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Biosocial Interactions and Correlates of Crime

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Abstract

Every level of analysis of biological factors - from molecular genetics, to brain structure and function, to neuropsychological performance - has found links between biology and antisocial behavior. Likewise, a number of social or environmental factors - maltreatment, socioeconomic status, education, and so on - are believed to contribute to crime and aggression. Over the past two decades, increasing interest in the interaction between biological and social factors in various behaviors and disorders has led to several fruitful lines of research. A great deal of such research has supported the interacting roles of nature and nurture in the development of criminality. This chapter will provide an overview of some of the major biosocial findings in research on crime and antisocial behavior.

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Biosocial Interactions and Correlates of Crime

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Introduction

Every level of analysis of biological factors – from molecular genetics, to brain structure and function, to neuropsychological performance – has found links between biology and antisocial behavior. Likewise, a number of social or environmental factors – maltreatment, socioeconomic status, education, and so on – are believed to contribute to crime and aggression. Over the past two decades, increasing interest in the interaction between biological and social factors in various behaviors and disorders has led to several fruitful lines of research. A great deal of such research has supported the interacting roles of nature and nurture in the development of criminality. This chapter will provide an overview of some of the major biosocial findings in research on crime and antisocial behavior.

Early Health Risks

Research on early health risk factors offers some of the most persuasive evidence that interactions of biological and social risk factors significantly increase risk for criminal behavior. Several pre- and peri-natal factors including minor physical anomalies, nicotine or alcohol exposure, and birth complications have been shown to predispose to crime, especially when combined with environmental risk factors.

Minor Physical Anomalies

Minor physical anomalies (MPAs) consist of fairly minor physical abnormalities such as adherent ear lobes, a single palmar crease, and a furrowed tongue. MPAs have been linked to pregnancy disorders and are viewed as biomarkers for fetal neural maldevelopment near the end of the first trimester (Firestone & Peters, 1983). Since the epidermis and the central nervous system (CNS) have shared embryological origins, MPAs are considered indicators of atypical CNS and brain development. While there may be a genetic component to MPAs, they may also be caused by environmental factors that affect the fetus, such as anoxia, bleeding, and infection (Guy, Majorski, Wallace, & Guy, 1983).

Primary Findings

A number of studies have found a relationship between elevated numbers of MPAs and increased antisocial behavior in children, adolescents, and adults (Raine, 1993). MPAs have been particularly linked to violent as opposed to nonviolent offending. For instance, Arseneault, Tremblay, Boulerice, Seguin, and Saucier (2000) showed that MPAs measured at age 14 in 170 males predicted violent but not nonviolent delinquency at age 17. The authors reported that these effects were independent of childhood physical aggression or family adversity. In another study, an increased level of MPAs was associated with recidivistic violent criminal behavior (Kandel, Brennan, Mednick, & Michelson, 1989). Kandel et al. (1989) assessed MPAs in 265 11- to 13-year-old Danish children, and found that recidivistic violent offenders had a greater number of MPAs compared with subjects with one or no violent offenses, according to police records of criminal behavior when the subjects were 20 to 22 years of age. These studies suggest that prenatal insults toward the end of the first three months of pregnancy may increase risk for violent behavior as a result of abnormal brain development.

Biosocial Interactions

Several studies have reported that MPAs interact with psychosocial factors in predisposing to crime. Mednick and Kandel (1988) assessed MPAs in 129 boys during visits to a pediatrician at age 12 and found that MPAs were related to violent crime, but not nonviolent property offenses when the subjects were 21 years old. However, when the authors examined family stability and intactness as a moderating factor, they found that MPAs only predicted later criminal involvement for those reared in unstable, nonintact homes. Brennan, Mednick and Raine (1997) reported a similar finding using a sample of 72 male offspring of parents with psychiatric diagnoses. Particularly high rates of adult violent crime were found in individuals who had both family adversity and MPAs compared to those who had only one of these risk factors. Another study, by Pine, Shaffer, Schonfeld, and

Davies (1997), examined whether MPAs interacted with environmental risk factors, such as low socioeconomic status (SES), spousal conflict, and marital disruption, in predicting later disruptive behavior disorders. Pine et al. (1997) found that individuals with both increased MPAs and environmental risk, assessed at age seven, had an elevated risk for disruptive behavior in general, and conduct disorder, in particular, at age 17. These three studies suggest that MPAs interact with adverse environmental experiences such that psychosocial factors predispose to criminal and violent behavior more strongly, and sometimes only, among individuals with high biological risk. Neurodevelopmental abnormalities such as MPAs thus appear to increase susceptibility to psychosocial risk factors for antisocial and violent behavior.

Prenatal Nicotine and Alcohol Exposure

Primary findings

A convergence of evidence from multiple sources has convincingly demonstrated that children who are exposed to maternal smoking during pregnancy have an elevated risk of later criminal behavior throughout the life-course (see Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002, for a review). Studies have shown that maternal prenatal smoking predicts childhood externalizing behavior, conduct disorder, delinquency, and adult criminal and violent offending (Brennan, Grekin, & Mednick, 1999; Brennan, Grekin, Mortensen, & Mednick, 2002; Fergusson, Horwood, & Lynskey, 1993; Fergusson, Woodward, & Horwood, 1998; Orlebeke, Knol, & Verhulst, 1997; Rantakallio, Laara, Isohanni, & Moilanen, 1992; Wakschlag et al., 1997; Weissman, Warner, Wickramaratne, & Kandell, 1999). Three studies have also documented a dose-response relationship between the degree of maternal smoking during pregnancy and the extent of later criminal behavior in offspring (Brennan et al., 1999; Maughan, Taylor, Caspi, & Moffitt, 2004; Maughan, Taylor, Taylor, Butler, & Bynner, 2001).

