Biosocial Studies of Antisocial and Violent Behavior in Children and Adults: A Review[1]

Adrian Raine[2,3]

Despite increasing knowledge of social and biological risk factors for antisocial and violent behavior, we know surprisingly little about how these two sets of risk factors interact. This paper documents 39 empirical examples of biosocial interaction effects for antisocial behavior from the areas of genetics, psychophysiology, obstetrics, brain imaging, neuropsychology, neurology, hormones, neurotransmitters, and environmental toxins. Two main themes emerge. First, when biological and social factors are grouping variables and when antisocial behavior is the outcome, then the presence of both risk factors exponentially increases the rates of antisocial and violent behavior. Second, when social and antisocial variables are grouping variables and biological functioning is the outcome, then the social variable invariably moderates the antisocial–biology relationship such that these relationships are strongest in those from benign home backgrounds. It is argued that further biosocial research is critical for establishing a new generation of more successful intervention and prevention research.

KEY WORDS: antisocial; violence; biosocial; psychophysiology; obstetric; genetics; neuropsychology.

INTRODUCTION

Over the past 50 years, important progress has been made in delineating replicable psychosocial risk factors for antisocial and violent behavior (Farrington, 2000; Hinshaw & Anderson, 1996; Loeber & Farrington, 1998; McCord, 2001; Rutter, Giller, & Hagell, 1998). Within the past 15 years, important progress has also been made in uncovering biological risk factors that predispose to antisocial behavior (Lahey, McBurnett, Loeber, & Hart, 1995; Moffitt, 1990a; Rutter et al., 1998; Susman & Finkelstein, 2001). Despite this progress, we know surprisingly little about how these different sets of risk factors *interact* in predisposing to antisocial behavior. Furthermore, although passing heuristic and theoretical references are frequently made to such interactional influences, there are

remarkably few investigators who are conducting serious empirical research on this interface in humans (Raine, Brennan, & Farrington, 1997).

The goal of this paper is to review the known facts on biosocial interaction effects in relation to antisocial and violent behavior in order to highlight this important yet underresearched field. The focus is placed on documenting *empirical* examples of interaction effects within the areas of genetics, psychophysiology, obstetrics, brain imaging, neuropsychology and neurology, hormones, neurotransmitters, and environmental toxins, rather than describing heuristic conceptualizations or theoretical perspectives. Only research on humans will be reviewed as perspectives from the animal literature are given in Miczek (2001), Suomi (1999), and Niehoff (1999). Although more complex transactional perspectives are potentially very important (Hinshaw & Anderson, 1996; Susman & Ponirakis, 1997), they are not the focus of this particular review because there are few empirical long-term outcome studies of serious antisocial and violent behavior.

The emphasis is placed on empirical knowledge because research in this area of antisocial behavior is sorely lacking hard empirical data on the nature of interactions, whereas in contrast speculation is rampant. By

---

[2]Department of Psychology, University of Southern California, Los Angeles, California.
[3]Address all correspondence to Adrian Raine, Department of Psychology, University of Southern California, Los Angeles, California 90089-1061; e-mail: raine@usc.edu.
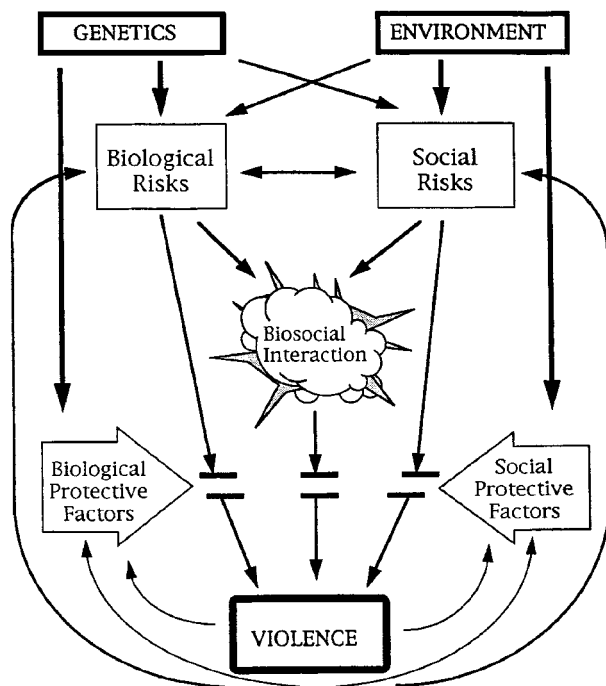
BIOSOCIAL MODEL OF VIOLENCE

Fig. 1. Heuristic biosocial model of violence.

documenting these findings at this point in time, the field will be better placed in the future to develop more sophisticated and specific biosocial theories of violence, which are empirically testable, as opposed to overly amorphous and all-encompassing. Nevertheless, good theory is also critical for scientific advance, and theoretical perspectives will be returned to in the conclusion section.

To serve as a heuristic guide to this review, Fig. 1 illustrates a simple biosocial model of violent behavior that highlights the key influences of genetic and environmental processes in giving rise to social and biological risk factors that both individually and interactively predispose to antisocial behavior. The model also incorporates social and biological protective factors, influences that will be touched upon briefly in this review. Inevitably, this model is overly simplistic, but it does provide a framework within which the research reviewed below can be viewed. It should also be noted that what constitutes a biological variable and what constitutes a social variable is open to question. There is much that is social about biological variables (e.g., head injuries leading to brain dysfunction are caused by the environment) and much that is biological about social variables (e.g., genetic factors, and their biological predispositions, contribute to bad

parenting). Although "biological" and "social" are in strict terms false dichotomies, they are retained here for illustrative purposes.

GENETICS

There is now clear evidence from twin studies, adoption studies, twins reared apart, and molecular genetic studies to support the notion that there are genetic influences on antisocial and aggressive behavior (Raine, 1993; Rowe, 2001; Rutter, 1997). The more challenging issue now concerns if and how genetic processes interact with environmental processes in predisposing to antisocial behavior. Twin studies find stronger evidence for heritability of antisocial behavior than adoption studies (Raine, 1993), and because interaction effects will influence heritability estimates from twin but not adoption designs, there is prima facie evidence that such interactions exist. Indeed, it is a truism that genetic processes need an environment in which to become expressed. As such, environmental changes will turn these genetic influences on and off across the life-span (Plomin & Rutter, 1998). Genetic factors likely give rise to biological risk factors for antisocial behavior such as low arousal, and if gene × environment interactions are found, this would suggest that interaction effects may well exist at the level of biological influences, a view that will be returned to later.

Gene by Environmental Interactions

One of the most striking examples of gene by environment interactions in genetic studies of crime is a cross-fostering analysis of petty criminality (Cloninger, Sigvardsson, Bohman, & von Knorring, 1982), results of which are illustrated in Fig. 2. Male Swedish adoptees ($N = 862$) were divided into four groups depending on the presence or absence of (a) a congenital predisposition (i.e., whether biological parents were criminal) and (b) a postnatal predisposition (how the children were raised by their adoptive parents). When both heredity and environmental predispositional factors were present, 40% of the adoptees were criminal compared to 12.1% with only genetic factors present, 6.7% for those with only a bad family environment, and 2.9% when both genetic and environmental factors were absent. The fact that the 40% rate for criminality when both biological and environmental factors are present is greater than the 18.8% rate given by a combination of "congenital only" and "postnatal only" conditions indicates that genetic and environmental factors interact. Further analyses indicated that occupational status of both biological and adoptive parents were
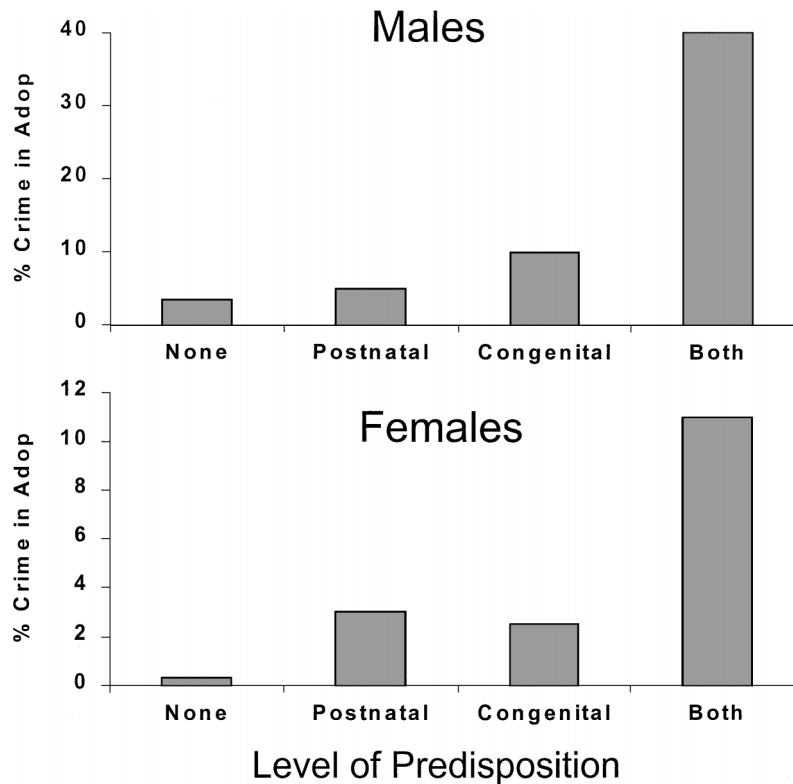
Fig. 2. Increased adult crime in adoptees when both genetic and environmental risk factors are present for both males (Cloninger et al., 1982) and females (Cloninger & Gottesman, 1987).

the main postnatal variables involved in this nonadditive interaction.

