part one

Aggression (Prenatal and Childhood)

chapter one

Biosocial Bases of Antisocial Behavior

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INTRODUCTION

Antisocial behavior has long been a topic of interest among researchers in the field of neuroscience, psychology, criminology, and sociology, whom for decades have attempted to uncover the biological and social bases of this complex behavioral problem. Independently, several biological and social risk factors have been identified to predispose one to antisocial behavior. Biologically, factors such as autonomic underarousal, obstetrical factors, brain deficits, and neuropsychological impairments have been strongly linked to antisocial behavior. Numerous social and environmental factors have also been associated with antisocial behavior, including low social class, peer influence, physical abuse, and parental rejection. Despite the establishment of these risk factors, the level of knowledge has been relatively limited to independent effects of either social or biological factors, with very little understanding of the interactions between these two factors. Indeed, across the literature it is noticeable that psychosocial researchers rarely use methods to

measure the biological variables in their antisocial samples, whereas biological researchers often use social factors as covariate variables instead of moderators. However, due to the multidimensional nature of antisocial behavior, a multidisciplinary approach may not only help but is critically needed to further the understanding of the underlying mechanisms of antisocial behavior.

Nonetheless, many challenges make the approach to address both the independent and interaction effects of biological and social factors extremely difficult. One of the main challenges is that biological risk factors tend to correlate significantly with social factors, resulting in the lack of statistical power to detect the interaction effects. This often leads to the false-negative conclusions in a study. However, several recent innovative studies that are explored in this chapter include large twin and adoption samples, which managed to significantly increase their statistical power. Many have successfully detected the interaction effects of social and biological factors on the outcome of antisocial behavior. Findings from these studies are mainly in line with previous hypotheses or models of the biosocial bases of antisocial behavior and suggest that **biosocial interactions** may indeed contribute to the development of antisocial behavior.

In this chapter the empirical findings on the known biological risk factors associated with antisocial behavior are first reviewed, with a focus placed in the research areas of psychophysiology, obstetrical factors, **brain imaging**, neuropsychology, neurology, hormones, neurotransmitters, and environmental toxins. Second, further discussions are conducted to review the evidence of biosocial interaction effects in relation to antisocial behavior in each of the key research areas. Finally, prior biosocial models of antisocial behavior are revisited and a model, extended from Raine's biosocial model of violence, is hypothesized for antisocial behavior.[1] It is worth mentioning that the term "**antisocial behavior**" is used throughout this chapter as an umbrella term for various behavioral problems, including violent, psychopathic, delinquent, and criminal behavior. We acknowledge there may be different underlying biosocial pathology among those behaviors; however, the approach of combining empirical data from studies on various antisocial-related behavior was used in the hope of providing a comprehensive review on the biosocial bases of antisocial behavior.

EMPIRICAL FINDINGS ON ANTISOCIAL BEHAVIOR

This section reviews major research findings on the physiological correlates of antisocial behavior.

Psychophysiological Impairments

Psychophysiological research has provided some of the most convincing evidence for a biological predisposition for antisocial behavior, including the repeatedly observed findings of reduced skin conductance and heart rate activity/reactivity, excessive slow-wave electroencephalogram (EEG), atypical EEG frontal asymmetry, and event-related potential (ERP) responses in antisocial children and adults.

Reduced Skin Conductance and Heart Rate Activity/Reactivity

Several studies and reviews provided solid evidence for lower skin conductance levels in antisocial individuals. For example, Kruesi and colleagues showed that low skin conductance levels measured at age 11 predicted institutionalization at age 13.[2] Deficits in reduced skin conductance responses to both neutral and emotional stimuli in antisocial individuals have also been reported. For example, Raine and Venables found antisocial adolescents with schizotypal features to show significantly lower skin conductance responsivity relative to those with their schizotypal-only counterparts.[3] Empirical studies also showed that antisocial populations exhibit poor skin conductance conditioning and that the association between poor skin conductance conditioning and antisocial behavior may be in place early on in childhood.[4]

One of the most replicable findings of psychophysiological impairments in antisocial individuals is that of low resting heart rate.[5] For children and adolescents, several studies reported that low resting heart rate is prospectively linked to antisocial behavior later in adulthood. For example, in a study involving more than 1,800 children in the United Kingdom, Wadsworth reported that low resting heart rate at age 11 predicted delinquency at age 21. Furthermore, it was suggested that this relationship of lower heart rate and increased antisocial behavior is diagnostically specific: No other psychiatric condition (e.g., alcoholism, depression, schizophrenia, anxiety disorder) has been linked to low resting heart rate. In addition, some studies reported reduced heart rate reactivity to negative stimuli in antisocial individuals; however, the findings are somewhat mixed.[6]

Summarizing the main findings, it is evident that reduced skin conductance and heart rate activity/reactivity suggest a pathological pattern of underarousal and reduced responsivity in antisocial individuals. Underarousal in antisocial individuals indicated by lower skin conductance levels and lower resting heart rate may promote a need for thrill and sensation-seeking in these individuals, whereas attenuated skin conductance responsivity and heart rate reactivity to aversive stimuli may suggest that antisocial individuals are less sensitive to the negative consequences of their behavior and thus mitigate the moral development and the obedience of social rule. Specifically, both underarousal and reduced responsivity reflect low levels of anxiety or fear, which may predispose to antisocial and violent behavior because such behavior in part requires a degree of fearlessness to execute. Lack of fear, especially in childhood, may explain poor socialization in antisocial individuals because a low fear of punishment would reduce the effectiveness of parental socialization processes. Alternatively, individuals with lower activity/reactivity levels may attempt to maintain an optimal level of arousal by seeking out thrill and excitement, which in turn may lead to the development of antisocial or criminal behavior.

