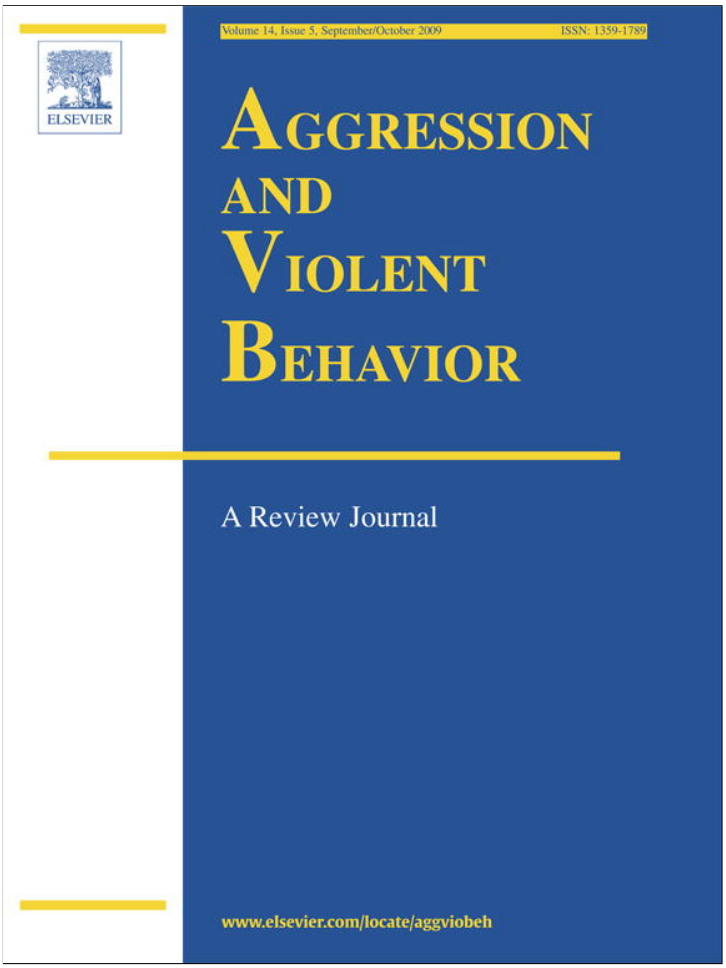

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Natural born killers: The genetic origins of extreme violence

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ABSTRACT

The current article examines the influence of genetics and evolution on acts of extreme and criminal violence among human primates. Moderate aggression can function to increase an organism's reproductive success; extreme violence can place the organism at unnecessary risk. Genetic polymorphisms that have been linked to extreme acts of violence are reviewed as is research elucidating how genetic risk and environmental stress may interact to increase risk of extreme violence. Extreme violence is viewed as high-end variance in an evolutionarily adaptive process in which the propensity for aggression and violent behavior, in moderate doses, has been adaptive for individual humans.

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The origin of human violence has been an issue of considerable concern and debate for centuries (e.g., Lombroso, 1876/2006). Violence is just one outcome that has been central to the "nature versus nurture" debate. Given technical limitations and predominating scientific views, much of the research produced on violence during the latter 20th century focused on social, family, and cultural influences on violence. More recent research has indicated that violent behavior has significant biological, genetic, and evolutionary origins as well. Several studies have identified gene polymorphisms that increase the risk for violent behavior. Increasingly, the evolutionary origins of violent behavior are being explored. The current paper seeks to provide a review of what is currently known about the genetic and evolutionary origins of extreme violent behavior.

1. Defining relevant terms

It is important to recognize that the terms used in the current paper, namely "aggression" "violence" and "extreme violence" should not be taken to be synonymous. For instance, when individuals in the general populace learn of research suggesting that, say, "Eating plums increases aggression," many such individuals may picture children or adults hitting, kicking, fighting, etc., or even imagine that such experimental results extend easily to criminally violent activities. In reality, participants in such studies may be merely filling in the missing letters of words, or delivering non-painful noise bursts to an ostensible consenting reaction-time game opponent (Savage, 2004). Many experimental measures of "aggression" do not predict violent acts or even physically aggressive behaviors (Ferguson & Rueda, 2009; Ritter & Eslea, 2005; Tedeschi & Quigley, 1996). As such, a proper understanding of relevant terms and how they are measured is necessary to prevent miscommunication.

Aggression has been defined as behavior produced to cause physical harm or humiliation to another person who wishes to avoid it (Baron & Richardson, 1994). Although this definition is functional, it does reflect a potential bias in assuming that aggression is inherently bad. In other words, the definition above is defined in such a way as to imply that the aggressor is a "perpetrator" and the aggression recipient is a "victim." As such, this is an incomplete definition of aggression. It is implied that aggression has no adaptive function and

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is always pathological and undesirable. This would appear to be naïve, and at best is an assumption. In moderate doses, aggression may very well be adaptive, guiding individuals toward many behaviors approved of by society including standing up for one's beliefs, assertiveness, defending others in need, careers in law enforcement, the military, business, legal affairs, etc., sporting activities, political involvement, debate and discourse indeed including scientific debate (Hawley & Vaughn, 2003; Smith, 2007). For purposes of this discussion a slightly altered definition of aggression is proposed, namely that aggression is "behavior which is intended to increase the social dominance of the organism relative to the dominance position of other organisms." Activities which met Baron and Richardson's (2004) definition of aggression would still fall within the current definition, although the current definition is stripped of moral implications. Aggression, then, is behavior intended to increase one's own dominance and, thus, reproductive success. Evidence does suggest that social dominance predicts reproductive success in contemporary humans (Jokela & Keltikangas-Järvinen, 2009). Other organisms may or may not be harmed depending on the form or intensity of the aggressive behavior. Violent behavior certainly would be aggressive, but not all aggressive behaviors are violent or even necessarily negative from a cultural perspective.

