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Socialization, Selection, or Both? The Role of Gene–Environment Interplay in the Association Between Exposure to Antisocial Peers and Delinquency

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Abstract

Objectives To better explain the near-universal association between peer and self-reported delinquency, three frameworks have been offered and have received varying degrees of support: (1) *socialization* or the social transmission of norms, attitudes, and behaviors among group members; (2) *selection* or the congregation of youth with similar traits and predispositions; and (3) *enhancement* or a combination of socialization and selection processes.

Methods Making use of sibling pairs and peer network data from the National Longitudinal Study of Adolescent to Adult Health, the current study compares all three frameworks using modified bivariate Cholesky models to simultaneously examine gene-environment correlations (rGE) and interactions (G × E).

Results Findings revealed that peer deviance (as reported by peers themselves) moderated underlying influences on delinquency such that genetic influences *decreased* and environmental influences *increased* as peer deviance increased. While previous studies have reported additional patterns of moderation (e.g., increases in both genetic and environmental influences), such studies have relied on subjective measures of peer behavior, more restrictive measures of delinquency, and samples comprised of young children.

Conclusions The results revealed preliminary evidence in favor of the selection hypothesis, but the overall patterns of moderation stemming from the examined G × E fall in line more closely with the enhancement hypothesis of peer influence.

Keywords Peers · Delinquency · Developmental theory · Gene–environment interplay

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Introduction

Peer behavior has been identified as a robust and influential factor in the development of a wide range of outcomes including physical (e.g., obesity; Chirstakis and Fowler 2007; Valente et al. 2009) and mental health problems (e.g., depression; Allen et al. 2006), age at sexual debut (Kinsman et al. 1998), and is widely considered one of the most important sources of influence on delinquency (Haynie et al. 2014; Hoeben et al. 2016; Pratt et al. 2010; Warr 2002). Despite the underlying consensus that behavioral patterns tend to cluster within peer groups, the more fundamental aspects of this association represented an important point of contention in the classic literature, with Hirschi and Gottfredson (1987) advancing the Glueck and Glueck's (1950) argument that "birds of a feather flock toge-ther" and Akers (2008) refuting that "if you lie down with dogs, you get up with fleas". In this way, the latter argument indicates that exposure to delinquent peers significantly increases the likelihood of subsequent delinquent and antisocial behaviors (commonly referred to as the *socialization hypothesis*), while the former indicates that delinquent youth are simply more likely to affiliate with one another (commonly referred to as the *selection hypothesis*).

While the socialization and selection hypotheses tend to represent opposite and extreme ends of the same continuum, a more tempered etiological pathway, primarily focused on the influence of gang membership on subsequent behavior, has also been proposed in the more contemporary literature (Gatti et al. 2005; Thornberry et al. 1993). The *enhancement hypothesis* posits that a combination of socialization and selection processes stemming from peer influences ultimately contribute to variability in subsequent behavioral patterns (de la Haye et al. 2013; Monahan et al. 2009). Individuals may affiliate with one another due to selection processes stemming from similarity in underlying traits, but the subsequent socialization processes that occur within peer groups effectively dampen or amplify pre-existing delinquent predisposition (Van Ryzin and Dishion 2014). Recent developments within the literature focused on further unpacking the association between peer and self-reported delinquency also find support for the enhancement hypothesis, with studies reporting findings that highlight a combination of both socialization and selection pro-cesses (Haynie et al. 2014; Matsueda and Anderson 1998; Samek et al. 2016; Wright et al. 1999). Also in line with these recent developments, Akers (1991) observed that "birds of a feather do flock together, but it [social learning theory] also admits that if the birds are humans, they also will influence one another's behavior, in both conforming and deviant directions" (p. 210).

This theoretical progress has been further bolstered through the introduction and implementation of innovative methodological techniques, with two particular advances shedding additional light on the mechanisms driving the association between peer and self-reported delinquency. Recent studies have demonstrated important differences between objective (typically measured using social network data) and subjective (typically mea-sured as respondents' assessments of their peers' behaviors) measures of peer behavior (Haynie and Osgood 2005; Hoeben et al. 2016; Meldrum and Boman 2013; Young 2014), with some studies suggesting that each measurement strategy taps a distinct latent con-struct (Meldrum and Boman 2013; Young et al. 2015). Studies have also more closely examined the potential role of genetic influences in the processes that ultimately result in the formation of peer groups and the transmission of behavioral norms within such groups (Brendgen 2012; Button et al. 2007; Connolly et al. 2015; Samek et al. 2017). These genetically-informed studies aim to examine the potential association between peer behaviors and self-reported delinquency after controlling for influences that tend to cluster

within families (which are typically both genetic and environmental in origin). While the results of this developing line of research have indicated that genetic influences are implicated in the association between peer behaviors and self-reported delinquency (Connolly et al. 2015; Hill et al. 2008; Samek et al. 2017), the overall pattern of findings provide support for both the socialization (Gaughan 2006; Watts and McNulty 2015) and selection hypotheses (Barnes et al. 2014; Gillespie et al. 2009; Hoffman et al. 2007; Iervolino et al. 2002; TenEyck and Barnes 2015). Also, and directly in line with previous theoretical development (Bronfenbrenner and Ceci 1994; Shanahan and Hofer 2005), additional studies have examined the enhancement hypothesis of peer influence (Agrawal et al. 2010; Guo et al. 2015; Harden et al. 2008; Hicks et al. 2009; Kendler et al. 2011; Samek et al. 2017), but such studies have commonly relied on subjective measures of peer behavior and more restrictive measures of delinquent behavior.

Taken together, findings from the current literature appear to provide near-universal support for the association between peer influences and self-reported delinquency (but see Brauer 2009), but there appears to be less consensus surrounding the underlying mechanisms that ultimately contribute to the association. In light of these observations, the current study aims to accomplish three interrelated objectives: (1) directly test the socialization, selection, and enhancement hypotheses of peer influence on delinquency within a genetically-informed framework; (2) more closely explore genetic and environmental pathways that contribute to the formation of peer groups; and (3) examine changes in genetic and environmental influences on self-reported delinquency across levels of an objective measure of peer group deviance.

Multiple Pathways of Peer Influence on Delinquency

Both classic and contemporary theoretical perspectives have emphasized the importance of peers in the development of behavioral outcomes, with some of the first formalized attempts to explain behavioral similarity within peer groups primarily focusing on the socialization hypothesis (Akers 1973; Burgess and Akers 1966; Sutherland 1947; Sutherland and Cressey 1955). Beginning with Sutherland's (1947; Sutherland and Cressey 1955) differential association theory and carrying through to Akers's social learning theory (1973; Burgess and Akers 1966), these perspectives proposed that delinquency was learned just like any other behavior and attempted to specify the process in which behaviors are transferred among members of peer groups. The connections between differential association and social learning theory are largely deliberate (Akers and Jensen 2006), but perhaps the primary similarity between these perspectives is their shared foundation in the socialization hypothesis. Both perspectives operate under the assumption that behavior must be learned from others, indicating that the socialization mechanisms outlined in both theories specify the processes in which attitudes, beliefs, and behaviors are passed from one individual (or group of individuals) to another. This shared characteristic of both perspectives represents an important point of distinction from other theoretical perspectives, in that it operates under the assumption that any preexisting internalized sources of predisposition toward delinquency are either nonexistent, or, at the very least, outweighed by the specified learning process.

On the other end of the theoretical continuum, competing perspectives have characterized the similarity between members of a given peer group in a different way, focusing on preexisting individual, internalized traits and the congregation of more similar

individuals (i.e., selection; Hirschi 1969). Previous studies have revealed that peer group members tend to more closely resemble one another across a wide range of physical characteristics (Chirstakis and Fowler 2007; Stevens et al. 1990; Valente et al. 2009), personality traits (Banikiotes and Neimeyer 1981; Bleda 1974), levels of academic achievement (Chen et al. 2008; Wentzel and Caldwell 1997), and other features compared to youth chosen at random. A related line of research has reported similar results for individual traits more directly related to delinquency, such as risk-seeking behaviors (Brechwald and Prinstein 2011), aggression (Espelage et al. 2003), and substance use (Harden et al. 2008). In, arguably, the most well-known example of the selection hypothesis in the classic criminological literature, Hirshi and Gottfredson (1987; and later, Gottfredson and Hirschi 1990; Hirschi and Gottfredson 2000) famously argued that socialization processes within peer groups have minimal impact on long-term trends in delinquency. Rather, they offered an argument rooted in a "hard-lined" form of selection in which delinquent youth were drawn to one another through similarity in overall levels of self-control, and subsequently, long-term behavioral trajectories.

