Journal of Criminal Justice 42 (2014) 471–482

Contents lists available at ScienceDirect

Journal of Criminal Justice

On the consequences of ignoring genetic influences in criminological research

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ARTICLE INFO

Available online xxxx

ABSTRACT

Purpose: Many criminological scholars explore the social causes of crime while giving little consideration to the possibility that genetic factors underlie the observed associations. Indeed, the standard social science method (SSSM) assumes genetic influences do not confound the association between X and Y. Yet, a nascent stream of evidence has questioned the validity of this approach by revealing many criminological variables are at least partially affected by genetic influences. As a result, a substantial proportion of the literature *may* be misspecified due to uncontrolled genetic factors. No effort has been made to directly estimate the extent to which genetic confounding has biased the associations presented in criminological studies.

Methods: The present study seeks to address this issue by drawing on simulated datasets.

Results/Conclusions: Results suggest genetic confounding may account for a negligible portion of the relationship between X and Y when their correlation (r_{yx}) is larger than the correlation between genetic factors and Y (i.e., $r_{yx} > r_{yg}$). Genetic confounding appears to be much more problematic when the correlation between X and Y is in the moderate-to-small range (e.g., $r_{yx} = .20$) and the genetic effect is in the moderate-to-large range (e.g., $r_{yg} \geq .30$).

Introduction

As a discipline, criminology has been dominated by sociological (i.e., environmental) explanations of human behavior (Cullen, 2011). Since Durkheim famously proclaimed that, "The determining cause of a social fact must be sought among antecedent social facts and not among the states of the individual consciousness" (Durkheim, 1982:134), sociologists and criminologists have argued that social forces are the most salient influence on human behavior (Udry, 1995). As a result, scholars in criminology often assume that the forces of nature and nurture are, for the most part, mutually exclusive with those factors believed to tap into nurture being most relevant and deserving of empirical observation. Such logic, however, has recently come under renewed scrutiny and no longer appears tenable (Sameroff, 2010).

Indeed, mounting evidence clearly indicates that *both* nature and nurture play a role in the etiology of many human behaviors (Carey, 2003; Pinker, 2002; Plomin, DeFries, Knopik, & Neiderhiser, 2013; Turkheimer, 2000) and biological explanations of such behaviors are gaining in popularity among criminologists (Rowe, 2002; Rowe & Osgood, 1984; Simons et al., 2011; Tibbetts, 2011; Tremblay, Hartup, & Archer, 2005). An emerging group of scholars—motivated by these modern assessments—have suggested that much of the criminological knowledge base should be revisited and perhaps reconsidered against the contrast of biosocial research that clearly implicates both genetic and environmental factors as sources of human variation (Cullen, 2011).

Although research into the genetic underpinnings of antisocial behavior has occasionally appeared in criminological journals (Barnes & Boutwell, 2012; Fishbein, 1990; Walsh, 2000), it has only been in the past decade or so that biosocial criminology has begun to gain prominence and a clear research agenda (see the special issue on genetics and antisocial behavior published in 2013 in *Journal of Criminal Justice* [Tuvblad & Beaver, 2013]). Biosocial criminologists have, over a relatively short time span, revealed that many of the long held "truths" of criminology are built on shaky foundations that should be recast alongside newly emerging evidence (Cullen, 2009; Walsh, 2002). While it may be an overstatement to say that biosocial criminology is leading the discipline toward a paradigm shift (Kuhn, 1962), an honest appraisal of the criminological discipline will no doubt reveal that the implications of biological and genetic research have recently piqued many scholars' attention (Rocque, Welsh, & Raine, 2013; Wright & Boisvert, 2009).[1]

Like all academic disciplines, criminology has drifted (Matza, 2009 [1964]) from one theoretical focus to the next (Elliott, 1985). One can

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http://dx.doi.org/10.1016/j.jcrimjus.2014.08.003
0047-2352/

look back over the decades of criminological thinking and see a shift—slight as it may have been at times—in the prevailing theoretical zeitgeist. Thus, criminology as a discipline has shown a tendency to change course when new evidence becomes available. The willingness to redirect attention is certainly a desirable trait of an academic area given that the laws of probability make it unlikely that any one theory (or group of theories) is "true" (Ioannidis, 2005). In general, the incremental phases in which a scientific knowledge base gets built makes an academic discipline like criminology well suited for regular shifts in focus. Nonetheless, a shifting theoretical landscape means that, on occasion, criminology will have to face certain "hard" questions. It would appear that time has, once again, arrived.

The growing body of evidence revealing a genetic influence on criminal/antisocial behavior has reached a critical mass. There are now hundreds of studies that reveal such a link (see any of the six recent meta-analyses on the topic: Burt, 2009a,b; Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002), indicating that the question of whether genes matter is no longer a question at all; they do (Barnes et al., in press). Nonetheless, we will review this literature in the sections that follow. More important, though, is our focus on two fundamental questions facing criminology. The first concerns how (and to what extent) evidence from biosocial criminology should be integrated into modern criminological research and theories. The second is whether and to what degree the evidence from biosocial criminology contradicts prevailing wisdom about the causes of crime. Both questions are addressed in the present study. The former question is addressed in the sections that follow by considering available evidence. The latter question provided the motivation for the analysis.

The standard social science method (SSSM)

Much of criminological research analyzes individual-level data to test hypotheses about the causes of crime (Harris, 1998; Rowe, 1994; Weisburd & Piquero, 2008; Wright & Beaver, 2005). Researchers commonly rely on the standard social science method (SSSM) to gather the data necessary to analyze such questions. The SSSM can be defined as any method of data gathering and/or data analysis that does not allow the researcher to account for genetic influences (Harris, 1998; Plomin et al., 2013). In the case of family-based research, the SSSM involves analysis of one child per household. To gather and analyze information from more than one child per household is rarely considered, primarily due to the statistical assumptions that would be violated by utilizing such a dataset. For instance, analyzing data from more than one child per household violates the assumption that regression errors are independent and normally distributed across all observations ($e \sim idN[0, \sigma^2]$). By taking this approach, however, criminologists are defaulting to the SSSM and thus making the tacit assumption that genetic effects are near zero. We notate this assumption as, $h^2 = 0$. (Note that h^2 represents a heritability coefficient, which will be explained below. Briefly, a heritability coefficient identifies the portion of variance in a trait that is attributable to genetic differences among the sampled observations).

While there has been no systematic effort to tally the frequency with which the SSSM is utilized in criminological research, it is safe to say that the approach is quite common. Yet, SSSMs may generate results that are systematically biased if the assumption of $h^2 = 0$ is not met. It is true that recent advances in statistical modeling have allowed for the mitigation of certain problems inherent to the SSSM. For instance, the application of multi-level modeling (e.g., HLM) to individual-level data allows for the analysis of *changes* in behavior over time, thus controlling *stable* influences by virtue of the research design. When considering the case for these methods, scholars are likely to rely on the assumption that genetic factors do not change over time and, therefore, cannot explain changes in an outcome like criminal involvement. In a narrow sense, this is a safe assumption; an individual's genetic code (their arrangement of DNA base pairs) does not change. However, genetic *expression* (and thus the influence of genes on certain outcomes) can, and does, change throughout the life course (e.g., Caspi et al., 2002; Plomin et al., 2013). Modeling within-person changes may mitigate certain problems outlined above but it does not rule them out entirely.

In summary, the problem of focus is that SSSMs may produce results that are biased due to omitted controls for genetic factors. This requires that two questions be confronted: 1) is the assumption that $h^2 = 0$ for antisocial behavior tenable?; and 2) is the assumption that $h^2 = 0$ for independent variables tenable? Indeed, the findings gleaned from an SSSM will not be biased if genetic factors only operate on one of the variables in the model (assuming the genetic effect does not explain 100% of the variance). The following section will review the empirical literature that has bearing on both questions.

