

Self-regulation and aggressive antisocial behaviour: Insights from amygdala-prefrontal and heart-brain interactions

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Abstract

Explanations of aggressive and antisocial behaviour often refer to impairments in self-regulation, or the ability to implement control over ones thoughts, emotions and behaviours. However, the evidence that impaired self-regulation is associated with antisocial behaviour is somewhat mixed, and is likely to depend on the type (e.g., reactive versus proactive aggression) and severity of the antisocial behaviour. The amygdala and prefrontal cortex are critically involved in the process of self-regulation, and neuroimaging and behavioural methods, including the role of executive functions, have been used to study abnormalities of prefrontal structure and function in individuals who display aggressive and antisocial behaviours. The functioning of these circuits is also influenced by activity of the autonomic nervous system, and a robust and consistent relationship has been observed between low resting heart rate and violent and non-violent crime. Understanding the mechanisms underlying this relationship may lead to the development of interventions aimed at reducing aggressive and antisocial behaviour based on a well-defined mechanism of change. Neuroimaging and physiology research on heart-brain interactions offers new insights in to the role of self-regulation in aggressive and antisocial behaviour, and for understanding who might benefit the most from interventions aimed at improving self-regulation.

Keywords: self-regulation; antisociality; violence; heart rate; heart rate variability (HRV); executive function; inhibition

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Self-regulation and aggressive antisocial behaviour

Broadly defined, self-regulation refers to one's ability to implement intentional cognitive control over thoughts, emotions, and behaviours. Where these abilities fail, behaviours may follow that are harmful for both the individual and those around them. Heatherton and Wagner (2011) highlight several adverse outcomes associated with self-regulatory failure; these include aggressive and antisocial behaviours, often in response to negative mood states, weight gain and obesity where temporary lapses have snowballed out of control, addiction, and poor financial decision making. The aim of this paper is to review the role of self-regulation in aggressive and antisocial behaviour, with a particular focus on the neural mechanisms underlying self-regulatory processes. The prefrontal cortex (PFC) plays a central role in self-regulation and exerts cortical control over lower level brain circuits involved in emotion and reward. Further, these neural circuits appear to be influenced by the functioning of the autonomic nervous system (ANS), that is, the system responsible for the regulation of breathing and heart rate. In this paper we will review findings that different cognitive functions that are associated with the functioning of the PFC are impaired among antisocial populations, and examine the relationship of prefrontal neural activity with activation of the ANS. We will end by summarising how a better understanding of the mechanisms underlying these relationships can progress understanding of aggressive and antisocial behaviour and inform intervention efforts.

A distinction between reactive and proactive aggression

Self-regulation has been considered to play a role in alcohol and substance abuse, acts of violence, sexual offences, and other impulsive, or risk taking behaviours (Davidson, Putnam, & Larson, 2000; Gillespie, Mitchell, Fisher, & Beech, 2012; Gillespie & Beech, 2016; Quinn & Fromme, 2010). In the current paper we will focus on the relationship of self-regulation with aggression and antisociality. Aggression can be defined as purposeful behaviour aimed at harming another person either physically or psychologically, and is typically classified into two major subtypes: proactive and reactive (Raine et al., 2006). Although these two types of aggression are not mutually exclusive, and an aggressive act may be characterized by elements of both, failure to take in to account these differences may lead to inconsistent findings.

Proactive aggression can emerge without any obvious antecedent. This form of aggression typically requires the presence of a plan and is not associated with impaired executive performance (Ellis, Weiss, & Lochman, 2009). By contrast, reactive aggression results from the interpretation of someone's intentions as being hostile and provocative (Dodge et al., 2015; Kempes, Matthys, De Vries, & Van Engeland, 2005), and is associated with difficulties in executive performance and self-regulation (Card & Little, 2006; Ellis et al., 2009; Marsee & Frick, 2007). For this reason, the reactive type is perhaps most relevant to the discussion here.

