three models from a perspective that is both genetically-informed and developmentally-sensitive ([for a general discussion about the benefits of a genetically sensitive design, see Bates & Lewis, 2012](#_ENREF_3); [as for the benefits of a developmental perspective, see LeBlanc & Loeber, 1998](#_ENREF_68)). Whenever possible, a distinction is made between *overt* (i.e., personal violence, aggression) and *covert* (i.e., vandalism or destruction of property, theft, lying, cheating, rule breaking behavior) delinquent behavior. The two types of delinquent behavior are partly independent from each other and they are associated with partly different risk factors ([Barker et al., 2011](#_ENREF_2); [Burt, 2009](#_ENREF_21); [Lacourse et al., 2010](#_ENREF_61)). The relative role of genetic and environmental influences is also different for the two types of delinquent behavior. Overt delinquent behavior is highly heritable and the genetic factors contributing to its development are first expressed during early-to-middle childhood ([Lacourse et al., sous presse](#_ENREF_62); [Niv, Tuvblad, Raine, & Baker, 2013](#_ENREF_75); [van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003](#_ENREF_92)). In contrast, environmental influences are more salient on covert delinquent behavior, despite a steady increase of genetic influences from childhood to adolescence ([Burt & Klump, 2009](#_ENREF_22)). In consequence, some of the processes that underlie the role of DPA and its interplay with genetic or environmental influences on delinquent behavior may vary depending on the type of delinquent behavior considered (i.e., overt vs. covert) or the developmental period under investigation (i.e., childhood vs. adolescence).

**The Usefulness of a Genetically-Informed Perspective**

The vast majority of empirical evidence for genetic effects on delinquent behavior comes from *quantitative genetic studies* (which are also often termed behavioral genetic studies) (see Beaver, Schwartz, & Gajos, this volume). In contrast to *molecular genetic* *studies*, which attempt to identify specific genes related to a phenotype such as delinquent behavior, quantitative genetic studies do not explicitly measure specific genes and many do not even include any specific measures of environmental influence. Instead, quantitative genetic studies statistically infer the relative strength of genetic and environmental influences on a phenotype by examining the similarity of family members with varying degrees of genetic relatedness in regard to that behavior in a specific population. This can be accomplished using a variety of research designs, such as through the comparison of adopted and biological siblings or the comparison of identical (i.e., monozygotic, MZ) and fraternal (i.e., dizygotic, DZ) twin pairs. In the case of the classical twin design, the genetic and environmental variance associated with a given phenotype (i.e., overt or covert delinquency; DPA) is decomposed by comparing the within pair similarity of MZ twins, who share 100% of their genes and who are raised together, to the within pair similarity of same-sex DZ twins, who are also raised together but only share on average 50% of their genes. The underlying assumption of all quantitative genetic designs is that inter-individual differences in a phenotype can be decomposed into three different sources of variance: *additive (or non additive) genetic factors*, *shared environmental factors*, and *nonshared environmental factors*. In the classical twin design, *genetic* influences are indicated when the phenotypic similarity (i.e., correlation) of MZ twin pairs is greater than the phenotypic similarity of DZ twin pairs. Shared environment refers to environmental factors ― both inside and outside of the family ― that siblings are jointly exposed to and that make them similar to each other (e.g., neighborhood crime level, family SES, parental mental health problems). *Shared environmental* influences are indicated when MZ pairs as well as DZ twin pairs are similar to each other and, in addition, the degree of similarity among DZ twins is comparable to that of MZ twins. Nonshared environmental factors refer to experiences within the family or outside the family that make siblings different from each other. *Nonshared environmental* influences are indicated by the extent to which even MZ twins, who are genetically identical and are raised in the same family, are different from each other (i.e., the degree to which the MZ correlation is less than 1). Nonshared environmental experiences can come from within the family, such as differential treatment by parents ([Conger & Conger, 1994](#_ENREF_31); [Dunn, Stocker, & Plomin, 1990](#_ENREF_43); [McHale, Crouter, McGuire, & Updegraff, 1995](#_ENREF_72)). However, the most important nonshared environmental influences are likely those experienced outside the family ([Dunn & Plomin, 1990](#_ENREF_42)). Such outside-of-the-family experiences, of course, refer to peers and friends as many children, including MZ twins, do not affiliate with the same friends as their sibling ([Pike & Atzaba-Poria, 2003](#_ENREF_78); [Rose, 2002](#_ENREF_81); [Thorpe & Gardner, 2006](#_ENREF_90)). Hence, depending whether DPA is shared or not shared by the twins of the same pair, it could be part of the shared or non-shared environmental factors affecting delinquent behavior, in line with the Social InfluenceModel.

Findings from quantitative genetic studies suggest that between 40 and 60% of the variance of overt or covert delinquent behavior is influenced by genetic factors; the remaining variation is influenced by nonshared and, to a much lesser degree, by shared environmental factors ([Harris, 1995](#_ENREF_51); [Moffitt, 2005](#_ENREF_74); [Rhee & Waldman, 2002](#_ENREF_80); [Tuvblad & Baker, 2011](#_ENREF_91); [Viding, Larsson, & Jones, 2008](#_ENREF_94)). Although quantitative genetic designs are typically used to estimate heritable and environmental influences on traits or behaviors, such as delinquent behavior, this type of analysis can be extended to any measured variable, including aspects of the environment such as DPA. It thus becomes possible to test whether, in line with the Social Selection Model, the same underlying genetic disposition that leads to delinquent behavior also leads to DPA. Such a phenomenon, where environmental experiences are influenced by individuals’ genetic disposition for certain traits or behaviors, is called *gene–environment correlation*, or *rGE*

**Testing the Social Influence and the Selection Models through Gene-Environment Correlation: Evidence from Quantitative Genetic Studies**

Several quantitative genetic studies have found significant genetic influences on youngsters’ propensity to affiliate with delinquent or aggressive peers (Baker & Daniels, 1990Baker & Daniels, 1990; [Beaver, DeLisi, Wright, & Vaughn, 2009](#_ENREF_5); [Beaver, Shutt, et al., 2009](#_ENREF_7); [Button et al., 2007](#_ENREF_25); [Cleveland, Wiebe, & Rowe, 2005](#_ENREF_28); [Kendler, Jacobson, Myers, & Eaves, 2008](#_ENREF_55); [Manke, McGuire, Reiss, Hetherington, & Plomin, 1995](#_ENREF_71); [Rose, 2002](#_ENREF_81); [Rowe & Osgood, 1984](#_ENREF_82); [Tarantino et al., 2014](#_ENREF_88)). In these studies, between 20 and 40% of the variance in DPA is explained by genetic factors. Interestingly, all of the previously mentioned studies that found genetic effects on DPA used adolescent samples. In contrast, studies using samples of children or pre-adolescents often found no or very weak genetic effects and instead a moderate contribution of shared environmental factors and a large contribution of nonshared environmental factors. For example, a study of 12 year-old Finnish male twins ([Rose, 2002](#_ENREF_81)) showed that participants’ genetic make-up was not related to their friends’ externalizing – or, for that matter, internalizing - problems, and only weak and inconsistent genetic effects were found for girls. An even clearer lack of genetic effects on DPA for both boys and girls was found in a study of even younger identical and fraternal twins (mean age = 10 years) based on teacher-ratings and direct observational assessments of children’s and their close friends’ antisocial behaviors ([Bullock, Deater-Deckard, & Leve, 2006](#_ENREF_20)). And, finally, the same picture emerged in data drawn from the Quebec Newborn Twin Study (QNST), a longitudinal sample of identical and fraternal twins for whom friendship nominations were obtained over multiple time points. In kindergarten, teacher and peer ratings of physical aggression were obtained for each twin child and his or her nominated best friends in the classroom ([van Lier, Wanner, & Vitaro, 2007](#_ENREF_93)). As in other studies ([DiLalla, 2002](#_ENREF_35)), children’s own physical aggression was highly heritable. In contrast, their friends’ physical aggression was unrelated to children’s genetic make-up. A lack of genetic effect was also found in a follow-up study in grade 1, not only with respect to children’s affiliation with physically aggressive friends, but also with respect to their affiliation with socially aggressive friends ([Brendgen et al., 2008](#_ENREF_17)). Overall, existing empirical evidence from different studies thus suggests that, whereas friends’ characteristics are unrelated to individuals’ genetic disposition in younger children and pre-adolescents, rGE seems to emerge in early-to mid-adolescence and increase thereafter.