Cloninger and Gottesman (1987) later analyzed data for females to compare with the findings for males. 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 (see Fig. 2).

Evidence for gene × environmental interactions is also provided by Cadoret, Cain, and Crowe (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 × environment interaction in his analysis of adopted-away offspring of female prisoners, although this trend was only marginally significant ($p < .10$). Cadoret, Yates, Troughton, Woodworth, and Stewart (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, an adverse adoptive home environment was found to *interact* with adult antisocial personality in predicting increased aggression in the offspring, that is, a gene × environment interaction effect.

Gene by Environmental Correlation and the Moderating Effects of Demographics

A related but different concept is that of gene–environment correlation. An interesting example is provided in a study by Ge, Conger, Cadoret, and Neiderhiser (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 compared to the adopted away offspring of nonantisocial, nonsubstance abusing parents. This helps establish genetic transmission of childhood antisocial behavior, but 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 examine the interplay between genes and environment in this fashion further. More generally, there are likely to be future exciting developments with respect to identifying the specific genes, which give rise to the risk factors that shape criminal behavior.

An interaction of a different kind was also reported by Christiansen (1977) in an analysis of Danish twin data on criminality. Although overall he found significant heritability for crime, he also found that such heritability was greater in (a) those from high socioeconomic backgrounds and (b) those who were rural born. In other words, these sociodemographic variables moderated heritability for criminal behavior. This finding is of interest because, as will be seen below, it has also been found on several occasions with respect to psychophysiological and brain imaging studies. This suggests that stronger biology–antisocial findings can be found in social contexts where social predispositions to crime are minimized.

PSYCHOPHYSIOLOGY

Psychophysiological characteristics are prime candidates for possible interaction effects with psychosocial variables because, as the name implies, they tap the dynamic interface between psychological processes and physiological processes. They also have significant heritability and are likely to provide one of the routes through which genetic influences on antisocial behavior find expression.

Moderating Effect of Benign Home Backgrounds: The Social Push Perspective

A number of studies have found that psychophysiological factors show stronger relationships to antisocial behavior in those from benign social backgrounds that lack the classic psychosocial risk factors for crime. For example, although in general resting heart rate level is lower in antisocial individuals, it is a particularly strong characteristic of antisocial individuals from higher social classes (Raine & Venables, 1984b), those from privileged

middle class backgrounds attending private schools in England, (Maliphant, Hume, & Furnham, 1990), and those from intact but not broken homes (Wadsworth, 1976). One prospective study found that low resting heart rate at age 3 years related to aggression at age 11 years in Creole Mauritians from high but not low social classes (Raine, Reynolds, Venables, & Mednick, 1997). Increased heart rate variability (indexing increased vagal tone and related to reduced heart rate level) was found to relate positively to aggression in young adults who had not been victims of violence, but not in those who were victims of violence; in this study moderating effects were not observed for resting heart rate (Scarpa, Romero, Fikretoglu, Bowser, & Wilson, 1999). Similarly, with respect to electrodermal classical conditioning, reduced skin conductance activity characterizes antisocial adolescents from high but not low social classes (Raine & Venables, 1981), criminals without a childhood history broken by parental absence and disharmony (Hemming, 1981), and "privileged" (high SES) offenders who commit crimes of evasion (Buikhuisen, Bontekoe, Plas-Korenhoff, & Van Buuren, 1984). In children, reduced skin conductance orienting to neutral tones at age 3 years is related to aggressive behavior at age 11 years, but only in those from high social class backgrounds (Raine, Reynolds, Venables, & Mednick, 1997). Similarly in adults, schizoid criminals from intact but not broken early home environments show reduced SC orienting (Raine, 1987).

One explanation for this pattern of results is the "social push" hypothesis. Under this perspective, where an antisocial child *lacks* social factors that "push" or predispose him/her to antisocial behavior, then biological factors may more likely explain antisocial behavior (Mednick, 1977; Raine & Venables, 1981). In contrast, social causes of criminal behavior may be more important explanations of antisociality in those exposed to adverse early home conditions. This is not to say that antisocial children from adverse home backgrounds will never evidence biological risk factors for antisocial and violent behavior—they clearly will. Instead, the argument is that in such situations the link between antisocial behavior and biological risk factors will be weaker (relative to antisocial children from benign social backgrounds) because the social causes of crime camouflage the biological contribution. Conversely, in the case of antisocial children from benign home backgrounds, the "noise" created by social influences on antisocial behavior are minimized, allowing the biology–antisocial behavior relationship to shine through.

The importance of this perspective is that biological researchers would be well-advised to take into account psychosocial risk factors if they want to uncover stronger biology–antisocial relationships. Reversing the approach,

psychosocial researchers may find stronger links between psychosocial influences and antisocial behavior in children who lack biological risk factors for antisocial behavior, although to date no one appears to have tested this proposition.

Interactions Between Psychophysiological and Social Risk Factors

These examples of biosocial interactions are unusual in that the psychophysiological variable is the dependent variable. In these cases, the question that is being asked is whether antisocial individuals with good or bad social backgrounds differ in psychophysiological functioning. When antisocial behavior becomes the dependent variable however, a different conceptual question is posed, namely, is antisocial behavior greatest in those with both social and biological risk factors? Put another way, do psychophysiological factors interact with social factors in explaining the outcome of antisocial behavior?

There have been fewer examples of studies addressing this question in the psychophysiological literature. One particularly thorough analysis is given by Farrington (1997) with respect to statistically significant interactions between resting heart rate and psychosocial variables. Boys with low resting heart rates are more likely to become violent adult offenders if they also have a poor relationship with their parent, and if they come from a large family (Farrington, 1997). Similarly, boys with low heart rates are especially likely to be rated as aggressive by their teachers if their mother was pregnant as a teenager, if they come from a low SES family, or if they were separated from a parent by age 10 (Farrington, 1997).

Protective Factors

Until recently, nothing was known about biological factors that can protect against antisocial outcome, but there is now some evidence that heightened autonomic arousal may play such a role. Adolescent antisocial behavior is a risk factor for later criminal behavior, but some antisocial adolescents desist from further antisocial behavior. These individuals, compared to both antisocial boys who become criminal and never antisocial controls show increased electrodermal and cardiovascular arousal and orienting in an English sample (Raine, Venables, & Williams, 1995, 1996). In an independent extension of these findings, Brennan et al. (1997) found that Danish boys who had a criminal father but who did not become criminal themselves were characterized by increased electrodermal and cardiovascular orienting compared to both nonantisocial offspring of noncriminal controls, and criminal offspring

with criminal fathers. This latter study is particularly interesting because it illustrates how the social risk factor of having a criminal father moderates the protective role of heightened autonomic functioning in relation to crime outcome.

Interpretation of Reduced Autonomic Activity in Antisocial Children

Why should low autonomic activity predispose to antisocial and criminal behavior? There are at least two main theoretical interpretations. Fearlessness theory indicates that low levels of arousal are markers for low levels of fear (Raine, 1993). A fearlessness interpretation of low arousal levels assumes that subjects are not actually at "rest," but that instead the rest period of psychophysiological testing represents a mildly stressful paradigm. Low arousal during this period is taken to indicate lack of anxiety and fear. Lack of fear would predispose to antisocial and violent behavior because such behavior (e.g., fights and assaults) requires a degree of fearlessness to execute, whereas lack of fear, especially in childhood, would help explain poor socialization because 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 (Fowles, Kochanska, & Murray, 2000; Kagan, 1994; Scarpa, Raine, Venables, & Mednick, 1997b).

A second theory explaining reduced arousal is stimulation-seeking theory (Eysenck, 1977; Quay, 1965; Raine, 1993; Raine, Reynolds, Venables, Mednick, & Farrington, 1998). This theory argues that low arousal represents an unpleasant physiological state, and that antisocial individuals 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. Stimulation-seeking and fearlessness theories may be complementary perspectives in that a low level of arousal may predispose to crime because it produces some degree of fearlessness, and also because it encourages antisocial stimulation-seeking. Indeed, behavioral measures of stimulation-seeking and fearlessness, both taken at age 3 years in a large sample, predict to aggressive behavior at age 11 years (Raine, Reynolds, et al., 1998). The combined effect of these two influences may be more important in explaining antisocial behavior than either influence taken alone.

In contrast to arousal deficits, explanations of reduced orienting activity have centered on attentional and prefrontal dysfunction theory. Raine and Venables (1984a)

proposed an attention deficit hypothesis in which it was argued that antisocial individuals were characterized by a fundamental deficit in the ability to allocate attentional resources to environmental events. In discussing this perspective, Fowles (1993) alternatively suggested that there may be two attentional deficits in antisocial individuals, one deficit with respect to attending to neutral stimuli, and other deficit with respect to the anticipation of aversive events.