The World Health Organization (2002) has defined violence as "the intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment or deprivation." By and large this appears to be a reasonable definition of violence. It is worth noting that not all violent acts are negative. Violent acts motivated by self-defense, or defense of one's family, social group or culture are generally deemed as acceptable. Such violent acts may also be adaptive due to the protection of kin (Queller & Strassman, 2002; Smith, 1964) or, in the case of cultural violence, the advancement of one's cultural group and prestige for oneself, with consequential improvement of potential mating options. Although violent behaviors tend to carry significant risk of injury, at times they may be adaptive with the risks of *not* being violent greater than for engaging in violent behavior.

By contrast use of the term *extreme violence* specifically refers to violent behavior for which the risks outweigh potential benefits. Risks either of personal injury or to one's social esteem through disapproval, retaliation, or incarceration are of likelihoods greater than any anticipated benefits. Extreme violence then is rather synonymous with criminal violence. Yet because criminal codes vary from state to state, country to country and from one time to another, use of the term "criminal violence" may be too subjective to be truly meaningful.

2. Social science's resistance to evolution and genetics

It has been noted that social science, through much of the latter half of the 20th century, has focused more exclusively on "learning" explanations of behavior at the expense of biological explanations of human behavior (Buss & Shackelford, 1997; Okami & Shackelford, 2001). As one example, the American Psychological Association's brochure on youth violence states that "There is no gene for violence. Violence is a learned behavior…" (APA, 1996). The brochure later notes that genetically influenced factors including learning disabilities and impulsivity interact with learned violence. Yet, this initial line appears to suggest that there are no genetic alleles that increase violence risk. The statement itself is essentially a "straw man" by setting up a false argument. After all, there clearly is no *single* gene for violence. Furthermore, genes and environment certainly interact to produce behavior (Moffitt, 2005). Thus, the APA's pamphlet "shoots down" an argument few behavioral geneticists or evolutionary psychologists would be likely to argue. Similarly, it has been noted that the National Institutes of Health have historically de-emphasized genetic, evolutionary, or other biological studies of violence behavior (Enserink, 2000) although this trend may be slowly reversing (Glenn, 2008).

Critiques of biological theories of aggression are perhaps epitomized by Berkowitz (1993), who claimed that aggression is not linked to brain structures for aggressive instinct, and that aggression is provoked by external stimuli. Berkowitz appears to claim that aggression would be biological in origin only if it were univariate, purposeless, and unprovoked. Tooby and Cosmides (1992) argue that perspectives, such as Berkowitz's, are indicative of the "Standard Social Science Model" (SSSM), which postulates the brain as a general-purpose learning device, devoid of content at birth, with behavior solely a product of subsequent learning. As a consequence, much of 20th century social science had focused on "pitfalls" of modern life, such as media violence, toy guns and Western values, although violence and homicide rates are found to be high among non-advanced cultures without access to these modern accruements (Buss & Shackelford, 1997). Beliefs in the value of such environmental variables may persist dogmatically long beyond their empirical value. For instance, recent meta-analytic reviews of media violence have found their effects to be negligible (Ferguson & Kilburn, 2009; Savage & Yancey, 2008).

The recent reluctance of social science to embrace genetic and evolutionary explanations of behavior may be related to several phenomena. The first may be related to historical abuses of genetic explanations of human behavior to promote racism, sexism, eugenics, and the belief in racial differences in intelligence (Kamin, 1974). These concerns may be inflamed due to the occasional "just so story" by scholars purporting ostensible evolutionary explanations for a behavior that are not based on empirical evidence. Ramachandran's (1997) purposefully facetious "Gentlemen prefer blondes" satire of evolutionary psychology is one such example. Careless "just so stories" may promote the false belief that evolutionary psychology and behavioral genetics are not data-based.

Second, misunderstandings about evolutionary theory, evolutionary psychology, and behavioral genetics may increase resistance. Two common misconceptions include the "naturalistic fallacy" and biological hard determinism. The naturalistic fallacy is the belief (or fear) that if something is caused by biology, this provides moral justification for the behavior. In other words, "natural" behavior is equated with "morally desirable" behavior. Similarly biological hard determinism implies that human behavior is due only to genetic or other biological effects, and is not influenced by the environment, nor open to the effects of agency. However, evolutionary psychologists have indicated clearly that they do not endorse either the naturalistic fallacy or biological hard determinism (see Wilson, Dietrich, & Clark, 2003 for a discussion).