While both the socialization and selection hypotheses have been the subject of a considerable amount of research examining peer effects in the classic literature (for general overviews see Hoeben et al. 2016; Pratt et al. 2010), a third, more moderate, theoretical framework has also been offered. This perspective, often referred to as the enhancement hypothesis, posits that socialization and selection processes collectively contribute to increased similarity between peer group members (Caspi 2002; Kiuru et al. 2010). While the enhancement hypothesis has been most closely linked to research examining gang formation and delinquency (Gatti et al. 2005; Thornberry et al. 1993), it also directly aligns with the theoretical concept of *homophily*, which highlights the importance of both selection and socialization processes in the development of peer similarities (Brechwald and Prinstein 2011; Flashman and Gambetta 2014; McPherson et al. 2001; Turnovic and Young 2016; Young 2014). Specifically, selection processes may contribute to the congregation of more similar individuals, but subsequent interactions between group members work to amplify or dampen preexisting individual traits, resulting in increased behavioral similarity among peer group members (Van Ryzin and Dishion 2014). Three distinct observations from the existing literature provide additional support for the enhancement hypothesis. First, previous studies have found that various characteristics of peer network structure (e.g., density; Haynie 2001, 2002; Haynie and Osgood 2005; McGloin and Shermer 2009) as well as the attitudes and behaviors of individual group members (Agnew 1991; Megens and Weerman 2012) significantly moderate the association between peer behavior and self-reported delinquency. These findings indicate that the association between peer influences and subsequent delinquent behavior may be conditioned by the nature and frequency of group contact. Second, studies employing longitudinal social network data have revealed that similarity among peer group members across multiple traits and behaviors increases over time (Chirstakis and Fowler 2007, 2008). Third, additional studies employing social network data have also made use of sophisticated modeling strategies that allow for the simultaneous estimation of socialization and selection processes (e.g., stochastic agent based models) and have found evidence of the co-occurrence of both sets of influences (Haynie et al. 2014; Kiuru et al. 2010; Lomi et al. 2011; Turnovic and Young 2016; Young et al. 2014).

Gene–Environment Interplay and Peers

A sizable literature has also explored the potential combined contribution of genetic and environmental influences on the association between peer and self-reported delinquency (Beaver et al. 2009; Button et al. 2007; Fergusson et al. 1999; Iervolino et al. 2002; Kendler et al. 2011; Samek et al. 2013). This line of research is rooted in contemporary developmental theory, which posits that biological and social influences are systematically integrated and fluctuate across various stages of human development (Lerner and Castellino 2002). Directly in line with developmental theory, a sizable number of studies have found evidence suggesting that affiliating with delinquent peers is influenced by a combination of both genetic and environmental influences (Agrawal et al. 2010; Kendler et al. 2007; Samek et al. 2013), with such studies indicating that heritability estimates increase across the life course.

While this pattern of results may seem counterintuitive at first glance, genetic influences on peer deviance are likely the result of an active gene–environment correlation (typically symbolized as *r*GE; Plomin et al. 2013; Rutter et al. 2006; Scarr 1992; Scarr and McCartney 1983). Active *r*GE[1] provides greater insight into the potential mechanisms that ultimately contribute to genetic influences on peer deviance (Brendgen 2012; Button et al. 2007). As mentioned previously, individuals from the same peer group tend to more closely resemble one another across a wide variety of traits and characteristics compared to individuals chosen at random. An active *r*GE can be described as the process in which individuals who have more genetically-influenced traits in common would be more likely to cluster together. According to the logic of active *r*GE, similarity in such traits directly translates to increased levels of genetic similarity (or shared genetic material) within peer groups since variability in virtually all phenotypes is the result of a combination of both genetic and environmental influences (Polderman et al. 2015; Turkheimer 2000). Within the context of the current study, the presence of an active *r*GE would most closely align with the selection hypothesis, since such processes imply that genetically influenced sources of selection contribute to similarities in delinquency among peer group members.

Directly in line with this possibility, a developed line of research has provided support for the presence of active *r*GE in the development of peer groups (Barnes et al. 2014; Button et al. 2007; Connolly et al. 2015; Fowler et al. 2007). For example, the results of a recent study indicated that approximately one-third (37%) of the covariance between changes in peer delinquency and developmental trajectories in delinquent behavior was explained by additive genetic influences (Connolly et al. 2015). Additional evidence of genetically-influenced selection processes (i.e., active *r*GE) was observed by Fowler and colleagues (2011), who examined a small set of genetic polymorphisms and found that members of a peer group tend to share more genetic variants (and, in turn, more genetically influenced phenotypes) compared to individuals chosen at random. In a follow-up study, Christakis and Fowler (2014) examined a larger number of genetic variants (over 400,000 single nucleotide polymorphisms or SNPs) within a sample of nearly 2000 individuals from the Framingham Heart Study. Once again, the results indicated that members of the same friendship pair more closely resembled one another across the examined genetic

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[1] This discussion is limited to active *r*GE since this form of *r*GE has been most closely linked to peer group formation and selection processes in previous research (TenEyck and Barnes 2015). However, two additional forms of *r*GE have been discussed previously—passive and evocative. Additional information on all three forms of *r*GE can be found elsewhere (Plomin et al. 2013; Rutter et al. 2006; Scarr and McCartney 1983).

variants compared to randomly paired individuals. These findings indicate that selection stemming from genetically-influenced traits (such as impulsivity, delinquency, or attitudes favorable toward delinquent behavior) represents one possible source of influence contributing to homophily. In addition, these findings indicate that, due to selection processes, behavioral patterns within peer groups are not free from internalized sources of influence. This is an important caveat as it would allow for the possibility that any underlying predisposition toward self-reported delinquency may be conditioned by peer influences and also allows for the potential of an over- or underestimation of the resulting association due to underlying genetic influences on delinquent behavior (Turkheimer and Harden 2014).

Due to this possibility, previous studies have employed genetically-informed research designs to further examine associations between peer influences and various outcomes including externalizing behavior problems (Button et al. 2007; Kendler et al. 2011; Samek et al. 2017; TenEyck and Barnes 2015), substance use (Agrawal et al. 2010; Gillespie et al. 2009; Harden et al. 2008; Hill et al. 2008; Rhee et al. 2003; Samek et al. 2013), academic achievement (Barnes et al. 2014), and internalizing problems like depression (Brendgen et al. 2009; Hicks et al. 2009). The primary advantage of employing a genetically-informed modeling strategy in this context is that selection processes related to active rGE are effectively controlled, better isolating the association between peer and self-reported delinquency.

In addition to active rGE, a second form of gene–environment interplay related to the association between peer influences and self-reported delinquency is gene–environment interaction (symbolized as G × E). This form of gene–environment interplay refers to the phenomenon in which genetic (or environmental) influences on a given phenotype are moderated by the simultaneous presence of environmental (or genetic) influences (Duncan 2014; Plomin et al. 2013; Rutter et al. 2006; Shanahan and Hofer 2005).[2] In this way, G × Es may be directly involved with the enhancement hypothesis, as any underlying genetic predisposition toward delinquent behavior (including those involved with active rGE) may be moderated by socialization processes within peer groups. In other words, genetically influenced traits (such as self-control or delinquency) may influence selection into a peer group, but those underlying influences may be effectively moderated based on subsequent interactions with peer group members, effectively increasing or decreasing predisposition toward engaging in delinquent behavior.

