The Relationship between Low Resting Heart Rate and Antisocial Behavior: Correlation or Causation?

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Based on a meta-analysis of 45 independent effect sizes from 40 studies, Ortiz and Raine (2004) concluded “low resting heart rate appears to be the best-replicated biological correlate to date of antisocial and aggressive behavior in children and adolescents” (p. 154). Research included in the Ortiz and Raine (2004) meta-analysis and subsequent studies show that low resting heart rate is predictive of crime and antisocial behavior among adults (Farington, 1997; Raine et al., 1990, 1995; Wadsworth, 1976) and that the link between resting heart rate and antisocial behavior remains net of controls for variables from traditional criminological theories (Armstrong et al., 2009; Cauffman et al., 2005; Farrington, 1997). Recent research also indicates that low resting heart rate (LRHR) is related to serious and violent antisocial behavior (SVASB), but not to more general delinquency (Armstrong et al., 2009). In their meta-analysis, Ortiz and Raine (2004) find an average effect size of 0.44 among studies testing the relationship between low resting heart rate (LRHR) and antisocial behavior (ASB). The effect size for the subset of studies testing the relationship between low heart rate reactivity (LHRR) and antisocial behavior was in an effect size of 0.76. This suggests that particular attention to LHRR may be warranted.

Raine (2002) reviewed a number of hypotheses regarding the relationship between resting heart rate and antisocial behavior. These hypotheses were organized around two main causal mechanisms. In the first, low autonomic arousal (low resting heart rate) is a marker for psychological states which in turn lead to increased antisocial behavior. In the second, physiological characteristics cause both low resting heart rate and increased antisocial behavior.

Psychological characteristics proposed as mediators between resting heart rate and antisocial behavior include stimulation seeking and fearlessness.
seeking suggests low autonomic arousal constitutes an unpleasant psychological state, with acts of antisocial behavior, including crime and delinquency, undertaken in an effort to increase arousal levels. Low resting heart rate may also be a marker of fearlessness, which leads to increased antisocial behavior through a disregard for the negative consequences of this behavior. Physiological processes potentially mediating the relationship between resting heart rate and antisocial behavior included right hemisphere dysfunction, reduced noradrenergic functioning and increased vagal tone.

In an effort to shed light on the relationship between resting heart rate and antisocial behavior, this chapter will first discuss the physiological processes that determine heart rate with specific attention to the process that may influence heart rate reactivity. Next, the chapter will identify brain regions that may explain the relationship between heart rate and antisocial behavior. Activity in these brain regions are associated with both heart rate and antisocial behavior, as well as being linked to empathy, fear conditioning, and the experience of emotion. Subsequently the chapter will review evidence for a direct influence of heart rate on antisocial behavior net of brain activity predictive of both heart rate and antisocial behavior.

Physiological Structures and Processes Contributing to Heart Rate and Changes in Heart Rate

Heart rate is determined largely by a series of structures ranging from the prefrontal cortex to direct inputs to the heart’s sino-atrial node through the autonomic nervous system (ANS). The ANS has two major divisions: the parasympathetic and sympathetic nervous systems. Broadly conceived the parasympathetic nervous system (PNS) is responsible for vegetative and restorative function, while the sympathetic nervous system (SNS) is associated with the mobilization of energy under stress or the “fight or flight” response. Regarding heart rate, increases in parasympathetic nervous system activity lead to deceleration in heart rate, while relative increases in sympathetic nervous system activity are associated with heart rate increases.

Input to the ANS is provided by cortical and subcortical structures through key areas in the brain stem. Together the cortical and subcortical structures that influence heart rate along with areas in the brain stem are referred to as the central autonomic network (CAN) (Benarroch, 1993, 1997). The parasympathetic and sympathetic nervous systems influence heart rate through input to the sino-atrial node. The sino-atrial node serves as the heart’s pacemaker, generating electrical

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1 The neuroanatomical structures and interconnections identified in the CAN are demonstrated through studies of animal models and through neuroimaging studies in humans. For a review of the animal literature see Cechetto (2004) and Saper (2002). For a review of neuroimaging studies in humans see Cechetto and Shoemaker (2009).
impulses that trigger heart contraction. Parasympathetic input to the sino-atrial node is conveyed by the tenth cranial or ‘vagus’ nerve and originates in the dorsal motor nucleus and the nucleus ambiguous in the brain stem (Levy and Warner, 1994). Sympathetic influences originate in the rostral ventrolateral medulla and are conveyed to the sino-atrial node through stellate ganglia by way of the intermediolateral (IML) column of the spinal cord (Levy and Martin, 1979).

The CAN includes cortical, limbic, and midbrain structures. Cortical structures in the CAN include the medial prefrontal cortex, and orbitofrontal cortex. Limbic structures in the CAN include the cingulate cortex, insular cortex, amygdala and the hypothalamus; midbrain structures include the parabrachial complex, nucleus of the solitary tract (NTS), and the rostral and caudal ventrolateral medulla. In the ANS, efferent neurons carry information away from the CAN and afferent neurons carry information toward the CAN. Afferent neurons suggest that autonomic arousal (or underarousal) may have a direct influence on emotion and cognition, and by extension, behavior. Feedback from these neurons ultimately returns to the insular cortex (IC) and other higher cortical structures, where central representations of bodily states are thought to influence emotion and cognition (Craig, 2002 and 2009).

