EXAMINING THE IMPACT OF PEER GROUP SELECTION ON SELF-REPORTED DELINQUENCY

A Consideration of Active Gene–Environment Correlation

MICHAEL TENEYCK
J. C. BARNES
University of Cincinnati

Research has yet to discount all sources of confounding in the relationship between an individual's delinquent behavior and that of his or her peers. One approach is to control for an active gene–environment correlation (*r*GE). Active *r*GE occurs when one selects into an environment based on genetic propensities. The current study utilizes twin data from the National Longitudinal Study of Adolescent Health to examine the impact of a direct measure of peer delinquency on self-reported delinquency while controlling for active *r*GE. The final analytic sample ranged between 456 and 524 dizygotic and 286 and 350 monozygotic twins, depending on the measures being analyzed. Using an augmented version of the DeFries–Fulker model, results revealed the peer effect was no longer statistically significant once genetic confounding (active *r*GE) was controlled. These findings support selection arguments and run counter to learning theory explanations.

Keywords: peer effect; peer delinquency; social learning; selection; genetic effects

There has been a great deal of research examining the role peers play in the etiology of delinquent and antisocial behavior. Indeed, a large body of work has revealed a strong correlation between group behavior and the behaviors of individual members of the group. Scholars have shown, for instance, that peers tend to resemble one another on things such as drug use, sexual behaviors, and delinquency (Pratt et al., 2010). In addition, research has

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revealed that specific behavioral outcomes such as alcohol consumption, obesity, depression, and smoking all spread through social networks, hinting that group dynamics have a causal influence on the behaviors of individual group members (Christakis & Fowler, 2007, 2008; Rosenquist, Fowler, & Christakis, 2011; Rosenquist, Murabito, Fowler, & Christakis, 2010). Based on these findings, there is little doubt that individuals resemble their peers on a range of characteristics. The association has been so robust and consistent that it has become a mainstay in criminological research, though the most popular interpretation of the peer effect (that the causal arrow flows from group to individual behavior) remains tenuous and shrouded by debate (Young, Rebellon, Barnes, & Weerman, 2013a). Two issues with the available research have been the subject of recent discourse: (a) the measurement of peer delinquency (Young, Rebellon, Barnes, & Weerman, 2013b) and (b) the impact of self-selection on the peer effect (Weerman, 2011; Young et al., 2013a).

Debate over how to measure peer delinquency has spread over the past decade (Haynie, 2002). Traditionally, researchers have relied on respondent-reported peer behavior, known as the "indirect" method (Akers, 1998; Warr & Stafford, 1991). One classic study employing the indirect method was conducted by Warr and Stafford (1991). Using data obtained from the National Youth Survey (NYS), these authors measured peer behavior by asking the target respondent how many times their friends had participated in certain delinquent acts in the past year. Indirect measures of peer delinquency such as the one used by Warr and Stafford (1991) are obtained via the focal respondent and, therefore, may be subject to projection bias (Gottfredson & Hirschi, 1990). To avoid this potential pitfall, many scholars have begun using the "direct" or "social network" method of measuring peer delinquency. Using this method, respondents identify their peers, and measures of delinquency are then obtained directly from the peers (i.e., peers report on their own behavior; Haynie, 2002; Meldrum, Young, & Weerman, 2009; Young, Barnes, Meldrum, & Weerman, 2011). Researchers have advocated for measuring peer delinquency in this fashion by arguing that direct measures avoid the projection bias and, therefore, provide a more accurate view of the peer groups' actual level of involvement in delinquency. The extent to which the target respondent's self-reported delinquency correlates with direct measures of the peers' delinquency has, however, received relatively little attention (Boman, Strogner, Miller, Griffin, & Krohn, 2011; Haynie, 2001, 2002; Meldrum et al., 2009), representing an important area for future work. The current study will consider the impact of a direct measure of peer behavior on self-reported delinquency among a large sample of American youth.

The second topic of debate in the peer delinquency literature is a classic one that, while not unique to this line of research, is particularly salient: the debate over causality versus selection. Some researchers have argued that delinquent individuals self-select into deviant peer groups, thereby offering an alternative explanation of the observed correlation between a respondent's behavior and that of his or her peers (Gottfredson & Hirschi, 1990). If youth who are more inclined toward delinquency seek out the companionship of similar others, then the extent to which peers cause future delinquency becomes cloaked by endogeneity concerns. Some scholars maintain that peers have a causal impact on one another's behavior even though selection effects may bolster the observed correlation between the individual and the group (Akers, 1998). The debate over causality versus selection has thrived for at least six decades, with little headway being made by either side (Glueck & Glueck, 1950). The current study will present an analysis that addresses this issue and offers insight from a

new perspective: behavioral genetics and active gene–environment correlation (*r*GE). Prior to doing so, we will introduce the bedrock of literature, both theoretical and empirical, upon which the current study builds.

THEORETICAL DISCUSSION OF THE PEER EFFECT

Numerous theories have been proffered to explain the correlation between an individual's behavior and that of his or her peers. Three of the most prominent explanations are differential association (Sutherland, 1947), social learning theory (Akers, 1998), and group socialization theory (J. R. Harris, 1998). Because each of these theories builds on prior work, it is useful to consider them in chronological order. As we review the theories, however, note that each posits the peer group will affect the individual, even in light of known selection effects.

DIFFERENTIAL ASSOCIATION AND SOCIAL LEARNING THEORY

Differential association and social learning theories both acknowledge that individuals learn from their peers and these theories attempt to explain the learning process. Sutherland (1947) suggested that criminal behavior is learned much like any other behavior; mainly, the individual is exposed to a behavior and then imitates the behavior. Sutherland also argued that people learn definitions favorable to crime through interactions with others primarily within intimate personal groups (e.g., friends and family). Drawing from psychological explanations of learning, namely operant conditioning (Skinner, 1953), Burgess and Akers (1966; and later, Akers, 1998) extended Sutherland's (1947) theory, developing what is known as social learning theory. While this theory retained the differential association processes of Sutherland's theory, Burgess and Akers's social learning theory expanded on Sutherland's work by discussing three additional concepts: differential definitions, imitation, and differential reinforcement. Differential definitions are transferred from peers to the individual and can take on a variety of forms, ranging from prosocial to antisocial. Imitation, which involves the observation and repeating of others' behavior, can depend on the characteristics of the model, the behavior itself, and the resulting consequence of the behavior. The last point brought to bear by Akers (1998) centers on differential reinforcement. Differential reinforcement highlights the point that both the actual and anticipated costs and rewards of a given behavior are important for determining whether the behavior will be repeated. In short, these theories propose that individuals learn delinquency from their peers (i.e., the peer effect of delinquency goes in one direction, from the peer group to the individual), and to the extent that selection occurs, it is not enough to fully account for the observed level of peer similarity.