The neurobiology of self-regulation and aggression

The neurobiology of trait aggressiveness appears to be underpinned by brain structures related to self-regulation, with much attention being paid to the role of the amygdala, and areas of the prefrontal cortex [PFC] (Davidson et al., 2000). These areas include the ventromedial PFC [VMPFC], the orbitofrontal cortex [OFC], and the dorsolateral PFC [DLPFC]. The amygdala is located in the medial parts of the temporal lobe and can be

broadly subdivided into two major components, the basolateral amygdala and the centromedial amygdala (Sah, Faber, De Armentia, & Power, 2003). It has been estimated that the basolateral component, which can be subdivided into the lateral amygdala nuclei and the basal nuclei, acts as the major input territory for the amygdala. The basal nucleus receives particularly strong afferent inputs from the PFC. However, inputs to the lateral nuclei from the thalamus and high level sensory cortical areas have also been described. On the other hand, the centromedial is more characterised by its outputs. Several reviews of morphological and functional brain imaging studies suggest that amygdala abnormalities may be present among individuals who are prone to reactive aggression, with reports of reduced volume and lower resting activity, as well as phasic increases in activity in response to both aggressive tasks and images of faces (Rosell & Siever, 2015; Siever, 2008; Nelson & Trainor, 2007; Coccaro, Sripada, Yanowich, & Phan, 2011). By contrast, hypoactivation of the amygdala in response to fearful faces in youth with conduct problems appears to mediate the relationship of callous-unemotional traits with instrumental/proactive aggression (Lozier, Cardinale, VanMeter, & Marsh, 2014). However, it should be noted that neuroimaging studies typically do not distinguish between the different components of the amygdala.

Importantly, the amygdala makes extensive reciprocal connections with the PFC, and areas of the PFC can be seen in functional terms as inhibiting the amygdala (Stein et al., 2007). Consequently, failure of these circuits causes the amygdala to be released from inhibition and a tendency for behaviours associated with poor self/emotion-regulation can be observed. The VMPFC plays a key role in interpreting the physiological changes that accompany emotional reactions. The VMPFC is continuous with the adjacent OFC. Damage to the OFC can be accompanied by major personality changes, as in the case of Phineas Gage, a railway engineer who experienced severe personality changes following an accident in which his OFC was penetrated by an iron rod. The OFC, particularly its medial area, is

believed to play a significant role in the evaluation of social and emotional cues (Coccaro et al., 2011). By contrast, the DLPFC plays fundamental roles in high level cognitive actions including abstract reasoning, planning and working memory. Strongly related to the PFC, but not strictly part of it, is the anterior cingulate cortex (ACC). The ACC has typically been associated with error detection and monitoring, although accumulating evidence suggests a relationship of ACC activity with effortful cognitive and motor control, or task difficulty (see Critchley et al., 2003; Holroyd & Yeung, 2012). Heightened ACC activation has previously been found to inhibit amygdala responses (Ochsner & Gross, 2005). The quality of functional and structural connectivity between substructures of the PFC and the amygdala nuclei has a key impact on emotional responding (Ochsner & Gross, 2005; Banks, Eddy, Angstadt, Nathan, & Phan, 2007).

Given that the PFC plays a pivotal role in self-regulation, it has not surprisingly been spoken of as a target for treatments and interventions that aim to improve self-regulation among antisocial populations. However, different types of self-regulatory failure are related to impairments in different cognitive functions (e.g., inhibition, working memory), and these are associated with differing areas of PFC (Hofmann, Schmeichel, & Baddeley 2012). Specifying and elaborating on these relationships is essential for understanding the causes of aggressive and antisocial behaviour, and the potential mechanisms of change in offending behaviour programs (see Ward, Wilshire, & Jackson, this issue). Below, we will review the literature on executive performance, PFC structure and function, and antisociality.