Reduced skin conductance orienting has also been interpreted with respect to a prefrontal dysfunction hypothesis of antisocial behavior (Raine, 1997). Briefly, this perspective argues that damage to the prefrontal region of the brain leads to psychophysiological abnormalities (reduced orienting and arousal) that predispose to traits and characteristics (e.g., stimulation-seeking, disinhibition, attention deficits), which in turn predispose to antisocial behavior. Support for this model stems from research showing that the prefrontal cortex is involved in the generation of skin conductance orienting responses (Damasio, Tranel, & Damasio, 1990; Williams et al., 2000) and is also involved in arousal regulation and stress responsivity (see Raine, 1997 for further details). Reduced autonomic arousal is in turn associated with increased stimulation-seeking (Gatzke, Raine, Loeber, Steinhauer, & Stouthamer-Loeber, 2002), fearlessness (Raine, 1993), and disinhibition (Fowles et al., 2000; Scarpa, Raine, Venables, & Mednick, 1997a), traits that in turn have been associated with antisocial behavior (Raine, Reynolds, et al., 1998).

OBSTETRIC FACTORS

Of all the subfields of biological research on antisocial behavior, obstetric influences show the most compelling evidence for biosocial interactions, with at least 11 studies from five different countries finding evidence for statistical interactions. These obstetric studies fall into three domains: minor physical anomalies, prenatal nicotine exposure, and birth complications.

Pregnancy Complications

Minor Physical Anomalies (MPAs)

At least six studies have found an association between increased MPAs and increased antisocial behavior in children (Raine, 1993). Minor physical anomalies have been associated with disorders of pregnancy and are thought to be a marker for fetal neural maldevelopment toward the end of the first 3 months of pregnancy. As such, they may
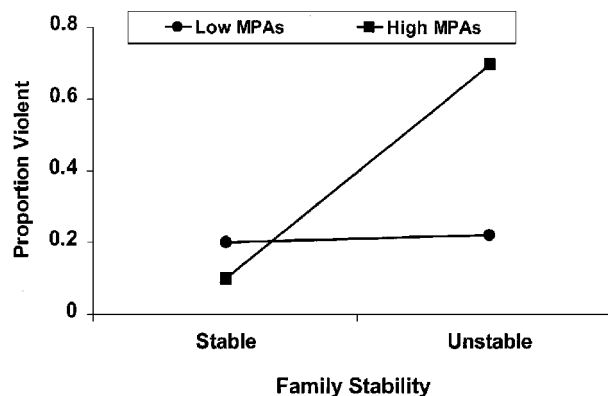

Fig. 3. Interaction between family instability and high minor physical anomalies in the prediction of adult violence (Mednick & Kandel, 1988).

be viewed as an indirect marker of abnormal brain development. MPAs are relatively minor physical abnormalities consisting of such features as low-seated ears, adherent ear lobes, and a furrowed tongue. Although MPAs may have a genetic basis, they may also be caused by environmental factors acting on the fetus such as anoxia, bleeding, and infection (Guy, Majorski, Wallace, & Guy, 1983).

At least three studies have found that MPAs interact with social factors in predicting antisocial and violent behavior. Mednick and Kandel (1988) assessed MPAs in a sample of 129 12-year-old boys seen by an experienced pediatrician. MPAs were found to be related to violent offending as assessed 9 years later when subjects were aged 21 years, although not to property offenses without violence. However, as illustrated in Fig. 3, when subjects were divided into those from unstable, nonintact homes and those from stable homes, a biosocial interaction was observed. MPAs only predicted violence in those individuals raised in unstable home environments. Similarly, Brennan, Mednick, and Raine (1997) found that those with both MPAs and family adversity had especially high rates of adult violent offending within a sample of 72 male offspring of psychiatrically ill parents. This interaction was again confirmed by Pine, Shaffer, Schonfeld, and Davies (1997) who found that MPAs in 7-year-olds combined with environmental risk in predisposing to conduct disorder at age 17. These findings are similar to those on birth complications reported above; in both cases 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 of MPAs to violence, Arseneault, Tremblay, Boulerice, Seguin, and Saucier (2000) found that MPAs assessed at age 14 predicted to violent delinquency at age 17 in 170 males, but not to nonviolent delinquency. In this study, effects were independent of family adversity.

Nicotine Exposure

The effect of fetal exposure to alcohol in increasing risk for conduct disorders is well known (e.g., Fast, Conry, & Loock, 1999; Olson et al., 1997; Streissguth, Barr, Bookstein, Sampson, & Olson, 1999), but recently a spate of studies has established beyond reasonable doubt a significant link between smoking during pregnancy and later conduct disorder and violent offending (see Raine, in press, for a review). Three of these studies have also observed interactions between nicotine exposure and psychosocial variables in the prediction of later violent offending, and are impressive in terms of their size, the prospective nature of data collection, long-term outcome, and control for third factors such as antisocial behavior in the parents, other drug use, and low social class. Brennan, Grekin, and Mednick (1999) using a birth cohort of 4,169 males found a twofold increase in adult violent offending in the offspring of mothers who smoked 20 cigarettes a day, and also found a dose–response relationship between increased number of cigarettes smoked and increased violence. However, a *fivefold* increase in adult violence was found when nicotine exposure was combined with exposure to delivery complications—there was no increase in violence in those who were nicotine-exposed but lacking delivery complications. Brennan et al. (1999) observed that effects were specific to persistent offending and did not apply to adolescent-limited offending. Similarly, Rasanen et al. (1999) found a twofold increase in violent criminal offending at age 26 in the offspring of women who smoked during pregnancy. In addition, nicotine exposure lead to an 11.9-fold increase in recidivistic violence when combined with single-parent family, and a 14.2-fold increase when combined with teenage pregnancy, single-parent family, unwanted pregnancy, and developmental motor lags. Again, odds ratios were stronger for recidivistic violence than for violence in general or property offending. Gibson and Tibbetts (2000) also found that maternal smoking interacted with parental absence in predicting early onset of offending in a U.S. sample.

Maternal smoking during pregnancy may be an important contributory factor to the brain deficits that have been found in adult offenders. Animal research has clearly demonstrated the neurotoxic effects of two constituents of cigarette smoke—carbon monoxide (CO) and nicotine (see Olds, 1997 for a detailed review). Prenatal nicotine exposure even at relatively low levels disrupts the development of the noradrenergic neurotransmitter system and disrupts cognitive functions (Levin, Wilkerson, Jones, Christopher, & Briggs, 1996). Reduction of noradrenergic functioning caused by smoking would be expected to disrupt sympathetic nervous system activity, consistent with

evidence outlined earlier for reduced sympathetic arousal in antisocial individuals (Raine, 1996). Pregnant rats exposed to nicotine have offspring with an enhancement of cardiac M2-muscarinic cholinergic receptors that *inhibit* autonomic functions (Slotkin, Epps, Stenger, Sawyer, & Seidler, 1999). This would help to explain the well-replicated finding of *low* resting heart rate in antisocial individuals outlined above (Raine, 1993).

Birth Complications

Several studies have shown that babies who suffer birth complications are more likely to develop conduct disorder, delinquency, and commit impulsive crime and violence in adulthood when other psychosocial risk factors are present. Specifically, obstetric factors *interact* with psychosocial risk factors in relation to adult violence. Werner (1987) found that birth complications interacted with a disruptive family environment (maternal separation, illegitimate child, marital discord, parental mental health problems, paternal absence) in predisposing to delinquency. Similarly, Raine, Brennan, and Mednick (1994) prospectively assessed birth complications and maternal rejection at age 1 year in 4,269 live male births in Copenhagen, Denmark. Birth complications significantly interacted with maternal rejection of the child in predicting to violent offending at age 18 years (see Fig. 4, upper half). Only 4% of the sample had both birth complications and maternal rejection, but this small group accounted for 18% of all the violent crimes committed by the entire sample.

In this latter study, the 4,269 babies were followed up to age 34 when outcome for violent crime was reassessed (Raine, Brennan, & Mednick, 1997). It was found 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 attempt to abort the fetus were the key aspects of maternal rejection found to interact with birth complications in predisposing to violence.