Fetal alcohol exposure is also an established risk factor for antisocial behavior in children, adolescents, and adults (Fast, Conry, & Looock, 1999; Olson et al., 1997; Streissguth, Barr, Kogan, & Bookstein, 1996). Fetal Alcohol Syndrome (FAS) is characterized by a host of cognitive, behavioral, social, and physical deficits and results from heavy alcohol consumption during pregnancy. However, deficits are observed even in those who have been prenatally exposed to alcohol yet do not meet diagnostic criteria for FAS (Schonfeld, Mattson, & Riley, 2005). For example, two studies found high rates of delinquency in children and adolescents with heavy fetal alcohol exposure, even if they did not have FAS (Mattson & Riley, 2000; Roebuck, Mattson, & Riley, 1999). In addition, research has demonstrated that adolescents who were prenatally exposed to alcohol are overrepresented in the juvenile justice system (Fast et al., 1999). One study found that 3 percent of adolescents in a juvenile inpatient forensic psychiatry unit were diagnosed with FAS, and 22 percent were diagnosed with fetal alcohol effects (Fast et al., 1999). Another study reported that 61 percent of adolescents, 58 percent of adults, and 14

percent of children between the ages of six to 11 with fetal alcohol exposure had a history of trouble with the law (Streissguth et al., 1996).

Biosocial interactions

A number of studies have revealed interactions between maternal smoking during pregnancy and psychosocial risks in the prediction of later crime. Importantly, these studies had large sample sizes, assessed long-term outcomes, prospectively collected data, and controlled for potential confounds such as parental antisocial behavior, drug use, and low SES. In one study using a sample of 4,169 males born between 1959 and 1961 in Denmark, Brennan et al. (1999) found a dose-response relationship between the number of cigarettes subjects' mothers had smoked daily while pregnant, and the extent of subjects' nonviolent and violent crime at 34 years of age. Moreover, Brennan et al. (1999) reported that these effects were specific to persistent criminal behavior, rather than delinquency in adolescence. Arrest records revealed that subjects whose mothers smoked 20 cigarettes a day while pregnant had a two-fold increase in adult violent offending. However, the authors found that when maternal prenatal smoking was combined with delivery complications, there was a five-fold increase in adult violent offending; in contrast, prenatal nicotine exposure without delivery complications did not lead to increased violence in offspring. Another study, by Rasanan et al. (1999), found a two-fold increase in violent crime at age 26 in the offspring of women who smoked during pregnancy, and showed that prenatal nicotine exposure combined with being raised in a single-parent family, led to an 11.9-fold increase in recidivistic violent offending. Furthermore, prenatal nicotine exposure combined with four psychosocial risk factors (teenage pregnancy, single-parent family, unwanted pregnancy, and developmental motor delays), led to a 14.2-fold increase in recidivistic violence. As in the previous study, this study found increased risk particularly for persistent violent offending, rather than violence in general or property crime. Finally, a study by Gibson and Tibbetts (2000) documented an interaction between prenatal nicotine exposure and parental absence in predisposing to early onset of antisocial behavior and offending.

Birth Complications

Biosocial interactions

Several methodologically rigorous studies have demonstrated that obstetric complications interact with psychosocial risk factors in predicting conduct disorder, delinquency, and impulsive crime and violence in adulthood. In one study, birth complications combined with a disruptive family environment (which included such experiences as maternal separation, illegitimacy, marital discord, parental mental health problems, and paternal absence) predisposed to delinquency over and above either biological or psychosocial risk factor independently (Werner, 1987). Raine, Brennan, and Mednick (1994, 1997) also demonstrated the importance of biosocial

interactions in predicting violent crime in two prospective longitudinal studies. In the first of these studies, Raine, Brennan, and Mednick (1994) investigated whether the early experience of extreme maternal rejection (e.g., unwanted pregnancy, attempts to abort the fetus, and institutional care of the infant during the first year of life) interacted with birth complications in predisposing to adult violent crime in a sample of 4,269 males born in Copenhagen, Denmark, between 1959 to 1961. Raine, Brennan, and Mednick (1994) demonstrated that birth complications significantly interacted with maternal rejection in predisposing to violent crime at 18 years of age, and that while only 4 percent of the sample experienced both birth complications and maternal rejection, this group was responsible for 18 percent of the violent offenses perpetrated by the whole sample. In a follow-up study, Raine, Brennan, and Mednick (1997) reassessed criminal violence in the same sample at age 34. The authors replicated the biosocial interaction for violent but not nonviolent crime. Moreover, they showed that the results applied specifically to serious violence, rather than violent threats, and to early-onset as opposed to late-onset violence. Obstetric complications have been shown to interact with various psychosocial risk factors (parental mental illness, poor parenting, familial adversity) in a number of studies using large samples from around the world (Arseneault, Tremblay, Boulerice, & Saucier, 2002; Brennan, Mednick, & Mednick, 1993; Hodgins, Kratzer, & McNeil, 2001; Piquero & Tibbetts, 1999).

