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George B. Palermo MD MScCrim PhD

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Biological and Environmental Correlates of Aggressive Behavior

GEORGE B. PALERMO, MD, MScCrim, PhD
University of Nevada Medical School, Henderson, Nevada

Aggressive behavior is influenced by the interplay of multiple genes that operate on brain biochemical pathways, neurotransmitters, and hormones, producing a certain psychological predisposition that, in contact with environmental risks within or outside the family, is conducive to antisocial aggressive behavior. This brief review of the scientific research shows that such behavior is the result of these predisposing, but not causal, factors. The importance of the individual’s unique decisional capacity in such behavior is a question that requires further research.

KEYWORDS aggression, neuroimaging, MAOA gene, neurotransmitters, amygdala

INTRODUCTION

Aggressive behavior and violence against others are not only ubiquitous but have been present since the beginning of humankind. Their presence reached high peaks in different historical periods and, in any of those periods, the violence was believed to be the worst. Therefore, they should be viewed not only from an evolutionary perspective but as relative to the historical period taken into consideration.

At present, aggressive violent behaviors in their diverse manifestations are at high levels in society. Frequently, it is present within the family—between husband and wife, parents and children, children and parents, and between other cohabitants. This is not new, as history is replete with consanguineous crimes, and spousal killing and filicides are the frequent subject of research. In 2002, the United States Bureau of Justice Statistics reported

Address correspondence to George B. Palermo, University of Nevada Medical School, 2169 Silent Echoes Drive, Henderson, NV 89044. E-mail: palermogb@juno.com
that about 20% of the people in the Western world are victimized by violent and nonviolent behavior every year. However, generally

fewer that ten percent of the families in any community account for more than 50% of that community's criminal offenses . . . . Parents may transmit to their child a genetic liability for aggression and simultaneously provide an environment of violent, abusive maltreatment that is symptomatic of the parents' genetic liability for aggression” (Moffitt, 2005, pp. 533, 538).

Nevertheless, although maltreated children are at risk of later criminality, the majority of them do not show delinquent or criminal acting out in adolescence or adulthood.

Animal studies on aggression have stimulated the curiosity of scientists regarding any correlation to human behavior, promoting genetic and biological studies in an attempt to understand human violence. Early genetic studies in humans and animals did not support a definitive association among impulsivity, aggression, and reduced 5-hydroxytryptamine (5HT) activity. Indeed, the Maudsley rat study demonstrated that a strain of rat (MR) showed high impulsivity, a low level of inhibition, and a higher limbic level of 5HT (Sudack & Maas, 1964). Recently, Janssen et al. (2005) reported that mice genetically altered in their MAOA system show extreme aggressive behavior, killing their mates.

Researchers are interested in finding whether, in addition to the usual contributory factors of psycho-social-economic variables, there is a constitutional predisposition to aggressive behavior. Understanding the biology and the environmental risks behind human conduct is also important in the attempt to predict and prevent aggression and violence. The importance of genetics and of the genome, with its biological information, is evident, and it seems logical to think that the resultant phenotype is a product of the interaction between the genotype and the environment. More than half a century ago, Carmichael (1947) and, two decades later, Hirsch (1967), both psychologists, rejected the idea that the environment is the only determinant of human behavior, and historian Degler (1991) asserted that behaviorists did not give importance to the inherited determinants of behavior. It has been suggested that that genetic factors may exert some influence on criminogenic traits that, in turn, might affect an individual’s propensity to engage in antisocial behaviors (Beaver, DeLisi, Vaughn, Wright, 2008). Behavior, however, is looked upon as an interplay of genetics, biology, and the environment. Individuals seem to be predisposed to react to the environment according to their basic biological predisposition. This obviously raises the eternal problem of nature versus nurture. In this regard, Willhoit (1976) aptly stated,

We are genetically programmed to learn and persist in certain kinds of behaviors much more readily than in the case with other possible
behaviors . . . This does not necessarily mean that a particular behavior is inheritable, rather that heredity significantly affects the probability of its development (p. 1010).

Much later, Moffitt and Caspi (2006) supported the interaction between genes and the family-wide environment and “in such interactions the effect of an environmental risk may be even larger than previously reported among the subgroup of individuals having a vulnerable genotype” (p. 52).

**HERITABILITY OF A BIOLOGICAL PREDISPOSITION TO AGGRESSIVE ACTING OUT**

In an attempt to determine the heritability of a genetic/biological predisposition to psychophysiological factors that may influence aggressive acting out, various family, twin, and adoption studies have been conducted. As Moffitt (2005) reported, “Once genetically influenced behavior has brought a person into contact with an environment, the environment may have unique causal effects of its own, cutting off the opportunity to develop alternative prosocial behaviors, promoting the persistence of antisocial behavior, and exacerbating its seriousness” (p. 540). However, it is unlikely that genes completely determine a behavioral disorder. Most probably, under natural conditions, the general effects of genetics and the environment are evenly divided. Bad parenting might contribute to antisocial behavior, and such parenting might be under some degree of genetic influence because parents’ personality traits, known to be genetically influenced, also influence styles of parenting (Spinath & Conner, 2003).

