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ARTICLE

# Aggressivity, Violence, Sociability and Conflict Resolution: What Genes Can Tell Us

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## Abstract

Conflicts are inherent to the human condition, as they are for all living beings. Disputes about resources or access to mating partners are among the most common causes of conflict. Conflict is herein defined as a struggle or contest between individuals or parties, and may involve a variety of aggressive behaviours. In humans, aggressiveness, violence and conflicts, including individual predisposal to conflict resolution, have traditionally been said to have deep cultural roots, but recent research in both neuroscience and genetics has shown the influence of genes on such complex behavioural traits. In this paper, recent data on the genetic aspects of these interrelated behaviours will be put together, including the effects of particular genes, the influence of stress and gender on gene regulation, and gene–environment interactions, all of which may influence biological predisposal to conflict resolution. Other genetically influenced behavioural aspects involved in conflicts and conflict resolution, such as sociability, will also be discussed. The importance of taking into account genetic and biological data to provide strategies for conflict resolution will be highlighted.

## Keywords

behaviour heritability, aggressiveness, sociability, conflicts, neurotransmitters

## INTRODUCTION: GENETIC VARIANCE AND HERITABILITY OF AGGRESSIVENESS

Aggressiveness is a prominent behavioural trait in most animal species including humans, the expression of which must be carefully modulated to assure the success of individuals, small groups and large societies. As it has so many facets, aggressiveness has been studied from a wide variety of disciplines including sociology, anthropology, biology and pharmacology. When taken together, the vast body of work produced to date indicates that the modulation of aggressiveness is influenced by a complex set of biological, psychological and social variables.

In the field of genetics, in order to discuss gene contribution to a particular character or trait, a certain degree of heterogeneity in the expression of that character within individuals in the analysed population must first be demonstrated. Thus, in order to discuss the genetic and biological contributions to human aggressiveness, it has to be acknowledged that the human population is not homogeneous with respect to the development of aggressive behaviours (Eley et al., 1999, 2003). That is, some individuals may be more prone to aggressiveness or may show different thresholds to violent behaviour. This does not imply that these complex traits are solely determined by particular gene variants, but that some gene variants (alleles) of particular genes have different effects on the overall threshold for aggressiveness and predisposal for conflict resolution, in conjunction with other genes and multiple cultural and educational factors.

For students of conflictology unfamiliar with genetics, it should be noted that a gene is a segment of DNA coding for the synthesis of a particular protein, which in turn has a specific biochemical function in the organism, thus conferring a particular character. Complex traits,

however, such as behavioural traits, are usually influenced by a number of different genes, each contributing to the final character, as well as by the environment. Within this general scheme, each gene may exhibit several different alleles coding slightly different genetic messages, leading to variations in the corresponding trait. Alleles for a particular gene may differ in their coding sequences, producing slightly different proteins that in turn confer a particular trait, and/or they may differ in the non-coding sequences that regulate the expression of that gene, conferring slightly different manifestations of the trait despite exhibiting exactly the same corresponding protein. A discussion of the particular mutations that generate the sets of alleles of the genes discussed below is beyond the scope of this paper but can be found in the cited literature.

Coming back to human population heterogeneity, and in order to demonstrate the genetic contribution to this heterogeneity, the variance and heritability of aggressive behaviours have to be quantified. In genetics, variance measures individual deviations from the mean of the analysed trait, which is mathematically calculated as the mean of squares of individual deviations from the mean. Heritability measures the proportion of total phenotypic variation in a particular trait, at the population level, that is attributable to variation in the genotype. Phenotype refers to the visible, or otherwise measurable, physical or biochemical characteristics of an organism, and the genotype is the precise genetic constitution of an organism; the phenotype results from the interaction of genotype and environment.

In this regard, a detailed evaluation of a meta-analysis of 24 genetics studies concerning aggression concluded that heritability accounted overall for about 50% of the variance of the trait – 44% from genetic effects and 6% from shared environmental factors. The remaining 50% of variance can only be explained by unshared environmental factors (Rhee and Waldman, 2002). From these results it can be concluded that particular genotypes account for some of the individual differences with respect to aggressiveness. It has also been demonstrated that this heritability changes with age; whilst genetic factors and a common environment are equally important in childhood, heritability becomes even more prominent in adulthood (Miles and Carey, 1997).