In contrast to these findings, two studies failed to document an interaction between birth complications and environmental risk factors (Cannon et al., 2002; Laucht et al., 2000). However, these studies differed in several important ways from the studies cited above. Cannon and colleagues (2002) examined 601 individuals with schizophrenia spectrum disorders; thus, other differences in brain functioning in this sample may have obscured findings related to violence. Laucht et al. (2000) only followed a small sample of 322 children until eight years of age. Raine and colleagues (Raine, 2002a and 2002b; Scarpa & Raine, 2007) have argued that CNS insults resulting from perinatal complications may be particularly related to life-course persistent antisocial behavior, rather than child antisocial behavior. A majority of evidence thus suggests that birth complications interact with psychosocial risk factors in predisposing to violent crime.

Genetics

Compelling evidence for genetic influences on criminal behavior comes from studies using a diverse set of methodological approaches (Beaver, DeLisi, Wright, & Vaughn, 2009; Popma & Raine, 2006). Researchers attempting to determine the relative importance of genetic contributions to crime have employed twin, adoptive, and molecular genetic designs. Although studies have found heritability estimates that range from 7 to 85 percent, the majority of studies have reported heritability estimates in the 40 to 60 percent range. (Arsenault et al., 2003; Beaver et al., 2009; Jaffee et al., 2005; Jaffee, Caspi, Moffitt, & Taylor, 2004; Lyons et al., 1995;

Miles & Carey, 1997; Moffitt, 2005; Rhee & Waldman, 2002; Rowe, 1986; Slutske et al., 1997). Methodological differences between studies, such as how antisocial behavior is operationalized, sample age, age of offending onset, and gender have all likely contributed to variation in heritability estimates. For example, some researchers have operationalized antisocial behavior in terms of behavior (i.e. the violation of legal or social norms), while others have defined it as the presence of a psychiatric diagnosis such as conduct disorder, while still others have used aggression severity.

One important moderator of the magnitude of genetic and environmental influences on delinquency appears to be sample age. Research has revealed that the relative importance of genes and environment in the etiology of criminal behavior shifts across the lifespan (Goldman & Ducci, 2007). The majority of studies have reported lower heritability estimates and higher shared environmental contributions to delinquency and conduct problems in childhood compared to adolescence (Jacobson, Prescott, & Kendler, 2002; Lyons et al., 1995; Miles & Carey, 1997). This is consistent with the notion that genetic influences on delinquency tend to increase with age while shared environmental effects tend to decrease (Goldman & Ducci, 2007). Studies have also found that early and persistent delinquent behavior is more heritable than childhood-limited antisocial behavior. In addition, while some genes have been found to influence criminal behavior across the lifespan, others only have an effect during adolescence or adulthood (Goldman & Ducci, 2007).

Research has also suggested that heritability estimates differ for aggressive versus non-aggressive offending. For instance, studies have found that aggressive offending, which includes physical acts of aggression such as fighting, is more heritable than non-aggressive offending, which includes behavior such as rule-breaking and theft (Eley, Lichtenstein, & Moffitt, 2003). Conversely, shared environmental factors, such as family criminality, family poverty, and poor parenting, appear to have a greater effect on non-aggressive offending, although genetic influences have also been found to influence some of these factors (Moffitt, 2005).

Studies have implicated several candidate genes in the development of offending and antisocial behavior; however, none of these genes have been found to account for a large percentage of the phenotypic variance in crime (Goldman & Ducci, 2007). This suggests both that many genes are involved in increasing vulnerability to antisocial behavior, and that gene–environment interactions are involved in the etiology of criminal behavior. One particularly influential study documenting a gene–environment interaction in the prediction of antisocial behavior was performed by Caspi and colleagues (2002). In a large sample of male children in New Zealand, the authors examined a functional polymorphism in the gene encoding monoamine oxidase A (MAO-A; a neurotransmitter-metabolizing enzyme) and childhood maltreatment. Maltreated children with a genotype conferring high levels of MAO-A expression were less likely to develop conduct disorder in adolescence or engage in violent crime in adulthood than maltreated children with a genotype conferring low levels of MAO-A expression.

Caspi et al.'s (2002) study was one of the first to reveal that children's susceptibility to environmental risk factors may be moderated by specific genotypes. However,

not all studies have revealed the same pattern of results. Nilsson et al. (2008) found that female adolescents with high expression of MAO-A were *more* likely to exhibit impulsive aggression when exposed to an unfavorable environment. Similarly, a recent study found that childhood physical abuse was associated with increased violent recidivism in individuals with the high MAO-A activity genotype (Tikkanen et al., 2010). In this study, MAO-A genotype alone had no main effect on the risk of recidivism. These studies emphasize that biological and social factors considered alone are often not able to predict criminal behavior, but must be considered within the same context.

Overall, these studies show that a commonly studied social factor such as childhood adversity may result in different patterns of antisocial behavior depending on the biological makeup of the individual. Caspi et al.'s (2002) findings may also help explain why some children who experience maltreatment do not become violent or abusive and display resilience in the face of environmental adversity, whereas others perpetuate the cycle of violence in later life. Thus, research employing a wide array of methodologies has persuasively shown that there are genetic influences on criminal and antisocial behavior.

Hormones and Neurotransmitters

At the molecular level, there have also been findings of interactions between biological and social factors, particularly in studies of hormones and neurotransmitters. These studies have generally found that the combination of biological and social variables is better able to explain antisocial behavior than either type of variable alone.

