Assessment of emotional processes and psychopathy among offenders using both behavioural and physiological measures

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Summary

Psychopathy is a personality disorder, the boundaries and content of which lack clarity and consensus. Researchers and clinicians tend to agree on one key aspect: affective hypo-responsivity. Recent evidence suggests that this disposition may be specific to certain features of psychopathy, and that affective deficits may be specific to aversive stimuli. Recognising that existing tests of emotion processing in relation to psychopathy have tended to rely on facial affect recognition, the present work delineated emotion processing into several components which were assessed in relation to the psychopathy dimensions broadly labelled *primary* and *secondary psychopathic traits*. Use of the same pictorial stimulus set allowed for an examination of processing of affective cues in terms of categorisation of affect, physiological response assessed through pupillometry, and influence on behaviour. Emotion experience was assessed through self-report. This approach allowed an examination of whether deficient threat reactivity is consistently found across the different manifestations of emotion. Moreover, by assessing psychopathy in terms of *primary* and *secondary psychopathic traits*, the generalisation of threat deficits across the variants could also be examined. Recognising the existing debate regarding the content of psychopathy, the present work also utilised two alternative measures of the disorder, the Psychopathy Checklist: Screening Version, and the Triarchic Psychopathy Measure. Assessing components of emotion processing in relation to the psychopathy dimensions within a sample of 94 adult male offenders revealed the specificity of threat deficits in relation to PCL:SV Factor 1. Contrary to hypotheses, physiological reactivity to affect was intact for offenders high on *primary psychopathic traits*. For offenders high on *secondary psychopathic traits,* affective responses across the components were intact but an atypical pattern of autonomic activity was found. Assessment of multiple components of affect therefore allowed a more subtle pattern of psychopathic emotion processing to emerge and highlighted the multifaceted nature of psychopathy.

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Chapter 1: Introduction

1.1 Psychopathy

1.1.1 Why study psychopathy

Psychopathy is both a dangerous and an expensive personality disorder whose importance in the criminal justice system has become increasingly recognised (Hakkanen-Nyholm and Hare, 2009). With a prevalence of about 1% in the general population (Hare, 2003), individuals with high levels of psychopathy have a large societal and economic impact. Although most (approximately 80%) criminal offenders would qualify for a diagnosis of Anti-Social Personality Disorder (ASPD; DSM-5, American Psychiatric Association, 2013), relatively few (15 – 20%) would meet the criteria for a diagnosis of psychopathy and yet offenders with psychopathy account for a disproportionate amount of all offences committed (Hare, 2003). Offenders with high levels of psychopathy are charged with more offences, more violent offences, and a greater diversity of offences than other inmates (Hare, 2003). They are more likely than other offenders to fail treatment (Ogloff, Wong and Greenwood, 1990), and a clinical diagnosis of psychopathy is a strong predictor of criminal recidivism (Laurell and Daderman, 2005) and violence (Walsh, Swogger and Kosson, 2009). A better understanding of psychopathy is essential in order to reduce its negative impact.

1.1.2 What is psychopathy

Although there is ongoing disagreement regarding the exact nature and boundaries of psychopathy (Lilienfeld, Watts, Smith, Berg and Latzman, 2015b), psychopathic personality disorder can be characterised by a distinct pattern of affective, interpersonal and behavioural symptoms. Recent evidence supports the presentation of psychopathy as a dimensional construct (Edens, Marcus, Lilienfeld and Poythress, Jr., 2006; Guay, Ruscio, Knight and Hare, 2007). Individuals with many

psychopathic traits typically display interpersonal-affective deficits underscored by a lack of empathy (Brook and Kosson, 2013), as well as poor impulse control and antisocial behaviour (Cima and Raine, 2009). While the broader behavioural aspects of psychopathy overlap with other personality disorders (such as ASPD), personality traits unique to psychopathy such as callous-unemotionality make this disorder distinct from others (Rutter, 2005). One of the principle methods of assessing the disorder is the Psychopathy Checklist (Revised; PCL-R, Hare, 1991, 2003; and Screening Version, PCL:SV; Hart, Cox and Hare, 1995), with established self-report measures including the Triarchic Psychopathy Measure (TriPM; Patrick, 2010). The following section will discuss historical conceptions of psychopathy which led to the main theories of psychopathy, before introducing the approach of the presented work.

1.2 Theoretical Grounding

1.2.1 Historical conceptions

1.2.1.1 Cleckley's Clinical Profile

Cleckley's clinical descriptions and insights on personality (1955) sought to narrow the concept of psychopathy variously described earlier by Pinel, Prichard, Kraeplin and Schneider (Yildirim and Derksen, 2015). Cleckley's finalised *Clinical Profile* of psychopathy was composed of 16 features (see Table 1) which can be grouped into three categories: positive adjustment indicators, behavioural deviance indicators and indicators of emotional unresponsiveness (Patrick, 2006). Cleckley gained his insights primarily through patient contact in a psychiatric hospital; his illustrative case studies often featured well-educated individuals from middle to upper class families.

Table 1.

Cleckley's Clinical Profile of psychopathy (adapted from Cleckley, 1955; Patrick, 2006)

Item category	No.	Description
Positive adjustment	1	Superficial charm and good "intelligence"
	2	Absence of delusions and other signs of irrational thinking
	3	Absence of "nervousness" or psychoneurotic manifestations
	14	Suicide rarely carried out
Emotional-interpersonal deficits	5	Untruthfulness and insincerity
	6	Lack of remorse or shame
	10	General poverty in major affective reactions
	9	Pathologic egocentricity and incapacity for love
	11	Specific loss of insight
	12	Unresponsiveness in general interpersonal relations
Behavioural deviance	7	Inadequately motivated antisocial behaviour
	8	Poor judgement and failure to learn by experience
	4	Unreliability
	13	Fantastic behaviour with drink and sometimes without
	15	Sex life impersonal, trivial, and poorly integrated
	16	Failure to follow any life plan.

Cleckley's psychopath presents with general affective deficits, meaning an absence of emotional depth across both positive and negative affective states (General Poverty in Major Affective Reactions). Indeed, Cleckley queried a connection between a lack of affect and a lack of emotional connection to others, wondering whether emotions can be experienced within the self if emotion has not been experienced in connection to someone outside the self. Psychopathic individuals do not experience the higher emotions of empathy or guilt and have no sense of genuine remorse or responsibility for their actions (Lack of Remorse or Shame), displaying a complete self-centredness (Pathologic Egocentricity and Incapacity for Love). Affections are limited and short-lived and simulations of love are only undertaken when they benefit the self (Pathologic Egocentricity and Incapacity for Love). Cleckley also noted a lack of insight, an inability to understand how others feel

in response to you or to subjectively feel anything comparable in interpersonal interactions, and importantly a lack of awareness of this deficit (Specific Loss of Insight). This exists alongside thoughtless interpersonal relations (Unresponsiveness in General Interpersonal Relations), whereby appreciation and care are only displayed when they will benefit the individual.

In terms of behavioural deviance, Cleckley's psychopath has no sense of responsibility (Unrealibility) and disregards obligations and consequences, but not necessarily consistently – leading to a lack of predictability. Such individuals are routinely dishonest with ease (Untruthfulness and Insincerity) - however Cleckley considered such falsehoods to be simply delivered without over-emphasis or obvious glibness. At other times Cleckley's psychopath admits to responsibility with apparent honesty, indicating an unpredictable individual prone to disarming displays of (shallow) fortitude. Such individuals also engage in a variety of antisocial behaviours, without clear need or purpose (Inadequately Motivated Antisocial Behaviour) and fail to learn from punishment or experience (Poor Judgement and Failure to Learn from Experience). Cleckley noted an intact ability to make objective moral or emotional decisions, both for the individual and for others, that did not translate to real-life decision making; indicating a disconnect between what is understood objectively and what informs (or fails to inform) personal actions.

Several features of Cleckley's *Clinical Profile* indicate an individual with positive adjustment characteristics: a well-adjusted, happy person without signs of affectation or excessive affability (Superficial Charm and Good "Intelligence"), presenting with excellent logical reasoning (Absence of Delusions and Other Signs of Irrational Thinking), and extraordinary poise, immune from anxiety (Absence of "Nervousness" or Psychoneurotic Manifestations). These superficial positive adjustment characteristics mask the affective, interpersonal and behavioural deficits, creating the 'mask of sanity' and allowing individuals with psychopathy to seem normal and even engaging despite the disorder.

Cleckley referred to this analogically as semantic aphasia; reflecting the disconnect between the appearance of typical functioning and the underlying deficits. Cleckley also frequently referred to an inherent semantic dementia, in that individuals with psychopathy fail to extract emotional meaning from events and stimuli. The repeated use of 'semantic' has often lead to a misinterpretation that Cleckley believed the core abnormality to be related to the use of language (Patrick, 2006), whereas Cleckley used 'semantic' to refer to the 'meaning' of things. Debate exists in the literature regarding the relevancy of the positive adjustment features and also the extent to which these features are contained (or should be) within the PCL measures (see Patrick, 2006; Patrick, Fowles and Krueger, 2009; see Hare and Neumann, 2008, for a response), and have recently led to the development of alternative measures of psychopathy (see section 3.3.3.2).

1.2.1.2 Primary and Secondary Psychopathy

Cleckley's *Clinical Profile* presented psychopathy as a configuration of disparate tendencies, but the case studies predominantly presented one variant of psychopathy[1]. By contrast, Karpman's (1948) writings detailed two psychopathic variants: *primary*, and *secondary*. Like Cleckley, Karpman's writings were based on clinical observation. Karpman differentiated *primary*, essential or idiopathic psychopathy from symptomatic or *secondary psychopathy* believing that the underlying etiology, motivations, dynamics, and response to treatment of the two variants differed. In Karpman's view, *primary psychopathy* is underpinned by a heritable affective deficit, whereas *secondary psychopathy* represents an environmentally acquired affective disturbance. The hostile, callous

[1] A construct that many now refer to as *primary psychopathy* (Poythress and Skeem, 2006; Skeem, Polaschek, Patrick and Lilienfeld, 2011).

behaviour of the secondary psychopath is viewed as unresolved negative emotions surrounding early environmental adversity (such as parental rejection or abuse). His behaviour has motivations, which therefore makes him amenable to treatment (Karpman, 1946a). Trait anxiety, the disposition to feel anxious across time and situations, is the key distinction between Karpman's primary and secondary psychopaths. The secondary psychopath for Karpman experienced extreme neuroses (stress and guilt) and intense anxiety stemming from early psychosocial learning. By contrast primary psychopaths are marked by a profound lack of anxiety. Their behaviour lacks discernible motivation, making treatment difficult if not impossible (Karpman, 1946a). Primary psychopaths engage in instrumental, premediated violence in order to gain power and dominance over others. Karpman's (1948) secondary psychopath is more hostile, hot-headed, impulsive, and engages in reactive violence (Karpman, 1948). Lykken (1995) built on Karpman's work and also described primary psychopaths as exhibiting relatively little anxiety, with secondary psychopaths vulnerable to anxiety and other negative emotions.

Research has supported this distinction between *primary* and *secondary psychopathy* in terms of levels of anxiety (Hicks, Markon, Patrick, Krueger and Newman, 2004; Skeem, Johnsson, Andershed, Kerr and Louden, 2007). For example, Hicks et al. (2004), in a sample of highly psychopathic adult offenders (PCL:R Total $\geq$ 30), found two clusters consistent with theoretical descriptions of *primary* and *secondary psychopathy*. The *primary psychopathy* group presented with high social dominance, fearlessness, low anxiety, low harm avoidance and aggression. The *secondary psychopathy* group reported high aggression, reactive hostility, impulsiveness and negative emotions. Secondary psychopaths also reported more childhood and adult fights, greater levels of alcohol misuse, lower socialisation and higher trait anxiety relative to primary psychopaths – yet the two groups did not differ on psychopathic traits as assessed by the PCL:R (Total and Factor 1 scores).

Mealey (1995a, 1995b) has proposed an evolutionary model to account for *primary* and *secondary psychopathy* (what she refers to as sociopathy). Primary psychopaths appear to be genetically disposed to display antisociality, which results in an inborn temperament and pattern of autonomic arousal that together allow the individual to be selectively unresponsive to environmental cues which would otherwise facilitate socialization and moral development (i.e., they are incapable of experiencing social emotions such as guilt or empathy), and actively to seek highly arousing, often deviant, environmental stimuli (Mealey, 1995a). Existence of these individuals reflects genetically based individual differences in the employment of life strategies, such as a cost-benefit approach based on immediate personal outcomes. Primary psychopaths should be predominantly male, stem from all socioeconomic backgrounds, exhibit behavioural propensities that are persistent throughout the lifespan, and be relatively uncommon.

Genetic heritability, for Mealey, plays less of a role in the development of *secondary psychopathy*, whereby individuals are more susceptible to environmental pressures and risk factors (Mealey, 1995a). Their presence in society reflects genetically based individual differences in response to the environment, resulting in differential use by individuals of environmentally contingent strategies. The secondary psychopaths' life strategies will involve frequent, but not necessarily emotionless, cheating. Secondary psychopaths should come predominantly from lower-class backgrounds, be found among both men and women, exhibit patterns of antisocial behaviour that vary across the lifespan, and their prevalence should fluctuate according to societal conditions across time or culture. Recent work has supported the former supposition: in a cluster analysis, Olver, Sewall, Sarty, Lewis and Wong (2015) found that more than half the individuals high on *secondary psychopathy* in their sample were from non-White backgrounds which is likely to have overlap with social disadvantage in a number of respects. Mealey (1995b) however also proposed that *primary*

psychopathy was a type while *secondary psychopathy* was a strategy, an approach inconsistent with more recent etiological work (see section 1.2.2.3).

Karpman (1946b) further described two forms of *primary psychopathy* that shared similar motivations and dynamics but differed in their interactions with others: *aggressive/predatory* and *passive/parasitic* (see also Book, Quinsey and Langford, 2007). These predatory and passive subtypes have recently been validated in a latent class analysis by Mokros, Hare, Neumann, Santilla, Habermeyer and Nitschke (2015; although note that these authors describe the third subtype from this analysis primarily in terms of sociopathy, in line with Mealey (1995a), as opposed to *secondary psychopathy*). Yildirim and Derksen (2015) have also theoretically clarified *primary* and *secondary psychopathy* into *controlled primary psychopathy* and *disinhibited primary psychopathy*, *detached secondary psychopathy* and *unstable secondary psychopathy*, respectively.

1.2.1.3 The Low-Fear and Low-Punishment Hypotheses[2]

Lykken (1995) conceptualised the emotional-reactivity deficit relative to *primary psychopathy* more specifically as a low-fear temperament: the *low-fear hypothesis*. In situations involving both rewards and punishments that are contingent on behaviour, failure of threat anticipation to inhibit approach behaviour produces reward dominant, impulsive behaviour. Low-fear renders these

[2] LeDoux (2014) makes an important distinction between using the term "fear" to describe non-conscious threat detection and defence response mechanisms and "fear" to describe the conscious experience that occurs when an organism is threatened. LeDoux (2014) proposes that the term "fear conditioning" is not useful and should be replaced with another, for example "threat conditioning", while "fear" should be reserved for the conscious labelling of the outcome of threat conditioning reactivity. In the present work, threat conditioning refers to tasks typically described in the psychopathy literature as fear conditioning, and fear refers to the conscious, subjective experience of an emotional state. Where theories and models of psychopathy formally refer to fear (e.g., *low-fear hypothesis*), this term is retained in order to be consistent with the literature.

individuals less responsive than others to the kind of threats that parents or caregivers apply in trying to shape their behaviour and instil prosocial attitudes and internal constraints (i.e., conscience). Using measures of skin conductance, Lykken demonstrated poor threat conditioning in psychopaths (see also Hare and Craigen, 1974; Hare and Quinn, 1971) and a failure to inhibit responses that would be punished. Individuals high on psychopathy presented with low levels of tonic skin conductance when in a 'resting' state, smaller unconditioned skin conductance responses to shock and smaller anticipatory responses to shock (Hare and Craigen, 1974; Hare and Quinn, 1971); psychopaths were said to experience little fear arousal prior to encountering an aversive stimulus. Lykken's (1995) primary and secondary psychopaths were distinguished by differences in extreme temperament, with *primary psychopathy* typified by impaired threat sensitivity and *secondary psychopathy* characterised by impaired reward sensitivity.

The *low-fear hypothesis* was the dominant theory of psychopathy for many years and is closely linked to the *low-punishment hypothesis* (Fowles, 1980). Both hypotheses effectively modelled Gray's theoretical model of arousal onto psychopathy (Gray's Reinforcement Sensitivity Theory, 1970, 1987). Gray (1970, 1987) suggested a behavioural activation system (BAS), a behavioural inhibition system (BIS), and a nonspecific arousal system receiving excitatory inputs from both the BAS and BIS. The BIS is responsible for the inhibition of behaviour in punishment and extinction situations. The BIS can be viewed as the substrate for anxiety as it responds to threat stimuli (punishment cues) by producing anxiety. The BIS acts in opposition to a system which activates behaviour in response to conditioned stimuli for rewards, the BAS. This latter system can be viewed as an appetitive, reward-seeking or approach system which responds to incentives, but also mediates escape and active avoidance behaviour. Gray (1970, 1987) proposed that both the BIS and BAS have input to a non-specific generalised arousal system, indicating that both aversive and appetitive stimuli can be arousal or drive-inducing.

A weak BIS would explain psychophysiological evidence that individuals with *primary psychopathy* display an absence of anxiety in the presence of normally threatening stimuli and an inability to inhibit behaviour in the face of threats of punishment. A weak BIS in this case would result in impulsive, reward driven behaviour. This lead to the development of the *low-punishment hypothesis* (Fowles, 1980), primarily with respect to *primary psychopathy*. Reviewing relevant skin conductance and heart rate studies, Fowles (1980) noted that in terms of skin conductance, psychopaths show poor classical conditioning of threat stimuli, and a smaller increase in skin conductance level and nonspecific fluctuations in anticipation of threat stimuli. The major finding for heart rate was acceleration followed by deceleration in anticipation of a shock or an aversive noise (see also Hare and Craigen, 1974). The overall pattern of skin conductance and heart rate was interpreted as an absence of fear arousal and an adaptive process that minimises emotional impact. A weak or deficient BIS may therefore relate to emotional (lack of anxiety) and behavioural (approach and active avoidance unrestrained by threat of punishment or frustrative non-reward) characteristics (Fowles, 1980).

Secondary psychopathy reflects a combination of high BAS and normal-underactive BIS functioning (Lykken, 1995). Secondary psychopaths may be hypersensitive to reward and their hypersensitivity to reward undermines the processing of BIS information (Baskin-Sommers, Wallace, MacCoon, Curtin and Newman, 2010). This bias appears to be exacerbated under conditions of high task load with the result that they are especially unlikely to process BIS cues and modulate approach behaviour as cognitive demands increase.

1.2.2 Theories of modern psychopathy

1.2.2.1 The Amygdala Dysfunction Hypothesis

The *amygdala dysfunction hypothesis* focuses on the affective poverty associated with psychopathy and emphasises the absence of any guiding force of emotion; resulting in an inability to appropriately process punishment information. In humans, the amygdala is located in the anterior aspect of the temporal lobe and consists of two parts, the basolateral and central nuclei (Blair, 2006). The amygdala has long been implicated in both emotion processing and learning functions (Blair, 2006).

Patrick (1994) first suggested a role for the amygdala in psychopathy. Psychopathy has been associated with underactivity of the amygdala (Birbaumer, Veit, Lotze, Erb, Hermann, Grodd, et al., 2005), smaller amygdala volume, and structural abnormalities (Weber, Habel, Amunts and Schneider, 2008; Yang, Raine, Narr, Colletti and Toga, 2009). However, psychopathy has also been associated with overactivity of the amygdala (Muller, Sommer, Wagner, Lange, Taschler, Roder et al., 2003). Blair (2006) has argued that, as a result of amygdala related dysfunction, psychopaths have an impaired neuronal system for affect representations, with ineffective coding of emotional stimuli. For example, individuals with psychopathy display impaired aversive conditioning (Flor, Birbaumer, Hermann, Ziegler and Patrick, 2002), reduced modulation of the startle reflex by visual threat stimuli (Patrick, Bradley and Lang, 1993) and impaired fearful facial affect categorisation (see Dawel, O' Kearney, McKone and Palermo, 2012, for a recent review).

Blair (2006) focuses on the dysfunction of, and between, the amygdala and areas interconnected with the amygdala (regions of orbital frontal cortex and ventrolateral prefrontal cortex) suggesting an *integrated emotions system* (IES) model that combines the learning (i.e., poor threat

conditioning) and affective deficits of individuals with psychopathic traits. This neurocognitive position is laid out in *The Psychopath: Emotion and The Brain* (R.J.R. Blair, Mitchell and Blair, 2005; see also Blair, 2005, 2006). The specific role of the amygdala is controversial (Bonnet, Comte, Tatu, Millot, Moulin and de Bustos, 2015) but it is considered to be involved in the formation of conditioned stimulus associations, which can be either appetitive or aversive (Blood and Zatorre, 2001). According to the IES, individuals with psychopathy are impaired in the formation of both appetitive and aversive conditioned stimulus associations, with a greater impairment for the latter (Blair, 2006). Blair has clearly emphasised the role of the amygdala in connecting with other regions of the brain; the observed emotional deficits in psychopathy should not be considered equivalent to amygdala lesions in typical individuals (Blair, 2006).

Blair argues that the role of the amygdala is to represent the affective components of stimuli and thus allow emotional learning to occur, particularly in relation to the affective processes involved in socialisation. Emotionally salient stimuli, such as fearful or sad expressions, represent distress cues or social unconditioned stimuli (Blair, 2005). If an individual does not have an empathic reaction to, e.g., threat or distress cues, an associative link will not be formed between the cue and, e.g., the causal transgression. The transgression will never become inhibited and may become the preferred route if it provides a quicker method for achieving goals. Individuals with psychopathy do not form associations to these distress cues, which impedes other-oriented, positive (i.e., prosocial, Eisenberg, 2000) interactions with others (Blair et al., 2005; Blair, 2005). The IES model therefore brings together deficits in social function with those of associative learning. This position is supported by neuroimaging data indicating limbic system abnormalities in relation to psychopathy (see Blair, 2006). Blair (2006) has argued that the emotional dysfunction shown by individuals with psychopathy makes them more likely to learn antisocial strategies to reach goals. Research has indicated that empathic deficiencies, exemplified by psychopathic individuals, contribute to dysfunctional moral development

(Rogstad and Rogers, 2008). This dysfunction, in turn, would lead to their apparent lack of conscience and guilt when repeatedly committing violent crimes (Woodworth and Porter, 2002). Disinhibited behaviour is permitted due to a lack of fear and remorse.

The IES model proposes deficits in both threat and reward related processing, with greater impairments in the former. Some evidence has been found for impaired reward and threat processing in relation to psychopathy (Lorenz and Newman, 2002; Mitchell, Richell, Leonard and Blair, 2006; Williamson, Harpur and Hare, 1991; see Blair, 2013) but studies have also found typical modulation of the startle reflex by appetitive visual stimuli (Levenston, Patrick, Bradley and Lang, 2000; Pastor, Molto, Vila and Lang, 2003; Patrick et al., 1993). Amygdala dysfunction may be selective (Blair, 2006). Recent evidence examining reward processing has linked certain psychopathic traits to hyperactivity, as opposed to underactivity, of the ventral striatum (Buckholtz, Treadway, Cowan, Woodward, Benning, Li et al., 2010), an area of the brain involved in reward expectancy. Blair (2006) has suggested that pathways for reward related processing are likely to be refined in future research with some, but not all, aspects of reward processing impaired in relation to psychopathy.

The *amygdala dysfunction hypothesis* fails to explain why individuals with psychopathy are not impaired at judgements of trustworthiness (Richell, Mitchell, Peschardt, Winston, Leonard, Dolan et al., 2005) or judging complex emotions from the eyes (Richell, Mitchell, Newman, Leonard, Baron-Cohen and Blair, 2003), two social cognitions which appear to require amygdala input (see Blair, 2006). Work by Dawel, McKone, O'Kearney, Sellbom, Irons and Palermo (2015) also found that the relationship between callous-unemotional traits and impaired processing of fearful facial stimuli was best explained by an attentional theory of psychopathy (see section 1.2.2.2). Finally, a recent review paper (Blair, 2013) referred solely to the unitary construct of psychopathy and did not consider differential amygdala activity or structural integrity between the two established variants of

psychopathy (i.e., *primary* or *secondary psychopathy*). To the best of knowledge Blair has yet to explicitly (i.e., beyond footnote or supplementary analysis) apply his IES model to the underlying dimensions of psychopathy.

Recent work by Moul and colleagues (Moul, Killcross and Dadds, 2012) has clarified the *amygdala dysfunction hypothesis* into the *differential amygdala activation model* (DAAM) in light of mixed evidence for amygdala dysfunction in relation to psychopathy (e.g., Birbaumer et al., 2005; Muller et al., 2003). The DAAM model proposes that the basolateral amygdala is underactive, thus impairing reflexive shifts in attention, while the central amygdala is normally or overactive in individuals high on psychopathic traits, implying relatively intact explicit recognition of emotion. The DAAM attempts to refine the IES and more explicitly combine attentional (see section 1.2.2.2) and emotion-based models of psychopathy.

1.2.2.2 The Response Modulation Hypothesis

The *amygdala dysfunction hypothesis* is accompanied by other attempts to explain deficits within psychopathy. The *response modulation hypothesis*, by contrast, states that the core psychopathic deficit is cognitive in nature. Psychopathic individuals are capable of normal emotional responses but have difficulty processing affective information when it is peripheral (secondary) to their primary attentional focus (Newman, Schmitt and Voss, 1997; Glass and Newman, 2009, Zeier, Maxwell and Newman, 2009; Zeier and Newman, 2013; see Smith and Lilienfeld, 2015, for a recent review). Whereas emotion-based models predict weaker responses to affective cues regardless of attentional focus, the *response modulation hypothesis* holds that attentional factors determine the quality of processing of both affective and neutral cues.

Response modulation involves a rapid and involuntary shift of attention from goal directed behaviour to a stimulus which is outside of central attention. Attentional impairments in psychopathy are characterised in terms of deficient response modulation. When a dominant response is set, individuals with psychopathy are unable to accommodate or switch to alternative goals. This narrows the attentional focus, reducing adequate processing of extraneous stimuli, including - but not limited to - threat or punishment stimuli. Therefore, when emotion-related information is within their attentional focus, the *response modulation hypothesis* predicts that psychopathic individuals will be normally responsive (i.e., experience the same increase in arousal) to threat and other emotion-related information. Psychopathy is thus said to be related to an impaired ability to process the meaning of contextual environmental cues when engaged in goal directed behaviour (Newman et al., 1997). The *response modulation hypothesis* has undergone various developments and alterations since its original presentation (see Smith and Lilienfeld, 2015) but has maintained its central hypothesis of impaired integration of top-down (goals) and bottom-up (environment) control of attention.

Two lines of evidence support the *response modulation hypothesis.* First, psychopathic dysregulation is not limited to affective information: there are impairments in processing secondary neutral information also (Hiatt and Newman, 2006; Zeier et al., 2009). For example, individuals high on *primary psychopathy* show less interference of response-incongruent information placed in peripheral attention as compared to controls, but comparable performance under conditions where attention is directed towards both central (target) and peripheral (distractor) locations (Zeier et al., 2009). The specificity of this deficit to individuals high on *primary psychopathy* indicates a tendency for these individuals to maintain a rigid, goal-directed focus of attention. This impairs the ability to consider alternative adaptive responses and to regulate behaviour.

Second, individuals with psychopathy display typical affective reactivity when attention is explicitly directed to the affective cues (e.g., Baskin-Sommers, Curtin and Newman, 2011). In the study of Baskin-Sommers et al. (2011), the eyeblink startle reflex was measured while participants categorised coloured letter stimuli under four experimental conditions that crossed threat versus alternative (i.e., threat-irrelevant) focus of attention with early versus late presentation of the threat-relevant cues. Across these conditions, psychopathy was negatively related to the eyeblink startle reflex in response to threat only in the early-alternative focus condition. There was no evidence for a psychopathy-related deficit in the threat-potentiated startle when the threat-relevant dimension of the stimulus was goal relevant or presented later in the trial sequence. This lead to the development of the *early-attentional bottleneck hypothesis*, an elaboration of the *response modulation hypothesis*. Individuals with psychopathy show minimal interference when early selection allows them to filter out distractors, but normal or greater interference when accuracy requires late selection. Results such as this imply an impairment in cognitive control. For example, a recent study from Newman and colleagues (Zeier, Baskin-Sommers, Newman and Racer, 2012) assessed cognitive control through a flanker task: distractors appeared to either side of a centrally placed target. Participants had to categorise the target as either a letter or a number. Distractors were either congruent or incongruent in content to the target. Psychopathic participants displayed greater interference on incongruent trials as compared to non-psychopathic participants, regardless of level of anxiety (i.e., *primary* or *secondary psychopathy*).

Similarly, in the study of Baskin-Sommers et al. (2011), findings for both the interpersonal-affective and impulsive-antisocial features of psychopathy were very similar to findings for the overall psychopathy score indicating that this early selection effect is a general feature of the psychopathy construct. Individuals with high scores on both interpersonal-affective and impulsive-antisocial features of psychopathy (using both self-report and interview measures of psychopathy) also show less

distractor interference on the Picture-Word Stroop task (Zeier and Newman, 2013), indicating that in this experimental context the *early-attentional bottleneck* related to the broad concept of psychopathy as opposed to the unique variance of either variant. By contrast, a study by Baskin-Sommers, Zeier and Newman (2009) showed that a self-report measure of attention was positively correlated with interpersonal-affective deficits but negatively correlated with impulsive-antisocial behaviours. Notwithstanding the limitations of self-report, this contrasting result implied that the *response modulation hypothesis* was more relevant for individuals high on *primary psychopathy* (see also Zeier and Newman, 2013).

The fact that very few studies assessing the *response modulation hypothesis* examine the psychopathy dimensions makes these conflicting results difficult to resolve. Smith and Lilienfeld (2015) note that, in general, the reliance on total psychopathy scores as opposed to indices of the underlying dimensions to examine response modulation deficits may be problematic in the face of growing evidence of the multidimensionality of psychopathy. Furthermore, the use of extreme groups designs can result in inflated effect sizes (MacCallum, Zhang, Preacher and Rucker, 2002), suggesting that the *response modulation hypothesis* may not explain as much variability as it seems to. Recent studies assessing the *early-attentional bottleneck* have not included a measure of anxiety (e.g., Baskin-Sommers, Curtin, Li and Newman, 2012b; Baskin-Sommers et al., 2011) while others have failed to find an interaction between psychopathy and anxiety (Zeier et al., 2012). Therefore the extent to which the *response modulation hypothesis'* findings are robust across different operationalisations of psychopathy is not yet clear (Smith and Lilienfeld, 2015).

One of the major challenges to the *response modulation hypothesis* lies in that the hypothesis predicts deficits in response to both motivationally neutral and relevant stimuli, but psychopathy is in general related to a reduction in skin conductance activity in response to threat stimuli only (see Lorber,

2004, for a meta-analysis). Furthermore, the *response modulation hypothesis* cannot account for a number of replicated findings in the psychopathy literature that do not require a shift of attention, for example, deficient classical conditioning to aversive stimuli (e.g., Flor et al., 2002).

1.2.2.3 The Dual-Process Model

Extensive empirical work indicating both affective (*low-fear hypothesis*, *amygdala dysfunction hypothesis*) and cognitive (*response modulation hypothesis*) impairments in relation to psychopathy can be said to culminate in the *dual-process model* (referred to as the Two-Process theory, Patrick and Bernat, 2009; or the Dual-Deficit model, Dindo and Fowles, 2011; Fowles and Dindo, 2006; Fowles and Dindo, 2009; referred to cumulatively as the *dual-process model*, see, e.g., Esteller, Poy and Molto, 2016; Schulreich, Pfubiga, Derntl and Sailer, 2013), a etiologic theory of psychopathy focusing on two alternate processes in the emergence of psychopathy.

The *dual-process model* reflects a growing consensus that the source trait of psychopathy is unlikely to arise from a single etiology (Lilienfeld et al., 2015b), and also that psychopathy can be best divided into two underlying dimensions. Patrick and Bernat (2009) refer to these pathways as *trait fearlessness* and *externalising vulnerability*, whereas Dindo and Fowles (2011) refer to these pathways as *low-fear temperament* and *regulatory dyscontrol*. Both pathways link to existing theoretical models of *primary* and *secondary psychopathy* respectively and are thought to reflect etiologic pathways already found in childhood psychopathology (Fowles and Dindo, 2009). The model proposes that separate neural mechanisms contribute differentially to the interpersonal-affective and impulsive-antisocial features of psychopathy representing different temperamental risk factors and distinct etiological pathways to the manifestation of antisocial behaviour (Dindo and Fowles, 2011). Specifically, impaired threat reactivity is suggested to be specific to *trait fearlessness/low-fear*

temperament, whereas impaired cognitive-executive functioning contributes to the development of *externalising vulnerability/regulatory dyscontrol*.

One of the most common methods of assessing psychopathy is Hare's PCL:R (Hare, 1991, 2003) which was originally designed to measure psychopathy as a unitary construct. The finding of two related but separable factors[3] contained within the PCL:R, Factor 1 Interpersonal/Affective deficits and Factor 2 Impulsive/Antisocial Lifestyle and Behaviour, contributed to the development of this *dual-process model*. Based on their review of the literature (see also subsequent sections here), Patrick and Bernat (2009) have suggested that Factor 1 and Factor 2 (and indeed the two factors of the self-report Psychopathy Personality Inventory: Revised, PPI-R, Lilienfeld and Widows, 2005) can be thought of as imperfect, latent manifestations of *trait fearlessness/low-fear temperament* and *externalising vulnerability/regulatory dyscontrol* respectively.

Hare and Neumann (2008) have, however, argued that the role of anxiety and fear in clinical accounts of psychopathy is unclear, and would likely take issue with the explicit labelling of certain psychopathic traits as *trait fearlessness* or *low-fear temperament*. Cleckley's third characteristic of psychopathy, absence of nervousness or psychoneurotic manifestations, has been considered equivalent to low trait anxiety and fearlessness. Although Neumann, Hare and Johansson (2013) used structural equation modelling to demonstrate that low anxiety and fearlessness are broadly captured by the PCL-R and are not specific to Factor 1 traits (Factor 1 does not equate to fearlessness), other

[3]Alternative three factor (Cooke and Michie, 2001) and four-facet (Hare, 2003) models of the PCL-R have also been proposed. The present research limits analysis to the traditional two-factor model. The majority of research using the PCL and its derivatives has used the two-factor model, therefore results will be applicable to as broad a segment of the literature as possible.

publications from these authors (see Hare and Neumann, 2008) state that the more accurate interpretation of Cleckley's work reflects a lack of problematic anxiety, as opposed to a low level of anxiety. Similarly, Neumann et al. (2013) also suggest that psychophysiological data collected during threat conditioning paradigms (see section 1.2.1.3) should be interpreted as reflecting poor impulse inhibition rather than low fear arousal.[4]

As will be discussed, the present work used two measures of psychopathy, the PCL:SV (Hart et al., 1995) and the Triarchic Psychopathy Measure (TriPM, Patrick, 2010) and considered psychopathy results in terms of the two psychopathic dimensions. For ease of communication, the present work considered results from PCL:V Factor 1 and TriPM Boldness/Meanness, and PCL:SV Factor 2 and TriPM Disinhibition/Meanness, alongside one another in two broad dimensions.. However, labelling the first dimension *trait fearlessness* or *low-fear temperament* as suggested by the *dual-process model* would be problematic, as this would imply that indices of this dimension, i.e., PCL:SV Factor 1, are equivalent to fearlessness. An alternative approach, that of referring to the first dimension as interpersonal-affective deficits and the second as impulsive-antisocial behaviours, would equate the TriPM scales with the PCL:SV Factors, which is equally problematic given that the TriPM was designed to operationalise psychopathic features the PCL measures are said to miss. In order to avoid introducing new terms into an already swollen literature, the present work refers to the first dimension as *primary psychopathic traits*, reflected in PCL:SV Factor 1, TriPM Boldness and TriPM

[4] Although recent work from Hare (e.g., Neumann et al., 2013) has moved away from interpreting impaired conditioned threat responses in psychopathy as *low fear* arousal, the original papers on these studies did discuss these findings in such terms. For example, in Hare and Quinn (1971) the authors state "avoidance learning is conceptually related to fear arousal"; and in Hare and Craigen (1974); "psychopaths experience little fear arousal prior to reception of an aversive stimulus" and "small electrodermal responses that precede aversive stimuli reflect the relative absence of fear arousal".

Meanness, and the second dimension as *secondary psychopathic traits*, reflected in PCL:SV Factor 2, TriPM Meanness and TriPM Disinhibition. There are two important caveats in this respect: 1) the labelling of the dimensions as primary and secondary does not imply that one set of traits is more fundamentally psychopathic than the other. Both dimensions are considered essential to the psychopathy construct. 2) The use of such labels does not equate Factor 1/Boldness/Meanness with *primary psychopathy*, nor Factor 2/Disinhibition/Meanness with *secondary psychopathy* as described in section 1.2.1.2. As Neumann et al. (2013) note, the tendency to equate Factor 1 and Factor 2 with *primary* and *secondary psychopathy* is misleading. Again, it is stressed that these labels were chosen to divide the psychopathic traits into broad specifications as suggested by both theoretical models and empirical work, while avoiding explicit labels particularly referencing fear that may be considered problematic by some researchers.

1.2.2.3.1 Primary psychopathic traits

Primary psychopathic traits are suggested to reflect an underlying impairment in emotional responsiveness, particularly in terms of deficient activation of defensive systems in response to threat stimuli, which may be moderated by attention: an early selection bias which restricts the processing of contextual, secondary information (*response modulation hypothesis*; see section 1.2.2.2; Baskin-Sommers, Curtin and Newman, 2015b; Baskin-Sommers and Newman, 2014).

Empirically, *primary psychopathic traits* are indexed by deficits in threat conditioning (e.g., Flor et al., 2002), reduced amygdala reactivity to fearful facial expressions (e.g., Carre, Hyde, Neumann, Viding and Hariri, 2013), reduced threat-potentiated startle (e.g., Patrick et al., 1993) and impaired passive avoidance learning (e.g., Newman and Kosson, 1986); specific to the affective-interpersonal measurement of psychopathy when assessed with the PCL. Affective-interpersonal deficits are also associated with low levels of neuroticism, an arrogant interpersonal style, higher levels

of social dominance and pursuit of physically dangerous, thrilling behaviour (Patrick, 2010); this suggests underlying processes of strong reward-approach motivation combined with weak threat-based inhibitory control (see Dindo and Fowles, 2011). Levels of *primary psychopathic traits* are also associated with enhanced executive functioning (although this may be dependent on the measure used; see, e.g., Baskin-Sommers, Brazil, Ryan, Kohlenberg, Neumann and Newman, 2015a), and associated with enhanced error detection as assessed through event-related potential (ERP) responses (Pasion, Cruz and Barbosa, 2016). This suggests that aspects of this psychopathy dimension are associated with adaptive, successful behaviour (i.e., positive adjustment features; see section 3.3.3.2.1 for a discussion of boldness).

A commonly used physiological index of trait fear is that of the previously mentioned, eyeblink startle reflex potentiation. Threat-potentiated startle reflex, a measure of defensive threat-reactivity, is negatively related to the affective-interpersonal psychopathy factor but does not change as a function of scores on the impulsive-antisocial factor (Baskin-Sommers, Curtin and Newman, 2013; Patrick et al., 1993; Vaidyanathan, Hall, Patrick and Bernat, 2011; although see Vanman, Mejia, Dawson, Schell and Raine, 2003, for an interactive effect of the two factors on startle potentiation). Recent work indicates that impaired threat-potentiated startle may be specific to the positive adjustment features of *primary psychopathic traits*, as opposed to the callous-unemotional traits (at least in undergraduate samples; Esteller et al., 2016).

The visual complexity and attentional demands of aversive stimuli used in startle reflex tasks also moderate the observed effects: individuals high in interpersonal-affective traits showed reduced startle reflex to aversive, visually complex pictures and required more attentional resources to process emotion when pictures were visually complex (Sadeh and Verona, 2012; see also Baskin-Sommers et

al., 2013, for a manipulation of picture familiarity; and Dadds, Perry, Hawes, Merz, Riddell, Haines et al., 2006 for an attention to the eyes manipulation).

1.2.2.3.2 Secondary psychopathic traits

Secondary psychopathic traits, by contrast, describe a regulatory deficit (Fowles and Dindo, 2006). This dimension reflects the combination of impulsive antisocial behaviour and poor emotional control; a difficulty in inhibiting responses once activated (Fowles and Dindo, 2006) and a 'vulnerable narcissism' characterised by neurotic conflicts, stress reactivity, violence and depression (Yildirim and Derksen, 2015).

Secondary psychopathic traits are associated, through criterion-based (Dindo and Fowles, 2011) and empirical (Venables, Hall, Yancey and Patrick, 2015) evidence, with externalising disorders more broadly (Patrick, Hicks, Krueger and Lang, 2005) such as ASPD and substance misuse (Fowles and Dindo, 2011). In contrast to the early attentional deficits associated with *primary psychopathic traits*, the impulsive-antisocial dimension is associated with deficits in dynamic cognitive processes that control behaviour ('executive dysfunction'; Carlson, Thai and McLarnon, 2009; Sadeh and Verona, 2008) particularly in relation to a reduced P300 ERP response (Venables et al., 2015; see Patrick and Bernat, 2009; but see Anderson, Steele, Maurer, Bernat and Kiehl, 2015, for an association between reduced amplitude of the early component of the P300 in response to target stimuli in an oddball task and levels of *primary psychopathic traits* [PCL:SV Facet 1: Interpersonal]). Decreased amplitude of the P300 is thought to reflect a deficit in general cognitive efficiency in individuals high on impulsivity. The impulsive-antisocial factor of psychopathy has a neuropsychological link with the dispositional liability to externalising: the reduction in P300 response appears to underlie a vulnerability common to substance dependency and antisocial syndromes (see Patrick and Bernat, 2009). Levels of *secondary psychopathic traits* have also been associated with decreased amplitude of

the Error-Related Negativity (ERN) ERP response reflecting error detection (Pasion et al., 2016); intact error detection is theoretically linked with adjustment and improvement of behaviour.

Secondary psychopathic traits are associated with increased emotion reactivity in the context of reward (Buckholtz et al., 2010; Endres, Rickert, Bogg, Lucas and Finn, 2011; Martin and Potts, 2004) and other motivationally significant cues (i.e., drug cues, Volkow and Li, 2004). Individuals high on this dimension also display weaker BIS functioning (Baskin-Sommers et al., 2010), often in terms of an over-prioritization of attention toward motivationally salient stimuli (Baskin-Sommers et al., 2012a). For example, individuals high on impulsive-antisocial psychopathy traits showed greater interference on an emotion-word Stroop task to positive words (Sadeh, Spielberg, Heller, Herrington, Engels, Warren et al., 2013).

Results in relation to threat stimuli are mixed: *secondary psychopathic traits* are unrelated to deficits in threat categorisation – although levels of antisocial behaviour have been related to miscategorising neutral faces as angry (Dadds et al., 2006) and greater interference by negative stimuli (Sadeh et al., 2013), suggesting weak control over both behaviour and emotions (Dindo and Fowles, 2011), and possibly a hostility bias (see Dadds et al., 2006). *Secondary psychopathic traits* appear unrelated to the observed reduction in threat-potentiated startle seen in those high on *primary psychopathic traits* (Sadeh and Verona, 2012; Vaidyanathan et al., 2010), but enhanced threat processing has been found specific to focused-attention task conditions in relation to *secondary psychopathic traits* (Baskin-Sommers et al., 2012a).

Secondary psychopathic traits relate positively to measures of stress reactivity, anxiety, neuroticism, depression and suicidality (Douglas, Lilienfeld, Skeem, Poythress, Edens and Patrick, 2008; Hicks and Patrick, 2006; Hyde, Byrd, Votruba-Drzal, Hariri and Manuck, 2014; Patrick, 1994; Verona, Patrick and Joiner, 2001). With regard to stress reactivity, individuals high on impulsive-

antisocial traits of psychopathy showed increased skin conductance responses while delivering a speech focused on their faults (Dindo and Fowles, 2011). Although more focus has been given to the affective-interpersonal traits of psychopathy, accumulating evidence suggests dysfunction in cognitive-executive systems in the affective and behavioural control problems displayed by individuals high on *secondary psychopathic traits* (Venables et al., 2015; see, e.g., Baskin-Sommers and Newman, 2014). Recent work directly testing the *dual-process model* has found differential neurocognitive mechanisms between the two dimensions (e.g., feedback processing; Schulreich et al., 2013). Patrick and colleagues (Skeem et al., 2011) do however emphasise that these dimensional variants are also likely to have areas of overlap and share significant traits (see, e.g., Anderson et al., 2015; Baskin-Sommers et al., 2015a). Patrick's work on the dual processes underlying psychopathy lead to the development of the Triarchic Model of Psychopathy (see section 3.3.3.2).

1.3. Emotion

1.3.1 Why is it important to study emotion in psychopathy

Emotion is a major driver of all human and animal behaviour, particularly social behaviour – it is emotion that literally drives us to seek or escape positive and negative consequences (LeDoux, 2012). Affective deficits and dysregulation are key in psychopathy. In terms of deficits, the clearest is deficient threat responding. A recent review (Brook, Brieman and Kosson, 2013) reported psychopathy to be related to deficits in recognition of threat stimuli most reliably (studies of facial affect categorisation using morphed facial stimuli and tasks integrating facial and vocal affect cues, see section 4.2.1). Individuals with psychopathic traits, particularly those high on *primary psychopathic traits*, exhibit abnormal startle reflex to noise stimuli administered while viewing unpleasant scenes, relative to neutral and pleasant scenes (Patrick et al., 1993). Subjective experiences of fear are also reduced (see Brook et al., 2013, also section 4.2.2).

In terms of *secondary psychopathic traits*, the literature is less extensive but is beginning to establish a pattern of affective dyregulation. Levels of impulsive-antisocial traits are associated with high negative affect (Hicks and Patrick, 2006), increased anxiety (Newman, MacCoon, Vaughn and Sadeh, 2005) and hyper-sensitivity to reward stimuli (Baskin-Sommers et al., 2010). Impulsive-antisocial traits predicted neurochemical and neurophysiological hyper-reactivity of the reward system in response to pharmacological and monetary reinforcers (Buckholtz et al., 2010). Hicks and Patrick (2006) have also found positive associations between anger and impulsive-antisocial traits.

Guilt, a self-conscious moral emotion (Eisenberg, 2000), is also important in relation to psychopathy. Guilt has been utilised as an operationalisation of remorse, a key construct which is emphasised in forensic psychopathy assessments. The developmental psychology definition of guilt is regret over wrongdoings. Guilt often has been operationalised as a response that involves concern for the feelings of others and is associated with self-reported, other-oriented empathic responsiveness (Tangney, 1991). Guilt has also been defined as a focus on an event or behaviour, as opposed to a negative evaluation of the self in response to an event or behaviour (see Spice, Viljoen, Douglas and Hart, 2015). In the psychopathy literature, guilt is ambiguously treated as being similar to remorse (Hare, 2003).

The development of guilt relies on the ability to discern social approval and disapproval (Lazarus, 1991a). Low-levels of guilt have long been associated with psychopathy, particularly *primary psychopathic traits* (e.g., Cleckley, 1955). Guilt stands contrary to items contained within psychopathy measurements which index an inability to take responsibility for wrongdoings. As a strong self-conscious emotion (Eisenberg, 2000) guilt is also contrary to the shallow affect typified by individuals high on *primary psychopathic traits.* Dispositional guilt that is more ruminative and chronic is positively related to psychopathology, including externalising problems (Eisenberg, 2000).

The combination of negative emotionality and low regulation may be particularly problematic in regard to *secondary psychopathic traits.* Extant literature is not conclusive, however. Levels of both *primary* and *secondary psychopathic traits* have been negatively associated with guilt in an adolescent offender sample (Spice et al., 2015) and an adult offender sample (Johnsson, Andersson, Wallinius, Hofvander, Stahlberg, Anckarsater et al, 2014) whereas levels of *primary psychopathic traits* were negatively associated and levels of *secondary psychopathic traits* were not associated with guilt in a community sample (Lyons, 2015; see section 4.5.2).

Other negative emotions, such as sadness, have received less focus in the psychopathy literature; sadness, however, has relevance for prosocial and empathic responding (Blair, 2005), a lack of which may facilitate both callous and antisocial behaviour. In relation to positive emotions, there is not a large amount of evidence indicating consistent impairments in categorisation of appetitive stimuli in relation to psychopathy (see Dawel et al., 2012); heightened reward seeking may be enhanced in relation to psychopathy, particularly *secondary psychopathic traits* (see Buckholtz et al., 2010). A consideration of emotion processing in relation to the psychopathy dimensions would naturally require the inclusion of both positive and negative affect. Additionally, in order to consider the uniqueness of any threat deficits, an alternative negative emotion (in this case, sadness), would also be useful.

1.3.2 What is emotion

In the emotion literature, there is no generally accepted theoretical framework for emotion (Phillips, Dreverts, Rauch and Lane, 2003; Russell, 2003) – ongoing debates question whether different emotions are qualitatively distinct representing basic emotions (Ekman and Cordaro, 2011), the product of cognitive appraisals (Scherer, 2001, 2009), or if emotions can be more accurately described dimensionally in terms of arousal (low – high) and valence (bad – good, Bradley, Codispoti, Cuthbert and Lang, 2001).

Basic emotion models hold emotions to be qualitatively distinct states which can be distinguished from each other, and which also evolved through environmental adaptation (Ekman and Cordaro, 2011) implying an evolutionary/functional view of emotion (Christie and Friedman, 2004). Ekman does not propose that each emotion is a single affective state, but that there are families of related states connected by common factors that differ between emotion families (see Table 2 for a list of the characteristics of basic emotions). Among these characteristics are distinctive universal signals, distinctive physiology, automatic appraisal and distinctive subjective experience. Similarly, the central tenet of James' (1884) model of emotion processing was that 'standard' emotions are made manifest through distinct patterns of autonomic activity ("bodily sensations", page 189) and produce an appropriate response to an environmental demand (see also the *somatic marker hypothesis*, Damasio, 1994, Damasio, Everitt and Bishop, 1996; but see Schachter and Singer, 1962, for a view of undifferentiated autonomic activity in emotion)[5]. As James (page 190) stated: we are afraid because we tremble.

Basic emotion models therefore hold a bottom-up view of emotion, with autonomic and other peripheral activity shaping subjective feeling states and cognitive functioning (James, 1884; Levenson, 2014). Although, in general, there is limited empirical support for the idea of differentiated autonomic activity (Adolphs, 2013), recent work (Kreibig, Wilhelm, Roth and Gross, 2007) has indicated differential physiological response patterns for neutral films and films designed to elicit subjective experiences of fear and sadness. Both types of affective film clips elicited distinct autonomic activation

[5] Scherer (2005) has argued that James' use of the term 'emotion' in his 1884 essay should in fact be replaced with 'feeling', denoting the subjective element of emotion.

patterns as compared to the neutral film, with differences in heart rate, diastolic blood pressure and repiration rate between the fear and sad film clips.

Table 2

Characteristics of basic emotions (from Ekman and Cordaro, 2011, page 365)

Distinctive universal signals
Distinctive physiology
Automatic appraisal
Distinctive universals in antecedent events
Presence in other primates
Capable of quick onset
Can be of brief duration
Unbidden occurrence
Distinctive thoughts, memories and images
Distinctive subjective experience
Refractory period filters information available to what supports the emotion
Target of emotion unconstrained
The emotion can be enacted in either a constructive or destructive fashion.

Ekman has suggested (Ekman and Cordaro, 2011) that the following 7 emotions are universal: anger, fear, surprise, sadness, disgust, contempt and happiness. Guilt includes nearly all of the required characteristics – according to Ekman it is as yet unclear whether guilt has a distinctive signal different from, e.g., sadness. Subcortical structures of the midbrain, striatum and limbic system commonly linked to emotion produce basic emotions which are elaborated by higher-level cognitive processes and develop into finer gradations (Marsh, 2013).

For appraisal theorists, emotions come from evaluations (appraisals; Lazarus, 1984, 1991a, 1991b; Scherer, 2001, 2005, 2009). Emotion is always a response to a meaning or an understanding, but the flow between emotion and cognition is dynamic and recursive (Lazarus, 1991b). Emotions are the result of appraisals, action tendencies (approach or avoid), physiological changes and subjective experiences (Lazarus, 1991b). For Lazarus (1991b), appraisals are both primary – broadly assessing the stimulus in terms of goal relevance, congruence and content – and secondary, related to options

and prospects for coping. In this way, appraisal theories are relational, motivational and cognitive: emotions relate to person-environment relationships that involve harms or benefits, relate to goals, and involve knowledge and appraisal of situations (Lazarus, 1991b).

For example, the Component Process Model of emotion (CPM, Scherer, 2001; 2005; 2009) posits dynamic, recursive emotion processes in response to an event which is *relevant* to an individual's major concerns – their needs, goals and values. The concept of emotion refers to a series of changes in inter-related components (see Table 3); cognitive processes, autonomic changes, motivational aspects, expressive behaviour, action tendencies and subjective feeling states (Scherer, 1984; 2001). Events are subjected to multilevel evaluations, which assess the event in terms of relevance, implications, coping potential and normative significance. This top-down view of emotion places emphasis on these cognitive evaluations, which initiate emotion. Strongly influenced by individual differences, different evaluations of the same event can result in different emotions (Scherer, 2001). The results of these evaluations have a motivational effect, producing action tendencies as well as physiological response patterns and motor expression. These components are centrally integrated and continuously updated (i.e., subject to reappraisal, Scherer, 2001) as events and evaluations change. Parts of this centrally integrated representation may then become conscious and subject to assignment to vague emotion categories, as well as being labelled with emotion words. The fundamental of appraisal theories is that evaluation results drive the response patterning in other components by triggering outputs designed to produce adaptive reactions that are in line with the current evaluation results (often mediated by motivational changes; Scherer, 2009).

Table 3

Relationships between the functions and components of emotion (adapted from Scherer, 2001)

Emotion function	Emotion component
Evaluation of objects and events	Cognitive component (appraisal)
System regulation	Neurophysiological component (bodily symptoms)
Preparation and direction of action	Motivational component (action tendencies)
Communication of reaction and behavioural intention	Motor expression component (facial and vocal expression)
Monitoring of internal state and organism-environment interaction	Subjective feeling component (emotional experience)

In line with basic emotion theorists, some appraisal theorists have also categorised emotions into broad categories. For example, Lazarus (1991b) has categorised emotions as either resulting from harms, losses and threats (negative emotions: anger, anxiety, fear, guilt, shame, sadness, envy, jealousy and disgust), or resulting from benefits (positive emotions deriving from goal attainment or a movement towards such: happiness, joy, pride, gratitude and love). Similarly, Scherer (2005) has distinguished between 'utilitarian' emotions (anger, fear, joy, disgust, sadness, shame and guilt) that facilitate adaptation, such as action tendencies, to relevant events, and aesthetic emotions (awe, wonder, solemnity) that are often elicited in response to art or music.

Dimensional models of emotion represent an alternative approach to conceptualising affect. Such models posit that emotions like fear, anger and happiness can be described as points on core dimensions of arousal and valence. Bradley et al. (2001) present a motivational model, which consists of two systems, to account for these parameters of arousal and valence: a defence (avoidance) system is activated in threat contexts, with a behavioural repertoire built on avoidance and attack. An appetitive (approach) system is activated in survival contexts that promote approach and nurturance. These two systems therefore represent motivational activation: valence judgements indicate which

motivational system is active or should be activated, and judgements of arousal indicate the intensity of motivational activation (Bradley et al., 2001; Bradley, 2009).

Arranged orthogonally, the dimensions of arousal and valence form a circumplex upon which emotions can be plotted and quantitatively compared. When activation of the appetitive and defensive systems are low, arousal is minimal and stimuli are typically rated as neutral or non-emotional suggesting a weak action tendency (Bradley, 2009). As approach or avoid activation increases, reports of arousal also increase. For example, sadness is low in arousal and negative in valence, fear is high arousal and strongly negative. Threat stimuli elicit the greatest evidence of defensive activation as compared to other aversive stimuli, evoking the largest skin conductance responses and the greatest startle reflex potentiation (Bradley et al., 2001). Erotic stimuli also produce the largest skin conductance changes and most inhibited startle reflex as compared to other pleasant stimuli (Bradley et al., 2001). Autonomic systems change at different levels of motivational engagement (Bradley et al., 2001). Further distinctions among emotions are thought to reflect individual differences in evaluations of the events surrounding the basic changes in arousal and valence. The specific emotion an individual experiences (or perceives themselves to experience) may be shaped by interpretations of neurophysiological changes in arousal and valence in light of the eliciting stimulus and the individual's idiosyncratic stores of semantic knowledge, memories, and behavioural responses that shape the subjectively experienced state (Russell, 2003).

The present work does not attempt to provide evidence for any particular emotion model, but rather emphasises that what these different models of emotion highlight is the importance of delineating emotion components. The emotion process, occurring after the initial presentation of an affective stimulus, may therefore be understood in terms of the following components: 1) the appraisal and identification of the emotional significance of the stimulus; 2) the elicitation of various states,

including autonomic, neuroendocrine and somatomotor responses, by the stimulus; as well as 3) conscious emotional feeling (Phillips et al., 2003). For individuals high on the psychopathy dimensions, the key emotion deficit may lie in one or more of these components. Tasks broadly assessing these components may highlight the specificity of emotion deficits when the emotion process is delineated with respect to psychopathy.

1.4 Summary

Although the field of psychopathy research is characterised by disagreements and competing hypotheses (Lilienfeld et al., 2015b), several early and modern theories of psychopathy highlight the faceted nature of the disorder and consider a focus on the underlying variants to be both informative and necessary for the advancement of both research and treatment. *Primary psychopathic traits* have been most usefully illustrated by tests of the *low-fear* and *low-punishment hypotheses*, as well as tests of impaired *response modulation* and theories of genetic dispositions towards fearlessness. *Secondary psychopathic traits* have, more recently, been described in terms of impaired executive function, susceptibility to environmental risk factors and emotional dysregulation.

The key role of *negative emotionality* has emerged across a variety of models and theories (see also section 4.2.2). Cleckley (1955) focused on low emotional distress as one of his markers of psychopathy, detailing features such as suicide rarely carried out, a failure to experience guilt or shame and a lack of anxiety. Cleckley's (1955) *Clinical Profile* also extended this to a more general low trait anxiety, and noted that strong anger reactions are uncharacteristic. Karpman (1941, 1948) suggested a taxonomy of psychopathy distinguished by the presence or absence of negative emotionality. *Primary psychopathy* was characterised by low anxiety and lack of conscience, while *secondary psychopathy* was characterised by heightened emotional distress and depression. Later, Lykken (1995) presented *low-fear* as the primary psychological deficit in psychopathy. Finally, the *dual-process model*

illustrated the lack of defensive responses to threat stimuli specifically in relation to *primary psychopathic traits* and the heightened levels of negative emotionality in relation to *secondary psychopathic traits*.

Emotion perception may be best understood in terms of a dynamic process, occurring following initial presentation of an affective stimulus, which involves the identification of the stimulus's relevance and the elicitation of an affective state in response (Phillips et al., 2003). This affective state will have autonomic, somatomotor and labelled responses, amongst others (Phillips et al., 2003; Scherer, 2001, 2009). The key emotion deficit in relation to psychopathy may lie in one or more of these components. Use of the same stimulus set would allow for an examination of processing of affective cues in terms of categorisation, physiological response and influence on behaviour in relation to the two psychopathy dimensions.

Categorisation of affect, for example categorising threat related stimuli as fearful, includes key processes including knowledge of the concept of fear, the lexical label 'fear' and perception of the fear response in the individual (Adolphs, 2002). Tasks of facial affect categorisation are typically used in psychopathic populations (see section 4.2.1) with deficits in performance taken as indicative of deficient emotionality. However, tasks assessing emotion categorisation in relation to psychopathy typically do not also assess subjective experiences of emotion which indicate diverging relationships with the underlying psychopathy variants (see section 4.2.2), nor has any study (to the best of knowledge) assessed categorisation of and physiological reactivity to the same emotional stimuli. Research combining all three (categorisation of, reactivity to, and subjective experiences of, emotion) may go some way to delineating emotion deficits and dysregulation in relation to the psychopathy dimensions. Tasks assessing the impact of emotional information on behavioural responses may further highlight the specificity of psychopathy-related deficits. The present work assessed the

psychopathy dimensions and affective processing through various components of emotion (categorisation, reactivity, behaviour) and measurement of self-reported emotion experience. In this way, several hypotheses were explored: the specificity of categorising threat stimuli (Affect Categorisation task), the role of *negative* and *positive emotionality* (self-report measure of emotions), emotional modulation of autonomic reactivity (pupil reactivity measure) and use of affective cues (Affective and Semantic Priming tasks).