Some evidence for such an emerging rGE with respect to DPA comes from a retrospective study with a sample of 373 adult male twins. Specifically, Kendler and his colleagues ([Kendler et al., 2007](#_ENREF_56)) found that genetic effects on DPA (measured as the proportion of respondents’ friends who engaged in specific delinquent behaviors) increased substantially and steadily across five age periods: 8-11, 12-14, 15-17, 18-21, and 22-25. In contrast, the effects of shared environmental influences on DPA, while substantial at age 8-11, decreased over the first three age periods before increasing again moderately at ages 18-21 and 22-25. Using a prospective longitudinal design, Tarantino et al. (2014) also found that genetic influences steadily increase from age 15 to age 21.

**Overlap between the Genetic Factors Influencing DPA and Delinquent Behavior**

The finding of genetic influences on DPA suggests that youngsters’ deviant friendship choices are at least in part influenced by heritable characteristics, which, in turn, is consistent with the Selection Model. Even clearer support for the Selection Model comes from findings that a significant portion of these genetic influences on DPA comes from genetic factors related to delinquent behavior or similar behaviors such as substance use ([Boisvert, Boutwell, Vaske, & Newsome, 2013](#_ENREF_12); [Button et al., 2007](#_ENREF_25); [Harden, Hill, Turkheimer, & Emery, 2008](#_ENREF_50); [Rowe & Osgood, 1984](#_ENREF_82)). To illustrate, Boisvert et al. ([2013](#_ENREF_12)) used the twin and full-sibling subsample from the National Longitudinal Study of Adolescent Health (Add Health) to show that common genes explain more than 70% of the moderately high covariation between DPA and delinquency in adolescence, with the remaining covariation explained by common nonshared environmental factors operating on both variables. These results are similar to those reported by Button et al. (2007) showing that 86% of the correlation between DPA and antisocial behavior was due to common genetic factors, with common nonshared environmental factors explaining the rest. Again, however, these findings are typically based on adolescent samples. There is one study, however, that examined this issue over the course of three developmental periods (i.e., late childhood: ages 8-11; early adolescence: ages 12-14 years; middle-to-late adolescence: ages 15-18 years) ([Kendler, Schmitt, et al., 2008](#_ENREF_57)). This study used a retrospective design and is based on the same sample of 373 participants described earlier in reference to the Kendler et al. ([2007](#_ENREF_56)) study. The results showed that the same genetic factors that influenced delinquent behavior at each developmental period also influenced concurrent DPA as well as increases in DPA from one period to the next. The overall strength of these gene-environment correlations was considerably greater in mid-to late adolescence than in late childhood. In addition to common underlying genetic factors explaining both delinquent behavior and DPA, Kendler and his colleagues also found evidence of shared environmental factors that influenced both delinquent behavior and DPA. Since shared environmental experiences could include exposure to similar friends or the same friends at school or in the neighborhood, this result could reflect a direct effect of DPA. It could also reflect the impact of family processes such as a lack of monitoring or joint family activities, which in turn may lead to exposure to deviant friends. It is noteworthy that these shared environmental influences common to both delinquent behavior and DPA were observed only during late childhood and early adolescence, but not during middle-to-late adolescence. SEE ALSO TARANTINO ET AL. IN JACP

Together, these findings support the notion that rGE involving DPA is less likely (or at least less strong) during childhood than during adolescence. This developmental perspective is, in turn, concordant with the notion proposed by Dishion and colleagues ([Dishion, Patterson, & Griesler, 1994](#_ENREF_39)) that, over the course of childhood and into adolescence, youngsters progressively select –and are selected by– friends who share and positively reinforce their own values to the exclusion of others. If indeed DPA in adolescence results from rGE, then the association between DPA and delinquent behavior in adolescence may not reflect an environmental influence of DPA on delinquent behavior during that developmental period ([see also Jaffee & Price, 2007 for a similar suggestion](#_ENREF_53)). In contrast, the association between DPA and delinquent behavior in childhood may reflect a true environmental influence because the association between DPA and delinquent behavior is only partly, if at all, explained by common underlying genetic factors. In other words, the Socialization model may apply mostly to childhood whereas the Selection model may apply more strongly to adolescence.

It is important to note that these tentative conclusions rest on few studies, most of which may have significant methodological limitations. For example, Kendler, Schmitt et al. ([2008](#_ENREF_57)) used a retrospective life calendar method to collect their data. Such data are not entirely free from retrospective recall bias, particularly given the long time interval between the earliest recall period (age 8) and the actual time of data collection (age 40). In addition, in all studies both friends’ delinquent behavior and participants’ own delinquent behavior were rated by the same source, thus creating an additional bias towards inflated similarity ([Berndt & Keefe, 1995](#_ENREF_10); [Kandel, 1978](#_ENREF_54)). Not distinguishing between overt and covert delinquent behavior might also have affected the results since genetic influences on overt delinquent behavior are stronger and more developmentally constant than genetic influences on covert delinquent behavior. Notwithstanding these limitations, the previously mentioned studies cast some doubt on the notion that the association between DPA and delinquent behavior simply reflects an environmental influence of the former on the latter, at least not in adolescence when antisocial youth increasingly express their genetic dispositions and shape their own social world. A similar conclusion in favor of the Selection Model was made more than 30 years ago by Scarr and McCartney ([1983](#_ENREF_84)).

This conclusion is also supported by two prospective studies that explored the role of DPA in samples of adolescent twins while controlling for possible rGEs through the use of the MZ difference method. Since MZ twins share 100% of their genes (and the same family environment when raised together), the MZ difference method affords a unique opportunity to examine the role of nonshared environmental experiences that make the two twins of a pair different from each other, while controlling for genetic and shared environmental influence. This is achieved by correlating differences in the measured environment (e.g., DPA) with later differences in the measured behavior (e.g., delinquent behavior), while controlling for baseline differences in delinquent behavior and differences in other types of relevant environmental experiences ([see Vitaro, Brendgen, & Arseneault, 2009, for a full description of the method](#_ENREF_97)). As a consequence, the MZ difference method allows testing the premise of the Social Influence Model that DPA predicts delinquent behavior even when possible selection processes and other familial influences are controlled. The two studies that used the MZ difference method with adolescent samples found that within-pair differences in DPA (a general score of peers’ delinquent behavior) were unrelated to increased within-pair differences in (combined overt and covert) delinquent behavior, which stands in contrast to what would be expected according to the Socialization model ([Beaver, 2008](#_ENREF_4); [Burt, McGue, & Iacono, 2009](#_ENREF_24)). On the other hand, Vitaro, Brendgen et al. ([2011](#_ENREF_98)) found that within pair differences in friends’ overt externalized problems (i.e., physical aggression towards others) at age 6 years predicted an increase in within pair differences in twins’ overt externalized problems from age 6 to age 7 years, while controlling for possible confounders such as within-pair differences in peer rejection by normative peers and coercive parenting. Overall, the findings from studies that controlled possible rGE through the use of the MZ-difference method are thus similar to those from classical quantitative genetic studies that directly tested for rGE: In line with the Social Influence Model, DPA significantly contributes to the development of delinquent behavior in childhood, even when controlling for potential selection processes. This does not seem to be the case in adolescence, however, as DPA was unrelated to delinquent behavior once controlling for selection processes through rGE. It is important to note, however, that the three studies using the MZ-difference method may not be directly comparable because of their methodological differences, which may be confounded with developmental issues.