The case for $h^2 \neq 0$

Human beings, or *Homo sapiens*, are animals. By most estimates, modern humans originated in Africa roughly 150/200,000 years ago, and following our migration off the continent, began the process of global colonization. *Homo sapiens* would go on to establish agricultural practices, wade through industrial revolutions, witness large scale social enlightenments, and achieve technological advances utterly foreign in other species. Indeed, humans are unique in many respects as compared to other animal species. We are not, however, "unique in our uniqueness" (Wilson, 1975[2000]). Ultimately, what our species represents is yet another animal, subject to the exact same selection pressures which have molded and shaped every other species on the planet (Wilson, 1975[2000]). Research outside of the human lineage has always seemed to proceed under the assumption that the evolutionary past and biological makeup of the organism mattered, at least in some respect. Even the strictest adherence to behaviorism (Skinner, 1953) allowed for the idea of an organism's brain as having at least some mechanistic role in learning—if for no other reason than operant conditioning schedules had to affect something in the body (Pinker, 2002).

For decades, the null hypothesis regarding *human* nature and behavior has been that the influence of biology is minimal to non-existent (Pinker, 2002). Certainly, there are reasons why this view might have taken root—ranging from a limited understanding of biological concepts (van den Berghe, 1990) to concerns regarding the potential ramifications of biologically informed research (Pinker, 2002). Whatever the reason, social scientists studying human behavior have tended to begin with the assumption that biology is unimportant, hence the SSSM as the default methodological approach. Consistent with other disciplines studying animal behavior, however, one might argue that the opposite assumption should have been made all along. Anything that exists in human nature such as behavior and personality would ultimately have to exist—directly or indirectly—because of natural selection and biological evolution. By extension, this dictates that genes are involved, at least to some degree, given that natural selection exerts its influence at the level of the genome (Dawkins, 1976; Williams, 1966).

Despite the prevailing assumption that biological factors have negligible influences on human behavior, the past few decades have been witness to an emerging body of evidence that suggests genetic factors indeed underlie the variation observable in any human trait (Plomin et al., 2013). As was noted in the preceding section, there are two points that must be given careful consideration by contemporary criminologists. The first is whether genetic factors play a role in the etiology of criminal behavior. The second concerns whether genetic factors underlie the etiology of common independent variables used in criminological research.[2] Some of the most robust predictors that come to mind are self-control, differential association/social learning, strain, and parenting factors. The next two sections will present an overview of the

current evidence on whether genetic factors influence 1) antisocial behavior and 2) common criminological variables.

Genetic influences on antisocial behavior

In order to assess the evidence of genetic influences on human behavior it is necessary to review research conducted with behavioral genetic methods. Behavioral genetic methods employ statistical analyses to study the genetic and environmental influences on phenotypes (Plomin et al., 2013). A phenotype can be defined as any measurable trait or behavior including levels of self-control or involvement in delinquency. Behavioral geneticists analyze phenotypes by estimating the amount of variance that is explained by environmental *and* genetic factors. Specifically, the variance in a phenotype is decomposed into three latent components: heritability (h^2), shared environment (c^2), and nonshared environment (e^2). The heritability component (h^2) measures the amount of variance in the phenotype that can be attributed to differences in genetic material among the individuals in the sample. For example, a heritability estimate of .25 would mean that 25 percent of the variance in the phenotype is attributable to differences in genetic material among the respondents. (See Barnes et al. (in press) for a discussion of behavior genetic assumptions.) The environmental components (i.e., c^2 and e^2) estimate the amount of variance in the phenotype that can be attributed to environmental factors. It is important to point out that behavioral geneticists distinguish between two types of environmental influences: shared (c^2) and nonshared (e^2). The shared environment captures any environmental influence that makes two siblings more alike. Nonshared environmental influences, on the other hand, capture environmental effects that make siblings different from one another. As the name implies, nonshared environments are events that are either experienced separately or are interpreted differently between siblings (Turkheimer & Waldron, 2000).

In quantitative behavioral genetics, the three components (h^2, c^2, and e^2) are estimated simultaneously as latent factors. In this way, behavioral genetic models do not suffer from the specification problems that may afflict SSSMs (i.e., omitted variable biases) because all factors that can influence variance in the phenotype are included in the model (albeit as latent measures). In order to perform such an analysis, however, one must analyze data that includes more than one child per household.[3] In this way, cross-sibling comparisons can be used to estimate the amount of variance in a trait that is attributable to genetic and environmental influences.

Behavioral genetic methods have been used extensively to study the relative contributions of genetic and environmental factors to the variance in antisocial phenotypes. Currently, there are six meta-analyses and at least four major literature reviews that will allow for a straightforward summary of the current knowledge base (Burt, 2009a,b; Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Moffitt, 2005; Raine, 1993, 2002; Rhee & Waldman, 2002).

In one of the first systematic reviews of the behavioral genetic literature bearing on antisocial behavior, Raine (1993:71) stated:

> One approximate deduction that can be drawn from twin and adoption studies is that genetic influences may well account for roughly half of the *explained* variance in crime. That is, genetic influences are nontrivial and probably account for as much variance as environmental influences in relation to crime (emphasis in original).

It is quite clear from Raine's comment that the research available in 1993 indicated that genetic differences across individuals was responsible for approximately half of the variance in criminal outcomes. Raine's (1993) review primarily spoke to variance in adult criminal behavior but his review of the evidence on juvenile delinquency reached a similar conclusion, though he argued that the genetic effect might be weaker for juvenile delinquency.

Since Raine's (1993) review, several groups of scholars have conducted meta-analyses to generate a mean h^2 estimate for crime, delinquency, and/or antisocial behavior (Burt, 2009a,b; Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002). Although each meta-analysis was unique in the studies it covered, in the time period it examined, and the operationalization of antisocial behavior, a remarkably consistent pattern of findings emerged; each of the meta-analyses concluded that genetic factors play a salient role in the etiology of antisocial behavior. When the estimates from these studies are combined, a confidence interval for the heritability estimate appears to be somewhere between .30 and .60 (Burt, 2009a; Ferguson, 2010) with an average of .50 (see generally, Moffitt, 2005). Thus, the available evidence indicates that genetic influences account for about half of the variance in antisocial behavior (i.e., $h^2 \approx .50$ for antisocial behavior).

Genetic influences on common criminological variables

Behavioral geneticists have consistently concluded that genetic influences on human complex traits are greater than zero (i.e., $h^2 > 0$) and this appears to apply to most phenotypes, not just antisocial behavior (Turkheimer, 2000). A long line of developmental research, for example, has concluded that self-control and related phenotypes such as self-regulation and ADHD have heritability estimates of approximately .40 (Beaver, Connolly, Schwartz, Al-Ghamdi, & Kobeisy, 2013; Bezdjian, Baker, & Tuvblad, 2011; Boisvert, Wright, Knopik, & Vaske, 2012), leading contemporary scholars to search for the underlying genetic variants linked to these traits via technologies that scan the entire genome in search of causal variants (e.g., Stergiakouli, Hamshere, Holmans, et al., 2012). Additionally, there is a nascent line of research that seeks to uncover whether variables typically identified as "social" or "environmental" influences are in fact partially underpinned by genetic influence. In order to grasp how this might be possible, it is informative to consider the role of a phenomenon known as gene-environment correlation (rGE), among which there are three types: passive rGE, evocative rGE, and active rGE (DiLalla, 2002; Kendler & Baker, 2007; McAdams, Gregory, & Eley, 2013; Scarr, 1992; Scarr & McCartney, 1983).