Thayer et al. (2009) provide an overview of the function of the CAN and its relationship to the ANS, with medial prefrontal cortex (MPFC) and orbitofrontal cortex influencing autonomic output through interconnections with both the cingulate cortex and the insular cortex. These regions connect directly and indirectly (through the amygdala) with hypothalamus, periaqueductal gray matter, and the parabrachial poinine nuclei, which then influence both parasympathetic and sympathetic tone through the NTS and sympathetic tone through the rostral ventrolateral medulla (RVLM). It is important to note that the connections described by Thayer et al. (2009) represent the primary connections between structures in the CAN. There are numerous interconnections between structures in the CAN. As described by Benarroch (1993), the components of the CAN tend to be reciprocally interconnected “allowing continuous feedback interactions and integration of autonomic responses” (p. 990).

The RVLM appears to be the major contributor to pre-ganglionic sympathetic nervous system tone (Guyenet, 1990; Dampney et al., 2000). Stimulation of the RVLM causes an increase in heart rate and blood pressure (Dampney, 1994). There is also evidence that pharmacological blockade of the RVLM reduces depressor effects that normally occur during the stimulation of the prefrontal cortex or areas within the lateral hypothalamus (Cechetto and Chen, 1992). The RVLM influences sympathetic tone through projections into the intermediolateral (IML) cell column (Cabot, 1990).

The RVLM and the NTS receive projections from the central nucleus of the amygdala (CeA) (Saha, 2005). The CeA is likely a key mediatary structure in mechanisms resulting in cardiovascular changes as the result of environmental stimuli. Electrical and chemical stimulation of the CeA results in responses consistent with an increase in sympathetic nervous system activity including
increases in heart rate (Saha, 2005). The NTS also receives projections from the MPFC and the IC (Verberne and Owens, 1998).

**Evidence Linking CAN Function to Autonomic Function Including Heart Rate**

The influence of structures in the CAN on autonomic function is supported by research based on animal models (for recent reviews see Cechetto, 2004 and Saper, 2002) and by research in humans (for recent reviews see Cechetto and Shoemaker, 2009 and Thayer et al., 2009). Animal model studies rely primarily on electrical simulation/chemical activation, pharmacological blockade, and tract tracing methods. Electrical and chemical activation studies examine the effect on autonomic arousal of the activation of brain structures in animals that are analogous to those in humans. For example, in a review of research on the role of amygdala in the control of blood pressure, Saha (2005) notes that electrical stimulation of the amygdala in the cat and rat has been found to produce increases in heart rate, arterial blood pressure and muscle blood flow (p. 451). Similarly, Verberne and Owens (1998) review studies exploring cortical modulation of the cardiovascular system including work showing electrical stimulation of the MPFC results in depressor responses. Verberne (1996) suggests depressor responses may be a function of the inhibition of the sympathetic nervous system through the influence of the MPFC on the RVLM.

Applied to the study of autonomic function pharmacological blockade studies assess changes in autonomic function consequent to the chemical blockade of structures in the CAN and the ANS. Tavares et al. (2009) explored the effect of the pharmacological blockade of areas in the MPFC on the relationship between acute restraint stress and increases in heart rate in the rat. Blockade was achieved with microinjection of an unspecified synaptic blocker (CoCl2). Blockade of the prelimbic cortex (in MPFC) resulted in increased heart rate response, while blockade of the infralimbic cortex resulted in significant reductions in heart rate response. No changes occurred with blockade of other regions in MPFC. Importantly this shows different MPFC regions have distinct effects on heart rate.

Tract tracing methods explore interconnections between neuronal structures through the injection of a traceable substance in the area of interest. In anterograde tract tracing the substance is “picked up” by the cell body of a neuron and transported forward to the synapse. Retrograde tract tracing is used to trace connections from synapse to cell body. Cechetto et al. (1983) used retrograde tract tracing with horseradish peroxidase to test the structures that feed information forward to the amygdala in cats. Injections to the medial central region of the amygdala were carried by neurons to the paraventricular and ventromedial nuclei in the hypothalamus, as well as the parabrachial nuclei, showing that afferent information from the ANS is conveyed to the amygdala through the hypothalamus and brain stem.

Taken as a group, animal studies demonstrate that structures in the CAN influence autonomic nervous system function including heart rate. These studies
also demonstrate profound interconnectivity between these structures. This interconnectivity is juxtaposed with primary interconnections that serve as the major pathways by which information is conveyed among CAN and ANS structures. Some of these primary interconnections are between structures in the forebrain, including the orbitofrontal cortex (OFC) and the MPFC, and structures in the limbic system including the amygdala. There is some suggestion among animal models that prefrontal cortices exhibit an inhibitory influence on lower brain structures. Verberne and Owens (1998) find that MPFC exerts a sympathoinhibitory influence on the ANS through the RVLM. Evidence of an inhibitory influence prefrontal cortices is coupled with evidence indicating that activity in the amygdala and insular cortex is predominantly associated with increases in sympathetic nervous system (SNS) arousal including increases in heart rate (Nagai et al., 2010; Saha, 2005).

Studies based on animal models are complemented by lesion studies and neuroimaging research in humans. Neuroimaging studies relate changes in the ANS activity in response to a given stimulus to activity in the brain. Many of these studies used stimuli directly related to autonomic arousal. Other studies used stimuli related to cognitive or emotional function. Examples of stimuli directly related to ANS function include isometric hand grip exercises and the Valsalva maneuver. Here I will focus on neuroimaging studies of the relationship between brain activity and autonomic function that use a cognitive or affective stimuli.

