Error-monitoring in antisocial youth with and without callous unemotional traits: an event–related potential study

Michael Eisen

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has be
derived from other sources, I confirm that this has been indicated in the thesis.

Signature: 

Name: Michael Eisen

Date: 15/06/2015

Overview

Antisocial behaviour is a subject of considerable clinical and research interest, especially when it is associated with psychopathy. This thesis aimed to explore the role of outcome monitoring and processing in such behaviour, by examining it in individuals with and without psychopathy.

The literature review examined the evidence on psychopathic abnormalities in the processing of reward and punishment. The evidence was reviewed for: an emotional account of such processing; a cognitive/attentional account; and two neural accounts. A review of seventeen studies found evidence for both the emotional processing and cognitive/attentional accounts, and for one of the neural accounts. These three accounts are all compatible with one another, and so the evidence may indicate more than one abnormality in psychopathic reward and punishment processing.

The empirical study tested the relations in young people between externalising/antisocial behaviour, psychopathic traits, and the error-related negativity – an event-related potential component related to error-monitoring. 34 antisocial/externalising and 39 control adolescents were tested using a combined flanker/Go/No-Go task. As predicted, the externalising group showed a reduced negativity after errors and, within the externalising group, psychopathic traits were associated with reduced negativity after errors.

The critical appraisal noted the challenges of the research process, and considered the academic and clinical implications of the findings.

This study was conducted as a joint project.

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Part one: Literature review

Reward and punishment processing in psychopathy: psychophysiological data

A theoretical and empirical review

Abstract

Aims

Reward and punishment processing in psychopathy is an area of research interest due to its probable role in psychopathic criminality and recidivism. Research in this area has frequently made use of psychophysiological data to develop and support relevant models. This data was reviewed, together with its implications for these models.

Methods

A systematic review was conducted of online databases to identify relevant studies published in peer-reviewed journals.

Results

Seventeen published papers were reviewed in detail. They provided a mixture of support and contrary evidence for both the "fear deficit" and "response modulation" models of reward and punishment processing in psychopathy, and support for the "paralimbic hypothesis" regarding the neural substrates underlying such processing.

Conclusions

It is unclear whether either or both of the fear deficit and response modulation models are correct, and further research is needed to clarify this. The paralimbic hypothesis of neural dysfunction in psychopathy is better supported by this review than a more parsimonious alternative.

Introduction

The construct of psychopathy has proved to be of enduring interest, in part because psychopaths are disproportionately likely to commit crime (Blair, Mitchell & Blair, 2005). Much of this criminality consists of repeat offending - psychopaths appear particularly unresponsive to legal sanctions and other adverse consequences of their actions (Blair, 2013), and so their responses to reward and punishment have been the subject of considerable research attention. Whilst much of this research has made use of behavioural measures, psychophysiological data offers the prospect of elucidating the mechanisms that lie behind psychopathic behaviour, and identifying its neural substrates. It will be the aim of this review, therefore, to evaluate the psychophysiological findings of this research, and assess their implications for current models of reward and punishment processing in psychopathy.

Psychopathy

The modern construct of psychopathy was first delineated by Cleckley (1941), who described a category of individuals with superficial charm and a lack of anxiety but also a lack of guilt and empathy; who were dishonest, egocentric, promiscuous, and unable to plan ahead, appreciate the impact of their behaviour on others, and learn from punishment. More recent descriptions have built upon this description, continuing to emphasise emotional deficits (in guilt and empathy) and a lack of planning and consistency, resulting in a tendency towards antisocial behaviour (Blair, Mitchell & Blair, 2005). The disorder is now thought to originate early in life, and to be neurodevelopmental (Blair, Mitchell & Blair, 2005).

Psychopathy is currently best described by the Psychopathy Checklist – Revised (Hare, 1991), which is widely used to diagnose the disorder. The PCL-R consists of 20 items that may be grouped into two factors: Factor 1, capturing deficits

in emotions such as empathy and guilt, and the callous interpersonal style that results; and Factor 2, capturing an antisocial and impulsive lifestyle (Hare, 1991).

Reward and punishment processing in psychopathy

Reward and punishment processing is of interest in part because of its relation to socialisation and the regulation of behaviour: individuals learn to suppress antisocial behaviour through coming to associate it with negative contingencies such as punishment (Trasler, 1978). In psychopaths, it has been suggested, disruption of these processes produces a tendency towards antisocial behaviour that persists even in the face of adverse consequences (Newman, Patterson, Howland, & Nichols, 1993). Psychophysiological measures have frequently been used to investigate this.

One broad framework within which to understand reward and punishment processing is Gray's two-process model, which proposes a "behavioural activation system" (BAS) that processes information indicating the availability of reward and initiates appropriate goal-directed behavior, and a behavioural inhibition system (BIS), which is concerned with processing threat-related information, suppressing goal-directed action, and initiating action to avoid punishment (Gray, 1987). This model has been applied directly in considering reward and punishment processing in psychopathy (Newman, MacCoon, Vaughn, & Sadeh, 2005), but generally has been used to inform the development of disorder-specific theories. These may be divided into those that focus on: emotional processing; attention and cognition; and neural structures and processes.

Emotional processing deficits

These accounts suggest that whilst psychopaths may be fully able to anticipate impending punishment, their emotional response to it is deficient (Fowles, 1988; Patrick, 1994). Such "fear deficit" models have drawn upon findings of other

emotional deficits in psychopathy: failure to recognize the emotional expressions of others (Dawel, O'Kearney, McKone, & Palermo, 2012), or to show normal autonomic responses to others' expressions of pain and distress (Blair, Jones, Clark, & Smith, 1997).

The possibility of deficits in punishment processing was first raised by studies showing that psychopaths anticipating an electric shock show lower levels of electrodermal response than non-psychopaths (Hare, 1965; Hare & Quinn, 1971; Hare, 1978). Explanations that have been advanced for this lack of fear when anticipating punishment include: insensitivity to punishment when it actually takes place (Hare, 1965); an inability to develop an emotional response to the cognitive awareness of impending punishment (Sommer et al., 2006); or a successful "coping" response that enables the aversive response to impending punishment to be managed or suppressed (Hare, Frazelle, & Cox, 1978). The second of these accounts is currently most favoured: that whilst psychopaths may be fully able to cognitively anticipate punishment, they are unable to represent the emotional significance of it. This account predicts that psychopaths should show normal cognitive processing but reduced fear in response to cues to impending punishment, regardless of whether task demands focus participants' attention on those cues or elsewhere.

Cognitive/attentional abnormalities

Newman and colleagues have developed the Response Modulation Hypothesis (RMH), an account in which psychopathic deficits in responding to punishment cues are due to a failure to attend fully to these cues. (Patterson & Newman, 1993).

The RMH arose from observations that psychopaths' deficits in reward and punishment processing emerge only under certain conditions: punishment learning

and avoidance amongst psychopaths was found to be normal when subject only to punishment with no competing reward contingency (Newman & Kosson, 1986), and when forced to pause and (presumably) reflect between punishment and their next action (Newman, Kosson & Patterson, 1987). To account for this, the RMH proposes that psychopaths are relatively unable to interrupt a dominant goal-seeking response set in order to process the outcomes of their actions and adjust their behaviour accordingly, and instead respond to punishment with increased arousal and a more rapid reward-seeking response to the next stimulus presented (Patterson & Newman, 1993). This is thought to entail both excessive focus upon the pursuit of reward, such that environmental stimuli signaling negative outcomes are "screened out", and a failure, due to increased arousal, to pause and reflect when negative outcomes take place (Patterson & Newman, 1993). The RMH predicts that deficits in punishment processing should be seen when psychopaths are engaged in goal-directed behaviour that focusses their attention on stimuli other than cues to punishment (Patterson & Newman, 1993).

Neural models

Finally, neural imaging work has resulted in competing accounts of the neural structures and processes associated with abnormal reward and punishment processing in psychopathy (Blair, 2013). These may be consistent to varying degrees with emotional and cognitive accounts.

Kiehl's paralimbic hypothesis

In a review drawing on comparisons with brain damage patients, EEG data, and studies of language, attention, orienting, and affective processing, Kiehl (2006) implicates a number of different brain regions in the deficits observed in psychopathy. These regions are spatially dispersed within the brain but may, on the

basis of a cytoarchitectonic approach, be thought of together as the paralimbic system: the orbitofrontal cortex; insula; posterior and anterior cingulate cortex (ACC); amygdala; parahippocampal gyrus; and anterior superior temporal gyrus (Kiehl, 2006). It is dysfunction in this system, Kiehl's model suggests, that produces deficits in reward and punishment processing in psychopathy (Kiehl, 2006).

The model has been criticized by Blair, who points to sMRI findings that reductions in gray matter volume in psychopathy are confined to the posterior cingulate cortex, rather than the whole of the cingulate cortex as the paralimbic hypothesis would predict, and to neuropsychological evidence that psychopaths do not in fact show the impairments that would be predicted by the paralimbic hypothesis (e.g. episodic memory impairments due to parahippocampal dysfunction, conflict monitoring deficits due to ACC dysfunction, and Theory of Mind deficits due to superior temporal cortex and temporal pole dysfunction) (Blair, 2013). Blair has proposed, as an alternative, the integrated emotion systems model (Blair, 2013).

Integrated emotion systems

Blair's model takes a more conservative approach than the paralimbic hypothesis, specifying as dysfunctional only those brain regions whose activity has been shown to be aberrant *and* whose functions have been found to be disrupted (Blair, 2013). The model posits dysfunction only of the amygdala and its communication with the ventromedial prefrontal cortex, resulting in impaired stimulus-reinforcement learning and use of reinforcement expectancy information (Blair, 2013).

It is of note that both leading neural models of psychopathic deficits in reward and punishment processing posit dysfunction in the amygdala, a structure that is thought to play a key role in the processing of negative emotion (LeDoux, 2003).

Amygdala dysfunction might, therefore, offer a neural substrate for fear deficits in psychopathy (Blair, 2008). Dysfunction in the ACC, meanwhile, is posited only by the paralimbic hypothesis but would be consistent with difficulties in cognitive/attentional processing of punishment cues, as the ACC is thought to be involved in the cognitive processing of feedback during tasks (Holroyd & Coles, 2002).

Psychophysiological measurement

Psychological states frequently have physiological correlates. Measurement of physiological states, therefore, can provide information about psychological states, often finer-grained information than could be obtained with behavioural measures, self-report, or observation (Cacioppo, Tassinary, & Berntson, 2007). In the studies reviewed here, psychophysiological measures have been used to investigate: emotional processing by psychopaths of reward and punishment; the cognitive processes that lie behind poor performance by psychopaths on some tasks involving reward and punishment; and the neural substrates that may lie behind both of these. The measures used will be discussed below.

Summary

In seeking to account for the observed poor performance of psychopaths on some tasks involving reward and punishment, and perhaps, by extension, psychopaths' real world recidivism, three kinds of explanation have been proposed: emotion-processing/fear deficit accounts, in which psychopaths fail to produce a normal emotional response to cues to impending pain; cognitive/attentional accounts, in which psychopaths fail to attend fully to cues to impending punishment; and neural accounts, which seek to identify the features of brain function that underlie psychopathic deficits in reward and punishment processing. These three kinds of

explanation are not necessarily incompatible with each other, but have been supported by different studies, often conducted using different measures and experimental paradigms. This research has made frequent use of psychophysiological measures to specify in detail the relevant psychological processes or states, and relevant structures and processes within the brain.

The present review

Research into reward and punishment processing has important implications for reducing antisocial behaviour and rehabilitating those who engage in it (Trasler, 1978). Behavioural data may not always be suitable for illuminating the fine details of cognitive and emotional processing that underlie behaviour, whilst self-report data may not capture distinctions between emotional and cognitive aspects of participants' experience, and is vulnerable to respondent bias, which may be particularly problematic in those with psychopathic tendencies (Cacioppo, Tassinary, & Berntson, 2007). A review of relevant psychophysiological findings, therefore, seems appropriate.

The review will aim to answer the following questions:

What does psychophysiological research indicate regarding:

(i) Emotional deficits in the processing of reward and punishment by psychopaths?

(ii) Cognitive/attentional abnormalities in the processing of reward and punishment by psychopaths?

(iii) Neural abnormalities affecting the processing of reward and punishment by psychopaths?

Method

Inclusion/exclusion criteria

Studies were selected for the review according to the following criteria:

- *Published in a peer-reviewed journal*: this criterion was adopted as a guarantee of the general quality of studies.
- *Participants aged over 18*: the diagnosis of psychopathy should not be applied below the age of 18.
- *Participants not known to be substance-dependent*: studies have suggested that substance dependence is associated with abnormalities in reward and punishment processing. These could confound findings in respect of reward and punishment processing in psychopathy.
- *Study includes a group of participants diagnosed as psychopathic using the PCL-R*: restricting the review to studies making use of a single diagnostic measure will limit the variance between samples and maximize the generalizability of findings across studies.
- *Study made use of unambiguous punishment (e.g. electric shock or monetary loss)*: some studies investigating reward and punishment processing have made use of stimuli that may not in fact be aversive for some individuals, e.g. criticism, or affective images. Monetary loss and physically uncomfortable stimuli are assumed to be aversive, to at least some degree, to the vast majority of individuals, whether psychopathic or not.
- *Participants' processing of reward and/or punishment examined using a psychophysiological measure*.

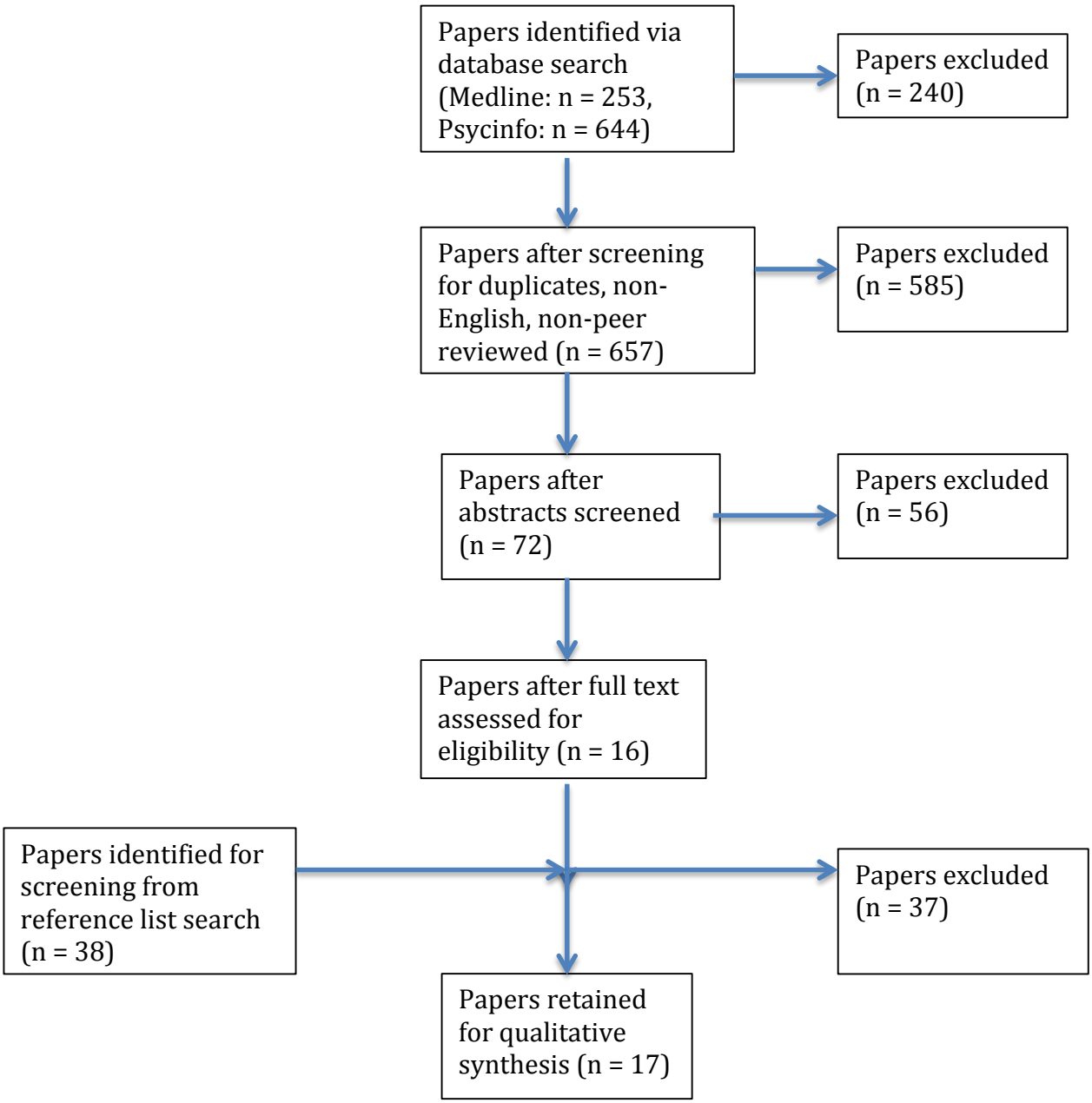
Search strategy

Keyword searches were conducted on Medline and Psycinfo, using search terms adapted from those used in a published review of the literature on reward and punishment processing in youth exhibiting psychopathic traits and/or antisocial behaviour (Byrd, Loeber & Pardini, 2013). The search terms were as follows, combined into searches as follows:

1. learning or conditioning
2. reward or punishment
3. learning or conditioning OR reward or punishment
4. psychopath* or "CU traits" or "callous unemotional" not psychopatholog*
5. learning or conditioning OR reward or punishment
   AND
   psychopath* or "CU traits" or "callous unemotional" not psychopatholog*

17 papers were identified as meeting as criteria for inclusion in this review (see Figure 1).

