Biosocial Criminology

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The idea that biological *and* environmental factors are related to criminal behavior is central to biosocial criminology. Biosocial criminology is best understood as a general paradigm of research that analyzes all factors related to the etiology of antisocial behavior, meaning that genetic influences, biological influences such as hormone levels, and neurological factors are considered in combination with environmental influences like socialization, exposure to poverty, and external sources of control. This entry will introduce the biosocial criminology framework by dividing the discussion into two sections. The first section will provide a brief discussion of evolutionary criminology, biological criminology, and neurocriminology In the second section, behavioral genetics will be explored in detail because it has been the main workhorse of biosocial criminology to date. This section will provide an introduction to biosocial criminology, with a specific focus on the differences between influences attributed to "nature" and those attributed to "nurture." Note, however, that these are overlapping domains and that modern biosocial criminologists understand the dichotomy between "nature" and "nurture" as a false one. Both influences are known to matter in the etiology of behavior and, in most cases, "nature" interacts with "nurture" to produce variance in behavioral outcomes (Beaver, Barnes, & Boutwell, 2015).

Introduction to Biosocial Criminology

Biosocial criminology incorporates the effects of genetics, physiological and neurological factors, as well as influences of society and family in the causes of antisocial behavior. In this way, biosocial criminology includes at least four major domains: evolutionary criminology, biological criminology, neurocriminology, and behavior genetics (Barnes, Boutwell & Beaver, 2015). In this section, an overview of the first three domains is provided. Specifically, this section will introduce the concepts and logic underlying evolutionary criminology, biological criminology, and neurocriminology. A detailed overview of behavior genetics will be provided in a later section.

Evolutionary criminology

One perspective on criminal behavior stems from an evolutionary focus. In this sense, the evolutionary criminology perspective seeks the ultimate causes of criminal behavior, which address the broader questions that ask "why?" Evolutionary criminologists understand that humans are the product of selection pressures that were present throughout our ancestral history (Daly & Wilson, 1988). Therefore, evolutionary criminology seeks to explain human behavior (especially "universal" behaviors that are displayed in many human cultures) in the context of evolutionary pressures and responses (i.e., adaptations) to those influences (Barnes et al., 2015). Both evolutionary psychology and evolutionary criminology aim to identify ultimate causes of behavior that can be tied to selection pressures that prevailed in our ancestral environment (Daly & Wilson, 1988; Tooby & Cosmides, 2005). With this in mind, evolutionary criminology may provide answers to questions criminologists have, such as "Why are males overinvolved in violence?" and "Why does risky behavior peak during adolescence?" (Barnes et al., 2015). The answers to these questions may lie in the differential mating goals of males and females, especially as they emerge in the adolescent period of development (Boutwell, Barnes, Deaton, & Beaver, 2013; Ellis et al., 2012).

From an evolutionary view, aggressive behavior may be seen as an adaptation to pressures that were common in the environment of our ancestors. That is, human ancestors with genotypes that supported higher behavioral aggression may have been more likely to survive or reproduce

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as compared to those with other genotypes (Boutwell et al., 2013). In this way, aggressive behavior may have been passed from generation to generation via intergenerational genetic transmission. Moreover, recent evidence corroborates these claims by revealing that high-rate, persistent offenders bear more offspring compared to other members of the population (Boutwell et al., 2013). Taken together, these results suggest that evolutionary explanations of antisocial behavior can provide important insight into questions concerning why humans commit antisocial acts – even some of the most atrocious and incomprehensible acts, such as infanticide (Daly & Wilson, 1988) – and why these behaviors are notoriously difficult to eradicate at a population level.

Biological criminology

Biological criminology focuses on the physiological factors, not just genetic factors, which may be related to antisocial behavior. Although this perspective is broad, there are certain types of biological criminology research that tend to appear more often than others: hormonal associations (e.g., testosterone) with antisocial behavior (Mazur, 2009); resting heart-rate levels, which are thought to influence autonomic arousal levels that can also influence sensation-seeking behaviors (Portnoy et al., 2014); and the role of pubertal onset/development in the etiology of delinquency in adolescence (Barnes & Beaver, 2010).