Finally, evolution and behavioral genetics may offer fewer practical solutions to a problem such as violence, in comparison to social learning explanations. Learned behavior can (presumably) be unlearned. However, genetic sequences cannot be ethically or practically altered. Yet, blinding research to the influence of genetic elements on behavior, by necessity, blinds science also to gene/environment interaction effects, which may offer some solutions for the reduction of negative behavior. Understanding the genetic influences on behavior, and identifying these genetic risks within individuals, may result in treatments that theoretically could be targeted early and preventatively toward individuals who may have this genetic risk.

In fairness, resistance to genetic and evolutionary theories appears to be slowly abating. Articles covering evolutionary psychology and behavioral genetics approaches to violence have appeared in leading criminological and psychological journals, including APA journals with increasing frequency (e.g., Caspi et al., 2004; Ellis, 1991; Ellis & Walsh, 1997; Larsson, Andershed, & Lichtenstein, 2006; Wright & Beaver, 2005). As such, the social science of violence may be in the process of self-correction. In all likelihood, dogmatic debates will

continue for some time, even as evidence in favor of genetic influences mounts, before genetics based research is more fully accepted.

3. Genetic polymorphisms associated with violence

A rich line of behavioral genetic research has analyzed samples of kinship pairs (e.g., twins) to estimate the proportion of variance in antisocial phenotypes that is due to genetic influences. Results of these studies, which have been based on thousands of sibling pairs, have pointed to the inescapable conclusion that genetic factors are implicated–at least to some degree–in the etiology of violence. Precisely how influential genetic factors are, however, is difficult to garner when examining single studies because heritability estimates wax and wane from study to study based on sample characteristics and methodological differences. A number of meta-analyses (Ferguson, in press; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002) and literature reviews (Moffitt, 2005) have thus been conducted as a way of summarizing the findings from these extant behavioral genetic studies. Overall, the conclusions reached by these studies have been highly consistent in showing that approximately 50% of the variance in antisocial phenotypes is the result of genetic factors.

The information gathered from this line of behavioral genetic research has been of utmost importance in establishing the genetic foundations to antisocial behaviors. At the same time, however, analyzing samples of kinship pairs is limited in that it cannot reveal precisely which genetic polymorphisms are implicated in the development of violence. A different research strategy–one that examines DNA sequences and their relation to violent phenotypes–is needed to address this line of inquiry. During the past decade, a rapidly growing body of research has tested for associations between measured genetic polymorphisms and various types of antisocial behaviors. Although this line of research is still in its infancy, a number of genetic polymorphisms have been identified as perhaps being involved in the etiology of extreme violence (Morley & Hall, 2003). Most of the genes thought to be related to extreme violence are involved in the detection, transportation, and breaking down of neurotransmitters, especially dopamine and serotonin.

Genes of the dopaminergic system have been a source of a considerable amount of research attention. Part of the reason for focusing on dopaminergic genes is because the dopaminergic system is part of the pleasure/reward system of the human body. Dopamine acts as a natural reinforcement because the release of dopamine generates euphoric feelings in the human body. As a direct result, behaviors that stimulated the release of dopamine are likely to be repeated again in the future. Eating, sexual intercourse, and the use of certain drugs, such as cocaine, all are associated with an increase in dopamine; hence they are repeated time and again. Dopamine levels, however, sometimes fall outside the normal range of variation and when they do, deleterious outcomes are often evident. For example, variation in dopamine levels has been tied to the development of psychosis, schizophrenia, bulimia, and depression. There is even some research indicating that high dopamine levels are associated with involvement in violent and aggressive acts (Niehoff, 1999; Raine, 1993). The studies revealing an association between dopamine levels and antisocial behavior were used as a springboard from which researchers hypothesized that dopaminergic genes might also be related to violence.

One dopaminergic gene that has been the focus of a number of studies examining violence is the dopamine transporter gene (DAT1). DAT1 is located on chromosome 5 and codes for the production of the dopamine transporter protein, which is partially responsible for terminating dopamine activity from the synapse. DAT1 has a polymorphism in the 3′ untranslated region of the gene that arises from a variable number of tandem repeats (VNTR) that can be repeated between 3 and 11 times. This polymorphism has been shown to affect genetic expression (Fuke et al., 2001); and some research has singled out the 10-repeat allele as coding for a dopamine transporter protein that is extremely efficient at removing dopamine from the synapse (Swanson et al., 2000). Consequently, researchers have identified the 10-repeat allele as the "risk allele" that is thought to increase violent, aggressive, and various other antisocial behaviors.

As Table 1 shows, empirical research has linked this polymorphism to criminal and delinquent behaviors. Of particular relevance are two recent studies–both of which used data drawn from the National Longitudinal Study of Adolescent Health (Add Health)–that document a link between DAT1 and violence. In the first study, Guo, Roettger, and Shih's (2007) analysis of the Add Health revealed that the 10R allele was associated with increased involvement in acts of violent delinquency among adolescents and young adults. Similarly, Beaver, Wright, DeLisi, and Vaughn (forthcoming), using a slightly different measure of violence, also found that the 10R allele conferred an increased risk of violence among males. Although replication studies need to be undertaken, these two pieces of research provide initial evidence that DAT1 may play some role in the commission of extreme violence.

