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Review

Psychophysiological correlates of aggression and violence: an integrative review

Christopher J. Patrick*

Department of Psychology, University of Minnesota, Elliott Hall, 75 East River Road, Minneapolis, MN 55455, USA

This paper reviews existing psychophysiological studies of aggression and violent behaviour including research employing autonomic, electrocortical and neuroimaging measures. Robust physiological correlates of persistent aggressive behaviour evident in this literature include low baseline heart rate, enhanced autonomic reactivity to stressful or aversive stimuli, enhanced EEG slow wave activity, reduced P300 brain potential response and indications from structural and functional neuroimaging studies of dysfunction in frontocortical and limbic brain regions that mediate emotional processing and regulation. The findings are interpreted within a conceptual framework that draws on two integrative models in the literature. The first is a recently developed hierarchical model of impulse control (externalizing) problems, in which various disinhibitory syndromes including aggressive and addictive behaviours of different kinds are seen as arising from common as well as distinctive aetiologic factors. This model represents an approach to organizing these various interrelated phenotypes and investigating their common and distinctive aetiologic substrates. The other is a neurobiological model that posits impairments in affective regulatory circuits in the brain as a key mechanism for impulsive aggressive behaviour. This model provides a perspective for integrating findings from studies employing different measures that have implicated varying brain structures and physiological systems in violent and aggressive behaviour.

Keywords: psychophysiology; aggression; violence; psychopathy; externalizing

1. OVERVIEW

The paper is organized into three sections. The first section discusses alternative methodological approaches to investigating the neurobiological bases of aggressive behaviour, highlighting the key role that psychophysiological studies can play in this area of research. The second section provides a review of major lines of evidence emerging from psychophysiological investigations of aggressive and violent behaviour, which have been published to date. The third section presents an integrative conceptual framework for interpreting available evidence from studies of this kind, based on the two aforementioned models, and discusses avenues for future research.

2. APPROACHES TO INVESTIGATING THE NEUROBIOLOGICAL BASES OF AGGRESSIVE BEHAVIOUR: THE ROLE OF PSYCHOPHYSIOLOGICAL MEASUREMENT

A variety of different methodological approaches exist for studying the role that neurobiological factors play in violent and aggressive behaviour. These approaches contribute to knowledge in differing, but complementary ways. They can be grouped according to the particular sorts of questions they address regarding violent/aggressive behaviour.

One approach consists of *aetiological studies* that address the following question: 'What constitutional and environmental factors contribute *causally* to the occurrence of violence/aggression?' For example, behavioural genetic studies address this question by comparing the degree of similarity in tendencies towards aggression between identical versus fraternal twins, or between identical twins who are reared in the same versus differing family environments. Studies of this type can be used to estimate the proportion of variance in a behavioural phenotype that is attributable to additive genetic influence compared with shared (family) or non-shared environmental influence. Twin research designs can also be used to test for moderating effects of varying environments on the proportional contribution of genetic factors to a behavioural phenotype (cf. Purcell 2002).

Molecular genetic studies provide another means for investigating aetiologic contributions to behavioural phenotypes such as aggression, contributions made by specific genes or gene sequences, or by interactions between specific genes and environmental factors (e.g. Caspi *et al.* 2002). In addition to behavioural and molecular genetic investigations, a third approach to investigating aetiologic contributions of neurobiological variables to aggressive behaviour is longitudinal–developmental research. Studies of this kind provide

*cpatrick@umn.edu

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a basis for inferring aetiologic influences because temporal relations between putative causal factors and outcome variables can be specified with precision in quantitative models of longitudinal data (cf. Card & Little 2007).

In addition, it is important to understand what neurobiological systems are affected by these basic aetiologic factors and how individual differences in the structure and functioning of neurobiological systems dispose towards and give rise to aggressive actions. To do so, it is necessary to measure characteristics of neurobiological systems within individuals. Psychophysiological and neurochemical assessment methods provide this capability.

In this regard, a second type of approach to the investigation of neurobiological factors in aggressive behaviour consists of *marker* studies. The basic question addressed by studies of this type is: 'What biological differences are characteristic of aggressive individuals compared with non-aggressive individuals?' Methods for addressing this question include (i) peripheral and electrocortical psychophysiology (to index visceral/autonomic, somatic/muscular and brain activity differences), (ii) structural and functional neuroimaging (to detect differences in brain anatomy and activity), and (iii) neurochemical assays (to assess differences in brain neurotransmitter levels and activity). Some biological variables on which individuals who exhibit (or are at risk for) aggressive behaviour differ reliably from non-aggressive individuals include resting heart rate levels, amplitude of brain event-related potential (ERP) responses, volume and activity in specific brain regions and levels of the brain neurotransmitter serotonin. Marker studies are valuable because they point to biological systems and functions that may be targets of genetic and/or environmental influence and because they can provide biologically based indicators of vulnerability that can be used to identify candidates for prevention programmes.

A third type of approach to investigating neurobiological factors in aggression consists of *process* studies. The question addressed by studies of this type is: 'How do aggressive individuals differ in their psychological processing of stimuli and events?' The emphasis is on understanding the functional role that neurobiological reactivity differences play in dispositions towards aggression and in instigating aggressive acts. Cognitive and affective neuroscience studies contribute to understanding at this level by measuring brain activation within a performance task designed to index some specific psychological process or processes. The focus of such studies is on understanding differences in online cognitive or emotional processing that appear relevant to some clinical phenomenon of interest, such as aggression. Measurement techniques used to study brain activity associated with online processing include functional neuroimaging (positron emission tomography (PET) and functional magnetic resonance imaging (fMRI)) and electroencaphalography (EEG)/ERP recording.