The first type of rGE is known as passive rGE. Passive rGE recognizes that parents pass along an environment *and* genes to their offspring. Since the child's environment and the child's genes both originate from the same source (i.e., their parents) the two are likely to be correlated. Passive rGE offers a viable alternative explanation to the mounds of research that has investigated the correlation between parenting and childhood behavioral outcomes (Harris, 1998; Pinker, 2002; Wright & Beaver, 2005). The second type of rGE is referred to as evocative rGE. Evocative rGE occurs when a person elicits responses from the environment due to his or her genetic propensities, many of which will be expressed via personality traits. In this way, evocative rGE offers a unique perspective for interpreting particular associations such as the one between victimization frequency and delinquency involvement (Barnes & Beaver, 2012; Vaske, Boisvert, & Wright, 2012). The third type of rGE is known as active rGE. Active rGE occurs when a person seeks out environments to suit his or her genetic tendencies/propensities (i.e., personality). Similar phenomena are often referred to as *niche picking* or *self-selection* by criminological scholars (Gottfredson & Hirschi, 1990). Active rGE offers a framework for understanding how genetic factors might influence the nonrandom selection into particular environments such as marriage (Burt et al., 2010), delinquent peer groups (Cleveland, Wiebe, & Rowe, 2005), and perhaps even broader social contexts (Reiss, Plomin, Neiderhiser, & Mavis Hetherington, 2003).

Consideration of rGEs is critical to the current focus because they provide a theoretical scaffold that can help explain why the association between a criminological predictor (X) and an antisocial outcome (Y) might be biased in criminological research. In this respect, the findings reported by Kendler and Baker's (2007) review of the rGE literature are critical. Kendler and Baker analyzed 55 studies to determine the

range of genetic influences on myriad environmental variables thought to be important for psychiatric and drug use disorders. The authors divided the sample of studies into different sections in order to reduce the effects of measurement heterogeneity on the heritability estimates. The different sections tapped various environmental factors including stressful life events and the family environment. Though there was a wide range of heritability estimates observed across the sections and across individual studies (i.e., different environmental variables), Kendler and Baker (2007: 620) concluded that:

> The literature we have reviewed suggests that genetic influences on measures of the environment are pervasive in extent and modest to moderate in impact. Every aspect of the environment that we were able to examine was significantly influenced by genetic factors. However, the role of genetic influences on these behaviors was far from overwhelming…with most falling between 15% and 35%.

In short, the findings from Kendler and Baker's (2007) analysis revealed that genetic factors influence variance in measures of the environment. Thus, these authors provided evidence that many criminological variables, even those traditionally viewed as purely environmental in origin, might be under genetic influence. In short, it appears $h^2 \neq 0$ for independent variables often employed in criminological research (see also McAdams et al., 2013).

The current study

Over the past decade, criminology has witnessed an unprecedented growth in empirical findings indicating moderate-to-large genetic influences on criminal, delinquent, and antisocial behavior (Burt, 2009a,b; Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Moffitt, 2005; Raine, 1993; Rhee & Waldman, 2002). Further, behavioral genetic research has revealed that nearly all human phenotypes are under partial genetic influence (Turkheimer, 2000), including phenotypes often used as independent variables by criminological scholars (Kendler & Baker, 2007). Though these two converging lines of research bring with them many potential implications, most prominent is the argument for model misspecification due to omitted variables bias in SSSMs. As is commonly known, one of the most important assumptions of any statistical model is no endogeneity between the error term and the included covariates. If endogeneity arises due to an uncontrolled third variable(s), the possibility that the correlation between X and Y is biased will increase as a function of the correlation between the omitted variable(s) and X and Y (though it is worth noting omitted variables can also have suppressor effects).

We submit that the SSSM approach of omitting controls for genetic factors (i.e., assuming $h^2 = 0$) violates the endogeneity assumption of inferential statistical models because behavioral genetic research has shown that both antisocial behaviors (i.e., Y) and criminological variables (i.e., X) such as parenting (Kendler & Baker, 2007; Reiss et al., 2003) and self-control (Boisvert et al., 2012) are under genetic influence. Utilizing a series of simulated datasets, the following analysis will demonstrate the various situations in which omitted variables bias is likely to have affected criminological research due to the lack of controls for genetic factors. In essence, we seek to provide criminological scholars with a resource to assist in the assessment of whether an observed correlation is biased and to what degree it may have impacted the substantive conclusions of the study.

Simulation strategy

This analysis unfolded in three phases. The first phase dealt with data generation/creation. The statistical software package, *R*, was used to create simulated observations for three variables: 1) an outcome variable which will be referred to as "Y"; 2) an independent variable which will be referred to as "Criminological Variable"; and 3) a variable tapping omitted genetic factors which will be referred to as "Genetic Factors." A vector of correlation coefficients used to define the correlation structure of the three variables to be generated was created first. Meta-analyses and systematic reviews were consulted in order to choose the pre-defined correlations between Y and the Criminological Variable. This correlation was set to range between .10 and .30 based on the conclusions gleaned from comprehensive reviews of the criminological literature (e.g., Derzon, 2010; Elliott, 1985; Hoeve et al., 2009; Pratt & Cullen, 2000; Pratt et al., 2010; Weisburd & Piquero, 2008).

Pratt and Cullen (2000), for example, estimated the average correlation between self-control and delinquency to be in the range of $r \sim .20$-.30, leading the authors to conclude that self-control was "…one of the strongest known correlates of crime" (p. 952). A recent review of the role of family in the etiology of problem, aggressive, criminal, and violent behavior reported a grand mean effect size of $r = .15$ (Derzon, 2010). Weisburd and Piquero (2008: 479), moreover, reported "Studies of individuals anchor the low part of the distribution with an average R^2 of .302." Taken at face value, this may lead one to believe that the correlation between Y and the Criminological Variable should be approximately .55 (i.e., $\sqrt{.30} = .55$). Note, however, that Weisburd and Piquero reported R^2 rather than *r*. This is an important distinction because R^2 will typically include the influence of more than one variable. Thus, our choice of .30 as the upper bound for the correlation is likely to be a reasonable estimate of the correlation between any *one* Criminological Variable and Y.

The correlation between the Genetic Factors and the other variables (i.e., Y and the Criminological Variable) was manipulated for the analysis and ranged between .10 and .70 based on behavior genetic findings of *heritability* (i.e., h^2) estimates ranging between 30% and 60%, on average, for antisocial behavior (Burt, 2009a,b; Kendler & Baker, 2007; Rhee & Waldman, 2002). Specifically, taking the square root of a heritability estimate ($r \approx \sqrt{h^2}$) provides an approximation of the correlation between genetic factors and the variable of interest.[4] Thus, the average h^2 of .50 for antisocial behavior approximately translates into $\sqrt{.50} = .71$. Recall that h^2 reveals the impact of a "global" measure of genetic factors on the outcome of interest. Thus, the correlation between Genetic Factors and Y will represent the correlation between a comprehensive measure of genetic effects and the outcome of interest.

The next step was to specify the desired sample size ($N = 1{,}000$ for all analyses presented below) and the means and standard deviations of the three variables.[5] All data analyzed here were simulated using the "mvrnorm" command which is part of the MASS package in *R*. The mvrnorm command simulated the values for each of the three variables so that they would have a multivariate normal distribution, each with a mean of 0.00 and a standard deviation of 1.00 on average. We revisit this issue in the Discussion section.

The second phase to the analysis utilized Monte Carlo routines of estimation where the number of repetitions was set to produce 1,000 regression parameter estimates that were then saved to generate a sampling distribution of coefficients. This process was carried out twice, representing the two regression models of interest:

$$Y = \hat{b}_0 + \hat{b}_1(\text{Criminological Variable}) + \hat{e} \quad (1)$$

$$Y = \hat{b}_0 + \hat{b}_1'(\text{Criminological Variable}) + \hat{b}_2(\text{Genetic Factors}) + \hat{e} \quad (2)$$

Eq. (1) is a bivariate regression of Y on the Criminological Variable. Note that standardized parameters were estimated due to the specification of both variables as standard normal variables, though any one estimate will vary slightly from a true standardized estimate due to the stochastic component of the Monte Carlo method (i.e., the regression estimates are samples drawn from the larger sample space of regression estimates for a given relationship). The substantive description of the

Criminological Variable is unnecessary beyond noting that we referred to the criminological literature when determining the correlation value between Y and the Criminological Variable (see above). After estimating Eq. (1) via Monte Carlo simulation, the distribution of the parameter estimates for b_1 was saved along with the average value for b_1 that was gleaned from all of the simulations. Next, Eq. (2) was estimated which included both the Criminological Variable from Eq. (1) and the Genetic Factors considered as the confounder. Again, the parameter estimates can be considered standardized coefficients due to the specification of all variables as standard normal variables. Eq. (2) was estimated via Monte Carlo simulation and the distribution of the b_1' parameter estimates was saved along with the average b_1' parameter estimate observed across all 1,000 simulations.