A G × E involving exposure to peer deviance resulting in increases in genetic influences on self-reported delinquency would indicate that underlying predispositions are maximized when paired with a social context that encourages delinquent behaviors. This pattern of results would more directly align with the *diathesis stress* or *contextual triggering* models, such that genetic predisposition for delinquency is increased when paired with a deleterious environmental context (Shanahan and Hofer 2005). Alternatively, a G × E involving peer deviance that results in increases in environmental influences (and decreases genetic influences) on delinquent behaviors would indicate that the social interactions between peer group members are driving increased levels of delinquency, above and beyond underlying genetic predispositions. This pattern of moderation has been

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[2] Importantly, multiple interpretations of G × Es have been offered in the existing literature, with at least three different theoretical models proposed previously: (1) *diathesis-stress* (Monroe and Simons 1991; Zuckerman 1999); (2) differential susceptibility (Belsky and Pluess 2009; Ellis et al. 2011); and (3) *average and expectable environments hypothesis* (Scarr 1992). These models, along with additional but similar models [e.g., Guo et al.'s (2015) *swing model*], have been described elsewhere (for a more comprehensive summary see Shanahan & Hofer, 2005), but an additional summary is also included in the online supplement.

previously referred to as *social context as enhancement* by Shanahan and Hofer (2005), and indicates that "positive" genetic predispositions will be enhanced in more positive or enriching environmental contexts, but these same genetic predispositions should be dampened in negative contexts. The social context as enhancement hypothesis is rooted in the bioecological model presented by Bronfenbrenner and Ceci (1994), which posits that genetic influences on positive attributes (e.g., prosocial behavior) increase as *proximal processes* increase in magnitude. Proximal processes refer to "mechanisms…through which genetic potentials for *effective* psychological functioning are actualized" (p. 568, emphasis in original), and refer to optimal environmental context. In this way, greater exposure to prosocial peers would be more likely to enhance genetic influences on prosocial behavior, but greater exposure to deviant peers should result in significant decreases in genetic influences on delinquent behavior. These two competing patterns of moderation (diathesis stress/contextual triggering and social context as enhancement) both characterize the enhancement hypothesis of peer influence, but do so in different ways, with the former expecting increases in both genetic and environmental influences as peer deviance increases and the latter expecting decreases in genetic influences and increases in environmental influences as peer deviance increases.

Previous studies examining the potential moderating influences of peer behaviors on underlying genetic and environmental influences on self-reported delinquency have revealed at least three patterns of results. First, the majority of existing literature reports findings consistent with the diathesis stress model or contextual triggering model, wherein exposure to negative peer behavior results in a simultaneous increase in both genetic and environmental influences on self-reported antisocial behavior. For example, Button and colleagues (2007) found that greater exposure to delinquent peers significantly increased overall genetic influences on conduct problems. This same pattern of moderation has also been observed for substance use (Agrawal et al. 2010; Harden et al. 2008) and physical aggression in childhood (Brendgen et al. 2008). Second, the results of two recent studies examining G × Es involving delinquent peer exposure and antisocial behavior across multiple periods of development reported age-graded moderating effects wherein G × Es were observed in adolescence but not early adulthood (Kendler et al. 2011; Samek et al. 2017). Third, the results of another recent study found support for the social context as enhancement model wherein genetic influences on childhood delinquency decreased as delinquent peer affiliation increased (Burt and Klump 2013). Hicks et al. (2009) reported a similar pattern of moderation wherein greater exposure to antisocial peers resulted in significant increases in nonshared environmental influences on internalizing disorders, but additive genetic and shared environmental influences remained stable across levels of peer behavior. This pattern of moderation is consistent with the social context as enhancement model (Shanahan and Hofer 2005).

In addition to studies examining G × Es involving latent genetic influences, a number of studies have also examined whether peer delinquency moderates the influence of measured polymorphisms (or vice versa) on delinquent or antisocial outcomes (Beaver and Holtfreter 2009; Brendgen 2012; DiLalla et al. 2009; Latendresse et al. 2011; Lee 2011; Lu and Menard 2017). These studies have examined G × Es involving deviant peer affiliation and several candidate genotypes including the DRD4 dopamine receptor gene (DiLalla et al. 2009), the muscarinic acetylcholine receptor M2 gene (CHRM2; Latendresse et al. 2011), and the monoamine oxidase A (MAOA) gene (Beaver and Holtfreter 2009; Lee 2011; Lu and Menard 2017). Despite the contributions of these studies, recent methodological innovations have voiced caution in the interpretation of candidate G × E (cG × E) studies (Dick et al. 2015, Duncan and Keller 2011; McGue and Carey 2017) due

to a lack of sufficient statistical power, high false positive rates, and an inability to fully control for the simultaneous occurrence of G $\times$ E and rGE. Based on these limitations, the current study employs a biometric, or latent, G $\times$ E (as opposed to a cG $\times$ E) approach that makes use of twin and sibling pairs.

The Current Study

The current study aims to compare the socialization, selection, and enhancement hypotheses of peer influence within the confines of a genetically-informed modeling strategy utilizing twin and sibling pairs from the National Longitudinal Study of Adolescent to Adult Health (Add Health). While previous studies have directly examined all three etiological hypotheses of peer influence (Gatti et al. 2005), fewer have employed genetically-informed research strategies, allowing for the potential over- or underestimation of the examined associations. This oversight could be important, as failing to consider a more moderate etiological pathway may potentially mask more nuanced processes that ultimately contribute to similarity in delinquent behaviors among peer group members. For example, selection processes could provide preliminary support for the enhancement hypothesis, but failing to directly consider this particular etiological pathway may result in the misinterpretation of such results as evidence in favor of the selection hypothesis.

Other methodological limitations are also present among the genetically informed studies currently comprising the literature including the exclusion of one or more possible hypotheses of peer influence (TenEyck and Barnes 2015), and perhaps even more importantly, a reliance on *subjective* (as opposed to objective) measures of peer behavior (Agrawal et al. 2010; Burt and Klump 2013; Button et al. 2007; Hicks et al. 2009; Kendler et al. 2011; Samek et al. 2017). The generalizability of the remaining studies is limited in that such studies are either focused on behavior in childhood (Brendgen et al. 2005) or on a distinct facet of antisocial behavior like substance abuse (Agrawal et al. 2010; Gillespie et al. 2009; Guo et al. 2015; Harden et al. 2008). The current study aims to address these limitations, makes use of objective measures of peer delinquency and a more comprehensive indicator of delinquent behavior during adolescence.

Methods

Data

The current study analyzed data from the Add Health, a longitudinal, nationally-representative sample of American youth enrolled in middle or high school during the 1994–1995 academic year (Harris et al. 2009). Participants were targeted using a multistage cluster sampling design that resulted in the selection of 132 middle and high schools in the United States. Approximately 90,000 students attending the selected schools and present at the time of data collection completed the in-school portion of the study (Udry 2003). Participants provided information on various topics including health-related behaviors, participation in school activities, and substance use. Participants were also presented with a roster of students enrolled at their school and were asked to identify up to five of their closest male and female friends (up to 10 friends total). Students were also allowed to nominate friends at other schools (Harris 2013). The collection of this

information allowed for the identification of specific peer networks nested within the Add Health sample (Harris 2013; discussed in more detail below).

After the completion of the in-school portion of the study, a subsample of 20,745 students agreed to participate in the in-home portion of the study, which was aimed at collecting more detailed information. The first wave of the in-home portion of the study (i.e., Wave I interviews) was completed in 1995 and included information on a wide range of topics including overall physical and mental health, interpersonal relationships, and behavior. Approximately 1–2 years after the completion of Wave I interviews, nearly 15,000 of the Wave I respondents agreed to participate in the Wave II interviews. The third wave of data collection was completed between 2001 and 2002 and included 15,197 participants from the original in-home sample of youth. The most recent wave of data collection (Wave IV) was completed between 2007 and 2008 when participants were between 24 and 34 years old and included nearly 16,000 of the participants from the Wave I in-home interviews. In total, the Add Health includes information spanning approximately 14 years of the life course and provides insight into a wide range of topics directly related to multiple domains of development (Harris 2011).