Figure 1. Paper selection and screening process



Results

The methodologies and measures used by the studies in the review will be presented, followed by the findings of the studies as they relate to the three questions that the review seeks to answer. All results are summarised in Table 1.

Paradigms/tasks

Paradigms used in the studies were: Pavlovian fear conditioning (five studies); instructed fear conditioning (five studies); active/passive avoidance (two studies); risk-taking (one study); a "countdown" procedure (two studies); Taylor Aggression Paradigm (TAP) (one study); and a task in which participants passively won or lost money, while being asked to predict their wins and losses (one study).

Pavlovian fear conditioning

In Pavlovian fear conditioning, a neutral stimulus (conditioned stimulus, CS+/-) comes through repeated pairings to be associated with an aversive stimulus (unconditioned stimulus, US) and thus comes to elicit fear (Pavlov & Anrep, 2003). This paradigm seems well-suited to investigating fear deficit theories of psychopathy, as it examines the process of association formation, which in turn forms the basis of a fear response: an association must be formed between a stimulus and punishment for that stimulus to elicit fear (Pavlov & Anrep, 2003). All five studies that made use of this methodology did so in order to test fear deficit theories (Birbaumer et al., 2005; Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Rothemund et al., 2012; Veit et al., 2002; Veit et al., 2013)

Instructed fear conditioning

In instructed fear conditioning, participants are told that the CS+ will precede the US, so that fear conditioning is achieved immediately via conscious knowledge, rather than through repeated exposure (Olsson & Phelps, 2004). This paradigm is

well-suited to investigating cognitive/attentional accounts of deficits in punishment processing, as these do not imply any difficulty in forming associations between stimuli and punishment, but rather a difficulty, under some circumstances, in attending to stimuli that are already associated with punishment.

All five studies in the present review that made use of instructed fear conditioning tasks did so in order to test Patterson and Newman's (1993) RMH (Anton, Baskin-Sommers, Vitale, Curtin, & Newman, 2012; Baskin-Sommers, Curtin, & Newman, 2011; Baskin-Sommers, Curtin, Wen-Li, & Newman, 2012; Baskin-Sommers, Newman, Sathasivam, & Curtin, 2011; Newman, Curtin, Bertsch, & Baskin-Sommers, 2010).

Passive avoidance

In passive avoidance tasks, participants are presented with stimuli that, if responded to, might result in either reward or punishment, and must learn which are associated with reward and which with punishment, in order to withhold responses to those that are associated with punishment (Newman, Widom, & Nathan, 1985). It was from such studies that the RMH arose (Newman & Kosson, 1986; Newman et al., 1987) but because associations are both learnt and responded to in these tasks, the two processes, and deficits in them, cannot easily be dissociated. These tasks were used in three studies (published as two papers) included in this review (Arnett, Howland, Smith, & Newman, 1993; Arnett, Smith, & Newman, 1997).

Countdown

Two studies made use of "countdown" procedures, in which psychophysiological measures are taken whilst participants are awaiting an impending event. In a study by Ogloff and Wong (1990), the impending event was punishment. In another by Forth and Hare (1989), the impending event was a signal

to which participants had to react as quickly as possible in order to win money or avoid losing it. These studies form part of the line of research, mainly predating the studies included in this review, that demonstrated psychopathic insensitivity to punishment cues and so gave rise to the fear deficit model (Hare, 1965; Hare & Quinn, 1971; Hare, 1978).

Risk-taking

Prehn et al. (2014) used the Behavioural Investment Allocation Strategy task, in which participants are required to make repeated choices between a "risky" option offering a lower probability of a large reward and a "safe" option offering a higher probability of a small reward (Kuhnen & Knutson, 2005). This task allows examination of reward and punishment processing under conditions of uncertainty. A reward-only version of the task was used here, meaning that the study could not offer any data relevant to fear deficit theories of reward and punishment processing.

TAP

In the TAP (Taylor, 1967), two participants compete against each other in a reaction time task, with the winner of each round being able to impose punishment upon the loser. Veit et al. (2010) used this paradigm to investigate participants' responses to anticipated and actual punishment.

Passive gain/loss of money

In one study (Pujara, Motzkin, Newman, Kiehl, & Koenigs, 2014), participants were presented with stimuli associated with differing probabilities of reward/punishment and asked to predict after each stimulus which stimulus would come next, while measures were taken of participants' neural responses to reward and punishment.

Psychophysiological measures

Studies made use of: measures of autonomic responding; measures of facial reaction to emotion; neural imaging; and event-related potentials (ERP). Most studies made use of more than one psychophysiological measure.

Autonomic

Five studies used measures of both heart rate and skin conductance response (SCR) (Arnett et al., 1993; Arnett et al., 1997; Flor et al., 2002; Ogloff & Wong, 1990; Rothemund et al., 2012), whereas a further three used SCR together with another psychophysiological measure (Birbaumer et al., 2005; Veit et al., 2010; Veit at al., 2013).

Heart rate is in general taken to reflect non-specific arousal, such that it has various possible interpretations in the context of reward and punishment processing: when accelerated heart rate precedes anticipated punishment, it has been interpreted as reflecting the mobilisation of a "coping" response (Lykken, 1967); when it occurs in the context of availability/anticipation of reward, it may reflect activity of the BAS (Fowles, 1980). Skin conductance response, meanwhile, has been theorised to index anxiety and/or the activity of the BIS (Siddle & Trasler, 1981), so that attenuated SCRs before anticipated punishment would indicate a lack of fear and/or a weak BIS. Just such a pattern of responding was found amongst psychopaths in early work on punishment processing, which gave rise, in part, to the fear deficit model (Hare, 1978).

Facial reactions

Six studies measured fear-potentiated startle (FPS) (Anton et al., 2012; Baskin-Sommers et al., 2011a; Baskin-Sommers et al., 2011b; Flor et al., 2002; Newman et al., 2010; Rothemund et al., 2012), a reflexive eyeblink reaction that is

taken to index fear, and is a standard measure in fear conditioning paradigms (Davis, Falls, Campeau, & Kim, 1993). All six studies made use of Pavlovian or instructed fear conditioning paradigms.

Electromyography of the corrugator supercilli muscles of the face was used in two studies (Flor et al., 2002; Rothemund et al., 2012). This technique measures the activity of muscles involved in frowning and so, like FPS, is taken to index emotional reactivity to stimuli, with muscle activity increasing with negative emotional response (Dimberg, 1990). Both studies that used this technique did so in the context of Pavlovian conditioning.

Neural imaging

Five studies made use of functional magnetic resonance imaging (fMRI), using it across a range of experimental paradigms to investigate the neural correlates of a range of hypothesized psychological processes (Birbaumer et al., 2005; Prehn et al., 2014; Pujara et al., 2014; Veit et al., 2002; Veit et al., 2010). fMRI measures activity in different brain regions by using the contrast seen between oxygen-rich and oxygen-poor blood (the blood-oxygen-level dependent contrast, or BOLD) to show changes in blood flow related to energy use in brain cells (Huettel, Song, & McCarthy, 2004). In indicating the activity of particular brain regions at particular stages in tasks, fMRI may indicate the operation of the processes thought to be associated with those regions, and so may produce evidence relevant to any of the models of reward and punishment processing set out above.

One study, by Pujara et al. (2014), made use of volumetric analysis, a technique used to measure the volume of brain regions and thus illuminate differences in brain structure between individuals and groups, which may in turn suggest differences in brain function and psychological processes (Raz, Gunning-

Dixon, Head, Dupuis, & Acker, 1998). Again, data of this sort may be relevant to any model of reward and punishment processing.

ERP

ERPs are changes in the voltage measurable at the scalp (by means of electroencephalography), that take place as a result of a particular sensory, motor, or cognitive event (Luck, 2014). ERP data is extremely fine-grained in respect of the timing of events, and so offers the possibility of exploring the fine detail of cognitive processes (Luck, 2014). ERPs are, therefore, particularly suited to testing cognitive/attentional theories of reward and punishment processing deficits in psychopathy. Six studies made use of ERP data, three of them in Pavlovian conditioning studies (Flor et al., 2002; Rothemund et al., 2012; Veit et al., 2013), two in instructed fear conditioning studies (Anton et al., 2012; Baskin-Sommers et al., 2012), and one in a countdown task (Forth & Hare, 1989). ERP components investigated were:

N100: Indexes early attention (Woldorff et al., 1993), with larger amplitudes indicating selective attention (Luck, 2000).

P200: Indexes higher-order perceptual processing, modulated by attention (Siegel, 1997).

P300: Associated with stimulus evaluation and categorisation, the P300 is sensitive to changes in the salience of information (Sutton, Braren, Zubin, & John, 1965) and to late attentional processes (Schupp, Junghofer, Weike, & Hamm, 2004).

Contingent negative variation (CNV): correlates with selective attention and arousal, but is also sensitive to expectancy and motivational aspects of stimuli (Tecce, 1972). The CNV may be decomposed into two subcomponents – the initial and terminal CNV (iCNV and tCNV). The iCNV seems to reflect an orienting

response associated with stimulus evaluation (Rockstroh, 1989), whereas the tCNV may reflect motor preparation and the emotional salience of stimuli, and is particularly pronounced in anticipation of intense aversive stimuli (Birbaumer, Elbert, Canavan, & Rockstroh, 1990).

P140: A very early ERP component, peaking at around 140ms post-stimulus, that indicates selective attention (Hillyard, Simpson, Woods, Van Voorhis & Munte, 1984).

Late positive complex (LPC): The LPC has been found to differentiate reactions of psychopaths from those of non-psychopaths in respect of affective stimuli vs neutral (Williamson, Harpur, & Hare, 1991).

Table 1. Studies included in the review: characteristics and results

Study	Participants	Groups	Task	Measures	Outcome
Forth & Hare, 1989	29 white male inmates aged 18-45	Psychopaths (Ps) and controls (Cs)	Countdown to reaction time task, for money	ERP	Early CNV of Ps was significantly larger than that of Cs
Ogloff & Wong, 1990	32 male inmates aged 18-42	Ps and Cs	Countdown to shock	Heart rate, SCR	Cs had higher SCR when could not avert punishment; Ps did not, and had lower SCR in general. Ps had higher heart rate when could not avert punishment; Cs did not
Arnett et al., 1993	63 white male inmates aged 18-40	Ps and Cs, subdivided into low-anxious and high-anxious groups	Passive avoidance: Go/No-Go	Heart rate, SCR	Ps had lower heart rate and fewer (but not smaller) SCRs after punishment than controls
Arnett et al., 1997	63/71 (study 1/study 2) white male inmates aged 18-40	Ps and Cs, subdivided into low-anxious and high-anxious groups	Passive/active avoidance	Heart rate, SCR	Ps had smaller SCRs to punishment cues than Cs, but no differences in heart rate
Flor et al., 2002	9 non-criminal male Ps; 12 male community Cs	9 non-criminal male Ps; 12 male community Cs	Pavlovian conditioning: foul odor	ERP, heart rate, SCR, corrugator EMG, FPS	Cs showed CS+/CS- differentiation; Ps didn't. ERPs showed Ps not deficient in information processing and have better anticipatory responding
Veit et al., 2002	4 criminal Ps, 4 social phobics, 7 community Cs, all male	4 criminal Ps, 4 social phobics, 7 community Cs	Pavlovian conditioning: painful pressure	fMRI	Ps only showed brief amygdala activation to anticipated pain; Cs showed activation in whole limbic pre-frontal circuit

Birbaumer et al., 2005	10 emotionally detached offender Ps; 10 community controls. All male	10 emotionally detached offender Ps; 10 community controls	Pavlovian conditioning: painful pressure	fMRI, SCR	Ps showed no activity in limbic-prefrontal circuit, and no conditioned SCR
Veit et al., 2010	10 male Ps from forensic psychiatric institutions	No groups	TAP	fMRI, SCR	Ps lacked amygdala activation when anticipating pain
Newman et al., 2010	125 white male inmates	No groups	Instructed fear conditioning: electric shock. Manipulated attentional focus	FPS	Ps have reduced FPS but only under alternative-focus conditions. This is driven by PCL-R Factor 1
Baskin-Sommers et al., 2011a	92 African-American male inmates	No groups	Instructed fear conditioning	FPS	FPS indicated no fear deficit in psychopathy
Baskin-Sommers et al., 2011b	87 white male inmates	No groups	Instructed fear conditioning: shock. Manipulated attentional focus	FPS	FPS deficit in early-alternative-focus condition. Ps with high working memory capacity had reduced FPS in late-alternative-focus condition.
Anton et al., 2012	84 white female offenders	Ps; ASPD sufferers	Instructed fear conditioning. Manipulated attentional focus and cognitive load	FPS, ERP	Psychopathy and ASPD associated with distinct cognitive and affective patterns
Rothemund et al., 2012	11 offender Ps; 11 community controls. All male	11 offender Ps; 11 community controls	Pavlovian conditioning	ERP, FPS, SCR, heart rate, corrugator EMG	Ps didn't condition, as indexed by FPS and SCR

Baskin-Sommers et al., 2012	101 white male inmates	No groups	Instructed fear conditioning. Manipulated attentional focus	ERP	Ps showed larger P140 under alternative-focussed vs threat-focussed conditions
Veit at al., 2013	14 offender Ps in forensic psychiatric institutions	No groups	Pavlovian conditioning	ERP, SCR	High Factor 1 = less conditioned fear and increased information processing; whereas Factor 2 = decreased attention and interest to CS+
Prehn et al., 2014	23 male offenders	11 emotionally hyporeactive (high Factor 1 PCL-R); 12 emotionally hyperreactive	Risk-taking	fMRI	Hyporeactive showed diminished activation in rACC in response to uncertainty, and diminished activation in prefrontal cortex when choosing safe options in response
Pujara et al., 2014	41 inmates	Ps and Cs	Passive gain/loss of money	fMRI	Psychopathy severity correlated with ventral striatum activation and volume amongst Ps but not Cs

Findings

Study findings, and issues bearing on the weight to be given to them, will be discussed in relation to each of the review questions.

(i) What does psychophysiological research indicate regarding emotional deficits in the processing of reward and punishment by psychopaths?

Pavlovian fear conditioning studies

The majority of findings supporting the fear deficit model were generated by the five studies that used this paradigm, conducted by what appears to be a single research team (Birbaumer et al., 2005; Flor et al., 2002; Rothemund et al., 2012; Veit et al., 2002; Veit et al., 2013). These studies made use of small samples of between four (Veit et al., 2002) and 14 (Veit et al., 2013) psychopathic offenders, apart from the study by Flor et al. (2002), which used a sample of non-criminal psychopaths. Psychopaths were compared to healthy community control groups or, in the case of the study by Veit et al. (2013), no control group.

Community control groups may not be ideal for comparison to psychopathic comparison groups because they may differ systematically on variables such as intelligence (although these studies did match psychopathic and control participants for education level and/or employment status) and prior experience of physical pain. The latter variable has obvious relevance for studies investigating physiological responses to the expectation of pain, whereas intelligence has been identified as a major potential confound in neural imaging work on psychopathic reward and punishment processing (Blair, 2013). The results of the study by Flor et al. (2002) must also be treated with some caution in evaluating a fear deficit account of reward

and punishment because it made use of a foul odor, rather than pain, as the aversive stimulus, which may evoke disgust in participants rather than fear.

Pavlovian fear conditioning studies used a range of measures to produce data relevant to fear deficit theories.

Autonomic and facial responding

Consistent with fear deficit theories, psychopaths failed to show conditioning in respect of SCRs (Birbaumer et al., 2005; Flor et al., 2002; Rothemund et al., 2010; Veit et al., 2010; Veit et al., 2013); heart rate (Flor et al., 2002; Rothemund et al., 2012); and FPS and corrugator EMG (Flor et al., 2002; Rothemund et al., 2012). Further, psychopaths showed no deficit in responsiveness to unconditioned stimuli, whether measured using SCRs (Flor et al., 2002), FPS (Flor et al., 2002), or corrugator EMG (Rothemund et al. 2012), suggesting that failure to condition was due to reduced responsiveness to impending rather than actual punishment, as predicted by current fear deficit theories.

Birbaumer et al. (2005) and Veit et al. (2013) investigated the relationship between the factor structure of the PCL-R and failure to condition, and showed, respectively, that the emotionally detached dimension of psychopathy is associated with conditioning deficits and that it is the affective facet of Factor 1 that drives this relationship.

ERP data

ERP data was used in these studies mainly to test whether cognitive/attentional deficits could account for observed deficits in conditioning. Results suggested that it could not: good attention and processing during conditioning was found using the N100 (Flor et al., 2002), P200 (Flor et al., 2002; Rothemund et al., 2012), CNV (Flor et al., 2002; Rothemund et al., 2012) and LPC

(Flor et al., 2002), indicating that any deficit in conditioning was not due to a cognitive deficit. Rothemund et al. (2012), meanwhile, interpreted a finding of a reduced tCNV at frontal sites in psychopaths compared to controls as indicating reduced activity in the limbic-prefrontal circuit, consistent with fear deficit theories.

Veit et al. (2013) explored the relationship between the factor/facet structure of psychopathy (as captured by the PCL-R) and cognitive/attentional processes during conditioning, as indexed by the N100 and P300 ERP components, concluding that the interpersonal facet of psychopathy is associated with superior information processing, whereas the antisocial facet is associated with reduced attention to the CS+.

