Human Aggression's Social Neuroendocrinology

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Introduction

Human research on the neuroendocrine basis of violent behaviour has traditionally followed a one-way approach, focusing on the function of testosterone in fostering aggression. In response to and in anticipation of competitive and aggressive interactions, testosterone levels fluctuate fast. Some researchers believe that acute testosterone fluctuations (rather than baseline testosterone concentrations) are more significant to our knowledge of individual differences in aggressive behavior.

Human Aggression and Testosterone Levels

Aggression is defined as any behaviour aimed at damaging or injuring another living being who is trying to avoid it.

The link between individual variances in testosterone and human aggression is relatively minimal, in contrast to animal studies. First, unlike animal studies, most human studies rely on self-report measures that are only weakly connected with actual violence. Furthermore, when examining the association between baseline testosterone concentrations and self-reported aggression, researchers have often failed to distinguish between reactive and proactive aggression. Another drawback is that these questionnaires only look at general behavioural tendencies in a variety of scenarios (i.e., trait aggression). This is a particularly troubling issue because research on non-human animals suggests that the association between testosterone and aggressive behaviour is highly situation dependent.

Other research have analysed testosterone levels in prisoners convicted of violent and non-violent crimes, in addition to self-report measurements. Men and women convicted of violent crimes often had higher testosterone levels than those convicted of non-violent crimes, according to these research. The fact that these research are predicated on correlations between present testosterone levels and historical aggressive actions is the most significant restriction. This research technique assumes that testosterone levels remain stable over time and that current testosterone levels should mirror testosterone levels at the time of the crime. Although testosterone levels are largely steady across days, weeks, and months, testosterone levels fluctuate in reaction to social interactions, especially aggressive conduct. It is impossible to say whether high testosterone levels lead men and women to commit violent crimes, or whether aggressive behaviour in prison causes high testosterone levels. Apart from methodological limitations, another probable explanation for the smaller effects reported in humans is that aggression is not as well controlled by testosterone as it is in other species. While brain regions concerned with higher-order cognitive capacities (such as the neocortex) have grown in relative size across phylogenies, those involved in controlling the hormonal control of fundamental motivated behaviours (e.g., sex, parental behaviour, aggression) have shrunk (e.g., hypothalamus, septum).

As a result, the link between testosterone and human aggression may be less since human behaviour is generally free of the neuroendocrine system's restrictions. As a result, the link between testosterone and human aggression may be less since human behaviour is generally free of the neuroendocrine system's restrictions. The 'Challenge Hypothesis' and the 'Biosocial Model of Status,' two of the most significant theoretical models describing the context-dependent relationship between testosterone and aggressive behaviour, are presented in the following section.

Challenge Hypothesis

The original goal of the 'Challenge Hypothesis' was to explain intra- and inter-species variation in testosterone secretion in birds. During the season, testosterone levels oscillate between three levels, according to Wingfield, et al. (1990): constitutive baseline, breeding baseline, and physiological maximum. During the non-breeding season, testosterone levels in monogamous males who give paternal care are kept low at Level A. At the start of the breeding season, concentrations rise to Level B, signalling the commencement of spermatogenesis, secondary sex expression, and full exhibition of male reproductive activity. Finally, as a method of facilitating aggressive behaviour, concentrations may rise to Level C in response to male-to-male competitive encounters. When inter-male competition falls, testosterone levels fall to Level A, encouraging paternal care. Thus, there appears to be a trade-off between mating and paternal efforts in male birds that give paternal care, which appears to be mediated by testosterone levels. The costs of maintaining elevated testosterone concentrations throughout the season (e.g., decreased paternal care, increased risk of physical injury/death, depressed immune function, increased energetic demands) may have resulted in a highly flexible endocrine system capable of modulating testosterone concentrations in response to changes in the social environment. Although the 'Challenge Hypothesis' was first developed to explain hormone-behavior links in birds, its major hypotheses have been supported in a wide range of species, including fish, non-human primates, humans, and insects.

Status based on the biosocial model

The 'Biosocial Paradigm of Status,' like the 'Challenge Hypothesis,' is a theoretically comparable theoretical model. The 'Challenge Hypothesis' differs from the 'Biosocial Model of Status' in that the latter predicts that testosterone levels during competition will vary as a function of the competitive interaction's outcome: winners will have higher testosterone levels, while losers will have lower testosterone levels. Mazur (1985) postulated that winners of competitive interactions may face more status problems, and that the increase in testosterone promotes competitive and violent behaviours aimed at defending one's status. The decrease in testosterone in The social neuroendocrinology of human aggression response to defeat, on the other hand, encourages submissive behaviours to avoid further loss of status and/or physical harm. Although evidence for the effect of competition results on testosterone release exists, few studies have looked into Mazur's important claim that competition-induced testosterone alterations modulate future social behaviour.Individual differences in testosterone responses to competitive interactions are not predicted by any theoretical model. To put it another way, neither model predicts how individual differences in aggressive behaviour are related to testosterone responses to competitive interactions.

The relationship between testosterone dynamics and aggression in animals and animal models

Animal models are very valuable for examining the causal mechanisms that shape complex social behaviour.

Male California mice given testosterone (without prior winning experience) were more aggressive, but not more likely to win future competitive exchanges, according to a recent experiment. After winning a competitive engagement, mice given testosterone became more aggressive and more likely to win subsequent interactions. Castrated male California mice given testosterone after a successful aggressive contact were considerably more aggressive in subsequent aggressive encounters than mice given a saline injection, according to another study by the same group. Similarly, Oliveira et al. looked at how testosterone affected the 'winning' and 'loser' effects in male Mozambiquan tilapia. Winners of the initial aggressive engagement were more likely to win a subsequent aggressive interaction (88 percent won second fight) in control fish that had not been given any pharmacological challenge, whereas losers were more likely to lose subsequent aggressive interactions (87 percent lost second fight). Winners who were given an anti-androgen medicine to block the natural increase in testosterone in response to aggressive encounters were less likely to win another hostile interaction (relative to control males). Losers who were given an androgen (11-ketotestosterone) were not more likely to win the next aggressive engagement. These data suggest that the 'winning effect' (but not the 'loser effect') is highly reliant on testosterone changes. Fuxjager et al (2010) found that the 'winner effect' (i.e., the idea that winning an aggressive interaction increases one's chances of winning a subsequent interaction) was due to an up-regulation of androgen receptors in several key brain regions involved in reward and motivation (e.g., nucleus accumbens and ventraltegmental area) as well as social aggression (e.g., nucleus accumbens and ventralte (bed nucleus of the stria terminalis). These studies, taken together, provide persuasive evidence for the involvement of testosterone dynamics produced by competition in modulating current and/or future social behaviour. The next step is to figure out what brain mechanisms are at work when testosterone levels influence violent behaviour.

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