Nested within the Add Health sample is a subsample of twin and sibling pairs. Adolescents who participated in the Wave I in-home portion of the study were asked whether they had a co-twin or lived with a sibling. To maximize the number of sibling and twin pairs included in the Add Health study, unrelated pairs, half-siblings, full siblings, and co-twins who lived in the same household as the target participant were also included in the sample. Twin zygosity for same-sex pairs was determined with a confusability index along with the supplemental comparison of 11 genetic polymorphisms (Harris et al. 2006). The final analytic sample was restricted to sibling pairs in which at least one sibling had complete data on the peer deviance measure (discussed in more detail below) and consisted of monozygotic (MZ; $n = 106$ pairs) and dizygotic (DZ; $n = 155$ pairs) twins, full siblings ($n = 445$ pairs), half siblings ($n = 70$ pairs), and unrelated pairs ($n = 45$ pairs), resulting in a final analytic sample of 821 twin and sibling pairs.[3]

Measures

Self-Reported Delinquency

Following the lead of previous studies analyzing the Add Health, self-reported delinquency was measured using 13 items from the Wave II portion of the in-home interviews (Haynie and Osgood 2005; McGloin and Shermer 2009; Thomas 2015). The Wave II measures are used (as opposed to the Wave I measures) in an effort to preserve causal order. Items from the in-school portion of the study were used to construct the peer deviance measures (described in more detail below) and while the Wave I in-home portion of the study was completed after the in-school portion of the study, the delinquency items measured at each

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[3] The decision to include both twin and sibling pairs in the final analytic sample was primarily driven by the desire to obtain acceptable levels of statistical power and variability in the examined peer deviance and self-reported delinquency measures. More specifically, limiting the final analytic sample to MZ and DZ twin pairs with valid information on the study measures would result in a final sample size of $N = 261$ pairs ($n = 106$ MZ pairs and $n = 155$ DZ pairs). In addition, the results of a recent study examining criminal behavior within Swedish population data (twins and siblings born between 1958 and 1991) found that biometric modeling results derived from full and half siblings directly aligned with results from models examining MZ and DZ twin pairs (Kendler et al. 2015). For these reasons, the final analytic sample includes both twin and sibling pairs.

wave were retrospective, tapping behaviors within the past 12 months allowing for potential overlap in responses across the two time points. For this reason, items from the Wave II in-home portion of the study were used to prevent overlap in the peer deviance and self-reported delinquency measures. Participants were asked to report how often they had engaged in various delinquent activities including shoplifting, stealing something worth more or less than $50, participating in a serious physical fight, and using or threating to use a weapon against someone in the past 12 months. Due to a limited amount of variation, the resulting responses for each item were coded dichotomously, where 0 = *none* and 1 = *one or more times*. Self-reported delinquency was estimated as a factor score derived from confirmatory factor analysis (CFA) and was *z*-transformed (mean = 0, standard deviation = 1). The results of the estimated CFA revealed that a single factor solution provided an acceptable fit to the data (comparative fit index [CFI] = .94; Tucker Lewis index [TLI] = .93; and root mean square error of approximation [RMSEA] = .06), with standardized factor loadings ranging between .55 (lying to parents about whereabouts) and .91 (stealing something worth less than $50). Since all the included indicators were dichotomous, the CFA was performed using a weighted least squares estimator with a probit link and robust standard errors (WLSMV). Missing values on the self-reported delinquency measure were handled using a modified version of full information maximum likelihood (FIML) estimation. Univariate statistics for the self-reported delinquency measure, along with all other study measures, are reported in Table 1.

Table 1 Descriptive statistics for all study measures

Study measures	Mean/%	SD/n	Range
Self-reported delinquency			
Wave 2 self-reported delinquency (mean)	−.01	.97	−1.04 to 3.69
Peer deviance			
Send network peer deviance (mean)	−.03	.99	−4.40 to 2.56
Covariates			
Proportion of shared peers (mean)	.05	.11	.00 to .70
Sibling nominated as peer			0 to 1
Yes (%)	12.18	200	
No (%)	87.82	1442	
Age (mean)	16.08	1.67	13 to 21
Sex			0 to 1
Male (%)	48.60	798	
Female (%)	51.40	844	
Race			1 to 3
Caucasian (%)	71.19	1169	
African-American (%)	16.99	279	
All other races (%)	11.81	194	
N	1642		

Peer Deviance

Based on the design features of the Add Health, it is possible to make use of direct or *objective* measures of peer deviance in which peers report on their own behaviors, as opposed to indirect or subjective measures in which participants report on their peers' behaviors. Previous studies have illustrated improved validity when using direct measures as compared to indirect forms of measurement (Meldrum and Boman 2013; Young et al. 2014). Similar to previous studies analyzing the Add Health peer network data (Haynie 2001; McGloin and Shermer 2009; TenEyck and Barnes 2015; Thomas and McGloin 2013), peer deviance measures were created using up to 10 (five male and five female) nominated peers' responses to seven items assessed during the in-school portion of the study and tapping risky and delinquent behaviors over the past 12 months: (1) smoking cigarettes; (2) drinking alcohol; (3) getting drunk; (4) skipping school without an excuse; (5) doing something dangerous on a dare; and (6) lied to parents or guardians; and (7) getting into a physical fight. Responses to the first six items were coded such that $0 = never,$ $1 = once\ or\ twice;$ $2 = once\ a\ month\ or\ less;$ $3 = two\ or\ three\ days\ a\ week;$ $4 = once\ or\ twice\ a\ week;$ $5 = three\ to\ five\ days\ a\ week;$ and $6 = nearly\ every\ day,$ while responses for the physical fighting measure were coded $0 = never;$ $1 = one\ or\ two\ times;$ $2 = three\ to\ five\ times;$ $3 = six\ or\ seven\ times;$ and $4 = more\ than\ seven\ times.$ Responses to the seven items were averaged across all nominated peer group members to create the peer deviance measure. The peer deviance measure was log transformed due to positive skew, and then z-transformed to aid in interpretation.

Covariates

In line with previous research employing similar analytic approaches, age (measured continuously in years), sex ($0 = female;$ $1 = male$), and race ($0 = Caucasian;$ $1 = all\ other\ races$) were included in all estimated models as covariates (Hill et al. 2008; Tuvblad et al. 2006). Additionally, to account for the possibility that siblings from the same household were able to nominate the same individual as a member of their peer group (resulting in overlap in peer groups between siblings), an additional covariate indicating the proportion of overlapping peers was also included in all estimated models ($M = .05,$ $SD = .11$). Finally, since siblings were also able to nominate one another as members of their peer group, a dummy indicator variable was used to distinguish siblings that nominated their co-sibling as a member of their peer group (12.18% of the final analytic sample) from those that did not (87.82% of the final analytic sample).

Plan of Analysis

The current study employed a three-step analytic plan aimed at directly addressing the proposed study goals. The first step in the analysis included the examination of phenotypic and cross-sibling correlations for the peer deviance and self-reported delinquency measures. This stage of the analysis was aimed at examining the extent to which peer deviance and self-reported delinquency are associated and the level of similarity across such measures across various subgroups of sibling pairs. A pattern of findings indicating a positive association between the magnitude of the estimated cross-sibling correlation coefficients and levels of genetic relatedness (i.e., the amount of genetic material shared by sibling pairs) would provide preliminary evidence of a combination of both genetic and environmental influences on the examined measures.

