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*AREA REVIEW*

# An Evolutionary Approach to Understanding Violent Antisocial Behavior: Diagnostic Implications for a Dual-Process Etiology

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AREA REVIEW

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# An Evolutionary Approach to Understanding Violent Antisocial Behavior: Diagnostic Implications for a Dual-Process Etiology

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**ABSTRACT.** This article proposes an evolutionary theory for understanding the etiology of violent antisocial behavior. From this approach, aggressive behavior is understood as an evolutionary adaptation that has provided a selective advantage for members of the human species that demonstrated this trait. Similarly, aggression control is also viewed as an adaptive trait. Antisocial behavior is described as aggressive behavior that is excessive or uncontrolled and results from either of two pathways. Instrumental antisocial behavior may arise from genetic contributions to aggressive personality traits whereas reactive antisocial behavior may result from damage or deficiencies in a biological "impulse control devise." Supporting evidence from behavioral genetic and other genetic research is presented and discussed in the context of gene-environment interaction. Diagnostic implications of this approach suggest that risk

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prediction techniques that rely specifically on antisocial personality traits may miss a subgroup of potentially high-risk individuals who do not have antisocial personalities but who do have deficiencies in the impulse control device. Diagnostic recommendations for antisocial subgroups are provided.

**KEYWORDS.** Q1

Since the latter part of the twentieth century, violent antisocial behavior has been perceived by the psychological discipline, as a whole, as caused mainly by environmental learning-based influences. For instance, the American Psychological Association's (APA) pamphlet on youth violence states, "There is no gene for violence. Violence is a learned behavior . . ." (American Psychological Association [APA], 1996). Explanations of violent antisocial behavior that focus exclusively on learning-based approaches such as modeling of family violence (Straus & Yodanis, 1996) or exposure to media violence (Anderson et al., 2003) garnish popular support among psychologists, yet genetic or evolutionary explanations of violent behavior remain controversial (Wasserman & Wachbroit, 2001). This is somewhat surprising in light of the general "nature/nurture compromise" adopted by the field to defuse debate between biological and non-biological influences on debate. One such example of the "nature/nurture compromise" is the "diathesis stress" model that attributes behavior to both genetic and environmental causes (Zubin & Spring, 1977). This article contends that the "nature/nurture compromise" can be effective only when serious attempts are made to understand the genetic and evolutionary roots of behavior to best elucidate the interactional nature of genetics and environment. In relation to violent antisocial behavior, this article reviews evidence in support of a genetic and evolutionary etiology and provides practical diagnostic implications from this framework for diagnosing subgroups of individuals at high risk for violent antisocial behavior.

## *THE GENETICS OF VIOLENT ANTISOCIAL BEHAVIOR*

Before discussing evidence in support of a genetic etiology of violent antisocial behavior, it is important to acknowledge that genetic and non-genetic theories of behavior need not be in competition. Indeed most

behavioral genetics researchers understand behavior as arising through interactions between genes and environment (e.g., Moffitt, 2005). It is beyond the scope of this article to fully review learning-based influences on violent antisocial behavior, although there appears to be solid evidence to support that direct exposure to violence in the family can act as a causal influence (Straus & Yodanis, 1996). Nonetheless it is worth noting that not all individuals exposed to violence in the family develop into violent antisocial individuals (Capsi et al., 2002). On the other hand, although some media violence researchers suggest media violence may "cause" increases in "aggression" (Anderson et al., 2003), how this relates to violent antisocial behavior is actually poorly understood. For example Tedeschi and Quigley (2002) note that aggression measures used in many of these studies lack validity. Others such as Pinker (2002) have suggested the effects attributed to media violence exposure may actually be due to "third" variables such as personality or family environment. Similarly, two recent meta-analyses of video game violence effects found no support for a causal relationship with aggressive behavior (Ferguson, in press; Sherry, 2007). Q2

As such, though learning-based etiologies of violent antisocial behavior are an important part of the developmental picture, they are not adequate in and of themselves to fully understand the etiology of violent antisocial behavior. It is thus reasonable to suggest that genetics may provide additional information to any comprehensive etiological model. Evidence for a genetic etiology of violent antisocial behavior comes from two main sources: from research on specific genes and their relationship with violent antisocial behavior and from behavioral genetic studies of antisocial behavior.

Research on specific genes in human populations has begun to provide some evidence for how genes may contribute in the development of antisocial behavior. For example, in one study of 240 children with attention deficit hyperactivity disorder, children with the valine/methionine variant in the catechol O-methyltransferase (COMT) gene showed greater antisocial behaviors, including violent behaviors, than those without this variant (Thapar et al., 2005). This variant of the COMT gene also appeared to interact with neonatal risk to increase APB. The COMT gene variant may have influenced the development of the prefrontal cortex, potentially reducing control over aggressive impulses. Q3

Caspi et al. (2002) used a longitudinal design to examine the impact of the MAOA gene located on the X-chromosome and its interaction with exposure to maltreatment in the family. Results indicated that males with both a low-MAOA activity genotype and exposure to maltreatment were

significantly more likely to exhibit child conduct disorder and adult antisocial behavior, including criminal arrests, than were high-MAOA activity genotype males who had been similarly maltreated. Although the low-MAOA genotype on its own did not increase APB, it appears that its presence places individuals at risk for APB, which can be triggered by maltreatment in the family. The presence of this gene on the X-chromosome may also help to explain why males are more aggressive, on average, than are females, particularly if the low MAOA activity genotype is recessive in nature.

Retz, Retz-Junginger, Supprian, Thome, & Rosler (2004) found a relationship between the serotonin transporter promoter gene (5-HTT) and impulsive violence in a forensic sample of 153 males. Specifically, a deletion/insertion polymorphism on this gene predicted impulsively violent behavior within this population. In and of itself, polymorphism on this gene was not able to explain the majority of violent behavior, but it appears that this gene may be one part of a larger genetic puzzle.

It is worth noting that having one of the above genotypes does not, in and of itself, appear to deterministically cause violent antisocial behavior. Rather, in combination with environmental influences, the likelihood or risk of violent antisocial behavior is increased due to specific genotypes.