Another approach that was used to demonstrate the overall contribution of genes to aggressiveness involved the generation of several different rat strains by 50 consecutive selective reproduction cycles, in which, for each generation, individuals showing higher and lower aggressive responses were selectively mated. Comparison of the basal aggressive behaviours between strains under the same environmental conditions clearly showed the heritability of this trait (Rebollo-Mesa et al., 2010). Moreover, experiments in which a particular gene named MAOA (see below) was knocked out in mice (as spontaneously occurs in some humans), causing lack of function of the corresponding gene, showed the contribution of some of its alleles to impulsive aggressiveness (Scott et al., 2008). Thus, as individual differences in aggressiveness have an important genetic basis, in order to analyze its involvement in human conflicts as well as to increase our understanding of conflict resolution strategies, it is also necessary to consider this trait from evolutionary, physiological and genetic points of view, alongside its cultural, social and educational roots.

## AGGRESSIVENESS AND SOCIABILITY AS AN EVOLUTIONARY ADAPTATION

The presence of genes and their variants in populations is the result of long and complex evolutionary processes. They include fortuitous mutation events that generate alleles and new genes, as well as natural selection, not only within our current species but also in our ancestors and the ancestral species of our lineage from which we have inherited our genetic constitution. The various facets of human aggression, as well as of sociability, a behavioural trait of which the genetic contribution will be discussed latter, should be considered as a balance between traits that are evolutionarily advantageous in a competitive world based on social cohesion.

Aggression, particularly for males living in communities, as analysed in a wide range of model species including zebra fish, snakes, dogs, cats, elephants, mice, rats and primates, including humans, provides a competitive edge in securing resources and in intra-sexual competition through combat. High levels of aggression may also compensate for a lack of physical prowess in establishing hierarchy and dominance with respect to reproductive success, one of the key aspects in evolutionary processes.

In females, aggression can help to protect offspring against a range of threats. As this behaviour represents a high-risk strategy, due to the associated possibilities of injury or death, there is a high likelihood of both potentially positive and negative selective discrimination throughout evolution, which may explain why human aggression appears to have a strong genetic underpinning (Maynard Smith et al., 1988). Interestingly, with respect to aggression as a response to threat – reactive aggression –there appears to be a complex relationship in mammals between anxiety and aggression, and strong evolutionary conservation of the brain regions involved in both, including the amygdala circuitry, the anterior cingulated cortex and regions of the prefrontal cortex on the other hand (Lesch, 2005).

## AGGRESSIVENESS AND GENDER

It appears that heritability of aggressiveness is also related to gender, although the situation in humans is not completely

clear and there are conflicting data. In humans, very clear cut distinctions between sexes have been made on the basis of crime and a wealth of studies has demonstrated a correlation between testosterone levels and aggression (Archer, 1991), but contradictory observations have also been made (Turner, 1994).

More recently, a meta-analysis of 45 independent studies, reflecting a range of both positive and negative correlations, provided support for an overall weak positive effect of testosterone levels on human aggressiveness (Book et al., 2001). Differences between males and females are likely to be due to the different evolutionary aggression strategies, which result in different proactive and reactive responses in terms of level and frequency, based on the interaction of hormones and neurotransmitters with their receptors and in the subsequent signal transduction mechanisms, as well as epigenetic modifications (see below). Interpretation of the relationship is, however, complicated in humans, not only because of methodological problems but also because of fluctuations in hormone levels in response to environmental conditions and circadian rhythm. Age is also a confounder, with regard to both the changing response and hormone levels.

Despite this, male-associated aggression suggests the possible involvement of genes for androgen synthesis and function, possibly via the Y chromosome, as it contains the gene involved in triggering male development and male-hormone synthesis (named *Sry*). A tendency has been observed in healthy Swedish males for a correlation between particular alleles for the androgen receptor gene and muscular tension and verbal aggression (Jonsson et al., 2001), along with a significant correlation with respect to violent criminal activity in Indian males (Rajender et al., 2008).

However, it appears that not only the Y chromosome is important in contributing to aggression through its role in male determination, but that other genes are also involved. Thus, if the normal male-determining *Sry* gene is deleted from the Y chromosome in mice and is subsequently provided as an autosomal copy, the strains created vary in aggressiveness (Gatewood et al., 2006). This indicates that other candidate genes are required in order to study this trait from the genetic point of view.

