CRIMINOLOGY

PULLING BACK THE CURTAIN ON HERITABILITY STUDIES: BIOSOCIAL CRIMINOLOGY IN THE POSTGENOMIC ERA*

CALLIE H. BURT and RONALD L. SIMONS
School of Criminology and Criminal Justice, Arizona State University

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Unfortunately, the nature-versus-nurture debate continues in criminology. Over the past 5 years, the number of heritability studies in criminology has surged. These studies invariably report sizeable heritability estimates (~50 percent) and minimal effects of the so-called shared environment for crime and related outcomes. Reports of such high heritabilities for such complex social behaviors are surprising, and findings indicating negligible shared environmental influences (usually interpreted to include parenting and community factors) seem implausible given extensive criminological research demonstrating their significance. Importantly, however, the models on which these estimates are based have fatal flaws for complex social behaviors such as crime. Moreover, the goal of heritability studies—partitioning the effects of nature and nurture—is misguided given the bidirectional, interactional relationship among genes, cells, organisms, and environments. This study provides a critique of heritability study methods and assumptions to illuminate the dubious foundations of heritability estimates and questions the rationale and utility of partitioning genetic and environmental effects. After critiquing the major models, we call for an end to heritability studies. We then present what we perceive to be a more useful biosocial research agenda that is consonant with and informed by recent advances in our understanding of gene function and developmental plasticity.

Questions about nature versus nurture have been a perennial topic of debate in the social sciences. Since the 1970s, these questions have been addressed by a field of study known as behavioral genetics. A major focus of behavioral genetics research has been to partition the variation in an outcome of interest into a proportion caused by genes (heritability) and a proportion caused by the environment (e.g., DiLalla, 2004; Plomin et al., 2012). These heritability studies (also called quantitative genetics, nonmolecular genetics, or biometrics) have compared phenotypes (observed characteristics of individuals) within

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and between families that vary in genetic relationships to estimate the influence of genes. The general conclusion emerging from heritability studies is that variance in almost every human characteristic—including preferences and social behaviors—is shaped significantly by genetic influences (Plomin et al., 2012; Turkheimer, 2000). For example, studies have shown substantial heritability (usually between 40 and 60 percent) of everything from time spent watching television (Plomin et al., 1990), breastfeeding (Colodro-Conde, Sanchez-Romera, and Ordonana, 2013), and breakfast eating patterns (Keski-Rahkonen et al., 2004) to political ideology and party affiliation (Alford, Funk, and Hibbing, 2005) and delinquency (Wright et al., 2008).

Since 2008, behavioral genetic studies by criminologists investigating the heritability of criminal behavior and factors associated with crime have been published at a rapid and seemingly increasing pace. Indeed, every few months new studies estimate the heritability of some aspect of criminality, such as self-reported delinquency (e.g., Boisvert et al., 2012; Wright et al., 2008), arrests (Beaver et al., 2012), self-control (e.g., Beaver et al., 2008, 2009), and gang membership (Barnes, Boutwell, and Fox, 2012). Notably, behavioral geneticists have been estimating the heritability of antisocial behavior, delinquency, and criminal convictions for years prior to this recent surge (e.g., Mednick, Gabrielli, and Hutchings, 1984; Rowe and Osgood, 1984; see Moffitt, 2005, for a review). These studies have revealed ostensibly that genetic factors explain a substantial portion—between 30 and 90 percent of the variance—of every examined crime-related phenotype with shared environmental factors playing a minimal role (often reported to be 0 percent).

We are surprised that these somewhat astonishing findings reported in recent studies, such as the reports of more than 50 percent heritability for such complex social behaviors as crime and victimization, have not generated more critical attention in criminology. We also are perplexed by the lack of response to the heritability study finding that so-called shared environmental factors play a minor role in explaining variation in crime-related phenotypes (e.g., Barnes, Boutwell, and Fox, 2012; Beaver et al., 2008; Beaver, Ferguson, and Lynn-Whaley, 2010; Boisvert, Wright, et al., 2013). Indeed, the conclusion from many of these heritability studies that little—if any—of the variance in criminal behavior is due to shared environments, often interpreted to include parenting and community factors, contradicts a wealth of research conducted during the past century as well as the major theories of crime. As the renowned psychiatrist and behavioral genetics practitioner Michael Rutter (2006: 11) noted, "[The] sweeping assertions on the irrelevance of the family environment are not supported by research evidence. It is quite striking that behavioral genetics reviews usually totally ignore the findings on environmental influences. It is almost as if research by non-geneticists is irrelevant."

The lack of critical attention to heritability studies in criminology is even more conspicuous given their known limitations. Since at least the early 1930s, scholars—including prominent geneticists, neuroscientists, and molecular biologists—have been warning about the fallibility of heritability studies in human populations, especially for complex social behaviors such as crime (e.g., Joseph, 2004; Kamin, 1974; Lewontin, Rose, and Kamin, 1984; Wahlsten, 1990; Wilson, 1934). As we discuss in this article, these methodological problems are not merely a sampling or measurement issue that can be corrected with various model adjustments but are *inherent to the method itself*. Remarkably, as we discuss, many of the recent criminology studies have failed to mention crucial assumptions and technical limitations, and even fewer have discussed the implications of their violation, especially for assumptions whose violations bias toward heritability.

It is not merely the techniques of heritability studies that are suspect, but also the basic conceptual framework of heritability studies is unsound (e.g., Charney, 2008; Crusio, 2012; Turkheimer, 2011). Heritability studies rest on a model of gene function that views genetic effects as independent and separable from the environmental context in which they operate. Genes, however, do not work this way. Genes are not a self-activating code that can be understood apart from environmental inputs but are only one part of an interactive, developmental biopsychosocial system. As we will discuss, genes and the environment are not separate analytical entities, and thus, it is biologically nonsensical to attempt to partition genetic from environmental influences on phenotypes (e.g., Gottlieb, 2001; Greenberg, 2011).

Although the problems of quantitative genetic methods have been discussed elsewhere (see, e.g., Charney, 2008, 2012; Joseph, 2004, 2006; Lewontin, Rose, and Kamin, 1984; Rutter, 2006), and in criminology several decades ago or more (e.g., Walters and White, 1989), as yet there has been no critical response to the recent profusion of heritability studies in criminology. Given the significant advances in our understanding of gene function and the profound theoretical and policy implications of this work, such a response is sorely needed. It is our objective in this article to provide such a response. Notably, most of the arguments in this article are not original but are those of prominent scientists, many of whom we cite, whose criticisms have been largely unheeded by the criminological community in recent years. We hope to renew a dialogue in criminology about heritability studies and stimulate what we view as a much-needed debate about the utility of heritability studies for crime and related phenotypes.

The objectives of this article are twofold. First, we critique heritability study methods and assumptions so that the criminological community can be more informed consumers of these findings. Our critique is grounded in both methodological and conceptual issues. Regarding the methodological critique, we argue that heritability studies are seriously flawed and, thus, do not provide useful estimates of genetic influences on criminal phenotypes. As we will show, these technical flaws have the effect of biasing estimates toward inflating heritability and underestimating shared environmental effects. Second, we argue that, methodological problems aside, partitioning individual differences into genetic versus environmental influences is a misguided endeavor in the first place. Drawing on a variety of sources, including the arguments of prominent behavioral geneticists (e.g., Rutter, 2006; Turkheimer, 2011), we call for an end to heritability studies in criminology and recognition of the problematic nature of existing heritability estimates for criminal phenotypes.

To be clear at the outset, it is not the position of this article that genes do not contribute to individual differences in behavior. Criminal behavior, like all other behavior, results from a combination of factors, including environmental and genetic ones. Any claim to the contrary is patently false. We do not wish to move the field toward extreme cultural or social determinism. Instead, we attempt to tackle these issues and put them in a new perspective, not by denying the role of genes or other biological factors but by recognizing the complexity of the biopsychosocial system. Heritability studies do not resolve the outworn nature-versus-nurture debate; they promote it. Successfully leveraging the advances of the genomic era in the new era of postgenomics requires that we move beyond heritability, transcending the outdated question of how much significance to attribute to genetics versus the environment in the development of particular behaviors and traits (Lickliter, 2009).

Thus, in the final sections of the article, we challenge scholars interested in biosocial work in criminology to move beyond heritability toward research grounded in the reality that genetic effects on human behavior are meaningful only when considered in combination with environmental influences. Notably, many criminologists who are involved in heritability studies also engage in biosocial research consistent with the postgenomic biosocial agenda encouraged in this article. With this work, these scholars have advanced biosocial criminology and our own understanding of biosocial mechanisms. This article is not a criticism of scientists but a critique of a particular method.

Our argument unfolds in four sections. First, after a brief primer on genetics, we discuss the concept of heritability—a concept that is often and easily misunderstood—and its quantified form, the heritability coefficient, as well as the foundational assumptions of heritability studies. Next, we turn our attention to the various methods that behavioral geneticists use to estimate heritability. We begin with the most common design, the twin study. The fundamental assumptions of this approach are detailed, followed by a discussion of the way that using this method to study criminal and antisocial behavior violates crucial assumptions. Then, we discuss the other prominent method in quantitative genetics used to estimate the heritability of criminal phenotypes: adoption studies. We also briefly discuss the third heritability model, twins reared apart studies, which is less relevant to criminology given its lack of use in this domain but is still cited as convergent support.

Although evidence from the different methods are used to "provide convergent findings," given that "each of the primary designs used by behavioral geneticists has its own Achilles heel(s)" (Moffitt, 2005: 57), we show that all of these models are biased toward *inflating heritability* and *underestimating shared environmental influences*. After this discussion of the methodological limitations in heritability studies, we turn our attention to the misguided idea that we can partition genetic and environmental influences on complex phenotypes. Finally, having presented what we believe to be compelling arguments for abandoning heritability studies in criminology, we briefly present examples of biosocial research that are consonant with and informed by the postgenomic paradigm that views genes and the environment as inextricably linked and in a dynamic relationship.

HERITABILITY

GENES AND ALLELES

Genes are segments of DNA coded for the production of RNA molecules and specific proteins organized on chromosomes. Individuals possess two copies of each gene in all cells, with a couple of exceptions; one copy comes from the egg and one from the sperm. Each copy of the gene is known as an allele of that gene. For the overwhelming majority of genes, only one allele exists. However, some genes vary across the population, such that there are at least two "versions" of the allele (Beaver, 2008). Polymorphisms are versions that occur in more than 1 percent of the population, whereas versions that occur in less than 1 percent of the population are known as mutations. The term "genotype" is commonly used to refer to whether an individual possesses a particular allele or the alleles for a particular gene, and the term "genome" refers to an individual's entire DNA sequence (Charney, 2012; Schaffner, 2006).

