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The aggressive brain: insights from neuroscience

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Aggression is a complex, multifaceted behavior often caused by numerous factors and expressed in innumerable ways. Like all behaviors, aggression represents the outcome of sets of biological and physiological processes emerging from the brain. Although this may seem obvious, discovering the specific neural circuits and neurophysiological processes responsible for engendering aggressive responses has proven anything but simple. The purpose of this review is to provide a brief overview of discoveries in both human cognitive neuroscience and animal behavioral neuroscience that have begun to shed light—literally in some cases—on the heretofore mysterious neural processes and connections responsible for producing aggressive behavioral responses.

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Aggressive behavior, defined as any action that is intended to harm (or threaten harm to) another individual [1], is nearly ubiquitous across species [2]. Although specific expressions of aggression (*i.e.*, phenotypes) often differ across species, the organization and function of relevant brain structures is often highly similar. In particular, the neuronal organization of limbic structures in rat and mouse brains is very comparable to that of human brains [4^{**}]. Also, multiple neurochemical systems that regulate species-specific aggressive behaviors have co-evolved in humans and mice, for example, making animal models very useful analogues of human neural function in this context [4^{**}]. Thus, considerable research has focused on discovering the neurophysiological and neurochemical mechanisms responsible for aggression in animals, one aim of which is to apply this knowledge to understanding human aggression [5].

Neuroanatomy of aggression

Prefrontal cortex

Given the extreme complexity and multi-functionality of neural systems, researchers face a daunting challenge in trying to understand the brain basis for complex behaviors like aggression. Historically, knowledge on this topic has come from case studies of individuals who suffered neural lesions as the result of disease or injury. For example, following the destruction of large portions of his medial prefrontal cortex during a horrific accident, 19th Century railroad worker Phineas Gage became fitful and irreverent, “indulging at times in the grossest profanity (which was not previously his custom),” and “at times pertinaciously obstinate” [6, p. 338]. Because some of Gage’s mental abilities were spared (*e.g.*, his memory; basic functions like walking and talking), whereas his personality and social behaviors were drastically altered, his case produced some of the earliest discoveries related to neural specialization. In particular, the location of Gage’s injury provided some of the first clues concerning the importance of the *prefrontal cortex* (PFC; primarily, Brodmann areas 8–11 and 44–47) in regulating anger and aggression [7,8].

The PFC is associated with high-level cognition and executive functioning [9], suggesting that aggressive actions often result from a failure of self-regulatory control [10,11]. A large body of research has supported this idea. For example, in one study, men who performed poorly on cognitive tests thought to rely on PFC functioning behaved more aggressively after being provoked in a laboratory setting, relative to their better-performing peers [12]. Another study linked increased human aggression to poor functioning in the orbital PFC [13], an area severely damaged in Gage’s accident, confirming earlier reports of increased aggression in rats following orbital PFC lesions [14]. Other studies have used lesion [15,16] and brain imaging data [17] to document the negative association between PFC functioning and aggression.

Social behavior network

The areas of the brain that appear to control aggression are not specialized for this purpose, leading some to suggest that aggression is an emergent property of a larger neural network involved in the regulation of social behaviors generally [18]. This proposed network includes the anterior hypothalamic nucleus (AHN), ventromedial hypothalamus (VMH), medial amygdala (MA), bilateral septum (BLS), periaqueductal gray (PAG), and the bed nucleus of the stria terminalis (BNST). PFC structures are thought to interact with social-behavior network structures by inhibiting or modulating their activation

[3]. Evidence supporting this interpretation has come from studies with rats showing that lesions of the BLS, BNST, AHN and MA tend to reduce aggression [19], whereas lesions of the orbitofrontal cortex tend to enhance aggression [14].

The hypothalamus appears to have particular significance for aggression. Early lesion studies with cats established the hypothalamus as important for the control of rage [20]. Focused electrical stimulation of the AHN has been shown to increase aggression in both rats [21,22] and cats [23], whereas micro-injection of a vasopressin-receptor antagonist into the AHN reduces aggression in hamsters [24]. Also, electrical stimulation of the AHN in rhesus monkeys and the VHA in marmosets increases aggressive displays and attacks on subordinate males [3]. Research with humans likewise supports an important role for the hypothalamus in triggering aggression [25].