GROUP SOCIALIZATION THEORY

J. R. Harris (1995, 1998) proposed a theory of group socialization, focusing mainly on how peer influences shape an adolescent's personality development. The author challenged prior parenting literature that argued parents were the predominant force in determining a child's personality by stating:

> Experiences in childhood and adolescent peer groups modify children's personalities in ways they will carry with them to adulthood. Group socialization theory makes this prediction: That

> children would develop into the same sort of adults if we left their lives outside the home unchanged—left them in their schools and their neighborhoods—but switched all the parents around. (J. R. Harris, 1998, p. 339)

Drawing on research from evolutionary psychology, J. R. Harris (1998) makes the compelling argument that throughout our history as a species we have relied on the peer group to socialize the adolescent to become a functioning adult. She argues that even now, when children are out of the home, which is where adolescents spend most of their time, they identify with and assimilate into a group. This requires that the child take on the group's attitudes and behavioral norms. As a result, it is reasonable to assume that children are still mainly socialized by their peers. It is their identification with this group that shapes their environment and is responsible for environmental influences on the adolescent's personality characteristics. Adolescent peer groups create their own culture by selecting or rejecting various characteristics of the adult culture and making cultural advances of their own. As the child moves through this sequence of peer cultures, they become more similar to their peers in some ways, yet simultaneously different in other ways.

J. R. Harris (1998) also relied on the tenants of evolutionary psychology to aid in explaining how adolescents act differently across situations, often referred to as code switching (Anderson, 1999) or context-specific socialization. For example, an adolescent may be the class clown at school, the smart kid on the street corner, and the quiet child at home. As the child develops and gets older, the "outside-the-home behavior system" begins to take priority over the "inside-the-home behavior system" and becomes an integral part of the personality. The child's niche-picking (i.e., peer group selection) eventually aids in forming their adult personality. In short, J. R. Harris notes the importance of selection effects but maintains that peer effects are the predominant factor affecting personality development in adolescence and adulthood.

To be sure, J. R. Harris's theory informs traditional learning theories in two crucial ways. First, J. R. Harris (1995, 1998) adds a genetic component to the effect of peer behavior on one's own behavior by discussing niche-picking. Specifically, children are born with certain temperaments and will seek out niches within their group that fit their genetic predispositions. This is known as active *r*GE. J. R. Harris refers to this process as the indirect effects of genes or "the effects of the effects of the genes" (J. R. Harris, 1998, p. 28). J. R. Harris also notes that as children get older they have the freedom to choose their own peer group. These individuals may belong to the same peer groups or cliques because they have similar attitudes. Once in these roles, the individual's characteristics tend to become more exaggerated. J. R. Harris (1998) solidifies this when discussing aggression:

> As birds of a feather flock together, aggressive teens and those who are attracted to excitement and danger find others like themselves. Such personality characteristics are partly genetic, so when kids seek out other kids who are similar to themselves, to some extent they are seeking out those with similar genes. (pp. 267-268)

In other words, a smart child will get smarter, an athlete will become more athletic, and a delinquent will become more delinquent.

The second way J. R. Harris's theory informs traditional learning theories is by incorporating contextual effects in which individuals change their behavior depending on where they are, who is present, and with which group they identify. J. R. Harris (1998) argues that

"most children have at least two distinct environments: the home environment and the world outside the home. Each has its own rules of behavior, its own punishments and payoffs" (p. 55). The author points out that a child cannot expect the same response from their parents as they receive from their peers. For instance, if a child cries at home they may be coddled, but if they cry on the playground they will be ridiculed. Therefore, an individual may be the dominant sibling at home but submissive in their peer group. In addition, J. R. Harris suggests that if there is any correlation between a child's behavior in the home and out of the home, or between different contexts, it would be due to the child's genetic predisposition (i.e., the direct and indirect effects of genes). In short, J. R. Harris's group socialization theory would predict that delinquent individuals, because of genetic predispositions, will be more likely to join delinquent peer groups. Once in these peer groups, learning still occurs and behavioral tendencies may be exacerbated or blunted.

EMPIRICAL TESTS OF THE PEER EFFECT

The effect of peer behavior on one's own delinquency has received an impressive amount of attention from scholars (Cao, 2004; T. Fowler et al., 2007; Pratt et al., 2010; Thomas & McGloin, 2013). Peer effects are usually tested by examining one or more of the tenets of social learning and differential association theories. Some aspects of each theory have been found to be more salient predictors of delinquent behavior than others. Nonetheless, the overarching conclusion to be drawn is that the correlation between peer delinquency and personal delinquency has been supported by empirical research.

In a recent meta-analysis of 133 empirical studies published between 1974 and 2003, Pratt et al. (2010) concluded that the effect sizes for differential association predictors were large and that peer behavior was a strong and significant correlate of personal delinquent behavior. Thus, the results demonstrated that measures tapping aspects specified by social learning theory have received general support in the literature. Pratt et al. (2010) suggest that the strong evidence for differential association measures may be partially attributable to the fact that most studies used indirect measures of peer delinquency. Had the studies included in the meta-analysis used direct measures of peer delinquency, the effect sizes may have been smaller, a point that has been the topic of recent inquiry (e.g., Haynie, 2001; Meldrum & Boman, 2013).

For example, Weerman and Smeenk (2005) analyzed a sample of 1,978 Dutch youth from age 12 to 16 years to examine the association between an individual's delinquency and that of their friends (i.e., peers). In comparing the indirect method of measuring peer delinquency with the direct method, Weerman and Smeenk (2005) noted that employing the latter method resulted in larger percentages of offending and higher mean scores, demonstrating that respondents often underestimate their peer's delinquency when measured via the indirect method (see also Young et al., 2011). Furthermore, Weerman and Smeenk (2005) stated that using indirect measures might seriously overestimate the similarity between respondents and their peers. More recent studies have found similar results, concluding that the relationship between peer delinquency and one's own delinquency decreases significantly when a direct measure is used (Boman & Gibson, 2011; Meldrum et al., 2009). These findings provided the motivation for the current study to rely on the direct method of measuring peer delinquency as relatively few studies have utilized this method within a genetically sensitive research design.