These brain measurement techniques each have relative advantages and disadvantages. fMRI provides fine-grained spatial resolution, which permits activity to be precisely localized within specific regions of the brain, but its temporal resolution is limited. EEG provides fine-grained resolution in both temporal and spectral (frequency) domains, but its spatial resolution is limited in comparison with fMRI. However, the spatial resolution of EEG can be improved through multi-electrode, dense array recording, which provides for more precise estimation of signal sources at and below the brain's surface ('source modelling'). The resolution of EEG can be improved even further by referencing EEG data to structural or functional neuroimaging data collected from the same participant, either concurrently or in separate test sessions. In conjunction with continuous measurement of activity along dimensions of time and frequency, this technique provides for even finer grained localization of underlying sources of brain activity ('source imaging') because EEG source models can be constrained to accommodate known neuroanatomic structures or regions of brain activation established by MRI recording.

Section 3 provides a brief overview of findings from studies that have used psychophysiological measurement techniques to investigate neurobiological factors contributing to the behaviour of persistently aggressive individuals. It will be seen that most published studies of this sort, including the majority of studies that have employed neuroimaging methods, fall into the category of marker studies.

3. REVIEW OF FINDINGS FROM AUTONOMIC, ELECTROCORTICAL AND BRAIN IMAGING STUDIES OF AGGRESSIVE INDIVIDUALS

(a) *Autonomic response measures: cardiovascular and electrodermal activity*

Studies of children and adolescents exhibiting antisocial behaviour (i.e. conduct problems encompassing non-aggressive as well as aggressive behaviour) have yielded consistent evidence of lower resting levels of autonomic activity—most notably heart rate (HR) and also to some extent skin conductance (SC)—in comparison with control youth (Lorber 2004; Ortiz & Raine 2004). The finding of low resting heart rate in particular appears to be especially robust among children with aggressive behavioural tendencies (Scarpa & Raine 1997). Findings with respect to autonomic *reactivity* to noxious or threatening stimuli have been more mixed, but as a whole the available findings point to *enhanced* HR and SC responses to stressors in children exhibiting aggressive conduct problems specifically (Lorber 2004), particularly in those exhibiting *reactive* aggression; proactively aggressive children if anything tend to show attenuated reactivity to stressors compared with control children (Hubbard *et al.* 2002).

In addition, studies of parasympathetic versus sympathetic mediation of cardiovascular activity have yielded some evidence of weaker vagal–parasympathetic regulation of HR activity in children and adolescents exhibiting aggressive conduct problems (Mezzacappa *et al.* 1997; Beauchaine *et al.* 2001). Investigators in this area have posited that this lack of vagal control, evidenced by enhanced HR variability under circumstances involving stressors or challenges, combines with chronic underarousal and weak inhibitory capacity (reflected by lower resting HR and fewer spontaneous

SC responses, respectively) to lower the threshold for impulsive aggressive behaviour (Beauchaine *et al.* 2001). Other research has identified weak vagal control as a variable associated with the development of both internalizing (emotional dysregulation) and externalizing problems in at-risk children (e.g. El-Sheikh *et al.* 2001). Taken together, these findings are consistent with the idea that aggression in children entails difficulties regulating anger and other emotional reactions—with consequent enhancement of defensive reactivity under conditions of threat.

In studies of adults, one prominent focus has been on differences in autonomic (particularly cardiac) activity in individuals high on aggression-related traits, such as hostility, anger expression and type A personality. A meta-analysis of the association between trait hostility and cardiovascular reactivity by Suls & Wan (1993) reported that although effect sizes as a whole in studies of this kind tended to be small, robust positive effects were evident in studies that examined relations between trait hostility (particularly when defined by overt expressions of anger, such as verbal and physical aggression) and cardiovascular (particularly blood pressure) reactivity in situations involving interpersonal stress or provocation (i.e. as opposed to physical stressors). Studies published since this meta-analysis have not yielded positive findings in all cases (e.g. Gallo *et al.* 2000), but when obtained significant effects have generally been in the direction of heightened autonomic reactivity for high trait-aggressive individuals during interpersonal stress (e.g. Smith & Gallo 1999; Peters *et al.* 2003).

Studies comparing autonomic reactivity in adults with and without a history of violent behaviour have yielded less consistent results. For example, physiological studies of men who have assaulted their romantic partners have not revealed consistent differences in relation to non-assaultive men. A study by Gottman *et al.* (1995) suggested a possible explanation for this in terms of two distinctive subgroups of male batterers, one who showed decreases in HR activity during a marital interaction (type 1) and the other who showed increases in HR (type 2). Type 1 batterers scored higher on antisocial traits and were more hostile and contemptuous towards their spouses and more assaultive towards other people in general, whereas type 2 batterers scored higher in interpersonal dependency. However, this pattern of results has not been replicated in subsequent studies (Meehan *et al.* 2001; Babcock *et al.* 2004). Few studies have examined physiological response differences in individuals with a history of physical abuse towards children. One study by Frodi & Lamb (1980) reported enhanced autonomic reactivity to infant emotional displays, whether positive or negative (i.e. smiling or crying), in women with a history of abuse compared with non-abusive women.

Most published studies involving autonomic response qualify as marker studies because they have focused on detecting simple reactivity differences between aggressive and non-aggressive individuals. In some cases, speculative inferences have been made about underlying psychological processes based on the use of particular measures (e.g. cardiac variability as an index of vagal regulation) or particular stimulus manipulations (e.g. aversive versus non-aversive cues and interpersonal versus physical stressors). However, studies of this kind for the most part have not set out to measure specific processing differences online. One exception is recent work by Verona and colleagues examining the mediating role of negative emotional activation in enhancing punitive behaviour among aggression-prone individuals. Verona *et al.* (2002) used increased startle reactivity to index unpleasant activation associated with threat versus absence of threat in a laboratory aggression paradigm. Individuals high on self-report traits of anxiousness, alienation and aggressiveness showed enhanced unpleasant activation during shock-threat periods (as evidenced by heightened baseline startle reactivity), and in conjunction with this, enhanced aggressive behaviour (i.e. delivery of stronger shocks to a confederate). This finding was interpreted as supporting Berkowitz's (1990) theory that negative emotional activation operates to prime aggressive behaviour. Subsequent work (e.g. Verona & Curtin 2006) has indicated that this facilitative effect of negative emotion on aggression may be stronger in men than in women, in line with prior research demonstrating moderating effects of gender on stress-induced aggression (cf. Hokanson 1970).