Chapter 2: Preparatory Work

2.1 Task Development and Progression

At the outset of the PhD, a four task structure was devised based on Phillips et al. (2003). Tasks were to assess categorisation of an affective stimulus, the cognitive (priming) and physiological (pupillometry) responses to an affective stimulus, and the regulation of an emotional state in relation to the psychopathy dimensions. Over the course of piloting in year 1 and 2 of the PhD, an extensive restructuring of the experimental procedure occurred and the final task structure was decided upon. A final stimulus set of happy, sad, fear, and neutral images was decided upon following extensive piloting[6]. The following sections will discuss the stimulus selection procedure, as well as present pilot data from the behavioural (Affect Categorisation, Affective and Semantic Priming) and psychophysiological tasks, in order to demonstrate task efficacy.

2.2 Stimulus Selection Progression: Development of main stimulus set

The majority of studies of emotion have used visual stimuli, most often pictures of expressive human faces or complex images, to elicit emotional responses and reactivity (Sabatinelli, Fortune, Qingyang, Siddiqui, Kraft, Oliver et al., 2011). Studies utilising images have largely used the International Affective Picture System (IAPS; Lang, Bradley and Cuthbert, 2008), a dataset of static images based on a dimensional model of emotion that has been characterised across multiple

[6] 'Fear' images are threat depicting stimuli, in line with the distinction between threat and fear raised in Chapter 1. However, in selecting stimuli for the threat category, pilot participants rated these images as to how 'frightening' they were. In results and figures these images are therefore referred to as fear in order to be consistent with the coding that was used. When discussing results, 'fear' images are referred to as threat stimuli.

populations with respect to both valence and arousal. There is a precedent for using the IAPS to study both discrete emotions and the dimensions of emotions (Mikels, Fredrickson, Larkin, Lindberg, Maglio and Reuter-Lorenz, 2005). Affective images depict emotion-laden scenes that contain a range of content reflecting the variability of both the natural and man-made environment. These images may have more ecological validity compared to highly-edited facial stimuli (typically only the oval face is presented to minimize variation across exemplars; Sabatinelli et al., 2011).

Facial expressions of emotion represent universal, specific social signals, communicating the affective state of the sender and alerting the receiver to relevant events. Affective images communicate differently to the observer; the interaction is more indirect and the affective content requires greater processing (Sabatinelli et al., 2011). Responses to certain types of affective images, such as those containing potentially dangerous animals, thus represent an alternative social cue to facial stimuli. Responses to other types of affective images, such as a pointed gun, may reflect a 'learned' (i.e., non-biological) threat, understood as such through experience and social learning. Relative to facial stimuli, affective images elicit comparable amygdala activity (Sabatinelli et al., 2011), significantly greater neural responses in particular areas of the brain including the anterior cingulate cortex and ventral prefrontal cortex (Hariri, Tessitore, Mattay, Fera and Weinberger, 2002), orbitofrontal cortex, and subsections of the thalamus (Sabatinelli et al., 2011), likely reflecting the more cognitive nature of processing image stimuli. Studies assessing affective image processing are thus distinct from studies assessing facial affect processing. The present research utilised images from the IAPS to assess emotion in relation to the psychopathy dimensions. Images which contained a close up of a face as the dominant feature in a scene were avoided, due to the categorisation advantage for happy over fear faces (see Milders, Sahraie and Logan, 2008).

2.2.1 Year 1 Stimulus Set

Thirty affective (10 happy, 10 sad, 10 fear) and 10 neutral images were chosen from the IAPS[7] (Lang et al., 2008) and constituted the first stimulus set. Images were converted to greyscale due to research highlighting the impact of colour on the pupil irrespective of affective content (as discussed in Snowden, O' Farrell, Burley, Erichsen, Newton and Gray, 2016).

Throughout the behavioural tasks (Affect Categorisation, Affective and Semantic Priming), the test images were backwardly masked in order to restrict awareness. In the general affective literature, backward masking is used to manipulate awareness during the viewing of emotional stimuli. The reliability of backward masking in eliminating visual awareness has been questioned (Bacon-Mace, Mace, Fabre-Thorpe and Thorpe, 2005; Pessoa, Japee and Ungerleider, 2005); particular issues have been raised by studies investigating the utility of a mosaic noise mask versus a neutral face mask (Milders et al., 2008). In the current set of (behavioural) experiments, the use of a backward masking procedure placed emphasis on restricted processing, as opposed to any claim of eliminating visual awareness (i.e., preconscious or subliminal processing). This restriction was intended to reduce floor and ceiling effects, raise task difficulty and make any psychopathy-related individual differences in task performance more apparent. In this context, the exact type of mask used was not a major design or theoretical issue. A visual noise mask, of the same dimensions as the test images, was therefore created from 100 randomly selected and arranged rectangular portions of the 10 neutral images from

[7] IAPS reference numbers of stimuli used: Fear: 1220, 1300, 1301, 1321, 1930, 1932, 6250, 6300, 6370, 9405; Sad: 2800, 3230, 3350, 2730, 2710, 3180, 9040, 9410, 9250, 3266; Happy: 8501, 8161, 2216, 8034, 8200, 8350, 2208, 4599, 8470, 8490; Neutral: 7002, 7004, 7006, 7009, 7010, 7020, 7025, 7030, 7031, 7150.

each iteration of the stimulus set (see also Hoffman, Lipka, Mothes-Lasch, Miltner and Straube, 2012). The use of a scrambled pattern noise mask (as opposed to an intact neutral image) would reduce the risk of perceptual artefacts, based on unintended interactions between the image and the mask (see Hoffman et al., 2012). Backward masking was deemed unnecessary during the collection of pupil data, as the intention here was not to increase task difficulty but rather to allow natural processing of the images to occur.

The IAPS manual includes arousal ratings per image, obtained in norming studies, with arousal reflecting the intensity of motivation system activation; from calm to high activation (Lang et al., 2008). The 30 affective images in the first stimulus set were matched on these normative ratings of arousal and all affective lists had higher arousal ratings than neutral. In discussion with a consultant clinical and forensic psychologist, the decision was made to exclude positive images that were highly sexual. Action and adventure shots were included as happy images as an appropriate high arousal, positive valence alternative.

The set of 30 affective and 10 neutral images was used for piloting conducted during Year 1 of the PhD. This piloting produced conflicting results: In the Affect Categorisation task, participants categorised sad and happy images with significantly reduced discrimination accuracy as compared to neutral and fear images. This potentially indicated that happy and sad images were not considered as exemplars of these respective categories by participants. A by-items analysis was conducted on the Affect Categorisation task data; images were coded as to whether they were correctly categorised, incorrectly categorised and included, correctly categorised but excluded as outliers due to slow response times (RT), or incorrectly categorised and excluded as outliers.

This by-items analysis highlighted a number of problematic images[8] and indicated three potential issues: action versus happy; gist versus detail; and disparate complexities. For happy images, action and adventure shots were not reliably categorised as 'happy' but rather were miscategorised as 'neutral' or 'frightening'. In turn, many of the sad images featured emotional detail (i.e., a woman with a black eye in an otherwise neutral scene) and were miscategorised as 'neutral'. This can be contrasted with the threat images, many of which featured an entirely negative scene (i.e., a close up shot of a snarling dog). Participants may have been unable to use the emotional gist, the global (overall) emotionality (Humphrey, Underwood and Lambert, 2012), to accurately categorise the sad images. Results of this stage of piloting also indicated that the outcome of the Affect Categorisation task was possibly due to a stimulus complexity confound. The neutral images consisted of single item objects often on plain backgrounds. The affective images often featured complex interactional scenes. These neutral images were likely categorised with greater sensitivity than the affective images due to structural as opposed to emotional content, but this complexity confound represented a significant design problem.

2.2.2 Year 1 Ratings Task

To assess the potential confound of complexity, and to better select images that were more appropriate for the affective categories, a ratings task was run. Participants ($N = 7$) were asked to rate a set of 70 images (all the images from the Year 1 stimulus set that were not deemed problematic, plus a set of alternative images considered to address the issues of gist/detail and stimulus complexity).

[8] IAPS reference numbers of images identified as problematic: Fear: 1040, 1931, 6212; Sad: 3220, 6315, 6415, 9050, 9252, 9400, 9570; Happy: 1750, 5621, 5626, 8080, 8160, 8186, 8300, 8341, 8502.

Stimulus selection for the alternative images was also based on data from Mikels et al. (2005). Mikels et al. (2005) present descriptive emotional category data on the IAPS, identifying images that are uniquely categorised as eliciting positive or negative norms: of interest here were images that elicited fear (threat), sadness and positive affect. Mikels et al. (2005) do not present norms for "happiness", as they consider this to be a broad emotional term with diverse meanings that is nonspecific with respect to positive affect; rather they categorise positive images as eliciting amusement, awe, contentment or excitement (Mikels et al., 2005). However, in the present research, participants would be required to categorise the images as either 'happy', 'sad', 'frightening' or 'neutral'; therefore, for the sake of consistency, pilot rating participants were asked to consider the images in terms of how 'happy' they were. For the present research, of interest were images that elicited a state of positive affect as opposed to excitement. Pleasant images were selected from the IAPS (Lang et al., 2008) that reflected the Mikels et al. (2005) norms of amusement, contentment and to a lesser degree excitement. A selection of pleasant images (puppies, kittens) were selected from the internet using a Google Image search for "happy pictures" and included in the rating task.

Pilot participants were asked to rate each of the images on 7 separate scales: how happy, how sad, how fearful, how neutral, how pleasant, how arousing and how complex each image was. Each dimension was rated on a scale of 1 (not at all) to 10 (completely). Participants received the following instructions for the happy, sad and fear scales: 'A value of 1 on the scale says that you think the picture does not show this particular emotion at all, while a value of 10 on the scale says that you think the picture is a very strong example of that particular emotion'. For the neutral scale, the following instructions were given: 'A value of 1 on the scale says that you think the picture is not at all neutral – so it probably shows a picture which is very emotional. A value of 10 on the scale says that you think the picture is completely neutral – it has no emotional content at all'. For the valence scale, participants received the following instructions: 'A value of 1 on the scale says that you think the picture is very

unpleasant, while a value of 10 on the scale says that you think the picture is very pleasant'. For the arousal scale, the following instructions were given: 'Please indicate how calm or exciting/energetic (arousing) you think the picture is. By exciting/energetic, we mean how strongly you feel while you view the picture. So if you think a picture is very frightening or sad, and you feel this emotion quite strongly then this picture would get a higher rating on the calm/arousing scale. If you think a picture is quite neutral with not a lot of emotional content, then this picture would get a lower rating on the calm/arousing scale'. Finally, for the complexity scale, participants received the following instructions: 'Please indicate how simple or complex you think the picture is, in terms of the picture content. By simple, we mean that the picture is quite basic, and has maybe just one thing or person on a plain background in it. By complex, we mean that there is lots going on in the picture – maybe there are several people doing different things with other activities in the background. A rating of 1 on the scale says that you think the picture is very simple in terms of content, while a rating of 10 on the scale says that you think the picture is very complex in terms of content'.

Scores for each scale per image were averaged across participants and then sorted in Microsoft Excel for highest scores on happy/sad/fear/neutral lists. For each image, the mean appropriate affective/neutral categorisation needed to be at least 3 points higher than any other categorisation in order for that stimulus set to be deemed exemplars of that affective category. For example, if, averaged across participants, a stimulus had a mean happy rating of 7, a mean sad rating of 1.5, a mean fear rating of 1, and a mean neutral rating of 3, that image was considered to represent a happy image. Based on these pilot ratings, 10 images per stimulus type were selected.

2.2.3 Year 2 Stimulus Set

Results of the Year 1 ratings task produced the Year 2 stimulus set. This second stimulus set again consisted of 30 affective (10 happy, 10 sad, 10 fear) and 10 neutral images.[9] Three happy images were sourced from Google Images. All lists were matched on rated complexity. The affective lists were matched on IAPS (Lang et al., 2008) ratings of arousal but all significantly different from neutral. All lists were significantly different on normative IAPS (Lang et al., 2008) manual ratings of valence (unpleasantness versus pleasantness) which reflects the dominant motivation system activated (avoidance or approach).

This stimulus set was used to conduct Year 2 piloting. Analysis of the results from this undergraduate sample indicated a problem: happy images did not elicit greater pupillary dilation than neutral images (see section 2.4.2.1.2.2), a pattern in contrast with the literature (e.g., Bradley, Miccoli, Escrig and Lang, 2008). There were thus concerns that the happy images were not sufficiently arousing. It was also noted that, based on pilot ratings, the neutral images now had a slightly greater (non-significant) complexity as compared to the affective images. This complexity might be producing greater cognitive load, producing pupillary dilation. Therefore, a second ratings task was conducted to inform the new choice of neutral images and examine possibly replacing the happy set with more arousing ("adrenaline") images.

[9] IAPS reference numbers for stimuli: Fear: 1220, 1300, 1301, 1321, 1930, 1932, 6370, 6510, 6560, 9405; Sad: 2345.1, 2375.1, 2661, 2800, 3220, 3301, 3350, 6570, 9050, 9410; Happy: 1920, 2208, 4608, 4609, 4623, 4626, 8470; Neutral: 2383, 2745.1, 7036, 7037, 7130, 7140, 7217, 7550, 7595, 7700.

2.2.4 Year 2 Ratings Task

Thirteen images[10] that represented viable alternatives to the neutral and happy images were selected from the IAPS (Lang et al., 2008) for a second identical ratings task with the same (N = 7) pilot participants. The alternative happy images (labelled 'adrenaline') were selected based on examination of an existing study assessing pupil reactivity to affective visual stimuli (Bradley et al., 2008). Bradley et al.'s (2008) pleasant images nearly all represented awe or excitement, therefore the adrenaline images were selected to reflect this content (four adventure images, 5 erotic/romantic images, 1 contentment/affiliative image). The set of adrenaline images was matched on mean IAPS (Lang et al., 2008) arousal ratings to the existing affective sets. Within the adrenaline set, erotic images were included alongside adventure images. Previous work has however indicated that adventure stimuli elicit smaller psychophysiological responses as compared to erotic stimuli (e.g., Weinberg and Hajcak, 2010). This potential limitation is acknowledged. Three alternative neutral images were selected based on simplicity of content and low arousal and valence.

Results of the year 2 ratings task indicated that the adrenaline stimuli were matched on complexity ratings with the other stimulus lists and that the stimuli, particularly the erotic stimuli, were rated as pleasant and happy. The three alternative neutral images were, as expected, rated as low in complexity. These three images were substituted into the existing neutral set, replacing three images

[10] IAPS reference numbers for stimuli assessed in this task: Adrenaline: 4640, 4653, 4659, 4687, 4694, 2260, 8420, 8161, 8280, 8300, 8420, 8280; Neutral: 7004, 7009, 7010.

high in complexity, to create an alternative neutral list (labelled neutral-simple) to be contrasted with the existing neutral list (labelled neutral-complex).

Reliability of the adrenaline and neutral-simple lists was assessed through pupil dilation (see section 2.4.2.2). Results indicated that all affective images (happy, sad, fear, adrenaline[11]) elicited greater dilation as compared to the set of neutral-simple images. The neutral-simple images (referred to from hereon as neutral) were therefore used in the final stimulus set. Adrenaline stimuli elicited a strong physiological response, however the number of highly erotic stimuli was again considered a confound for the offender sample as previously discussed. In previous piloting of the Affect Categorisation task, adventure images were also not reliably categorised as happy as mentioned, despite participants in the current rating task scoring similar images as 'happy'. Therefore, based on these two concerns, the existing set of happy images, featuring a mix of contentment, romance and amusement scenes, which now elicited greater dilation as compared to the improved neutral set, was retained. Means (*M*) and standard deviations (*SD*) of the valence, arousal and complexity ratings per stimulus list in the final stimulus set are presented in Table 4[12].

P values for pairwise comparisons between the lists on the three variables (valence, arousal and complexity) are provided in Table 5. Arousal ratings, based on normative ratings provided by the IAPS (Lang et al., 2008), of the affective images did not differ significantly from one another (all *p*

[11] Adrenaline is not an affective state; this term is used to reflect exciting images.

[12] IAPS reference numbers for stimuli: Fear: 1220, 1300, 1301, 1321, 1930, 1932, 6370, 6510, 6560, 9405; Sad: 2345.1, 2375.1, 2661, 2800, 3220, 3301, 3350, 6570, 9050, 9410; Happy: 1920, 2208, 4608, 4609, 4623, 4626, 8470; Neutral: 2383, 2745.1, 7004, 7009, 7010, 7130, 7140, 7217, 7550, 7595. Three Happy stimuli were sourced online and can be viewed below.

values > .05) but did differ from the neutral images (all *p* values < .001, see Table 5). Stimulus lists were further matched in terms of rated complexity (all *p* values > .05). All lists were significantly different on valence (all *p* values < .05), based on normative ratings provided by the IAPS (Lang et al., 2008). Images were matched for luminance and matched for contrast (defined as the standard deviation of all pixel values: Moulden, Kingdom and Gatley, 1990; all *p* values > .05). Luminance was calculated using Adobe Photoshop Elements 12.0, under the histogram function. The overall luminance across the set of pictures was calculated (*M* = 69) and all images were adjusted to be within this mean (Range = 68 - 69.50). For a point of comparison, the ratings for neutral-complex (original set of neutral images) are also provided (Table 6.e). For information, the ratings for the adrenaline images are also provided (Table 6.f).

Table 4

Descriptive statistics [M (SD)] for each stimulus list

Stimulus List	Valence	Arousal	Complexity
Neutral	4.99 (.30)	2.99 (.73)	3.72 (2.23)
Happy	7.36 (.42)	5.62 (.69)	4.69 (.86)
Sad	2.24 (.66)	5.78 (.63)	4.68 (1.19)
Fear	3.18 (.83)	6.37 (.44)	3.98 (.68)

Note: Valence and arousal ratings were taken from normative ratings provided by the IAPS manual; Lang et al., 2008; complexity ratings were taken from pilot ratings tasks.

Table 5

P values for pairwise comparisons between final stimulus lists

Comparison	Valence	Arousal	Complexity
		p	
Happy-Neutral	0.001	0.001	1.00
Sad-Neutral	0.001	0.001	0.84
Fear - Neutral	0.001	0.001	1.00
Happy-Sad	0.001	1.00	1.00
Happy-Fear	0.001	0.12	1.00
Sad-Fear	0.01	0.25	1.00

Note: Valence and arousal ratings were taken from normative ratings provided by the IAPS manual; Lang et al., 2008; complexity ratings were taken from pilot ratings tasks.

Individual ratings for each image, ordered per stimulus list, are provided in Tables 6a – 6f.

Table 6.a

Happy image list: Mean ratings for each individual image, following pilot ratings tasks[1]

IAPS ID	Happy[2]	Sad[2]	Fear[2]	Neutral[2]	Complexity[2]	IAPS Valence[3]	IAPS Arousal[3]
4626	8.67	1.00	1.00	2.34	5.00	7.6	5.78
alt3[4]	8.50	1.67	1.00	3.50	3.17		
alt4[4]	7.34	1.00	1.00	4.34	4.17		
alt6[4]	8.00	1.00	1.00	3.34	4.17		
1920	8.00	1.17	1.17	5.34	3.84	7.9	4.27
2208	6.83	1.50	1.50	4.17	6.17	7.35	5.68
4623	8.50	1.00	1.00	3.00	5.25	7.13	5.44
8470	7.67	1.00	1.17	3.00	3.84	7.74	6.14
4608	7.50	1.50	1.50	2.50	5.00	7.07	6.47
4609	7.50	1.00	1.00	4.25	4.75	6.71	5.54

Note: [1] Mean ratings were averaged across participants. [2] How Happy, How Sad, How Fearful, How Neutral and How Complex ratings were completed on a 1-10 scale with 1 meaning "Not at all" and 10 meaning "Extremely". [3] IAPS Valence and IAPS Arousal ratings were taken from normative ratings provided by the IAPS manual (Lang et al., 2008). [4] Three images were sourced online and so have no corresponding IAPS ratings.

Table 6.b

Sad image list: Mean ratings for each individual image, following pilot ratings tasks[1]

IAPS ID	Happy[2]	Sad[2]	Fear[2]	Neutral[2]	Complexity[2]	IAPS Valence[3]	IAPS Arousal[3]
2345.1	2.00	8.00	5.00	2.50	4.00	2.26	5.50
2800	1.00	8.84	2.84	3.00	2.67	1.78	5.49
3350	3.34	6.84	3.00	1.84	6.84	1.88	5.72
2375.1	2.25	6.25	3.00	4.00	4.00	2.20	4.88
2661	1.50	8.25	3.00	2.00	4.75	3.90	5.76
9410	2.25	7.25	2.25	3.75	5.50	1.51	7.07
3301	2.00	6.50	4.75	2.00	4.75	1.80	5.21
6570	1.50	8.50	7.00	1.50	3.50	2.19	6.24
3220	2.25	7.25	2.25	3.75	5.50	2.49	5.52
9050	1.00	8.50	4.00	1.84	5.25	2.43	6.36

Note: [1] Mean ratings were averaged across participants. [2] How Happy, How Sad, How Fearful, How Neutral and How Complex ratings were completed on a 1-10 scale with 1 meaning “Not at all” and 10 meaning “Extremely”. [3] IAPS Valence and IAPS Arousal ratings were taken from normative ratings provided by the IAPS manual (Lang et al., 2008).

Table 6.c

Fear image list: Mean ratings for each individual image, following pilot ratings tasks[1]

IAPS ID	Happy[2]	Sad[2]	Fear[2]	Neutral[2]	Complexity[2]	IAPS Valence[3]	IAPS Arousal[3]
6510	2.00	4.00	9.00	1.00	3.50	2.46	6.96
6560	1.34	4.00	7.84	3.00	4.50	2.16	6.53
6370	1.17	2.50	7.84	1.67	3.34	2.70	6.44
1220	1.34	2.50	6.17	2.00	5.17	3.47	5.57
1301	1.00	3.00	7.17	3.17	3.34	3.70	5.77
1930	1.34	2.34	7.00	2.17	3.67	3.79	6.42
1321	1.50	2.17	6.67	2.00	3.34	4.32	6.64
9405	1.00	4.67	6.17	1.00	4.84	1.83	6.08
1300	1.00	3.67	7.34	1.67	3.84	3.55	6.79
1932	1.50	2.50	6.17	3.34	4.34	3.85	6.47

Note: [1] Mean ratings were averaged across participants. [2] How Happy, How Sad, How Fearful, How Neutral and How Complex ratings were completed on a 1-10 scale with 1 meaning "Not at all" and 10 meaning "Extremely". [3] IAPS Valence and IAPS Arousal ratings were taken from normative ratings provided by the IAPS manual (Lang et al., 2008).

Table 6.d

Neutral image list: Mean ratings for each individual image, following pilot ratings tasks[1]

IAPS ID	Happy[2]	Sad[2]	Fear[2]	Neutral[2]	Complexity[2]	IAPS Valence[3]	IAPS Arousal[3]
7217	1.67	1.17	1.00	9.17	2.84	4.82	2.43
2745.1	2.34	1.34	1.17	8.84	7.50	5.31	3.26
7550	2.67	1.50	1.00	8.84	5.84	5.27	3.95
7010	5.00	1.00	1.00	10.00	1.50	4.94	1.76
7130	3.00	1.67	1.67	8.50	4.00	4.77	3.35
2383	2.17	1.34	1.00	8.50	4.67	4.72	3.41
7595	1.84	1.17	1.34	8.34	6.00	4.55	3.77
7140	2.50	2.17	1.00	8.34	2.34	5.50	2.92
7004	5.00	1.00	1.00	10.00	1.50	5.04	2.00
7009	5.00	1.00	1.00	10.00	1.00	4.93	3.01

Note: [1] Mean ratings were averaged across participants. [2] How Happy, How Sad, How Fearful, How Neutral and How Complex ratings were completed on a 1-10 scale with 1 meaning "Not at all" and 10 meaning "Extremely". [3] IAPS Valence and IAPS Arousal ratings were taken from normative ratings provided by the IAPS manual (Lang et al., 2008).

Table 6.e

Neutral-Complex image list: Mean ratings for each individual image, following pilot ratings tasks[1]

IAPS ID	Happy[2]	Sad[2]	Fear[2]	Neutral[2]	Complexity[2]	IAPS Valence[3]	IAPS Arousal[3]
7217	1.67	1.17	1.00	9.17	2.84	4.82	2.43
2745.1	2.34	1.34	1.17	8.84	7.50	5.31	3.26
7550	2.67	1.50	1.00	8.84	5.84	5.27	3.95
7700	1.50	1.50	1.00	8.84	7.17	4.25	2.95
7130	3.00	1.67	1.67	8.50	4.00	4.77	3.35
2383	2.17	1.34	1.00	8.50	4.67	4.72	3.41
7595	1.84	1.17	1.34	8.34	6.00	4.55	3.77
7140	2.50	2.17	1.00	8.34	2.34	5.5	2.92
7037	1.84	1.17	1.00	8.17	5.67	4.81	3.71
7036	2.34	1.67	1.34	8.00	8.34	4.88	3.32

Note: [1] Mean ratings were averaged across participants. [2] How Happy, How Sad, How Fearful, How Neutral and How Complex ratings were completed on a 1-10 scale with 1 meaning "Not at all" and 10 meaning "Extremely". [3] IAPS Valence and IAPS Arousal ratings were taken from normative ratings provided by the IAPS manual (Lang et al., 2008).

Table 6.f

Adrenaline image list: Mean ratings for each individual image following pilot ratings tasks[1]

IAPS ID	Happy[2]	Sad[2]	Fear[2]	Neutral[2]	Complexity[2]	IAPS Valence[3]	IAPS Arousal[3]
2260	6.67	1.67	1.00	4.34	5.67	8.06	4.26
4640	6.34	1.00	1.00	2.00	6.00	7.18	5.52
4653	7.00	1.00	1.00	2.00	4.67	6.56	5.83
4659	6.00	1.00	1.00	1.67	5.67	6.87	6.93
4687	6.34	1.00	1.34	1.67	5.00	6.87	6.51
4694	7.00	1.00	1.00	1.67	6.00	6.69	6.42
8161	5.34	1.00	3.00	5.67	4.00	6.71	6.09
8280	4.00	1.34	1.34	7.34	4.00	6.38	5.05
8300	6.00	2.34	3.34	3.67	6.34	7.02	6.14
8420	6.67	1.00	1.67	4.67	7.00	7.76	5.56

Note: [1] Mean ratings were averaged across participants. [2] How Happy, How Sad, How Fearful, How Neutral and How Complex ratings were completed on a 1-10 scale with 1 meaning “Not at all” and 10 meaning “Extremely”. [3] IAPS Valence and IAPS Arousal ratings were taken from normative ratings provided by the IAPS manual (Lang et al., 2008).

Happy images sourced online (Figures 1 -3).

Figure 1. Happy image sourced online, labelled Alt 3

Figure 2. Happy image sourced online, labelled Alt 4

Figure 3. Happy image sourced online, labelled Alt 6

2.3 Affective Priming Targets

In the Affective Priming task, test trials consisted of image primes and target words. Targets for the Affective Priming task were refined across the course of year 1 and year 2. A set of 10 positive and 10 negative adjectives were selected from the Affective Norms for English Words (ANEW, Bradley and Lang, 2010) database[13]. Words were selected so that average word length, frequency and arousal (using ANEW normative ratings) were equivalent across word groups (all *p* values > .05; see Table 7). Length was measured in number of letters per word and words were rated for arousal and valence on a 9 point scale with 1 being negative valence/low arousal and 9 being positive valence/high arousal.

Table 7

Word length, arousal, frequency and valence [(M, SD)] for positive and negative targets: Affective Priming task, original set

	Positive Targets	Negative Targets
Word Length	8.0 (1.63)	7.50 (1.72)
Arousal	6.25 (0.86)	6.27 (0.69)
Frequency	27.40 (30.67)	21.67 (15.84)
Valence	8.14 (1.32)	2.65 (1.50)

Note: Arousal, frequency and valence ratings are based on normative ratings provided by the ANEW manual (Bradley and Lang, 2010).

[13] Original negative targets: afraid, anxious, defeated, despairing, distressed, helpless, miserable, nervous, panic, scared; Original positive targets: confident, delight, enjoyment, ecstasy, excitement, friendly, happy, joyful, terrific, triumphant.

Following piloting, targets were reviewed in light of the offender population. Discussions with a consultant clinical and forensic psychologist led to age of acquisition (Kuperman, Stadthagen-Gonzalez and Brysbaert, 2012) being considered more relevant to an offender sample than frequency. Given the low levels of educational attainment and reading age within offender populations (Devitt, 2011), it was essential to choose target words which the offenders would have in their general vocabulary and would not struggle to recognise during brief stimuli presentations. Impaired linguistic recognition would be an additional confound to the priming task; this was dealt with by setting a relatively low age of acquisition threshold of approximately 6 years.

The final set of targets was comprised of 10 positive and 10 negative adjectives, selected from the ANEW (Bradley and Lang, 2010) database.[14] Affective lists were measured for average word length, valence, arousal and age of acquisition (Kuperman et al., 2012; means and standard deviations presented in Table 8).

As seen in Table 8, the positive word list was not significantly different from the negative word list in word length, arousal (ANEW arousal ratings; Bradley and Lang, 2010) and age of acquisition (Kuperman et al., 2012; all p values > .05). Positive and negative lists were

[14] Finalised positive targets: beautiful, brave, cheerful, friendly, handsome, happy, joyful, merry, relaxed, terrific. Finalised negative targets: afraid, awful, boring, disgusted, dreadful, lonely, nervous, upset, useless, scared

Positive targets removed: confident, delight, enjoyment, ecstasy, excitement, triumphant. Positive targets added: beautiful, brave, cheerful, merry, handsome, relaxed. Positive targets maintained: friendly, happy, joyful, terrific

Negative targets removed: anxious, despairing, distressed, defeated, helpless, panic, miserable. Negative targets added: useless, upset, boring, dreadful, awful, lonely, disgusted. Negative targets maintained: afraid, nervous, scared.

significantly different on normative ANEW (Bradley and Lang, 2010) ratings of valence ($p <$ .001).

Table 8

Arousal, valence, word length and Age of Acquisition ratings for target words [M (SD)] used in each priming task: final version

Target type	Arousal	Valence	Word Length	Age of Acquisition
Affective Priming task				
Positive	5.67 (1.15)	7.87 (.45)	7.00 (1.61)	5.98 (1.41)
Negative	5.46 (1.28)	2.51 (.48)	6.50 (1.20)	5.92 (1.19)
Semantic Priming task				
Semantic				
People	5.58 (.74)	4.92 (2.43)	4.90 (.74)	6.32 (1.36)
Animals	4.88 (1.13)	4.88 (1.61)	5.00 (1.70)	5.21 (1.15)
Affective				
Positive	4.91 (.98)	6.74 (.55)	4.60 (1.07)	5.45 (1.19)
Negative	5.55 (.95)	3.06 (.70)	5.30 (1.42)	6.07 (1.49)

Note: Arousal and valence ratings are based on normative ratings provided by the ANEW manual (Bradley and Lang, 2010); Age of Acquisition ratings are provided by Kuperman et al. (2012).

2.4 Final Task Structure and Undergraduate Results

The following task structure was finalised:

- Affect Categorisation task
- Physiological reactivity
- Affective Priming task
- Semantic Priming task

2.4.1 Affect Categorisation Task

Piloting of this task was undertaken in order to create an Affect Categorisation task, with briefly presented images, without floor and ceiling effects. For brevity, and because apart

from the change to stimulus selection no design changes occurred over these rounds of piloting, only the final instance of undergraduate data collection prior to collection of forensic data will be presented for this task.

2.4.1.1 Method

2.4.1.1.1 Participants

A sample of 16 university students (3 male, *M* age = 19.13, *SD* = .81, Range 18-20) took part in the Affect Categorisation task (Year 3 data collection).

2.4.1.1.2. Materials, Design and Procedure

Images were presented using Direct RT software in the centre of the monitor, against the light grey background of a 14" Syncmaster 171N monitor (75Hz) which was connected to a hard drive (identical materials for other task piloting). The task was a repeated measures design. Image content (neutral, happy, sad or fear) was manipulated and image categorisation was measured in the form of a sensitivity index. As a preferred alternative to percentage correct, this sensitivity score took into account response biases. Hit rates and false alarms were calculated to create an index of discrimination sensitivity. For each emotion discrimination score, hit rate was defined as images correctly categorised, e.g., happy images categorised as 'happy'. False alarm rate was defined as all other images incorrectly categorised as that particular emotion, e.g., neutral, sad and fear images incorrectly categorised as 'happy'. Discrimination for each stimulus was calculated as the proportion of correctly identified, e.g., happy, images minus the proportion of all remaining images misidentified as, e.g., 'happy' (i.e., hits – false alarms; see Doerksen and Shimamura, 2001; Snodgrass and Corwin, 1988). This sensitivity score was calculated on a scale of 0 – 1, with a score of 1 indicating perfect

categorisation sensitivity – the ability to correctly discriminate, e.g., happy images from, e.g., sad images. A discrimination task was preferable to a detection task, i.e., detecting whether an image was affective or neutral, in order to assess sensitivity for specific affective categories.

Participants were provided with on screen instructions prior to the task, which were reiterated by the experimenter as well as a visual prompt (laminate) for response options. Participants were instructed to categorise the emotional content of each observed image through a forced choice identification of emotion: happy, neutral, sad or frightening. Instructions required participants to press Z for happy, C for neutral, B for sad or M for frightening[15].

The task consisted of two phases, the practice phase and the test phase. Practice and test trials were identical in structure (see Figure 4). Practice trials utilised different images to avoid familiarity with test images and data from practice trials were not included in analyses. Each trial started with a fixation point 'o' (100ms). Each image was then presented for 100 ms, followed by the noise mask for 100 ms. A brief (100 ms) stimulus presentation created stringent task requirements removing the likelihood of observing floor and ceiling effects. Of the 22 studies covered in a recent meta-analysis (Dawel et al., 2012) of categorisation deficits for facial expressions in relation to psychopathy, in both adult and child/adolescent samples, only one study (Book et al., 2007) utilised a stimulus exposure of less than one second (duration of

[15] Threat images were categorised as 'frightening' as this label was more appropriate than 'fear' as a response option. Response keys were not counterbalanced as evidence that responding with the right as opposed to the left hand increases affect categorisation deficits in psychopathy is lacking (Wilson, Juodis and Porter, 2011).

100 ms). The stimulus presentation rate of 100 ms in the present work was chosen to make the image difficult to see, but still visible, thus avoiding any possibility of subliminal presentations (Milders et al., 2008) which was not of interest. Research indicates that a large amount of meaningful information can be extracted from a single glance at a scene (Bacon-Mace et al., 2005). In line with this, it was not deemed necessary to record subjective confidence ratings of responses. However, natural eye movements still warranted consideration. Saccades are limited under brief presentation conditions (e.g., 150ms) when a fixation point is continuously visible (Doyle and Snowden, 2001; Fischer and Weber, 1993). Thus, stimulus presentation was necessarily briefer than this window in order to remove the possibility of potential eye movements which could obscure perception.

Immediately following the noise mask four forced-choice response options were displayed across the centre of the screen for 1500 ms: happy, neutral, sad, or frightening. The response screen was cleared following this 1500 ms response window but the response was still recorded if received outside this time. The next trial began following an inter-trial interval (ITI) of 1000 ms. The task consisted of a practice phase of 8 trials and a test phase of 160 randomised trials (four presentations of each test stimulus).

Undergraduate participants read the information sheet before giving consent and providing basic demographic details (age and gender). The study was introduced and participants were informed that they could take a break at any stage, or if they found any of the test stimuli distressing. The experimenter remained in the experimental room during the practice phase only to ensure task familiarity. The task took approximately 5 minutes.

Following completion, participants were fully debriefed with the opportunity to ask more questions and received £6 for their participation in the full task battery.[16]

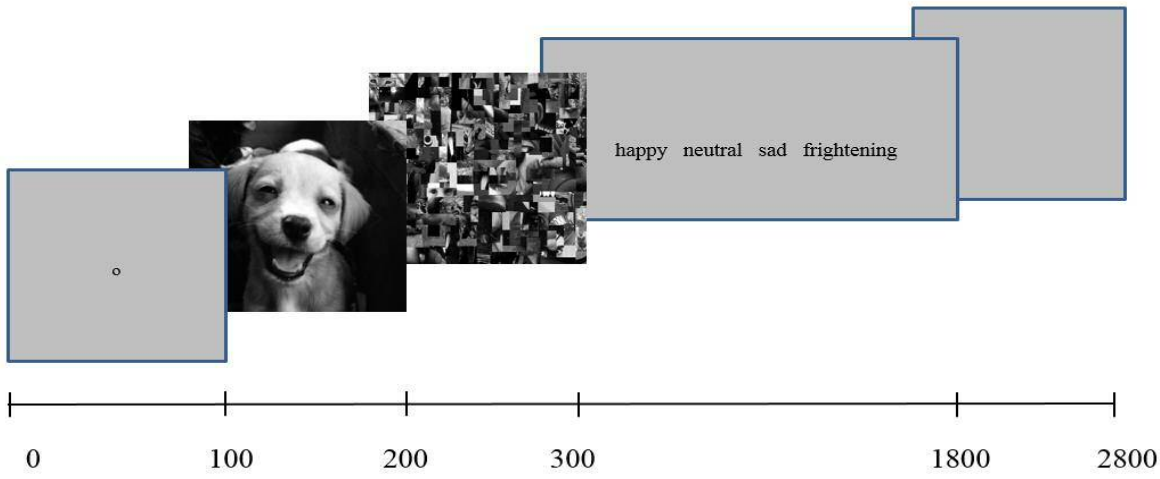


Figure 4. Affect Categorisation task: Finalised trial structure

2.4.1.2 Results and Discussion

Outliers (responses recorded under 300 ms and after 3000 ms) were excluded from the analysis resulting in 2.27% of data removed. Figure 5 shows the pattern of results for the sample. A repeated measures analysis of variance (ANOVA) showed a significant effect of stimulus affective content on sensitivity when categorising the images ($F(3, 45) = 14.24$, $p < .001$, $\eta_G^2 = .35$). Planned comparisons indicated greater sensitivity when categorising neutral as compared to sad ($t(15) = 5.64$, $p < .001$, $d_z = 1.41$) or fear ($t(15) = 3.25$, $p < .01$, $d_z = 0.81$) images. There was no difference in discrimination sensitivity between neutral and happy

[16] This experimental procedure - informed consent, demographic details, supervised practice trials and debrief - was identical across piloting sessions.

images ($t(15) = 1.07$, $p > .05$). Exploratory analysis on the affective categories using paired sample *t*-tests indicated greater sensitivity when categorising happy as compared to sad ($t(15) = 5.53$, $p < .001$, $d_z = 1.38$) and fear images ($t(15) = 2.11$, $p < .05$, $d_z = 0.53$). There was also greater sensitivity for fear as compared to sad images ($t(15) = 2.81$, $p < .01$, $d_z = 0.70$).

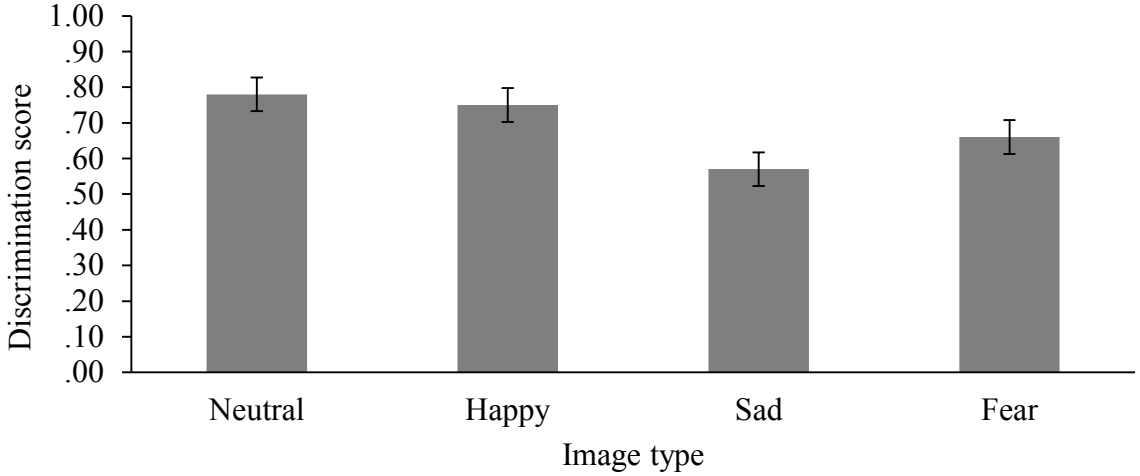


Figure 5. Discrimination indices representing mean categorisation sensitivity per stimulus type based on year 3 stimulus set sampling. Error bars display 2 *SE* (correction for repeated measures designs, Cousinea (2005) and Morey (2008)).

A consideration of these results is important considering that this paradigm was utilised for testing in the offender sample. Discrimination sensitivity for all three affective categories did not show an advantage as compared to neutral images. The results, therefore, did not clearly adhere to the emotionality hypothesis (see Calvo, Avero and Lundqvist, 2006; Humphrey et al., 2012; Lang, Greenwald, Bradley and Hamm, 1993), whereby affective stimuli or events are more salient than non-affective, neutral stimuli or events. The results also did not provide support for the negativity hypothesis (see Calvo et al., 2006), whereby negative stimuli attract attention due to their biological salience. Comparisons did not provide support for the more specific threat hypothesis (Ohman, Lundqvist and Esteves, 2001) which suggests that stimuli

and events that are associated with danger and threat, such as the threat images in the present work, are more salient and therefore may be more easily recognisable. Given that the neutral images tended to not contain people, while the affective images tended to contain either people or animals as subjects, it was likely that this went some way toward explaining the ease of neutral classification.

Between the affective categories, happy stimuli were better discriminated than the sad/threat images. Recent work (Becker, 2012) has identified that, under brief presentation rates (60 ms), participants were better at identifying positive as compared to negative images when identification was assessed through a recognition memory task or through an immediate identification of two simultaneously presented images. The results suggest that additional time is needed to categorise negative images. Image confounds such as complexity were also not responsible for this positivity advantage (Becker, 2012).

The pattern of results could also suggest that when categorising briefly presented stimuli, more false alarms are created when assigning an affective category label to target stimuli, e.g., sad images, which share properties with distractor stimuli, e.g., threat images. The affective similarity of sad and threat images in the present work may have resulted in affective interference (see Nummenmaa and Calvo, 2015). The comparative affective uniqueness of happy images may have facilitated their discrimination as they were the only clearly pleasant image category. Discrimination sensitivity was, however, above chance for all classes of image with no observable floor or ceiling effects.

2.4.2 Physiological reactivity

Extensive piloting of the pupil paradigm was undertaken, which greatly influenced the stimulus selection. Thus this section presents undergraduate data collection completed during year 2, with a discussion of the issues that led to the undergraduate data collection completed prior to collection of forensic data. Piloting of the pupil task aimed to create a paradigm whereby affective images elicited different pupil reactivity as compared to neutral images.

2.4.2.1. Year 2 data collection

2.4.2.1.1. Method

2.4.2.1.1.1. Participants

A sample of 36 university students (11 male, *M* age = 22.00, *SD* = 2.86, Range = 18-29) took part.

2.4.2.1.1.2. Materials

This round of piloting utilised the Year 2 stimulus set (see section 2.2.3). A greyscale image with the mean luminosity computed across all images comprised the background screen for fixation, baseline and response window screens. Stimuli were presented using E-Prime Professional software and pupil diameter was assessed by the Tobii X2-60 Hz eye system; see section 2.4.1.1.2 for further details of hardware.

2.4.2.1.1.3. Design

The task was a repeated measures design assessing the impact of image content (neutral, happy, sad, fear) on pupil reactivity. Pupil dilation (average dilation, as a change from baseline,

in the time window 1000 – 2000 ms post stimulus onset) was measured. The trial structure is detailed in Figure 6.

The pupil task utilised a stimulus presentation of 2000 ms. Existing literature measuring emotional modulation of the pupil has typically presented stimuli for many seconds (e.g., Bradley et al., 2008); however recent work has established that image duration (100, 300, 1000 or 3000 ms) has no effect on the emotional modulation of the pupil, suggesting a comparable dilation due to emotional content at even very brief, unmasked stimulus presentations (Snowden et al., 2016). A study assessing ERP, autonomic, and facial responses to unmasked affective and neutral images, presented at varying rates from 25 to 6000 ms, found no interaction between responses and stimulus presentation time, suggesting that emotional engagement is similar in magnitude across both short and long presentation times (Codispoti, Mazzetti and Bradley, 2009). In the present research it was felt that very brief stimulus presentations across all the experimental tasks might place the offender participants under undue stress and possibly increase rates of attrition. Similarly, it was felt that a presentation rate of several seconds would excessively increase the length of the task and also possibly increase rates of attrition. The 40 images were each presented once in a randomised order with no practice trials.

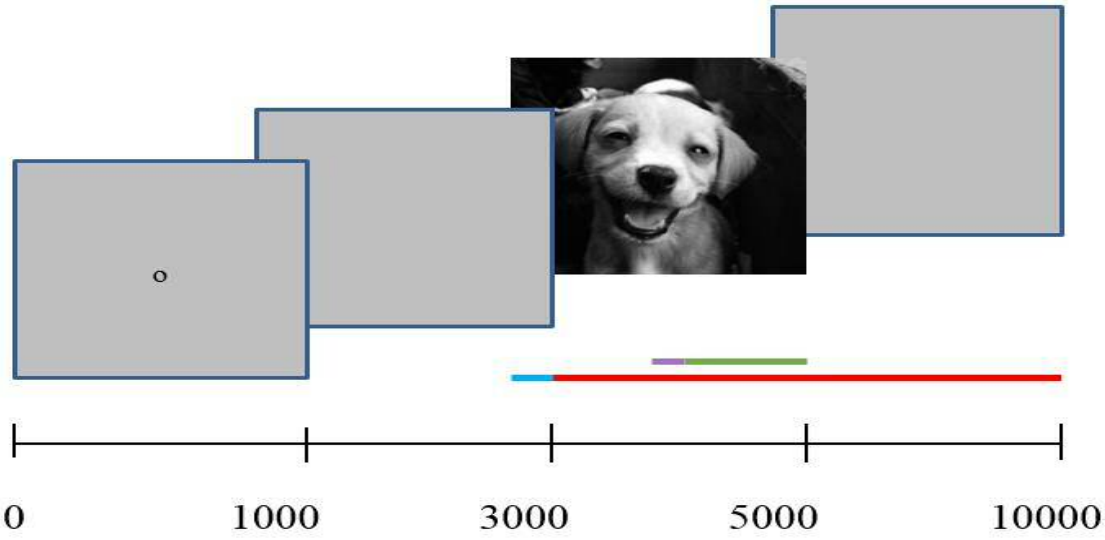


Figure 6. Task trial structure. *Note:* the red line represents the period of pupil diameter used to visually inspect the data in a graph; the blue line represents the last 200ms of the baseline period used to calculate change from baseline measures. This blue line also represents the initial pupil diameter used in subsequent forensic analysis. The green line represents the period of pupil dilation. The purple line represents the period of initial constriction response (subsequent forensic analysis).

2.4.2.1.1.4 Procedure

The participant was seated in an upright chair approximately 60 cm directly in front of the eye tracker which was placed just below the stimulus presentation screen. A calibration procedure was conducted for each participant using a 5-point calibration screen: the participant was instructed to view a moving target as it moved sequentially between 5 points over the course of 10 seconds. This calibration procedure then located the participant's pupils in three dimensional space, therefore allowing for small head movements to occur. Participants were then instructed to view each stimulus as it was presented on the screen and not look away.

Participants were asked to keep as still as possible but were reassured they could blink freely. The task took approximately 6 minutes.

Previous work (Snowden et al., 2016) established no difference in emotional modulation of pupil response between free viewing (as in the present paradigm) and active processing (indicating whether the image was affective or neutral) conditions. Although that particular study does not represent a thorough assessment of the potential moderating role of attention on pupil responses, the result is sufficient to suggest that the emotional modulation of pupil response generalizes from basic attention conditions to free viewing conditions. Given that this was the first work to utilise pupil activity in this population, the free viewing condition was preferred in order to establish the pattern of pupil response in relation to psychopathy, in the absence of any attentional manipulations or task demands (such as placing stimuli in the peripheral visual field; see De Cesarei, Codispoti and Schupp, 2009).

2.4.2.1.2. Results and Discussion

2.4.2.1.2.1. Data reduction

Pupil size was continuously sampled by the Tobii system at 60 Hz throughout each trial from the onset of the baseline screen to the offset of the recovery screen. Pupil data was converted offline through E-Prime software from arbitrary units to millimetres. Pupil diameter was determined as the mean diameter of both pupils due to consensual reflex (Lemaire, Aguillon-Hernandez, Bonnet-Brilhault, Martineau and El-Hage, 2014). Using the procedures described in previous studies (e.g., Arriaga, Adriao, Madeira, Cavaleiro, Silva, Barahona et al., 2015; Bradley et al., 2008; Partala and Surakka, 2003), any pupil diameter change of +/- .38

mm within a 20 ms interval was interpreted as a random fluctuation and removed. Data points surrounding missing data (within 33.34 ms) were also deleted to avoid anomalous readings.

A pre-stimulus baseline pupil size average of 200 ms (as per Kuchinke, Schneider, Kotz and Jacobs, 2011; Leknes, Wessberg, Ellingsen, Chelnokova, Olausson and Laeng, 2013; Lemaire et al., 2014; Van der Meer, Beyer, Horn, Foth, Bornemann, Ries et al., 2010; blue line in Figure 6) was calculated for each trial and subtracted from each subsequent data point to establish change from baseline pupil response for each data point within the trial. Mean, change from baseline, pupil diameter at every data time point (every 16.67 ms) across trials was thus calculated for each class of image. For example, on a within subjects basis, change from baseline pupil diameter at every time point was calculated for each happy image and the mean diameter across each time point was calculated across each happy image in order to create one grand mean for happy images per participant. These data points were then plotted over time in order to create the pupil waveform.

On a within subjects basis, mean pupil diameter was deleted at data time-points where there was missing data at that time-point for more than 50% of trials per image type. Common procedures for data smoothing were used (Beatty and Lucero-Wagoner, 2000). Linear interpolation, i.e., creating a value for a missing data point based on the average of the preceding three and following three data points, was used to estimate pupil diameter where a missing pupil sample led to fluctuations in the mean pupil change for the relevant class of image, usually around image-offset. Linear interpolation was done at a sample level (i.e., Figure 7) in order to aid visual interpretation of the data pattern and not at the subject level, therefore subject means used in inferential analyses were not manipulated. Change from baseline pupil dilation was calculated on a within subjects basis as the mean pupil diameter

change from initial pupil diameter levels 1 – 2 s post stimulus onset (corresponding to the green line on Figure 6)[17].

Table 9

Mean (SD) pupil diameter, in millimetres, per stimulus content, for period of pupil dilation

	Neutral[1]	Happy	Sad	Fear
Pupil dilation	-.74 (.03)	-.77 (.03)	-.52 (.03)	-.59 (.03)

Note: Standard deviation corrected for repeated measures designs (Cousineau, 2005; Morey, 2008). [1] Year 2 stimulus set.

2.4.2.1.2.2 Pupil dilation

Reliability of the pupil dilation response was established through zero-order split-half correlations of mean diameter (odd versus even trials) during this time window (corrected by the Spearman-Brown formula: $2r$ / $1+r$) indicating good reliability for each stimulus type: neutral, $r(36) = .84$, $p < .001$; happy, $r(36) = .87$, $p < .001$; sad, $r(36) = .81$, $p < .001$; fear, $r(36) = .78$, $p < .001$. Missing or deleted data accounted for 10.12% of all data points in this time window.

Data averaged across participants is shown in Figure 7 (means and *SE* are presented in Table 9). A repeated measures ANOVA was conducted on the average dilation to each stimulus type 1000 - 2000 ms post stimulus onset. This analysis found a main effect of stimulus

[17] Mean pupil diameter over this time window was chosen as the measure of interest as it is less vulnerable to random variations than taking a measure of peak dilation in a particular time window (Beatty and Lucero-Wagoner, 2000) and trial length was identical across participants. However, this measure is subject to the number of data points recorded in the time window.

emotional content, $F(3, 105) = 25.52$, $p < .001$, $\eta_G^2 = .08$. Planned comparisons indicated that fear ($t(35) = 4.24$, $p < .001$, $d_z = 0.71$) and sad ($t(35) = 6.67$, $p < .001$, $d_z = 1.11$) images elicited greater dilation as compared to neutral stimuli. There was no difference in dilation between neutral and happy images $t(35) = 1.11$, $p > .05$.

Exploratory analysis on the affective categories showed that the dilation elicited by sad images was greater as compared to happy images ($t(35) = 7.04$, $p < .001$, $d_z = 1.17$) and marginally greater than that of fear images ($t(35) = 1.94$, $p = .06$, $d_z = 0.32$). The dilation elicited by fear images was also greater as compared to happy images ($t(35) = 5.96$, $p < .001$, $d_z = 0.99$).

Results for the sad and threat images were comparable to those found by Bradley et al. (2008) when they compared pupil response to neutral images versus 'unpleasant' images. This indicated that sad and threat images were both capable of changing pupil size. However, results for the happy images did not match those of Bradley and colleagues for their 'pleasant' images. Recent work has demonstrated differing patterns of physiological responses for specific emotion categories contained within categories of pleasant or unpleasant (e.g., Bradley et al., 2001, Sarlo, Palomba, Buodo, Minghetti and Stegagno, 2005; Weinberg and Hajcak, 2010). For example, highly unpleasant and defensive-response inducing images, such as pictures featuring mutilation and threat, produced larger SCR and enhanced startle blink patterns (Bradley et al., 2001; Sarlo et al., 2005) as compared to other unpleasant images. These differing patterns of physiological reactivity, often for stimuli that are matched on self-reported arousal ratings, suggest that certain images are more motivationally relevant (Cunningham and Brosch, 2012) than others and are more likely to activate appetitive or defensive motivational systems (Weinberg and Hajcak, 2010). As discussed, pupil dilation to a larger set of images (happy, adrenaline, sad, fear, neutral-simple and neutral-complex) was then assessed.

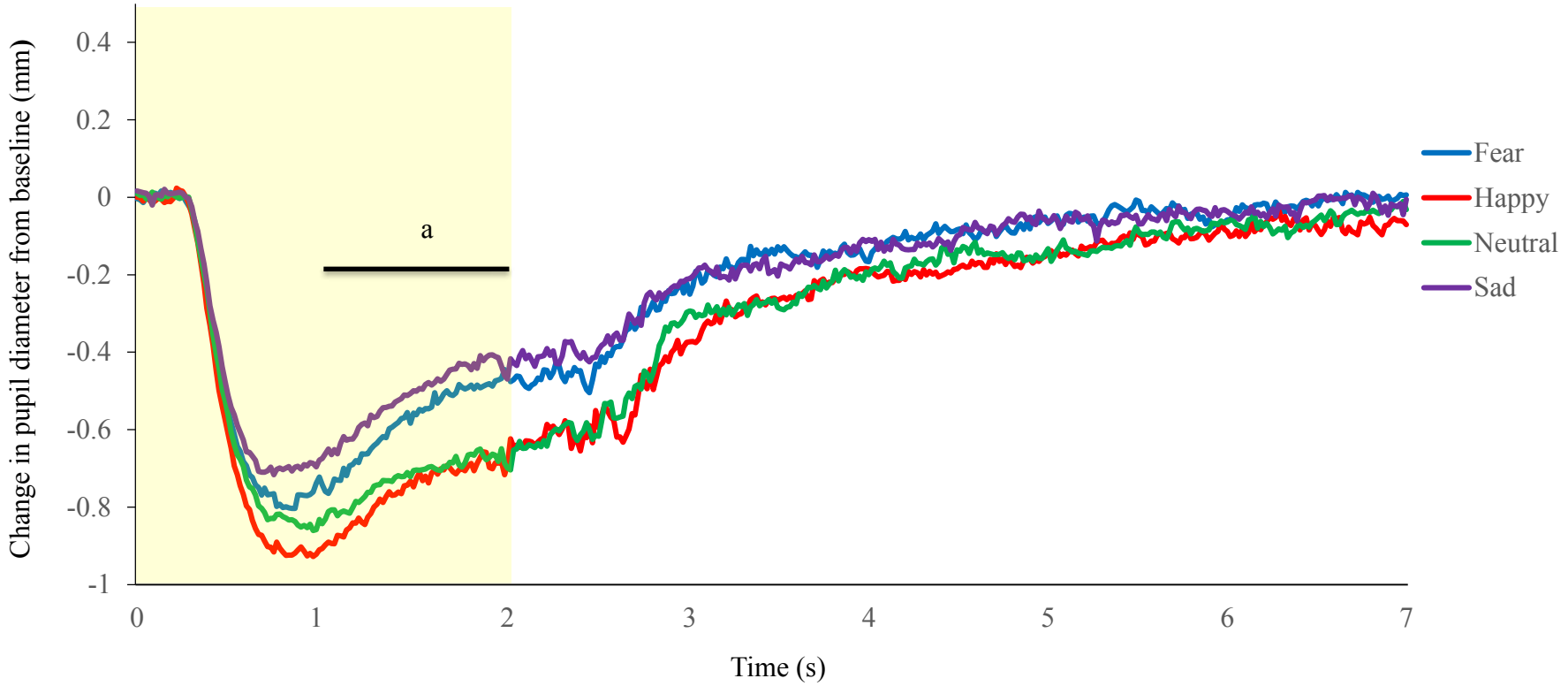


Figure 7. Change (mm) in pupil diameter from a 200 ms baseline preceding picture onset when viewing fearful, happy, sad or neutral images (Year 2 stimulus set). The shaded section represents interval of target presentation. Pupil dilation (a) was averaged in a window from 1.0 to 2.0 s following target onset

2.4.2.2. Year 3 data collection: 6 conditions study

This round of piloting aimed to test the efficacy of the new stimulus set ahead of forensic data collection. In this experiment, six sets of stimuli were presented: neutral-complex (existing set of neutral stimuli), neutral-simple (alternative set of neutral stimuli), happy, sad, fear and adrenaline. The aim was to provide a better neutral set for comparisons, and assess any potential differences in the impact of happy and adrenaline stimuli on pupil dilation.

2.4.2.2.1. Method

2.4.2.2.1.1 Participants

Twenty seven university students and community members (11 male) participated, aged 18 – 29 (M = 22.67 years).

2.4.2.2.1.2 Materials, Design and Procedure

Materials were 40 affective (10 happy, 10 sad, 10 fear, 10 adrenaline) and 20 neutral images, as detailed in section 2.2.4. Study design and procedure were identical to the year 2 data collection original task. As well as the measure of pupil dilation, two additional measures of pupil reactivity were recorded: initial pupil diameter (average diameter -200 – 0 ms pre stimulus onset) and initial constriction response (average constriction in the time window 500 – 1000 ms post stimulus onset, as a change from baseline levels). Calibration procedure was identical to that reported in section 2.4.2.1.1.4.

2.4.2.2.2. Results

Methods of data reduction are detailed in section 2.4.2.1.2.1. Means and *SD* for each of the time windows are presented in Table 10.

Table 10

Mean (SD) pupil diameter, in millimetres, per stimulus content, for specific time windows

Initial pupil diameter	5.19 (.62)	
Stimulus content	Initial Constriction Response	Later dilation
Neutral-Simple[1]	-.60 (.05)	-.55 (.05)
Happy	-.59 (.02)	-.50 (.03)
Sad	-.48 (.02)	-.39 (.02)
Fear	-.54 (.02)	-.42 (.02)
Adrenaline	-.45 (.05)	-.31 (.05)
Neutral-Complex	-.61 (.02)	-.53 (.02)

Note: Standard deviation calculated using corrected sample variance for repeated measures designs as per Cousineau (2005) and Morey (2008). [1]Finalised stimulus set.

2.4.2.2.2.1. Initial pupil diameter

The initial pupil diameter (IPD) was calculated as the average pupil diameter across all 40 trials, in the time window -200 – 0 ms pre-stimulus onset. Reliability of the IPD in this undergraduate sample was assessed through a zero-order correlation with the initial diameter -200 – 0 ms prior to the onset of the first test image, producing a near perfect correlation r = .94, p < .001. Missing or deleted data accounted for 15.07% of all data points in this time window.

In the forensic sample, IPD was regressed onto the psychopathy factors and scales. IPD was therefore used in the forensic analysis as a criterion variable. Pupil diameter from the same time window (i.e., -200 – 0 ms pre-stimulus onset) was also used to calculate change from baseline (reactivity) constriction responses and pupil dilation responses on a trial by trial basis.