Relationship of prefrontal structure and function with antisocial behaviour

PFC and tests of executive function

Behavioural tasks are commonly used to test several known functions of the PFC. These functions, commonly termed *executive functions* (EFs), can be best understood according to

an influential taxonomy that specifies three basic EFs (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000): working memory operations, behavioural inhibition, and mental set switching. In a review of EF and self-regulation, Hoffman et al. (2012) highlight the ways in which different EFs may aid self-regulation, and provide an overview of basic tasks on which performance may serve as an indicator of an individual's capacity for these different EFs. For example, working memory capacity may be tested using operation span and n-back measures, where participants are required to maintain and update information (e.g., numbers), while inhibition may be tested using the Stroop task (Stroop, 1935), where participants are required to name the colour ink that colour naming words are printed in, inhibiting the automatic response to read the colour naming word (e.g., the word 'RED' printed in blue ink).

Performance on these tests of EF is associated with the functioning of particular sub-regions of the PFC. For example, a relationship of executive performance with lateral and medial, but not orbital, PFC structure has been highlighted in a meta-analysis of structural neuro-imaging studies (Yuan & Raz, 2014). However, the strength of this relationship varied with the task used, with a stronger association reported for the Wisconsin Card Sorting Test, than with digit backwards span, Trail Making Test and verbal fluency (Yuan & Raz, 2014). It has also been shown that particular functions may be associated with activation in particular regions, with Stroop performance associated with activation in the inferior frontal gyrus and the anterior cingulate gyrus (Laird et al., 2005), and WM performance associated with activity in lateral PFC (Rottschy et al., 2012).

Executive dysfunction and antisocial behaviour

Results of studies that have tested the association of antisocial behaviour with frontal lobe and EF impairments have been equivocal, and Morgan and Lilienfeld (2000) note that many studies are plagued by methodological concerns. However, a meta-analysis on the association

of antisociality with executive performance showed a medium to large weighted effect size of .62, with antisocial groups performing worse than their non-antisocial counterparts (Morgan & Lilienfeld, 2000). Effects were greatest when antisocial behaviour was operationalised as criminality or delinquency, and groups were best differentiated by the number of errors on the Porteus Mazes that ostensibly reflected impulsivity (e.g., crossed lines, direction changes). Other tests including Stroop and verbal fluency tasks showed a medium effect size. However, differences in performance were also noted for other neuropsychological tests, raising the possibility that difficulties may extend beyond EF to other neuropsychological impairments.

A more recent meta-analysis also yielded a medium weighted effect size of .44, showing greater executive dysfunction among antisocial compared with non-antisocial groups (Ogilvie, Stewart, Chan, & Shum, 2011). Effect sizes were again greatest for groups classified according to criminality, and the largest effect sizes were reported for the self-ordered pointing task, a test of non-spatial WM. Other tests that differentiated antisocial from comparison groups with medium-to-large effect sizes included the mazes test, the delayed matching to samples task, the go/no-go task, and the spatial working memory task (Ogilvie et al., 2011). Tests that failed to differentiate groups with at least a small effect size included the Tower of London task, the Simon task, the Paced Auditory Serial Addition Test, and the Two-Back Test.

Although the results suggest that antisocial groups do show impaired performance on tests of EF, the pattern of results appears to vary with the EF measures used, and with the operationalisation of antisociality. A recent study also found that out of six tests included from the Cambridge Automated Neuropsychological Test Battery (CANTAB; Cambridge Cognition, UK), only response inhibition distinguished violent from non-violent offenders, with the violent group showing more pronounced deficits (Meijers, Harte, Meynen, &

Cuijpers, 2017). These findings highlight the importance of investigating and reporting associations with specific EFs, rather than looking for and reporting on deficits in executive performance more generally. Single studies also indicate that the persistence and severity of antisocial behaviour may be associated with executive performance, with more pronounced deficits observed among children with co-morbid oppositional-defiant/conduct disorder, and attention-deficit hyperactivity disorder (Clark, Prior, & Kinsella, 2000). Those with life-course persistent antisocial behaviour also appear to show more pronounced impairments compared with less severe comparison groups (Raine, Moffitt, Caspi, Loeber, Stouthamer-Loeber, & Lynam, 2005). However, while these tests provide an approximation of PFC function, more direct evidence for abnormalities in PFC structure and function has been gained from neuroimaging studies.