This finding from Denmark has recently been replicated in four other countries (Sweden, Finland, Canada, U.S.A.) in the context of a variety of psychosocial risk factors. Piquero and Tibbetts (1999) in a prospective longitudinal study of 867 males and females from the Philadelphia Collaborative Perinatal Project found that those with both pre/perinatal disturbances and a disadvantaged familial environment were much more likely to become adult
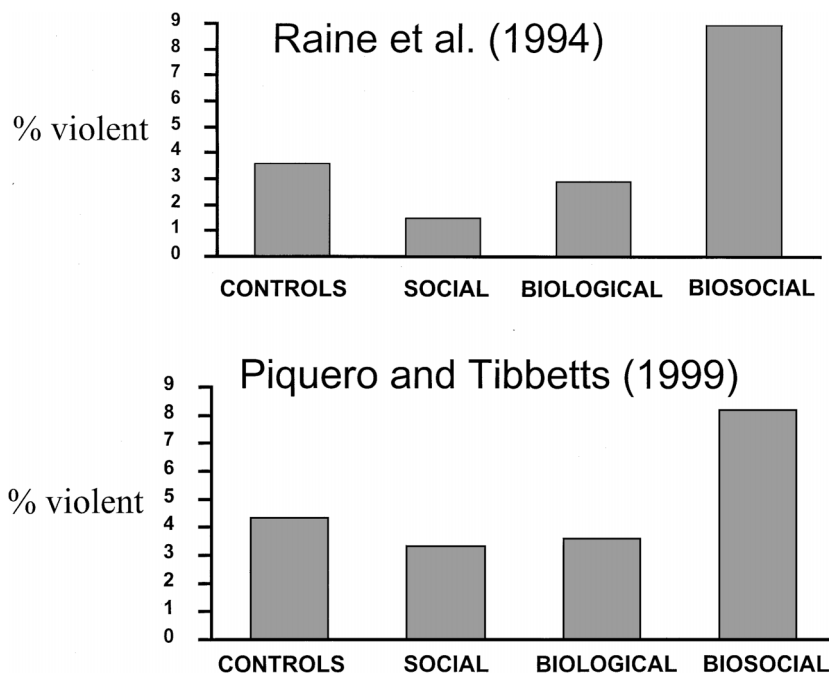
Fig. 4. Interaction between birth complications and negative family outcome in the prediction of adult violence in both Danish (Raine et al., 1994) and U.S. (Piquero & Tibbetts, 1999) samples.

violent offenders (see Fig. 4, lower half). Similarly, pregnancy complications interacted with poor parenting in predicting adult violence in a large Swedish sample of 7,101 men (Hodgins, Kratzer, & McNeil, 2001). In a Canadian sample of 849 boys, Arsenault, Tremblay, Boulerice, and Saucier (in press) found an interaction between increased serious obstetric complications and family adversity in raising the likelihood of violent offending at age 17 years. In a Finnish sample perinatal risk interacted with being an only child in raising the odds of adult violent offending by a factor of 4.4 in a sample of 5,587 males (Kemppainen, Jokelainen, Jaervelin, Isohanni, & Raesaenen, 2001). On the other hand, being an only child is not obviously linked to psychosocial adversity, and the meaning of this interaction requires further elucidation.

A fifth study reported by Brennan, Mednick, and Mednick (1993) and also from Denmark showed that birth complications interacted with parental mental illness in predicting violent crime in the male offspring (see Fig. 5). On the other hand, no interaction between perinatal insult and family adversity was found for a smaller sample of German children ($N = 322$) where outcome was restricted to follow-up at age 8 years (Laucht et al., 2000). This last failure may be due to the fact that neurological deficits stemming from birth complications may particularly influence the more severe outcome of life-course

persistent antisocial behavior rather than the more common outcome of child antisocial behavior (Moffitt, 1993; Moffitt & Caspi, 2001). Indeed, several of the above studies find that interaction effects involving birth complications and family factors show evidence of linkage to what may be broadly termed life-course persistent violent behavior rather than adolescent-limited antisocial
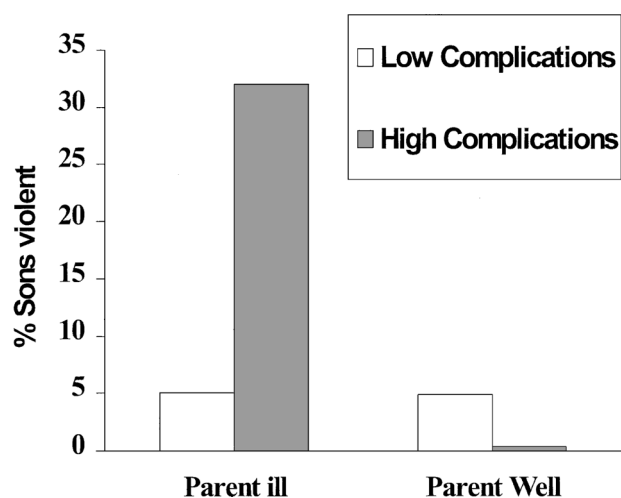

Fig. 5. Interaction between birth complications and parental mental illness in the prediction of violent crime in the offspring (Brennan et al., 1993).

behavior. In addition to these interactions with psychosocial variables, low Apgar scores at birth have been found to interact with maternal smoking in the prediction of adult violent offending (Gibson & Tibbetts, 1998).

Birth complications such as anoxia (lack of oxygen), forceps delivery, and preeclampsia (hypertension leading to anoxia) are thought to contribute to brain damage, and they may be just one of a number of early sources of brain dysfunction observed in child and adult antisocial groups. On the other hand, as indicated above, birth complications may not by themselves predispose to crime, but instead may require the presence of negative environmental circumstance to trigger later adult crime and violence. Furthermore, although they are likely to contribute to prefrontal damage, their effects would not be specific to this brain area but would impact multiple brain sites, including the hippocampus. Interestingly, recent brain imaging studies have shown that the hippocampus shows abnormal functioning in murderers (Raine, Buchsbaum, & LaCasse, 1997), shows structural abnormalities in psychopaths (Laakso et al., 2001) and is particularly susceptible to anoxia.

BRAIN IMAGING

Only two brain imaging studies of antisocial, violent behavior appear to have tested for interaction effects with social influences. Nevertheless they are described here because brain imaging is a growing and increasingly influential subarea within biological research on antisocial behavior and because their findings complement those from other areas.

Positron Emission Tomography (PET)

Previous research has indicated that violent offenders have reduced functioning of the prefrontal cortex (Raine, 1993; Volkow et al., 1995). One PET (positron emission tomography) study addressed the issue of how psychosocial deficits moderate the relationship between prefrontal dysfunction and violence (Raine, Stoddard, Bihrle, & Buchsbaum, 1998). A sample of murderers were divided into those who came from relatively good home backgrounds and those who came from relatively bad homes. 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 to normal controls, deprived murderers showed relatively good prefrontal functioning, whereas nondeprived murderers showed significantly reduced prefrontal function-

ing. In particular, murderers from good homes had a 14.2% reduction in the functioning of the right orbitofrontal cortex; damage to this brain area results in reduced fear conditioning as well as personality and emotional deficits that parallel criminal psychopathic behavior, or what Damasio and colleagues have termed "acquired sociopathy" (Damasio, 1994). These results extend findings from several psychophysiological studies showing especially reduced autonomic functions in those from benign home backgrounds, and again suggest that biology–violence relationships are potentiated in those lacking psychosocial risk factors for violence.

Functional Magnetic Resonance Imaging (fMRI)

A second brain imaging study found an interaction of a different type in seeking an answer to a different type of question. Although the relationship between physical child abuse and violence is well established (Lewis, Pincus, Bard, & Richardson, 1988; Tarter, Hegedus, Winsten, & Alterman, 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. The study in question (Raine, Park, et al., 2001) asked two main questions: (a) 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? (b) what characterizes those who experience severe physical abuse but who refrain from serous violence? Four groups of participants were recruited from the community: (i) nonviolent controls who had not suffered abuse, (ii) severe physical child abuse only (i.e., had suffered severe physical or sexual abuse in the first 11 years, but were not violent), (iii) serious violence only (violence that either caused bodily injury or trauma, or were life-threatening acts), and (iv) severely abused, seriously violent offenders. All underwent functional magnetic resonance imaging (fMRI) while performing a visual/verbal working memory task. Results showed that violent offenders who had suffered severe child abuse show reduced right hemisphere functioning, particularly in the right temporal cortex. Abused individuals who had refrained 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 indicate that a biological risk factor (initial right hemisphere dysfunction), when combined with a psychosocial risk factor (severe early physical abuse) predisposes to serious violence. They also suggest that

relatively good right hemisphere functioning *protects* against violence in physically abused children.

NEUROPSYCHOLOGY AND NEUROLOGY

Neuropsychological and neurological deficits, especially those associated with executive function deficits, are a reasonably well-established risk factor for antisocial behavior in children, adolescents, and adults (Moffitt, 1990a; Morgan & Lilienfeld, 2000; Raine, 1993). Although most neuropsychological and neurological research has not explored the interaction between neuropsychological dysfunction and psychosocial factors in predisposing to violence, a few studies are beginning to suggest that this may be a promising avenue for future research.

Prospective, Longitudinal Findings

Lewis, Lovely, Yeager, and Femina (1989) in a follow-up study of 15-year-old juvenile delinquents found that although having only neurocognitive deficits was associated with an average of 2.1 adult violent offenses, and only experiencing child abuse was associated with an average of 1.9 adult offenses, the combination of three neurocognitive indicators combined with child abuse was associated with an average of 5.4 violent offenses in adulthood. Consequently, the combination of neurocognitive and psychosocial risk factors was associated with particularly high rates of violence in adulthood.

Other prospective longitudinal research is broadly consistent with this finding. Moffitt (1990b) reports that boys with both low neuropsychological performance and family adversity had aggression scores four times higher than boys with either adversity only or neuropsychological deficits only. Similarly, Raine, Brennan, Mednick, and Mednick (1996) found that those with both early neuromotor deficits (including birth complications) and unstable family environments later went on to have higher rates of teenage behavior problems and adult criminal and violent offending compared to those with only social or only biological risk factors. The biosocial group with both sets of risk factors accounted for 70.2% of all violence committed by the entire cohort.