This does not represent a baseline-correction, rather it is considered to represent a change score. As will be discussed in section 5.4.4, IPD (average pre-stimulus pupil diameter across all 40 trials) was also included as a covariate in regression analyses regressing constriction responses onto the psychopathy scales and factors, given the inverse relationship between IPD and reactivity. This inverse relationship may represent a mechanical limitation; a small resting pupil may be limited in its capacity for further constriction. In a sense the magnitude of the constriction response may therefore be confounded by its average resting point (initial diameter). For this reason, IPD was included as a covariate in constriction regression models.

2.4.2.2.2.2. Initial constriction response

The initial constriction response (ICR) was calculated as the mean diameter, as a change from baseline, in the time window 500 – 1000 ms post-stimulus onset. This time window was chosen based on visual inspection of the data. Reliability of the ICR was established through zero-order split-half correlations of mean diameter during this time window (see section 2.4.2.1.2.2) indicating good reliability for each image type: neutral-simple, $r(27) = .91$, $p < .001$; neutral-complex, $r(27) = .90$, $p < .001$; happy, $r(27) = .92$, $p < .001$; sad, $r(27) = .89$, $p < .001$; fear, $r(27) = .90$, $p < .001$; adrenaline, $r(27) = .97$, $p < .001$. Missing or deleted data accounted for 8.90% of all data points in this time window.

As can be seen in Figure 8, the pupil's response follows a pattern in line with that previously established (compare to Figure 7) with an onset for all images at around 300 ms post-stimulus onset. The response for the adrenaline and the sad images begin to diverge from that of the other images at around 700 ms, with these affective images eliciting notably less constriction.

A repeated measures ANOVA on the ICR to each image type showed a main effect of stimulus affective content, $F(5, 130) = 22.56$, $p < .001$, $\eta_G^2 = .07$. Results will focus on neutral-simple as opposed to neutral-complex images, given that the former were included in the finalised stimulus set. Planned comparisons indicated that fear ($t(26) = 3.19$, $p < .01$, $d_z = 0.61$), sad ($t(26) = 6.14$, $p < .001$, $d_z = 1.18$) and adrenaline images ($t(26) = 10.47$, $p < .001$, $d_z = 2.01$) elicited an attenuated ICR as compared to neutral-simple images. There was no difference in the magnitude of the ICR between neutral-simple and happy stimuli $t(26) = .76$, $p > .05$.

Exploratory analysis on the affective categories showed that the ICR elicited by sad images was attenuated as compared to fear images ($t(26) = 4.02$, $p < .001$, $d_z = 0.77$) and happy images ($t(26) = 6.30$, $p < .001$, $d_z = 1.21$). The ICR elicited by adrenaline images was also attenuated as compared to fear ($t(26) = 4.98$, $p < .001$, $d_z = 0.96$) and happy images ($t(26) = 5.38$, $p < .001$, $d_z = 1.03$). The ICR elicited by fear was marginally attenuated as compared to happy images ($t(26) = 1.93$, $p = .07$, $d_z = 0.37$). There was no difference in the magnitude of the ICR elicited by adrenaline or sad images ($t(26) = 1.36$, $p > .05$).

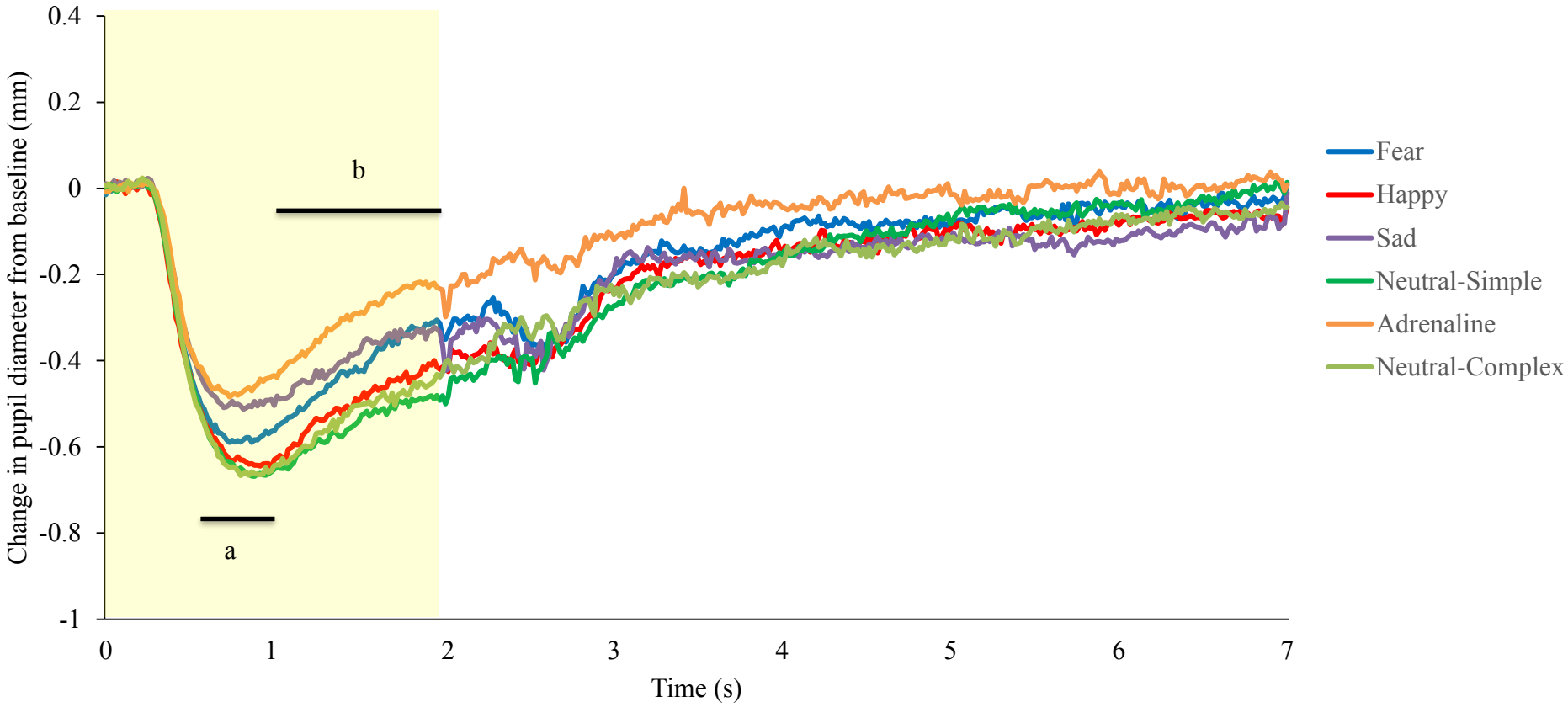


Figure 8. Change (mm) in pupil diameter from a 200 ms baseline preceding picture onset when viewing fearful, happy, sad, adrenaline, neutral-simple or neutral-complex images (finalised stimulus set). The shaded section represents interval of target presentation. ICR (a) was averaged in a window from 0.5 to 1.0 s following target onset and pupil dilation (b) was averaged in a window from 1.0 to 2.0 s following target onset.

2.4.2.2.2.3. Pupil dilation

Pupil dilation was calculated as discussed in section 2.4.2.1.2.1. Reliability of the pupil dilation response was established through zero-order split-half correlations of mean diameter during this time window (see section 2.4.2.1.2.2) indicating good reliability for each image type: neutral-simple, $r(27) = .78$, $p < .001$; neutral-complex, $r(27) = .86$, $p < .001$; happy, $r(27) = .78$, $p < .001$; sad, $r(27) = .90$, $p < .001$; fear, $r(27) = .80$, $p < .001$; adrenaline, $r(27) = .94$, $p < .001$. Missing or deleted data accounted for 9.36% of all data points in this time window.

A repeated measures ANOVA on the average dilation to each image type 1000 - 2000 ms post stimulus onset showed a main effect of content, $F(4.20, 109.12) = 24.48$, $p < .001$, $\eta_G^2 = .12$. Planned comparisons indicated that all affective images (happy: $t(26) = 2.26$, $p < .05$, $d_z = 0.43$; sad: $t(26) = 5.43$, $p < .001$, $d_z = 1.04$; fear: $t(26) = 4.41$, $p < .001$, $d_z = 0.85$; adrenaline: $t(26) = 13.58$, $p < .001$, $d_z = 2.61$) elicited greater dilation than neutral-simple images. Exploratory analysis showed that adrenaline images also elicited greater dilation than happy $t(26) = 6.91$, $p < .001$, $d_z = 1.33$; sad $t(26) = 3.06$, $p = .005$, $d_z = 0.59$; and fear $t(26) = 4.00$, $p < .001$, $d_z = 0.77$, images. Sad images elicited greater dilation than happy images $t(26) = 3.67$, $p = .001$, $d_z = 0.71$.

The below Figure 9 illustrates the effectiveness of the finalised stimulus set in eliciting pupil reactivity. The attenuation of the ICR in response to affective images was found for aversive images only, with no difference in the magnitude of the ICR between happy and neutral images. This is in line with recent results indicating the specificity of the threat-inhibited ICR (see section 5.2.1). All affective images elicited greater pupil dilation as compared to neutral images, indicating the effectiveness of the paradigm ahead of data collection in the forensic sample.

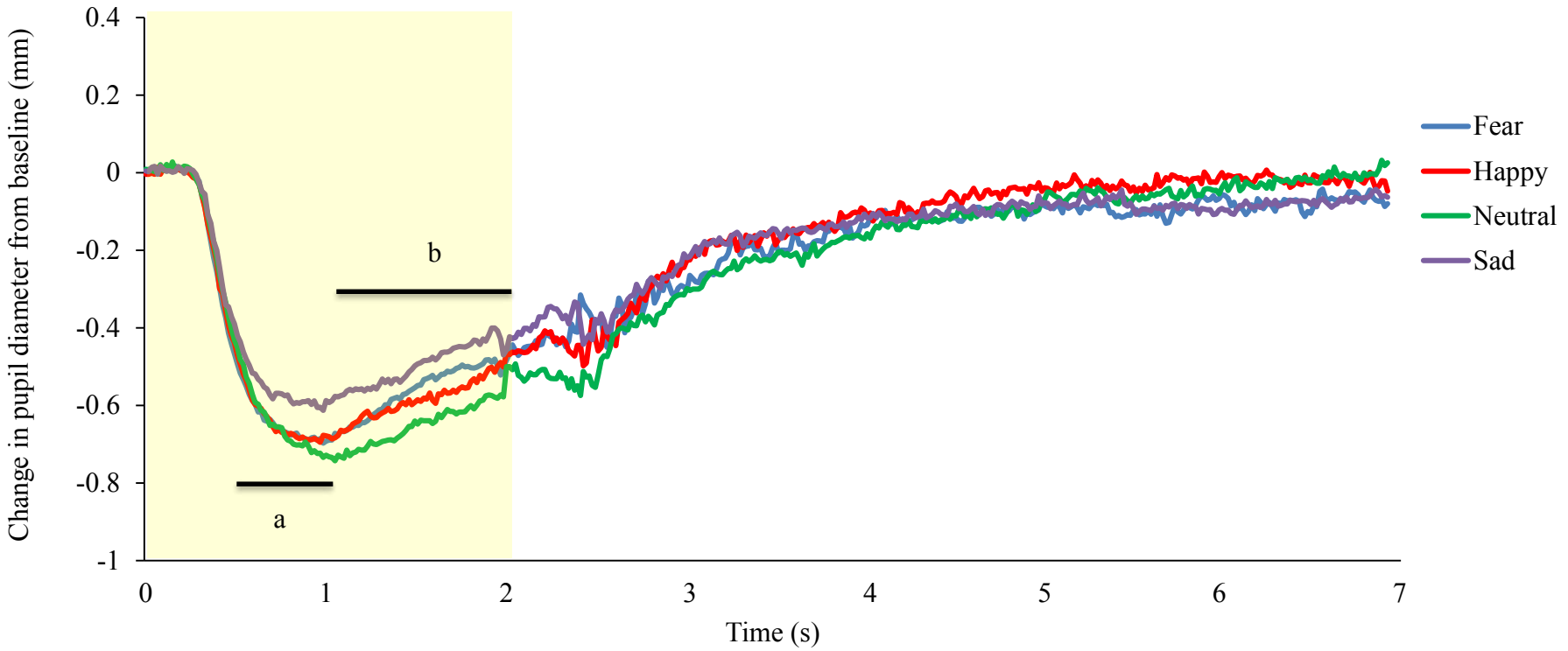


Figure 9. Change (mm) in pupil diameter from a 200 ms baseline preceding picture onset when viewing fearful, happy, sad or neutral images (finalised stimulus set). The shaded section represents interval of target presentation. ICR (a) was averaged in a window from 0.5 to 1.0 s following target onset and pupil dilation (b) was averaged in a window from 1.0 to 2.0 s following target onse

2.4.3. Affective Priming task

The Affective Priming task was originally designed as a between-groups experiment (year 1 piloting) before being revised to a within-subjects experiment (year 2 and 3 piloting). Due to this, piloting from year 1 and year 3 are presented. Little methodological changes occurred in the task itself between year 2 and year 3 (apart from stimulus selection) therefore results for the former are not presented.

2.4.3.1. Year 1 data collection

2.4.3.1.1. Method

2.4.3.1.1.1. Participants

A sample of 30 university students (12 male, *M* age = 22.87, *SD* = 2.54, Range = 19-29) were placed into one of two groups; Low Attention group *n* = 16 (7 male), *M* age= 23.06, *SD* = 2.91, Range = 18 - 29; High Attention group *n* = 14 (5 male), *M* age = 22.64, *SD* = 2.13, Range = 20 - 26. Data from one participant was excluded from the High Attention group for extremely poor performance on both congruent and incongruent trials.

2.4.3.1.1.2. Materials

Images from the Year 1 stimulus set were presented as primes (see section 2.2.1 for details) with year 1 affective targets (see section 2.3 for details).

2.4.3.1.1.3. Design

The study used a mixed model design. The dependent variable was response time (RT), measured in milliseconds, within a response window deadline (1000 ms) assessed through a forced choice response. Two independent variables were manipulated. The first independent variable was a repeated measures variable of prime: congruent, neutral and incongruent. The second independent variable was a between-group measure of group: Low or High Attention

The task began with on screen instructions and consisted of two phases, the practise phase and the test phase. All participants were informed that a picture would be presented on the screen, followed by a scrambled image, then a word. Instructions for the Low Attention group said that "you will see some pictures presented first, but your response is based on the word that you see afterward."

Participants in the High Attention group also received a 'prime only' manipulation (Attend trials). Instructions for the High Attention group said that "you need to try to pay attention to the picture that appears first, as well as the word that you see afterward." Participants were instructed that on some trials the target word would not be presented and a screen would instruct them to report the valence ('good' or 'bad') of the prime image. All participants were required to press Z if the target word was 'good' and M if it was 'bad'. Response options were counterbalanced across participants.

The presentation order of test trials was randomised throughout both tasks for each participant. Practice trials were identical in structure to the test trials but utilised different primes and targets (Low Attention group = 28 practice trials, High Attention group = 32 practice trials).

The task utilised backwardly-masked prime stimuli, briefly presented and followed by a visual noisemask in order to prevent further processing of the prime stimulus.[18] Prime stimuli were presented for 100 ms in order to create a stimulus onset asynchrony (SOA) of 200ms, considered optimal to elicit priming effects (Avero and Calvo, 2006, experiment 2). Priming effects have been observed to decrease with an SOA greater than 300 ms (Hermans, De Houwer and Eelen, 2001). Combining the 4 types of prime and 2 types of target allowed 8 trial structures to be created (Neutral trials: neutral-positive, neutral-negative; Congruent trials: happy-positive, sad-negative, fear-negative; Incongruent trials: happy-negative, sad-positive, fear-positive). Neutral trials were included in the Affective Priming task to assess whether priming effects were caused by facilitation or inhibition processes of positive and negative primes. These 8 trials were repeated in 20 blocks resulting in 160 trials for the Low Attention group. An additional 30 trials (approximately 20%, affective primes only) were added to the High Attention group in which only the prime was presented, resulting in a total of 190 trials. All primes and targets were presented throughout the task. Targets and primes were randomly assigned to each other; the restriction in this instance was that there should be equal number of affectively congruent and incongruent trials. Trial structure for the task utilised a 50/50 congruent/incongruent trial ratio and targets and primes were randomly paired.

[18] No suggestion is made here for subliminal priming, rather the noisemask serves to restrict further elaboration of the prime stimulus and facilitate the desired stimulus onset asynchrony (SOA, see section 6.2.1).

Each trial started with a fixation point 'o' (100 ms). At the offset of the fixation point, the prime stimulus was presented for 100 ms followed by a noise mask for 100 ms producing an SOA of 200 ms. The target immediately followed the noise mask and was presented for 1000 ms; targets were cleared if an RT (i.e., a button press) was collected within this window. The response screen displayed the options for 1000 ms following the target, after which the screen returned to blank. Responses were still collected if made outside of the 1000 ms response window. If the RT was collected outside of the 1000ms response window, an error message of "Too Slow!" was displayed upon collection of the RT. The next trial began after an ITI of 1000 ms (see Figure 10a).

For trials in which only the prime was presented (High Attention group, see Figure 10b) the trial structure was as follows: Using prime durations of 100 ms, the noise mask followed for the standard 100 ms display. A response screen of "What was the image?" immediately followed the mask (1000 ms); if the RT was collected within this 1s response deadline the next trial began after the ITI of 1000 ms. The response screen cleared after 1000 ms, if the RT was taken after this deadline an error message of "Too Slow!" was displayed upon collection of the RT. RT measurements provide an upper estimate of processing time, as they include the time required for image processing but also time required for decisional and motor mechanisms (Bacon-Mace et al., 2005). Despite this limitation, priming tasks utilising measures of RT have delivered robust effects (see section 6.2.1).

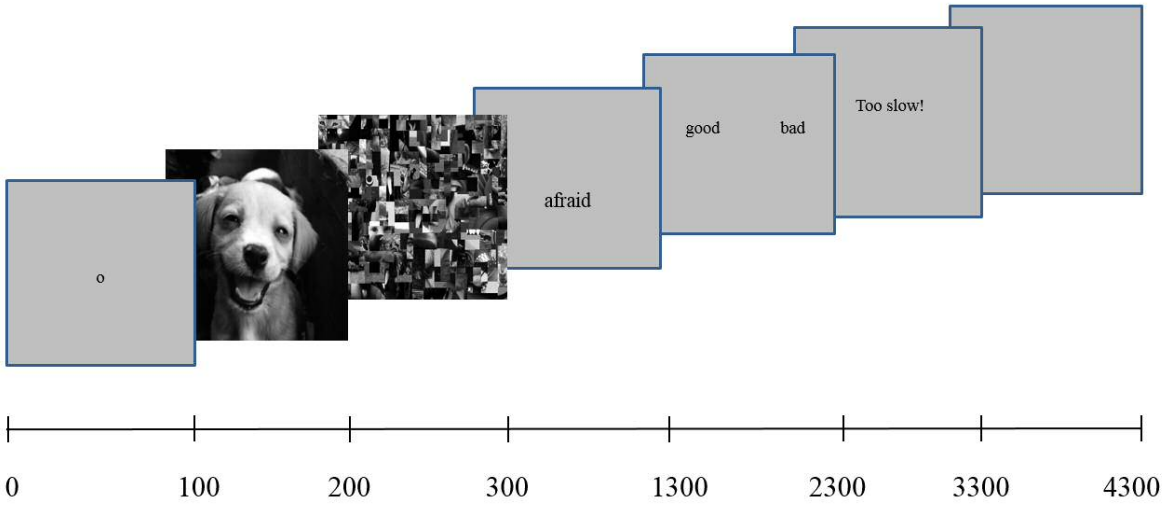


Figure 10a. Affective Priming: Trial structure for prime and target trials**.** Participants in both the Low and High Attention groups completed these trials.

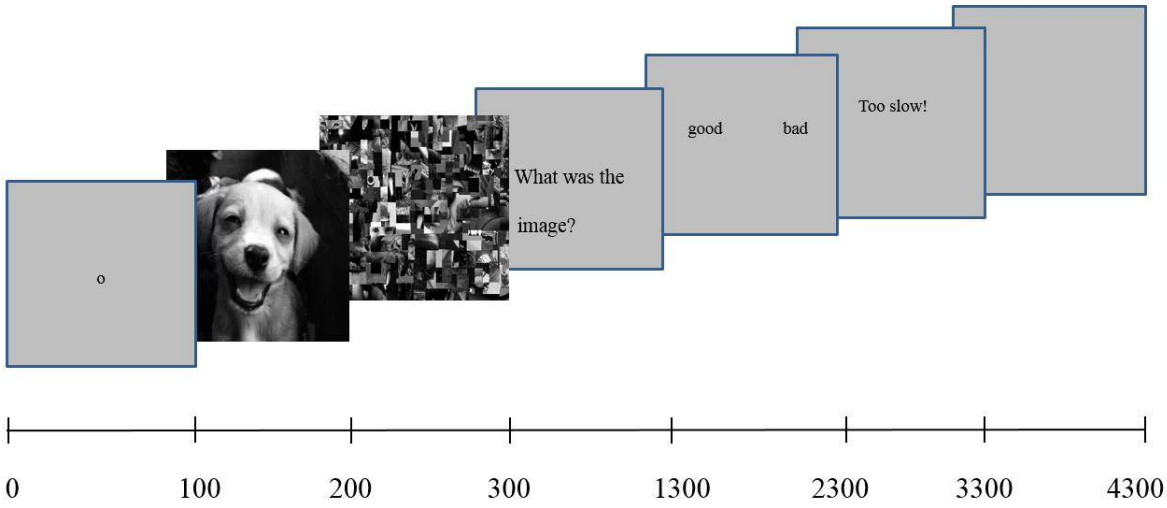


Figure 10b. Affective Priming: Trial structure for prime-only (Attend) trials. Only participants in the High Attention group completed these trials.

2.4.3.1.1.4 Procedure

Participants were provided with on screen instructions prior to the task, which were reiterated by the experimenter as well as a visual prompt (laminate) for response options. Participants were either instructed to prioritise attention to the target word (Low Attention group); or instructed to attend to both the word and the image (High Attention group) as the response would be based on whichever was last presented. Instructions required participants to press Z if the response stimulus was 'good' and M if the response stimulus was 'bad' (counterbalanced response options). Instructions emphasised speed of response. The task took approximately 6 minutes.

2.4.3.1.2. Results and Discussion

Response times on trials on which participants correctly classified the valence of the target stimulus were screened individually and outliers deviating from the mean by more than 3 *SD* were excluded (Low Attention group = 2.77% of total trials; High Attention group = 1.74% of total trials). Analyses were conducted on correct responses only. Data from practice trials were not included in analyses.

A mixed model 3 (prime: congruent, neutral, incongruent) x 2 (group: Low Attention, High Attention) ANOVA was conducted which showed a significant main effect of prime, $F(1.77, 47.85) = 25.43$, $p < .001$, $\eta_G^2 = .06$, but no main effect of group, $F(1, 27) = 2.06$, $p > .05$. There was a significant prime by group interaction, $F(1.77, 47.85) = 19.91$, $p < .001$, $\eta_G^2 = .04$. Analysis of the interaction effect indicated that no significant differences were found within the Low Attention group, whereas there was an affective priming effect within the High Attention group: RTs to congruent trials were

faster than incongruent ($t(12) = 5.84$, $p < .001$, $d_z = 1.62$) and neutral trials ($t(12) = 14.65$, $p < .001$, $d_z = 4.06$). There was no significant difference between incongruent and neutral trials ($t(12) = .84$, $p > .05$; see Figure 11).

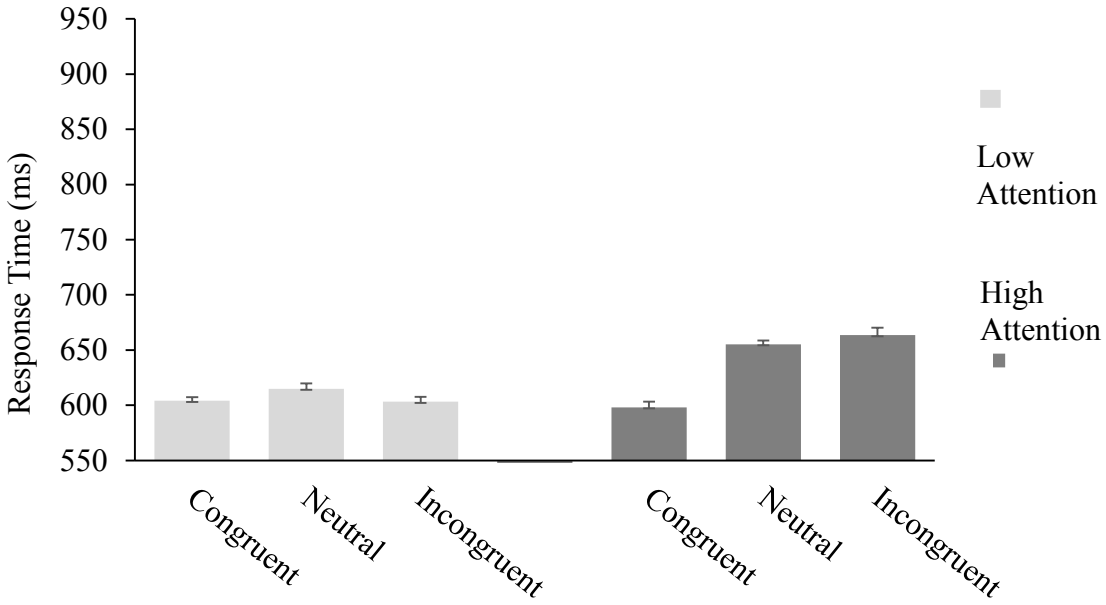


Figure 11. Response times to congruent, neutral and incongruent trials on a group basis: Affective Priming Task, Year 1 data collection. Error bars display 2 *SE* (correction for repeated measures designs, Cousineau (2005) and Morey (2008)).

The failure to find an affective priming effect within the Low Attention group was surprising given that the procedure was similar to those used elsewhere. Low Attention instructions were designed to discourage participants from processing the primes in a strategic way – participants were informed that pictures would be presented, but were asked to concentrate on the target word. It is possible that priming effects are more likely to emerge when participants receive specific instructions on how to deal with the primes (De Houwer and Randell, 2002). It may be that the instructions for

participants in the Low Attention group in relation to the primes were not explicit enough; previous studies in which participants were explicitly instructed to ignore the prime resulted in affective priming effects (Hermans et al., 2001) as well as studies where participants were explicitly instructed to report the prime valence (Andrews, Lipp, Mallan and Konig, 2011). Studies in which instructions with regard to the prime were less explicit, for example De Houwer and Randell (2002), have also failed to find an effect of affective priming.

Within the High Attention group, RT to congruent trials was significantly faster than incongruent and neutral trials. There was no significant difference between incongruent and neutral trials. Slower evaluation times were also observed in the High Attention group, as compared to the Low Attention group. Previous studies (e.g., Andrews et al., 2011) noted that the overall slower evaluation times in their reporting conditions suggested that reporting the prime expression required more cognitive resources than did the no reporting conditions, which possibly reflected the identification and encoding of the prime for later. Present results were also similar to Fazio, Sanbonmatsu, Powell and Kardes (1986; experiment 3) in which participants were asked to consciously evaluate a series of positive and negative words 5 times, while for another set of words they had to decide whether or not the word was a one-syllable word. The manipulation of Fazio et al. (1986; experiment 3) had a significant effect on the results of the subsequent affective priming task in which these words were used as primes. For primes that had been repeatedly evaluated prior to the priming phase, the affective priming effect was stronger than for primes for which a non-evaluative decision was asked. This result is easily explained if one assumes that repeated conscious evaluation of an attitude object strengthens the object-evaluation association (Powell and Fazio, 1984), which in turn might facilitate automatic attitude activation.

In light of these results, the decision was made to move forward using only the High Attention condition for future testing in the offender sample. The pattern of results also brought into question the typically assumed uncontrollability of affective priming, suggesting a vulnerability to explicit strategies. It therefore was a concern that future participants could alter their pattern of affective priming by attending to the valence of the primes and using this knowledge to strategically manipulate the speed of their response to the target. It seemed likely though that such manipulations would require participants to be aware of the purpose of the task and the relevance of the prime to the target. Studies examining the susceptibility of affective priming to conscious manipulation have indicated that, even armed with such knowledge and with instructions focused on response speed, attempts at manipulation were less successful if response deadlines were used (Degner, 2009). In the present work, this method was utilised.

2.4.3.2 Year 3 data collection

The third round of piloting aimed to assess the utility of the final stimulus set ahead of testing the offender population. With the between-groups manipulation now removed, the task was now a repeated-measures design with a single within-subjects independent variable (prime). All participants therefore completed the High Attention condition with the following changes: the number of practice trials was reduced to 8, the number of typical test trials was reduced to 80 with 16 prime only trials, the emphasis on speed of response was removed and the "Too Slow!" error message was removed. An analysis of prime evaluation scores is also presented here.

2.4.3.2.1. Method

2.4.3.2.1.1 Participants, Materials, Design and Procedure

The sample was the same as for the Affect Categorisation task Year 3 data collection sample. Data from one participant was removed from the Affective Priming task due to less than 66% accuracy on average; chance levels of accuracy in this task are 50% therefore levels above 66% were considered to represent appropriate levels of performance[19]. Materials were the year 3 stimulus set (see section 2.2.4) and year 3 affective targets (see section 2.3). Figures 12a and 12b detail the finalised trial structure.

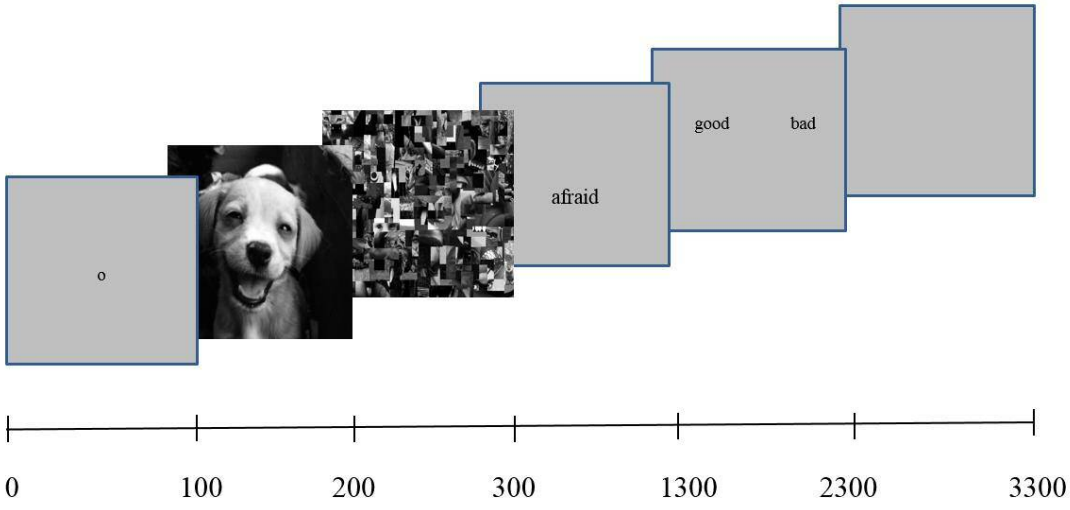


Figure 12a. Affective Priming: Finalised trial structure for prime and target trials**.**

[19] This approach was applied to the offender sample also.

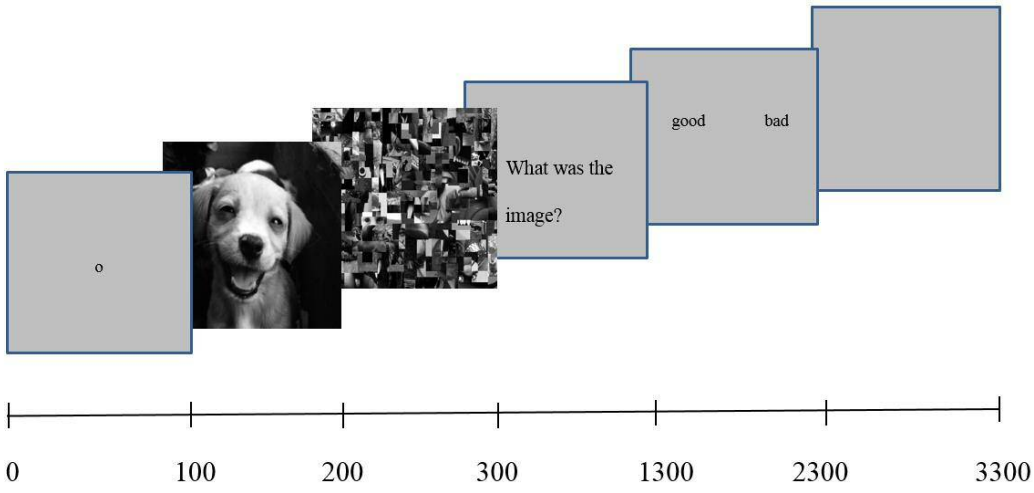


Figure 12b. Affective Priming: Finalised trial structure for prime-only (Attend) trials.

2.4.3.2.2. Results and Discussion

Data cleaning was as per the Year 1 data collection protocol (3.47% of trials were excluded as outliers).

2.4.3.2.2.1. Prime evaluation data

Participants were instructed to attend to both the prime and the target. As a manipulation check on the prime-only trials, prime evaluation scores were calculated for each participant by subtracting the proportion of errors (primes incorrectly evaluated) from the proportion of correctly evaluated primes. Perfect performance would be indicated by a score of 1 on this evaluation index. Participants performed at better than chance levels; the mean score was .84 (*SD* =.14; Range = .56 – .84) which a one sample *t*-test indicated was significantly different from zero, $t(14) = 22.95$, $p < .001$, $d_z = 5.93$.

2.4.3.2.2.2 Response Time data

Overall RT to congruent, neutral and incongruent trials were assessed for skew and kurtosis. All *z*-scores for skewness (Skew/SE Skewness) and kurtosis (Kurtosis/SE Kurtosis) were below 2.58 ($p > .01$; criteria for small samples, see Field, 2005, page 72) therefore no transformations were necessary. A repeated measures (prime: congruent, neutral, incongruent) ANOVA was conducted on the RT data. There was a main effect of the prime condition, $F(2, 28) = 4.27$, $p < .05$, $\eta_G^2 = .02$ (see Figure 13). Planned comparisons indicated that RT to congruent trials was significantly faster than neutral ($t(14) = 2.25$, $p < .05$, $d_z = 0.58$) and incongruent trials ($t(14) = 2.75$, $p < .05$, $d_z = 0.71$). There was no significant difference in RT between the neutral and incongruent trials ($t(14) = .05$, $p > .05$). The paradigm was considered to elicit affective priming effects ahead of the forensic data collection.

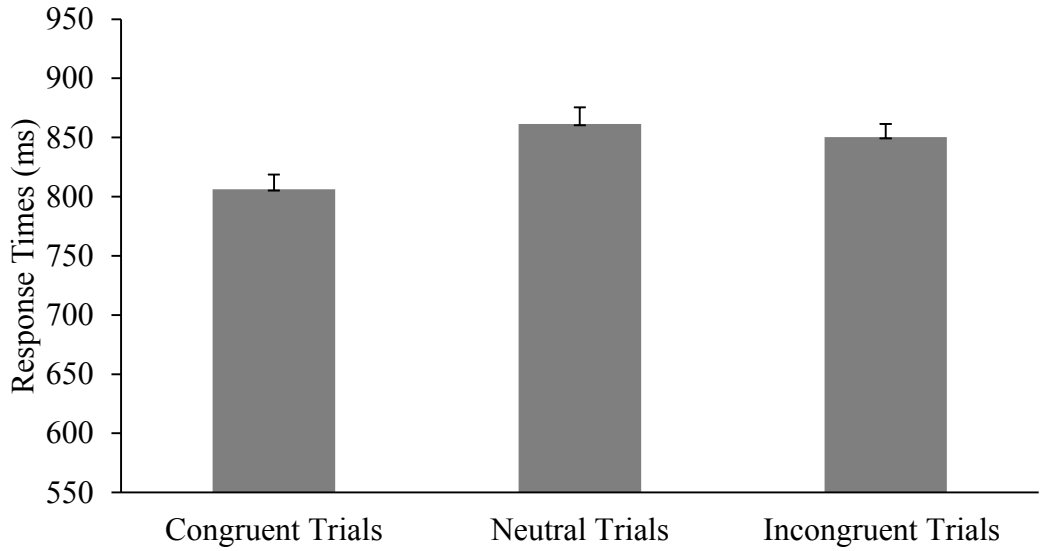


Figure 13. Response times to congruent, neutral and incongruent trials: Affective Priming Task, Year 3 data collection. Error bars display 2 *SE* (correction for repeated measures designs, Cousineau (2005) and Morey (2008)).

2.4.4 Semantic Priming task

The Semantic Priming task was piloted in year 2 and 3 to assess for efficacy ahead of data collection in the forensic sample. Given that there were no methodological changes to the task between year 2 and 3, apart from the stimulus selection which did not alter the pattern of results, for brevity only piloting from year 3 is presented here. The rationale for including a semantic priming task is presented in section 6.2.2. The Semantic Priming task differed from the Affective Priming task in that there were no neutral trials; the response required attention to the semantic content (person versus animal) which did not allow a logical neutral comparison. Studies examining semantic priming typically only include congruent and incongruent trials (see Gulan and Valerjev, 2010).

2.4.4.1. Method

2.4.4.1.1. Participants

The sample was the same as the Affective Priming: Year 3 sample.

2.4.4.1.2. Materials

Data collection for the Semantic Priming task utilised the Year 3 stimulus set (see section 2.2.4). Methods of presentation were identical to the Affective Priming task: year 3 data collection, with the below noted differences. Primes and targets could be either a picture of, or a word related to, an animal or person, and also either positive or negative. This produced four categories of both prime and target: positive animal, positive person, negative animal, negative person.

Prime stimuli were 16 affective (8 positive, 8 negative) images, selected from the existing set of 40 primes, containing either a person or an animal. Four images were selected for each category. [20] Using ratings as detailed in section 2.2.4, animal and person primes were matched on stimulus complexity, valence and arousal (all *p*'s > .05). Means and standard deviations of arousal, valence and complexity for these primes are presented in Table 11.

Table 11

Mean (SD) arousal, valence and complexity for Semantic Priming task primes

	Arousal	Valence	Complexity
Animal	5.98 (1.03)	4.65 (1.84)	3.69 (.38)
Person	5.69 (.49)	4.65 (2.85)	4.60 (1.21)

Note: Arousal and valence ratings are based on normative ratings provided by the IAPS manual; Lang et al., 2008; complexity ratings are based on the results of pilot research.

Targets were 10 animal-related and 10 person-related words, selected from the ANEW (Bradley and Lang, 2010) database. Word valence (positive or negative) was varied orthogonally to the semantic category, producing 4 word categories: positive animal targets, negative animal targets, positive person targets, negative person targets.

[20] IAPS reference numbers for negative animal primes: 1300, 1321, 1930, 1301; Positive animal prime: 1920; Negative person primes: 2800, 2375.1, 3350, 6560; Positive person primes: 4609, 4623, 4626, 8470. Three positive animal primes were sourced online.

The development of the semantic priming task was secondary to the affective priming task. As such, the semantic categorisation (person versus animal) was devised based on a natural division of the existing affective priming primes which necessarily limited the number of primes in each category.

Five stimuli were selected for each category.[21] Using ratings as per the Affective Priming task, when ordered semantically (positive and negative people versus positive and negative animals) target lists were matched on arousal, valence, word length and age of acquisition (all p values > .05; means and *SD* are presented in Table 8). When ordered affectively (positive animals and people versus negative animals and people), target lists were significantly different on valence ($p < .001$). Word length, arousal and age of acquisition were matched across affectively ordered lists (all p values > .05).[22]

2.4.4.1.3. Design

The semantic priming task used a 2x2 repeated measures design. Independent variables were created in line with Voss, Rothermund, Gast and Wentura (2013). The first within subjects' independent variable was labelled semantic: congruent/incongruent trials. Semantically congruent trials consisted of semantically matched prime-target trials while semantically incongruent trials paired semantically mismatched primes and targets. The second within subjects' independent variable was labelled affective: congruent/incongruent trials. Affectively congruent trials consisted of affectively

[21] Positive animal targets: cat, dove, pony, rabbit, swan. Positive person targets: actor, bride, hero, king, queen. Negative animal targets: cockroach, snail, snake, spider, wasp. Negative person targets: idiot, liar, loser, moron, slave.

[22] Note: Attempts were also made to match across tasks for all Affective Priming target words and all Semantic Priming target words on the four categories. Tasks were matched on target valence (p =.71; Affective Task: Valence M = 5.19, SD = 2.79; Semantic Task: Valence M = 4.90, SD = 2.00); target arousal (p = .35; Affective Task: Arousal M = 5.57, SD = 1.25; Semantic Task: Arousal M = 5.23, SD = 1.00); and age of acquisition (AoA; p =.67; Affective Task: AoA M = 5.95, SD = 1.34; Semantic Task: AoA M = 5.76, SD = 1.35). Target words were significantly longer in the Affective Priming Task (Length M = 6.75, SD = 1.48) as compared to the Semantic Priming Task (Length M = 4.95, SD = 1.28, $p < .001$).

matched prime-target pairs, affectively incongruent trials consisted of affectively mismatched prime target pairs.

Four overall trial categories were created: affective and semantic congruency (e.g., positive animal primes with positive animal targets); affectively congruent/semantically incongruent (e.g., negative animal primes with negative person target); affectively incongruent/semantically congruent (e.g., negative person primes with positive person target) and affective and semantically incongruent (e.g., positive person prime with negative animal target). With four possible combinations within each trial category, a block of trials consisted of 16 trials. This block was repeated 5 times, producing 80 typical test trials. Sixteen prime only trials were also presented (4 positive animal primes, 4 positive person primes, 4 negative animal primes, 4 negative person trials) making 96 test trials in total. These prime-only trials required an 'animal/person' response to the picture. Figure 14a and Figure 14b below detail the trial structure.

The task began with on screen instructions and consisted of two phases, the practise phase and the test phase. Participants were instructed to attend to both the word and the image as the response would be based on whichever was last presented. Participants were provided with on screen instructions prior to the task, which were reiterated by the experimenter as well as a visual prompt (laminate) for response options. Participants completed 8 practice trials. Response options were either person (press the Z button) or animal (press the M button, response options counterbalanced across participants). The use of this semantic response category, a superordinate-level semantic categorization of a natural category, allowed a comparison with a super-ordinate level affective categorization (good versus bad, i.e., pleasant versus unpleasant) in the affective priming category (see also Nummenmaa,

Hyona and Calvo, 2010). The response categories clearly differed however in that the Affective Priming task required an evaluation of abstract target words whereas the Semantic Priming task required a categorisation of concrete target words.

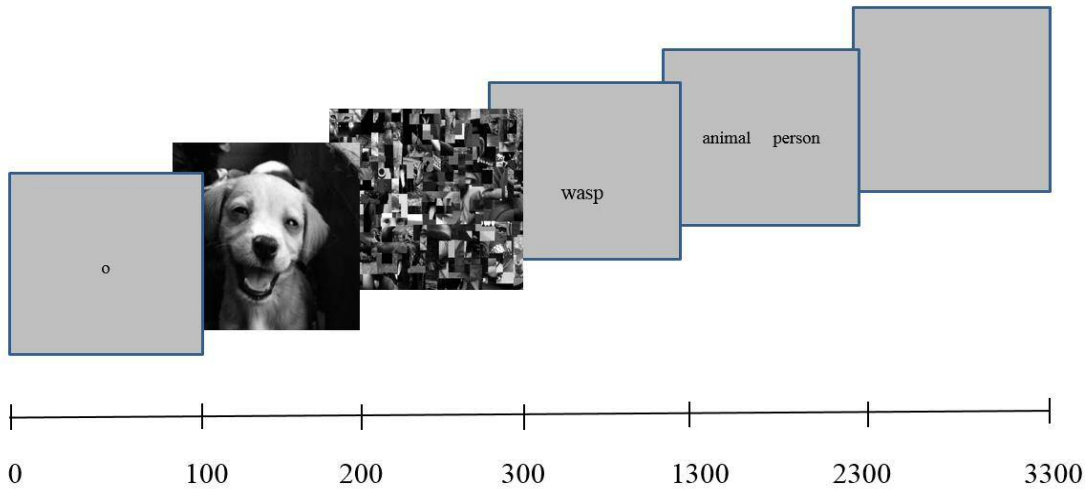


Figure 14a. Semantic Priming: Finalised trial structure for prime and target trials

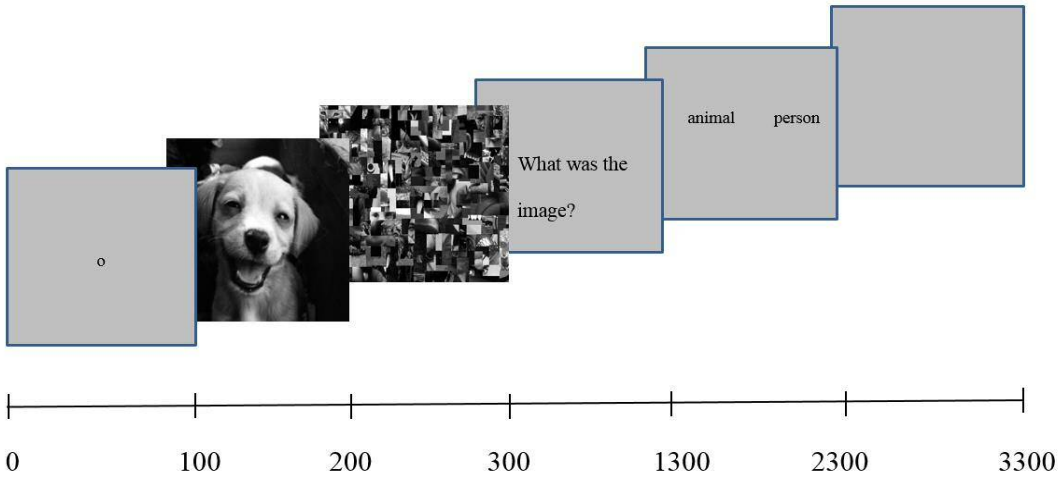


Figure 14b. Semantic Priming: Finalised trial structure for prime-only (Attend) trials

2.4.4.2. Results and Discussion

Response times were dealt with as in the Affective Priming task (3.45% of all trials removed as outliers).

2.4.4.2.1. Prime evaluation data

Participants performed at better than chance levels; the mean score was .84 (*SD* =.17; Range = .50 – 1.00) which a one sample *t*-test indicated was significantly different from zero, $t(14) = 20.26$, $p < .001$, $d_z = 5.41$.

2.4.4.2.2. Response Time data

Overall RT to all trial configurations were assessed for skew and kurtosis. All z-scores for skewness and kurtosis were below 2.58 ($p > .01$) therefore no transformations were necessary. A 2x2 repeated measures ANOVA was conducted on the RT for semantic (congruent, incongruent) and affective (congruent, incongruent) categories and indicated a significant main effect of semantic category: $F(1, 14) = 10.62$, $p < .01$, $\eta_G^2 = .17$, see Figure 15. There was no main effect of affective category ($F(1, 14) = .00$, $p > .05$) and no significant affective by semantic interaction ($F(1, 14) = .1.41$, $p > .05$). Planned analysis indicated a significant difference between semantically congruent and semantically incongruent trials: $t(14) = 3.26$, $p < .01$, $d_z = 0.87$.

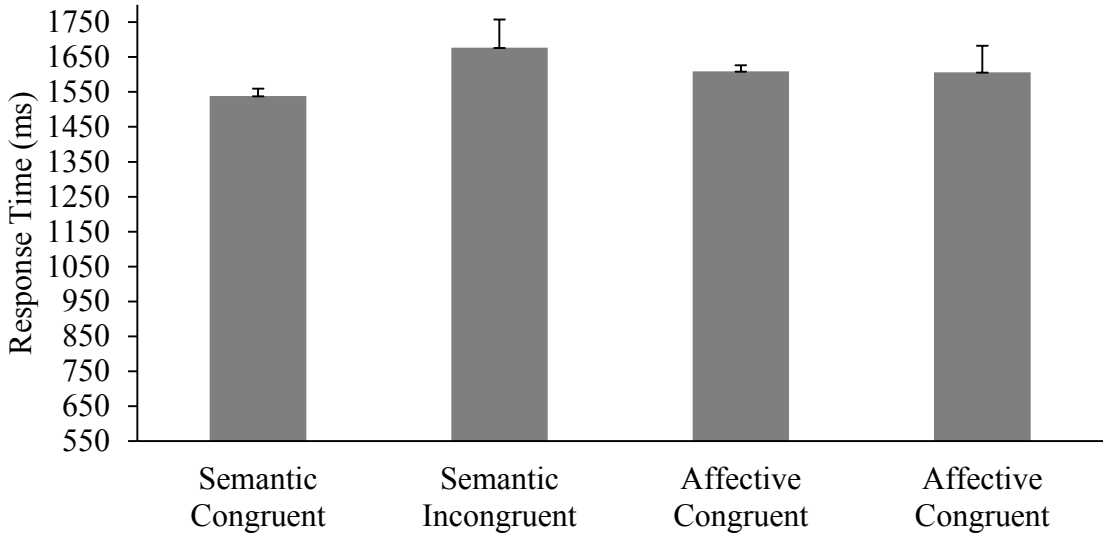


Figure 15. Mean response times (ms) per trial category: Semantic Priming task year 3 data collection. Error bars display 2SE (correction for repeated measures designs, Cousineau (2005) and Morey (2008)).

With only a main effect of semantic priming, the results indicate the utility of the Voss et al. (2013) design for separating affective and semantic priming. The essential separation of affective and semantic priming necessarily depends on how the relevant content is defined (Avero and Calvo, 2006). The semantic task required a categorical identification, bringing about a more refined analysis of specific features of the stimulus (Avero and Calvo, 2006).

2.5 Summary

Extensive preparatory work resulted in a stimulus set matched on various dimensions, and a group of experimental tasks that produced robust effects, allowing for the examination of performance modulated by individual differences.

Chapter 3: General Methods

This chapter lays out the common methodology in relation to the forensic sample, providing information on participants, materials, data treatment and experimental procedure. Subsequent experimental chapters are ordered by paradigm.

3.1 Power analysis

The statistical power of a test is the probability that it will correctly reject the null hypothesis when the null hypothesis is false; the probability of not committing a Type II error (Rothman, 1990). This probability is referred to as the false negative rate (β), and power is equal to 1 – β (Button, Ioannidis, Mokrysz, Nosek, Flint, Robinson et al., 2013). In order to have sufficient power to detect an effect of individual differences (psychopathy) on task performance using multiple regressions analyses, with a medium effect size and 6 or less predictors, Miles and Shevlin (2001) recommend a sample of approximately 100 participants. This was supported by G*Power (Faul, Erdfelder, Lang and Buchner, 2007) calculations. The G*Power calculations utilised a power of 80%, meaning that if there were 100 genuine non-null effects to be discovered, these studies expected to discover 80 of them (considered to represent adequate power, see Button et al., 2013, but see Bacchetti, 2013, for criticisms); an effect size of 0.13 (but see section 3.4.2.4), alpha level set at $p < .05$ and number of predictors set at 6 (based on the largest planned model of 3 predictors and 3 covariates). Results of these G*Power calculations indicated a required sample size of 98. Thus the present research set out to recruit a sample of approximately 100 participants.

3.2 Participants

All adult male offenders from a Category C UK prison (HMP Channings Wood) were invited to participate in the research, provided they were not currently being supported by the Assessment, Care in Custody and Teamwork (ACCT) policy (offenders identified as at-risk for self-harm). An offender population, as opposed to a community population, was chosen due to the higher prevalence of psychopathy among criminal populations and the availability of the collateral information required for reliable assessments (Hare and Neumann, 2010). Offenders were excluded from participation if settled within the resettlement and drug therapeutic units due to them being potentially different in therapeutic experiences when compared to the remaining prison population. Study participants (N = 94) voluntarily participated in the research and due to Ministry of Justice regulations received no financial or other incentives for participation. Specific sample sizes for each task are provided in the experimental chapters. Sample characteristics are provided in Table 12.

The study was advertised through leaflets distributed throughout the prison. Interested offenders then volunteered by returning the leaflets with their name and location details added. After a check as to whether the offenders were presently supported by the ACCT policy, offenders were sent an information sheet compiled in line with National Offender Management Service (NOMS) guidelines, which provided full details of the study and emphasised that participants would receive no benefit (i.e., reduced sentence or financial compensation) from study completion. Participants also

received a movement slip according to site policy providing them with details of the study session time and location[23].

The sample was predominantly White and of average intelligence. Years of education were calculated as follows: no formal qualifications, years approximated by participant; GCSE or equivalent, 12 years in education; A-Level or equivalent, 14 years in education; undergraduate degree, 17 years in education; postgraduate degree, 18 years in education. Five participants refused to complete the intelligence assessment due to disinterest but were deemed eligible, based on file information, to consent to such an assessment. The majority of participants were right handed (Right = 92.55%, Left = 5.32%, Ambidextrous = 2.13%). The number of prior convictions ranged from 0 to 93. Current sentence length ranged from 1 to 444 months (M = 58.19, SD = 83.56). Over half the sample were serving a sentence for a sexual offence (breach of sex offender order, arranging/facilitating a child sex offence, making/possessing/distributing indecent images/pseudo images of a child or children, sexual grooming, familial sexual offences, indecent/sexual assault, sexual offence against a child, gross indecency with child under 16, rape) at the time of study completion. The remainder of the sample were serving sentences for violent, fraud or drug related offences at the time of study completion: possessing an offensive weapon in a public place, affray, assault, wounding and other acts endangering life, grievous bodily harm (GBH), aggravated bodily harm (ABH), manslaughter, arson, false

[23] Approximately five potential participants declined to participate after receiving the full information sheet. As consent had not been granted for a file review of these offenders, it is impossible to ascertain whether these offenders differed in meaningful ways from the study participants.

imprisonment, theft, burglary, aggravated burglary, robbery, dishonesty, fraud, misuse of drugs, production of/being concerned in the production of a controlled drug, drug import, driving offences, or breach of licence. Over half the sample had a history of violent offending.

Twenty-four participants were receiving psychotropic medication for issues ranging from depression to psychosis. Precise details of dosages were not available from psychological or prison file notes; access to medical files was not requested prior to study commencement and seeking consent was not possible following study completion for practical reasons, therefore these details are not available. Two participants had epilepsy and were receiving appropriate medication, two participants had a clinical diagnosis of schizophrenia and four participants had a clinical diagnosis of personality disorder. Three participants disclosed an attention disorder (Attention Deficit Hyperactivity Disorder; ADHD) diagnosis to the researcher; an official diagnosis was not found on their psychological or prison notes but this information was retained for descriptive purposes.

As discussed further in section 5.3.1, psychotropic medications have a varied impact on pupil reactivity; use of psychotropic medications is also associated with subjective reports of blunted affect (Price, Cole and Goodwin, 2009), increased neural activation to positive affect and decreased activation to negative affect (Ma, 2015). Absence/presence of psychotropic medication was therefore included as a broad, dummy-coded covariate in all regression models. Descriptive information on task performance as a function of individual medications is presented in the appendices.

Table 12

Descriptive statistics for the sample (N = 94)

	M (*SD)* [reliability][b]
Age	40.41 (12.70)
PCL:SV[a]	
Total	12.70 (4.60) [.79]
Interpersonal-Affective	5.76 (2.80) [.78]
Impulsive-Antisocial	6.91 (2.99) [.77]
TriPM[c]	
Boldness	29.14 (7.97) [.79]
Meanness	10.50 (9.12) [.89]
Disinhibition	23.97 (12.80) [.90]
Years of education	9.56 (3.28)
2-item WASI IQ (*n* = 89)	99.47 (15.05)
Age at first conviction	22.79 (10.68)
Number previous offences	16.43 (24.03)
Number previous convictions	10.34 (15.27)
Ethnicity	%
Caucasian	91.00
Black	4.00
Mixed Race	4.00
Middle Eastern	1.00
Index offence	
Sexual	54.00
Violent	24.00
Acquisition	14.00
Drug related	5.00
Fraud/deception	3.00
History of violence	54.00
Medication intake[d]	*n*
SSRI[e]	10
AED[f]	4
Antipsychotics	4
NaSSA[g]	3
Benzodiazepine	3
Comorbidity	
Depressive disorder	10
Learning difficulty	5
Personality Disorder	4
ADHD	3
Schizophrenia	2
Epilepsy	2

Note: [a]Psychopathy Checklist: Screening Version. [b]Cronbach's α values. [c]Triarchic Psychopathy Measure. [d]Participants may have had more than one medication intake or no medication at all. [e]Selective Serotonin Reuptake Inhibitor. [f]Anti Epiletic Drugs. [g]Noradrenergic and Specific Serotonergic antidepressants.

3.3 Materials

3.3.1 Presentation hardware and software

A Direct RT program controlled presentation of the stimuli and recorded response latencies for all behavioural tasks (Affect Categorisation task, Affective Priming task and Semantic Priming task). Stimuli in the task assessing physiological reactivity were presented using E-Prime Professional software and pupil diameter was assessed by the Tobii X2-60 Hz eye system. All stimuli were presented in the centre of the monitor, against the light grey background of a Toshiba TECRA W50-A-102 laptop (17" screen).

3.3.2 Stimuli

Full details of the 30 affective and 10 neutral test images used are provided in section 2.2.4.

3.3.3 Measurement of psychopathy

3.3.3.1. The Psychopathy Checklist: Screening Version

The Psychopathy Checklist: Screening Version (PCL:SV, Hart et al., 1995) is a derivative of the Psychopathy Checklist: Revised (PCL:R, Hare, 1991, 2003), initially designed for use in non-forensic contexts but also used as a standalone instrument for assessing psychopathy. The PCL:SV is strongly related to the PCL-R, both conceptually and empirically (Cooke, Michie, Hart and Hare, 1999, Guy and Douglas 2006) and is validated in both community and forensic samples (Hart et al., 1995) with good internal consistency (see, e.g., Hughes, Dolan and Stout, 2013).

As the PCL:SV was derived from the PCL:R, details of the latter are presented here. The PCL-R is a clinical construct rating scale that uses a semi-structured interview, case history information and specific scoring criteria to rate each of 20 items on a 3 point scale (0, 1, 2) according to the extent to which it applies to a particular individual. These 20 items were derived from traditional clinical writings on psychopathy, combining personality traits and antisocial behaviours and are described in detail in the PCL:R manual. The items are summed (and prorated if items were omitted) to produce a total psychopathy score; this total score ranges from 0 - 40 reflecting the degree to which a given individual matches the prototypical psychopath. A cut-off score of 30 or greater is used to diagnose psychopathy. The items can also be summed to produce the different dimensions of psychopathy. Eighteen of the items form four facets, which combine to form two factors (Hare, 2003): Facet 1: Interpersonal and Facet 2: Affective contribute to the PCL-R Factor 1: Interpersonal/Affective; Facet 3: Lifestyle and Facet 4: Antisocial contribute to PCL-R Factor 2: Lifestyle/Antisocial (see Table 13 for item loadings). Two items (promiscuous sexual behaviour, many short-term relationships) do not load onto either factor but contribute to the overall PCL-R score. Empirical studies provide considerable support for the dimensions of psychopathy as represented by the PCL-R, for both the 4-facet structure and a super ordinate factor (PCL-R total), across diverse and large samples of male and female offenders, forensic and civil psychiatric populations, youth offenders, and community samples (see Hare and Neumann, 2008).

Table 13

Items in the Hare Psychopathy Checklist: Revised

Item	Factor loading
Glibness/superficial charm	1
Grandiose sense of self-worth	1
Need for stimulation/proneness to boredom	2
Pathological lying	1
Conning/manipulative	1
Lack of remorse or guilt	1
Shallow affect	1
Callous/lack of empathy	1
Parasitic lifestyle	2
Poor behavioural controls	2
Promiscuous sexual behaviour	-
Early behaviour problems	2
Lack of realistic, long term goals	2
Impulsivity	2
Irresponsibility	2
Failure to accept responsibility for own actions	1
Many short-term marital relationships	-
Juvenile delinquency	2
Revocation of criminal release	2
Criminal versatility	2

Note. Factor 1 = Interpersonal-Affective, Factor 2 = Impulsive-Antisocial. A dash indicates the item does not load on either factor.