Excessive Slow-Wave EEG, Atypical EEG Frontal Asymmetry, and ERP Responses

Several extensive reviews were conducted that suggested fairly consistent findings of higher rates of EEG abnormalities in antisocial individuals relative to control subjects.[7] In general, these reviews indicate that higher levels of slow-wave activity (particularly delta wave, frequency < 4 Hz) have been repeatedly found in aggressive, antisocial individuals. For example,

one quantitative EEG analysis conducted by Convit, Czobor, and Volavka[8] found significant correlations between EEG delta activity and the number of violent incidents among psychiatrically hospitalized patients. In addition, a few prospective longitudinal studies demonstrated that excessive slow-wave EEG (i.e., delta wave) precedes the onset of significant criminal behavior in both high-risk (i.e., parents with schizophrenia or personality disorders) and community samples. For example, Raine, Venables, and Williams reported that slower frequency EEG activity at age 15 predicted antisocial behavior at age 24. It is generally considered that the enhanced, slow-wave EEG activity is an indication of cortical immaturity among those with antisocial behavior.[9]

Another line of research has revealed that atypical frontal EEG asymmetry (right > left) is associated with antisocial/externalizing behavior problems in children and adult populations.[10] In general, relatively greater left frontal activity (i.e., relatively reduced left alpha wave activity) is associated with positive affect and approach behavior, whereas relatively greater right frontal activity (i.e., relatively reduced right alpha wave activity) is related to negative affect and withdrawal behavior.[11] It is therefore considered that atypical EEG frontal asymmetry is an indication of aberrant emotional regulation among those with antisocial behavior.

Regarding the ERP, one postulated neurobiological marker for antisocial behavior is reduced amplitude of the P300 component of the ERP in "oddball" tasks.[12] The P300 has been viewed as an orienting response ("what is it?"), and the reduction of its amplitude has been considered to indicate inefficient deployment of neural resources to process cognitive task-relevant information.[13] A recent **meta-analysis** confirmed the presence of reduced P300 amplitudes in antisocial individuals.[14] Because P300 is generated maximally over the parietal lobe (a region important for working memory),[15] it is suggested that a reduction in P300 amplitude may imply a higher level cognitive impairment in antisocial, violent individuals, such as poor decision making and behavioral disinhibition.

Biosocial Interactions

Increasing evidence shows that social factors may interact with psychophysiological predispositions in the development of antisocial and violent behavior. For example, Farrington reported that boys with low resting heart rate are more likely to become adult violent criminals if they also have a poor relationship with their parents and come from a large family. Similarly, boys with low resting heart rates are 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.[16] Alternatively, a number of studies found that biological impairments, including 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. For example, Hemming observed poor skin conductance conditioning among criminals from relatively good social backgrounds.[17] Similarly, Raine and Venables found poor skin conductance conditioning specifically in antisocial children from higher social class but not in those from lower social class.[18] In a prospective longitudinal study, Raine et al. found that low heart rate at

age 3 years predicted aggression at age 11 years in children from high but not low social classes. These findings, as argued by the "social push" hypothesis, suggest that psychophysiological impairments may assume greater importance when social predispositions to crime are minimized.[19] In contrast, social causes may be more important explanations of antisocial behavior in those exposed to adverse early home conditions.

Obstetrical Factors

Of all the areas of biological research on antisocial behavior, studies on obstetrical factors have provided the most compelling evidence for biosocial interaction effects on antisocial behavior. Several prenatal and perinatal factors, including minor physical anomalies, prenatal nicotine or alcohol exposure, and birth complications, have been most closely linked to antisocial behavior and thus are the focus in this section.

Minor Physical Anomalies

Minor physical anomalies (e.g., low-seated ears, adherent ear lobes) have been associated with pregnancy disorders and are considered to be indicators of fetal neural maldevelopment near the end of the first or the beginning of the second trimester of pregnancy.[20] Because the integument and the central nervous system have shared embryological origins, minor physical anomalies are seen as indirect markers of atypical central nervous system and brain development. Several studies found a relationship between more minor physical anomalies and increased antisocial behavior in children, adolescents, and adults. In particular, minor physical anomalies have been linked to violent as opposed to nonviolent offending. For instance, Arseneault and her colleagues showed that minor physical anomalies 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.[21] In another study, Kandel, Brennan, Mednick, and Michelson assessed minor physical anomalies in 265 12-year-old Danish children and found that recidivistic violent offenders had a greater number of minor physical anomalies compared with subjects with one or no violent offenses at an average age of 21 years.[22] These studies suggest that prenatal insults toward the end of the first 3 months of pregnancy may increase risk for violent behavior as a result of abnormal brain development.