A number of neuroimaging studies link MPFC activity and autonomic function in humans (Gianaros et al. 2004; Lane et al., 2009; Wager et al., 2009a and 2009b). Lane et al. (2009) explored the relationship between brain and autonomic function during the induction of happiness, sadness, and disgust. Emotion was induced by through the viewing of emotionally laden film clips and through the recall of personal experience. HF-HRV (parasympathetic nervous system tone) had substantial overlap with emotion specific regional cerebral blood flow (rCBF) in the MPFC, indicating MPFC activity was associated with increased parasympathetic tone and a depressive effect on heart rate. Paralleling these results, Critchley et al. (2000a) found decreased activity in prefrontal and medial temporal regions during an arithmetic mental stressor task was associated with increased heart rate.

Activity in the MPFC and the OFC is related to increases in heart rate during social stress (Wager et al., 2009b) and activity in the MPFC is associated with decreased HF-HRV during increasingly difficult working-memory tasks (Gianaros et al., 2004). HF-HRV indexes parasympathetic influences on heart rate and decreases

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2 The Valsalva maneuver is an attempt to exhale against a closed airway.
3 Studies of autonomic function often use analysis of the heart rate power spectrum to measure parasympathetic activity. The high-frequency range (> 0.15 Hz) of the heart rate power spectrum is uniquely influenced by parasympathetic outflow. The low-frequency range component (<0.15 Hz) is jointly influenced by both the parasympathetic and sympathetic nervous system activity (Saul, 1990). Given the confluence of parasympathetic and sympathetic influences on the low frequency range of the heart rate power spectrum, research using power spectral analysis often focuses on changes in parasympathetic nervous system activity as indexed by the high frequency range of the heart rate power spectrum or high frequency heart rate variability (HF-HRV).
would tend to cause increases in heart rate. Neumann et al. (2006) find activity in the right frontal (premotor) cortex is related to increased HF-HRV during a face recognition task and decreased HF-HRV during an response inhibition paradigm. A positive correlation between prefrontal cortex function and HF-HRV was also found by Lane et al. (2009) when decreases in prefrontal activation were associated with decreases in HF-HRV activity. Differences in results across studies in the relationship between given cortical areas and cardiac activity may be caused by the different stimuli used in the various experiments and may reflect the activation of particular locations with a given cortical structure.

Wager et al. (2009b) find pregenual anterior cingulate cortex (ACC) activity is correlated with heart rate under conditions of social threat (public speech preparation task). Other studies have also found ACC activity was related to positive influences on heart rate. Gianaros et al. (2004) found task difficulty in working memory tasks resulted in decreased HF-HRV that overlapped with increased rCBF in the right ACC. Critchley et al. (2003) found increased activity in dorsal ACC function during a stressful cognitive task was related to increased sympathetic nervous system influences on heart rate. Right ACC function is also related to increases in mean arterial pressure during mental arithmetic stressor tasks (Critchley et al., 2005), and to increases in heart rate during the presentation of pictures of faces depicting emotion. In contrast to work showing a positive relationship between heart rate and ACC activity (or changes in autonomic function associated with increases in heart rate), Matthews et al. (2004) found activation of the left ventral ACC positively correlated with HF-HRV during a counting Stroop task presenting incongruent and congruent stimuli.

Activation of the amygdala is consistently associated with increased heart rate or changes in autonomic functioning associated with increased heart rate. The strong majority of studies finding a relationship between amygdala activity and autonomic function use some sort of affective stimuli. Increases in heart rate associated with increased amygdala activity have been found during the presentation of faces portraying negative affect (Critchley et al., 2005; Kuniecki et al., 2003). Gianaros et al. (2004) found task difficulty in working memory tasks results in decreased HF-HRV that overlapped with increased rCBF in the amygdala. Yang et al. (2007) found that in adolescents, activity in the right amygdala was associated with increased sympathetic dominance of heart rate during the viewing of angry faces. Mujica-Parodi et al. (2009) found that diminished coupling (correlation) between time series based on activity in limbic structures was associated with greater sympathetic activation of the ANS.

Activity in the insular cortex (IC) is associated with increases in heart rate during the presentation of emotional faces and increased sympathetic nervous system influences on heart rate during a stressful cognitive task (Critchley et al., 2005).

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4 Sympathetic dominance of heart rate is indicated by the ratio of LF HRV/HF HRV. Higher values are associated with increasing sympathetic nervous system effects on heart rate. The ratio is proportional to heart rate itself, with higher values indicating increased heart rate (Bootsma et al., 1994).
Gianaros et al. (2004) found decreases in HF-HRV were associated with increased rCBF in left insula during task difficulty in working memory tasks. In contrast Lane et al. (2009) found emotion specific rCBF in the mid-insula positively correlated with HF-HRV.

Based on a review of the research Thayer et al. (2009) argue activity in the prefrontal cortices associated with ANS function commonly leads to increases in HF-HRV (increased parasympathetic tone). All else being equal, increased parasympathetic tone should be associated with decreased heart rate. Forebrain influences on parasympathetic tone are supported by research using retrograde tract tracing in rats to identify forebrain neural pathways specifically involved in the parasympathetic control of heart rate (Ter Horst and Postema, 1997). Thayer et al. (2009) argue prefrontal activity associated with parasympathetic increases may serve to support behavior inhibition. While many neuroimaging studies support the proposed relationship between forebrain function, others have found function in the MPFC and OFC is related to increases in heart rate/decreased HF-HRV (Gianaros et al., 2004; Wager et al., 2009b). One possible explanation for this pattern of results is that activity in PFC structures can serve to both increase HF-HRV to support effortful cognition or withdraw HF-HRV to support the experience of emotion.