One nonprospective study of 64 violent juvenile offenders took a different approach by dividing subjects into members of gangs versus nongang members (Sparling & Cohen, 1997). Nongang members compared to gang members were much more likely to have neurobehavioral deficits such as history of head injury (57.1% vs. 11.1%) and intermittent explosive disorder (71.4% vs. 11.4%). These pilot findings are intriguing because they suggest that the social variable of belonging to a gang (and the consequent social-affiliative nature of these networks) moderates neurobehavioral–violent relationships. Conceivably, violent offenders with neurobehavioral deficits are less able to sufficiently modulate their aggressive tendencies for good functioning in gangs, which value controlled, proactive aggression.

Protective Influence of Stable Home Environment

Although it is reasonable to hypothesize that the negative effects of biological risk factors in predisposing to antisocial behavior may be ameliorated by the benefits of a positive home environment, there appear to be few tests of this hypothesis. Streissguth, Barr, Kogan, and Bookstein (1996) in a study of 473 individuals with fetal alcohol syndrome found that a stable home environment protected the child from an antisocial outcome. An intriguing case study from Spain of a man who had an iron spike pass through his head, selectively destroying the prefrontal cortex, showed that unlike the case of Phineas Gage, this individual did not have an outcome over the next 60 years of antisocial or criminal behavior (Mataro et al., 2001). Mataro et al. (2001) concluded that prefrontal damage can be followed by stable psychosocial functioning, but a different interpretation can also be made. It is intriguing to note that the subject in question had wealthy parents who owned a family business in which he would be employed for the rest of his life, and that his fiancé (a childhood sweetheart) stood by him after the accident and married him, producing two good children and a family which, in the words of one of the children "protected" him throughout his life. It can be argued that this individual did not develop antisocial behavior and psychosocial dysfunction because his family environment buffered him from these negative outcomes. Without such psychosocial support, a very different outcome may have resulted.

Social Demands of Adolescence Overloading Executive Functions

One interactional neuropsychological model of antisocial behavior concerns the notion that the social and executive function demands of late adolescence overload the late developing prefrontal cortex, giving rise to prefrontal dysfunction and a lack of inhibitory control over antisocial, violent behavior that peaks at this age (Raine, in press). Prior to adolescence, children live in relatively structured environments where complex, life-changing planning and decision-making is not the norm. In contrast,

late adolescence is a stage in life where enormous social demands are being placed on the rapidly growing teenager, a load that calls on resources of the frontal cortex and its associated executive functions. Such adolescents need to regulate, control, and inhibit a growing sex drive, juggle the threats and challenges to their social status that arise within their peer groups, deal with the complexities of relations with the opposite sex, and increasingly sustain attention at school to maximize chances of academic success, and plan and organize for a future career.

The prefrontal cortex bears the burden of this magnified cognitive load that requires multiple executive functions—sustained attention, behavioral flexibility to changing contingencies, working memory, self-regulation and inhibition, abstract decision-making, planning and organization. Yet this processing load occurs at a time when the prefrontal cortex is still maturing, with myelination of the frontal cortex continuing into the 20s and beyond. A minority of individuals with early damage to or dysfunction of the prefrontal cortex would be particularly likely to suffer an information overload during this time period, resulting in further dysfunction of the prefrontal cortex, less regulatory control, and further lifelong antisocial behavior. Others with a late-maturing but intact prefrontal cortex may be antisocial during childhood and adolescence, but with further maturation of the frontal lobes in early adulthood may eventually discontinue their antisocial behavior. Still others may have frontal dysfunction, but may be protected from antisocial behavior by having more social support or fewer social-transitional demands placed on them, as was argued above in the Spanish head trauma case. Yet another group of late-onset offenders (Hamalainen & Pulkkinen, 1996; Ishikawa, Raine, Lencz, Bihrle, & LaCasse, 2001) may have neither significant executive function deficits nor antisocial behavior until early adulthood when life stressors at this time overload a prefrontal cortex with latent functional impairments.

This theoretical perspective would lead to a number of predictions. In addition to expecting that those with prefrontal dysfunction and poor executive functions would be predisposed to antisocial behavior, this outcome would be most likely in those with a less structured, less stable psychosocial environment. Second, those with significant executive functions who resist becoming antisocial would be expected to have a particularly well-structured, protective social environment or alternatively high intelligence that minimizes the impact of executive function deficits. Third, those with initial early antisocial behavior who desist in life would be expected to show initially poor executive functions but later better executive functioning.

HORMONES, NEUROTRANSMITTERS, AND TOXINS

Hormones

Research on links between hormones and antisocial, aggressive behavior illustrates the complexities of biology–behavior relationships, and clearly demonstrates the influence of the social context on biological functioning. Detailed reviews of the bidirectional relationship between hormones and behavior and of the influence of social context on hormones may be found in Dabbs (1992), Mazur and Booth (1999), Susman (1993), Susman and Ponirakis (1997), and Tremblay et al. (1997).

Although links between high testosterone and self-report measures of aggression are relatively weak (Archer, 1991), there is now convincing evidence from a wide number of *behavioral* studies for a link between high testosterone and increased aggressive and violent behavior (see reviews by Archer, 1991; Dabbs, 1992; Harris, 1999; Mazur & Booth, 1999; Raine, 2002). Particularly persuasive are experimental randomized, placebo-controlled, crossover trials in normal men that show that testosterone administration increases aggression (Pope et al., 2000). Nevertheless, it also appears that although aggression–testosterone links are well-established with respect to adult aggressive and violent behavior, this relationship may be absent, or even reversed, with respect to aggression during childhood (Susman & Ponirakis, 1997; Tremblay et al., 1997).

An understanding of why the aggression–testosterone relationship washes out in childhood may be gained from consideration of social influences on testosterone. It is well-established that high testosterone is associated with both high dominance and high socioeconomic status, and while experience of success increases testosterone, failure reduces it (Dabbs, 1992; Mazur & Booth, 1999). Aggressive children are more likely to be rejected by their peers in school (Dodge, Lochman, Harnish, & Bates, 1997), and it may be this social ostracization and failure in academia that artificially reduces their otherwise high testosterone levels. This interpretation is supported by two pieces of evidence. First, Tremblay et al. (1997) report that 13-year-old boys who are both physically tough *and* who are well-liked have high testosterone levels. Second, although Tremblay et al. (1997) found that aggressive boys have low testosterone at ages 13 and 14, follow-up at age 16 after 30% had dropped out of school show them to have substantially higher testosterone than nonaggressive boys. It may be that aggression–testosterone links are found in violent offenders during adulthood because they are better able to use their

aggression to raise their dominance status within their antisocial subcultures and achieve some degree of social success.

Despite the clear evidence that contextual and environmental influences alter both testosterone and cortisol, few researchers have tested for biosocial interactions in the way that they have been tested for psychophysiological and obstetric factors. Dabbs and Morris (1990) found that in low SES subjects, those with high testosterone had higher levels of childhood and adulthood delinquency, whereas these effects were not found for high SES subjects. On the other hand, risk ratios for some measures of antisocial behavior (military AWOL, marijuana use, many sex partners) were equally high in both SES groups, and consequently these initial findings should be viewed with caution. Nevertheless, Scarpa et al. (1999) found that children who both gave large cortisol responses to a provocation task and who also had been physically abused had the highest aggression scores. Both of these hormonal studies are therefore similar in that biology–antisocial relationships were more marked in those from negative environments (low SES, abuse), and lie in contrast to studies measuring psychophysiological and brain imaging risk factors (low heart rate, low skin conductance, increased vagal tone, low prefrontal activity), which find antisocial–psychophysiology relationships strongest in those from *benign* home backgrounds. It remains to be seen whether further hormone studies find stronger hormone–antisocial links in those from negative rather than positive home backgrounds.

Neurotransmitters and Toxins

Neurotransmitter and toxin research is also beginning to provide evidence of interactions with social and environmental processes. Moffitt et al. (1997) found that although violent offenders had higher blood serotonin levels than controls, those with both high blood serotonin and a conflicted family background were over three times more likely to become violent by age 21 compared to men with only high serotonin or only conflicted family background. Similarly, Masters, Hone, and Doshi (1998) in an analysis of violent crime rates in 1,242 counties in the United States found a three-way interaction between environmental lead or manganese exposure, high population density, and alcoholism rates, with highest rates of violence in counties with high densities, exposure to toxins, and alcoholism. As with research on hormones, there is a dearth of empirical data to support or refute a biosocial interaction hypothesis of antisocial and violent behavior with respect to toxins, and further tests of this proposition are required.

CONCLUSIONS AND RECOMMENDATIONS

Summarizing the key findings of this review, there has in recent years been growing evidence for replicable interactions between biological and social factors in relation to antisocial and violent behavior. In the last review of this area (Brennan & Raine, 1997), only nine studies could be drawn on to illustrate biosocial interactions. In the current review, 39 studies have been outlined that illustrate interactions between biological and social factors in relation to antisocial and violent behavior. Clearly, there is recent, growing evidence that social and biological processes do interact in predisposing to antisocial behavior. To date, the best-replicated biosocial effect appears to consist of birth complications interacting with negative home environments in predisposing to adult violence, and there is also evidence that this effect particularly characterizes life-course persistent antisocial behavior.