**Testing the Social Influence and Selection Models through Gene-Environment Correlation: Evidence from Molecular Genetic Studies**

Only a handful of molecular genetic studies have been published that examined potential rGE linking DPA with specific genes. These studies cover different age groups, different genes and different deviant behaviors, which makes it difficult to draw definite conclusions. Nevertheless, these studies help shed additional light on a possible rGE involving DPA and delinquent behavior. One of the first studies to examine a potential rGE between measured genes and DPA was published by Beaver, Wright, and Delisi ([2008](#_ENREF_8)). Using genotypic data (*N* = 1,816) from the Add Health Study, these authors examined whether a specific variant of the Dopamine transporter gene DAT1 – the 10-repeat allele (10R) – is associated with adolescents’ affiliation with substance-using peers. The dopamine transporter DAT1 is the primary mechanism for reuptake of released dopamine in the brain and individuals who carry the 10R allele of DAT1 have been found to show a lack of inhibitory control and to be more susceptible to dopamine-related disorders, notably disruptive behaviors ([Cornish et al., 2005](#_ENREF_32)). Adolescents were asked how many of their three closest friends smoke at least one cigarette per day, drink alcohol once a month, and smoke pot at least once a month. The results revealed that – for male adolescents from problematic family backgrounds – those with a greater number of the risk allele reported more affiliation with substance using friends (rGE = .13), despite controlling for own delinquent behavior, lack of self-control, and drug and alcohol use. No correlation was found for female adolescents or those from less problematic family backgrounds. Although these findings may indicate presence of selective rGE, the interpretation is somewhat hampered by the fact that friends’ behavior was based on adolescents’ perceptions instead of friends’ own reports, and that the measure of friends’ “deviancy” actually constitutes rather normative behavior compared to, for example, interpersonal violence or serious delinquency (i.e., using a weapon or arson).

Perhaps clearer evidence for selective rGE in regard to friendship affiliation comes from a study by Fowler, Settle, and Christakis ([2011](#_ENREF_45)). In this study, adult participants (mean age = 38 years, range = 21 to 70), were asked to nominate up to two close friends during seven repeated assessment waves over a 32 year period. They were also genotyped for six genetic markers. After Bonferroni correction and control for population stratification, the results did not show similarity between friends with regard to DAT1, but friends were significantly similar (r = .11) with respect to the DRD2 genotype. Frequency of the minor (A1) allele of DRD2 has been associated with antisocial behavior and with alcoholism ([Hill, Zezza, Wipprecht, Locke, & Neiswanger, 1999](#_ENREF_52); [Le Foll, Gallo, Le Strat, Lu, & Gorwood, 2009](#_ENREF_67)). By showing that friends resemble each other (albeit weakly) on a genotypic level, these results suggest that individuals (i.e., adults in this case) with a genetic predisposition for deviant behavior may actively seek out friends with similar traits.

The weak and inconsistent evidence regarding possible rGE involving specific genes and DPA is further challenged by three studies that found no association between DPA and genotypic variants on MAOA (implicated in the breakdown of synaptic neurotransmetters such as dopamine, norepinephrine, and serotonin and associated with antisocial behavior), CHRM2 (implicated in neurocognitive processes related to sensation seeking and dishinibition), and BDNF (implicated in the regulation of responses to stress) ([Kretschmer, Vitaro, & Barker, 2014](#_ENREF_59); [Latendresse et al., 2011](#_ENREF_66); [Lee, 2011](#_ENREF_69)). Since an interaction was found between DPA and genotype in all three studies, a more complete description of each study is presented later.

The inconsistency of the results from molecular genetic studies may cast doubt on the validity of the gene-environment correlations found in quantitative genetic studies. It is important to keep in mind, however, that the likelihood of finding significant associations between measured genes and another variable such delinquent behavior or DPA depends on the selection of appropriate candidate genes. In contrast to the few single-gene disorders such as cystic fibrosis or sickle cell anemia, a vast number of candidate genes are functionally relevant for complex social behaviors or traits – let alone for the environmental experiences that may be influenced by such traits. Any individual gene is thus likely to only have a very small effect and many samples may be underpowered to detect such small effect sizes. The effect of genes may also operate indirectly rather than directly on the environment, mediated by the outwardly expressed behavior or trait associated with a constellation of genes. Findings of rGE in support of the Social Selection Model – or lack thereof – derived from the findings from the few existing molecular studies are thus necessarily very preliminary and thus need to be interpreted with utmost caution.

**Testing the Social Interaction Model through Gene-Environment Interaction: Findings from Quantitative Genetic Studies**

According to the Social interactional model, DPA during adolescence may exert its influence on delinquent behavior not so much directly, but rather in an interactive fashion by facilitating the expression of a pre-existing personal disposition for delinquent behavior. Findings from non-genetically informed studies indeed show that DPA moderates (i.e., exacerbates) the effect of personal characteristics such as disruptiveness ([Lacourse et al., 2003](#_ENREF_64); [Vitaro, Brendgen, & Tremblay, 2000](#_ENREF_99)) or low self-regulation ([Gardner, Dishion, & Connell, 2008](#_ENREF_46)) on delinquent behavior. Because these behaviors are partly heritable ([Bornovalova, Hicks, Iacono, & McGue, 2010](#_ENREF_15)), such an interaction effect would be in line with a mechanism known as a gene environment interaction (GxE). While GxE may arise through different processes ([Brendgen, 2012](#_ENREF_16)), the Social Interactional model specifically suggests a Trigger or Enhancement process of GxE, which occurs when the presence of an environmental risk factor such as DPA triggers or exacerbates the expression of a genetic disposition for delinquent behavior. It is important to note that GxE and rGE processes can co-occur, such that the same environmental factor may simultaneously be involved in both GxE and rGE. Thus, failure to account for possible rGE may lead to biased estimates of GxE ([Purcell, 2002](#_ENREF_79)).

So far, all evidence from genetically-informed studies uniformly suggests that genetic influences on delinquent behavior (or on related behaviors such as substance use) are indeed amplified in adolescents who affiliate with deviant peers compared to those who do not. These findings are observed even when controlling for rGE ([Button et al., 2009](#_ENREF_26); [Dick et al., 2011](#_ENREF_34)). Importantly, this pattern is also confirmed when rigorous measures of peers’ deviancy are employed. A case in point is the aforementioned study by Harden et al. ([2008](#_ENREF_50)) with the Add Health data where peers’ tobacco and alcohol use was assessed through the peers’ self-reports (instead of through participants’ reports). These authors found that adolescents with a stronger genetic propensity for substance use (i.e., drinking and smoking) were more likely than others to have substance-using friends (reflecting rGE). Moreover, adolescents with a higher genetic liability drank and smoked even more if their friends did as well (reflecting GxE). Using data from the same sample, Guo, Elder, Cai, and Hamilton ([2009](#_ENREF_49)) reported a similar GxE specifically with respect to friends’ and adolescents’ own alcohol use. Finally, findings by Boardman, Saint Onge, Haberstick, Timberlake, and Hewitt ([2008](#_ENREF_11)) with the Add Health data suggest that genetically vulnerable youth may not only be influenced by their close friends’ behavior. They are also more likely to smoke when they attend schools where the most popular students are also smokers.