Although in the mid-twentieth century it was thought that genes code for proteins in a straightforward manner, we now know this is not the case. DNA is not a self-activating code. Foreshadowing our later discussion, the process of gene activation, often referred to as "gene expression," is a multistep, multifactorial process. Genes are now understood to operate more as catalysts; they exist in cells that have many components and "function in a manner akin to chemicals in a test tube. Everything in the test tube affects everything else in the test tube; so too, everything in the cell affects genes" (Greenberg, 2011: 177). Importantly, the chemistry of the cell is influenced by environmental factors, such as a person's diet, exercise, exposure to various elements, and the like. Thus, environmental influences are important determinants of gene activation or expression (Jablonka and Lamb, 2006).

Moreover, evidence suggests that the genome itself is dynamic. DNA is composed of transposable elements—deletions, insertions, and rearrangements—that can alter the genetic sequence (the "genetic code"; Charney, 2012). Indeed, one of the more remarkable findings from the Human Genome Project is that approximately 45 to 48 percent of the genome is composed of such transposable elements (Lander et al., 2001). Evidence suggests that changes in DNA sequencing continue throughout life, can be inherited, and seem to be environmentally responsive (see Charney, 2012). Although there is much that scientists still do not know, it is increasingly clear that partitioning genetic and environmental influences is biologically nonsensical. Yet, attempts to do just that continue in criminology.

HERITABILITY CONCEPT

The heritability concept was originally designed for use in agricultural research to predict the outcomes of controlled breeding and thereby to assist animal and plant breeders in selecting for desirable traits. Since the 1970s, this concept has been promoted as a "nature–nurture ratio" of the relative influence of heredity (via genes) and environmental experiences on particular traits. However, this concept is frequently misunderstood (Joseph, 2006).

Heritability was defined by Wahlsten (1990: 244) as "the proportion of variance in a measure of behaviour or other phenotype in a breeding population that is attributable to genetic variation" and by Plomin et al. (2012: 87) as "the proportion of phenotypic variance that is accounted for by genetic differences among individuals." The heritability coefficient is the numeric index of heritability ranging from .0 (no genetic contribution) to 1.0 (complete heritability). The estimation of this parameter involves a model based on Mendelian principles as well as on several assumptions (discussed later).

Notably, heritability refers to the genetic contribution to trait variability in a population, not to the import of genetic factors as they influence the individual (Joseph, 2004; Plomin et al., 2012). Heritability estimates are often wrongly interpreted by laypersons (and some scholars) as the proportion of a trait that is caused by genetic influence rather than as the proportion of population variance in a trait. The methods of Mendelian genetics are responsive only to the slight portion of genes that are polymorphic and make us different; they do not allow for conclusions about the role of heredity in general (Wahlsten, 1990). As Joseph (2004: 138–9) noted, "This leads to a paradoxical situation in which a trait could be 100% inherited, yet have a heritability of zero—human beings having two eyes for example." Genetics, of course, explains humans' "eyedness"; we have two eyes

because of our genetic endowment. Because everyone (or nearly everyone) who has fewer than two eyes is that way as a result of life experiences, the heritability of "eyedness" is zero (see also Lewontin, Rose, and Kamin, 1984).

Although in the past some behavioral geneticists have used genetic findings as a basis for arguing that heritability estimates within groups can be used to explain differences between groups (e.g., Herrnstein and Murray, 1994; Jensen, 1969), these arguments are now widely understood to be fallacious. Heritability estimates are time and population specific. As such, findings of genetic influences on differences within populations do not extrapolate into explaining differences observed in traits across populations (Plomin et al., 1997). This aspect of heritability is wonderfully illustrated by Lewontin, Rose, and Kamin (1984). Suppose one takes two handfuls of heterogeneous corn seed and plants one handful on a field of nutrient-rich soil and the other in a field of nutrient-poor soil. When the seeds have grown, we can observe that there is variation within fields in plant height as well as variation between fields, specifically with lower plant height for the poor-quality-soil field. Not allowing any environmental variation within the respective fields (equal light, water, etc.), differences in the resulting plant height *within* the fields are totally the result of genetic factors (heritability = 1.0). Differences in plant height *between* the two fields, however, are completely the result of environmental factors, specifically, soil quality (heritability = 0). This example demonstrates that even for trait variance that is entirely heritable within a population, the cross-population variance may be completely caused by environmental factors (Joseph, 2004).

Importantly, heritability estimates do not speak to the responsiveness of a phenotype to environmental intervention. Traits can be highly heritable and yet be drastically altered or eliminated by changes in the environment (e.g., Lewontin, 1974; Plomin et al., 2012). Moreover, the heritability of a trait can change as a result of changes in the environment. Returning to the cornfield example, if one were to plant trees around the corn field, thereby producing unequal sun exposure among the corn plants within a field, then heritability would decrease as the resulting variation in sunlight exposure would account in part for differences in plant height.

HERITABILITY STUDIES

According to the prevailing behavioral genetics methodology, the heritability of a given phenotype is determined by comparing concordances and discordances between subjects relative to their presumed degree of genetic similarity. As noted, the index used to capture the relative strength of genetic influence on the population variance of a phenotype is the heritability coefficient. Although the word "heritability" has been around since the 1800s, the concept as it is used today was introduced by Lush in 1936, who was interested in facilitating agricultural breeding for economically desirable traits (Bell, 1977). Animal and plant breeders needed a quantifiable method for predicting the results of programs of selective breeding. For Lush (1949: 359), knowing "whether heritability is high or low is important when *making efficient breeding plans*" (emphases added). In human populations, selective breeding is known as eugenics, and although some early behavioral geneticists were interested in heritability for eugenics purposes (e.g., Fisher, 1918), currently few to none advocate the use of heritability estimates for eugenics programs to prevent

or control crime or other behaviors.[1] Although some behavioral geneticists have argued that the "only practical application of the heritability coefficient is to predict the results of a program of selective breeding" (Wahlsten, 1990: 119), others obviously believe that the estimates have other practical uses given the continued estimations of heritability on humans.

Even if the heritability concept is appropriate for use in humans in nonselective breeding programs, the methods used to derive these estimates are problematic in ways that render the estimates from the model ambiguous *at best* (e.g., Charney, 2012; Goldberger, 1979; Joseph, 2004, 2006; Wahlsten, 1990). Because each of these methods has its own set of problems, we discuss each in turn focusing in particular on the relevance of the methods' assumptions for conclusions about the heritability of crime. Although the methods are based on several different assumptions, in general, they are united by the fact that all compare individual phenotypes across varying degrees of genetic relationships and use these comparisons to estimate genetic and environmental influences *without actually measuring either*.

THE TWIN STUDY (STUDIES OF TWINS REARED TOGETHER)

The main "workhorse" used in behavioral genetics to estimate heritability is the classic twin-based research design (or the "twin study"; Plomin et al., 2012). Identical (monozygotic [MZ]) twins come from one fertilized egg and are assumed to share 100 percent of their genetic material (genetic clones), whereas fraternal (dizygotic [DZ]) twins come from two fertilized eggs and are presumed to share on average half of their genetic material (the same amount as other nontwin biological siblings). Given this and several assumptions, researchers have used MZ and DZ twins as a natural experiment to separate genetic and environment influences on variation in phenotypes (for a review, see Plomin et al., 2012).

The twin study separates phenotypic variation into three components: additive genetic (h), shared environment (c), and unshared environment (e). The unshared environment also includes model error. Notably, the terms "shared" and "unshared" environment do not correspond directly to common sense interpretations. The so-called shared environment consists of all nongenetic influences that make twins similar to each other, whereas "unshared" environmental influences consist of all nongenetic factors that make twins different (Plomin, 2011; Suhay and Kalmoe, 2010). Whether "shared" and "unshared" environments are actually shared is not at issue; instead, they refer to "'effects' rather than 'events'" that twins experience (Plomin, 2011: 582). Scholars frequently have failed to describe clearly what is meant by these terms, and others have made inappropriate conclusions about the insignificance of parental or community factors based on shared environment estimates (Harris, 1998; Rowe, 1994). It is important to remember that, in general, twin studies *do not actually measure* the shared or unshared environments; rather,

1. It is worth noting that it was not so long ago (as late as 1979 in Virginia) that compulsory sterilization as a means of crime prevention and/or punishment was practiced in the United States. More than two thirds of states adopted sterilization laws in the twentieth century, and probably many more than the widely cited figure of 63,000 Americans were involuntarily sterilized from the 1890s through the 1970s (Largent, 2007). Isolated instances of "voluntary" sterilization for convicted criminals in exchange for a reduced sentence continue in the twenty-first century and not only for sexual offenses (Largent, 2007).

these parameters are estimated based only on concordance rates or correlations between MZ and DZ twins.

The basic logic of the twin study is to compare twin concordances for phenotypes and, based on several assumptions, assign the greater phenotypic similarity of MZ relative to DZ co-twins to their greater genetic similarity. Through the formula explained in more detail in the subsequent discussion, heritability is usually estimated from twice the MZ–DZ difference in correlations. Although in recent years the twin study model has become more sophisticated, using latent variable models, the basic logic underlying these more advanced models is identical to that of the earlier twin studies, in which the twins' correlations are inserted into a series of simple equations and the heritability coefficient is calculated with elementary algebra (Suhay and Kalmoe, 2010). The basic assumptions that underlie the twin-study method have remained largely unchanged since the 1920s and are still central to these models given that neither genetic nor environmental influences are measured. Some of these assumptions are generally unproblematic, whereas others are dubious. The assumptions are as follows (Charney, 2012; Joseph, 2006; Plomin et al., 2012):

1. Researchers can reliably and accurately determine twin type (DZ vs. MZ).
2. The genes of MZ twins are 100 percent identical and are approximately 50 percent identical for DZ twins.
3. The percentages of genes shared by different types of twin pairs remain the same over the life course.
4. Phenotypic variation can be demarcated into genetic (G), shared environmental (C), or unshared environmental (E) components.
5. The relevant genes exert effects additively.
6. The likelihood of receiving a diagnosis or label for a phenotype (e.g., criminal conviction) is the same among the twin and the nontwin population (generalizability).
7. The risk of receiving the diagnosis or label is the same among MZ and DZ co-twins.
8. The phenotype (e.g., criminality or self-control) can be modeled as a quantitative trait.
9. The environments of MZ co-twins are no more similar than that of DZ co-twins ("equal environment assumption").