Although these findings give reason to take biosocial perspectives on antisocial behavior seriously, study counts and documentation of findings by themselves will not advance knowledge within a field. The establishment of interaction effects is not an end process, but merely the end of a beginning in understanding antisocial behavior. Interaction effects need to be explained with respect to their underlying mechanisms. For example, birth complications consistently interact with negative home environments in predisposing to adult violence, and this effect seems specific to violence, and to violence with an early onset—but why? What are the processes that operate in negative home environments, which trigger the deleterious effects of birth complications? Do birth complications predispose to violence by mild impairment to brain functioning, or does family adversity instead predispose to both birth complications such as pregnancy-induced hypertension and also childhood conduct disorder? That is, are birth complications merely a marker of a third factor and are by themselves unrelated to violence? Although these questions are difficult to answer, the next generation of biosocial research needs to go beyond simple interaction effects and research the fundamental mechanisms and processes underlying the interactions.

Theoretical perspectives are needed to help guide future research on biosocial interaction effects. Moffitt (1993) has argued that life-course persistent offending is a product of early interactions between neuropsychological and family factors and as such would predict early biosocial interaction effects in relation to life-course persistent offending, a prediction that seems to be generally supported by the data. In addition, the social push perspective argues that the link between antisocial behavior and biological risk factors will be weaker in adverse

homes because the social predispositions to crime camouflage the biological contribution. In contrast, in benign home environments the role of biological predispositions can be more easily detected. The prefrontal dysfunction–executive overload theory outlined above is more specific in specifying conditions under which antisocial behavior will result as a function of the interplay between an immature prefrontal cortex and psychosocial demands. Ultimately, biosocial perspectives of antisocial behavior will be maximally helpful when a complementary and balanced theoretical–empirical approach is developed.

In analyzing the pattern of interaction effects observed above, there are two main conclusions that can be drawn and that can guide future hypothesis-testing. When the biological and social factors are the grouping variable and antisocial behavior is the outcome, then the presence of both risk factors appears to increase rates of antisocial and violent behavior. When social and antisocial variables are the grouping variables and biological functioning is the outcome, then the social variable invariably (but not always) moderates the antisocial–biology relationship such that these relationships are strongest in those from benign home backgrounds. A question for future biosocial studies is whether these two patterns of findings, which ask different questions, can be substantiated, or whether different patterns emerge. In addition, studies conducted to date are relatively simplistic, and the question of whether these biosocial interactions are carried by conditions comorbid with antisocial behavior such as hyperactivity need to resolved (Hinshaw, Lahey, & Hart, 1993).

There are also statistical as well as theoretical difficulties facing future research into the biosocial bases of antisocial behavior. Statistically speaking, interactions and moderator effects are notoriously difficult to obtain due to problems of measurement error (McClelland & Judd, 1993), with power in the type of studies reviewed above being less than 20% of experimental studies. As such, Type II error is frequently more of a problem than Type I error in biosocial studies. Even where no statistically significant effect is observed, it would be advisable for researchers to report the means and standard deviations for groups on the biological variable broken down by the social variable (e.g., high vs. low social class) and compute effect sizes so that future meta-analyses can be conducted with the ensuing benefit of increased power.

A practical barrier to future biosocial research is the simple fact that few psychosocial researchers take into account biological variables, and few biological researchers take into account social variables. Even when they do, psychosocial variables are often conceptualized as covariates or nuisance variables by biological researchers rather than as moderators. Researchers need to face the fact that

there will likely be diminishing returns from further research on currently known risk factors and that more will be gained from examining interactions between risk factors. Indeed, researchers who do not test for interaction effects that may well exist, and who obtain nonsignificant main effects, will erroneously conclude that the variables in question are of no etiological significance. Although the measurement of biological variables poses significant challenges for psychosocial researchers, low resting heart rate is the best-replicate biological correlate of antisocial behavior in child and adolescent samples (Raine, 1993), takes only 1 minute to asses, and can be measured cheaply and easily using a stethoscope, digital heart rate meter, or by taking a pulse manually. Incorporating even this simple biological measure into psychosocial longitudinal research would be a step in the right direction.

Finally, biosocial studies of antisocial behavior should not be restricted to risk and protective factors but also need to consider prevention implications at two levels. First, environmental manipulations can be used to alter biological risk factors. For example, an environmental enrichment at ages 3–5 years using a randomized, stratified design resulted in significant increases in psychophysiological arousal and attention 8 years later at age 11 years (Raine, Venables, et al., 2001). Second, psychosocial influences may moderate the effects of a prevention program on antisocial behavior. As one example, Olds et al. (1998) demonstrated that a prenatal and early postnatal early health prevention program was more successful in reducing delinquency at age 15 years in unmarried, low SES mothers than less disadvantaged mothers. Biosocial research on risk and protective factors themselves should clearly be a priority for establishing a new generation of more biosocially-informed prevention and intervention programs.

ACKNOWLEDGMENTS

REFERENCES

Archer, J. (1991). The influence of testosterone on human aggression. *British Journal of Clinical Psychology*, *82,* 1–28.

Arseneault, L., Tremblay, R. E., Boulerice, B., & Saucier, J. F. (2002). Obstetrical complications and violent delinquency: Testing two developmental pathways. *Child Development, 73,* 496–508.

Arseneault, L., Tremblay, R. E., Boulerice, B., Seguin, J. R., & Saucier, J. F. (2000). Minor physical anomalies and family adversity as risk factors for violent delinquency in adolescence. *American Journal of Psychiatry*, *157,* 917–923.

Brennan, P. A., Grekin, E. R., & Mednick, S. A. (1999). Maternal smoking during pregnancy and adult male criminal outcomes. *Archives of General Psychiatry*, *56,* 215–219.

Brennan, P. A., Mednick, B. R., & Mednick, S. A. (1993). Parental psychopathology, congenital factors, and violence. In S. Hodgins (Ed.), *Mental disorder and crime* (pp. 244–261). Thousand Oaks: Sage.

Brennan, P. A., Mednick, S. A., & Raine, A. (1997). Biosocial interactions and violence: A focus on perinatal factors. In A. Raine, P. A. Brennan, D. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 163–174). New York: Plenum.

Brennan, P. A., & Raine, A. (1997). Biosocial bases of antisocial behavior: Psychophysiological, neurological, and cognitive factors. *Clinical Psychology Review*, *17,* 589–604.

Brennan, P. A., Raine, A., Schulsinger, F., Kirkegaard-Sorensen, L., Knop, J., Hutchings, B., et al. (1997). Psychophysiological protective factors for male subjects at high risk for criminal behavior. *American Journal of Psychiatry*, *154,* 853–855.

Buikhuisen, W., Bontekoe, E. H., Plas-Korenhoff, C., & Van Buuren, S. (1984). Characteristics of criminals: The privileged offender. *International Journal of Law and Psychiatry*, *7,* 301–313.

Cadoret, R. J., Cain, C. A., & Crowe, R. R. (1983). Evidence for gene-environment interaction in the development of adolescent antisocial behavior. *Behavior Genetics*, *13,* 301–310.

Cadoret, R. J., Yates, W. R., Troughton, E., Woodworth, G., & Stewart, M. A. (1995). Genetic-environmental interaction in the genesis of aggressivity and conduct disorders. *Archives of General Psychiatry*, *52,* 916–924.

Christiansen, K. O. (1977). A preliminary study of criminality among twins. In S. A. Mednick & K. O. Christiansen (Eds.), *Biosocial bases of criminal behavior* (pp. 89–108). New York: Gardner Press.

Cloninger, C. R., & Gottesman, I. I. (1987). Genetic and environmental factors in antisocial behavior disorders. In S. A. Mednick, T. E. Moffitt, & S. A. Stack (Eds.), *The causes of crime: New biological approaches*. Cambridge: Cambridge University Press.

Cloninger, C. R., Sigvardsson, S., Bohman, M., & von Knorring, A. L. (1982). Predisposition to petty criminality in Swedish adoptees: II. Cross-fostering analysis of gene-environmental interactions. *Archives of General Psychiatry*, *39,* 1242–1247.

Crowe, R. R. (1974). An adoption study of antisocial personality. *Archives of General Psychiatry*, *31,* 785–791.

Dabbs, J. M. (1992). Testosterone measurements in social and clinical psychology. *Journal of Social and Clinical Psychology*, *11,* 302–321.

Dabbs, J. M., & Morris, R. (1990). Testosterone, social class, and antisocial behavior in a sample of 4,462 men. *Psychological Science*, *1,* 209–211.

Damasio, A. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: GP Putnam's Sons.

Damasio, A. R., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. *Behavioural Brain Research*, *41,* 81–94.

Dodge, K. A., Lochman, J. E., Harnish, J. D., & Bates, J. E. (1997). Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. *Journal of Abnormal Psychology*, *106,* 37–51.

Eysenck, H. J. (1977). *Crime and personality* (3rd ed.). St. Albans: Paladin.

Farrington, D. P. (1997). The relationship between low resting heart rate and violence. In A. Raine, P. A. Brennan, D. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 89–105). New York: Plenum.

Farrington, D. P. (2000). Psychosocial predictors of adult antisocial personality and adult convictions. *Behavioral Sciences and the Law*, *18,* 605–622.

Fast, D. K., Conry, J., & Loock, C. A. (1999). Identifying Fetal Alcohol Syndrome among youth in the criminal justice system. *Journal of Developmental and Behavioral Pediatrics*, *20,* 370–372.