In summary, research to date has generally revealed lower baseline levels of autonomic arousal, but higher autonomic reactivity to stressful events, in aggressive children and adolescents. Findings with adult samples have been less consistent, but in general have indicated enhanced autonomic reactivity to stressors (interpersonal stressors in particular) in hostile–aggressive individuals. As discussed further in the third major section below, the general finding that aggression-prone individuals show enhanced autonomic reactivity to stressful events fits with the hypothesis that violent behaviour entails a breakdown in normal affective regulatory capacity (Davidson *et al.* 2000). By contrast, markedly different results are evident in the psychophysiological literature on adult psychopathy. Most of these studies have relied on diagnoses of psychopathy based on Cleckley's (1941) criteria for the disorder or Hare's (2003) Psychopathy Checklist-Revised (PCL-R). Adult psychopathic offenders, relative to non-psychopathic offenders, tend to show *reduced* electrodermal response to aversive cues and during anticipation of stressful events (Hare 1978; Arnett 1997; Lorber 2004). On the other hand, psychopathy tends not to be consistently associated with differential HR reactivity to aversive or stressful stimuli (Lorber 2004), or with differential baseline levels of either HR or electrodermal arousal (Hare 1978; Arnett 1997; but see Hansen *et al.* 2007).

These contrasting results are noteworthy because higher levels of psychopathy in offender samples are reliably associated with increased tendencies towards violence and violent recidivism (Porter & Woodworth 2006). The construct of psychopathy as it has been studied in adults entails, in part, tendencies towards impulsivity, recklessness and aggression—and it is these antisocial deviance ('factor 2'; Hare 2003) features that are most predictive of violent offending and recidivism (Hemphill *et al.*1998; Walters 2003). However, a full diagnosis of psychopathy additionally requires the presence of distinctive affective–interpersonal features, including absence of remorse, deficient

empathy, shallow affectivity, glibness, grandiosity and manipulativeness (Hare 2003). While at odds with the general trend of results for impulsive–aggressive individuals, the finding of diminished autonomic (in particular electrodermal) reactivity to stressors is consistent with theories that have emphasized insensitivity to punishment or reduced capacity for fear in psychopathy (e.g. Lykken 1995)—especially in relation to its affective–interpersonal features (Patrick 1994). These distinctive features of psychopathy tend to be more related to instrumental–proactive aggression (Porter & Woodworth 2006) than to the impulsive–reactive type that has been emphasized historically in physiological studies of aggression. Thus, individuals who exhibit the core-affective features of psychopathy need to be considered separately from other types of violent offenders in attempting to understand neurobiological substrates of aggressive behaviour.

(b) *Electrocortical response measures: EEG and ERP*

Early investigations of brain differences in violent criminal offenders focused on abnormalities in EEG activity. A relatively consistent finding in these early studies was enhanced cortical slow wave activity, particularly in the delta (less than 4 Hz) frequency range (cf. Volavka 1990). This finding was not linked systematically to any particular scalp region, although some investigators focused on frontal or temporal regions. A good deal of this early literature suffered from methodological problems including subject selection biases and non-quantitative scoring of EEG data. However, subsequent work using improved methodologies has successfully replicated this finding and some evidence has emerged that increased slow wave EEG activity in adolescence predicts the emergence of antisocial behaviour later in life (i.e. official criminal convictions of any kind; Raine *et al.* 1990). Theoretical interpretations of the association between enhanced slow wave EEG and violent offending or criminal offending more broadly have focused on cortical immaturity resulting in impaired inhibitory control (Volavka 1990), and cortical underarousal that predisposes towards compensatory stimulation seeking (Raine *et al.* 1990). In addition to this EEG slow wave research, a newer line of work by Harmon-Jones and colleagues has examined differences in left versus right frontal EEG asymmetry in relation to induced states of anger and trait differences in anger proneness as assessed by self-report (for a review of this work, see Harmon-Jones 2003).

The published literature on ERP response in aggressive individuals is less extensive than the EEG literature. Nonetheless, associations with aggression have been reported for various components of the ERP. The most consistent association has been found for reduced amplitude of the P300 response component in oddball tasks in which participants respond to intermittent target stimuli interspersed with more frequently occurring non-targets. Diminished P300 has been reported especially among individuals exhibiting aggression of the impulsive variety (e.g. Branchey *et al.* 1988; Barratt *et al.* 1997; Gerstle *et al.* 1998). In view of theoretic models that interpret P300 response as reflecting the online updating of cognitive representations (Donchin & Coles 1988), reduced P300 amplitude in impulsively aggressive individuals implies some impairment in higher (perhaps working memory related) cognitive function.

Reduced P300 amplitude has also been reported in individuals diagnosed with antisocial personality (Bauer *et al.* 1994), a disorder that often includes impulsive aggressive behaviour. In addition, however, reduced P300 has been found for a variety of other impulse control problems, most notably alcohol dependence (cf. Polich *et al.* 1994), and also drug dependence, nicotine dependence, child conduct disorder and attention deficit hyperactivity disorder (cf. Iacono *et al.* 2002). The implication is that reduced P300 amplitude may reflect something these disorders have in common, rather than what is unique to any one of them. As discussed in the third major section below, reduced P300 amplitude appears to be an indicator of the broad 'externalizing' factor (Krueger 1999) that these disorders share. I hypothesize that this broad factor, which has been conceptualized as a dispositional vulnerability to impulse control problems of various kinds (Krueger *et al.* 2002), represents a key to understanding aggressive behaviour, in particular impulsive–reactive aggression.

