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The Biological Basis of Crime

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Recognition is increasing that biological processes are at some level implicated in the development of criminal behavior. There is certainly debate about the precise contribution of such factors to crime outcome, and there is considerable debate about the precise mechanisms that these biological factors reflect. Yet few serious scientists in psychology and psychiatry would deny that biological factors are relevant to understanding crime, and public interest in and understanding of this perspective are increasing. The discipline of criminology, on the other hand, has been reluctant to embrace this new body of knowledge. Part of the reason may be interdisciplinary rivalries, part may simply be a lack of understanding, and part may be due to deep-seated historical and moral suspicions of a biological approach to crime causation. For whatever reason, these data have been largely ignored by criminologists and sociologists.

It is hoped that this chapter will go some way to allaying these suspicions. Certainly many reasons exist to take this body of knowledge seriously. Biology is not destiny, and we *can* benignly change many of the biological predispositions that shape the violent offender. One of the reasons why we have been so unsuccessful in preventing adult crime is because interventions to date have systematically ignored the biological side of the biosocial equation that produces crime. If we are to be truly serious about tackling crime and violence in society, we need to give more attention to the biological factors that cause crime.

This chapter will first outline the evidence for a genetic predisposition to crime. If genetic factors are indeed involved, then there has to be some biological basis to crime (although it should also be made clear that environmental factors in addition give rise to biological risk factors for crime). It then turns to a discussion of psychophysiological factors that

predispose to crime and how one heritable influence (low physiological arousal) is thought to be the best-replicated biological correlate of antisocial behavior in child and adolescent samples. Technical advances have led to the ability to look directly at the brains of violent and criminal offenders, and the next section reviews the area of brain imaging and findings of prefrontal functional and structural deficits in adult offenders. Other biological processes are then briefly reviewed, including birth complications, minor physical anomalies, nutrition, hormones, neurotransmitters, and molecular genetics.[1] Finally, policy implications of this research will be outlined, covering intervention and prevention programs aimed at reducing the effect of biological risk factors, and also implications of brain imaging research for the criminal justice system.

GENETICS

Twin Studies

The twin method for ascertaining whether a given trait is to any extent heritable makes use of the fact that monozygotic (MZ) or "identical" twins are genetically identical, having 100 percent of their genes in common with one another. Conversely, dizygotic (DZ) or "fraternal" twins are less genetically alike than MZ twins, and are in fact no more alike genetically than non-twin siblings.

When the trait being measured is a dichotomy (for example, criminal/noncriminal), "concordance" rates are calculated for MZ and DZ twins separately. A 70 percent concordance for crime in a set of MZ twins, for example, would mean that if one of the MZ pair is criminal, then the chance of the co-twin being criminal is 70 percent. Similar concordance rates can be calculated for DZ twins. If MZ twins have higher concordance rates for crime than DZ twins, then this constitutes some evidence for the notion that crime has a heritable component. The difference between these correlation coefficients, when doubled, gives an estimate of heritability, or the proportion of variance in criminality that can be attributed to genetic influences (Falconer 1965).

Are identical twins more concordant for criminality than fraternal twins? The answer from many reviews conducted on this expanding field is undoubtedly yes. As one example, a review of all the twin studies of crime conducted up to 1993 showed that although twin studies vary widely in terms of the age, sex, country of origin, sample size, determination of zygosity, and definition of crime, nevertheless all thirteen studies of

crime show greater concordance rates for criminality in MZ as opposed to DZ twins (Raine 1993). If one averages concordance rates across all studies (weighting for sample sizes), these thirteen studies result in concordances of 51.5 percent for MZ twins and 20.6 percent for DZ twins. Furthermore, the twin studies that have been conducted since 1993 have confirmed the hypothesis that there is greater concordance for antisocial and aggressive behavior in MZ relative to DZ twins (for example, Slutske et al. 1997; Eley, Lichenstein, and Stevenson 1999).

Twin studies have methodological limitations that restrict the conclusions that can be drawn from individual studies. A very common criticism of twin studies is that MZ twins may share a more common environment than DZ twins. For example, parents may treat MZ twins in a more similar fashion than DZ twins, thus artificially raising concordance rates in MZ twins. If this were true, the greater concordance for crime in MZ twins may be due more to environmental than genetic factors. Some evidence indicates that this may be the case (Allen 1976).

Criticisms such as these tend to lead researchers to discount results from twin studies as showing evidence for heritability, but there is also counterevidence. Grove et al. (1990) studied thirty-two sets of monozygotic twins who were separated and reared apart shortly after birth, and found statistically significant heritabilities for antisocial behavior in both childhood (0.41) and adulthood (0.28). Such evidence for heritability cannot be due to being raised in the same environment. Furthermore, one has to consider the methodological problems with twin studies, which can *decrease* estimates of heritability as opposed to artificially increasing them. For example, there is evidence that some twins make attempts to "de-identify" or be different from one another (Schacter and Stone 1985), while other twin pairs develop opposite (for example, dominant-submissive) role relationships (Moilanen 1987). These effects are expected to be greater in MZ pairs, with the result of artificially *reducing* heritability estimates. Though MZ twins are genetically identical, identical twinning can result in biological differences that can accentuate human differences. For example, there is a greater discrepancy in the birthweights of MZ twins relative to DZ twins, and birth complications have been linked to differences in behavior and cognition. This nongenetic, biological factor will result in an exaggeration of behavioral differences in MZ twins and a reduction in heritability estimates. The methodological problems of twin studies are just as likely to *decrease* heritability estimates as opposed to *inflating* them; in all probability these effects tend to cancel each other out.

Adoption Studies

Adoption studies also overcome the problem with twin studies because they more cleanly separate out genetic and environmental influences. We can examine offspring who have been separated from their criminal, biological parents early in life and sent out to other families. If these offspring grow up to become criminal at greater rates than foster children whose biological parents were not criminal, this would indicate a genetic influence with its origin in the subject's biological parents.

A variation of this type of study is the "cross-fostering" technique that has been used extensively in experimental genetic studies of animals. Applied to humans, the offspring whose biological parents are criminal or noncriminal are raised by parents who themselves are either criminal or noncriminal. This 2 x 2 design capitalizes on what is effectively a natural experiment, and allows for a more systematic exploration of genetic and environmental influences. As will be seen later, this method also allows an assessment of possible interactions between genetic and environmental influences.

A good example of a cross-fostering adoption study is a classic study conducted by Mednick et al. (1984), illustrated in Table 3.1. These researchers based their analyses on 14,427 adoptions that took place in Denmark between 1927 and 1947. Infants were adopted out immediately in 25.3 percent of cases, 50.6 percent within one year, 12.8 percent in the second year, and 11.3 percent after age two. Court records were obtained on 65,516 biological parents, adoptive parents, and adoptees in order to assess which subjects had convictions. When both adoptive and biological parents were noncriminal (neither genetic nor environmental predispositions present), 13.5 percent of the adoptees had a criminal record. This increased to 14.7 percent when adoptive parents only were criminal, meaning that an environmental but not genetic effect was operating. When only the biological parents were criminal, the conviction rate in the adoptees increased to 20.0 percent. When both adoptive and biological parents were criminal (both genetic and environmental predispositions present), the conviction rate increased to 24.5 percent. The effect of an adopted child having a criminal biological parent was associated with a statistically significant increase in the likelihood of the adoptee becoming criminal.

While this is but one example, a review of fifteen other adoption studies conducted in Denmark, Sweden, and the United States shows that all but one find a genetic basis to criminal behavior (Raine 1993). Importantly, evidence for this genetic predisposition has been found by several independent research groups in several different countries. These data, therefore, provide evidence that the basic finding is robust. Interestingly, the three studies that

Table 3.1
Results of Cross-Fostering Analyses (Percentages refer to the proportion of adoptees who had court convictions.)

		Are biological parents criminal?	
		Yes	No
Are adoptive parents criminal?	Yes	24.5%	14.7%
	No	20.0%	13.5%

SOURCE: Mednick, Gabrielli, and Hutchings (1984).