Prenatal Nicotine and Alcohol Exposure

Extensive evidence on prenatal nicotine exposure has established beyond a reasonable doubt that children who are exposed to maternal smoking during pregnancy are at increased risk for later antisocial behavior that extends over the life course.[23] Specifically, prenatal exposure to nicotine has been linked to childhood externalizing behavior, conduct disorder, delinquency, and adult criminal and violent offending.[24] Several studies also reported a dose–response relationship between the extent of maternal smoking during pregnancy and the severity of later antisocial behavior in offspring.[25]

In addition to nicotine exposure, it has also been established that fetal alcohol exposure significantly increases risk for antisocial behavior in children, adolescents,

and adults.[26] Heavy alcohol consumption during pregnancy can result in fetal alcohol syndrome, which is characterized by a host of cognitive, behavioral, social, and physical deficits. However, deficits are observed even in those who have been prenatally exposed to alcohol but do not meet diagnostic criteria for fetal alcohol syndrome.[27] For example, research found high rates of delinquency in children and adolescents with heavy fetal alcohol exposure, even if they do not have fetal alcohol syndrome.[28] In addition, studies showed that adolescents who were prenatally exposed to alcohol are overrepresented in the juvenile justice system. For example, Fast and colleagues[26] found that 3 percent of adolescents in a juvenile inpatient forensic psychiatry unit were diagnosed with fetal alcohol syndrome, and 22 percent were diagnosed with fetal alcohol effects. Another study reported that 61 percent of adolescents, 58 percent of adults, and 14 percent of children between the ages of 6 and 11 years with fetal alcohol exposure had a history of trouble with the law.

Birth Complications

Complications at birth (e.g., pre-eclampsia, deprivation of oxygen) have been demonstrated in several studies to predict future engagement of antisocial behavior. The first study was conducted by Pasamanick and colleagues in which a significant link between birth complications and behavior disorders in children was established.[29] The findings were replicated by several studies.[30] However, other studies also questioned the independent effects of birth complications on the development of antisocial behavior by showing that only when combined with social risk factors (e.g., maternal rejection, disadvantageous family background) did birth complications associate with violent behavior.[31]

Biosocial Interactions

Multiple studies showed a significant interaction effect between obstetrical and social risk factors in predisposing one to antisocial behavior. Regarding minor physical anomalies, for example, one study by Mednick and Kandel measured minor physical anomalies in 129 boys during visits to a pediatrician at age 12. The authors found that minor physical anomalies were associated with violent behavior when the subjects were 21 years old. Interestingly, however, when the authors divided subjects into those from unstable, nonintact families and those from stable families, they found that minor physical anomalies only predicted later criminal involvement for those reared in unstable, nonintact homes.[32] A similar finding was reported by Brennan, Mednick, and Raine, who evaluated adult violent offenses in a sample of 72 male offspring of parents with psychiatric diagnoses. The authors found particularly high rates of violent offending in individuals who had both family adversity and minor physical anomalies compared with those who had only one of these risk factors.[33] In another study, Pine et al. investigated the interaction of minor physical anomalies and social risk factors, such as low socioeconomic status, spousal conflict, and marital disruption, in predicting later disruptive behavior disorders. The authors found individuals who had both increased minor physical anomalies and social risk at age 7 to show greater antisocial behavior at age 17.[34]

In terms of prenatal nicotine/alcohol exposure, several studies also documented interactions between maternal prenatal smoking and social risks in the prediction of later violence. These studies are notable for their large sample sizes, assessment of long-term outcomes, prospective data collection, and control for potential confounds such as drug use and socioeconomic status difference. One striking study conducted by Rasanan et al. found that the offspring of women who smoked during pregnancy had a twofold increase in violent crime at age 26, and when combined with being raised in a single-parent family, recidivistic violent offending increased 11.9 times. Moreover, prenatal nicotine exposure led to a 14.2 times increase in recidivistic violence when combined with a number of other social risk factors (i.e., teenage pregnancy, single-parent family, unwanted pregnancy, and developmental motor delays).[35] It is worth mentioning that in this study the biosocial effect was more prominent for persistent violent offending rather than violence in general or property crime.

A number of well-designed studies also demonstrated that birth complications interact with social risk factors in predicting antisocial behavior in adulthood. For example, Werner found that birth complications combined with a disruptive family environment (i.e., maternal separation, illegitimacy, marital discord, parental mental health problems, paternal absence) predisposed to delinquency over and above either biological or psychosocial risk factor independently.[36] Two prospective longitudinal studies by Raine, Brennan, and Mednick also provide evidence for the presence of biosocial interactions in antisocial behavior.[37] In brief, Raine et al. evaluated whether the early experience of extreme maternal rejection (e.g., unwanted pregnancy, attempts to abort the fetus, institutional care of the infant during the first year of life) interacted with birth complications predisposed antisocial behavior in a sample of 4,269 males born in Copenhagen, Denmark between 1959 and 1961. The authors found that birth complications significantly interacted with maternal rejection in predisposing one to antisocial behavior at age 18 years. The importance of this finding is highlighted by the fact that whereas 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 reassessing this sample at age 34, the authors replicated the biosocial interaction and found the effect to be specifically strong for serious and early-onset antisocial behavior. Similar biosocial interactions between birth complications and various social risks have been reported in studies using large samples from around the world.[38]

Overall, findings from these studies provide robust evidence suggesting that obstetrical factors, particularly minor physical anomalies, prenatal nicotine/alcohol exposure, and birth complications, may significantly increase the likelihood of antisocial behavior when combined with social risk factors. Also, the presence of both biological and social risk factors may be needed to predispose one to severe antisocial behavior.

Brain Deficits

A significant body of evidence has accumulated suggesting that brain deficits, regional structural/functional abnormalities or brain damage, may act as precursors to the development of antisocial behavior.

Structural and Functional Abnormalities

Brain imaging has become increasingly popular among researchers in recent years. As a relatively objective method for assessing the structural and function of the brain, several studies used brain imaging to reveal the neuropathological impairments in antisocial children and adults. Most of these studies used positron emission tomography (PET; measures glucose metabolism), single photon emission computed tomography (SPECT; assesses blood flow), and functional magnetic resonance imaging (fMRI; measures blood oxygen level changes) to evaluate brain function, and anatomical magnetic resonance imaging (aMRI) to assess global and regional brain structural alterations in individuals with antisocial behavior.