In general, many recent twin studies in criminology often fail even to mention these assumptions, much less discuss the adequacy of them. Although each assumption is potentially subject to some qualification, the final assumption, the equal environment assumption (EEA), has drawn the most attention among scholars criticizing the technical problems of heritability studies. As discussed, this crucial assumption and its modified form are clearly violated when the outcome of interest is criminal and related behaviors.

EEA Assumption

To observe the relevance of the EEA, it is useful to look at the basic equations underlying the model (see Plomin et al., 2012; Suhay and Kalmoe, 2010). Note in the equations that follow, the terms are squared because they represent the proportion

of variance explained.

$$\cdot r_{MZ} = h^2 + c^2$$

represents trait correlations between MZ co-twins as a function of genes (h) and shared environment (c).

$$\cdot r_{DZ} = h^2/2 + c^2$$

represents trait correlations between DZ co-twins as a function of genes (h), half that of MZ co-twins, and shared environment (c).

To calculate the heritability estimate, the DZ correlation is subtracted from the MZ correlation:

$$\cdot r_{MZ} - r_{DZ} = (h^2 + c^2) - (h^2/2 + c^2)$$

To move past this equation, the EEA assumption is necessary. By assuming that the shared environment (c) is equivalent for MZ and DZ co-twins (the EEA assumption), the equation can be simplified to the following equation:

$$\cdot r_{MZ} - r_{DZ} = h^2 - h^2/2$$

then

$$2(r_{MZ} - r_{DZ}) = 2(h^2 - h^2/2)$$

and finally

$$h^2 = 2(r_{MZ} - r_{DZ})$$

The shared environmental influence is calculated by starting with the first equation and solving for c^2.

$$\cdot c^2 = r_{MZ} - h^2 \text{ (or: } c^2 = 2r_{DZ} - r_{MZ})$$

Given the assumption (#4) that all effects are genetic, shared environmental, or unshared environmental effects, one can calculate the effects of the unshared environment (e^2) with the residual variance. Given that $h^2 + c^2 + e^2 = 1$, then:

$$\cdot e^2 = 1 - h^2 - c^2 \text{ (or, alternatively: } e^2 = 1 - r_{MZ})$$

As we can observe, these calculations are crucially dependent on the EEA assumption that the environments of MZ co-twins are no more similar than that of DZ co-twins. Every twin-based study is based on this EEA assumption to assign the greater concordance rates among MZ co-twins to genetics. Without this assumption, the greater concordance rates of MZ twins could be caused by more similar environments and/or genetics, and thus, h^2 could not be calculated (e.g., Joseph, 2004).

As it happens, and likely not surprising to those who have experience with MZ and DZ twins, this central assumption is flatly contradicted by both empirical evidence and common sense. Research clearly demonstrates that MZ co-twins experience more similar social environments than DZ co-twins. For instance, MZ twins are more likely to

be treated similarly by their parents (Evans and Martin, 2000), to have the same friends (Cronk et al., 2002; Horowitz et al., 2003), to share the same classroom (Cronk et al., 2002; Morris-Yates et al., 1990), to spend time together (and therefore experience the same social environments more frequently; Horowitz et al., 2003; Rende et al., 2004), and to go out together than DZ twins (Kendler and Gardner, 1998). Not surprisingly, MZ co-twins also report greater closeness and identification with one another (Jackson, 1960; LaBuda, Svikis, and Pickens, 1997; Segal, 2000) as well as mutual influence (Ainslie, 1997; Sandbank, 1999). For example, studies reveal that MZ twins are more likely than DZ twins to share bedrooms and clothes, and to share experiences like identity confusion (91 percent vs. 10 percent), being inseparable as children (73 percent vs. 19 percent), being brought up as a unit (72 percent vs. 19 percent), and having a high level of closeness (65 percent vs. 19 percent) (Richardson, 2011). Perhaps at the most basic level, MZ twins are treated more similarly and experience situations more similarly given their more similar appearance (attractiveness, height, physicality, and the like; Horowitz et al., 2003; Joseph, 2004).[2]

As persuasive evidence that the environments are more similar for MZ than DZ co-twins began to mount in the 1960s, behavioral geneticists acknowledged that the EEA was invalid (Joseph, 2004). Rather than concede that the twin study was ill suited for estimating heritability, they adopted a redefined "trait-relevant EEA," which grants that MZ co-twins might experience more similar social environments than DZ co-twins but assumes that these differences are irrelevant for the trait being studied (e.g., Carey and DiLalla, 1994; Kendler, 1983). For example, Kendler et al. (1993: 21) defined the equal-trait relevant EEA as the assumption "that monozygotic (MZ) and dizygotic (DZ) twins are equally correlated for their exposure to environmental influences that are of etiological relevance to the trait under study." Although this assumption may be reasonable for some phenotypes such as diabetes or heart disease, it is clear that it is not tenable for phenotypes such as crime given evidence that the more similar environments of MZ co-twins are trait *relevant*. Indeed, many of the shared environmental influences for which MZ co-twins have been shown to be more similar than DZ co-twins (e.g., parenting, peers, and leisure time together) represent some of the most potent predictors of the outcomes of concern to criminologists.

Given these clear findings, among others, of more similar social environments for MZ co-twins than DZ co-twins, many scholars have asserted that the EEA, including its trait-relevant form, is invalid and that the more similar environments of MZ than DZ co-twins bias heritability estimates upward to a significant degree (e.g., Beckwith and Morris, 2008; Horowitz et al., 2003; Joseph, 2004; Lewontin, Rose, and Kamin, 1984; Richardson, 2011). Even minor violations of the EEA can produce substantial overestimations of heritability (and thus underestimates of the shared environment). For example, if the shared environmental effect is .3 for MZ twins and .2 for DZ twins, then heritability will be inflated by 20 percent (Suhay and Kalmoe, 2010). Thus, it is a plausible interpretation of twin-study findings that surprisingly high heritability and unexpectedly low shared environmental estimates are caused in no small part by MZ co-twins having more similar environments than DZ co-twins (e.g., Jackson, 1960; Joseph, 2006).

2. Although some behavioral geneticists argue that treatment similarity based on similar appearance should be treated as a genetic effect (e.g., Bouchard et al., 1990), we disagree (see Joseph, 1998, for a discussion).

Table 1 displays a list and description of 20 twin studies we identified that have been published since 2008 examining crime and related phenotypes from a criminological perspective.[3] These studies were compiled from a search of the literature, primarily using Google Scholar. Criteria for inclusion were that the twin-study method was used to estimate the heritability of crime or a related phenotype (e.g., self-control, deviant peers, and antisocial behavior) from a criminological perspective (indicated by the use of criminological terms and theories). No study was excluded for any reason other than the criminological criteria just mentioned. Our critique of recent criminological twin studies is based on these 20 studies.

Given the centrality of the EEA, it is particularly surprising that most (19 of 20) of the recent criminological twin studies displayed in table 1 fail even to mention the EEA or its trait-relevant form at all, much less discuss its adequacy or the implications of its violation. Thus, readers who are unfamiliar with the assumptions of the model are not presented with this highly dubious assumption that is crucial for making genetic inferences, is likely violated, and results in inflated heritability and underestimated shared environmental estimates.

The EEA is even less reasonable for twin studies that combine same-sex and opposite-sex twins into their models (e.g., Beaver et al., 2008; Boisvert et al., 2012; Boutwell et al., 2013; Vaske, Boisvert, and Wright, 2012). As shown in table 1, most of the criminological twin studies we identified since 2008 included opposite-sex DZ pairs, and nine of the ten studies since 2012 did. (The percent of DZ co-twins in these studies that were opposite sex was approximately 45 percent.) In these cases, same-sex MZ twins are being compared with same-sex and opposite-sex DZ twins, with the assumption that (same-sex) MZ co-twins are treated *no more similarly* than opposite-sex DZ co-twins. Given the voluminous research on sex/gender differences in experiences, this assumption seems patently invalid. Evidence supporting this interpretation is found in a recent study by Meier et al. (2011), who compared the correlation for childhood conduct disorder among opposite-sex and same-sex DZ twins and found that the opposite-sex correlation was significantly smaller (approximately half, $r = .15$) than that of the same-sex DZ twins (see also Saudino, Ronald, and Plomin, 2005).

An even more surprising practice in a few recent criminology heritability studies is the use of kinship pairs, which includes MZ twins, DZ twins, siblings (full and half), and cousins (Barnes and Beaver, 2012; Barnes and Boutwell, 2012; Barnes, Boutwell, and Fox, 2012). [These extended twin-study designs are included in the category of "twin studies" given that they use the same model and assumptions extended for different degrees of genetic relatedness (Plomin et al., 2012)]. In these models, kinship pairs are compared with twins based on their average genetic relatedness, ranging from 1.0 for MZ co-twins to .125 for cousins. While these models often are not described clearly, by necessity they rely on the EEA to infer genetic influence. Thus, heritability estimates in these studies

3. Given that we had to select some cutoff date, the year 2008 was chosen for three reasons. First, the recent surge in heritability studies in criminology started in this year. Second, this is after the time at which recent critiques of the method had been published (e.g., Horowitz et al., 2003; Joseph, 2004; 2006) and even prominent scholars (e.g., Rutter) in the behavioral genetics field had highlighted the problems in the models and recommended that we should move beyond heritability. Finally, we wanted to keep the list manageable so that the reader can get an idea of the characteristics of the recent studies on which we focus without being overwhelmed.