Hormones

Hormones are molecules that are released into the bloodstream and travel to act at a different location in the body. Common hormones that have been associated with antisocial behavior are cortisol and testosterone. Cortisol is a glucocorticoid hormone that is part of the body's stress reactivity network and serves to mobilize the body's resources and to provide energy in times of stress (Kudielka & Kirschbaum, 2005). Biologically-based studies have generally found cortisol levels to be reduced in antisocial children (van Goozen, Matthys, Cohen-Kettenis, Thijssen, & van Engeland, 1998), adolescents (Loney et al., 2006), and adults (Cima, Smeets, & Jelicic, 2008; Holi et al., 2006), suggesting that these individuals may be less responsive to stressors and may be less fearful of negative consequences such as potential punishment.

Testosterone is a sex hormone that is part of the hypothalamic-pituitary-gonadal (HPG) axis. It is primarily released by the testes in males and the ovaries in females. Males have several times the amount of testosterone as females. Because there are large sex differences in antisocial behavior, with the male-to-female ratio being

about 4:1 for antisocial personality disorder and as large as 10:1 for violent crimes (van Honk & Schutter, 2007), it has been hypothesized that testosterone may be involved in aggressive behavior. Elevated testosterone levels have been associated with antisocial behavior and violent crime in adults (Banks & Dabbs, 1996; Dabbs, Frady, & Carr, 1987).

Only a few studies have explored whether there are interactions between hormone measurements and social factors. This may be partly due to the fact that social factors are not measured or are not analyzed in many biologically-based studies. The findings from the following studies suggest that social factors may be important in gaining a complete picture of the role of hormones as a risk factor for antisocial behavior.

Dabbs and Morris (1990) found that high testosterone was associated with higher levels of childhood and adult delinquency in participants of low socioeconomic status (SES), but this relationship was much weaker in participants with high SES. Although the mechanism of this effect is unknown, one possibility is that individuals with high SES have protective factors that help them to avoid antisocial behavior, despite their high levels of testosterone. In another study, Mazur (1995) found that a combined biosocial model involving the hormones cortisol, testosterone, and thyroxin, in combination with social factors including age, education, and income better predicted delinquent behavior than a biological or social model alone. This emphasizes the importance of including both biological and social factors in order to understand how antisocial behavior develops.

An exciting new area of hormone research has begun to explore whether social factors, in the form of a psychosocial intervention, may be able to *change* hormone responses in order to reduce antisocial behavior in those at risk. Brotman et al. (2007) conducted a 22-week family-based intervention of preschool-age children at risk for antisocial behavior, and found that relative to controls, children in the intervention condition had increased cortisol levels in anticipation of a social challenge; the experimentally-induced change in cortisol levels resembled patterns found in normally developing children at low risk for antisocial behavior. This suggests that changes in external, social factors are able to alter an individual's hormonal responding, providing direct evidence that biological and social factors interact in predisposing for antisocial behavior. Contrary to the popular belief that biological factors are fixed and cannot be changed, this study shows that socially based interventions may be effective largely because of their ability to change biological responding; this study showed that changes in cortisol accounted for 69 percent of the intervention effect on reducing childhood aggression.

Furthermore, this research group found that the intervention had a stronger effect on cortisol among families demonstrating lower warmth in the home (O'Neal et al., 2010). They conducted a follow-up study of these youth and found that among lower warmth families, the intervention-induced cortisol response was predictive of later aggression levels. This suggests that the family environment is also an important factor to consider when attempting to improve biological responding. Taken together, these studies suggest that hormones act within an environmental

context to predispose to antisocial behavior, and that changes in the environment can have an impact on hormones.

Neurotransmitters

Neurotransmitters are molecules that are used to signal between neurons in the central nervous system. A neurotransmitter released from one neuron affects the functioning of adjacent neurons. Common neurotransmitters include serotonin, dopamine, and norepinephrine. Related molecules include the metabolites of neurotransmitters, such as the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA).

Numerous studies have examined the neurotransmitter serotonin in criminal groups. In a review of the literature, Berman and Coccaro (1998) conclude that reduced serotonin activity is related to aggressive behavior, particularly in those who commit or attempt to commit crimes with significant potential for harming others, such as arson and homicide. In a meta-analysis by Moore, Scarpa, and Raine (2002), the effect size for the relationship between the serotonin metabolite 5-HIAA levels and antisocial behavior was -0.45. In addition to studies that have established a relationship between serotonin and antisocial behavior, one study found that antidepressants, which increase serotonin, reduce aggression in humans (Coccaro & Kavoussi 1997).

Biosocial interactions have also been observed in studies of serotonin as well as genes that code for it. Moffitt et al. (1997) found that violent offenders with high blood serotonin levels who came from a conflicted family background were more than three times more likely to become violent by age 21 than men with only the biological or only the social risk factor. Similarly, Marks et al. (2007) found that children with low central serotonin reactivity were increasingly likely to exhibit increased conduct problems as the magnitude of psychosocial risk increased. A molecular genetics study by Sadeh et al. (2010) found that the psychopathic traits "callous-unemotional" and "narcissism" were associated with SES in youth with two "long" variants of the serotonin transporter gene polymorphism. This gene variant is thought to alter the serotonin system, although the exact effect on serotonin transmission in the brain is not clear. Overall, these studies suggest that alterations in serotonergic function may confer risk for antisocial behavior by heightening vulnerability to environmental stress.

Overall, these findings suggest that hormones and neurotransmitters often interact with social and environmental factors to increase the likelihood of antisocial behavior. The mechanisms by which hormones and neurotransmitters may interact with social factors to result in antisocial behavior are not well understood, so future studies will be necessary to further elucidate these relationships. Increasing our understanding of how biological and social factors interact will likely have implications for the prevention and treatment of crime.