Research from behavioral genetics studies of antisocial behavior typically attempt to associate similarities in behavior to either genotype or environment. Given that identical (monozygotic [MZ]) twins share all of their genetic material and fraternal (dizygotic [DZ]) twins share approximately half of their genetic material, but (it is assumed) share similar environments, it is concluded that the behavior of MZ twins should be twice as similar to that of DZ twins (Moffitt, 2005). Any variations from this observation can be attributed to non-genetic effects.

The number of behavioral genetic studies conducted to examine antisocial behavior is quite large, although it is worth noting that such studies do not always distinguish between violent and nonviolent antisocial behaviors. As such this review will focus specifically on meta-analytic reviews of these studies. Meta-analytic studies have consistently concluded that approximately 50% of the variance in antisocial behavior can be attributed directly to genetic effects (Ferguson, 2007; Miles & Carey, 1997; Rhee & Waldman, 2002). These results acknowledge both that genetic effects are a substantial contributor to the development of antisocial behavior but also that "non-genetic" factors are crucial to our understanding of antisocial behavior. The majority of non-genetic factors appear to be "unique" rather than shared, suggesting that family influences, which are best represented by shared non-genetic variances, account for the smallest

percentage of variance in antisocial behavior. The remaining variance is attributed to unique non-genetic influences, which may include unique learning experiences but also may include non-genetic biological influences such as head injuries, infections, and the like. Similarly, this variance may be attributed, in part, to gene-environment interactions, wherein the behavior of the individual is adapted to unique environmental pressures.

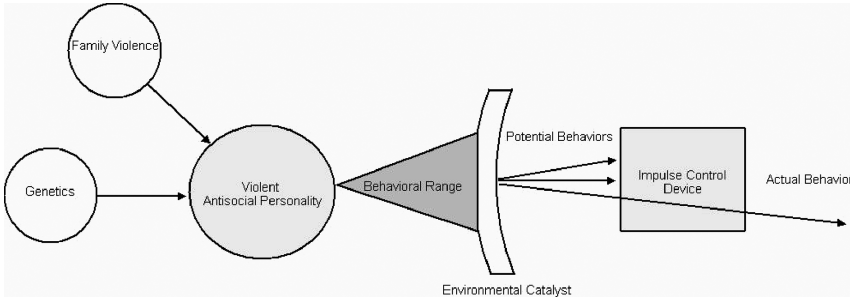
## *AN EVOLUTIONARY MODEL OF VIOLENT ANTISOCIAL BEHAVIOR*

Most current models of antisocial or aggressive behavior minimize the contribution of genetics or evolution to antisocial behavior. For example, may non-evolutionary theories of aggression focus on social modeling approaches (e.g., the General Aggression Model; Bushman & Anderson, 2002). Although such theories may vaguely allow for the contribution of "personality" or "internal" factors, such factors are seldom elucidated or incorporated into social learning models. As a result, such models typically exclude significant explanatory power from their analysis. Not surprisingly, the effect sizes from tests of these models have generally been weak (Savage, 2004), and they lack concordance with real-world phenomenon (Olson, 2004). In one test of a model of antisocial behavior that focused on a combination of personality factors and family violence, compared to a model that focused exclusively on external factors such as family and media violence, the model that included internal non-learned influences was a better fit to the data (Ferguson et al., in press). Last, many current theories of aggression separate out "biology" and "culture" as if these factors were distinct from each other. Yet from an evolutionary perspective, culture itself could be viewed as a product of, and intractably linked with, biology (Ferguson, 2007; Richerson & Boyd, 2005). Arguably, much of social science research has focused on "proximal" causes of aggressive behavior, which themselves are simply products of more "ultimate" causes of behavior. For example one could theorize that an antisocial personality leads to aggressive behavior (a proximal cause) although antisocial personality is itself the product (in part at least) of genetics (the ultimate cause).

From an evolutionary perspective, a behavior that is fairly ubiquitous such as aggression can be viewed as an evolutionary adaptation (although evolutionary theory can itself be viewed through multiple perspective; see Laland & Brown, 2002). In other words, human beings display aggression

because it is adaptive for them to do so, and they are more likely to display aggression under environmental circumstances where it is more adaptive, and less likely to display aggression under environmental circumstances where it is not adaptive. This fairly basic statement highlights the gene-environment interaction. From an evolutionary perspective, this involves the development of a brain "module" that seeks to identify environmental stimuli that are provocative of an aggressive response. Genetics need not result in deterministic behavior but rather provide the organism with a behavioral repertoire with which the organism may meet specific environmental needs. Aggression, here, is considered a non-pathological behavior. In this model, use of the term *aggression* is similar to that proposed by Baron and Richardson (1994). Specifically, aggressive behavior is here defined as behavior intended to cause physical harm or humiliation to another organism that wishes to avoid the harm. Considering aggression to be non-pathological does not necessarily imply that such behavior is morally desirable or ought to be encouraged (to do so would be to engage in the naturalistic fallacy). Aggression, as defined above, includes a wide range of behaviors, including self-defensive behaviors, hunting behaviors, behaviors in competitive sports, competition in occupational realms such as politics or business, many play behaviors between children, behaviors intended to secure dominance over others, as well as violent criminal behaviors that are not sanctioned by an individual's social group. Buss and Shackelford (1997) provide an excellent review of the underlying premises of an evolutionary understanding of aggressive behavior. Buss and Shackelford argue that aggression can be thought of as an adaptive response that can result in certain gains such as co-opting or defending resources, increase in mating options and mate fidelity, and increase in status. Aggression is not unitary or context-blind but rather modular and context-specific, and one would expect aggression to be provoked by external stimuli. It is important to point out that the terms *aggression* and *antisocial behavior* are not synonymous, nor are *aggression* and *violence* (defined below). Aggressive behaviors may include many behaviors that are socially sanctioned and beneficial both to individuals and to the social group. Likewise, antisocial behaviors include many behaviors that are neither necessarily aggressive nor violent but rather may include a wider range of behaviors that are risky, manipulative, thrill-seeking, hedonistic, or otherwise considered morally negative by the individual's society.