Table 1. List of Identified Criminology Twin Studies and Kinship Studies Published Since 2008

Authors	Year	Journal[a]	Outcome(s)	Data	Twins vs. Kinship	OS and SS DZ Pairs	Mention EEA	Note Gene Additivity /Discuss	Mention G × E	Mention Shared "Effects"[b]	Present CIs/ SDs	Any Envir. Measured[c]
1. Viding et al.	2008	DS	ASB	Twins Early Dev. Study	Twins	SS	No	Yes/no	No	No	Yes	Yes
2. Ball et al.	2008	CP&P	Bullying, bully-victimization	E-Risk Long. Twin Study	Twins	SS	No	Yes/no	No	Yes	No	Yes
3. Beaver et al.	2008	JCJ	SC, SC stability	Add Health	Twins	OS + SS	No	No/no	No	No	No	No
4. Wright et al.	2008	JQ	SC, DPeers, delinquency	Add Health	Twins	OS + SS	No	Yes/no	No	No	No	Yes
5. Beaver, Schutt, et al.	2009	CJ&B	SC, DPeers	Add Health	Twins	SS	No	No/no	No	No	No	Yes
6. Beaver, Boutwell, et al.	2009	YV&JJ	Victimization	Add Health	Twins	SS	No	No/no	No	No	No	Yes
7. Beaver, Ferguson, and Lynn-Whaley	2010	CJ&B	SC	Add Health	Twins	SS	No	No/no	No	No	No	Yes
8. Meier et al.	2011	JAP	Conduct disorder, ASB	Australian Twin Reg	Twins	OS vs. SS[e]	Yes	Yes/no	Yes	No	Yes	Yes
9. Beaver	2011b	JQC	Parenting, SC, delinquency	Add Health	Twins	SS	No	No/no	Yes	No	Yes	Yes
10. Beaver, Gibson, et al.	2011	C&D	DPeers, DPeers stability	Add Health	Twins	SS	No	No/no	No	No	No	No
11. Barnes and Beaver	2012	JIV	Victim–offender overlap	Add Health	Kinship	OS + SS	Yes	No/no	Yes	No	Yes	No
12. Barnes, Boutwell, and Fox	2012	YV&JJ	Victimization, gang membership, overlap	Add Health	Kinship	OS + SS	No	No/no	No	No	Yes	Yes
13. Barnes and Boutwell	2012	JCJ	Delinquency, stability	Add Health	Kinship	OS + SS	No	No/no	Yes[g]	Yes	Yes	No
14. Boisvert et al.	2012	JQC	SC, delinquency, overlap	Add Health	Twins	OS + SS	No	No/no	No	No	Yes	No

(Continued)

Table 1. Continued

Authors	Year	Journal[a]	Outcome(s)	Data	Twins vs. Kinship	OS and SS DZ Pairs	Mention EEA	Note Gene Additivity /Discuss	Mention G × E	Mention Shared "Effects"[b]	Present CIs/ SDs	Any Envir. Measured[c]
15. Vaske, Boisvert, and Wright	2012	JIV	Victim, delinquency, overlap	Add Health	Twins	OS + SS	No	No/no	Yes	No	Yes	No
16. Boisvert, Wright, et al	2013	JQ	SC (sex differences)	Add Health	Twins	SS	No	Yes/no	No	Yes	Yes	No
17. Boisvert et al.	2014	JCJ	SC, subuse, overlap	Add Health	Twins	OS + SS	No	Yes/no	Yes[h]	Yes	Yes	No
18. Boutwell et al.	2013	JA	SC, victim, overlap	Add Health	Sibling[d]	OS + SS	No	No/no	No	No	Yes	No
19. Boisvert et al.	2013	CJ&B	DPeers, delinquency, overlap	Add Health	Sibling	OS + SS	No	Yes/no	No	Yes	Yes	No
20. Beaver, Boutwell, and Barnes	2013	CJ&B	Social support, SC	Add Health	Twins	OS + SS[f]	No	Yes/no	No	No	Yes	Yes

ABBREVIATIONS: ASB = antisocial behavior; CI = confidence interval; DPeers = delinquent peers; OS = opposite sex; SC = self-control; SD = standard deviation; SS = same sex.

[a]Journal abbreviations/acronyms are as follows (in order of appearance): DS: Developmental Science; CP&P: Journal of Child Psychology and Psychiatry; JCJ: Journal of Criminal Justice; JQ: Justice Quarterly; CJ&B: Criminal Justice and Behavior; YV&JJ: Youth Violence and Juvenile Justice; JAP: Journal of Abnormal Psychology; JQC: Journal of Quantitative Criminology; C&D: Crime and Delinquency; JIV: Journal of Interpersonal Violence; JA: Journal of Adolescence.

[b]This column indicates whether the study mentioned that "shared environmental estimates" actually capture shared environmental "effects." No study goes beyond a brief mention.

[c]Whether an environmental factor is measured is determined broadly, such that factors that could conceivably include environmental effects are considered "environmental" here, such as sex and age.

[d]Cousins were excluded. The authors noted: "[P]arameter estimates were substantively unchanged when only MZ and DZ twins were analyzed" (p. 660).

[e]OS twins were not combined but assessed separately for comparison.

[f]Does not specify OS, but given the reported sample size, we determined that OS twins were included.

[g]The authors mentioned in a footnote.

[h]Explained to the reader how interactions were treated in the model.

are based on the assumption that the environments of pairs of opposite-sex cousins are no less similar than that of identical twins for the outcomes under study. Quite simply, the EEA seems preposterous for studies of kinship pairs. That such studies report high heritabilities and nil shared environmental effects is not surprising.

Statements that ignore the EEA and its potential violation, such as "the only reason MZ twins should be more similar than DZ twin pairs is because they share twice as much genetic material," are found in recent heritability studies (e.g., Beaver, 2011b: 86; Beaver, Ferguson, and Lynn-Whaley, 2010). As we have discussed, MZ co-twins may be more similar than DZ co-twins for criminal phenotypes for two reasons: genetics and/or more similar shared environments. Given the evidence of more similar treatment for MZ co-twins as opposed to DZ co-twins (and especially in the case of opposite-sex DZ twins and kinship pairs), we think it is unquestionably the case that violations of the EEA are inflating heritability and decreasing shared environmental effects to a substantial degree.

Genetic Additivity Assumption

The additivity assumption (assumption #5) also is implicated in the accuracy of heritability estimates. Although the evidence of nonadditivity of genetic and environmental influences is discussed more below, this assumption refers to the additivity of genetic influences on a phenotype. Genetic variance can be separated into additive and nonadditive components.[4] Additive genetic variance, which is that assumed by twin studies, is a model of gene combinatorial effects where many genes each contribute small individual effects that add up to shape the phenotype. Nonadditive genetic variance is that which arises because of the interactions between genes such that the resulting quantitative phenotype is significantly different from the sum of the individual genetic effects (Stoolmiller, 1999). Nonadditive variance can be of two kinds: dominance and epistasis. Dominance is that occurring when the alleles at a given locus (one from each parent) interact to produce a phenotype. This interaction occurs among genes that operate with a strict dominance-recessive mode of inheritance. Epistasis occurs when several genes (alleles at different loci) interact to produce a behavior. Although the extent of nonadditive genetic variance involved in criminal phenotypes is not known, there is good reason to believe it is operative for all complex traits and behaviors, including crime (Plomin et al., 2012).

Violations of the additivity assumption have consequences for heritability estimates. Several decades ago, Grayson (1989) showed that in twin studies, undetected nonadditive genetic variance inflates heritability and deflates shared environmental influences. The population genetic correlation between first-degree relatives, such as DZ co-twins, on a trait where all genetic influences are additive is .50. Conversely, nonadditive genetic influences can, at most, produce a correlation of .25 if the nonadditivity is caused entirely by dominance interactions. As the nonadditivity shifts to epistasis, the expected genetic correlation declines toward zero (Grayson, 1989; Stoolmiller, 1999). Genetic nonadditivity will thus have the effect of reducing the genetic correlation for DZ co-twins but not for

4. Given the rarity of monogenic traits, there is consensus that in nearly all cases, genes combine to influence phenotypes. For example, eye color, which was once thought to be determined by around three genes or less, has been shown to be influenced by at least 20 and possibly hundreds of genes (Liu et al., 2010).

MZ co-twins (because MZ co-twins have all of the same interacting alleles), which will lead to a reduction in the overall correlation for DZ twins but not for MZ twins. Twice the MZ–DZ difference in correlations (the estimate of heritability) is thus an overestimate, and twice the DZ correlation minus the MZ correlation (the estimate of shared environment) is thus an underestimate (Grayson, 1989).

Thus, similar to violations of the EEA, violations of the additivity assumption serve to "maximize the potential role of genetic influences and minimize the potential contribution of shared/familial environmental influences" (Grayson, 1989: 594). Although dominance interactions can be modeled in twin-study designs, we have only observed this attempted once in criminology (Boisvert, Boutwell, et al., 2013), and doing so has its own set of problems (for example, shared environmental effects are ignored because they cannot be estimated simultaneously; Neale and Cardon, 1992). Moreover, modeling epistatic interactions with human kinship data is generally considered impracticable (Eaves, 1988). Regarding nonadditivity, Plomin et al. (2012: 401) stated: "These types of effects complicate model fitting because there are many forms in which they could occur. Normal twin study designs do not offer much hope for identifying them."

Recent published twin studies in criminology often, but not always, have noted that additive genetic and environmental influences were being estimated; however, as shown in table 1, these studies invariably have not explained what this means or the implications of the likely violation of the assumption of additive effects (deflated shared environmental estimates and inflated heritability). As such, the resulting biases caused by the likely violation of the additivity assumption have not been made clear.

Additional Issues

Although not technical limitations of the twin-study model, we believe that three additional characteristics of recent criminology twin studies are worth noting. As can be observed in table 1, of the identified 20 criminological twin studies published since 2008, 17 used the Add Health data. We do not argue that the genetic twin sample in the Add Health is deficient; indeed, the quality of the data seems to be extraordinary (Harris et al., 2006). We do believe, however, that reproducing findings of similar heritabilities for various criminal-related traits on the same set of 289 MZ and 452 DZ twin pairs is problematic. Moreover, this means that most recent heritability estimates in criminology have been based on the same imperfect measures (self-control, delinquent peers, delinquency, and victimization) that are available in the Add Health data. The measure of delinquent peers, for example, only includes respondents' perceptions of their three closest friends' use of three substances (e.g., Beaver, Schutt, et al., 2009; Beaver et al., 2011; Boisvert, Boutwell, et al., 2013), and the measure of victimization only includes experiences with physical violence (Boisvert et al., 2014; Vaske, Boisvert, and Wright, 2012). To be sure, we are not encouraging replication of these findings in other data sets as we believe the twin-study method has too many problems to provide valid or reliable estimates. We do wish to draw the reader's attention to the fact that similar twin-study findings in criminology, at least over the past 6 years, have been based largely on the same group of twins (and kinship pairs) with generally the same measures taken at the same wave.