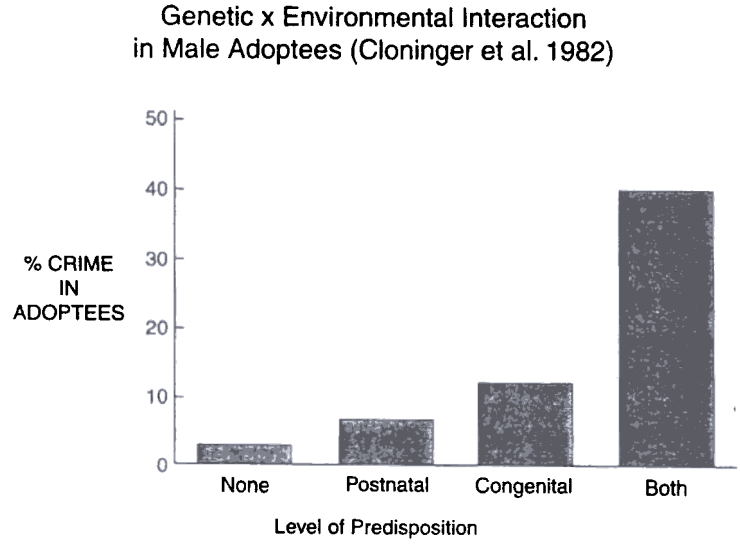
had a large enough sample size to separate violent from nonviolent, petty property crime found that there is heritability for petty property crimes but not for violent crimes (Bohman et al. 1982; Mednick et al. 1984; Sigvardsson et al. 1982). On the other hand, an adoption study by van den Oord, Boomsma, and Verhulst (1994) found heritability of 70 percent for aggressive behavior compared to 39 percent for delinquency. Consequently, while there is very clear evidence for a genetic basis to adult criminal offending, there is currently some question as to whether adoption studies of violent adult offending in particular show a genetic basis.

One of the key themes of this chapter is the notion that the *interaction* between biological and social factors may be particularly important. This concept is well illustrated in a cross-fostering analysis of petty criminality (Cloninger et al. 1982), results of which are illustrated in Figure 3.1. Swedish adoptees ($N = 862$) were divided into four groups depending on the presence or absence of (1) a congenital predisposition (that is, whether biological parents were criminal) and (2) a postnatal predisposition (how the children were raised by their adoptive parents). When both heredity and environmental predispositional factors were present, 40 percent of the adoptees were criminal compared to 12.1 percent with only genetic factors present, 6.7 percent for those with only a bad family environment, and 2.9 percent when both genetic and environmental factors were absent. The fact that the 40 percent rate for criminality when both biological and environmental factors are present is greater than the 18.8 percent rate given by a combination of "congenital only" and "postnatal only" conditions indicates that genetic and environmental factors are interacting.

Further analyses indicated that the occupational status of both biological and adoptive parents were the main postnatal variables involved in this nonadditive interaction. Cloninger and Gottesman (1987) later analyzed data for females with larger sample sizes. As would be expected, these crime rates in female adoptees are much lower than for males, but the same interactive pattern is present: Crime rates in adoptees are greatest when both heritable and environmental influences are present, with this interaction accounting for twice as much crime as is produced by genetic and environmental influences taken alone.

Evidence for gene x environment interaction is also provided by Cadoret et al. (1983), who presented data from three adoption studies. When both genetic and environmental factors are present, they account for a greater number of antisocial behaviors than either of these two factors acting alone. Crowe (1974) also found some evidence for a gene x environment interaction in his analysis of adopted-away offspring of female prisoners, though this trend was only marginally significant ($p < .10$). Cadoret et al. (1995) in an adoption study of 95 male and 102 female adoptees whose parents had either antisocial personality and/or alcohol abuse showed that parental antisocial personality predicted increased aggression and conduct disorders in the offspring—illustrating evidence for genetic processes. But in addition, adverse adoptive home environment was found to *interact* with adult antisocial personality in predicting increased aggression in the offspring, that is, a gene x environment interaction effect.

A related but different concept is that of gene-environment correlations. An interesting example of this is a study by Ge et al. (1996), who showed that the adopted-away offspring of biological parents who had antisocial personality / substance abuse were more likely to show antisocial and hostile behaviors in childhood. This helps establish genetic transmission of childhood antisocial behavior. In addition, an association was found between antisocial behavior in the biological parent and the parenting behaviors of the adoptive parents. This can be explained by a transmission pathway in which the biological parent contributes a genetic predisposition toward antisocial behavior in the offspring. The antisocial offspring then in turn elicit negative parenting behaviors in the adoptive parents. This study provides direct evidence of an "evocative" gene-environment correlation, and suggests that the association between negative parenting in the adoptive parent and antisocial behavior in the child is mediated by genetic processes. One of the goals of future behavior genetic studies should be to further examine the interplay between genes and environment in this fashion. More generally, there are likely to be future exciting developments with



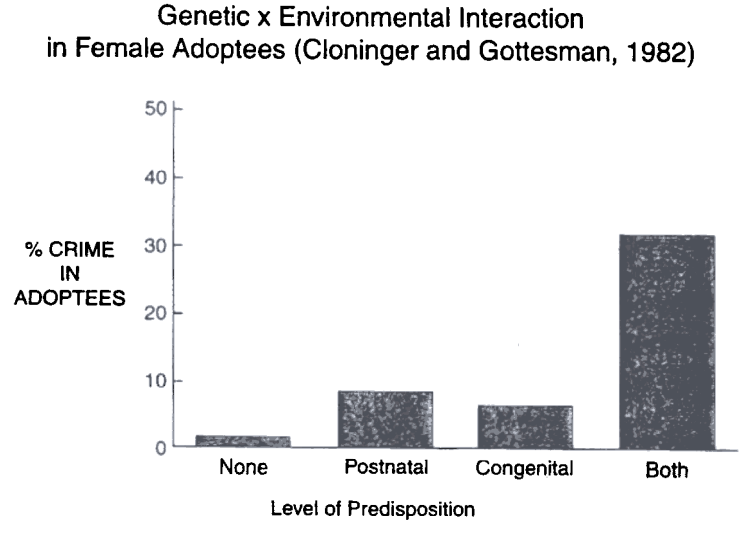


Figure 3.1
Results of a Cross-Fostering Analysis Indicating Evidence for an Interaction between Genetic and Environmental Factors in (a) Males and (b) Females.
SOURCES: Cloninger and Gottesman (1987); Cloninger et al. (1982).

respect to identifying the specific genes which give rise to the risk factors that shape criminal behavior (see section on neurogenetics).

PSYCHOPHYSIOLOGY

Since the 1940s an extensive body of research has been built up on the psychophysiological basis of antisocial, delinquent, criminal, and psychopathic behavior. For example, there have been at least 150 studies on electrodermal (sweat rate) and cardiovascular (heart rate) activity in such populations, and in electroencephalographic (EEG) research alone there have been hundreds of studies on delinquency and crime (Gale 1975). This body of research has received little attention in the broader field of criminology, and is rarely referred to in textbooks on crime. One purpose of this chapter is to bring this body of knowledge to the attention of this more general audience.

Definitions of psychophysiology vary, but one useful perspective outlined by Dawson (1990) is that it is "concerned with understanding the relationships between psychological states and processes on the one hand and physiological measures on the other hand" (p. 243). Psychophysiology is uniquely placed to provide important insights into criminal behavior because it rests at the interface between clinical science, cognitive science, and neuroscience (Dawson 1990). Thus, it is sometimes easier to see the relevance of this research for crime relative to biochemistry research because concepts in psychophysiology are more easily linked to broader concepts such as learning, emotion, arousal, and cognition.

There are many psychophysiological correlates of antisocial, criminal, and psychopathic behavior.[2] The focus here will lie with one particular psychophysiological construct, low arousal, because—as will become clear—it is the strongest psychophysiological finding in the field of antisocial and criminal behavior.

EEG Underarousal

One influential psychophysiological theory of antisocial behavior is that antisocial individuals are chronically underaroused. Traditional psychophysiological measures of arousal include heart rate, skin conductance activity, and electroencephalogram (EEG) measured during a "resting" state. Low heart rate and skin conductance activity, and more excessive slow-wave EEG (delta activity with a frequency of 1–4 cycles per second [cps], 4–7 cps theta activity, and 8–10 cps slow alpha) indicate underarousal, that is, less than

average levels of physiological arousal. Most studies tend to employ single measures of arousal, although studies that employ multiple measures are in a stronger position to test an arousal theory of antisocial behavior.