Studies have shown that testosterone levels in the brain at various stages in development are quite influential on offending probabilities (Mazur, 2009). Furthermore, on average, the resting heart and pulse rates of convicted offenders are lower than the general population, including adult and juvenile offenders (Portnoy et al., 2014). Research has also provided evidence to support the notion that pubertal onset impacts delinquent behavior in adolescence, but these influences are not direct and likely interact with social factors in predicting behavioral outcomes (Barnes & Beaver, 2010; Moffitt, 1993).

Neurocriminology

Neurocriminology links brain structure and function to behavioral outcomes and, in a general sense, provides a "clear" causal pathway between genes, the brain, and behavior. We use quotations around "clear" to indicate that the logic of the causal chain is obvious. Genetic factors influence brain functioning/structure, which goes on to impact behavioral outcomes. Note, however, that this relatively simple causal chain is, in reality, one of the most complex puzzles known to man. Indeed, the brain acts as a mediator variable between "risk" factors identified above and the behavioral outcomes related to antisocial behavior. It has only recently been demonstrated in neuroscience research that certain regions of the brain may be critically important for understanding the causes of antisocial behavior (Raine, 2013). Though this is an emerging area of interest that has received relatively less attention than the other aspects of biosocial criminology, recent evidence provides evidence of a link between direct measures of neurological functioning and criminality (Aharoni et al., 2013; Pardini, Erickson, Loeber, & Raine, 2014). Although neuroscience research has matured in a relatively short time span, exactly how the brain works to impact behavioral outcomes remains shrouded in mystery.

Behavioral Genetics: Nature versus Nurture

This section provides an overview of the research using behavioral genetic techniques. Specific emphasis will be placed on the three components of behavioral genetics: heritability, the shared environment, and the nonshared environment. A synopsis of several studies is included, though not all are exclusive to the field of criminology.

Behavioral genetics is a field of study that aims to unpack the genetic and environmental influences on human behavioral outcomes (Barnes et al., 2014b; Beaver, 2013). Research in behavioral genetics typically comes in one of two forms. One form of behavioral genetic research estimates a latent genetic effect by studying kinship pairs. If the assumptions of behavioral genetic models are met (Barnes et al., 2014b), a latent measure of genetic influences can be inferred if siblings who share more genetic material are more similar to one another than siblings who share less genetic material. The second form of behavioral genetics utilizes molecular genetic markers

(i.e., specific genes) to estimate the impact of a gene on a behavioral outcome. In this way, behavioral genetics offers scientists an avenue by which both genetic and environmental influences on human behavior and personality traits can be analyzed. Behavioral genetics has been a centerpiece of biosocial criminology and it represents the integrative nature of biosocial criminology by giving equal weight and attention to biological *and* environmental factors.

Nature: Heritability

Behavioral genetic studies estimate the proportion of variance in a trait that is due to three different components: a heritability component, a shared environmental component, and a nonshared environmental component. Heritability (symbolized as h^2) captures the estimated amount of variance in a trait that is attributable to additive genetic factors. Heritability explains variance at the group level but does not provide information about genetic factors for individuals. In other words, heritability estimates allow one to estimate whether and how much genetic variance in the population contributes to behavioral variance in the population. Heritability does not, however, tell us how much genetic factors influence the behavioral traits of a single individual. Heritability estimates, therefore, should not be equated with a fixed constant value because they can fluctuate over time and over developmental periods (Ferguson, 2010).

Research indicates that heritability estimates fluctuate over the life course. For instance, heritability estimates may be higher during childhood and lower during adolescence; or relatively low during adolescence, then become much stronger in adulthood (Ferguson, 2010). The effects under h^2 are not based solely on genetic factors; the estimates also include the effects of gene x environment interactions (GxE), which may involve genetic factors interacting with shared environmental factors. Additionally, heritability estimates comprise the effects of gene x environment correlations (rGE). Gene x environment correlations occur when genetic factors are antecedent predictors of environmental exposure (Barnes et al., 2014b).