SELECTION VERSUS CAUSATION

One of the most debated issues in the social learning literature is whether the correlation observed in prior research is due to individuals selecting into peer groups or whether it is due to the influence of those peers (Akers, 1977; Hirschi, 1969; Steglich, Snijders, & Pearson, 2010; Sutherland, 1947). Still other researchers maintain that both learning and selection processes occur (Matsueda & Anderson, 1998; Warr, 2002). If individuals select into peer networks, it is likely that a number of different characteristics will be targeted for selection (Weerman, 2011). For instance, research has often shown that the relationship between peer delinquency and one's own delinquency is robust to different modeling strategies (Pratt et al., 2010). However, research has yet to rule out many sources of confounding (i.e., selection effects) between the two key variables (i.e., peer and personal behavior), making the interpretation of available evidence tenuous.

Scholars have recently begun to apply stochastic actor-based models to social network data in an attempt to address the selection versus causation question. Stochastic actor-based models (see Snijders, 2005) use a continuous-time Markov chain Monte Carlo estimator to model the most probable sequence of individual events explaining the total change in a social network between two or more measurement periods (Mathys, Burk, & Cillessen, 2013). The method does this by controlling for influences such as homophilic selection based on age, race, and gender (Snijders, 2005; Snijders, van de Bunt, & Steglich, 2010; Steglich et al., 2010). Scholars have used this method to examine the selection and socialization effects on substance use, nonsuicidal self-injury, happiness, marijuana initiation, and emergence of alcohol use (de la Haye, Green, Kennedy, Pollard, & Tucker, 2013; Giletta, Burk, Scholte, Engels, & Prinstein, 2013; Mathys et al., 2013; Osgood et al., 2013). This approach offers a unique way to capitalize on longitudinal network data in an attempt to simulate changes in the network structure over time. Nonetheless, this modeling strategy may not be able to address all sources of selection, especially those that are unobserved such as genetic factors.

*r*GE

Considering that individuals often select into certain environments, and building on modern behavioral genetic research as well as J. R. Harris's (1998) theory, the idea that environmental variables such as peer group exposure are influenced by genetic factors becomes a possibility that must be entertained. To understand how this could be the case, it is necessary to introduce the concept of *active* r*GE* (see Scarr, 1992; Scarr & McCartney, 1983). To understand active *r*GE, consider the impact of "niche-picking" on peer group selection processes. Specifically, it is commonly known that people choose environments or niches that suit their temperaments (McPherson, Smith-Lovin, & Cook, 2001). That niche-picking may be partially directed by genetic factors is the central point of active *r*GE discussions (Scarr & McCartney, 1983). Personality is highly heritable (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990; Turkheimer, 2000), and if one chooses environments that suit his or her personality, the genetic influences will have an indirect effect on environmental variables via their impact on personality development.

Findings from a recent study by Christakis and Fowler (2014) will aid in making these points more clear. Specifically, data drawn from a large pool (more than 400,000) of single

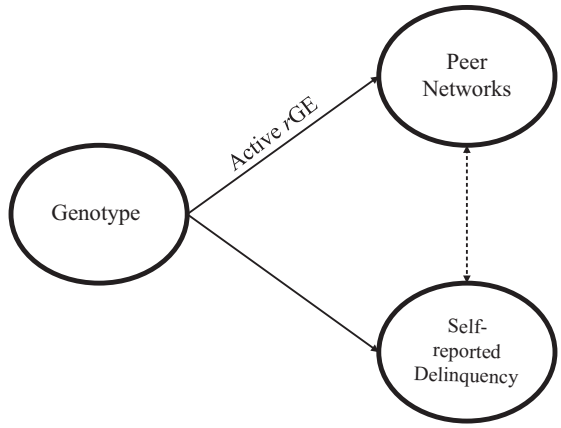

Figure 1: Diagram of Active *r*GE With Respect to the Peer Effect
Note. The figure represents the hypothesized relationship between peer network influences and self-reported delinquency. *r*GE = gene–environment correlation.

nucleotide polymorphisms (SNPs) in nearly 2,000 individuals was analyzed to determine whether peers tended to share more genetic material than randomly paired strangers. If so, evidence in favor of active *r*GE would be found. Christakis and Fowler (2014) summarized the findings of their study by stating,

> Pairs of friends are, on average, as genetically similar to one another as fourth cousins, which seems noteworthy because this estimate is above and beyond mean ancestry and background relatedness. Acquiring friends who resemble oneself genotypically from among a group of strangers may reflect a number of processes, including the selection of particular friends or particular environments. (p. 5)

In short, evidence of active *r*GE was found.

When examining the effects of peer delinquency on one's own delinquency, active *r*GE could aid in identifying potential confounding influences. This is because an individual's genotype might affect both an individual's selection of peers (Christakis & Fowler, 2014; Yun, Cheong, & Walsh, 2011) as well as his or her own level of self-reported delinquency (Beaver, Wright, & DeLisi, 2008; Burt, 2009a, 2009b; Rhee & Waldman, 2002). Recent evidence suggests a significant portion of the variance in peer network deviance is attributable to genetic factors (Beaver et al., 2009; Cleveland, Wiebe, & Rowe, 2005; Harden, Hill, Turkheimer, & Emery, 2008), that an individual's genotype predicts exposure to peer deviance (Beaver, DeLisi, Vaughn, & Barnes, 2010; Beaver et al., 2008), and that genotypes cluster in peer networks (Barnes, Beaver, Young, & TenEyck, 2014; Christakis & Fowler, 2014; J. H. Fowler, Settle, & Christakis, 2011; but see Boardman, Domingue, & Fletcher, 2012). Because all behavior is likely to be under some genetic influence (Turkheimer, 2000), controlling for active *r*GE, or genetic self-selection, is arguably one of the most comprehensive ways to rule out potential sources of confounding (i.e., selection) in a causal modeling framework (Cleveland, Beekman, & Zheng, 2011). Figure 1 provides a graphical depiction of this relationship as it pertains to the present study.

THE CURRENT STUDY

There is now considerable evidence that individuals share a wide range of characteristics with their peers (McPherson et al., 2001), including involvement in delinquent activities (Christakis & Fowler, 2008; Pratt et al., 2010; Rosenquist et al., 2011). Nonetheless, problems with confounding due to selection effects have not been completely ruled out (Young et al., 2013b), due in large part to researchers' inability to randomly assign youth to peer networks.[1] Thus, the distinction between selection and causal effects remains a central concern for social learning theorists specifically and criminologists more generally (Young et al., 2013a). Gottfredson and Hirschi (1990) argued that delinquent individuals self-select into delinquent peer groups and that if this selection effect is controlled, the relationship between one's own delinquency and that of their peers will disappear. In the words of modern behavioral genetic research, Gottfredson and Hirschi's (1990) position can be easily recast as an argument that active *r*GE will explain the behavioral covariance between peer group members. Alternatively, learning theorists like J. R. Harris (1998) and Akers (1998) contend that peers will have a causal impact on one's behavior, even in the presence of selection effects. It should be noted, however, that J. R. Harris (1998) identified the importance of genetic self-selection processes while Sutherland (1947) and Akers (1998) took more of a sociological approach to the issue. Recognizing these problems, the present study advances the concept of active *r*GE as an alternative way to conceptualize the self-selection problem in criminological research. Most important is that active *r*GE provides a built-in solution to the problem of confounding due to selection effects. Specifically, if genetic influences account for a portion of one's decisions to select into a particular peer group (and recent evidence suggests they do; see Beaver et al., 2009; Boisvert, Boutwell, Vaske, & Newsome, 2013; Christakis & Fowler, 2014; Cleveland et al., 2005; J. H. Fowler, Dawes, & Christakis, 2008; Harden et al., 2008), many sources of confounding can be ruled out by controlling for those genetic factors. As will be described below, the current study will analyze the impact of peer delinquency on self-reported delinquency before and after controlling for genetic sources of confounding due to selection.