The second step of the analysis involved fitting a series of univariate biometric models (Neale and Cardon 1992; Plomin et al. 2013). These specialized structural equation models (SEM) estimate the proportion of overall variance in the examined peer deviance and self-reported delinquency measures explained by genetic and environmental influences. Consistent with the classic twin model (Neale and Cardon 1992), univariate biometric models decompose the overall variance in peer deviance and self-reported delinquency into three latent factors: additive genetic influences (symbolized as A); shared environmental influences (symbolized as C); and all residual variance, which includes both nonshared environmental influences and measurement error (symbolized as E). In this way, A refers to the sum of the average independent effects of all genes that influence the examined phenotype. C refers to environmental influences that cluster within families and result in greater similarity in the examined trait among sibling pairs. Alternatively, E refers to environmental influences that vary between siblings from the same household and result in greater levels of within-pair phenotypic variance. More in-depth summaries of the differences (both theoretical and analytical) between C and E can be found elsewhere (Turkheimer and Waldron 2000).

A is estimated as the covariance between siblings from the same household on a given measure (e.g., peer deviance). Importantly, the resulting within-pair correlations were constrained in line with additive genetic theory and reflect the proportion of genetic material shared between siblings, with the resulting correlations constrained as follows: $r = 1.00$ for monozygotic (MZ) twins; $r = .50$ for dizygotic (DZ) twins and full siblings; $r = .25$ for half siblings; and $r = .00$ for unrelated pairs. Since shared environmental sources of influence vary between, but not within, families and ultimately result in greater similarities among siblings from the same household, the within pair correlation for shared environmental influences was constrained to 1.00 for all sibling pairs. Finally, since nonshared environmental influences are expected to vary both within and between families and ultimately result in behavioral differences between siblings from the same household, E is estimated as the residual variance in each examined measure. In this way, the resulting latent factor also contains all variance resulting from measurement error.

The third and final step of the analysis was directly aimed at assessing the socialization, selection, and enhancement hypotheses and involved the estimation of a series of modified bivariate Cholesky models that simultaneously estimate rGE and G × E,[4] with the resulting model presented in Fig. 1 (Brendgen et al. 2009; Purcell 2002). As can be seen in the figure, the bivariate Cholesky model expands the univariate biometric model by estimating both common (A_C, C_C, and E_C) and unique (A_U, C_U, and E_U) latent factors, with the former tapping the *covariance* between peer deviance and self-reported delinquency and the latter tapping influences that are unique to self-reported delinquency. The paths labeled a_R, c_R, and e_R are factor loadings for peer deviance on the respective latent factors. The paths labeled a_C, c_C, and e_C are the factor loadings of self-reported delinquency on the common factors, and the paths labeled a_U, c_U, and e_U are factor loadings for self-reported delinquency on the unique factors. In this way, the A_C parameter would reflect additive genetic influences that contribute to the covariance between peer deviance and self-reported delinquency, while the A_U parameter would reflect additive genetic influences that

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[4] This is a necessary extension and common practice when using modified bivariate Cholesky models as Purcell (2002) reported that failing to account for the presence of rGE increases the likelihood of detecting a false positive when testing for the presence of G × E. An undetected rGE may also result in multicollinearity between genetic and environmental influences, effectively increasing the likelihood of a false negative.
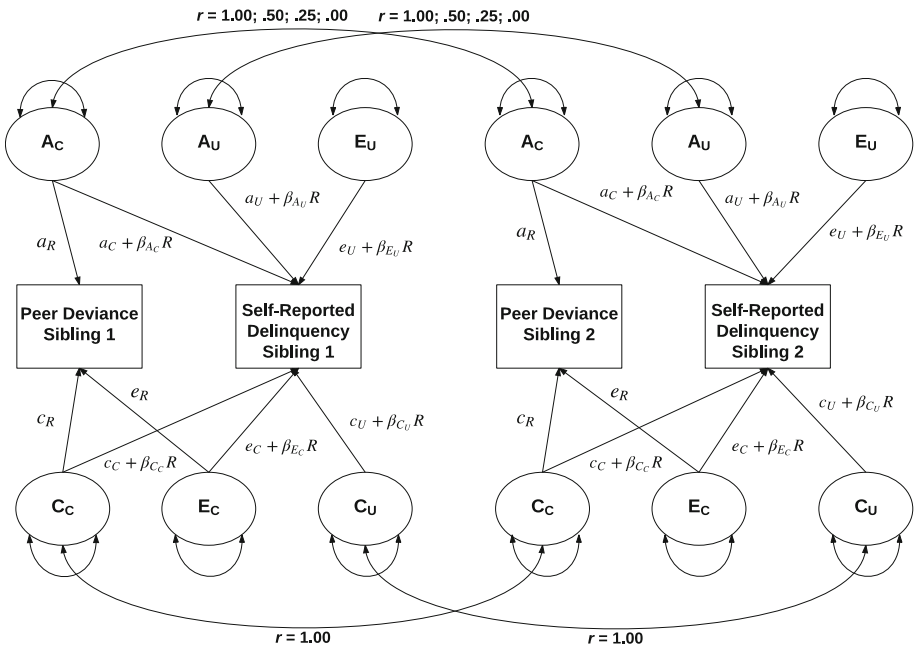
Fig. 1 Bivariate biometric model with interaction terms

are unique to self-reported delinquency (with the additional parameters interpreted similarly). A significant A_C parameter would indicate the presence of an active rGE, and provide support for the selection hypothesis, as genetic influences would contribute to selection into more deviant peer groups along with self-reported delinquency. Alternatively, a nonsignificant A_U parameter would provide preliminary evidence in favor of the socialization hypothesis, as this pattern of results would indicate that any genetic influences involved in the formation of peer groups is unique from genetic and environmental influences that contribute to the development of self-reported delinquency.

As can be seen in Fig. 1, the modified bivariate Cholesky model also includes interaction terms examining the potential moderating effects of peer deviance on genetic and environmental influences on self-reported delinquency. A significant interaction term between A_C and peer deviance ($\beta_{A_C}R$) would indicate that a similar set of genetic influences are implicated in both rGE and G × E. Alternatively, a significant interaction between A_U and peer deviance ($\beta_{A_U}R$) would indicate that genetic influences implicated in rGE and G × E are distinct. The same general interpretation would apply to interaction terms involving shared ($\beta_{C_C}R$ and $\beta_{C_U}R$) and nonshared ($\beta_{E_C}R$ and $\beta_{E_U}R$) sources of environmental influence. In either case, the presence of a significant interaction term would provide evidence in favor of the enhancement hypothesis of peer influence, as such findings would indicate that underlying predispositions and additional sources of environmental influence are effectively moderated by exposure to deviant peers.

All analyses were estimated in M*plus* 7.4 (Muthén and Muthén 2012) using full information maximum likelihood estimation, which estimates values for all cases that include at least partial information on the estimated latent variables and has been found to be an acceptable strategy for handling missing data (Acock 2005). For all analyses, aside from the latent G × E models, model fit was assessed using Satorra-Bentler scaled χ^2, the

comparative fit index (CFI; values .95 or greater indicate a close fit and values .90 or greater indicate an acceptable fit), the Tucker Lewis Index (TLI; interpreted similarly to the CFI), and the root mean square error of approximation (RMSEA; values less than .05 indicate a close fit and values less than .10 indicate an acceptable fit; Hu and Bentler 1999). For models estimating interaction terms that include a latent variable, traditional fit indices such as χ^2 and CFI are not available, but nested models can be compared using a likelihood ratio test (LRT), which is distributed as χ^2 units. In this way, a nonsignificant change in χ^2 would indicate that the estimation of the nested (or more restricted) model would not result in a significant loss of overall model fit (Purcell 2002). Finally, covariates were included in all biometric models as statistical controls.

Results

The first step of the analysis involved the estimation of phenotypic and cross-sibling correlations for the peer deviance and self-reported delinquency measures. The phenotypic (i.e., zero order) correlation between peer deviance and self-reported delinquency was $r = .10$ ($p < .001$), indicating a small, but significant bivariate association in the expected direction (i.e., greater exposure to peer delinquency was associated with greater overall levels of self-reported delinquency). The results of the cross-sibling correlations are reported in Table 2, and revealed that within-pair similarity on the peer deviance measure decreased in a mostly stepwise fashion in line with additive genetic theory, with MZ twins being most similar to one another ($r = .52$, $p < .01$) and half-siblings being the least similar to one another ($r = .16$, $p = .19$), with the cross-sibling correlation among unrelated pairs ranking as the second lowest coefficient ($r = .18$, $p = .23$). A similar pattern of findings emerged for the self-reported delinquency measure and also revealed that MZ twins were most similar ($r = .52$, $p < .01$) and half-siblings were least similar ($r = -.13$, $p = .33$) and the second smallest cross-sibling correlation coefficient was found among unrelated pairs ($r = .00$, $p = .98$). Taken together, these findings provide preliminary evidence of a combination of additive genetic and environmental influences on both peer deviance and self-reported delinquency.