Researchers have also examined whether dopamine receptor genes are associated with antisocial behaviors. In particular, two dopamine receptor genes–DRD2 and DRD4–have emerged as leading candidate genes for violence and aggression. DRD2 is located on chromosome 11 and is implicated in the production of D2 receptors, which are involved in the postsynaptic detection of dopamine. D2 receptors are highly concentrated in neurons found in the midbrain, the caudate, the nucleus accumbens, the amygdala, the hippocampus, and the cerebral cortex—areas of the brain that have been linked to violence and aggression (Wright, Tibbetts, & Daigle, 2008).

DRD2 is a polymorphic gene that contains two alleles: the A1 allele and the A2 allele. Existing research has indicated that carriers of the A1 allele are at an increased risk for various psychopathologies, including victimization (Beaver, Wright, DeLisi, Daigle et al., 2007), alcoholism (Connor, Young, Lawford, Ritchie, & Noble, 2002), and pathological gambling (Comings et al., 2001). Most applicable to the current review, however, are the studies examining whether A1 is related to extreme violence and aggression. Although the evidence is limited, it appears as though the A1 allele of DRD2 is associated with increased involvement in acts of serious physical violence and aggression (Beaver, Wright, DeLisi, & Walsh et al., 2007; Guo et al.,

Table 1
Genes associated with antisocial behaviors.

Gene	Functionality	Types of antisocial behaviors
Dopamine transporter gene	Codes for the production of a transporter protein that is implicated in the reuptake of dopamine	Crime, delinquency, violence
Dopamine receptor genes	Involved in the detection of dopamine at the postsynaptic neuron	Alcoholism, crime, delinquency, drug use, gambling
Serotonin transporter gene	Codes for the production of a transporter protein that is implicated in the reuptake of serotonin	ADHD, aggression, conduct disorder, nicotine dependence, violence
Catechol-*O*-methyltransferase gene	Codes for the production of the COMT enzyme, which is partially responsible for breaking down neurotransmitters	Aggression, violence
Monoamine oxidase A gene	Codes for the production of the MAOA enzyme, which is partially responsible for metabolizing neurotransmitters	Aggression, conduct disorder, violence

2007). These findings should be tempered by the fact that the dearth of studies bearing on the association between DRD2 and violence makes it difficult to draw any firm conclusions about the true nature of this relationship.

DRD4 is another dopamine receptor gene that has been identified as a likely contributor to violence and other antisocial behaviors (Rowe, 2002) and like DRD2, DRD4 codes for the production of receptors that facilitate postsynaptic detection of dopamine. DRD4 is found on chromosome 11 and has a polymorphism that arises from a 48 base-pair VNTR in the third exon. Although the alleles for this polymorphism can be repeated between 2 and 11 times, the 4-repeat allele and the 7-repeat allele are the two most common (Wang et al., 2004). This polymorphism has been found to be functional, where the 7-repeat allele codes for receptor proteins that are not as efficient at binding dopamine when compared to the receptor proteins produced by the 4-repeat allele (Kluger, Siegfried, & Ebstein, 2002). As a result, the 7-repeat allele has been identified as the risk allele for antisocial behaviors, including extreme violence and physical aggression.

A considerable amount of research has examined whether carriers of the 7-repeat allele are at-risk for various psychopathologies. Results of these studies indicate that DRD4 is related to ADHD (Faraone, Doyle, Mick, & Biederman, 2001), conduct disorder (Rowe et al., 2001), and gambling (Comings et al., 2001). Given that these types of outcomes covary significantly with violence and aggression, it is likely that the 7-repeat allele would confer an increased risk to serious physical violence. A study carried out by Schmidt, Fox, Rubin, Hu, and Hamer (2002) provides partial support for this possibility. This team of researchers examined whether DRD4 was associated with aggressive behaviors in a sample of young children. Results of their analysis revealed some evidence linking longer alleles (e.g., the 7-repeat allele) to maternal reports of aggression. There was no relation between DRD4 and observed aggressive behavior. Whether these findings would apply to violence committed by adolescents and adults remains an open empirical issue. It should be noted, however, that one study has found that DRD4 is associated with serious violence in adult males, but only for males who also possess the A1 allele of DRD2 (Beaver, Wright, DeLisi, & Walsh et al., 2007).

Genes from the serotonergic system have also been identified as being potentially involved in the etiology of extreme violence and serious aggression. Serotonin is a neurotransmitter that has inhibitory properties that act as the body's natural brake system. The release of serotonin works to modulate behaviors, dampen innate drives and instincts, and curtail impulsive behaviors. Given that extreme violence is often unplanned and spontaneous (Gottfredson & Hirschi, 1990), there has been a lot of interest in examining the precise role that the serotonergic system plays in the development of antisocial behaviors. A body of research has examined whether variation in serotonin levels corresponds to variation in behavioral problems (Raine, 1993). Although the findings have been mixed (Rowe, 2002), a relatively recent meta-analysis found a statistically significant and negative association between serotonin levels and extreme violence (Moore, Scarpa, & Raine, 2002). In other words, lower levels of serotonin were found to correspond with greater involvement in acts of extreme violence.