In contrast with findings from studies of impulsive aggressive individuals, Stanford *et al.* (2003) reported no difference in P300 amplitude to auditory target stimuli in psychiatric outpatients characterized as 'premeditated aggressors' compared with controls. Similarly, Barratt *et al.* (1997) found no evidence of a relationship between premeditated aggression and P300. Results from these studies indicate that the association between reduced P300 and aggression may be specific to individuals who manifest aggression of an impulsive nature. Studies examining the association between psychopathy and P300 amplitude have yielded mixed results, with some showing a negative association, others a positive association and still others no association (see reviews by Raine 1989, 1993, along with more recent work by Kiehl *et al.* 1999).[1] As noted earlier, these inconsistent findings could reflect the fact that a diagnosis of psychopathy includes affective–interpersonal features in addition to antisocial deviance symptoms. The affective–interpersonal features, which tend to be associated more with proactive rather than impulsive aggression, may moderate the association between psychopathy and brain potential response in some samples.

As with studies using autonomic measures, most published EEG and ERP studies of aggressive individuals consist of marker studies aimed at detecting differences in brain activity at rest or in simple task procedures. Given the fine-grained information available from EEG/ERP measurement in time and frequency domains, together with advances in EEG source localization methods, there is potentially much to be learned in the future from process-oriented studies of aggression-prone individuals using electrocortical measures.

(c) *Neuroimaging measures: CT, SPECT, PET and MRI.*

Three older neuroimaging studies of violent individuals employed computerized tomography (CT), a technique in which fluctuations in X-ray beams passed

through the brain, associated with regional differences in tissue density, are used to index brain structure. Tonkonogy (1991) and Wong *et al.* (1994) reported evidence mainly of temporal lobe abnormalities in psychiatric patients with violent behaviour. Blake *et al.* (1995) found evidence of abnormalities in frontal as well as temporal brain regions in a sample of 31 homicide offenders.

In parallel with this, studies using single photon emission computerized tomography (SPECT), a procedure in which photons emitted by a radioactive isotope injected into the brain are measured to index activity in specific regions of interest, have consistently revealed evidence of abnormalities in the prefrontal cortex and the temporal lobes. Three studies that used SPECT to assess neuronal activity at rest in psychiatric patients exhibiting aggression involving severe antagonism, physical attack or property destruction (Amen *et al.* 1996; Hirono *et al.* 2000), or individuals convicted of impulsive violent offences (Soderstrom *et al.* 2000) found evidence of reduced blood flow in both the prefrontal cortex and the temporal lobes (the left temporal lobe, specifically, in two of the three studies). In contrast with the activity reductions in these brain regions, Amen *et al.* (1996) reported evidence of *increased* activity in basal ganglia and subcortical (limbic) regions in their aggressive patient sample. One other SPECT study (Kuruoglu *et al.* 1996) reported reduced blood flow in frontal brain regions in alcoholic individuals with comorbid antisocial personality relative to non-alcoholic controls. SPECT has also been used to examine differences in neurotransmitter function in impulsively violent offenders. Studies of this kind have revealed evidence of abnormal dopaminergic neurotransmission in the striatum and diminished serotonin transporter density in the midbrain (Tiihonen *et al.* 1995, 1997).

Positron emission tomography (PET), like SPECT, relies on measurement of particles (positrons, in this case) from an injected radioisotope to index neural activity or neurotransmitter function in specific brain regions. Studies using PET comprise the largest subset of published neuroimaging studies of aggression. The majority of these studies have reported evidence of prefrontal brain dysfunction in violent individuals compared with controls (for a detailed review, see Patrick & Verona 2007). Some of these studies have measured brain activity at rest (e.g. Volkow & Tancredi 1987), others during simple (e.g. continuous performance) tasks designed to activate the prefrontal cortex (e.g. Raine *et al.* 1994, 1997). Raine *et al.* (1998) subdivided violent participants from the Raine *et al.* (1997) study, consisting of 41 convicted murderers, into predatory (proactive) and affective (impulsive) subgroups based on the nature of their crimes, and found that prefrontal dysfunction was specific to the affective subgroup. Other studies have used PET imaging to investigate brain reactivity to drugs that activate the serotonin system in aggressive and non-aggressive individuals (e.g. Siever *et al.* 1999; New *et al.* 2002). These studies have reported blunted reactivity to serotonin agonists (as evidenced by lower levels of glucose metabolism) among impulsive–aggressive patients compared with controls in regions of prefrontal cortex, particularly orbitofrontal and ventromedial regions. Other brain regions implicated with some consistency in these and other PET imaging studies include temporal cortex, anterior cingulate cortex, and to a lesser degree, hippocampus and amygdala.

With regard to the amygdala and hippocampus, Raine *et al.* (1997) found evidence of abnormal asymmetry (i.e. decreased functioning on the left side and increased functioning on the right one) in both these structures in murderers compared with controls. In a PET study of serotonin-binding potential, Parsey *et al.* (2002) reported a significant negative relationship between reported lifetime aggression and binding in brain regions including the amygdala (but not hippocampus). George *et al.* (2004), in a study of domestic abusers with comorbid alcoholism, reported decreased correlations between glucose activity in the amygdala and glucose activity in various cortical structures compared with non-violent controls. The authors postulated that these decreased associations reflected a lack of cortical input to the amygdala associated with increased sensitivity to environmental stressors among impulsively violent individuals.

Comparatively, few studies have used structural magnetic resonance imaging (MRI) to investigate neuroanatomic differences associated with impulsive aggressive behaviour, and these studies have yielded less consistent results than PET studies. Two studies, one involving temporal lobe epilepsy patients with aggressive–assaultive behaviour (Woermann *et al.* 2000) and the other female patients diagnosed with borderline personality disorder (van Elst *et al.* 2003), reported evidence of reduced grey matter volume in regions of prefrontal cortex, and the latter of these also reported volume reductions in anterior cingulate cortex, hippocampus and amygdala. However, another study by Dolan *et al.* (2002) that compared impulsive–aggressive patients with controls reported a significant reduction in temporal lobe volume in the patients but no reduction in frontal lobe volume, and two other studies that examined subcortical structures (Laakso *et al.* 2000; van Elst *et al.* 2000) found no difference between violent and non-violent patient groups in hippocampal or amygdala volume.