While ERP data from Pavlovian fear conditioning studies may provide support for fear deficit theories, it cannot undermine the RMH, which predicts that cognitive/attentional abnormalities will affect punishment processing only where attention is directed away from punishment cues, which was not the case in these studies.

Neural imaging

Both Veit et al. (2002) and Birbaumer et al. (2005) found that controls but not psychopaths showed differential activation in the limbic prefrontal circuit during Pavlovian conditioning, in line perhaps with ERP findings by Rothemund et al. (2012), whereas psychopaths showed only activation in the amygdala. This activation was brief in the Veit et al. (2002) study, and was of only the right amygdala in the Birbaumer et al. (2005) study. Veit et al. (2002) suggest that psychopaths show impairments in anterior cingulate-orbitofrontal connectivity that may be crucial for emotional responding. These results seem consistent with fear deficit accounts of psychopathy.

Instructed fear conditioning studies

These studies have been mainly concerned with developing and testing the RMH, and have generally found data contrary to fear deficit theories: FPS to threat-related stimuli was found to be normal when the psychopathic participant's attention was focussed on those stimuli (Anton et al., 2012; Baskin-Sommers et al., 2012; Baskin-Sommers et al., 2011a; Newman et al., 2010), and these results were found to be the same for either factor of the PCL-R, as well as for the total score (Baskin-Sommers et al., 2011b; Newman et al., 2010), whereas a fear deficit account would predict reduced FPS to punishment cues regardless of where attention was focussed.

It is not clear, however, how differences between instructed fear and Pavlovian conditioning methods might contribute to the conflicting results produced by studies using them: it is possible that emotional processing deficits in psychopathy might affect only conditioned associations formed experientially via Pavlovian conditioning, and not associations formed verbally via instructed fear conditioning (Newman et al., 2010 – see below for further details). This raises the possibility that deficits in reward and punishment processing in psychopathy might be due to either or both of emotional deficits or cognitive/attentional abnormalities, depending upon the learning processes involved in a given situation.

Passive avoidance studies

One of the two passive avoidance studies included here found (limited) differences in psychopathic SCR and heart rate responses to actual punishment (Arnett et al., 1993), offering some support to older fear deficit theories that suggest psychopaths' reduced responsiveness to cues to punishment is due to reduced responsiveness to actual (rather than anticipated) punishment (Hare, 1965), and

conflicting with current fear deficit theories and their supporting evidence (Flor et al., 2002; Rothemund et al., 2012).

The weight to be given to data from this study may be questioned, however, on the grounds that: it failed to find differences in performance between psychopaths and non-psychopaths (in contrast to other passive avoidance studies: Lykken, 1957; Newman et al., 1990; Schmauk, 1970); the differences in SCRs related to their number, rather than amplitude; the correct interpretation of heart rate data is not obvious, as heart rate seems to index non-specific arousal; and the other passive avoidance study included in this review found no differences in either SCRs or heart rate between psychopaths and controls following punishment (Arnett et al., 1993).

Other studies

A "countdown" study by Ogloff and Wong (1990) found an absence of increased SCR when anticipating electric shock, which could indicate a fear deficit, but also an increase in heart rate, which is more ambiguous, given that increases in heart rate indicate non-specific arousal.

Veit et al. (2010) used the TAP paradigm to investigate the fear deficit model and, consistent with that model, found that psychopaths' SCRs did not correlate with the degree of punishment anticipated. fMRI data from the study provided further support for a fear deficit by showing that anticipation of punishment was associated amongst psychopaths with activation in a range of brain areas, but not with any activation in the amygdala, ACC, or prefrontal areas: Blair (2013) has suggested that a fear deficit in psychopaths could derive from amygdala dysfunction.

(ii) *What does psychophysiological research indicate regarding cognitive/attentional abnormalities in the processing of reward and punishment by psychopaths?*

Instructed fear conditioning studies

The majority of relevant findings were generated by five studies that used this methodology to test Patterson and Newman's (1993) RMH by requiring participants to make responses to the conditioned stimulus, under conditions that varied participants' attentional focus and the cognitive load upon them (Anton et al., 2012; Baskin-Sommers et al., 2011a; Baskin-Sommers et al., 2011b; Baskin-Sommers et al., 2012; Newman et al., 2010).

Three of these studies, in particular, build up a detailed picture in which psychopaths show deficits in responding to punishment cues only where attention is already focussed elsewhere (Newman et al., 2010), as under those circumstances they screen out the punishment cues at an early stage of attentional processing (Baskin-Sommers et al., 2011a), and this is indexed by the P140 ERP component (Baskin-Sommers et al., 2012).

These studies made use of larger samples than the Pavlovian conditioning studies that have provided support to the fear deficit model, and used only offender samples rather than community controls. Although they did not match psychopathic and control participants for IQ, the study by Baskin-Sommers et al. (2011b) instead matched them for working memory, the aspect of cognitive functioning theorised to be most relevant to the experimental task. The studies are critiqued by Blair (2013), however, on the grounds that none of them matched psychopaths and controls for IQ, and their findings contrast with other work on attentional processing in psychopaths and healthy individuals (Desimone & Duncan, 1995; Pessoa & Ungerleider, 2004).

Instructed fear conditioning studies made use of FPS and ERP measures to produce data relevant to the RMH.

Facial reactions

Two studies (Baskin-Sommers et al., 2011b; Newman et al., 2010) found evidence that FPS to threat-related stimuli is normal in psychopaths when task conditions direct attention to those stimuli, but attenuated when attention has already been directed to alternative stimuli by the time punishment cues are presented, and that these results are unchanged when either factor score for the PCL-R is used instead of the overall score.

This is consistent with the difficulties in reallocating attention that are predicted by the RMH (Patterson & Newman, 1993), but, as set out above, not with the findings from Pavlovian conditioning paradigms (Flor et al., 2002; Rothemund et al., 2012), which could be due to differences between the paradigms: Newman et al. (2010) point out that in instructed conditioning, associations are mediated verbally, and may rely less on amygdala function, which is theorised to be disrupted in fear deficit accounts of psychopathy (Becharia, Damasio, Damasio, & Lee, 1999; Mitchell, Colledge, Leonard, & Blair, 2002). Whilst the authors judge that this explanation is not the most parsimonious account of their findings, it may be the most parsimonious account that can reconcile those findings with those from Pavlovian conditioning tasks in the studies by Flor et al. (2002) and Rothemund et al. (2012). On the other hand, the instructed fear conditioning studies discussed here used considerably larger samples than did the Pavlovian conditioning studies (125 and 87 participants, as compared to 21 and 22 participants), and so their data is perhaps more reliable.

Findings from Baskin-Sommers et al.'s (2011a) study with a sample of African American psychopaths, meanwhile, are incompatible not only with fear deficit theories but also with the RMH: testing for attenuated FPS to threat-related stimuli when attention had already been allocated to alternative stimuli revealed no evidence of an FPS deficit in any condition. Anton et al. (2010) applied similar methodology with a female sample of offenders and found that high scores for psychopathy were associated with reduced FPS when attention was directed towards non-threat-relevant stimuli, consistent with the RMH.

ERP

Baskin-Sommers et al. (2011b) produced ERP data that supports an "early attentional bottleneck" variant of the RMH, in which psychopaths' superior abilities to screen out stimuli that are not relevant to their goals are deployed at an early stage of processing, resulting in unresponsiveness to (non-goal relevant) punishment cues. The study examined the P140, a very early ERP component indexing attention, where participants were presented with cues to impending punishment under two conditions: one where their attention was focussed on those cues; and another where it was focussed on alternative stimuli. Psychopaths showed an enhanced P140 to alternative stimuli as compared to controls, and this finding appeared to be driven by high Factor 1 psychopathy scores.

This data must be treated with some caution, however, as the findings in respect of the P140 emerged unexpectedly in a study designed to investigate the P300 component, and the authors note, moreover, that these findings could instead indicate that psychopaths are simply less engaged by threat-relevant information due to a fear deficit. Whilst the authors seek support for the first interpretation by pointing to Newman et al.'s (2010) finding of normal FPS amongst psychopaths

when attending to threat-relevant stimuli, this must be set against the evidence from Pavlovian conditioning experiments that psychopaths fail to condition and thus fail to produce a normal FPS response to the CS+ (Flor et al., 2002; Rothemund et al., 2012). Again, however, the far larger sample used by Newman et al. (2010) may lend credibility to that study's findings.

Findings in relation to the P140 were consistent with those from Anton et al.'s (2010) study using a female sample: participants with high total psychopathy scores showed larger P100 and P300 components when attention was focussed on alternative stimuli, and smaller P100 and P300 components during the threat-focussed condition, which appears consistent with findings that in conditions where attention is focussed on non-threat-relevant stimuli, psychopaths show reduced FPS (Baskin-Sommers et al., 2011; Newman et al., 2010).

Pavlovian fear conditioning studies

Pavlovian conditioning studies by Flor et al. (2002) and Rothemund et al. (2012) produced ERP data indicating that the attentional/cognitive aspects of conditioning were intact in psychopaths or even superior, even as the psychopaths failed to show conditioned emotional responses to threat cues. As set out above, however, these results are not inconsistent with the RMH, as these studies did not include demands for attentional focus on non-threat related stimuli, and so the RMH would not predict that they would reveal psychopaths' deficits in punishment processing.

Regarding the relationship between the facet/factor structure of psychopathy and cognitive/attentional processing, meanwhile, Veit et al. (2013) found evidence that the interpersonal-affective factor was associated with reduced early attentional processing and good late attentional processing. This data supports the "early

attentional bottleneck" version of the RMH. Unfortunately, this study made use of a small sample that did not offer the range of PCL-R scores necessary to examine the dimensional effects of the factor/facet structure of psychopathy, and it did not make use of a control group.

Passive avoidance studies

It was in the context of passive and active avoidance studies that the RMH evolved from theories based around the BIS and the BAS (Newman & Kosson, 1986; Newman et al., 1987), and in three studies Arnett and colleagues used this methodology to evaluate the hypotheses of a weak BIS, a strong BAS, or difficulties in switching between response sets (as in the RMH) in psychopathy.

Results provide only limited support for the RMH: Arnett et al. (1997) conducted two studies in which psychopaths were exposed to punishment cues after being primed to seek rewards, and found reduced SCRs to punishment cues (in line with the RMH) in one study but not the other; and Arnett et al. (1993) found that psychopaths showed lower heart rate after punishment than did controls, inconsistent with their RMH-based prediction that psychopaths would not decrease their BAS activation after punishment and so would show faster heart rate than controls. These studies had a number of limitations, however, that are set out above in reference to the evidence for fear deficit theories.

(iii) What does psychophysiological research indicate regarding neural abnormalities affecting the processing of reward and punishment by psychopaths?

Studies found evidence for dysfunction in brain areas that are specified by both Kiehl's paralimbic hypothesis (2006) and Blair's integrated emotion systems model (2013), but also areas that are specified only by the paralimbic hypothesis.

The paralimbic hypothesis is compatible both with fear deficit theories of psychopathy and with the RMH. As the hypothesis posits dysfunction in both the amygdala, a crucial site of emotional processing, and the ACC, which is implicated in cognitive/attentional processing, it could even be taken to suggest that psychopaths show abnormalities in both their emotional and cognitive/attentional processing of cues to punishment, i.e. that both fear deficit theories and the RMH are correct.

Pavlovian conditioning studies

Pavlovian conditioning studies revealed abnormalities in brain areas and systems associated with emotional responding: during conditioning, psychopaths showed abnormal limbic prefrontal activity (Veit et al., 2002; Rothemund et al., 2012; Birbaumer et al., 2005), reduced amygdala functioning (Birbaumer et al., 2005; Viet et al., 2002), and evidence of impaired ACC-orbitofrontal connectivity (Veit et al., 2002). The amygdala is thought to be involved in punishment processing (LeDoux, 2003), and the ACC in outcome evaluation and representing the value of reward and punishment (Cardinal, Parkinson, Hall, & Everitt, 2002). These results are consistent with Kiehl's (2006) paralimbic hypothesis but not with Blair's (2013) integrated emotion systems model.

Other studies

Using the TAP paradigm, Veit et al. (2010) found that psychopaths showed no activation in the amygdala, ACC, or prefrontal areas when anticipating punishment. These results are consistent with the existence of a fear deficit in psychopaths deriving from amygdala dysfunction, as specificed by Blair (2013), and with the paralimbic hypothesis (Kiehl, 2006).

Pujara et al. (2014) investigated the role of the ventral striatum (VS) in responses to reward and punishment in psychopaths using fMRI and volumetric analysis, finding that the groups showed different patterns of correlation between PCL-R scores and VS activity and volume: amongst psychopaths, PCL-R scores correlated positively with VS activity to reward vs loss and with the volume of the right accumbens area of the VS, whereas amongst non-psychopaths there were no such correlations.

Prehn et al. (2014) used fMRI to show that individuals scoring highly on Factor 1 of the PCL-R showed diminished activity in the right rostral ACC in response to uncertainty, and diminished activity in the right inferior frontal gyrus (prefrontal cortex) when choosing low-risk, low-reward options (Prehn et al., 2014). These results are in line with Kiehl's paralimbic hypothesis, which specifies dysfunction in the ACC as related to the affective processing deficits in psychopathy (Kiehl, 2006).

Discussion

This review aimed to answer the following questions:

What does psychophysiological research indicate regarding:

(i) Emotional deficits in the processing of reward and punishment by psychopaths?

(ii) Cognitive/attentional abnormalities in the processing of reward and punishment by psychopaths?

(iii) Neural abnormalities affecting the processing of reward and punishment by psychopaths?

Summary and conclusions

The studies reviewed here offer support both for a deficit in emotional anticipation of punishment and for cognitive/attentional abnormalities in psychopathy, although the far larger samples and use only of offender samples in the studies supporting cognitive/attentional accounts render their findings more convincing.

As stated above, emotional processing deficits and cognitive/attentional abnormalities could coexist. It is possible that psychopaths both fail to form emotional associations between punishment and cues of impending punishment and struggle to attend to threat-related information when their attention is focussed elsewhere; and/or that they struggle to form conditioned associations through repeated pairings of stimuli (as in Pavlovian conditioning) but not through verbal instructions (as in instructed fear conditioning). These possibilities cannot be confirmed or rejected at present, as the evidence supporting and undermining the two accounts is mainly drawn from different experimental paradigms and/or tasks:

Pavlovian conditioning studies tend to support fear deficit accounts and undermine attentional accounts, whereas instructed fear conditioning tasks involving attentional manipulations do the opposite. Given that the two sets of studies that make use of these methodologies seem to have been produced by two different research teams, it must be wondered whether developer effects have influenced their results.

Regarding neural models of reward and punishment processing, meanwhile, studies found differences between psychopaths and controls in neural functioning that are predicted by Kiehl's paralimbic hypothesis (2006) but not Blair's (2013) integrated emotion systems model. The paralimbic hypothesis is, therefore, to be preferred. This hypothesis is potentially consistent with either or both of fear deficit theories and the RMH.

Gaps in the evidence base, and future directions

Further research is needed to investigate the possibility that psychopathic reward and punishment processing is influenced by deficits in both emotion and attention. An obvious way to explore this possibility would be to test Pavlovian conditioning responses under conditions that contrast a focus on threat-relevant stimuli with a focus on alternative stimuli.

Further specification is needed of the relationships between the two factors of the PCL-R and the fear deficit and cognitive/attentional abnormality models of reward and punishment processing in psychopathy. Three studies examined these relationships (Baskin-Sommers et al., 2011b; Newman et al., 2010; Veit et al., 2013), and produced interesting results that suggest that the results of other studies reviewed here may have been affected by their failure to analyse the psychopathy factors separately.

Finally, models and research in this area need to be extended to demographic groups other than white males: only one study reviewed here made use of a female sample (Anton et al., 2012), and only one made use of a non-white (African American) male sample (Baskin-Sommers et al., 2011a), both finding differences between these demographic groups and white males. This difference was striking in the case of the African American sample, highlighting possible difficulties in interpreting studies that make use of ethnically mixed samples, and the need for detailed investigation of reward and punishment processing in different ethnic groups.

Limitations

The principal limitation of this review is that its scope excluded studies that were relevant to the theoretical models discussed here but that did not make use of psychophysiological measures: these models cannot be fully evaluated without reference to the large amount of behavioural and self-report data that has been produced on reward and punishment processing in psychopaths. Further, there was considerable research carried out in this area before the development of the PCL-R, which again was excluded from this review. Whilst it may be hoped that that older research has been fully elaborated upon or critiqued by the studies reviewed here, only a fuller review could with certainty establish that to be the case.

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Part two: Empirical paper

Error-monitoring in antisocial youth with and without callous unemotional traits: an event–related potential study

Abstract

Aims

Reduced error-related negativity (ERN) is a possible biomarker of risk for externalising psychopathology, including antisocial behaviour. This study aimed to confirm this amongst young people with a history of antisocial behaviour, and to explore, within this group, the relationship between the ERN and callous unemotional traits.

Method

An externalising group of young people with a history of antisocial behaviour ($N = 39$) and a community sample matched for age and sex ($N = 34$) completed a combined Go/No-Go/flanker task in which they could win or lose money, whilst EEG recordings were taken. Measures of the ERN were taken and analysed together with questionnaire measures of callous unemotional traits and antisocial behaviour.

Results

ERN after errors was reduced amongst externalising young people, particularly those externalising young people who scored highly for callousness (a subfactor of callous unemotional traits). This relationship was due in part to the relationship between callousness and externalising, but perhaps also to a unique relationship between callousness and the ERN.