Fowles, D. C. (1993). *Electrodermal activity and antisocial behavior: Empirical findings and theoretical issues* (pp. 223–237). New York: Plenum.

Fowles, D. C., Kochanska, G., & Murray, K. (2000). Electrodermal activity and temperament in preschool children. *Psychophysiology*, *37,* 777–787.

Gatzke, L., Raine, A., Loeber, R., Steinhauer, S., & Stouthamer-Loeber, M. (2002). *Low autonomic arousal, sensation seeking, and antisocial behavior*. Manuscript under review.

Ge, X., Conger, R. D., Cadoret, R. J., & Neiderhiser, J. M. (1996). The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology*, *32,* 574–589.

Gibson, C. L., & Tibbetts, S. G. (1998). Interaction between maternal cigarette smoking and Apgar scores in predicting offending behavior. *Psychological Reports*, *83,* 579–586.

Gibson, C. L., & Tibbetts, S. G. (2000). A biosocial interaction in predicting early onset of offending. *Psychological Reports*, *86,* 509–518.

Guy, J. D., Majorski, L. V., Wallace, C. J., & Guy, M. P. (1983). The incidence of minor physical anomalies in adult male schizophrenics. *Schizophrenia Bulletin*, *9,* 571–582.

Hamalainen, M., & Pulkkinen, L. (1996). Problem behavior as a precursor of male criminality. *Development and Psychopathology*, *8,* 443–455.

Harris, J. A. (1999). Review and methodological considerations in research on testosterone and aggression. *Aggression and Violent Behavior*, *4,* 273–291.

Hemming, J. H. (1981). Electrodermal indices in a selected prison sample and students. *Personality and Individual Differences*, *2,* 37–46.

Hinshaw, S. P., & Anderson, C. A. (1996). Conduct and oppositional defiant disorders. In E. J. Mash & R. A. Barkley (Eds.), *Child psychopathology* (pp. 113–149). New York: Guilford Press.

Hinshaw, S. P., Lahey, B. B., & Hart, E. L. (1993). Issues of taxonomy and comorbidity in the development of conduct disorder. *Development and Psychopathology*, *5,* 31–49.

Hirono, N., Mega, M. S., Dinov, I. D., Mishkin, F., & Cummings, J. L. (2000). Left frontotemporal hypoperfusion in associated with aggression in patients with dementia. *Archives of Neurology*, *57,* 861–866.

Hodgins, S., Kratzer, L., & McNeil, T. F. (2001). Obstetric complications, parenting, and risk of criminal behavior. *Archives of General Psychiatry*, *58,* 746–752.

Ishikawa, S. S., Raine, A., Lencz, T., Bihrle, S., & LaCasse, L. (2001). Autonomic stress reactivity and executive functions in successful and unsuccessful criminal psychopaths from the community. *Journal of Abnormal Psychology*, *110,* 423–432.

Kagan, J. (1994). *Galen's prophecy: Temperament in human nature*. New York: Basic Books.

Kemppainen, L., Jokelainen, J., Jaervelin, M. R., Isohanni, M., & Raesaenen, P. (2001). The one-child family and violent criminality: A 31-year follow-up study of the northern Finland 1966 birth cohort. *American Journal of Psychiatry*, *158,* 960–962.

Laakso, M. P., Vaurio, O., Koivisto, E., Savolainen, L., Eronen, M., Aronen, H. J., et al. (2001). Psychopathy and the posterior hippocampus. *Behavioural Brain Research*, *118,* 187–193.

Lahey, B. B., McBurnett, K., Loeber, R., & Hart, E. L. (1995). Psychobiology. In G. P. Sholevar (Ed.), *Conduct disorders in children and adolescents* (pp. 27–44). Washington, DC: American Psychiatric Press.

Laucht, M., Esser, G., Baving, L., Gerhold, M., Hoesch, I., Ihle, W., et al. (2000). Behavioral sequelae of perinatal insults and early family adversity at 8 years of age. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39,* 1229–1237.

Levin, E. D., Wilkerson, A., Jones, J. P., Christopher, N. C., & Briggs, S. J. (1996). Prenatal nicotine effects on memory in rats: Pharmacological and behavioral challenges. *Developmental Brain Research*, *97,* 207–215.

Lewis, D. O., Lovely, R., Yeager, C., & Femina, D. D. (1989). Toward a theory of the genesis of violence: A follow-up study of delinquents.

Journal of the American Academy of Child and Adolescent Psychiatry, *28*, 431–436.

Lewis, D. O., Pincus, J. H., Bard, B., & Richardson, E. (1988). Neuropsychiatric, psychoeducational, and family characteristics of 14 juveniles condemned to death in the United States. *American Journal of Psychiatry*, *145*, 584–589.

Loeber, R., & Farrington, D. P. (1998). Never too early, never too late: Risk factors and successful interventions for serious and violent juvenile offenders. *Studies on Crime and Crime Prevention*, *7*, 7–30.

Maliphant, R., Hume, F., & Furnham, A. (1990). Autonomic nervous system (ANS) activity, personality characteristics, and disruptive behaviour in girls. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *31*, 619–628.

Masters, R. D., Hone, B., & Doshi, A. (1998). Environmental pollution, neurotoxicity, and criminal violence. In J. Rose (Ed.), *Environmental toxicology: Current developments* (pp. 13–48). New York: Gordon and Breach.

Mataro, M., Jurado, M. A., Garcia-Sanchez, C., Barraquer, L., Costa-Jussa, F. R., & Junque, C. (2001). Long-term effects of bilateral frontal brain lesion 60 years after injury with an iron bar. *Archives of Neurology*, *58*, 1139–1142.

Mazur, A., & Booth, A. (1999). The biosociology of testosterone in men. In D. Franks & S. Smith (Eds.), *Mind, brain, and society: Toward a neurosociology of emotion* (Vol. 5, pp. 311–338). Stamford, CT: JAI Press.

McClelland, G. H., & Judd, C. M. (1993). Statistical difficulties of detecting interactions and moderator effects. *Psychological Bulletin*, *114*, 376–390.

McCord, J. (2001). Psychosocial contributions to psychopathy and violence. In A. Raine & J. Sanmartin (Eds.), *Violence and psychopathy* (pp. 141–170). New York: Kluwer Academic/Plenum.

Mednick, S. A. (1977). A bio-social theory of the learning of law-abiding behavior. In S. A. Mednick & K. O. Christiansen (Eds.), *Biosocial bases of criminal behavior*. New York: Gardner Press.

Mednick, S. A., & Kandel, E. S. (1988). Congenital determinants of violence. *Bulletin of the American Academy of Psychiatry and the Law*, *16*, 101–109.

Miczek, K. A. (2001). Research on animal aggression: Emerging successes for understanding determinants of human violence. In M. E. Carroll & J. B. Overmier (Eds.), *Animal research and human health: Advancing human welfare through behavioral science* (pp. 41–61). Washington, DC: American Psychological Association.

Moffitt, T. E. (1990a). The neuropsychology of delinquency: A critical review. In M. Tonry & N. Morris (Eds.), *Crime and justice: A review of research* (pp. 99–169). Chicago: University of Chicago Press.

Moffitt, T. E. (1990b). Juvenile delinquency and Attention Deficit Disorder: Boys' developmental trajectories from age 3 to age 15. *Child Development*, *61*, 893–910.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, *100*, 674–701.

Moffitt, T. E., & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Development and Psychopathology*, *13*, 355–375.

Moffitt, T. E., Caspi, A., Fawcett, P., Brammer, G. L., Raleigh, M., Yuwiler, A., & Silva, P. (1997). Whole blood serotonin and family background relate to male violence. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 231–249). New York: Plenum.

Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, *20*, 113–156.

Niehoff, D. (1999). *The biology of violence*. New York: Free Press.

Olds, D. (1997). Tobacco exposure and impaired development: A review of the evidence. *Mental Retardation and Developmental Disabilities Research Reviews*, *3*, 257–269.

Olds, D., Henderson, C. R. J., Cole, R., Eckenrode, J., Kitzman, H., Luckey, D., et al. (1998). Long-term effects of nurse home visitation on children's criminal and antisocial behavior: 15-year follow-up of a randomized controlled trial. *JAMA*, *280*, 1238–1244.

Olson, H. C., Streissguth, A. P., Sampson, P. D., Barr, H. M., Bookstein, F. L., & Thiede, K. (1997). Association of prenatal alcohol exposure with behavioral and learning problems in early adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 1187–1194.

Pine, D. S., Shaffer, D., Schonfeld, I. S., & Davies, M. (1997). Minor physical anomalies: Modifiers of environmental risks for psychiatric impairment? *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 395–403.

Piquero, A., & Tibbetts, S. (1999). The impact of pre/perinatal disturbances and disadvantaged familial environment in predicting criminal offending. *Studies on Crime and Crime Prevention*, *8*, 52–70.

Plomin, R., & Rutter, M. (1998). Child development, molecular genetics, and what to do with genes once they are found. *Child Development*, *69*, 1223–1242.

Quay, H. C. (1965). Psychopathic personality as pathological stimulation-seeking. *American Journal of Psychiatry*, *122*, 180–183.