Against this backdrop, researchers have also examined whether genes involved in the functioning of serotonin are associated with antisocial behaviors. The most widely studied serotonergic gene–at least as it relates to behavior–is the serotonin transporter (5HTT) gene. The 5HTT gene is located on chromosome 17 and has a 43 base-pair insertion/deletion found in the 5′ regulatory region of the gene (Heils et al., 1996). This polymorphism, symbolized as 5HTTLPR, contains two groups of alleles: low expressing alleles and high expressing alleles. The 5HTTLPR polymorphism is functional, where the low expressing alleles have been found to suppress transcription of the serotonin transporter protein (Hu et al., 2006; Lesch et al., 1996). The end result is that carriers of the low expressing alleles could have diminished levels of serotonin available in the brain, which has led most researchers to conclude that the low expressing alleles are the risk alleles for antisocial behaviors.

A number of studies have documented a statistically significant association between 5HTTLPR and antisocial outcomes. For example, carriers of the low expressing alleles are at-risk for displaying ADHD symptoms (Cadoret et al., 2003), consuming large amounts of alcohol (Herman, Philbeck, Vasilopoulos, & Depetrillo, 2003), and having childhood conduct disorder (Cadoret et al., 2003). Of particular interest are studies that have examined the relation between the 5HTTLPR polymorphism and aggression and violence. Once again, there are a limited number of studies that have explored this topic, but there are two showing that the low expressing alleles are associated with increased involvement in aggressive acts in samples of children (Beitchman et al., 2006; Haberstick, Smolen, & Hewitt, 2006). This is particularly important because one of the best predictors of extreme violence in adolescence and adulthood is childhood aggression and conduct problems (Wright et al., 2008). Thus, it is quite possible that the low expressing alleles differentially set persons onto a violent antisocial pathway very early in the life course.

Two additional studies examined the effect that the 5HTTLPR polymorphism has on extreme violence in adults. In the first study, Retz, Retz-Junginger, Supprian, Thome, and Rosler (2004) analyzed the distribution of 5HTTLPR alleles in a sample of violent and nonviolent offenders. Results of their analysis revealed that the low expressing alleles were more prevalent among violent offenders than nonviolent offenders. This is a particularly compelling study because it showed that the 5HTTLPR polymorphism could be used to distinguish different types of offenders. In the second investigation, Liao, Hong, Shih, and Tsai (2004) also explored the nexus between 5HTTLPR and extreme violence. They analyzed genotypic data from a sample of Chinese males. Results of their analysis indicated that extreme violence was more common among males who carried the low expressing alleles. Collectively, these studies hint at the very real possibility that the origins of extreme violence may be partially tied to the 5HTTLPR polymorphism.

Other genes from the serotonergic system, including several serotonin receptor genes (e.g., 5HTR2A, 5HTRiB, and 5HTR2C) and the tryptophan hydroxylase (TPH) gene, have also been studied. Although there is some research linking these genes to antisocial behaviors, the small number of studies examining extreme violence and the inability to replicate some of the findings leaves the effects of these genes unresolved. Future researchers need to explore in greater detail whether these and other genes of the serotonergic system are implicated in the development of extreme violent behaviors.

The last set of genetic polymorphisms that have been hypothesized to relate to extreme violence are genes that are involved in metabolizing neurotransmitters. Two of these genes–the catechol-*O*-methyltransferase (COMT) gene and the monoamine oxidase A (MAOA) gene–have consistently been shown to relate to antisocial behaviors (Volavka, Bilder, & Nolan, 2004). The COMT gene is located on chromosome 22 and codes for the production of the COMT enzyme. This enzyme is partially responsible for breaking down neurotransmitters, such as dopamine, epinephrine, and norepinephrine, and thus plays a pivotal role in terminating the synaptic activity of certain neurotransmitters. The COMT gene has a polymorphism that arises from a single nucleotide difference. This polymorphism is functional, where one allele codes for the production of the amino acid methionine (i.e., the Met allele) and the other allele codes for the production of the amino acid valine (i.e., the Val allele). The Met allele, in comparison with the Val allele, is associated with lower COMT activity. Because COMT metabolizes neurotransmitters that are thought to be positively related to violence, the lower COMT activity associated with the Met allele points to the likelihood that the Met allele is the risk allele for antisocial behaviors.

The available research strongly suggests that carriers of the Met allele display more signs of violence and aggression, including

aggressive personality traits, when compared to carriers of the Val allele (Rujesco, Giegling, Gietl, Hartmann, & Möller, 2003). In one of the first studies to examine the effect of the COMT polymorphism on extreme violence, Lachman, Nolan, Mohr, Saito, and Volavka (1998) examined violence in a sample of schizophrenics. Their analysis revealed that patients with a history of extreme violent behaviors were more likely to carry two copies of the Met allele when compared to patients lacking a history of extreme violence. These findings have been upheld in the analysis of other samples of schizophrenic patients (Kotler et al., 1999; Strous, Bark, Parsia, Volavka, & Lachman, 1997). It should be noted, however, that in one study of schizophrenics the Val allele, not the Met allele, was related to an increased use of aggression (Jones et al., 2001). The precise reasons for these countervailing findings remain unknown. Thus, future researchers need to examine more fully the nexus between COMT and extreme violence.