**Twin Studies**

To determine the genetic influences or the environmental causes of aggression and violence, scholars depend on twin and adoption studies, because from these studies either the genetic relatedness or the environmental causation can be inferred (Tremblay, Hartup, & Archer, 2005). Twin studies make use of the fact that monozygotic (MZ)—or identical—twins share a 100% genetic identity, whereas dizygotic (DZ)—or fraternal—twins do not. By calculating the concordance rate for criminal behaviors (criminal versus noncriminal), scholars have found that MZ twins have a higher concordance rate for crime than DZ twins. These findings are supportive of the heritability of variables that may lead to criminal behavior in particular circumstances. However, the fact that a gene or multiple genes are involved in the liability for antisocial behavior or aggression does not make the gene the cause of it, because “the gene products operate via one or more biochemical pathways
that only indirectly lead to the psychological outcome, normal or abnormal” (Rutter, 2006, p. 224).

A study by Christiansen (1977) of 3,586 pairs of Danish male twins suggested that some MZ twins inherit biological variables, characteristics, and predispositions toward a risk for criminal behavior. The concordance rate in the study was 35% for MZ twins and 12% for DZ twins. In a later review of twin studies addressing psychopathy and criminality in MZ and DZ twins, the concordance rate for the first was 67%, whereas that for the latter was approximately 30% (Mednick & Finello (1983). Earlier studies, including those of Lange in Bavaria (1931) and Yoshimasu in Japan (1961), had found similar results.

In a meta-analysis by Walters (1992), 11 family studies, 14 twin studies, and 13 adoption studies were taken into consideration. The analysis revealed that the genetic influence on criminality varied from low to moderate. A review of 13 studies regarding twins and crime (Raine, 1993) found that MZ twins had a concordance rate of 51.5% and DZ twins a rate of 20.6%, regardless of variations in age, gender, country, and definition of crime. In addition to demonstrating two genetic pathways to drug abuse, a twin/adoption study by Cadoret, Yates, Troughton, Woodworth, & Stewart (1995) supported the possible heritability of aggressive behavior with a greater concordance rate for MZ twins than for DZ twins, with genetic factors being more involved than environmental ones. In a meta-analysis of 24 twin studies and four adoption studies of aggressive people, Miles and Carey (1997) concluded that although “environmental factors provide models and reinforcements such that a child learns to act aggressively . . . genetics must be seriously considered when explaining the similarity found in the levels of aggression among family members” (p. 215.). A greater concordance for aggressive behavior in MZ twins than in DZ twins also was reported in a study by Slutske et al. (1997) and by Eley, Lichtenstein, and Stevenson (1999). A study by Vernon, McCarthy, Johnson, Jang, and Harris (1999) and one by Taylor, Loney, Bodabilla, Oacono, and McGue (2003) on MZ twins supported a significant degree of genetic heritability for antisocial behaviors and aggression. Tehrani and Mednick (2000) suggested that if there is a higher concordance rate for MZ twins than for DZ twins in criminal behavior, it can be assumed that there is a genetic influence.

A meta-analysis by Rhee and Waldman (2002) of 51 twin and adoption studies, comprising 87,152 persons who exhibited antisocial behaviors, concluded that “genetic variation . . . appears to cause approximately 40% of the individual differences across various constructs of aggressiveness, whereas about 60% of the variants is explained by environmental factors” (in Tremblay et al., 2005, p. 229). Also, in the studies analyzed, aggression was assessed either by the individual or reported by others. When the assessment of aggression was done by the self, heritability of aggression was 39%; it was 53% when done by others. In a later reanalysis by Tremblay et al. (2005) of the meta-analysis of Rhee and Waldman, heritability varied from 45% for
complete dependency (information obtained from parents and teachers) to 29% for partial dependency (information given by the individual) to 5% for no dependency (information given by other people).

Mention should be made of Lyon’s ongoing twin study begun in 1996, comprising 3,226 pairs of twins, subjects from the Vietnam Era Twin Registry, 55% of which were MZ. The racial variations were white (90.4%), non-Hispanic (4.9%), African-American (2.7%), Hispanic (2.7%), First Nations (1.3%); and others (0.7%). Each twin answered a questionnaire regarding arrests and felonious criminal behavior prior to age 15 and afterward. The results showed that environmental influences were stronger in early criminal behaviors (up to 15 years of age), whereas genetic factors had a greater influence on later criminal behaviors.

Adoption Studies

Adoption studies provide, as Rutter (2006) stated, “the most straightforward approach [to gene and environment correlations] because of their clear separation between the biological and the adoptive parents who did not provide the genes but who did provide the rearing environment” (p. 189). Such studies indicate a genetic influence on criminal behaviors but not on violent behaviors. Indeed, Raine (1993) reported a meta-analysis of 15 adoption studies previously conducted in Sweden, Denmark, and the United States, drawing the conclusion that all except one of the studies supported a genetic basis for aggression and criminal behavior in MZ twins and that that behavior was mostly property crime.

A study by Crowe (1974) of adoptees from Iowa found an increased rate of criminality in those who had a criminal biological mother. Cadoret, Cain, and Crowe (1983), taking into consideration Iowa children adopted at birth, found that they had a higher level of antisocial behavior when their biological parents also showed antisocial behavior. Three separate studies were used to test for gene-environment interaction in the development of adolescent antisocial behaviors. The researchers found that when an adoptee had both genetic factors and exposure to an adverse environment, his antisocial behaviors increased. They concluded that these behaviors were due to genetic and environmental factors acting together. This finding was replicated and extended in another Iowa adoption study in which the adoptive parents’ adversities were defined according to the presence of marital and legal problems, substance abuse and/or mental disorders (Cadoret et al., 1995). These adversities reportedly interacted significantly with the biological genetic predisposition, predicting elevated rates of childhood aggression, conduct disorder, and adolescent aggression in the adoptees.