In a general sense, there are two different types of heritability, broad heritability (h_B^2) and narrow heritability (h^2). All sources of genetic variance (additive, dominance, and epistasis, explained below) are captured by broad (or broad-sense) heritability (h_B^2). Narrow (or narrow-sense) heritability (h^2) only taps into the additive genetic variance and, therefore, is a more restricted measure of heritability (Beaver, 2013). Because genetic effects come in several different types, it is important to understand the differences between additive effects, dominant effects, and epistatic effects. Additive genetic variance is simply the sum of each gene's contribution to phenotypic variance. There is a presumption that the effect of each gene on the phenotype does not depend on the effects of the other genes (Barnes et al., 2014b). In other words, the additive component is typically estimated by summing up the average effects of individual genes across the entire genome (Lemery & Goldsmith, 1999; Plomin, DeFries, Knopik, & Neiderhiser, 2013). Note that alternative copies of a gene are called "alleles" and the term "genotype" references the specific grouping of alleles for a particular gene. Dominance effects and epistasis effects are two sources of non-additive genetic variance. Dominance effects capture the interaction between the alleles of one gene (Beaver, 2013). Dominance deviations are most commonly understood as the Mendellian inheritance patterns where a person's two alleles at one spot on the genome are important for predicting variance in an outcome. The second type of non-additive genetic variance is epistasis. Epistasis refers to the interaction of alleles at two different places in the genome (Plomin et al., 2013).

Research conducted under the framework of behavioral genetics has indicated that certain behaviors and disorders are influenced by genetic factors, including antisocial personality disorder, substance abuse, impulsivity, low self-control, and attention deficit hyperactivity disorder (Beaver, 2013). There are varying definitions for antisocial behaviors, but Ferguson (2010, p. 2) describes the term as including "both the innate traits and motivation that directs individuals toward antisocial behavior (i.e., antisocial personality disorder, psychopathy) and antisocial behavior itself (i.e., aggression, violence, lying, stealing, etc.)." Several personality–kinship studies have shown that the similarity between individuals can often be predicted based on their percentage of shared genes and not the shared environment (Barnes

et al., 2014b; Lemery & Goldsmith, 1999; Plomin et al., 2013). In their international adoption study, van den Oord, Boomsma, and Verhulst (1994) reported strong genetic influences on both delinquency and aggression. Genetic effects were key for externalizing scales and attention problems but were nearly nonexistent for internalizing scales (van den Oord et al., 1994, p. 201). Mednick, Gabrielli, and Hutchings' early adoption study (1984, p. 891) also paved the way for later behavioral genetic research into the etiology of antisocial behavior. For example, in regard to property crime convictions, the authors found a statistically significant correlation between the adoptees and their biological parents; however, this was not true for violent crimes. Mednick and colleagues compared adoptees whose biological parents did not have a criminal record with adoptees whose biological parents had been arrested, and found that the latter group was more likely to report antisocial behavior as compared to the former group. Summarizing their findings, Mednick and colleagues (1984, p. 893) concluded that "some factor transmitted by criminal parents increases the likelihood that their children will engage in criminal behavior." Additionally, they noted the findings "imply biological predispositions are involved in the etiology of at least some criminal behavior" (Mednick et al., 1984, p. 893).

The above discussion raises the question of how much genetic factors influence variance in antisocial behavior. Luckily, there are several reviews and meta-analyses that offer guidance on this point. Mason and Frick's (1994) meta-analysis examined 12 twin studies and three adoption studies of antisocial behavior. The overarching conclusion from their study was that roughly 50% of the variance in antisocial behavior was attributable to genetic influences while the remaining 50% was attributable to environmental factors. This conclusion has since been supported by other meta-analyses and systematic literature reviews (Burt, 2009a, 2009b; Ferguson, 2010; Moffitt, 2005; Rhee & Waldman, 2002).