Two final issues must be addressed before moving to the present analysis. First, recall that there are two different ways in which scholars measure peer delinquency: the indirect method and the direct method. Recent evidence suggests that each method taps into separate latent constructs (Young et al., 2013b), and logical insight suggests the direct method may be the most desirable measurement strategy given the possibility for projection bias in the indirect method (Young et al., 2011). As a result, the current efforts will rely on a measure of peer delinquency that gathers information directly from respondents' peers (i.e., the direct method).

Second, the present study draws on data from the National Longitudinal Study of Adolescent Health (Add Health), which have been used in several prior studies on closely related topics (e.g., Beaver, 2008; Beaver et al., 2009; Boisvert, Boutwell, et al., 2013; Cleveland et al., 2005; Harden et al., 2008). Thus, it is important to note the unique features of this study as compared with those already available. While there are many features of this study that are shared by those cited above, no study of which we are aware has utilized the Add Health peer network files to analyze whether active *r*GE explains a portion of the correlation between peer delinquency and self-reported delinquency. Several articles have utilized the twin data in the Add Health to estimate the influence of genetic factors on peer delinquency, but these studies have drawn on the indirect measure (Beaver, 2008; Beaver

et al., 2009; Boisvert, Boutwell, et al., 2013), relied on best-friend data (Harden et al., 2008), or utilized items that differ from ours in constructing the peer delinquency scale (Cleveland et al., 2005). The latter article by Cleveland et al. (2005) analyzed the full peer network but only gathered information on peer substance use. Our measure, which is discussed in detail below, captures a wider range of risky and problem behavior in the peer group.

METHODS

DATA

Data for the current study were taken from the Add Health (K. M. Harris, 2009). The Add Health is a nationally representative sample of adolescents who were in Grades 7 through 12 during the 1994-1995 school year. At the beginning of the project, schools served as the primary sampling unit. Schools were first stratified by size, type, region, urbanization, and percent White. Next, a sampling frame consisting of 26,666 schools was identified. From this sampling frame, 132 middle, junior high, and high schools were selected. Every student attending these 132 schools was asked to complete a self-report questionnaire (the in-school survey), resulting in a sample of 90,118 students. A portion of these participants was contacted and asked to complete a follow-up interview in their homes (the in-home surveys). A total of four waves of in-home surveys were collected (K. M. Harris et al., 2009).

The Add Health is a unique data source because it incorporates information from multiple respondents and across multiple time points. For instance, data were collected from target respondents, the respondents' parents, and the respondents' peers. In the current study, we draw information from three data files: the Wave 1 in-school survey, the Wave 1 in-home survey, and the genetic subsample. As previously stated, the in-school questionnaire was completed by 90,118 students during normal school hours. These surveys tapped information about the participants' school life, peers, family life, school work, recreational activities, health status, and involvement in deviant or delinquent behaviors. One feature of the in-school data is that peer networks can be identified and constructed. During the in-school interviews, participants were asked to select up to five of their best friends (male and female) from their school's student roster. With this information, it is possible to assess features of the target respondent's peer network by linking their survey answers with responses from their nominated peers (or peers who nominated the target respondent).

The second data file that will be utilized is the Wave 1 in-home questionnaire. Drawing from the pool of 90,118 subjects who participated in the in-school survey, 20,747 students in Grades 7 through 12 were sampled to take part in a subsequent interview conducted in their home (i.e., Wave 1 in-home survey). During these interviews, more extensive information was obtained about the adolescent's personality traits, social relationships, and behaviors.

Finally, the third data file that will be used is the subsample of sibling pairs who were living in the same household during Wave 1 interviews (Rowe & Jacobson, 1998). Included in this subsample was any student who identified himself or herself as an identical twin (monozygotic [MZ]) or a fraternal twin (dizygotic [DZ]), along with his or her co-twin. The sibling subsample consists of 900 DZ twins and 578 MZ twins. Twin zygosity was determined by Add Health researchers by first classifying all mixed-sex twins as DZ. Next, same-sex twins were rated based on their similarity (i.e., "confusability") of appearance. Most twins were successfully categorized based on this measure, but a small number of

respondents were classified as having an uncertain zygosity. These twin pairs were categorized as MZ or DZ based on the results from a genetic test at seven loci. This test has an error rate of approximately 4 out of 1,000 (K. M. Harris, Halpern, Smolen, & Haberstick, 2006; Rowe & Jacobson, 1998). All individuals with an unassigned zygosity were removed from the twin sample prior to the analysis (n = 43 twin pairs). Drawing on this subsample of twins and after eliminating cases with missing values, the final analytic sample ranged between 456 and 524 DZ and 286 and 350 MZ twins, depending on the measure(s) being analyzed.

MEASURES

Peer Network Delinquency

Peer delinquency is measured using the direct method which allows peers to report on their own behavior. Following the lead of prior Add Health researchers (Haynie, 2001; Morris & Johnson, 2010), a measure of peer delinquency was constructed by averaging peers' responses to seven questions that tap involvement in delinquency and/or risky behavior (a well-established correlate of deviance; Gottfredson & Hirschi, 1990). Specifically, peers were asked to report how often they smoked cigarettes, drank alcohol, got drunk, did something dangerous because they were dared to, lied to parents or guardians, skipped school, and got into a physical fight. All seven items were coded on a scale ranging from 0 (*never*) to 4 (*3 to 5 days a week or more than 7 times*). Other researchers have used similar measures (see Young, 2014).