In contrast to the influence of forebrain structures on heart rate, activity in the limbic structures including the ACC, amygdala, and insular cortex, tends to result in increased sympathetic tone, a shift towards sympathetic dominance of heart rate, and increased heart rate. Neuroimaging studies show that brain activity leading to changes in autonomic function attendant to the processing of emotional faces or passive viewing of emotional faces results tends to be concentrated in structures in the limbic system (Critchley et al., 2005; Kuniecki et al., 2003; Yang et al., 2007). Studies of the relationship between brain activity and autonomic function during tasks related to emotion processing do not tend to find activation of prefrontal cortical structures including OFC and MPFC. While activity in the ACC is related to increased heart rate/decreased HF-HRV it does not tend to be related to emotional processing/passive emotion viewing tasks (Lane et al., 2009).

The Physiology of Low Heart Rate Reactivity

Forebrain influences on heart rate bypass limbic structures in some cases (Ter Horst and Postema, 1997), but can also cause changes in heart rate by influencing the function of the limbic structures including the amygdala. Structures in the forebrain including the medial prefrontal cortex and the orbitofrontal cortex are connected with the central nucleus of the amygdala through the cingulate cortex and the insula (Saha, 2005). Forebrain structures can exert an inhibitory influence on the limbic system in part through pathways to GABAergic neurons in the amygdala (Barbas et al., 2003; Shekhar et al., 2003). Reductions in prefrontal activity lead to the activation (disinhibition) of the central nucleus of the amygdala and increases in heart rate.
Thayer et al. (2009) have identified three different pathways through which increases in activity in the central nucleus of the amygdala (CeA) can lead to increases in heart rate. Two of these pathways involve the activation of inhibitory influences on SNS activity through the NTS. In the first of these, CeA activity leads to NTS inhibition, which leads to a reduction in inhibitory influences on sympathetic activity generated by the RVLM through a reduction in caudal ventrolateral medullary (CVLM) inhibitory inputs to the RVLM. The net effect is an increase in sympathetic nervous system activity (through a decreased inhibition of the rostral ventrolateral medullary sympathoexcitatory neurons) and an increase in heart rate. In the second pathway involving inhibitory processes mediated by the NTS, inhibition of the NTS leads to a reduction of parasympathetic nervous system activity when inputs to the nucleus ambiguous and dorsal vagal motor nucleus are reduced by the inhibition of the NTS. The third pathway through which increases in activity in the central nucleus of the amygdala can lead to increased heart rate is though direct activation of the RVLM by the CeA. Direct activation leads to increased sympathoexcitatory activity.

The limbic-hypothalamus-pituitary-adrenal (LHPA) axis also influences heart rate reactivity. This influence includes an influence on the sympathetic nervous system through connections between the paraventricular nucleus (PVN) of the hypothalamus and the IML and the RVLM. Activations of the PVN lead to increases in heart rate (Kc and Dick, 2010). Neurons in the hypothalamus that are associated with sympathetic nervous system activity overlap with those regulating the hormonal cascade that occurs during the fight or flight response (Jansen et al., 1995). The hypothalamus is directly connected with the amygdala, supporting a central role for the amygdala in heart rate increases (Risold et al., 1997).

The Relationship of Structures in the CAN with Empathy, Fear Conditioning, Emotion Recognition, and SVASB

Here I argue that an important part of the LHRR/SVASB relationship is spurious with both LRHR and cognitive characteristics leading to ASB caused by a common set of structures in the CAN. Specifically the amygdala, insular cortex, and ACC are all related to heart rate and also related to the shared experience of pain and fear conditioning (Lamm et al., 2011; Sehmeyer et al., 2009). As indicated earlier, hypo reactivity in the limbic CAN, in particular the amygdala and the ACC, also leads to lower autonomic arousal, including reduced HRR, and has been implicated in recent explanations of ASB emphasizing callous unemotional traits (Shirtcliff et al., 2009) and in explanations of psychopathy (Blair, 2005).

A key part of the proposed relationship between LHRR and ASB is the suggestion that structures in the CAN that influence HRR also influence ASB. More specifically, differential function of structures in the CAN result in both LHRR and a lack of sensitivity to negative consequences for others. CAN structures have been related to a number of phenomena that may serve to contribute to a lack of sensitivity to negative consequence for others. These include, reduced sensitivity
to pain in others, fear conditioning deficits, and disrupted emotion recognition. In addition to playing a key role in autonomic arousal, limbic structures including the amygdala, the IC, and the ACC are related to emotion (Damasio, 1998; LeDoux, 2000), empathy (Decety and Jackson, 2004 and 2006; Singer and Lamm, 2009), pain perception (Apkarian et al., 2005; Davis, 2000; Price, 2000), and fear conditioning ( Büchel and Dolan, 2000; Sehlmeyer et al., 2009), and, most critically, antisocial and criminal behavior (DeLisi et al., 2009; Raine and Yang, 2006).

A growing body of research finds that a common set of structures underpins both responses to the direct experience of pain and the observation of pain in another. Lamm et al. (2011) conducted two meta-analyses of studies relating observed pain to brain activity as measured by fMRI. The first was based on nine studies for which fMRI images were available. The second was coordinate-based meta-analysis of 32 studies. Areas activated both in experienced pain and observed pain included the border of the medial cingulate cortex and the ACC, the IC, and the amygdala. Activation of regions in the ACC and the AI have been found during the imagination of another in a painful situation (Jackson et al., 2006), in relation to the presentation of photos depicting body parts in painful everyday life situations (Jackson et al., 2005), in response to the observation of applied thermal stimuli (Hutchison et al., 1999), and in the observation of facial expressions of pain (Saarela et al., 2007).