All the above studies used samples of adolescents. The few studies with children have so far produced equivocal findings. Thus, in one study that used mother and father ratings to assess both the target children’s and their peers’ delinquent behavior, the results showed that DPA indeed played an enhancement role even when controlling for rGE. However, it was the shared environmental influences rather than the genetic influences on delinquent behavior that were exacerbated by DPA ([Burt & Klump, 2013](#_ENREF_23)). In contrast, evidence of true GxE was found in a study using the QNTS sample, for whom teacher- and peer rated generalized aggression was available both with respect to the twins themselves and with respect to each twin child’s three reciprocal classroom friends in kindergarten ([van Lier et al., 2007](#_ENREF_93)). In line with an enhancement process of GxE, children were most likely to display high levels of aggression if they were at high genetic risk for such behavior and, at the same time, were exposed to highly aggressive friends. A follow-up study conducted with data collected in grade one ([Brendgen et al., 2008](#_ENREF_17)) revealed that this GxE may only hold for the link between friends’ and children’s physical aggression but not relational aggression, a more insidious type of aggression that includes social exclusion or malicious gossiping*.* Instead, affiliation with relationally aggressive friends seemed to foster relational aggression independently of genetic effects on this behavior.

To summarize, quantitative genetic studies that examined GxE unanimously found that a genetic disposition for delinquent behavior is more likely to be expressed when adolescents affiliate with deviant peers, thus supporting the Social Interaction model. It is still unclear, however, whether the same also holds true for children prior to adolescence or for all forms of delinquent behavior. Prospective longitudinal quantitative genetic studies using a variety of sources to measure DPA throughout childhood and adolescence and distinguishing between subtypes of delinquent behavior are needed to clarify a possible developmental change of GxE linking DPA and delinquent behavior.

**Testing the Social Interaction Model through Gene-Environment Interaction: Findings from Molecular Genetic Studies**

The findings from quantitative genetic studies showed that DPA may influence delinquent behavior either directly or by fostering the expression of youngsters’ genetic risk for delinquency. However, these studies cannot determine which specific genes are involved. To this end, molecular genetic studies are needed. Of note, although statistically it is irrelevant which of the two variables involved in an interaction is considered the moderator, GxE in molecular genetic studies are often interpreted using the genotype as the moderator of the effect of DPA on delinquent behavior. Again, only very few studies to date have investigated the interactive effect of DPA and specific genes on delinquent behavior. Thus, using a subsample of male Caucasian adolescents and young adults from the Add Health study, Lee (2011) examined a potential interaction between the monoamine oxidase-A (MAOA) gene and DPA in predicting delinquent behavior. The MAOA gene is implicated in the breakdown of synaptic neurotransmitters such as dopamine, norepinephrine, and serotonin and has been associated with antisocial behavior in some studies (e.g., [Buckholtz & Meyer-Lindenberg, 2008](#_ENREF_18); [Caspi et al., 2002](#_ENREF_27); [Kim-Cohen et al., 2006](#_ENREF_58)). Deviant peer affiliation was assessed based on participants’ reports of their three best friends’ smoking, drinking, and marijuana use, and of how often they fought together with their peers against others. The results showed a significant GxE in line with a trigger/enhancement process. Specifically, perceived peer deviancy was more strongly associated with participants’ overt antisocial and criminal behavior for carriers of the high risk (i.e., high-activity) MAOA genotype than for carriers of the low risk (low-activity) genotype. Similar results were reported in a study with Caucasian male and female adolescents ([Latendresse et al., 2011](#_ENREF_66)) with respect to the muscarinic acetylcholine receptor M2 gene (CHRM2), which is implicated in neurocognitive processes related to sensation-seeking and disinhibition ([Dick et al., 2011](#_ENREF_34)). Specifically, the likelihood of showing moderate to high levels of self-reported externalizing behavior from ages 12 through 22 years increased with each additional copy of the minor allelic (‘G’) variant of the CHRM2 gene, compared to individuals who only carried the ‘A’ allele. Moreover, this association was exacerbated in individuals who reported affiliating with highly antisocial peers at age 12 years. Finally, Kretschmer et al. ([2014](#_ENREF_59)) found that exposure to deviant peers during childhood (i.e., at age 10) was related to a greater risk of aggression in adolescence (i.e., at age 15) for carriers of the met/met variant than for carriers of the val/val variant of the BDNF polymorphism, which is implicated in the regulation of responses to stress ([Colzato, van der Does, Kouwenhoven, Elzinga, & Hommel, 2011](#_ENREF_30)).

To our knowledge, only one study has tested a possible interaction between DPA and specific measured genes in young children. The study focused on the DRD4 polymorphism, which had been associated with ADHD in children in previous research ([Gornick et al., 2007](#_ENREF_47); [Li, Sham, Owen, & He, 2006](#_ENREF_70)). The authors observed preschoolers while interacting with an unfamiliar same-age, same-sex peer during a free play paradigm in a laboratory setting ([DiLalla, Elam, & Smolen, 2009](#_ENREF_36)). The results showed that, when the peer was not aggressive, children with at least one long allele of DRD4 (i.e., the risk allele) were significantly more aggressive than children without the risk allele. In contrast, when the peer behaved aggressively, children with and children without the risk allele were equally highly aggressive. Interestingly, this pattern indicates a suppression process – rather than an enhancement process – of GxE: In a benign peer environment, interindividual differences in aggressive behavior could be explained by children’s genetic vulnerability, whereas exposure to an aggressive peer environment was sufficient to elicit high aggression even in children without genetic risk. These results are more in line with the predictions made by the Social Influence Model than those made by the Social Interaction Model.

Overall, the few existing molecular genetic studies thus support the findings from the quantitative genetic studies that delinquent behavior is influenced by an interactive effect of DPA and genetic factors. At least in adolescence, this interactive effect seems to correspond to an enhancement process, thus supporting the Social Interaction Model. Whether the same holds true for young children is still unclear, however, as the few existing studies have produced equivocal results that are more in line with the Social Influence Model than with the Social Interaction Model.

**Mechanisms that Could Account for a Main or Moderating Effect of DPA**

The results from the molecular and quantitative genetic studies thus give credence to the findings from non-genetically informed studies showing that DPA may foster delinquent behavior, either directly (most likely in younger children) or by facilitating the expression of pre-existing personal disposition for delinquent behavior (most likely in older children or adolescents). What mechanisms can account for the main or moderating effect of DPA? One mechanism that may explain how exposure to DPA predicts an increase in delinquent behavior is observational learning through modeling of rule-breaking or aggressive behaviors ([Berndt, 1999](#_ENREF_9)). Clear modeling effects have been shown by Cohen and Prinstein ([2006](#_ENREF_29)) in an experiment in which adolescents were randomly exposed to virtual peers in a laboratory setting. Adolescents conformed to the virtual peers’ aggressive/risky behaviors, particularly if the peers had a high (versus a low) social status. Similar experiments as well as observational studies showed that exposure to peers committing aggressive or deviant acts resulted in an increase in children’s similar behaviors already during the preschool years ([see Boivin et al., 2005](#_ENREF_14)).

A second process that can explain how DPA can impact children’s or adolescents’ delinquent behavior is differential reinforcement by deviant peers. This process, labeled “deviancy training” has received substantial empirical support ([Dishion, Spracklen, Andrews, & Patterson, 1996](#_ENREF_41)). Specifically, deviant peers tend to reinforce, through laughter or positive verbal or nonverbal support, rule-breaking talk and deviant acts. They also tend to ignore or punish normative behaviors ([Buehler, Patterson, & Furniss, 1966](#_ENREF_19)). This differential reinforcement of deviant behaviors has been found to result in an increase in youngsters’ subsequent delinquent behavior and substance use ([Dishion, Poulin, & Burraston, 2001](#_ENREF_40)). Deviancy training may already occur among kindergarten children. Engaging in deviant talk and positive reinforcement of deviant behaviors with same-gender peers predicted an increase in both overt and covert delinquent behavior over a 1-year interval ([Snyder et al., 2005](#_ENREF_86)) and covert delinquent behavior over a 3-year interval ([Snyder et al., 2008](#_ENREF_85)).