The next step of the analysis involved the estimation of a series of univariate biometric models, the results of which are presented in Table 3. The first set of rows report the results of a univariate biometric model decomposing the variance in the peer deviance measure. The resulting model provided an acceptable fit to the data ($\chi^2 = 108.76(91)$, $p = .10$; CFI = .93; TLI = .93; RMSEA = .03), and indicated that approximately 41% of the overall variance in peer deviance was explained by additive genetic influences, 14% ($p = .08$) was explained by shared environmental influences, and the remaining 46% of the overall variance was explained by nonshared environmental influences (and measurement error). Additional analyses revealed that constraining the C parameter to zero did not significantly worsen overall fit ($\Delta\chi^2 = 3.89(1)$, $p = 1.00$). The results of this trimmed (or best fitting) model largely converged with the full model and indicated that approximately 59% of the overall variance in peer deviance was explained by additive genetic influences with the remaining 41% of the variance explained by nonshared environmental influences. The second set of rows present results from models examining the self-reported delinquency measure. The full model fit the data closely ($\chi^2 = 74.15(91)$, $p = .90$; CFI = 1.00; TLI = 1.00; RMSEA = .00) and indicated that approximately 36% of the overall variance in self-reported delinquency was explained by additive genetic influences and the remaining 64% of the variance was explained by nonshared environmental influences,

Table 2 Descriptive statistics and cross-sibling correlations for peer deviance and self-reported delinquency measures across levels of genetic relatedness

Study measures	Peer deviance	Self-reported delinquency
Monozygotic twin subsample		
Mean (SD)	−.10 (.99)	−.07 (.98)
Cross-twin correlation	.52**	.36**
N (pairs)	106	95
Dizygotic twin subsample		
Mean (SD)	−.02 (.97)	−.05 (.99)
Cross-twin correlation	.47**	.26**
N (pairs)	155	142
Full sibling subsample		
Mean (SD)	−.03 (1.01)	−.01 (.98)
Cross-sibling correlation	.33**	.20**
N (pairs)	445	397
Half sibling subsample		
Mean (SD)	.08 (1.03)	.11 (1.06)
Cross-sibling correlation	.16	−.13
N (pairs)	70	59
Unrelated pairs subsample		
Mean (SD)	.22 (.94)	.05 (1.02)
Cross-sibling correlation	.18	.00
N (pairs)	45	35
Full sample		
Mean (SD)	.00 (1.00)	.00 (1.00)
Cross-sibling correlation	.35**	.19**
N (pairs)	821	728

The phenotypic (i.e., zero order) correlation between peer deviance and self-reported delinquency was $r = .10$ ($p < .001$) for the full sample

**$p < .01$; *$p < .05$; †$p < .10$

while shared environmental influences failed to explain any variance. Constraining the C parameter to zero did not worsen overall model fit ($\Delta\chi^2 = .00(1)$, $p = 1.00$) or result in any change to the parameter estimates.

The third step of the analysis was directly aimed at examining the socialization, selection, and enhancement hypotheses with modified bivariate Cholesky models. Prior to the estimation of the Cholesky models, it is first necessary to examine the extent to which rGE contributes to the underlying association. This preliminary step of the analysis involved the estimation of a series of baseline bivariate Cholesky models. The results of the models examining both common and unique sets of influence on peer deviance and self-reported delinquency are presented in the online supplement and provided evidence of a sizable rGE between peer deviance and self-reported delinquency (rGE = .728, 95% CI = .58–.88, $p < .001$).[5] In addition, the baseline bivariate Cholesky models also revealed a lack of overlap in nonshared environmental influences on peer deviance and

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[5] rGE can be calculated using the following formula and parameter estimates from the baseline Cholesky model (presented in the online supplement):

Table 3 Results from univariate biometric models

	A	C	E	χ^2 (df)	$-2LL$(np)	$\Delta - 2LL$(np)	CFI	TLI	RMSEA
Peer deviance									
Full model	.41**	.14†	.46**	108.76 (91)†	2209.52 (9)	–	.93	.93	.03
	(.13)	(.08)	(.07)						
Best-fitting model	.59**	–	.41**	113.11 (92)†	2211.44 (8)	3.89 (1)	.91	.92	.03
	(.05)	–	(.05)						
Self-reported delinquency									
Full model	.36**	.00	.64**	74.15 (91)	2059.39 (9)	–	1.00	1.00	.00
	(.07)	(.00)	(.07)						
Best-fitting model	.36**	–	.64**	74.96 (92)	2059.39 (8)	.00 (1)	1.00	1.00	.00
	(.07)	–	(.07)						

Standardized parameter estimates presented and reflect the proportion of overall variance explained in each phenotype. All estimated models included controls for proportion of shared peers, siblings nominated as a peer, age, sex, and race. Robust standard errors presented in parentheses. Final sample sizes: peer deviance models ($n = 821$ pairs); self-reported delinquency models ($n = 728$ pairs)

$-2LL$ = log-likelihood, CFI = comparative fit index, TLI = Tucker–Lewis index, RMSEA = root mean error of approximation

**$p < .01$; *$p < .05$; †$p < .10$

self-reported delinquency, indicating that the remaining variance in self-reported delinquency not explained by genetic influences was explained by unique sources of nonshared environmental influence (and measurement error).

The results of the final step of the analysis, which estimated an additional set of Cholesky models that included interaction terms, are presented in Table 4. The results of the baseline model revealed a nonsignificant E_C parameter ($E_C = -.09$, $p = .19$) and accompanying interaction ($\beta_{E_C} = -.06, p = .22$), and constraining both parameters to zero did not significantly worsen overall model fit ($\Delta\chi^2 = 2.36(2)$, $p = .31$). Additionally, the results of the baseline bivariate Cholesky models (presented in the online supplement) indicated that the E_C parameter was problematic and constraining this parameter to zero did not worsen overall fit ($\Delta\chi^2 = .00(1)$, $p = 1.00$). Based on this pattern of findings, the E_C and β_{E_C} parameters were fixed to zero in the final, best-fitting model. The results of the best fitting model indicate that genetic influences unique to self-reported delinquency decrease as peer deviance increases ($\beta_{A_U} = -.16$, $p = .03$). Alternatively, nonshared environmental influences that were unique to self-reported delinquency ($\beta_{E_U} = .12$, $p = .003$) increased as levels of peer deviance increased. Genetic influences that were common to both peer deviance and self-reported delinquency were also significantly moderated by exposure to peer deviance, but the resulting interaction term was positive indicating that genetic influences increased as exposure to peer deviance increased ($\beta_{A_C} = .06$, $p = .04$).