Using PET, several studies found metabolic abnormalities during resting states in antisocial individuals. For example, Volkow et al. observed significantly reduced glucose metabolism in both prefrontal and medial temporal regions in violent adult patients with antisocial personality disorder (APD) compared with normal control subjects.[39] Similar results have been reported for children with antisocial behavior. For example, in a PET study on aggressive children with epilepsy, Juhasz et al. found a significant correlation between higher severity of aggression and lower metabolism in the bilateral medial prefrontal and left temporal cortex in this sample of antisocial children. On the other hand, instead of using a resting state, some studies examined the metabolic response in antisocial individuals during a challenge task (e.g., a continuous performance task).[40] For example, using an auditory continuous performance task, Goyer et al. showed that the number of impulsive-aggressive acts in patients with APD and other personality disorders was negatively associated with glucose metabolism in the orbitofrontal, left anterior frontal, and anterior medial frontal cortices.[41] Reduced glucose metabolism has also been found in the anterior medial prefrontal, orbitofrontal, and superior frontal cortex in murderers compared with normal control subjects during a continuous performance task. Similar indications of abnormal functioning in the frontal and temporal regions have been reported in studies using single photon emission computed tomography. For example, Amen et al. found decreased regional cerebral blood flow activity in the prefrontal cortex, and increased activity in the anterior medial frontal and left temporal cortex in aggressive psychiatric patients.[42] Using 21 individuals convicted of impulsive violent offenses, Hirono et al. also found violent patients with dementia to show reduced regional cerebral blood flow in the left anterior temporal, bilateral dorsofrontal, and right parietal cortex compared with nonviolent dementia patients.[43]

The use of fMRI has furthered knowledge by revealing both cognitive and emotional impairments in antisocial children and adults. For example, during the viewing of negative affective pictures, decreased activation in the amygdala–hippocampal complex and increased activation in the frontotemporal region were observed in criminal psychopaths.[44] Sterzer et al. also found reduced activation in the amygdala and hippocampus in aggressive children with conduct disorders while viewing negative emotional pictures. In addition, abnormal conditioning response was found in antisocial individuals.[45] By using an aversive conditioning task, Schneider et al. found an increase in activation in the dorsolateral

prefrontal cortex and amygdala during the acquisition phase of aversive conditioning in individuals with APD.[46] To assess cognitive functioning in antisocial individuals, Raine et al. used a working memory task and revealed reduced activation in the right temporal cortex in violent offenders with a history of abuse compared with control subjects.[47] Similar findings have been reported by Kumari et al., showing activation deficits in the left frontal gyrus and anterior cingulate cortex in violent patients compared with normal control subjects during a working memory task.[48]

In terms of structural abnormalities, aMRI has become the most common method to be used in examining brain structure in recent years. Several studies to date have found volumetric abnormalities in the prefrontal and temporal regions in antisocial individuals. For example, Raine and colleagues found that individuals with APD show a significant gray matter volume reduction in the prefrontal cortex compared with control subjects.[49] Woermann et al. also found reduced left prefrontal gray volumes in aggressive epileptic patients compared with nonaggressive epileptic patients.[50] Similarly, Laakso et al. reported reduced gray matter volume in the dorsolateral prefrontal, medial frontal, and orbitofrontal cortex in alcoholics with APD compared with control subjects.[51] Another study conducted by Dolan et al. found a volume reduction in the temporal lobe in violent APD patients, but no such reduction was found in the prefrontal cortex.[52] More recently, Yang et al. found a volume reduction in prefrontal gray matter in psychopaths with prior convictions (i.e., unsuccessful psychopaths) compared with both those without convictions (i.e., successful psychopaths) and nonpsychopathic control subjects. These findings further suggest that the relatively intact prefrontal cortex may act as a protective factor in preventing successful psychopaths from getting convicted.[53] In addition to frontal and temporal regions, structural abnormalities have also been found for the hippocampus and corpus callosum in antisocial individuals.[54] However, more replications are needed to confirm the effects in these regions.

Overall, functional abnormalities and volumetric reductions in the frontal and temporal cortex have been repeatedly linked to antisocial behavior. Due to the crucial roles of both frontal and temporal regions in the process of decision making, emotional regulation, and moral judgment, deficits to these two areas may therefore predispose one to antisocial violent behavior.

Brain Lesions

The examination of patients with brain lesions has proven to be promising in establishing a causal link between brain damage and antisocial behavior. In fact, some of the most striking evidence for the role of certain brain regions in antisocial behavior comes from descriptions of patients with acquired brain damage who have subsequently developed antisocial and psychopathic-like behavior. A number of case studies point to the role of the frontal lobe in antisocial behavior. One of the earliest cases is that of Phineas Gage, a railway foreman who had a tamping iron blown through his frontal lobe in an accident involving explosives. He survived the injury and recovered his physical and intellectual abilities, but his personality changed dramatically and he became callous, irritable, obnoxious, and irresponsible.[55] Similar personality changes have been observed in various case studies of

frontal lobe damage.[56] Common features after prefrontal damage include lack of empathy, difficulties with emotion regulation, impulsivity, disinhibited behavior, poor planning, and blunted emotions. In essence, these individuals develop a psychopathic-like personality or what has been referred to as "acquired sociopathy."[57] Antisocial or psychopathic characteristics seem to develop particularly when damage occurs to the orbital or ventromedial regions of the frontal lobe. For example, Grafman et al. examined a large group of Vietnam War veterans and found that aggressive and violent attitudes were heightened in veterans who had suffered lesions to the ventromedial region of the frontal lobe when compared with control subjects and individuals with damage to other brain regions.[58]