EEG is recorded from scalp electrodes that measure the electrical activity of the brain. Literally hundreds of studies assessing EEG in criminals, delinquents, psychopaths, and violent offenders have been done over the past sixty years, and it is clear that a large number of them implicate EEG abnormalities in violent recidivistic offending. As examples, Bach-y-Rita et al. (1971) and Hill and Pond (1952) examined large samples of violent offenders and observed EEG abnormalities in about 50 percent of the cases, with the most common abnormality being excessive slow-wave EEG (underarousal). These findings have been supported by studies of murderers and other violent offenders (for example, Mark and Ervin 1970; Williams 1969). Fishbein et al. (1989), in a sample of 124 adult male drug abusers, found that aggression was associated with increased slow-wave theta activity and decreased alpha, indicating underarousal. Convit, Czobor, and Volavka (1991) observed the same pattern within a sample of psychiatric inpatients; the number of instances of violence on wards was related to increased levels of delta activity and lower levels of alpha. Drake, Hietter, and Pakalnis (1992) found that, while none of 24 depressed patients and only 1 of 20 headache control patients had abnormal EEGs, 7 of 23 patients with either intermittent explosive disorder or episodic dyscontrol (brief periods where the individual lacks control) had diffuse or focal slowing in EEG. Murderers have more recently been shown to have more EEG deficits in the right than the left hemisphere of the brain, with multiple abnormalities being especially present in the right temporal cortex (Evans and Park 1997). On the other hand, Pillmann et al. (1999) showed greater abnormalities in the *left* temporal region of repeat violent offenders.

Generally speaking, the prevalence of EEG abnormalities in violent individuals in this large literature ranges from 25 percent to 50 percent, with the rate of abnormalities in normals estimated as ranging from 5 percent to 20 percent. The bulk of this research implicated the more frontal regions of the brain, areas that regulate executive functions such as planning and decision making. Similar conclusions are drawn by Volavka (1987) and Milstein (1988) for crime in general and violent crime in particular.

Cardiovascular Underarousal

Data on resting heart rate provides striking support for underarousal in antisocials. Indeed, the findings for heart rate level (HRL) on non-

institutionalized antisocials are believed to represent the strongest and best replicated biological correlate of antisocial behavior. A detailed review of these studies and full theoretical and methodological considerations are given in Raine (1993) and Raine (1996a). Briefly, twenty-four studies of resting heart rate and conduct-disordered, delinquent, and antisocial children and adolescents performed between 1971 and 1996 yielded twenty-nine independent samples of antisocials and a total of thirty-eight effect sizes (a measure of the strength of the effect). Of the thirty-eight effect sizes, thirty-two were significant and positive (that is, low heart rate associated with antisocial behavior), while only one was significant and in the negative direction. This latter, unexpected finding (Zahn and Kruesi 1993) was interpreted by its authors as possibly due to biased referral to the clinic by anxious, easily stressed parents who worried about their child and who may genetically transmit high HRL to their child.

A low resting heart rate is the best-replicated biological marker of antisocial and aggressive behavior in childhood and adolescent community samples. Resting HRL was measured in a wide variety of ways, including polygraphs, pulse meters, and stopwatches. A wide number of definitions of antisocial behavior are used, ranging from legal criminality and delinquency to teacher ratings of antisocial behavior in school, self-report socialization measures, diagnostic criteria for conduct disorder, and genetically inferred law breaking (i.e. offspring of criminals). Subjects were also assessed in a wide variety of settings, including medical interview, study office, school, university laboratory, and hospital. In the light of such variability, it is surprising that consistency in findings have been obtained, attesting to the robustness of the observed effects. Importantly, there has also been good cross-laboratory replication of the finding, and it has also been found in six different countries—England, Germany, New Zealand, the United States, Mauritius, and Canada—illustrating invariance to cultural context.

The link between low heart rate and crime is not the result of such things as height, weight, body bulk, physical development, and muscle tone (Raine, Venables, and Mednick 1997; Raine, Buchsbaum and La Casse 1997; Wadsworth 1976; Farrington 1997); scholastic ability and IQ (Raine, Venables, and Williams 1990; Farrington 1997); excess motor activity and inattention (Raine, Venables, and Mednick 1997; Farrington 1997); drug and alcohol use (Raine, Venables, and Mednick 1997); engagement in physical exercise and sports (Wadsworth 1976; Farrington 1997); or low social class, divorce, family size, teenage pregnancy, and other psychosocial adversity (Raine, Venables, and Williams 1990; Wadsworth 1976; Farrington 1997). Intriguingly, an unusual and important feature of the relationship is

its diagnostic specificity. No other psychiatric condition has been linked to low resting heart rate. Other psychiatric conditions, including alcoholism, depression, schizophrenia, and anxiety disorder, have, if anything, been linked to *higher* (not lower) resting heart rate.

Low heart rate has been found to be an independent predictor of violence. Out of forty-eight psychosocial and individual measures, only two risk factors were related to violence independently of all other risk factors in all six analyses: low resting heart rate and poor concentration (Farrington 1997). Indeed, low heart rate was more strongly related to both self-report and teacher measures of violence than having a criminal parent. These findings led Farrington (1997) to conclude that low heart rate may be one of the most important explanatory factors for violence (p. 99). There is also a substantial heritability for resting heart rate, suggesting that it may be a genetic marker for antisocial and criminal behavior (Raine et al. 1990). Furthermore, the offspring of criminal parents have been shown to have low resting heart rate in two separate studies (Farrington 1987; Venables 1987). Low heart rate characterizes female as well as male antisocial individuals. Several studies, including two that are prospective, have now established that, *within* females, low heart rate is linked to antisocial behavior (Raines et al. 1990; Maliphant, Hume, and Furnham 1990; Raine, Venables, and Mednick 1997; Moffitt and Caspi In press).

Prospective Studies of Underarousal

One of the major difficulties in trying to draw conclusions on the psychophysiological basis of criminal behavior is that most studies conducted to date have been nonprospective and have utilized institutionalized populations. In addition, most studies report results from only one of the three most commonly measured psychophysiological response systems (electrodermal, cardiovascular, and cortical). Prospective longitudinal research—that is, research that follows people forward through their lives—allows for much more powerful statements to be made about predispositions for criminal behavior and to elucidate cause and effect relationships; but because prospective research is more difficult to execute, there are few such studies.

Regarding heart rate levels, five prospective studies of heart rate alone have confirmed that low heart rate is predictive of later antisocial behavior, while five additional prospective studies also show significant effects for electrodermal and electrocortical arousal. Wadsworth (1976) found that lower resting heart rate in unselected eleven-year-old schoolboys predicted delinquency measured from ages eight to twenty-one. The very lowest

HRLs were found in those who committed nonsexual violent criminal offenses as adults. Similarly, Farrington (1987) found that resting heart rate measured at age eighteen to nineteen in noninstitutionalized males predicted to violent criminal offending at age twenty-five. With respect to EEG, two separate studies have shown that slow alpha frequency predicts to adult thievery in a sample of 129 Danish thirteen-year-old boys (Mednick et al. 1981) and 571 Swedish one- to fifteen-year-old boys (Petersen et al. 1982). Low heart rate characterizes life-course-persistent antisocial individuals in particular; Moffitt and Caspi (In press) have recently found that low resting heart rate assessed at ages seven, nine, and eleven is particularly characteristic of life-course-persistent offenders, a group who have been hypothesized as having early neurobiological deficits (Moffitt 1993).

As with most other studies, evidence in these prospective studies for prediction to antisocial behavior is based on only one measure of arousal. One nine-year prospective study of crime by Raine, Venables, and Williams (1990) has shown, however, that low resting heart rate, low resting skin conductance activity, and excessive slow-wave theta EEG (indicating underarousal) measured at age fifteen in normal unselected schoolboys predicted criminal behavior at age twenty-four. These three measures correctly classified 74.7 percent of all subjects as criminal/noncriminal, a rate significantly greater than chance (50 percent). In the total population, the three arousal measures were statistically independent; the fact that they all independently predicted to criminal behavior indicates strong support for an arousal theory of criminal and antisocial behavior (although this finding also cautions against the use of a simplistic, unitary arousal concept in explaining crime). Group differences in social class, academic ability, and area of residence were not found to mediate the link between underarousal and antisocial behavior.