Despite the positive and significant β_{A_C} term, the overall pattern of moderation indicated that genetic influences on self-reported delinquency decreased (and nonshared

Footnote 5 continued

$$rGE = \frac{a_R a_c}{\sqrt{a_R^2(a_C^2 + a_U^2)}} = \frac{.74(.34)}{\sqrt{.74^2(.34^2 + .32^2)}} = .728$$

environmental influences increased) as exposure to peer deviance increased. This pattern of results is primarily driven by the size of the β_{A_U} parameter relative to the size of the β_{A_C} parameter. Since the former was larger in magnitude than the latter, the net moderating effect is negative. To aid in the interpretation of the interaction models, the results are also presented in Fig. 2. As can be seen, the variance in self-reported delinquency explained by genetic and nonshared environmental influences varies across levels of peer deviance such that genetic influences on self-reported delinquency are greatest when paired with overall

Table 4 Results from bivariate models including interaction terms

	A	β_A	E	β_E	$-2LL(np)$	$\Delta - 2LL(np)$
Full model					4150.22 (25)	
Common effects	.24**	.10*	−.09	−.06		
	(.06)	(.04)	(.07)	(.05)		
Unique effects	.35**	−.16†	.83**	.09†		
	(.06)	(.08)	(.03)	(.05)		
Best fitting model					4151.34 (23)	2.36 (2)
Common effects	.17**	.06*	–	–		
	(.04)	(.03)	–	–		
Unique effects	.35**	−.16*	.85**	.12**		
	(.06)	(.07)	(.03)	(.04)		

Unstandardized parameter estimates presented. All estimated models included controls for proportion of shared peers, siblings nominated as a peer, age, sex, and race. Robust standard errors presented in parentheses

$**p < .01$; $*p < .05$; $^\dagger p < .10$
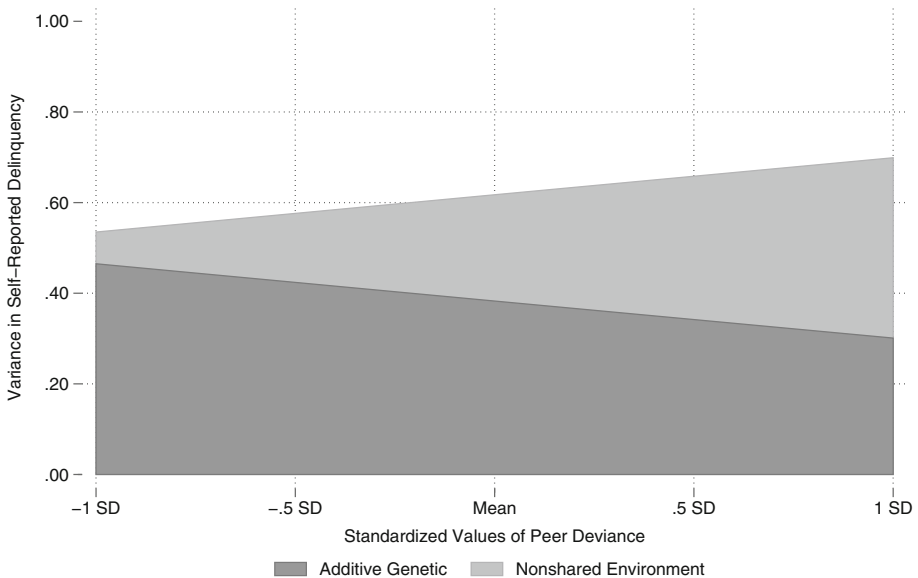
Fig. 2 Results from bivariate interaction models. All estimated models included controls for proportion of shared peers, siblings nominated as a peer, age, sex, and race

lower levels peer deviance (e.g., values below the mean) and lowest when paired with overall greater levels of peer deviance (e.g., values above the mean). The exact opposite pattern is observed for nonshared environmental influences on self-reported delinquency, which explain less variance when peer delinquency is relatively low and more variance as overall levels of peer delinquency increase.

Sensitivity Analyses

A series of sensitivity analyses were also estimated to examine the robustness of the findings reported in the primary analysis. The results of the sensitivity analyses are presented in the online supplement. First, to ensure that the findings were not sensitive to the employed self-reported delinquency measure, an alternative measure in which all 13 Wave II delinquency items were summed to create a variety index was substituted for the factor score employed in the primary analyses. The resulting models revealed a similar pattern of results in which genetic influences explained approximately 37% and nonshared environmental influences explained the remaining 64% of the variance in the variety index. The results of the bivariate Cholesky models were also similar to the findings from the primary analysis and revealed that as exposure to peer deviance increased, genetic influences on self-reported delinquency decreased ($\beta_{A_U} = -.21$, $p = .005$) and nonshared environmental influences increased ($\beta_{E_U} = .16$, $p < .001$).

The second set of sensitivity analyses included an alternative measure of peer deviance. More specifically, the peer deviance measure employed in the primary analyses included individuals (up to 10 peers in total) nominated by the target respondent and is referred to as the send peer network deviance measure. An alternative measure, referred to as the send-receive peer network deviance measure, was created using scores from peer group members who were either nominated by the target participant *or* nominated the target participant, resulting in a potentially broader peer group as well as increased statistical power ($N = 1054$). Once again, the overall pattern of findings was similar to those from the models employed in the primary analyses. The univariate biometric model decomposing the variance in the send-receive peer deviance measure indicated that genetic influences explained 71% of the variance with nonshared environmental influences explaining the remaining 29% of the variance. The results of the bivariate Cholesky models were also similar to those employed in the primary analyses with one minor exception. While genetic influences unique to self-reported delinquency decreased ($\beta_{A_U} = -.18$, $p = .008$) and nonshared environmental influences unique to self-reported delinquency increased ($\beta_{E_U} = .85$, $p < .001$) as exposure to deviant peers increased, common genetic ($\beta_{A_C} = -.12$, $p = .001$) and nonshared environmental ($\beta_{E_C} = .20$, $p < .001$) influences were also significant and trended in the same direction. This pattern of results provides additional evidence of the general pattern of moderation detected in the primary analyses, genetic influences decreased and nonshared environmental influences increased as exposure to peer deviance increased.

Discussion

The role of peers in the etiological development of delinquency has been a topic of interest within criminology for decades and has culminated into a substantial literature aimed at identifying the mechanisms connecting peers and delinquency (Hoeben et al. 2016). While

findings from the classic literature tend to represent more extreme ends of the theoretical continuum, with studies citing evidence of both the socialization (Akers 1973; Burgess and Akers 1966; Sutherland 1947; Sutherland and Cressey 1955) and selection hypotheses (Hirshi and Gottfredson 1987, 2000; Gottfredson and Hirschi 1990), recent studies have revealed a more nuanced pattern of results in which socialization and selection processes are better integrated, effectively providing evidence of the enhancement hypothesis of peer influence (Haynie et al. 2014; Matsueda and Anderson 1998; Samek et al. 2016; Wright et al. 1999). In light of these findings, the current study aimed to compare all three hypotheses within the context of contemporary developmental theory (Lerner and Castellino 2002) by making use of a sample of sibling pairs from the Add Health to examine the role of both genetic and environmental influences on the association between peer deviance and self-reported delinquency. The results of the biometric models revealed three key findings, all of which warrant additional explanation.

First, the results of the biometric models failed to provide support for a "pure" or "hard-lined" version of the socialization hypothesis. The results of the univariate biometric models revealed that a significant portion (ranging between 41 and 59%, depending on the examined model) of the variance in peer group deviance was explained by genetic influences unique to the examined *respondents*. While this finding may seem counterintuitive at first glance, it is a preliminary indicator of selection stemming from genetically-influenced traits. In other words, peers with similar traits (which are genetically influenced to some extent) congregate, resulting in the formation of peer groups. This finding directly aligns with a series of studies from Christakis and Fowler (2014; Fowler et al. 2011), indicating that members of peer groups share a greater number of genetic variants, and, in turn, lower overall levels of within-group variance across behavioral phenotypes relative to individuals paired at random. In this way, selection processes that result in similarity between group members are, at least to some extent, genetically influenced. This finding fails to support a "hard-lined" version of the socialization hypothesis in that internalized sources of influence are involved in the development of peer groups, indicating that peer group members are typically more similar to one another *prior to* the transfer of any social influence.

Second, the results of the modified bivariate Cholesky models found preliminary support for the selection hypothesis. More specifically, the results revealed that a significant proportion of the covariance between peer deviance and self-reported delinquency was explained by genetic influences (i.e., a significant A_C parameter). In addition, using the parameter estimates from the baseline bivariate Cholesky model (results presented in the online supplement), the rGE between peer deviance and self-reported delinquency was moderately sized (.728), providing preliminary evidence for the selection hypothesis. Importantly, these findings align with previous studies identifying significant genetic influences on affiliating with delinquent peers (Agrawal et al. 2010; Kendler et al. 2007; Samek et al. 2013) as well as the association between peer behaviors and antisocial behaviors (Connolly et al. 2015; Hill et al. 2008; Samek et al. 2017). This pattern of results indicates that the overlap in genetic influences that collectively contribute to selection into peer groups and delinquency are not only associated, but also play an important role in both processes. While a similar pattern of results has been recognized in previous studies (Barnes et al. 2014; Connolly et al. 2015; Hill et al. 2008; TenEyck and Barnes 2015), future research would benefit from the continued integration of genetically informed modeling strategies when examining associations involving peer influences.