The third phase of the analysis involved comparing the distribution of the parameter estimates for b_1 that was gleaned from Eq. (1) against the estimates for b_1' gleaned from Eq. (2). We present several different configurations of the results including graphical displays of the sampling distributions of b_1 and b_1', comparison of the mean of the parameter estimates, the percentage of bias expected under each condition, and the percentage of tests that reached statistical significance with a t statistic of +1.96 or greater. In short, if the parameter estimates gleaned from b_1' (Eq. (2)) have a smaller mean, a wider distribution of scores, a distribution of scores that does not substantially overlap with the distribution gleaned from b_1 (Eq. (1)), or fewer tests that reach statistical significance, then evidence of omitted variable bias will be found.

Findings

Table 1 shows the correlation structure of the three variables utilized in the analyses. Below the diagonal are three letters used to guide the reader through the simulation process. The letter "a" is used to identify the correlation between the Criminological Variable and Y, the dependent variable. Here it is important to realize the Criminological Variable is conceptualized as a *single* variable that a criminologist may be interested in studying. For instance, one may consider the Criminological Variable to be a proxy for a measure of self-control. The letter "b" represents the correlation between Y and the Genetic Factors. Recall the Genetic Factors variable represents a comprehensive measure of *all* (or most all) genetic effects on Y. The letter "c" identifies the correlation between the Criminological Variable and the Genetic Factors. All three correlations will be manipulated in the simulations that follow. Of most interest, however, is the effect of correlation "c" on the results from the simulation. As will be shown, the amount of bias present in any correlation between the Criminological Variable and Y (i.e., "a" from Table 1, b_1 from Eq. (1), and b_1' from Eq. (2)) will increase as a function of the correlation between the Genetic Factors and the Criminological Variable (i.e., "c").

Table 2 displays the results from the first set of simulations. To begin, both correlation "a" and correlation "b" were set to equal .20 (see the first column of the table). Correlation "a" was set to this value based on our review of meta-analyses and systematic reviews of key criminological variables such as self-control and delinquent peers (e.g., Pratt & Cullen, 2000; Pratt et al., 2010; Weisburd & Piquero, 2008). Correlation "b" (the correlation between the Genetic Factors and Y) was set to .20 in order to provide a meaningful, yet conservative, starting point for the analysis. Setting this value to .20 is almost certainly an *under*estimate given that behavior genetic research has found antisocial behavior to be approximately 50% heritable (see above), which might suggest a correlation of .70 is more realistic. We will consider higher—and more realistic—values of correlation "b" in later analyses. For the first simulation, however, it was important to establish a baseline set of findings where the effect of the Criminological Variable on Y and the effect of the Genetic Factors on Y were equal in magnitude.

Table 1
Correlation Matrix Utilized for Simulations

	Y	Criminological Variable	Genetic Factors
Y	-		
Criminological Variable	a	-	
Genetic Factors	b	c	-

Table 2
Parameter Estimates for b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .20, b = .20, c

Simulated Correlations		Results from Eq. (1)		Results from Eq. (2)		Comparison across Equations	
		mean of b_1	% $p < .05$	mean of b_1'	% $p < .05$	% Spurious	Over Estimate
a	0.20	0.20	100	0.18	100	0.10	1.11
b	0.20						
c	0.10						
a	0.20	0.20	100	0.16	99.7	0.20	1.25
b	0.20						
c	0.25						
a	0.20	0.20	100	0.14	98.2	0.30	1.43
b	0.20						
c	0.40						
a	0.20	0.20	100	0.13	94	0.35	1.54
b	0.20						
c	0.55						
a	0.20	0.20	100	0.12	76.5	0.40	1.67
b	0.20						
c	0.70						

The parameter value of interest in Table 2 was correlation "c" and the different rows in the table display the simulation results gleaned when "c" was set between .10 and .70. The Monte Carlo simulation was conducted five separate times: where (1) "c" = .10; (2) "c" = .25; (3) "c" = .40; (4) "c" = .55; and (5) "c" = .70. Column 2 ("mean of b_1") presents the mean effect of the Criminological Variable on Y in a bivariate regression (i.e., b_1) observed from 1,000 Monte Carlo simulations for Eq. (1). As can be seen, the average correlation across all of the simulated datasets was estimated to be .20, exactly as specified. Column 3 reveals that 100 percent of the estimates were statistically significant at the .05 significance threshold.

Column 4 ("mean of b_1'") reveals the average *semipartial* correlation between the Criminological Variable and Y after the influence of the Genetic Factors had been parsed out (i.e., b_1' from Eq. (2)). As can be seen, the mean effect of b_1' is, in every case, closer to zero than the corresponding effect of b_1. The impact of the Criminological Variable on Y ranges between .18 when "c" = .10 and .12 when "c" = .70. Column 5 displays an interesting pattern in terms of the percentage of cases that produced a statistically significant association between the Criminological Variable and Y after the Genetic Factors was included. Specifically, more than 90 percent of all cases were statistically significant when the "c" correlation ranged between .10 and .55. When "c" was set to .70, however, more than 20 percent of the cases failed to reach statistical significance. This suggests that in cases where the Criminological Variable and the Genetic Factors are highly correlated with one another, the effect of the Criminological Variable on Y may fail to reach statistical significance in roughly one out of four trials when the Genetic Factors variable is included in the model.

Column 6 in Table 1 reveals the percentage of the effect of the Criminological Variable on Y that was explained away (on average) after introducing the Genetic Factors—therefore, this column is labeled "% Spurious" to reveal the total amount of bias that was present before controlling for genes (i.e., $[b_1 - b_1']/ b_1$). Finally, Column 7 ("Over Estimate") reveals the average amount of bias present in the effect of the Criminological Variable on Y as a factor of the semipartial correlation (i.e., b_1/b_1'). Any value over 1 reveals the amount of bias that was present in terms of a factor change. For example, a result of 2.0 would indicate that b_1, as

compared to b_1', was an overestimate by a factor of 2. The simulation results reported in Table 2 revealed that omitted variables bias was present at all levels of "c" (i.e., the correlation between Genetic Factors and the Criminological Variable). For instance, when "c" was set to .10, the effect of the Criminological Variable on Y was overestimated by a factor of 1.11; the effect size dropped by 10% after genetic factors were controlled. When "c" was set to .70, the amount of bias present was increased to a factor of 1.67; a 40% drop in the parameter estimate from Eq. (1) to Eq. (2).

In order to aid in the interpretation of the results, a series of combined histograms were generated and are presented in Fig. 1. The figure has five panels and each corresponds to a simulation where "c" was set to a different value. Thus, the panels directly correspond to the rows presented in Table 2. Within each panel, two distributions are displayed. Chartered in red is the distribution of the b_1 parameter estimate (i.e., the effect of the Criminological Variable on Y) *before* the Genetic Factors was included in the model (i.e., Eq. (1)). The scores presented in black reflect the distribution of the b_1' parameter estimate (i.e., the effect of the Criminological Variable on Y) *after* the Genetic Factors variable was included in the model (i.e., Eq. (2)). Two points should be immediately obvious. First, the distributions show substantial overlap in all five panels. This reveals that, while a certain level of bias may be present in the specified conditions, it is unlikely that b_1 will be completely explained away after controlling for genetic factors in a regression model when the correlation structure is "a" = .20, "b" = .20, and "c" ~ .10-.70. The second point to notice, however, is that the two distributions share less overlap as the correlation value for "c" is set to higher (yet, realistic) values. For instance, when "c" = .10, the distributional overlap is nearly complete. When "c" = .70, however, only the tails of the distributions remain overlapping. These figures graphically illustrate the pattern of findings that were presented in Table 2.