Interpretations of Low Arousal: Fearlessness and Stimulation-Seeking Theories

Why should low arousal and low heart rate predispose to antisocial and criminal behavior? There are two main theoretical interpretations. Fearlessness theory indicates that low levels of arousal are markers of low levels of fear (Raine 1993; Raine 1997). For example, particularly fearless individuals such as bomb disposal experts who have been decorated for their bravery have particularly low HRLs and reactivity (Cox et al. 1983; O'Connor, Hallam, and Rachman 1985), as do British paratroopers decorated in the Falklands War (McMillan and Rachman 1987). A fearlessness

interpretation of low arousal levels assumes that subjects are not actually at "rest," but that instead the rest periods of psychophysiological testing represent a mildly stressful paradigm and that low arousal during this period indicates lack of anxiety and fear. Lack of fear would predispose to antisocial and violent behavior because such behavior (for example, fights and assaults) requires a degree of fearlessness to execute, while lack of fear, especially in childhood, would help explain poor socialization since low fear of punishment would reduce the effectiveness of conditioning. Fearlessness theory receives support from the fact that autonomic underarousal also provides the underpinning for a fearless or uninhibited temperament in infancy and childhood (Scarpa et al. 1997; Kagan 1994).

A second theory explaining reduced arousal is stimulation-seeking theory (Eysenck 1964; Quay 1965; Raine 1993; Raine, Reynolds, Venables, et al. 1998). This theory argues that low arousal represents an unpleasant physiological state; antisocials seek stimulation in order to increase their arousal levels back to an optimal or normal level. Antisocial behavior is thus viewed as a form of stimulation-seeking, in that committing a burglary, assault, or robbery could be stimulating for some individuals. Before leaving this theory, the possibility has to be considered that fearlessness theory and stimulation-seeking theory may be complementary rather than competing theories. That is, low levels of arousal may predispose to crime because it produces some degree of fearlessness, and also encourages antisocial stimulation-seeking. Indeed, behavioral measures of stimulation-seeking and fearlessness, both taken at age three in a large sample, predict to aggressive behavior at age eleven (Raine, et al. 1998). The combined effect of these two influences may be more important in explaining antisocial behavior than either influence taken alone.

Psychophysiological Protective Factors against Crime Development

Until recently, there had been no research on biological factors that *protect* against crime development, but that is changing. We are discovering that *higher* autonomic activity during adolescence may act as a protective factor against crime development. Raine, Venables, and Williams (1995, 1996) report on a fourteen-year prospective study in which measures of arousal, orienting (sweat rate and heart rate responses to tone stimuli), and classical conditioning (learning through association) were taken in 101 unselected fifteen-year-old males. Of these, seventeen adolescent antisocials who desisted from adult crime (Desistors) were matched on adolescent antisocial

behavior and demographic variables with seventeen adolescent antisocials who had become criminal by age twenty-nine (Criminals), and seventeen non-antisocial, noncriminals (Controls). Desistors had significantly higher HRLs as well as higher scores on the other psychophysiological measures than did the Criminals. Findings suggest that boys who are antisocial during adolescence but who do not go on to adult criminal offending may be protected from such an outcome by their high arousal levels.

Findings from a second study on adults provide some support for this initial finding in adolescents. Brennan et al. (1997) report on a study of protective factors in fifty men predisposed to crime by virtue of having a seriously criminal father who had been imprisoned. Of these men, twenty-four developed a criminal record and were imprisoned themselves, while the other twenty-six did not show any criminal offending. Heart rate and skin conductance measures of reactivity to fourteen orienting tones were measured at age thirty-five. The group who desisted from crime was found to have significantly higher levels of physiological orienting relative to those who exhibited criminal behavior, and to a noncriminal control group who had noncriminal fathers. Desistors seem to have a nervous system particularly sensitive to forming associations between signals of punishment and the punishment itself. In a similar fashion, higher resting heart rates in Desistors may be interpreted as indicating higher levels of fearfulness in these individuals.

Overall, the initial profile that is being built up on the psychophysiological characteristics of the Desistor is one of heightened information processing (better orienting), greater responsivity to environmental stimuli in general (fast recovery), greater sensitivity to cues predicting punishment in particular (better classical conditioning), and higher fearfulness (high HRLs). The importance of research on psychophysiological protective factors such as these is that they offer suggestions for possible intervention and prevention strategies.

BRAIN IMAGING

Advances in brain imaging techniques in the past fifteen years have provided the opportunity to gain dramatic new insights into the brain mechanisms that may be dysfunctional in violent, psychopathic offenders. In the past, the idea of peering into the mind of a murderer to gain insights into his or her acts was the province of pulp fiction or space-age movies. Yet now we can literally look at, and into, the brains of murderers using functional and structural imaging techniques that are currently revolutionizing our understanding of the causes of clinical disorders.

Brain imaging studies of violent and psychopathic populations have been reviewed by Raine (1993), Raine and Buchsbaum (1996), and Henry and Moffitt (1997). These reviews (that cover studies up to 1994), while showing variability in findings across studies, concur in indicating that violent offenders have structural and functional deficits to the frontal lobe (behind the forehead) and the temporal lobe (near the ears). Between 1994 and 1997, six more studies support this key finding of anterior brain dysfunction (Goyer et al. 1994; Volkow et al. 1995; Kuruoglu et al. 1996; Seidenwurm et al. 1997; Intrator et al. 1997; Soderstrom et al. 2000). Taken together, these later studies show continued support of the notion that poor functioning of the frontal and temporal regions may predispose to crime. Out of the six more-recent studies, five showed evidence for frontal dysfunction and four showed evidence for temporal lobe dysfunction. Despite some discrepancies, the first generation of brain imaging studies supports earlier contentions from animal and neurological studies implicating the frontal (and to some extent temporal) brain regions in the regulation and expression of aggression.

Prefrontal Dysfunction in Murderers

In the first published brain imaging study of murderers (Raine, Buchsbaum, Stanley et al. 1994), we scanned the brains of twenty-two murderers pleading not guilty by reason of insanity (or otherwise found incompetent to stand trial) and compared them to the brains of twenty-two normal controls who were matched with the murderers on sex and age. The technique we used was positron emission tomography (PET), which allowed us to measure the metabolic activity of many different regions of the brain including the prefrontal cortex, the frontalmost part of the brain. We had subjects perform a task that required them to maintain focused attention and be vigilant for a continuous period of time, and it is the prefrontal region of the brain that in part subserves this vigilance function.

The key finding was that the murderers showed significantly poorer functioning of the prefrontal cortex, that part of the brain lying above the eyes and behind the forehead. It is thought that poorer functioning of the prefrontal cortex predisposes people to violence for a number of reasons. Reduced prefrontal functioning can result in a loss of the ability of this part of the brain to control deeper and more primitive subcortical structures, such as the amygdala, which are thought to give rise to aggressive feelings. Prefrontal damage also encourages risk-taking, irresponsibility, rule breaking, emotional and aggressive outbursts, and argumentative

behavior that can also predispose to violent criminal acts. Loss of self-control, immaturity, lack of tact, inability to modify and inhibit behavior appropriately, and poor social judgment could predispose to violence as well. This loss of intellectual flexibility and problem-solving skills, and reduced ability to use information provided by verbal cues can impair social skills essential for formulating nonaggressive solutions to fractious encounters. Poor reasoning ability and divergent thinking that results from prefrontal damage can lead to school failure, unemployment, and economic deprivation, thereby predisposing to a criminal and violent way of life. A further study showed that it was especially the impulsive, emotionally undercontrolled murderers who were especially likely to show prefrontal deficits (Raine, Meloy et al. 1998). Nevertheless, it should be recognized that, while there is an association between poor prefrontal function and impulsive violence, this brain dysfunction may be essentially a predisposition only, requiring other environmental, psychological, and social factors to enhance or diminish this tendency.

Corpus Callosum, Left Angular Gyrus, and the Subcortex

What other brain deficits, apart from prefrontal dysfunction, characterize murderers? We took this imaging research a step further by expanding our sample from twenty-two to forty-one murderers, and also by increasing the size of our control group to forty-one. This increase in sample size gave us more statistical power to detect group differences, and in 1997 we reported our updated findings (Raine, Buchsbaum, and La Casse 1997). The results were interesting for a variety of reasons. First, we confirmed that there was a significant reduction in the activity of the prefrontal region in murderers.