Finally, antisocial children and adolescents have been found to be bossier with their peers, including their friends, and are more frequently involved in coercive exchanges than non-antisocial children ([Deptula & Cohen, 2004](#_ENREF_33); [Dishion, Andrews, & Crosby, 1995](#_ENREF_37)). These conflict-ridden interactions could set in motion a “coercive interactional process” ([Boivin & Vitaro, 1995](#_ENREF_13); [Patterson, Reid, & Dishion, 1992](#_ENREF_77)) whereby coercing or threatening one’s friends can increase the likelihood of similar coercive behaviors from the friends. Consistent with this notion, conflict with a best friend predicted delinquency beyond peer rejection and best friend’s aggressiveness ([Kupersmidt, Burchinal, & Patterson, 1995](#_ENREF_60)). In another study, coercion by the best friend partially explained the predictive association between the best friend’s aggressiveness and an increase in children’s aggressiveness, controlling for other negative experiences such as parent coercion and peer rejection ([Vitaro et al., 2011](#_ENREF_98)). Coercion by the best friend, however, is not associated with increased aggression when the friendship bond and friends’ conflict resolution skills are high ([Salvas et al., sous presse](#_ENREF_83)). Interestingly, different processes may be related to different outcomes. As shown by Snyder, Schrepferman, Stoolmiller, and Brooker ([2007](#_ENREF_87)), deviancy training and modeling may foster covert delinquent behavior, whereas coercion may be help explain overt delinquent behavior. Similar processes may explain how DPA moderates the expression of youngsters’ genetic disposition for overt or covert aggression and delinquency.

**Summary**

* Genetic influences on DPA have been found in several studies. These genetic influences, and consequently the likelihood for rGE, seem to increase with age, but no study yet examined this issue using a prospectively longitudinal design.
* In light of the current genetically informed literature, the Socialization model could be in effect during childhood whereas the Selection model would (increasingly) apply during adolescence.
* In accordance to the Interaction model, DPA seems to amplify the expression of genetic dispositions towards delinquent behavior in adolescents. This appears to be the case in children also, but it is family and neighborhood effects that are enhanced by DPA.
* In turn, the role of DPA is modified by individual characteristics, including genetic polymorphisms. The exact nature of these moderating effects, i.e., enhancement or suppression, is still unsettled and may involve a developmental dimension.
* A number of mechanisms can account for the main or the moderating effect of DPA. These mechanisms can differ whether covert or overt delinquency is concerned.

**Future Research Needs**

* Longitudinal quantitative genetic studies using the same definition and the same sources to measure DPA throughout childhood and adolescence are needed to resolve the issues raised in this review chapter in reference to rGE or GxE.
* These studies should distinguish between overt and covert delinquency for both the peers and the participants and the severity of these behaviors (e.g. use of weapons) ([Lacourse et al., 2010](#_ENREF_61); [Lacourse, Dupéré, & Loeber, 2008](#_ENREF_63)). Further refinements between physical vs. social, proactive vs. reactive aggression are possible ([see Vitaro & Brendgen, 2012](#_ENREF_96)).
* Future studies also need to use a multi-source, multi-method approach with respect to both DPA and delinquency. Most studies reported in the chapter used one source only to report on both DPA and delinquent behavior. They may also need to distinguish and compare what DPA refers to and how it is measured. Potential halo effects or assumed similarity might be increased if the characteristics of the friendship group are evaluated as a whole instead of judging the behavior of specific, nominated friends and if the measures reflect the participants’ perceptions of their friends’ behavior instead of friends’ self-reported behavior ([Kandel, 1978](#_ENREF_54)).
* Future studies need to adopt a developmental perspective and distinguish between childhood/onset (overt and covert) delinquency and adolescence/onset (overt and covert) delinquency. Childhood/onset and adolescence/onset delinquent behaviors may have different gene-environment architectures as suggested by several authors ([Moffitt, 1993](#_ENREF_73); [Patterson, DeBaryshe, & Ramsey, 1989](#_ENREF_76)). A growth-curve or a latent class trajectory approach would help empirically define the different developmental patterns of delinquent behavior and relate them to different patterns of DPA ([Lacourse et al., 2008](#_ENREF_63); [Lacourse et al., 2003](#_ENREF_64); [Lacourse et al., 2006](#_ENREF_65)). Furthermore, they should address stability and intra-individual change and also how DPA evolves into more organized deviant clique (i.e., gang membership) or organized crime.
* Future studies need to verify whether the pattern of results is similar for males and females. Other variables such as ethnicity or macro socio-demographics could also be tested as moderators.

**Recommended Readings**

Bates, T. C. and Lewis, G. J. (2012). Towards a genetically informed approach in the social sciences: Strengths and an opportunity. *Personality and Individual Differences, 53*, 374-380.

Brendgen, M. (2012). Genetics and peer relations: A review. *Journal of Research on Adolescence, 22*, 419-437.

Kendler, K. S., Jaffee, S., & Romer, D. (2011). *The dynamic genome and mental health: The role of genes and environments in youth development*. Oxford University Press: Oxford.

Freese, J. (2008). Genetics and the Social Science Explanation of Individual Outcomes. *American Journal of Sociology, 114,* S1-S35*.*

**References**

Baker, L. A., & Daniels, D. (1990). Nonshared environmental-influences and personality-differences in adult twins. *Journal of Personality and Social Psychology, 58*(1), 103-110. doi: 10.1037//0022-3514.58.1.103

Barker, E. D., Tremblay, R. E., van Lier, P. A. C., Vitaro, F., Nagin, D. S., Assaad, J.-M., & Séguin, J. R. (2011). The neurocognition of conduct disorder behaviors: Specificity to physical aggression and theft after controlling for ADHD symptoms. *Aggressive Behavior, 37*(1), 63-72.

Bates, T. C., & Lewis, G. J. (2012). Towards a genetically informed approach in the social sciences: Strengths and an opportunity. *Personality and Individual Differences, 53*(4), 374-380. doi: 10.1016/j.paid.2012.03.002

Beaver, K. M. (2008). Nonshared environmental influences on adolescent delinquent involvement and adult criminal behavior. *Criminology, 46*(2), 341-369.

Beaver, K. M., DeLisi, M., Wright, J. P., & Vaughn, M. G. (2009). Gene-environment interplay and delinquent involvement evidence of direct, indirect, and interactive effects. *Journal of Adolescent Research, 24*(2), 147-168. doi: 10.1177/0743558408329952

Beaver, K. M., Ratchford, M., & Ferguson, C. J. (2009). Evidence of genetic and environmental effects on the development of low self-control. *Criminal Justice and Behavior, 36*(11), 1158-1172. doi: 10.1177/0093854809342859

Beaver, K. M., Shutt, J. E., Boutwell, B. B., Ratchford, M., Roberts, K., & Barnes, J. C. (2009). Genetic and environmental influences on levels of self-control and delinquent peer affiliation. *Criminal Justice and Behavior, 36*(1), 41-60. doi: 10.1177/0093854808326992

Beaver, K. M., Wright, J. P., & DeLisi, M. (2008). Delinquent peer group formation: Evidence of a gene x environment correlation. *Journal of Genetic Psychology, 169*(3), 227-244. doi: 10.3200/gntp.169.3.227-244

Berndt, T. J. (1999). Friends' influence on students' adjustment to school. *Educational Psychologist, 34*, 15-28.

Berndt, T. J., & Keefe, K. (1995). Friends' influence on adolescents' adjustment to school. *Child Development, 66*, 1312-1329.