Brain Imaging

In the last few decades, an increasing amount of research has been directed toward understanding the neurobiological etiology of criminal behavior. Recent brain imaging studies have been extremely informative in providing the empirical evidence connecting structural and functional deficiencies in several brain regions with criminal behavior. Regarding structural abnormalities associated with criminal behavior, studies have largely focused on regions involved in decision-making (e.g. prefrontal cortex) and emotion regulation (e.g. amygdala, hippocampus). For example, Woermann et al. (2000) found reduced left prefrontal gray volumes in aggressive epileptic patients compared to non-aggressive epileptic patients. Supportively, Yang et al. (2005; Yang, Raine, Colletti, Toga, & Narr, 2010) also found reduced gray matter volume and thickness in the middle frontal and orbitofrontal cortex and reduced volume and surface deformations in the amygdala in psychopaths with prior convictions (i.e., unsuccessful psychopaths) compared to psychopaths without convictions (i.e., successful psychopaths) and non-psychopathic controls. In one recent study, Yang et al. (2010) revealed reduced hippocampal and parahippocampal volumes in murderers with schizophrenia compared to schizophrenia patients and non-violent controls, which is in line with another study by Boccardi et al. (2010) showing abnormal hippocampal morphology in habitually violent offenders. Using *vivo* diffusion tensor magnetic resonance imaging tractography, one recent study by Craig et al. (2009) further showed impaired amygdala-orbitofrontal connections in psychopaths with convictions. These studies have provided some initial evidence for brain morphological alterations associated with criminal behavior.

In common with structural imaging studies, several functional imaging studies have presented evidence suggesting impaired brain functioning in criminal offenders in several brain regions, especially in the prefrontal and temporal cortex. For example, Raine, Buchsbaum, Stanley, Lottenberg, Abel, & Stoddard (1994) found reduced glucose metabolism in the anterior medial prefrontal, orbitofrontal and superior frontal cortex in murderers compared to normal controls during a continuous performance task. Similarly, Hirono, Mega, Dinov, Mishkin, and Cummings (2000) also found individuals convicted of impulsive violent offenses to show reduced regional cerebral blood flow activity in the left anterior temporal, bilateral dorsofrontal, and right parietal cortex compared to non-violent dementia patients. Another study by Raine, Park, Lencz et al. (2001) employed a working memory task and revealed reduced activation in the right temporal cortex in violent offenders with a history of abuse compared with controls. Recently, Lee, Chan, & Raine (2009) showed that domestic violence offenders had increased brain activity to threat stimuli in the hippocampus, orbitofrontal cortex, and other regions compared with controls. These findings have provided a foundation for understanding how brain predispositions may contribute to criminal behavior, however many of these studies have focused solely on neurobiological factors for criminal behavior while providing very little if any discussion on the importance of other contributing factors, specifically social risk factors, in their explanations of

criminal outcome. Such lacking in examining both biological and social etiological variables in the brain imaging field is surprising as it has been made clear that nature (genes) and nurture (environment) are inextricably intertwined in the development of the brain. Furthermore, the impact of environmental effects on the brain is not restricted to prenatal development, but continues throughout the lifetime. Thus, one would only get a partial story by investigating biological factors outside the context of the environmental influences. Here, we will review some rare instances of empirical studies with a particular focus on incorporating brain imaging and social factors that advance our understanding of how environment interacts with brain development in predisposing to criminal behavior.

The first direct examination of potential biosocial interactions in predicting crime was conducted in 1998 by Raine, Stoddard, Bihle, and Buchsbaum, using positron emission tomography to address the issue of how social deficits moderate the relationship between brain function and criminal behavior. In brief, they divided a sample of murderers into those with and those without psychosocial deprivation. Specifically, ratings of psychosocial deprivation in this study took into account several social risk variables including early physical and sexual abuse, neglect, extreme poverty, foster home placement, having a criminal parent, severe family conflict, and a broken home. Compared to normal controls, murderers with psychosocial deprivation showed relatively good prefrontal functioning, whereas non-deprived murderers showed significantly reduced prefrontal functioning. Specifically, a 14.2 percent reduction in functioning in the right orbitofrontal cortex was found in murderers from good homes. These results suggest that the association between biological impairment and antisocial behavior are more prominent in those lacking social risk factors for antisocial behavior. Using functional magnetic resonance imaging, Raine, Park, Lencz et al. (2001) found a similar effect of biosocial interactions by comparing violent individuals with and without a child abuse history. More specifically, they found that violent offenders who had suffered severe child abuse show reduced right hemispheric functioning, particularly in the right temporal cortex. Further analyses revealed that abused individuals who had refrained from serious violence showed relatively lower left, but higher right, activation of the temporal lobe. The results further suggest that a higher functioning right temporal region may act as a protective factor in preventing one with social risk factors to commit a criminal act.