Even fewer studies of violent individuals have been conducted using fMRI. Raine *et al.* (2001) examined brain activation during a working memory task in small groups of community participants with histories of serious violent behaviour and/or early abuse ($n=4$–5) relative to a healthy control group ($n=9$). When compared with controls, violent individuals who had been abused as children showed reduced right hemisphere activation (particularly in right temporal regions), whereas abused individuals without violence showed lower left, but higher right activation of the superior temporal gyrus. In addition, both of these groups showed generally reduced cortical activation during task processing, particularly in the left hemisphere. The authors interpreted these findings as indicating a unique role of right hemisphere dysfunction, when combined with exposure to early abuse, in violent behaviour. However, the findings of this study must be interpreted with caution due to the small sample sizes.

In addition to studies of violent individuals, a number of published neuroimaging studies have examined brain differences in individuals diagnosed as psychopathic using Hare's (2003) PCL-R (for a review, see Raine & Yang 2006). Aside from the fact that these studies did not focus specifically on aggressive behaviour, their findings are difficult to compare with those already reviewed owing to differences between individuals diagnosed as psychopathic and individuals who tend to exhibit impulsive (reactive) aggression. In particular, psychopathy is believed to involve a deficiency in negative emotion (in particular, anxiety or fear; Patrick 1994; Lykken 1995) and it has been theorized that psychopaths differ from other aggressive–antisocial individuals in subcortical brain structures that mediate basic emotional processing (e.g. the amygdala; Intrator *et al.* 1997; Blair 2006). The few neuroimaging studies that have separated violent offender participants into emotionally overreactive versus predatory-psychopathic subgroups (e.g. Raine *et al.* 1998) have reported different patterns of results for these groups. For these reasons, it is important to consider psychophysiological (including neuroimaging) findings for impulsively violent individuals separately from findings for individuals diagnosed as psychopathic or exhibiting mainly predatory (proactive) aggression.

From the foregoing summary of findings from published neuroimaging studies of aggressive individuals, it is apparent that most studies of this kind fall into the category of marker studies. Overall, the findings of these studies point to deviations in the structure and functioning of frontal, temporal and anterior cingulate brain regions as relevant to an understanding of impulsive–aggressive behaviour. However, the specific cognitive and affective processes that are disrupted in connection with these brain abnormalities remain to be established. Clearly, PET and neuroimaging methods have been used extensively in basic cognitive neuroscience research and hold great potential to contribute to understanding of such processes in relation to aggressive behaviour. An illustration of the potential of neuroimaging techniques in this regard comes from two recent PET imaging studies that examined brain activation during states of anger induced using imagery manipulations. Pietrini *et al.* (2000) reported evidence of significant decreases in blood flow in the prefrontal cortex (especially the ventromedial region) among healthy control participants during imaginal processing of a scenario in which they expressed unrestrained aggressive behaviour compared with reactivity during a neutral scenario. By contrast, Drexler *et al.* (2000) reported *increased* activity in inferior frontal cortex during imagery of a personal anger scene versus a neutral scene in a sample of nicotine-dependent men included as control participants in a study of cocaine-dependent men. The fact that participants in the Pietrini *et al.* study imagined themselves actively aggressing may account for the difference in findings for these two studies. Activation of ventromedial prefrontal cortex may occur primarily during anger states in which aggressive urges are suppressed rather than enacted (cf. Davidson *et al.* 2000). Notably, cocaine-dependent men in the Drexler *et al.* study showed relative decreases in frontal brain activation during anger imagery. The authors' interpretation was that drug-dependent individuals exhibit deficits in anger regulation that dispose them towards aggressive behaviour and relapse under circumstances of stress.

4. AN INTEGRATIVE CONCEPTUAL FRAMEWORK FOR INTERPRETING RESULTS FROM PSYCHOPHYSIOLOGICAL STUDIES OF AGGRESSION AND VIOLENCE

To provide a basis for thinking integratively about existing research on the psychophysiology of aggression, two conceptual models are considered: (i) a hierarchical model of externalizing syndromes that conceives of various types of impulse control problems, including aggressive-antisocial behaviour, as manifestations of a common dispositional vulnerability and (ii) a neurobiological model that views persistent aggression as arising from dysfunction in a set of interconnected brain systems (including the prefrontal cortex, anterior cingulate cortex and amygdala) that function to regulate affective states, including anger.

(a) *Hierarchical model of the externalizing spectrum*

An integrative hierarchical model of the externalizing spectrum—encompassing child and adult antisocial deviance, alcohol and drug dependence and impulsive personality traits—has recently been developed to account for the systematic covariance (comorbidity) known to exist among these phenomena. The foundation for this model was a behaviour genetic study by Krueger *et al.* (2002). These authors performed confirmatory factor analyses on diagnostic and psychometric data from a large mixed-gender sample of monozygotic and dizygotic twins ($N=1048$) recruited from the community. Variables in the analysis included symptom scores for four DSM disorders (child conduct disorder, adult antisocial behaviour, alcohol dependence and drug dependence) along with a self-report measure of disinhibitory personality traits consisting of reversed scores on the constraint factor of Tellegen's (1982) multidimensional personality questionnaire. The best-fitting model of the data was a common pathways model in which a shared general factor (externalizing) contributed aetiologically to all primary variables in the model. Over 80% of the variance in this common factor was found to be attributable to additive genetic influence. Non-shared environmental influence accounted for most of the residual variance in each primary variable not accounted for by the broad externalizing factor, although for conduct disorder a significant contribution was also found for shared environment. Based on these findings, Krueger *et al.* (2002) advanced a hierarchical model in which a largely heritable general vulnerability contributes to the development of various traits and disorders in the externalizing spectrum, but the distinctive expression of this underlying vulnerability (i.e. as disinhibitory personality traits, antisocial behaviour of different sorts or as alcohol or drug problems) is determined by other specific aetiologic influences.