Sehlmeyer et al. (2009) reviewed 46 studies using fMRI or positron emission tomography (PET) to study fear conditioning and/or extinction in healthy volunteers. The reviewed studies used fMRI and/or PET to identify the neurological correlates of fear conditioning. Fear conditioning paradigms include the presentation of a previously neutral stimulus with an aversive unconditioned stimulus. In normal individuals conditioning occurs when the previously neutral stimulus begins to elicit physiological reactions (e.g., change in skin conductance, increase in heart rate) that accompany the experience of the unconditioned stimulus. Extinction occurs when the conditioned stimulus no longer elicits this physiological reaction and is caused by the repeated presentation of the conditioned stimulus in the absence of the unconditioned stimulus. Sehlmeyer et al. (2009) found frequent activation of a network of fear condition related brain areas including the amygdala, insula, and ACC. Other areas also activated included hippocampus, thalamus, the cerebellum, and the sensory cortices. Other reviews of neuroimaging research report similar results in humans (Büchel and Dolan, 2000) as have reviews of fear conditioning research in animals (Fendt and Fanselow, 1999).

Phan et al. (2002) conducted a meta-analysis of 55 PET and fMRI activation studies of emotion. Emotion induction methods included visual, auditory, and recall. Results show that MPFC was commonly activated across a range of emotions. The amygdala was specifically recruited during fear, while the ACC was the region most commonly activated during anger. Activity in the IC was differentially associated with disgust. Critchley et al. (2005) support a link between amygdala reactivity and both the processing of emotional faces and increases in heart rate, finding activity in the amygdala and the IC was associated with both response to emotional face stimuli and heart rate acceleration. Sad and angry expressions
resulted in greater heart rate increases than happy and disgusted facial expression. The central role of the amygdala in the recognition of fear in others is reviewed by Adolphs (2008). Key studies show lesions of the amygdala profoundly impair the ability to recognize fearful faces (Adolphs et al., 1995; Graham et al., 2007).

Based on the above evidence I argue that amygdala, ACC, and IC are all central to the understanding of the relationship between LRHR and SVASB. The CeA serves as a key area for the translation of the psychologically induced fear and stress into cardiovascular changes. Saha (2005) notes that there is “ample evidence that the CeA plays a critical role in the integration and coordination of cardiovascular responses to acute emotional or threatening stimuli” (p. 2005). The role of CeA in determining cardiovascular responses extends to heart rate. Simulation of the CeA results in behavioral and physiological signs of anxiety and fear including increased heart rate (Iwata et al., 1987). Beyond a relationship with ANS arousal to fear inducing stimuli, under-reactivity in the amygdala has been tied to psychopathy and callous-unemotional traits (Blair, 2005; Shirtcliff et al., 2009). Studies have found reductions in the volume of the amygdala in violent offenders (Wong et al., 2007; Tiihonen et al., 2000) and psychopaths (Yang et al., 2006). Reduced activity in the amygdala has been found in adolescents with conduct disorders (Sterzer et al., 2005), violent patients with mild retardation (Critchley et al., 2000b), and psychopaths (Birbaumer et al., 2005; Kiehl et al., 2001; Veit et al., 2002).

The IC plays a central role in cardiac function. Increases in left IC activity are associated with SNS activity and increased heart rate (Nagai et al., 2010). Afferent neurons in the SNS and PNS relay information regarding the body’s physiological condition to the IC. This information is then re-represented in the anterior insular cortex (AIC) where it is available for integration in emotion and decision making (Craig, 2002, 2009). In decision making, this information can provide a predicted set of physiological reactions corresponding to different behavioral possibilities. Accordingly, a number of recent reviews have ascribed a central role for the AIC in the central representation of autonomic and visceral responses (Craig 2002, 2009; Critchley, 2005; Singer et al., 2009).

Sterzer et al. (2007) found adolescents with conduct disorder had significantly less volume in left amygdale and bilateral anterior cortex. Bilateral insular gray matter volume was also significantly correlated with empathy scores, but amygdala volume was not. Insular gray matter volume and left amygdala gray matter volume both showed a correlation with aggressive behavior, but the correlation between insular gray matter volume and aggressive behavior was stronger. Studies contrasting the fear conditioning of psychopaths and controls show that psychopaths do not experience activity in the IC and the ACC while controls do (Birbaumer et al., 2005; Veit et al., 2002).

The ACC is commonly interpreted as playing a role in the evaluation of responses (Gehring and Fencsik, 2001; Swick and Turken, 2002) (from Critchley et al., 2003). ACC lesions in humans are related to difficulties in response selection and modification (Ochsner et al., 2001; Swick and Turken, 2002). In a meta-analysis of PET studies of ACC activity, Paus et al. (1998) found task difficulty was related to ACC activation. Critchley et al. (2003) suggest that evidence regarding function
in the dorsal ACC regions can be parsimoniously interpreted as indicating dorsal ACC contributes to peripheral autonomic response to meet concurrent behavioral demands. The neuroimaging studies exploring ANS reactivity to cognitive and emotional challenge show that ACC activity is consistently related with increases in heart rate or ANS changes consistent with increases in heart rate. Reduced ACC activation has been found in psychopaths (Kiehl et al., 2001; Birbaumer et al., 2005), antisocial personality disorder patients (Kumari et al., 2006; Vollum et al., 2004), conductor disorder patients (Sterzer et al., 2007), and is related to aggression and defiance among boys (Boes et al., 2008).