Second, we now found in this larger sample that the left angular gyrus was functioning more poorly in the murderers. The angular gyrus lies at the junction of the temporal (side of head), parietal (top and back of head), and occipital (very back of head) regions of the brain and plays a key role in integrating information from these three lobes. Reductions in activity of the left angular gyrus have been correlated with reduced verbal ability, while damage to this region has been linked to deficits in reading and arithmetic. Such cognitive deficits could predispose to educational and occupational failure which in turn predisposes to crime and violence. The fact that learning deficits have been found to be common in violent offenders lends further support to this interpretation.

Third, we found reductions in the functioning of the corpus callosum, the band of white nerve fibers that provide lines of communication between the left and right hemispheres. Although we can only speculate at the present time, we think that poor connection between the hemispheres may mean that the right hemisphere, which is involved in the generation of negative emotion (Davidson and Fox 1989), may experience less regulation and control by the inhibitory processes of the more dominant left hemisphere, a factor that may contribute to the expression of violence. Furthermore, researchers have commented on the inappropriate nature of emotional expression and the inability to grasp long-term implications of a situation in split-brain patients who have had their corpus callosum surgically severed. This implies that the inappropriate emotional expression of violent offenders and their lack of long-term planning may be partly accounted for by poor functioning of the corpus callosum. Nevertheless, callosal dysfunction by itself is unlikely to cause aggression. Instead, it may only contribute to violence in those who also have other brain abnormalities.

Effect of the Home Environment on Brain-Violence Relationships

How do psychosocial deficits moderate the relationship between prefrontal dysfunction and violence? We addressed this question by dividing our sample of murderers into those who came from relatively good home backgrounds and those who came from relatively bad ones (Raine, Stoddard, et al. 1998). In this study, ratings of psychosocial deprivation took into account early physical and sexual abuse, neglect, extreme poverty, foster home placement, a criminal parent, severe family conflict, and a broken home. The results of the study showed that, while the deprived murderer shows relatively good prefrontal functioning, it is the nondeprived murderer who shows the characteristic lack of prefrontal functioning. In particular, we found that murderers from good homes had a 14.2 percent reduction in the functioning of the right orbitofrontal cortex, a brain area that is of particular interest. Damage to this brain area results in personality and emotional deficits that parallel criminal psychopathic behavior, or what Damasio and colleagues have termed "acquired sociopathy" (Damasio 1994).

These findings are at one level counterintuitive, but from another perspective they make some sense. If a seriously violent offender comes from a bad home environment, then it seems likely that the cause of the offender's violence is due to that bad environment. But if they come from a good home background, then environmental causation seems less likely

and instead biological deficits may be a better explanation. Consistent with these brain imaging findings, previous research has shown that poor fear conditioning in schoolchildren is related to antisocial behavior especially in those from a *good* home environment (Raine and Venables 1981). That is, the biological deficit (poor conditioning) is found in those who *lack* a social predisposition to antisocial behavior. Perhaps not surprisingly, it is the right orbitofrontal cortex (situated above the eye orbits) that has been found to play an important role in the development of fear conditioning.

Reduced Prefrontal Gray Matter in Antisocial Personality Disorder

Ranging from single case studies (Damasio et al. 1994) to series of neurological patients (Damasio, Tranel, and Damasio 1990; Stuss and Benson 1986), those who have suffered demonstrable damage to both gray and white matter within the prefrontal region of the brain proceed to acquire an antisocial, psychopathic-like personality. These patients also show autonomic arousal and attention deficits to socially meaningful events (Damasio 1994, Damasio, Tranel, and Damasio 1990), a finding consistent with the role played by the prefrontal cortex in modulating emotion, arousal, and attention (Stuss and Benson 1986; Davidson 1993; Raine, Reynolds, and Sheard 1991).

We recently conducted a structural magnetic resonance imaging (MRI) study on volunteers from the community with Antisocial Personality Disorder and made volumetric assessments of prefrontal gray and white matter (Raine et al. 2000). Skin conductance and heart rate activity during a social stressor was also assessed in addition to psychosocial and demographic risk factors for violence. Subjects were drawn from temporary employment agencies in Los Angeles and consisted of twenty-one males with Antisocial Personality Disorder, a normal control group of thirty-four males, and a psychiatric control group of twenty-seven males with substance dependence. Antisocials had significantly lower prefrontal gray volumes than both Controls and Substance Dependents. In contrast, groups did not differ on white prefrontal volume, indicating specificity of the deficit to gray matter (neurons). Furthermore, Antisocials also showed reduced autonomic reactivity during the social stressor compared to both Controls and Substance Dependents.

When prefrontal gray matter was expressed as a function of whole brain volume, groups were again found to differ significantly, so the results are not due to general difference in brain size but a specific difference in

prefrontal size. Further analyses indicated that the three prefrontal and autonomic variables (prefrontal gray/whole brain, heart rate, skin conductance) predicted group membership with an accuracy of 76.9 percent. These prefrontal and autonomic deficits were independent of psychosocial deficits in the Antisocial group. After ten demographic and psychosocial risk factors for antisocial personality were statistically controlled for, the prefrontal and autonomic deficits added substantially to the prediction of Antisocial vs. Control group membership. As such, the brain deficits cannot be easily accounted for by psychosocial factors, and instead appear to reflect a different risk process. When both biological and social measures were used together, they correctly classified to 88.5 percent, indicating the importance of a biosocial perspective that integrates biological and social factors.

Brain Deficits in Violent Offenders with a History of Childhood Abuse

While the relationship between physical child abuse and violence is well established (Lewis et al. 1988; Tarter et al. 1984; Widom 1997), there appears to be little or no research, biological or social, on factors that differentiate abused victims who go on to perpetrate violence from those who refrain from adult violence. We recently conducted a study (Raine, Park, et al. In press) in which we used functional magnetic resonance imaging (fMRI) to address two important gaps in our knowledge of brain functioning and violence: (1) What are the brain correlates of adults in the community who have suffered severe physical abuse early in life and who go on to perpetrate serious violence in adulthood? (2) What characterizes those who experience severe physical abuse but who refrain from serious violence?

We recruited four groups of participants from the community: (1) nonviolent controls who had not suffered abuse; (2) participants with severe physical child abuse only (that is, had suffered severe physical or sexual abuse in the first eleven years, but were not violent); (3) ones with serious violence only (violence that caused either bodily injury or trauma, or that were life-threatening acts); and (4) severely abused, seriously violent offenders. All underwent fMRI while performing a visual/verbal working memory task. Functional magnetic resonance imaging measures blood flow within brain tissue, and thus allows one to assess the functional properties of the brain. In this sense, it is like PET, but unlike PET there is no exposure to radioactivity. Furthermore, it detects activity in brain regions that are as small as 1 millimeter. The task involved subjects hold-

ing in short-term memory pictures shown very briefly and pressing a response button any time a picture was repeated, a task known to activate the temporal and frontal regions of the brain.

Violent offenders who had suffered severe child abuse show reduced right hemisphere functioning, particularly in the right temporal cortex. Abused individuals who refrain from serious violence showed relatively lower left, but higher right, activation of the temporal lobe. Abused individuals, irrespective of violence status, showed reduced cortical activation during the working memory task, especially in the left hemisphere. These findings constitute the first fMRI study of brain dysfunction in violent offenders and indicate that initial right hemisphere dysfunction, when combined with the effects of severe early physical abuse, may predispose to serious violence. They also suggest that relatively good right hemisphere functioning *protects* against violence in physically abused children.

OTHER BIOLOGICAL PROCESSES: BIRTH COMPLICATIONS, MINOR PHYSICAL ANOMALIES, NUTRITION, AND NEUROCHEMISTRY

Birth Complications

Several studies have shown that babies who suffer birth complications are more likely to develop conduct disorder, delinquency, and impulsive crime and violence in adulthood (see Raine 1993 for a detailed review). Birth complications such as anoxia (getting too little oxygen), forceps delivery, and preeclampsia (hypertension leading to anoxia) are thought to contribute to brain damage, and this damage in turn may predispose to antisocial and criminal behavior. On the other hand, birth complications may not by themselves predispose to crime, but may require the presence of negative environmental circumstance to trigger later adult crime and violence.