Krueger *et al.* (2007) extended this work by undertaking a more comprehensive analysis of personality trait and behavioural constructs within the domain of externalizing. Self-report items were developed to index constructs embodied in the diagnostic criteria for conduct disorder, adult antisocial behaviour, alcohol dependence, drug dependence (cf. Krueger *et al.* 2002), along with other constructs (e.g. impulsivity and aggression of varying types, nonconformity and stimulation seeking) identified as relevant to externalizing through a review of existing literatures. Quantitative methods including item-response modelling and confirmatory factor analysis were applied across multiple rounds of data collection to refine the item set as well as clarify the nature of constructs linked to the broad externalizing factor. The final result was a set of 23 constructs, each assessed by a distinctive scale. Confirmatory factor analyses of these 23 construct scales yielded evidence of one superordinate factor (externalizing) on which all scales loaded and two subordinate factors that accounted for residual variance in particular scales. The two subordinate factors were independent from one another statistically and also from the superordinate externalizing factor. Table 1 lists the 23 scales of the externalizing inventory along with loadings of each scale on the general externalizing factor and on each of the two subfactors. It can be seen that all of the scales loaded 0.45 or higher on the general factor, with irresponsibility and problematic impulsivity scales demonstrating the highest loadings. The table also shows that variance in some of the scales not accounted for by the general externalizing factor was associated with one or the other subfactor. One subordinate factor was defined by residual variance in subscales measuring aggression (all three types), callousness, excitement seeking, rebelliousness and (low) honesty; the other subfactor was defined by residual variance in subscales indexing alcohol/drug use and substance-related problems. It is important to note that the variance defining each of these subfactors consisted of residual variance from particular scales unrelated (orthogonal) to the broad externalizing factor.

Table 1. Loadings of 23 scales of the externalizing inventory on general factor and residual subfactors. (Note. Loading coefficients reflect standardized parameter estimates from a hierarchical model with two subfactors fit using semiparametric maximum-likelihood estimation (for details, see Krueger *et al.* 2007). Loadings above 0.30 are in bold. Loadings listed as zero to two decimal values were fixed at this value and not estimated.)

externalizing scale	general factor	first subfactor	second subfactor
irresponsibility	**0.93**	0.00	−0.01
dependability	**−0.66**	−0.15	0.00
problematic impulsivity	**0.91**	0.00	-0.04
impatient urgency	**0.73**	0.22	0.00
planful control	**−0.66**	−0.07	0.00
theft	**0.87**	0.00	0.13
alienation	**0.49**	0.01	0.00
blame externalisation	**0.51**	0.24	0.00
relational aggression	**0.62**	**0.68**	0.00
destructive aggression	**0.65**	**0.55**	0.00
physical aggression	**0.74**	**0.41**	0.00
empathy	**−0.48**	**−0.55**	0.00
excitement seeking	**0.56**	**0.46**	0.00
rebelliousness	**0.79**	**0.31**	0.00
boredom proneness	**0.59**	0.28	0.00
honesty	**−0.54**	**−0.31**	0.00
fraud	**0.87**	0.26	0.00
marijuana use	**0.73**	0.00	**0.61**
marijuana problems	**0.75**	0.00	**0.48**
other drug use	**0.79**	0.00	**0.49**
other drug problems	**0.87**	0.00	**0.30**
alcohol use	**0.45**	0.00	**0.36**
alcohol problems	**0.69**	0.00	0.24

The findings of this research provide further support for the idea that a broad dispositional factor contributes to the emergence of a wide range of externalizing problems as well as to disinhibitory personality traits that have been linked to such problems. In addition, the findings of this work have important implications for conceptualization and understanding of aggressive behaviour. A sizable portion of the variance in all the three types of aggression represented in the externalizing inventory was accounted for by the general factor on which all scales loaded. In addition, residual variance in each aggression scale went towards defining a separate, statistically independent factor on which low empathy (callousness) and excitement seeking also loaded prominently. Notably, among the three aggression scales, it was the physical aggression scale (which emphasizes reactive–angry aggressive tendencies more so than proactive–instrumental tendencies) that loaded most strongly on the general externalizing factor (i.e. 0.74 versus 0.62/0.65), whereas it was the relational aggression scale (which emphasizes proactive–instrumental aggressive tendencies in particular) that loaded most strongly on the callous-aggression subfactor (i.e. 0.68 versus 0.41/0.55).

The findings of this research are consistent with the idea that differing types of aggressive behaviours are interrelated (e.g. Bushman & Anderson 2001), yet meaningfully distinct from one another (e.g. Dodge 1991). The findings of Krueger *et al.* (2007) suggest that a general propensity towards impulse control problems contributes to aggression in varying forms, in particular, to physically aggressive acts prompted by provocation or stress. From this standpoint, the broad externalizing factor represents an important target of investigation in the study of impulsive–reactive aggression in particular. In addition, a separate propensity involving deficient empathy and stimulation-seeking tendencies appears to contribute independently to aggressive behaviour, particularly aggression that involves instrumental coercion and abuse of others. Other constructs that loaded to some degree on this callous-aggression subfactor included dishonesty,

fraudulence, rebelliousness and a tendency to externalize blame (table 1). These various features are reminiscent of the core affective–interpersonal component of psychopathy in Hare's PCL-R, which tends to be associated more so with proactive–instrumental aggression than with reactive–impulsive aggression. This callous-aggression subfactor may be important for reconciling divergences in findings for aggressive individuals diagnosed as psychopathic compared with those low in core psychopathic features. In sum, the hierarchical model of externalizing traits and behaviours provides a framework for isolating distinctive factors contributing to varying subtypes of aggression that can be targeted separately in neurobiological studies.