While strong evidence linking reduced ACC activation to both heart rate and SVASB suggests ACC is important for the explanation of the relationship between LRHR and SVASB, the nature of this relationship is at issue as ACC does not appear to be related to heart rate specific to emotion (Critchley et al., 2005; Lane et al., 2009), but is related to heart rate and ANS changes consistent with increases in heart rate during cognitive challenge and stressful tasks (Critchley et al., 2003; Critchley et al., 2005; Gianaros et al., 2004; Matthews et al., 2004; Wager et al., 2009b).

While prefrontal cortex function is related to both heart rate and SVASB, it does not appear to be central to the explanation of the relationship between SVASB and LRHR. The amygdala, IC, and ACC all play a key role in explaining heart rate. Further, these structures are related to SVASB and fear conditioning, and are identified in studies of the neural commonalities in the experience of pain in one’s self and the observation of pain in another. In contrast, structures in the prefrontal cortex do not tend to be related to fear conditioning or implicated in the overlap between experienced and observed pain (Lamm et al., 2011; Sehlmeyer et al., 2009). Similarly, prefrontal cortex function is clearly related to ASB and has been linked to psychopathy (Glenn and Raine, 2008); however, as Blair (2007a, 2007b) notes it does not appear to be linked to the cognitive dispositions that are an important part of the definition of psychopathy (i.e., lack of concern for others). Further, a number of studies have found that psychopaths show increased activation in forebrain areas including dorsolateral prefrontal cortex (Glenn and Raine, 2008) and lesions of MPFC led to impulsive antisocial behavior but not the instrumental antisocial behavior that is the hallmark of the psychopath (Blair, 2007b). While cortical structures implicated in the control of heart rate, but located in the PFC, are not directly implicated in the LRHR/ASB relationship, PFC deficits are likely a part of a distinct substrate for impulsive and undercontrolled ASB. Where, consistent with the model of emotion regulation in the explanation of violence suggested by Davidson et al. (2000), structures in the PFC provide “top down” control of “bottom up” impulses.

The research reviewed above fairly clearly establishes a role for lower limbic structures including the amygdala, ACC, and IC in heart rate, empathy and the experience of emotion, and antisocial behavior. With regard to the relationship between LRHR/LHRR and SVASB the most parsimonious interpretation of this evidence is that hypo-reactivity in these lower limbic structures causes both LRHR/LHRR and SVASB. Absent strong evidence for an influence of LRHR/LHRR on SVASB that is causal rather than correlational it may be best to assume that the relationship
between LRHR/LHRR and SVASB is spurious, given that this explanation has the advantage of parsimony. However, there is indeed some evidence suggesting that ANS system activity can have a causal influence on behavior. This evidence, reviewed below, suggests that while some of the relationship between LRHR/LHRR and SVASB may be caused by the shared influence of lower limbic structures on both LRHR/LHRR and SVASB, it is also possible that LRHR/LHRR does have a direct causal influence on SVASB.

Explaining the Low Resting Heart Rate/Antisocial Behavior Relationship

Beyond an association due to the shared influence of CAN structures, it is also possible that ANS under-arousal including LRHR and LHRR has a causal influence on SVASB. Causal effect of the LRHR/LHRR activity on behavior can occur through autonomic feedback to limbic and higher cortical structures. Feedback on peripheral physiological states occurs through ANS afferents that return information on autonomic function to structures in the brain stem. This information is then relayed to limbic and frontal lobe areas where it is incorporated in higher order processing and exerts an influence on decision making (Craig, 2002, 2009). Afferent influences on cognitive processes are an important part of several prominent explanations of emotion and emotional influences on cognition (Damasio, 1994, 1998; Critchley, 2005; Craig, 2002, 2009; Levenson, 2003).

The argument that the ANS has a causal influence on behavior is supported by the structure of the ANS itself. Sympathetic nervous system influences on heart rate include homeostatic feedback mechanisms and what have been characterized as “feed forward” mechanisms (Dampney et al., 2002). Feedback mechanisms include the cardiopulmonary and gastrointestinal afferents that carry information from the gastrointestinal system and the cardiopulmonary system back to the brain (Cechetto and Shoemaker, 2009). Cardiopulmonary feedback mechanisms include the baroreceptor and chemoreceptor reflexes. The baroreceptor reflex occurs when feedback from arterial baroreceptors signal changes in blood pressure. Chemoreceptors are sensitive to a decrease in oxygen in the blood.

Cardiopulmonary and gastrointestinal afferents end in the nucleus of the solitary tract (NTS). Neurons conveying feedback project from the NTS directly to the rostral ventrolateral medulla (RVLM) and indirectly to the RVLM via the caudal ventrolateral medulla (CVLM). The NTS also conveys visceral feedback to higher forebrain centers including the insular cortex and the amygdala (Cechetto and Shoemaker, 2009). Information from cardiovascular and gastrointestinal afferents is relayed to the amygdala and insular cortex through relays in the parabrachial complex and through the ventral basal thalamus (Cechetto, 1987; Cechetto and Saper, 1987; Saper, 2002; Pattinson et al., 2009; Topolovec et al., 2004). It is through feedback mechanisms to the limbic structures that ANS function may causally influence limbic and higher order processes.