One example of this "biosocial interaction" is a study of birth complications and maternal rejection in all 4,269 live male births that took place in one hospital in Copenhagen, Denmark (Raine, Brennan, and Mednick 1994). Birth complications were assessed by obstetricians and midwives. When the baby was one year old, the mother was interviewed by a social worker and the degree of maternal rejection was assessed by three measures: mother did not want the pregnancy, mother made an attempt to abort the fetus, and the baby was institutionalized for at least four months in the first year of life. Babies were then followed for eighteen years, when

their arrests for violent crimes were assessed. A highly significant interaction was found between birth complications and maternal rejection. Babies who only suffered birth complications or who only suffered maternal rejection were no more likely than normal controls to become violent in adulthood. On the other hand, those who had both risk factors were much more likely to become violent. Only 4 percent of the sample had both birth complications and maternal rejection, but this small group accounted for 18 percent of all the violent crimes committed by the entire sample. This finding from Denmark was replicated by Piquero and Tibbetts (1999) in a prospective longitudinal study of 867 males and females from the Philadelphia Collaborative Perinatal Project; those with both prenatal/perinatal disturbances and a disadvantaged familial environment were much more likely to become adult violent offenders.

This sample was studied again when they were age thirty-four (Raine, Brennan, and Mednick 1997). The results indicate that the biosocial interaction previously observed holds for violent but not nonviolent criminal offending. Furthermore, the interaction was found to be specific to more serious forms of violence and not threats of violence. The interaction held for early onset but not late onset violence and was not accounted for by psychiatric illness in the mothers. Rearing in a public care institution in the first year of life and an attempt to abort the fetus were the key aspects of maternal rejection found to interact with birth complications in predisposing to violence.

Minor Physical Anomalies

Minor physical anomalies (MPAs) have been associated with disorders of pregnancy and are thought to reflect maldevelopment of the fetus (including brain maldevelopment) toward the end of the first three months of pregnancy. MPAs are relatively minor physical abnormalities consisting of such features as low seated ears, adherent ear lobes, furrowed tongue, curved fifth finger, single transverse palmar crease, gaps between the first and second toes, unusually long third toes, and fine hair that doesn't easily comb down. They are not stigmatizing as they are not obvious unless a careful physical examination is done. While MPAs may have a genetic basis, they may also be caused by environmental factors of the fetus such as anoxia, bleeding, and infection (Guy et al. 1983).

Minor physical anomalies have also been found to characterize preadult antisocial behavior and temperament. Paulus and Martin (1986) found more MPAs in aggressive and impulsive preschool boys, while

Halverson and Victor (1976) also found higher levels of MPAs in elementary male schoolchildren with problem school behavior. MPAs have even been linked to peer aggression as early as age three (Waldrop et al. 1978). Although MPAs have generally characterized behavior disorders in children drawn from the normal population (see Pomeroy, Sprafkin, and Gadow 1988 for a review), at least one study failed to observe a link between MPAs and conduct disorder within a mixed group of emotionally disturbed children (Pomeroy, Sprafkin, and Gadow 1988).

Mednick and Kandel (1988) assessed MPAs in a sample of 129 twelve-year-old boys examined by an experienced pediatrician. MPAs were found to be related to violent offending as assessed nine years later when the subjects were age twenty-one, though not to property offenses without violence. However, when subjects were divided into those from unstable, nonintact homes versus those from stable homes, a biosocial interaction was observed. MPAs only predicted violence in those individuals raised in unstable home environments. A similar interactive relationship was also observed for birth weight and family stability (Kandel and Mednick, 1991). These findings are quite similar to those on birth complications reported above; in both studies the presence of a negative psychosocial factor is required to "trigger" the biological risk factor, and in both cases the effects are specific to violent offending. In a study confirming specificity to violence, Arseneault et al. (2000) found that MPAs predicted to violent delinquency during adolescence in 170 teenagers, but not to nonviolent delinquency. Furthermore, effects were independent of family adversity.

Nutrition

Although deficiency in nutrition itself has been rarely studied in relation to childhood aggression, several studies have demonstrated the effects of related processes including food additives, hypoglycemia, and more recently cholesterol on human behavior (Rutter, Giller, and Hagell 1998; Raine 1993; Fishbein and Pease 1994). In addition, some studies have shown associations between overaggressive behavior and vitamin and mineral deficiency (Breakey 1997; Werbach 1995). Furthermore, one study (Rosen 1996) claimed that nearly a third of a population of juvenile delinquents (mostly males) showed evidence of iron deficiency. Nevertheless, these findings remain both conflicting and controversial (Rutter, Giller, and Hagell 1998).

One intriguing study illustrates the potentially causal role of malnutrition as early as pregnancy in predisposing to antisocial behavior. Toward the end of World War II when Germany was withdrawing from Holland,

they placed a food blockade on the country that led to major food shortages and near starvation in the cities and towns for several months. Women who were pregnant at this time were exposed to severe malnutrition at different stages of pregnancy. The male offspring of these women were followed up into adulthood to ascertain rates of Antisocial Personality Disorder and were compared to controls who were not exposed to malnutrition. Pregnant women starved during the blockade had 2.5 times the rates of Antisocial Personality Disorder in their adult offspring compared to controls (Neugebauer, Hoek, and Susser 1999).

Initial evidence also shows relationships between both protein and zinc deficiency and aggression in animals (Tikal, Benesova, and Frankova 1976; Halas, Reynolds, and Sanstead, 1977). Recent studies of humans support these animal findings. Protein and zinc deficiency may lead to aggression by negatively impacting brain functioning. There is extensive experimental evidence in animals that the offspring of rats fed a diet containing marginal levels of either zinc or protein throughout pregnancy and lactation showed impaired brain development (Oteiza et al. 1990; Bennis-Taleb et al. 1999). In humans, zinc deficiency in pregnancy has been linked to impaired DNA, RNA, and protein synthesis during brain development, and congenital brain abnormalities (Pfeiffer and Braverman 1982). Similarly, protein provides essential amino acids for the rapid growth of fetal tissue. PET studies of violent offenders have revealed deficits to the prefrontal cortex and corpus callosum (Raine, Buchsbaum, and La Casse 1997; Volkow et al. 1995), and it is of interest that the offspring of rats fed a low-protein diet during pregnancy show a specific impairment to the corpus callosum (Wainwright and Stefanescu 1983) and reduction in DNA concentration in the forebrain (Bennis-Taleb et al. 1999). The amygdala, which also shows abnormal functioning in PET imaging of violent offenders (Raine, Buchsbaum, and La Casse 1997; Raine, Meloy, et al. 1998; Davidson, Putnam, and Larson 2000), is densely innervated by zinc-containing neurons (Christensen and Frederickson 1998), and males with a history of assaultive behavior were found to have lower zinc relative to copper ratios in their blood compared to nonassaultive controls (Walsh et al. 1997). Consequently, protein and zinc deficiency may contribute to the brain impairments shown in violent offenders which in turn are thought to predispose to violence.

Environmental Pollutants and Neurotoxicity

It has long been suspected that exposure to pollutants, particularly heavy metals that have neurotoxic effects, can lead to mild degrees of brain

impairment which in turn predisposes to antisocial and aggressive behavior. One of the best studies to date is that of Needleman et al. (1996) who assessed lead levels in the bones of 301 eleven-year-old schoolboys. Boys with higher lead levels were found to have significantly higher teacher ratings of delinquent and aggressive behavior, higher parent ratings of delinquent and aggressive behavior, and higher self-report delinquency scores. These findings do not occur in isolation: Similar links between lead levels and antisocial, delinquent behavior and aggression have been found in at least six other studies in several different countries (see Needleman et al. 1996 for a review). Furthermore, experimental exposure to lead during development increases aggressive behavior in hamsters (Delville 1999), thus suggesting a causal link.

Less strong to date, but nevertheless provocative, are findings with respect to manganese. At high levels, manganese has toxic effects on the brain and can damage the brain so much that it can even lead to Parkinson-like symptoms. Furthermore, it reduces levels of serotonin and dopamine, neurotransmitters that play a key role in brain communication (see later "Neurotransmitters" section). One study by Gottschalk et al. (1991) found that three different samples of violent criminals compared to controls had higher levels of manganese in their hair. On the other hand, not all studies have found this association (Schauss 1981). It may be that low levels of calcium intake interact with high manganese in predisposing to violent behavior because animal research indicates that the neurotoxic effect of manganese is particularly strong when the animal is deficient in calcium.