A meta-analytical study by Mednick, Gabrielli and Hutchings (1984) of 14,427 adoptees, adopted at 1 year, 2 years, or after 2 years, whose criminal convictions were registered together with those of their biological
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or adoptive parents, found a higher rate of convictions in the adoptees and their biological parents. When only the biological parents were criminal, the conviction rate of the adoptees was 20%; when only the adoptive parents were criminal, the conviction rate was 14.7%; and when both biological and adoptive parents had criminal records the adoptee conviction rate was 24.5%.

Van den Oord, Boomsma, and Verhulst (1994), in a study of adopted twins, found a 70% heritability for aggressive behavior and a 39% for delinquency. The Colorado Adoption Project (O’Connor, Deater-Deckard, Fulker, Rutter, and Polmin, 1998) found that children whose biological parents were antisocial often behaved in a disruptive way with their adoptive parents, eliciting negative parenting from the adoptive parents. A study by Mason and Frick (1994) consisted of a meta-analysis of 12 twin studies, totaling 3,795 twin pairs, and three adoption studies, totaling 338 adoptees. It found that there was a medium-to-large effect size for genetic influence across the studies and 50% variance in measures of antisocial personality due to genetic effects, more significant for severe manifestations. A 1996 report by Bohman of an ongoing, Swedish longitudinal study on adoption, begun in 1960 (The Stockholm Adoption Study), found a high correlation in the alcohol abuse of adopted sons with the alcohol abuse of their biological fathers. The same correlation was found to exist regarding criminal activity, with or without alcohol. Bohman also found that the alcoholic criminals in his study often recidivated. A study by Riggins-Caspers, Cadoret, Knutson, and Langbehn (2003) pointed out that the antisocial behavior of adoptees, possibly due to parental-biological psychopathology, provoked harsh discipline from the adoptive parents who themselves had encountered adversity in their lifetimes. In sum, a biological background for criminality increased the risk of criminality for adopted children.

It can be concluded from the preceding studies that rates of crimes of aggression and violence are greatest when, as Raine (2002) wrote, “both heritable and environmental influences are present” (p. 48). Although somewhat controversial because of methodological problems, including the difficulty of separating environmental from genetic factors, twin and adoption studies are nevertheless very important in the examination of such factors and their influence on behavior.

Electroencephalography Studies

Early studies of electroencephalographic (EEG) abnormalities in aggressive and violent people by Hill and Watterson (1942) and by Silverman (1944) reported various nonspecific abnormalities in the EEGs of the criminals they tested. Gibbs, Bagchi, and Bloomberg (1947) compared the EEG findings of 452 inmates and 1,432 non-prisoner controls and found no differences between the tracings of the two groups. They concluded that
brain dysrhythmia was noncontributory in the genesis of aggressive violence. However, Mednick and Volavka (1980), reanalyzing the study by Gibbs et al., found a great number of abnormalities in the tracings of the inmates, which they believed could explain their aggression.

Studies by Buikhuizen (1982) found a higher proportion of abnormalities in the EEG tracings of offenders, especially aggressive offenders. Ample evidence in support of EEG tracing brain abnormalities in aggressive offenders, especially in cases of episodic aggression or violence, is supported by earlier studies. Monroe (1970) reported focal or generalized abnormalities in two tracings in 58% of 93 aggressive criminals. Bach-y-Rita, Lion, Climent, and Ervin (1971) reported 50% spiking in the temporal region EEG tracings of 123 offenders, 13 of whom had undiagnosed temporal lobe epilepsy. Later, Elliott (1987) noted significant EEG abnormalities in 60% of aggressive criminals studied.

Offenders show not only slower brain waves than those found in the general population but a greater dip in P300 response (Costa et al., 2000). In modern computerized brain waves, the P300 amplitude-evoked potential shows a spike in electrical voltage, usually followed by a dip after the presentation of a test stimulus. This is idiosyncratic for each individual.

AROUSAL AND HYPOAROUSAL THEORIES OF AGGRESSION

In the case of aggression, the chain of events begins in the hypothalamus and involves the hypothalamic pituitary adrenal (HPA) axis and the autonomic nervous system (ANS). The hypothalamus, located below the thalamus and behind the optic chiasma, has many functions, the primary one being to maintain body homeostasis (within minimal variations, blood pressure, body temperature, fluids and electrolytes, and body weight are basically fixed). The hypothalamus not only sends stimulating messages to various body receptors but receives inputs that basically enable it to reassess the body status and reestablish equilibrium when necessary. The hypothalamus, via nervous pathways, communicates with the reticular activating formation, the limbic system (amygdala and hippocampus), the endocrine system, and the autonomic nervous system. The ANS is formed by parasympathetic and sympathetic nuclei and long-axon chains. It controls the heart rate and the sweating mechanism, and it is in contact with the reticular formation.