## THE SEROTONINERGIC SYSTEM: SOME CANDIDATE GENES FOR AGGRESSIVENESS

Research pointing to the key role of the neurotransmitter serotonin in aggression suggests that the genetic control of all aspects of serotonin metabolism, and particularly of its synthesis, its release from neurons, and its action via the various receptors, represents a rich field for the selection of candidate genes involved in aggressiveness. One aspect of this is the observation that the level of arginine vasopressin (AVP), a peptide hormone in the cerebrospinal fluid (CSF) within brain ventricles, correlates with the life history of aggression, and that a reduced level of the serotonin metabolite, 5-hydroxy-indole acetic acid (5-HIAA), within this fluid is associated with violent behaviour (Coccaro et al., 1997, 1998). These molecules are also present in human and rodent CSF during embryonic and foetal development (Parada et al., 2005 and 2007; Zapaterra et al., 2007; Bueno et al., personal communication), possibly contributing to neural pathways for later behaviours. However, the complex composition and the dynamics of CSF make it difficult to determine whether the levels of these gene products are the cause of aggressive behaviour or the consequence, which may be reflected in the composition of the CSF.

Other known candidate genes, reported to be involved in the regulation and/or heterogeneity of aggressiveness, that are also related to serotonin pathways are the serotonin transporter SLC6A4, the monoamine oxidase enzymes (MAOA and MAOB), tryptophan hydroxylases (TPH1 and TPH2), and the serotonin receptors 5-HT1A and 5-HT1B. MAOA and MAOB are two closely related enzymes that play an important role in the metabolism of biogenic amines in the central nervous system, including serotonin, norepinephrine and epinephrine (MAOA) and dopamine (MAOB). An important watershed of the candidature of MAOA followed the above mentioned correlation of null mutations and aggression in both mice and humans (Brunner et al., 1993; Cases et al., 1995). More recently, mutations that modify its level of expression have also been associated with aggression and violence (D'Souza and Craig, 2008), although the results show some inconsistencies due to the complex interactions between functional alleles, exposure to environments and sex hormones.

It has been reported that maltreated males are significantly more likely to develop antisocial behaviour if they had MAOA alleles exhibiting low activity (Caspi et al., 2002), and that the high activity variants conferred some protection against a stressful and abusive childhood (Foley et al., 2004; Nilsson et al., 2006; Widon and Kim-Cohen, 2007). Moreover, a significant correlation between MAOA alleles exhibiting high expression and lower impulsivity has been found, suggesting a complementary role of this gene in the heterogeneity of aggressiveness: MAOA may act on impulsivity, and subsequently on aggressive behaviour (Huang et al., 2004, Nilsson et al., 2007). More recently, a significant interaction between exposure to moderate trauma and the low activity of MAOA, with respect to aggression scores, has been confirmed, although exposure to extreme levels of trauma results in high aggression scores regardless of the gene variant for MAOA expression (Weder et al., 2009).

However, results from studies relating functional variants of MAOA and female behaviour are intriguing. For example, it has been reported that girls with alleles that show high rather than low activity appeared to be at risk of engaging in criminal behaviour in the presence of psy-

chosocial risk (Sjoberg et al., 2007). There is evidence that MAOA transcription may be regulated by both androgens, including testosterone, and glucocorticoids, linking aggressiveness with both gender and stress (Craig, 2007; Ou et al., 2006; Sjoberg et al., 2008). These results make it increasingly obvious that it is necessary to consider the impact of genes not in isolation, but as part of a multifactorial miasma including other genetic factors and the environment.

With respect to other genetic factors that are related to the serotonin pathway mentioned earlier, an interaction between the impact of high adversity in childhood on later-life violence has been reported, though only in people exhibiting low activity alleles for the serotonin transporter SLC6A4 (Reif et al., 2007). On the other hand, as the action of serotonin is mediated by a range of receptors, e.g. 5-HT1A and 5-HT1B, these receptors should also be considered as candidates for generating heterogeneity in human aggressiveness. It has been demonstrated that up-regulation of these receptors in rats is associated with high levels of aggression, behaviour that is further enhanced following victorious aggressive experiences (Caramaschi et al., 2007). In humans, more than 20 correlation studies linking gene variants of these receptors with aggressiveness and antisocial behaviours have yielded varying results (Sanders et al., 2002), ranging from antisocial alcoholism (Hasegawa et al., 2002; Lappalainen et al., 1998; Soyka et al., 2004) to pervasive aggression in children (Davidge et al., 2004), and including a range of behaviours such as psychosis, agitation, aberrant motor behaviour and depression (Prichard et al., 2008).