Boardman, J. D., Saint Onge, J. M., Haberstick, B. C., Timberlake, D. S., & Hewitt, J. K. (2008). Do schools moderate the genetic determinants of smoking? *Behavior Genetics, 38*(3), 234-246. doi: 10.1007/s10519-008-9197-0

Boisvert, D., Boutwell, B. B., Vaske, J., & Newsome, J. (2013). Genetic and environmental overlap between delinquent peer association and delinquency in adolescence [Published online]. *Criminal Justice and Behavior*.

Boivin, M., & Vitaro, F. (1995). The impact of peer relationships on aggression in childhood: Inhibition through coercion or promotion through peer support. In J. McCord (Ed.), *Coercion and punishment in long-term perspectives* (pp. 183-197). New York, NY: Cambridge University Press.

Boivin, M., Vitaro, F., & Poulin, F. (2005). Peer relationships and the development of aggressive behavior in early childhood. In R. E. Tremblay, W. W. Hartup, & J. Archer (Eds.), *Developmental origins of aggression* (pp. 376-397). New York, NY: Guilford Press.

Bornovalova, M. A., Hicks, B. M., Iacono, W. G., & McGue, M. (2010). Familial transmission and heritability of childhood disruptive disorders. *American Journal of Psychiatry, 167*(9), 1066-1074. doi: 10.1176/appi.ajp.2010.09091272

Brendgen, M. (2012). Genetics and peer relations: A review. *Journal of Research on Adolescence, 22*(3), 419-437. doi: 10.1111/j.1532-7795.2012.00798.x

Brendgen, M., Boivin, M., Vitaro, F., Bukowski, W. M., Dionne, G., Tremblay, R. E., & Pérusse, D. (2008). Linkages between children's and their friends' social and physical aggression: Evidence for a gene-environment interaction. *Child Development, 79*(1), 13-29.

Buckholtz, J. W., & Meyer-Lindenberg, A. (2008). MAOA and the neurogenetic architecture of human aggression. *Trends in Neurosciences, 31*(3), 120-129.

Buehler, R. E., Patterson, G. R., & Furniss, J. M. (1966). The reinforcement of behavior in institutional settings. *Behaviour Research and Therapy, 4*, 157-167.

Bullock, B. M., Deater-Deckard, K., & Leve, L. D. (2006). Deviant peer affiliation and problem behavior: A test of genetic and environmental influences. *Journal of Abnormal Child Psychology, 34*, 29-41.

Burt, S. A. (2009). Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. *Clinical Psychology Review, 29*(2), 163-178. doi: 10.1016/j.cpr.2008.12.004

Burt, S. A., & Klump, K. L. (2009). The etiological moderation of aggressive and nonaggressive antisocial behavior by age. *Twin Research and Human Genetics, 12*(4), 343-350.

Burt, S. A., & Klump, K. L. (2013). Delinquent peer affiliation as an etiological moderator of childhood delinquency. *Psychological Medicine, 43*(6), 1269-1278. doi: 10.1017/s0033291712000013

Burt, S. A., McGue, M., & Iacono, W. G. (2009). Nonshared Environmental Mediation of the Association Between Deviant Peer Affiliation and Adolescent Externalizing Behaviors Over Time: Results From a Cross-Lagged Monozygotic Twin Differences Design. *Developmental Psychology, 45*(6), 1752-1760. doi: 10.1037/a0016687

Button, T. M. M., Corley, R. P., Rhee, S. H., Hewitt, J. K., Young, S. E., & Stallings, M. C. (2007). Delinquent peer affiliation and conduct problems: A twin study. *Journal of Abnormal Psychology, 116*(3), 554-564. doi: 10.1037/0021-843X.116.3.554

Button, T. M. M., Stallings, M. C., Rhee, S. H., Corley, R. P., Boardman, J. D., & Hewitt, J. K. (2009). Perceived peer delinquency and the genetic predisposition for substance dependence vulnerability. *Drug and Alcohol Dependence, 100*(1-2), 1-8. doi: 10.1016/j.drugalcdep.2008.08.014

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*, 851-854.

Cleveland, H. H., Wiebe, R. P., & Rowe, D. C. (2005). Sources of exposure to smoking and drinking friends among adolescents: A nehavioral-genetic evaluation. *Journal of Genetic Psychology, 166*(2), 153-169.

Cohen, G. L., & Prinstein, M. J. (2006). Peer contagion of aggression and health risk behavior among adolescent males: An experimental investigation of effects on public conduct and private attitudes. *Child Development, 77*(4), 967-983.

Colzato, L. S., van der Does, A. J. W., Kouwenhoven, C., Elzinga, B. M., & Hommel, B. (2011). BDNF Val(66)Met polymorphism is associated with higher anticipatory cortisol stress response, anxiety, and alcohol consumption in healthy adults. *Psychoneuroendocrinology, 36*(10), 1562-1569. doi: 10.1016/j.psyneuen.2011.04.010

Conger, K. J., & Conger, R. D. (1994). Differential parenting and change in sibling differences in delinquency. *Journal of Family Psychology, 8*, 287-302.

Cornish, K. M., Manly, T., Savage, R., Swanson, J., Morisano, D., Butler, N., Grant, C., Cross, G., Bentley, L., & Hollis, C. P. (2005). Association of the dopamine transporter (DAT1) 10/10-repeat genotype with ADHD symptoms and response inhibition in a general population sample. *Molecular Psychiatry, 10*(7), 686-698. doi: 10.1038/sj.mp.4001641

Deptula, D. P., & Cohen, R. (2004). Aggressive, rejected, and delinquent children and adolescents: A comparison of their friendships. *Aggression and Violent Behavior, 9*, 75-104.

Dick, D. M., Meyers, J. L., Latendresse, S. J., Creemers, H. E., Lansford, J. E., Pettit, G. S., Bates, J. E., Dodge, K. A., Budde, J., Goate, A., Buitelaar, J. K., Ormel, J., Verhulst, F. C., & Huizink, A. C. (2011). CHRM2, Parental Monitoring, and Adolescent Externalizing Behavior: Evidence for Gene-Environment Interaction. *Psychological Science, 22*(4), 481-489. doi: 10.1177/0956797611403318

DiLalla, L. F. (2002). Behavior genetics of aggression in children: Review and future directions. *Developmental Review, 22*, 593-622.

DiLalla, L. F., Elam, K. K., & Smolen, A. (2009). Genetic and gene-environment interaction effects on preschoolers' social behaviors. *Developmental Psychobiology, 51*(6), 451-464. doi: 10.1002/dev.20384

Dishion, T. J., Andrews, D. W., & Crosby, L. (1995). Antisocial boys and their friends in early adolescence: Relationship characteristics, quality, and interactional processes. *Child Development, 66*(1), 139-151.

Dishion, T. J., & Patterson, G. R. (2006). The development and ecology of antisocial behavior. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Risk, disorder, and adaptation* (vol. 3, pp. 503-541). New York, NY: Wiley.

Dishion, T. J., Patterson, G. R., & Griesler, P. C. (1994). Peer adaptations in the development of antisocial behavior: A confluence model. In L. R. Huesmann (Ed.), *Aggressive behavior: Current perspectives* (pp. 61-95). New York, NY: Plenum Press.

Dishion, T. J., Poulin, F., & Burraston, B. (2001). Peer group dynamics associated with iatrogenic effects in group interventions with high-risk young adolescents. In D. W. Nangle & C. A. Erdley (Eds.), *New directions for child and adolescent development: Friendship and psychological adjustment* (pp. 79, ch. 92). San Francisco, CA: Jossey-Bass.

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

Dunn, J., & Plomin, R. (1990). *Separate lives: Why siblings are so different*. New York: Basic Books.

Dunn, J., Stocker, C., & Plomin, R. (1990). Nonshared experiences within the family: Correlates of behavior problems in middle childhood. *Development and Psychopathology, 2*, 113-126.