Arousal Theory

The arousal theory explains differences in arousal in various personalities on the basis of physiological correlates. The different emotional components
are reflected in variations of the ANS arousal pattern. The ANS, stated Walsh and Hemmens (2008) “carries out the basic housekeeping of the body by funneling messages, which never reach our conscious awareness, from the environment to the various internal organs. . . .” (p. 240). The ANS also contributes to conditioning and to learning the emotions of shame and guilt and eventually to the formation of one’s moral conscience, giving one knowledge of the expectations of society. “Individuals with a readily aroused ANS are easily socialized; they learn their moral lessons well, because ANS arousal . . . is subjectively experienced as fear and anxiety . . . a protective factor against antisocial behavior” (Walsh & Hemmens, p. 241). Brennan et al. (1997) also had supported the protective factor of hyperarousal. Subjects who have a hypoarousability of ANS, on the contrary, socialize with difficulty and experience minimal fear, shame, or guilt. Thus, people cannot be adequately conditioned to behave properly socially, even though they might learn the difference between right and wrong (Raine, 1997).

In addition to the ANS, the reticular activating system (RAS) is involved in arousal. There are people who, under the same circumstances, are highly sensitive to the RAS, called augmenters, and others who are insensitive to it, called reducers. Augmenters have a hyperactive ANS and reducers a hypoactive one. The reducers need more stimulation because they are hypoactive. They are thoughtless risk-takers, fearless, and sensation-seeking and are frequently bored, as shown by EEG tracings, resting heart rate, and skin conduction tests (Ellis, 2003).

Low Arousal Theory

The low arousal theory (hypoarousal) supports the tenet that psychopaths, or aggressive people in general, have a low level of ANS arousal and a low cortical arousal, which promotes hyperactive, aggressive, violent behavior. Psychopaths “will be in a chronic state of stimulation and sensation seeking . . . [and] do not become automatically aroused to stimuli that would otherwise be stressful, exciting or frightening to non-psychopaths” (Walsh & Hemmens, 2008, p. 264). Low arousal is a manifestation of the psychopathic personality, and the aggressive psychopath appears calm during his crimes.

A longitudinal study by Raine, Venable, and Williams (1990) demonstrated that criminals differ in arousal from non-criminals. Kiehl et al. (2004) showed that psychopaths have abnormalities in the right hemisphere, which undermined the semantic processing of abstract material. The same authors also claimed that psychopaths have less affective-related activity in the amygdala (2001).
Heart Rate
A strong biological correlate of antisocial aggressive violent behavior is the heart rate. A low resting heart rate is the best replicated biological marker of antisocial and aggressive behavior, according to Raine, Venable, & Mednick (1997). The finding is consistent in a variety of social settings and in different countries, and it seems to be specific for the antisocial personality disorder. It is an independent predictor for violence. A low resting low heart rate is a physiological indicator of suboptimal arousal in convicted offenders. Because of low arousal, offenders need more stimulation, and they better tolerate unpleasant environmental stress. The slow reactivity of their ANS modulates their calm, cool, and collected behavior during aggressive episodes or criminal acting out.

Raine, Venables, and Williams (1990) opined that a slow heart rate may be a genetic marker, present in both aggressive antisocial males and females. They reported a prospective study that lasted 9 years, which took into consideration the triad for low arousal: a low resting heart rate, low resting skin conductance, and excessive slow-wave theta waves in the electroencephalogram. The variables were measured at age 15 in a group of normal, unselected boys. The boys were followed up for 9 years to assess whether the triad was predictive of aggressive criminal behavior. At age 24, on the basis of the three variables, 74.5% of the subjects in the study were classified as having aggressive behavior.

Skin Conductivity
Psychopathic offenders exhibit lower skin conductivity under stress, such as when frightened or feeling hostile, part of the general slow reactivity of their ANS. While they are stressed, they perspire less and, because of that, less sodium is available on the skin. That creates a weaker galvanic skin response. The slow reactivity of the ANS brings about suboptimal arousal, changes in skin conductivity, and heart and pulse rate changes at rest and under stress and influences testosterone and cortisol levels. The slow dissipation of fear was pointed out by slower skin conductance recovery in psychopaths (Hare, 1978a) and also appeared to be a predictor of recidivism (Hare, 1978b).

Low Arousal and Psychopathic Aggression
Low arousal indicates a low level of fear (fearlessness theory), a low level of anxiety, and low fear of punishment and supports risk-taking and stimulation-seeking behavior. All of these are underpinnings of antisocial
behavior, indicating that a low arousal level may predispose the individual to aggressive acting out. On the contrary, a high arousal level protects against aggressive psychopathic behavior.

The psychopathic feels significantly less anticipatory anxiety in the face of unpleasant and traumatic situations and lacks empathy and the capability of identifying with another’s emotional pains (Meloy, 1996). It appears that decreased cortical arousal inhibits the fear response and produces a lack of shame, guilt, and remorse. Psychopaths have difficulty in emotion processing along with poor prefrontal functioning and dysfunctioning of the amygdala (Herpertz & Sass, 2000). They tend to show marked levels of instrumental aggression, which is modulated by the amygdala, and orbital frontal cortex dysfunction is involved in the modulation of reactive aggression (Blair, 2004). In addition, poor inter-hemispheric integration and lateralization deficits create in the psychopath abnormal processing asymmetries. A study by Raine et al. (2003), taking into consideration the corpus callosum of 15 psychopaths, found that “psychopathic antisocial individuals compared with controls had significantly increased callosal white matter and whole-brain volumes” (p. 1137), possibly reflecting “an early arrest of axonal pruning or increased white myelinization” (p. 1134). That could be the cause of poor inter-hemispheric integration.