Conclusions

Results were not conclusive, possibly due to a lack of power in the study, but indicate that externalising young people show reduced ERN, and that callous externalising young people have a particularly reduced ERN, possibly due to the effect of empathy deficits upon the ERN.

Introduction

Disorders characterised by disinhibition and impulsivity tend to co-occur and appear to be underpinned by a common latent factor, known as the externalising dimension or spectrum (Krueger, 1999). Recent research has linked externalising problems to a potential biomarker: reduced amplitude of the error-related negativity (ERN) (Olvet & Hajcak, 2008), an event-related potential that occurs after errors (Gehring, Goss, Coles, Meyer, & Donchin, 1993). The ERN may be of use in understanding and identifying risk of externalising psychopathology in young people, but this has not been investigated experimentally with young people with a documented history of antisocial behaviour. Amongst externalising young people, those showing callous unemotional traits are of particular interest, because they may constitute a clinically useful subgrouping that is at risk for showing particularly severe and persistent antisocial behaviour, and even developing adult psychopathy (Frick & White, 2008; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007). Investigating the ERN amongst externalising young people, including those who show callous unemotional traits, offers hope of better understanding and identification of externalising psychopathology and callous unemotional traits as well as better understanding of the ERN (Hall, Bernat, & Patrick, 2007).

Externalising problems

Disinhibited conditions such as antisocial behaviour and substance abuse tend to co-occur (Krueger, 1999). Factor analytic methods have indicated that the covariance between these conditions is systematic, which indicates that they are underlain by a common factor – labeled externalising – that captures tendencies towards disinhibition, impulsivity, aggression, and negative emotionality (Krueger, 1999; Krueger et al., 2002). This factor applies both to adult and child

psychopathology (via the diagnosis of conduct disorder) and is more than 80% heritable (Krueger et al., 2002), which has been taken to indicate that it has a strong neurobiological basis (Hall et al., 2007). In contrast, conditions characterised by low mood and anxiety are united by a factor labeled internalising, which again has been applied successfully in childhood as well as in adults (Kovacs & Devlin, 1998; Krueger, 1999).

The internalising and externalising spectra offer a coherent framework within which to think about psychological and behavioural disorders as arising from common core processes, rather than as wholly discrete entities (Krueger, 1999). In describing underlying vulnerabilities that may be expressed in a range of different presentations or disorders, depending on other etiologic factors, these constructs offer the prospect of identifying risk for psychopathology before its acute manifestation (Krueger, 1999; Hajcak, 2012).

The likelihood of a neurobiological basis for externalising raises the possibility of identifying biomarkers associated with it, that may differentiate it from internalising vulnerability (Olvet & Hajcak, 2008). One possible biomarker that has been the focus of considerable recent research is the ERN, an event-related potential component that appears after errors in experimental tasks, and is reduced in externalising and enhanced in internalising conditions (Gehring et al., 1993; Olvet & Hajcak, 2008).

In respect of externalising, reduced ERN has been linked to: substance misuse (Franken, Van Strien, Franzek, & van de Wetering, 2007); impulsivity (Potts, George, Martin & Barratt, 2006); low socialization (as measured by the California Psychological Inventory – Gough, 1975) amongst adults (Dikman & Allen, 2000)

and children (Santesso, Segalivitz, & Schmidt, 2005); impulsive-antisocial traits of psychopathy (Heritage & Benning, 2013); and externalising itself (Hall et al., 2007).

Conversely, in respect of internalising, enhanced ERN has been linked to OCD in adults (Gehring, Himle, & Nisenson, 2000; Ruchsow et al., 2005) and children (Hajcak, Franklin, Foa, & Simons, 2008), GAD in children (Ladouceur, Dahl, & Carter, 2004), and related personality traits such as pathological anxiety (Hajcak, McDonald, & Simons, 2003) and negative affect (Luu, Collins, & Tucker, 2000). No study has, as yet, tested the ERN in a sample of young people with a documented history of antisocial behaviour.

There is evidence that the ERN, as an index of externalising and internalising vulnerability, might serve to index traits that increase risk or liability to disorder, rather than the conditions themselves, offering the prospect of using it to identify risk for these states prospectively: amongst children with OCD, enhanced ERN was found even following successful treatment (Hajcak et al., 2008); and amongst spider phobics, ERN amplitude did not increase during active provocation of symptoms (Moser, Hajcak, & Simons, 2005).

The ERN

The ERN is a neural response to the commission of errors in experimental tasks (Gehring et al., 1993). It may be measured at the scalp using EEG, and appears as a sharp negative-going deflection in the ERP that is time-locked to the participant's responses, peaking around 50ms after an incorrect response is given, and is maximal at frontal-central midline recording sites (Falkenstein, Hohnsbein, Hoorman, & Blanke, 1991; Gehring et al., 1993).

The ERN originates in the anterior cingulate cortex (ACC), which is known to be involved in adjusting behaviour according to feedback (Dehaene, Posner, &

Tucker, 1994; Holroyd, Dien, & Coles, 1998). However, the ACC is active following both errors and correct responses, and there have been observations of a smaller negative ERP following correct responses, labeled the Correct Response Negativity (CRN), leading to suggestions that the ERN and CRN may in fact reflect overlapping, or the same, processes, which are accentuated following errors (Falkenstein et al., 1991; Ford, 1999). To take account of the CRN, the ERN is often measured using the difference between the ERP after errors and after correct responses. Some experimental work on error monitoring in externalising conditions, however, has examined the ERN using its amplitude after errors, sometimes alongside CRN amplitude, rather than the difference between these amplitudes (Hall et al., 2007; Heritage & Benning, 2013).

The ERN can be elicited in a range of experimental tasks using various stimulus and response modalities (Bernstein, Scheffers, & Coles, 1995; Falkenstein et al., 1991), and so is understood to index the activity of a general error-monitoring system (Weinberg et al., 2012). There is some debate, however, as to the exact processes indexed by the ERN, and this has relevance for understanding the mechanisms that underlie internalising and externalising vulnerabilities. Current theories either emphasise cognitive processes in generating the ERN, or combine these with an affective/motivational element (Weinberg et al., 2012).

Cognitive neuroscientific accounts

There are two dominant cognitive accounts of the ERN, both of which are concerned with cognitive control (Weinberg et al., 2012). On one of these, the ERN reflects conflict monitoring: the simultaneous activation of tendencies towards two different responses in a task (an incorrect response, and the correct response) (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999); this conflict is detected by the

ACC, which sends signals to the prefrontal cortex calling for greater cognitive control (Yeung, Botvinick, & Cohen, 2004). A reinforcement learning account, meanwhile, suggests that the ACC receives feedback from the basal ganglia on the results of behaviour, and uses this feedback to shape future behaviour, the ERN being produced when the outcomes of behaviour are worse than was expected (Holroyd & Coles, 2002). On either of these accounts, reduced ERN in externalising presentations could reflect cognitive deficits due to fronto-cortical dysfunction, a deficit frequently observed in individuals with disorders or traits of disinhibition (Davidson, Putnam, & Larson, 2000; Dinn & Harris, 2000).

The affective/motivational account

Drawing on evidence that the ERN is influenced by motivational factors (Dikman & Allen, 2000; Pailing & Segalovitz, 2004), Hajcak and colleagues suggest that while conflict monitoring and reinforcement learning theories might explain the basic processes leading to the generation of the ERN, variation in its magnitude across individuals and situations is due to an affective component (Hajcak, 2012; Olvet & Hajcak, 2008; Weinberg, Riesel, & Hajcak, 2012). On this account, the ERN reflects evaluation of the motivational salience of an error, and is the earliest stage of a defensive response to threat, which response includes a range of neural and physiological changes, such as fear-potentiated startle and increased amygdala reactivity (Weinberg et al, 2012). This response has been labelled "defensive reactivity" and varies between individuals (Weinberg et al., 2012).

Indicators of defensive reactivity have been found to be higher in individuals with internalising psychopathology and lower in individuals with externalising psychopathology (Patrick & Bernat, 2010), and so defensive reactivity may be thought of as an endophenotype underlying externalising vulnerability (Olvet &

Hajcak, 2008). The ERN, as a part of that endophenotype, offers both insight into the processes that comprise internalising and externalising, and a means to identify these broad personality factors and risk for their associated forms of psychopathology (Olvet & Hajcak, 2008). If it is in part an index of affective/motivational processes, it may be of particular relevance to an externalising presentation that is characterised by affective deficits: psychopathy.

Externalising psychopathology: psychopathy and callous unemotional traits

Amongst externalising disorders, few generate as much interest and concern as psychopathy, which is associated with particularly severe and chronic antisocial behavior (Hemphill, Hare, & Wong, 1998), and with an apparent failure to learn from adverse experience (Walters, 2003). This is true amongst both adults with psychopathy and young people with psychopathic traits (the full disorder can only be diagnosed in adulthood) (Frick, Barry, & Bodin, 2000), and researchers have been able to use some psychopathic traits, labeled callous unemotional (CU) traits, to identify a subgroup of antisocial young people whose antisocial behaviour may be particularly severe and persistent and have a distinctive etiology, and who may be at risk for developing adult psychopathy (Frick & White, 2008; Lynam et al., 2007).

CU traits are characterised by deficits in guilt, empathy, and remorse (Frick & Ellis, 1999). They represent the affective facet of the multidimensional construct of psychopathy (Essau, Sassagawa, & Frick, 2006). The others are an interpersonal facet, characterised as a deceitful and arrogant interpersonal style with a narcissistic view of the self and conning and manipulative behaviour, and a lifestyle facet that captures an impulsive, irresponsible and antisocial behavioural style (Cooke, Michie, & Hart, 2006). The interpersonal and affective facets are combined in some

conceptualisations of psychopathy into a higher-order interpersonal-affective factor (Hare, 1991).

It is the affective facet of psychopathy that is typically considered to be the core or cardinal feature of the disorder, because it is the one that is most specific to adults with the disorder, whereas the other facets are shared to a considerable extent with other antisocial individuals (Cooke & Michie, 1997). There is evidence that the same is true amongst young people, and that CU traits can be used to designate a meaningful subgroup amongst antisocial young people, with a distinct etiology, presentation, and, importantly, prognosis (Frick & White, 2008). CU traits among antisocial young people are associated with pre-adolescent onset of conduct problems (which is in turn associated with greater likelihood of criminality in adulthood and higher levels of aggression, neuropsychological and cognitive disturbance, impulsivity, and alienation) (Silverthorn, Frick, & Reynolds, 2001), and with particularly severe and stable conduct problems as compared to other young people with severe conduct problems (Christian, Frick, Hill, Tyler, & Frazer, 1997; Frick, Stickle, Dandreaux, Farrell, & Kimonis, 2005). They are relatively stable from adolescence into adulthood, and are predictive of adult psychopathy (Lynam et al., 2007).

CU traits are related to externalising behaviour, but this is largely due to their correlation with the impulsive-antisocial traits of psychopathy, which are in turn highly correlated with externalising (Patrick, Hicks, Krueger, & Lang, 2005). However, a recent study that measured externalising using a specialized measure – the Externalising Inventory (Krueger, Markon, Patrick, Benning, & Kramer, 2007) – found that while the affective traits of psychopathy were not strongly related to an overarching externalising factor or a disinhibition subfactor (these being more

strongly related to the lifestyle aspect of psychopathy), they were robustly associated with a callous aggression subfactor (Venables & Patrick, 2012). CU traits may, then, have a distinctive relationship with some aspects of externalising.

CU traits, psychopathy, and the ERN

Evidence has been found linking psychopathy and its traits, including CU traits, to reduced, enhanced, or normal ERN. As will be seen, complicating factors may include the affective/motivational aspects of experimental tasks, and differential influences of the factors/facets of psychopathy.

Findings of reduced ERN in psychopaths or individuals with psychopathic traits have tended to suggest that ERN is only reduced in these individuals under certain affective and/or motivational circumstances: Munro et al. (2007) found that ERN amongst psychopaths was reduced relative to that of controls only on a task involving the processing of emotional expressions, whereas it was not reduced on a task that was otherwise identical but emotionally neutral; Von Borries et al. (2010) found reduced ERN on a learning task in which the motivational salience of errors was enhanced by the availability of monetary gain (or loss); and Dikman and Allen (2000) found that low-socialised individuals (which the researchers adopted as an analog for psychopathy) showed reduced ERN under conditions where errors were punished, but not where correct responses were rewarded.

Two studies found no difference between the ERN in psychopaths and healthy controls (Brazil et al., 2009; Brazil et al., 2011). These studies, however, used small samples and did not offer rewards or punishments, which may have limited the motivational salience of the task and thus, on an affective/motivational account of the ERN, prevented differences in the ERN from being observed. Studies that have examined the factors of psychopathy separately also failed to find clear

evidence of reduced ERN: one study investigating psychopathic traits using a community sample found that the lifestyle facet of psychopathy was related to both reduced ERN and reduced CRN, perhaps indicating poor response monitoring in general rather than reduced error monitoring, whereas the affective and interpersonal facets were unrelated (Heritage & Benning, 2012), whilst another found that in an offender sample the interpersonal-affective dimension of psychopathy was positively related to ERN amplitude (Bresin, Finy, Sprague, & Verona, 2014). Again, however, this study did not make use of reward and punishment in the experimental task.

It seems, then, that under insufficiently motivating circumstances, psychopaths, especially those scoring highly for the interpersonal-affective traits of the disorder, show no reduction in ERN, but that this emerges under conditions involving reward and/or punishment contingencies and/or emotional processing.

The present study

Reduced ERN is associated with externalising psychopathology (Dikman & Allen, 2000; Franken et al., 2007; Hall et al., 2007; Heritage & Benning, 2013; Potts et al., 2006, Santesso et al., 2005), and so the ERN offers a means to identify risk for such psychopathology, together with insights into the deficits that underlie it (Olvet & Hajcak, 2008). This may be of particular value in research on children who show high levels of externalising, including antisocial behaviour – a group at high risk of developing externalising disorders in adulthood (Hofstra & Verhulst, 2000), and amongst whom early intervention may prevent this. Notable amongst these young people are those with callous unemotional traits, as they may constitute a clinically useful subgrouping who show a particularly severe and chronic pattern of antisocial behaviour, and are at risk for developing psychopathy – a disorder with significant

costs to sufferers and those who are the victims of their violent or antisocial acts (Christian et al., 1997; Frick et al., 2005; Hemphill et al., 1998; Lynam et al., 2007).

Research is limited, however, on the ERN in externalising young people and, amongst that group, young people with CU traits. No study has examined the ERN in young people with a documented history of antisocial behaviour, with or without CU traits. This study aims to fill that gap in the literature.

In the adult literature, meanwhile, there is a wealth of evidence to suggest that externalising is linked to reduced ERN (Dikman & Allen, 2000; Franken et al., 2007; Hall et al., 2007; Heritage & Benning, 2013; Potts et al., 2006, Santesso et al., 2005) – providing a strong basis for predicting the same in young people – but studies of psychopathy or psychopathic traits and the ERN provide no such clear guidance: studies have found the ERN to be both normal (Brazil et al., 2009; Brazil et al., 2011) and reduced (Dikman & Allen, 2000; Munro et al., 2007; Von Borries, 2010) in psychopathy or the presence of psychopathic traits. Most studies, however, have failed to examine the facets or factors of psychopathy separately, to take account of possible affective/motivational influences on the ERN, or to consider whether psychopathy or psychopathic traits have a relationship to the ERN that is independent of their shared variance with externalising.

In answer to the need for data on the ERN in antisocial young people, and clarity regarding its relation to core psychopathic traits, this study used a sample of young people with extensive histories of antisocial behaviour, comparing them to a control group on a task involving monetary reward and punishment, and taking questionnaire measures of CU traits and of present levels of antisocial behaviour. In keeping with many studies in the literature, we used a flanker task to measure ERN responses (Yeung et al., 2004).

In order to confirm that high levels of antisocial behaviour amongst young people are associated with a reduced ERN, we compared the ERN of the "externalising" group to that of the control group. In order to investigate whether, amongst highly externalising young people, those with CU traits constitute a clinically meaningful subgroup whose externalising behaviour is underlain by distinct etiology and processes and is associated with risk for adult psychopathy, we examined the relationship between CU traits and the ERN within the externalising group, controlling for present levels of antisocial behaviour. The relevant literature posits such a subgroup only amongst antisocial/externalising individuals, and so the relationship between the ERN and CU traits was investigated only within the externalising group.

Drawing on the associations between CU traits, externalising, and the ERN, and evidence for reduced ERN amongst psychopaths under certain affective/motivational conditions, together with the evidence for reduced ERN amongst the wider group of externalising young people, we advanced two hypotheses:

1) *Young people with a documented history of antisocial behaviour will show a reduced ERN compared to healthy controls*.
2) *Amongst young people with a documented history of antisocial behaviour, CU traits will be associated with reduced ERN*.

In respect of Hypothesis 2, we remained agnostic as to:

(i) Which of the ICU subscales would be associated with reduced ERN.

(ii) *Whether an association between CU traits and reduced ERN would be due to a unique effect of CU traits or wholly accounted for by the association between CU traits and high levels of externalising behaviour*.

Method

Ethics

Ethical approval was obtained via NHS ethics procedures (ref: 12-LO-0733) and Research & Development clearance from the Anna Freud Centre.

Design

The study used a cross-sectional correlational design, making use of between-groups analysis to test for relationships between externalising and ERN amplitude, and within-group analyses to test for relationships between CU traits and ERN amplitude.