Two measures were created for the analysis: (a) a send measure and (b) a send-&-receive measure. The send measure is the least restrictive because it counts all nominated peers as friends, regardless of whether the nomination was reciprocated. The send-&-receive measure only includes adolescents who reciprocated each other's nomination as a friend. Thus, this measure is more conservative in who gets paired with whom. To generate these two measures, the target respondent's peers' answers were averaged for each individual question. For example, imagine a respondent who identified four peers during the in-school survey. These four individuals were asked about their cigarette smoking and the average of these four responses was calculated and saved as a new variable for the target respondent. The second step was to average the mean responses to each of the seven items to generate the peer delinquency scale, where higher values indicate more peer delinquency (α = .79 for the send measure, α = .81 for the send-&-receive measure).

Self-Reported Delinquency

During Wave 1 in-home interviews, respondents were asked a series of questions referring to their involvement in delinquent activities. Specifically, respondents were asked to indicate (0 = *never*, 1 = *one or two times*, 2 = *three or four times*, and 3 = *five or more times*) how frequently they painted graffiti, damaged property, lied to their parents, stole from a store, got into serious fights, hurt someone badly enough that the person required medical attention, ran away from home, stole a car, stole something worth less than $50, stole something worth more than $50, broke into a house or building, used a weapon to get something from someone, sold drugs, took part in a group fight, acted loud or unruly in public, carried a weapon to school, and used a weapon in a fight (the latter two items were coded 0 = *no*, 1 = *yes*). For the analysis, each of the 17 items were combined into a frequency scale by

creating an additive index where 0 indicated that the respondent had not participated in any of the activities and higher values reflected a greater involvement in delinquency ($\alpha = .84$).

ANALYSIS PLAN

The analysis unfolded in two broad steps. First, cross-twin correlations for the peer network delinquency scales and for the self-reported delinquency scale were analyzed to determine whether genetic factors impacted the measures. Cross-twin correlations are an important first step in establishing whether genetic influences impact a phenotype. MZ twins share twice as much genetic material (100%) as compared with DZ twins who share, on average, 50% of their distinguishing DNA. Both sets of twins are assumed to share an equal amount of their environment, known commonly as the equal environments assumption (EEA). While it is beyond the scope of the present article, recent work suggests the EEA is upheld in twin-based research and that any violations of the assumption are likely to be offset by countervailing concerns (Barnes, Boutwell, Beaver, Gibson, & Wright, 2014; Barnes, Wright, Boutwell, Schwartz, Connolly, Nedelec, & Beaver, 2014; Carey, 2003; Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013; Miles & Carey, 1997). Thus, to the extent that MZ twins resemble one another more closely than DZ twins, genetic factors are likely to play a role in the etiology of that phenotype (Plomin, DeFries, Knopik, & Neiderhiser, 2013). As noted earlier, recent evidence (Barnes, Beaver, et al., 2014; Christakis & Fowler, 2014) and the concept of active *r*GE suggests that peers will resemble one another genetically. Thus, to the extent that the measures of peer network delinquency are more similar among MZ twin pairs than DZ twin pairs, evidence in favor of active *r*GE will be found.

The second step to the analysis focused on estimating the impact of peer network delinquency on self-reported delinquency in two unique ways using a regression-based approach. The first regression model estimated the impact of peer network delinquency on self-reported delinquency *before* controlling for genetic factors that impact self-reported delinquency. This model can be thought of as a baseline estimate of the correlation between peer network delinquency and self-reported delinquency because sources of genetic confounding have not been removed. In this way, the baseline model represents the standard social science method (SSSM) of analyzing the correlation between two measures (Barnes, Boutwell, et al., 2014). Model 1 was carried out by estimating a negative binomial regression where the dependent variable was the measure of self-reported delinquency and the independent variable was peer network delinquency. The negative binomial model can be represented as follows:

$$\ln\left(\lambda_i\right) = \hat{b}_0 + \hat{b}_1\left(PeerNet_i\right) + a + e_i, \tag{1}$$

where the subscript i reveals that each respondent will provide information to the analysis, $\ln(\lambda_i)$ represents the natural log of the expected average value on the self-reported delinquency measure for each observation, $PeerNet_i$ represents the observed value on the peer network delinquency measure for each observation, a is the negative binomial variance parameter, and e_i is the unobserved error term (see MacDonald & Lattimore, 2010).

The second negative binomial model represents an adaptation of the DeFries–Fulker (DF) model (DeFries & Fulker, 1985; Rodgers, Buster, & Rowe, 2001), which is an established strategy for estimating genetic influences on a phenotype in a regression-based framework (Cherny, DeFries, & Fulker, 1992).

$$\ln\left(\lambda_{1j}\right) = \hat{b}_0 + \hat{b}_1\left(SRD_{2j} - \bar{SRD}\right) + \hat{b}_2\left(R_j \times [SRD_{2j} - \bar{SRD}]\right) + \hat{b}_3\left(PeerNetDiff_{1j}\right) + a + e_{ij}. \quad (2)$$

In Equation 2, the subscripts now reveal that Twin 1's expected value for self-reported delinquency (i.e., $\ln(\lambda_{1j})$) is being predicted by Twin 2's grand-mean centered self-reported delinquency score (i.e., $SRD_{2_j} - \bar{SRD}$). Note that the negative binomial DF model is a twin pair analysis, and therefore the subscript *j* has been added to the equation to denote that the twin pair structure of the data is being taken into account (standard errors were adjusted for the clustering and each twin will appear once as Twin 1 and again as Twin 2). Important to note is that three new variables have been entered into Equation 2 that were not present in Equation 1. First is R_j, which represents the genetic relatedness between the twins who provided data. This variable is set to .50 if the twins are DZ and 1.00 if the twins are MZ. Second is the product term for, $R_j \times [SRD_{2_j} - \bar{SRD}]$, which captures the multiplicative interaction between the genetic relatedness variable and Twin 2's (from twin pair *j*) self-reported delinquency score. The parameter estimate for $\hat{b}_2$ captures the influence of genetic factors on the outcome variable by estimating the degree to which Twin 2's score is a stronger predictor of Twin 1's score for MZ twins as compared with DZ twins. Rodgers and McGue (1994) demonstrated that the DF model is a consistent and efficient estimator of genetic influence.

The third new variable to appear in Equation 2 will be the focal point of the analysis. Specifically, the parameter estimate for $\hat{b}_3$ captures the impact of the $PeerNetDiff_{1j}$ variable. The $PeerNetDiff_{1j}$ variable is constructed by subtracting Twin 2's score on the peer network delinquency measure from Twin 1's score on the same measure, where both twins come from twin pair *j* (i.e., $PeerNetDiff_{1j} = PeerNet_{1j} - PeerNet_{2j}$). In this way, the augmented negative binomial DF model will estimate the impact of peer network delinquency on self-reported delinquency *after* controlling for genetic influences on the latter. Thus, this model can be thought of as estimating the correlation between peer network delinquency and self-reported delinquency after all sources of genetic confounding (including active *r*GE) have been removed from variance in the dependent variable. To the extent that the peer network delinquency measures are significantly associated with self-reported delinquency in Equation 1 but *not* in Equation 2, evidence for active *r*GE being a source of confounding will be obtained.