Aggression may be affective or predatory. Affective aggression is the result of internal or external stimuli that provoke an autonomic system reaction such as fight, flight, or freezing. Contrary to the “threatening vocalizations and attacking or defending posture [which involve] the spinal thalamic tract and the periaqueductal gray [matter],” typical of affective aggression, the predatory aggression of the psychopath involves “minimal autonomic arousal and vocalization and no elaborate behavioral rituals” (Meloy, 1996, p. 25). That indicates that in affective aggression, there is autonomic arousal whereas in predatory aggression there is no arousal, but the predator quietly and methodically prepares and carries out an attack by focusing attention on the prey.

**NEUROTRANSMITTERS**

At a biological level, behavior is regulated by two opposing systems. The first is the behavioral activating system associated with the neurotransmitter dopamine, which facilitates goal-directed behavior (Gove & Wilmoth, 2003). The second system is the behavioral inhibitory system, with the neurotransmitter serotonin, a general modulator of behavior and of brain memory areas (Walsh & Hemmens, 2008).

Neurotransmitters are chemical substances in the brain that transmit signals from one neuron to another. They may be hypo- or hyperfunctioning. Serotonin, with its own circadian and ultradian rhythm, which inhibits
both types of aggression, affective and predatory (Valzelli, 1981), is also responsible for a relaxed mood and feelings of contentment. Virkkunen, Goldman, and Linnoila (1996) found low serotonin blood levels in individuals who had committed impulsive crimes. It is thought that serotonin influences the limbic system and prefrontal area of the brain, facilitating the functioning of the prefrontal cortex and enabling it to discharge its restraining function in cases of impulsive aggressive behavior. Various areas of the emotional regulating system have receptors for serotonin. Serotonin is low in antisocial behavior. Already decades ago, Greenberg and Coleman (1976) found that aggressive behaviors are usually associated with low serotonin metabolites. A study by Hariri et al. (2002) confirmed the effect of the serotonin transporter gene (SERT-5-HTT) on the physiological response to stress. Nitric oxide has been implicated in aggression in mice, possibly because of an effect on serotonin levels. Mice lacking the gene essential for its production have been found to be highly aggressive. In addition, agents inhibiting it appear to lead to aggressive behavior (Chiavegatto, Dawson, Mamounas, Koliatsos, Dawson, & Nelson, 2001; Trainor, Workman, Jessen, & Nelson, 2007).

It has been postulated that in aggressive behaviors, there is an imbalance between the excitatory neurotransmitter glutamate and the inhibitory γ-aminobutyric acid (Swann, 2003). Other neurotransmitters implicated in aggression are norepinephrine, which enhances affective aggression; dopamine, which enhances affective and negatively affects predatory aggression; and acetylcholine, which positively correlates with both types of aggression (Eichelman, Elliott, & Barchas, 1981). The norepinephrine level was noted to be high in the cerebrospinal fluid of aggressive criminals in one study (Brown, Ballanger, Minichiello, & Goodwin, 1979). The effects of these neurotransmitters correlate well with the calm, detached behavior of the predatory aggression of the psychopath and with affective aggression in general.

GENES

Genes do not cause a specific behavior but make the individual sensitive to environmental noxae and modulate responses to them. They influence personality traits and, indirectly, they contribute to a person’s criminal behavior through traits such as low IQ, impulsivity and low empathy, all of which are contributory factors in aggressive crime.

Monoamine Oxidase A

Monoamine oxidase A (MAOA) is an enzyme present in the mitochondria, whose function consists of metabolizing neurotransmitter molecules, or part
of them, at the synaptic level by activating special nerve cells. It is also found in blood platelets. The MAOA gene exerts an effect on all neurotransmitter systems, including norepinephrine and dopamine, by converting amine groups into aldehyde groups. MAOA is low among violent, aggressive offenders and seems to correlate with high levels of testosterone and low levels of serotonin. The gene for MAOA, which influences the level of messengers in the brain, may be present in two different forms: a short form and a long form. People with the short form of the gene have higher levels of neurotransmitters. The gene for MAOA is located on the X chromosome, and some individuals may have mutations and/or deletions of the gene; these defects are gender-linked and result in cognitive deficits. A MAOA defect has been associated with violence in males.

Brunner syndrome (Brunner, 1996) is due to a MAOA gene mutation and consists of mental deficiency and impulsive aggression. Brunner et al. (1993) studied a large Dutch family over four generations and reported that 14 males of the family were borderline mentally retarded and exhibited numerous aggressive behaviors, mostly verbal but with a propensity to physical violence. The participants had a history of mistreatment in childhood. Crimes reported to have been committed by them included rape, assault, and arson. Only the males were afflicted, and the transmission of the condition was through the X chromosome. The studies revealed that the behaviors were due to a minor mutation of the gene that produces MAOA. The males affected by the condition could not produce MAOA, which affected their ability to break down serotonin and dopamine. Even though the level of serotonin was very high, which should impede aggressive behavior, they had a reduced number of serotonin receptors in their brains.