Participants

73 participants were used in this study: an externalising group of 39 young people with a documented history of antisocial behaviour, and 34 healthy controls. The two groups were matched for sex (externalising group: 19 females and 20 males; control group: 16 females and 18 males), age, with ages ranging from 13 to 19 (externalising group: $M = 16.46$; $S.D. = 1.64$; control group: $M = 16.11$; $S.D. = 1.70$), and socioeconomic status, being recruited from the same geographical areas. These groups were derived from a pool of 99 participants from whom data was originally collected. Of these, seven were not used in the study because errors in EEG recording had resulted in poor quality data, and 19 were not used because the necessary questionnaire data had not been collected from them (see below).

The externalising group was recruited from the participants in the Systemic Therapy for At-Risk Teens trial: a clinical intervention trial of multi-systemic

therapy (an intervention for antisocial behaviour) (Henggeler, 1999) that made use of a sample of young people who met criteria to be considered at high risk of requiring out-of-home care, specifically when this risk was associated with antisocial behaviour including conviction as a young offender (University College London, date unknown). Recruitment into this study was via research assistants who visited the homes of participants in the trial for six-month follow-up assessments after the multi-systemic therapy intervention had been completed, and offered them the chance to take part in this further study. The majority of the control group was recruited by visiting schools in the geographical areas where the externalising group had been recruited (in order to match the two groups for socioeconomic status) and offering students the chance to take part in the study, or by contacting teachers at schools in these areas and asking them to approach students who might be interested in taking part in the study. Two control group participants were contacted and recruited via a sibling who had already taken part in the study, and three were recruited via a community drama group.

Data collection was carried out collaboratively by a research team of three doctoral psychology students, producing this and two other studies (forthcoming). The team was supervised by Dr Pasco Fearon, doctoral research supervisor of all members of the team.

Measures

CU traits were assessed using the self-report version of the *Inventory of Callous Unemotional Traits* (ICU) (Essau et al., 2006), a 24-item questionnaire in which respondents score statements (e.g. "I do not show my emotions to others") on a four-point Likert scale from 0 (Not at all true) to 3 (Definitely true). The measure is divided into three subscales: callous; uncaring; and unemotional. These subscales

load on an overarching general factor. The measure was developed on the basis of four items that have been found to be highly indicative of the construct of CU traits using different assessment methods (Forth, Kosson, & Hare, 2014; Frick et al., 2000), and has been validated with samples in Germany (Essau et al., 2006), the USA (Kimonis et al., 2008), Cyprus (Fanti, Frick, & Georgiou, 2009), the Netherlands (Roose, Bijttebier, Decoene, Claes, & Frick, 2010), and Italy (Ciucci, Baroncelli, Franchi, Golmaryami, & Frick, 2014). The factor structure of the measure has been found to be invariant between the sexes (Essau et al., 2006; Ciucci et al., 2014). No cut-off scores for clinical significance have been established for the measure, and whilst some studies have found scores to be significantly higher in offender than community samples, others have not (Feilhauer, Cima, & Arntz, 2012; Pihet, Etter, Schmid, & Kimonis, 2014).

Data on current levels of antisocial behaviour was collected using a self-reported delinquency scale (SRD) (Smith & McVie, 2003). The questionnaire asks respondents about 29 types of delinquent/antisocial behaviour that they might have engaged in during the preceding six months (e.g. "During the last six months, how often did you do these things at school? Arrive late for classes…Fight in or outside school…"), requiring them to give scores on either a four-point or seven-point scale (depending on the item) for the frequency with which they have engaged in these behaviours. The questionnaire produces separate scores for the volume and variety of antisocial behaviour engaged in. The questionnaire was developed as part of a longitudinal study of transitions and changes during adolescence and early adulthood that aimed to explain why some young people with criminal inclinations become offenders, and some more persistent offenders than others, and was developed using a systematic analysis of previously existing relevant instruments, and a review of

questions used in similar studies (Smith and McVie, 2003). No norms have been established for the measure, and no data has been published on mean scores for volume and variety of antisocial behaviour (the scores used in this study) obtained by clinical samples.

Tasks and procedure

Testing took place in the Developmental Neuroscience Unit at the Anna Freud Centre in London. Prior to visiting the laboratory for testing, participants and their parents/guardians were sent information sheets (Appendices C and D) providing full details of the study. Upon arrival at the laboratory, each participant was asked to read and sign a form giving consent to take part (Appendix C), as was their parent/guardian if the participant was aged under 16 (Appendix D). Participants were then prepared for EEG recording.

Testing began with a two-minute baseline recording. Participants then completed an imitation/inhibition task that was not included in the present study. After a break for refreshments, participants were given instructions for the task used in the present study, both on a monitor screen and verbally by one of the experimenters, and completed several practice rounds of the task, observed by the same experimenter. Participants were then introduced via the monitor to their "opponent" for the task (in fact the "opponent" was a video recording of a young person of the same sex and similar age, and the real opponent was the computer running the task) and began the task.

The experimental task was a combined Go/No-Go/flanker task, in which participants saw, on a monitor, a central arrow coloured either red or green, surrounded by grey arrows pointing either in a congruent or a non-congruent direction, with participants being required to press one keyboard button for green

central arrows pointing to the left, another button for green central arrows pointing to the right, and to refrain from pressing any buttons for red central arrows. The aim of the task was to execute all button presses with greater speed and accuracy than the opponent, in order to win money.

The task was organized into four blocks, with two blocks being played against one (fictional) opponent and two against another. Each block consisted of 120 trials. The trials were grouped into sub-blocks of 20 trials, at the beginning of which participants were presented with a slide asking them to select a level of monetary punishment for their opponent, to be imposed should they win the following sub-block of trials by responding faster and more accurately than the opponent. The possible levels of punishment were 10p, 20p, 30p, 40p, 50p, and 60p. In the middle of each sub-block of trials, participants were presented with a "blink" slide, offering them an opportunity to blink without affecting the recording of experimental data, and at the end of each sub-block they were presented with a slide saying either "you win!" or "you lose!" followed, in the case of the "you lose!" slide by a slide telling them the level of the financial punishment that had been imposed upon them. In the case of sub-blocks where they were told that they had won, participants were always rewarded with 35p. Participants were told that they started the task with £3.50, which they could add to or lose by winning or losing against their opponent.

The four blocks of the task were fixed so that participants would lose roughly 50% and win roughly 50% of trials, and would be punished heavily by one opponent (an average of 50p per trial) and lightly by the other (an average of 20p per trial).

After completing the experimental task, participants completed a number of questionnaires, two of which were the ICU and the SRD used in this study.

Participants were then debriefed as to the purpose of the experiment and (in the case of the clinical group but not the control group, many of whom knew one another and so might have shared the information) the deception regarding their fictitious "opponents". Participants were paid £30 for their time, and the amount that they had won in completing the task. Travel costs were reimbursed where receipts were provided.

EEG recording

Continuous EEG recordings were collected using a Hydrocel high-density array of 128 AD/AgCl electrodes soaked in a solution of H_20, KCL, and baby shampoo. Data was collected using the Netstation v.4.4.2 software package and high-impedance filters, sampling at 250Hz, with online filters set to 1-100Hz. Impedances were below 50KΩ and were checked with the Netstation impedance tool.

EEG analysis

To detect and reject artifacts, data was band-pass filtered with cutoffs of 0.3 and 40 Hz. The EEG was segmented around participants' responses (i.e. trials without a response were discarded) from 500ms before the participant's response to 600ms post-response, with a 100ms window from -500ms to -400ms serving as the baseline. Correct and error trials were averaged separately. For each subject the ERN was quantified as the average activity around Cz from 0-100ms after the participant's response.

Data analysis

Statistical analyses were conducted with SPSS (version 2.2). An ERN difference amplitude was calculated for each participant by subtracting the negativity after correct responses (i.e. the CRN) from the negativity after incorrect responses (i.e. the ERN after errors), and statistical analyses were conducted on ERN amplitude

after errors, CRN amplitude, and ERN difference amplitude.

A paired-samples t-test was used to test for the presence of the ERN within the whole sample after an error: mean CRN amplitude was compared to mean ERN amplitude after errors.

As the two experimental groups were already matched for demographic variables (age, sex, socioeconomic status), these were not included as covariates in between-group analyses. Independent samples t-tests were used to look for differences between the externalising and control groups in respect of ICU subscale scores, SRD scores for variety and volume of antisocial behaviour, ERN amplitude after errors, CRN amplitude, and ERN difference amplitude.

For the externalising group, partial Pearson correlations were calculated between: ERN amplitude after errors; CRN amplitude; ERN difference amplitude; and participants' scores on the ICU sub-scales. Regression analyses were then conducted to identify the unique effects within the externalising group of ICU subscale scores and demographic variables (as independent variables) upon ERN amplitude after errors, CRN amplitude, and ERN difference amplitude.

Further to the results from those tests, post-hoc correlational and regression analyses were conducted to identify Pearson correlations between the callous subscale of the ICU and the volume and variety of antisocial behaviour engaged in by participants (as measured by the SRD), and the unique predictive effect of the callous subscale of the ICU on ERN amplitude after errors, CRN amplitude, and ERN difference amplitude, over and above the contribution of the volume and variety of antisocial behaviour, as measured by the SRD.

Results

The ERN

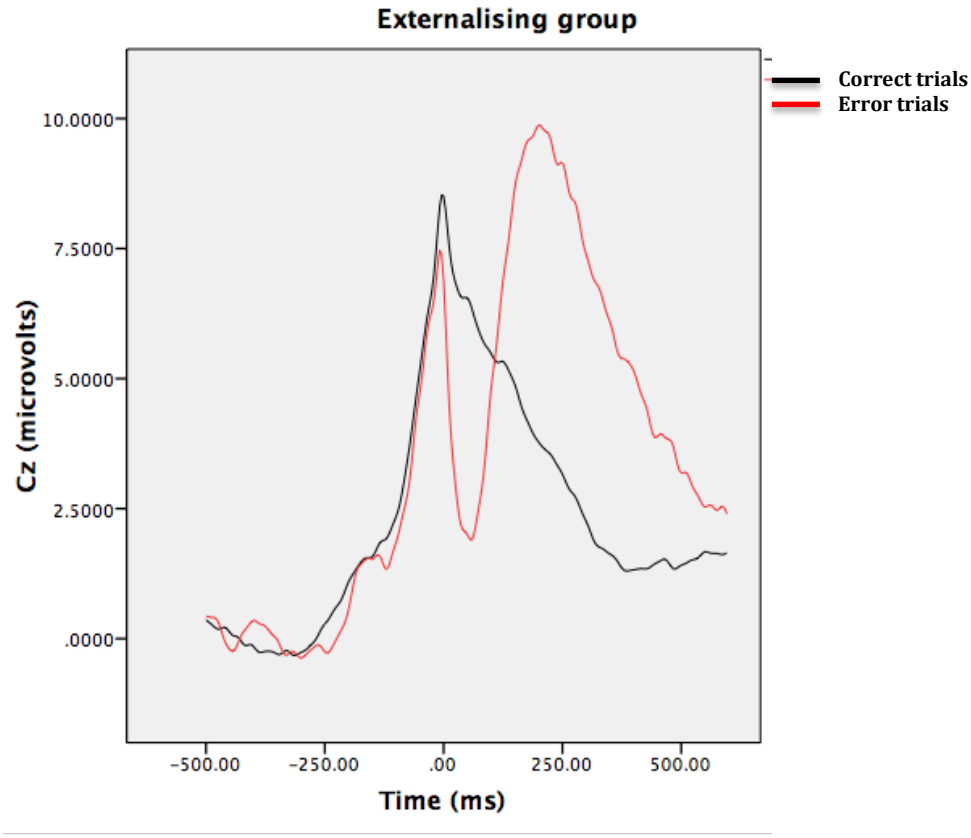
Table 1 shows descriptive statistics for the participants in the study.

Table 1. Descriptive variables of interest: mean (S.D.)

Variables	Externalising	Control
Age	16.46	16.02
	(1.64)	(1.70)
ICU callous	7.13	7.20
	(3.87)	(4.51)
ICU unemotional	8.46	7.97
	(3.24)	(2.75)
ICU uncaring	8.23	7.00
	(4.65)	(3.75)
SRD volume	7.74	1.65
	(8.86)	(4.01)
SRD variety	1.82	.56
	(1.99)	(1.58)

Figure 1 presents the average waveforms recorded at Cz for correct and incorrect trials for both groups. The ERN after errors can be seen as a sharp negative-going deflection that peaks 0-100ms post-response. The scalp topography maps shown in Figure 2 confirm that ERN amplitude was maximal at Cz for both groups.

Figure 1 – grand average ERP waveforms



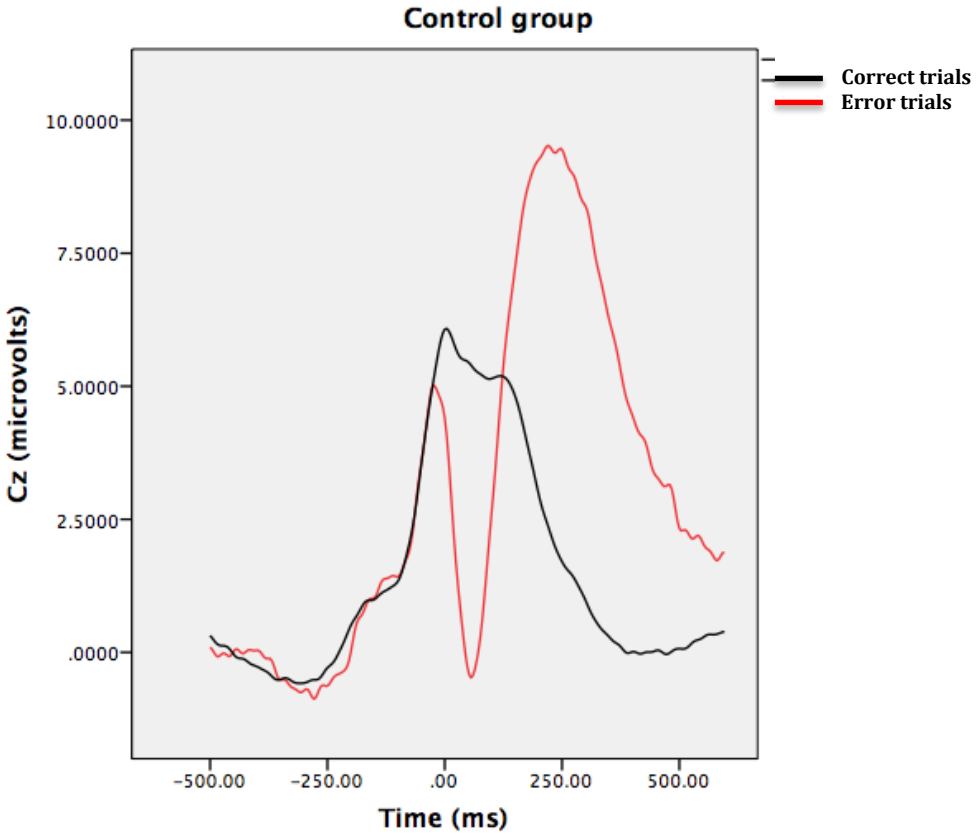
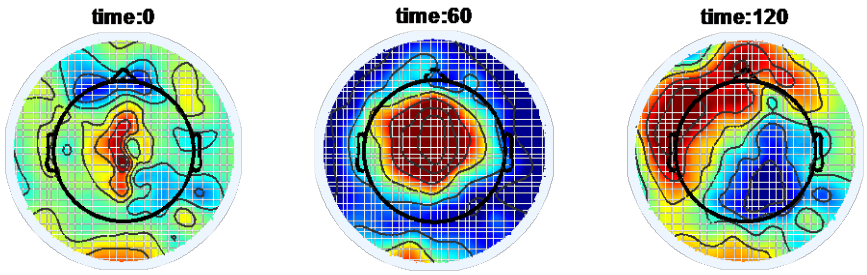
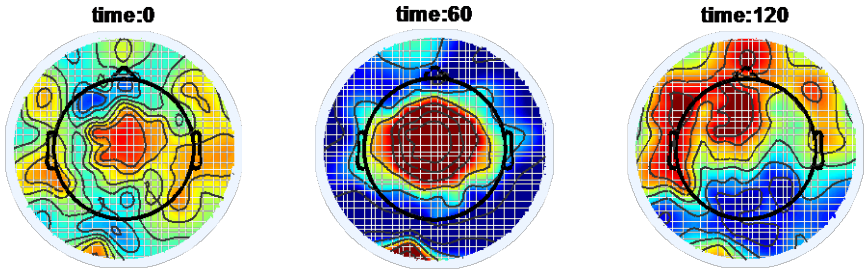

Control group
Correct trials
Error trials
10.0000
7.5000
5.0000
2.5000
.0000
Cz (microvolts)
-500.00
-250.00
.00
250.00
500.00
Time (ms)

Figure 2 – scalp topography maps (time in ms)

Externalising group



Control group



As expected, negativities for the whole sample were larger after errors (M = 2.19, $S.D.$ = 4.04) than after correct responses (M = 5.35, $S.D.$ = 3.45), confirming the presence of the ERN ($t(72) = 10.01$, $p < .001$).

Comparing groups

The two groups did not differ significantly in their scores for the callous subscale of the ICU, $t(71) = -.08$, $p = .94$, the uncaring subscale of the ICU, $t(71) = 1.22$, $p = .23$, or the unemotional subscale of the ICU, $t(71) = .69$, $p = .49$. The two groups differed significantly in their scores on the SRD for volume of delinquent behaviours engaged in, $t(71) = 3.87$, $p < .001$, and variety of delinquent behaviours engaged in, $t(71) = 2.97$, $p = .004$.