(b) *Neurobiological model of impulsive aggression*

The other model I consider is the neurobiological model of aggressive behaviour formulated by Davidson *et al.* (2000). In this model, impulsive aggression is viewed as arising from dysfunction in a set of interrelated brain structures that function to regulate emotional processing and reactivity, including the prefrontal cortex (in particular, its orbitofrontal and ventromedial subdivisions), the anterior cingulate cortex and subcortical-limbic structures (in particular, the amygdala, hippocampus and hypothalamus). The subcortical elements of this circuit play a primary role in activating emotional states, whereas the anterior cingulate and prefrontal cortices operate to detect circumstances under which affective control is needed and to implement control processes, respectively. From the standpoint of this model, repetitive episodes of impulsive aggression (as tend to be characteristic of high-externalizing individuals) reflect a breakdown in the normal capacity to recognize and respond to signals of possible provocation as they arise and/or to modulate defensive reactivity in the face of immediate provocation or threat.

(c) *Interpretation of results from existing psychophysiological studies*

A number of consistent findings have emerged from psychophysiological studies of aggression and aggressive individuals. One of these is the finding of a robust association between low levels of resting HR and antisocial-aggressive behaviour. Research by Raine and colleagues has shown that low resting HR prospectively predicts the emergence of antisocial deviance in at-risk individuals, suggesting that this variable could represent a biological marker (endophenotype) for antisociality. This reduced baseline HR in antisocial-aggressive individuals has been interpreted as reflecting low dispositional arousal, which is assumed to promote stimulation seeking and disinhibited behaviour (Raine 1993, 2002; Ortiz & Raine 2004). However, this interpretation must be regarded as speculative, since no research has yet been conducted to directly assess the functional role of low cardiac arousal in the disinhibited behaviour of antisocial-aggressive individuals.

Two findings of related interest from the electrocortical literature are those of enhanced EEG slow wave activity and reduced P300 brain potential response in antisocial-aggressive individuals. Enhanced EEG slow wave, like low resting HR, has been theorized to reflect low dispositional arousal that motivates stimulation seeking (Eysenck 1967; Zuckerman 1979). Differing interpretations have been attached to the finding of reduced P300 response amplitude in individuals with externalizing problems (e.g. Iacono 1998; Begleiter & Porjesz 1999). One interpretation that fits with the evidence for low resting HR and enhanced EEG slow wave, as well as with evidence for diminished non-specific electrodermal activity (cf. Raine 1993), is that anticipatory and preparatory activities are reduced in such individuals, resulting in a more stimulus-driven processing style (Taylor *et al.* 1999). Relevant to this, Malone *et al.* (2002) reported that high-externalizing individuals showed reduced vigilance, as indexed by alpha band EEG activity, prior to the occurrence of stimuli in a visual oddball task in which reduced P300 was observed.

Although findings pertaining to P300 amplitude reduction in antisocial-aggressive individuals have been emphasized in this review, it should be noted that, historically, reduced P300 has been investigated more extensively as an indicator of risk for alcohol problems (cf. Polich *et al.* 1994). Reports of reduced P300 amplitude in relation to other externalizing syndromes (e.g. conduct disorder, antisocial personality and drug dependence) have appeared more recently. These more recent findings raised the possibility that P300 amplitude reduction might reflect a disposition towards externalizing problems generally rather than to specific problem(s) within this spectrum. Patrick *et al.* (2006) tested this hypothesis in a large sample of adolescent male twins and found that the general factor representing the overlap among various externalizing syndromes (conduct disorder, adult antisocial behaviour, and alcohol, nicotine and other drug dependence) fully accounted for bivariate associations between individual syndromes and reduced P300 amplitude. In a follow-up study that capitalized on the twin composition of this sample, Hicks *et al.* (2007) undertook biometric analyses to examine the aetiologic basis of the relationship between externalizing and P300 amplitude and established that the relationship was mediated primarily by genes.

This work is important because it demonstrates that a known physiological indicator of impulsive aggressive behaviour, reduced P300 response amplitude, is in fact an endophenotype marker of the externalizing vulnerability factor that contributes to various problems of impulse control, including impulsive–reactive aggression. In turn, this raises the question of whether low resting HR and increased EEG slow wave activity are specific to antisocial-aggressive individuals, or if these physiological indicators might be associated with externalizing vulnerability more generally. To address this question, it will be valuable in future research to systematically investigate relations between these physiological indicators and other problems that fall within the externalizing spectrum (e.g. alcohol, drug and nicotine dependence). In addition, the finding that P300 represents a marker of general externalizing vulnerability rather than an indicator of specific problems within this spectrum highlights the importance of the broad externalizing factor as a target for

investigation in the study of aggressive behaviour. This broad factor plays an important role in aggression of various types (physical, relational and destructive; Krueger *et al.* 2007) and the hierarchical model of externalizing provides a strategy for isolating this broad aetiologic factor and investigating its correlates and mechanisms separately from other aetiologic variables. For example, using a groups approach, violent individuals can be subdivided into those who score very high on the general externalizing factor but below the median on the callous-aggression subfactor versus those who score very high on the callous-aggression subfactor but below the median on the general externalizing factor. Using a correlational (e.g. structural modelling or hierarchical regression) strategy, predictive relations with neurobiological criterion measures can be evaluated separately for variance components corresponding to the general externalizing factor, the callous-aggression subfactor and the unique residual variance in each specific subtype of aggression (physical, relational and destructive).