An additional reason to view twin-study heritability estimates with caution is that they tend to have large confidence intervals (DiLalla, 2002). As shown in table 1, several recent

studies in criminology have not reported the confidence intervals or the standard errors for the estimates, which serves to reify an inherently imprecise estimate.

A final caution about twin-study findings has to do with interpretations of the results. Several recent criminological twin studies draw unjustified conclusions from insignificant estimates of shared environmental effects (e.g., Beaver, Boutwell, et al., 2009; Beaver, Schutt, et al., 2009). For example, Beaver, Ferguson, and Lynn-Whalley (2010: 1060) concluded: "The null results for the shared environment across various measures of parenting indicate that parents do not have a causal effect on shaping and molding their offspring's level of self-control." However, a basic principle of hypothesis testing is that failing to reject the null hypothesis does not mean that the null hypothesis is true. As such, failing to reject the null hypothesis that the shared environmental effect equals zero is not evidence that the effect is, in fact, zero. All one can say from such null findings is that we do not have enough evidence to say that the shared environmental effect is not different from zero. As such, the evidence does not show that shared environmental effects, such as parenting, do not have a causal effect on variation in various phenotypes.

ADOPTION STUDIES

Adoption studies have been deemed a powerful design for investigating genetic and environmental influences. Adoption studies investigate correlations for phenotypes between adoptees and their biological parents and genetically unrelated adoptive parents. Several different adoption designs exist (see Joseph, 2004, 2010; Rutter et al., 1990, for overviews), but all make use of this "genetic parent"–"social parent" contrast for inferring genetic influences.

In theory, correlations between adoptees and biological parents indicate the influence of genes, whereas those between adoptees and social parents represent the influence of environmental factors. As such, adoption studies have been promoted as "natural experiments in which the effects of genetics and rearing influences may be separated to a high degree" (Mednick and Kandel, 1988: 103). Many scientists unswayed by the findings from twin studies have pointed to adoption studies as powerful confirming evidence, and scholars have pointed to the salient role of adoption study findings in shifting the tide toward genetic explanations (Joseph, 2004; Rowe and Jacobson, 1999). Like twin studies, however, adoption studies suffer from several limitations that render their estimates questionable and seem to distort estimates toward inflating heritability and underestimating environmental influences, especially the shared environment (e.g., Joseph, 2004; Rutter, 2006; Stoolmiller, 1999). Included in these limitations are late separation, nonrepresentativeness and range restriction, selective placement, and prenatal environment confounding.

Late Separation

Late (nonbirth and beyond) separation of the child from his or her biological parent is an obvious environmental confound, for two reasons. First, late separation confounds the allegedly pure genetic influence of the biological parent (e.g., Faraone et al., 1999), especially given the large body of research evincing that early childhood is a crucial period for development. In addition, late separation itself alters the natural experiment of adoptions. Adoptions can have a significant disruptive effect on child development when separations occur after the formation of an attachment relationship (e.g., Baumrind, 1993). Moreover,

in addition to the stress of adoption, the status of being an adoptive child is itself a source of strain (Brodzinsky, Singer, and Braff, 1984; Steinhauer, 1983).

Nonrepresentativeness and Range Restriction

Perhaps the most serious problem with adoption studies is their nonrepresentativeness. Adoptive parents severely underrepresent high-risk social environments as they tend to be more affluent, more highly educated, and live in better communities than the general population (e.g., Lewontin, Rose, and Kamin, 1984; Stoolmiller, 1999). Thus, the range of environmental (especially familial) quality in adoption studies is both well above average and more restricted in range than in the general population (Kamin, 1981; Miles and Carey, 1997). As Stoolmiller (1998) noted, this restricted range results from at least three different selection mechanisms: 1) Adoptive parents are carefully screened by adoption agencies before they are allowed to adopt, 2) adoptive families always choose to have a child, and 3) adoptive families volunteer to participate in the study (presumably adoptive families who provide a poor family environment would not agree to participate in an adoption study). It seems clear that "powerful selection forces are at work in determining what kind of families will be present in an adoption study" (Stoolmiller, 1999: 395). Indeed, evidence suggests that *at least* the bottom 60 percent of the general population's range of family environmental quality is excluded from adoption studies (Stoolmiller, 1998).

Given that range restriction reduces correlations, the effect of this significant restriction of range in family environment quality in adoptive samples will be to attenuate the correlation between adoptive parents and children, thereby decreasing shared environmental estimates and inflating genetic estimates (Lewontin, Rose, and Kamin, 1984; Miles and Carey, 1997). Applying a correction for range truncation to analyses of the Texas Adoption Project, Stoolmiller (1998) estimated that shared family environment accounted for 55 percent of the variance in child IQ scores, noting that conventional estimates are usually less than half that amount. Based on his research, Stoolmiller (1999: 393) concluded: "This pattern of differential attenuation and inflation is the reason for the apparent lack of relative importance of SE [shared environment] in behavior-genetic adoption studies." Moreover, range restriction may account for the ostensibly contradictory findings between individual and group studies of adoption (Stoolmiller, 1999). In contrast to individual studies, group studies of adoption clearly indicate a beneficial effect of being adopted from higher risk environments to lower risk environments (e.g., lower class to middle class) on IQ and deviance, which is consistent with a strong effect of the shared environment (Turkheimer, 1991).

Selective Placement

Another problematic issue with adoption studies is the "no selective placement" assumption, which is the assumption that there is no association between characteristics of the biological parents and the adoptive family (Joseph, 2010; Kamin, 1985; Lewontin, Rose, and Kamin, 1984). In regard to criminality, selective placement could bias the separation of genetic and environmental effects if the adopted children of the least criminally inclined biological parents were adopted by the families who provided an environment least conducive to criminality (and vice versa). If selective placement occurs, then genetic effects will be inflated. Selective placement can result from adoption agencies "fitting the

home to the child" (attempting to maximize similarities between biological and adoptive families to increase the child's chance of fitting in; Munsinger, 1975: 627) or children of criminal parents being considered less desirable adoptees and therefore ending up in less desirable adoptive families (Joseph, 2004; Munsinger, 1975).

Evidence suggests that selective placement related to criminal characteristics is a factor in adoption studies (see the review in Joseph, 2004). For example, evidence shows that children whose biological parents had a history of criminal behavior or mental disorders were more likely to be placed in inferior adoptive family environments (within the restricted range) compared with adoptees without convictions or disorders among their biological parents (Joseph, 2004; Kamin, 1985). In addition to selective placement, it seems that selective *late* placement is also a biasing factor. For example, Bohman (1978: 275), an adoption study proponent, found that adoptees with criminal or alcoholic biological parents were placed on average 2 to 3 months later than controls, noting that "later placement is associated with selective factors that contributed independently to poorer social adjustment later in life and an increased risk of appearance in the [criminal] registers." In sum, it seems that selective placement is operating in adoptive studies, especially in regard to factors such as criminality, in violation of random assignment, which valid adoption studies require (Joseph, 2004).

Prenatal Influences

A final notable limitation in adoption studies concerns prenatal influences. Birth mothers in adoptive studies provide not only genes but also a prenatal environment; thus, similarity between biological mothers and children—even those adopted at the moment of birth—can be caused by both genetic and (prenatal) environmental influences (e.g., Plomin et al., 2012). Importantly, poor obstetric care, exposure to toxins, stress, and poor nutrition during pregnancy are not randomly distributed among the population of pregnant women. Instead, some of the same social factors associated with criminality and/or consequences of criminality (e.g., poverty, neighborhood disadvantages, and substance use) are associated with factors known to have an influence on fetal development and postnatal cognitive and behavioral developmental (e.g., lead, illicit drugs, nicotine, and endocrine disrupters). Inasmuch as prenatal exposure to these factors increases the likelihood of crime (e.g., Raine, 2002a, 2002b), children of criminal parents would be at a higher risk for crime for nongenetic reasons. Such shared environmental factors between mother and child (in utero and after birth in the case of late placement) will be included in the genetic estimate in adoption studies. Given this evidence, Conley (2011) noted:

> [W]e know, ipso facto, that families who adopt are a distinct social group on unobservables—as are the adoptees themselves… The only adoption study that would avoid such [problems] would be one in which adoptees were randomly selected from the new-born population and then randomly assigned to parents, with both groups blind to the treatment (i.e., not knowing whether they were adopted or not)—all while prenatal environment was held constant. In other words, it is an impossibility to reliably estimate genetic heritability using [the adoption method]. (p. 597)

Adoption Studies of Crime-Related Phenotypes

Heritability estimates of criminality and related phenotypes (e.g., aggression and antisocial behavior) from adoption studies are lower than those from twin studies (Joseph, 2004; Moffitt, 2005), and not all find evidence of the heritability of crime (Bohman, 1978).[5] In general, all of the adoption studies contain various invalidating flaws, including but not limited to those mentioned previously. For example, the most frequently cited adoption study in support of genetic effects on crime by Mednick, Gabrielli, and Hutchings (1984) suffers from selective placement, late placement, and nonrepresentativeness, among others (see Joseph, 2004; Kamin, 1985). Criticisms of particular features of all but the most recent studies have been detailed elsewhere (see Gottfredson and Hirschi, 1990; Joseph, 2001, 2004; Walters and White, 1989). We have not yet found critical attention to the latest adoption studies. Thus, we briefly comment on two recent studies.

Beaver (2011a: 282) investigated "genetic influences on being processed through the criminal justice system" using the subsample of adoptees included in the Add Health Study. Although Beaver (2011a) found that adoptees whose biological parents "had ever spent time in jail or prison" were significantly more likely to have contact with the criminal justice system, this finding is vitiated by several serious limitations, including those mentioned. Perhaps the most significant of these is the study sample. The only requirement for inclusion in the study sample was that the respondent indicated that he or she was adopted sometime before the survey (which took place when youth were in grades 7 through 12) and did not currently live with a biological parent. Because of data limitations, Beaver (2011a) could not ascertain the age at which the children were adopted and did not control for contact with biological parents. Additionally, information about the biological parents' incarceration or lack thereof came from the adoptees themselves, and only respondents who were aware of their biological parents' jail or prison experiences were included in the analyses. (Adoptees who answered "I don't know" to the question of biological parents' prison or jail experience were excluded.) As such, those respondents who had no knowledge about their biological parents' jail or prison status—almost certainly those who had the least contact with their biological parents (and could not be influenced by potential labeling processes involved in having a criminal parent)—were not included in the analyses. This same Add Health adoption subsample and model also was used to "estimate genetic influences on victimization" (Beaver et al., 2013: 149).