The next set of simulation results is presented in Table 3. The table format is identical to Table 2. The key difference between Tables 3 and 2 is that the "b" correlation was set to .30 for the simulations presented in Table 3. Recall that the "b" correlation taps the relationship between the Genetic Factors and Y. Thus, Table 3 analyzes the association between the Criminological Variable and Y when the correlation between the Genetic Factors and Y is .30 and the "c" correlation ranges between .10 and .70. As before, the "b" correlation is, in all likelihood, an *under*estimate of the genetic influence on Y (if Y is conceptualized as antisocial behavior). Nonetheless, the pattern of results is substantively similar to those presented above with one key exception: the association between the Criminological Variable and Y is much more sensitive to omitted variables bias in this condition (i.e., when correlation "b" is set to a higher and more realistic value). This point can be easily grasped by observing the values in the % Spurious column. Note that even under weak correlation between the Criminological Variable and the Genetic Factors (i.e., correlation "c" = .10), the b_1 parameter will be overestimated by a factor of 1.18 on average as compared to b_1'; 15% spurious. As the "c" correlation is adjusted upward the percent spurious value increases drastically, hitting 100% when "c" = .70.

As before, the parameter estimates for b_1 and b_1' were plotted alongside one another and are presented in Fig. 2. Looking from left to right, the figures clearly show the divergence in the distribution of the parameter estimates before (red bars) and after (black bars) the Genetic Factors variable is included in the regression model. Note that the

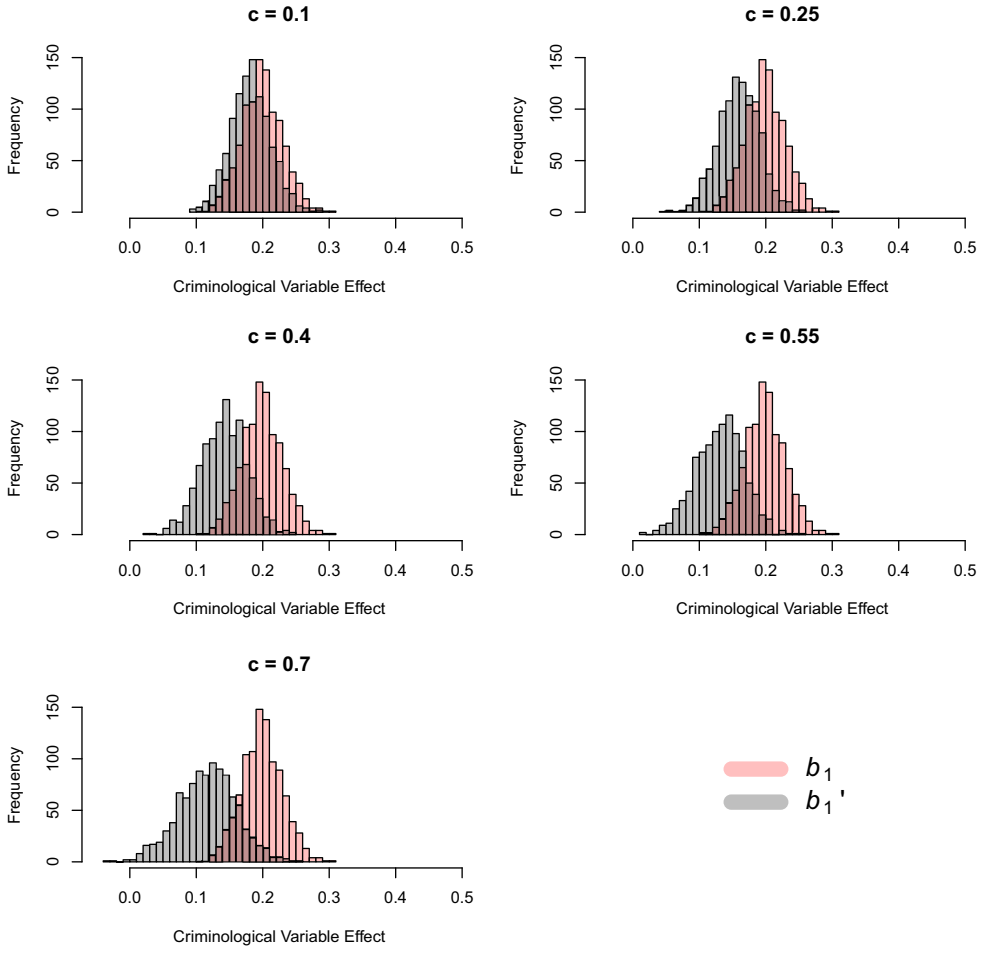

Fig. 1. The Distribution of b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .20, b = .20, c.

Table 3
Parameter Estimates for b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .20, b = .30, c

Simulated Correlations		Results from Eq. (1)		Results from Eq. (2)		Comparison across Equations	
		mean of b_1	% $p < .05$	mean of b_1'	% $p < .05$	% Spurious	Over Estimate
a	0.20	0.20	100	0.17	100	0.15	1.18
b	0.30						
c	0.10						
a	0.20	0.20	100	0.13	98.7	0.35	1.54
b	0.30						
c	0.25						
a	0.20	0.20	100	0.09	83.3	0.55	2.22
b	0.30						
c	0.40						
a	0.20	0.20	100	0.05	31.6	0.75	4.00
b	0.30						
c	0.55						
a	0.20	0.20	100	-0.02	8.6	1.00	-
b	0.30						
c	0.70						

distributions are more differentiated in Fig. 2 than they were in Fig. 1. In short, the likelihood that the relationship between a Criminological Variable and Y is spurious will increase as a function of the proportion of variance in Y that is attributable to genetic factors. Taking the results from the first two sets of simulations into consideration leads to one obvious conclusion: correlation "a" may be upwardly biased—and in some cases completely spurious—when the genetic influence on both variables (i.e., Y and the Criminological Variable) is in the moderate-to-high range.

Reported in Table 4 are the results of five additional simulations. As before, the "c" correlation was adjusted between .10 and .70. What is unique about the simulation results displayed in Table 4 is that the "a" and "b" correlations differ from those presented above. These five simulations were estimated when correlation "a" was set to .30 (i.e., a larger criminological influence) and correlation "b" was set to .20 (i.e., less genetic influence on Y). An interesting pattern of results emerged under these conditions. Specifically, when "c" was set to values between .10 and .55, the b_1 parameter was found to be an overestimate of b_1' roughly by a factor of 1.10—arguably a negligible amount. Interestingly, however, when the "c" correlation was set to .70 (substantial overlap between the Criminological Variable and the Genetic Factors), the findings suggest that omitted variable(s) bias is not a concern. Indeed, the b_1' parameter was, on average, larger than the b_1 parameter. To summarize the results in Table 4, it appears that genetic confounding is less problematic under conditions where the correlation between the Criminological Variable and Y is larger in magnitude than the correlation between the Genetic Factors and Y (i.e., $r_{yx} > r_{yg}$). The five combined distributions presented in Fig. 3 align well with this conclusion by revealing a substantial degree of overlap in the distribution of b_1 and b_1'.