Shedding light on neural function

Electrical and chemical stimulation studies represent a major advance over earlier ‘knife cut’ lesion studies or other neural ablation techniques, which often afford little precision in targeting specific groups of neurons for study. Still, even these more advanced techniques can be problematic because the electrical current, for example, activates both the neurons of interest and the fibers connecting them with other structures, making it difficult to pinpoint causal effects. Thankfully, *optogenetics* provides a solution to this problem. By delivering a gene that encodes light-sensitive protein onto the cells of interest, scientists can engineer neurons in a targeted location to be activated by specific frequencies of light delivered via an implanted optic fiber [26]. Critically, any photons that inadvertently shine on nearby but not-genetically-altered cells will have no effect on their activation.

Using this technique, recent studies have shown that very specific neurons in the ventrolateral portion of the VMH (VMHvl) in mice, a microscopic area comprised of only around 10 000 cells, control male attack behaviors. One study found that attack responses – but not social investigation responses – are strongly suppressed by VMHvl inhibition, and that aggression returns to normal levels when VMHvl activity is restored [27]. More recent work provided evidence that aggression is an emergent property of a neural network subserving a range of social behaviors [28]. By adjusting the intensity of the light delivered to VMHvl neurons, the researchers could control whether mice engaged in sexual mounting behaviors (low-intensity light) or attack behaviors (high-intensity light). These data show both the exquisite sensitivity of VMHvl neurons to varying levels of stimulation and that the functional significance of their activation ranges dramatically, from the highly prosocial to the extremely antisocial.

Neurochemistry of aggression

Even within a circumscribed cluster like VMHvl, there is variation with respect to the presence of receptors for differing neurotransmitters. Thus, not all cells within a given structure are responsive to the same kind of neurochemical signaling and, ultimately, are not all responsible for regulating the same kinds of behaviors. Indeed, Lee *et al.* [28] found it was a relatively small subset of VMHvl neurons, distinguished by the presence of a receptor for the hormone estrogen, that were responsible for the scalable mounting-to-attacking behaviors elicited by optical stimulation (see also Ref. [29]).

Considerable research implicates the monoamine serotonin in regulating aggressive responses. Serotonin plays an important role in regulating affective responses, including those implicated in reactive, angry aggression. In very simple terms, too little serotonin can make people irritable and less able to control anger, indirectly leading to aggression. In correlational studies, brain serotonin levels have been negatively related to violence in both humans [30,31] and primates [32,33]. More direct support for the role of serotonin in aggression comes from experimental laboratory studies showing that short-term reduction in serotonin levels, achieved by decreasing dietary tryptophan, increases aggressive responding, whereas increasing serotonin levels via tryptophan supplements decreases aggressive responding [34,35]. Brain imaging studies show a potential mechanism for this effect. One study found that tryptophan-depleted participants showed weaker co-activation of limbic (amygdala) and prefrontal structures while viewing angry faces, suggesting that prefrontal regulation of anger-related responses is more difficult when serotonin levels are low [36]. Drug studies similarly have shown that acutely increasing serotonin levels via pharmacotherapy reduces aggression in the short term [37,38], and prolonged exposure to medications that increase serotonin levels chronically reduce impulsive aggression in patients with personality disorders [39,40]. Importantly, however, the serotonin–aggression link appears to hold only for high-arousal, impulsive or reactive aggression and not for low-arousal, planned or proactive aggression [25,41], consistent with the more general role of serotonin in regulating irritability (also see Ref. [42]).

Neural responses associated with aggression

Findings reviewed thus far have come from studies in which neural structure and function have been manipulated. Another class of studies involves measuring naturally occurring neural responses either as aggressive behaviors are enacted or as environmental cues associated with aggressive behaviors are processed. This cognitive neuroscience approach can establish links between aggression-related triggers in the environment and the neural processes that give rise to overt behavioral expressions of aggression.

A considerable amount of human aggression research is concerned with factors in the environment (*e.g.*, perceiving

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