Developmentally, it appears that when brain damage occurs very early in life, the effects on antisocial behavior can be even more pronounced. Anderson et al. found that patients who incurred brain damage before the age of 16 months developed irresponsible and criminal behavior, abusive behavior toward others, and a lack of empathy or remorse.[59] These antisocial characteristics and behaviors were more severe than those observed in patients who suffer ventromedial prefrontal damage in adulthood. It has been suggested that intact functioning of the ventromedial prefrontal cortex is important for moral development. When this region is damaged very early in life, the process of moral socialization may be disrupted. Brain damage during childhood has also been found to lead to clinical diagnoses of antisocial behavior. Pennington and Bennetto found that seven of nine patients who had incurred damage to the frontal lobes before the age of 10 developed conduct disorder.[60]

Together these studies demonstrate that brain impairments, particularly in the orbitofrontal/ventromedial region of the prefrontal cortex, may lead to antisocial behavior. However, not all patients with brain damage become antisocial, suggesting that other factors may influence whether an individual with brain impairment becomes antisocial.

Biosocial Interactions

Very few brain imaging studies have been conducted to date that evaluated interactions between social influences and brain deficits in antisocial individuals. The first was conducted in 1998 by Raine, Stoddard, Bihrle, and Buchsbaum using PET to address the issue of how social deficits moderate the relationship between brain function and antisocial behavior. In brief, the authors divided a sample of murderers into those with and those without psychosocial deprivation. In addition, ratings of psychosocial deprivation took into account early physical and sexual abuse, neglect, extreme poverty, foster home placement, having a criminal parent, severe family conflict, and a broken home. Compared with normal control subjects, murderers with psychosocial deprivation showed relatively good prefrontal functioning, whereas nondeprived 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.[61] By using fMRI, Raine and colleagues 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 develop antisocial behavior.[62]

The interaction effect found in brain imaging studies on antisocial individuals is consistent with evidence in patients with brain damage, suggesting that social factors, in combination with the biological risk factor of brain impairment, can influence whether patients develop antisocial behavior. For example, studies by Lewis et al. in young children, adolescents, and adults found that across the life span, exposure to violence and abuse in the family is the strongest factor that leads to violence 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 becoming antisocial.[63] Mataró et al. 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, he showed no signs of hostility, outbursts, or irritability. Such a different outcome may have been because 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.[64] 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 antisocial.

Overall, these findings suggest that structural or functional brain deficits when combined with a social risk factor can predispose one to antisocial behavior. They also suggest that biological or social factors may protect against the outcome of antisocial behavior.

Neuropsychological Impairments

Neuropsychological research has contributed significantly to our understanding of the pathological bases of antisocial behavior. Neuropsychological investigations of violence, crime, and aggression have generally focused on different domains of cognitive functioning in an attempt to understand these phenomena by identifying associated behavioral expressions of brain dysfunction. As a result, several neuropsychological impairments have been identified, including lower intelligence and impaired executive functioning in antisocial individuals, suggesting a dysfunction in the brain, particularly the prefrontal cortex.

Lower Intelligence

Literature available to date suggests that lower intelligence (i.e., IQ or Full Scale IQ) is one of the best replicated cognitive correlates of antisocial behavior among non–mentally ill individuals.[65] In addition, both Verbal and Spatial/Performance IQ have been examined in adults and children with antisocial behavior. Regarding antisocial children and adolescents, lowered Verbal IQ appears to be a crucial characteristic of this population. For example, in a study of New Zealand birth cohort children, Moffitt, Lynam, and Silva reported that verbal deficits at age 13 predicted delinquency with persistent, high-level offenses at age 18.[66]

The findings are consistent with prior arguments that verbal deficits may affect the development of self-control,[67] leading ultimately to socialization failure and antisocial behavior.[68] Findings for Spatial/Performance IQ have been inconclusive; however, a study by Raine et al. using a community sample of 325 adolescents linked both Verbal and Spatial IQ deficits to antisocial behavior. Furthermore, one prospective longitudinal study showed that low Spatial (but not Verbal) IQ at age 3 years predisposed to life-course persistent offending, whereas several other studies of childhood antisocial behavior have also observed spatial ability impairment.[69] These authors proposed that early visuospatial deficits may potentially interfere with mother–infant bonding and may reflect right hemisphere dysfunction that disrupts the processing and regulation of emotions, in turn contributing to life-course antisocial behavior. Although similar deficits in Spatial/Performance or Verbal IQ have not been reported in adult antisocial individuals, reduced Verbal as opposed to Performance IQ has been reported in adult antisocial populations.[70]

Executive Dysfunction

Executive functioning (EF) refers to the cognitive processes that allow for goal-oriented, contextually appropriate behavior and effective self-serving conduct.[71] Executive dysfunction is thought to represent frontal lobe impairment and is indicated by poor performance on neuropsychological measures of strategy formation, cognitive flexibility, or impulsivity. Neuropsychological investigations of EF deficits in antisocial behavioral research have typically focused on diagnostic categories (i.e., APD, conduct disorder, psychopathy) and legal/judicial concepts (i.e., criminality and delinquency). A prominent meta-analysis found overall EF deficits in antisocial individuals compared with control subjects, with the strongest effects found for the Porteus Mazes test and criminal behavior. More recently, EF deficits have been found in a variety of adult antisocial populations including male and female violent and nonviolent criminals and individuals with APD.[72] However, results for executive dysfunction in children and adolescent with antisocial behavior are less than conclusive. Earlier studies of EF in children reported mixed evidence for a link between delinquency and EF deficits, although this may be attributable to methodological weaknesses, inconsistent definitions of EF, or both.[73] More recent findings are also mixed, with EF deficits characterizing some antisocial youths and not others.[74]