Elliott, D. S., Huizinga, D., & Ageton, S. S. (1985). *Explaining delinquency and drug use*. Beverly Hills, CA: Sage.

Fowler, J. H., Settle, J. E., & Christakis, N. A. (2011). Correlated genotypes in friendship networks. *Proceedings of the National Academy of Sciences of the United States of America, 108*(5), 1993-1997. doi: 10.1073/pnas.1011687108

Gardner, T. W., Dishion, T. J., & Connell, A. M. (2008). Adolescent self-regulation as resilience: Resistance to antisocial behavior within the deviant peer context. *Journal of Abnormal Child Psychology, 36*(2), 273-284. doi: 10.1007/s10802-007-9176-6

Gornick, M. C., Addington, A., Shaw, P., Bobb, A. J., Sharp, W., Greenstein, D., Arepalli, S., Castellanos, F. X., & Rapoport, J. L. (2007). Association of the dopamine receptor D4 (DRD4) gene 7-repeat allele with children with attention-deficit/hyperactivity disorder (ADHD): An update. *American Journal of Medical Genetics Part B-Neuropsychiatric Genetics, 144B*(3), 379-382. doi: 10.1002/ajmg.b.30460

Gottfredson, M. R., & Hirschi, T. (1990). *A general theory of crime*. Stanford, CA: Stanford University Press.

Guo, G., Elder, G. H., Cai, T. J., & Hamilton, N. (2009). Gene-environment interactions: Peers' alcohol use moderates genetic contribution to adolescent drinking behavior. *Social Science Research, 38*(1), 213-224. doi: 10.1016/j.ssresearch.2008.04.002

Harden, K. P., Hill, J. E., Turkheimer, E., & Emery, R. E. (2008). Gene-environment correlation and interaction in peer effects on adolescent alcohol and tobacco use. *Behavior Genetics, 38*(4), 339-347. doi: 10.1007/s10519-008-9202-7

Harris, J. R. (1995). Where is the child's environment: A group socialization theory of development. *Psychological Review, 102*(3), 458-489.

Hill, S. Y., Zezza, N., Wipprecht, G., Locke, J., & Neiswanger, K. (1999). Personality traits and dopamine receptors (D2 and D4): Linkage studies in families of alcoholics. *American Journal of Medical Genetics, 88*(6), 634-641. doi: 10.1002/(sici)1096-8628(19991215)88:6<634::aid-ajmg11>3.0.co;2-m

Jaffee, S. R., & Price, T. S. (2007). Gene-environment correlations: a review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry, 12*(5), 432-442. doi: 10.1038/sj.mp.4001950

Kandel, D. B. (1978). Homophily, selection, and socialization in adolescent friendships. *American Journal of Sociology, 84*, 427-436.

Kendler, K. S., Jacobson, K., Myers, J. M., & Eaves, L. J. (2008). A genetically informative developmental study of the relationship between conduct disorder and peer deviance in males. *Psychological Medicine, 38*(7), 1001-1011. doi: 10.1017/s0033291707001821

Kendler, K. S., Jacobson, K. C., Gardner, C. O., Gillespie, N., Aggen, S. A., & Prescott, C. A. (2007). Creating a social world - A developmental twin study of peer-group deviance. *Archives of General Psychiatry, 64*(8), 958-965. doi: 10.1001/archpsyc.64.8.958

Kendler, K. S., Schmitt, E., Aggen, S. H., & Prescott, C. A. (2008). Genetic and environmental influences on alcohol, caffeine, cannabis, and nicotine use from early adolescence to middle adulthood. *Archives of General Psychiatry, 65*(6), 674-682. doi: 10.1001/archpsyc.65.6.674

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W., & Moffitt, T. E. (2006). MAOA, maltreatment, and gene-environment interaction predicting children's mental health: new evidence and a meta-analysis. *Molecular Psychiatry, 11*(10), 903-913. doi: 10.1038/sj.mp.4001851

Kretschmer, T., Vitaro, F., & Barker, E. D. (2014). The association between peer and own aggression is moderated by the BDNF val-met polymorphism. *Journal of Research on Adolescence, 24*(1), 177-185. doi: 10.1111/jora.12050

Kupersmidt, J. B., Burchinal, M., & Patterson, C. J. (1995). Developmental patterns of childhood peer relations as predictors of externalizing behavior problems. *Development and Psychopathology, 7*, 825-843.

Lacourse, E., Baillargeon, R., Dupéré, V., Vitaro, F., Romano, E., & Tremblay, R. (2010). Two-year predictive validity of conduct disorder subtypes in early adolescence: A latent class analysis of a Canadian longitudinal sample. *Journal of Child Psychology and Psychiatry, 51*(12), 1386-1394.

Lacourse, E., Boivin, M., Brendgen, M., Petitclerc, A., Girard, A., Vitaro, F., Paquin, S., Ouellet-Morin, I., Dionne, G., & Tremblay, R. E. (sous presse). A longitudinal twin study of physical aggression during early childhood: Evidence for a developmentally dynamic genome [Epub ahead of print]. *Psychological Medicine*, 1-11.

Lacourse, É., Dupéré, V., & Loeber, R. (2008). Developmental trajectories of violence and theft. In R. Loeber, D. Farrington, M. Stouthamer-Loeber, & H. R. White (Eds.), *Violence and serious theft: Development and prediction from childhood to adulthood* (pp. 231-268). New York, NY: Routledge/Taylor & Francis Group.

Lacourse, É., Nagin, D., Tremblay, R. E., Vitaro, F., & Claes, M. (2003). Developmental trajectories of boys' delinquent group membership and facilitation of violent behaviors during adolescence. *Development and Psychopathology, 15*(1), 183-197.

Lacourse, É., Nagin, D. S., Vitaro, F., Côté, S., Arseneault, L., & Tremblay, R. E. (2006). Prediction of early-onset deviant peer group affiliation: A 12-year longitudinal study. *Archives of General Psychiatry, 63*(5), 562-568.

Latendresse, S. J., Bates, J. E., Goodnight, J. A., Lansford, J. E., Budde, J. P., Goate, A., Dodge, K. A., Pettit, G. S., & Dick, D. M. (2011). Differential Susceptibility to Adolescent Externalizing Trajectories: Examining the Interplay Between CHRM2 and Peer Group Antisocial Behavior. *Child Development, 82*(6), 1797-1814. doi: 10.1111/j.1467-8624.2011.01640.x

Le Foll, B., Gallo, A., Le Strat, Y., Lu, L., & Gorwood, P. (2009). Genetics of dopamine receptors and drug addiction: a comprehensive review. *Behavioural Pharmacology, 20*(1), 1-17. doi: 10.1097/FBP.0b013e3283242f05

LeBlanc, M., & Loeber, R. (1998). Developmental criminology upgraded. In M. Tony (Ed.), *Crime and justice handbook* (pp. 115-198). Chicago, IL: Chicago University Press.

Lee, S. S. (2011). Deviant peer affiliation and antisocial behavior: Interaction with Monoamine Oxidase A (MAOA) genotype. *Journal of Abnormal Child Psychology, 39*(3), 321-332. doi: 10.1007/s10802-010-9474-2

Li, D. W., Sham, P. C., Owen, M. J., & He, L. (2006). Meta-analysis shows significant association between dopamine system genes and attention deficit hyperactivity disorder (ADHD). *Human Molecular Genetics, 15*(14), 2276-2284. doi: 10.1093/hmg/ddl152

Manke, B., McGuire, S., Reiss, D., Hetherington, E. M., & Plomin, R. (1995). Genetic contributions to adolescents extrafamilial social interactions: Teachers, best friends, and peers. *Social Development, 4*, 238-256.