Figure 1 presents the "Catalyst Model" that has been developed to understand the etiology by which aggressive behaviors may develop into




Q7

FIGURE 1. A catalyst model for violent antisocial behavior.

violent antisocial behaviors. In contrast to aggressive behaviors, violent antisocial behaviors are here defined as intentional behavior intended to cause physical harm or humiliation to another organism that contrastingly wishes to avoid the harm *and the behavior is out of proportion with precipitating or provoking stimuli and carried out with disregard to the welfare or rights of others.* Thus, all violent antisocial behaviors are aggressive, but not all aggressive behaviors are necessarily antisocial nor violent. This model is viewed as differing from the traditional paradigm of antisocial personality disorder in which violent antisocial behavior (by and large) originates from a single uniform condition. By contrast, the current model posits two separate processes that, together, may result in several variants of antisocial traits and behavior as discussed further.

The Catalyst Model is built on several assumptions that are consistent with evolutionary psychology:

1. Human aggression is a normative and adaptive response that has a selective advantage for our species.
2. Restraining aggression (i.e., impulse control) also is a normative and adaptive response that has a selective advantage for our species.
3. Aggressive and impulse control modules respond to environmental stimuli, or catalysts, that are cognitively processed to select the most adaptive response to an environmental stressor.
4. The human brain has evolved separate systems, or "devices or modules," to manage separate aggression and aggression-reduction impulse control drives. These devices may, at times, compete, particularly when environmental catalysts are ambiguous.

This article has already considered the first point: that aggressive modules influence behaviors that are individually and adaptive species-wide. The second point highlights that, just as aggressive modules are adaptive, so too restraining or controlling aggressive behaviors is adaptive, particularly when the costs of such behaviors are potentially high or the gains are relatively low. For example, using fatal violence to slay an assailant who is assaulting one's children is adaptive, as the benefits of such behaviors (survival of offspring) are high, despite equally high costs (being killed or injured oneself). However, using fatal violence in response to a verbal insult would best be restrained or controlled since the costs of such behaviors (once again injury/death, or potential social isolation due to incarceration) are much higher than the potential benefits (temporary dominance over a relative stranger). Thus, it is not accurate to say that aggressive behaviors are universally adaptive. Rather it would be accurate to suggest that aggressive behaviors are adaptive to the degree that they maximize the potential benefits from an environmental situation and minimize the potential costs. As mentioned, the adaptation of aggression occurs through the development of a specific brain module designed to produce aggressive behavior in response to provoking stimuli. Such modules are developed through natural selection wherein organisms with advantageous prewired brain modules are more likely to survive to successful reproduction (also see Buss, 1999 for a discussion). These brain modules may "turn on" at differing points of the life span (such as for courtship after puberty) and may differ between genders (or other groups) based on differing selective pressures (Tooby & Cosmides, 2002; Ward & Siegert, 2002).

It is suggested here that, in response to this reality, humans have evolved an "impulse control device" to limit expression of the aggressive drive (e.g., Lorenz, 1963). This impulse control device may be synonymous with what is often referred to as "executive functioning" (the impulse control device may best be thought of as a potential physiological structure or set of structures from which the process of executive functioning originates). Evidence would suggest that this device is located, at least in part, within the frontal lobes (Kennedy & Coelho, 2005). For instance, Rowe, Bullock, Polkey, and Morris (2001) found executive functioning deficits to be common in individuals with frontal lobe lesions. This idea is well supported in the literature, as deficits in portions of the brain (i.e., frontal lobes of the cortex) related to executive functioning have been demonstrated to predict overly aggressive (i.e., violently antisocial) behavior (Brower & Price, 2001;

Donovan & Ferraro, 1999; Mercer & Selby, 2005; Soderstrom et al., 2002).

The catalyst model presented in Figure 1 presents a developmental pathway for violent antisocial behavior. Consistent with the observations of Caspi et al. (2002), variations in genotype as well as exposure to family violence are responsible for the development of excessively aggressive personality styles (i.e., violent antisocial personality). This initial causal pathway is consistent with data from Caspi et al. regarding the etiology of antisocial personality. Although there are a variety of other "risk factors" identified in the literature for the development of antisocial behavior (see Claire, Faille, & Penn, in press), it is argued here that most of these risk factors are sequela of the antisocial personality, not causal. Some later external influence, such as peer group, may contribute to the environmental catalyst portion of the current model, whereas others such as media violence exposure appear to lose all predictive value once family violence exposure is adequately controlled (Ferguson et al., in press). Similarly this model is consistent with the observation that considerable variance in antisocial behavior can be explained through genetic factors (Ferguson, 2007; Rhee & Waldman, 2002). It is this personality style (as represented by internal cognitive or physiological states) that produces a behavioral range, or a behavioral repertoire with which a person responds to his or her environment. Individuals who are higher in violent antisocial personality traits (i.e., who test higher on measures of psychopathy or antisocial personality) are more likely to respond aggressively to situations where aggression is not warranted (Hare, 1993). This is particularly true under circumstances of higher environmental stress or strain as indicated by the environmental catalyst. As such, individuals higher in violent antisocial personality traits are more likely to perceive ambiguous environmental stimuli as hostile or threatening and respond aggressively. Similarly, such individual are likely to consider the fault to lie with those environmental stimuli (i.e., other people) rather than their own inclinations and, consistent with the DSM-IV-TR, express little remorse for their actions (American Psychiatric Association, 2000).

The interaction between the individual personality and environmental catalysts may still produce several behavioral options regarding how to respond to the perceived environmental strain. These options may range from highly aggressive to less highly aggressive. These behavioral options are filtered through the impulse control device, which weighs the perceived costs and benefits of each and ultimately is responsible for selecting the behavior that is perceived as likely to maximize benefits and

minimize costs. The impulse control device thus allows for individuals to restrain the aggressive modules when the environmental costs of aggressive behaviors are likely to be higher than the benefits of such behaviors. This process is represented in Figure 1 by two arrows representing rejected options being blocked by the impulse control device while a third selected behavioral option passes through to become actual behavior.