Biosocial Interactions

Neuropsychological studies suggested a possible interaction of lower intelligence/executive dysfunction and adverse social influences with significantly increased levels of antisocial behavior later on in life. For example, Lewis et al. conducted a study on juvenile delinquents at age 15 and found that a combination of neuropsychological impairments and child abuse was associated with a significant increase in violent offenses in adulthood compared with those with either neuropsychological impairments alone or child abuse alone. These findings are supported by several recent longitudinal studies. For example, in a group of 435 children, Moffitt reported that those with both low neuropsychological performance and family adversity had aggressive scores four times higher than those with

either neuropsychological deficits or family adversity only. Using a high-risk sample of 370 Australian adolescents, Brennan et al. also found that although the independent presence of biological risk factors including neuropsychological impairments (e.g., low age 5 vocabulary ability, poor age 15 Verbal IQ and EF) or social risk factors (e.g., lack of paternal control or acceptance, poor educational background of the mother, poverty, harsh discipline style) predicted later antisocial behavior, an interaction of early social risks with later biological risks predicted *persistent* antisocial violent behavior.[75] Furthermore, a lifetime cumulative interaction of these risks is a stronger predictor of persistent antisocial behavior than when the risks were presented only in childhood or adolescence. Results from these studies suggest that a combined focus on neuropsychological impairments and social risk factors may be influential in the developmental patterns of antisocial behavior.

Abnormal Hormones, Neurotransmitters, and Toxins

Although very few studies have been conducted, several additional biological risk factors have been suspected to predispose one to antisocial behavior. These risk factors are abnormal levels of hormones, neurotransmitter dysfunctions, and the presence of high-level environmental toxins.

Hormonal Imbalances

Common hormones associated with antisocial behavior are cortisol and testosterone. There is considerable evidence suggesting that cortisol levels are reduced in antisocial children, adolescents, and adults. In children, low cortisol levels have been associated with aggression, externalizing behavior and low anxiety, and symptoms of conduct disorder. Low cortisol has been observed in adolescents with conduct disorder, callous and unemotional traits, and conduct problems.[76] In a 5-year longitudinal study, Shoal et al. found that low cortisol in preadolescent boys (ages 10–12 years) was associated with low harm avoidance, low self-control, and more aggressive behavior during adolescence (ages 15–17 years). Finally, low cortisol levels have been found in violent adults and psychopathic offenders.[77] Lower levels of cortisol may indicate that individuals are less responsive to stressors and may be less fearful of negative consequences such as potential punishment.

Testosterone has also been associated with aggressive behavior. Males have several times the amount of testosterone as females. Because there are large gender differences in antisocial behavior, with a male-to-female ratio of about 4:1 for APD and as large as 10:1 for violent crimes, it has been hypothesized that testosterone may be involved in aggressive behavior.[78] Elevated testosterone levels have been linked to antisocial behavior and violent crime in adults, yet studies of aggressive children and adolescents have yielded mixed results.[79] Nevertheless, in a meta-analysis by Brook, Starzyk, and Quinsey, a modest but robust association between testosterone levels and antisocial behavior was confirmed.[80] It has been argued that testosterone may not be linked to aggression per se but to social dominance, whether it be within healthy or antisocial groups, which may account for some of the mixed results.[81]

Neurotransmitter Dysfunction

One of the neurotransmitters found to be particularly linked to antisocial behavior is serotonin and the related molecules, including the enzyme that metabolizes serotonin (i.e., monoamine oxidase A [MAO-A]) and the serotonin metabolite 5-hydroxyindoleacetic acid. In a review of the literature, Berman and Coccaro concluded 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.[82] Lower concentrations of the serotonin metabolite 5-hydroxyindoleacetic acid in the cerebrospinal fluid have been found among antisocial populations.[83] In a meta-analysis by Moore, Scarpa, and Raine, the authors concluded that there is a significant association between 5-hydroxyindoleacetic acid levels and antisocial behavior.[84] Levels of MAO activity, although an indirect measure of metabolization of serotonin and dopamine, have been associated with violent, antisocial behavior. In addition to these studies, several studies also found that antidepressants, which increase serotonin, are linked to reductions in aggressive behavior.[85]

Environmental Toxins

The toxins discussed here are metals found in the environment and known to be harmful to humans when exposure is high. Research has demonstrated a possible link between metal toxicity and criminal behavior. For example, studies of prison inmates found that hair levels of manganese, lead, and cadmium were significantly higher in violent offenders than in nonviolent offenders or control subjects.[86] Environmental lead exposure has been associated with aggressive behavior in children, adolescents, and adults.[87] For example, Burns et al. found that school children from a lead smelting community had increased externalizing behavior problems, when controlling for other variables. It has been suggested that metal toxins may be related to antisocial behavior because they can affect neurotransmitter levels.[88]

Biosocial Interactions

Although the interaction between the biological factors of abnormal levels of hormones, neurotransmitters, and toxins and social risk factors has rarely been studied, results from a few studies suggest such interactions may indeed influence the outcome of antisocial behavior. For example, Dabbs and Morris found that high testosterone was associated with higher levels of childhood and adult delinquency in subjects with low socioeconomic status but not in those with high socioeconomic status. Another study conducted by Mazur found that biological factors of abnormal levels of hormones, including cortisol, testosterone, and thyroxin, in combination with social factors, including age, education, and income, were better at predicting delinquent behavior than either factor alone.[89]