Prefrontal structure and antisocial behaviour

Although several studies have used neuroimaging to assess brain structure and function in antisocial and aggressive groups, like results for EFs, these findings have also proven equivocal. In an influential review and meta-analysis of 43 brain imaging studies in antisocial, violent, and psychopathic individuals, Yang and Raine (2009) found significantly reduced prefrontal structure and function in antisocial individuals. In particular, these effects were localised to the right OFC, right ACC, and left DLPFC (Yang & Raine, 2009).

However, these findings have been contested in a recent systematic review and voxel based meta-analysis. Here it was found that only the parietal lobe showed consistent reductions in grey matter volume, and a meta-analysis showed no significant clusters of reduced grey matter volume associated with interpersonal violence (Lamsma, Mackay, & Fazel, 2017). As noted by the authors, these results contrast with several theories that focus on potential brain markers for antisocial behaviour and violence. However, it is worth noting that this review

and meta-analysis focussed on structural imaging findings, and a focus on brain lesion or head injury studies (Bannon, Salis, & O'Leary, 2015; Farrer, Frost, & Hedges, 2012), or studies that use functional imaging, may reveal a different pattern of results.

Future studies that aim to examine the relationship of PFC structure and function with interpersonal violence should attempt to differentiate between different types of aggression. For example, the distinction outlined near the start of this paper, between reactive/impulsive and proactive/instrumental aggression, may be associated with differential patterns of PFC structure and function. In particular, the more reactive type is considered to better reflect impairments in the conscious control of emotion, self-regulation, and impulse inhibition. On the other hand, proactive aggression would be expected to entail intact abilities for self-regulation, emotion control, and impulse inhibition (Card & Little, 2006; Ellis et al., 2009; Marsee & Frick, 2007). Such a distinction is important to note in the development of new interventions aiming to reduce the likelihood that a given person will offend again in the future.

A better understanding of the underlying mechanisms for reactive and proactive aggression may be gained from not only studying neural function and executive performance, but by also studying how neural function is related to the functioning of the ANS. Measurements of ANS activity, including resting heart rate (rHR) and variability in the interbeat intervals of the heart, known as heart rate variability (HRV), have provided further insights in to self-regulation and antisocial behaviour. The functioning of the ANS represents an important factor for understanding the behavioural and emotional responses to threat, and levels of activity in the ANS may influence activation of neural circuits involved in self-regulation and behavioural inhibition. In the below section we will review findings that point to a consistent and robust relationship of aggressive and antisocial behaviour with low rHR,

briefly highlight two theoretical accounts that have been proposed to account for this relationship, and examine the strength of the evidence to support these theories.

The physiology of aggression and antisocial behaviour

Low resting heart rate and antisocial behaviour

A relationship between low resting heart rate (rHR) and antisocial behaviour has been postulated for some time, with low rHR now considered to be one of the best replicated physiological correlates of antisocial behaviour in children and adolescents (Glenn & Raine, 2014). This relationship is supported by the results of two meta-analyses, both of which highlight that aggressive and antisocial behaviours in children are associated with a lower rHR compared with their non-antisocial or aggressive counterparts (Lorber, 2004; Ortiz & Raine, 2004). A further meta-analysis also confirms this relationship, and shows it to be generalizable with respect to sex in both child and adult samples (Portnoy & Farrington, 2015). More recently, this link was emphatically supported in a longitudinal study of heart rate and antisociality in a sample of more than 700,000 men (Latvala, Kuja-Halkola, Almqvist, Larsson, & Lichtenstein, 2015). In this study, Latvala et al. showed that in models adjusted for physical, cardiovascular, psychiatric, cognitive, and socioeconomic covariates, there was a relationship of low rHR with violent and non-violent criminality, as well as with assaultive and unintentional injuries. However, the relationship did not extend to the commission of sexual crimes.