While most of the behavioral genetic literature has focused on the impact of genetic factors on measures of antisocial behaviors, some scholars have begun to expand their focus toward key *predictors* of antisocial behavior such as peer behavior. Cleveland, Wiebe, and Rowe (2005, p. 153) reported that the variance in adolescents' exposure to friends who smoke and drink could be explained by genetic influences ($h^2 \approx .64$). In other words, the formation of friendships with substance-using peers may be influenced by genetic factors, which then contributes to adolescents' exposure to substance use behaviors. The authors concluded that these findings provide evidence of either evocative or active gene–environment correlations, or both (Cleveland et al., 2005).

Nurture: Shared and nonshared environments

An important development from behavioral genetic research is the idea that environmental influences on antisocial/criminal behaviors can come in two forms: shared environments or nonshared environments. During the twentieth century, environmental explanations, especially those focusing on learning, became more prominent in the field of psychology, eventually finding their way into criminological research (Ferguson, 2010). Initial studies on behavior genetics divided phenotypic variance into environmental and genetic components, the former being further separated into shared and nonshared environment (DiLalla, 2002). Shared environmental (symbolized as c^2) factors tap the environmental influences that make siblings more similar to one another. More specifically, shared environments may include growing up in the same household, in the same neighborhood, and going to the same schools.

In contrast, nonshared environmental (symbolized as e^2) factors are the environmental effects that make siblings differ from one another (Beaver, 2013). For example, nonshared environments may encompass random events or environmental effects in which siblings differ in their perceptions or subjective interpretations of an incident (Turkheimer & Waldron, 2000). Common examples are different prenatal environments, different schools, and different peers. Shared and nonshared environmental components capture the effects of socialization factors and the effects, for example, of nonsocialization, nongenetic factors. These include birth complications, diseases, and head injuries (Plomin et al., 2013).

Shared environments When working from a behavioral genetic focus, it is easy to see that criminologists typically study shared environmental effects (Beaver, 2013), while simultaneously ignoring nonshared environmental effects. However, several decades of behavior genetic studies have been consistent in finding that almost none of the parent–child behavioral similarities, and very little of sibling similarities, is due to having been reared by the same parents, in the same home. Much of the similarity between siblings is, instead, likely to be caused by genetic overlap (Plomin et al., 2013; Udry, 1995). In other words, the shared environment often accounts for little to no variance in measures of antisocial behavior. Most sociological theories of family influence, however, appear to focus on shared environments (Udry, 1995). Indeed, many sociological theories make explicit assumptions or hypotheses that emphasize the significance of familial influences on the socialization of children in creating individual differences (Udry, 1995). This complicates efforts to interpret the validity of this body of evidence because genetic factors, until recently, have been ignored by sociological and criminological research (Moffitt, 2005).

A classic example can be found in one of the most influential theories in criminology, Gottfredson and Hirschi's (1990) *General Theory of Crime*. Gottfredson and Hirschi wrote that low self-control is the personality trait responsible for much of criminal and antisocial behavior. A great deal of research has supported this hypothesis, including the meta-analysis by Pratt and Cullen (2000). The results of the meta-analysis indicate that, "regardless of measurement differences, low self-control is an important predictor of crime and of 'analogous behaviors'" (Pratt & Cullen, 2000, p. 931). Furthermore, according to Gottfredson and Hirschi (1990), parental socialization techniques, parental discipline, and parental recognition of a child's transgressions all point toward the beginning of self-control. The results from Hay's (2001) study show that self-control theory receives some measure of empirical support for Gottfredson and Hirschi's (1990) parental socialization thesis. When Wright and Beaver (2005) tested the parental socialization hypothesis using behavioral genetic methods, however, they found no consistent association. In other words, once genetic influences were taken into account, the impact of parental influences on the child's level of self-control was found to be near zero. Confounding occurs when the relationship between the original independent variable (cause) and dependent variable (effect) disappears after another variable (called the confounding variable) is introduced into the equation. The analysis by Wright and Beaver (2005) reveals that the parental socialization hypothesis may have been confounded by the omission (but subsequent introduction) of genetic factors into the model (see the analysis by Barnes, Boutwell, Beaver, Gibson, and Wright, 2014a for a more detailed discussion and simulation of the problem of genetic confounding in criminological research).