Third and finally, while the preceding findings provided preliminary evidence in favor of the selection hypothesis, the results of the G × E models provided convincing support for the enhancement hypothesis of peer influence. More specifically, as peer group

deviance increased, environmental influences on self-reported delinquency also increased, effectively overshadowing underlying genetic influences. This pattern of results is consistent with Shanahan and Hofer's (2005) enhancement model as well as the bioecological model proposed by Bronfenbrenner and Ceci (1994) such that as proximal processes increase in magnitude (i.e., peer deviance decreases) genetic influences on antisocial behavior increase and environmental influences decrease. Alternatively, as proximal processes become weaker (i.e., increased levels of peer deviance), genetic influences decrease and are overpowered by adverse environmental influences. While the bioecological model represents only a single pattern of G × E influences (see the online supplement for a brief summary of alternative models), the results of the current study seem to conform most closely to this particular hypothesis.

While previous studies have reported a similar pattern of moderation (Burt and Klump 2013; Hicks et al. 2009), the majority of previous studies have reported moderation patterns that are more directly aligned with the diathesis stress model and include a simultaneous increase in genetic and environmental influences (Agrawal et al. 2010; Button et al. 2007; Brendgen et al. 2008; Harden et al. 2008) and age-graded patterns with moderating effects confined to a specific stage of the life course (Kendler et al. 2011; Samek et al. 2017). These discrepancies may be the result of methodological differences across studies. For example, the majority of previous studies that have examined the potential moderating influence of peer deviance on self-reported forms of antisocial behavior have employed subjective indicators of peer behavior (Agrawal et al. 2010; Burt and Klump 2013; Button et al. 2007; Hicks et al. 2009; Kendler et al. 2011; Samek et al. 2017), examined more restrictive forms of antisocial behavior (Agrawal et al. 2010; Gillespie et al. 2009; Guo et al. 2015; Harden et al. 2008), or have examined behavior confined to childhood (Brendgen et al. 2005). In this way, the pattern of moderation detected in the current study may be unique to the methodological parameters employed and the demographic characteristics of the Add Health sample. Future research would benefit from a closer examination of the manner in which such factors effectively moderate findings from G × E research.

The pattern of moderation detected in the current study also diverges from patterns observed in cG × E studies, particularly those examining the MAOA genotype (Beaver and Holtfreter 2009; Lee 2011; Lu and Menard 2017). More specifically, several studies have reported a significant G × E in which the high-activity allele of the MAOA gene and exposure to delinquent peers increases the likelihood of engaging in fraudulent behaviors (Beaver and Holtfreter 2009), serious criminal behavior (Lu and Menard 2017) and more general forms of antisocial behavior (Lee 2011). These findings are somewhat surprising since low MAOA activity has been more directly implicated in the development of antisocial behaviors (for a recent meta-analysis see Ficks and Waldman 2014), particularly when paired with adverse social environments (Byrd and Manuck 2014). In this way, the findings from the current study, which illustrate a decrease in genetic influences as peer deviance increases, do not align with previous cG × E studies. While only speculation, this discrepancy in findings may be a result of some of the methodological limitations that accompany such studies including limited statistical power, high false positives, and perhaps, most importantly within the context of the current study, an inability to fully control for the simultaneous occurrence of rGE and G × E (Dick et al. 2015, Duncan and Keller 2011; McGue and Carey 2017). More specifically, while it is possible to test for an rGE involving the examined environmental influence (e.g., peer deviance) and genotype (e.g., MAOA), cG × E studies are typically not able to address rGEs involving the examined environmental influence and additional genetic influences. One of the primary

advantages of the latent G × E approach applied in the current study is that since this approach takes into account all observed variance in both the environmental influence and the examined outcome, it allows for a more comprehensive estimation of both rGE and G × E (Purcell 2002). This is an important extension, as previous simulation studies have indicated that failing to sufficiently account for the simultaneous occurrence of rGE and G × E may increase the likelihood of detecting false positives when testing for the presence of G × E (Purcell 2002). In addition, an undetected rGE may also result in multicollinearity between genetic and environmental influences, effectively increasing the likelihood of a false negative. Despite these limitations, the continued use of cG × E approaches may still be appropriate in some context (for a comprehensive overview see Dick et al. 2015) and should be explored in more detail in future research.

Despite these contributions to the existing literature, the current study is not without its own limitations. First, while the current study relied on an objective measure of peer deviance, the measure was limited to relatively minor forms of delinquency like substance use and truancy. While previous studies analyzing the Add Health have employed similar measures (Haynie 2001; McGloin and Shermer 2009; TenEyck and Barnes 2015; Thomas and McGloin 2013), future research would benefit from the use of a more comprehensive measure. The decision to examine peer deviance using items from the in-school survey was aimed at accomplishing two objectives: (1) maintaining temporal order; and (2) capturing information on as many peer group members as possible. In regard to the first objective, peer deviance reported during the in-school portion of the study reflected peer behaviors in the year prior to the data collection period, while self-reported delinquency was measured during Wave II of the in-home portion of the study and reflected behaviors for the previous 12 months. In this way, the resulting peer deviance measure reflects peer behaviors that occurred prior to the self-reported delinquency measures. In regard to the second objective, all students who participated in the in-school portion of the study (approximately 90,000 in total) were eligible for inclusion in identified peer groups so long as they were identified as a member of each target individual's peer group. Alternatively, the in-home portion of the study included information on a broader range of delinquent behaviors, but due to the limited number of students sampled from each school, the resulting possible pool of peer group members available for nomination was significantly truncated, resulting in excessive missingness. This is primarily due to the fact that a sizable portion of students that participated in the in-home portion of the study nominated peers who were not included in the same portion of the study, resulting in missing data.

Second, and directly related, the current study examines peer deviance at a single time point and self-reported delinquency subsequently, but due to the limited amount of time that elapsed between each wave of data collection, the reported findings only reflect the association between peer and self-reported delinquency during a single period of development—adolescence. While adolescence may represent a developmental period in which peer influence becomes more salient (Gardner and Steinberg 2005; Harris 2009), additional research has recognized the importance of examining peer influence at other stages of development (Brendgen et al. 2009; Warr 2002). In addition, calls from developmental criminology, including a recent Sutherland Address from Cullen (2011), have urged researchers to focus on periods of development other than adolescence. Future research would certainly benefit from examining associations between peer deviance and self-reported delinquency at additional time points. For example, the results of two recent studies indicate that the potential moderating effects of peer delinquency on externalizing behavior problems varies over different developmental periods (Kendler et al. 2011; Samek et al. 2017). Despite the usefulness of these findings, both previous studies relied on

subjective measures of peer deviance, leaving open the possibility for a different pattern of findings when employing an objective measure.

Despite these limitations, the results of the current study contribute to a growing body of literature suggesting that the manner in which peer influences contribute to individual behavioral trends are likely far more complex than previously thought. In addition, theoretical perspectives that adopt "hard-lined" versions of the socialization and selection hypotheses appear to be incomplete. Growing evidence indicates that both processes are intricately involved in the association between peer and self-reported delinquency (Guo et al. 2015; Haynie et al. 2014; Samek et al. 2017; Snijders and Baerveldt 2003), effectively providing support for the enhancement hypothesis of peer influence. In light of the findings from the current study, we echo Haynie et al.'s (2014) observation that "selection and influence [or socialization] perspectives need not be opposed to one another, but they can complement each other and operate together to explain peer–delinquency homophily" (p. 713).

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