MAOA is another polymorphic gene that is involved in the metabolism of neurotransmitters. The MAOA gene codes for the production MAOA, which is an enzyme that breaks down certain neurotransmitters, including serotonin and dopamine. This gene is located on the X chromosome and, as a result, males have only one copy of this gene while females have two copies. The MAOA gene has a polymorphism that is the result of a 30 base-pair VNTR in the promoter region of the gene. The alleles for this polymorphism are typically grouped into two categories: one group contains alleles that that correspond to low MAOA activity and one group contains alleles that correspond to high MAOA activity. Importantly, the low MAOA activity alleles are not as effective as the high MAOA activity alleles at metabolizing neurotransmitters. As a consequence, the low MAOA activity alleles are typically considered the risk alleles for various psychopathologies and extreme violence.

Initial evidence linking the MAOA gene to extreme violence in humans was discovered by Brunner et al. when they studied a Dutch kindred (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993; Brunner et al., 1993). Fourteen males from this family lineage were affected by an unknown disorder that was typified by borderline mental retardation, impulsive and abnormal behaviors, and, in some instances, serious physical violence. Interestingly, this disorder only affected males; females from the family were immune to it. Brunner and his colleagues sought to uncover the genetic factors that caused this disorder and they reasoned that since only males were affected, the gene would be found on the X chromosome. They performed genetic linkage analysis to test their hypothesis and they found that all of the males with this disorder had an MAOA gene that was malfunctioning and could not produce the MAOA enzyme.

Although Brunner's studies tied the MAOA gene to extreme violence, there are not any other documented cases of persons (outside of this single family pedigree) that have this mutated MAOA gene (Mejia, Ervin, Palmour, & Tremblay, 2001). More contemporary research, however, has examined whether variants of the MAOA gene are linked to violence. Most of the research has revealed that there is not a direct, main association between MAOA and antisocial behaviors. However, there is an impressive amount of research showing that the low MAOA activity alleles can increase violence and aggression in the presence of detrimental environmental conditions. To illustrate, in a landmark study, Caspi et al. (2002) examined the interrelationships among MAOA, childhood maltreatment, and antisocial phenotypes in a sample of males from the Dunedin Multidisciplinary Health and Development Study. Results indicated no main effect of MAOA on antisocial phenotypes; however, further analysis revealed that MAOA was associated with aggression and violence in males who had been maltreated as child. Although only about 12% of the sample had been maltreated and had the low MAOA activity allele, they were responsible for 44% of all the violent convictions in the cohort. Follow-up studies have since been conducted in an attempt to replicate this finding and the results of a recent meta-analysis indicated that across a range of studies, the association between MAOA and psychopathology is contingent on the presence of an adverse environment (Kim-Cohen et al., 2006). Taken together, the available evidence suggests that the MAOA gene is perhaps the one gene that is most consistently related to extreme violence.

4. An evolutionary approach to understanding violence

Among biologists, there is broad agreement that natural selection is the primary driving force, aside from mutation, for the selection of genes and the phenomenon of population genetics (Gottschalk & Ellis, 2009). Put simply, if a behavior provides organisms with a selective advantage, the genes that promote such behavior are more likely to be passed down to future generations of organisms. Although natural selection occurs at the individual level (although there is some debate about the appropriate level of evolutionary analysis, genetic, individual or species), for organisms of a particular species experiencing identical selective pressures, the result is often a general pattern of physical characteristics and behavior, although some variance between individuals typically remains. For members of the species that drift apart to dissimilar environments with differing selective pressures, the result can be gradual separation into subspecies and different species altogether. Charles Darwin's observation of the specialized beaks among subgroups of Galapagos finches provides one of the most famous examples of this phenomenon (Darwin, 1859). Among humans, living in diverse environments has clearly produced physiological differences in skin, hair and eye color, bone and facial structure, musculature, fat composition, etc. Similar behavioral differences due to living in diverse environments may form the foundation of what we understand as “culture” although there are likely more behavioral similarities across cultures than differences overall.

In order to understand the mechanism by which some humans become genetically at risk to extreme violence it is first important to understand the evolutionary and biological mechanisms of normal, adaptive aggression from which extreme violence stems. Although aggression is often thought of as “bad”, particularly by social scientists, there is considerable evidence that aggression in moderate doses is adaptive (Ferguson, 2008; Hawley & Vaughn, 2003; Smith, 2007). As noted earlier, possessing a modicum of aggression directs us toward increased social dominance and consequent reproductive success. Many activities that benefit from aggression in humans including sports participation, defense of young, active pursuit of school and career success, etc., are considered socially acceptable. Individuals lacking utterly in healthy aggression may be diagnosed with mental health conditions such as Avoidant Personality Disorder or Dependent Personality Disorder (American Psychiatric Association, 2000).

Although levels of aggression may vary somewhat from one culture to another, aggression is ubiquitous to the human species (McCall & Shields, 2008). Archaeological evidence from pre-historical human cultures reveals evidence of the use of fatal violence in these cultures (McCall & Shields, 2008). Humans' closest genetic relative, the chimpanzee, has been observed engaging in mass intergroup fatal violence (Goodall, 1979) and fatal abuse of infants (Goodall, 1977). Given that greater sexual competition exists among males (Gottschalk & Ellis, 2009), and that females are more invested in the care of young (Buss & Duntley, 2006), males engage in greater levels of aggression than do females, as is the case with most other mammalian species (Gottschalk & Ellis, 2009; Okami, & Shackelford, 2001). This *sexual selection* of male aggression and violence may also be related to the division of labor between males and females in prehistoric hunter–gatherer societies in which males typically undertook the riskier activity of hunting (Morris, 1999). Aggressive males are much more likely to attack unrelated children than they are their own children

(Daly & Wilson, 1994, 1996). The evidence that aggression is, in large part, the product of evolution thus comes from multiple sources.