The final set of simulation results is presented in Table 5 and the distributional comparisons are presented in Fig. 4. This set of simulations was estimated on a correlation structure where "a" = .30, "b" = .70, and "c" ranged between .10 and .70. It is important to note the "b" correlation was raised substantially as compared to the previous analyses. The correlation of .70 was chosen because it represents the most

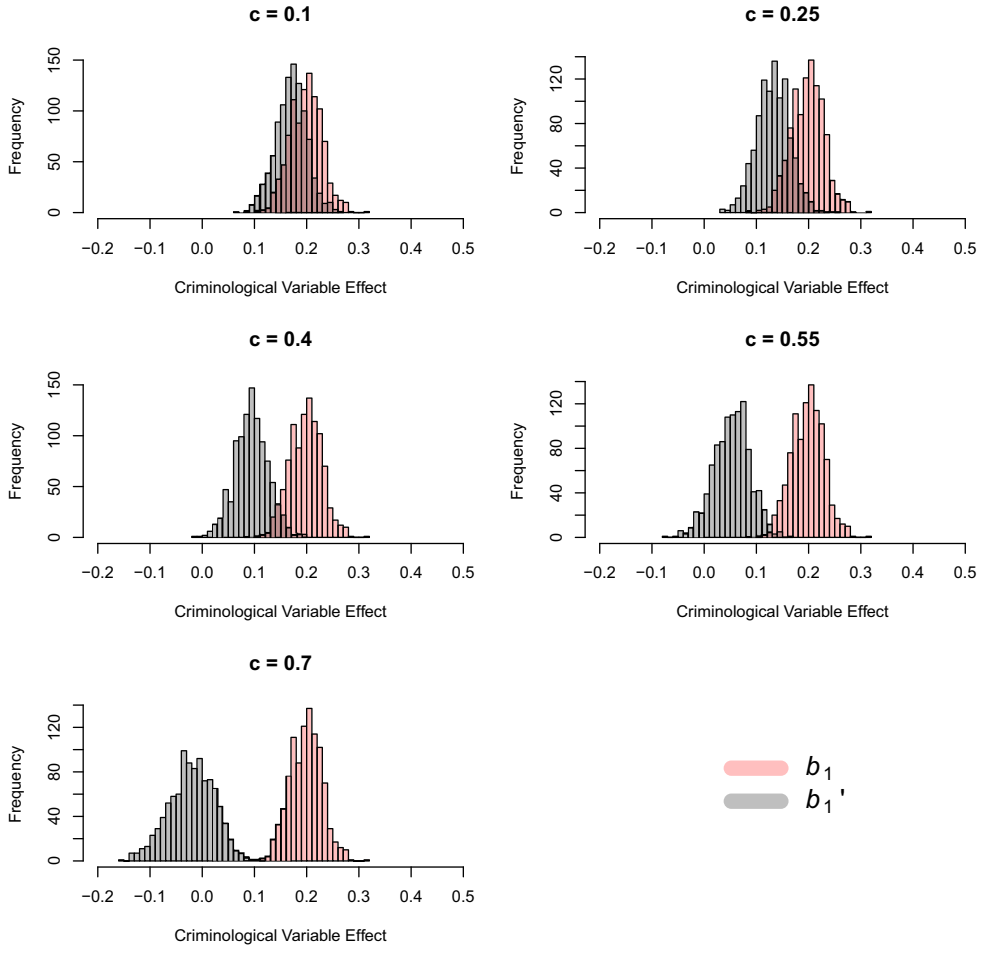

Fig. 2. The Distribution of b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .20, b = .30, c.

Table 4
Parameter Estimates for b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .30, b = .20, c

Simulated Correlations		Results from Eq. (1)		Results from Eq. (2)		Comparison across Equations	
		mean of b_1	% $p < .05$	mean of b_1'	% $p < .05$	% Spurious	Over Estimate
a	0.30	0.30	100	0.28	100	0.07	1.07
b	0.20						
c	0.10						
a	0.30	0.30	100	0.27	100	0.10	1.11
b	0.20						
c	0.25						
a	0.30	0.30	100	0.26	100	0.13	1.15
b	0.20						
c	0.40						
a	0.30	0.30	100	0.27	100	0.10	1.11
b	0.20						
c	0.55						
a	0.30	0.30	100	0.31	100	-0.03	-
b	0.20						
c	0.70						

Table 5
Parameter Estimates for b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .30, b = .70, c

Simulated Correlations		Results from Eq. (1)		Results from Eq. (2)		Comparison across Equations	
		mean of b_1	% $p < .05$	mean of b_1'	% $p < .05$	% Spurious	Over Estimate
a	0.30	0.30	100	0.23	100	0.23	1.30
b	0.70						
c	0.10						
a	0.30	0.30	100	0.13	100	0.57	2.31
b	0.70						
c	0.25						
a	0.30	0.30	100	0.02	15.0	0.93	15.00
b	0.70						
c	0.40						
a	0.30	0.30	100	-0.12	0.00	1.40	-
b	0.70						
c	0.55						
a	0.30	0.30	100	-0.37	0.00	2.23	-
b	0.70						
c	0.70						

realistic estimate of the correlation between the Genetic Factors variable and Y given that $h^2 = .50$ for antisocial behavior and $\sqrt{.50} = .71$. For this reason, results from the final set of simulations are probably the most meaningful to the larger discipline. The pattern of results under these conditions—with one important distinction—are in line with those obtained in Tables 2 and 3. Specifically, as the "c" correlation increases, the divergence between b_1 and b_1' grows. What is distinct about this set of results is that the divergence between b_1 and b_1' expands at a much faster rate than was seen previously. To be sure, at even modest levels of the "c" correlation (e.g., "c" = .40), the % Spurious column reveals a nearly completely spurious result (93%). In the latter two simulations, where "c" = .55 and "c" = .70, the b_1 parameter was

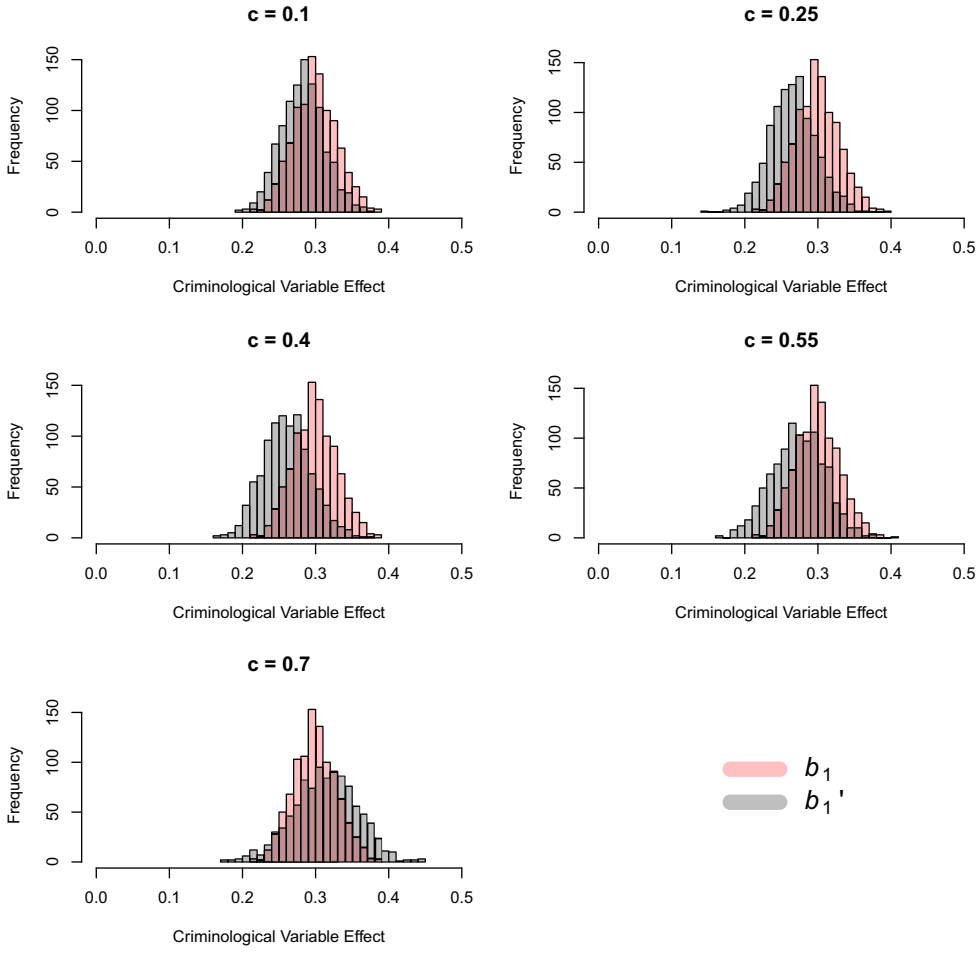

Fig. 3. The Distribution of b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .30, b = .20, c.