The underlying mechanisms for the relationship of low rHR with aggressive and antisocial behaviour remain poorly understood. However, two popular theories have been put forward. The first of these is the stimulation-seeking hypothesis (Quay, 1965; Raine, 2002), which suggests that low rHR is experienced as an aversive state, and that under-aroused individuals take risks in order to boost levels of arousal to a more optimal level. The second

theory revolves around low rHR as an indicator of a fearless temperament. According to this theory, low rHR may reflect a reduced fear response when confronted with the mild stressor of undergoing physiological recording in an unfamiliar laboratory setting, and this relative fearlessness predisposes these individuals to risk-taking and antisocial behaviours (Raine, 2002). Although support for either of these theories is slim, a couple of recent studies show support for the stimulation-seeking hypothesis (Portnoy, Raine, Chen, Pardini, Loeber, & Jennings, 2014; Sijtsema et al., 2010). For example, impulsive sensation seeking, but not state fear, mediated the association of rHR with both aggression and non-violent delinquency in a sample of adolescent boys (Portnoy et al., 2014). In a similar fashion, sensation seeking in adolescent boys mediated the relationship of rHR with rule breaking (Sijtsema et al., 2010). Thus, the stimulation-seeking theory appears to be the better supported.

Together with theoretical accounts, these findings indicate that the rHR-antisocial relationship may be mediated by impulsive sensation seeking. Thus, it would be hypothesized that low rHR is associated with increased impulsivity, as indicated by poor performance on tests of inhibition, as well as structural and functional abnormalities in areas of the PFC related to inhibition and self-regulation (e.g., medial regions of PFC). In order to understand these relationships it is important to consider (i) the functioning of the ANS, including the separable influences of the sympathetic and parasympathetic branches, and (ii) heart-brain interactions, that is, the relationship that exists between the activity of the ANS and the functioning of specific neural circuits involved in impulse inhibition, self-regulation, and the conscious control of emotion.

Organisation of the ANS

The physiological changes that typically accompany emotional responses are mediated by the functioning of the ANS, that is, the elements of the nervous system which form extensive

neural links between the internal organs, including the heart, and the brain. The neural connections from the viscera provide the brain with vital information about the physiological state of the body. Based on this data, the brain can send information back to the viscera and adjust the activity of target organs to current demands. For example, an imminent risk of being physically harmed may be accompanied by changes in the cardiovascular system. This may be followed by increased blood flow to the muscles to increase the chance of survival, through either the so-called 'fight' or 'flight' responses. These physiological changes can also be associated with complex behaviours that accompany emotional and cognitive responses. However, the precise neuroanatomical pathways of this heart-brain-behaviour arrangement are still to be fully elucidated.

The heart receives connections from the ANS which can be divided into two divisions, the sympathetic division and the parasympathetic division (Berntson et al., 1997). When the former operates, it tends to lead to increased expenditure of energy, whereas an increase in activity of the latter is linked with relaxation and conservation of resources. Organs are usually innervated by both the sympathetic and parasympathetic branches of the ANS. The heart rate at rest is predominantly controlled via the vagus nerve of the parasympathetic nervous system. The vagus nerve of the heart descends from the nucleus ambiguus of the medulla oblongata located in the brainstem. The descending vagal preganglionic fibres travel to the heart where short postganglionic fibres innervate the sinoatrial node (Critchley, 2005). The sinoatrial node can be described as a group of cells able to produce electrical impulses causing the heart to contract. The slowing of contractions is achieved by release of acetylcholine from the vagal pre and postganglionic synapses. This effect can be diminished with increased activity of the noradrenaline based sympathetic innervations to the heart. Therefore, the rate of the heart is dictated by the interplay of the two subsystems of the ANS. However, the relative dominance of sympathetic and

parasympathetic (vagal) influences on mean HR appears to vary in important ways with participant sex (Koenig & Thayer, 2016).