Finally, alleles for the tryptophan hydrolase 1 and 2 (TPH1 and TPH2) enzymes, involved in serotonin synthesis in the brain, have also been associated with lower levels of 5-HIAA in the CSF in control males, but not in females (Nielsen et al., 1994), producing higher scores for aggression and a tendency to experience unprovoked anger (Henning et al., 2005). In mice, an allele of TPH2 has also been associated with inter-male aggressiveness (Kulikov et al., 2005; Osipova et al., 2009). These results indicate the participation of gene variants in aggressiveness and violence regulation and heterogeneity, although as part of the multifactorial miasma, as argued above.

## CANDIDATE GENES IN THE STRESS RESPONSE PATHWAY

As discussed in terms of its relationship with aggressiveness, the influence of MAOA also depends on stress levels. Two separate elements exist in the stress pathway: the neuroendocrine stress response and the autonomic reaction to stressful situations (commonly typified by the *fight or flight paradigm*). Thus, genetic factors interacting with these systems are also candidates for regulating aggressiveness and violence in conflicts. For example, dopamine-beta-hydroxylase (DBH), a key enzyme in the synthesis of norepinephrine, may underpin aspects of antisocial behaviour, including increased neuroticism scores and impulsive and/or aggressive behaviour (Hess et al., 2009).

Another candidate gene is catechol-O-methyl transferase (COMT), an enzyme involved in dopamine and epinephrine metabolism. A lack of COMT function in mice leads to increased aggressive behaviour but only in males (Gogos et al., 1998). In humans, some authors have found a correlation between a particular COMT allele and increased aggressiveness, particularly for males, and frequently associated with schizophrenia. In women, however, this allele correlates with the least aggressive behaviour (Kulikova et al., 2008). The dopamine receptor DRD2 has also been associated with stress disorders, and a particular allele of DRD2 has been recently associated with higher risk of posttraumatic stress disorder in war veterans (Voisey et al., 2009). In rodents and monkeys there is also a positive relationship between noradrenergic activity and fighting/biting behaviour, and a positive relationship between aggressiveness and the CSF level of norepinephrine or its metabolites is also found in most human studies (Craig and Halton, 2009; Placidi et al., 2001).

## OTHER CANDIDATE GENES

Other genes have been associated to aggressiveness and violence. This includes genes encoding the enzyme, nitric oxide synthase (NOS1), recently associated with traits relating to impulsivity, including hyperactive and aggressive behaviour (Reif et al., 2009), and the arginine vasopressin receptor (AVPR1A), whose ligand, arginine vasopressin, has been mentioned above as being implicated in aggressiveness (Ferris et al., 1997). In humans, this receptor has been associated with social communication and autistic traits, but no direct relationship with aggression has yet been demonstrated (Bachner-Melman et al., 2005).

Various physiological changes can also promote increased aggressive behaviour. One of the most analysed is the lowering of sugar levels in the blood, which provokes fatigue, dizziness, headaches and irritability. Various studies have attempted to link serotonin mechanisms, insulin levels and glucose metabolism with aggression and impulse control (Linnoila and Virkkunen, 1992; Virkkunen et al., 2007). A variation in the serotonin transporter gene was found to affect nutritional intervention on fasting blood glucose levels in non-diabetic females (Yamakawa et al., 2005). Furthermore, glucose transporters are also thought to have an affect on hypoglycaemia and the resultant behaviour. This is supported by a familial study, in which six out of the eight family members were found to have a mutation in the glucose transporter SLC2A1 gene, resulting in irritability and impulsive behaviour (Weber et al., 2008).

## THE ROLE OF EPIGENETICS IN AGGRESSIVENESS AND VIOLENCE

New findings in epigenetics have to be taken into account, as these may help to link environmental conditions with genetic traits and explain the apparently contradictory gender-dependent influence of some alleles. Epigenetic modifications of the genetic material consist of biochemical modifications of particular DNA nucleotides and/or of some of the proteins involved in chromatin structure, such as histones. These modifications, which under certain conditions are reversible and are genetically controlled, may affect the regulation of gene expression.