In sum, the adoption method was promoted as a powerful model for separating genetic and environmental influences that avoided limitations of twin studies by "more cleanly [separating] genetic and environmental influences" (Raine, 1993: 60; also Mednick and Kandel, 1988; Plomin and DeFries, 1985) and as such has played a crucial role in bolstering findings from twin studies. It is clear, however, that the adoption method suffers from several of its own invalidating flaws, which—like twin studies—seem to bias estimates systematically toward genetic influences and against shared environmental ones (e.g., Joseph, 2004; Stoolmiller, 1999).

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5. For a more thorough critical review of adoption studies of crime, see Joseph (2004), and for a list of heritability estimates and brief methodological features of the studies prior to 2004, see Moffitt (2005).

STUDIES OF TWINS "REARED APART"

The third heritability model, which has been used less frequently than the other two, largely because of the rarity of the situation, is known as the twins reared apart (TRA) study. TRA studies compare the phenotypes of reared-apart MZ pairs (MZAs) with a control group of MZ pairs raised together. The rationale for these studies is in large part to avoid the problems generated by the EEA (Joseph, 2010). Despite the paucity of studies, TRA findings often are cited as providing crucial support for claims that dismiss the import of shared environmental factors on psychosocial development and in support of the EEA (e.g., Alford, Funk, and Hibbing, 2005; Harris, 1998; Pinker, 2002). Although only one systematic study of TRAs has investigated antisocial behavior (Grove et al., 1990), this study often is cited in criminological work as support for the findings obtained from classic twin studies regarding the heritability of crime/antisocial behavior (e.g., Moffitt, 2005; Raine, 2002b). In general, TRA studies have received considerable attention from both the scientific community and the media over the past several decades, partly because of the interest in the compelling stories of eerie similarities among reared-apart twins (see Joseph, 2004).

Despite their intuitive appeal and publicity, however, TRA studies suffer from a host of limitations that render their heritability estimates highly problematic at best (e.g., Joseph, 2004; Rutter, 2006). The limitations include the following: 1) Many common environmental factors shared by MZAs could lead to greater concordance; 2) many if not most twins were only reared "partially apart"; and 3) biases in sampling, which favor recruitment of MZA pairs that are more similar than the MZA population (e.g., Charney, 2008; Farber, 1981; Joseph, 2004). As a result of the dearth of TRA studies of criminal behavior, a detailed discussion of these limitations is not possible in this article. However, given many behavioral geneticists' assertions that TRAs provide crucial support for twin-study findings, we provide a discussion of this method and its limitations in the online supporting information.[6] There, we provide evidence supporting our position that TRA studies also suffer from serious problems that render their results highly dubious and biased toward inflated heritability.

CONCEPTUAL PROBLEM: (THE FALLACY OF) PARTITIONING GENETIC AND ENVIRONMENTAL INFLUENCES

Aside from their methodological pitfalls, an equally serious problem with heritability studies is the notion that genetic and environmental effects can be partitioned into separate additive influences in the first place (assumption #4). Obviously, an estimation of heritability requires that one can in fact separate genetic from environmental influences on behavior. Reality is not so simple; next, we attempt to show that quantitative genetics uses an outdated model of genetic structure and function that is explicitly rejected by the scientists who study the actions of genes directly (e.g., Gottlieb, 1992; Meaney, 2010).

From a classical genetic perspective, two contributions to phenotypic variance preclude the separation of G and E: genetic–environmental covariance and genetic–environment

6. Additional supporting information can be found in the listing for this article in the Wiley Online Library at http://onlinelibrary.wiley.com/doi/10.1111/crim.2011.52.issue-2/issuetoc.

interaction. *Genetic–environmental covariance* occurs when certain genotypes are associated with particular environments (Plomin et al., 2012). Scholars have used the example of children's genetically influenced behavioral problems evoking harsh parental discipline, which, in turn, could increase behavior problems. It is an ongoing matter of debate as to how to classify this covariance in the calculation of heritability; certainly many, if not most, scholars would agree that it fits neatly in neither the G nor the E category. Several behavioral geneticists have argued that such gene–environment correlations should be classified as genetic effects (e.g., Fowler, Baker, and Dawes, 2008; Segal and Johnson, 2009); however, as Rutter (2002: 4) noted, "it is misleading to suppose that just because genetic factors influence the occurrence of an environmental risk factor, this must mean that the risk process is genetically mediated. This assumption does not follow because there is no necessary connection between the causes of the origin of a risk factor and its mode of risk mediation." This can be illustrated with real-world examples such as skin pigmentation (Billings, Beckwith, and Alper, 1992; Joseph, 2004).

In the United States, a person genetically coded to have darker skin will experience a different social environment, on average, than one with lighter skin. Darker skin pigmentation is associated with exposure to social criminogenic risk factors, such as racial discrimination, lower socioeconomic status (SES), and disadvantaged communities, among others (e.g., Burt, Simons, and Gibbons, 2012; Sampson and Wilson, 1995). Surely, most scientists believe that classifying offending that results from racial discrimination because of skin pigmentation as solely "genetic" is preposterous. But even in less manifestly genetically spurious cases, the classification is still not clear and rests on algebra and statistics, which are not up to the task of classifying interactional biopsychosocial relationships (e.g., Spencer and Harpalani, 2004; Wahlsten, 1990).

In addition, the idea that the effects of nature and nurture can be partitioned into percentages requires one to assume that genetic and environmental influences are competing and noninteractional or at least that interaction effects are trivial (Wahlsten, 1990). Mounting evidence suggests that this not the case. Rather than the rare exception, gene–environment (G × E) interactions seem to be the rule (e.g., Bagot and Meaney, 2010; Charney, 2008; Rutter, 2007). As with many of the other problems we have discussed in quantitative genetics, this criticism of heritability studies is not new. Indeed, Hogben (1932: 201) first identified the problem that G × E posed for efforts to estimate heritability more than 80 years ago, criticizing the "false antithesis of heredity and environment," while demonstrating the effects of G × E on heritability estimates (for a historical overview, see Tabery, 2007).

Although the debate over the problems posed by G × E interactions for heritability studies continued for years after Hogben's initial critique, perhaps reaching its height during the IQ controversies (e.g., Lewontin, 1974; Wahlsten, 1979), this matter is much less controversial currently, as the idea that heritability studies attempt the impossible is more widely accepted among scholars, including prominent behavioral geneticists who had previously been proponents of heritability studies (e.g., Rutter, 2006; Turkheimer, 2011). Importantly, interactions between shared environmental effects and genetic influences are captured in the heritability estimate (Alper and Beckwith, 1993; Miles and Carey, 1997; Moffitt, 2005). Such G × E interactions are widespread, especially for complex socially mediated phenotypes such as crime, and they result in substantial underestimates of the shared environment in twin studies (Rutter, 2002). In sum, from the classical genetics perspective, strong evidence invalidates the rationale of heritability studies.

POSTGENOMIC CHALLENGES TO HERITABILITY STUDIES

Perhaps the most decisive evidence against the validity of partitioning G and E influences comes from recent advances in molecular genetics, which demonstrate among other things that the human genome is dynamic and environmentally responsive. A wealth of evidence has accumulated in recent years supporting an interactional, bidirectional model of gene and environmental function (e.g., Charney, 2012; Jablonka and Lamb, 2006). These developments in molecular genetics have altered the scientific understanding of heredity, the role of the gene, and the relationship between genotype and phenotype and mark a shift from the classical "gene-centric" paradigm, on which existing behavioral genetics methodologies are grounded, to a "postgenomic" view.

A key theme of the postgenomic view is the idea that genes do not stand outside the developmental system of which they are a part. Humans develop through a process of dynamic relations involving factors from the biological to the sociocultural levels of organization. Influences from *all* levels contribute integratively to the structure and function of human development (Gottlieb, 2001; Greenberg, 2011; Lickliter, 2009). It is not merely that genes and environments are independent and interact to influence a phenotype (as in G $\times$ E) but that genes and environments are not separate analytical entities in the first place. Perhaps the most compelling evidence for the interactional relationship between genes and environments in cellular function emerges from the relatively recent field of study known as epigenetics. Research from this burgeoning field exemplifies the blurred boundaries between environments and genes and illustrates the interactional relationship among genes, biology, and environments even at the level of cellular molecules (e.g., Bagot and Meaney, 2010; Charney, 2012).

A Primer on Epigenetics

Our environment influences us by regulating our genetic activity. As noted, DNA is not self-activating. DNA has to be transcribed to produce RNA and proteins, but before DNA can be transcribed, it must be activated by the *epigenome*—the complex, biochemical regulatory system that can turn on, silence (leave off), or change the transcriptional activity of genes (Charney, 2012; Martiensen, Riggs, and Russo, 1996). As such, the mere presence of a gene does not ensure that it is going to be used (activated by the cell), and changes in the epigenome can alter the phenotype without any underlying change to the genome (Bernstein, Meissner, and Lander, 2007). In this way, the epigenome regulates gene expression. As the epigenome is responsive to environmental input (both internal and external to the cell), the environment influences gene expression through the epigenome (e.g., Charney, 2012; Jablonka and Lamb, 2006).

Most gene regulation is a response to the immediate demands of the environment, takes place in time spans ranging from minutes to weeks, and differs between specialized cells, which contain identical DNA (Francis, 2011; Plomin et al., 2012). For example, our liver cells will react one way to food poisoning, our intestinal cells will react another way, and many cell types will not react at all. In recent years, however, researchers have devoted much attention to a type of gene regulation that takes place over much longer intervals. Epigenetics focuses on the mechanisms of gene regulation implicated in changes in gene expression and phenotype, which can last for months, years, or across the life span and can even be transmitted onto future generations (Bagot and Meaney, 2010; Bollati and Baccarelli, 2010; Charney, 2012).