Briefly, according to models of heart-brain interactions (e.g., Critchley & Harrison 2013; Smith, Thayer, Khalsa, & Lane, 2017; Thayer & Lane, 2009), sensory information from the heart is conveyed to the nucleus of the solitary tract (NTS) of the brainstem by ascending branches of the vagus nerve. The NTS in turn sends efferents to the nucleus ambiguus which gives rise to the vagal parasympathetic input to the heart. Other brainstem structures to which the NTS is reciprocally connected include the periaqueductal gray (PAG), and the parabrachial nucleus. The NTS also projects to the hypothalamus, a limbic structure which plays a critical role in maintaining homeostasis. The PAG, parabrachial nucleus and the hypothalamus relay visceral related information to the basal forebrain and to the amygdala. Equivalent information is also sent to the thalamus from where it is relayed to parts of the cerebral cortex. Activity in all the mentioned brain structures contribute to the strength of the descending vagal control over the rate of the heart.

HRV and the heart-brain connection

As well as activity in the structures outlined above, vagal control can also be affected by breathing. Breathing out results in minor increases in the frequency of vagal nerve discharges (Yasuma & Hayano, 2004). Consequently, expiration is associated with a short duration slowing of the heart rate. This physiological phenomenon is referred to as the respiratory sinus arrhythmia. A slow-paced pattern of breathing is associated with a stronger vagal nerve response and thus an exaggerated slowing of the heart rate (Grossman & Taylor, 2007). This increases the variability in the interbeat intervals of the heart, or HRV. Resting HR and HRV, not surprisingly, are closely connected to each other, with the values of these two parameters

typically being highly negatively correlated. Thus, emotional reactions which result in increased HR simultaneously cause a correlated decrease in HRV.

HRV appears to be closely connected with self-regulatory functions, and low levels of HRV have been linked with negative psychological characteristics (Kemp & Quintana, 2013; Thayer & Lane, 2009), including depression (Kemp, Quintana, Gray, Felmingham, Brown, & Gatt, 2010), anxiety disorders (Chalmers, Quintana, Abbott, & Kemp, 2014), and stress (Vrijkotte, Van Doornen, & De Geus, 2000; Hall et al., 2004). Conversely, elevated levels of HRV have been positively linked with good emotion regulation (Thayer & Lane, 2009; Thayer, Åhs, Fredrikson, Sollers III, & Wager, 2012; Thayer, Hansen, Saus-Rose, & Johnsen, 2009), increased performance on tests of EF (Hansen, Johnsen, & Thayer, 2009; Hansen, Johnsen, & Thayer, 2003; Thayer, Hansen, Saus-Rose, & Johnsen, 2009), and good physical fitness (Buchheit & Gindre, 2006; Oliveira, Barker, Wilkinson, Abbot, & Williams, 2017). To summarize, lower levels of HRV are associated with increased emotion dysregulation and affective difficulties, while higher levels are associated with better regulation over emotional states and improved EF performance.

The results outlined above have been confirmed in a recent meta-analysis that examined existing findings on the relationship of HRV indices with aspects of top-down self-regulation (e.g., EF performance, emotion regulation, effortful control) (Holzman & Bridgett, 2017). Results from this analysis showed a small but significant effect, with indices of higher HRV associated with better self-regulation, indicative of a relationship between HRV and PFC function. This view is consistent with a recent meta-analysis which showed that vagally mediated HRV is associated with activity in structures including the medial PFC and the amygdala (Thayer et al., 2012). One recent study also suggests that higher HRV is associated with VMPFC activity and greater self-regulation, measured as increased resistance to temptation in dietary self-control challenges (Maier & Hare, 2017). The relationship between

prefrontal activation and indices of HRV may reflect an inhibitory influence of the PFC over subcortical structures, including the amygdala, which affects vagal input on the sinoatrial node of the heart (Holzman & Bridgett, 2017; Thayer et al., 2009). In support of this theory, it has been shown that HRV is associated with greater functional connectivity between medial PFC and amygdala, such that increases in medial PFC activity are associated with correlated decreases in amygdala activity in younger and older adults (Sakaki, Yoo, Nga, Lee, Thayer, & Mather, 2016). Thus, increased HRV appears to be associated with activity in a network of areas that involves top-down regulation of the amygdala by regions of PFC.