One disadvantage of the PCL:R is that administration requires access to both file information and the completion of a lengthy interview. The PCL:R is therefore costly to administer in terms of time and effort (Cooke et al., 1999; Hart et al., 1995). These considerations led to the development of the PCL-SV. The PCL:SV consists of 12 items indicative of psychopathic traits completed through a review of institutional file information. Each trait is scored on a 3-point scale (0 = clearly not present; 1 = may be present; 2 = clearly present); the items are summed (and prorated if up to two items are omitted) to give a maximum score of 24. A cut-off score of 18 or greater, approximately equivalent to a score of 30 on the PCL:R, is used to indicate psychopathy. The measurement produces both a total

score and two dimensions. In the present work, PCL:SV Interpersonal-Affective (Factor 1) reflects *primary psychopathic traits*, whereas PCL:SV Impulsive-Antisocial (Factor 2; see Table 14 for item loadings) [24] reflects *secondary psychopathic traits*. Factor 1 and Factor 2 are, typically, moderately correlated. The item descriptions in the PCL:SV manual are brief as compared to the PCL:R manual and require less information to score. The descriptions are not exhaustive; the rater is encouraged to use the descriptions to form a prototypical idea of the characteristic and compare the individual being rated to the prototype (Hart et al., 1995). Many of the PCL:SV items were born out of the PCL:R items, by simplifying and shortening the latter without losing their meaning (see Table 14).

Table 14

Items in the Hare Psychopathy Checklist: Screening Version

Item	Factor loading	Corresponding PCL:R item(s)
Superficial	1	1
Grandiose	1	2
Deceitful	1	4, 5
Lacks remorse	1	6
Lacks empathy	1	7, 8
Doesn't accept responsibility	1	16
Impulsive	2	3, 14
Poor behaviour controls	2	10
Lacks goals	2	9, 13
Irresponsible	2	15
Adolescent antisocial behaviour	2	12, 18
Adult antisocial behaviour	2	19, 20

Note. Factor 1 = Interpersonal-Affective, Factor 2 = Impulsive-Antisocial.

[24] As noted, the present work utilises the two factor structure. Reliability of the four facets in the present work was inconsistent as compared to reliability for the two factors: Facet 1: Interpersonal $\alpha = .70$, Facet 2: Affective $\alpha = .64$, Facet 3: Lifestyle $\alpha = .80$, Facet 4: Antisocial $\alpha = .50$.

Means, standard deviations and reliability scores for the PCL:SV in the present sample are provided in Table 12, correlations with the self-report psychopathy measure and intelligence are provided in Table 15. Average psychopathy scores compare well to previous studies utilising the PCL:SV in adult male offender samples (Hughes et al., 2013; PCL:SV Total M = 12, Factor 1 M = 5.20, Factor 2 M = 6.70).

PCL:SV's were rated, on the basis of the offenders lifetime functioning, based on file review (criminal record, presentence report, Offender Assessment System (OASys) reports, behavioural notes, mental health evaluations, victim statements, treatment programme reports), behavioural observations and collateral information from wing officers and treatment programme staff, by trained doctoral level raters whose individual reliabilities had been checked via the Darkstone programme of PCL:R/SV training (intra-class correlation coefficient for the two raters total PCL:R was .88, $F(5, 5) = 47.40$, $p < .001$). Raters received training on the nature and assessment of psychopathy; the PCL:R/SV assessment procedure, and PCL:R/SV scoring. Ratings on eight practice cases were completed prior to administration of the PCL:SV during data collection[25].

[25] The researcher did not conduct a semi-structured interview in order to score the PCL:SV items. Similar approaches have been taken when assessing psychopathy in offender (Kropp, Hart, Lyon and Storey, 2011), mentally disordered sex offender (Craissati and Blundell, 2013) and forensic psychiatric samples (Arbach-Lucioni, Andres-Pueyo, Pomarol-Clotet and Gomar-Sones, 2011; Desmarais, Nicholls, Wilson and Brink, 2012). This approach was taken in order to undertake data collection efficiently, in light of the richness of collateral and file information available for each offender. Hart et al. (1995) have stated that scoring of the PCL:SV without the semi-structured interview is possible but that the administration of the PCL:SV under these non-standard conditions be noted.

3.3.3.2. The Triarchic Model

As well as assessing psychopathy through the PCL:SV, levels of the disorder were measured through the Triarchic Psychopathy Measure (TriPM, Patrick, 2010). The TriPM was of interest due to the proposed ability of the measure of illustrate adaptive aspects of psychopathy which other measures are said to miss, as well as its short administration time.

As discussed in section 1.2.2.3, Patrick's work on dual processes within psychopathy led to the development of the TriPM. Patrick, Hicks, Nichol and Krueger (2007) found support for a bifactor model of psychopathy as assessed by the PCL-R; results indicated a general factor on which all items of the PCL-R loaded significantly, along with three separate subfactors on which particular subsets of PCL-R items loaded. In particular, the Interpersonal subfactor reflected interpersonal surgency and a relative absence of experienced anxiety and fear. According to Patrick et al. (2007), the Interpersonal subfactor tapped something of the positive adjustment features of psychopathy emphasized by Cleckley.

Following on from this, a review (Patrick et al., 2009) of historic and contemporary efforts to conceptualize psychopathy revealed three prominent recurring themes: boldness, meanness and disinhibition. These three constructs although occasionally empirically inter-related have distinct phenotypic identities and can be conceptualized, measured and understood separately. Patrick (2010) developed the TriPM to index these three domains through three scales (TriPM Boldness, TriPM Meanness and TriPM Disinhibition). The development of the measure aimed to operationalize facets of psychopathy in physiological terms with biobehavioural referents and neurophysiological indicators

(Patrick and Drislane, 2015). Patrick and colleagues (Skeem et al., 2011) have emphasised that the triarchic model is a descriptive model and should be used to inform etiologic models of psychopathy such as the *dual-process model*. From the *dual-process model* perspective, boldness and, to a lesser extent, meanness, reflect a temperamental deficit in low fear, whereas *secondary psychopathic traits* (referred to as *externalising vulnerability* by Patrick and Bernat, 2009) reflect disinhibition and meanness (Skeem et al., 2011). This approach has been taken in existing research mapping the Triarchic Model onto the *dual-process model* (e.g., Pasion et al., 2016). Patrick et al. (2009) situate the psychopathy dimensions within the developmental psychopathology literature, in the context of the early-onset pathway to antisocial behaviour posited by Moffitt (1993). Boldness and meanness have roots in dispositional fearlessness, while difficult temperament, failure of secure attachment and coercive exchanges are relevant to disinhibition and also meanness (Patrick et al., 2009).

The TriPM has been used in a range of populations, including male and female offenders, drug treatment programme patients, mixed gender undergraduate samples and community samples (Craig, Gray and Snowden, 2013; Drislane, Patrick and Arsal, 2014; Sellbom and Phillips, 2013; Stanley, Wygant and Sellbom, 2013; Strickland, Drislane, Lucy, Krueger and Patrick, 2013). Research findings indicate that the TriPM domains capture a substantial amount of variance in established self-report psychopathy measures and established personality measures within female (Sellbom and Phillips, 2013) and male (Stanley et al., 2013) offender samples and in mixed gender undergraduate samples (Donnellan and Burt, 2016; Drislane et al., 2014; Poy, Segarra, Esteller, Lopez and Molto, 2014). While the majority of existing work utilising the TriPM has focused on supporting the validity of the TriPM operationalisation (see Evans and Tully, 2016, for a review), findings also indicate that beyond

criterion validation work the TriPM has utility in correlational (Craig et al., 2013) and imaging studies (Vieira, Almeida, Ferreira-Santos, Barbosa, Marques-Teixeira and Marsh, 2015).

3.3.3.2.1. Boldness

Boldness is hypothesised to be represented by low fear and weak defensive reactivity, with impaired threat potentiated startle and amygdala reactivity to fearful faces as neurophysiological indicators. TriPM Boldness encompasses *primary psychopathic traits*. Related to the PCL-R Interpersonal facet (Charm, Grandiosity, Deceitfulness and Manipulativeness) and the PPI-R Fearless Dominance factor (see Stanley et al., 2013), TriPM Boldness is epitomised by high self-assurance and social efficacy (social dominance), a tolerance for unfamiliarity and danger (thrill-adventure seeking; Sellbom and Phillips, 2013) and the capacity to remain calm and focused in situations involving pressure or threat. Boldness manifests behaviourally as imperturbability, social poise, assertiveness and persuasiveness, bravery and venturesomeness (i.e., low negative emotionality) and is characterised by increased BAS activity (Donahue and Caraballo, 2015). Research indicates a strong correlation between TriPM Boldness and positive emotionality (Sellbom and Phillips, 2013) in the form of psychobiological approach systems, emphasising Cleckley's positive adjustment characteristics that contribute to the mask of sanity. TriPM Boldness is most related to features involving resilience and social efficacy as indexed by other measures of psychopathy (Patrick and Drislane, 2015). A recent study by Wall, Wygant and Sellbom (2015) used hierarchical regression analyses to demonstrate that TriPM Boldness contributed incrementally to prediction of PCL:R Factor 1, and the Interpersonal facet specifically, over and above variance accounted for by ASPD symptoms.

3.3.3.2.2. Meanness

Meanness is considered to represent the intersection between externalising psychopathology and the core interpersonal-affective features of psychopathy (Venables, Hall and Patrick, 2014). TriPM Meanness is contained in both *primary* and *secondary psychopathic traits*. It is likely that environmental factors such as failed attachment and early abuse as well as genes for low affiliation underscore the difference between the phenotypic expression of Meanness and Boldness. TriPM Meanness is central to conceptions of psychopathy in criminal and delinquent samples. With tendencies towards callousness and deficient empathy (Sellbom and Phillips, 2013), narcissistic feelings of entitlement and egocentricity (Stanley et al., 2013), empowerment through cruelty, predatory aggression, rebelliousness and excitement seeking, Meanness indicates a profound lack of social connectedness (Patrick and Drislane, 2015). TriPM Meanness is most related to the deficient affect features of other psychopathy measures (see Patrick and Drislane, 2015): the PCL-R Affective facet (Callousness, absence of remorse, shallow affect and blame externalisation) and the Coldheartedness subscale of the PPI. TriPM Meanness manifests behaviourally in the active exploitation of and confrontation with others, arrogance and verbal decisiveness, defiance of authority, lack of close personal relationships, aggressive competitiveness, physical cruelty toward people and animals, predatory (proactive, premeditated) aggression, strategic exploitation of others for gain, and excitement seeking through destructiveness. TriPM Meanness, when underpinning *secondary psychopathic traits,* is also associated with hostility, social withdrawal and depression (Strickland et al., 2013). Individuals high on TriPM Meanness are characterised by increased BIS and decreased BAS activity (Donahue and Caraballo, 2015).

3.3.3.2.3. Disinhibition

Secondary psychopathic traits are reflected in TriPM Disinhibition. Disinhibition is characterised by impaired regulation of affect and urges (impulsiveness, oppositionality, anger/hostility, i.e., high negative emotionality), insistence on immediate gratification and deficient behavioural restraint (irresponsibility). It is associated with the impulsive lifestyle features of psychopathy, as indexed by other psychopathy measures: the Antisocial deviance of the PCL-R Factor 2 and the impulsive antisocial component of the PPI-R (PPI-R-II; see Patrick and Drislane, 2015). Disinhibition in the context of psychopathy is hypothesised to stem from deficient inhibitory control, indicated neurophysiologically by P300 and ERN responses (Nelson, Patrick and Bernat, 2011) and increased BAS activity (Donahue and Caraballo, 2015). Disinhibition manifests behaviourally as aggressive acting out (angry reactive-aggression), proneness to substance misuse, engagement in illicit or other norm-violating activities, alienation and distrust. Research links TriPM Disinhibition with lower levels of dependability and self-discipline and higher degrees of emotional instability (Stanley et al., 2013) as well as impulsive and socially deviant behaviour (Sellbom and Phillips, 2013). Patrick et al. (2009) note that high scores on Disinhibition alone would not qualify an individual to be considered high on psychopathic traits as this might only be a reflection of broad externalising psychopathology; an individual would also need to present with high levels of dispositional Boldness and/or Meanness.

3.3.3.2.4 The Triarchic Psychopathy Measure

The TriPM (Patrick, 2010) is a 58-item self-report measure designed to assess the three distinct components of psychopathy described in the Triarchic model (Patrick et al., 2009). Items are scored using a 4 point scale: False = 0, Somewhat False = 1, Somewhat True = 2 and True = 3. The TriPM yields scores on three subscales reflecting Boldness, Meanness and Disinhibition. TriPM Meanness and Disinhibition are typically positively correlated, with a more modest positive correlation between TriPM Meanness and Boldness and a negative relationship or lack of correlation between TriPM Boldness and Disinhibition (Patrick and Drislane, 2015). Previous work utilising the TriPM has indicated good reliability, with Cronbach's alpha values in a female offender sample of Boldness α = .89, Meanness α = .90, and Disinhibition α = .89 (Sellbom and Phillips, 2013). Means and standard deviations of the TriPM scales in the present sample are provided in Table 12; correlations with the PCL:SV and intelligence are provided in Table 15; the TriPM Meanness scale was positively skewed therefore this scale was square root transformed for use in all inferential statistics[26]. Average psychopathy scores are lower than those reported in existing offender samples (e.g., Stanley et al., 2013: Boldness *M* = 50.70; Meanness *M* = 41.50; Disinhibition *M* = 53.70). However, Stanley et al. (2013) utilised a Likert scale with a 1 – 4 scoring whereas the present research utilised a Likert scale with a 0 – 3 scoring. The TriPM manual (Patrick, 2010) does not specify as to how to score the item

[26] Due to the skewed distribution, descriptive statistics for TriPM Meanness were calculated by taking the mean and standard deviation of the square root transformed scale and back-transforming these values (i.e., through squaring; see Miles and Shevlin, 2001, page 3).

scale. Other work has scored the TriPM items on both a 0 – 3 scale (Esteller et al., 2016; Vieira et al., 2015; Strickland et al., 2013; non-offender samples), a 1 - 4 scale (Donnellan and Burt, 2016; Sellbom and Phillips, 2013) or not specified (Almeida, Seixas, Ferreira-Santos, Vieira, Paiva, Moreira et al., 2015; Drislane et al., 2014; Crego and Widiger, 2014; Poy et al., 2014). Thus in this instance comparison with an appropriate offender sample is difficult. However, as the present work takes a dimensional approach to psychopathy, absolute values are perhaps not as relevant as the reliability of the scales – which in the present sample are good (see Table 12).

3.3.3.3. Measure coherence

Zero-order correlations between the psychopathy measures are presented in Table 15. Scales broadly reflecting *secondary psychopathic traits* were positively correlated (PCL:SV Factor 2 and TriPM Disinhibition). Scales broadly reflecting *primary psychopathic traits* (PCL:SV Factor 1 and TriPM Boldness) were not significantly correlated. TriPM Meanness was positively correlated with PCL:SV Factor 2, TriPM Disinhibition and TriPM Boldness. When examining partial correlations to control for the shared variance between the PCL:SV factors, PCL:SV Factor 1 was positively correlated with TriPM Boldness (partial $r = .21$, $p < .05$) but remained uncorrelated with TriPM Meanness (partial $r = .06$, $p > .05$). The magnitude of the associations between PCL:SV Factor 2 and TriPM Meanness and between Factor 2 and TriPM Disinhibition did not change when controlling for the shared variance with PCL:SV Factor 1. The only psychopathy measure to show a relationship with age was TriPM Disinhibition.

3.3.4 Measurement of intelligence

3.3.4.1 The WASI

Participants completed the 2 sub-test Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999) at the end of the experimental session to assess for levels of intelligence (IQ). The 2 sub-test WASI is a brief measure of IQ based on the respondent's scores on a Vocabulary and Matrix Reasoning task and produces the FSIQ-2 (Full Scale IQ). The WASI Vocabulary subtest requires participants to orally define words and measures an examinees word knowledge and verbal concept formation. The Matrix Reasoning subtest consists of incomplete gridded patterns which the participant completes by choosing the correct response; this subtest taps fluid intelligence, broad visual intelligence, classification and spatial ability, knowledge of part-whole relationships, simultaneous processing and perceptual organisation. The WASI was chosen for inclusion due to its brief administration time (< 15 minutes) whilst demonstrating convergence with more intensive indices of IQ (e.g., the WISC-III and WAIS-III; see Wechsler, 1999). Mean IQ for the sample is presented in Table 12, and relationships with the two psychopathy measures are presented in Table 15. WASI scores were not obtained from five participants due to refusal. These participants were deemed eligible to consent but refused to undertake the WASI assessment at the end of the test session due to disinterest.

Table 15

Zero-order correlations between PCL:SV and TriPM scales, age and intelligence

	1	2	3	4	5	6	7	8
1. PCL:SV Total	---							
2. PCL:SV Factor 1	.78**	--						
3. PCL:SV Factor 2	.81**	.27**	--					
4. TriPM Boldness	.02	.13	-.13	--				
5. TriPM Meanness	.32**	.14	.36**	.23*	--			
6.TriPM Disinhibition	.46**	.07	.66**	-.13	.63**	--		
7. IQ	-.11	.09	-.27*	.16	-.20	-.24*	--	
8. Age	.02	.19	-.15	.07	-.13	-.28**	.29**	--

Note: PCL:SV Factor 1 = Interpersonal and Affective deficits; PCL:SV Factor 2 = Impulsive and Antisocial Behaviours.

* $p < .05$ ** $p < .01$

3.3.4.2. Intelligence and psychopathy

Behavioural work on psychopathy often includes a measure of IQ as a covariate in analyses, particularly where measures of IQ are correlated with task performance (e.g., Book et al., 2007). IQ scores are obtained to ensure that intelligence differences do not account for relationships between study variables. Studies that use a between groups design may include a measure of IQ in order to match psychopathy and control groups on this variable, e.g., on operant response tasks (Mitchell et al., 2006), emotion categorization tasks (Anderson and Stanford, 2012), facial affect recognition tasks (Glass and Newman, 2006), tasks assessing the neural processing of facial affect (Decety, Skelly, Yoder and Kiehl, 2014) and affective and semantic priming tasks (Blair, Richell, Mitchell, Leonard, Morton and Blair, 2006).

Some have suggested that the use of a covariate such as IQ in analyses is often misguided (Miller and Chapman, 2001). Analyses of covariance are suggested to be inappropriate for pre-existing

groups (i.e., patient versus control groups) that naturally differ on the covariate. Dennis, Francis, Cirino, Schachar, Barnes and Fletcher (2009) further suggest that the use of IQ in particular as a covariate, in analyses of neurodevelopmental disorders, is inappropriate and has resulted in overcorrected results about neurocognitive functioning. They repeat the point of Miller and Chapman (2001) that attempting to adjust for the effects of IQ when it is an attribute of the disorder is meaningless.

The use of covariate variables is appropriate when the covariate is theoretically and empirically related to the independent variable (i.e., psychopathy), or if covariate is empirically related to the dependent variable (i.e., task performance). In his classic work, Cleckey (1955) described the psychopath as being of superior general intelligence, with the ability to charm, manipulative and deceive others. However, Hare and Neumann (2008) have suggested that the inclusion of this characteristic may have been a consequence of Cleckley's sample population: many of his patients were well-educated from wealthy backgrounds. For Hare and Neumann (2008), there is no obvious theoretical reason why psychopathy should be related to measures of intelligence.

The empirical evidence for IQ as a consistent attribute of psychopathy remains unclear. Psychopathic traits and verbal IQ have been found to positively related (DeLisi, Vaughn, Beaver and Wright, 2010). IQ and psychopathy have also previously been found to be negatively related (Walsh, Swogger and Kosson, 2004). Greater levels of violent activity occur among low IQ psychopathic offenders (Walsh et al., 2004). Neumann and Hare (2008) found that all psychopathy facets were negatively related to IQ across male and female samples, while others have found differential relationships between the psychopathy facets and IQ (Salekin, Neumann, Leistico and Zalot, 2004,

Vitacco, Neumann and Jackson, 2005). In the present sample, IQ was inversely related to indices of *secondary psychopathic traits*; PCL:SV Factor 2 and TriPM Disinhibition. TriPM Meanness was inversely related to IQ at a trend level (p = .06).

Relations between IQ and task performance are presented in each experimental chapter. No consistent relationship emerged, with only one significant relationship and one marginally significant relationship between IQ and 22 indices of task performance across the thesis. Therefore all multiple regression analyses presented in the experimental chapters were run without including IQ as a covariate; analyses re-run with IQ as an additional covariate are presented in the appendices. Any significant differences between these two sets of analyses are reported in the relevant experimental chapter.

3.4 Data treatment

Analyses were conducted using IBM SPSS Statistics 20.0 (referred to hereafter as SPSS).

3.4.1 Descriptive statistics and graphs

Bar charts present the mean +2 Standard Error (*SE*; repeated measures correction, Morey, 2008, Cousineau, 2005) of appropriate variables. The *SE* of the mean is the standard deviation (variability) of the means if the study was replicated multiple times (Streiner, 1996). In a normal distribution, ~95% of the data points falls between -1.96 and +1.96 *SD* of the mean (Cumming, Fidler and Vaux, 2007). This range of values can be referred to as a confidence interval (CI); defined by Morey, Hoekstra, Rouder, Lee and Wagenmakers (2016) as an interval generated by a procedure that, on repeated sampling, has a fixed probability of containing the parameter. Notwithstanding the concerns raised by

Morey et al. (2016) regarding the fallacies of using CI to assign probabilities (e.g., the probability that the observed interval contains the 'true' value), means are presented with +2 *SE* in all bar charts, as this method essentially shows the 95% CI (Streiner, 1996; Cumming et al., 2007)[27]. *SE* was calculated using the repeated measures correction suggested by Cousineau (2005) and refined by Morey (2008).

3.4.2. Inferential statistics

3.4.2.1. Repeated measures Analysis of Variance

Task efficacy (basic effects of experimental manipulations) were assessed using one-way or factorial repeated measures analyses of variance (ANOVA). Planned comparisons were conducted using paired *t*-tests and are labelled as such, specifically in the case of affective versus neutral content comparisons. Further exploratory comparisons, for example comparing affective categories to each-other, were labelled as such and conducted using paired *t*-tests. Corrected degrees of freedom are reported using the Huynh and Feldt correction where appropriate; i.e., if the sphericity assumption of repeated measures designs was violated (Field, 2005). The correction of Huynh and Feldt was applied as it is considered more powerful, and some researchers consider Greenhouse-Geisser to be too conservative (Abdi, 2010).

[27] Confidence intervals were not generated for effect sizes, a practice which is increasing in popularity, in an effort to avoid the pitfalls of this approach as discussed by Morey et al. (2016).

3.4.2.2 Correction for multiple comparisons

When a large number of statistical significance tests are conducted in a particular study, there is a common understanding that the alpha level (the Type I error rate) should be adjusted to reflect the number of tests being conducted. This correction for multiple comparisons is often undertaken through a Bonferroni correction – dividing the alpha level by the number of comparisons.

Several oft-cited papers (O' Keefe, 2003; Perneger, 1998; Rothman, 1990) have argued that the use of such alpha adjustments is unnecessary and should not generally be undertaken. Alpha adjustments are useful in certain circumstances such as when the universal null hypothesis is of interest and sequential testing of trial results (Perneger, 1998). However, statistical adjustment for multiple tests is incorrectly applied beyond these specific situations (Perneger, 1998) and also inconsistently applied (O' Keefe, 2003) being typically a feature of comparisons between cells but not in, e.g., hierarchical regression models. This inconsistent application raises queries as to whether there is a principled basis for applying alpha-adjustments (O' Keefe, 2003). For example, Bonferroni corrections imply that a given statistical test will be interpreted differently depending on how many other tests were performed, indicating that a difference between two groups on, e.g., task accuracy, may or may not be significant depending on whether or not, e.g., task response time and length of task completion time are also assessed. Any result could be deliberately undermined by additional significance testing, thus threatening the security of all research results (O' Keefe, 2003). Perneger (1998) believes that this 'defies common sense', and also notes that the conservative nature of Bonferroni corrections which directly target the Type I error problem do so at the expense of reducing the power to find a statistical effect where one exists (Type II error; Rothman, 1990). Although loss of power alone is not a

justification to avoid alpha adjustments (O' Keefe, 2003), the cost of Type I error risk inflation also cannot be localised (O' Keefe, 2003; Perneger, 1998). Type I error risk inflation is presented as a risk of the number of tests performed, but not a function of the datasets over which the tests are performed, the identity of the person who performs the tests, the time frame over which tests are conducted, or whether tests are performed sequentially or simultaneously (O' Keefe, 2003). Because there is no justifiable way of defining the localisation of Type I error risks, alpha adjustments based on the number of tests conducted are not justified (O' Keefe, 2003). Rather, each statistical association should be subject to critical evaluation (Rothman, 1990). Perneger (1998) suggests that the ideal approach is to integrate prior beliefs (i.e., to present results as planned and linked to specific hypotheses, or to present results as exploratory) and statistical approaches when discussing the possible interpretations of each result. For planned analyses, focused and specific hypotheses will be pursued in the present research. Significant results will be discussed in relation to effect size, adherence to specified hypotheses and existing literature.

3.4.2.3 Alpha levels

Levels of significance are reported to the nearest alpha level: $p < .001$, $p < .01$, $p < .05$. In the recent statement from the American Statistical Association regarding the use of *p* values (Wasserstein and Lazar, 2016), six principles regarding the context, process and purpose of *p* values were discussed, e.g., that scientific conclusions should not be based only on whether a *p* value passes a specific (arbitrary) threshold, that proper inference requires not just a report of *p* values but full reporting such as the extent of exploratory analysis on the data (see section 3.4.2.2), and that good science requires an assessment of the effect size found in an experiment (see section 3.4.2.4).

Relatedly, Pritschet, Powell and Horne (2016) note that the practice of describing results as marginally significant, although prevalent in psychology, is not supported by journal guidelines and also differs across different fields of psychology research. In an attempt to constrain the 'grey area' of marginal significance, but also to acknowledge the arbitrary nature of the $p < .05$ threshold, p values falling between $p = .05$ and $p = .07$ are reported as 'marginally significant' or at 'trend level'; the reader is invited to consider the effect size associated with these results. P values falling above $p = .07$ are considered non-significant and all non-significant p values are reported as $p > .05$. Results with a p value at trend level are reported in the appropriate Results section, however only significant results are reported in the relevant Discussion section.

3.4.2.4 Effect sizes

Effect sizes represent the magnitude of the effect of an independent variable on a dependent variable in a standardised metric (Lakens, 2013). Effect sizes for one sample and paired sample *t*-tests were reported using Cohen's d_z which describes the standardised mean difference of an effect for dependent designs. These effects were calculated using the effect size spreadsheet provided by Lakens (2013).

For ANOVA, eta squared (η^2) is one of the most widely used effect sizes in psychological research (Lakens, 2013). η^2 reflects the magnitude of effect within a single factor experiment. η^2 is based upon the proportion of total variability in the dependent variable accounted for by variation in the independent variable; the ratio of the between groups sums of squares to the total sums of squares (Levine and Hullett, 2002). η^2 can be interpreted in terms of the percentage of variance accounted for

by a model or variable (Levine and Hullett, 2002) given that the summed effects for all portions of the variance, including the error variance, comes to 1.00.

With a simple repeated measures design (i.e., one repeated measures variable), η^2 should be reported (Lakens, 2013). However, η^2 cannot be easily compared between studies, because the total variability in a study depends on the design of the study (Olejnik and Algina, 2003). Partial eta squared (η_p^2) may then improve the comparability of effect sizes across studies – this effect size relates the treatment effect to some (partial) of the variance, not affected by other features of the experiment. However, η_p^2 can only be used to compare effects between studies with similar experimental designs (Olejnik and Algina, 2003). Bakeman (2005) and Olejnik and Algina, (2003) suggest the use of generalised eta squared (η_G^2) as the preferable effect size for repeated measures designs (see also Levine and Hullett, 2002, for a criticism of both η^2 and η_p^2). η_G^2 excludes variation from other experimental factors to allow the magnitude to be comparable to other studies in which these factors were not manipulated. Effect sizes for ANOVA were therefore reported using η_G^2, calculated using the effect size spreadsheet provided by Lakens (2013). Lakens (2013) and Olejnik and Algina (2003) discuss the benchmarks provided by Cohen (1988) to define small (.02), medium (.13) and large (.26) effect sizes in relation to ANOVA, and note that using these benchmarks when interpreting η_G^2, as with all effect sizes, should be done with caution as this is not wholly consistent with the original considerations of these benchmarks.

3.5 Psychopathy: Correlational and regression analyses

3.5.1 The dimensional structure of psychopathy

MacCallum et al. (2002) provide statistical arguments against the dichotomization of individual difference measures. The authors note that this split results in a loss of effect size and power, making the detection of effects more difficult, as well as a loss of information about individual differences. The often variable cut-off scores used to subdivide participants into psychopathic or non-psychopathic groups could either hide or exaggerate effects, depending on the cut off used (Smith and Lilienfeld, 2015). The alternative method of creating extreme groups – comparing participants from the top and bottom of a distribution – can also exaggerate group differences. The unreliability of extreme - group designs also increases the risk of a Type I error (Smith and Lilienfeld, 2015).

At the measurement level, the latent structure of psychopathic traits is dimensional as opposed to taxonic (Edens et al., 2006; Guay et al., 2007). Psychopathic personality traits can be viewed as an extreme variant of multiple dimensions of personality (Hare and Neumann, 2008), consistent with literature indicating that personality disorders in general are dimensional in nature (Hare and Neumann, 2008). Hare and Neumann (2008), therefore, argue that it is more efficient to assess participants in terms of levels of psychopathic traits, as opposed to creating psychopathic and non-psychopathic groups. Psychopathic individuals differ from others in degree rather than kind (Edens et al., 2006). Guay et al. (2007) state that particularly in legal matters, individuals should be referred to

as being 'high on measures of psychopathy' as opposed to being 'a psychopath'[28]. In this regard, Skeem et al. (2011) have also noted that there is no consensus definition of symptom criteria that would allow an individual to be diagnosed as a psychopath.

3.5.2. Correlations and regression models

In order to assess relationships between task performance and the psychopathy dimensions, zero-order correlational analyses were used. Correlation coefficients (*r*) are the measure of the size and direction of the linear relationship between two variables (Miles and Shevlin, 2001). Multiple regression models, with measures of task performance regressed onto the psychopathy factors, were then used in order to examine the robustness of correlational results in the presence of covariates, and also adjusting for common variance between psychopathy scales.

Regression models allow for the analysis of the relationship between two variables – in this instance, a particular predictor (X) and criterion (Y) variable. Regression models produce both beta coefficients and partial and semi-partial coefficients, which are used to assess the relationship between the predictor and criterion variables. The beta coefficient for each predictor variable produced by the regression model reflects the number of standard deviations that the criterion will change as a result of one standard deviation change in the predictor. Therefore by looking at the betas of different

[28] Hare (2003) however believes that it is important to remember that the dimensionality of a personality disorder does not prevent the use of "diagnostic" thresholds for making clinical decisions.

predictor variables within one model it is easy to compare the relative importance of predictors in a model.

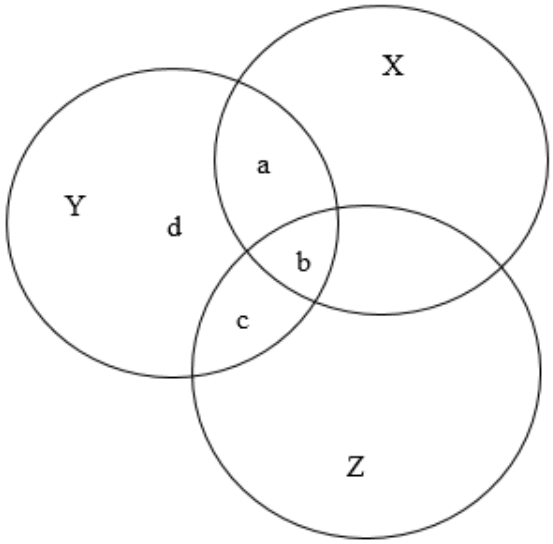
Regression models can also produce the semi-partial correlation for each predictor variable and the criterion variable. The semi-partial correlation shows the strength and direction of the relationship between a predictor and criterion variable while holding the effects of further predictor variables constant for the predictor only. After removing variance that the predictor has in common with other predictors, the semi-partial correlation expresses the correlation between the residualized predictor and the unaltered criterion variable. The square of the semi-partial correlation therefore reflects the proportion of the criterion associated uniquely with the predictor. Squared semi-partial correlations are more useful than partial correlations for measuring the importance of a predictor variable (Tabachnick and Fidell, 2007).

In standard multiple regression models, all predictor variables enter the equation at once: each one is assessed as if it had entered the regression after all other predictors had entered. Each predictor variable is evaluated in terms of what it adds to prediction of the criterion variable that is different from the predictability afforded by all the other predictor variables (Tabachnick and Fidell, 2007). In sequential or hierarchical multiple regression models, the most theoretically or statistically important predictor variables are entered first (step 1), followed by other less important variables (step 2). Alternatively, less important variables are entered first (step 1, the covariate approach) with theoretically important variables entered second (step 2). The hierarchical model is useful for assessing the incremental validity of a new predictor over and above existing predictors already in the model. Although the individual predictor betas produced by these two approaches are identical, reflecting the

ratio of standard deviations of the criterion and the predictor of interest, the interpretation of the squared semi-partial correlation varies depending on the type of regression model used as illustrated in Figure 16. In standard multiple regression, the squared semi-partial correlation more accurately reflects the unique contribution of each predictor to the criterion variable, as compared to hierarchical multiple regression, whereby the variable that is entered first (X) receives both its unique variance and the variance it shares with a second predictor variable (Z).

The present research is primarily interested in, with hypotheses focused on, the general and unique contributions of each predictor variable, i.e., each PCL:SV factor and each TriPM scale, as opposed to the overall contribution of either complete measure of psychopathy, to the task criterion variables. Therefore interest is focused on the beta coefficients and the squared semi-partial correlation coefficients as opposed to the model R^2. The individual contributions of each predictor variable are best expressed by the semi-partial correlations provided by standard multiple regression models, therefore the present research utilises standard regression models[29]. Hierarchical regression models are more appropriate for assessing the incremental validity of one predictor over and above the variance already explained by existing predictors (Miles and Shevlin, 2001), as represented by the change in R^2 (ΔR^2), which is not the focus of the present research.

[29] Lynam, Hoyle and Newman (2006) present a cautionary argument against the partialling out of predictor variables from each-other in regression models with reference to aggression and psychopathy; this issue is given further consideration in section 7.2.3.



		Standard Multiple	Hierarchical
Squared correlation	X	(a+b) / (a+b+c+d)	(a+b) / (a+b+c+d)
	Z	(c+b) / (a+b+c+d)	(c+b) / (a+b+c+d)
Squared semi-partial correlation	X	a / (a+b+c+d)	(a+b) / (a+b+c+d)
	Y	c / (a+b+c+d)	c / (a+b+c+d)
Squared partial correlation	X	a / (a+d)	(a+b) / (a+b+d)
	Y	c / (c+d)	c / (c+d)

Figure 16. Areas representing squared correlation, squared semi-partial correlation and squared partial correlation in standard multiple and hierarchical regression (where X is given priority over Z). Adapted from Tabachnick and Fidell, 2007, page 145.

Multiple regression models were conducted as follows, after the prioritised entry of any covariates: in an individual model, an index of emotion processing was regressed onto a psychopathy

scale, e.g., PCL:SV Factor 1, producing a beta coefficient for this scale. In a separate multiple regression model, the same index of emotion processing was regressed onto PCL:SV Factor 2 producing the beta coefficient for this scale. In a final multiple regression model, the index of emotion processing was regressed onto Factor 1 and Factor 2 simultaneously producing controlled beta coefficients for each scale and the appropriate semi-partial correlations which were then squared. This approach has been taken in existing work (Baskin-Sommers et al., 2015a).

Appropriate checks for multicollinearity were made for each regression model. Multicollinearity occurs when there is a strong correlation between two or more predictors in a regression model which reduces the ability to assess the importance of individual predictors. Through SPSS, multicollinearity can be evaluated with the variance inflation factor (VIF) and tolerance statistic. The VIF indicates whether a predictor has a strong relationship to other predictors, and the tolerance statistic is its reciprocal (1/VIF). In the present research the mean VIF across all regression models was 1.11 (Range = 1.01 – 1.16) and the mean tolerance statistic was .90 (Range = .86 - .99) indicating acceptable values (see Field, 2005).

3.5.3 Suppression effects

Multiple regression models also allowed for the examination of suppressor effects, by comparing the beta coefficients for each factor or scale of psychopathy individually and when present with the other factor or scales. A suppressor variable (Z), when entered into a multiple regression equation, removes or suppresses criterion-irrelevant variance from the initial variable (X; Paulhus, Robins, Trzesniewski and Tracy, 2004) and enhances the predictive validity of X (Friedman and Wall,

2005). The first formal discussion of suppression was by Horst (1941; as cited in Friedman and Wall, 2005) and would be referred to now as 'classical suppression' (see Figure 17). In this situation, a suppressor variable is an independent variable that is uncorrelated with the criterion variable but is correlated with the other predictor variable and when entered into the regression equation, increases the variance explained (R^2). The term suppression has since been applied to wider contexts (see Friedman and Wall, 2005) but essentially, a suppressor situation is one where the simultaneous inclusion of predictor variables improves the validities of both as compared to when each variable is entered alone. In a three variable framework (criterion variable Y and two predictor variables X and Z), Paulhus et al. (2004) detail three types of suppressor situations, illustrated in Figure 17.

a) Classical suppression: the original predictor (X) benefits from the entry of the new predictor (Z) that appears to have no predictive power. Z is composed entirely of variance irrelevant to the criterion, and X and Z overlap. Entering Z into the regression equation removes the irrelevant variance (manifested in its negative beta weight) and the underlying efficacy of X is revealed.
b) Reciprocal/cooperative suppression: the validities of the two predictors have opposite signs. Because the predictors are positively correlated, however, both validities are being restricted. Including them together in a regression equation controls for their overlap and their mutual suppression is revealed by boosts in both regression weights.
c) Net suppression: the three variables are all positively correlated, suggesting a redundancy situation rather than a suppressor situation. Its true identity as a suppressor situation is not

apparent until the regression weights are calculated and the smaller beta shows an opposite sign to its validity.

d) Redundancy situation (non-suppressor): positively correlated predictors have similar validities both separately and entered together.

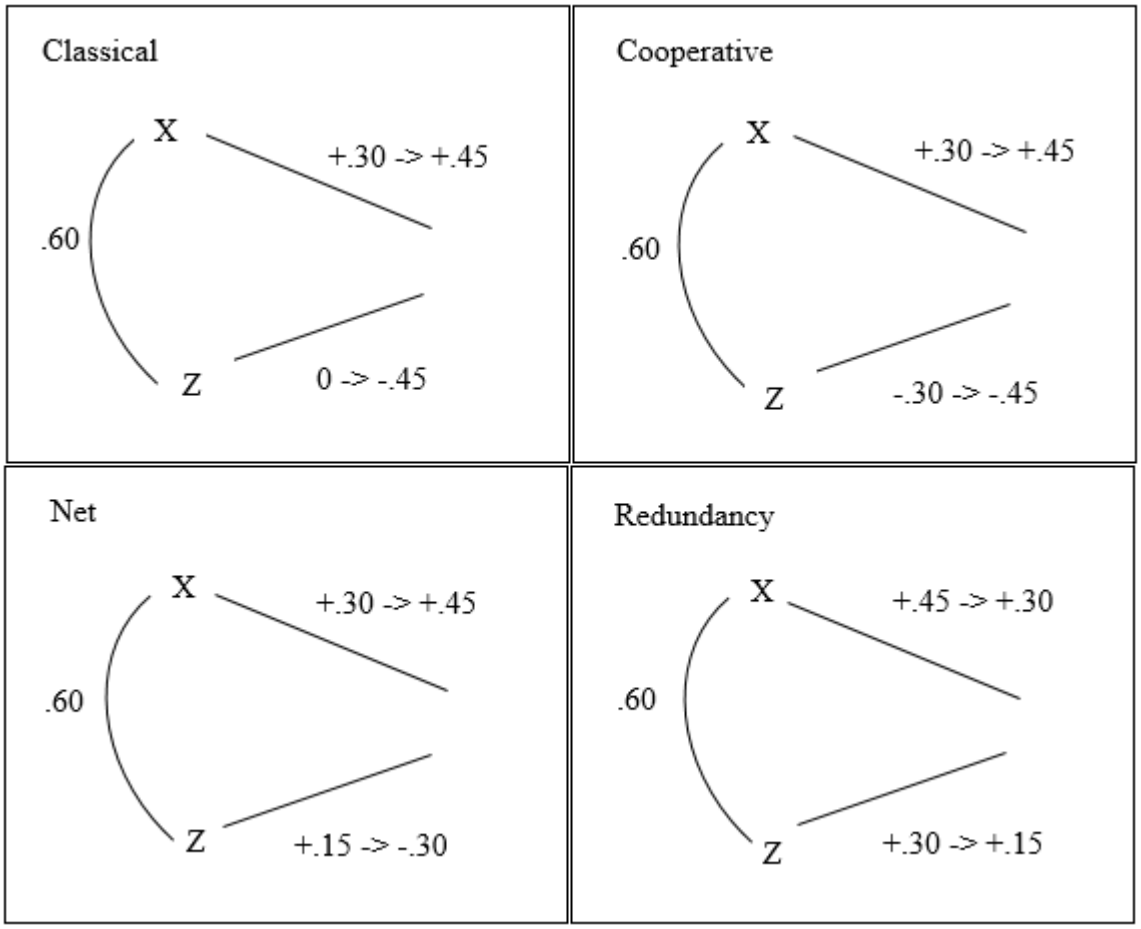


Figure 17. Graphical view of suppressor situations (Classical, Cooperative and Net) and redundancy situation. Adapted from Paulhus et al. (2004).

Suppression is statistically similar but conceptually distinct to mediation and confounding (MacKinnon, Krull and Lockwood, 2000). Mediation occurs when the effect of an independent variable on a dependent variable occurs indirectly through a third mediator variable, while confounding occurs when a third variable related to two other variables of interest falsely obscures or enhances the relationship between them (MacKinnon et al., 2000). Both these situations assume that the statistical adjustment for the third variable reduces the magnitude of the relationship between the independent and dependent variables. Suppression occurs when the statistical adjustment for the third variable enhances the magnitude of the relationship between the independent and dependent variables (MacKinnon et al., 2000).

3.5.4 Suppressor situations and psychopathy

If replicable, suppressor effects have important implications for the assessment of psychopathy (Blonigen, Patrick, Douglas, Poythress, Skeem, Lilienfeld et al., 2010). Detection of suppression effects is important because it helps to reconcile disparities between theory and empirical observations (Paulhus et al., 2004). The presence of cooperative suppressor effects in particular signifies the presence of highly distinctive underlying constructs embedded within a common measurement (Paulhus et al., 2004).

Verona, Hicks and Patrick (2005) detected a cooperative suppressor effect when evaluating the association between suicidal behaviour and the PCL:R Factors in female offenders. When history of suicide attempts was predicted using both Factor 1 and Factor 2, the beta coefficients for both were greater than when the Factors were used individually (e.g., the beta for Factor 1 increased from -.12 to

-.32; the beta for Factor 2 increased from .24 to .40). Cooperative suppressor effects have also been observed when evaluating the associations between negative emotionality and the PCL:R Factors. For example, Hicks and Patrick (2006) found that when fearfulness was predicted using both Factor 1 and Factor 2, the beta coefficients for both were greater than when the Factors were used individually (e.g., the beta for Factor 1 increased from -.21 to -.34; the beta for Factor 2 increased from .07 to .24). A net suppressor situation was also observed when the PCL:R Factors predicted levels of anger-hostility (Hicks and Patrick, 2006). Simultaneous entry of the two PCL:R Factors as predictors resulted in a change in the direction of association for Factor 1 and an increase in the positive association for Factor 2. Models based on the two factors were also superior to those based on the PCL:R Total scale alone. Finally, a more recent study by Blonigen et al. (2010) showed a clear cooperative suppression effect: the magnitude of relationships between PCL:SV Factors 1 and 2 and internalising were increased when both factors were included as predictors of internalising compared to when either factor was a single predictor. Results such as these support the idea of dissociable dimensions within the psychopathy construct.

3.6 Experimental session procedure

Upon arrival at the test session, the researcher verbally summarised the session content, highlighted key points of information from the information sheet (emotional content, confidentiality, no financial or other reward for participation, access to prison records and psychology files) and gave the participant the opportunity to ask any questions. Participants then completed the tick-box consent form and provided a signature of consent. All participants were considered competent to give consent. The consent form specifically detailed the following four points; that the participant had read the

information sheet, had had time to ask questions and that any questions had been answered satisfactorily; that participation was voluntary and the participant could withdraw at any stage without giving reason or without their treatment at the prison being affected; that by agreeing to take part the researcher had permission to access all relevant psychology and prison files; and that any information provided in relation to behavioural infractions or plans to harm themselves/others would be shared with the relevant staff. Participants provided brief demographic information (age, highest level of education reached and ethnicity) and gave an approximate daily caffeine and nicotine intake (if applicable).

The experimental procedure was as follows: Affect Categorisation task, Physiological reactivity, Affective and Semantic Priming tasks, TriPM and PANAS-X questionnaires, WASI. Order of completion of priming tasks and order of completion of questionnaires was counterbalanced across participants. The Affect Categorisation task was always presented before the Physiological reactivity task as the opposite order of presentation would have facilitated performance on a task that was designed to be difficult. Prior to each experimental task, participants received verbal instructions from the researcher. Instructions were also presented on screen. A laminated sheet of paper reminding participants of response options for each behavioural task was placed beside the testing laptop prior to each task. The researcher observed the participants progress during the practice phase of each behavioural task and reiterated instructions if appropriate. The researcher sat facing the participant during the test phase of each task. Participants completed each questionnaire independently but received verbal instructions prior to completion. Each questionnaire included written instructions also, and participants were encouraged to ask for clarity on items if unsure. If the researcher suspected the

participant would struggle with the questionnaires, the researcher offered to read each item aloud and record the responses on behalf of the participant. This was offered on two occasions and was declined by each of these participants. Following questionnaire completion, the researcher administered the WASI. At the end of the test session, the participant was given the opportunity to ask any questions. Participants were verbally debriefed and provided with a short debrief sheet to retain. This debrief sheet provided participants with details of how to get further information on the study and also of how to make a complaint if they wished.

Chapter 4: Affect Categorisation and Experience

4.1 Abstract

The ability to feel emotion may be related to the ability to perceive emotion; labelling of affective experiences and categorisation of affective cues thus represent two key components of emotion. Adult male offenders (n = 93) completed a forced choice affect categorisation task and a self-report measure of emotion experience. Participants completed an abbreviated version of the Positive and Negative Affect Schedule – Expanded Form (PANAS-X), reporting to what degree, generally or on average, they experienced emotion items indexing four continuous scales of emotion experience: guilt, joviality, sadness and fear. The categorisation task utilised briefly (100 ms) presented, backwardly masked affective (happy, sad, fear) and neutral images. Ability to categorise each image type was operationalised though a discrimination sensitivity score. The assessment-based measure of *primary psychopathic traits* (PCL:SV Factor 1) predicted reduced experiences of negative emotionality (fear, sadness and guilt) and reduced sensitivity when categorising threat images. The self-report measure of *primary psychopathic traits* (TriPM Boldness) predicted increased experiences of positive emotionality (joviality) and reduced experiences of negative emotionality, and greater sensitivity when categorising happy images. PCL:SV Factor 2, as an index of *secondary psychopathic traits,* did not significantly predict affect experience or categorisation: however, the unique variance of TriPM Meanness and TriPM Disinibition exhibited diverging relationships with categorisation of sad images. The pattern of results for *primary psychopathic traits* suggested a link between subjective experience and objective recognition of emotion.

4.2 Introduction

Two key components of emotion are the initial stage of affective stimulus categorisation, and the labelling of self-experienced emotion in response to stimuli and events (Scherer, 2009). This experimental chapter presents these two components together in relation to the psychopathy dimensions. Research (e.g., Buchanan, Bibas and Adolphs, 2010) indicates a link between the categorisation of affective cues and self-reported affective experience: the self-reported intensity of personal experience of happiness and fear is related to greater facial categorisation accuracy of these respective emotions. These results support the hypothesis that own emotional experience may play a role in the emotion process, either through on-line simulation or through effects during development.

Simulation Theory suggests that accurate attribution of another's emotional state, through the observation of, e.g., facial affect, occurs at least in part through the simulation, replication or reproduction (or the attempt to do so) in the observer's own mind of the same state as the target's (Goldman and Sripada, 2005). Accurate recognition or categorisation of emotion may therefore require the experience, either concurrently or through past experience, of that particular emotional state (see Buchanan et al., 2010). Lesion studies support the existence of co-occurring deficits in the production of affect and the ability to identify expressions of that affective state in others (see Goldman and Sripada, 2005). More specific models of Simulation Theory propose the use of reverse simulation with an 'as if' loop bringing a somatosensory representation of 'what it would feel like' were the observer to make that expression; as well as emotional contagion (Heberlein and Atkinson, 2009). Through the latter, observing emotion is suggested to somehow produce an empathic emotional experience in the perceiver. As such, models of Simulation Theory are consonant with theories of embodied cognition,

which emphasize the utilisation of body behaviours and are reliant on the idea of shared substrates (overlapping neural circuitry; i.e., mirror neurons) between categorisation and experience (see Heberlein and Atkinson, 2009). Simulation Theory would, therefore, suggest that the processing and categorisation of affective cues would relate to the experience of similar affective states. Impaired processing of e.g., threat cues, might suggest a corresponding reduction of self-reported fear in relation to e.g., *primary psychopathic traits*. For individuals high on *secondary psychopathic traits,* their proposed heightened affective experiences may indicate relatively intact affect categorisation.

4.2.1 Emotion Experience

Self-reported affect represents the labelling component of emotion. The nature of this measure makes it necessarily subjective and reliant on several different processes which are not always measureable. These processes may include the level of insight a subject holds into their own affective responses, how motivated they are to give such responses a particular label, and a subject's knowledge of emotional language. The degree of interpretation of self-reported emotion is therefore necessarily limited, and becomes even more complex in relation to psychopathic traits.

Emotion experience can be broadly differentiated into *negative* (NEM) or *positive emotionality* (PEM). NEM (see also section 1.3.1) combines anxiousness/neuroticism, alienation and hostility (see Verona et al., 2001) and describes individual differences in the tendency to experience unpleasant emotional states such as nervousness, worry, fear, sadness, guilt, anger, irritability, contempt and nervous tension (Hicks and Patrick, 2006). Hicks and Patrick (2006) refer to Buss and Plomin's (1994) model of childhood temperament and Tellegen's (Tellegen and Waller, 1992) temperament-based

structural model of adult personality when highlighting the multifaceted nature of NEM. NEM is proposed to differentiate into two broad traits of fearfulness and anger-hostility. PEM describes individual differences in the tendency to experience positive states, including those associated with extraversion, dominance, ambition, and engagement with others (Del Gazio and Falkenbach, 2008). High PEM also reflects a lack of psychological distress.

The majority of research examining the role of emotion experience in psychopathy has focused on NEM, with differential relations emerging between the two psychopathy dimensions and experience of negative affect. Measures of *primary psychopathic traits* are negatively related to self-reported fear and emotional distress (Hicks and Patrick, 2006; Patrick, 1994), anger-hostility (Hicks and Patrick, 2006) and broader measures of NEM (Del Gaizo and Falkenbach, 2008), independent of impulsive-antisocial traits (Hicks and Patrick, 2006; Patrick, 1994). In other work, *primary psychopathic traits* were not associated with low anxiety sensitivity or trait anxiety (Hale, Goldstein, Abramowitz, Calamari and Kosson, 2004).

By contrast, levels of *secondary psychopathic traits* are positively related to greater NEM (Del Gaizo and Falkenbach, 2008), self-reported fear and emotional distress (Hicks and Patrick, 2006; Patrick, 1994) and anger-hostility (Hicks and Patrick, 2006), independent of interpersonal-affective traits (Hicks and Patrick, 2006; Patrick, 1994). NEM and constraint (impulsivity, sensation seeking, socialization and psychoticism; Verona et al., 2001) completely accounted for the relationship between the impulsive-antisocial factor of psychopathy and suicidal behaviour (suicidal ideation, gestures, attempts and completions); while

Measures of *primary psychopathic traits* relate positively to social potency and achievement, reflecting agentic PEM (Verona et al., 2001) and broader measures of PEM (Del Gaizo and Falkenbach, 2008). Impulsive-antisocial psychopathic behaviours are negatively related to PEM (Verona et al., 2001). Other results indicate higher self-reported NEM and PEM for psychopaths as compared to controls. Gullhaugen and Nottestad (2011) found that offenders with possible and strong indications of psychopathy self-reported higher levels of NEM as measured by the Positive and Negative Affect Schedule (PANAS; Watson, Clark and Tellegen, 1988), as compared to non-personality disordered and noncriminal controls. Offenders with strong indications of psychopathy scored significantly higher on PEM compared to controls.

4.2.2 Affect Categorisation

The present work utilised affective and neutral image scenes as opposed to affective facial stimuli, and required participants to categorise the gist of each presented scene. Scene gist recognition may be operationalised in terms of the ability to categorise the global content of a briefly flashed scene image at a level ranging from the highly specific (e.g., a baby reaching for a butterfly; Fei-Fei, Iyer, Koch and Perona, 2007), to the superordinate (e.g., natural; Greene and Oliva, 2009), to the scene's affective valence (Calvo, Nummenmaa and Hyona, 2008). The ability to recognise and categorise the gist of a scene affects later processes including attentional selection (Torralba, Oliva, Castelhano and Henderson, 2006). However, as the vast majority of affect categorisation tasks in the psychopathy literature have utilised facial stimuli, the following section will primarily discuss these studies in relation to the categorisation of affective cues in psychopathy.

The assumption that psychopathy is related to impairments in the processing of affective cues, specifically in categorising facial displays of affect, is pervasive in research (Almeida et al., 2014). However, the behavioural evidence regarding these deficits is not robust. Meta-analyses have found a specific deficit in recognising fearful facial expressions in antisocial populations (Marsh and Blair, 2008) with individuals with high levels of psychopathic traits displaying impairments in categorising fearful faces as compared to controls (Iria and Barbosa, 2009; Blair, Mitchell, Peschardt, Colledge, Leonard, Shine et al., 2004). A more recent meta-analysis has indicated that psychopathy is associated with small deficits for categorisation of all emotions, with the largest deficits for fear and sadness facial expressions (Wilson et al., 2011; see also Dolan and Fullam, 2006, and Hastings, Tangney and Stuewig, 2008, for deficits for sad faces). Finally, Dawel et al. (2012) in their meta-analysis found that psychopathy was associated with significant impairments for positive and negative facial cues. Significant deficits were found for fear, happiness and surprise facial expressions. For example, psychopathic participants have shown a reduced ability to distinguish happy faces from sad as compared to control participants (Habel, Kuhn, Salloum, Devos and Schneider, 2002) and poorer categorisation of happy faces (Hastings et al., 2008).

Other studies have indicated no psychopathy related deficits on forced-choice tasks using static facial stimuli (Pham and Phillipot, 2010; Book et al., 2007; Glass and Newman, 2006). Book et al. (2007) found no correlation between psychopathy scores and performance on a forced-choice task categorising static facial affect (happiness, disgust, sadness, anger and fear). Glass and Newman (2006) found no difference between psychopathic and non-psychopathic inmates in both cued and un-cued conditions of a forced choice static facial affect task. No significant correlations were found between

psychopathy factors and categorisation accuracy in a sample of French offenders (Pham and Phillipot, 2010).

Studies assessing psychopathy in terms of its underlying dimensions have also found varied results regarding the categorisation of affective cues (facial affect). *Primary psychopathic traits* have been inversely associated with accuracy for fearful faces (Blair and Coles, 2000; Dadds et al., 2006; Prado, Treeby and Crowe, 2015), inversely associated with accuracy for anger, disgust, sad or shame facial expressions (Prado et al., 2015), unrelated to accuracy for anger, disgust, fear or sad facial expressions (Gillespie, Rotshtein, Wells, Beech and Mitchell, 2015) and positively associated with discriminating happy from sad faces (Habel et al., 2002). *Secondary psychopathic traits* have been inversely associated with accuracy for fearful faces (Blair and Coles, 2000), inversely associated with accuracy for neutral faces (Dadds et al., 2006), inversely associated with accuracy for happy faces (Dolan and Fullam, 2006; Hastings et al., 2008), inversely associated with accuracy for sad faces (Hastings et al., 2008), positively associated with ability to categorise facial disgust (Hansen, Johnsen, Hart, Waage and Thayer, 2008) and unrelated to accuracy for facial expressions (Prado et al., 2015, Gillespie et al., 2015).

These mixed results are problematic and, as of yet, no definitive relation between psychopathy, or the psychopathy dimensions, and processing of affective facial cues has been found. The difficulty of comparing across tasks likely lies in varying samples but also in methodological variability; some studies take dependent measures of number of errors (Book et al., 2007; Del Gaizo and Falkenbach, 2006; Blair et al., 2004, Hastings et al., 2008; Glass and Newman, 2006) or intensity threshold

(Hastings et al., 2008; Blair et al., 2004). Furthermore, many of the reported studies show total sample or control group ceiling effects for accuracy on various emotions (see Dawel et al., 2012).

The present study aimed to move away from these disparate results utilising facial displays of affect, by testing categorisation of affective image stimuli. There is a paucity of studies that have examined the ability to categorize the emotional content of scenes. Anderson and Stanford (2012, task 2) previously presented affective image stimuli to community participants in a simplistic version of a categorisation task which required participants to indicate whether each presented image was emotional or neutral. Levels of psychopathy, as assessed with the PPI-R (Lilienfeld and Widows, 2005), were not related to behavioural task performance. To the best of knowledge, the current study is the first study to assess the psychopathy-related affect categorisation, using a task similar to facial affect paradigms but utilising complex affective images, in an offender population.

4.2.3 Present study

The present study evaluated the relationship between affect categorisation, emotion experience (*positive* [PEM] and *negative* [NEM] *emotionality*) and psychopathic traits in an adult male offender sample. Simulation Theory would predict a relationship between experience and categorisation of affect. The *dual-process model* suggests that deficits in threat sensitivity are specific to *primary psychopathic traits,* whereas emerging evidence suggests heightened affective experiences in relation to *secondary psychopathic traits.* Taking support from the *dual-process model* and Simulation Theory, the following hypotheses were made: 1), that PCL:SV Factor 1 and TriPM Boldness, reflecting *primary psychopathic traits*, would predict reduced experiences of NEM and reduced sensitivity when

categorising affective cues, specifically aversive stimuli. Reduced experiences of NEM, specifically fear, among individuals high on *primary psychopathic traits* may go some way to underpinning their deficits in recognising this affective cue. By contrast, it was hypothesised that 2), PCL:SV Factor 2 and TriPM Disinhibition would predict heightened self-reported NEM in light of the associated emotional lability. Simulation Theory would predict that *secondary psychopathic traits* would therefore also predict enhanced negative affect categorisation; however this is not supported by the literature (see section 4.2.2) and as such analyses were exploratory. Analyses regarding both psychopathy dimensions and PEM were also exploratory.

TriPM Meanness reflects both *primary* and *secondary psychopathic traits*. As such, hypotheses for this measure of psychopathy were exploratory which would necessarily limit the interpretation of any significant results in relation to Meanness. Any results would therefore be interpreted in relation to the other psychopathy measures. For example, if a similar pattern of results were found between affect categorisation and Meanness, and affect categorisation and Disinhibition, that would be considered informative in relation to *secondary psychopathic traits.* If similar results were found between affect categorisation and Meanness, and affect categorisation and Boldness, that would be considered in light of *primary psychopathic traits.*

4.3. Method

4.3.1 Participants

Categorisation data was not collected from one participant due to refusal, resulting in a sample of 93 offenders with complete data for both the Affect Categorisation task and the emotion experience

questionnaire. Twenty six offenders were receiving a form of psychotropic medication. As is consistent throughout the thesis, results here control for psychotropic medication. Descriptive details of the impact of individual medications on task performance are presented in Appendix A.

4.3.2 Materials, Design and Procedure

The Affect Categorisation task stimulus set is described in section 2.2.4, the presentation hardware and software are detailed in section 3.3.1. Details of the IQ measure are provided in section 3.3.4.1 and details of the psychopathy measures are provided in section 3.3.3. Specifics of the Categorisation task design are provided in section 2.4.1.1.2. Details of the experimental session procedure are provided in section 3.6 and expansion of methods of analysis are provided in section 3.4. To summarise, during the Affect Categorisation task participants viewed briefly presented, backwardly masked affective (happy, sad, fear) and neutral images made up of complex scenes featuring both animals and people. Participants were required to categorise each image as either happy, neutral, sad or frightening in a forced choice response. Participants also completed a self-report measure of emotion experience. The items on this questionnaire contributed to four affective scales: guilt, sadness, fear and joviality.