Although new epigenetic mechanisms are still being uncovered, perhaps the best understood fall into a class known as chromatin markers, often referred to as "epigenetic markers." Two of the most well known are histone modification and methylation.[7] These markers affect cellular activity and phenotypes by influencing the accessibility of DNA to transcription factors, thereby shaping gene expression. Evidence is accumulating for the effects of environmental factors on these epigenetic markers, thereby illustrating ways in which gene expression is influenced by the environment (Bagot and Meaney, 2010; Francis, 2011). Perhaps most important for our purposes, evidence for the effects of epigenetic factors in neural development is accumulating rapidly. Studies have shown, for example, that epigenetic modification influences perception, emotion, memory, cognition and learning, and neural and behavioral plasticity (e.g., Allen, 2008; Crews, 2008). Indeed, gene expression is an essential step in the numerous cellular processes involved in learning and memory and in altering the growth and organization of nerve and especially neural cells (Kaplan and Rodgers, 2003; Molfese, 2011).

Neogenome and Heritability

What relevance do these recent postgenomic advances have for heritability studies? The implications of these phenomena, which Charney (2012) collectively referred to as the *neogenome*, are several. First, the research on epigenetics provides powerful evidence that differences in phenotypes cannot be separated into genetic versus environmental influences. The environment influences gene function, and the neogenome behaves like neither the G nor E category (Charney, 2012). Instead of genome/neogenome versus environment, research evinces that the system is bidirectional and interactive. As Kaplan and Rodgers (2003: 5) noted, "development is not merely a process involving a battle between nature (genes) and nurture (experience) but the interweaving of dynamic processes within a system that is inseparably both the organism and its environment." Epigenetics illuminates the flexibility of the biological organism and the complexity of the relationship between genes and the environment during development.

Grounded in the now outdated (oversimplified and incorrect) paradigm pitting nature versus nurture and emphasizing differences in the genetic code as the way in which nature shapes phenotypic variance, heritability studies have no place in a postgenomic paradigm. As research in epigenetics makes clear, the mere presence of a gene as part of a genotype does not ensure that it is going to be activated; rather, changes in the environment can alter the activation of genes and, in turn, shape or change a phenotype without any underlying change to the genome (Bernstein, Meissner, and Lander, 2007). Moreover, although evidence suggests that environmental events that occur early in life (e.g., prenatal factors or child abuse) tend to produce more pronounced epigenetic effects than those that occur later (Bradley et al., 2008; Trembley, 2010), methylation and other epigenetic processes continue throughout the life span (Francis, 2011; Weaver, Meaney, and Szyf, 2006). In sum, these recent advances provide clear evidence that the genome is immensely flexible in its expression, and this expression is responsive to context, experience, and developmental history. Several assumptions are necessary for heritability studies to be meaningful, but the most crucial of these is the model of gene function as separable and

7. For a friendly introduction to epigenetics, see Francis (2011), and for a more technical overview, see Charney (2012).

independent from the environment (Charney, 2012). Evidence is clear that genes do not work in this way.

Heritability Studies: Concluding Remarks

Research has evinced that human behavior is a function of the interplay of biology and the environment. As we have argued, we believe that this evidence clearly demonstrates that quantitative genetics is a misguided endeavor that asks the wrong questions and uses flawed methods to try to answer them.[8] What remains unclear is why this enterprise continues. The question of nature *versus* nurture no longer makes any sense whatsoever in the context of modern genetics. These recent advances in molecular genetics "really should be the final nail in heritability's coffin" (Crusio, 2012: 362).

Moreover, it is now widely accepted—even by prominent behavioral geneticists—that heritability estimates are of little practical relevance (Rutter, 1997; Turkheimer, 2011). Even if estimated accurately, which is impossible for the reasons we mention, they do not predict the likely developmental endpoints for individuals or groups, the consequences of interventions, or the causal processes or mechanisms involved in phenotypic variations (Rutter, 1997). Given all of these limitations, we recommend an end to heritability studies in criminology. In addition, we urge scholars to recognize that existing heritability estimates are the result of models biased toward inflating genetic influences and underestimating shared environmental ones, and that using these rough and biased heritability estimates to undergird specious debates about the irrelevance of shared environmental factors, such as the family, neighborhoods, and SES (e.g., Harris, 1998; Rowe, 1994), does a disservice to both scientific and public knowledge.

As we have noted, however, we are not trying to push the criminology community away from biosocial theory and research. Instead, we recommend a different research agenda for criminologists interested in doing biosocial research, one that is consistent with developments in the postgenomic paradigm, with its emphasis on the interactional, bidirectional, and multifactorial relationships among genes, biology, environment, and behavior. Our recommendations have two main underpinnings. First, we believe research must go beyond simply recognizing or paying lip service to the fact that behavior, including crime, is a function of the interplay of genetic and environmental influences. Too often, this interplay has been wrongly translated into the misleading notion that "bad genes" (or a "bad brain" in the case of neuroscience) combined with a "bad environment" produce antisocial behavior, with the assumption of a causal influence from genes to brain to behavior. This notion of "bad genes" and a unidirectional effect from genes to brain and behavior is misguided, as research suggests a bidirectional relationship from behavior and environment to the brain through the epigenome. Furthermore, there is the usually unstated implication that genetic and environmental influences are equally important (because heritability has been estimated at 50 percent) and that both exert main effects in addition to their interaction. Accumulating molecular genetics research suggests, however, that environmental influences are more salient in explaining individual differences

8. Had we unlimited space we would discuss other problems with heritability studies, such as phenotype ambiguity and classification issues. Given space constraints, we point the reader elsewhere to excellent discussions of these and other limitations (e.g., Charney, 2008; Duster, 1990, 2006; Joseph, 2006; Lewontin, Rose, and Kamin, 1984).

in complex phenotypes such as criminal behavior, with genetic influences usually being limited to moderating the effect of the environment (e.g., Moffitt, Caspi, and Rutter, 2005; Simons, Beach, and Barr, 2012).[9]

For example, candidate G × E studies, which investigate the interaction between genetic polymorphisms and environmental risk factors, invariably find that the genetic influence on criminal or related phenotypes is limited to its moderation of the environmental effect. Caspi et al.'s (2002) now famous *Science* article, which marked the beginning of the wave of G × E studies, examined the effects of childhood mistreatment and the MAOA polymorphism on antisocial behavior among a sample of White males. The MAOA risk allele, which was found in 37 percent of the sample, had no direct effect on antisocial behavior but rather served to augment the likelihood of antisocial behavior in the presence of childhood maltreatment. Importantly, *regardless of MAOA status*, childhood maltreatment increased the likelihood of antisocial behavior. Subsequently, scores of recent candidate G × E studies have shown that polymorphisms associated with various genes (e.g., DRD2, DRD4, MAOA, GABRA2, and 5-HTT) interact with environmental risk factors to increase the likelihood of various types of internalizing and externalizing problems (see Belsky and Pluess, 2009; Simons, Beach, and Barr, 2012, for reviews).[10]

In addition to research highlighting the interplay of biology and the environment, we believe that work that seeks to elucidate processes or pathways of influence should be given priority among biosocial criminologists. As Rutter (2002: 6) has noted, although G × E studies are a useful first step toward this end, they are "the relatively easy part"; the challenge is understanding "what these genes do" (i.e., their effects on proteins and the process through which these proteins lead to various cellular and biological activities) and understanding how these various biological activities shape responses to environmental factors. An integrated, interdisciplinary research approach is required for a clear understanding of these mechanisms. Moreover, such work should be informed by evidence that the neogenome is itself not a fixed or static entity but that it changes in response to environmental conditions (Charney, 2012). In other words, biosocial researchers in criminology need to incorporate the effects of the environment on changes in biological and/or neogenomic factors into their models.

Several ongoing lines of biosocial research are consistent with this research agenda (e.g., Walsh and Beaver, 2009). We wish to draw attention to epigenetics and social neuroscience as promising avenues of future research in biosocial criminology. Work in these two overlapping areas is rooted in the postgenomic paradigm and has the potential to elucidate the complex biological pathways linking environmental factors to criminal

9. Although a few studies have shown that candidate genes such as MAOA have direct effects on antisocial outcomes (e.g., Beaver, Delisi et al., 2010; Beaver et al., 2012), these models have several limitations that vitiate the implications of their main effects findings, not the least of which is omitted variable bias (e.g., Charney and English, 2013). Few to no environmental risk factors are included in the models in these studies; therefore, both the main effect for the environment and any G × E effects are omitted from the analyses, and the model is misspecified.

10. Although out of the scope of this article, see Charney and English (2012, 2013) for an excellent critique of G × E studies, including their lack of replicated findings. Notably, G × E studies are based in the classical genetic paradigm, whereby a snapshot of DNA (not the epigenome) is examined and interacted with measures of the environment. Recent advances suggest the wisdom of moving beyond this more static approach and attending to epigene–environment interplay and interactions (e.g., Bollati and Baccarelli, 2010; Charney, 2012).

behavior. (To be sure, much of this work is preliminary and should be subject to various critiques and to the caveat that the findings require replication before being accepted.) In the final section of the article, we briefly discuss how these endeavors might be incorporated into criminological research in a manner that complements current work being done in the field.

EPIGENETICS AND SOCIAL NEUROSCIENCE

The postgenomic paradigm, with its emphasis on gene regulation and epigenetics, contributed to a paradigm shift in neuroscience. Prior to the new century, conventional wisdom in neuroscience held that the adult mammalian brain is fixed in two respects: No new neurons are created (as neurons die there is no replacement), and the fundamental structure of an individual's brain is dictated by genes and is largely immutable (Doidge, 2007). A wealth of recent evidence suggests that these conventional dogmas are incorrect and informs the current neuroplasticity paradigm (Adophs, 2010). This new view holds that neurogenesis—the manufacture of new neurons within the brain—takes place well into old age (Kempermann, 2011). Furthermore, this view asserts that repeated experiences, activities, and thoughts alter gene expression, which influences the wiring of the brain (Adolphs, 2010; Davidson and McEwen, 2012). This process involves growth in cortical (brain) space devoted to processes and functions that are used more frequently and a corresponding decrease in cortical space devoted to rarely performed processes. Thus, "the very structure of our brain—the relative size of different regions, the strength of connections between one area and another—reflects the lives we have led" (Begley, 2007: 8–9).