This presents a view of the human brain as separate modules that may come into conflict with one another (Sagan, 1986). Thus, an individual may, under some circumstances, experience both a drive to act aggressively as well as the instinct to control or suppress that drive owing to the perceived costs. It is important to note that violent antisocial behaviors, in this model, may result from two sets of etiological systems. First, an individual may have a genotype (and perhaps exposure to family violence) that predisposes him or her to an aggressive personality. Second, an individual may have a deficiency in (due to genetics) or receive damage to (due to environmental insults such as head injuries or toxin exposure) the impulse control device.

## *DIAGNOSTIC CONSIDERATIONS FOR A DUAL PROCESS OF VIOLENT ANTISOCIAL BEHAVIOR*

It has been observed for some time that antisocial behavior can be thought of as existing along an instrumental/reactive dichotomy (Atkins, Stoff, Osborne, & Brown, 1993; Buss, 1961). Instrumental antisocial behavior (or instrumental aggression as it is often referred to) can be thought of as behavior that is directed at a specific goal and comes at the cost to another person. Theft of valuable objects, or violent behavior used to elicit submissiveness in others, are examples of instrumental aggression. By contrast, reactive antisocial behaviors occur in response to an environmental stressor, are not preplanned, and serve little purpose other than the expression of anger or rage at the expense of another person. A man who shoots his wife after learning that she has had an affair and then shoots himself is an example (though perhaps extreme) of reactive aggression.

### *Implications for Practice*

As noted, violent antisocial behavior may arise through two processes, which are not mutually exclusive. First, an individual may be genetically

prone to violent antisocial behavior through the development of a violent antisocial personality. Second, an individual may experience deficiencies or damage in the impulse control device such as through frontal lobe deficits or injury (Kant, Smith-Seemiller & Zeiler, 1998; Warnken, Rosenbaum, Fletcher & Hoge, 1994), which limits his or her ability to modify or control aggressive drives. This dual process model essentially classifies four potential outcomes for individuals. These categories are suggested for the purposes of future research, and such research will be necessary to fully understand the diagnostic differences between these categories.

### *Low-Average Aggression–High Impulse Control*

This first category of individual is essentially the "normal" default category. These individuals make up the majority of the population and, in regard to antisocial behavior at least, are considered normal or non-pathological. However, individuals who are very low in aggression may require interventions for assertiveness, shyness, and the like. Diagnostic outcomes for these individuals, in regard to tests of antisocial behavior or violence risk, would likely be within the normal range. Similarly, neuropsychological tests of executive functioning are most likely to return normal results.

### *High Aggression–High Impulse Control*

This group of individuals is likely to have a high drive for aggression. However, their impulse control device is functional and generally prevents them from engaging in extreme, violent antisocial behavior. These individuals are more likely to find "prosocial" outlets for their aggression, including recreation and career options. In relation to personality tests such as the Minnesota Multiphasic Personality Inventory–2 (MMPI; Hathaway, & McKinley, 1989), these individuals are likely to score moderately high on scales such as the 4–Psychopathic Deviate scale. Similarly, measures of psychopathy such as the Psychopathy Checklist (PCL; Hare, 1991) are likely to show small to moderate elevations. Neuropsychological tests of executive functioning (such as the Wisconsin Card Sort, Stroop or Trails B; Lezak, 1983) are likely to be within the normal range. Actuarial, violence risk measures such as the HCR-20, (Webster, Douglas, Eaves, & Hart, 1997) may be clinically insignificant or in the low range of clinical significance owing to the relatively low incidence of associated risk factors. Nonetheless, violent antisocial behaviors may

appear in these individuals when under periods of intense strain. When violent antisocial behaviors do emerge, expression of remorse is unlikely and may focus on costs to the individual (i.e., the costs associated with being caught).

### *High Aggression–Low Impulse Control*

This group of individuals is likely to be perceived of as highly antisocial, self-destructive, and poorly functioning. Frontal lobe deficits and lesions to the frontal lobe, coupled with high aggression motivation, are likely to be causal indicators for aggressive behaviors in these individuals. Violent antisocial behaviors may emerge from these individuals with little to no provocation and are followed by little or no expression of remorse. Regarding diagnostic testing, on personality tests such as the MMPI, these individuals are likely to score high on both measures of antisocial behavior or trait aggression as well as impulsiveness (i.e., the 4–9 profile on the MMPI). Tests on psychopathy measures are also likely to be high, and these individuals are the most likely to score in the clinically significant range on violence risk assessment measures, such as the HCR-20. Neuropsychological tests of executive functioning will likely reveal significant deficits in these abilities. These individuals are likely to be perceived of as bullying, cruel, inconsistent, and demeaning. Diagnostic criteria such as "unsocialized" or "childhood onset" conduct disorder (APA, 2000) or sadistic personality disorder (Millon, 1995) would be consistent with this group.

### *Low Aggression–Low Impulse Control*

This fourth group, despite being at relatively high risk for violent antisocial behavior, is arguably the most likely group to be missed during diagnosis. Absence of antisocial personality traits makes diagnosis difficult; nonetheless, frontal lobe deficits place this group at some risk for aggressive behaviors. Such individuals are likely to score within the normal range on tests such as the MMPI and PCL, with the exception of the hypomania scale (9 scale) on the MMPI or similar scales on other measures of personality. Similarly. violence risk assessment measures such as the HCR-20 are unlikely to register in the clinically significant range, particularly when they include the PCL as part of the risk assessment. The most significant clue diagnostically of this group of individuals would be poor performance on neuropsychological impulse control or executive functioning measures. Similarly, neuropsychological tests that

provoke frustration such as the Paced Auditory Serial Addition Task (PASAT; Tombaugh, 2006) are likely to elicit more extreme behavioral responses related to agitation from this group than might otherwise have been expected, although use of tests for this purpose should be done with caution and with care to provide specific informed consent prior to administration. Individuals from this group are likely to, more often than not, appear to behave normally with little inclination toward antisocial behavior. However, during periods of strain, aggressive responses will appear with unexpected frequency and often in a self-destructive manner, with little premeditation or concern for consequences. These individuals are also likely to sincerely express remorse following an incident of violent antisocial behavior and revert to "normal" behavior once environmental strains have passed. This group may also be at significant risk for depression or suicidal ideation following violent antisocial behaviors in which other persons have been seriously harmed.