It has been reported that rat pups deprived of maternal care exhibit an epigenetic modification affecting the glucocorticoid receptor promoter, a particular methylation, which subsequently produces behavioural deficits and increased inter-male aggressiveness (Weaver et al., 2004; Veenema et al., 2007). In humans, an analogous epigenetic methylation of the glucocorticoid receptor promoter has been associated with violent suicide victims who had experienced adverse events early in life (McGowan et al., 2009). Similarly, rat pups raised by stressed mothers exhibit increased epigenetic methylation of the gene coding for the brain-derived neurotrophic factor (BDNF), a neural growth factor in the brain's prefrontal cortex, which tends to reduce the level of BDNF. It has also been reported that this methylation pattern is passed on to the subsequent generation (Roth et al., 2009).

Interestingly, epigenetic methylation of the MAOA promoter region has been reported in females but not in males; a difference that may be related to the different aggressive responses related to gender (see above), although the extent of this remains to be elucidated (Pinsonneault et al., 2006). Epigenetics is a new field that could contribute to our understanding of the association between genetics and beha vioural aspects such as aggressiveness and violence, linking the environment with the expression of particular genes.

## SOCIABILITY

Although most of the results discussed in this paper refer to aggression and violence, conflicts may be caused by a variety of aggressive behaviours which, in turn, complicate its resolution, so these aggressive behaviours have to, at the least, be taken into account. This is just the "tip of the iceberg" as there are many other behavioural traits that may have some genetic basis, as they originate in the brain, a biological structure that develops and works under the direction of gene networks. These gene networks propitiate neural plasticity, which also depends on a number of environmental factors – although these will not be discussed here. The other behavioural traits include an inclination to many different degrees of optimism, empathy, sociability, etc.

For example, it is known that the level of neurotransmitters such as serotonin and dopamine is related to depression, and that some pharmacological treatments for depression act on these neurotransmitters. Thus, different alleles of the relevant genes may result in different degrees of optimism in different individuals. Moreover, different alleles of a gene called VMAT2, whose protein product is involved in packing neurotransmitters such as serotonin and dopamine within neurons, have been found to be associated with human spiritual capacity (Zimmer, 2004), which in turn influences many behaviours.

Twin and family studies suggest that uniquely human characteristics such as empathy, altruism, a sense of equity, love, trust, music, economic behaviour, and even politics are partially hardwired (reviewed by Epstein et al., 2010). The leap from twin studies to identifying specific genes engaging the social brain has occurred in the past decade, aided by new insights on social behaviour in lower mammals. Remarkably, genes such as the arginine vasopressin receptor and the oxytocin receptor contribute to social behaviour in a wide range of species from voles to humans (Donaldson and Young, 2008). Several studies have allowed the heritability of social behavioural phenotypes to be determined for a number of traits (Table 1). This implies that although most of the genes and alleles involved in such traits may still be unknown, or at least much less known than those for the abovementioned serotoninergic pathway that is associated with aggressiveness, research in these areas is of vital importance to fully understand conflicts, and to more effectively apply conflict resolution.

**Table 1.** Heritability of social behaviour traits. Modified from Ebstein et al. (2010)

| Trait | Genetic effects | Shared environment effects | Unshared environment effects |
|---|---|---|---|
| Prosocial behaviour (boys) | 57% | 12% | 31% |
| Prosocial behaviour (girls) | 55% | 4% | 41% |
| Psychosocial stress (males) | 57% | 12% | 31% |
| Infidelity (females) | 50% | 0% | 50% |
| Social responsiveness (boys) | 50% | 25% | 25% |
| Social responsiveness (girls) | 39% | 42% | 19% |
| Empathy | 47% | 0% | 53% |
| Political attitudes | 42% | 23% | 35% |
| Aggression | 40% | 17% | 43% |
| Leadership | 40% | 0% | 60% |
| Parental warmth | 38% | 0% | 62% |
| Risk | 37% | 0% | 63% |
| Secure attachment | 37% | 0% | 63% |
| Dictator giving | 30% | 0% | 70% |

## CONCLUSION

The impressive amount of data available demonstrates that genes play a role in human conflicts: the variety of aggressive and violent behaviours that derive from as well as provoke them. For this reason, and to develop strategies for conflict resolution, it is necessary to consider the impact of genes as part of a multifactorial miasma, including other genetic and epigenetic, as well as environmental factors. In other words, it is necessary to consider not only the cultural, social and educational environment, but also the genetic and other biological factors related to evolutionary human traits such as aggressiveness and violence, sociability and empathy, altruism and optimism.

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