Caspi et al. (2002) studied the association between violent criminal behavior in maltreated children and MAOA activity in the brain. Caspi et al. analyzed data from 442 adult New Zealanders and identified 154 who had been abused as children, including 33 who had been severely abused. They reported that 85% of those who had been severely abused and developed some form of antisocial behavior were carriers of a MAOA low-level activity variant. The study also indicated that maltreated children with a genotype conferring high levels of MAOA expression were less likely to develop antisocial problems. Caspi et al. provided initial evidence that there is a functional polymorphism in the promoter region of the gene encoding the neurotransmitter-metabolizing enzyme MAOA with various levels of activity. Obviously, further research is needed. Caspi et al. asserted that “high MAOA activity exerts a protective influence against maltreatment for girls as well as boys and raises the possibility that further research into X-linked genotypes may help explain one of the least understood facts about serious antisocial behavior: the sex difference” (p. 853).

As stated by Rutter, longitudinal study data all tend to point to a gene-environment interaction but suffer from “the disadvantage of having to rely on an inferred genetic liability rather than a measure genetic liability in
relation to an identified susceptibility gene” (p. 196), a limitation that has been remedied in findings from the Dunedin Longitudinal Study. Research from that study found that antisocial behavior is a function of MAOA activity and a childhood history of maltreatment (Caspi et al. 2002); that the effect of life stress on depression is moderated by the 5-HTT gene (Caspi et al.); and that the effect of maltreatment in childhood on liability to depression was moderated by the same gene (Caspi et al.).

Other genes thought to be involved in predisposing the individual to react aggressively are catechol-O-methylytransferase, an enzyme that degrades catecholamines such as dopamine; DRD4, a particular dopamine receptor; and 5HTT. In a recent study, Beitchman et al. (2004) found that persistent and pervasively aggressive children were more likely to carry “a particular MAOA allele that results in higher level of MAOA transcription than in normal adults . . . . [and] that the increased MAOA might affect persistent aggressive behavior by having a constant degradative effect on other neurotransmitters, such as serotonin, dopamine and norepinephrine” (cited in Anderson, 2006, p. 193). These genetic deficiencies of MAOA activity were also found in aggressive mice (Rowe, 2001). All these genes influence the development of the brain, the neurotransmitters, and their receptors. They do not inevitably lead to traits but only predispose some persons to have certain reactions to others and to events in general.

Testosterone

The hormone testosterone is an important correlate of aggression and criminal behavior. The testosterone level changes throughout the day, resulting in a different predisposition to aggression at different times. It follows an ascending curve during early childhood/adolescence, reaching a peak around 18 to 24 years, and slowly declines thereafter. It seems to coincide with a peak in antisocial aggressive behavior, which is higher in males 16 to 28 years of age. Its major effect is at the brain level.

Testosterone production takes place in two distinct phases of development, the organizational or perinatal phase and the activational or post-pubertal phase. Petronis (2001) suggested that “prenatal-neonatal sex hormones might bring about effects on gene expression in humans . . . . [and] on later psychological functioning” (cited in Rutter, 2006, p. 213), even though the effects are modest and have individual variations. Indeed, as Walsh and Hemmens (2008) stated, “Most of the permanent effects of testosterone occur perinatally. If levels of testosterone are high, the brain will be masculinized; if they are low, the brain will remain in its default feminine mode” (p. 309).

The evolutionary neuroandrogenic theory of criminal behavior suggests that brain levels of testosterone induce suboptimal arousal or diminish brain sensitivity (Ellis, 2003). This brings about a search for a higher level of
sensory stimulation, which is what is observed in psychopaths. The theory proposes that exposure to testosterone may cause hyperactivity of the right hemisphere of the brain—a rightward shifting of neocortical functioning—which has to do with the calculation of risk and reward probabilities, as opposed to the language and empathic reasoning proper of the left hemisphere. The evolutionary neuroandrogenic theory posits that males are more violent and aggressive than females because their brains are exposed to much higher levels of testosterone.

Dabbs, Frady, Carr, and Besch (1987) measured free testosterone levels in the saliva of 89 male prisoners. Their findings showed “mean saliva testosterone concentrations to be higher among inmates convicted of violent crimes than among those convicted of nonviolent crime.” (p. 179). A later study, measuring salivary testosterone levels in 692 adult male prisoners, supported those findings (Dabbs, Carr, Frady, & Riad, 1995). Taking into consideration 230 male prisoners from the same study, Dabbs, Riad, and Chance (2001) also found that the behavior of the high-testosterone-level prisoners was more ruthless than that of those whose testosterone levels were low. A study of a group of rapists and child molesters found that salivary testosterone was associated with an index of antisocial behaviors (Aromaki, Lindmann, & Erikson, 2000).

In a 1993 study, Raine reported that testosterone is probably one of several hormones involved in aggressive behavior and the link to aggression mediated especially by serotonin. Similar findings were found by Soler, Vinayak, and Quadagno (2000) and by Sanchez-Martin, (2000). Giotakos, Markianos, Vaidakis, and Christodoulou (2003) found high testosterone levels and low levels of 5-HIAA in rapists, suggesting that the relationship between testosterone and aggression might be mediated via serotonin.

Mazur (1985) and Mazur and Booth (1998) suggested that testosterone is primarily related to status and dominance and secondarily to aggression when the individuals are frustrated in being unable to achieve status and dominance. A study by Bernhardt (1997) also found that testosterone promotes dominance-seeking behavior. Although testosterone, as a cause of aggression, seems to be controversial, removing it, either by castration or with anti-hormone drugs, reduces recidivism in sexual offenders (Anderson, 2006).