Neuroimaging studies demonstrate that afferent activity is represented in anterior insular cortex (AIC). Craig (2002, 2009) identified the primate lamina I...
spinothalamocoritical pathway which conveys information from fine sympathetic afferents innervating all tissues in the body. Lamina I projects to the sympathetic cell columns in the spinal cord, and to the rostral ventrolateral medulla, parabrachial nucleus, periaqueductal gray matter, and catecholomieric cell groups. Sympathetic feedback to the mid/posterior insula is conveyed directly through lamina I projections to the posterior ventromedial nucleus in the hypothalamus and indirectly through the parabrachial nucleus to the basal part of the ventromedial nucleus. Parasympathetic feedback is conveyed to the mid/posterior insula through the basal part of the ventromedial nucleus which receives projections from the NTS and from the parabrachial nucleus. The IC is then the initial cortical destination for information on the body’s physiological state.

Reduced feedback regarding LHRR (autonomic arousal) can lead to reduced concern for the impact of one’s actions on another through an inability to impute these consequences or more proximally through reduced HRR (autonomic arousal). A reduced concern for the impact of one’s actions on another is potentially attributable to the lack of a link between negative affect on the part of another and autonomic responses commonly paired with personal negative affect. In those with LHRR, autonomic arousal typically associated with fear and pain in the self fails to get linked with environmental cues indicating the experience of these emotions on the part of another. The lack of a link between negative affect in another and autonomic responses results consistent with fear and pain in the self, results in a decrease in the inferred consequences of one’s actions (including antisocial behavior) when these actions potentially result in negative emotional consequences for others. This explanation of the LHRR/SVASB can easily be conceptualized as an application of Blair’s (2005) integrated emotion system (IES) model of psychopathy. In the IES model amygdala dysfunction provides the neurological substrate for the lack of empathy and guilt that accompany the behavioral manifestations of psychopathy.5

The idea that physiological experiences form the underpinning of the experience of emotion is generally traced back to the work of William James (1894) and Carl Lange (1885). The James–Lange position holds that central representation of physiological states serve as an important component in the experience of emotion. This position assumes that autonomic arousal provides a key physiological substrate to the experience of emotion. More recently, Damasio (1994, 1998) has offered the “somatic marker hypothesis,” arguing for a role of the central representation of bodily arousal in emotion and cognition. Somatic markers represent how we feel under general classes of conditions and influence cognition in complex and

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5 One important difference between the model proposed here and Blair’s IES model is the application of limbic dysfunction to SVASB in general and not just to patterns of SVASB that meet the definition of psychopathy. Limbic deficits are viewed on a continuum with minor deficits leading to minor changes in behavior; moderate deficits leading to moderate changes in behavior; and serous deficits leading to profound patterns of SVASB potentially meeting the definition of psychopathy.
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uncertain situations. In everyday language somatic markers are described as “gut feelings.”

Kreibig (2010) reviewed 134 studies relating autonomic function to emotion. Autonomic changes including increases in heart rate were correlated with a range of distinct emotions. Kreibig’s review and other key studies in this area show that there is considerable variation in the autonomic correlates of different emotions. This suggests that specific emotions have unique autonomic signatures (see also Ekman et al., 1983). Similarly, many of the fMRI studies of the influence of the CAN on cardiac function that were reviewed earlier show that emotion induction and cognitive challenges result in changes in ANS function.

While suggestive, these studies do not definitively demonstrate that autonomic function has a causal influence on emotion and cognition. Other research, albeit limited, provides more direct evidence of a causal role for autonomic arousal. Levenson and colleagues explored the influence of peripheral action on autonomic arousal through the Directed Facial Action task. This task provides subjects step-by-step instruction to create faces that reflect emotion. The directed facial action task produced subjective emotional experience and autonomic changes associated with emotional experience (Ekman et al., 1983; Levenson et al., 1990). The causal influence of the directed facial action task on autonomic arousal was supported by differences in arousal across emotion type. For example, greater arousal during fear or anger versus disgust (Levenson et al., 1990).

Additional evidence for the importance of the central representation of autonomic states comes from studies of primary autonomic failure (PAF). PAF occurs as a consequence of loss of neuronal cells in the peripheral autonomic nervous system and results in an inability to regulate the bodily state through ANS activity (Mathias, 2000). Those with PAF show reduced mid-insula activity during effortful mental arithmetic and exercise (Critchley et al., 2001). PAF patients also show reduced response to threat stimuli during fear conditioning related activity in their insula and amygdala (Critchley et al., 2002).

Collectively, the research reviewed above provides reasonably convincing evidence that autonomic feedback can influence the experience of emotion and cognition. In normally functioning individuals, this feedback may facilitate the internal simulation of other’s autonomic states and allow the integration of this information in decision making. In those where autonomic feedback is impaired at some stage, the internal simulation of other’s autonomic states is also impaired, resulting in a less accurate understanding of the consequences of one’s actions for another. Consistent with the suggestion that the ability to simulate other’s autonomic states is key for the understanding of affective consequences, Levenson and Ruef (1992) found the accuracy of predictions of negative affect in another increased as the similarities in autonomic arousal increased.

Given the complex nature of the CAN it is difficult to localize the potential causes of impaired autonomic feedback, but there are three general sets of CAN/ANS structures that should be considered. The first is the set of structures that are responsible for the generation of the ANS response itself. I have argued that these are amygdala, ACC, and IC. Reduced function in these areas should lead to
reduced sympathetic outflow, reduced heart rate increases, and reduced afferent feedback regarding autonomic arousal, as a consequence of lower arousal itself. This reduced afferent feedback may play an important part in explaining the link between reduced activity in these structures and increases in SVASB. It is also possible that impaired function of the sympathetic and parasympathetic nervous system afferents that return information on peripheral physiological arousal may result in reduced autonomic feedback and a reduced ability to infer affective consequences. Lastly, the central representation of autonomic feedback for emotional and cognitive processes may be impaired when brain stem and cortical structures responsible for the representation of afferent feedback function poorly.