As indicated, the theoretical model proposed here postulates three separate potential outcomes leading to violent antisocial behavior. The DSM-IV-TR does not distinguish between groups two and three, as both of these would likely be classified with "antisocial personality disorder." However, group three may be consistent with Hare's (1993) concept of psychopathy or Millon's (1995) concept of the sadistic personality. Group four is most likely consistent with the intermittent explosive disorder in the DSM-IV-TR. The distinctions above provide diagnostic suggestions for distinguishing between these groups of individuals at high risk for antisocial behavior.

## *Implications for Policy*

Perhaps the most obvious implication and potential pitfall for policy is the potential misinterpretation of the evolutionary development of antisocial behavior as fully deterministic. Pinker (2002) has noted that misinterpreting evolutionary theory as "hard" determinism has been both a roadblock to the acceptance of evolutionary theory in the social sciences and also may be cynically used to rationalize away responsibility for bad acts. As the genetic and evolutionary basis of antisocial behavior becomes better understood (and harder to rationalize as a purely learned behavior), there is a risk that the science may have a deleterious effect on the legal system.

The classical school of criminology, which arguably still underlies much of Western judiciary and legal systems, is founded on the idea that

humans, though hedonistic, are capable of rational choice (Jenkins, 1984). At first glance, an evolutionary theory of antisocial behavior would appear to contradict this, as one might reasonably assume that if we have no control over our genes and our genes control much of our behavior, we thus have little control over our behavior. The natural consequence of this logic is to conclude that antisocial offenders are genetically "disordered" and thus not responsible for their criminal actions. The repercussions of this logic to a criminal justice system could involve a widespread increase in the use of Not Guilty by Reason of Insanity or Mental Defect (NGI) defenses based on the belief that antisocial individuals, by virtue of their genetic defects, cannot be held accountable for their actions.

However, Pinker (2002) notes that this sort of logic is in error. Although genes do indeed play a role in the etiology of antisocial behavior (Ferguson, 2007; Rhee & Waldman, 2002), a fair amount of the variance in antisocial behavior remains unexplained. Although some of this variance is likely due to idiosyncratic events (head injuries, infections, traumatic experiences, etc.), it is not unreasonable to suggest that some portion of this variance might be due to what has been called "agency" or "free will" (Bandura, 2006; Ferguson, 2000; Rychlak, 1999). Indeed, one could argue that the capacity for behavioral flexibility (i.e., free will) is itself an evolutionarily adaptive trait, as organisms with this capacity would be better able to respond to novel environmental stimuli. One way of considering this, albeit simplified, is to consider that I (perhaps more than even the average hominid) am attracted to sugary foods. It is quite likely that the motive to eat sugary foods is largely genetic, yet I am not forced by my genes to act upon this inclination. Should I be presented with, say, a doughnut, I have the option of responding to my genetically determined motivation or not responding to it. Though the motivation may be genetic, the decision to act upon it is still my own and thus "freely" chosen. Of course, were I to receive lesions to my frontal lobes and thus damage my impulse control device, my responses to the same impulses may be different. In effect, damage to the frontal lobes may rob individuals of some level of "free will".

As such, most antisocial individuals who come to the attention of the criminal justice system would not qualify for special consideration vis-à-vis the NGI defense. The potential exception would be in cases in which significant frontal lobe lesions could be documented to have impacted the individual's behavior. This would be most evident in cases in which behavior had demonstrably changed from a non-antisocial to antisocial-prone state subsequent to an injury of lesion. Unlike an individual with a

psychotic mental illness or mental retardation (more typical diagnoses relevant to NGI), an individual with frontal lobe injury may be able to voice "right from wrong" but still be unable to act upon these cognitions. It is recommended that NGI be evaluated in such cases through careful combination of a neuropsychological functional exam along with medical or neurological exams of structural lesions or dysfunction. The presence of considerable deficits (standard "IQ" scores of less than 70, for instance) in executive functioning may present reasonable evidence of mental defects that may limit control over aggressive behaviors, information that may be valuable to an NGI assessment.

## *THE ROLE OF THE FORENSIC PSYCHOLOGIST/PSYCHIATRIST*

In most jurisdictions, the primary standard for the admission of evidence related to genetics, brain damage and the NGI defense is the Daubert standard (Daubert v. Merrell Dow Pharmaceuticals, 1993). Perhaps contrary to its intent (to remove emphasis on the "general acceptance" of scientific evidence within the scientific community, a hallmark of the earlier Frye standard, to allow for "cutting edge" research), Daubert is thought by some to have resulted in a more conservative standard to the admissibility of expert testimony (e.g. Dixon & Gill, 2002). This impact is primarily documented in civil cases, and the impact of Daubert on expert testimony pertaining to NGI has been less well documented. Within social science, resistance to evolutionary theory has been both considerable and somewhat perplexing (Pinker, 2002), with professional organizations asserting that violence has no genetic basis (e.g. American Psychological Association, 1996), despite evidence to the contrary. Nonetheless, evidence of a genetic contribution to violence is considerable, demonstrating stronger effects than is common for much of social science (e.g., Rhee & Waldman, 2002), and support for evolutionary theory, although resisted by some elements of social science, is widely accepted in the general scientific community. Thus, particularly when such evidence provides the basis of a defense (when such evidence is based on replicable studies using valid scientific techniques), it is argued here that genetic or brain injury based arguments for NGI would likely survive a Daubert motion. Genetic tests for gene alleles predictive of antisocial behavior and violence are not yet widely employed, and it is not yet clear whether their utility will advance to the point that they meet the Daubert

standard but, with greater refinement of these tests, their inclusion in a forensic examination may increase. If such tests are ultimately shown to have validity coefficients equal to or better than standard psychological tests already used in forensic examinations, they may increasingly see use in the courts. Since validity coefficients as low as .2 may sometimes be accepted (Anastasi & Urbino, 1996), this possibility is not as remote as some psychologists may think (or hope). If validity coefficients for genetic tests as predictors of violence reached the more generally acceptable .4 level for validity coefficients (similar to those found for existing violent risk assessment measures, such as the HCR-20; Webster et al., 1997), the grounds for admissibility under Daubert would be all but assured. Neuropsychological tests for frontal lobe injury are, of course, already commonly employed in some forensic examinations.