RESULTS

Presented in Table 1 are summary statistics for each of the variables utilized in the analysis. Note that the table presents these statistics for the full sample, for the MZ twin subsample, and for the DZ twin subsample. Also presented in Table 1 are the cross-twin correlations for each of the measures within each group. As noted above, the cross-twin correlations (i.e., *r*DZ and *r*MZ) are a preliminary step in estimating whether genetic influences account for variance in a measure. Two points are important to take away from the statistics presented in Table 1. First, across all three measures, the cross-twin correlation for MZ twins (i.e., *r*MZ) was larger than the cross-twin correlation for DZ twins (i.e., *r*DZ). For example, the send-&-receive peer network delinquency measure revealed that *r*MZ = .71 was considerably stronger than *r*DZ = .45, a pattern of results that hints genetic factors may explain a portion of the variance in exposure to peer group delinquency. Second, note that the cross-twin correlations do not diverge as much when the send version of the peer network delinquency

TABLE 1: Summary Statistics and Cross-Twin Correlations for Add Health Study Variables

	Full Sample					MZ Twins					DZ Twins				
	M	*SD*	Minimum	Maximum	*r*Full	*M*	*SD*	Minimum	Maximum	*r*MZ	*M*	*SD*	Minimum	Maximum	*r*DZ
Peer network delinquency															
Send	1.03	0.62	0	4.86	.48*	1.00	0.61	0	4.86	.54*	1.05	0.62	0	3.71	.43*
Send-&-receive	1.02	0.59	0	5.71	.55*	1.00	0.60	0	5.71	.71*	1.03	0.57	0	3.75	.45*
Self-reported delinquency	4.14	5.30	0	47	.42*	4.32	5.83	0	47	.46*	4.03	4.93	0	43	.39*

Note. *M*=mean; *SD*=standard deviation; MZ = monozygotic; DZ = dizygotic.
*$p < .05$, two-tailed.

measure was analyzed. This finding suggests genetic factors may not be as influential for the send measure. Perhaps most important are the cross-twin correlations for the self-reported delinquency measure. As the table reveals, MZ twins were more similar in their involvement in delinquent behavior (rMZ = .46) compared with DZ twins (rDZ = .39). This pattern of findings suggests genetic factors explain a portion of the variance in each of the three measures. Given these findings, it is important to consider the impact of peer network delinquency on self-reported delinquency before and after controlling for any genetic influences on the latter. The next phase of the analysis will address this point.

Table 2 provides estimates from four negative binomial regression models. Note that the table is split into two panels. Panel A analyzes the impact of the send measure of peer network delinquency on self-reported delinquency. Note that two sets of results are presented in Panel A. The first set of results reveals the impact of peer network delinquency (send) prior to including a control for genetic factors. The next set of results in Panel A analyzes the impact of peer network delinquency (send) on self-reported delinquency after genetic factors have been controlled. The first set of results in Panel A of Table 2 reveals that the send measure of peer network delinquency was positively associated with self-reported delinquency in a bivariate negative binomial regression model. The coefficient of relationship was $b = .29$ with an incidence rate ratio (IRR) of 1.34 and this association was statistically significant ($p < .01$). The latter coefficient indicates that a one-unit increase in peer network delinquency (send) is associated with a 34% increase in the rate of self-reported delinquency, on average.

The next set of results in Panel A of Table 2 reveals the association between peer network delinquency (send) and self-reported delinquency *after* controls for genetic factors have been taken into account. Two important points distinguish this analytic model from the bivariate model just discussed. First, the present analytic model included a measure to control for genetic influences on self-reported delinquency by utilizing the DF regression approach laid out in the Analytic Plan (Rodgers et al., 2001). Second, the peer network delinquency variable is analyzed as a within-twin pair difference score in the DF model but was not transformed in the preceding model. For these reasons, the regression coefficients presented in the second set of results in Panel A cannot be directly compared with the first set of results presented in Panel A. What can be contrasted across the two sets of results is whether the impact of peer network delinquency (send) maintains a statistically significant influence on self-reported delinquency *after* genetic controls are included in the model. As can be seen from the results in Panel A, this was not the case. To be sure, the effect of the send measure of peer network delinquency was no longer statistically significant after the influence of genetic factors were controlled for ($b = -.10$, IRR = .90, *ns*).

Results for the send-&-receive measure of peer network delinquency are presented in Panel B of Table 2. Though subtle differences in the coefficient values emerged, the overall substantive conclusions garnered from the send-&-receive measure (Panel B) were identical to those reported for the send measure (Panel A). Specifically, results in Panel B indicate that the send-&-receive version of peer network delinquency was positively associated with self-reported delinquency ($b = .37$, IRR = 1.45, $p < .01$) in the bivariate model but the relationship was no longer statistically significant in the DF model ($b = -.03$, IRR = .97, *ns*). As before, these results indicate that the inclusion of genetic factors led the effect of peer network delinquency (send-&-receive) on self-reported delinquency to drop from statistical significance.

TABLE 2: The Effect of Peer Network Delinquency on Self-Reported Delinquency

	Before Genetic Control				After Genetic Control (DF Model)			
	b	*SE*	IRR	*p*	*b*	*SE*	IRR	*p*
Panel A: Peer network delinquency (send)								
Genetic factors	—	—	—	—	.15	.02	1.16	<.01
Peer network delinquency								
Send	.29	.11	1.34	.01	—	—	—	—
Send (difference score)	—	—	—	—	−.10	.07	.90	.14
n		520				520		
Panel B: Peer network delinquency (send-&-receive)								
Genetic factors	—	—	—	—	.14	.02	1.15	<.01
Peer network delinquency								
Send-&-receive	.37	.11	1.45	<.01	—	—	—	—
Send-&-receive (difference score)	—	—	—	—	−.03	.09	.97	.71
n		668				668		

Note. Negative binomial regression and negative binomial DeFries–Fulker (DF) regression of self-reported Delinquency on Peer Network Delinquency (standard errors were corrected to account for the clustering of twins within families). IRR = incidence rate ratio.