Raine, A. (1987). Effect of early environment on electrodermal and cognitive correlates of schizotypy and psychopathy in criminals. *International Journal of Psychophysiology*, *4*, 277–287.

Raine, A. (1993). *The psychopathology of crime: Criminal behavior as a clinical disorder*. San Diego: Academic Press.

Raine, A. (1996). Autonomic nervous system activity and violence. In D. M. Stoff & R. B. Cairns (Eds.), *Aggression and violence: Genetic, neurobiological, and biosocial perspectives* (pp. 145–168). Mahwah, NJ: Erlbaum.

Raine, A. (1997). Antisocial behavior and psychophysiology: A biosocial perspective and a prefrontal dysfunction hypothesis. In D. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 289–304). New York: Wiley.

Raine, A. (2002). The biological basis of crime. In J. Q. Wilson & J. Petersilia (Eds.), *Crime: Public policies for crime control* (pp. 43–74). San Francisco: ICS Press.

Raine, A. (2002). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior. *Journal of Child Psychology and Psychiatry*, *43*, 417–434.

Raine, A., Brennan, P., & Farrington, D. P. (1997). Biosocial bases of violence: Conceptual and theoretical issues. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 1–20). New York: Plenum.

Raine, A., Brennan, P., & Mednick, S. A. (1994). Birth complications combined with early maternal rejection at age 1 year predispose to violent crime at age 18 years. *Archives of General Psychiatry*, *51*, 984–988.

Raine, A., Brennan, P., & Mednick, S. A. (1997). Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: Specificity to serious, early-onset violence. *American Journal of Psychiatry*, *154*, 1265–1271.

Raine, A., Brennan, P., Mednick, B., & Mednick, S. A. (1996). High rates of violence, crime, academic problems, and behavioral problems in males with both early neuromotor deficits and unstable family environments. *Archives of General Psychiatry*, *53*, 544–549.

Raine, A., Buchsbaum, M., & LaCasse, L. (1997). Brain abnormalities in murderers indicated by positron emission tomography. *Biological Psychiatry*, *42*, 495–508.

Raine, A., Park, S., Lencz, T., Bihrle, S., LaCasse, L., Widom, C. S., et al. (2001). Reduced right hemisphere activation in severely abused violent offenders during a working memory task: An fMRI study. *Aggressive Behavior*, *27*, 111–129.

Raine, A., Reynolds, C., Venables, P. H., & Mednick, S. A. (1997). Biosocial bases of aggressive behavior in childhood. In A. Raine,

P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 107–126). New York: Plenum.

Raine, A., Reynolds, C., Venables, P. H., Mednick, S. A., & Farrington, D. P. (1998). Fearlessness, stimulation-seeking, and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. *Archives of General Psychiatry*, *55,* 745–751.

Raine, A., Stoddard, J., Bihrle, S., & Buchsbaum, M. (1998). Prefrontal glucose deficits in murderers lacking psychosocial deprivation. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *11,* 1–7.

Raine, A., & Venables, P. H. (1981). Classical conditioning and socialization—a biosocial interaction. *Personality and Individual Differences*, *2,* 273–283.

Raine, A., & Venables, P. H. (1984a). Electrodermal nonresponding, antisocial behavior, and schizoid tendencies in adolescents. *Psychophysiology*, *21,* 424–433.

Raine, A., & Venables, P. H. (1984b). Tonic heart rate level, social class and antisocial behaviour in adolescents. *Biological Psychology*, *18,* 123–132.

Raine, A., Venables, P. H., Dalais, C., Mellingen, K., Reynolds, C., & Mednick, S. A. (2001). Early educational and health enrichment at age 3–5 years is associated with increased autonomic and central nervous system arousal and orienting at age 11 years: Evidence from the Mauritius Child Health Project. *Psychophysiology*, *38,* 254–266.

Raine, A., Venables, P. H., & Mednick, S. A. (1997). Low resting heart rate at age 3 years predisposes to aggression at age 11 years: Evidence from the Mauritius Child Health Project. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36,* 1457–1464.

Raine, A., Venables, P. H., & Williams, M. (1995). High autonomic arousal and electrodermal orienting at age 15 years as protective factors against criminal behavior at age 29 years. *American Journal of Psychiatry*, *152,* 1595–1600.

Raine, A., Venables, P. H., & Williams, M. (1996). Better autonomic conditioning and faster electrodermal half-recovery time at age 15 years as possible protective factors against crime at age 29 years. *Developmental Psychology*, *32,* 624–630.

Rasanen, P., Hakko, H., Isohanni, M., Hodgins, S., Jarvelin, M. R., & Tiihonen, J. (1999). Maternal smoking during pregnancy and risk of criminal behavior among adult male offspring in the northern Finland 1996 birth cohort. *American Journal of Psychiatry*, *156,* 857–862.

Rowe, D. C. (2001). *Biology and crime*. Los Angeles: Roxbury Publishing.

Rutter, M. L. (1997). Nature-nurture integration: The example of antisocial behavior. *American Psychologist*, *52,* 390–398.

Rutter, M., Giller, H., & Hagell, A. (1998). *Antisocial behavior by young people.* Cambridge: Cambridge University Press.

Scarpa, A., Raine, A., Venables, P. H., & Mednick, S. A. (1997a). Heart rate and skin conductance in behaviorally inhibited Mauritian children. *Journal of Abnormal Psychology*, *106,* 182–190.

Scarpa, A., Raine, A., Venables, P. H., & Mednick, S. A. (1997b). Heart rate and skin conductance in behaviorally inhibited Mauritian children. *Journal of Abnormal Psychology*, *106,* 182–190.

Scarpa, A., Romero, N., Fikretoglu, D., Bowser, F. M., & Wilson, J. W. (1999). *Community violence exposure and aggression: Biosocial interactions*. Paper presented at the meeting of the American Society of Criminology, Toronto, Canada.

Slotkin, T. A., Epps, T. A., Stenger, M. L., Sawyer, K. J., & Seidler, F. J. (1999). Cholinergic receptors in heart and brainstem of rats exposed to nicotine during development: Implications for hypoxia tolerance and perinatal mortality. *Brain Research*, *113,* 1–12.

Sparling, Y. A., & Cohen, R. (1997). Neurobehavioral influences on propensity for juvenile violence. *Journal of Neuropsychiatry*, *9,* 134–135.

Streissguth, A. P., Barr, H. M., Bookstein, F. L., Sampson, P. D., & Olson, H. C. (1999). The long-term neurocognitive consequences of prenatal alcohol exposure: A 14-year study. *Psychological Science*, *10,* 186–190.

Streissguth, A. P., Barr, H. H., Kogan, J., & Bookstein, F. L. (1996). *Understanding the occurrence of secondary disabilities in clients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE)*. Seattle: Washington Publication Services.

Suomi, S. J. (1999). Developmental trajectories, early experiences, and community consequences: Lessons from studies with rhesus monkeys. In D. P. Keating & C. Hertzman (Eds.), *Developmental health and the wealth of nations: Social, biological, and educational dynamics* (pp. 185–200). New York: Guilford.

Susman, E. J. (1993). Psychological, contextual, and psychobiological interactions: A developmental perspective on conduct disorder. *Development and Psychopathology*, *5,* 181–189.

Susman, E. J., & Finkelstein, J. W. (2001). Biology, development, and dangerousness. In G. F. Pinard & L. Pagani (Eds.), *Clinical assessment of dangerousness: Empirical contributions* (pp. 23–46). New York: Cambridge University Press.

Susman, E. J., & Ponirakis, A. (1997). Hormones—context interaction and antisocial behavior in youth. In A. Raine, P. A. Brennan, D. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 251–269). New York: Plenum.

Tarter, R. E., Hegedus, A. M., Winsten, N. E., & Alterman, A. I. (1984). Neuropsychological, personality, and familial characteristics of physically abused delinquents. *Journal of the American Academy of Child Psychiatry*, *23,* 668–674.

Tremblay, R. E., Schaal, B., Boulerice, B., Arseneault, L., Soussignan, R., & Perusse, D. (1997). Male physical aggression, social dominance, and testosterone levels at puberty: A developmental perspective. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 271–291). New York: Plenum.

Volkow, N. D., Tancredi, L. R., Grant, C., Gillespie, H., Valentine, A., Mullani, N., et al. (1995). Brain glucose metabolism in violent psychiatric patients: A preliminary study. *Psychiatry Research: Neuroimaging*, *61,* 243–253.

Wadsworth, M. E. J. (1976). Delinquency, pulse rate and early emotional deprivation. *British Journal of Criminology*, *16,* 245–256.

Werner, E. E. (1987). Vulnerability and resiliency in children at risk for delinquency: A longitudinal study from birth to young adulthood. In J. D. Burchard & S. N. Burchard (Eds.), *Primary prevention of psychopathology* (pp. 16–43). Newbury Park, CA: Sage.

Widom, C. S. (1997). Child abuse, neglect, and witnessing violence. In D. M. Stoff, J. Breiling, & J. D. Maser (Eds.), *Handbook of antisocial behavior* (pp. 159–170). New York: Wiley.

Williams, L. M., Brammer, M. J., Skerrett, D., Lagopolous, J., Rennie, C., Kozek, K., et al. (2000). The neural correlates of orienting: An integration of fMRI and skin conductance orienting. *Neuroreport*, *11,* 3011–3015.