The interaction effect found in brain imaging studies on criminals are consistent with evidence in patients with brain damage suggesting that social influences, in combination with the biological risk factor of brain impairments, can predict criminal, violent behavior. For example, Lewis, Pincus, Feldman, Jackson, and Bard (1986) have demonstrated that exposure to violence and abuse in the family is the strongest factor for violent behavior in individuals with neurological impairment. Alternatively, there is also evidence that social factors can act as protective factors to prevent individuals with brain damage from future involvement in crime. For example, Mataró et al. (2001) describe a patient in Spain who suffered a similar accident to Phineas Gage when his frontal lobe was impaled by the spike of an iron gate in 1937. However, unlike Gage who later developed antisocial behavior,

this patient showed no signs of hostility, outbursts, or irritability. Mataró and colleagues suggested that such a different outcome may be due to the fact that his childhood sweetheart stood by him and married him after the accident, and his family was highly protective and caring, and gave him a job in his father's factory where he could be supervised. This finding suggests that social factors such as a nurturing family environment may be able to lower the risk that an individual with brain impairments will become criminal.

Although findings are rare, these previous studies have provided initial evidence suggesting that structural or functional brain deficits, when combined with a social risk factor, may predispose one to be engaged in criminal behavior while a social protective factor may prevent a potential criminal outcome and vice versa. However, findings to date are still a long way from elucidating the complicated interactional processes of the brain and environment as they relate to outcomes of criminal behavior. One specific goal for future studies will be to determine more specifically which brain morphological/functional factors play an important role in this biosocial interactive process of leading to criminal behavior. Another goal will be to determine whether particular social risk factors interact with particular brain predispositions to produce criminal outcomes (e.g., environmental stress may be more likely to trigger criminal behavior if a biological predisposition of amygdala deficits is present that lower the person's ability to handle stressful events). Future developmental studies with more sophisticated assessments on the environment and the brain will provide invaluable information for understanding the etiological equation of criminal behavior.

Psychophysiology

Although an extensive body of research has been built up on the psychophysiological basis of antisocial and criminal behavior (see reviews by Lorber, 2004; Patrick, 2008), relatively fewer studies have been conducted to examine the possible interaction effects with psychosocial variables. Most psychophysiological research in the biosocial studies has focused on autonomic nervous system functioning at a baseline level or in response to external stimuli using measures such as skin conductance activity and heart rate. Skin conductance is usually measured from electrodes placed on the fingers or palm of the hand and is controlled exclusively by the sympathetic nervous system. Skin conductance activity captures small fluctuations in the electrical activity of the skin, with enhanced conductivity (i.e., activity) elicited by increased sweating. Heart rate measures the number of heart beats per minute and reflects the complex interactions between sympathetic and parasympathetic nervous system activity.

With the advantages of ease of data collection (especially heart rate) and their noninvasive features, psychophysiological measures have been valuable in filling the gap between genetic risk for crime and the brain abnormalities which interact with environmental factors in predisposing some individuals to antisocial, violent,

and psychopathic behavior. In this section, empirical examples of interaction effects within the area of psychophysiology and relevant theoretical perspectives will be reviewed, followed by clinical implications of these findings.

First, psychophysiological risk factors have been found to interact with psychosocial variables in predisposing certain individuals to antisocial and criminal behavior. In this line of research, psychophysiological variables are dependent variables and the moderating effects of psychosocial factors are investigated. A number of studies have found that psychophysiological factors, particularly measures of skin conductance and heart rate, show stronger relationships to antisocial behavior in those from *benign* social backgrounds that lack the classic psychosocial risk factors for crime (Raine, 2002a). For example, studies have shown that poor skin conductance conditioning (see below) is a characteristic for antisocial individuals from relatively good but not bad social backgrounds (Hemming, 1981; Raine & Venables, 1981). In a longitudinal study, low heart rate at age three years has been found to predict aggression at age 11 years in children from high but not low social classes (Raine, Venables, & Mednick, 1997). These findings, as argued by the “social push” hypothesis, suggest that psychophysiological risk factors may assume greater importance when social predispositions to crime are minimized. In contrast, social causes may be more important explanations of antisocial behavior in those exposed to adverse early home conditions (Raine, 2002a).

Reduced conditioning has been a key concept of antisocial and psychopathic behavior and considered an index of emotional deficits and a proxy for amygdala dysfunction (Blair, 2007; Patrick, 2008). Among all the psychophysiological indexes, conditionability as measured by skin conductance responses in a laboratory-based classical conditioning paradigm has been studied most extensively in biological or biosocial research. For example, in a sample of 101 15-year-old male school children in England, Raine and Venables (1981) found that when subjects were from the high social classes, antisocial individuals had poor conditionability, whereas antisocial individuals from low social classes exhibited good conditionability. In a pilot study by Mednick et al. (1977) on psychophysiological functioning of antisocial individuals who were raised in criminogenic versus noncriminogenic environments, preliminary analyses suggested that criminal sons with noncriminal fathers were poor conditioners, whereas criminal sons with criminal fathers were good conditioners. These results are consistent with Eysenck’s concept of “antisocialization” (Eysenck, 1977) in which Eysenck argued that individuals from criminogenic backgrounds who have good conditionability might be socialized into antisocial habits that are not punished by their parents; in contrast, individuals with poor conditionability who are raised in criminogenic environments may not develop the antisocial tactics as well as their highly conditionable counterparts.

Alternatively, some studies have documented the “dual-hazard” effect in the psychophysiological research of antisocial behavior. In these studies, the biosocial interactions are examined with antisocial and criminal behavior as dependent variables. For example, boys with a low resting heart rate have been found more likely to be rated as aggressive by their teachers if their mother was pregnant as a teenager, if they were from a low social class family, or if they were separated from

a parent before age 10. They are also more likely to become adult violent criminals if they also have a poor relationship with their parents and come from a large family (Farrington, 1997). In a recent study on a community sample of 7–13-year-olds, community violence victimization was found to be positively related to proactive aggression only in children with low heart rate, and witnessed community violence was positively related to reactive aggression only in conditions of high heart rate variability (Scarpa, Tanaka, & Haden, 2008). These findings are in line with the biosocial theories of crime which predict that negative social environments in combination with deficits in biological functioning predispose to criminal outcome (Mednick, 1977).