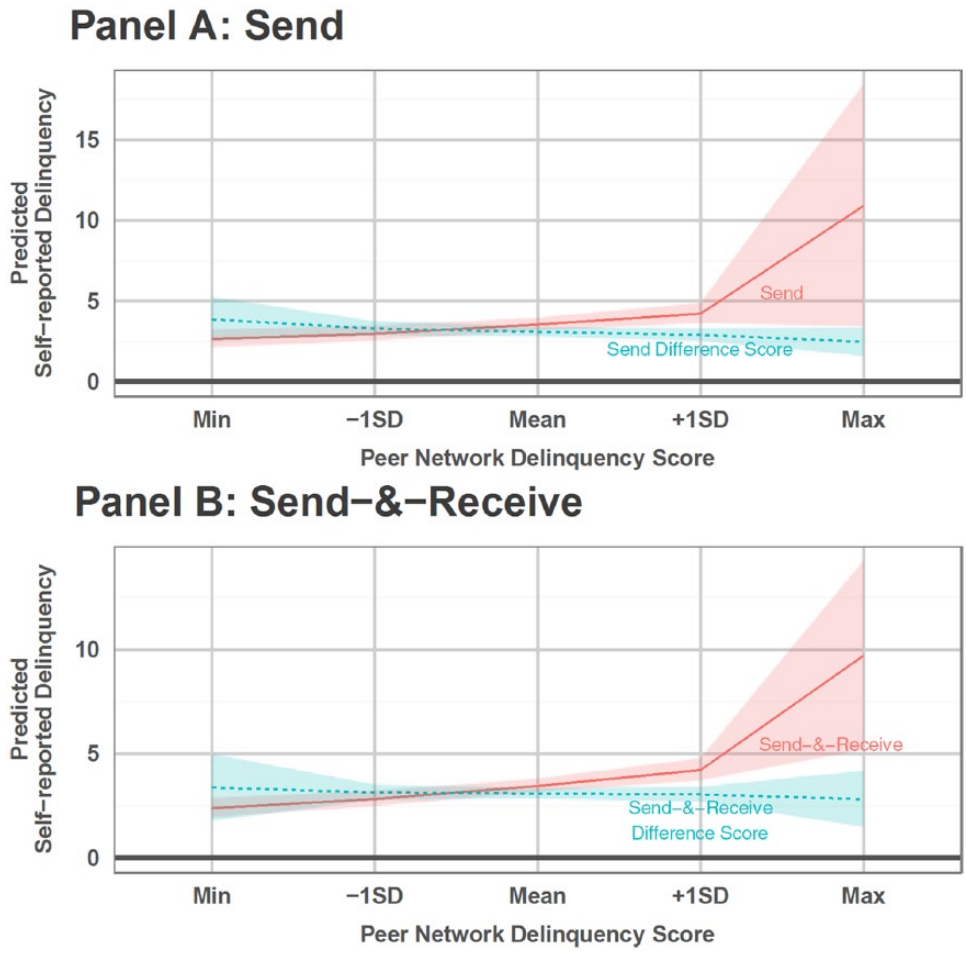

Figure 2: Predicted Self-Reported Delinquency as a Function of Peer Network Delinquency
Note. The solid lines represent the impact of peer network delinquency prior to controlling for genetic influences. The dashed lines represent the impact of peer network delinquency after controlling for genetic influences.

To ease the interpretation of the results, a graphical representation of the findings are provided in Figure 2. Specifically, predicted counts of self-reported delinquency were generated for each of the negative binomial models that were estimated in Table 2. Due to the different scales used for each of the peer network delinquency measures, meaningful comparisons could only be made at standardized intervals such as the minimum value, standard deviation units, the mean, and the maximum value. The figure, which is broken into two panels that correspond with the panels in Table 2, plots the observed values of peer network delinquency along the *x*-axis and the predicted rate of self-reported delinquency on the *y*-axis. Within each figure box, two lines are presented. The solid line reveals the predicted self-reported delinquency scores as a function of peer network delinquency *before* controlling for genetic factors. As can be seen, the solid line reveals a positive association in both

of the panels. The broken line plots the relationship between peer network delinquency and self-reported delinquency *after* genetic factors were controlled. Consistent with the results in Table 2, the broken line has a weak negative slope that does not appear to differ significantly from zero.

DISCUSSION

The relationship between an individual's behavior and that of his or her peers has been the focus of a large body of criminological, psychological, and sociological research. Studies have found that peers resemble one another on a host of behaviors such as delinquency, victimization, tobacco use, alcohol use, and drug use (Christakis & Fowler, 2008; Rosenquist et al., 2011). Still, there remains a debate over whether and how much the peer group affects the behavior of an individual group member. Whether the peer effect observed in prior research captures peer group influences via learning mechanisms or is due to selection processes remains an open empirical question. Some of the most popular criminological theories suggest the effect represents a causal influence where the peer group affects the individual's behavior through mechanisms linked to learning. Sutherland (1947) argued that one's involvement in delinquency or criminal behavior would vary to the extent that they associate with peers who have definitions favorable to antisocial behavior and who behave in antisocial ways. Akers (1998) extended this by adding the concepts of imitation and reinforcement, and J. R. Harris (1995, 1998) added her group socialization theory to the discussion. J. R. Harris (1998) agrees with Sutherland (1947) and Akers (1998) that adolescents take on the attitudes and behaviors of their peer group, but she notes that the individual may behave differently across various contexts (e.g., code switching; Anderson, 1999) and that adolescents often pick their peer groups based on their own genetic predispositions. J. R. Harris's (1998) theory concludes that the peer group is influential but that the relationship is likely to be complicated by *r*GE. In short, J. R. Harris allows for selection effects but contends that peer effects are the dominant factor in shaping an adolescent's personality.

With these theoretical explanations in mind, the present study sought to analyze the effect of peer group delinquency on self-reported delinquency after accounting for a primary source of endogeneity, active *r*GE. Recall that active *r*GE points to the importance of genetic factors in understanding the role of self-selection into an environment such as a peer group. As J. R. Harris (1998) pointed out, one's genes may lead to selection into one group versus another. Recent evidence supports this claim (Barnes, Beaver, et al., 2014; Christakis & Fowler, 2014; Cleveland et al., 2005). Thus, our analysis examined the impact of a direct measure of peer delinquency on self-reported delinquency while controlling for sources of confounding due to genetic self-selection. Several important findings emerged from the current analysis. First, the cross-twin correlation for MZ twins was larger than the cross-twin correlation for DZ twins for all three measures included in the analysis. These results suggested genetic factors partially underlie the etiology of each measure, but they also left room for social learning mechanisms to explain a portion of the variance because the cross-twin correlations did not indicate genetic factors were the only influential mechanism. In this respect, J. R. Harris's (1998) group socialization theory may be most informative because it allows for both selection effects due to *r*GE and peer influence effects.