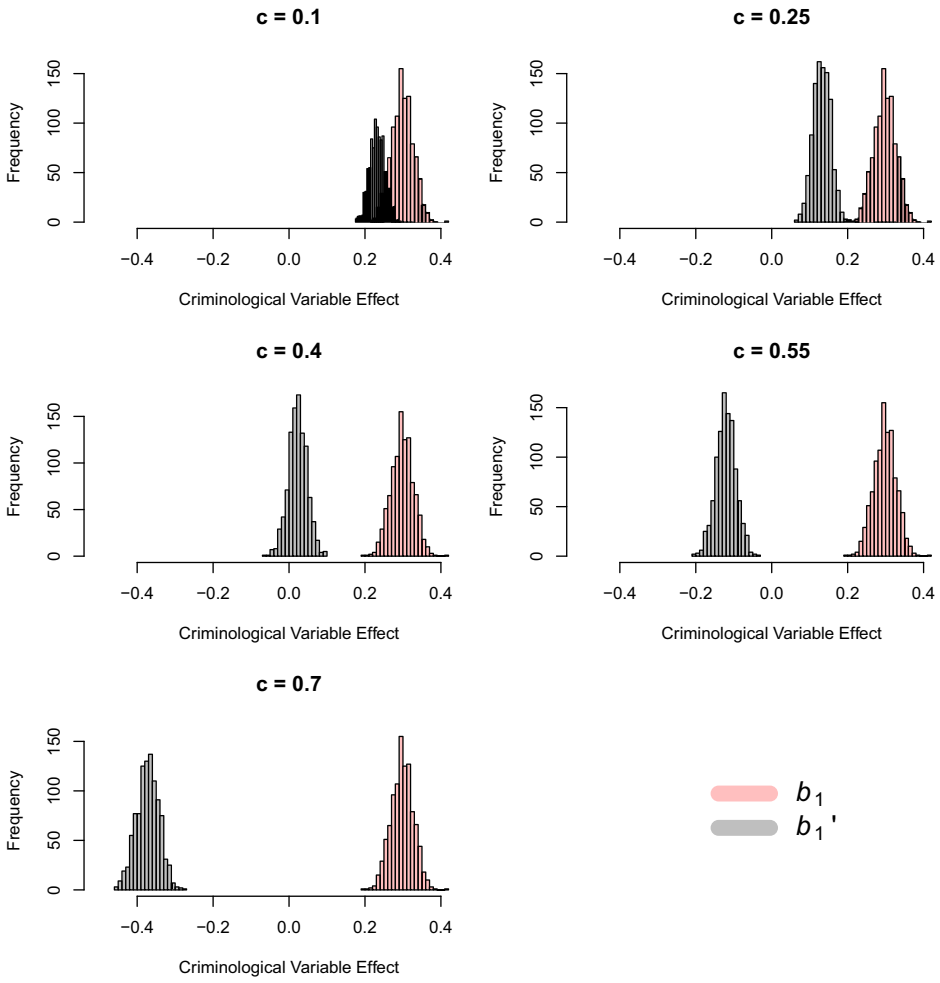

Fig. 4. The Distribution of b_1 and b_1' at Different Levels of Genetic Confounding When Correlation Values Set to: a = .30, b = .70, c.

not only overestimated but the analysis indicated that b_1' was *negative*. As before, the distributions of b_1 and b_1' are presented in Fig. 4. These joint histograms reveal, graphically, the same pattern of findings shown numerically in Table 5. Note that the two distributions no longer overlap once "c" is set to be greater than or equal to .40.

Discussion

By many accounts, the criminological discipline was born out of an attempt to associate biology with criminality (Lombroso, 1895; Raine, 2013). Although many suggestions made by early biologically oriented researchers represented logic that was off the mark in varying degrees, science progresses iteratively and with the passage of time biosocial research has achieved important insight into the etiology of human behavior (Pinker, 2002; Raine, 2013). With these points in mind, the purposes of the current paper were twofold. First, evidence from contemporary biosocial research has forced several "hard" questions upon the broader criminological discipline and, therefore, our goal was to provide an objective review of the issues at hand.

As was demonstrated in the literature review, there are hundreds of papers that reveal a genetic influence on antisocial behaviors. By itself, this finding poses little problem for criminologists. Indeed, behavior genetic research has shown that, on average, only about half of the variance in antisocial behavior is attributable to genetic influences, leaving the other 50% open to environmental explanations (e.g., Burt, 2009a,b; Rhee & Waldman, 2002). Our review of the literature did not stop with research into the etiology of antisocial behavior, however. Instead, empirical research into the association between genetic influences and environmental exposures (via rGE mechanisms) was also considered. This line of research indicates that many of the outcomes typically considered purely social in origin may be partially attributable to genetic factors as well (Kendler & Baker, 2007; Turkheimer & Waldron, 2000). These two streams of evidence—one showing genetic influences on antisocial behavior and another showing genetic influences on variables typically used as predictor variables by criminologists—lead naturally to the second goal of the current study.

In particular, the second goal of the current paper was to address a central concern raised by biosocial research: to what degree is criminological research biased when controls for genetic factors are omitted? Recall that the default methodological approach is to utilize a research design that cannot fully (if at all) account for genetic influences (i.e., the SSSM). The problem of statistical endogeneity is endemic to social science research, but it is our position that a prominent confounder variable—genetic influences—has largely gone overlooked. To briefly summarize our concern: if genetic influences underlie the etiology of antisocial behavior (see the meta-analyses discussed above Burt, 2009a,b; Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002) *and* the etiology of common criminological variables such as self-control (Boisvert et al., 2012; Nikolas & Burt, 2010), peer group formation (Cleveland et al., 2005), and broader social

environments (Kendler & Baker, 2007; Reiss et al., 2003), then the possibility that a researcher will *over*estimate the correlation between X and Y (where X is a common criminological variable and Y is antisocial behavior) is non-zero unless an explicit attempt to control for genetic factors via modern behavior genetic designs such as the discordant twins model is made (see Cleveland, Beekman, & Zheng, 2011).[6]

Summary of findings

In order to provide a meaningful assessment of the problem of genetic endogeneity in criminological research, the current analysis drew on information from a series of simulated datasets and Monte Carlo estimation techniques. The goal for the analysis was simple: estimate the degree to which the correlation between common criminological variables and antisocial behavior is *over*estimated due to the omission of control variables tapping genetic influences. By consulting meta-analyses (e.g., Pratt & Cullen, 2000; Pratt et al., 2010), the range of correlations between standard criminological variables and antisocial behavior (i.e., correlation "a") was established to be between $r = .20$ and $r = .30$. Next, drawing on the behavior genetic literature, it was established that the correlation between genetic factors and antisocial behavior (i.e., correlation "b") would likely range somewhere between $r = .50$ and $r = .70$. Despite these high correlations, the simulation procedures consistently used values of $r = .20$ and $r = .30$, with the exception of the final set of simulations which utilized the most defensible correlation value of $r = .70$. This strategy was used in order to provide meaningful, yet conservative, estimates of the problem of confounding (more on this below). Finally, the correlation between genetic factors and criminological variables (i.e., correlation "c") is likely to range between $r = .30$ and $r = .60$. These values were calculated based on the conclusions reached by Kendler and Baker (2007). In order to provide an examination of a broad range of possibilities, the correlation between the genetic variable and the criminological variable ranged between $r = .10$ and $r = .70$ in the simulation analysis. This strategy provided estimates of the problem of confounding with the range of possibilities likely facing criminologists.