There was a difference between groups in ERN amplitude after error trials, $t(71) = 2.13$, $p = .037$, indicating that the ERN was smaller (i.e. less negative) after error trials in the externalising group (M = 3.11, $S.D.$ = 4.04) than in the control group (M = 1.14, $S.D.$ = 3.84). There was no difference in CRN amplitude $t(71) = 1.48$, $p = .567$ between the externalising (M = 5.90, $S.D.$ = 3.41) and control (M = 4.71, $S.D.$ = 3.44) groups. For ERN difference amplitude, there was no significant difference between the externalising (M = -2.79, $S.D.$ = 2.32) and control (M = -3.57, $S.D.$ = 3.05) groups, $t(71) = 1.24$, $p = .22$, but the non-significant difference indicated that ERN amplitude was smaller in the externalising group.

CU traits analyses

Table 2 sets out partial Pearson correlations within the externalising group between the subscales of the ICU, ERN amplitude after error, CRN amplitude, and ERN difference amplitude, with age, sex, and SES as covariates. A significant positive partial correlation was found between ERN amplitude after error and the callous subscale of the ICU (Pearson's $r = .37$, $p = .029$), indicating a smaller (more

positive) ERN amongst those scoring highly on the callous subscale, whereas no other partial correlations between ICU subscale scores and ERN amplitude after error, CRN amplitude, or ERN difference amplitude were significant. The partial correlation between callousness and ERN difference amplitude approached significance, however, and indicated that callousness was associated with smaller ERN (Pearson's $r = .30$, $p = .08$).

Table 2. Pearson correlations (with *p* values) between ICU subscales and ERPs – externalising group

Variables	1.	2.	3.	4.	5.	6.
1. ICU callous	-	.20 (.256)	.59 (.000)	.37 (.029)	.23 (.184)	.30 (.08)
2. ICU unemotional		-	.14 (.43)	.11 (.53)	.05 (.80)	.12 (.48)
3. ICU uncaring			-	-.02 (.91)	-.14 (.44)	.17 (.33)
4. ERN amplitude after errors				-	.82 (.000)	.52 (.001)
5. CRN					-	-.07 (.701)
6. ERN difference amplitude						-

Covariates: age; sex; SES

A series of regression analyses were conducted. Simultaneous multiple regressions were used, in order to control for variable effects by only allowing unique variation attributed to each variable in the model.

In order to establish the predictive effects of the subscales of the ICU upon the ERN, we conducted three regression analyses, which each took as their independent variables the three subscales of the ICU together with age, sex, and SES of participants, and took as their dependent variables (respectively) ERN amplitude after errors, CRN amplitude, and the ERN difference amplitude. Where ERN amplitude after errors was the dependent variable, the overall model was not

predictive ($F(6,31) = 1.95$, $p = .104$, $R^2 = .27$). The callous subscale of the ICU, however, did predict ERN amplitude after errors ($B = .64$, $t(37) = 2.90$, $p = .007$), with a smaller ERN being predicted by higher scores for callousness. Where CRN amplitude was the dependent variable, the overall model was not predictive ($F(6,31) = 1.22$, $p = .323$, $R^2 = .19$), but callousness was predictive ($B = .45$, $t(37) = 2.30$, $p = .028$), with higher scores for callousness predicting smaller CRN. Where ERN difference amplitude was the dependent variable, the overall model was not predictive ($F(6,31) = .92$, $p = .49$, $R^2 = .15$), and nor was the callous subscale of the ICU ($B = .19$, $t(37) = 1.38$, $p = .179$), but its effect was in the same direction as its (significant) predictive effect upon ERN amplitude after errors.

Further to the finding that the callous subscale of the ICU was a significant predictor of ERN amplitude after errors, we conducted six post-hoc regression analyses to investigate the relationships between this subscale, the volume and variety of current antisocial behaviour, and the ERN.

Three of these were intended to investigate the relationship between callousness and the ERN when variety of current antisocial behaviour was controlled for. Each of them took as its independent variables the callous subscale of the ICU (but not the other subscales, in order to retain any variance shared with them), the age, sex, and SES of participants, and the variety of antisocial behavior engaged in by participants, and took as their dependent variables (respectively) ERN amplitude after errors, CRN amplitude, and ERN difference amplitude.

Where ERN amplitude after errors was the dependent variable, the overall model was not predictive ($F(5,32) = 1.68$, $p = .169$, $R^2 = .21$), but the callous subscale of the ICU was predictive ($B = .47$, $t(37) = 2.41$, $p = .022$), with a smaller ERN being predicted by higher scores for callousness. Variety of antisocial

behaviour did not predict ERN amplitude after errors (B = -.29, t(37) = -.81, *p* = .429). Where CRN amplitude was the dependent variable, the overall model was not predictive (F(5,32) = .57, *p* = .722, R^2 = .08), and neither were callous subscale score (B = .22, t(37) = 1.21, *p* = .24) or variety of antisocial behaviour (B = .02, t(37) = 0.07, *p* = .943). Where ERN difference amplitude was the dependent variable, the overall model was not predictive (F(5,32) = 1.64, *p* = .179, R^2 = .20) but the score for the callous subscale of the ICU was predictive (B = .26, t(37) = 2.27, *p* = .03), with a smaller ERN difference amplitude being predicted by higher scores for callousness, whilst the score for variety of antisocial behaviour was not predictive (B = -.32, t(37) = 1.5, *p* = .143).

The remaining three regression analyses were intended to investigate the relationship between callousness and the ERN when volume of antisocial behaviour was controlled for. They each took as their independent variables the callous subscale of the ICU, the age, sex, and SES of participants, and the volume of antisocial behaviour engaged in by participants, and took as their dependent variables (respectively) ERN amplitude after errors, CRN amplitude, and the ERN difference amplitude.

Where ERN amplitude after errors was the dependent variable, the overall model was not predictive (F(5,32) = 1.55, *p* = .202, R^2 = .20), but the callous subscale score was predictive (B = .44, t(37) = 2.25, *p* = .032), with higher scores for callousness predicting smaller ERN amplitude. The volume of antisocial behaviour did not predict ERN amplitude after errors (B = -.03, t(37) = -.37, *p* = .714). Where CRN amplitude was the dependent variable, the overall model was not predictive (F(5,32) = .57, *p* = .72, R^2 = .08), and neither were the callous subscale score (B = .21, t(37) = 1.21, *p* = .235) or the volume of antisocial behaviour (B = .01, t(37) =

1.14, p = .893). Where the ERN difference amplitude was the independent variable, the overall model was not predictive (F(5,32) = 1.27, p = .3, R^2 = .17), and nor was the callous subscale score (B = .224, t(37) = 1.97, p = .057), although this result very closely approached significance and was in the same direction as the significant predictive effect of the callous subscale on ERN amplitude after errors. Volume of antisocial behaviour was not predictive (B = -.04, t(37) = -.84, p = .41).

Discussion

Externalising behaviour represents a broad dimension of vulnerability linking together disorders of disinhibition, including psychopathy (Krueger, 1999; Venables & Patrick, 2012). Externalising is associated with a reduced ERN (Dikman & Allen, 2000; Franken et al., 2007; Hall et al., 2007; Heritage & Benning, 2013; Potts et al., 2006, Santesso et al., 2005), such that, although there is debate to as exactly which aspect of monitoring and responding to errors is indexed by the ERN (Botvinick et al., 1999; Holroyd & Coles, 2002; Olvet & Hajcak, 2008), it has potential as a biomarker for externalizing (Olvet & Hajcak, 2008). This has not yet been demonstrated, however, amongst young people with a documented history of antisocial behaviour. Amongst this group are young people who show high levels of CU traits – the most distinctive feature of adult psychopathy. This subgroup of antisocial young people is of clinical interest, as it has been found to show particularly severe and chronic antisocial behaviour that may have a distinct etiology from that of other antisocial young people (Frick & White, 2008; Hemphill et al., 1998). Studying the relationship of CU traits to the ERN amongst young people with high levels of externalising behaviour could consolidate the validity and usefulness of the high-CU subgrouping amongst antisocial young people by demonstrating

difficulties with error monitoring (as indexed by the ERN) that go beyond those accounted for by high levels of externalising.

For these reasons we used a competitive Go/No-Go/flanker task to compare the ERN in a group of young people who have a documented history of antisocial behaviour with that in a control group, and then examined the relationship between CU traits and ERN amplitude within the externalising group.

We predicted that:

1) *Young people with a documented history of antisocial behaviour would show a reduced ERN compared to healthy controls*.
2) *Amongst young people with a documented history of antisocial behaviour, CU traits would be associated with reduced ERN*.

In respect of Hypothesis 2, we remained agnostic as to:

(i) *Which of the ICU subscales would be associated with reduced ERN*.

(ii) *Whether an association between CU traits and reduced ERN would be due to a unique effect of CU traits or wholly accounted for by the association between CU traits and high levels of externalising behaviour*.

In respect of Hypothesis 1, we found that the externalising group showed greater volume and variety of antisocial behaviour than did the control group, and that ERN amplitude after errors was smaller in the externalising group than in the control group. The ERN difference amplitude was also smaller in the externalising group than in the control group, but this difference was not significant.

In respect of Hypothesis 2, we found: that the two groups showed similar levels of CU traits; that, amongst the externalising group, callousness predicted both reduced ERN amplitude after errors and CRN amplitude; that the relationship

between callousness and ERN amplitude after errors remained when variety and volume of antisocial behaviour were controlled for, but the relationship between callousness and CRN amplitude did not; and that when variety (but not volume) of antisocial behaviour was controlled for, callousness predicted not only reduced ERN amplitude after errors but also reduced ERN difference amplitude.

Interpretation of findings

Hypothesis 1 – Young people with a documented history of antisocial behaviour will show a reduced ERN compared to healthy controls.

The results of comparison between the two groups were consistent with this hypothesis, in that the externalising group was found to engage in significantly more antisocial behavior (both a greater volume and variety of behaviours), and ERN amplitude after errors was found to be reduced in this group. This evidence was not conclusive, however, because the finding of reduced ERN difference amplitude in the externalising group as compared to the control group was not significant. It is the negativity after errors relative to the negativity after correct responses that reveals processes distinctive to error monitoring, and so the ERN is best measured by subtracting the amplitude of the negativity after correct responses (i.e. the CRN) from the negativity after errors (i.e. the ERN after errors). A significant finding in respect of the ERN difference amplitude, therefore, would have provided stronger evidence in respect of this hypothesis.

One possible interpretation of these findings is that the reduced ERN amplitude after errors in the externalising group reflected not a difference in processing of errors, but rather reduced processing of responses in general – this was the interpretation given by Heritage & Benning (2013) to a finding of reduced ERN amplitude after errors together with reduced CRN amplitude amongst individuals

scoring highly for the impulsive-antisocial traits of psychopathy (which are closely related to externalising – Patrick et al., 2005). Unlike that study, however, our results did not find a reduced CRN amplitude, as might be expected in the case of reduced general response monitoring. An alternative account of these findings would be that the externalising group did show reduced neural activity to errors specifically, such as would be captured by the ERN difference amplitude, but that this study lacked the necessary power to demonstrate this. Consistent with this interpretation, the non-significant difference between the groups in ERN difference amplitude indicated smaller ERN difference amplitude in the externalising group. Such an interpretation would also be in line with other findings linking externalising to the ERN, and so is perhaps to be preferred.

Hypothesis 2 – Amongst young people with a documented history of antisocial behaviour, CU traits will be associated with reduced ERN.

Findings in respect of this hypothesis present a complex picture, in that they indicate similar levels of CU traits between the two groups and, within the externalising group, differential relationships between callousness and ERN amplitude after errors, CRN amplitude, and ERN difference amplitude, which are driven in part by variance that is shared with the volume and variety of antisocial behaviour.

The similarity of ICU scores between the two groups may indicate either a failure to detect elevated CU traits in the externalising group, perhaps due to socially desirable responding, or actual parity in levels of CU traits between the two groups, which may in turn indicate either that the externalising sample was not representative of antisocial young people more broadly, or that CU traits are not in general higher amongst antisocial young people than their non-antisocial peers (Feilhauer, Cima, &

Arntz, 2012), which last possibility may have implications for the construct of the high-CU subgrouping amongst antisocial individuals (See "Limitations" and "Implications and future directions" below).

As regards the relationship between callousness and the ERN, the most consistent element of the results was a finding that callousness predicted reduced ERN amplitude after errors, even when volume and variety of antisocial behaviour were controlled for. As with Hypothesis 1, the question arises as to whether this indicates reduced general response monitoring, or a reduced ERN that the study failed to detect via the ERN difference amplitude, due to either a lack of power in the study or a failure to accurately measure CU traits in the externalising group.

In support of the first interpretation: when neither volume nor variety of antisocial behaviour was controlled for, callousness predicted not only reduced ERN amplitude after errors but also reduced CRN amplitude, and failed to predict reduced ERN difference amplitude, suggesting that callous individuals had reduced general response monitoring but not reduced error monitoring, specifically.

In support of the second interpretation (that the study failed to detect the relationship between callousness and ERN difference amplitude): first, when either volume or variety of antisocial behaviour was controlled for, the relationship between callousness and CRN amplitude disappeared, whereas the relationship between callousness and ERN amplitude after errors did not; second, when variety (but not volume) of antisocial behaviour was controlled for, callousness predicted not only reduced ERN amplitude after errors, but also reduced ERN difference amplitude, giving a more conclusive finding of reduced error monitoring as indexed by the ERN; and, third, the partial Pearson correlation between callousness and ERN difference amplitude approached significance, as did the predictive effect of

callousness upon ERN difference amplitude when volume of antisocial behavior was controlled for, and both were in the same direction as the (significant) equivalent findings in respect of ERN after errors.

A plausible synthesis of these two interpretations is that, among externalising young people, callous individuals show both reduced general response monitoring and reduced error monitoring as indexed by the ERN. Reduced general response monitoring is due to levels of externalising: callousness did not have any unique predictive effect on CRN amplitude when antisocial behaviour was controlled for. Reduced error monitoring by callous individuals, meanwhile, was accounted for at least in part by externalising, in the form of volume of antisocial behavior: the significant effect of callousness on ERN difference amplitude disappeared when volume of antisocial behaviour was controlled for. There may also, however, have been a unique predictive effect of callousness upon the ERN: callousness had a unique predictive effect upon ERN amplitude after errors even when both volume and variety of antisocial behaviour were controlled for, and a nearly significant predictive effect on the ERN difference amplitude when volume of antisocial behavior was controlled for. It may be that with a larger sample or more accurate measurement of CU traits, this analysis would have yielded a significant effect in respect of the ERN difference amplitude, and thus a conclusive finding that callousness predicts reduced ERN over and above the variance that it shares with externalising.

It is unsurprising that callousness should have been the subscale of the ICU that was predictive of reduced ERN, as this relationship seems to have been at least in part mediated by externalising/antisocial behaviour, and callousness has been found to show the strongest associations, among the ICU subscales, with

externalising and conduct disorder symptoms (Essau et al., 2006). However, if callousness predicted reduced ERN over and above the effect of antisocial behaviour (as these results suggest), then this requires explanation, which may shed light on the processes that underlie the ERN.

One such explanation would be that it is lack of empathy, the aspect of CU traits that the callous subscale of the ICU was designed to capture, that accounts for the unique relationship between callousness and reduced ERN. This would be consistent with findings by Munro et al. (2007) that psychopaths showed reduced ERN in a flanker task involving processing the emotional expressions of others (but not in an emotionally neutral task), and by Santesso and Segalovitz (2009) that ERN amplitude was positively correlated with empathy amongst young people.

Such an explanation is consistent, moreover, with a motivational/affective account of the ERN as an index of defensive reactivity (Hajcak, 2012; Olvet & Hajcak, 2008;Weinberg et al., 2012): following Blair (2013), deficits in processing the emotional expressions of others may be understood as deficits in the ability to pair stimuli with outcomes, and to effectively represent, and therefore respond to, the likely value of outcomes (Blair argues that emotional expressions are reinforcers serving to rapidly transmit information on the valence of objects and actions between people). Deficits in empathy may, therefore, be integral to deficits in defensive reactivity amongst high-CU externalising young people, and thus contribute to a reduced ERN.

It is perhaps surprising, however, that no relationship was found between the uncaring subscale of the ICU and ERN, given that this subscale captures, amongst other things, a lack of concern regarding performance on tasks (Essau et al., 2006): if the ERN is influenced by motivational factors, then a lack of concern for

performance on tasks might be expected to predict reduced ERN.

The relationship between motivation and the ERN is complex, however: Dikman and Allen (2000) found that low-socialised individuals (a measure that was used due to its similarity to psychopathy) showed reduced ERN only in a punishment-only condition where reward was not available, and not in a reward-only condition. This finding seems to suggest that psychopaths would be adequately motivated in a task, such as the one used here, where rewards are available, and so would not show a reduced ERN (insofar as ERN amplitude reflects motivation).

On that basis, it could be that the failure to find conclusive evidence of reduced ERN in most of the analyses in this study was due to the motivational conditions of the experimental task: this suggests directions for future research (see "Implications and future directions" below).

The finding of a possible relationship between the callous but not the uncaring subscale of the ERN, moreover, suggests a dissociation between empathy-related and motivational influences on the ERN, which again should be investigated further (see below).

Limitations

The most substantial limitation in the study was that it may have been underpowered, and thus failed to find convincing results in respect of the ERN difference amplitude. A larger study making use of similar methods may be able to produce such results.