- Molecular and behavioral genetics as discussed earlier in the article.
- Cross-species similarity comparison with other mammals including primates.
- Cross-cultural and cross-historical similarity.
- Sex differences in aggression are consistent across cultures, across history and in the expected evolutionary direction.

The evidence in favor of evolved aggression in humans is so strong that it is difficult to imagine that humans could have transcended evolution, yet revolved back to exactly the set of behaviors that would have been expected from an evolutionary perspective, but did so only through the coincidental non-biological process of socialization. This is not to say that the environment is inconsequential. Environmental stress and strain can serve as catalysts for aggressive behavior, with organisms becoming more aggressive under increased strain.

If aggression can thus be considered adaptive, evolutionarily derived behavior, extreme violent behavior can be understood as resulting from two primary mechanisms. The first is due to normal variation. In this sense extreme violence may be synonymous with melanism in the English pepper moth (Steward, 1977). Melanism was a rare coloring variation in the pepper moth which ultimately became adaptive once much of England's trees became darkened by domestic soot pollution. Although melanism wasn't initially adaptive (the dark color was easily visible against tree branches, alerting birds to a tasty snack), a small number of moths were nonetheless born with this extreme variation in coloration. Once the tables turned and tree branches were stained black by soot, the frequency of melanism among pepper moths skyrocketed, with lighter colors becoming rare. Once the pollution was cleaned, the frequencies reversed yet again. Similarly, although extreme violence is not currently adaptive, relatively small numbers of humans may be born with gene variations that place them at high risk for extreme violent behaviors.

The second mechanism is that adaptive aggression inhibition systems may be damaged due to head injuries or reduced in efficiency due to genetic variations (Beaver et al., 2009). Being able to restrain aggression to situations in which the benefit outweighs the risks is highly adaptive. Damage to the restraint system can impair this process. Put more simply, for pathologically violent individuals, either the aggression drive may be too strong, or the aggression inhibition drive too weak.

Lorenz (1963) presents one of the earliest evolutionary models for aggressive and violent behavior, although it was not specifically generalized to violent crime. According to Lorenz, aggression is a natural instinct or drive that accumulates over time, particularly in response to environmental stress. Lorenz advocated the general idea, ultimately associated with catharsis, that periodic releases of the aggression drive keep it to manageable levels, much as periodic orgasm helps diminish the sex drive, at least temporarily. The influence of catharsis has been difficult to observe in humans (Geen & Quanty, 1977), although others have argued that it has not been properly studied by social scientists (Kutner, Olson, Warner, & Hertzog, 2007).

Darwin's (1859) model of sexual selection is also helpful in explaining the preponderance of male involvement relative to female involvement in extreme violence cross culturally (Gottschalk & Ellis, 2009). As females have greater investment in the care of young, owing to the nine-month pregnancy and feebleness of the human infant and child, females are both relatively averse to high-risk activities and exert greater selective pressure over males in the traits they look for when selecting mates. This allows for somewhat parallel evolutionary pathways for males and females with different traits being selected in males, due to females' preferences, than are selected for in females themselves. Gottschalk and Ellis (2009) take this a step further and argue for an explanation of variance in male aggression and violence called the "Dads and Cads" model. Briefly, the authors argue that females select for traits in males that will increase the survivability of their own young. As such, females are more inclined to select mates who will assist with childrearing and provide resources for them and their offspring. Males with such traits are reproductively successful "dads". For those males lacking in such traits, the alternatives are lying, deceit, the violent elimination of the "dad" rivals, and violent rape of unwilling females. These "cad" strategies are higher-risk than the "dad" strategies and, as such, are less frequent in the population. Nonetheless they are successful enough, from a reproductive sense, to continue some frequency of the relevant gene alleles into the next generation. Thornhill and Palmer's controversial theory of male rape (Thornhill & Palmer, 2000) posits a fairly similar view.

Ferguson (2008) has presented an evolutionary model of violent behavior that describes the interaction of genetic and environmental influences on extreme violence. Referred to as the Catalyst Model, this model is presented in Fig. 1. Briefly, this model posits that extreme violence is the product of interactions between specific gene alleles and environmental abuse or neglect (e.g. Caspi et al., 2002). Most individuals possess either gene alleles that allow for normative levels of aggression, or are not exposed to physical abuse during their formative years. For those with both the at-risk gene alleles and a history of family violence exposure, the consequence may be a personality which is prone to extreme violent behavior.