Other studies have focused on psychophysiological correlates as protective factors against the development of antisocial and criminal behavior. For example, in a prospective longitudinal study, 15-year-old antisocial adolescents who did not become criminals by age 29 showed higher resting heart rate levels, higher skin conductance arousal, and better skin conductance conditioning when compared to their antisocial counterparts who became adult criminals (Raine, Venables, & Williams, 1995, 1996). In another study on adolescents who had criminal fathers and thus were at higher risk for antisocial outcomes, those who desisted from crime had higher skin conductance and heart rate orienting reactivity in comparison with those who eventually became criminals (Brennan et al., 1997). Therefore, enhanced autonomic nervous system functioning, as indexed by higher levels of arousal, better conditioning, and higher orienting responses, may serve as biological protective factors that reduce the likelihood that an individual will become an adult criminal.

Some researchers have attempted to integrate biosocial findings into prevention and intervention programs. One line of research concerns directly altering one's psychophysiological functioning by manipulating environmental factors. For example, in one longitudinal study, better nutrition, more physical exercise, and cognitive stimulation from ages three to five years was shown to produce long-term psychophysiological changes six years later at age 11 years, including increased skin conductance level, more orienting, and a more aroused EEG profile (Raine, Venables, Dalais et al., 2001). This environmental enrichment was also found to reduce criminal offending at age 23 years (Raine, Mellinger, Liu, Venables, & Mednick, 2003).

In summary, psychophysiological risk factors interact with psychosocial variables in predisposing certain individuals to antisocial and criminal behavior. Meanwhile, evidence of psychophysiological protective factors against the development of antisocial behavior has emerged. Finally, prevention and intervention programs aimed at reducing antisocial behavior might benefit enormously by directly improving their psychophysiological functioning through environmental manipulations. Certain psychophysiological measures, including heart rate activity, can be recorded relatively easily (e.g., using portable equipment or taking a pulse), and as such they are especially valuable to the criminologists who are attempting to explore the biosocial etiology of crime.

Neuropsychology

Neuropsychological impairments have long been known to be associated with criminal and antisocial behavior. Many areas of functioning have been found to be impaired in criminal populations, and research has focused on overall intellectual ability (especially verbal abilities) as well as executive functioning.

Intelligence

Evidence for decreased intellectual functioning (as measured by IQ) in association with antisocial behavior has been found by many researchers. In fact, at a very broad level, estimates of the average U.S. state IQ have been found to be significantly and negatively correlated with FBI crime statistics in each state (Bartels, Ryan, Urban, & Glass, 2010). Furthermore, antisocial behavior and low IQ have been found to have a common genetic basis (Koenen, Caspi, Moffitt, Rijdsdijk, & Taylor, 2006). Most research on intellectual functioning in this population, however, has highlighted a discrepancy between Verbal IQ and Performance IQ. Measures of Verbal IQ include tests which require participants to answer orally and often cover fact-based or crystallized knowledge, such as defining words or solving math problems aloud. Performance IQ tasks, in contrast, involve measures of spatial and non-verbal reasoning, such as manipulating blocks or searching for symbols on a page. Studies have found decreased Verbal IQ compared to Performance IQ in children (e.g., Loney, Frick, Ellis, & McCoy, 1998; McHale, Obrzut, and Sabers 2003), adolescents (e.g., Archwamety & Katsiyannis, 2000; Dougherty et al., 2007), and adults with antisocial behavior (e.g., Kirkpatrick et al. 2007; Vitacco, Neumann, & Wodushek, 2008). However, some studies have found no relative deficit in Verbal IQ in antisocial populations (e.g., Cadesky, Mota, & Schachar, 2000; Rispens et al., 1997), and another has found deficits in spatial, but not verbal, abilities (Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). A recent meta-analysis of this Performance > Verbal discrepancy found that, while the results were statistically significant for all age groups, the effect size of reduced Verbal IQ was negligible in children and small in adults (Isen, 2010). In contrast, the effect size for the discrepancy in antisocial adolescents was closer to medium in size, and reflected a six-point difference on average between Verbal and Performance IQ in delinquent adolescents (Isen, 2010). Therefore, the deficit in Verbal compared to Performance IQ appears to characterize antisocial populations, especially during adolescence.

In addition to these age-based differences in the discrepancy between Verbal and Performance IQ measures, there appear to be some differences based on race or type of antisocial behavior. For example, Isen (2010) reported that effect sizes for Black participants were smaller than for White or mixed race samples. Additionally, in one sample, the highest rates of violent delinquency were found in those adolescents who were both high in callous-unemotional (psychopathic-like) traits and high in verbal abilities (Muñoz, Frick, Kimonis, & Aucoin, 2008). Similarly, in a group of boys with conduct disorder, those who had a biological

parent with antisocial personality disorder and who had Verbal IQ scores above 100 had the highest number of conduct disorder symptoms three years later (Lahey et al., 1995). Thus, it is likely that the Performance > Verbal IQ pattern is not found in all antisocial groups.