Nonshared environments Research indicates that most of the environmental variance in antisocial behaviors is attributable to the influence of nonshared environmental factors (Ferguson, 2010). On average, research has repeatedly found that nonshared factors account for approximately 30–50% of the variance, depending again on the sample and the study's design, such as twin-based studies using monozygotic (MZ) and dizygotic (DZ) twins (Beaver & Barnes, 2012; Moffitt, 2005). Nonshared environments are often comprised of environments outside of the home such as differential associations (Akers, 1998). However, this is not necessary; nonshared environments are also found within families. For example, research has indicated that parents often treat their own children quite differently (Harris, 1998). Indeed, the premise of Harris's (1998) work was that nonshared environmental effects emerge even in ostensibly *shared* environments. In short, the emergence and importance of nonshared environments is a critical component to understanding the developmental etiology of antisocial behavior across the life course.

The Future of Biosocial Criminology: Combining Genetic and Environmental Effects

While the above discussion may lead the reader to believe the world works in an "either/or" fashion, meaning that influences are either genetic or they are environmental, nothing could be further from

the truth. As biosocial criminology has matured over the past few decades, one piece of evidence has repeatedly come to light. Specifically, it is now glaringly obvious that human behavior, such as antisocial behavior, is the product of a complex arrangement of both genetic *and* environmental influences. It is, for all intents and purposes, useless to discuss an outcome as being genetic or not. Biosocial criminologists are acutely aware that antisocial behavior requires genetic and environmental factors to coalesce, to interact, and to mediate one another in their impact on behavior (Barnes et al., 2014b).

A study by Jaffee and colleagues (2005) provides an example of this line of thinking. These researchers tested whether the effect of physical maltreatment on risk for behavior issues was strongest among those who had a genetic risk toward antisocial outcomes. They found that "the experience of maltreatment was associated with an increase of two percent in the probability of a conduct disorder diagnosis among children at low genetic risk for conduct disorder, but an increase of 24% among children at high genetic risk" (2005, p. 67). Lastly, they found that some genotypes may stimulate opposition to trauma (Jaffee et al., 2005).

Another noteworthy topic within the realm of gene–environment interplay is epigenetics. The epigenome has chemical markers along the strands of DNA (Beaver, 2013). The gene's "job" consists of coding for the production of protein, but the epigenome controls the activation and inactivation of genetic markers. To understand this better, an explanation must be provided about ribonucleic acid (RNA). The chemical markers attributed to the epigenome affect gene activity because they alter the ability of DNA to be duplicated onto ribonucleic acid (RNA). The nucleotides on RNA have all the information necessary to assemble the amino acids that will eventually end in the production of proteins. Once the genetic information has been copied into RNA, RNA departs from the cell nucleus and goes into the cytoplasm (Beaver, 2013). Regarding the epigenetic chemical markers, Beaver (2013) explains that some of them enhance gene activity, but other chemical markers silence or decrease gene activity (i.e., the DNA → RNA process). In response to the environment, the chemical markers on the epigenome can change throughout life. These epigenetic changes are able to alter genetic expression. Thus, the epigenome is also partly accountable for creating variance in phenotypes. Interestingly, these epigenetic modifications can be passed to later generations.

By analyzing MZ twin pairs, studies have detected epigenetic modifications. Because MZ twins, though they share 100% DNA, do not always turn out (behaviorally) alike, the differences could be a result of exposure to different environments that have altered their epigenome. Hence, these alterations may cause certain genes to be differentially expressed, creating phenotypic variations (Beaver, 2013). Though the study of epigenetics remains in its infancy, it is perhaps the best example of the complex relationship between genetic and environmental influences. Epigenetics, at its core, reveals that the human genome can be influenced by the prevailing social/environmental world. In a very real sense, the environment can "turn on" or "turn off" certain genes and, therefore, can impact behavior via genetic regulation. We are excited to see what the future holds for biosocial criminology, especially as it concerns epigenetic effects.

SEE ALSO: Aggression and Crime; Antisocial Behavior and Crime; Biology and Crime; Personality and Crime.

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