Hormones

Testosterone. Excellent reviews and discussions of the potential role played by testosterone in both animals and man can be found in Olweus (1987), Brain (1990), Archer (1991), and Susman and Ponirakis (1997). Animal research suggests that the steroid hormone testosterone plays an important role in the genesis and maintenance of some forms of aggressive behavior in rodents, and early exposure to testosterone has been found to increase aggression in a wide range of animal species (Brain 1990).

A key question generated by this literature is whether testosterone is involved in aggression and violent crime in man. Studies correlating questionnaire measures of aggression in normals to testosterone levels have generally produced weak or nonsignificant findings (Rubin 1987). Studies of violent incarcerated inmates on the other hand have been more consis-

tent in producing significant effects of moderate to large strength (Rubin 1987). This theme is reiterated by Archer (1991) in a rigorous and critical review of the literature, arguing effects are small or negligible when aggression is measured using personality inventories, but strong and significant when groups high and low on behavioral measures of aggression are compared. Five studies of prisoners reviewed by Archer resulted in substantial effects. Furthermore, female prisoners also show high testosterone levels, and interestingly this effect was found to be specific to females who committed unprovoked assault but not those who reacted violently when physically assaulted (Dabbs et al. 1988).

The critical question in this literature concerns whether testosterone-violence relationships are causal. Little doubt exists that castration decreases aggression in animals and administration of testosterone increases aggression. Few experimental studies have been conducted in humans, but there is nevertheless evidence of a causal relationship. Olweus et al. (1988) assessed their finding of higher testosterone in male adolescents with high levels of self-report aggression using path analysis and concluded that testosterone had causal effects on both provoked and unprovoked aggressive behavior. One study that comes close to such an ideal experiment is that of Wille and Beier (1989), who showed that ninety-nine castrated German sex offenders had a significantly lower recidivism rate eleven years postrelease (3 percent) compared to thirty-five noncastrated sex offenders (46 percent). There is in addition some limited evidence that less drastic methods of reducing testosterone levels such as administration of anti-androgens and progesterone derivatives have some effect in lowering violence and sexual aggression (Rubin 1987; Brain 1990; Archer 1991). One double-blind, crossover hormone replacement study administered testosterone to male adolescents with delayed puberty and found that medium doses increased aggressive behavior twenty-one months later (Susman and Ponirakis, 1997). It could be argued that extreme alterations in testosterone are not a good model for the less severe variability found in the general population, and that moderate changes of this hormone do not significantly influence aggression. On the other hand, Loosen, Purdon, and Pavlou (1994) found that mild reductions in testosterone in men were associated with reductions in outwardly directed anger, thus suggesting that mild changes in testosterone can modulate aggression.

Testosterone levels are in part heritable (Turner et al. 1986), and it is conceivable that the genetic predisposition to crime may in part be expressed through the hormonal system. On the other hand, it is also

known that environmental influences such as success in competition, the perception of winning, exposure to erotic films, and social dominance can increase circulating levels of testosterone (Brain 1990; Archer 1991). Clearly, links between testosterone and aggression are complex, and simplistic explanations of this link are probably incorrect. By the same token, it would be equally erroneous to discount the evidence for the role of hormones in influencing aggression merely because hormones are influenced by the environment. In this context, theoretical perspectives should take into account reciprocal influences between behavior and aggression and the roles hormones play in the regulation of arousal (Susman et al. 1996).

Cortisol. Cortisol (also called a glucocorticoid) is a hormone produced by a corticotropin-releasing factor (CRF), a peptide, which is in turn produced by a brain area called the hypothalamus, which regulates autonomic functions (like heart rate and skin conductance) and emotional responses. Individuals who are aroused or stressed show an increase in cortisol. As such, one might expect reduced cortisol levels in antisocials who, as we saw earlier, are thought to be relatively underaroused and fearless.

A number of studies in a wide variety of contexts have now shown that there is indeed a link between low resting cortisol and antisocial, aggressive behavior. McBurnett et al. (1991) found that boys with conduct disorder without comorbid anxiety disorder showed significantly lower concentrations of cortisol than did boys with CD and comorbid anxiety disorder. Cortisol has similarly been reported to be low in habitually violent incarcerated offenders (Virnkunnen 1985), in aggressive schoolchildren (Tennes and Kreye 1985), in adolescents with conduct problems (Susman and Petersen 1992), boys with disruptive behavior (McBurnett et al. 2000), disinhibited children, and boys with oppositional defiant disorder (van Goozen et al. 1998).

Taken together, these studies suggest that resting cortisol may play a nontrivial role in mediating antisocial, violent, and criminal behavior. In this context it supports and extends the findings described earlier for resting heart rate, confirming the finding of autonomic arousal deficits in antisocial populations. On the other hand, the link between antisocial behavior and cortisol *reactivity* to a stressor (that is, the change in cortisol before and after a stressful event) is less clear, with some studies finding decreased reactivity in antisocial children (for example, van Goozen et al. 1998) while other studies find increased reactivity (for example, Susman et al. 1997). Even accepting that there are differences in antisocial children in basal cortisol levels, it must be remembered that, as with many other

biological measures, cortisol is part of a dynamic system that is responsive to environmental changes and demands (Susman, Dorn, and Chrousos 1991; Susman and Ponirakis 1997). As such, integration with social and environmental factors must be a primary aim of future studies in this area.

Neurotransmitters

Basic neurotransmitters such as dopamine, norepinephrine, and serotonin form the basis to information processing and communication within the brain, and in this sense underlie all types of behavior, including sensation, perception, learning and memory, eating, drinking and, more controversially, antisocial behavior. Neurotransmitters are chemicals stored in the synaptic vesicles (small globules) of a communicating cell's axon; this axon carries the nerve impulse of the cell body to other cells. During cellular communication, these chemicals are discharged into the synaptic cleft (the space between two neurons) and are taken up by special receptors in the postsynaptic membrane of the recipient cell, initiating what is termed a postsynaptic potential. Thus, neurotransmitters form the basis to the transmission of information throughout the brain.

Paradoxically, the well-known and best-studied neurotransmitters (serotonin, dopamine, norepinephrine) account for only a small proportion of cell firing in the brain. For example, serotonin is thought to be the transmitter substance in fewer than 0.1 percent of brain synapses. Nevertheless, they are thought to be highly important in the context of brain and behavior; for example, serotonin, norepinephrine, and dopamine have been implicated in the etiology of major disorders such as schizophrenia and depression.

A meta-analysis of twenty-nine studies examined the relationship between norepinephrine, dopamine, and serotonin, and antisocial behavior (Raine 1993). Results indicated a relatively large effect size (-0.75) between reduced central serotonin and antisocial behavior, and a medium effect size of -0.41 between reduced norepinephrine and antisocial behavior, with no effect for dopamine. Subanalyses were conducted in an attempt to specify to which subgroups of antisocials these findings pertain. These analyses indicated that serotonin was lowest in antisocials with a history of alcohol abuse, borderline personality disorder, and violence, while cerebrospinal fluid norepinephrine was lowest in those with alcohol abuse, borderline personality disorder, and depression.

Increased impulsivity has been associated with lower serotonin, and there is some evidence that aggressive individuals who are impulsive have

particularly lower serotonin. Humans low in social class have also been found to have reduced serotonin, and it is possible that reductions in serotonin produced by a fall in dominance triggers impulsive aggression as a way of raising the individual in the dominance hierarchy. In evolutionary terms this would be adaptive as increased dominance gives greater access to food and sex, thus increasing the individual's ability to reproduce and pass on their genes. Yet again, poor diet may play a role. Diets low in, or otherwise blocking, the uptake of tryptophan or tyrosine (the precursors of serotonin and norepinephrine respectively) have been found to lower the levels of these transmitters in the brain (see Weisman 1986). In addition, even when returned to a normal diet, brain serotonin levels are never fully compensated (Timiras, Hudson, and Segall 1984). Poor nutrition possibly occurring in individuals of lower socioeconomic status (including poor dietary care during pregnancy) could conceivably influence neurotransmitter levels throughout life.

