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Gender differences in violence and aggression – a neurobiological perspective

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Abstract

Violence is a ubiquitous phenomenon, which has been part of the experience of humanity since its inception. Violence has classically been viewed as being associated with being male. In general population, men are reported to commit violent acts significantly more frequently than women. As the interdisciplinary research data point to, violence is a complex phenomenon that could be approached from different perspectives, such as social, economic, political, religious, biological, and genetic. We hereby provide a review of the neurobiological mechanisms underlying the gender distinctions in violence and aggression in both general and psychiatric population.

Keywords: antisocial personality disorder; conduct disorder; theory of mind; epigenetic; genetic polymorphism

1. Introduction

Violence and aggression have traditionally been viewed as being associated with being male. This long-standing lay perception has been supported by several studies which have shown that in general population males perpetrate violent acts approximately ten times more frequently than females (Tardiff & Sweillam, 1980). The gender differences in violence persist among patients with psychiatric diagnoses, although in a more attenuated form (Staniloiu & Markowitsch, in press). They could have various underpinnings (as biological and socio-cultural). Several hypotheses for the neurobiologically-mediated gender variations in violence have been advanced. One assumption is that they are linked to genetic or hormonal factors (Strüber, Lück, & Roth, 2008). This explanatory avenue was suggested by developmental studies of the trajectory of aggression, which revealed that sex differences in the level of aggression become obvious at an early age. These studies have shown that the so-called "normative" or

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"developmental" physical aggression in the form of biting, kicking or hitting is a common behaviour in childhood, which starts around one year of age and arguably peaks around 3.5 years of age (Tremblay, 2008). It decreases afterwards, in the context of the emergence of social skills and language abilities, being partly channelled to other forms aggression, such as verbal aggression and being modulated by both child's genetic predispositions and environmental (parental) variables. Additional evidence for sex differences in aggression comes from epidemiological studies of conduct disorder. Conduct disorder (DSM-IVTR, 2000) is a serious mental disorder of childhood or adolescence that features a long-standing pattern of rules' and laws' violations. It may lead to antisocial personality disorder, a condition that is diagnosed after 18 years of age. The prevalence of conduct disorder in children and adolescents was estimated to range between 6 % and 10 % (Staniloiu & Markowitsch, in press). This disorder was found to afflict males twice more frequently than females (Staniloiu & Markowitsch, in press). Given that seven out the fifteen DSM-IVTR (2000) criteria for conduct disorder code for physical aggression, the rates of conduct disorder were opined to provide indirect indications about the frequency of pathological physical aggression in children and teenagers. While these numbers indeed might reflect a true higher risk for violence and antisocial behaviours in males in comparison to females, this risk may be spuriously overestimated due to a deficiency in the gender sensitivity of the DSM-IVTR diagnostic criteria for conduct disorder (Kjelsberg & Friestad, 2009).

2. Genetic, epigenetic and gender variations in liability to violence and aggression

A slighter heritability of physical aggression was reported in males in comparison to females by some studies, but not all (Brendgen et al., 2005). Quantitative genetic studies typically estimate the heritability of physical aggression as being around 50 % (ranging from 40 % to 80 %, depending on methods and subjects), underscoring that this complex behaviour is underlain by gene-environment interplays.

Several gene variants were postulated to increase the susceptibility for violent behaviour (Craig & Halton, 2009). Except for a minority, these variants are common in general population and are not solely implicated in the causal string leading to violence, but together with other genes and interacting environmental factors: social, economical, familial etc. The link between XYY genotype and aggression is still a source of heated debate, although a significantly higher prevalence of XYY genotype among the sexually motivated homicide perpetrators than among other male offenders was relatively recently reported (Briken, Habermann, Berner, & Hill, 2006). An androgen receptor gene variant was found to be associated with violent crimes in a group of convicted Indian males (Rajender et al., 2008). The investigation of the relationship between aggressive behaviours and testosterone levels yielded inconsistent results (Staniloiu & Markowitsch, in press). One study showed an association between testosterone saliva levels and aggression in delinquent males with low saliva cortisol level, but not with high cortisol level, advancing therefore a role for the cortisol in mediating the observed testosterone-overt aggression relationship (Popma et al., 2007). Several abnormalities of neurotransmitters (serotonin, but also dopamine, norepinephrine and acetylcholine, glutamate, gamma amino butyric acid and nitric oxide), neurohormones (such as arginine-vasopressin, oxytocin and melatonin) and neuromodulators (opioids) have been linked to violence and aggression. The genes of monoamine oxidase A (MAOA) enzyme (that metabolizes serotonin, which is involved in impulsive aggression and violence) and androgen receptor are located on X-chromosome. The hemizygous state of male carriers for X-linked genes increases their susceptibility for certain X-chromosome-linked disorders, as it is the case with a rare mutation of the MAOA gene that causes a functional knockout of the gene in affected men, leading to impulsive violent behaviours (Brunner et al., 1993). The promoter polymorphism associated with lower expression of the MAOA gene (MAOA-L) is common in general population, but was found to predict higher rates of impulsive violence in New Zealender Caucasian male carriers only in combination with a history of early