In terms of neurotransmitters, Moffitt et al. found that violent offenders with high blood serotonin levels and a conflicted family background were over three times more likely to become violent by age 21 than men with only the biological or the social risk factor.[90] Caspi and his colleagues, using a large sample of 1,037 children, found maltreated

children with a genotype conferring low levels of MAO-A expression were more likely to exhibit antisocial behavior. High levels of MAO-A expression appeared to act as a biological protective factor in maltreated individuals, as the rates of antisocial behavior were lower.[91] A similar pattern was found in anther study of 514 twins conducted by Foley et al. showing that family adversity (e.g., parental neglect, exposure to interparental conflict, and inconsistent parental discipline) was more significantly associated with conduct disorders in those with low MAO-A activity than in those with high MAO-A activity.[92]

Overall, it appears that abnormal levels of hormones, neurotransmitters, and toxins, when combined with social risk factors, may increase the likelihood of developing antisocial behavior. However, future studies are needed to further elucidate moderator effects of social factors on the relationship between hormones, neurotransmitters, and toxins and antisocial behavior.

BIOSOCIAL MODEL OF ANTISOCIAL BEHAVIOR

Several researchers proposed a biosocial model for antisocial behavior. The first model proposed by Eysenck suggests that certain biological factors increase the risk for antisocial outcomes, particularly when a particular social upbringing is present.[93] This model suggests that antisocial behavior is intrinsically rewarding, and the family and social environment inhibit antisocial behavior by repeatedly pairing antisocial behavior with punishment (i.e., classical conditioning). This process should effectively reduce antisocial behavior if the child has a nervous system that responds normally to conditioning. This model, although intriguing and supported in some studies, has not been rigorously tested.

Later models of antisocial behavior focused instead on a "dual hazards" interaction of antisocial behavior, predicting that a negative social environment in combination with biological deficits increases the likelihood of predisposing one to antisocial behavior. For example, Mednick proposed that children with deficits in the autonomic nervous system (i.e., the biological factor) who are also raised in inadequate family environments (i.e., the social factor) are at the highest risk for developing poor avoidance conditioning and an inability to learn law-abiding behavior. It was argued that passive avoidance (i.e., avoiding committing an act that has previously been punished) occurs because of the child's fear of punishment, a necessary "civilization" process in normal development. The model suggests that a social environment, when presented with consistent and adequate punishment for antisocial acts (to induce the fear) or a well-functioning autonomic nervous system (to quickly dissipate the fear), will result in fast learning of passive avoidance and a successful inhibition of the nonpreferable (i.e., antisocial) behavior. If both biological and social components are absent, the child is more likely to display antisocial behavior.[94] This model receives some support from the empirical studies, particularly the biological component of poor conditioning in antisocial individuals. However, this model did not fully explain some results of biosocial interaction effects, such as why antisocial individuals from benign families show greater biological impairments.

A "social push" model proposed by Raine incorporated empirical findings and highlighted the key influences of genetic and environmental processes in giving rise to social and

biological risk factors that both individually and interactively predispose one to antisocial behavior. Striking evidence suggested a strong interaction effect between genetic and environmental factors in predisposing one to antisocial behavior. One of the earliest studies by Cloninger et al. showed that 40 percent of adoptees with both genetics (i.e., biological parents were criminals) and environmental risk factors (i.e., negative parenting in adoptive parents) were criminals compared with 12.1 percent of those with only genetic factors present, 6.7 percent of those with only a bad family environment, and 2.9 percent for those with neither risk factor.[95] Results show that genetic and environmental factors indeed interact and that the interaction results in a nonadditive increase in antisocial behavior in individuals. The interaction effect was consistent with findings of several other adoption studies confirming the strong influence of these two basic variables in the development of antisocial behavior.[96] This model also incorporated several social and biological protective factors, many of which were mentioned earlier in the review of the empirical findings. The involvement of additional protective biological (e.g., increased prefrontal volume, higher EF) or social factors (e.g., higher social class, supporting family) further explains why some individuals with biosocial precursors did not exhibit antisocial behavior.

In this chapter a biosocial model extending Raine's model is proposed, suggesting that three key factors, social/environmental, genetics/biological, and protective factors, independently and interactively influence the outcome of antisocial behavior (**Figure 1-1**). This biosocial model hypothesizes that genetic predispositions could lead to a variety of