Neurogenetics

As outlined earlier, genetic studies are now beginning to progress from simply demonstrating that there is indeed a genetic basis to crime. A new generation of neurogenetic studies is now beginning to identify specific genes that give rise to abnormally aggressive behavior, often via abnormal functioning of specific neurotransmitters.

Some of the first clues have come from animal research where "knockout" mice have been genetically engineered to lack individual genes that normally give rise to specific neurotransmitters. One of the neurotransmitter systems implicated to date is nitric oxide. Mice that lack the gene essential for nitric oxide in the brain have been found to become highly aggressive, and furthermore agents which inhibit nitric oxide also lead to very aggressive behavior in normal mice (Demas et al. 1997).

Particularly interesting are findings on the knockout of the gene that codes for monoamine oxidase-A (MAOA), an enzyme that metabolizes brain neurotransmitters including serotonin. Adult mice who are deficient in MAOA are abnormally aggressive to other males and are much rougher and aggressive in their mating behavior with females (Cases et al. 1995; Nelson et al. 1995). These mice also showed very high levels of serotonin. What makes animal MAOA findings such as these more provocative is that they have also been found in humans. Brunner et al. (1993) have shown that a family in the Netherlands with a history of highly aggressive behavior have a mutation in the MAOA gene. They also showed impaired IQs, and like the MAOA mice, had very high levels of serotonin.

Animal findings such as these which are also found in humans are particularly interesting. Nevertheless, such genetic mutations affecting the MAOA gene are rare in humans and therefore it will be difficult to test whether Brunner's findings will be replicated in other samples. Furthermore, deletion of this produces high, not low, serotonin levels, a finding not consistent with the finding of *lower* serotonin in the cerebrospinal fluid of impulsive violent offenders. Nevertheless, it is not unlikely that the field of neurogenetics will produce multiple breakthroughs in the next few years on the molecular genetic basis of crime and violence.

POLICY IMPLICATIONS

One of the biggest and widely held myths in criminology research is that biology is destiny. Instead, the reality is that the biological bases of crime and violence are amenable to change through benign interventions. In the past fifty years, intervention programs have not been as successful in reducing crime and violence as had been hoped, and it is possible that part of their failure has been due to the fact that they have systematically ignored the biological component of the biosocial equation.

Brain damage and poor brain functioning have been shown to predispose to violence, and one possible source of this brain damage could be birth complications (Raine, Brennan, and Mednick 1997). The implication is that providing better pre- and postbirth health care to poor mothers may help reduce birth complications and thus reduce violence. Alternatively, rather than attempting to reduce birth complications, interventions could focus on the psychosocial half of the biosocial equation and attempt to reduce early maternal rejection which intensifies the effect of birth complications (Raine, Brennan, and Mednick 1994; Raine, Brennan, and Mednick 1997). Consideration might be given to multiple efforts across time to reduce maternal rejection by, for example, making parenting skills classes compulsory in high school to the next generation of mothers with unwanted pregnancies; providing more pre-birth visits from nurses to monitor both the pregnancy and the parent's attitude to the unborn baby; and providing home visits from pediatricians specifically to mothers who suffered birth complications to monitor the mother-infant bonding process, assess the physical and cognitive development of the infant, and provide appropriate remediation of cognitive and physical deficits which are known to follow from perinatal complications (Liu and Raine 1999; Raine and Liu 1998).

Another source of brain damage could be poor nutrition; and as has been seen earlier, there is evidence for a link between poor nutrition during pregnancy and later crime. Furthermore, cigarette and alcohol usage during pregnancy have been linked to later antisocial behavior (for example, Brennan, Grekin, and Mednick [1999]). In this context, Olds et al. (1998) in a methodologically strong randomized controlled trial showed that improving the quality of pregnant mothers' prenatal diet (among other factors) reduced offspring criminal and antisocial behavior fifteen years later. The intervention was also shown to improve the quality of the mothers' prenatal diet. Similarly, Lally, Mangione, and Honig (1988) showed that advice to pregnant women on good nutrition, health, and child rearing leads to a reduction in juvenile delinquency at age fifteen. These studies provide more support to the notion that nutrition plays a causal role in the development of childhood aggression, but future prevention trials that focus explicitly on the specific role of nutrition are required to further support the specific role of malnutrition.

It has been shown that low physiological arousal is the best-replicated biological correlate of antisocial behavior in child and adolescent samples. An important question from a prevention perspective concerns whether low arousal is amenable to change using noninvasive procedures. Recent findings from Mauritius suggest that it is. A nutritional, physical exercise, and educational enrichment from ages three to five resulted in increased psychophysiological arousal and orienting at age eleven compared to a matched control group (Raine et al. In press). Furthermore, a longer-term follow-up of these children to age seventeen showed a reduction in conduct disorder ($p < .01$) and motor excess scores ($p < .03$) in the experimental group compared to the control group (Raine et al. 1999). It should be noted, however, that these latter behavioral effects are sleeper effects; no effect of the intervention was noted on age-eleven aggression scores. Some initial reports have shown the possible efficacy of using biofeedback to increase physiological arousal in hyperactive children (Lubar 1989), while more recent pilot work has indicated that this technique shows some short-term behavioral improvement in children with conduct problems. Biofeedback training, as part of a larger multimodal treatment package, could conceivably help to reduce antisocial and violent behavior in adolescents.

The policy implications of biological research on crime also extend to the criminal justice system. One question raised by these and other studies is whether any of us have freedom of will in the strict sense of the term. If brain deficits make it more likely that a person will commit violence, and if the cause of the brain deficits was not under the control of the individ-

ual, then the question becomes whether or not that person should be held fully responsible for the crimes. Of course we have to protect society, and unless we can treat this brain dysfunction, we may need to keep violent offenders in secure conditions for the rest of their lives; but do they deserve to be executed given the early constraints on their free will? It could be argued that if an individual possesses risk factors that make him disproportionately more likely to commit violence, then he has to take responsibility for these predispositions. Just like an alcoholic who knows he suffers from the disease of alcoholism, the person at risk for violence needs to recognize his risk factors and take preventive steps to ensure that he does not harm others. These persons have risk factors, but they still have responsibility and they have free will.

This makes good, practical sense, but responsibility and self-reflection are not disembodied, ethereal processes but are rooted firmly in the brain. Patients who have damage to the ventromedial (lower) prefrontal cortex are known to become irresponsible, lack self-discipline, and fail to reflect on the consequences of their actions. The ability to take responsibility for one's actions is damaged in violent offenders. It can be argued that they are no longer able to reflect on their behavior as others do and take responsibility for their predispositions. It is not just that the brain mechanisms subserving responsibility and internal soul-searching are damaged in the violent offender and prevent him from taking action to rectify the causes of his violence.

Brain scan information is increasingly being used in capital cases, most frequently in the punishment stage of a trial as a mitigating circumstance against the death penalty, but it has also been used successfully in the guilt phase of a trial (D'Agincourt 1993). Nevertheless, use of brain imaging data in law courts is hotly debated, with some arguing against their use. Of course, brain scans are not diagnostic in that they do not perfectly predict who is violent and who is not. On the other hand, brain imaging data such as PET and fMRI constitute more direct indexes of brain function than EEG and neuropsychological test data, which have frequently been used in courts. Still, others worry that brain imaging data will be inappropriately used to "excuse" violent crimes.

Yet perhaps brain imaging research on violence is most troubling to some because it challenges the way we conceptualize crime. It questions our treatment of murderers in just the same way that we now look back 200 years and question the way in which the mentally ill were kept in shackles and chains, treated little better than animals. The history of civilization has shown that as time progresses, society becomes wiser and more humane.

Two hundred years from now, we may have reconceptualized recidivistic serious criminal behavior as a clinical disorder with its roots in early social, biological, and genetic forces beyond the individual's control (Raine 1993). Will we look back aghast at our current practices of execution and inhumane treatment of seriously violent offenders? Will we view this execution of prisoners as barbaric and unjustified, as we now view the burning of witches?

Biological research is beginning to give us new insights into what makes a violent criminal offender. It is hoped that these early findings may lead us to rethink our approach to violence and goad us into obtaining new answers to the causes and cures of crime while we continue to protect society.