For those jurisdictions that still employ the Frye v. United States (1923) standard, which focuses on the "general acceptance" of scientific evidence within the scientific community, the outlook for the admissibility of evolutionary/genetics testimony is somewhat murkier. As has been mentioned, within the social science community, resistance to evolutionary approaches to understanding violence continue (e.g., American Psychological Association, 1996), although arguably the same may not be true for the medical or biological science communities (e.g., American Academy for the Advancement of Science [AAAS], 2002). Yet general science defense of evolutionary theories seldom is applied to human violence per se, where the scientific community has been largely content to allow the social sciences to "fight it out." Ultimately, admissibility under the Frye standard would arguably hinge upon two key interpretations. First, which "scientific community" is most relevant . . . the social science community or the general science community? And second, when is a theory sufficiently "generally accepted" within such a community? Arguably within the social science community, evolutionary explanations of evolutionary explanations of violence are not yet, "generally accepted" (Pinker, 2002), despite that evidence in support of such theories is reasonably strong (thus allowing admission under Daubert) and a considerable subset of social scientists would arguably endorse such theories at least to some degree. Thus, although some have argued that the Daubert standard has resulted in generally more conservative results regarding the admissibility of expert testimony (e.g. Dixon & Gill, 2002), the opposite may prove true for evolutionary theory as applies to NGI cases.

The role of the forensic psychologist or psychiatrist will come in helping the courts and juries make sense of these data. As mentioned earlier, one

problem for evolutionary approaches has been the interpretation by the general populace and thus potential jurors (as well as much of the social science community) that genetic or evolutionary contributions to behavior imply "hard" determinism (Pinker, 2002). Put another way, non-scientist jury members may interpret genetic/evolutionary explanations of violence as implying, "if the person has this gene he/she was *doomed from birth* to engage in violent criminal behavior." In fact, this sort of belief is not supported by the model suggested here (nor endorsed by most evolutionary psychologists) and can most easily be dispelled by noting that many individuals with the same gene allele do not engage in criminal behavior. In other words, genes may play some role in setting up our motivations, but most individuals retain the free will to indulge or not indulge those motivations (this can be easily related to "real-life" situations in jurors' own lives to which they might easily related, such as craving sweet foods and dieting). Similarly, many individuals in the community may feel similar general motivations as a criminal defendant (i.e., thrill seeking, hedonism, aggression) but find pro-social (or at least legal) outlets for such motivations (i.e., extreme sports, careers in which aggression is an advantage). Similarly, jurors may have difficulty distinguishing between types and severity of brain damage and dysfunction and may prove either unquestioningly accepting or unduly skeptical of scientific evidence that they do not well understand. Part of the role of forensic psychologists or psychiatrists giving expert testimony may come in explaining the ways in which genes and brain damage do and do not influence behavior in a manner that can be more easily understood by a lay audience. Expert witnesses may also help explain the complex way in which genes interact with one another, with the external environment, and with personality to produce behavior. Thus, part of the role of the expert witness may come in assisting jurors or the courts in understanding scientific evidence presented so that more effective and sophisticated decisions can be made by these bodies.

According to the presented model, an individual with gene alleles predictive of violence retains his or her capacity for free will (and thus criminal responsibility) so long as his or her brain function related to impulse control is demonstrably intact. This is, in fact, not terribly distinct from ways in which personality and mental illness are considered currently in NGI defenses: An "antisocial" individual is not considered to have diminished capacity per se, although an individual with psychosis may. Forensic psychologists or psychiatrists working for the prosecution could counter an NGI defense most successfully by demonstrating adequate frontal lobe functioning through a battery of neuropsychological

tests (i.e., Stroop, Wisconsin Card Sort, Trails). By contrast, examiners would bolster a defense case for NGI by demonstrating impaired impulse control functioning. Much in the same way that IQ scores below 70 argue for diminished mental capacity, I argue that standard scores on neuropsychological tests below 70 (using the IQ standard score with mean of 100 and 15-point standard deviation) argue for diminished capacity in regard to impulse control, particularly if demonstrated consistently across a variety of neuropsychological tests. As can be seen, it is unlikely that the "typical" non-brain-damaged defendant is likely to score so low on these tests (although tests for malingering are also advised), and thus such scores argue for significant brain impairment and thus support for an NGI defense.

Both the "low aggression/low impulse control" and "high aggression/low impulse control" types of defendants discussed in this article may demonstrate evidence consistent with NGI. I argue that forensic psychologists and psychiatrists have a further ethical duty (both for the protection of society and the welfare of the defendant) to clearly distinguish the risks for future violence and treatment course for these two types of defendants. Specifically, prognosis for the "low aggression/low impulse control" type is better (although not perfect), as this type may prove more motivated to participate in clinical interventions for improving impulse control, and being less consistently aggressive is less likely to see treatment continually interrupted by frequent "setbacks." It is recommended that assessment of impulse control/executive functioning become a routine part of violence risk assessments as the information provided by such tests are likely to add to the predictive utility of violence risk assessments. Evidence for the utility of tests of executive functioning as predictors of violence risk is already available (Donovan & Ferraro, 1999; Mercer & Selby, 2005). To the extent that impairments to executive functioning are organic in nature, remedy through treatment currently may be difficult. However, the adoption of clear, concrete decision-making strategies as well as concrete "thought-stopping" strategies, particularly those based on empirically validated treatments, may be beneficial in providing a "concrete" alternative to the kind of impulse control that functions automatically in non-impaired individuals.