Several hypotheses have been advanced to explain the discrepancy between verbal and non-verbal measures of ability in antisocial populations. One prominent theory is that pre-existing deficits in verbal ability lead to underachievement in school, which is, in turn, a risk factor for antisocial behavior (Moffitt, Lynam, & Silva, 1994). Others argue that antisocial behavior in childhood and adolescence often includes school refusal or gaps in education due to suspensions or incarceration, and these gaps in education are what account for deficits in Verbal IQ scores (Isen, 2010). That is, Verbal IQ tests often tap knowledge learned in school (e.g., word definitions), whereas Performance IQ tests are less reliant on education. Yet another hypothesis is that verbal deficits may lead to socialization failure (Eriksson, 2005) by affecting the development of self-control (Luria, 1980). Thus, it is unclear what underlies the relationship between Verbal IQ deficits and antisocial behavior, but researchers are continuing to probe this question.

Executive Function

In comprehensive neuropsychological batteries, executive functioning (EF) is also often found to be impaired in antisocial populations. EF is conceptualized as those cognitive processes which maintain representations of and allow the achievement of goals, and these processes are believed to be controlled largely by the prefrontal cortex (Miller & Cohen, 2001). Tasks commonly used to measure EF usually require mental flexibility, strategy formation, selective attention, and suppression of habitual responses. EF tasks are often used to identify patients with frontal lobe damage.

Thus far, research has supported the hypothesis that antisocial populations have impaired scores on tests of EF. A meta-analysis of this literature found that the effect size for EF deficits in antisocial populations was in the medium to large range, and this relationship held across all ages and all EF tests included (Morgan & Lilienfeld, 2000). More recent studies have supported these results, in adult male (Kavanagh, Rowe, Hersch, Barnett, & Reznik, 2010; Dolan & Park, 2002) as well as female offenders (Giancola, Shoal, & Mezzich, 2001), and in relation to violent (Hancock, Tapscott, & Hoaken, 2010), but not nonviolent (Barker et al. 2007; Hancock et al., 2010; Levi, Nussbaum, & Rich, 2010), offending. Although not all the evidence for EF impairment in youths with conduct problems is consistent (e.g., Nigg et al., 2004), the same deficit has generally been found in recent studies of antisocial children and adolescents (Beaver, DeLisi, Vaughn, & Wright, 2010; Hughes, Zagar, Busch, Grove, & Arbit, 2009; Raine et al., 2005; Syngelaki, Moore, Savage, Fairchild, & van Goozen, 2009).

Biosocial Interactions

A number of studies have found interactions between neuropsychological deficits and social risk factors in the prediction of antisocial behavior, especially in children and adolescents. An early study by Lewis, Lovely, Yeager, and Femina (1989) found that adult offending was better predicted using neuropsychological measures and abuse history than by juvenile offending alone. High IQ has been found to be protective in the context of environmental adversity, such that children and adolescents with high intellectual functioning do not show the typical increase in antisociality in risky environments (Masten et al., 1999; Vanderbilt-Adriance & Shaw, 2008). One feature of adverse environments that has been found to negatively impact children's intellectual functioning is neighborhood violence (Sharkey, 2010). Neighborhood violence may indeed be a significant factor in predicting antisocial behavior, as a strong relationship between exposure to violent media and poor EF has been found in children with disruptive behavior disorders (Kronenberger et al., 2005).

Parent traits have also been found to interact with neuropsychological functioning. Grekin, Brennan, and Hammen (2005) found that the relationship between parental alcohol use disorders (AUD) and offspring violent delinquency was mediated by the child's executive functioning, such that parental AUD leads to poor EF in the child, thereby increasing violence in the child. Parent diagnosis of antisocial personality disorder has also been found to interact with verbal IQ in the child. Lahey et al. (1995) found that (relatively) high IQ was only protective for conduct disorder in those children who did not have a biological parent with antisocial personality disorder. One longitudinal study of women followed from childhood into adulthood and motherhood found that the women who were aggressive as children provided poorer cognitive stimulation of their own children, who in turn had poorer verbal and abstract/visual abilities (Saltaris et al., 2004). Given the link between low IQ and crime, this study suggests one mechanism by which parents may pass on antisocial behavior may involve the poor cognitive environment provided at home.

Conclusions

In sum, there is a large body of evidence supporting the interacting roles of biological and social factors in criminal behavior. Pre- and peri-natal factors, such as prenatal exposure to nicotine and alcohol and birth complications, have been found to predict crime, particularly in the context of familial adversity and other psychosocial risk factors. Genetics research has reported heritability estimates of about 40–60 percent for crime, and specific genotypes, such as that conferring high levels of MAO-A, may be protective in adverse environments, at least for some populations. High levels of testosterone and low levels of cortisol may predispose to crime, and these hormones appear to interact with each other and with social risk factors

to predict antisocial behavior. Brain imaging research has found an association between decreased prefrontal cortex function and violence, and this pattern has been reported to interact with psychosocial adversity, such that murderers from good homes are more likely to show this brain deficit. Psychophysiological studies have found a similar relationship, in which the relationship between factors such as low heart rate and aggression is found only in those from benign backgrounds. Additionally, poor neuropsychological functioning is a risk factor for antisocial behavior, but good neuropsychological functioning is protective in the context of adversity. Although more work remains in clarifying these findings, especially with respect to how they apply to different types of offending (e.g., violent versus nonviolent, premeditated versus impulsive), the discovery of biosocial interactions using such a wide variety of measures lends support to the biosocial perspective on crime.

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