McHale, S. M., Crouter, A. C., McGuire, S., & Updegraff, K. A. (1995). Congruence between mothers' and fathers' differential treatment of siblings: Links with family relations and children's well-being. *Child Development, 66*, 116-128.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100*(4), 674-701.

Moffitt, T. E. (2005). Genetic and environmental influences on antisocial behaviors: Evidence from behavioral-genetic research. In J. Hall (Ed.), *Advances in genetics* (vol. 55, pp. 41-104). Amsterdam, The Netherlands: Elsevier Science Publishers.

Niv, S., Tuvblad, C., Raine, A., & Baker, L. A. (2013). Aggression and rule-breaking: Heritability and stability of antisocial behavior problems in childhood and adolescence. *Journal of Criminal Justice, 41*(5), 285-291. doi: 10.1016/j.jcrimjus.2013.06.014

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist, 44*(2), 329-335.

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *A social learning approach: IV. Antisocial boys*. Eugene, OR: Castalia Publishing Co.

Pike, A., & Atzaba-Poria, N. (2003). Do sibling and friend relationships share the same temperamental origins? A twin study. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 44*(4), 598-611.

Purcell, S. (2002). Variance components models for gene-environment interaction in twin analysis. *Twin Research, 5*(6), 554-571.

Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin, 128*, 490-529.

Rose, R. J. (2002). How do adolescents select their friends? A behavior-genetic perspective. In L. Pulkkinen & A. Caspi (Eds.), *Paths to successful development: Personality in the life course* (pp. 106-125). New York: Cambridge University Press.

Rowe, D. C., & Osgood, D. W. (1984). Heredity and sociological theories of delinquency: A reconsideration. *American Sociological Review, 49*, 526-540.

Salvas, M.-C., Vitaro, F., Brendgen, M., Dionne, G., Tremblay, R. E., & Boivin, M. (sous presse). Friendship conflict and the development of generalized physical aggression in the early school years: A genetically informed study of potential moderators [Epub ahead of print]. *Developmental Psychology*.

Scarr, S., & McCartney, K. (1983). How people make their own environments: A theory of genotype greater than environment effects. *Child Development, 54*(2), 424-435.

Snyder, J., Schrepferman, L., McEachern, A., Barner, S., Johnson, K., & Provines, J. (2008). Peer deviancy training and peer coercion: Dual processes associated with early-onset conduct problems. *Child Development, 79*, 252-268.

Snyder, J., Schrepferman, L., Oeser, J., Patterson, G., Stoolmiller, M., Johnson, K., & Snyder, A. (2005). Deviancy training and association with deviant peers in young children: Occurrence and contribution to early-onset conduct problems. *Development and Psychopathology, 17*, 397-413.

Snyder, J., Schrepferman, L., Stoolmiller, M., & Brooker, M. (2007). The roles of anger, conflict with parents and peers, and social reinforcement in the early development of physical aggression. In T. A. Cavell & K. T. Malcolm (Eds.), *Anger, aggression, and interventions for interpersonal violence* (pp. 187-214). Mahwah, NJ: Erlbaum.

Tarantino, N., Tully, E. C., Garcia, S. E., South, S., Iacono, W. G., & McGue, M. (2014). Genetic and environmental influences on affiliation with deviant peers during adolescence and early adulthood. *Developmental Psychology, 50*(3), 663-673. doi: 10.1037/a0034345

Thornberry, T. P., Krohn, M. D., Lizotte, A. J., & Chard-Wierschem, D. (1993). The role of juvenile gangs in facilitating delinquent behavior. *Journal of Research in Crime and Delinquency, 30*, 55-87.

Thorpe, K., & Gardner, K. (2006). Twins and their friendships: Differences between monozygotic, dizygotic same-sex and dizygotic mixed-sex pairs. *Twin Research and Human Genetics, 9*(1), 155-164. doi: 10.1375/183242706776402984

Tuvblad, C., & Baker, L. (2011). Human aggression across the lifespan: Genetic propensities and environmental moderators (ch. 8). In R. Huber, P. Brennan, & D. Bannasch (Eds.), *Advances in genetics: Aggression* (vol. 75, pp. 171-214). Boston, MA: Elsevier Press.

van Beijsterveldt, C. E. M., Bartels, M., Hudziak, J. J., & Boomsma, D. I. (2003). Causes of stability of aggression from early childhood to adolescence: A longitudinal genetic analysis in Dutch twins. *Behavior Genetics, 33*(5), 591-605. doi: 10.1023/a:1025735002864

van Lier, P. A. C., Wanner, B., & Vitaro, F. (2007). Onset of antisocial behavior, affiliation with deviant friends, and childhood maladjustment: A test of the childhood- and adolescent-onset models. *Development and Psychopathology, 19*, 167-185.

Viding, E., Larsson, H., & Jones, A. P. (2008). Quantitative genetic studies of antisocial behaviour. *Philosophical Transactions of the Royal Society B: Biological Sciences, 363*(1503), 2519-2527. doi: 10.1098/rstb.2008.0037

Vitaro, F., Boivin, M., & Bukowski, W. M. (2009). The role of friendship in child and adolescent psychosocial development. In K. Rubin, W. M. Bukowski, & B. Laursen (Eds.), *Handbook of peer interactions, relationships, and groups* (pp. 568-588). New York, NY: Guilford Press.

Vitaro, F., & Brendgen, M. (2012). Subtypes of aggressive behaviors: Etiologies, development, and consequences. In T. Bliesener, A. Beelmann, & M. Stemmler (Eds.), *Antisocial behavior and crime: Contributions of developmental and evaluation research to prevention and intervention* (pp. 17-38). Cambridge, MA: Hogrefe Publishing.

Vitaro, F., Brendgen, M., & Arseneault, L. (2009). The discordant MZ-twin method: One step closer to the holy grail of causality. *International Journal of Behavioral Development, 33*(4), 376-382. doi: 10.1177/0165025409340805

Vitaro, F., Brendgen, M., Boivin, M., Cantin, S., Dionne, G., Tremblay, R. E., Girard, A., & Pérusse, D. (2011). A monozygotic twin difference study of friends' aggression and children's adjustment problems. *Child Development, 82*(2), 617-632. doi: 10.1111/j.1467-8624.2010.01570.x

Vitaro, F., Brendgen, M., & Tremblay, R. E. (2000). Influence of deviant friends on delinquency: Searching for moderator variables. *Journal of Abnormal Child Psychology, 28*, 313-325.

Vitaro, F., Tremblay, R. E., & Bukowski, W. M. (2001). Friends, friendships, and conduct disorders. In J. Hill & B. Maughan (Eds.), *Conduct disorder in childhood* (pp. 346-378). Cambridge: Cambridge University Press.

Vitaro, F., Tremblay, R. E., Kerr, M., Pagani, L. S., & Bukowski, W. M. (1997). Disruptiveness, friends' characteristics, and delinquency: A test of two competing models of development. *Child Development, 68*(4), 676-689.

# Genetic and Environmental Overlap Between Delinquent Peer Association and Delinquency in Adolescence

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## Abstract

Delinquent peer association and criminal/delinquent behaviors are highly intertwined. The directionality and mechanisms underlying this relationship, however, have been debated in the literature for decades. The current study seeks to further inform this debate by examining whether individual differences at the level of the genome can help to explain the association between delinquent peer affiliation and delinquency. Using the twin and full-sibling subsample from the National Longitudinal Study of Adolescent Health (Add Health), behavioral genetic methodology is used to examine whether delinquent peer affiliation and delinquency in adolescence covary as the result of common genetic factors. Results indicate that delinquent peer association and delinquency are moderately influenced by additive genetic factors, and that common genes are in fact influencing the covariance between the two outcomes. The importance of incorporating genetic explanations into traditional theories of delinquency is discussed.