There is strong evidence for neuroplasticity during childhood and some indication that the types of childhood environments and psychological characteristics that criminologists have linked to crime are associated with distinct neurological patterns (Davidson and McEwen, 2012; Walsh and Bolen, 2012). Magnetic resonance imaging (MRI) and functional magnetic resonance imaging (fMRI) studies indicate, for example, that exposure to harsh and unpredictable childhood conditions (e.g., parental neglect) is associated with greater volume and reactivity of the amygdala, a portion of the brain that is responsible for vigilance and emotional responsiveness to threat (e.g., Mehta et al., 2009), and alteration of the prefrontal cortex, the area responsible for executive control (Hanson et al., 2010; Wilson, Hansen, and Li, 2011). The amygdala and prefrontal cortex, as well as their interconnection, are implicated in emotional regulation (e.g., Wager et al., 2008), impulsivity (e.g., Kim and Lee, 2011; Raine, 2002b), and reactive aggression (Crowe and Blair, 2008). This research might be viewed as identifying the neurological underpinnings of criminological research showing that childhood adversity decreases self-control.

Other neuroscientific studies have linked callused instrumental aggression to decreased amygdala volume and low fear response (e.g., Fairchild et al., 2011; Raine, 2013). Importantly, evidence shows that this pattern of underresponsiveness may develop in response to severe adversity (Del Guidice et al., 2012). Rutter (2012) labeled this "experience-adaptive programming." For example, surviving in extremely harsh environments requires that one can act calmly even when danger is high (Del Guidice, Ellis, and Shirtcliff, 2011). Evidence suggests that methylation of the oxytocin receptor gene is associated with behavior deemed callous and unemotional (Kumsta et al., in press). Thus, it seems that severe adversity calibrates the biological system and molds the brain so that it becomes better adapted to deal with the dangers of a highly unpredictable and threatening

environment. Unfortunately, these adaptations also might increase the chances of aggression and crime as a response to perceived dangers and exploitation.

Moreover, evidence suggests not only that neuroplasticity takes place in childhood and is influenced by environmental variables identified as important in traditional criminology theory and research but also that it continues in adulthood (Bloss et al., 2010; Davidson and McEwen, 2012). Studies, for example, reveal the growth of areas in the brain associated with context and space among taxicab drivers (Maguire, Woolett, and Spiers, 2006) and areas associated with finger movements in virtuoso violinists (Ebert et al., 1995). Indeed, simply imagining oneself playing a simple five-note sequence repeatedly on the piano has been shown to increase the space in the motor cortex devoted to the fingers (Pascual-Leone et al., 2005). Importantly, given our concern with deviant behavior, hundreds of studies have shown that cognitive behavior therapy (CBT) can effectively change the (disturbed) thought processes and behaviors of adults with various types of psychopathology (Hofmann et al., 2012). Moreover, strong evidence suggests that these cognitive and behavioral changes are associated with neurological changes assessed with MRI and fMRI (see Jokic-Begic, 2010). CBT also has been shown to be effective in changing cognitive and behavioral patterns of antisocial persons, including prisoners (Cullen and Jonson, 2011; Lipsey, Landenberger, and Wilson, 2007), and presumably these changes brought about by CBT are mediated by changes in the brain.

Similarly, both mindfulness and compassion-focused meditation have been shown to be effective in reducing anxiety, depression, and anger while enhancing emotion regulation, empathy, and psychological well-being (e.g., Eberth and Sedimeier, 2012; Hofmann, Grossman, and Hinton, 2011). These changes have been linked to changes in gene expression (e.g., Kaliman et al., 2014; Sharma et al., 2008) and in brain function and structure (Hofmann, Grossman, and Hinton, 2011; Izel et al., 2013). As with CBT, evidence shows that meditation promotes improved mood and behavior among prison inmates (Himelstein, 2011; Perelman, Miller, and Clements, 2012). Based on the findings with nonprisoner populations, these emotional and behavioral changes are likely mediated by interrelated changes in inmates' thinking, gene expression, and neurological patterns.

In recent years, neuroscientists have published several studies of conduct-disordered youth and a few of antisocial adults showing that these individuals manifest minor differences in brain structure or function compared with more conventional individuals (see Portnoy et al., 2013; Raine, 2013). This research often is viewed as contradicting criminology's emphasis on the importance of social environmental influences insofar as these neurological differences are considered to be genetically determined. However, as we have noted, genetic determinism is a flawed and outdated perspective. The postgenomic model, with its emphasis on gene–environment interplay, argues for a *social* and *developmental* neuroscience that recognizes the effect of environmental influences on brain structure and function. It highlights the lifelong plasticity of the human developmental system and calls for research that examines the manner in which environmental (social and psychological) factors linked to crime are associated with epigenetic and neurological changes.

Summarizing, we believe that research in this postgenomic era suggests the following biosocial paradigm: The social environment, especially during the critical periods of childhood and adolescence, becomes biologically embedded. Through processes like gene expression, biological systems such as the sympathetic nervous system and hypothalamic-pituitary-adrenal axis are calibrated and the brain sculpted in a manner that prepares the individual to function and survive in existing environmental conditions. These biological

systems, then, influence the way that an individual responds to subsequent situations and events.

Importantly, new environments and experiences can change gene expression and alter biological systems. In other words, systems can be recalibrated and the brain can be resculpted. As such, the new paradigm is a biosocial life-course perspective. Under this paradigm, the challenge for biosocial criminologists entails: 1) identifying how adverse environments sculpt an individual's physiology, especially the brain, to respond to environmental events with aggression, coercion, or violence, and 2) ascertaining how environments or experiences (whether naturally occurring changes or interventions) can change the person's biological systems and, in turn, their response to situations. Importantly, we believe that not only are such integrative research efforts potentially valuable for understanding the biological processes through which the environment influences development and behavior, but also such knowledge has the potential to inform social interventions to prevent crime and humanely reform offenders (e.g., meditation, CBT, exercise, and nutrition).

This marriage of social–behavioral science, epigenetics, and neuroscience offers a more thorough multilevel and processual understanding of the etiology of offending, as well as its onset, persistence, and desistence. For example, several criminological theories emphasize the way that adverse childhood experiences give rise to cognitive/psychological traits that increase the likelihood of offending (e.g., Moffitt, 1993; Simons and Burt, 2011). Recasting these theories within a postgenomic biosocial model would involve investigating the biological pathways, including epigenetic and neurological changes, through which such adverse experiences shape changes in cognitions and behavior. Such a model would explain why criminogenic traits are difficult to change as they have epigenetic and neurological concomitants or underpinnings. But that is only part of the story.

Although the new paradigm focuses on the biological embedding of experiences, it also emphasizes the possibility of change. As noted, rehabilitative programs have been shown to modify "criminal ways of thinking" and such cognitive changes tend to be associated with changes in neurological patterns. Consonant with these findings, criminological work has shown that changes in crime are associated with changes in cognitions, including traits and schemas. Giordano, Cernkovich, and Rudolph (2002) presented evidence that desistence from crime is associated with a change in cognitive style, and Simons and Barr (in press) recently reported that romantic relationships lead to desistance because they foster a change in psychological traits and schemas. The new biosocial paradigm would suggest that these cognitive changes are likely associated with epigenetic reprogramming and resculpting of brain circuitry. In highlighting the lifelong plasticity of the biopsychosocial system, this approach emphasizes the ability of interventions to enhance the life course for all individuals.

CONCLUSION

We have argued that there is compelling evidence that heritability studies are methodologically flawed, especially for complex social behaviors such as crime. We have argued also that heritability studies are based on an oversimplified and incorrect model of gene function and that the goal of partitioning genetic versus environmental influences on variance in phenotypes is biologically unsound. We therefore recommend an end to heritability studies in criminology. Moreover, given the many flaws in heritability studies, we also

call for an end to the use of the oft-repeated version of the phrase: "We know from a wealth of behavioral genetic studies that the heritability of [insert crime or related phenotype] is roughly 50 percent." Based on the arguments and research discussed in this article, it is apparent that we unequivocally do not know this to be the case. Furthermore, no amount of quantitative genetic research can establish the validity of such heritability estimates or their putative support for the irrelevance of shared environmental factors. Technically flawed and conceptually unsound models—no matter how often published or repeated—do not by virtue of their numbers make for sound evidence.

Notwithstanding our strong objections to heritability studies, we are enthusiastic about truly biosocial research programs that focus on the interactional, bidirectional relationship between social and biological factors and do not rely on an outdated gene-centric paradigm. The postgenomic paradigm shift has brought about changes in genetics that recognize the inextricable interplay between the environment and cellular processes in the organism, as evidenced in research in epigenetics and neuroplasticity. The challenge becomes harnessing the rapid advances in these areas to enhance our understanding of the etiology of crime. As such, social scientists, geneticists, and neuroscientists are confronted with the opportunity to take steps toward forging a broader interdisciplinary understanding of how the concepts central to their disciplines influence human behavior. To achieve this deeper, integrated understanding, social scientists need the assistance of geneticists and neuroscientists, and vice versa. Transcending disciplinary silos to form such interdisciplinary alliances will enable criminologists to develop more comprehensive explanations of criminality.

At the same time, we wish to underscore that criminologists who are uninterested in biological influences or pathways and who prefer to focus on social influences should understand that their work and the importance of social factors are not undermined by biological findings at the current state of knowledge. Although some scholars have argued that "the sociological conceptualization of environmental influences, which is the sine qua non of sociological criminology, are both incorrect and not very useful" (Cleveland, Beekman, and Zheng, 2011: 249), we do not believe the evidence even faintly supports such an argument. As we have noted, the identified effects of genetic and biological factors on crime and related behaviors are consistently limited to the role of mediating or moderating the effects of environmental factors. Furthermore, it seems unlikely that social models will be undermined in the future by biological research, as the more we learn about biological and genetic influences and mechanisms, the more consequential and intertwined social influences become. However, as we point out, incorporating biological influences can make our explanations more precise and more comprehensive; thus, as a discipline, we ignore them at our own peril.

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Callie H. Burt is an assistant professor in the School of Criminology and Criminal Justice at Arizona State University. Her research focuses on criminological theories, with particular emphasis on elucidating the social, psychological, and biological mechanisms through which social factors, such as racial discrimination, community crime, parenting practices, and peers, influence criminal offending across the life course. Her recent work also has appeared in *American Journal of Sociology*, *American Sociological Review*, and *Justice Quarterly*.

Ronald L. Simons is a foundation professor in the School of Criminology and Criminal Justice at Arizona State University and a fellow in the Institute for Behavioral Research at the University of Georgia. His research has focused on onset, amplification, and desistance from various externalizing and internalizing problems. Recently, he has expanded his research program to include genes and other biological variables.

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