life severe maltreatment (Caspi et al., 2002).This suggests an epigenetic effect, which may be both hormone- and experience-mediated and may take place during a particular developmental window of vulnerability. Indeed, both animal and human data point nowadays to the role of early life experiences in modifying gene expression via epigenetic mechanisms (McGowan et al., 2009). Several epigenetic mechanisms might enable environmental factors (including maternal rearing practices) to produce modifications of gene expression, which occur in the absence of a change in the underlying DNA sequence and may a times be transmitted to the next generations (Staniloiu and Markowitsch, in press). These epigenetic mechanisms include DNA covalent changes (methylation) and post-translational modifications of the histone tails. In women, random X-chromosome inactivation or epigenetic methylation of the promoter region of the MAOA gene may compensate for a double allelic load. A protective effect against genetic factors that enhance liability for violence may be provided in women by estrogens. Estrogens could affect MAOA gene transcription and an inverse relationship between MAOA brain levels and estrogens blood levels exists. The presence of a particular genetic polymorphism might lead to subtle molecular abnormalities, which, in conjunction with other genetic, hormonal and environmental factors, might influence physiological processes, synaptic plasticity and information processing, which may in turn predispose individuals to violent behaviour. Employing structural and functional magnetic resonance imaging in normal subjects, Meyer-Lindenberg et al. (2006) showed for example that MAOA-L carrier status was associated with gender-differentiated morphological and functional changes of brain regions where sex hormones are expressed, such as structural changes of amygdala, cingulate cortex, orbitofrontal cortex and hyperactivity of the amygdala during emotional tasks and reduced regulation of the amygdala by orbito-frontal and anterior cingulate cortex.

3. Violence and the sexually dimorphic brain

Sexual brain dimorphism resulting from differential prenatal exposure to sex hormones may partly account for gender differences in theory of mind functions, empathy and altruistic cooperativeness and risk taking behaviour. Evidence indeed points to biological underpinnings of sex differences in these capabilities, such as increased recruitment of mirror system in women in comparison to men during tasks tapping on empathy (Schulte-Rüther et al., 2008), larger relative total gray matter volume in women correlating with higher cooperativeness and altruistic behaviour (Yamasue, Kuwabara, Kawakubo, & Kasai, 2009), distinct functional neural correlates during risk taking tasks (Lee et al., 2009), gender differentiated effects of so-called prosocial peptides, such as oxytocin and arginin-vasopressin/AVP (Thompson et al., 2006). Cognition and behaviour are also shaped by life experiences and environmental influences, which, depending on the degree of brain's plasticity, can impact on brain function and structure as well. Parental rearing of girls versus boys may differ, especially across cultures. Traditionally, girls have been more often educated to look after others. Also, mothers tend to engage in more detailed reminiscing about emotional events with their daughters in comparison to their sons, which might facilitate the unfolding of the female capacities for episodic-autobiographical memory as well as for perceiving and getting attuned to the inner world of others. Women are more likely than men to display a self-focused ruminative style (Nolen-Hoeksema, 2000), while men tend to employ more distracting strategies. Women might therefore be more prone to engage in inward directed forms of aggression, in comparison to men who might choose more outward directed ways of aggression.

4. Gender differences in violence and psychiatric disorders

Psychiatric illnesses could impact on the biological factors that confer protection against violence in women and subsequently narrow the gender variance observed in general population (Bradford, 2008).

Women who suffer from psychiatric illness have higher rates of violence than women in general population (Stueve & Link, 1998). However, underestimations of the rates of violence and aggression in women in general population might exist. The targets of female violence are often family members and violent behaviours of women may lead to less physical injury in comparison to those of males. This may have as result an underreporting of female violent acts (Odgers, 2008).Furthermore, women may be more inclined than men to resort to verbal aggression as well as covert forms of social aggression, such as gossiping or spreading rumours with the intent to cause harm (Björkqvist, Lagerspetz, & Kaukiainen, 1992).

5. Conclusions

Gender differences in violence are a real phenomenon, which has a variety of neurobiological and socio-cultural underpinnings. A crucial task for future research is describing valid environmentally mediated factors that interact with biological factors to increase the risk for individual violent behaviour. Within this task, gaining further insights into the neurobiological underpinnings of gender differences in violence might prove to be a central piece for the development of optimal preventive interventions that timely target the liability for individual human violence in the future.

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