Ultimately then, NGI defenses related to this evolutionary approach hinge upon the documentation of two phenomena: the presence of gene alleles predictive of violence and (perhaps more critically) presence of significant impairment (i.e., standard scores below 70) in executive functioning/impulse control. Defense and prosecution strategies regarding

NGI would likely revolve around demonstrating or discrediting the existence of these two phenomena.

## *CONCLUSION*

The catalyst model presents several avenues of research that would be valuable in further understanding biological and evolutionary pathways to violent antisocial behavior. First, hierarchical cluster analysis and factor analysis procedures may be useful in examining the utility of antisocial personality measures and executive functioning measures in distinguishing subgroups of antisocial individuals within the general population, psychiatric populations as well as populations of offenders. It would also be valuable to examine how specific interventions may be tailored to address the divergent etiological pathways for these subgroups of individuals. Interventions that fail to acknowledge the evolutionary purposes of aggression and gene-environment interactions may show little evidence of success. Interventions that focus exclusively on learned behavior are likely to show only short-term gain, with behaviors reverting quickly back to baseline once the intervention is removed or completed. Interventions that acknowledge some immutability in the basic personality but focus on identifying environmental catalysts and preparing rehearsed alternate (to antisocial) behaviors for those catalytic circumstances may potentially be more promising. It is recommended also that clinical researchers take care to emphasize the impact of therapeutic interventions by examining effect size rather than relying only on "statistical" significance. Lipsey (1998) recommends a minimum effect size of $r = .20$ as demonstrating evidence for successful treatment impact. In the absence of such stringent criteria, time and funding may be spent on therapeutic interventions that appear to be "significantly" successful in treating antisocial behaviors but are not truly successful on a practical level.

It is hoped that the catalyst model with its diagnostic implications provided here will provide a platform for further research and discussion on the etiology and diagnoses of antisocial behavior. It is further hoped that this article will provide practical suggestions for the diagnosis of subgroups of violently antisocial individuals. Last, it is argued here that understanding the biological and evolutionary roots of aggressive behavior and how this etiology may relate to violent antisocial behavior is essential in advancing the diagnosis and treatment of antisocial individuals. Arguably,

evolutionary theory has met with resistance in its application to human behavior (Pinker, 2002). This is unfortunate, as the practical application of clinical procedures that may originate from advanced understanding of gene-environment interaction may be sacrificed by the intractability of a learning-exclusive scientific dogma.

## REFERENCES

American Academy for the Advancement of Science. (2002). Board resolution on intelligent design theory. Retrieved October 25, 2007, from http://www.aaas.org/news/releases/2002/1106id2.shtml.

American Psychiatric Association. (2000). *Diagnostic and statistical manual-IV-Text Revision*. Washington, DC: Author.

American Psychological Association. (1996). An APA brochure on youth violence. Retrieved February 7, 2007 from http://www.apa.org/ppo/issues/pbviolence.html.

Anastasi, A., & Urbina, S. (1996). *Psychological testing*. New York: Prentice Hall.

Anderson, C., Berkowitz, L., Donnerstein, E., Heusmann, L.R., Johnson, J., Linz, D., et al. (2003). The influence of media violence on youth. *Psychological Science in the Public Interest*, *4*, 81–110.

Atkins, M., Stoff, D., Osborne, M., & Brown, K. (1993). Distinguishing instrumental and hostile aggression: Does it make a difference? *Journal of Abnormal Child Psychology.* Retrieved February 8, 2007, from http://www.findarticles.com/p/articles/mi_m0902/is_n4_v21/ai_13240551.

Bandura, A. (2006). Toward a psychology of human agency. *Perspectives on Psychological Science*, *1*, 164–180.

Baron, R., & Richardson, D. (1994). *Human aggression*. New York: Plenum Press.

Brower, M., & Price, B. (2001). Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: A critical review. *Journal of Neurology, Neurosurgery and Psychiatry*, *71*, 720–726.

Bushman, B., & Anderson, C. (2002). Violent video games and hostile expectations: A test of the General Aggression Model. *Personality and Social Psychology Bulletin*, *28*, 1679–1686.

Buss, D., & Shackelford, T. (1997). Human aggression in evolutionary psychological perspective. *Clinical Psychology Review*, *17*, 605–619.

Buss, D. (1999). *Evolutionary psychology: The new science of the mind.* Needham Heights, MA: Allyn & Bacon.

Buss, A. (1961). *The psychology of aggression.* New York: Wiley.

Caspi, A., McClay, J., Moffitt, T., Mill, J., Martin, J., Craig, I., et al., (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*, 851–854.

Claire, M., Faille, L., & Penn, J. (in press). Prevention and treatment of violent offending/offenders. In C. Ferguson, (Ed.), *Violent crime: Clinical and social implications*. Thousand Oaks, CA: Sage.

Daubert v. Merrell Dow Pharmaceuticals. (1993). 509 U.S. 579.

Dixon, L., & Gill, B. (2002). *Changes in the standards for admitting expert evidence in federal civil cases since the Daubert cecision*. Santa Monica, CA: RAND Institute for Civil Justice.

Donovan, W., & Ferraro, R. (1999). Frontal lobe deficits in domestic violence offenders. *Genetic, Social and General Psychology Monographs*, *125*, 71–102.

Ferguson, C. J. (2007). Evidence for publication bias in video game violence effects literature: A meta-analytic review. *Aggression and Violent Behavior*, *12*, 470–482.

Ferguson, C. J. (2007). Genetic contributions to antisocial personality and behavior: A meta-analytic review from an evolutionary framework (unpublished manuscript).

Ferguson, C. J. (2000). Freewill: An automatic response. *American Psychologist*, *55*(7), 762–763.