In contrast with findings indicating reduced autonomic and electrocortical arousal at rest and in simple stimulus processing tasks, other research has demonstrated *enhanced* phasic reactivity to stressful or aversive stimuli in hostile, aggressive and abusive individuals—including enhanced cardiac and skin conductance reactivity to stressors, poor regulation of autonomic activity during anticipation of aversive events and reduced cardiac vagal tone. Furthermore, some evidence exists to indicate that this pattern of heightened reactivity to aversive cues or events, like reduced P300 brain response, may be generally characteristic of individuals with impulse control problems, rather than specific to impulsive–aggressive individuals (Taylor *et al.* 1999). Although the finding of enhanced reactivity to phasic stressors might seem inconsistent with data indicating low resting activation levels, the aforementioned hypothesis that externalizing (including proneness to impulsive–aggressive) entails a reactive, stimulus-driven processing style provides a framework for interpreting this overall configuration of results. From this perspective, high-externalizing individuals are more reactive to immediate stressors or challenges because they anticipate and prepare for them less effectively. This configuration of results is also consistent with Davidson *et al.*'s (2000) neurobiological conceptualization of impulsive aggression. Aggression-prone individuals are impaired in conflict detection and/or emotion regulation systems that mediate normal anticipation of events and proactive coping efforts; as a function of this, they exhibit reduced levels of activation until stressors/challenges are actually encountered. Indeed, it can be argued that impairments in the regulatory circuitry identified by Davidson *et al.* underlie the impulsive aggressive tendencies of individuals high on the broad externalizing factor.

A further point is that these findings for impulsive aggression (and externalizing more generally) are clearly at odds with findings for the syndrome of psychopathy. Adult psychopathic offenders do not show reliable differences in resting autonomic activity levels or P300 brain potential response (Raine 1993), whereas they do show consistent reductions in phasic reactivity to aversive cues, including diminished skin conductance response (cf. Hare 1978; Arnett 1997) and startle reflex potentiation (cf. Patrick 1994, 2007). The explanation for this divergence in findings almost certainly lies in the distinction between the affective–interpersonal versus the antisocial deviance features of psychopathy. It is the latter features that reflect heightened externalizing tendencies, including aggression and impulsiveness (Patrick 2007; Patrick *et al.* 2005). However, most EEG/ERP and brain imaging studies have not examined effects for these two components of psychopathy separately (for exceptions, see Laakso *et al.* 2001; De Oliveira-Souza *et al.* 2008). This is a crucial issue that needs to be addressed systematically in future research. Related to this, it will be important in future research to systematically examine alternative forms of aggression associated with differing underlying motives (e.g. proactive–instrumental versus reactive–impulsive) in relation to these two psychopathy factors in order to clarify relations with neurobiological measures. In particular, it is the impulsive–reactive subtype that appears to be most related to externalizing and to impairments in brain systems that govern emotion regulation. It will also be valuable in future studies to include multiple measures of physiological response (peripheral–autonomic along with electrocortical; EEG together with structural or functional neuroimaging) so that findings for different measures can be directly compared within the same task procedures.

With regard to neuroimaging studies, research of this kind—together with research on serotonin system function in aggressive individuals and studies of patients with lesions to distinct regions of the prefrontal cortex—served as the empirical basis for Davidson *et al.*'s (2000) neurobiological model of aggression. As described earlier, these authors postulated that impulsive aggressive behaviour arises from dysfunction in frontocortical and limbic brain regions that mediate affective reactivity and regulation. Specifically, these authors proposed that (i) the orbitomedial prefrontal cortex, which connects directly with limbic structures as well as other regions of frontal cortex, plays a crucial role in regulating (i.e. maintaining, inhibiting or enhancing) emotional states activated by subcortical structures such as the amygdala and (ii) the anterior cingulate cortex functions to signal the need for regulatory control on the part of the prefrontal cortex by detecting conflict among competing goals and response dispositions. Consistent with this formulation, neuroimaging studies have consistently yielded evidence of reduced activity in prefrontal brain regions (including orbitomedial cortex) in aggressive individuals, together with some evidence of reduced activity in anterior cingulate cortex. The finding that high-externalizing individuals show reduced brain error-related negativity (ERN) following incorrect responses in a speeded performance task (Hall *et al.* 2007) lends further support to the idea that impaired anterior cingulate function plays a role in impulsive aggression.

As a final point, it bears mention again that most published psychophysiological studies of aggressive individuals to date (including neuroimaging studies)

qualify as marker studies, in that they have focused on differences either in brain structure or in physiological activity at rest or in simple stimulus tasks. A pressing need exists for process-oriented studies aimed at elucidating differences in online cognitive and affective processing with functional relevance to aggression, including cortical psychophysiology studies that capitalize on the fine-grained temporal and frequency information afforded by EEG/ERP, and functional neuroimaging studies that capitalize on the fine-grained spatial information provided by MRI. In this regard, key questions for future research include the following: (i) What are the distinctive functional roles of brain regions that have been implicated in electrocortical and neuroimaging studies of aggression and how do these regions interact to achieve regulatory control over emotional states? Basic cognitive and affective neuroscience research is needed to elucidate this issue. (ii) What specific impairments in the functioning of these brain systems predispose individuals towards aggressive behaviour? To address this question, it will be necessary to conduct EEG/ERP and functional neuroimaging studies that examine online processing and brain reactivity within aggression-relevant task procedures, such as interpersonal provocation paradigms. (iii) Do different types of brain dysfunction underlie impulsive–reactive and callous–proactive manifestations of aggressive behaviour? The work of Krueger *et al.* (2007) and others suggests that these manifestations of aggression may have distinctive substrates. Thus, an important challenge for future research will be to delineate the nature of processing impairments or deviations that underlie impulsive aggression associated with externalizing compared with more callous–instrumental forms of aggression associated with psychopathic personality.

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ENDNOTE

[1]Relations with psychopathy have been reported for other components of the ERP besides P300. For example, studies by Kiehl and colleagues have yielded evidence of an abnormal late negativity, maximal over frontocentral scalp regions, among high PCL-R scoring offenders within a variety of stimulus-processing and decision-making tasks (for an overview of this work, see Kiehl *et al.* 2006).

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