In Sum

This chapter has a number of central points of emphasis. Perhaps the most important is the argument that investigations of the relationship between LHRR/LRHR and SVASB should consider the possibility that the relationship between LHRR/LRHR and SVASB is spurious, with both being explained by reduced lower limbic activity (amygdala, IC, ACC). Research has shown lower limbic structures are related to heart rate, fear conditioning, empathy for pain, and antisocial behavior, and has established a link between LHRR/LRHR and SVASB, but has not shown that this relationship exists after controls for lower limbic function. Beginning to tease out the direction of causality between ANS function, brain function, cognitive/emotional dispositions, and SVASB is likely to yield substantial advances to our understanding of the biological substrates of SVASB.

Beyond arguing for attention to the possibility of spuriousness, this chapter also reviews substantial, albeit indirect, evidence for the influence of ANS feedback on emotion, cognition and by extension SVASB. Further, the review of research in support of this argument suggests that it is decreased afferent feedback to cortical structures that potentially accounts for the unique contribution of ANS activity to SVASB. If this reduced feedback is indeed causally related to SVASB, it is likely through impaired ability to understand the affective consequences of one’s actions for another.

This argument, that a reduced sensitivity to consequence for others and for one’s self explains part of the relationship between LRHR/LHRR and SVASB, leads to eminently testable propositions. For example, if an important part of the relationship between LRHR/LHRR and SVASB is explained by sensitivity to other/self consequences, we should anticipate first that LRHR/LHRR will be related to a lack of empathy/concern for the consequences of one’s actions for another. We should also anticipate that the relationship between LRHR/LHRR and SVASB will be mediated by variation in these same empathy/concern measures. Encouragingly, these propositions are testable with little more than a carefully considered survey and some way to measure heart rate.

A final argument, implied in the above review and made expressly here, is that deficits leading to the relationship between LRHR/LHRR and SVASB are largely

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deficits of structures involved in the sympathetic nervous system tone, where underarousal in the SNS is both a corollary and cause of lower concern with the negative affective consequences of one’s actions for others and this leads to increased SVASB. This is not to argue that parasympathetic nervous system influences are entirely unimportant for the LRHR/LHRR and SVASB relationship, but rather that their importance is substantially diminished relative to SNS influences.

Recent models of social cognition and emotion regulation have argued that PNS plays an important role in emotion regulation and social decision making (Porges, 2001, 2007; Thayer and Lane, 2000). Porges (2001, 2007) argues that ‘orienting responses’ are a key feature of social engagement and that increases in parasympathetic tone support the ability to appropriately respond to social stimuli (Porges, 2001, 2007). Recalling that parasympathetic tone exerts downward pressure on heart rate, this indicates that an inability to lower heart rate through parasympathetic tone will be associated with weakened emotional and behavioral regulation. This suggests that the major function of the PNS may be the suppression of SNS in support of behavioral regulation. If this is the case PNS may still have a secondary but still important influence on the relationship between LHRR/SVASB, through the withdrawal of parasympathetic tone when the potentiation of emotion is appropriate. The argument for the primacy of the SNS in the explanation of the LHRR/SVASB relationship yields another testable proposition, that measures of SNS function should mediate more of the relationship between LHRR/SVASB than measures of PNS function. While the hardware associated with specific measures of the SNS (skin conductance) and the PNS (HF-HRV) are more expensive than the cost associated with the administration of a survey and the taking of the pulse, they are still relatively inexpensive (thousands rather than tens or hundreds of thousands).

The conceptualization of the relationship between LHRR/SVASB advanced here argues for a multidimensional criminal propensity. It is widely acknowledged that an important part of between individual differences in the tendency to engage in ASB is explained by impulsivity, or in the parlance of Gottfredson and Hirschi (1990), low self-control. Yet, impulsivity is also associated with a wide variety of acts that however ill advised are nonetheless not antisocial. And while impulsivity is clearly important in the explanation of ASB, it is when this characteristic is combined with an inability to appreciate and experience the consequences of one’s actions for others that a tendency toward impulsive acts gets translated into a profound and enduring pattern of antisocial behavior across the life course.

Enduring individual differences in criminal propensities then have two primary biological substrates. One linked to frontal lobe dysfunction, and the other to impaired limbic/ANS function. In the first, frontal lobe dysfunction leads to poor impulse control and in the second they lead to a lack of empathy or guilt. Yang et al. (2005) provide indirect evidence of these distinct substrates in comparisons of unsuccessful (arrested) psychopaths, successful psychopaths (not arrested), and community controls. Successful psychopaths have higher prefrontal gray matter volume than unsuccessful psychopaths. Differences between successful psychopaths and community controls in prefrontal gray matter volume were
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not significant. In the context of the above criminal behavior substrates, this suggests that successful psychopaths may have the lack of guilt or empathy that is the hallmark of the psychopath, but nonetheless restrain themselves from risky SVASB due to intact frontal lobe function. The successful psychopaths do not understand the negative affective consequences of their actions, and therefore are not deterred by them, but are nonetheless able to restrain themselves from acts of SVASB that would result in their arrest. In contrast, unsuccessful psychopaths with both the emotional hallmark of psychopathy and frontal lobe dysfunction neither understand the negative affective consequences of their actions nor are they able to restrain themselves in the face of the criminal justice system consequences.

References


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