The second finding of note emerged from the analysis of the impact of peer network delinquency on self-reported delinquency. Recall that these models were estimated twice for the

send version of the peer network delinquency measure and again for the send-&-receive measure. The results suggested that prior to controlling for genetic influences, peer network delinquency (both the send measure and the send-&-receive measure) was a salient predictor of self-reported delinquency. This finding is consistent with theoretical expectation and with the large body of research that has preceded this study (Pratt et al., 2010).

After genetic influences were controlled for, the pattern of findings shifted and no longer revealed a statistically significant impact of peer network delinquency (for the send and the send-&-receive measure) on self-reported delinquency. These results are supportive of J. R. Harris's (1998) argument that delinquent individuals self-select into deviant peer groups. Note, however, that J. R. Harris (1998) also made the case that once in the peer group, an adolescent would continue to be socialized by the group members. The present results do not appear to be consistent with this portion of J. R. Harris's (1998) explanation.

Although speculative at this point, two issues relating to the present findings are worth considering. First, our results may be consistent with the argument that context-specific socialization will occur within the peer group (see Anderson, 1999, and J. R. Harris, 1998). Specifically, one might expect adolescents to act differently around their parents than they do around their peers. This is an important point because the self-reported delinquency questions were asked to the respondent while they were being interviewed *in their home*. It may be difficult to find evidence of peer effects on adolescent delinquency when the delinquency measure is administered in the child's home, oftentimes with the parent present (at least in another room). It may be necessary to observe the child in the peer group to capture a more pure estimate of the effect of peer group delinquency on the child's delinquency (J. R. Harris, 1998).

The second point worth considering is that the present analysis relied exclusively on a direct measure of peer network delinquency and did not assess the impact of an indirect measure of peer behavior on respondent delinquency. This begs the question as to how the results may have changed if the indirect method was used rather than the direct method. Available evidence provides several clues to suggest that had we utilized the indirect measures, the impact of peer delinquency on self-reported delinquency would have emerged as a statistically significant influence. Specifically, Beaver (2008), drawing on Add Health data, utilized the MZ difference score method to analyze the impact of an indirect measure of peer delinquency on self-reported delinquency. Peer delinquency was a positive and statistically significant predictor of self-reported delinquency in most of the analytic models presented. Thus, when one compares the present findings to those available in prior research, it appears the use of the direct peer network measure of peer delinquency provides a substantively different set of results. Note, however, that these are only speculative conclusions because there are several major differences between Beaver's (2008) analysis and the present study, including the types of behaviors covered in the peer delinquency measures, the use of MZ and DZ twins in the present study (Beaver only drew on the MZ subsample), and the inclusion of other variables in the multiple regression models by Beaver.

Although the current study filled several gaps in the literature, there were certain limitations that must be kept in mind when weighing the evidence. First, the current analysis was limited to including network data on in-school friends. Participants were, however, able to nominate friends outside of school and prior analyses have revealed only a small number of outside connections (1.4 per student). As discussed by Haynie (2001), the out-of-school nominations did not differ between delinquent and nondelinquent adolescents and most

adolescents have friendship networks within schools rather than outside of the school. A second limitation is that there may be additional influences on an individual's delinquency that were not considered. For example, Shakya, Christakis, and Fowler (2012) found that social networks might extend beyond the traditional peer group. The authors concluded that while the behavior of an individual's friend was significantly associated with the individual's behavior, the parenting style of a peer's parent was also significantly correlated with the individual's behavior. Results revealed that peers' *parents* might impact the target individual's behavior, perhaps more so than the individual's own parents. Furthermore, the influences of positive parenting of a peer's mother were both direct (i.e., through contact with the parent) and indirect (i.e., mediated through the behavior of the peer). Our analysis did not account for any direct effects of peers' parents on self-reported delinquency, an issue that should be considered by future scholars.

A third limitation concerns the generalizability of the results from the Add Health twin subsample. As the sample consisted of twin pairs, results may not be generalizable to all adolescents. Addressing this point is a recent paper by Barnes and Boutwell (2013) who used Add Health data to compare twins and singletons. Their results indicated that twins did not differ significantly from the nationally representative sample of singletons on most measures analyzed, including delinquency and exposure to peer delinquency. Thus, this limitation may be of little concern for the present study. Finally, a fourth limitation is that sex differences were not considered in the present study. A budding line of research suggests genetic factors and their effects vary across males and females (e.g., Boisvert, Wright, Knopik, & Vaske, 2013). Building on this evidence base, we might expect the current findings to vary across sex. For instance, it may turn out that once genetic factors are controlled for, peer influences matter for males but not for females (or vice versa). We invite future scholars to analyze this possibility as it will open up many new avenues to expand criminologists' understanding of how, when, and for whom peers matter.

CONCLUSION

Despite the aforementioned limitations, this study represents one of the first attempts to estimate the impact of peer network delinquency on self-reported delinquency *after* accounting for sources of confounding due to genetic self-selection. Once genetic factors (i.e., active *r*GE) were controlled, the association between one's own behavior and that of his or her peer network was no longer statistically significant, drawing into question certain theories from the social learning tradition. To be certain, had we failed to control for genetic influences, our results would have supported the notion that peers play an influential role in the etiology of self-reported delinquency. Yet, this association was not present after genetic factors were taken into account, demonstrating the importance of controlling for genetic influences in traditional social science models (Barnes, Boutwell, et al., 2014).

NOTE

1. It is worth noting that researchers have been able to analyze dyadic peer relations formed based on random assignment of roommates at many universities (e.g., Sacerdote, 2001; Wagner, Li, Liu, & Guo, 2013). These studies revealed an association between the behaviors of roommates, offering support for the causation hypothesis. Nonetheless, neither study was able to rule out influences of the entire peer network and neither focused on the direct effect of the peer's deviance on the target individual's deviance. Note that Sacerdote's (2001) analysis addressed educational outcomes and decisions to join a fraternity/sorority while Wagner et al. (2013) analyzed alcohol consumption but did not report the main effect of the peer's drinking on the target individual's behavior.

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Michael TenEyck is a doctoral student in the School of Criminal Justice at The University of Cincinnati. His research interests include biosocial criminology, criminological theory, drugs and crime, and the psychological correlates of crime. His current research projects focus on the relative stability of impulse control over the transitional period between adolescence and adulthood. He has published in outlets such as *Social Networks, Journal of Psychiatric Research, American Journal of Criminal Justice*, and *PLoS ONE*.

J. C. Barnes is an associate professor in the School of Criminal Justice at the University of Cincinnati. He is a biosocial criminologist whose research seeks to understand how genetic and environmental factors combine to impact criminological phenomena. He has published in outlets such as *Aggressive Behavior, Behavior Genetics, Criminology, Developmental Psychology, Intelligence, Journal of Marriage and Family, Justice Quarterly, Journal of Theoretical Biology*, and *PLoS ONE*.