Implications for the action underlying the rHR-antisocial relationship

The findings reviewed above have implications for understanding the mechanisms underlying the robust relationship between low rHR and aggressive antisocial behaviour. Existing theories suggest that this relationship reflects either fearlessness or impulsive sensation seeking, but there is little evidence in support of either position. First, the findings reviewed here do not suggest an absence of the subjective experience of fear, although increased HRV may be associated with better prefrontal regulation of such threat related responses. Other evidence also suggests that psychopathy – a disorder characterised by a phenotypic increase in proactive aggression – is associated with impaired detection and responding to threat, but not reduced subjective experience of fear (Hoppenbrouwers, Bulten, & Brazil, 2016). Second, despite some limited evidence for the impulsive sensation seeking hypothesis, the negative relationship that exists between rHR and HRV would suggest that individuals characterised by low rHR should actually show *good* impulse inhibition. Instead, we would suggest that the relationship between low rHR and aggressive antisocial behaviour may better reflect high levels of HRV and associated increases in prefrontal inhibition over lower level circuits (see Figure 1). Although this explanation may seem paradoxical, Thayer et al. (2012) review

evidence which suggests that the threat response is by default ‘on’ and that areas of medial PFC may integrate perceptions of environmental threat and internal perceptions of control to regulate or inhibit threat representation in the amygdala. Thus, those behaviours associated with low rHR (and high HRV) that appear to be disinhibited or risk-taking, may actually reflect increased inhibition over emotional reactions to threatening or fear inducing situations, rather than failures of impulse control or behavioural inhibition. As such, these behaviours might better reflect calculated risks, rather than impulsive reactions.

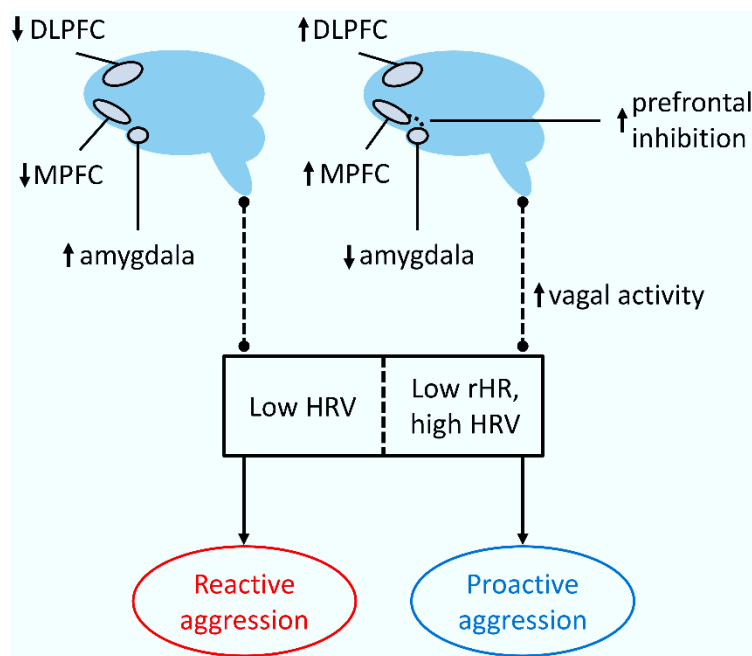


Figure 1. Pathways to reactive and proactive aggression in humans. Left: Reactive aggression is associated with poor impulse inhibition, emotional reactivity and impaired self-regulation. This pattern appears to reflect reduced prefrontal inhibition over lower level threat circuits including the amygdala. Right: Proactive aggression is characterised by low resting heart rate (rHR) and elevated vagally mediated heart rate variability (HRV). This pattern of autonomic functioning is associated with increased activation in medial prefrontal cortex (MPFC), increased prefrontal inhibition over the amygdala, and increased functional connectivity between MPFC and amygdala. This pattern suggests that the relationship of low

rHR with proactive aggression is reflective of good self-regulation, and a pattern of calculated rather than impulsive risk taking.