Similarly, the use of the self-report version of the ICU may have obscured the relationship between callousness and the ERN difference amplitude if, via socially desirable responding within the externalising group, it prevented accurate measurement of CU traits.

The samples used in the study may not have been fully representative of the populations from which they were drawn: both samples inevitably consisted of young people who volunteered for the study and then kept their appointments, and these young people may have differed systematically from those who did not. In the case of the externalising group, those who entered the study and attended for testing may have been those who felt more positive about the intervention study from which they were recruited, or who benefited more from it, or who were more conscientious and prosocial to begin with (which could account for the similar ICU scores between the two groups). Members of the control group may also have been more conscientious and prosocial than their peers, especially as some of them were selected by their teachers as likely to be interested in the study and keep appointments for testing.

As set out above, the availability of both reward and punishment during the experimental task could have obscured some of the effect of externalising and CU traits upon the ERN: Dikman and Allen (2000) found that low-socialised individuals showed a reduced ERN only in the punishment-only condition of a flanker task, and so it is possible that a task that separated reward and punishment into separate blocks of trials would have produced clearer findings.

Finally, the study employed a large number of statistical analyses, raising the possibility that one or more of the significant findings reported above might be the result of Type 1 error.

Implications and future directions

The ERN in externalising youth

The results here extend the finding of reduced ERN amongst externalising individuals to young people with documented histories of antisocial behaviour, but

are not conclusive, as the significant results were mainly in respect of ERN amplitude after errors, rather that the ERN difference amplitude. They provide partial support for the usefulness of the ERN as a biomarker or endophenotype for risk of externalising psychopathology. These findings are in need of replication in a larger study.

CU traits – a meaningful subcategory?

The results of this study suggest that there is a distinct contribution of CU traits to reduced ERN, over and above that of externalising, but do not provide conclusive evidence of it, again due to a failure to find significant results in respect of the ERN difference amplitude.

These results, moreover, are from a sample that that was no more callous and unemotional than the control sample, and so their implications for descriptions of a high-CU subgroup amongst antisocial young people are not entirely clear: if the clinically relevant features of this subgroup are thought to appear only with elevated CU traits (relative to the general population), then the results of this study have limited relevance to this subgroup. Alternatively, this and other studies showing parity of CU traits between antisocial and other individuals may suggest that the distinctive features of the high-CU subgrouping amongst antisocial individuals arise not from unusually high levels of CU traits, but rather from an interaction between these traits and antisociality.

In any case, this study makes some contribution to descriptions of high-CU antisocial young people, as it suggests a particular contribution of callousness to poor error monitoring, which may be related to reduced empathy amongst callous individuals.

A more powerful study, or one that does not rely wholly on self-report to measure CU traits, or one that separates reward-only and punishment-only conditions, might provide conclusive evidence as to whether CU traits uniquely predict reduced ERN, whilst work on the ERN with high-CU individuals in traditional emotion-processing paradigms could provide further information on CU traits, as well as on the ERN.

The ERN

Insofar as the results here can be taken as indicating a unique contribution of callousness to reduced ERN, they provide support for a motivational/affective account of the ERN. A finding that it is the callous subscale of the ICU that predicts ERN, whereas the uncaring subscale does not, may suggest that lack of empathy may make its own distinctive contribution to reduced ERN, separable from that of motivation.

Future research might explore the possible dissociation between empathic and motivational influences on the ERN by, again, investigating it in emotion-processing tasks under conditions that vary the availability of reward and punishment.

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Part 3: Critical reflections

These reflections will address three aspects of the thesis: first, the process of conducting the empirical study presented here, including design, data collection, and data processing; second, the theoretical relationship between the subject matter of the empirical paper (error-monitoring and externalising behaviour) and the literature review presented here (reward and punishment processing in psychopathy); and, third, the clinical implications of the empirical study.

The research process

Study design

The study was conceived of within the context of an already-ongoing larger project that involved numerous other researchers, which had implications for both its design and execution.

Data collection had already begun by the time I became involved with the project, meaning that the study questions/hypotheses had to be designed to fit the samples, experimental task, and measures rather than vice versa. This meant that it was very clear from the outset what would and would not be possible in terms of setting up and carrying out the study, so that no adaptations to the methodology were imposed by practical difficulties, but also that it was difficult to achieve a perfect fit between the study's aims and hypotheses and its methodology.

In particular, in light of evidence from a study by Dikman and Allen (2000) that reduced ERN amongst psychopaths emerged only under conditions where punishment but not reward was available, an optimal experimental task would have included punishment-only, reward-only, and reward-and-punishment conditions, to examine the effect of these different motivational contexts upon ERN amplitude. Whilst that finding alone was not enough, looking ahead before conducting the

study, to make it seem that it would not be worthwhile to proceed, in retrospect, in light of the results of the study, which did not include conclusive findings of reduced ERN, it seems possible that this area of poor fit between study aims and experimental task might have prevented such a finding.

Further, working within the context of a larger, pre-existing project made it difficult to be aware, at the time of designing the study, of all relevant details of data collection. In particular, questionnaire data (the Inventory of Callous Unemotional Traits and a Self-Reported Delinquency questionnaire) for the externalising group in my study had already been collected by the time the study was designed, but I did not have access to it, and so was not aware that this questionnaire data was in fact missing for 19 participants. This meant that those participants could not be included in the externalising group, which may have resulted in the study being underpowered, which may again have contributed to the failure to produce conclusive findings.

Data collection

The process of recruitment into the control group (for which I was jointly responsible, the externalising group having already been recruited by the time I joined the project) was relatively unproblematic, in that it was ultimately possible to recruit the required number of participants, of the required age and gender. It made very clear, however, the importance of early preparation and a large margin of error in the number of potential avenues for recruitment that were explored. At every stage of the recruitment process, there was a high rate of attrition. We contacted a large number of schools in order to recruit participants from amongst their pupils, but in only a minority were we able to contact the appropriate member of staff. When we did speak to that person, only a minority expressed interest in allowing us to contact

their students. Of those who did, only a minority responded to subsequent emails or phone calls. Of those who did, most were ultimately not able to facilitate our contacting their students, usually due to logistical difficulties, most notably the arrival of school holidays. Where we were able to visit schools to present the study to students and take the contact details of those who were interested, only a minority of those who expressed interest responded to our attempts to contact them and agreed to take part in the study. And of those who did, unfortunately, a large proportion failed to attend their testing sessions, without notifying us. It became clear that, even when it seemed that we had access to more than enough potential participants, it was necessary to maintain a large reserve pool of more potential participants, and of schools that might be able to supply yet more.

We learned with experience the optimal approach to booking sessions: stressing on the telephone and in emails the importance of either attending or letting us know if it was necessary to cancel or rearrange; booking in testing sessions no more than a month in advance, wherever possible; and making reminder phone calls not just the day before (by which time the participant, if they had forgotten the appointment or decided not to attend, would probably have made other plans), but a week before as well.

Testing sessions themselves proved challenging initially, largely due to the difficulty of learning and executing a fairly complex testing procedure: there were numerous minor errors, and on three occasions all data from the task used in this study were lost due to experimenter error (on one occasion a faulty EEG net was used for testing; on another the net was unplugged before data recording had been completed, causing it to be lost; and on a third a test version of the experimental task was accidentally used). We became aware that the relative infrequency of testing

sessions was inhibiting proper learning of the task: after several testing sessions in close succession, we found that errors reduced, and stayed at a low level. Working with our adolescent participants presented a different sort of challenge in testing sessions: whilst they were invariably polite and helpful, it was a struggle to maintain their interest and engagement during lengthy and repetitive testing, which may have had an adverse effect on the EEG data collected in some instances, owing to the effect of boredom on neural activity (Luck, 2005).

Working as part of a larger project, and as part of a smaller team within it, continued to present challenges beyond the design stage of the study, as well as many benefits: good co-ordination was required to ensure that all necessary tasks were done, and done in the most efficient manner possible. Inevitably there were logistical difficulties. Members of the smaller, immediate team were rarely physically together, and had no contact at all with many others involved in the project, and at times this led to inefficient use of time, duplication of effort, and/or necessary tasks not being done in a timely fashion.

Data processing and analysis

Finally, processing and analysis of EEG data required the use of unfamiliar software that was designed by other researchers and not with the inexperienced user in mind. This produced some delay in analysing the data from the study, which might have been avoided with more focussed effort on learning to use this software in advance.

Conclusions

The principal (and perhaps predictable) lesson that this researcher drew from the research process was the importance of getting thoroughly to grips with all aspects of the study at an early stage. Before formulating a study question and fitting

it to a methodology, it is necessary to become well-enough acquainted with the relevant literature that all possible obstacles to a true test of the hypotheses can be identified and avoided – for example, in this case, the possibility that positive findings might depend upon the correct configuration of reward and punishment contingencies in the experimental task. Upon becoming involved with any organisation or project through which data will be collected, it is necessary to become thoroughly acquainted with the structure and workings of that organisation/project, so as to be fully aware of what is to be done when, by whom, and where required information or study data is to be found, and how it is to be accessed. Before beginning processing and analysis of unfamiliar data (e.g. EEG data) with unfamiliar software, it is necessary to become thoroughly acquainted with the methods and technologies involved. At every stage, the temptation for the inexperienced researcher is to find out just enough to be able to proceed to the next step in the research, rather than to develop the more comprehensive overview that would allow potential difficulties to be identified, considered, and prepared for or addressed well in advance.

Research/academic implications

The most novel finding in the empirical study presented here was that of a reduced ERN amongst externalising young people with psychopathic traits. Discussion of these results in the empirical paper maintained a focus on the ERN and the processes that may underlie it. It may be helpful, however, to link these processes to others that play a role in learning and the regulation of behaviour in psychopathy. One obvious such link, given the focus of the literature review presented here, would be to theories of reward and punishment processing in psychopathy. There are

potential links with both the cognitive/attentional and emotional processing/fear deficit theories of such processing. As will be seen, on either account, the same processes that are theorised to underlie deficits in reward and punishment processing might also underlie reduced neural responsiveness to errors, as indexed by the ERN.

Cognitive/attentional abnormalities

The dominant cognitive/attentional account of reward and punishment processing abnormalities in psychopathy is the Response Modulation Hypothesis (RMH) (Patterson & Newman, 1993), the psychophysiological evidence for which was evaluated in the literature review presented here. According to the RMH, psychopaths do not have any deficit in their capacity to anticipate punishment per se, but rather: have difficulty in attending to cues to impending punishment when they are engaged in goal-directed behaviour that focusses their attention elsewhere; and, when actually experiencing punishment, experience an increase in arousal that intensifies their goal-directed behavior, preventing them from properly reflecting upon their current behavior and modifying it to avoid further punishment. Whilst the RMH focusses on attention and responses to external stimuli, there is no theoretical reason why it could not extend to attention and responses to one's own actions (such as is indexed by the ERN).

According to the RMH, individuals who are engaged in goal-directed behaviour form a reward-seeking response set, in which cognitive resources are directed towards organising goal-seeking behaviour. This focus is interrupted by momentary shifts of attention to assess environmental changes that could signal a need to adapt behaviour. Psychopaths, in this model, form reward-seeking response sets more readily and more intensely than other individuals, and interrupt them with fewer shifts of attention to assess changing environmental contingencies, such that

they may fail to attend to cues to impending punishment. The ERN could be readily incorporated into this account.

On a reinforcement learning account of the ERN, it indexes a similar process to that which the RMH posits is disrupted in psychopathy: attention to feedback. Whereas the RMH was formulated to account for unresponsiveness to feedback from the environment, however, the ERN, on a reinforcement learning account, indexes attention to internal feedback that is transmitted from the mesencephalic dopamine system to the anterior cingulate cortex when an error is made (Holroyd & Coles, 2002). The RMH could be adapted to include inattention to internal, as well as external, feedback signals, and thus to incorporate the ERN as an index of attention to (internal) contingencies that might signal a need to adapt behaviour.

If an affective/motivational account of the ERN is adopted, meanwhile, then a link may be drawn to another aspect of the RMH model. In the RMH, when punishment occurs, its aversiveness produces an increase in arousal, which amplifies the speed and intensity of the behavioural response to it. This appears to be an identical construct to the defensive reactivity that the ERN is said to index, on an affective/motivational account of the ERN (Weinberg, Riesel, & Hajcak, 2012). According to the RMH, however, disinhibited individuals (including psychopaths) show an usually strong reaction to aversive events, which causes them to become unusually aroused in response, and to respond more forcefully and urgently in pursuit of reward, rather than pausing to process the significance of the aversive feedback and adjust their behaviour accordingly. This contrasts sharply with the affective/motivational account of the ERN, which states that disinhibited individuals show reduced defensive reactivity, which results in their being less responsive to aversive events. The two accounts, then, rely on the same putative mechanism to

account for psychopathic disregard for punishment (namely defensive reactivity), but give diametrically opposed accounts of how it operates in psychopaths. There is no reason for the two theories not to agree that this mechanism – defensive reactivity – is indexed by the ERN, although clearly some theoretical work would be required to reconcile their opposite accounts of its impact on error monitoring.

Emotional processing deficits

Accounts of a fear deficit in psychopathy have taken various forms, but the one most consistent with current evidence focusses on a deficit in the ability to emotionally anticipate punishment, rather than a lack of responsiveness to it when it takes place (Hare & Quinn, 1971; Lykken, 1957; Veit et al., 2002). This potentially offers an interesting refinement to accounts of the ERN as indexing defensive reactivity, as it requires a distinction between reactivity to the prospect of punishment (which is what an error signals) and reactivity to punishment when it comes. In most conditions in which the ERN has been found to be abnormal, this distinction may not be of much significance, as those individuals who are strongly reactive to actual punishment will usually also be strongly reactive to the prospect of it - the ERN has been linked to high levels of punishment sensitivity, as well as to anxious presentations such as OCD and GAD (Boksem, Tops, Wester, Meijman, & Lorist, 2006; Gehring, Himle, & Nisenson, 2000; LaDouceur, Dahl, Birmaher, Axelson, & Ryan, 2007). In psychopathy, however, fear deficit theories posit a difficulty with emotionally representing in the present a punishment that is anticipated in the future. Fear deficit theories of psychopathy, then, would predict that psychopaths would show reduced ERN due not to a lack of defensive reactivity to actual aversive events, but rather due to reduced defensive reactivity to the anticipation of aversive events, such as when an error has been made.

Testing links between the ERN and reward and punishment processing

Future experimental work could attempt to develop the links sketched here between theories of the ERN and theories of reward and punishment processing in psychopathy. This may be complicated, however, by the availability of more than one plausible account of the ERN, and of reward and punishment processing. For instance, should an experiment designed to test for fear deficits in respect of anticipated punishment in psychopaths find a reduced ERN, then this could be taken to support both the fear deficit account of reward and punishment processing and an affective/motivational account of the ERN. If, however, there was no finding of reduced ERN, this result could still be taken as consistent with a fear deficit, provided a cognitive account of the ERN was adopted. Productive investigation of links between the ERN and reward and punishment processing may, therefore, not be possible until the state of knowledge on one or the other of them is clearer.

Clinical implications

The most important clinical implication of the findings presented here is as to the usefulness of the callous unemotional subgrouping amongst adolescents. The implications arising from the other study findings will be discussed first, however.

The finding of reduced ERN amongst externalising adolescents is too inconclusive to support the use of the ERN as a biomarker for externalising vulnerability in clinical practice, and in any case this study could only have demonstrated it in a sample who have already shown high levels of antisocial behaviour, i.e. in whom the risk of externalising pathology has already been realised. Whilst the presence of reduced ERN amongst this group may help to elucidate the processes underlying their antisocial behaviour, this cannot at present easily inform

the development of interventions for such behaviour, due to ongoing uncertainty as to what processes the ERN reflects.

The same is true of the tentative finding that callousness makes a contribution to reduced ERN amongst externalising adolescents over and above that of externalising itself: in the absence of certainty as to what processes the ERN represents, this finding cannot easily inform clinical practice. An account was developed in this study of ERN deficits among callous young people as being mediated by a lack of empathy, but, even beyond the inconclusivness of the finding on which it is based, this account is somewhat speculative. If it is correct, however, then it may be that the development of empathy should be a particularly important target in interventions for adolescents with CU traits (Kam, Greenberg, & Walls, 2003).

Finally, the study failed to provide strong support for CU traits as delineating a meaningful, and clinically useful, subcategory of externalising adolescents: only one subscale of the ICU was associated with reduced ERN within the externalising group, and this association was due in part, or perhaps wholly, to the variance shared by callousness and externalising. This does not seem sufficient to found clinical approaches that identify a subgroup of externalising young people as being of particular concern. Conversely, in light of the powerful stigma that attaches to the diagnosis of psychopathy, the therapeutic pessimism that surrounds it, and the absence of any established therapeutic interventions to address it, there is perhaps an argument for a presumption against importing it into clinical work with young people unless the evidence for doing so is clear (Edens, Skeem, Cruise, & Cauffman, 2001; Seagrave & Grisso, 2002).

Conclusion

This thesis has sought to extend and refine what is known regarding the processing of rewards, punishments, and errors in individuals prone to externalising. It has done so by examining the processing of rewards and punishments amongst a group noted for their very high levels of problematic antisocial/externalising behaviour – psychopaths – and by extending research on error monitoring, using the ERN, to externalising young people, including those with psychopathic traits. Finally, it has considered the possible links between the ERN and reward and punishment processing in psychopathy, and considered the clinical implications of the study presented here, alongside reflections on the research process. As the processing of reward, punishment, and error in externalising presentations including antisocial behaviour become better understood, it may be hoped that improved clinical assessments and intervention will be developed and offered to individuals with these presentations.