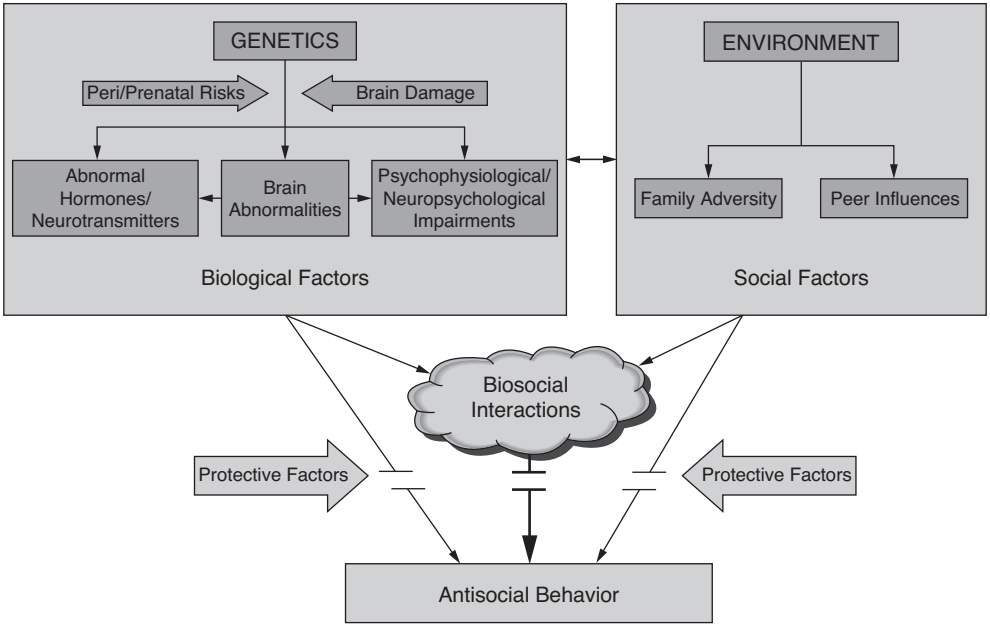

Figure 1-1 A Biosocial Model for Antisocial Behavior

biological risk factors, including abnormal hormones/neurotransmitters, brain abnormalities, and psychophysiological/neuropsychological impairments, which ultimately trigger antisocial behavior. The involvement of peri- and prenatal risks and brain damage could further contribute to the development of those biological deficits in this biosocial mechanism of antisocial behavior. It is proposed that the presence of both social and biological factors greatly increases the likelihood of developing a more severe form of antisocial behavior (i.e., repetitive violent offending). In addition, protective factors (e.g., intact prefrontal cortex, benign family) are also included in this model because they may interfere with the outcome of antisocial behavior.

Nonetheless, this model is unlikely to be applicable to different groups of antisocial individuals with a wide variety of symptom manifestations. Specifically, it has been argued that theoretical models involving biological vulnerabilities and maladaptive early home environments are better served in explaining the pathology underlying life-course persistent offenders (i.e., individuals with stable, continuous, lifelong antisocial behavior that begins in early childhood) but not their adolescent-limited counterparts (i.e., individuals with late-onset antisocial behavior who recover by early adulthood).[97] On the other hand, some unique subgroups of antisocial individuals, such as psychopaths, have also been found to show a different pathological pattern, such as the lack of executive dysfunction or prefrontal deficits, and thus may not share the same underlying biosocial mechanisms with other antisocial populations. Despite its limitations, this biosocial model provides several testable hypotheses that could guide future research in examining the epidemiology of antisocial behavior.

CONCLUSION

In general, studies on antisocial individuals presented convincing evidence for a biological contribution to antisocial behavior. Psychophysiological studies showed that lower skin conductance and heart rate activity/reactivity, excessive slow-wave EEG, atypical EEG frontal asymmetry, and ERP response are among the most robust findings on antisocial individuals. Prenatal and perinatal studies have linked increased numbers of minor physical anomalies, prenatal nicotine and alcohol exposure, and birth complications to antisocial behavior, particularly with respect to repetitive violent behavior. Imaging studies on antisocial individuals found promising results suggesting that abnormal brain structure and function, particularly in the prefrontal and temporal cortex, may predispose to antisocial behavior. These findings are consistent with lesion studies showing that damage to the frontal and temporal regions is followed by an increase in antisocial behavior. Neuropsychological findings of lower general intelligence as well as poorer performance on EF in antisocial individuals have also been observed. Other biological factors including hormones, neurotransmitters, and toxins have also been examined, and antisocial individuals have been found to show lower cortisol, higher testosterone, reduced serotonin activity, and a high level of metal toxicity. Despite some null findings, these biological risks were found to be associated with antisocial behavior in both children and adults.

The findings from studies examining biological and social interactions suggest that such interactions predispose to antisocial behavior. The significant number of biosocial studies on obstetrical factors provides the strongest evidence for such interaction effects, although supporting evidence was also accumulated in other areas of biological research in antisocial behavior. Overall, the findings suggest that biological risk factors, particularly low heart rate, obstetrical risks, and abnormal levels of hormones and neurotransmitters, when combined with social risk factors (i.e., low socioeconomic status, family adversity) significantly increase the likelihood of an antisocial outcome.

Using a different approach, a number of studies also found that antisocial individuals from relatively benign home backgrounds are more likely to exhibit higher biological risk factors compared with their counterparts from bad homes. Essentially, a greater degree of biological deficits may be needed to predispose an individual to antisocial behavior when their social backgrounds are otherwise relatively normal. Nonetheless, the most consistent findings in studies on biosocial interactions have demonstrated that individuals are most likely to engage in antisocial behavior when both the social and biological risk factors were present. In fact, these individuals are more likely to show persistent, violent antisocial behavior than those with only one risk factor. By reviewing the empirical findings and proposing a biosocial model for antisocial behavior, it is hoped that this chapter will encourage researchers to consider both biological and social factors in their research work, as such practice, if it becomes a standard, will generate a new body of knowledge for antisocial research and may lead to new insights on developmental pathways to antisocial behavior.

GLOSSARY

Antisocial behavior—an umbrella term for various behavioral problems, including violent, psychopathic, delinquent, and criminal behavior

Brain imaging—techniques used to examine the structure and functioning of the brain

Biosocial interactions—a process whereby the presence of a biological risk factor *and* a social risk factor increase the odds of antisocial behavior above and beyond the individual effects of either biological or social factors alone

Executive functioning—the cognitive processes that allow for goal-oriented, contextually appropriate behavior and effective self-serving conduct

Meta-analysis—a methodological technique used to quantitatively summarize all research conducted on a particular topic

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