Although there is a great deal of nuance that must be considered, the general conclusions of the present study can be summarized into three brief statements. First, genetic confounding may account for a negligible portion of any observed relationship between a criminological variable and antisocial behavior when the correlation between the criminological variable and antisocial behavior is larger than the correlation between genetic factors and antisocial behavior. Second, in cases where the influence of genetic factors on antisocial behavior is greater than or equal to the influence of a criminological variable, confounding becomes more problematic as the correlation between genetic factors and antisocial behavior increases. Recall that our simulations set the correlation between genetic factors and antisocial behavior to be either $r = .20$, $r = .30$, or $r = .70$. Estimates suggested that confounding was more problematic as the genetic effect on antisocial behavior was set to higher—and more realistic—values. It is important to also note that the final set of simulations—where "b" = .70—revealed that the actual influence of a criminological variable on antisocial behavior could not only be overestimated but that researchers may even conclude that the effect is in the *wrong direction* (see Harden, Mendle, Hill, Turkheimer, & Emery, 2008).

The third conclusion drawn from the present analysis is that the degree to which the correlation between a criminological variable and antisocial behavior is confounded is directly tied to the degree to which genetic factors influence the criminological variable. One pattern emerged across all but one of our simulations. As the correlation between genetic factors and the criminological variable increased, the degree to which the effect of the criminological variable on antisocial behavior was confounded increased as well. In some of the most extreme examples, 100% of the association between the criminological variable and antisocial behavior was explained away due to genetic confounding. In the future, scholars may wish to draw on the present results in an effort to estimate the degree to which a correlation is biased under particular conditions. This could easily be accomplished by simulating a single variable with a pre-defined correlation structure between the dependent variable and the primary independent variable.

Assumptions and limitations

It is important that we disclose the assumptions and limitations of the analysis. Four points should be revisited and made explicit here. First, recall that the analysis relied entirely on standard normal variables. Specifically, the simulated datasets were created such that the distribution of values for each variable was approximately normal. As any scholar of crime knows, however, the distribution of antisocial behavior scores in a dataset can rarely be classified as normal. We chose to simulate Y as a normal variable for two reasons. First, from a statistical standpoint, the normal distribution has desirable properties that allow for the estimation of parametric analyses such as the OLS regression model (which was relied upon in the current study). Second, the substantive conclusions were identical when the variables were coded in alternative ways. For instance, the substantive findings were identical when the Genetic Factors variable was coded as a positive integer with a uniform distribution. Also, substantive findings were unchanged when Y was coded as a dichotomous variable (where 1 = scores at or above 1 standard deviation from the mean, roughly the 80th percentile) and the simulations were re-estimated with a logistic regression model.

The second point to be kept in mind when considering the evidence presented here is that there may be an imperfect correspondence between a heritability estimate and a correlation between that trait and genetic factors. As noted earlier, taking the square root of a heritability estimate ($r \approx \sqrt{h^2}$) provides an approximation of the correlation between genetic factors and the variable of interest. In order to account for any imprecision we followed a two-pronged approach. First, we do not rely on one estimate of the genetic correlation when drawing conclusions. Quite the opposite; we relied on the preponderance of the evidence gleaned from simulations where the correlation was set to a wide range of different values. Second, when drawing conclusions we relied heavily on what were considered to be the most conservative estimates available. In this way, we have consciously attempted to avoid presenting the most radical set of findings and have instead opted for a set of results that have practical and substantive meaning.

Third, the present analysis assumes perfect correspondence between the genetic factors affecting the Criminological Variable and the genetic factors affecting Y. In the language of modern behavior genetic research, our simulations have assumed that the *genetic* correlation (i.e., *rg*) between the Criminological Variable and Y is 1.0. It is important that this assumption be kept in mind when generalizing our findings to one's own research because the degree to which genetic confounding is problematic will vary directly as a result of the genetic correlation between the Criminological Variable and Y. All other factors being equal, two variables linked by a high (e.g., 1.0) genetic correlation will be more susceptible to genetic confounding as compared to two variables linked by a low genetic correlation. This point highlights the importance of behavior genetic research that seeks to unpack the genetic correlation between common criminological predictors and outcomes. The analyses by Boisvert et al. (2012) and Fowler et al. (2007) serve as illustrious examples.

Finally, a fourth issue concerning the delineation of confounding and mediation must be considered. Mathematically, it is impossible to separate a confounder variable from a mediator variable in the context of a regression analysis. This means that the only way to separate a confounder variable from a mediator variable is by utilizing common sense, logic, and theoretical expectation. Our analysis has

assumed that genetic factors will always operate as a confounder variable but this need not be the case. On the contrary, genetic effects on Y can—and most certainly are—mediated by sociological phenomena. Here, the consideration of gene-environment correlations (*r*GE) is important and readers should be sensitive to this issue when seeking to generalize the results from the present study to their own work.

Recommendations and conclusion

The results summarized above reveal an important issue that can no longer be overlooked by criminological scholars. Though a common reaction on the part of researchers to findings such as this might be to include more control variables in their regression models, we caution that this approach may do little to circumvent the problem. Indeed, it is unlikely that a genetic influence can be "controlled away" by including covariates commonly available in criminological data sets. The best approach to controlling away genetic influences is more holistic research designs that include twins, siblings, or other individuals with known genetic relationships. By doing so, researchers will then be able to utilize behavior genetic modeling to rule out genetic influences. Specifically, research designs that allow for the estimation of *between-sibling* differences are a viable and rather convenient approach (see Cleveland et al., 2011). Other approaches such as fixed effects analysis where the respondent serves as his/her own control over many observation points may also provide a suitable—though incomplete (see footnote 6)—way to account for *stable* (but not *dynamic*) genetic influences.

The mounds of evidence indicating a genetic influence on antisocial behavior and the emerging evidence suggesting that common criminological variables also operate under partial genetic influence have forced criminology to a crossroads. As outlined above, the evidence is clear that genes play a role in the etiology of antisocial behavior (Barnes et al., in press). Moving forward, the most important questions facing the current generation of scholars are 1) how *much* do genes matter and 2) how much of the correlation between a given criminological variable and antisocial behavior is attributable to a shared genetic etiology (i.e., omitted variables bias). We see the latter as one of, if not *the*, most important issues facing the discipline today and we hope that the present study will spark an objective and honest assessment of where the discipline has been, where the discipline currently is, and where the discipline is headed.

Notes

[1] Indeed, as an anonymous reviewer pointed out, a paradigm change is often preceded by an "extinction burst" where advocates of the 'old' paradigm are hostile toward the 'new' paradigm with increasingly elaborate claims about the validity of 'their' theory.

[2] Note that our discussion will be limited to individual-level theories/variables. This will be important when we begin the analysis and certain average correlations are set.

[3] Although one could analyze siblings, specifically monozygotic twins, who do *not* live together to estimate heritability (see Bouchard et al., 1990). Another option is to analyze adoptees. Neither strategy is common.

[4] It is worth pointing out that it is unclear whether there is perfect overlap between heritability estimates and standardized regression coefficients (i.e., [semipartial] correlations). In other words, a correlation of .70 between a genetic factors variable and a criminological variable may not necessarily translate perfectly into a heritability of $.70^2 = 49\%$. This was one of the main reasons we opted to present different scenarios in the findings section and why we refer to these as *approximations*.

[5] *N* of 1,000 was chosen because this value is consistent with prior simulation studies in the criminological discipline (Skardhamar, 2010) and a sample of size of 1,000 is not uncommon in the criminological literature. Also, $N = 1{,}000$ allowed for the observation of variance in the parameter estimate distribution.

[6] Longitudinal modeling strategies do account for certain *stable* genetic influences but are unable to account for the full lot genetic factors. In short, analyses of intra-individual changes (i.e., dynamic analyses) will only control away genetic influences on stability in the observed outcome. The current understanding of the association between genetic factors and human complex traits (e.g., antisocial behavior) suggests that genetic influences can ebb and flow across the life course (Plomin et al., 2013). These influences are not controlled in longitudinal designs.

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