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Appendix A: Information sheet for participants

Information Sheet

The Neurobiological Correlates of Aggression and Empathy in Adolescence.

Version 1.1, 17.02.2014

London Queens Square REC reference Number : 12/LO/0733

We would like to invite you to take part in this research study. However, before you make your decision, we want to make sure you understand why the research is being done, and what your involvement means. Please take some time to read the following information about the study, and talk it through with anyone you wish. If there is anything that you don't understand, or if you would like to ask some more questions, please feel free to contact one of the researchers (contact details can be found at the end of this sheet).

What is the purpose of this study?

This study is looking at how problems with self-control and aggression might be related to activity in the brain. Part of this project involves studying a group of teenagers who have had significant difficulties in these areas, such as breaking the law or repeatedly getting into fights. We also need a group of teenagers that have not had these difficulties so that we can compare them. We are contacting you to be part of this second group of adolescents who have not experienced these difficulties. This study will be looking at brain activity that occurs when young people are dealing with several common situations, like winning or losing, dealing with stress and with situations requiring empathy. This will be done by looking at brain activity and behaviour whilst teenagers play two computer-based games. Ultimately, we hope this project will help us to find better ways of supporting teenagers that get into trouble in the future.

We measure brain activity using a completely safe and harmless net that is worn on your head. This net measures the tiny electrical changes (called EEG) that your brain naturally makes when you are thinking, perceiving, or responding. We are not looking to see if there is anything wrong with you, or to see if there is anything abnormal about your brain activity, and it is not possible for us to determine this. We are only interested in how brain activity relates to behaviour during the games, and comparing this between the two groups of teenagers.

Why have I been invited?

You have been invited because you are a teenager between the ages of 13 and 20.

Do I have to take part?

No, it's up to you. After reading this information sheet, we will go over all the tasks that you will be asked to complete, and you may ask any questions to help you decide whether you would like to participate. If you do, you will be asked to sign a consent form before the session begins. If at any point you want to stop, you can stop without giving us a reason. If you wish your data to be removed from the study upon your withdrawal, then we will do so. Any data that we do store will be kept strictly confidential.

What will I have to do if I take part?

We will invite you to a testing session at the Developmental Neuroscience Unit in the Anna Freud Centre, which is in North London, close to Finchley Road and Swiss Cottage Underground stations.

The study session is around two and a half hours long, and in that time you will complete 2 tasks on a computer whilst having an EEG recording. The first task, called the mirror neuron task, is a computer based reaction time game where you will be copying, or ignoring, hand movements as quickly as you can. This task helps us understand how teenagers are influenced by others and how they understand the actions of other people.

The second game is another reaction time game, which you will play against two other people, where you have a chance to win money. The player who is fastest will receive a small amount of money, and get to decide the punishment for the other player (how much money they lose). Depending on what you (or your opponent) chooses, the punishment will be accompanied by either a relatively loud or a quiet blast of white noise. The loud blast of white noise will not be painful, but it will be slightly uncomfortable. It is not loud enough to do any harm. This task helps us to see how children manage mildly challenging situations and competitive situations.

Finally, between the tasks, you will also be asked to complete a short questionnaire pack about your behaviour and how you get on with other people. This will take roughly 45 minutes to complete.

Description of the EEG recording

While you are doing the computer tasks, you will be wearing an EEG sensor net. The brain gives off small amounts of electricity at all times, and the EEG net lets us monitor and measure changes in these electrical signals, which can indicate changes in brain activity as you think of feel different things. However, you cannot tell *what* you are thinking!

The sensor net is made up of soft sponges sitting in small plastic tubes, which are held in place using an elastic net that stretches over your head. These sponges are placed in contact with your scalp and are what pick up the changes in electrical activity in the brain.

To place the net on you, we will not have to anything to your hair, but we will have to soak the net in a saline (salt water) and shampoo solution. This will help conduct the electrical signals across the scalp, letting us get a good reading of the brain's electrical activity. The whole process should take around 15 minutes.

The EEG itself is very safe and the net that we are using has been approved for safe use with human participants. Given that the net needs to be soaked in saline and shampoo solution before it is applied to your head, you will feel a mild dampness while it is there. Occasionally, some people report a mild itchiness whilst

the solution dries, but this will tends to disappear quickly.

Expenses and Payment

You will receive £30 for coming in and taking part, as well keeping the money you win in the competitive reaction time game. We will also refund your travel expenses, as long as you provide us with a receipt of travel.

What are the disadvantages of taking part?

As far as we can foresee, there shouldn't be any disadvantages from participating this study. The reaction game against another person may involve some mildly unpleasant sounds if you lose, which may be briefly uncomfortable, but will be played at a safe volume and won't be painful.

Will my participation in the study be confidential?

Yes. All the data that we collect will be kept completely anonymous and will only be used for research purposes. We will not store it with your name or any of your contact details, and once you have participated in the study, your data will be given an anonymous identification number and your name and contact details will be deleted. No one will be able to identify you based on the data you give us.

If you decide that you want to be contactable for future studies, your contact information will be stored completely separately from any data we gathered in relation to this study, and will be stored in a secure location (either a locked filing cabinet or a secure server).

Some study documents may also be looked at by authorised representatives from University College London (UCL) Research & Development Unit to check that the study is being carried out correctly. Professional standards of confidentiality will be followed by the authorised representatives. The handling, processing, storage and destruction of data will be in accordance with the UK Data Protection Act 1998.

What will happen to collected data?

All data that we collect during the study will be made anonymous, and will be stored securely, only accessible to the research staff who are working on the

study. Once we have collected all the data, we hope to report our findings in academic journals, and present the findings at conferences. There will be no way of identifying you in any of the reports or publications that result from this study.

If you would like to be informed of what the research team finds from the study, we would be more than happy to contact you with the findings. You will be asked to put your name and contact details on a list of those who would like to be contacted about the results of the study. This will be securely stored and then once the information has been sent to everyone, the list will be destroyed.

What happens if I want to make a complaint?

If you wish to complain, or have any concerns about any aspect of the way you and/or your child have been approached or treated by members of staff due to your participation in the research, National Health Service or UCL complaints mechanisms are available to you. Please ask the researchers if you would like more information on this.

If you still have concerns after you leave, or you wish to make a formal complaint, you may contact the principle investigator, Peter Fonagy, or the UCL Head of the Division of Psychology and Language science, David Shanks, all of whose details can be found at the bottom of this sheet.

Who is funding the research?

The research is being organised and funded by the Anna Freud Centre, a University College London affiliated research centre, and University College London.

Who has reviewed this study?

All research is reviewed by an ethics committee to ensure the protection and proper treatment of all who participate in the study. This study has been reviewed by the London Queen Square REC.

If you have any questions about the study or your participation in the study, please feel free to contact:

Vicki Chow, James Hanley, Michael Eisen

Phone: 020 7443 2240

Email: c.chow.12@ucl.ac.uk j.hanley.12@ucl.ac.uk
michael.eisen.12@ucl.ac.uk

To make a formal complaint, please contact one of the people below:

Professor Peter Fonagy

Phone: 0207 679 1943

Email: P.fonagy@ucl.ac.uk

Professor David Shanks

Phone: 0207 679 7588

Email: d.shanks@ucl.ac.uk

Appendix B – Information sheet (parent/guardian)

The Neurobiological Correlates of Aggression and Empathy in Adolescence.

Version 1.1, 17.02.2014

London Queens Square REC reference Number: 12/LO/0733

We would like to invite your child to take part in this research study. However, before you decide, we want to make sure you both understand why the research is being done and what your child's involvement means. Please take some time to read the following information about the study, and talk it through between the two of you, and anyone else you want. If there is anything that you don't understand, or if you would like to ask some more questions, please feel free to contact one of the researchers (contact details can be found at the end of this sheet).

What is the purpose of this study?

This study is looking at how problems with self-control and aggression might be related to activity in the brain. Part of this project involves studying a group of teenagers who have had significant difficulties in these areas, such as breaking the law or repeatedly getting into fights. We also need to see a group of teenagers that have not had these difficulties so that we can compare them. We are contacting you and your child to be part of this second group of adolescents who have not had these difficulties. The study will be looking at brain activity that occurs when young people are dealing with several common situations, like winning or losing, dealing with stress and with situations requiring empathy. This will be done by looking at brain activity and behaviour while teenagers play two computer-based games. Ultimately, we hope this project will help us to find better ways of supporting teenagers that get into trouble in the future.

We measure brain activity using a completely safe and harmless net that is

worn on your head. This net measures the tiny electrical changes (called EEG) that your brain naturally makes when you are thinking, perceiving, or responding. We are not looking to see if there is anything wrong with your child, or to see if there is anything abnormal about their brain activity, and it would not be possible for us to determine this. We are only interested in how brain activity relates to behaviour during the games, and comparing this between the two groups of teenagers.

Why has my child been invited?

Your child has been invited because they are a teenager between the ages of 13 and 20.

Do they have to take part?

Not at all. Their participation is up to the two of you. After reading this information sheet, we will go over all the tasks that your child will be asked to complete with both of you, and you can ask any questions to help both of you decide whether your child will participate or not. If you are both happy with the answers to your questions and would like to take part in the study, you will be asked to sign a consent form before the session begins. If at any point you or your child wants the session to stop, you can stop it without having to give any reason. If you want your child's data to be removed from the study upon your withdrawal, then we will do so. All your child's answers will be kept completely anonymous and will only be used for research purposes. Any data that we do store will be kept strictly confidential.

What will my child have to do if they take part?

We will invite you and your child to a session at the Developmental Neuroscience Unit in the Anna Freud Centre, which is in North London, close to Finchley Road and Swiss Cottage Tube stations.

The study session is around two and half hours long, and in that time your child will complete 2 tasks on a computer whilst having an EEG recording being taken. The first task is called the mirror neurone task. All that will be required of your child is to copy or ignore the action of a hand on a screen. This task helps us understand how teenagers are influenced by others and how they understand the

actions of other people.

The second task is a reaction time game where they will be playing against two other people, and the first one to press a correct key will get to decide how what kind of punishment the other player will get. Depending on what your child (or their opponent chooses) it will either be a relatively loud or quiet blast of white noise. The loud blast of white noise will not be painful, but it will be slightly uncomfortable. It is not loud enough to do any harm. This task helps us to see how children manage mildly challenging situations and competitive situations.

Between the behavioural tasks, we will also ask your child to complete a short questionnaire pack about their behaviour and how they get on with other people. These should take roughly 45 minutes to complete.

Description of the EEG recording

While they are doing the computer tasks, they will be wearing an EEG sensor net. The brain gives off small amounts of electricity at all times, and the EEG net lets us monitor and measure changes in these electrical signals, which can indicate changes in thoughts or in feelings. However, you cannot tell *what* they are thinking.

The sensor net is made up of soft sponges sitting in plastic tubes, which are held in place using an elastic net that stretches over your child's head. These sponges are placed in contact with your child's scalp and are what pick up the changes in electrical activity in the brain.

To place the net on them, we will not have to do anything to their hair, but we will have to soak the net in a saline (salt water) and shampoo solution. This helps us get a good reading of the brain's electrical activity. The whole process of applying the net should take around 15 minutes.

The EEG itself is very safe and the net that we are using has been approved for safe use with human participants. Given that the net needs to be soaked in a saline and shampoo solution before it is applied to your child's head, they will feel a mild dampness while it is there. Occasionally, some people report a mild itchiness

while the solution dries, but this disappears quickly.

Expenses and Payment

Your child will receive £30 for their participation in this study, as well as the money they win on the second reaction time game. We will also refund both of your travel costs to get here, as long as you provide us with a receipt of travel.

What are the disadvantages of taking part?

As far as we can foresee, there should not be any disadvantages for either of you from participating in this study. The reaction game against another person involves some mildly unpleasant noise if your child loses, which may be briefly uncomfortable, but will be played at a safe volume and will not be painful.

Will my child's part in the study be confidential?

Yes. All the data that we collect will be kept anonymous (stored with just a numerical code) and will only be used for research purposes. All your personally identifying information (e.g. name, address, telephone number) will be kept securely, not passed on to anyone else, and will be kept separate from the rest of the data that we collect as part of the study. Please note however that by law we are required to inform relevant authorities if we were to become extremely concerned about a child's safety. We would always endeavour to talk to you about this before taking any action.

Some study documents may also be looked at by authorised representatives from University College London (UCL) Research & Development Unit to check that the study is being carried out correctly. Professional standards of confidentiality will be followed by the authorised representatives. The handling, processing, storage and destruction of their data will be in accordance with the UK Data Protection Act 1998.

What will happen to collected data?

All data that we collect during the study will be made anonymous, and will be stored securely, only accessible to the research staff that are working on the study. Once we have collected all the data, we hope to report our findings in academic journals, and present the findings at conferences. There will be no way of identifying

either of you in any of the reports or publications that result from this study.

If you, or your child, would look to be informed of what the research team found from the study, we would be more than happy to contact you both with a summary of the findings. You will be asked to put your name and contact details on a list of those who would like to be contacted about the results of the study. This will be securely stored and then once the information has been sent to everyone, the list will be destroyed.

What happens if something goes wrong?

If you wish to complain, or have any concerns about any aspect of the way you and/or your child have been approached or treated by members of staff due to your participation in the research, National Health Service or UCL complaints mechanisms are available to you. Please ask your research doctor if you would like more information on this.

If you still have concerns after you leave, or you wish to make a formal complaint, you may contact the principle investigator, Peter Fonagy or the UCL Head of Division of Psychology and Language science, David Shanks, all of whose details can be found at the bottom of this sheet.

Who is funding the research?

The research is being organised and funded by the Anna Freud centre, a University College London affiliated research centre, and University College London.

Who has reviewed this study?

All research is reviewed by an ethics committee to ensure the protection and well treatment of all people who participate in the study. This study has been reviewed and given a favourable opinion by the London Queens Square REC.

If you have any questions about the study or your child's participation in the study, please feel free to contact:

James Sheffield

UCL Phone: 0207 679 1978

Anna Freud Centre Phone: 0207 443 2240

Email: James.Sheffield.11@ucl.ac.uk

To make a formal complaint, please contact one of the people below:

Professor Peter Fonagy

Phone: 0207 679 1943

Email: P.fonagy@ucl.ac.uk

Professor David Shanks

Phone: 0207 679 7588

Email: d.shanks@ucl.ac.uk

Appendix C – Consent form (participant)

Consent Form – Confidential

Project Title
The Neurobiological Correlates of Aggression and Empathy in Adolescence.

Researcher(s): Prof. Peter Fonagy, Prof. Pasco Fearon, James Sheffield, Chia Chi Chow, James Hanley, Michael Eisen.
Version 1.1, 17.02.2014
REC reference number: 12/LO/0733

Participant Identification number: _____

Please tick the box in front of each statement to indicate consent.

- ☐ I confirm that I have read and understood the information for the above study.
- ☐ I confirm that I have had time to think about and ask any questions about my participation in the above study.
- ☐ I understand that my participation in this study is voluntary and it's completely in my rights to withdraw any at point without needing to give a reason.
- ☐ I agree that the anonymous findings from this study can be used in scientific publications and reports. I understand that my identity will not be revealed, nor will I be identifiable from the data I provide.
- ☐ I agree to take part in the above study.

Please circle Yes or No for the following statements

1. I would like to be contacted in the future about opportunities to participate in research **Yes / No**
2. I would like to be contacted with information regarding the findings of this study **Yes / No**

_____	_____	_____
Participants name	**Participants signature**	**Date**

_____	_____	_____
Researchers name	**Researchers signature**	**Date**

Appendix D – Consent form (parent/guardian)

Caring for young minds
Anna Freud Centre

UCL

Parental Consent Form – Confidential

Project Title

The Neurobiological Correlates of Aggression and Empathy in Adolescence.

Researcher(s): Prof. Peter Fonagy, Prof. Pasco Fearon, James Sheffield, Chia Chi Chow, James Hanley, Michael Eisen.

Version 1.1, 17.02.2014

REC reference number: 12/LO/0733

Participant Identification number: _____

Please tick the box in front of each statement to indicate consent.

- ☐ I confirm that I have read and understood the information sheet provided for the above study.
- ☐ I confirm that I have had time to think about and ask any questions about my child's participation in the above study.
- ☐ I understand that my child's participation in this study is voluntary and it's completely in my and my child's rights to withdraw at any point without needing to give a reason.
- ☐ I agree that the anonymous findings from this study can be used in scientific publications and reports. I understand that my child's identity will not be revealed, nor will they be identifiable from the data they provide.
- ☐ I agree for my child to take part in the above study.

Please circle Yes or No for the following statements

3. It is ok for the researchers to contact me in the future about research opportunities my child could take part in. **Yes / No**

4. I would like to be contacted with information regarding the findings of this study **Yes / No**

_____	_____	_____
Parents name	**Parents signature**	**Date**

_____	_____	_____
Researchers name	**Researchers signature**	**Date**

Appendix E - Statement of contribution to joint research project

I conducted testing sessions, jointly with another researcher, to collect data from around two thirds of the 99 participants who took part in the study.

I took joint responsibility, with one other researcher, for recruitment of the control group used in the study. This entailed identifying and contacting appropriate schools, visiting them to present the study to the students, collecting contact details for the students and/or their parents, and then contacting them to arrange testing sessions.

I conducted all data analyses used in this study, with some assistance from the supervisor of this research.