4.3.2.1 Measurement of self-reported emotion experience

4.3.2.1.1 The PANAS-X

The Positive and Negative Affect Schedule – Expanded Form (PANAS-X; Watson and Clark, 1999) is a well-validated, self-administered questionnaire designed for adult (+18 years) populations.

The PANAS-X consists of a 60-item scale used to measure the examinees emotions on a variety of time scales (e.g., generally, today, within the past few days, etc.). The items are grouped into the following 4 subgroups and subscales: general dimension scales (negative affect, positive affect), basic negative emotion scales (fear, hostility, guilt, sadness), basic positive emotion scales (joviality, self-assurance, attentiveness), other affective states (shyness, fatigue, serenity, surprise). The participant is asked to read each item which describes feelings and emotions and enter a number that corresponds to the value on a scale. The 5-item scale ranges from '*very slightly or not at all'* with a value of 1, to '*extremely*' with a value of 5. For the purposes of the current study, an abbreviated version of the PANAS-X was devised. Following Watson and Clark (1999), under time constraints a restricted version of the questionnaire may be presented to participants. As such, items corresponding to fear (6 items), guilt (6 items), sadness (5 items) and joviality (8 items) were presented. The Fear scale was chosen due to the consistent evidence for threat deficiency in relation to *primary psychopathic traits*, the Sadness and Guilt scales were chosen due their importance in relation to empathic emotions and the Joviality scale was chosen as a measure of PEM. Fear, Sadness and Joviality scales were also selected in order to match the content of the stimulus set (i.e., fear, sad and happy images). Cronbach's alpha values for the four scales for this sample were good (Fear $\alpha = .90$; Guilt $\alpha = .90$; Sadness $\alpha = .89$, and Joviality $\alpha = .92$). Means and standard deviation for the scales were as follows: Fear: $M = 12.80$, $SD = 5.11$, maximum possible score = 30; Sadness: $M = 13.11$, $SD = 5.19$, maximum possible score = 25; Joviality: $M = 26.25$, $SD = 6.59$, maximum possible score = 40; Guilt: $M = 17.38$, $SD = 6.78$, maximum possible score = 30.

4.4. Results

4.4.1 Affect Categorisation task

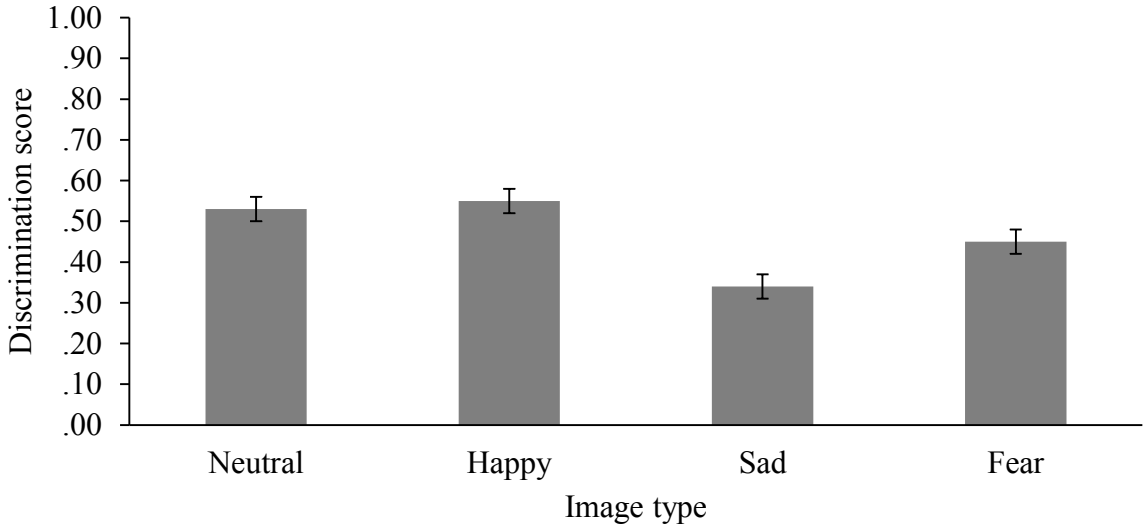


Figure 18. Discrimination indices representing mean categorisation sensitivity per stimulus type. Error bars show 2 *SE* (correction for repeated measures designs, Cousineau (2005) and Morey (2008)).

For each participant, outliers (responses less than 300 ms and more than 3000 ms) were excluded resulting in 7.18% of data removed overall. Mean categorisation per class of image is presented in Figure 18. Performance ranged from 0.34 to 0.55. Sensitivity for each class of image was above chance level of 0.25 as assessed through one-sample *t*-tests against zero (neutral: $t(92) = 21.57$, $p < .001$, $d_z = 2.24$; happy: $t(92) = 26.09$, $p < .001$, $d_z = 2.71$; sad: $t(92) = 17.03$, $p < .001$, $d_z = 1.77$; fear: $t(92) = 16.44$, $p < .001$, $d_z = 1.71$). Assessing for task efficacy, results of a one-way repeated measures ANOVA showed an effect of image content on categorisation sensitivity: $F(2.71, 249.56) = 44.18$, $p < .001$, $\eta_G^2 = .13$. Planned comparisons indicated greater sensitivity when categorising neutral

as compared to sad ($t(92) = 9.70$, $p < .001$, $d_z = 1.01$) or fear ($t(92) = 3.86$, $p < .001$, $d_z = 0.40$) images. Sensitivity for neutral and happy images was comparable ($t(92) = 1.30$, $p > .05$). Exploratory analyses indicated that categorisation sensitivity was greater for happy images as compared to sad ($t(92) = 11.26$, $p < .001$, $d_z = 1.17$) and fear ($t(92) = 4.16$, $p < .001$, $d_z = 0.43$) images. Fear images were also better recognised than sad images ($t(92) = 4.95$, $p < .001$, $d_z = 0.51$). Hence, it appeared the task produced overall results that were well away from floor and ceiling effects and provided a sensitive test for the influence of individual differences.

Task performance and scores on the PANAS-X were not consistently related to intelligence (Table 16). There was a strong inverse relationship between performance on the Affect Categorisation task and age. This age-related decline in affect categorisation has been well documented, mainly for facial affect (see Calder, Keane, Manly, Sprengelmeyer, Scott, Nimmo-Smith et al., 2003; Murphy and Isaacowitz, 2010; Orgeta, 2010) with little consistent evidence regarding the underlying mechanism, although restricted scanning patterns have been posited (Murphy and Isaacowitz, 2010). There is also an age-related decline in visual acuity, even in corrected visual acuity (see Salthouse, Hancock, Meinz and Hambrick, 1996) suggesting the decline in visual acuity relates to an age-related decrease in cognitive processing. This likely also contributes to the relationship between task performance and age in the present sample, particularly given the stringent task demands – task difficulty has been shown to moderate age differences in emotion recognition (Orgeta, 2010). All regression models assessing affect categorisation adjusted for the effects of age on task performance. Assessing for relationships among affective experience and categorisation, adjusting for age, a positive relationship was found between fear experience and fear categorisation (see Table 16).

Table 16

Zero-order and partial correlations between Affect Categorisation task performance, self-reported emotional experience, intelligence and age

	1	2	3	4	5	6	7	8	9	10
1.Neutral Categorisation	---									
2. Happy Categorisation	.62**	--								
3. Sad Categorisation	.47**	.41**	--							
4. Fear Categorisation	.65**	.43**	.39**	--						
5. Fear PANAS-X	**.00**	**.07**	**-.01**	**.24***	--					
6. Sadness PANAS-X	**.07**	**.03**	**.04**	**.13**	.62**	--				
7. Joviality PANAS-X	**-.10**	**-.10**	**.04**	**-.09**	-.31**	-.52**	--			
8. Guilt PANAS-X	**.05**	**-.02**	**.03**	**.09**	.52**	.60**	-.19	--		
9. Intelligence	.00	.29**	.07	-.08	-.06	.10	.01	-.02	--	
10. Age	-.45**	-.35**	-.33**	-.52**	-.04	-.07	.12	-.00	.29**	--

Note: PANAS-X = Positive and Negative Affect Schedule: Expanded Form. Partial correlations between task performance and PANAS-X scales, in bold print, adjusted for age.

*$p < .05$ **$p < .01$

4.4.1.2. Psychopathy analyses

4.4.1.2.1. PCL:SV

Zero-order correlations examined the relationship between the PCL:SV, and categorisation of affect (see Table 17). The general psychopathy factor (PCL:SV Total) was associated with reduced sensitivity when categorising threat images; this relationship appeared to be driven by levels of PCL:SV Factor 1. No other correlations reached significance; PCL:SV Factor 2 was not related to categorisation of affect.

In order to assess the robustness of this result, separate multiple regression models regressed discrimination scores per image type onto the PCL:SV Total and Factors 1 and 2 (see Table 17) while adjusting for age and medication. The values of interest from these models were the beta values for, e.g., Factor 1 alone (Factor 1 entered as a predictor variable) and with Factor 2 (Factor 1 and Factor 2 entered in the same step in order to control for their overlap). The unique sr^2 value is linked to the latter beta and reflects the unique contribution of, e.g., Factor 1, to the total variance of the criterion variable, in the presence of Factor 2. Levels of Factor 1 significantly predicted reduced sensitivity when categorising threat images, with the unique variance of Factor 1 marginally (p = .06) predicting reduced sensitivity when categorising threat images.

4.4.1.2.2. TriPM

Zero-order correlations among discrimination of affect and psychopathy as assessed with the TriPM (see Table 18) indicated that levels of TriPM Disinhibition were associated with greater sensitivity when categorising neutral images. However, this relationship was not maintained in regression models adjusting for age and presence of psychotropic medication (see Table 18). TriPM Boldness significantly predicted greater sensitivity when categorising happy images. A cooperative suppressor effect was revealed: the unique variance of TriPM Meanness predicted reduced sensitivity when categorising sad images, whereas the unique variance of TriPM Disinhibition predicted greater sensitivity when categorising sad images.

4.4.2 Emotion experience

4.4.2.1. Psychopathy analyses

4.4.2.1.1. PCL:SV

Zero-order correlations, presented in Table 17, indicated that PCL:SV Total and Factor 1 were related to reduced experiences of self-reported NEM (fear and guilt). Factor 1 was also related to reduced experience of sadness. These relationships were maintained in regression models adjusting for presence of psychotropic medication (see Table 17). Partialling out the common variance with Factor 2 indicated that the unique variance attributed to Factor 1 was a useful predictor of levels of self-reported NEM. Levels of Factor 2 did not significantly predict subjective emotion experience.

4.4.2.1.2. TriPM

Results of zero-order correlations between the TriPM scales and self-reported emotion experiences are presented in Table 18. TriPM Boldness was positively associated with self-reported PEM and inversely associated with self-reported NEM. All other correlations failed to reach significance. Significant relations between TriPM Boldness and self-reported emotional experiences were maintained when controlling for presence of psychotropic medication (see Table 18). Partialling out common variance with TriPM Meanness did not consistently increase the beta weights for Boldness, indicating that the complete scale was a useful predictor of self-reported affect. The complete TriPM Boldness scale accounted for between 11% (guilt) and 24% (fear) of the variance explained by the respective models (not reported in Table 18). The complete TriPM Meanness scale predicted reduced experiences of fear at a trend level (p = .06).

4.4.3 Covariates

Repeat analyses additionally controlling for IQ are presented in Appendix A and discussed in section 4.5.3. In relation to emotion experience, there were no changes to the results for either the PCL:SV or the TriPM when additionally controlling for intelligence. In relation to affect categorisation, PCL:SV Total and Factor 1 no longer significantly predicted reduced sensitivity for categorisation of fear images when additionally controlling for intelligence. The unique variance of TriPM Meanness and TriPM Disinhibition no longer predicted categorisation of sad images when additionally controlling for intelligence.

Table 17.

Relations between PCL:SV factors, affect categorisation and self-reported emotion experience

	PCL:SV Total		Factor 1				Factor 2			
Criterion Variable	*r*	β	*r*	β alone	β with F2	Unique sr^2	*r*	β alone	β with F1	Unique sr^2
Categorisation[a]										
Neutral	.01	.02	-.09	-.01	-.02	.00	.10	.03	.04	.00
Happy	-.05	-.06	-.01	.04	.10	.01	-.08	-.14	-.18	.03
Sad	-.12	-.11	-.16	-.10	-.07	.00	-.05	-.11	-.09	.01
Fear	-.21*	-.20*	-.29**	-.21*	-.18	.03	-.05	-.13	-.07	.01
Experience[b]										
Guilt	-.22**	-.25*	-.29**	-.30**	-.29**	.08**	-.07	-.11	-.02	.00
Joviality	-.02	-.03	.05	.05	.09	.01	-.10	-.12	-.14	.02
Fear	-.21*	-.21*	-.23*	-.23*	-.21*	.04*	-.13	-.13	-.07	.00
Sadness	-.17	-.17	-.26*	-.26*	-.27*	.07*	-.03	-.03	.05	.00

Note: PCL:SV= Psychopathy Checklist: Screening Version; F1 = Factor 1; F2 = Factor 2.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

[a]Adjusting for age and medication. [b]Adjusting for medication.

*$p < .05$ **$p < .01$

Table 18.

Relations between TriPM factors, affect categorisation and self-reported emotion experience

	Boldness				Meanness				Disinhibition			
Criterion Variable	*r*	β alone	β with D and M	Unique sr^2	*r*	β alone	β with D and B	Unique sr^2	*r*	β alone	β with M and B	Unique sr^2
Categorisation[a]												
Neutral	.03	.06	.10	.01	.16	.13	.00	.00	.27**	.16	.17	.01
Happy	.18	.19*	.22*	.04*	.04	-.00	-.08	.00	.06	-.05	.05	.00
Sad	.01	.04	.11	.01	-.02	-.11	-.31*	.06*	.14	.05	.29*	.04*
Fear	-.11	-.08	-.05	.00	-.05	-.09	-.11	.01	.11	-.04	.04	.00
Experience[b]												
Guilt	-.32***	-.33**	-.24*	.05*	-.18	-.16	-.23	.03	.05	.04	.17	.01
Joviality	.39***	.38***	.39**	.13**	-.00	-.05	-.13	.01	-.12	-.12	.01	.00
Fear	-.49***	-.49***	-.48***	.19***	-.17	-.20	-.16	.02	.05	.03	.11	.00
Sadness	-.42***	-.42***	-.37**	.12***	-.09	-.09	-.13	.01	.15	.15	.16	.01

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

[a]Adjusting for age and medication. [b]Adjusting for medication.

*$p < .05$ **$p < .01$ ***$p < .001$

4.5. Discussion

PCL:SV Factor 1 and TriPM Boldness evidenced diverging relationships with affect categorisation and experience. In line with the hypothesis, increasing Factor 1 predicted reduced sensitivity when categorising threat images, and reduced experiences of negative emotionality (NEM; fear, sadness and guilt). Higher levels of TriPM Boldness predicted greater sensitivity when categorising happy images, and both reduced experiences of NEM and greater experiences of positive emotionality (PEM). In contrast to the hypothesis, PCL:SV Factor 2 did not significantly predict affect categorisation, nor emotion experience. Suppression effects were observed in relation to TriPM Disinhibition and TriPM Meanness, with these scales evidencing diverging relationships with categorisation of sad images when adjusting for their shared variance. These results will now be discussed in turn.

4.5.1 Affect categorisation

4.5.1.1 Primary psychopathic traits

Higher levels of Factor 1 significantly predicted reduced sensitivity when categorising threat images. This result links to existing literature demonstrating impaired threat reactivity in relation to *primary psychopathic traits* across several experimental paradigms: e.g., aversive conditioning (Flor et al., 2002), eye-blink startle (Patrick et al., 1993; Vanman et al., 2003; Vaidyanathan et al., 2011), the identification of negative emotions from auditory stimuli (see Dawel et al., 2012) and passive avoidance tasks (Newman and Kosson, 1993). Previous work, which has identified reduced sensitivity

for fearful facial expressions in criminal and non-criminal psychopaths (Iria and Barbosa, 2009, see Marsh and Blair, 2008), may be interpreted as reflecting reduced sensitivity for stimuli which have inherent, biological relevance and are relatively homogeneous in content (Hariri et al., 2002; Sabatinelli et al., 2011). The present work represents a novel contribution to the literature by demonstrating that offenders high on Factor 1 traits also display reduced sensitivity for non-homogeneous images depicting learned threats to themselves and others. This interpretation is supported by empirical evidence demonstrating impaired aversive conditioning in relation to Factor 1 (Flor et al., 2002) – an impaired ability to establish an associative link between a cue and its associated aversive outcome. Deficient threat reactivity in relation to Factor 1 may reflect an impairment in aversive learning.

In contrast to previous work which has established suppressor effects between Factors 1 and 2 (Blonigen et al., 2010; Hicks and Patrick, 2006; Verona et al., 2005), traits unique to Factor 1 predicted threat categorisation at a trend level only. Recent work has suggested that the common variance between Factors 1 and 2 may have important explanatory value in characterising the relations between psychopathy and particular external correlates. For example, in a study from Baskin-Sommers, Brazil, Ryan, Kohlenberg, Neumann and Newman (2015), Factor 2 was negatively associated with executive function but after adjusting for Factor 1 traits, no association was found between Factor 2 and executive function (see also Anderson et al., 2015; Baskin-Sommers et al., 2015a). Results such as these are supportive of the assertion (Skeem et al., 2011) that the dimensions are also likely to have areas of overlap and share significant traits.

Individuals high on *primary psychopathic traits* are considered deficient in defensive system reactivity to threat situations, allowing them to exhibit fearless behaviour which is insensitive to punishment (Patrick, 1994). Affective deficits associated with this psychopathy dimension should be specific to aversive or threat-relevant stimuli, which is reflected in the present results in relation to PCL:SV Factor 1. By contrast, given the strong reward-approach motivation inherent to *primary psychopathic traits,* indices of this psychopathy dimension may also positively relate to responses to positive affect. This was borne out in the present sample under these task conditions: TriPM Boldness, an index of *primary psychopathic traits*, significantly predicted greater sensitivity when categorising happy images (see also Habel et al., 2002).

The present pattern of results suggests that TriPM Boldness and PCL:SV Factor 1 index different aspects of *primary psychopathic traits* (as TriPM Meanness also theoretically relates to *secondary psychopathic traits*, only Boldness and Factor 1 are compared here). Boldness is said to reflect Cleckley's positive adjustment characteristics that contribute to "the mask of sanity". As Patrick (2006) noted, several of Cleckley's item descriptions can be grouped together to reflect positive adjustment and immunity to internalizing psychopathology and experiences of fear (see section 1.2.1.1). Although both Boldness and Factor 1 describe an interpersonally dominant individual, the TriPM Boldness scale was designed to reflect calmness and poise in stressful situations and a high degree of uncertainty tolerance. By contrast, the interpersonal aspects of Factor 1 may be more reflective of maladjustment, such as deceitfulness and shallow affect. The results indicate the advantages of utilising alternative measures of the psychopathy dimensions.

The unique variance of TriPM Meanness predicted reduced sensitivity when categorising sad images. Meanness, reflecting both *primary* and *secondary psychopathic traits,* represents the intersection between externalising psychopathology and the interpersonal-affective features of psychopathy (Venables et al., 2014). The present result is intuitive in light of existing research linking TriPM Meanness with low empathy (Stanley et al., 2013) and callous-unemotional traits (Sellbom and Phillips, 2013). However, only the unique variance was associated with impaired categorisation of sad images. In the present sample, Meanness was positively correlated with both TriPM Boldness and Disinhibition, suggesting that the variance not shared with these overlapping traits was useful in predicting categorisation of sad images. The specific traits reflected by this variance may include a lack of social connectedness, but this is necessarily conjecture. Furthermore, if Meanness reflects *primary psychopathic traits*, it is not clear why this psychopathy scale was not associated with, or additionally associated with, impaired categorisation of threat images. Further work may be required to specify the external correlates of Meanness.

4.5.1.2 Secondary psychopathic traits

PCL:SV Factor 2 was not a significant predictor of categorisation of affective images. Previous work has similarly found indices of *secondary psychopathic traits* to be unrelated to accuracy for facial expressions (Prado et al., 2015, Gillespie et al., 2015).

By contrast, the unique variance of TriPM Disinhibition predicted greater sensitivity when categorising sad images. There is some support in the literature for increased ability to categorise affect

in relation to *secondary psychopathic traits* (e.g., categorisation of facial disgust, Hansen et al., 2008) but also evidence for impaired categorisation of facial displays of sadness (Hastings et al., 2008). Again, the specificity of this relationship warrants caution – only the unique variance, separate from the overlap shared with TriPM Meanness, was predictive of enhanced sensitivity when categorising sad images. In the only example of a cooperative suppression effect, Meanness, in the absence of Disinhibition and Boldness, predicted reduced sensitivity to sad images; Disinhibition, in the absence of Meanness, predicted greater sensitivity to these images. Given that Patrick et al. (2009) have stated that Disinhibition alone reflects broad externalising psychopathology and not explicitly psychopathic traits, the specificity of this result queries whether the unique variance of TriPM Disinhibition is relevant to *secondary psychopathic traits.*

4.5.2 Emotion Experience

4.5.2.1 Primary psychopathic traits

In line with existing results (Del Gaizo and Falkenbach, 2008; Hicks and Patrick, 2006), both the complete and unique variance of Factor 1 predicted reduced levels of self-reported negative emotionality (NEM); fear, sadness and guilt. There was a parallel between deficits in threat evaluation and subjective feelings of fear across the lifespan in relation to Factor 1; these results are supportive of Simulation Theory. Knowledge of affect may be underpinned by a low-level simulation process, using personal experiences of an emotion to understand external displays of that emotion (Goldman and Sripada, 2005). This suggests that the forming of associative links between perception and

experience may be key in relation to Factor 1. LeDoux (2014) has made a clear distinction between the systems underlying threat perception and detection, and the experience of fear. While the present work cannot inform on whether the systems are separate, or dissociable but related, results presented here suggest that both perceptions of threat and representations of fear are impaired in relation to particular psychopathic traits.

The accurate perception of another's state may contribute to the development of an empathic response. Deficits in threat categorisation may therefore relate to the empathy deficits characteristic of individuals high on *primary psychopathic traits*. For these individuals, the reduced ability to recognize displays of negative affect may be related to their own shallow experiences of NEM, which may impair their ability to empathize with these emotions in others. Offenders with high levels of psychopathy have shown reduced levels of activation in brain regions considered relevant to the affective component of empathy, while viewing images of individuals in painful situations and imagining others to be in that situation, indicating reduced vicarious experience (Decety, Chen, Harenski and Kiehl, 2013). While viewing the images and imagining themselves to be in that situation, high-psychopathy offenders showed greater activation relative to low-psychopathy offenders (Decety et al., 2013). There is also some evidence that individuals with psychopathy have a reduced ability to infer another's emotional state (empathic accuracy; see Brook and Kosson, 2013).

PCL:SV Factor 1 and TriPM Boldness both predicted reduced experiences of self-reported guilt. Guilt is a strong, self-conscious emotion which may reflect negative affect or regret in response to a behaviour or event, and concern over other's feelings in relation to such behaviours or events (see

section 1.3.1). Intact experience of guilt in typical individuals stands in contrast to the shallow affect, lack of remorse and inability to take responsibility for wrong-doings typified by individuals high on *primary psychopathic traits* (Hare, 2003). The present results are in line with a small literature specifically assessing levels of guilt in relation to the psychopathy dimensions: Spice et al. (2015) found that levels of both general and offence-related guilt were negatively related to interpersonal-affective deficits in a sample of adolescent offenders which implies reduced guilt across multiple domains. Levels of general and offence-related guilt were also negatively related to anger/irritability and depression/anxiety (Spice et al., 2015) suggesting an association between a lack of guilt and resilience against NEM more generally, as is reflected in the present study. Levels of guilt have also been negatively associated with *primary psychopathic traits* in community (Lyons, 2015) and adult offender (Johnsson et al., 2014) samples.

Levels of TriPM Boldness predicted reduced experiences of NEM (fear, sadness and guilt) and increased experiences of PEM (joviality). TriPM Boldness was not significantly related to categorisation of negative affect. In contrast to Simulation Theory, individuals high on TriPM Boldness reported significantly reduced experiences of, e.g., sadness, suggesting that these individuals showed relatively intact accuracy when observing displays of sadness that they themselves experience to a reduced extent. This links well to TriPM Boldness reflecting Cleckley's positive adjustment factors and the mask of sanity, with an outward expression of affective knowledge (intact sensitivity when categorising sad images) masking an inner deficit (reduced experiences of sadness). An intact ability to recognise sadness in others may go some way towards facilitating manipulative, dominant

behaviour (see Book et al., 2007), free from the likelihood of experiencing guilt as a result – given the reduction in experiences of this emotion reported by individuals high on Boldness.

Levels of TriPM Boldness also significantly predicted increased self-reported experiences of joviality. This may reflect the emotional resilience which characterises individuals high on Boldness and again suggests that PCL:SV Factor 1 and TriPM Boldness may index aspects of *primary psychopathic traits* to different extents. *Primary psychopathic traits* may reflect not just the absence of fear but the presence of resilience: the ability to remain strong in times of hardship (see Sandvik, Hansen, Hystad, Johnsen and Bartone, 2015). Cleckley's descriptions of the positive adjustment factors of psychopathy do not just reference a lack of anxiety, they also include the presence of psychological hardiness and adjustment (Patrick and Bernat, 2009). Previous work established that the relationship between PCL-R Factor 1 and low anxiety in a sample of adult male offenders was partially mediated by an index of psychological hardiness (Sandvik et al., 2015).

Simulation Theory would predict a relationship between affect categorisation and emotion experience. Scores on TriPM Boldness predicted both enhanced sensitivity for happy images and greater experiences of happiness (PANAS-X Joviality). Boldness manifests behaviourally as imperturbability, social poise, assertiveness and persuasiveness, bravery and venturesomeness and is characterised by increased BAS activity (Donahue and Caraballo, 2015). The BAS reflects sensitivities to appetitive stimuli associated with reward, therefore it seems intuitive that, in combination with the increased experiences of positive affect, individuals high on Boldness would also present with increased sensitivity towards positive stimuli. By comparison, the consistent relationship between

affect categorisation and emotion experience in relation to Factor 1 was for threat/fear, which again suggests that use of complimentary measures of *primary psychopathic traits* facilitates a nuanced examination of external correlates.

4.5.2.2 Secondary psychopathic traits

TriPM Disinhibition and PCL:SV Factor 2, and TriPM Meanness, did not significantly predict self-reported emotion experience. Previously, levels of PCL-R Factor 2 have been positively related to particular facets of NEM: greater anger-hostility, greater emotional distress and greater fearfulness (Hicks and Patrick, 2006), greater anxiety (Sandvik et al., 2015), and greater shame (Spice et al., 2015). PCL-R Factor 2 has also been associated with less guilt (Spice et al., 2015; Johnsson et al., 2014).

In assessing facets of NEM, Hicks and Patrick (2006) and Verona et al. (2001) used scales which measured alienation and aggression, neuroticism, stress reaction and anger expression. Hicks and Patrick (2006) also included the negative affect scale of the PANAS which includes items related to feeling ashamed and is broader than the subscales presented in the current study. Spice et al. (2015) found that shame was positively related to levels of behavioural features of psychopathy in an adolescent offender sample. As the present study was administered as part of a larger battery, time constraints required an abbreviated version of the PANAS-X to be presented. Administration of the complete version of this questionnaire would have allowed analysis in terms of general negative and positive affect, as well as further scales such as Hostility and Attentiveness. It is possible that measures of stress reaction, alienation, and anger/hostility, which more specifically index experience of affect

in more interpersonal contexts, as well as additional measures addressing emotional distress to supplement the PANAS-X, would produce predicted associations with the measures of *secondary psychopathic traits.*

Alternative measures of PEM may also be better placed than the PANAS-X Joviality scale to assess states of positive affect in relation to *secondary psychopathic traits.* Verona et al. (2001) found an inverse relationship between PCL-R Factor 2 and PEM when assessing levels of well-being, social potency, social closeness and achievement. Such measures may be more relevant to impulsive-antisocial behaviours than more basic measures of joviality as used here. Finally, analyses of emotion experience revealed little evidence of suppression effects across the two psychopathy measures.

4.5.3 Role of intelligence

When additionally controlling for intelligence, changes were seen in the pattern of results for the Affect Categorisation task. The pattern of significant results found in the main analysis – Factor 1 and impaired threat categorisation, TriPM Meanness and Disinhibition and diverging relations with sad categorisation – no longer passed the threshold for significance; although the predictive relationship between Factor 1 and threat categorisation (at $p = .08$) would perhaps be interpreted as 'approaching significance' by other work. Intelligence was not a significant predictor of the criterion variable; the inclusion of intelligence did not improve the model fit, resulting in a smaller R^2 value and a smaller adjusted R^2 value for each model, as compared to the corresponding model in the main analysis. Given that the adjusted R^2 value reflects the predictive improvement in the model taking into

account the number of variables, it appeared that the additional inclusion of intelligence made each of these models less parsimonious and detracted from the validity of the individual difference variables.

4.5.4 Conclusion

The results indicated that different indices of *primary psychopathic traits* were differentially related to the evaluation component (categorisation) of emotion and the subjective 'feeling' component of emotion. PCL:SV Factor 1 predicted reduced sensitivity when categorising threat images and reduced self-reported fear, sadness and guilt, supporting the suggestion that a key deficit underlying this psychopathy dimension is fearlessness (see Patrick and Bernat, 2009). TriPM Boldness predicted enhanced sensitivity when categorising happy images, greater PEM and reduced NEM. By contrast PCL:SV Factor 2 did not significantly predict affect categorisation or emotion experience. The relationship between the unique variance of TriPM Disinhibition and greater sensitivity when categorising sad images drew into question the relevance of this result for *secondary psychopathic traits* as opposed to externalising psychopathology more broadly. The pattern of results for TriPM Meanness did not allow a clear placing of this scale as either a sole indicator of *primary* or *secondary psychopathic traits,* which is perhaps to be expected given its theoretical spanning of these two dimensions.

Chapter 5: Physiological Reactivity

5.1 Abstract

Pupillometry, the measure of the change in pupil diameter in response to stimuli, represents an under-utilised method of assessing physiological reactivity to affective stimuli in relation to the two underlying dimensions of psychopathy. Pupil diameter was recorded in response to affective (happy, sad and fear) and neutral images in a sample of adult male offenders (n = 77). Initial pupil diameter, initial constriction responses and emotional modulation of pupil dilation were recorded. PCL:SV Factor 2 predicted a smaller initial pupil diameter indicating low arousal. *Secondary psychopathic traits* (PCL:SV Factor 2 and TriPM Disinhibition), as well as TriPM Meanness, also predicted restricted initial constriction responses to both affective and neutral stimuli, independent of initial pupil diameter. This pattern of autonomic activity for offenders high on *secondary psychopathic traits* may represent a psychophysiological risk factor for antisocial behaviour, i.e., increased offending. As expected, affective images elicited a dilation of the pupil in comparison to neutral images, but this affective modulation was not related to any measure of psychopathy. This suggests that under these task conditions, both *primary* and *secondary psychopathic traits* were characterised by typical physiological responses to affective stimuli. Discrepancies in the processing of affective information may be best indexed by alternative paradigms, such as those that utilise threat conditioning or attentional manipulations.

5.2 Introduction

5.2.1 Physiological reactivity

The third component of emotion assessed in this work was the elicitation of an affective state in response to a stimulus, specifically an autonomic or physiological response. Basic emotion models contending that physiological sensitivity is essential for successful emotional functioning are not new – James (1884) argued that emotion experiences arise directly from the perception of body changes. Under the Somatic Marker Hypothesis (Damasio, 1994; Damasio et al., 1996), the key element of emotional experience is held to be physiological reactivity. Appropriate autonomic functioning and sensitivity is critical to experiencing emotional states that guide prosocial behaviour and good decision making. 'Somatic markers' provide emotional experiences with affective value, reinforcing emotional knowledge and memory for key events. For example, somatic markers are unpleasant 'gut feelings' in response to external stimuli. Damage to the ventromedial prefrontal cortex and amygdala has been argued to result in an inability to experience this 'gut feeling' which, in turn, may predispose to psychopathic and antisocial behaviour (Damasio, 1994; Damasio et al., 1996, see also section 1.2.2.1).

The present study examined the two psychopathy dimensions in relation to a novel measure of psychophysiology, pupil activity. The study assessed initial levels of pupil diameter, the initial constriction response and emotionally modulated pupil dilation (difference between affective categories and neutral) to affective (happy, sad, fear) images.

5.2.2 Pupillometry

To the best of knowledge, this is the first assessment of the psychopathy dimensions through emotionally modulated pupil activity, aiming to contribute to a literature which increasingly defines these dimensions in terms of differing neuropsychological and psychophysiological profiles (Fowles and Dindo, 2009; Patrick and Bernat, 2009). Pupil activity can be remotely measured using discrete eye tracking equipment and is almost contact-free (Kuchinke et al., 2011), allowing a stress-free experience for the participant. The technique also requires only minimal cooperation or effort on the part of the participant and, as pupil activity is not affected by voluntary control (Beatty and Lucero-Wagoner, 2000), it may be a valuable method for investigations in populations that may have low levels of motivation or cooperation.

Changes in pupil diameter are underscored by both the parasympathetic (PNS) and the sympathetic (SNS; Beatty and Lucero-Wagoner, 2000, see Figure 19) branches of the autonomic nervous system. The SNS allows the body to function under stress; the release of norepinephrine prolongs the effects of sympathetic stimulation. The PNS controls vegetative functions and is in opposition to the SNS. Both the SNS and PNS directly control the activity of the iris muscles which determine pupillary movements (Beatty and Lucero-Wagoner, 2000).

The pupil is the opening in the middle of the contractile pigmented iris and controls the amount of luminance reaching the retina. The pupil is dynamic and responds to changing light levels; in bright light the pupil is small and constricted, while in dim light the pupil is dilated and relaxed. The resting

diameter of the pupil is, therefore, determined by the level of luminance reaching the retina, any accommodation responses which change the depth of visual field, and the individual's tonic state of arousal (Beatty and Lucero-Wagoner, 2000). The resting diameter of the pupil may be represented empirically by the initial pupil diameter; i.e., the diameter of the pupil prior to the onset of the test stimulus (Giakoumaki, Hourdaki, Grinakis, Theou and Bitsios, 2005).

The iris, which controls the movements of the pupil, is controlled by two antagonistic muscle groups, the sphincter and dilator muscles. The sphincter muscles are under PNS control; arranged concentrically, these muscles receive input to constrict the pupil; the dilator muscles, which are arranged radially, serve to enlarge the diameter of the pupil, through input from the SNS. At any moment in time, the size of the pupil is the integrated result of the ratio of activity occurring in the directly opposing pathways reciprocally innervating these two muscle groups (Sherrington's law of reciprocal innervation; as quoted in Beatty and Lucero-Wagoner, 2000).

Although closely related, parasympathetic inhibition and sympathetic excitation may be separated by way of their influence on pupil diameter (Giakoumaki et al., 2005, see Figure 19). Upon presentation of a visual stimulus, the pupil constricts, referred to here as the 'initial constriction response' (ICR). Clarke and Ikeda (1985) state that this ICR is almost entirely reliant on PNS control (see also Smith, 1992). The parasympathetic pathway underlying the constriction response is relatively simple (Beatty and Lucero-Wagoner, 2000). As illustrated in Figure 19, cells from the retina and optic nerve, responsive to luminance, transmit information through to the Edinger-Westphal nucleus. Constriction of the pupil is attributed to the weakening of the central sympathetic inhibition of the

parasympathetic Edinger-Westphal nucleus (Hou, Samuels, Langley, Szabadi and Bradshaw, 2007). The Locus Coeruleus, (LC), which is located in the sympathetic pathway in Figure 19, also plays a key role in the parasympathetic pathway by exerting an inhibitory influence on parasympathetic outflow.

From the Edinger-Westphal nucleus, fibres project to the iris sphincter muscles. Postganglionic fibres release the neurotransmitter acetycholine (ACh; cholinergic input) to activate the concentrically - arranged fibres of the iris sphincter muscle (Smith, 1992; see Figure 19), resulting in miosis or constriction. Inhibition of tonic activity of the Edinger-Westphal nucleus would lead to relaxation of the muscles, resulting in a smaller constriction (Smith, 1992). Smaller constrictions (attenuation of the ICR), presumably reflecting inhibition of PNS activity, have been observed in response to arousing stimuli as compared to neutral. This includes emotional arousal in the form of anticipation of aversive stimuli (Bitsios, Szabadi and Bradshaw, 1996; Giakoumaki et al., 2005) and arousal while viewing affective as compared to neutral images (Bradley and Lang, 2015; Henderson, Bradley and Lang, 2014).

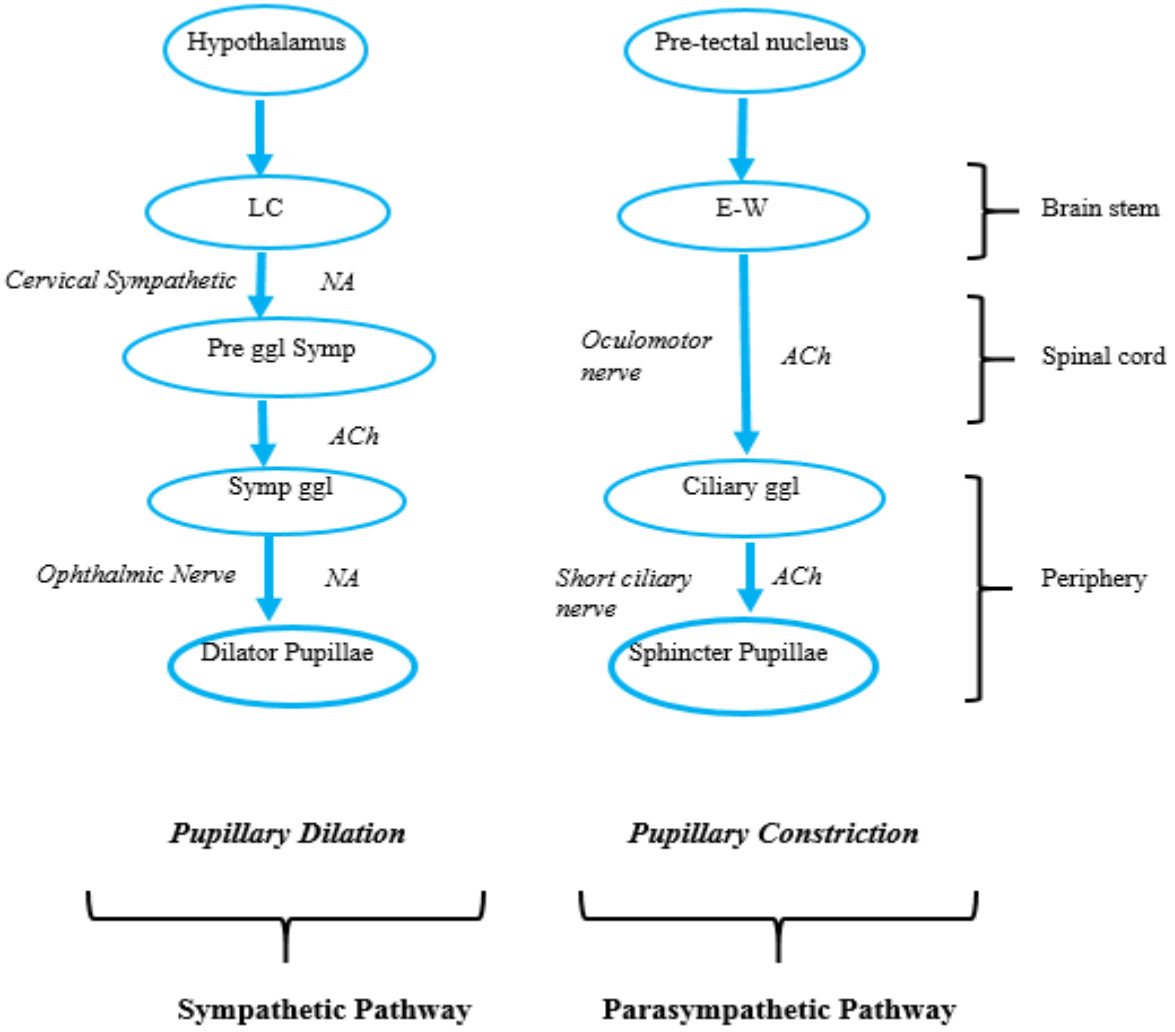


Figure 19. Schematic diagram of the neural pathways underscoring pupil diameter (adapted from Beatty and Lucero-Wagoner, 2000; Hou et al., 2007). Nuclei: *LC* Locus Coeruleus, *EW* Edinger-Westphal nucleus, *Pre ggl symp* preganglionic sympathetic neurones, *Symp ggl* sympathetic ganglia, *Ciliary ggl* ciliary ganglia. Neurotransmitters: *Ach* acetycholine, *NA* noradrenaline.

Following the ICR to a visual stimulus, subsequent dilation of the pupil is also influenced by various physical properties of a stimulus and its processing demands: colour, complexity and contrast

of the stimulus (Snowden et al., 2016). The pupil is particularly sensitive to the arousal content of stimuli; early work from Hess and Polt (1960) established pupil dilation in response to affective images, being larger in response to 'interesting' (mother and baby, nude figures) as compared to neutral images. This work has recently been extended to demonstrate larger pupil dilation to aversive and appetitive, as compared to neutral, images (see Bradley et al., 2008). This pupil dilation, as it returns to its original size, is greatly influenced by SNS activity (Bitsios et al., 1996; Smith, 1992).

The sympathetic system is more complex than the parasympathetic system (Beatty and Lucero-Wagoner, 2000). Neurons descend from the hypothalamus, which has projections from areas such as the cortex and central amygdala that influence sympathetic control of the pupil (Le Doux, 2012; Smith, 1992), then descend to the LC. The LC is the pivotal nucleus in Aston-Jones and Cohen's (2005) Adaptive Gain Theory of pupillary dilation and exerts an excitatory influence on sympathetic outflow by inhibiting the parasympathetic oculomotor complex or Edinger-Westphal nucleus (Laeng, Sirois and Gredeback, 2012).

From the LC, information is transmitted to the iris muscles via the ophthalmic nerve and the long ciliary fibres. ACh is also used as an internal transmitter for the sympathetic pathway (Hou et al., 2007; see Figure 19). Contraction of the dilator muscles enlarges the diameter of the pupil and necessarily reduces the size of the iris. The dilator muscles are thought to be activated primarily via stress-activated noradrenaline (NA) release from the LC, which increases levels of arousal by mobilizing the body and brain for action (Hou et al., 2007). Dilation (mydriasis) of the pupil occurs.

Bradley et al. (2008) assessed the effects of valence (appetitive/aversive) and arousal on pupil dilation using stimuli taken from the IAPS (Lang et al., 2008). Greater dilation in the period following the ICR was found for arousing as opposed to neutral pictures, with no significant difference between appetitive and aversive images. This basic finding has since been replicated in several studies (Arriaga et al., 2015; Bradley and Lang, 2015; Henderson et al., 2014; van Steenbergen, Band, and Hommel, 2011). In the original Bradley et al. (2008) study, pupillary changes varied directly with skin conductance responses (SCR), but not heart rate (HR), which suggests that pupil dilation elicited by affective pictures reflects SNS as opposed to PNS activity. SCR are determined by activity in the sympathetically innervated sweat glands and vary directly with increasing arousal independent of valence (Bradley et al., 2001; Bradley and Lang, 2000; Gomez, Stahel and Danuser, 2004). Thus, it is apparent that the ICR elicited by a stimulus onset (Bakes, Bradshaw and Szabadi, 1990; Giakoumaki et al., 2005) and the subsequent pupil dilation (Morley, Bradshaw and Szabadi, 1991) are both modulated by arousing (appetitive and aversive) as compared to neutral images.

Existing work has shown that pupil activity is a reliable index of individual differences in rumination (Duque, Sanchez and Vasquez, 2014), depression symptoms among at-risk children (Burkhouse, Siegle, Woody, Kudinova and Gibb, 2015), Aspergers Syndrome (Kuchinke et al., 2011), Autism (Nuske, Vivanti, Hudry and Dissanayake, 2014), Bipolar Disorder (Lemaire et al., 2014) and is reflected in administration of oxytocin (Leknes et al., 2013). This suggests that pupillometry will have utility as an index of the physiological component of emotion in relation to the psychopathy dimensions.

5.2.3. Psychopathy and physiological reactivity

A variety of physiological measures have been used across psychopathy studies, including SCR magnitude, HR reactivity or startle reflex, to assess the psychophysiological component of the emotion process. In a recent meta-analysis, psychopathy was associated with lower skin conductance magnitude at rest, during tasks, and as a change from baseline (reactivity, Lorber, 2004), indicating reduced SNS activity. Importantly, the negative association between psychopathy and SCR during tasks was in response to aversive stimuli only. This suggests psychophysiological deficits, in the SNS, when processing aversive stimuli. By contrast, the same meta-analysis indicated that psychopathy was not associated with measures of HR (Lorber, 2004), which is underscored by both SNS and PNS activity but may reflect more influence of the PNS through the vagus nerve (Porges, 2001). This would suggest that psychopathy would be negatively related to levels of initial pupil diameter (lower autonomic arousal), unrelated to changes in the initial constriction response (reactivity; PNS activity) and, most crucially, negatively related to the differentiation between the dilation elicited by aversive as compared to neutral stimuli (affective processing; SNS activity)[30].

[30] Emotionally modulated dilation, as compared to emotionally modulated constriction, was chosen as the third index of pupil activity due to the enhanced role of the SNS in dilation and the here reviewed literature suggesting deficits in sympathetically mediated affective processing in relation to *primary psychopathic traits*.

However, psychopathy-related results from Lorber (2004) tell us nothing about the dual dimensions of psychopathy previously described. Some recent studies assessing psychopathy in terms of its underlying dimensions have found reduced overall levels of skin conductance (Verona et al., 2004), reduced SCR during anticipation of a loud noise (Dindo and Fowles, 2011) and reduced skin conductance differentiation between affective and neutral sounds (Verona et al., 2004), indicating impaired affective processing in relation to the affective-interpersonal factor. This is consistent with Lorber (2004), indicating reduced SNS activity, particularly in relation to *primary psychopathic traits* which could lead to the hypothesis of 1) reduced initial pupil diameter indicating low arousal and 2) reduced differentiation between the dilation elicited by aversive as compared to neutral stimuli (SNS activity) indicating impaired aversive stimulus processing in relation to PCL:SV Factor 1 and TriPM Boldness.

By contrast, *secondary psychopathic traits* are characterised by exaggerated behavioural (e.g., aggressive) reactions (Baskin-Sommers et al., 2012a). Studies of aggression and measures of autonomic activity such as HR (strongly influenced by the PNS) have evidenced a reliable link (see Raine, 2015, Lorber, 2004). Adult aggression is associated with lower resting HR and greater autonomic reactivity (changes from baseline, both HR and skin conductance; Lorber, 2004; Patrick, 2008; although see Patrick et al., 1994, for contrasting results). This would suggest that the *secondary psychopathic traits* would be 3) negatively related to initial pupil diameter (low autonomic arousal) and 4) related to atypical initial constriction responses (reactivity; PNS activity). Results for emotional processing are mixed: high externalising offenders show typical brain response (P300) differentiation

between affective and neutral pictures indicating normal emotional modulation of response (Venables, Bernat, Hall, Steffen, Cadwallader, Krueger et al., 2005), but also show enhanced threat processing under focused attention conditions (Baskin-Sommers et al., 2012a) and enhanced processing of reward anticipation (Buckholtz et al., 2010). Given that the present task conditions did not manipulate attention or stimulus anticipation, 5) typical differentiation between affective and neutral images for offenders high on PCL:SV Factor 2 and TriPM Disinhibition was predicted (see also Venables et al., 2005).

TriPM Meanness reflects both *primary* and *secondary psychopathic traits*. In line with the Affect Categorisation task, hypotheses for this measure of psychopathy were exploratory. This would necessarily limit the interpretation of any significant results in relation to Meanness. However, as discussed in section 4.2.3, any significant results would be interpreted in relation to TriPM Boldness or TriPM Disinhibition.

Also of interest was how measures of pupil activity might relate to an index of antisocial behaviour: number of offences. In a longitudinal study of over 700,000 men, low resting HR at age 18 years predicted antisocial behaviour more than thirty years later – including severe violence, less-severe violence, drug related crime, property crime and traffic crime (controlling for physical, cardiovascular, psychiatric and socioeconomic effects; Latvala, Kuja-Halkola, Almqvist, Larsson and Lichtenstein, 2015). Impulsive-antisocial traits of psychopathy have strong links to criminal history variables such as frequency of offending (Hare, 2003). If a similar pattern of relations were found between pupil activity and number of offences, as between pupil activity and indices of *secondary*

psychopathic traits, this would go towards supporting a link between autonomic dysregulation as assessed by pupil response and both psychopathology and relevant behavioural outcomes.

5.3 Method

5.3.1 Participants

Of the sample of 94 offenders, pupil data was not collected from 13 participants due to failed calibrations. Data from a further three participants was excluded due to excessive data loss on more than 50% of the trials, and data from one further participant was excluded due to complete data loss on all trials featuring neutral stimuli. This resulted in a test sample of $n = 77$. All participants had normal or corrected to normal vision.

Nineteen offenders in this test sample were receiving a form of psychotropic medication. The impact of medication on pupillary reactions is varied: clonidine (administered to treat attention disorders) reduces pupil diameter (miosis) and prolongs the recovery time of the initial constriction response (Hou, Freeman, Langley, Szabadi and Bradshaw, 2005) while diazepam (from the benzodiazepine family) has no effect on pupil diameter nor any effect on the amplitude of the initial constriction response (Hou, Scaife, Freeman, Langley, Szabadi and Bradshaw, 2006). Administration of serotonin (for treating depression) reduces resting pupil diameter and reduces the maximum acceleration and velocity of the initial constriction response (Millson, Haworth, Rushton, Wilkinson, Hobson and Harry, 1991). Opioids (sedatives) typically cause constriction and reduce dilation (Novitskaya, Dean, Moore, Moore, Nagendran and Sharma, 2009) although large inter-individual

variability is found (Matouskova, Slanar, Chyril and Perlik, 2011) and the initial constriction response remains present and quantifiable (Rollins, Feiner, Lee, Shah and Larson, 2014). In general, pupillary responses to drugs show wide inter- and intra-individual variability (Smith, 1992). For this reason, a broad measure of absence/presence of psychotropic medication was included as a dummy-coded covariate in all regression models as was previously noted in Chapters 3 and 4. Previous work (e.g., Lemaire et al., 2014) found no effect of psychotropic medication treatment (anti-convulsants, anti-depressants, anti-psychotics and benzodiazepines) on pupil reactivity between a group of patients with Bipolar Disorder and matched controls. Descriptive information of the impact of individual medications on pupil activity are presented in Appendix B.

5.3.2 Materials, Design and Procedure

The physiological reactivity task stimulus set is described in section 2.2.4, presentation hardware and software are detailed in section 3.3.1. Details of the IQ measure are provided in section 3.3.4.1 and details of the psychopathy measures are provided in section 3.3.3. Specifics of the task design are provided in section 2.4.2.1.1.3. Details of the experimental session procedure are provided in section 3.6 and expansion of methods of analysis are provided in section 3.4. In brief, participants viewed affective (happy, sad, fear) and neutral images featuring complex scenes of both people,

animals and objects while pupil activity was recorded[31]. Images were matched on luminance, contrast and complexity. Affective images were matched on arousal. The following indices of pupil activity were extracted and calculated on a within-subjects basis: initial pupil diameter, initial constriction response elicited by each image type, and emotional modulation of pupil dilation.

5.3.3 Data cleaning

Full details of data reduction procedures are provided in section 2.4.2.1.2.1. Mean pupil diameter was excluded at data time-points where there was missing data for more than 50% of trials per class of image (deleted data per participant M = 8.00% of all data points, Range: 0 – 45.32%).[32] Any participant with greater than 50% of data deleted would have been excluded from the sample (see Burkhouse et al., 2015); all participants met this inclusion criteria. No outliers were identified for any of the measures of pupil activity. All figures present pupil diameter as a change from baseline.

[31] Due to the practicalities of on-site data collection, luminance levels in the testing rooms were not constant across participants or within individual testing sessions. In dim light conditions, parasympathetic tone is reduced with minimal constriction of the sphincter muscle, while the dilator muscle should receive excitatory input. In brighter light conditions, the sphincter muscle contracts resulting in constriction. Although this limitation is acknowledged, light conditions during testing sessions were not considered to vary greatly from "dark" to "bright", particularly since the immediate luminance levels from the testing screen were constant. Reliability of the pupil response was also good.

[32] Missing or deleted data per time window: IPD: 11.01% of total data points, ICR: 5.48% of total data points, Pupil dilation: 7.52% of total data points.

5.4 Results

Descriptive statistics for pupil activity across the various time windows are provided in Table 19; Figure 20 is provided in order to remind the reader of the different stages of pupil response over time. Initial constriction response and dilation values were adjusted for initial pupil diameter. Emotionally modulated pupil dilation was calculated as the difference in dilation, 1 – 2 s post stimulus onset, between the neutral images and each class of the affective images (mean affective diameter – mean neutral diameter). The larger this value, the greater the impact the affective content, as compared to neutral, had on pupil activity.

Table 19

Mean (SD) pupil diameter, in millimetres, per stimulus content, for specific time windows

Initial pupil diameter	3.53 (.07)		
Stimulus content	ICR	Later dilation	Amount of emotional modulation
Neutral	-.23 (.05)	-.24 (.06)	
Happy	-.22 (.05)	-.19 (.06)	.05 (.07)
Sad	-.17 (.06)	-.12 (.07)	.12 (.07)
Fear	-.20 (.05)	-.18 (.07)	.06 (.07)

Note: ICR = initial constriction response. Standard deviation calculated using corrected sample variance for repeated measures designs as per Cousinea (2005) and Morey (2008).

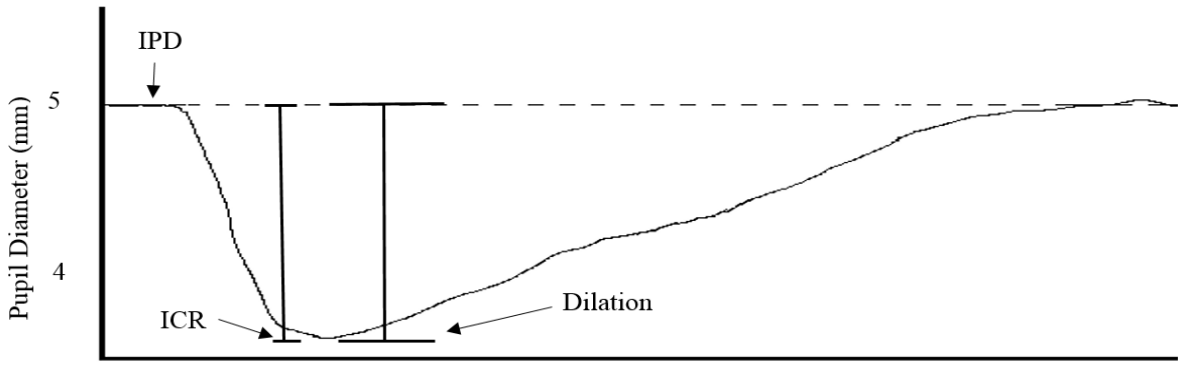


Figure 20. Schematic of pupil response and time windows of interest. IPD = Initial Pupil Diameter; ICR = Initial Constriction Response.

5.4.1 Behavioural indicators

Number of offences were correlated against the measures of IQ and pupil activity (initial pupil diameter [IPD], initial constriction response [ICR] and emotionally modulated dilation; see Table 20). Increasing numbers of offences were related to reduced IPD, restricted ICR to all images and reduced emotional modulation by sad images. IQ was marginally inversely related to increased number of offences (p = .05) and marginally positively related to IPD (p = .06).

Table 20

Zero-order correlations between number of previous offences, pupil activity, intelligence and age

Measure	1	2	3	4	5	6	7	8	9	10	11
1. Number of previous offences	---										
2. Initial Pupil Diameter	-.35**	---									
3. Neutral ICR	-.47***	.54**	---								
4. Happy ICR	-.44***	.58**	.94**	---							
5. Sad ICR	-.42***	.57**	.90**	.92**	---						
6. Fear ICR	-.50***	.54**	.93**	.91**	.91**	---					
7. Happy Modulation	-.12	.15	.26*	.05	.11	.19	---				
8. Sad Modulation	-.36**	.37**	.60**	.51**	.35**	.49**	.57**	---			
9. Fear Modulation	-.01	.14	.27*	.23*	.16	.04	.40**	.49**	---		
10. Intelligence	-.23	.22	.14	.17	.18	.19	.13	.00	.03	---	
11. Age	.06	-.19	.10	.04	.05	.08	-.24*	-.16	-.06	.29**	--

Note: ICR = initial constriction response.

*$p < .05$ **$p < .01$ ***$p < .001$

5.4.2 Psychopathy analyses

Table 21

Relations between PCL:SV factors and pupil activity: initial pupil diameter, initial constriction response and emotionally modulated pupil dilation

	PCL:SV Total		Factor 1				Factor 2			
Criterion Variable	*r*	β	*r*	β alone	β with F2	Unique sr^2	*r*	β alone	β with F1	Unique sr^2
IPD	-.15	-.14	.06	.06	.15	.02	-.27*	-.26*	-.31**	.10**
ICR										
Neutral	-.31**	-.24*	-.06	-.14	-.06	.00	-.27*	-.26**	-.24*	.05*
Happy	-.29*	-.21*	-.11	-.08	-.00	.00	-.39***	-.26**	-.26*	.05*
Sad	-.29**	-.21*	-.05	-.09	-.00	.00	-.40***	-.26**	-.26*	.06*
Fear	-.29**	-.22*	-.05	-.08	.02	.00	-.40***	-.29**	-.30**	.07**
Emotional Modulation										
Happy	-.08	-.07	-.09	-.10	-.10	.01	-.05	-.03	.00	.00
Sad	-.08	-.07	.01	.01	.06	.00	-.15	-.13	-.15	.02
Fear	-.04	-.04	-.02	-.03	-.02	.00	-.02	-.02	-.01	.00

Note: Total = Psychopathy Checklist: Screening Version Total score; F1 = Factor 1; F2 = Factor 2. IPD = initial pupil diameter. ICR = initial constriction response.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

*$p < .05$ **$p < .01$ ***$p < .001$

Table 22

Relations between TriPM factors and pupil activity: initial pupil diameter, initial constriction response and emotionally modulated pupil dilation

	Boldness				Meanness				Disinhibition			
Criterion Variable	*r*	β alone	β with D and M	Unique sr^2	*r*	β alone	β with D and B	Unique sr^2	*r*	β alone	β with M and B	Unique sr^2
IPD	.08	.07	.07	.00	-.12	-.11	-.07	.00	-.14	-.14	-.08	.00
ICR												
Neutral	-.05	-.06	-.09	.01	-.25*	-.25*	-.06	.00	-.29*	-.29*	-.27	.04
Happy	-.12	-.12	-.17	.02	-.29*	-.29*	-.06	.00	-.32**	-.32**	-.31*	.05*
Sad	-.11	-.11	-.15	.02	-.30**	-.30**	-.07	.00	-.33**	-.33**	-.32*	.05*
Fear	-.07	-.07	-.10	.01	-.34**	-.33**	-.11	.01	-.37**	-.37**	-.32*	.05*
Emotional Modulation												
Happy	.13	.12	.13	.01	-.03	.00	-.04	.00	-.02	-.02	.03	.00
Sad	.05	.03	.06	.00	-.13	-.10	-.13	.01	-.06	-.07	.02	.00
Fear	-.04	-.04	-.02	.00	.02	.02	-.04	.00	.08	.08	.10	.00

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness. IPD = initial pupil diameter; ICR = initial constriction response.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

*$p < .05$ **$p < .01$

5.4.3 Initial pupil diameter (IPD)[33]

5.4.3.1 PCL:SV

Analysis using zero-order correlations (see Table 21) indicated that levels of Factor 2 were associated with a reduced IPD. Subsequent analysis using multiple regression methods indicated that Factor 2 significantly predicted levels of IPD while adjusting for presence of psychotropic medication (see Table 21). Partialling out the common variance between Factor 1 and Factor 2 resulted in a boost in the beta values for both factors (Factor 1 changed from .06 to .15, Factor 2 changed from -.26 to -.31) suggesting a suppressor situation. The partialled influence of Factor 2 was responsible for 10% of the variance of IPD explained by the model.

5.4.3.2 TriPM

Correlational analyses indicated that reduced levels of IPD were not associated with the TriPM scales (see Table 22). Multiple regression models adjusting for the effects of medication similarly produced no significant effects (see Table 22).

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[33] As a reliability check the initial pupil diameter was correlated against the pre-task pupil diameter (mean diameter 200 ms prior to first trial stimulus onset). This produced a high correlation, $r(77) = .93$, $p < .01$.