In support of this hypothesis, some studies have reported increased parasympathetic activity, or vagally mediated HRV, in aggressive and antisocial samples. For example, Scarpa and colleagues distinguished between reactive and proactive aggression in a developmental sample, and recorded measurements of HR, HRV, and skin conductance (SC), an indicator of sympathetic activity (Scarpa, Haden, & Tankaka, 2010). Results showed that while reactive aggression was associated with reduced HRV and reduced SC, indicative of both reduced parasympathetic and sympathetic activity, proactive aggression was related to increased HRV and SC, indicative of increased parasympathetic and sympathetic activity. As such the findings suggest a rather complicated relationship of aggression with autonomic function. Somewhat surprisingly, the results showed no relationship of rHR with either reactive or proactive aggression, and it is suggested that in relation to aggressive behaviours, vagal influences on HR may play a stronger role than is reflected by the HR measure alone (Scarpa et al., 2010). Consistent with the model outlined in this article, the authors suggest that increased HRV in relation to proactive aggression may be indicative of increased self-regulatory abilities and frontal lobe function (Scarpa et al., 2010).

In a separate study, Hansen and colleagues examined the relationship of cognitive function, HR and HRV with interpersonal, affective, impulsive-lifestyle, and antisocial psychopathic traits in a sample of male prisoners (Hansen, Johnsen, Thornton, Waage, & Thayer, 2007). Results showed that lower rHR was associated with better WM performance among offenders, while the interpersonal facet of psychopathy was associated with lower rHR, higher HRV and also better WM performance (Hansen et al., 2007). Antisocial psychopathic traits also predicted greater levels of HRV. Interestingly, multiple regression

models showed no relationship of impulsive-lifestyle psychopathic traits with either rHR, HRV, or executive performance. Again, these findings support the hypothesis that the low rHR relationship with antisocial behaviour may better reflect higher HRV and associated increases in aspects of executive performance, rather than greater impulsive stimulation seeking.

Conclusion

Self-regulation plays an important role in aggressive and antisocial behaviours, and tests of executive performance and brain imaging techniques indicate that some aggressive and antisocial individuals are characterised by impulsive tendencies and impairments in the neural circuits underlying self-regulation. However, while these difficulties appear to provide an explanation for reactive, affectively driven aggression, they provide a more limited understanding of proactive, instrumental acts of aggression. Further insights in to proactive aggression may be gained by examining heart-brain interactions, particularly the relationships between rHR, HRV and brain function. Although a robust relationship has been identified between low rHR and aggressive antisocial behaviours, the precise mechanisms underlying this relationship are uncertain. Here, we present a model based on neuroimaging and psychophysiology that attempts to explain this relationship, hypothesising that those with a low rHR should show intact abilities for self-regulation, and good control over emotional reactions to fearful or threatening situations. According to the hypotheses presented here, the aggressive and antisocial behaviours that occur with greater frequency among those with low rHR (see Latvala et al., 2015), may better reflect increased inhibition, rather than PFC disruption/impulsive sensation seeking. Such an account would appear to better explain the proactive/instrumental type of aggression.

Through integrating results from different perspectives, including behavioural experiments, neuroimaging, psychophysiology, and neurochemistry, it may be possible to identify potential biomarkers for aggression, aid interpretation from multiple sources, and assist in developing models and informing prediction, intervention, and rehabilitation. Future research should tentatively employ HRV indices as bio-markers of top-down self-regulation in an effort to understand impairments in self-regulation, emotion control and impulse inhibition in aggressive and antisocial populations. Together with other measures of cardiovascular activity, including rHR, and measures of top-down self-regulation (e.g., tests of EF), such data could be instructive in better distinguishing between the underlying mechanisms for reactive and proactive aggression, and in informing treatment efforts in forensic settings. Techniques such as mindfulness meditation and HRV biofeedback have been recommended for improving self-regulation among those who display aggressive and antisocial behaviours, including those who have committed sexual offences (Gillespie et al., 2012; Gillespie & Beech, 2016). However, it is important that future research aims to identify those who would benefit most from such interventions, the mechanisms of change involved, and the psychological and physiological changes associated with practicing these techniques (see Brzozowski, Gillespie, Dixon, & Mitchell, 2017).

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