Just as producing aggression when practical can be adaptive, so can restraining aggression be adaptive when the costs of aggression are high and the benefits low. The Catalyst Model posits that humans have evolved an "impulse control device" to limit expression of the aggressive drive (e.g., Lorenz, 1963). Low levels of self control have been found to be among the strongest predictors of violent crime

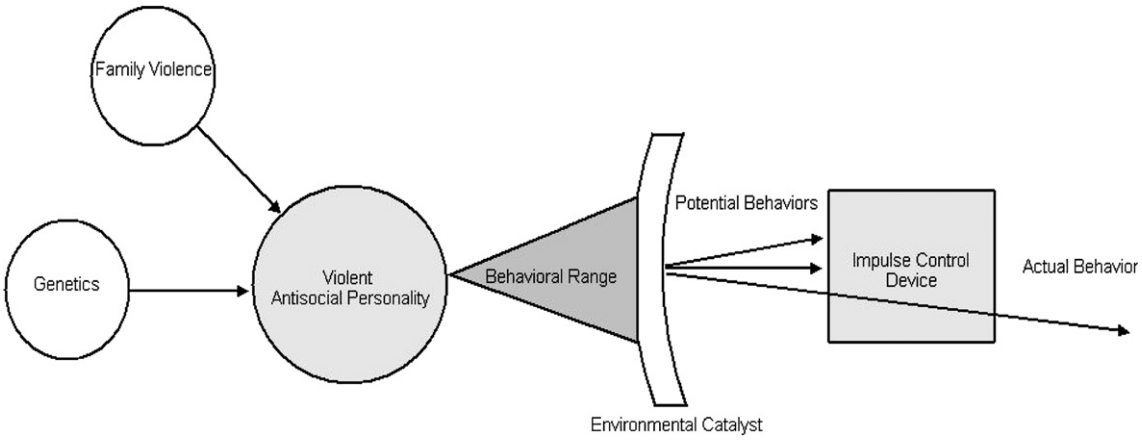

Fig. 1. A catalyst model for violent antisocial behavior.

commission (Pratt & Cullen, 2000, 2005; Séguin, Nagin, Assaad, & Tremblay, 2004). Self-control, like violent behavior, appears to be highly influenced by genetic factors which account for between 50–90% of the variance in self control (Beaver et al., 2009; Price, Simonoff, Waldman, Asherson & Plomin, 2001; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2003; Wright, Beaver, DeLisi, & Vaughn, 2008). This impulse control function is likely located in the frontal lobes of the brain. 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 extreme violent behavior (Brower & Price, 2001; Donovan & Ferraro, 1999; Mercer & Selby, 2005; Soderstrom et al., 2002). This impulse control device aids individuals in choosing how to respond to environmental strain. Individuals high in violent or antisocial personality traits are more prone to considering violent reactions to stress. Individuals with weakened impulse control may have difficulty restraining aggressive instincts when it would be appropriate to do so. Naturally, those individuals both high in violent and antisocial personality traits and low in impulse control will likely be most prone to extremely violent and high-cost risk taking behavior.

In short, the Catalyst model suggests that personality is shaped by a combination of genetics and learning, in which family or care-giving influences are predominant. People under stress seek out solutions for relieving that stress. Violent personalities are more likely to turn to violent solutions. People with intact impulse control will filter out more violent solutions in favor of lower-risk solutions when appropriate. Extreme violence, then, stems from too much aggression drive, too little impulse control or both in combination.

From these evolutionary perspectives several lines of reasoning emerge:

1) Extremely violent behaviors exist in the population to the extent that they are reproductively advantageous. Because they are higher-risk than moderately aggressive behaviors, extremely violent individuals are comparatively uncommon.
2) Greater female investment in young leads to greater risk-aversion among females, and hence less involvement in extreme violence.
3) Sexual selection by females and greater competition among males promotes male aggression. The separation of the sexes into "hunters" and "gatherers", both necessary for cooperative survival, has further ensconced male aggression as adaptive. Due to normal population genetics variations, some males (and females) will be at the extreme poles of the aggression continuum. Those at the higher pole for aggression are those most prone to extreme violent behavior.
4) Aggression and violence are both catalyzed by environmental stress and strain. The frequency of violent behaviors is likely to increase during times of environmental stress.
5) Because extreme violent behavior is high-risk, humans have evolved an impulse control device to limit high-risk violent behaviors. Individuals with damage to this impulse control device, located primarily in the frontal lobes of the brain, are at higher risk for engaging in extreme violent behaviors, whatever their pre-injury risk may have been.

Evolutionary explanations of violence are sometimes criticized for their "hopelessness" in that, if behavior is immutable, there is no hope offered by evolutionary psychology for behavioral change (Campbell, 2004). This is not precisely what an evolutionary model of violence offers, however. Understanding the evolutionary origins of extreme violence provides an understanding of the purpose of violence and the environmental stimuli that trigger such responses. Understanding and identifying those triggers provides the key to the practical applications of evolutionary theory. From behavioral genetics and evolutionary models of violence, we may more fully understand which individuals are at greatest risk for extreme violence. We can then begin to examine the interaction not only between genes and environmental catalysts for violence, but also the interaction between genes and *treatments* and *prevention efforts* for violence. This is the promise that evolutionary psychology may hold as it ultimately turns from treatment to outcome research. Future research on treatment outcomes for violence would benefit from evolutionarily and genetically informed models. The alternative appears to be to hold dear to what we wish to be true, rather than what is true.

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