Cortisol

The hypothalamus, which regulates emotional responses and the ANS, produces the corticotrophin-releasing factor. This is a peptide that stimulates the adrenal gland to produce cortisol. Cortisol, the so-called stress hormone, is a glucocorticoid that both during stress and during arousal increases in normal individuals. It makes available to the body increasing energy, enabling it to
better cope with stress for longer periods. It influences the brain by affecting mood. It is the ultimate product of the HPA axis (hypothalamus → pituitary → adrenal cortex). The activation of the HPA axis is under the influence of brain structures such as the amygdala. The corticotrophin-releasing factor leads to a subsequent increase in the heart rate and sweat gland activities (galvanic skin response).

Antisocial persons have low levels of cortisol under stress and also do not have a proper state of arousal or feelings of anxiety or fear. They could be described, then, as under-aroused, fearless, and without any anticipatory anxiety. Studies have linked low cortisol levels to aggressive behavior (e.g., Virkkunen, 1985). McBurnett, Lahey, Rathoua, and Loeber (2000) found that in a study of boys referred for disruptive behavior, “salivary cortisol concentrations sampled over time were inversely associated with several measure of aggression and disruptive behavior . . .” (p. 41). Pajer, Gardner, Robin, Perel, and Neals (2001) found significantly lower levels of cortisol in a “group of late-adolescent girls . . . who had exhibited a pattern of repeated antisocial acts for at least 1 year” (p. 300). In sum, a low cortisol level is a useful biological marker in persons who are prone to antisocial aggressive behavior.

**BODY TYPE**

Among the well-known body types, ectomorph, mesomorph, and endomorph described by Kretschmar (1970), the mesomorphic body type—a stocky, muscular, well-developed individual—shows a higher level of testosterone, which enhances the muscular mass and may give the individual greater offense probability (Blackson & Tarter, 1994).

**MATERNAL SMOKING**

Smoking in pregnant mothers was correlated by Räsänen, Hakko, Isohanni, Hodgins, Järvelin, and Tiihonen (1999) with a high probability of delinquent activity in offspring. The rationale given was that a high level of carbon monoxide and other neurotoxins may negatively influence the IQ of the child, leading to lower executive functioning. In other words, the aforementioned noxae will have negative effects on the frontal lobe and its functioning. The low IQ would affect the appreciation of a life within legal boundaries. In a later meta-analysis, however, Pratt, McGloin, and Fearn (2006) concluded that it was a moderately important but distal factor for criminal deviant behavior in offspring.
BLOOD GLUCOSE LEVELS

The hypothalamic-pancreatic axis is involved in the regulation of blood glucose, which is necessary for normal brain function through the production of insulin. Fluctuations in the blood glucose level (hypoglycemia or hyperglycemia) may cause mental confusion, irritability, and defects of reasoning. They may trigger violent mood swings in predisposed persons. During a hypoglycemic state, it is possible that the limbic system, and specifically the amygdala, may send unclear or incorrect messages to the frontal lobe, which then reacts accordingly, and aggression and violence may result. Malouf and Brust (1985) studied 125 persons presenting at emergency rooms for symptomatic hypoglycemia and observed 38 with bizarre confusional behavior and 65 with stupor. Signs and symptoms of hypoglycemia are autonomic (diaphoresis, tremor, tachycardia, sense of hunger) and neuroglycogenic (mental confusion, slurred speech, behavioral changes, lethargy, incoordination).

NUTRITIONAL FACTORS

Prenatal and childhood nutritional deficiency may predispose one to antisocial behavior, because it disturbs the production of serotonin and other neurotransmitters involved in the emotional regulatory system. A study conducted decades ago had found that diets high in corn, which is low in tryptophan, a precursor of serotonin, were associated with elevated homicide rates (Mawson & Jacobs, 1978). Serum cholesterol levels have also been hypothesized as related to aggression (Hillbrand, Spita, & Foster, 1995). One study of the effects of vitamin and mineral supplementation on juvenile delinquency in American schoolchildren found that those children who took the supplements had lower rates of antisocial behavior than those receiving placebos (Schoenthaler & Bier, 2000). The authors concluded that low concentrations of water-soluble vitamins in the blood impair brain functioning and may lead to serious antisocial behavior.

NEUROIMAGING AND AGGRESSION

Within the brain, there is an interplay between the prefrontal cortex and the amygdala. The prefrontal cortex exercises control of the amygdala, inhibiting aggressive behavior. Aggressive behavior is often due to a hypofunction of the frontal lobe leading to hyperfunction of the amygdala.
Prefrontal Dysfunction and Aggression in Adolescents

The prefrontal cortex undergoes an intense prepubescent period of synaptogenesis and a period of pruning of excess synapses during adolescence. The myelinization of the prefrontal cortex is not complete in adolescence nor is the speed and conductive efficiency of neurotransmission. This may be the cause of the irrationality of teen and young adolescent behavior.

The Amygdala

The amygdala is an almond-shaped region of the forebrain. It sends and receives projections from the frontal cortex. It is involved in a variety of behaviors, such as the reaction to facial expression, enhancing memory in emotional situations, and in aggression. Hariri et al. (2002, 2005), “using a functional imaging strategy, showed that humans with a short copy of the 5-HTT allele exhibited greater amygdala neuronal activity (i.e., in the part of the brain concerned with emotional reactivity) to fearful visual stimuli compared with individuals with two copies of the long allele” (in Rutter, 2006, p. 199).