Ferguson, C., Rueda, R., Cruz, A., Ferguson, D., Fritz, S., & Smith, S. (in press). Violent video games and aggression: Causal relationship or byproduct of family violence and intrinsic violence motivation? *Criminal Justice and Behavior.*

Frye v. United States (1923). 293 F. 1013.

Hare, R. (1993). *Without conscience*. New York: Guilford Press.

Hare, R. (1991). *The manual for the revised psychopathy checklist*. Toronto, Ontario: Multi-Health Systems.

Hathaway, S., & McKinley, J. (1989). *MMPI-2: Manual for Administration and Scoring*. Minneapolis: University of Minnesota Press.

Jenkins, P. (1984). Varieties of enlightenment criminology: Beccaria, Godwin, de Sade. *British Journal of Criminology*, *24*, 112–130.

Kant, R., Smith-Seemiller, L., & Zeiler, D. (1998). Treatment of aggression and irritability after head injury. *Brain Injury*, *12*, 661–666.

Kennedy, M., & Coelho, C. (2005). Self-regulation after traumatic brain injury: A framework for intervention of memory and problem solving. *Seminars in Speech and Language*, *26*, 242–255.

Laland, K., & Brown, G. (2002). *Sense and nonsense: Evolutionary perspectives on human behavior*. New York: Oxford University Press.

Lezak, M. (1983). *Neuropsychological assessment*. New York, NY: Oxford University Press.

Lipsey, M. W. (1998). Design sensitivity: Statistical power for applied experimental research. In L. Bickman & D. J. Rog (Eds.), *Handbook of applied social research methods* (pp. 39–68). Thousand Oaks, CA: Sage.

Lorenz, K. (1963). *On aggression*. New York: Harcourt, Brace and World.

Mercer, K., & Selby, M. (2005). The effects of psychopathy, violence and drug use on neuropsychological functioning. *American Journal of Forensic Psychology*, *23*, 65–86.

Miles, D., & Carey, G. (1997). Genetic and environmental architecture on human aggression. *Journal of Personality and Social Psychology*, *72*, 207–217.

Millon, T. (1995). *Disorders of personality: DSM-IV and beyond*. Hoboken, NJ: Wiley.

Moffitt, T. (2005). The new look of behavioral genetics in developmental psychopathology: Gene-environment interplay in antisocial behaviors. *Psychological Bulletin*, *131*, 533–554.

Olson, C. (2004). Media violence research and youth violence data: Why do they conflict? *Academic Psychiatry*, *28*, 144–150.

Pinker, S. (2002). *The blank slate: The modern denial of human nature*. New York: Penguin.

Retz, W., Retz-Junginger, P., Supprian, T., Thome, J., & Rosler, M. (2004). Association of serotonin transporter promoter gene polymorphism with violence: Relation with personality disorders, impulsivity and childhood ADHD psychopathology. *Behavioral Sciences and the Law*, *22*, 415–425.

Rhee, S., & Waldman, I. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin*, *128*, 490–529.

Richerson, P., & Boyd, R. (2005). *Not by genes alone: How culture transformed human evolution*. Chicago: University of Chicago Press.

Rowe, A., Bullock, P., Polkey C., & Morris, R. (2001). "Theory of mind" impairments and their relationship to executive functioning following frontal lobe excisions. *Brain: A Journal of Neurology*, *124*, 600–616.

Rychlak, J. (1999). Social constructionism, postmodernism, and the computer model: Searching for human agency in the right places. *Journal of Mind and Behavior*, *20*, 379–389.

Sagan, C. (1986). *The dragons of Eden: Speculations on the evolution of human intelligence*. New York: Ballantine.

Savage, J. (2004.) Does viewing violent media really cause criminal violence? A methodological review. *Aggression and Violent Behavior*, *10*, 99–128.

Sherry, J. (2007). Violent video games and aggression: Why can't we find links. In R. Preiss, B. Gayle, N. Burrell, M. Allen, & J. Bryant (Eds.). *Mass media effects research: Advances through meta-analysis*. Mahwah, NJ: L. Erlbaum.

Soderstrom, H., Hultin, L., Tullberg, M., Wikkelso, C., Ekholm, S., & Forsman, A. (2002). Reduced frontotemporal perfusion in psychopathic personality. *Psychiatry Research: Neuroimaging*, *114*, 81–94.

Straus, M., & Yodanis, C. (1996). Corporal punishment in adolescence and physical assaults on spouses in later life: What accounts for the link? *Journal of Marriage and the Family*, *58*, 825–841.

Tedeschi, J., & Quigley, B. (2000). A further comment on the construct validity of laboratory aggression paradigms: A response to Giancola and Chermack. *Aggression & Violent Behavior*, *5*, 127–136.

Thapar, A., Langley, K., Fowler, T., Rice, F., Turic, D., Whittinger, N., et al. (2005). Catechol O-methyltransferase gene variant and birth weight predict early-onset antisocial behavior in children with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, *62*, 1275–1278.

Tombaugh, T. (2006). A comprehensive review of the Paced Auditory Serial Addition Task (PASAT). *Archives of Clinical Neuropsychology*, *21*, 53–76.

Tooby, J., & Cosmides, L. (1992). The psychological foundations of culture. . In J. Barkow, L. Cosmides, & J. Tooby. (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp 19–36). New York: Oxford University Press.

Ward, T., & Siegert, R. (2002). Rape and evolutionary theory: A critique of Thornhill and Palmer's theory. *Aggression and Violent Behavior*, *7*, 145–168.

Warnken, W., Rosenbaum, A., Fletcher, K., & Hoge, S. (1994). Head injured males: A population at risk for relationship aggression? *Violence and Victims*, *9*, 153–166.

Wasserman, D., & Wachbroit, R. (2001). *Genetics and criminal behavior*. New York: Cambridge University Press.

Webster, C., Douglas, K., Eaves, S., & Hart, S. (1997). *The HCR-20: Assessing the Risk for Violence*. Vancouver: Simon Fraser University.

Zubin, J., & Spring, B. (1977). Vulnerability: A new view of schizophrenia. *Journal of Abnormal Psychology*, *86*, 103–126.

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