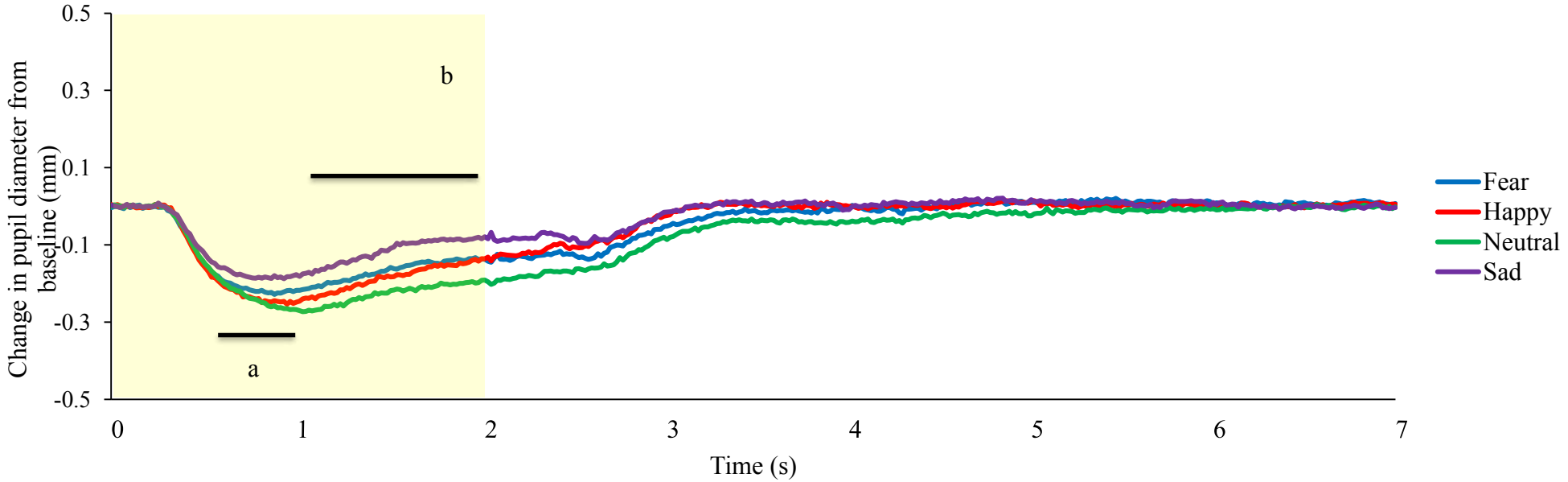

Figure 21. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset when viewing fearful, happy, sad and neutral images. The shaded section represents interval of image presentation. ICR (a) was averaged in a window from 0.5 to 1.0 s following image onset and pupil dilation (b) was averaged in a window from 1.0 to 2.0 s following image onset.

5.4.4. Initial constriction response (ICR)

Figure 21 illustrates the change in pupil diameter during trials. The ICR was inhibited when viewing affective compared to neutral images (see Table 19 for means). Results of a repeated measures ANOVA indicated a significant main effect of content ($F(2.78, 211.39) = 24.80$, $p < .001$, $\eta_G^2 = .03$). Planned analyses indicated that sad ($t(76) = 6.91$, $p < .001$, $d_z = 0.79$) and fear ($t(76) = 3.69$, $p < .001$, $d_z = 0.42$) images elicited an attenuated ICR as compared to neutral images. There was no difference in the ICR elicited by happy as compared to neutral images ($t(76) = 1.40$, $p > .05$). In exploratory analyses, the ICR elicited by sad images was inhibited compared to happy ($t(76) = 6.57$, $p < .001$, $d_z = 0.75$) and fear ($t(76) = 4.56$, $p < .001$, $d_z = 0.52$) images. The ICR elicited by fear images was significantly inhibited compared to happy ($t(76) = 2.26$, $p < .05$, $d_z = 0.26$) images[34].

5.4.4.1 PCL:SV

Correlational analyses, presented in Table 21, indicated that PCL:SV Total and PCL:SV Factor 2 were inversely associated with the magnitude of the ICR to each class of stimulus. As levels of total psychopathy and Factor 2 scores increased, the ICR became more attenuated.

[34] Reliability of the ICR elicited by each category of stimulus: neutral $r = .89$, $p < .001$; happy $r = .89$, $p < .001$; sad $r = .79$, $p < .001$; fear $r = .84$, $p < .001$ (see section 2.4.2.1.2.2 for further details of this reliability calculation).

As seen in Table 20 there was an inverse relationship between the ICR and the initial pupil diameter (IPD), which was apparent in all classes of images. As the IPD became smaller, the ICR became more attenuated. This may be a mechanical restriction, whereby a small pupil has limited scope for further constriction (Smith, 1992).

The IPD was therefore included as a covariate in multiple regression models regressing ICR to all images on to the psychopathy factors. Results suggested that the observed inhibition of the ICR to both affective and neutral images was associated with PCL:SV Factor 2 characteristics independent of the level of IPD and presence of psychotropic medication (see Table 21). The unique characteristics of Factor 2 explained 5 – 7% of the variance in ICR explained by the models, whereas the unique characteristics of Factor 1 explained 0% of the ICR variance across the models.

The relations between PCL:SV Factor 2 and pupil diameter are demonstrated by Figure 22. This figure illustrates the pupil response elicited by fear and neutral images for a subset of offenders scoring high on PCL:SV Factor 2 (> 6) against a subset of offenders scoring low on PCL:SV Factor 2 (< 6). Offenders with higher levels of Impulsive-Antisocial features displayed an inhibited ICR elicited by images with and without affective content as compared to low-Factor 2 offenders.

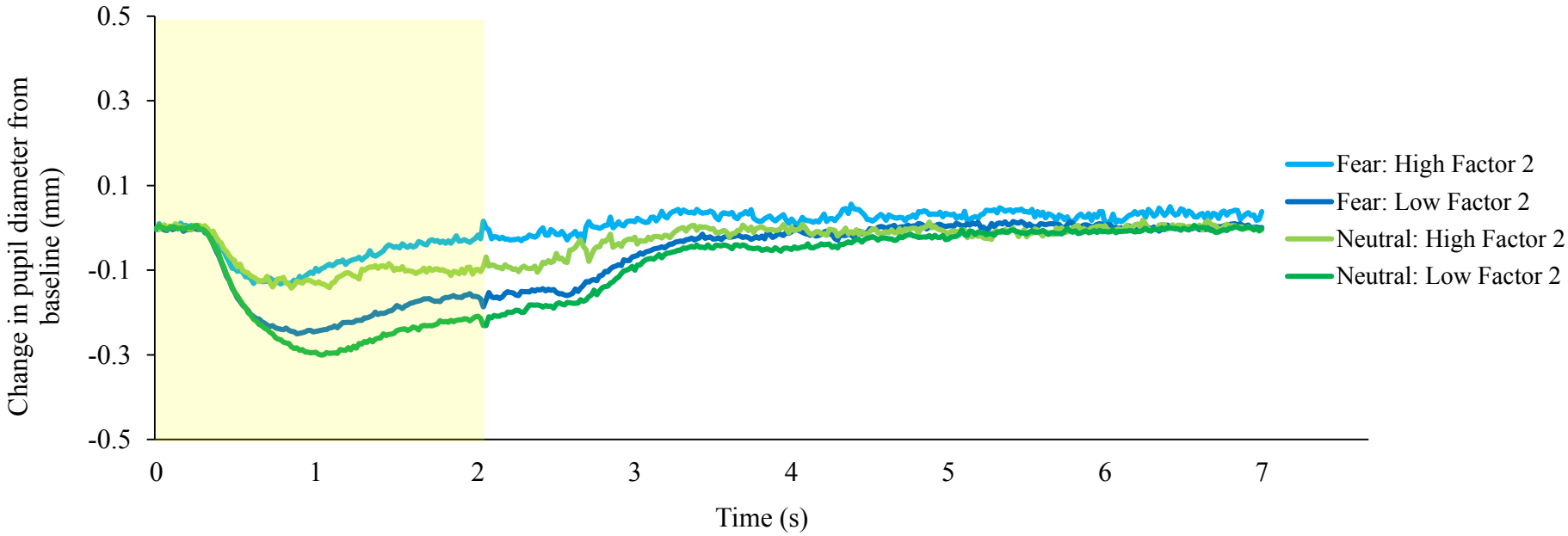

Figure 22. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset when viewing fearful and neutral images for subsets of high and low PCL:SV Factor 2 offenders. The shaded section represents interval of image presentation. The figure illustrates the inhibited constriction response and enhanced pupil dilation to fearful and neutral images for offenders with higher levels of Impulsive-Antisocial traits.

5.4.4.2. TriPM

Correlational analyses, presented in Table 22, indicated that TriPM Meanness and TriPM Disinhibition were inversely associated with the magnitude of the initial constriction response (ICR) to each class of stimulus. As levels of Meanness and Disinhibition increased, the ICR became more attenuated.

As can be seen in Table 22, with initial pupil diameter as a covariate, TriPM Meanness and Disinhibition were the most useful predictors of ICR to both affective and neutral images. The complete TriPM Disinhibition scale predicted an attenuated ICR to neutral images; the slightly larger betas for the complete as compared to partialled TriPM Disinhibition scale for the affective images suggests that the scale in its entirety, i.e., including the shared variance with Meanness, was a useful predictor. The unique variance of TriPM Disinhibition accounted for 4 – 5% of the ICR variance explained by each model, whereas the general variance of TriPM Disinhibition (not reported in Table 22) accounted for 9% (neutral) to 14% (fear) of the ICR variance explained by each model. Similarly, the complete TriPM Meanness scale predicted an attenuated ICR elicited in response to both affective and neutral images, but the partialled variance of Meanness did not predict this index of pupil activity. The unique variance of TriPM Meanness accounted for 0 – 1% of the ICR variance explained by each model, whereas the general variance of TriPM Meanness (not reported in Table 22) accounted for 6% (neutral) to 10% (fear) of the ICR variance explained by each model. This suggests that the full scales of TriPM Meanness and Disinhibition are robust predictors of autonomic activity, and does not support the idea of suppressor situations in relation to the TriPM and this measure of psychophysiology.

5.4.5 Pupil dilation

Pupil dilation following the initial constriction response was also modulated by image content, with larger changes when participants viewed emotional compared to neutral images (see Figure 21). Later pupil dilation was calculated as mean pupil diameter, from 1 – 2 s post stimulus onset for each stimulus type (means presented in Table 19). Assessing for task efficacy, results of a repeated measures ANOVA showed an effect of image content on pupil response: $F(3, 228) = 49.03$, $p < .001$, $\eta_G^2 = .05$. Planned comparisons indicated that all affective images elicited a significantly larger pupil response (happy: $t(76) = 5.69$, $p < .001$, $d_z = 0.65$; sad: $t(76) = 10.82$, $p < .001$, $d_z = 1.23$; fear: $t(76) = 6.29$, $p < .001$, $d_z = 0.72$) as compared to neutral (see Figure 21). Exploratory analyses indicated that sad images elicited a significantly larger pupil response as compared to happy ($t(76) = 7.60$, $p < .001$, $d_z = 0.87$) and fear ($t(76) = 5.48$, $p < .001$, $d_z = 0.62$) images. Happy and fear images elicited a comparable pupil response ($t(76) = 1.33$, $p > .05$).

A period of short secondary constriction followed by redilation is apparent in the time window approximately 2700 ms post stimulus onset (see Figure 21). This secondary response appears to be elicited by the change from target image to recovery screen which occurred at 2000 ms, despite the test images and background screen being matched on luminance. Existing pupillometry literature (Bradley et al., 2008; Bradley and Lang, 2015; Henderson et al., 2014) does not evidence this secondary constriction and redilation, possibly due to the longer interval of stimulus presentation (6000 ms for each of these three studies). Barbur (2004) notes that the processing of stimulus structure or motion can lead to small pupil constrictions. For example, stimuli comprised of equal luminance

gratings elicit a constriction despite being matched in overall luminance to the uniform background (Barbur, 1991). Heller, Perry, Jewett and Levine (1990) also suggest that this secondary response, found in their study following a 200 ms light stimulus, reflects a decrease in the level of the increased sympathetic inhibition of the Edinger-Westphal nucleus. The present work, in the absence of a pharmacological blockade manipulation, cannot inform on this possibility. It is possible that despite the link between pupil dilation and SNS activity (e.g., Bradley et al., 2008), pupil dilation may still involve a degree of parasympathetic relaxation (Heller et al., 1990).

5.4.5.1. PCL:SV

Correlational analyses, presented in Table 21, indicated no significant relationships between the PCL:SV factors and emotionally modulated pupil dilation. The results did not change when adjusting for presence of psychotropic medication (see Table 21).

5.4.5.2 TriPM

In line with results from the PCL:SV, there were no significant associations between the TriPM scales and emotional modulation of pupil dilation using either correlational or regression (see Table 22) methods of analysis.

5.4.6. Covariates

Multiple regression analyses additionally including IQ as a covariate are presented in Appendix B. When controlling for both IQ and medication, the unique variance of PCL:SV Factor 2 predicted a

reduced IPD at a trend level; TriPM Meanness did not significantly predict the ICR elicited by neutral images; the unique variance of TriPM Boldness predicted an attenuated ICR elicited by happy images and marginally significantly (p = .05) predicted an attenuated ICR elicited by sad images, and the complete Boldness scale marginally predicted an attenuated ICR elicited by happy images (p = .06). These changes in the pattern of results are discussed in section 5.5.4.

The size of a pupil at rest in darkness, when the SNS would be most active, has been found to decrease linearly with age from adolescence onwards (see Smith, 1992). Therefore, the role of increasing age in relation to measures of pupil activity was considered (see Table 20). However, in the present sample, age was not consistently related to measures of pupil activity; displaying an (inverse) relationship with the emotional modulation of pupil dilation elicited by happy images only. Age was not included as a covariate in analyses.

Nicotine is a cholinergic agonist which means that its intake will constrict the pupil (Erdem, Gundogan, Dinc, Yolcu, Ilhan and Altun, 2015). Nicotine intake (measured in number of cigarettes or hand-rolled cigarettes smoked a day) was recorded from participants and correlated against the measures of pupil activity, with increased nicotine intake associated with an attenuated ICR elicited by neutral ($r(77) = .30$ $p < .01$), sad ($r(77) = .23$, $p < .05$) and fear ($r(77) = .24$, $p < .05$) images. The pattern of results presented in the main analysis did not change when nicotine intake was included as a covariate. For brevity, these results are not presented in this work.

Caffeine is a stimulant which, in chronic users, has no effect on pupil size, but caffeine intake for non-users causes dilation (Wilhelm, Stuiber, Ludtke and Wilhelm, 2014). Caffeine intake (measured in number of caffeinated beverages consumed, such as tea, coffee or caffeinated soft drinks, a day) was recorded from participants and correlated against the measures of pupil activity, with increased caffeine intake associated with reduced emotional modulation of pupil dilation by sad images ($r(77) = -.23$, $p < .05$). The pattern of results presented in the main analysis did not change when caffeine intake was included as a covariate. Similarly to the checks for nicotine, these results are not presented in this work for brevity.

5.5 Discussion

Both the initial constriction response (ICR) and later pupil dilation were found to be emotionally modulated, lending support to recent results highlighting the early and continued impact of affective stimuli on pupil diameter (Bradley et al., 2008; Bradley and Lang, 2015; Henderson et al., 2014). The results adhered well to the pattern found in the undergraduate sample (see section 2.4.2.2.2).

In contrast to Bradley and Lang (2015) and Henderson et al. (2014), where the ICR was modulated in response to both appetitive (erotic) and aversive (violent) content, the ICR here was inhibited only in response to aversive (sad and fearful) content. The set of happy images did not inhibit the amplitude of the ICR as compared to neutral. This is suggestive of a negativity bias, where negative events or stimuli in general elicit more rapid and stronger bodily responses than positive events or

stimuli (Onorati, Barbieri, Mauri, Russo and Mainardi, 2013) – which has clear adaptive advantages (Rozin and Royzman, 2001) and coheres with the specificity of the threat-attenuated ICR (Bitsios et al., 1996; Giakoumaki et al., 2005). Studies utilising psychophysiological measures have indexed a negativity bias through event-related potential (ERP) responses (Carretie, Mercado, Tapia and Hinojosa, 2001) and pupil response to emotional stimuli (van Steenbergen et al., 2011). Although happy images did not elicit a differential ICR as compared to neutral, happy images did elicit a larger dilation as compared to neutral images. Pupil dilation, therefore, was modulated by both pleasant and unpleasant images.

The data was also highly reliable, as evidenced by the strong correlation between the initial pupil diameter (IPD) and pupil diameter prior to the first trial, and the strong split-half correlations for each class of image during the ICR and the window of dilation. Pupil diameter changes were consistent within-individuals across the task indicating that participants did not appear to become fatigued; attention was sustained and the pupil did not fluctuate greatly. Previous work (Snowden et al., 2016) has established that the differential pupil response elicited by neutral and affective images does not decrease (habituate) across multiple presentations of, e.g., different fearful images within blocks, and also does not habituate across several blocks. Results such as this suggest that emotional modulation of the pupil response is preserved across multiple presentations. Extant literature, therefore, suggests that the pupillary system is a sensitive, low-noise system for reliable, unobtrusive psychophysiological measurement (Beatty and Lucero-Wagoner, 2000).

With respect to psychopathy the following pattern of results was found. First, IPD was found to be smaller in those with higher levels of PCL:SV Factor 2 traits, whilst there was no relationship to PCL:SV Factor 1 traits. IPD was not related to psychopathy as assessed by the TriPM scales. Second, the ICR was smaller for those with higher levels of PCL:SV Factor 2 and TriPM Disinhibition, and TriPM Meanness, but there was no relation to PCL:SV Factor 1 and TriPM Boldness. This reduced ICR in relation to *secondary psychopathic traits* was found for both neutral stimuli and when the stimuli contained affective content and was independent of IPD. Third, none of the indices of psychopathy were related to changes in the amount of emotional modulation of the pupillary responses. Atypical pupil activity thus appeared specific to *secondary psychopathic traits.* These findings are now discussed in turn.

5.5.1 Initial pupil diameter

A small initial pupil diameter (IPD) is thought to correspond to a state of low-arousal (Gilzenrat et al., 2010; Laeng et al., 2012; Van der Meer et al., 2010). The pre-experimental/trial pupil diameter represents the tonic mode of pupil activity in Aston-Jones and Cohen's (2005) Adaptive Gain Theory. The tonic mode is considered an exploration mode that adaptively adjusts the scope of attention to optimise shifts of performance between tasks/events (Aston-Jones and Cohen, 2005). Individuals in tonic mode can more effectively abandon a current task for another (Gilzenrat et al., 2010; Laeng et al., 2012; Van der Meer et al., 2010). A relatively larger IPD can be seen as an indicator of a more pronounced tendency toward adaptive task-free exploring and scanning of the environment (Gilzenrat et al., 2010; Laeng et al., 2012; Van der Meer et al., 2010).

The reduced IPD in relation to PCL:SV Factor 2, therefore, suggests both atypical attentional states and reduced resting levels of exploratory behaviour, commensurate with *secondary psychopathic traits* reflecting a regulatory deficit (Dindo and Fowles, 2011). The reduced IPD in the current study may represent indirect psychophysiological evidence of cognitive-executive dysfunction in relation to offenders high on *secondary psychopathic traits.* Levels of impulsive-antisocial behaviours are often associated with reduced amplitude of the P300 ERP response, which is thought to reflect a deficit in general cognitive efficiency (Patrick and Bernat, 2009). Similarly, *secondary psychopathic traits* are associated with reduced amplitude of an ERP response which occurs following errors in laboratory tasks. The occurrence of the error-related negativity (ERN) is thought to reflect the detection of errors and the brain's automatic capacity to monitor and correct behavioural performance (Patrick and Bernat, 2009). Reduced ERN amplitude has been demonstrated in relation to trait impulsivity (Potts, George, Martin and Barratt, 2006). Collectively, studies examining the P300, the ERN and the present work suggest impaired monitoring of both the self and the environment in relation to Factor 2; possibly reflecting impairments in fronto-cortical systems that mediate anticipation, planfulness and behavioural control (Patrick and Bernat, 2009).

By contrast, the TriPM scales were unrelated to levels of IPD. This may be attributable to content differences between the PCL:SV and TriPM. Low resting autonomic activity, specifically low resting HR, is a significant risk factor for aggressive behaviour (Latvala et al., 2015). Reduced IPD may represent a similar risk factor, and as such may be most relevant in relation to measures that index such aggressive behavioural outcomes. The PCL items were extrapolated from exclusively criminal

samples; the PCL measures have been criticised as overly indexing maladjustment (Cooke and Michie, 2001). In this instance, this may be a strength rather than a limitation as the behaviourally-based PCL:SV may have been well constructed in relation to low IPD. By contrast, the TriPM items were designed to primarily index personality dispositions as opposed to overt behaviours, with the former perhaps being less relevant in relation to reduced IPD.

The inverse relationship between IPD and number of offences is also commensurate with low resting HR as an unequivocal risk factor for aggression (Latvala et al., 2015) – indicating that low autonomic arousal may be a risk factor for antisocial behaviour associated with *secondary psychopathic traits.* Explanatory constructs for this link between reactivity and behaviour are provided in the below section.

Some evidence was found for suppressor effects in the IPD analysis. There was evidence of cooperative suppressor effects for Factor 1 and Factor 2 (see also Blonigen et al., 2010; Hicks and Patrick, 2005; Verona et al., 2005). The beta for both factors increased in size when common variance shared with the other factor was partialled out as compared to the individual models (e.g., the beta for Factor 1 increased from .06 to .15; the beta for Factor 2 increased from -.26 to -.31). The unique variance of Factor 2 explained between 5 and 7% of IPD variance explained by the model. These results highlight the importance of assessing psychopathy in terms of the underlying dimensions in order to reveal diverging associations.

5.5.2 Initial constriction response

Levels of *secondary psychopathic traits* (PCL:SV Factor 2 and TriPM Disinhibition) and TriPM Meanness predicted inhibition of the initial constriction response (ICR) to all stimuli, including those with no affective content. This pattern was found to be independent of initial pupil diameter (IPD). Psychopathy-related changes in ICR were, therefore, not secondary to changes in IPD. The ICR is thought to predominantly reflect parasympathetic activity (Bitsios et al., 1996; Clarke and Ikeda, 1985; Giakoumaki et al., 2005); several authors have suggested that the inhibition of the ICR in response to arousal is consistent with inhibition of the parasympathetic oculomotor reflex arc, as opposed to sympathetic excitation (Bakes et al., 1990; Giakoumaki et al., 2005; Smith, 1992; Steinhauer, Condray and Kasparek, 2000; Steinhauer et al., 2015).

Support for inhibition of PNS activity comes from research where drugs are administered which block sympathetic innervation of the iris, allowing only parasympathetic activation (Giakoumaki et al., 2005, Steinhauer et al., 2015). The arousal-induced reduction in the amplitude of the ICR in the study of Giakoumaki et al. (2005) was identical for participants who had received either dapiprazole (a peripheral sympathetic blockade agent) or a placebo, suggesting that the blockade of sympathetic mediation did not affect the inhibition of the ICR by arousal (see also Steinhauer et al., 2015). The primary pathway for reduction of the ICR in response to arousal appears to be mediated by parasympathetic inhibitory processes at the Edinger-Westphal complex of the oculomotor nucleus. Hence, this result suggests greater inhibition of the PNS for those high on *secondary psychopathic traits.*

Number of offences was also related to inhibition of the ICR, reflecting inhibition of the PNS, elicited by both affective and neutral images. Inhibition of the PNS is, therefore, related to *secondary psychopathic traits* and, in the present study, with increased rates of offending.

According to Patrick (2008), robust physiological correlates of persistent aggressive behaviour include low resting autonomic activity and a lack of parasympathetic (vagal) control. This combination may be particularly risky for individuals with dispositional disinhibition such as those high on *secondary psychopathic traits*. Decreased PNS activity is associated with greater levels of hostility (Demaree and Everhart, 2004) and with individuals with ASPD (Raine, Lencz, Bihrle, LaCasse and Colletti, 2000). Studies of parasympathetic versus sympathetic facilitation of cardiovascular activity have provided evidence of weaker parasympathetic mediation of HR activity in children and adolescents with aggressive conduct problems (see Patrick, 2008). Reduced PNS activity, as measured by reduced amplitude of the ICR, is also associated with higher levels of anxiety (Bitsios et al., 1996; Bitsios, Szabadi and Bradshaw, 2002). Measures of anxiety have also been shown to positively correlate with PCL:R Factor 2 (Verona et al., 2001). The withdrawal of PNS activity may contribute to the observed emotional dysregulation in terms of the negative affect (both hostility, Demaree and Everhart, 2004; and anxiety, Bitsios et al., 1996; Bitsios et al., 2002) common to individuals high on *secondary psychopathic traits* that influences both behaviour and emotional reactions (Dadds et al., 2006; Hicks and Patrick, 2006). However, in the present work PCL:SV Factor 2 and TriPM Disinhibition were unrelated to measures of negative affect. As noted in section 4.5.2.2, alternative

measures of negative affect may better index the emotional dysregulation that possibly relates to withdrawal of PNS activity.

Two explanatory constructs, fearlessness theory and stimulation-seeking theory, have been put forward as the link between atypical autonomic activity and antisocial behaviour. Fearlessness theory views those with reduced autonomic activity as relatively fearless, even in unfamiliar contexts, compared with more fearful individuals. Fearless individuals are more likely to engage in antisocial behaviour because they are less fearful of the consequences of their actions and possible physical injury to themselves (Lykken, 1995). Stimulation-seeking theory suggests that states of physiological under-arousal are unpleasant and lead individuals to seek stimulation in an effort to increase their arousal levels to an optimal level. This produces sensation seeking behaviour (Zuckerman, 1990), which often involves engaging in particularly risky acts that the individual perceives as stimulating, possibly including violence. Beyond the association with increased numbers of offences, the present study cannot differentiate between these two theoretical accounts. Future work should include direct measures of anxiety and sensation seeking to test the underlying mechanism of the relationship between autonomic arousal and offending behaviour, as well as to clarify the role of inhibited autonomic activity in the cognitive-affective dysfunction associated with *secondary psychopathic traits.*

In terms of suppressor effects for the ICR, partialling out the common variance added little validity to Factor 2 as evidenced by the small increase in size between the beta coefficients. In relation to the TriPM scales, TriPM Meanness was only a significant predictor as a function of its overlap with

TriPM Disinhibition. Although the size of the partialled beta's for TriPM Disinhibition were robust, the pattern of results suggested the complete scale, including the variance shared with Meanness, was the better predictor. For both measures, the inclusion of the additional factor or scales did not provide clear evidence of suppression.

5.5.3 Emotionally modulated pupil dilation

In contrast to the hypothesis that PCL:SV Factor 1 and TriPM Boldness would be associated with reduced emotional modulation of the pupil response by threat images, no such relationship emerged. Similarly no relationships emerged between PCL:SV Factor 2, TriPM Disinhibition and Meanness, and emotionally modulated pupil dilation. By contrast, studies measuring startle reflex amplitudes in relation to psychopathy have reliably found reduced startle potentiation for aversive visual stimuli in relation to levels of *primary psychopathic traits* (e.g., Baskin-Sommers et al., 2013; Flor et al., 2002; Patrick et al., 1993; Vaidyanathan et al., 2011). The threat-potentiated startle reflex is an index of defensive reactivity in animals and humans; the pathway for this reflex descends from the amygdala (Patrick and Bernat, 2009). Blink potentiation in typical samples is strongest for directly threatening (e.g., a pointed weapon or aggressive animals) images and occurs to a less reliable extent for vicarious (victim) scenes involving physical injury or aggression (see Patrick and Bernat, 2009). In the present work, eight of the ten fear images represented a direct threat, which suggests that a significant relationship should have been found between levels of *primary psychopathic traits* and emotional modulation of pupil dilation by threat images.

Moul et al. (2012) note that empirical studies consistently evidence that individuals high on interpersonal-affective deficits have a lower conditioned threat response (e.g., Birbaumer et al., 2005, Flor et al., 2002; but see Schultz, Balderston, Baskin-Sommers, Larson and Helmstetter, 2016, for a suggestion that central presentation of the conditioned and unconditioned stimuli may ameliorate threat deficits in relation to *primary psychopathic traits*). Psychopathic individuals have also demonstrated impaired aversive conditioning across measures including skin conductance and corrugator responses (Flor et al., 2002). This deficit in the conditioned threat response indicates that individuals high on *primary psychopathic traits* have lower psychophysiological responses to neutral stimuli that have previously been paired with, and therefore are predictive of, fear-eliciting stimuli (Moul et al., 2012) suggesting impaired attentional processing of threat cues. By contrast, threat responses to unconditioned aversive stimuli are intact. In the study of Flor et al. (2002), for example, aversive conditioning to an unpleasant odour was impaired in a group of psychopaths, but this deficit was not related to reactivity to the stimulus itself as demonstrated by a lack of significant group differences in response to the unconditioned stimulus. Moul and colleagues (2012; see also Flor et al., 2002) suggest that these individuals may have a relatively intact aversive response, but an impaired ability to respond to cues that signal the occurrence of aversive stimuli. *Primary psychopathic traits* may be associated with normal responses to emotional events, while also being associated with attenuated reactivity to cues that predict such events. This suggests a primary deficit in aversive learning.

In relation to *secondary psychopathic traits,* high externalising participants show no reduction in brain response differentiation between affective and neutral pictures, indicating intact processing of

the affective content of appetitive and aversive pictures (Venables et al., 2005) which is supportive of the present results. By contrast, during experimental stressors, *secondary psychopathic traits* are related to enhanced psychophysiological reactivity (Dindo and Fowles, 2011; Gallo, Smith, and Kircher, 2000; Lorber, 2004; Patrick, 2008). In the study of Schultz et al. (2016), levels of *secondary psychopathic traits* were related to reduced threat conditioning despite the central presentation of the conditioned stimulus, a finding not easily explained by the *response modulation hypothesis*. The authors suggested that higher levels of anxiety may undermine threat responses, a hypothesis that requires further exploration. Patrick et al. (1994) found that deficits in SCR and HR changes (weaker differentiation between fearful and neutral remembered sentences) were specific to impulsive-antisocial behaviours and did not change as a function of interpersonal-affective traits. However, the same psychopathic sample showed intact SCR and HR changes to affective pictures (Patrick et al., 1993). Psychopathy-related differences in the physiological component of the emotion process may be context specific as opposed to global.

Number of offences was inversely related to modulation of pupil dilation by sad images, suggesting that offenders with an increasing criminal history showed, through pupil response, a reduced differentiation between the sad and neutral images. Given the links between an offending history and both callous-unemotional traits (Hare, 2003) and trauma (Fox, Perez, Cass, Baglivio and Epps, 2015), the desensitisation to images depicting sad scenes is, perhaps, unsurprising. A caveat that should be noted here is that the present work did not distinguish between types of offences in measuring offences, or, indeed, between reactive or instrumental aggression as features of offences.

Some research has suggested a link between indices of *primary psychopathic traits* and instrumental aggression, and between *secondary psychopathic traits* and reactive aggression (Porter and Woodworth, 2006; but see Blais, Solodukhin and Forth, 2014, for a meta-analysis identifying equal relationships between instrumental and reactive aggression and the psychopathy dimensions). A more refined analysis of aggression may be of benefit in future work.

In relation to this supplementary analysis, Cooke, Michie and Hart (2006) have argued that antisocial behaviour is a consequence, as opposed to a symptom, of psychopathy; such behaviour is argued to reflect acts rather than personality and not cohere with Cleckley'an psychopathy. Cleckley's case studies (1955), however, clearly refer to broadly defined, persistent antisocial behaviour – alcohol abuse, engaging in brawls, adultery, theft and fraud. There are also clear arguments for early and persistent patterns of antisocial behaviour which are central to psychopathy; Blonigen, Hicks, Krueger, Patrick and Iacono (2005) have shown that externalising features of psychopathy have significant heritability. More recent structural equation modelling work has identified antisocial behaviour as a core component of psychopathy (Neumann, Hare and Pardini, 2015). Thus, antisocial behaviour is considered relevant to the psychopathy construct and of relevance in relation to physiological reactivity. Furthermore, viewing behaviours and traits as static (i.e., behaviours as a consequence of traits) fails to consider the reciprocal interaction and influence between the two over the course of development. The present work cannot directly inform on the antisocial behaviour debate (see Skeem and Cooke, 2010; see Hare and Neumann, 2010, for a response) and does not try to. Number of

offences are presented as a correlate of the atypical autonomic activity associated with *secondary psychopathic traits*.

5.5.4 Role of intelligence

When adjusting for both IQ and medication, the unique variance of Factor 2 predicted a reduced initial pupil diameter (IPD) at a trend level only while the complete Factor 2 scale did not predict levels of IPD (see Appendix B). This is in contrast to the main analysis (IQ not included as a covariate), where both the complete and unique variance of Factor 2 significantly predicted a reduced IPD. As noted, the IPD represents the tonic, 'exploratory' mode of pupil activity in the Adaptive-Gain theory (Aston-Jones and Cohen, 2005). It is possible that individuals with increasing levels of intelligence would present with larger IPD, reflecting greater levels of exploratory behaviour. This was found to be the case by Heitz, Schrock, Payne and Engle (2006) and Van der Meer et al. (2010). In the present sample, IQ and Factor 2 were inversely associated (see section 3.3.4). It appears that when controlling for individual differences in IQ, which may be an aspect of Factor 2 psychopathy, the relationship between low IPD and Factor 2 was attenuated.

Similarly, when adjusting for both IQ and psychotropic medication, TriPM Meanness did not significantly predict the initial constriction response (ICR) elicited by neutral images. Intelligence and TriPM Meanness were also inversely (non-significantly) associated in the sample (see section 3.3.4). It is possible that offenders with higher levels of intelligence have increasingly typical responses to

neutral stimuli and present with less of a hostility bias (i.e., having a negative response to affectively neutral stimuli, see Dadds et al., 2006).

When adjusting for both intelligence and psychotropic medication the unique variance of TriPM Boldness predicted an attenuated ICR elicited by happy images (the complete TriPM Boldness scale was a marginally significant predictor at $p = .06$). This result is in contrast to the main analysis. Levels of TriPM Boldness and intelligence were not significantly associated in the sample (see section 3.3.4). Although this result seems relatively intuitive, Simmons, Nelson and Simonsohn (2011) emphasise caution when interpreting results which seem to hinge on the presence of a specific covariate for their detection.

5.5.5 Conclusion

PCL:SV Factor 1 and TriPM Boldness were unrelated to physiological activity to affective cues. The results suggest that, for indices of *primary psychopathic traits*, deficits in physiological reactivity to, e.g., threat, may be context specific. Levels of *secondary psychopathic traits* were inversely related to autonomic activity, but this was not affect specific; a similar pattern of physiological response to neutral images was also found. This reduced reactivity was also related to number of offences suggesting a pathway between general inhibition of parasympathetic nervous system activity and antisocial behaviour. Neither dimension of psychopathy was related to emotional modulation of pupil dilation, suggesting that atypical physiological responses in psychopathy to affective cues may be context specific as opposed to global.

Chapter 6: Affective and Semantic Priming

6.1 Abstract

Priming studies are ideal for assessing the impact of affective cues on the behavioural component of emotion. Adult male offenders completed Affective (n = 78) and Semantic (n = 83) Priming tasks which utilised the same set of priming stimuli in order to separate any potential overlap of these associative processes. In each task, participants were presented with first an image prime, typically followed by a target word, and were required to attend to both the prime and the target through directed attention. This was tested through a subset of prime-only trials. Overall patterns of response times (RT) in each task indicated the utility of the task designs. PCL:SV Factor 1 and TriPM Boldness, and PCL:SV Factor 2 and TriPM Disinhibition, did not predict performance on the Affective Priming task. However, levels of TriPM Meanness predicted reduced grand effects of fear primes on RT, underpinned by reduced facilitation of RT by fear primes; enhanced grand effects of happy primes on RT, underpinned by reduced inhibition of RT by happy primes; and reduced grand effects of sad primes on RT. These results would require replication before being considered further. The measures of *primary* and *secondary psychopathic traits* did not predict performance on the Semantic Priming task; levels of the general psychopathy factor (PCL:SV Total) did however predict increased errors on semantically incongruent trials. Specific experimental conditions may impact on finding affective or semantic processing irregularities in relation to the psychopathy dimensions.

6.2 Introduction

6.2.1 Affective Priming

This chapter presents two priming tasks reflecting the fourth assessed component of emotion: behavioural response. Priming tasks represent an under-utilised resource in examining the influence of affective stimuli on behaviour in relation to psychopathy; specifically, the effects of prior context on the categorisation of stimuli. The priming task was originally developed by Fazio, Jackson, Dunton and Williams (1995) to index automatic attitude activation and has since been developed to assess both semantic and affective priming. In the affective priming paradigm, participants are required to evaluate positively or negatively valenced target stimuli that have been preceded by briefly presented priming stimuli. Typically, the speed of target evaluation varies as a function of the match between prime and target valence. Response times (RT) are shorter for affectively congruent prime-target pairs (facilitation) than for affectively incongruent prime-target pairs (inhibition).

The response level account of affective priming suggests that the perception of the prime automatically triggers, via the activation of its valence, an evaluative response tendency that is either correct or incorrect in terms of the targets valence. That response tendency will then facilitate target response in the case of congruent trials, or interfere with target response in the case of incongruent trials (Degner, 2009; Voss et al., 2013).

Short-term affective priming effects[35] are most pronounced at short intervals between the onset of the prime and the onset of the target (stimulus onset asynchronies, SOAs) and even disappear at longer SOA (Hermans et al., 2001) – providing support for the notion that short-term affective priming is based on fast acting, automatic processes. The generality of the priming effect has been established for faces (Rohr, Degner and Wentura, 2012, Experiments 1 and 2), words (Bargh, Chaiken, Govender and Pratto, 1992), auditory tone stimuli (Tartar, de Almeida, McIntosh, Rosselli and Nash, 2012) and auditory speech stimuli (Degner, 2011) amongst others. Several studies have also investigated priming effects in relation to individual differences in alexythmia (Vermeulen, Luminet and Corneille, 2006), attachment avoidance (Suslow, Dannlowski, Arolt and Ohrmann, 2010) and depressive symptoms (Zetsche and Joormann, 2011).

6.2.1.1 Affective Priming and psychopathy

Affective priming has previously been examined in relation to psychopathy. Blair et al. (2006) presented prime words that could be positive, negative or neutral, followed by target words that could be positive or negative. Control offenders showed a significant prime by target interaction: typical facilitation and inhibition of RT was found for both positive and negative trial types. Offenders with

[35] Wentura and Rothermund (2014) distinguish between short-term priming effects, whereby trial by trial effects are used to investigate relationships between mental concepts, and long-term priming effects, where a participant is primed hours or sometimes days before behaviour is assessed. Short-term priming effects include both response priming tasks as in the present work and tasks such as the lexical decision task.

psychopathy showed a marginally significant (p = .05) prime by target interaction which was interpreted as non-significant and not followed up with further analysis.

A priming effect score was calculated by Blair et al. (2006) as the difference in RT between incongruent and congruent trials on a within-subjects basis. This effect was negatively correlated with both PCL:R Factor 1 and PCL:R Factor 2. The effect scores were calculated by collapsing across both positive and negative prime trials. Calculating a general effect score in this manner lacked the clarity required to assess specific relationships, such as whether the impairment was specific to positive or negative primes, or whether this effect was driven by reduced facilitation or inhibition of RT.

In the Blair et al. (2006) study, psychopathic and control offenders showed a similar pattern of errors on the affective priming task. This suggested that the individuals high on psychopathy had the ability to access the lexical content or meaning of the affective target words in order to accurately categorise the targets as positive or negative. However, the speed of this intact categorisation was not influenced by the affective context established by the prime, suggesting that individuals with psychopathy fail to use secondary cues. In light of the *response modulation hypothesis* (see section 1.2.2.2), it is important to establish whether, in priming paradigms, this insensitivity extends to non-affective cues. This was tested in the present work through a semantic priming paradigm.

6.2.2 Semantic Priming

Semantically related items belong to the same category and share semantic properties (e.g., cat and cow are both mammals) or are functionally related (e.g., broom and floor; Voss et al., 2013). As

in affective priming tasks, semantic priming paradigms require participants to categorise target stimuli that have been preceded by briefly presented prime stimuli. These primes are either from the same or an alternative category to the targets. RT is shorter when primes and targets are semantically matched compared to when they are mismatched (see Spruyt, Hermans, De Houwer and Eelen, 2002; Voss et al., 2013).

Semantic priming paradigms, as defined here, refer to priming paradigms which require a categorical evaluation of the target; such a definition is consistent with existing work (Avero and Calvo, 2006) defining semantic priming as the facilitation of the speed or accuracy of response to a target when a previous prime stimulus is related in meaning. In the general priming literature, however, semantic priming is also sometimes used to refer to tasks such as the lexical decision task[36] which are typically contrasted with response priming paradigms (see Lucas, 2000). Although both response priming paradigms and lexical decision tasks are used to assess associative structures or to investigate the mental basis of, e.g., attitudes, the two can be distinguished both structurally and theoretically. Response priming paradigms, which are utilised here, contain primes which are congruent or incongruent to both the target and the response (Wentura and Rothermund, 2014). These paradigms

[36] The lexical decision task is a word-non word discrimination task in which the presence of a prime activates the spreading of the network activation of the corresponding node, with faster RT to the target indicating greater network activation (Reidy, Zeichner, Hunnicutt-Ferguson and Lilienfeld, 2008).

are typically explained by response competition mechanisms while the processes underlying lexical decision tasks are most often explained by a facilitation of target processing.

Previous work in the priming literature has attempted to determine whether genuine affective content is obtained from a prime stimulus, and whether this affective content does, in fact, facilitate the response to the target independent of any semantic association – in short, whether affective and semantic priming as defined here can be separated. Storbeck and Robinson (2004) have suggested that non-affective processes, such as semantic categorisation, also occur automatically and might be involved in the effects attributed to affective priming. In their study, Storbeck and Robinson (2004) found semantic category priming, but not affective priming, with positive and negative images of different categories of animals. These results suggested that objects must be identified perceptually and semantically before their affective content can be extracted. This implied a confound of affective with non-affective (semantic) priming. By contrast, Avero and Calvo (2006; see also Carroll and Young, 2005, Experiment 2) demonstrated that the relative contributions of semantic category processing and affective processing can be separated. A significant affective priming effect was found, which generalized across different semantic categories and was not enhanced by category relatedness (there was no interaction between valence and semantic category; Avero and Calvo, 2006, Experiment 2).

Avero and Calvo (2006) noted that the ability to discern semantic from affective priming probably depends on task requirements. This supposition was recently extended by Voss et al. (2013) who demonstrated that the valence of a prime stimulus is not automatically processed if it is completely

irrelevant to the response category. In a task which orthogonally manipulated the valence and semantic category of both prime and target stimuli, only a main effect of semantic priming was found when the task required a semantic categorisation of the target, and a main effect of affective priming was found when the task required an affective categorisation (valence) of the target. Category relatedness did not facilitate the processing of affective content when it was not relevant to task requirements[37]. Thus, a semantic priming task would serve as a 'control' paradigm, assessing whether any predicted deficits or dysregularities in relation to *primary* and *secondary psychopathic traits* on the affective priming task extended to the semantic processing of stimuli also.

6.2.2.1 Semantic Priming and psychopathy

Blair et al. (2006) demonstrated intact semantic priming in relation to psychopathy. Participants were presented with prime and target words related to either animals or fruit. Both control and psychopathic offenders showed significant semantic priming for animal primes followed by animal targets. A semantic priming effect score was calculated as the difference between incongruent and congruent trials, collapsed across animal and fruit primes. Neither PCL-R Factor 1 nor Factor 2 were

[37] Results of Avero and Calvo (2006) and Voss et al. (2013) indicate only that affective and semantic priming can be separated; the authors do not suggest that affective evaluation precedes cognition. The relationship between affect and cognition is clearly relevant in the context of psychopathy. The specific temporal primacy of affect over cognition or vice versa, although interesting, is however not the central focus of this chapter.

related to the magnitude of the semantic priming effect. Levels of psychopathy were also unrelated to error rates.

The study of Blair et al. (2006) has some methodological limitations. The prime stimulus sets differed across the affective and semantic priming tasks so a direct performance comparison of affective versus semantic priming may not have been appropriate. Blair et al. (2006) noted that a direct copy of their affective priming task did not, during task piloting, elicit reliable semantic priming. To get reliable semantic priming, instructions required participants to indicate whether a target word belonged to a previously specified target word category (animal or fruit), thus requiring a binary yes or no response, as opposed to categorising the target word as either an animal or fruit. Lucas (2000) has noted that deciding whether a target word belongs to a specified category would require participants to focus explicitly on the meaning of the stimuli and possibly encourage strategic processing. This is in contrast to the instructions for the affective priming task (Blair et al., 2006) whereby participants were required to indicate what valence category a target word belonged to (positive or negative). As noted above, both psychopathic and control offenders in the Blair et al. (2006) study showed significant semantic priming for animal targets, but neither did for fruit targets. This methodological limit may be due to the 'fruit' category actually consisting of both fruit and vegetable related words.

Voss et al. (Experiment 2, 2013) present a more elegant design for assessing semantic and affective priming. Using the same set of stimulus materials, the semantic congruency (person or objects) of prime-target pairs was manipulated orthogonally to the affective congruency (positive or

negative). Four priming conditions were therefore created: Affective and Semantic Congruency; Affective Match/Semantic Mismatch; Affective Mismatch/Semantic Match, and Affective and Semantic Mismatch. In the affective condition, participants were required to make an evaluative decision (positive versus negative) to the target; in the semantic condition, targets were classified as a person versus an object. In each condition, only the task-relevant dimension (affective or semantic) had an influence on performance, as measured by RT. Given that the very same stimulus materials were used in both tasks, the pattern of results suggested that affective and semantic priming processes could be reliably separated.

6.2.3 Priming and attention

Although priming is suggested to be based on automatic processes, active attention to the prime can enhance the priming effect. In a meta-analysis of lexical decision tasks, Lucas (2000) found that all but one of the included studies required the participant to attend to the prime. Recent results (Andrews et al., 2011; see also Carroll and Young, 2005) found larger priming effects in conditions that required a report of the emotion expressed by the face primes (see also section 2.4.3.1.2 for a discussion of Fazio et al., 1986). In the study of Andrews et al. (2011), participants either reported whether the target word was pleasant or unpleasant, or reported the emotion of the prime in addition to the target evaluation. Overall, priming effects (incongruent – congruent RT) were larger in the conditions that required a report of the prime emotion. As would be expected, RT's were longer in the reporting condition as compared to the no reporting condition, which likely reflects the additional processes required to identify the prime content.

Thus, it seems that priming may not be as immune to explicit processes as previously assumed. These results bring into question whether or not priming is automatic. Automaticity is usually defined by a set of criteria that include fast processing of a stimulus without awareness, attention or inattention (Lucas, 2000). A number of studies have demonstrated that the effects found, e.g., in lexical decision tasks, only occur if participants are instructed to attend to the prime (Henik, Friedrich and Kellog, 1983; Stolz and Besner, 1999); priming can be reduced or eliminated by a task that affects the distribution of attention during processing of the prime word (Henik et al., 1983). It may be that attention acts to control how automatic activation is distributed across different levels of representation, but that attentional control does not necessarily imply a conscious process (Stolz and Besner, 1999). Whether or not automaticity requires attention is however tangential to the presented work. The current chapter assessed the impact of directed attention in relation to the psychopathy dimensions: when attending to both the prime and the target was relevant for task performance.[38]

In typical priming paradigms, the valence of the prime is irrelevant to the task goal of categorising the target, yet performance (as assessed through RT) on the task can be facilitated or inhibited by this irrelevant information. In the study of Blair et al. (2006), performance on the affective priming task for psychopathic participants was not affected by the prime information, with no

[38] Implicitly, attending to the target over the prime remained the dominant response as the majority of trials in both tasks were prime-target trials. However, participants were not aware of this and so in order to perform the task according to the set instructions had to attend to both prime and target on each trial.

inhibition or facilitation of RT as a function of prime word valence. This suggests that the dominant response – categorising the valence of the target – was not influenced by secondary affective information, the valence of the prime, in line with the *response modulation hypothesis* (see section 1.2.2.2). Performance on the semantic priming task was, however, affected by the prime information (Blair et al., 2006); individuals with psychopathy showed priming for animal primes followed by animal targets. This suggests that secondary non-affective information was appropriately attended to; a result which contradicts the *response modulation hypothesis*.

6.2.4 Present study

The present study aimed to assess affective and semantic priming in relation to the two psychopathy dimensions as assessed through alternate measures of psychopathy. By utilising the same set of prime stimuli, the present work aimed to address some of the methodological limits of Blair et al. (2006). By orthogonally rotating the affective and semantic dimensions of the prime stimuli in the semantic priming task, it would be possible to ascertain whether levels of *primary* and *secondary psychopathic traits* would be related to priming effects when participants were focused on the semantic component of an affective stimulus. The inclusion of an affective component made this task a more stringent test of semantic priming, statistically allowing for a separation of affective and semantic priming within the same task. The present work also extended the study of Blair et al. (2006) by examining specific negative affect primes (i.e., fear and sad primes) as opposed to the broader valence category of 'unpleasant'.

This final experimental chapter assessed the behavioural component of the emotion process. Measures of RT were taken as indicators of the extent to which the affective cue, the prime, influenced the speed of response, the behaviour, to the target stimulus. For the Affective Priming task, in order to be broadly comparable with the approach of Blair et al. (2006) grand effects of each prime type (happy, sad and fear) on RT were calculated on a within-subjects basis. Were significant results found, these grand effects would then be parsed into facilitation and inhibition scores in exploratory analysis, reflecting the facilitation and inhibition of RT by each prime type relative to neutral primes. For the Semantic Priming task, a grand effect of semantic priming was calculated on a within-subjects basis. In both priming tasks, the PCL:SV and TriPM were assessed in relation to errors committed on incongruent trials. These error rates may be indicative of an individual's ability to modulate the response established by the prime in favour of the response required by the target (Howland, Kosson, Patterson and Newman, 1993).

The inclusion of directed attention to the prime allowed an examination of competing hypotheses within psychopathy: affective (*amygdala dysfunction hypothesis*) versus attentional (*response modulation hypothesis*) impairments. The inclusion of the Semantic Priming task allowed a test of these competing hypotheses. As noted in Chapter 1, both the *amygdala dysfunction* and *response modulation* hypotheses typically refer to a unitary syndrome of psychopathy. Therefore, if levels of *primary* (PCL:SV Factor 1 and TriPM Boldness) and *secondary psychopathic traits* (PCL:SV Factor 2 and TriPM Disinhibition) were related to impaired performance on the Affective Priming task, but not the Semantic Priming task, while attending to contextual cues, that would be considered support for the *amygdala dysfunction hypothesis*. The *response modulation hypothesis*, by contrast, would

predict that any impairments in priming on both the Affective and Semantic Priming tasks in relation to *primary* and *secondary psychopathic traits* would be ameliorated by the attention manipulation.

As is consistent throughout the thesis, no specific hypotheses were devised in relation to TriPM Meanness. Again, this necessarily limits the interpretation of results in relation to Meanness, but as has been argued previously in Chapters 4 and 5, any significant results in relation to TriPM Meanness would be cautiously interpreted in light of other results more definitively supportive of either *primary* or *secondary psychopathic traits.*

6.3 Affective Priming task: Method

6.3.1 Participants

Priming data was not included from 18 participants, leaving a total n = 76. Of these 18 participants, data from 13 offenders were removed due to having less than 66% correct on average (percentage correct for these 13 participants M = 54%, Range = 35.42 – 61.46%). Data from a further two participants were removed due to having 0% accuracy on Fear-Positive trials. Data were not collected from a further three participants due to refusal. Nineteen offenders in this test sample were receiving a form of psychotropic medication. As is consistent throughout the thesis, multiple regression analyses adjust for psychotropic medication. Descriptive details of the impact of individual medications on task performance are presented in Appendix C.

6.3.2. Materials, Design and Procedure

Full details for the prime stimuli and target stimuli used in the Affective Priming task are provided in section 2.2.4 and section 2.3 respectively. Presentation hardware and software are detailed in section 3.3.1. Details of the IQ measure are provided in section 3.3.4.1 and psychopathy measures are described in section 3.3.3. Specific details of the Affective Priming task design are provided in section 2.4.3.1.1.3. Details of the experimental session procedure are provided in section 3.6 and expansion of methods of analysis are provided in section 3.4. In brief, participants viewed briefly presented affective (happy, sad, fear) and neutral prime images, made up of complex scenes containing both animals and people. On the majority of trials, the image was replaced by a target word which was either positive or negative. Participants were required to categorise the target word as good or bad. On a minority of trials, only the prime image was presented which the participant was required to categorise as good or bad. Practice targets were randomly selected from the list of test targets. This was to give the offender participants as much opportunity as possible to become familiar with and confident in responding to the test targets[39].

[39] This also applied in the Semantic Priming task.

6.4 Affective Priming task: Results

6.4.1 Data cleaning

RT for trials on which participants correctly classified the valence of the target stimulus were screened individually and outliers deviating from the mean by more than 3 *SD* were removed (6.81% of all trials). RT analyses were conducted on correct responses only. Error data were analysed separately and converted into percentage scores. Data from practice trials were not included in analyses. In both priming tasks, RT data exhibited a strong positive skew and were log transformed to achieve normalization. Analyses were conducted on transformed RT. In both priming tasks, graphs utilise untransformed RT in milliseconds for clarity.

6.4.2 Prime evaluation data

Participants were instructed to attend to both the prime and the target. As a manipulation check, prime evaluation scores were calculated for each participant by subtracting the proportion of incorrect responses from the proportion of correct responses on prime-only trials. Performance at chance levels would be indicated by a score of zero on this evaluation index. Participants performed at better than chance levels; the mean score was .65 (SD = .25) which a one sample t-test indicated was significantly different from zero, $t(75) = 22.74$, $p < .001$, $d_z = 2.61$.

6.4.3 Response Time data

RT to congruent, neutral and incongruent trials are presented in Figure 23. Assessing for task efficacy, a repeated measures (prime: congruent, neutral, incongruent) ANOVA indicated a main effect of the prime condition, $F(2, 150) = 11.83$, $p < .001$, $\eta_G^2 = .08$. Planned comparisons indicated that RT to congruent trials were significantly faster than neutral ($t(75) = 2.47$, $p < .01$, $d_z = 0.28$) or incongruent trials ($t(75) = 4.88$, $p < .001$, $d_z = 0.56$). Incongruent trials were slower than neutral ($t(75) = 2.39$, $p < .05$, $d_z = 0.27$).

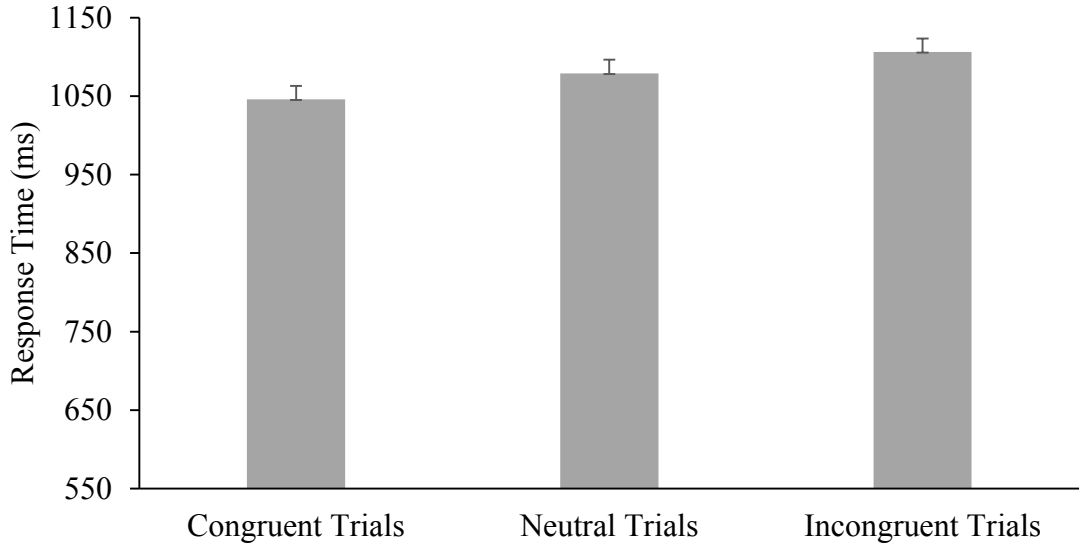

Figure 23. Overall pattern for Affective Priming task. Error bars show 2 *SE* (correction for repeated measures designs, Cousineau (2005) and Morey (2008)).

The priming effect by each prime type can be seen in Figure 24. In comparison to neutral primes, happy primes facilitated RT to positive targets while slowing down RT to negative targets. The sad

and fear primes did the opposite. In order to quantify this effect, three indices were calculated (in line with Vermeulen et al., 2006; referred to here as the grand effects of each prime on RT):

1. Happy prime grand effect = (RT for neutral-positive trials minus RT for happy-positive trials; facilitation) – (RT for neutral-negative trials minus RT for happy-negative trials; inhibition);
2. Sad prime grand effect = (RT for neutral-negative trials minus RT for sad-negative trials; facilitation) – (RT for neutral-positive trials minus RT for sad-positive trials; inhibition);
3. Fear prime grand effect = (RT for neutral-negative trials minus RT for fear-negative trials; facilitation) – (RT for neutral-positive trials minus RT for fear-positive trials; inhibition).

A one-sample *t*-test on each of these grand effect indices indicated that each was significantly different from zero (happy: $t(75) = 2.65$, $p < .05$, $d_z = 0.30$; sad: $t(75) = 3.83$, $p < .001$, $d_z = 0.44$; fear: $t(75) = 2.62$, $p < .05$, $d_z = 0.30$). Significant priming effects were elicited by each class of image.

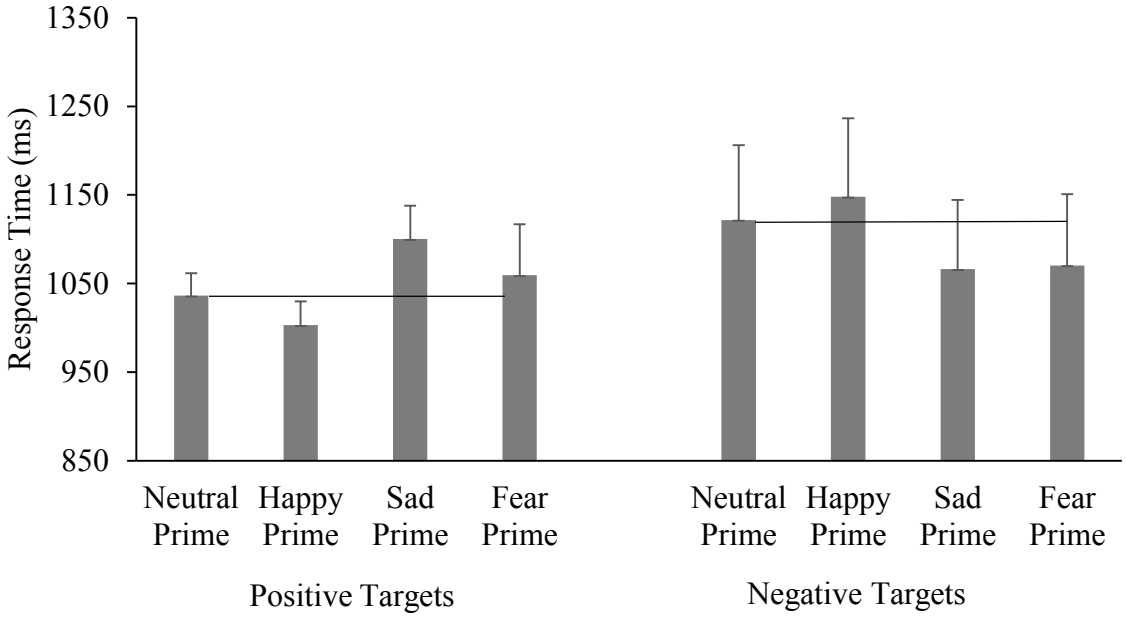

Figure 24. Response time by trial structure for affective priming task, illustrating creation of the grand effect indices. Error bars show 2 *SE* (correction for repeated measures designs, Cousineau (2005) and Morey (2008)).

As can be seen in Table 23, age and IQ were not related to measures of task performance therefore these variables were not included as covariates in regression analyses. Repeat analyses additionally controlling for IQ are presented in Appendix C.