The activity of the amygdala is modulated by three specific regions of the frontal lobe: the dorsolateral, the medial, and the orbitofrontal cortex. It plays a major role in abnormalities of behavior, especially when it is not controlled by the frontal lobe. In the amygdala, there are many receptors for testosterone. A dysfunction of the amygdala and the closely related hippocampus are associated with violent aggressive behavior. Fright activates the amygdala and stimulates the hypothalamus, which in turn stimulates the HPA axis to produce cortisol. The amygdala is mostly involved in aggression, especially instrumental aggression.

The Insula (Island of Reil)

The insula, or Island of Reil, is found at the posterior junction of the parietal, temporal, and occipital lobes. It contributes to the processing of emotional information and is involved in the experiencing of lust, disgust, pride, humiliation, guilt, atonement, and empathy, all of which are at times involved in aggressive behavior.

Brain Structural and Functional Correlates

Neuroimaging techniques give supporting objective evidence of structural abnormalities and dysfunction of brain regions that may contribute to
mental impairment, abnormal behavior, and even criminal acting out. These fast-evolving techniques—CAT, fMRI, sMRI, PET, and SPECT (computed axial tomography; functional magnetic resonance imaging; structural magnetic resonance imaging; positron emission tomography; single-photon emission computed tomography—are continuously improving our understanding of the neurobiology of normal and abnormal behavior.

Functional neuroimaging, by pointing out specific abnormalities in regions of the human brain, is one piece of evidence the expert should rely on in determining the extent of neuropathology of an individual. Neuroimaging may also have the potential to “identify correlates of illness which are sub-clinical, perhaps preceding the onset of clinical symptoms or persisting after an apparent remission” (Fu & McGuire, 1999, p. 1366). They may be used as a preventive measure against violence in predisposed persons.

Functional neuroimaging has clearly pointed out that irrational aggressive behavior is often the outcome of a top-down dysfunction of the brain in which, for example, the frontal cortex loses its control on lower structures, such as the amygdala. It can be envisioned that in the future, new, extremely precise, and more reliable high-resolution scanners will be devised and will be used to detect pertinent neuropathology in aggressive behavior. There is not only a lack of behavioral control in those persons with abnormalities of these brain areas but a dysfunction of thinking and planning. That is what is seen at times as irrational behavior in aggressive persons in particularly stressful situations.

Though the prevalence rate of brain dysfunction in the general population is 3%, as stated by Redding (2006),

neuropsychological studies show that the prevalence rate of brain dysfunction among the criminal population is extremely high, with prevalence rates of ninety-four percent among homicide offenders, sixty-one percent among habitually aggressive adults, forty-nine to seventy-eight percent among sex offenders, and seventy-six percent among juvenile offenders (p. 57).

The behavior of the person with an aggressive antisocial personality is reminiscent of the orbitofrontal syndrome: impulsive, irresponsible, lacking social inhibitions, impairment of insight and foresight. It has been suggested that a history of head injuries, from mild to severe, is more often reported by violent criminals than by non-violent criminals (see e.g., Volavka, 1995). Direct damage to the frontal lobes, however, is not even necessary to produce significant frontal lobe dysfunction. Damage to the upper brain stem may produce similar effects (Goldberg, 2001, p. 147). Further, Raine et al. (1998), in a study of the brains of convicted murderers, found PET scan abnormalities in the prefrontal cortex, with an 11% reduction in the gray matter. Patients with lesions of the temporal lobe/limbic system may show
violent repetitive behavior, with impulsivity, intense emotional arousal, and lack of control with repentance and depressive feelings after their actions.

Lesions of the frontal lobes may lead to an incapacity to distinguish right from wrong or to appreciate the moral implications of one’s behavior; lesions of the limbic system or mid-temporal regions may predispose one, because of poor impulse control, to random outbursts of rage and violence. Cognitive impairment and emotional disinhibition are often present in people who, basically personality-disordered, commit violent crimes.

Various investigators have found that the impulsive aggression of borderline personality disorder is most probably the consequence of a disruption of the emotional modulation circuits. These circuits include the anterior cingulate cortex (ACC), the orbital frontal cortex (OFC), the ventromedial prefrontal cortex (VMC), and the dorsolateral prefrontal cortex (DLPFC).

Structural magnetic resonance imaging (sMRI) findings in antisocial personality disorder show thinning of the prefrontal grey matter and that the volume of the amygdala is decreased, especially when the level of psychopathy is high (Raine, Lencz, Bihrl, LaCasse, & Colletti, 2000). One study found that in “criminal psychopaths, the fMRI showed decreased activity in the amygdala, hippocampal formation, parahippocampal gyrus, ventral striatum and anterior and posterior cingulated gyrus” (Goodman, Triebwasser, Shah, & New, 2007, p. 103).

CONCLUSION

In spite of the great forward strides made by scientific research during the recent past, definitive causative factors in the origins of crime have yet to be found, and many question marks remain. Aggressive behavior is influenced by the interplay of multiple genes that operate on brain biochemical pathways, neurotransmitters, and hormones, producing a certain psychological predisposition that, in contact with environmental risks within or outside the family, is conducive to antisocial aggressive behavior. Environmental risks are co-participants in such behavior. The importance of the individual’s unique decisional capacity in such behavior is a question that requires further research.

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Correlates of Aggressive Behavior


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