Table 23

Zero-order correlations between Affective Priming task performance, intelligence and age

	1	2	3	4	5	6	7	8	9	10	11	12
1.Happy Grand Effect	---											
2.Happy Facilitation	.65***	---										
3. Happy Inhibition	-.68***	.12	---									
4. Sad Grant Effect	-.50***	-.37**	.30**	---								
5. Sad Facilitation	-.44***	-.07	.51***	.70***	---							
6. Sad Inhibition	.30**	.45***	.05	-.76***	-.06	---						
7. Fear Grand Effect	-.50***	-.31**	.35**	.60***	.57***	-.31**	---					
8. Fear Facilitation	-.48***	-.11	.51***	.53***	.69***	-.11	.80***	---				
9. Fear Inhibition	.27*	.38**	.02	-.38**	-.15	.39**	-.73***	-.17	---			
10. Errors	.08	.06	-.05	.08	.17	.05	.04	.16	.11	---		
11. Intelligence	.06	.07	-.01	-.08	-.07	.04	-.03	-.04	.01	-.06	---	
12. Age	.15	.07	-.12	-.04	-.10	-.03	-.09	-.10	.03	.08	.26*	---

Note: Errors = error rates to incongruent trials.

*$p < .05$ **$p < .01$ ***$p < .001$

6.4.4 Psychopathy analyses

6.4.4.1 Response Time data

6.4.4.1.1. Analysis by prime type

6.4.4.1.2 PCL:SV

As seen in Table 24, no significant relationships emerged from correlational analyses between the PCL:SV psychopathy scales and the grand effects of each prime type on RT. A series of regression models regressed the grand effects of each prime type on RT onto the PCL:SV Total and factor scales (see Table 24) adjusting for psychotropic medication. No significant results emerged from these planned analyses, therefore exploratory analyses examining facilitation and inhibition of RT were not conducted in relation to the PCL:SV.

6.4.4.1.3 TriPM

Results of correlational analyses between the grand effects of each prime type on RT and the TriPM scales produced no significant associations (see Table 25). When regressing the grand effects of each prime type on RT onto the TriPM scales while adjusting for psychotropic medication (see Table 25), TriPM Meanness predicted greater impact of happy primes on RT, and a smaller effect of fear primes on RT. The magnitude of the beta weights suggested that the unique variance of TriPM Meanness underpinned this effect. The unique variance of TriPM Meanness predicted a smaller effect of sad primes on RT at a trend level (p = .07). The unique variance of TriPM Meanness accounted for 10%

of the variance in the grand effect of fear primes on RT explained by the model. No other effects in these planned analyses reached significance.

Exploratory analyses were conducted in order to assess facilitation and inhibition of RT by happy and fear primes in relation to TriPM Meanness. Facilitation and inhibition values were calculated on a within subjects basis using the values of facilitation and inhibition which formed the grand effect value, as above. As presented in Table 25, the association between grand effect of happy primes on RT and Meanness appeared to be driven by a smaller inhibition effect in relation to the unique variance of TriPM Meanness. The complete and unique variance of TriPM Meanness predicted significantly reduced facilitation of RT by fear primes relative to neutral primes.

6.4.4.2. Error data

Trials on which targets were incorrectly evaluated but not identified as outliers were converted into percentages. Errors rates were as follows: congruent $M = 3.16$, $SD = 6.04$; neutral $M = 3.46$, $SD = 4.60$; incongruent $M = 7.86$; $SD = 8.67$. Errors to incongruent trials were of interest as an index of modulation of responses. Given the floor effects, the error data was square root transformed to correct for skew. Analysis of error data should take into account the possibility of a speed-accuracy trade off, which is indicated by a negative correlation between RT on congruent trials and number of errors on incongruent trials (Howland et al., 1993). In the present sample, RT on congruent trials and errors committed on incongruent trials were not significantly associated ($r(76) = -.14$, $p > .05$). Analyses

including congruent RT as a covariate (not presented here for brevity) did not alter the pattern of results.

6.4.4.2.1 PCL:SV

Correlational analyses (see Table 24) indicated that the PCL:SV scales were not associated with error rates. Using regression analyses and adjusting for the effects of psychotropic medication, neither PCL:SV Total, Factor 1 or Factor 2 predicted error rates on affectively incongruent trials (see Table 24).

6.4.4.2.2 TriPM

Errors committed on incongruent trials were not significantly correlated with the TriPM scales; nor were any significant effects found when adjusting for psychotropic medication (see Table 25).

6.4.5. Covariates

Analyses additionally controlling for intelligence are presented in Appendix C; a similar pattern of significant results in relation to happy and fear primes was obtained in relation to TriPM Meanness. In an additional result to the main analysis, the unique variance of Meanness significantly predicted reduced effect of sad primes (see section 6.7.3 for a further discussion); again however this result did not survive the breakdown into facilitation and inhibition effects.

Table 24

Relations between PCL:SV factors and indices of affective priming: grand effects on response times and errors committed on incongruent trials.

	PCL:SV Total		Factor 1				Factor 2			
Criterion Variable	*r*	β	*r*	β alone	β with F2	Unique sr^2	*r*	β alone	β with F1	Unique sr^2
Grand Effects										
Happy	.10	.13	.02	.06	.03	.00	.12	.13	.12	.01
Sad	-.08	-.07	-.04	-.04	-.02	.00	-.08	-.07	-.07	.01
Fear	-.01	.02	.07	.08	.10	.01	-.08	-.06	-.08	.01
Incongruent Errors										
	.07	.09	.00	.03	.02	.00	.08	.09	.09	.01

Note: PCL:SV= Psychopathy Checklist: Screening Version; F1 = Factor 1; F2 = Factor 2.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

Table 25

Relations between TriPM factors and indices of affective priming: grand effects and effects of facilitation and inhibition on response times, and errors committed on incongruent trials.

	Boldness				Meanness				Disinhibition			
Criterion Variable	*r*	β alone	β with D and M	Unique sr^2	*r*	β alone	β with D and B	Unique sr^2	*r*	β alone	β with M and B	Unique sr^2
Grand Effects												
Happy	-.07	-.06	-.15	.02	.20	.25*	.36*	.06*	.12	.14	-.12	.01
Sad	-.09	-.10	-.01	.00	-.18	-.20	-.32	.04	-.05	-.04	.17	.01
Fear	.13	.09	.22	.04	-.18	-.25*	-.45**	.10**	-.08	-.09	.23	.02
Facilitation												
Happy					.19	.17	.11	.01				
Fear					-.18	-.27*	-.41**	.08**				
Inhibition												
Happy					-.10	-.17	-.38*	.07*				
Fear					.08	.09	.25	.03				
Incongruent Errors												
	-.02	-.00	.02	.00	.09	.09	-.02	.00	.12	.14	.15	.01

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

*$p < .05$ **$p < .01$

6.5 Semantic Priming task: Method

6.5.1 Participants

Semantic priming data was not included from 11 of 94 participants, leaving a test sample of *n* = 83. Of the 11 excluded participants, data from 9 participants were removed due to having less than 66% correct on average (average percentage correct for these 11 participants *M* = 53.70%, Range 33.33 – 64.58% correct). Data were not collected from a further two participants due to participant refusal. Nineteen offenders in this test sample were receiving a form of psychotropic medication. Results here control for psychotropic medication. Descriptive details of the impact of individual medications on task performance are presented in Appendix D.

6.5.2 Materials, Design and Procedure

Full details for the prime stimuli and target stimuli used in the Semantic Priming task are provided in section 2.2.4 and section 2.4.4.1.2 respectively. Presentation hardware and software are detailed in section 3.3.1. Details of the IQ measure are found in section 3.3.4.1 and psychopathy measures are described in 3.3.3. Specifics of the Semantic Priming task design are provided in section 2.4.4.1.3. Details of the experimental session procedure are in section 3.6 and expansion of methods of analysis are provided in section 3.4. In brief, participants viewed affective (happy, sad, fearful) prime images, made up of complex scenes containing both animals and people. On the majority of trials, the image was replaced by a target word which was either related to an animal or a person. Participants were required to categorise the target word as an animal or person word. On a minority of

trials, only the prime image was presented which the participant was required to categorise as containing an animal or person.

6.6 Semantic Priming task: Results

6.6.1 Data cleaning

RT were processed as in the Affective Priming task; 5.24% of trials were removed as outliers.

6.6.2 Prime evaluation data

Prime evaluation scores were calculated as in the Affective Priming task. The mean score was well above chance (M = .77, SD = .22) which a one sample t-test indicated was significantly different from zero, $t(82) = 32.07$, $p < .001$, $d_z = 3.54$, indicating that participants were processing the prime stimuli.

6.6.3 Response Time data

Figure 25 illustrates the overall pattern of RT. In order to check the effectiveness of the design, a 2x2 factorial repeated measures ANOVA (Semantic category: congruent, incongruent; Affective category: congruent, incongruent) was conducted. There was no main effect of affective category on RT ($F(1, 82) = .38$, $p > .05$) and no interaction between semantic and affective categories ($F(1, 82) = .28$, $p > .05$). There was a significant main effect of semantic category on RT: $F(1, 82) = 39.26$, $p <$

.001, $\eta_G^2 = .10$. A planned paired samples *t*-test indicated a significant difference between semantically congruent and semantically incongruent trials: $t(82) = 6.05$, $p < .001$, $d_z = 0.67$.

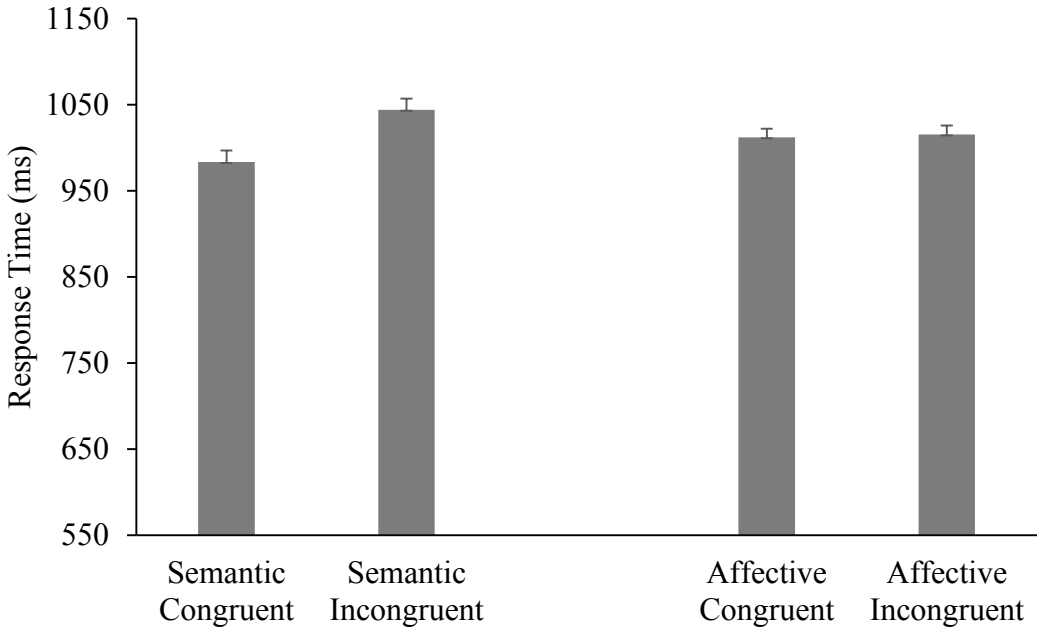

Figure 25. Main effect of semantic category in the Semantic Priming task. Error bars show 2 *SE* (correction for repeated measures designs, Cousineau (2005) and Morey (2008)).

In order to check for the consistency of this pattern of semantic priming across prime categories, a 2 (Prime: Animal, Person) x 2 (Target: Animal, Person) factorial repeated measures ANOVA was conducted. There was no main effect of Prime ($F(1, 82) = 1.13$, $p > .05$), a main effect of Target ($F(1, 82) = 34.71$, $p < .001$, $\eta_G^2 = .08$) and there was a significant Prime by Target interaction: $F(1, 82) = 25.87$, $p < .001$, $\eta_G^2 = .04$. Analysis of this interaction through planned paired *t*-tests indicated semantic priming by animal primes ($t(82) = 6.81$, $p < .001$, $d_z = 0.75$); RT to animal targets

preceded by animal primes was significantly faster than RT to animal targets preceded by person primes. There was no semantic priming for person targets preceded by person primes as compared to animal primes ($t(82) = .37$, $p > .05$).

A semantic priming effect score (RT for semantically incongruent trials – RT for semantically congruent trials) was calculated to reflect the magnitude of the semantic priming effect. A one-sample t-test indicated that this semantic priming effect was significantly different from zero: $t(82) = 6.05$, $p < .001$, $d_z = 0.66$.

Table 26

Zero-order correlations between semantic priming task performance, intelligence and age

	1	2	3	4
1. Semantic effect score	---			
2. Errors	.54***	---		
3. IQ	-.06	-.00	---	
4. Age	-.05	.22	.27*	---

Note: Errors = log-transformed error rates to semantically incongruent trials.

*$p < .05$ ***$p < .001$

As can be seen in Table 26, age and IQ were not related to measures of task performance therefore these variables were not included as covariates in regression analyses. There was a strong positive relationship between the semantic priming effect score and errors committed on semantically incongruent trials, suggesting that participants who were slower to respond were also committing more errors.

6.6.4 Psychopathy analyses

6.6.4.1 Response Time data

6.6.4.1.1 PCL:SV

Results of correlational analyses indicated that levels of the total psychopathy factor (PCL:SV Total) and Factor 2 were positively associated with the magnitude of the semantic priming effect (see Table 27). Regression analyses indicated that these relationships were reduced to trend level (p = .06 and p = .07, respectively; complete Factor 2 scale) when adjusting for presence of psychotropic medication.

6.6.4.1.2 TriPM

As seen in Table 28, the TriPM scales were not significantly associated with the magnitude of semantic priming either through correlational or regression analyses.

6.6.4.2 Error data

Trials on which targets were incorrectly evaluated but not identified as outliers were converted into percentages. Error rates were as follows: Semantically congruent trials M = 3.01, SD = 8.52; Semantically incongruent trials M = 9.34, SD = 8.52; Affectively congruent trials M = 5.90, SD = 4.88; Affectively incongruent trials M = 6.45, SD = 4.88. Error rates to semantically incongruent trials were log transformed. As in the Affective Priming task, RT on congruent trials and errors committed on

incongruent trials were not significantly associated ($r(83) = .11, p > .05$). Analyses including congruent RT as a covariate (not presented here for brevity) did not alter the pattern of results.

6.6.4.2.1 PCL:SV

Contrary to hypotheses, PCL:SV Total was significantly associated with a greater number of errors committed on semantically incongruent trials (see Table 27). This result was maintained ($p = .05$) in regression analyses adjusting for presence of psychotropic medication. Levels of Factor 1 were not significantly associated with error rates; levels of Factor 2 were correlated with error rates at a trend level ($p = .06$) when not adjusting for presence of psychotropic medication. When adjusting for medication, neither Factor 1 nor Factor 2 significantly predicted errors to incongruent trials.

6.6.4.2.2 TriPM

As seen in Table 28, the TriPM scales were not significantly associated with number of errors committed on semantically incongruent trials.

6.6.5 Covariates

Repeat analyses additionally controlling for IQ are presented in Appendix D. In contrast to the main analysis, no significant results were found between errors and PCL:SV Total when controlling for both IQ and psychotropic medication.

Table 27

Relations between PCL:SV factors and indices of semantic priming: grand effect and errors committed on semantically incongruent trials.

	PCL:SV Total		Factor 1				Factor 2			
Criterion Variable	*r*	β	*r*	β alone	β with F2	Unique sr^2	*r*	β alone	β with F1	Unique sr^2
Grand Effect										
	.22*	.22	.10	.13	.08	.01	.24*	.21	.19	.03
Incongruent Errors										
	.23*	.22*	.15	.16	.12	.01	.21	.18	.17	.03

Note: PCL:SV= Psychopathy Checklist: Screening Version; F1 = Factor 1; F2 = Factor 2.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

*$p < .05$

Table 28

Relations between TriPM factors and indices of semantic priming: grand effect and errors committed on semantically incongruent trials.

	Boldness				Meanness				Disinhibition			
Criterion Variable	*r*	β alone	β with D and M	Unique sr^2	*r*	β alone	β with D and B	Unique sr^2	*r*	β alone	β with M and B	Unique sr^2
Grand Effect												
	.03	.06	.06	.00	.13	.12	.06	.00	.14	.14	.12	.01
Incongruent Errors												
	.01	.03	.02	.00	.02	.03	.05	.00	-.01	-.01	-.04	.00

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

6.7 Discussion

Affective and semantic priming tasks assessed the impact of affective cues on the behavioural component of emotion in relation to levels of *primary* and *secondary psychopathic traits.* Within the Affective Priming task, both inhibition and facilitation of RT by prime valence was found. The results represented an improvement as compared to the undergraduate sample (see section 2.4.3.2.2.2) where only a weak effect of inhibition was found. Only a main effect of semantic priming was found in the second task, indicating the utility of the Voss et al. (2013) design for separating affective and semantic priming. The results are in line with those found in the undergraduate sample (see section 2.4.4.2.2) and consistent with existing literature (Carroll and Young, 2005, Experiment 2; Avero and Calvo, 2006, Experiment 6; Voss et al., 2013). The semantic task also produced a robust semantic priming effect; primarily driven by animal primes. This suggested that certain semantic categories may operate more effectively than others (see also Blair et al., 2006).

In relation to *primary psychopathic traits,* the pattern of results was broadly supportive of the *response modulation hypothesis*: both affective and semantic priming were intact in relation to levels of Factor 1 and TriPM Boldness during these directed attention paradigms. For *secondary psychopathic traits* (Factor 2 and TriPM Disinhibition), results indicated typical, not enhanced, task performance in the presence of directed attention for this psychopathy dimension. Two results were found which do not adhere to this pattern: TriPM Meanness significantly predicted reduced inhibitory effects of happy primes relative to neutral primes, and significantly predicted reduced facilitation effects of fear primes relative to neutral primes. The general psychopathy factor, PCL:SV Total,

predicted greater errors committed on semantically incongruent trials. These results are now discussed in turn.

6.7.1 Affective Priming task

6.7.1.1 Primary psychopathic traits

When directing attention towards affective cues, individuals high on PCL:SV Factor 1 and TriPM Boldness displayed normal responses to affective information. By contrast, an affective priming study which did not require explicitly attending to the prime (Blair et al., 2006; see also lexical decision tasks indicating impaired affective processing in relation to psychopathy: Lorenz and Newman, 2002; Reidy et al., 2008; Williamson et al., 1991) found reduced affective priming in relation to PCL:R Factor 1. In typical priming paradigms, the valence of the prime is irrelevant to the task goal of categorising the target, yet performance (RT) on the task can be facilitated or inhibited by this irrelevant information. Results of Blair et al. (2006) suggested that the dominant response – categorising the valence of the target – was not influenced by secondary or contextual information, the valence of the prime, in line with the *response modulation hypothesis*. In the present work, when attention was explicitly directed toward this secondary information, performance on the Affective Priming task was intact in relation to Factor 1. This result is consistent with the *response modulation hypothesis*.

Previous work utilising different paradigms has found that threat deficits in relation to *primary psychopathic traits* were ameliorated when attention was explicitly directed towards affective content. In an instructed threat conditioning task, levels of psychopathy were unrelated to the startle reflex

when participants were required to attend to the threat-relevant aspect of the stimulus, i.e., the colour that predicted an electric shock (Newman, Curtin, Bertsch and Baskin-Sommers, 2010). Baskin-Sommers et al. (2011) extended this study by demonstrating that levels of psychopathy were related to atypical startle reflex only in task conditions when attention was directed towards an alternative focus prior to presentation of the threat stimulus. When the threat relevant aspect of the stimulus was goal-relevant, no psychopathy related deficits in startle reflex were found. Results such as these suggest that individuals high on psychopathy can ignore threat-related distractors that are peripheral to their central goals, implying a difficulty in processing both goal relevant and peripheral information. The relative lack of attention to secondary information may be functionally similar to a lack of interest in the rights of others or displays of distress (Zeier et al., 2009). Recent work from Baskin-Sommers and colleagues (Baskin-Sommers et al., 2015b) has identified the potential role of cognitive remediation in redirecting attention to peripheral cues in order to address this bias.

Reduced priming, by fear and happy primes, was however found in relation to TriPM Meanness. It is important to consider that these results may reflect Type I error due to the number of analyses conducted, both planned and exploratory (but see section 3.4.2.2). Perneger (1998) notes that in such instances, prior beliefs should be integrated with evidence. Hypotheses regarding TriPM Meanness were undefined as previously discussed; however the data was partially supported by existing evidence (Sellbom and Phillips, 2013). Reduced facilitation by fear primes suggested that as levels of Meanness increased, participants were less likely to respond faster to a negative target preceded by a fear prime as compared to a negative target preceded by a neutral prime. The threat image had less of an impact

of RT; which is intuitive. Reduced impact of threat (fear) stimuli in relation to Meanness may facilitate antisocial behaviour and callous interpersonal reactions.

The unique variance attributed to TriPM Meanness predicted reduced inhibition by happy primes. Responses to a negative target preceded by a happy image are typically slower compared to responses to a negative target preceded by a neutral image. This effect was reduced in relation to the unique variance of Meanness. This suggests that features unique to Meanness, perhaps a lack of social connectedness, reflect blunting of positive affect. The general pattern here suggests deficient affect. Given that these results were found in relation to the TriPM scale and not psychopathy as assessed with the PCL:SV, the particular measurement used, as well as the impact of task complexity (see Baskin-Sommers and Newman, 2014), may play a role in the emergence of affective deficits. These findings must however be interpreted with great caution and require replication. Levels of Meanness are considered to be well represented within the PCL measures (Patrick and Drislane, 2015), so it unlikely that TriPM Meanness was capturing aspects of callous-unemotional traits not fully covered by PCL:SV Factor 1, which would otherwise explain the pattern of results. This raises the question of what form of meanness the TriPM assesses. It would be more conservative to infer that deficits typically associated with levels of *primary psychopathic traits* were not found when attention was directed to secondary affective cues.

6.7.1.2 Secondary psychopathic traits

Affective priming was intact in relation to Factor 2 and TriPM Disinhibition when attention was directed towards the prime. This contrasts with previous studies which have found, in relation to PCL-R Factor 2, significantly reduced affective priming, indicating reduced sensitivity for affective stimuli when attention was not explicitly directed towards the prime (Blair et al., 2006). As noted in section 1.2.2.2, the *response modulation hypothesis* predicts a rigid, goal-directed focus of attention as a feature of the overall psychopathy construct – impaired response modulation is often found in relation to levels of both *primary* and *secondary psychopathic traits* (Baskin-Sommers et al., 2011; Zeier et al., 2012; Zeier and Newman, 2013; but see Baskin-Sommers et al., 2009; Zeier et al., 2009). The present results are consistent with the *response modulation hypothesis:* processing of secondary affective cues was apparently intact in relation to *secondary psychopathic traits* during this directed attention paradigm.

By contrast, the use of Stroop tasks in relation to the two psychopathy dimensions have suggested enhanced affective sensitivity in relation to *secondary psychopathic traits,* providing support for the *dual-process model* over the *response modulation hypothesis.* In an emotion-word Stroop task (positive, negative and neutral words presented in different colours), impulsive-antisocial behaviours were related to greater interference of RT by affective relative to neutral words (Sadeh et al., 2013); indicating that as levels of these traits increased so did the degree of distraction by the arousing properties of the affective stimuli. Levels of *secondary psychopathic traits* also positively relate to increased emotion reactivity in the context of reward (Buckholtz et al., 2010; Endres et al.,

2011; Martin and Potts, 2004), threat (Baskin-Sommers et al., 2012a), and other motivationally significant cues (i.e., drug cues, Volkow and Li, 2004), often under conditions of directed attention or anticipation. However, the present results do not reflect enhanced sensitivity under conditions of directed attention for offenders high on *secondary psychopathic traits.* Affective dysregulation in relation to this psychopathy dimension may be moderated by experimental context; further work is needed to clarify this cognitive-affective profile.

It may be relevant to note that the priming literature describes the processes underlying priming response paradigms, i.e., response selection and execution, as occurring *early*, and the processes occurring in lexical decision tasks, i.e., facilitation of target processing, as occurring at a *later* stage of selection (Voss et al., 2013; Wentura and Rothermund, 2014). By contrast, within the psychopathy literature, tasks such as the lexical decision and Stroop task are used as indicators of processes occurring during the *early* stage of attentional selection. Although it is beyond the scope of this thesis to cover this in greater detail, this potential discrepancy may go some way to explaining the variation in result as a function of task paradigm.

6.7.2 Semantic Priming task

Indices of *primary* and *secondary psychopathic traits* were not related to performance on the Semantic Priming task. When focused on the semantic content of the prime, offenders were appropriately primed by the goal-relevant content as demonstrated by their typical RT. Thus, under conditions of directed attention, offenders were capable of processing and utilizing secondary semantic

and affective information to facilitate performance on both the Semantic and Affective Priming tasks. The results from the two tasks are in line with the *response modulation hypothesis* suggesting that impaired processing of both affective and non-affective cues can be ameliorated through goal-directed attention.

In the semantic priming task of Blair et al. (2006), individuals with psychopathy showed priming for animal primes followed by animal targets: performance on the task was affected by the prime information which suggests that even in the absence of an explicit attention manipulation, secondary (non-affective cues) were appropriately attended to. In that study, neither PCL-R Factors 1 nor 2 were related to the amount of semantic priming. This result contradicts the *response modulation hypothesis* (see also Brinkley, Schmitt and Newman, 2005, Experiment 1, whereby no psychopathy related differences in RT were found when making a word/non-word discrimination to target strings) and suggests that, in line with the *amygdala dysfunction hypothesis*, use of secondary semantic cues, irrespective of directed attention, is intact in relation to psychopathy. This questions the degree to which processing of non-affective cues is in fact impaired or altered in relation to the psychopathy dimensions. Future work should ideally present both typical (attend only to the target) and manipulated (attend to both prime and target) attention, affective and semantic priming tasks, as well as other response priming paradigms, in order to further clarify the nature of the cognitive-affective profiles of *primary* and *secondary psychopathic traits.*

The general psychopathy factor (PCL:SV Total) predicted greater errors committed on semantically incongruent trials, suggesting poor suppression of the prime content. Voss et al. (2013)

have noted that in some cases the response activation that is triggered by the prime is so strong that it elicits a response before the processing of the target is terminated. In such a case, the prime determines the response before target processing has reached a decision threshold. The present result has some support in the literature: in a flanker task, where centrally presented (non-affective) target stimuli were flanked on either side by (non-affective) distractor stimuli, levels of psychopathy were associated with greater errors on incongruent trials (Zeier et al., 2012). However, in the present study the relationship between errors and PCL:SV Total was reduced to non-significant when intelligence was included as a covariate (see Appendix D). Finally, as would be expected given the overall pattern of results, there was weak to no evidence of suppression effects in relation to the PCL:SV, the TriPM, and performance on the Affective and Semantic Priming tasks.

6.7.3 Role of intelligence

As noted in section 6.4.5, when additionally adjusting for the effects of IQ in the Affective Priming task, a pattern of significant results consistent with the main analysis was obtained for TriPM Meanness (see Appendix C). When controlling for both IQ and psychotropic medication, no significant results were found between Semantic Priming task performance and measures of psychopathy (see Appendix D). This suggested that in this instance, the relationship between the general psychopathy factor and increased errors on semantically incongruent trials was explained by levels of IQ as opposed to psychopathy.

6.7.4 Conclusion

The two priming studies effectively elicited affective and semantic priming, respectively, demonstrating the utility of the paradigms. The inclusion of the Semantic Priming task allowed a consideration of competing hypotheses within the psychopathy literature. The present results indicated that, in the context of directed attention, the use of secondary affective and semantic information to influence behaviour was intact in relation to levels of *primary* and *secondary psychopathic traits*. When the task focus included both secondary and central information, typical task performance was obtained, for goal-relevant affective and semantic information. Results related to TriPM Meanness would require replication before being considered further. The present results are consistent with the *response modulation hypothesis* that specifies an attentional rigidity inherent to psychopathy which can be ameliorated in specific conditions.

Chapter 7: General Discussion

7.1 Summary

Psychopathy is a personality disorder, the boundaries and content of which lack clarity and consensus. A precise definition of the disorder remains elusive; however academic researchers and clinicians, from Cleckley and Lykken to Patrick, tend to agree on one key aspect: affective hypo-responsivity. Recent evidence suggests that this disposition may be specific to certain *primary psychopathic traits*, and that affective deficits may be specific to aversive stimuli. Deficient threat reactivity has often been assessed through facial affect categorisation paradigms. The present work aimed to improve upon this approach by creating a well matched, ecologically valid stimulus set and assessing different components of emotion in addition to categorisation. This approach allowed an examination of whether deficient threat reactivity is consistently found across the different manifestations of emotion. Moreover, by assessing psychopathy in terms of primary and secondary traits, affective deficits and dysregulation across the variants could also be examined.

Presenting image, as opposed to facial, stimuli allowed an assessment of components of emotion elicited in response to complex, affective scenes. Such stimuli contain a range of subjects reflecting interpersonal situations and realistic content. The present work therefore afforded an examination of the processing of ecologically valid, affective scenarios, which may reflect learned affective associations (see section 2.2). As noted in Chapter 2, during a lengthy period of preparatory work the stimulus set was refined until a final set of images was selected, with all images matched on

luminance and complexity of content, and all affective images matched on ratings of arousal. Furthermore, by presenting the same stimulus set in each experiment, direct comparisons could be drawn between task performances in terms of psychopathy. This is a major strength of the presented work; existing studies (see Brook et al., 2013, for a review) assessing different indices of emotion processing in relation to psychopathy have attempted to make comparisons across static facial stimuli, dynamic facial stimuli, auditory stimuli and pictorial stimuli. The use of the same stimulus set throughout the present work ensured that any differences in psychopathy-related task performance were not secondary to variations in stimulus modality.

Like the field of psychopathy research, emotion research has yet to reach a consensus on what an emotion 'is': the order and nature of the events that bring about a conscious affective experience. The present work did not adhere to any particular model of emotion, but as noted in Chapter 1, took broad support from the many theories of emotion that emphasise different components of the emotion process. A general model of emotion, perhaps leaning more on appraisal theories of emotion than others, is presented in Figure 26. Upon presentation of an affective stimulus, e.g., a pointed weapon, a dynamic process beginning with stimulus evaluation brings about the various observable (facial, vocal, motor and autonomic) and internal (subjective feeling) components of, e.g., experienced fear and a threat response. As can be seen, studies which assess psychopathic affective reactivity solely in terms of affect categorisation, i.e., the cognitive evaluation component, would fail to consider several additional components of emotion which inform this appraisal. Studies which assess only the evaluation component tell an incomplete story. Similarly, tasks which assess the autonomic

component, through e.g., measures of skin conductance reactivity, could be informed through an additional consideration of how physiological reactivity compares to behavioural expression.

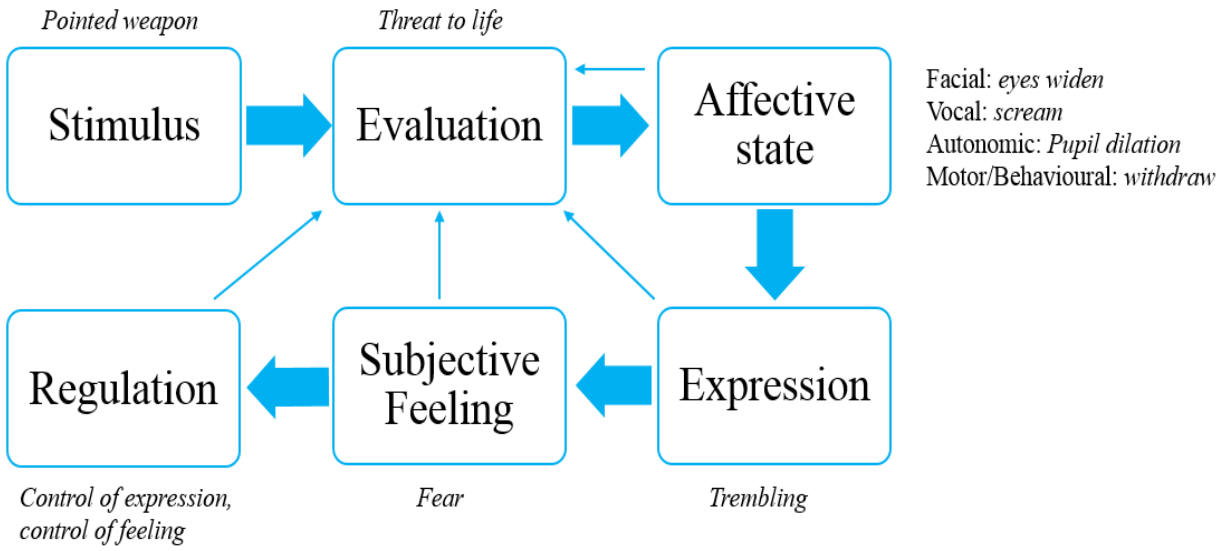

Figure 26. Broad model of components of emotion elicited in response to an affective stimulus. Major arrows indicate primary pattern of activation; minor arrows indicate feedback processes that inform subsequent activation and create affective memories.

The present work assessed psychopathy and affective processing through four components of emotion: evaluation, autonomic reactivity, behavioural response, and subjective feeling. This allowed an empirical examination of the specificity of threat evaluation deficits, emotional modulation of autonomic reactivity, use of affective cues, and the prevalence of *negative* and *positive emotionality*. The pattern of results showed that not all components of emotion were consistently related to

psychopathy, emphasising the importance of delineating the emotion process as illustrated by Figure 26. Assessing 'fear' in relation to PCL:SV Factor 1, for example, based solely on the results of the Affect Categorisation task, would have led to the conclusion that offenders with higher levels of Factor 1 present with impaired threat reactivity. This was not true for all assessed emotion components, however, notably for modulation of autonomic activity by threat stimuli. Assessment of multiple components of affect therefore allowed a more subtle pattern of psychopathic emotion processing to emerge.

7.1.1. Primary psychopathic traits

Offenders with higher levels of PCL:SV Factor 1 were less likely to categorise a threat or victim related image as frightening (Affect Categorisation task) and reported reduced experiences of fear (PANAS:X). In line with Simulation Theory, there was a parallel between deficits in threat evaluation and subjective feelings of fear in relation to Factor 1 (see section 4.5.1.1 for a consideration of the complete versus unique variance of Factor 1). This pattern is consistent with the theory that categorisation or knowledge of affect is underpinned by a low-level simulation process, using personal experiences of an emotion to understand external displays of that emotion (Goldman and Sripada, 2005). This suggests that the forming of associative links between perception and experience may be key in relation to *primary psychopathic traits.* It seems probable that impaired perceptions of threat alongside reduced representations of fear, in the presence of relevant biopsychosocial influences, impedes a compassionate response to distress in others.

Reduced sensitivity when categorising complex direct and victim-related threat images in relation to Factor 1 is a novel finding and extends existing literature which has predominantly focused on facial displays of fear as threat-signalling stimuli. The present work shows that not only do individuals high on *primary psychopathic traits* have impairments when categorising other peoples' faces as displays of fear, but that such individuals are also less likely to view stimuli representing a direct threat to themselves, or to others, as frightening.

The autonomic (Physiological Reactivity task) component of emotion appeared intact in relation to Factor 1 and TriPM Boldness. This suggests that threat deficits are context specific, not absolute. As was discussed in Chapter 5, deficits in threat-modulated autonomic activity may be specific to situations which require the learning of an associative link between the stimulus and its impact. Physiological responses to unconditioned threat stimuli, as in the present work, may be intact; autonomic deficits may be found in situations which signal the occurrence of a previously encountered stimulus that signals an aversive outcome. This suggests that it is the feedback process between the autonomic component of the affective state and the evaluation component (see Figure 26) of emotion that facilitates aversive learning, as opposed to the autonomic component in itself, which is impaired in relation to *primary psychopathic traits.*

Impact of affective information on the behavioural component of emotion (Affective Priming task) also appeared intact in relation to Factor 1 and TriPM Boldness. This result is supportive of the *response modulation hypothesis,* whereby directing attention towards secondary, affective cues alleviates impairments in affective reactivity. It may be feasible to bring about improvements in the

emotionally-modulated, behavioural component of the affective state through specific attentional interventions.

In the present work, PCL:SV Factor 1 and TriPM Boldness displayed differential relationships with (some of) the emotion components: Factor 1 was primarily related to reduced threat reactivity and fear experience, suggesting that aspects of *primary psychopathic traits* tapped by PCL:SV Factor 1 are more relevant to an absence of threat reactivity (but see Esteller et al., 2016). TriPM Boldness – perhaps specifically the unique variance - was significantly related to enhanced sensitivity for positive affect and increased experiences of positive affect, reflecting support for the inclusion of positive adjustment/resiliency features within *primary psychopathic traits,* particularly in terms of Cleckley's mask of sanity (see section 4.5.2.1). PCL:SV Factor 1 and TriPM Boldness were also only moderately correlated when common variance between Factor 1 and Factor 2 was controlled for (see section 3.3.3.3), suggesting modest overlap between these two measures (see also Lilienfeld et al., 2015a). This difference between the measures reiterates that it may not be appropriate for them to be used interchangeably as operationalisations of *primary psychopathic traits* – the best approach may be to use these measures in compliment.

7.1.2 Secondary psychopathic traits

Evidence for affective dysregulation in relation to *secondary psychopathic traits,* across any of the assessed components of emotion, was not found. Further work is needed to clarify the specific conditions under which components of emotion are intact, heightened or impaired in relation to

impulsive-antisocial behaviours. The profile for *secondary psychopathic traits* seemed best defined by atypical physiological arousal which was not affect specific.

Although it is always difficult to interpret null results, explanations have been offered throughout the thesis: affective deficits and irregularities may be context specific as opposed to global, alternative measures may be more suited to assessing emotion components particularly in relation to impulsive-antisocial behaviours. The present work may be considered a positive contribution to psychopathy research by clarifying the relative importance of certain aspects of NEM to secondary psychopathic traits. Aside from the Affective Priming task, the present work did not assess the possibility of cognitive-affective interactions as a key differential feature between the two dimensions. As discussed in section 1.2.2.3, accumulating empirical evidence suggests that *primary psychopathic traits* are best characterised by deficient attentional modulation to secondary cues while *secondary psychopathic traits* are typified by deficits in executive functioning. Assessing affective processing in the presence of attentional and anticipatory manipulations may be key.

7.2 Limitations and future directions

7.2.1. Theoretical

The present work assessed four components of emotion: the initial evaluation, behaviour, subjective report, and physiological change, based on Phillips et al. (2003; see also Lazarus, 1984). Not all theoretical components of emotion processing were assessed in the present work due to obvious time limitations. Emotion components not assessed included interoception (perception of

somatovisceral changes), expression, and emotion regulation. Within the psychopathy literature, some empirical work (e.g., Marsh, Finger, Schechter, Jurkowitz, Reid and Blair, 2011; Casey, Rogers, Burns and Yiend, 2012) has examined interoception and regulation, suggesting that further examination of these emotion components in relation to the psychopathy dimensions would be worthwhile.

Theoretical models of psychopathy, specifically in relation to *secondary psychopathic traits,* have emphasised features such as heightened anxiety and sensation seeking, and the potential role of early environmental trauma, with a large amount of empirical work supporting the link between impulsive-antisocial behaviours and anxiety in particular (see Chapter 1). The present work did not assess these variables and, as noted in section 4.5 and section 5.5, this absence necessarily limits the degree to which the pattern of results can specifically inform and support theoretical models of *secondary psychopathic traits*.

As in the present work, research on psychopathy predominantly focuses on White males therefore the extent to which the results generalise to other sample populations is unclear. Comparatively little research has assessed psychopathy in women or other ethnic groups (Skeem et al., 2011). As well as replication, future work should expand on typical methods of sampling.

7.2.2 Methodological

Two measures of psychopathy, the PCL:SV and the TriPM, were used in the present work. The PCL:SV in particular has been subject to much criticism; several researchers (Cooke et al., 2006; Skeem et al., 2011; Patrick, 2006; see Hare and Neumann, 2008, for a response) have judged the PCL

measures to overly index deviancy and maladjustment at the expense of positive adjustment features, given that the original PCL items were extrapolated through statistical analysis from exclusively criminal samples. Thus the PCL:SV may not reflect the classic description of psychopathy (e.g., Cleckley, 1955) which ought to be borne in mind as a limitation of this measure.

The TriPM, through its inclusion of the Boldness scale, was designed to operationalise positive adjustment features as described by Cleckley (1955). Although TriPM Boldness appeared to have an advantage over PCL:SV Factor 1 in indexing these traits, it is unclear why TriPM Boldness did not predict reduced threat sensitivity on the Affect Categorisation task, if bolder individuals require a higher threshold for activation of the threat system (Patrick et al., 2009; see also Lykken, 1995). The utility of TriPM Boldness as an index of empirically-derived low-fear, as compared to social potency, may need further assessment. In the Physiological reactivity task, the pattern of autonomic activity predicted by TriPM Meanness and TriPM Disinhibition was consistent, which suggests these scales together usefully predict *secondary psychopathic traits,* but this lack of specificity also brings into question their discriminant validity: the degree to which the TriPM scales index distinct constructs (see Evans and Tully, 2016).

It is also important to consider the inherent limitation of self-report measures: whether offenders would endorse questionnaire items assessing, in particular, meanness, and whether this had any impact on the restricted results. Indeed, TriPM Meanness was the only psychopathy measure to exhibit positive skew, indicating a floor effect in the scale. Aside from the requirement of insight when completing self-report measures (see Cleckley, 1955; Lilienfeld and Fowler, 2006), the risk of

deception is inherently problematic when assessing psychopathic traits amongst offender samples. The TriPM is limited in this regard, in that it does not contain a validity scale to assess consistency of responding (see, e.g., the PPI-R). Both Miller, Jones and Lynam (2011) and Ray, Hall, Rivera-Hudson, Pothress, Lilienfeld and Morano (2013) have suggested that individuals high on psychopathic traits may accurately report their symptoms in the absence of direct consequences or incentives of distorted responding; this suggests that self-report measures of psychopathy are not necessarily inevitably biased. Nevertheless, caution is required when using self-report measures. As noted, the most appropriate approach may be to use the TriPM in compliment with PCL measures.

As discussed in Chapter 4, existing work utilising facial affect stimuli has elicited conflicting results in the psychopathy literature. The present work aimed to move away from this by presenting affective and neutral images matched on various dimensions; nevertheless, no set of affective, or indeed neutral, stimuli can be considered 'perfect'. By including two types of negative stimuli, the stimulus set was necessarily unbalanced in terms of valenced content. The potential confound remained that affective images tended to contain people but neutral images tended to feature non-human content. The overall pattern of results in the Affect Categorisation task suggested that, despite attempts to match stimuli on complexity, neutral and happy images may have been more easily categorised due to simplicity of content. The pattern of results in the Physiological reactivity task suggested additional variance in psychophysiological response not accounted for by the dimensions of valence or arousal (see section 2.4.2.1.2.2). These issues are relevant to all empirical work which utilise images in emotion processing paradigms; however these issues may be particularly pertinent for

stimulus categories that intend to separate affect into discrete states as was the aim of the present work. In light of these considerations, future work may benefit from a more conservative categorisation of stimuli into appetitive and threat/distress categories.

Offender participants were not asked to provide ratings of stimulus valence and arousal. Previous work (Patrick et al., 1993; Verona et al., 2001; but see Pham and Phillippot, 2010) has established that levels of psychopathy are not related to subjective ratings of stimuli, whether visual or auditory, but are related to atypical autonomic responses to the same stimuli. This has been interpreted (Brook et al., 2013) as a dissociation between psychophysiological response and self-report ratings. The inclusion of such subjective ratings in the present work would have allowed a further assessment of Cleckley's (1955) mask of sanity and would be of interest in future work.

As noted in section 4.3.2.1, a short version of the PANAS-X questionnaire was utilised, presenting only items required for the four scales. Use of the complete PANAS-X scale would have additionally provided broad PEM and NEM scales, as well as scales more specifically assessing self-assurance and shame which may have been interesting in relation to *primary* and *secondary psychopathic traits* respectively. Furthermore, as noted in section 4.5.2.2, an additional measure assessing different aspects of PEM and NEM would have been valuable. The Multidimensional Personality Questionnaire: Brief Form (MPQ:BF; Patrick, Curtin and Tellegen, 2002) represents a viable measure in this respect: the MPQ:BF provides scores on stress reaction, alienation, aggression and harm avoidance. Future work could additionally include this measure.

The present work utilised the WASI as a measure of intelligence; this measure was the lab standard at the time of the projects beginning. A revised version of the WASI, the WASI II (Wechsler, 2011), was more recently designed with increased user-friendliness and better psychometric properties. To compare the relevant sections of the WASI II and the WASI: twenty items in the WASI II Vocabulary subtest were retained from the WASI; eight new items were added. The picture items were retained but the images were updated. The scoring criteria for the retained starting items was also refined to facilitate testing time. Twenty-three items from the WASI II Matrix Reasoning subtest were retained from the WASI with 7 new items added. This updated content is suggested to provide better floor and ceiling effects as well as closer links with other measures of intelligence; updated norms are also provided. As discussed in section 3.3.4.1, the WASI is a well-validated measure of intelligence; ideally however the most up to date versions of assessments should be used.

7.2.3 Statistical

The present work utilised multiple regression methods of analysis which partialled out common variance between independent variables. There was, however, little evidence for suppression effects over the body of research (with the exception of sensitivity when categorising sad images in relation to TriPM Meanness and Disinhibition). Although results indicated for the PCL:SV the utility of specifically assessing the factors as opposed to the total psychopathy score, there was little evidence that the unique variance of each factor, rather than the complete factor, was a better predictor of the emotion components. The common variance between *primary* and *secondary psychopathic traits* may have important explanatory value in characterising the relations between psychopathy and particular

external correlates. There is some support in the literature for this (see, e.g., Baskin-Sommers et al., 2015a); the present results do however contrast with previous work that revealed suppressor effects between the psychopathy dimensions (Blonigen et al., 2010; Hicks and Patrick, 2006; Verona et al., 2005). These studies had notably larger sample sizes than the present work (Blonigen et al., 2010: 1701 participants; Hicks and Patrick, 2006: 241 participants; Verona et al., 2005: 226 participants); such statistical power, beyond the scope of the present work, may be required in order to reveal the more subtle suppression effects between the psychopathy dimensions.

Lynam et al. (2006; see also Hare and Neumann, 2008) have cautioned that a suppression approach risks losing sight of the original construct under investigation. Statistical approaches utilising zero-order correlations may be more useful for clinicians interested in external correlates of, e.g., factor scales, while the regression approach may provide greater information to scientific researchers. Statistical preferences are also likely to differ depending on the theoretical approach taken: if a multidimensional disorder is suggested, methods of regression analysis may be preferred. Future work could extend the regression approach by assessing whether different indices of each psychopathy dimension, e.g., PCL:SV Factor 1 and TriPM Boldness, PCL:SV Factor 2 and TriPM Disinhibition, have incremental validity over each other in predicting criterion variables. Clarifying the understanding of the interaction between measure, dimension and functioning will aid understanding of psychopathy.

7.3 Conclusion

The present work assessed several components of emotion in relation to psychopathy. This approach afforded an examination of whether deficient affective reactivity generalises across the psychopathy dimensions, is consistent across the different manifestations of emotion, and whether affective deficits are in fact threat specific. The work was novel in its use of a well matched, ecologically valid stimulus set across all experimental paradigms and a new method of assessing psychophysiology in this population: pupillometry. Assessing emotion processing in relation to the psychopathy dimensions revealed the specificity of threat deficits in relation to *primary psychopathic traits* and also revealed sensitivity to positive affect for particular primary traits. In contrast to hypotheses, emotionally modulated physiological reactivity was intact for offenders high on *primary psychopathic traits.* For offenders high on *secondary psychopathic traits,* all assessed components of emotion were intact. The atypical pattern of autonomic activity in relation to *secondary psychopathic traits* suggested a link between arousal and adverse outcome; linking to research which has identified low resting heart rate as a risk factor for antisocial behaviour. Assessment of multiple components of affect allowed a more subtle pattern of psychopathic emotion processing to emerge and highlighted the multifaceted nature of psychopathy.

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Appendix A: Affect Categorisation task and PANAS-X

A.1. Affect Categorisation

A.1.1 PCL:SV

When additionally controlling for intelligence, the results differed from the main analysis. PCL:SV Factor 1 did not significantly predict categorisation of threat images (p = .08 for the beta value for Factor 1 on its own; see Table A1).

A.1.2 TriPM

When additionally controlling for intelligence, the unique variance of TriPM Meanness and TriPM Disinhibition no longer predicted categorisation of sad images as in the main analysis. In a change to the main analysis, the complete TriPM Disinhibition predicted greater sensitivity for categorising neutral images at a trend level (p = .06; see Table A2).

A.2 Emotion Experience

A.2.1 PCL:SV

When additionally controlling for intelligence, the results did not differ from the main analysis (see Table A1).

A.2.2 TriPM

When additionally controlling for intelligence, the results in relation to TriPM Boldness did not differ from the main analysis. In a change to the main analysis, the complete TriPM Meanness scale predicted reduced experience of fear at a trend level (p = .07; see Table A2).

Table A1

Relations between PCL:SV factors, affect categorisation and self-reported emotion experience: Additionally controlling for IQ

	PCL:SV Total	Factor 1			Factor 2		
Criterion Variable	β	β alone	β with F2	Unique sr^2	β alone	β with F1	Unique sr^2
Categorisation[a]							
Neutral	.04	-.01	-.03	.00	.07	.08	.01
Happy	-.03	.03	.07	.00	-.08	-.11	.01
Sad	-.07	-.08	-.07	.00	-.06	-.03	.00
Fear	-.15	-.16	-.15	.01	-.09	-.04	.00
Experience[b]							
Guilt	-.27*	-.31**	-.30**	.08**	-.13	-.03	.00
Joviality	.04	.11	.14	.01	-.07	-.11	.01
Fear	-.23*	-.25*	-.23*	.04*	-.14	-.07	.00
Sadness	-.22*	-.33**	-.35**	.11**	-.04	.07	.00

Note: PCL:SV= Psychopathy Checklist: Screening Version; F1 = Factor 1; F2 = Factor 2. *$p < .05$ **$p < .01$.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

[a]Adjusting for intelligence, age and medication. [b]Adjusting for intelligence and medication.

*$p < .05$ **$p < .01$

Table A2

Relations between TriPM factors, affect categorisation and self-reported emotion experience: Additionally controlling for IQ

	Boldness			Meanness			Disinhibition		
Criterion Variable	β alone	β with D and M	Unique sr^2	β alone	β with D and B	Unique sr^2	β alone	β with M and B	Unique sr^2
Categorisation[a]									
Neutral	.05	.05	.00	.16	.05	.00	.20	.17	.01
Happy	.16	.15	.01	.07	.04	.00	-.00	-.02	.00
Sad	-.01	.07	.00	-.05	-.22	.02	.09	.24	.02
Fear	-.11	-.08	.00	-.09	-.09	.00	-.02	.04	.00
Experience[b]									
Guilt	-.34**	-.29*	.07*	-.15	-.15	.01	.05	.12	.01
Joviality	.38***	.43***	.15***	-.05	-.20	.02	-.12	.05	.00
Fear	-.52***	-.49***	.18***	-.20	-.11	.01	.04	.07	.00
Sadness	-.44***	-.41***	.14***	-.05	-.07	.00	.18	.19	.02

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

[a]Adjusting for intelligence, age and medication. [b]Adjusting for intelligence and medication.

*$p < .05$ **$p < .01$ ***$p < .001$

A.3. Descriptive impact of medications on Categorisation and PANAS-X scores

A.3.1 Un-medicated participants

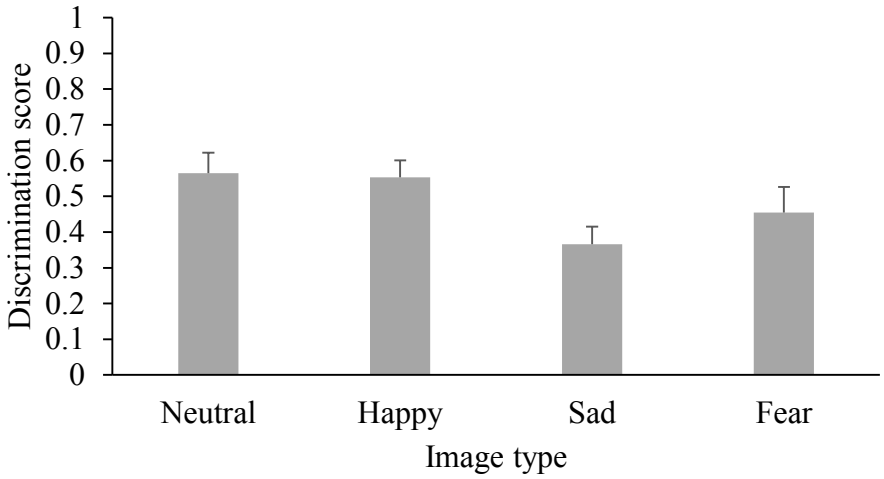

Figure A1. Mean discrimination sensitivity for un-medicated participants. Error bars show 2 *SE*.

Fifty seven participants were not receiving any form of psychotropic medication (see Figure A1). The pattern of results for the categorisation task for un-medicated offenders is similar to the main analysis, with greatest categorisation sensitivity for neutral images and least sensitivity for sad images. Average self-reported emotion experience, as assessed with the PANAS-X, for un-medicated participants was also similar to the means reported in the main analysis: Joviality: $M = 26.72$, $SD = 5.80$; Fear: $M = 12.00$, $SD = 4.96$; Sadness: $M = 12.63$, $SD = 4.84$; Guilt: $M = 16.81$, $SD = 6.59$.

A.3.2 Participants receiving SSRI medication

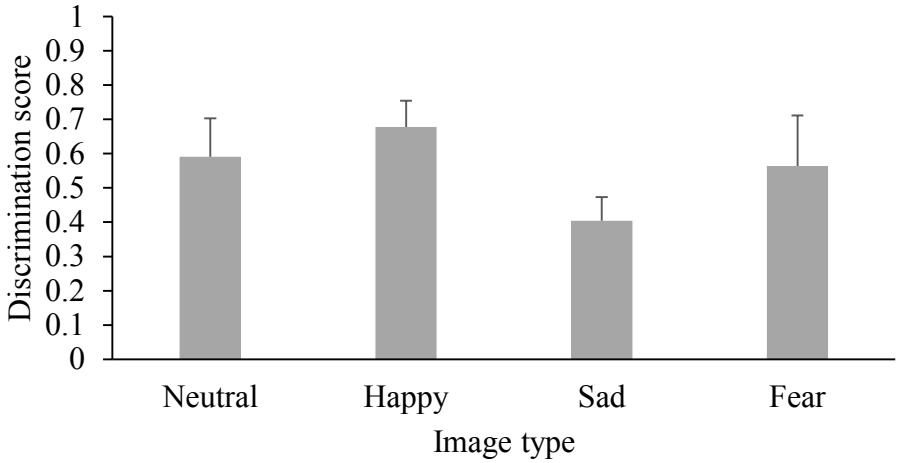

Figure A2. Mean discrimination sensitivity for participants receiving antidepressant medication, specifically, selective serotonin reuptake inhibitors. Error bars show 2 *SE*.

Ten participants were receiving Selective Serotonin Reuptake Inhibitor (SSRI) medication for relief of symptoms associated with depression (see Figure A2). SSRI increase the level of serotonin in the brain. In contrast to the main analysis, these participants showed greatest sensitivity when categorising happy images. Average self-reported emotion experience, as assessed with the PANAS-X, for participants receiving SSRI medication was slightly lower for positive affect and higher for negative affect as compared to the main analysis: Joviality: $M = 24.60$, $SD = 7.44$; Fear: $M = 15.70$, $SD = 6.41$; Sadness: $M = 14.40$, $SD = 5.72$; Guilt: $M = 20.20$, $SD = 8.28$.

A.3.3 Participants receiving AED

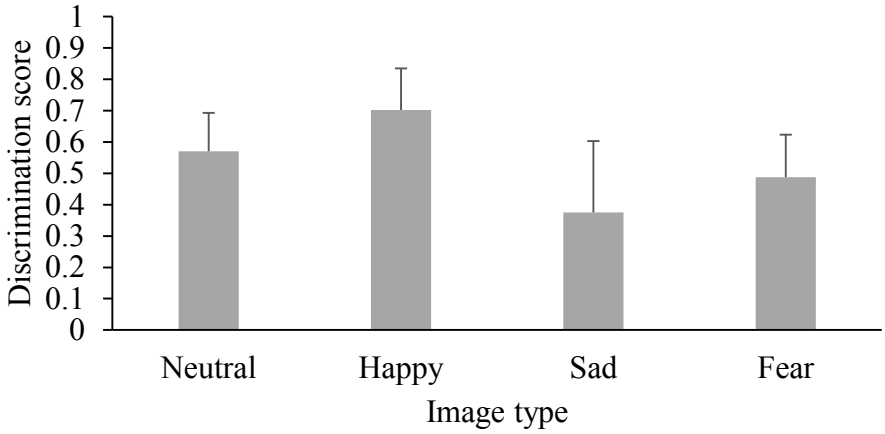

Figure A3. Mean discrimination sensitivity for participants receiving anti-epileptic drugs. Error bars show 2 *SE*.

Four participants were receiving pregabalin which is an anti-epileptic drug (AED). Pregabalin is also administered for nerve pain and for generalised anxiety disorder, and reduces the release of noradrenaline in the brain. Similar to participants in receipt of an SSRI, and in contrast to the main analysis, participants receiving pregabalin showed the greatest sensitivity when categorising happy images (see Figure A3). Average self-reported emotion experience, as assessed with the PANAS-X, for participants receiving pregabalin was higher for positive and negative affect as compared to the main analysis: Joviality: $M = 30.25$, $SD = 6.55$; Fear: $M = 15.50$, $SD = 4.80$; Sadness: $M = 14.25$, $SD = 4.57$; Guilt: $M = 18.25$, $SD = 4.86$.

A.3.4 Participants receiving NaSSA medication

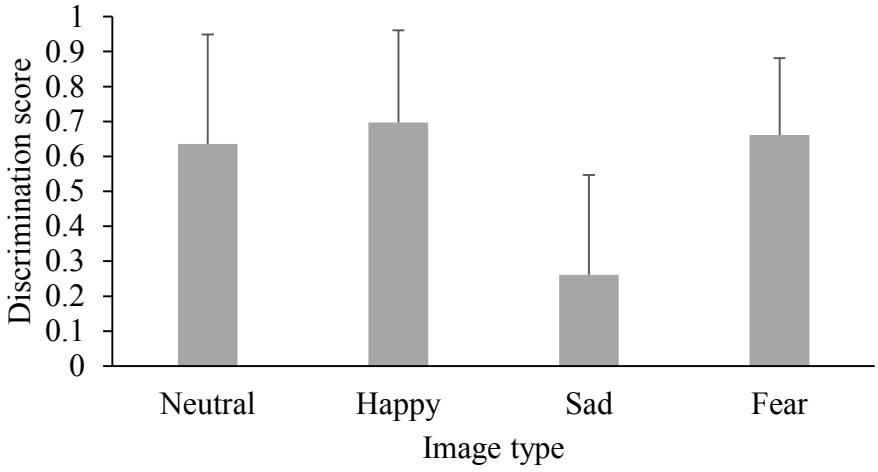

Figure A4. Mean discrimination sensitivity for participants receiving anti-depressant medication, specifically, noradrenergic and specific serotonergic antidepressants. Error bars show 2 *SE*.

Three participants were receiving Noradrenergic and Specific Serotonergic Antidepressant (NaSSA) medication for relief of symptoms associated with depression. These drugs work by increasing levels of noradrenaline and serotonin in the brain. As seen in Figure A4, discrimination sensitivity for happy and fear images was higher, and discrimination sensitivity for sad images was reduced, as compared to the main analysis. Average self-reported emotion experience, as assessed with the PANAS-X, for participants receiving NaSSA medication was lower for positive affect and higher for negative affect as compared to the main analysis: Joviality: $M = 23.33$, $SD = 1.53$; Fear: $M = 15.50$, $SD = 5.13$; Sadness: $M = 14.25$, $SD = 4.04$; Guilt: $M = 18.25$, $SD = 10.07$.

A.3.5 Participants receiving benzodiazepine medication

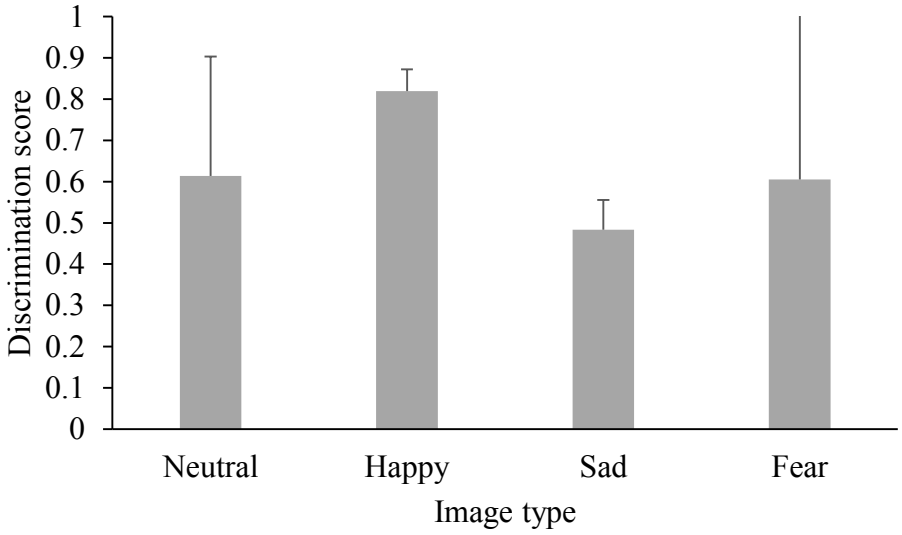

Figure A5. Mean discrimination sensitivity for participants receiving benzodiazepine medication. Error bars show 2 *SE*.

Three participants were receiving anti-anxiety medications from the benzodiazepine family (e.g., Lorazepam, Diazepam, Valium). These drugs have a calming, sedative influence on brain functions and can often mute the experience of negative affect. Participants receiving a benzodiazepine showed greater sensitivity for happy images, but good overall sensitivity for the other classes of images (see Figure A5). Average self-reported emotion experience, as assessed with the PANAS-X, for participants receiving benzodiazepine medication was higher for positive affect and guilt and lower for self-reported experiences of fear as compared to the main analysis. Levels of self-reported sadness was similar to the main analysis: Joviality: M = 31.00, SD = 4.00; Fear: M = 10.33, SD = 2.08; Sadness: M = 12.00, SD = 4.58; Guilt: M = 19.00, SD = 10.44.

A.3.6 Participants receiving medication for PTSD

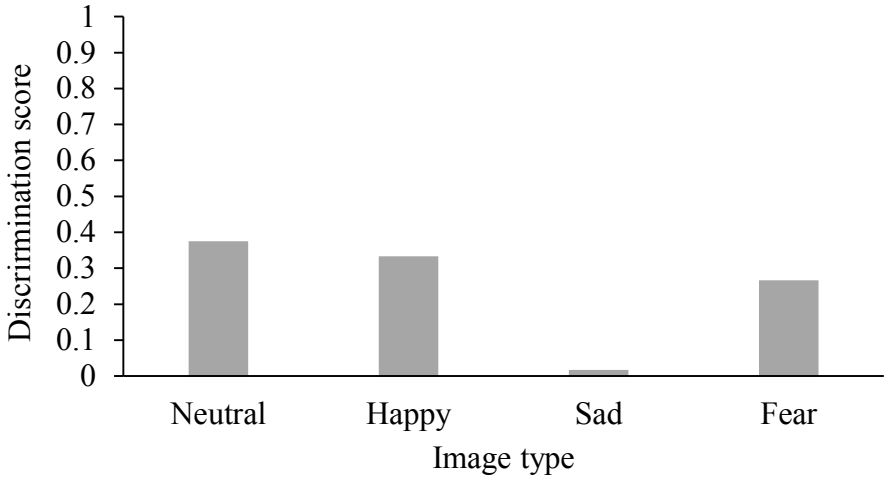

Figure A6. Mean discrimination sensitivity for a participant receiving medication for the symptoms of post-traumatic stress disorder.

One participant was receiving unspecified medication for relief of symptoms associated with Post-Traumatic Stress Disorder (PTSD). This participant displayed sharply reduced sensitivity for sad images (see Figure A6). Results of the PANAS-X indicated that the participant reported a similar level of positive affect and increased levels of negative affect as compared to the main analysis: Joviality: $M = 26.00$; Fear: $M = 19.00$; Sadness: $M = 15.00$; Guilt: $M = 23.00$.

A.3.7 Participants receiving anti-psychotic medication

Figure A7. Mean discrimination sensitivity for participants receiving anti-psychotic medication. Error bars show 2 *SE*.

Four participants were taking anti-psychotic medication; drugs of this sort typically have a sedating effect. As seen in Figure A7, these participants displayed reduced sensitivity when categorising sad images as compared to the main analysis. Average self-reported emotion experience, as assessed with the PANAS-X, for participants receiving anti-psychotic medication was slightly lower for positive affect, higher for self-reported sadness and similar for fear and guilt as compared to the main analysis: Joviality: $M = 24.00$, $SD = 8.76$; Fear: $M = 10.75$, $SD = 2.63$; Sadness: $M = 16.00$, $SD = 6.38$; Guilt: $M = 16.75$, $SD = 6.50$.

A.3.8 Participants receiving ADHD medication.

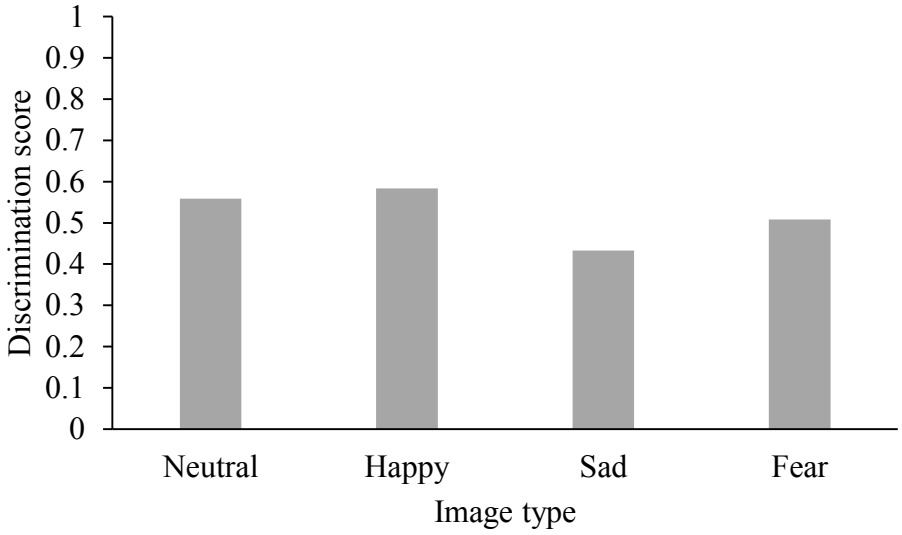

Figure A8. Mean discrimination sensitivity for a participant receiving medication for the symptoms of attention deficit hyperactivity disorder.

One participant was receiving stimulant medication (methylphenidate; trade name Ritalin) for symptoms associated with Attention Deficit Hyperactivity Disorder (ADHD) and, as presented in Figure A8, displayed a categorisation pattern similar to the main analysis. Through the PANAS-X, this participant reported higher levels of positive affect and fear, but lower levels of sadness and guilt as compared to the main analysis: Joviality: $M = 37.00$; Fear: $M = 19.00$; Sadness: $M = 7.00$; Guilt: $M = 13.00$.

Appendix B: Physiological reactivity

B.1 Initial pupil diameter

B.1.1. PCL:SV

Results are presented in Table B1. In a change to the main analysis, the unique variance of PCL:SV Factor 2 marginally predicted (p = .06) lower levels of IPD when adjusting for intelligence and psychotropic medication.

B.1.2. TriPM

In line with the main analysis, none of the TriPM scales significantly predicted IPD (see Table B2).

B.2 Initial constriction response

B.2.1. PCL:SV

When controlling for both IQ and presence of psychotropic medication, the results were in line with the main analysis (see Table B1).

B.2.2. TriPM

The pattern of results for TriPM Disinhibition adhered to the main analysis (see Table B2). When controlling for both IQ and presence of psychotropic medication, TriPM Meanness did not significantly predict the ICR elicited by neutral images. This result is in contrast to the main analysis. In line with the main analysis, TriPM Meanness was significantly associated with an attenuated ICR elicited in response to affective images. In results contrasting with the

main analysis, the unique variance of TriPM Boldness predicted an attenuated ICR elicited by happy images and marginally significantly (p = .05) predicted an attenuated ICR elicited by sad images. The complete Boldness scale marginally predicted an attenuated ICR elicited by happy images (p = .06).

B.3. Emotional modulation

B.3.1 PCL:SV

In line with the main analysis, when controlling for individual differences in IQ and presence of psychotropic medication, the psychopathy dimensions were not related to emotional modulation of pupil dilation (see Table B1).

B.3.2. TriPM

In line with the main analysis, all relationships with the TriPM as predictor variables failed to reach significance (see Table B2 below).

Table B1

Relations between PCL:SV factors and pupil activity: Additionally controlling for IQ: initial pupil diameter, initial constriction response and emotionally modulated pupil dilation

	PCL:SV Total		Factor 1			Factor 2	
Criterion Variable	β	β alone	β with F2	Unique sr^2	β alone	β with F1	Unique sr^2
IPD	-.10	.05	.12	.02	-.20	-.24	.05
ICR							
Neutral	.19	.10	.02	.00	.24*	.23*	.04*
Happy	.17	.05	-.03	.00	.24*	.25*	.05*
Sad	.17	.05	-.03	.00	.24*	.25*	.05*
Fear	.17	.04	-.05	.00	.26*	.28*	.06*
Emotional Modulation							
Happy	-.06	-.07	-.06	.00	-.04	-.02	.00
Sad	-.01	.06	.09	.01	-.08	-.11	.01
Fear	-.01	.00	.00	.00	-.00	-.00	.00

Note: Total = Psychopathy Checklist: Screening Version Total score; F1 = Factor 1; F2 = Factor 2. IPD = initial pupil diameter, ICR = initial constriction response.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

*$p < .05$

Table B2

Relations between TriPM factors and pupil activity: Additionally controlling for IQ: initial pupil diameter, initial constriction response and emotionally modulated pupil dilation

	Boldness			Meanness			Disinhibition		
Criterion Variable	β alone	β with D and M	Unique sr^2	β alone	β with D and B	Unique sr^2	β alone	β with M and B	Unique sr^2
IPD	.05	.06	.00	-.08	-.06	.00	-.09	-.04	.00
ICR									
Neutral	.12	.14	.02	.19	.02	.00	.22*	.23	.03
Happy	.19	.23*	.04*	.21*	-.02	.00	.24*	.28*	.04*
Sad	.18	.21	.03	.24*	.02	.00	.26*	.27*	.04*
Fear	.14	.16	.02	.26*	.05	.00	.29**	.29*	.04*
Emotional Modulation									
Happy	.15	.19	.03	-.04	-.13	.01	-.04	.07	.00
Sad	.03	.07	.00	-.08	-.13	.01	-.04	.05	.00
Fear	-.04	-.02	.00	.04	-.02	.00	.09	.10	.01

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness. IPD = initial pupil diameter, ICR = initial constriction response.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

*$p < .05$ **$p < .01$

B.4. Descriptive impact of medications on Physiological Reactivity

B.4.1 Un-medicated participants

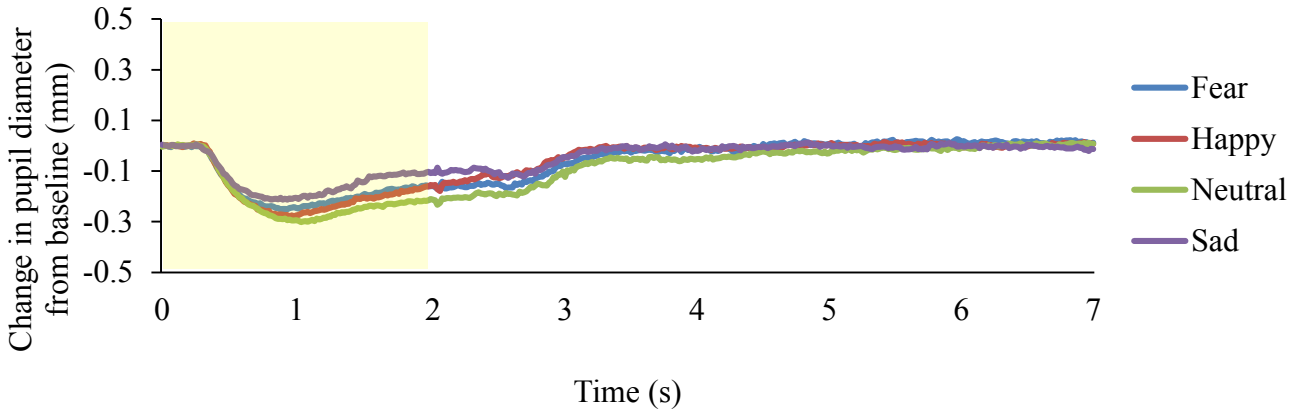

Figure B1. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset for un-medicated participants when viewing fearful, happy, sad and neutral images. The shaded area represents the interval of image presentation.

Participants not receiving any form of medication (n = 47, see Figure B1) displayed a similar pattern of pupil activity to the main analysis. The greatest attenuation of the ICR was elicited by sad images, with all affective images eliciting an attenuated constriction and greater dilation as compared to the neutral images.

B.4.2 Participants receiving SSRI medication

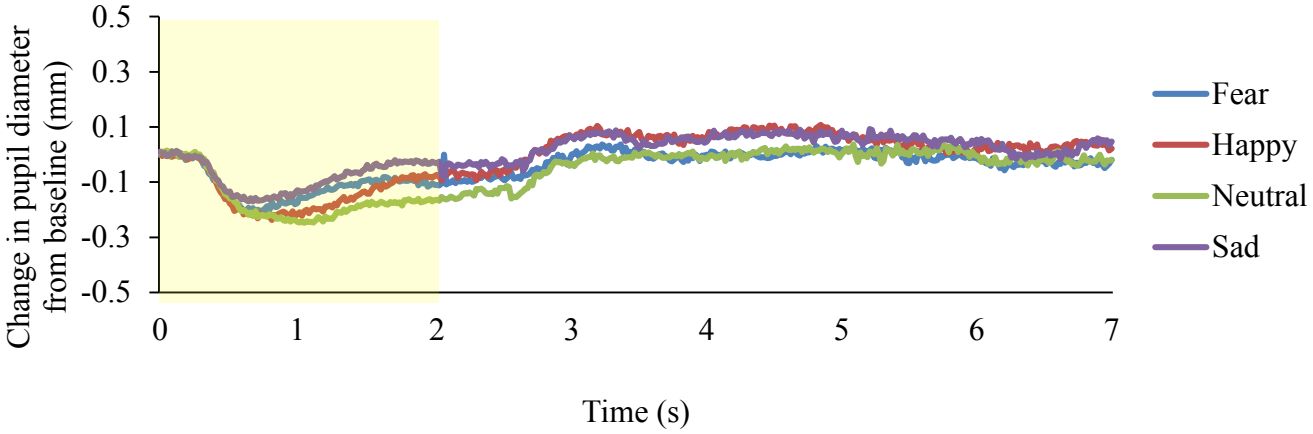

Figure B2. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset for participants receiving an SSRI when viewing fearful, happy, sad and neutral images. The shaded area represents the interval of image presentation.

Nine participants were receiving antidepressant medication in the form of an SSRI, which increases the levels of serotonin in the brain by inhibiting its reuptake into the presynaptic cell. This could lead to miosis (constriction). For the ICR, these participants showed a slightly attenuated pupil pattern compared to the main analysis. Following image offset, participants receiving SSRI medication showed a reduced dilation to fear stimuli as compared to the main analysis (see Figure B2).

B.4.3 Participants receiving AED

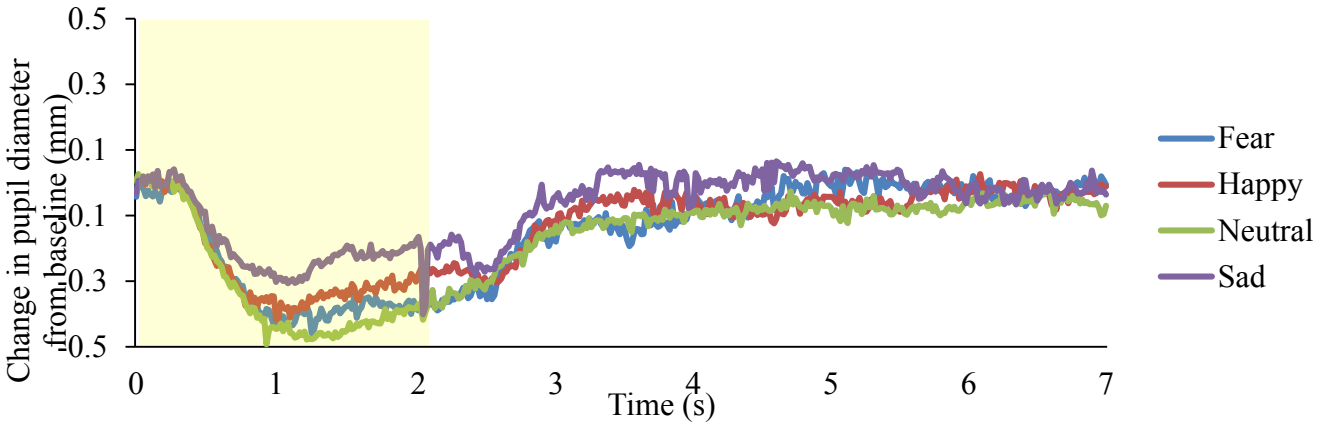

Figure B3. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset for participants receiving pregabalin when viewing fearful, happy, sad and neutral images. The shaded area represents the interval of image presentation.

Four participants were receiving pregabalin which is an anti-epileptic drug (AED). Pregabalin is also administered for nerve pain and for generalised anxiety disorder, and decreases the release of noradrenaline (NA) in the brain which could lead to miosis. The pattern of pupil activity for participants receiving pregabalin (see Figure B3) was similar to that of participants receiving NaSSA, with an attenuated ICR to sad images and a typical constriction response to the other classes of images. Dilation in the period following image onset was greatest for sad images.

B.4.4 Participants receiving NaSSA medication

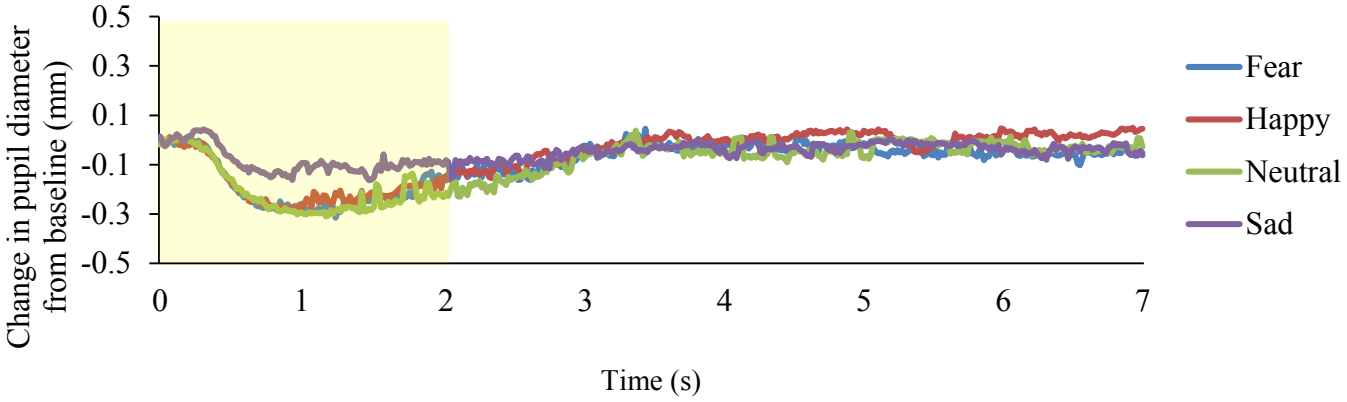

Figure B4. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset for participants receiving an NaSSA when viewing fearful, happy, sad and neutral images. The shaded area represents the interval of image presentation.

Three participants were receiving NaSSA (see Figure B4) which work by increasing levels of NA and serotonin, which could produce mydriasis (dilation) and miosis, respectively. These participants showed a sharply attenuated ICR to sad images and a comparable and typical ICR to the remaining images relative to the main analysis. All classes of images elicited a comparable amount of dilation in the period following image offset, with the possibility of happy images eliciting a slightly stronger dilation response.

B.4.5 Participants receiving benzodiazepine medication

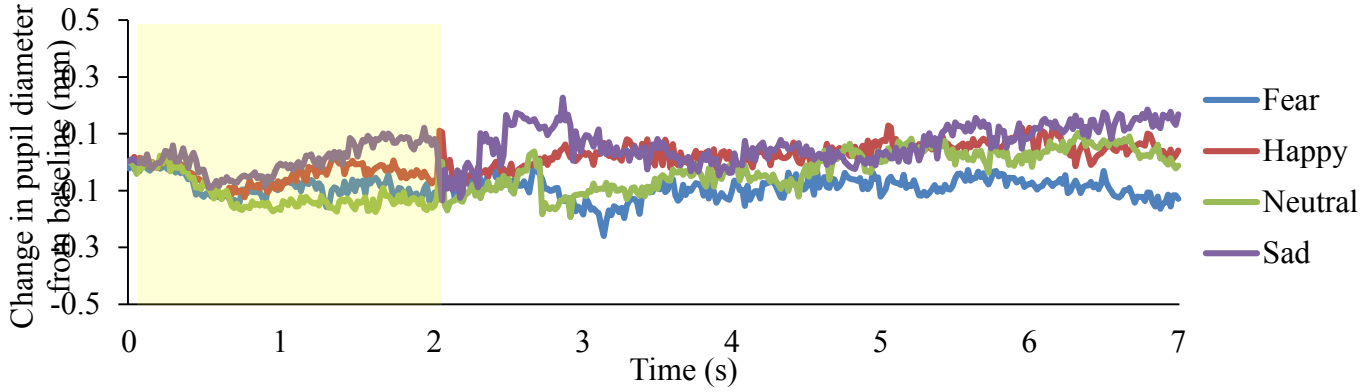

Figure B5. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset for participants receiving benzodiazepines when viewing fearful, happy, sad and neutral images. The shaded area represents the interval of image presentation.

Two participants were receiving benzodiazepines (e.g., Lorazepam, Diazepam); sedatives typically prescribed for the treatment of anxiety and panic disorders that often mute dampen sympathetic effects and cause miosis. These participants, in comparison to the main analysis, displayed an attenuated ICR to all images, although the largest ICR was to the neutral images. In the period following image offset, the dilation response was largest for sad and happy images, with the smallest dilation response for fearful images (see Figure B5).

B.4.6 Participants receiving medication for PTSD

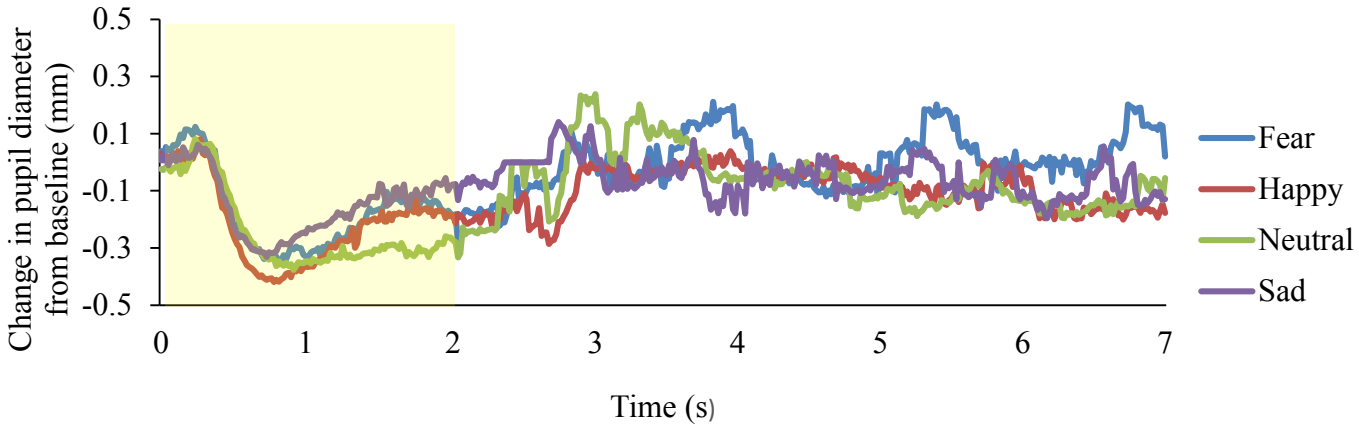

Figure B6. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset for a participant receiving medication for symptoms of PTSD when viewing fearful, happy, sad and neutral images. The shaded area represents the interval of image presentation.

One participant was receiving medication (unspecified) for relief of symptoms associated with PTSD (see Figure B6). This participant exhibited a typical ICR to all classes of images. The dilation of the pupil following the ICR appeared slightly erratic with dilation peaking at approximately 3 s post image onset and then an unstable pupil response over the remainder of the trial window, particularly in response to fearful images. Individuals with PTSD show greater dilation to threat as compared to neutral stimuli (Cascardi, Armstrong, Chung and Pare, 2015).

B.4.7 Participants receiving anti-psychotic medication

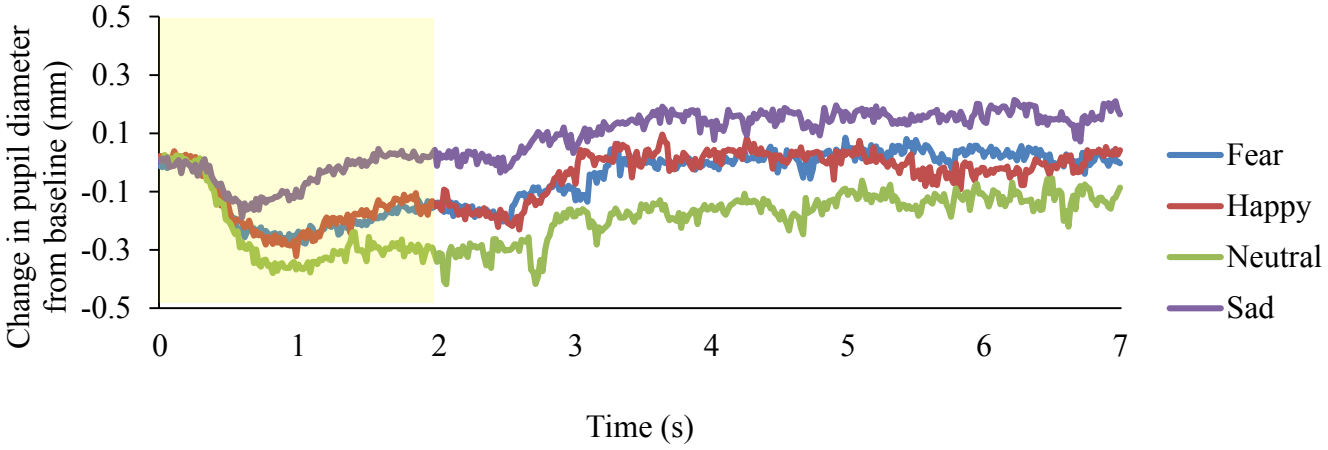

Figure B7. Pupil activity from stimulus onset to end of the recovery screen for a participant receiving anti-psychotic medication. The shaded area represents the interval of stimulus presentation.

One participant was in receipt of medication (atypical antipsychotic quetiapine; trade name Seroquel) for symptoms associated with psychosis. This drug has a sedating effect which would be expected to cause miosis. Although the ICR elicited by sad images was quite attenuated, the overall pattern was comparable to the main analysis with all affective images producing attenuated constriction and greater dilation as compared to the neutral images (see Figure B7).

B.4.8 Participants receiving ADHD medication

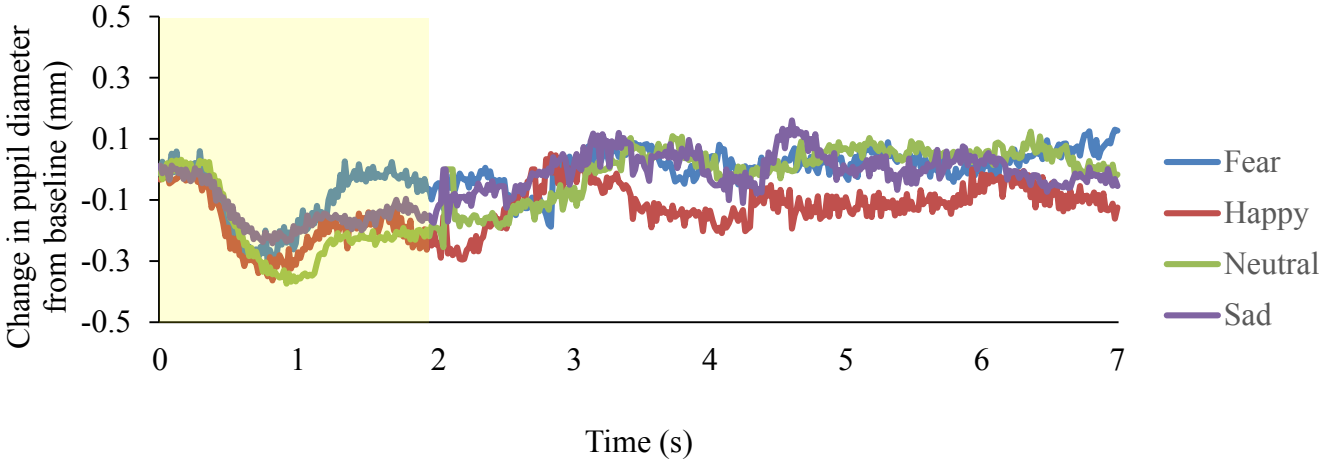

Figure B8. Change (mm) in pupil diameter from a 200 ms baseline preceding stimulus onset for a participant receiving medication for symptoms of ADHD when viewing fearful, sad, happy and neutral images. The shaded area represents the interval of image presentation.

One participant reported being in receipt of stimulant medication (methylphenidate; trade name Ritalin) for symptoms associated with ADHD. Stimulants often dilate the pupil by blocking re-uptake of NA causing mydriasis. The participant display a typical ICR to all classes of image, and a sharp dilation to fearful images relative to the main analysis. Dilation in the recovery period was lowest in response to happy images (see Figure B8).

Appendix C: Affective Priming task

C.1 Affective Priming effects

C.1.1 PCL:SV

When controlling for both IQ and psychotropic medication (see Table C1), the results for psychopathy as assessed with the PCL:SV adhered to the main analysis.

C.1.2 TriPM

When controlling for both IQ and medication, a similar pattern emerged as to that presented in the main analysis (see Table C2). In a change to the main analysis, the unique variance of Meanness significantly predicted reduced effect of sad primes (complete TriPM Meanness scale $p = .05$); there was also a trend ($p = .06$) for the unique variance of TriPM Boldness to predict a greater effect of fear primes on RT. At a trend level ($p = .07$) the unique variance of Meanness predicted reduced facilitation of RT by sad primes, greater facilitation of RT by happy primes and greater inhibition of RT by fear primes.

C.2 Error data

C.2.1 PCL:SV

As in the main analysis, the PCL:SV factors were not significantly associated with errors committed to incongruent trials (see Table C1).

C.2.2 TriPM

As in the main analysis, psychopathy as assessed with the TriPM was not associated with error rates (see Table C2).

Table C1

Relations between PCL:SV factors and indices of affective priming: Additionally controlling for IQ: grand effects on response times and errors committed on incongruent trials.

	PCL:SV Total	Factor 1			Factor 2		
Criterion Variable	β	β alone	β with F2	Unique sr^2	β alone	β with F1	Unique sr^2
Grand Effects							
Happy	.12	.04	.01	.00	.15	.12	.01
Sad	-.05	-.02	.00	.00	-.10	-.07	.00
Fear	.03	.11	.13	.01	-.06	-.10	.01
Incongruent Errors							
	.09	.05	.03	.00	.08	.08	.01

Note: PCL:SV= Psychopathy Checklist: Screening Version; F1 = Factor 1; F2 = Factor 2.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

Table C2

Relations between TriPM factors and indices of affective priming: Additionally controlling for IQ: grand effects and effects of facilitation and inhibition on response times, and errors committed on incongruent trials.

	Boldness			Meanness			Disinhibition		
Criterion Variable	β alone	β with D and M	Unique sr^2	β alone	β with D and B	Unique sr^2	β alone	β with M and B	Unique sr^2
Grand Effects									
Happy	-.06	-.17	.02	.27*	.40*	.07*	.15	-.12	.01
Sad	-.09	.02	.00	-.24	-.36*	.06*	-.05	.18	.02
Fear	.09	.24	.04	-.27*	-.50**	.11**	-.10	.25	.03
Facilitation									
Happy				.22	.16	.01			
Sad				-.19	-.33	.05			
Fear				-.29*	-.44*	.09*			
Inhibition									
Happy				-.16	-.40*	.07*			
Sad				.15	.18	.01			
Fear				.12	.30	.04			
Incongruent Errors									
	.01	.03	.00	.08	-.02	.00	.13	.15	.01

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

*$p < .05$ **$p < .01$.

C.3. Descriptive impact of medications on Affective Priming

C.3.1 Un-medicated participants

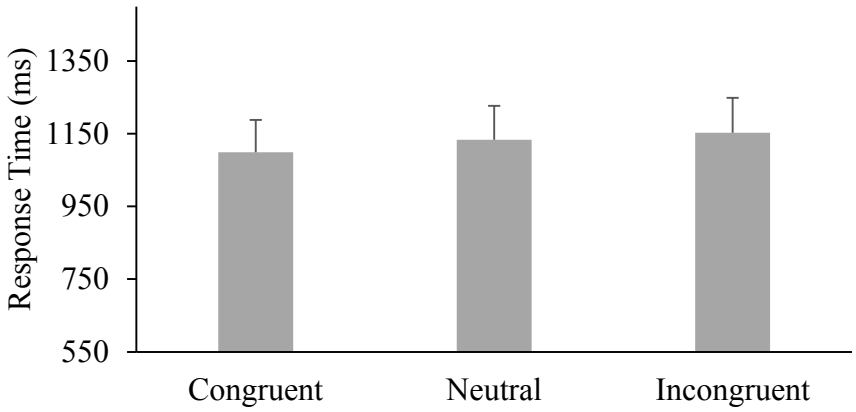

Figure C1. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for un-medicated participants. Error bars show 2 *SE*.

Fifty participants were not receiving any form of medication (see Figure C1). These participants showed a comparable RT to the main anlaysis with congruent trials faster than neutral (difference of 34 ms) and incongruent (difference of 54 ms). In contrast to the main analysis, neutral trials were 19 ms faster than incongruent trials.

C.3.2 Participants receiving SSRI medication

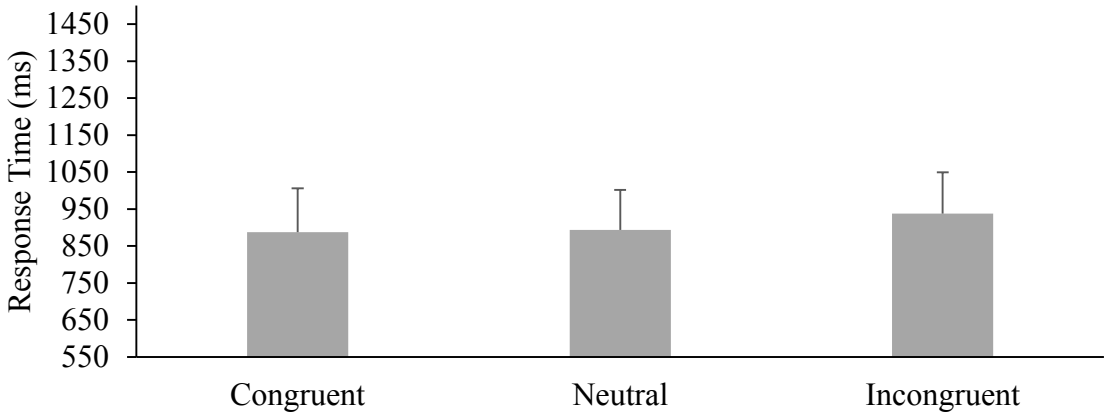

Figure C2. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for participants receiving an SSRI. Error bars show 2 *SE*.

Nine participants were receiving a Selective Serotonin Reuptake Inhibitor (SSRI) for relief of symptoms associated with depression. SSRI increase the level of serotonin in the brain. Participants receiving an SSRI displayed faster RT as compared to the main analysis (see Figure C2). Congruent trials were only 6 ms faster than neutral but were 51 ms faster than incongruent. In line with the main analysis, RT to neutral trials was 44 ms faster than to incongruent trials.

C.3.3 Participants receiving AED

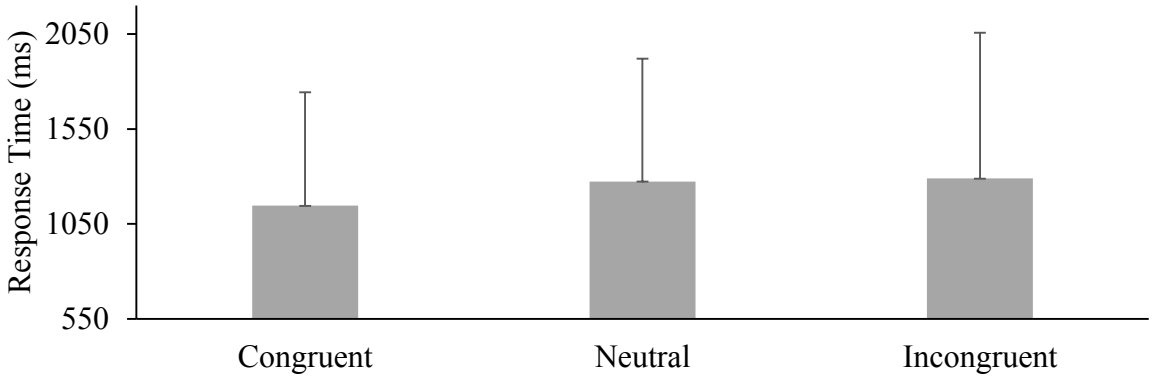

Figure C3. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for participants receiving anti-epileptic medication. Error bars show 2 *SE*.

Three participants were receiving pregabalin which is an anti-epilepsy drug (AED) also administered for pain relief and generalised anxiety disorder. As can be seen in Figure C3, these participants displayed the typical pattern of affective priming: congruent trials were 127 ms faster than neutral, congruent trials were 143 ms faster than incongruent and neutral trials were 16 ms faster than incongruent. Response latencies were comparable to the main analysis.

C.3.4 Participants receiving NaSSA medication

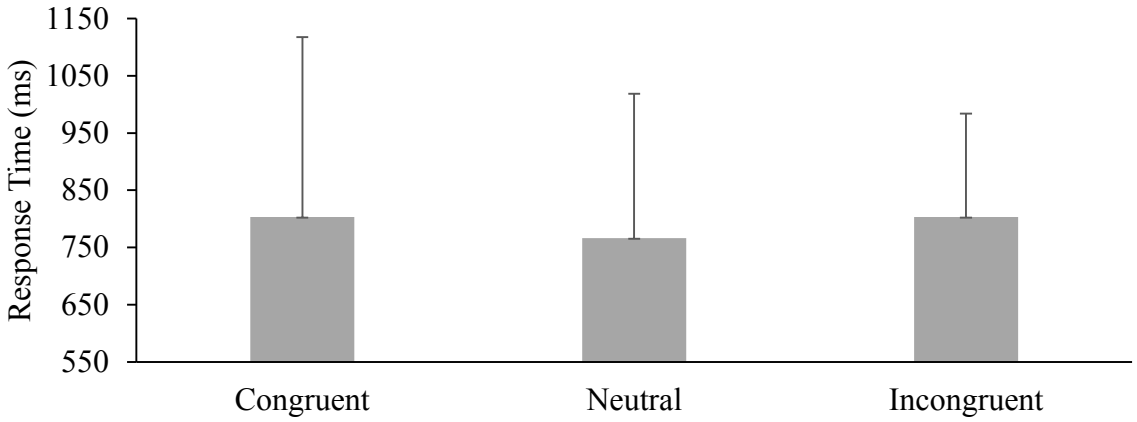

Figure C4. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for participants receiving an NaSSA. Error bars show 2 *SE*.

Two participants in the Affective Priming task were receiving this anti-depressant (Noradrenergic and Specific Serotonergic Antidepressant, NaSSA) medication. As can be seen in Figure C4, RT was much faster than in the main analysis. There was little evidence of affective priming: RT to congruent trials was 37 ms slower than RT to neutral trials, and RT to neutral trials was 37ms faster than RT to incongruent trials; there was no difference in RT between congruent and incongruent trials.

C.3.5 Participants receiving benzodiazepine medication

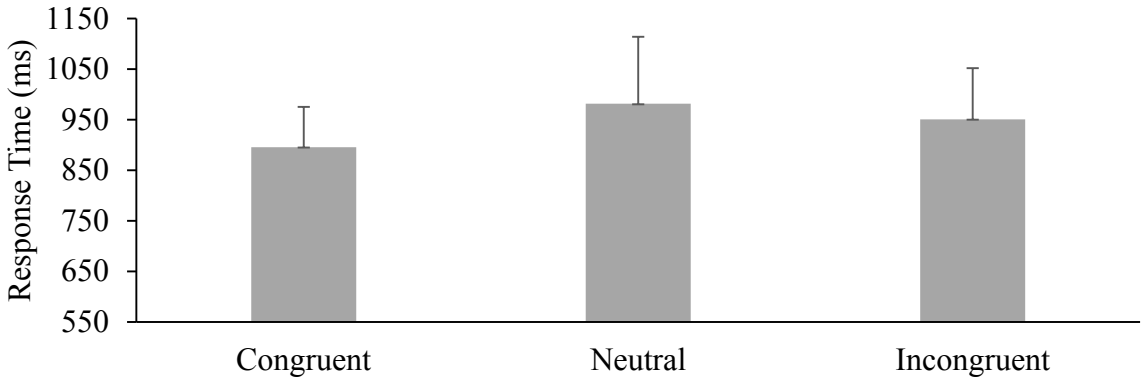

Figure C5. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for participants receiving a benzodiazepine. Error bars show 2 *SE*.

RT for the three participants receiving a Benzodiazepine was faster as compared to the main analysis (see Figure C5). Benzodiazepines have a calming, sedative effect on brain function. Despite this, a relative pattern of affective priming was found for these participants, with RT to congruent trials 86 ms faster than RT to neutral, 55 ms faster than RT to incongruent, and RT to incongruent trials 31 ms faster than RT to neutral trials.

C.3.6 Participants receiving medication for PTSD

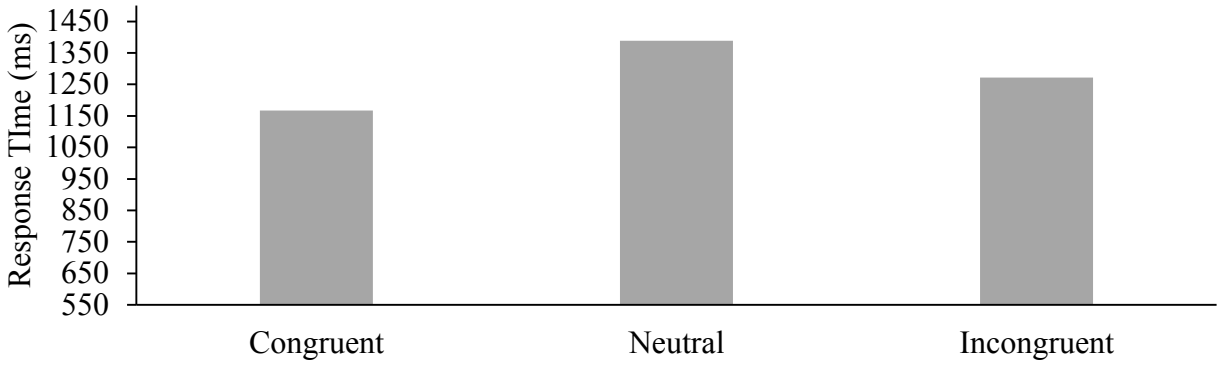

Figure C6. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for a participant receiving medication for relief of symptoms associated with PTSD.

One participant in the Affective Priming task was receiving unspecified medication for relief of symptoms associated with Post-Traumatic Stress Disorder (PTSD). This participant, whose response latencies are displayed above in Figure C6, had slightly slower RT as compared to the main analysis. Although RT to congruent trials was 221 ms faster than RT to neutral and 104 ms faster than RT to incongruent, RT to incongruent trials were 117 ms faster than RT to neutral.

C.3.7 Participants receiving anti-psychotic medication

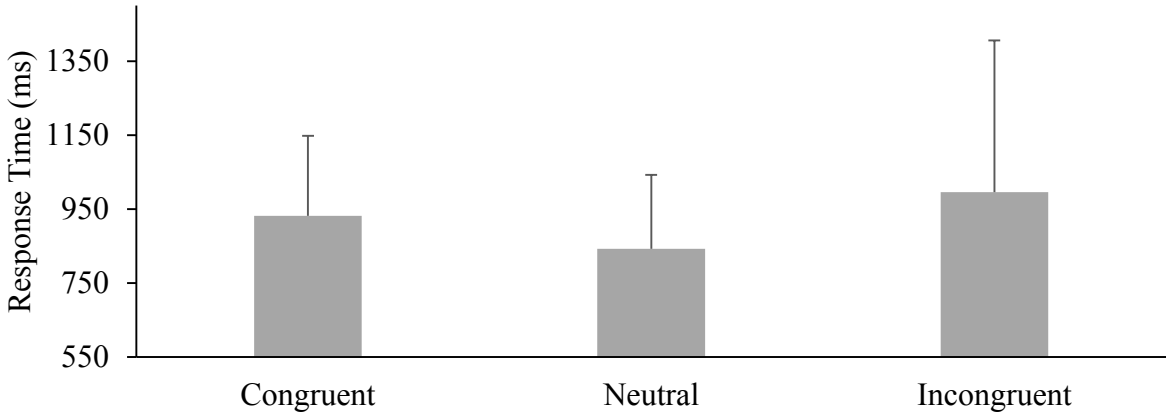

Figure C7. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for participants receiving anti-psychotic medication. Error bars show 2 *SE*.

Two participants in the Affective Priming task were receiving anti-psychotic medication (means presented in Figure C7); medication of this sort typically has a sedating effect on brain function. RT was faster as compared to the main analysis, and RT was fastest to neutral trials (89 ms faster than congruent trials and 153 ms faster than incongruent trials). RT to congruent trials was 64 ms faster than RT to incongruent trials.

C.3.8 Participants receiving ADHD medication

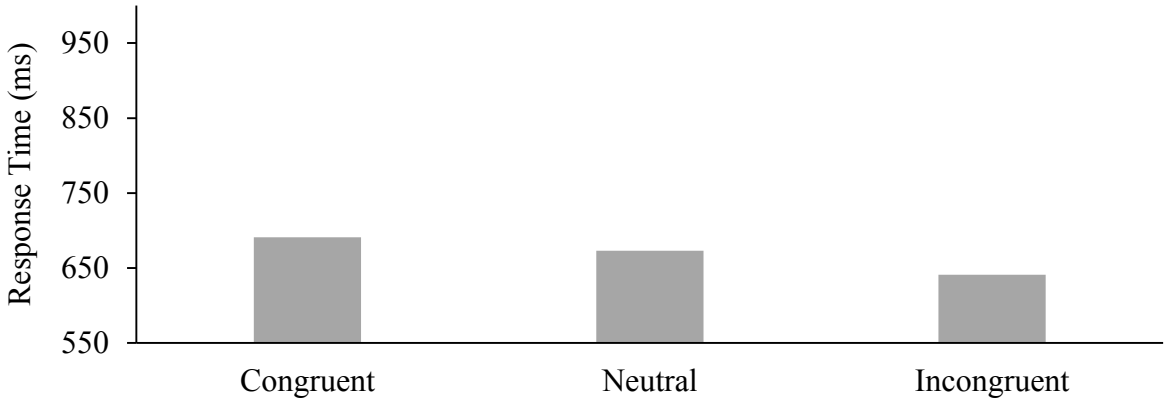

Figure C8. Mean response time, in ms, to congruent, neutral and incongruent trials in the Affective Priming task for a participant receiving medication for symptoms associated with ADHD.

One participant in the Affective Priming task was receiving stimulant medication (methylphenidate; trade name Ritalin) for symptoms associated with Attention Deficit Hyperactivity Disorder (ADHD). This participant, as can be seen in Figure C8, displayed extremely short response latencies as compared to the main analysis. The pattern of RT was not typical of affective priming: RT to incongruent trials was 32 ms than RT to neutral trials and 50 ms faster than RT to congruent trials; RT to neutral trials was 18 ms faster than RT to congruent trials.

Appendix D: Semantic Priming task

D.1 Semantic Priming effect

D.1.1 PCL:SV

In line with the main analysis, when controlling for both IQ and psychotropic medication psychopathy as assessed with the PCL:SV was not related to the magnitude of the semantic priming effect (see Table D1).

D.1.2 TriPM

In line with the main analysis, psychopathy as assessed with the TriPM was not related to the magnitude of the semantic priming effect (see Table D2).

D.2 Error data

D.2.1 PCL:SV

In contrast to the main analysis, PCL:SV Total was not related to number of errors committed on semantically incongruent trials when adjusting for both IQ and medication (see Table D1).

D.2.2. TriPM

In line with the main analysis, the TriPM scales were not associated with number of errors committed on semantically incongruent trials (see Table D2).

Table D1

Relations between PCL:SV factors and indices of semantic priming: Additionally controlling for IQ: grand effect and errors committed on semantically incongruent trials.

	PCL:SV Total	Factor 1			Factor 2		
Criterion Variable	β	β alone	β with F2	Unique sr^2	β alone	β with F1	Unique sr^2
Grand Effect							
	.14	.08	.04	.00	.15	.14	.02
Incongruent Errors							
	.13	.08	.04	.00	.12	.12	.01

Note: PCL:SV= Psychopathy Checklist: Screening Version; F1 = Factor 1; F2 = Factor 2.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

Table D2

Relations between TriPM factors and indices of semantic priming: Additionally controlling for IQ: grand effect and errors committed on semantically incongruent trials.

	Boldness			Meanness			Disinhibition		
Criterion Variable	β alone	β with D and M	Unique sr^2	β alone	β with D and B	Unique sr^2	β alone	β with M and B	Unique sr^2
Grand Effect									
	.10	.10	.01	.09	-.01	.00	.10	.11	.00
Incongruent Errors									
	.07	.09	.00	-.06	-.07	.00	-.08	-.03	.00

Note: D = TriPM Disinhibition; B = TriPM Boldness; M = TriPM Meanness.

Unique sr^2 = the unique contribution of the predictor variable to the total variance of the criterion variable, in the presence of the suppressor variable(s).

D.3. Descriptive impact of medications on Semantic Priming

D.3.1 Un-medicated participants

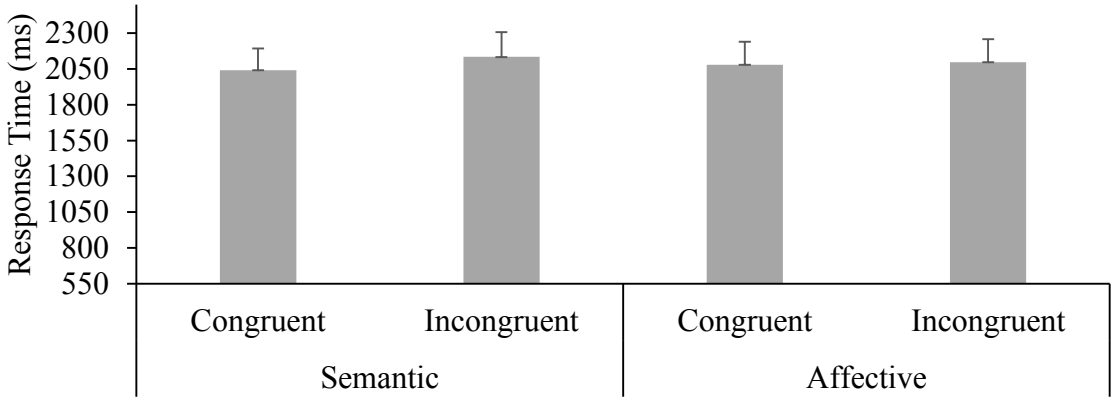

Figure D1. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for un-medicated participants. Error bars show 2 *SE*.

Figure D1 shows the mean RT for un-medicated participants (n = 52) in the Semantic Priming task. Participants showed the expected pattern with responses to semantically congruent trials 92 ms faster than those to semantically incongruent trials. Responses to affectively congruent trials were 18 ms faster than responses to affective incongruent trials. Overall, RT was slower as compared to the main analysis.

D.3.2 Participants receiving SSRI medication

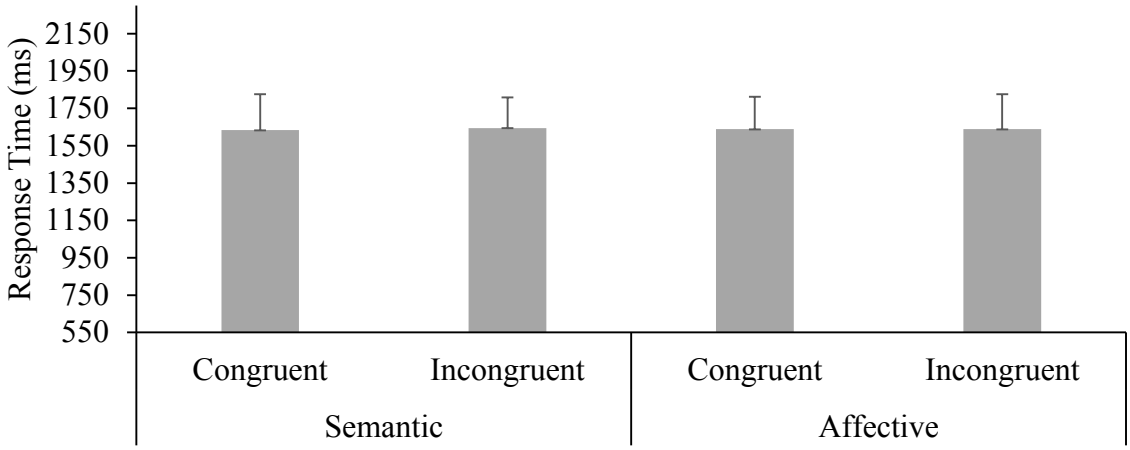

Figure D2. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for participants receiving an SSRI. Error bars show 2 *SE*.

Figure D2 shows the pattern of semantic priming RT for the nine participants receiving an SSRI. RT was overall slower as compared to the main analysis, but these participants showed the expected pattern of RT with semantically congruent trials 11 ms faster than semantically incongruent. There was no difference in RT between affectively congruent and incongruent trials.

D.3.3 Participants receiving AED

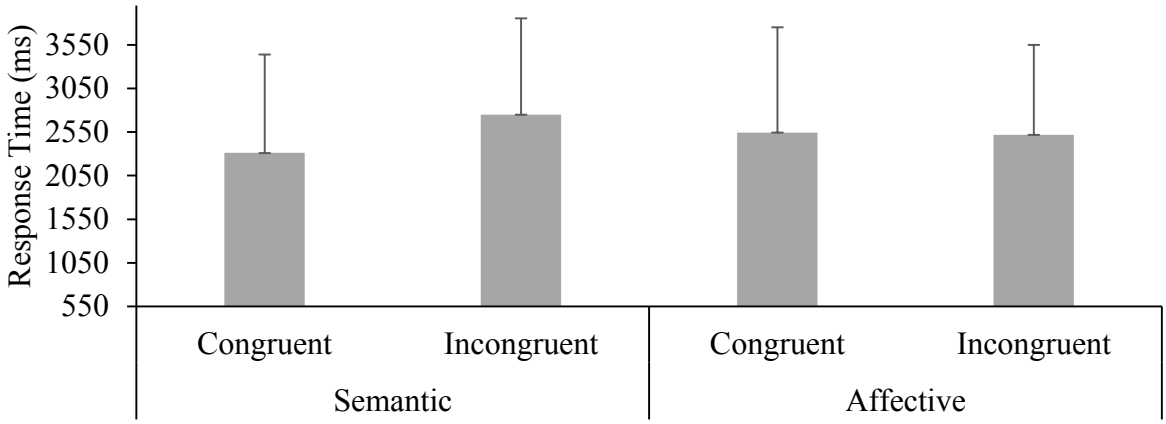

Figure D3. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for participants receiving anti-epileptic medication. Error bars show 2 *SE*.

Four participants in the Semantic Priming task were receiving pregabalin. Figure D3 presents the mean RT for these participants, with RT to semantically congruent trials 439 ms faster than semantically incongruent. RT to affectively congruent trials was 26 ms slower than to affectively incongruent trials.

D.3.4 Participants receiving NaSSA medication

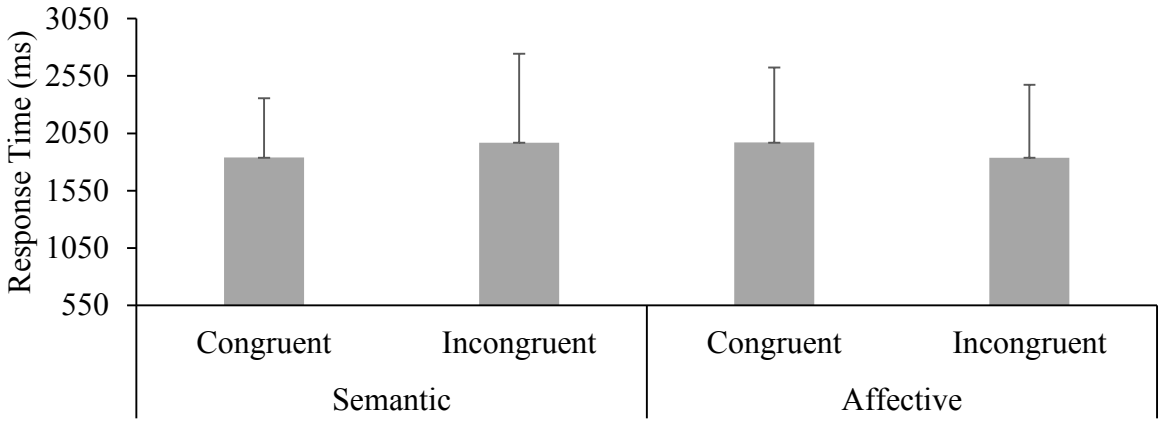

Figure D4. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for participants receiving a NaSSA. Error bars show 2 *SE*.

Three participants in the Semantic Priming task were receiving a NaSSA (mean RT presented in Figure D4). RT was slower than the means in the main analysis, but the expected pattern was found with RT to semantically congruent trials 323 ms faster than RT to semantically incongruent trials. RT to affectively congruent trials was 67 ms slower than RT to affectively incongruent trials.

D.3.5 Participants receiving benzodiazepine medication

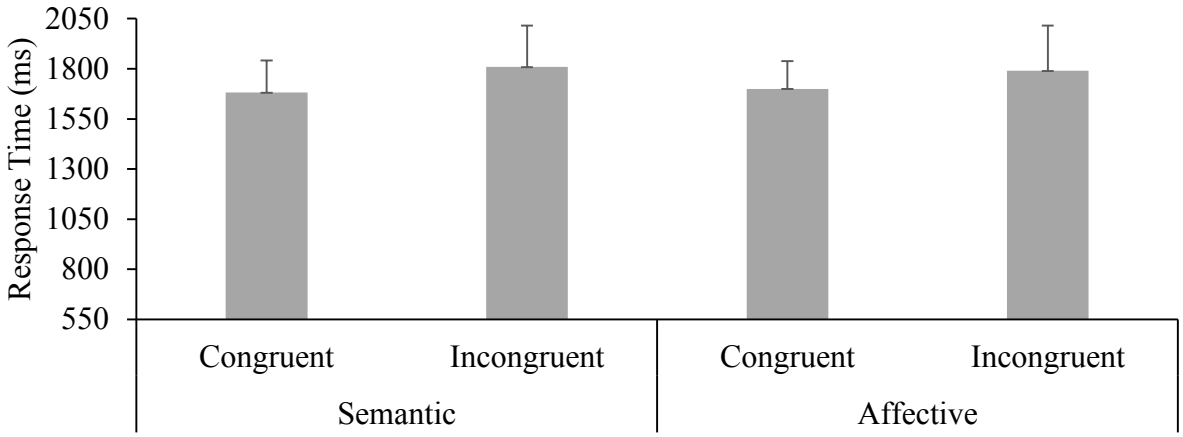

Figure D5. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for participants receiving a Benzodiazepine. Error bars show 2 *SE*.

The three participants receiving a Benzodiazepine displayed slower RT as compared to the main analysis (see Figure D5). The expected pattern of semantic priming was found with RT to semantically congruent trials 127 ms faster than RT to semantically incongruent trials. RT to affectively congruent trials was 91 ms faster than RT to affectively incongruent trials.

D.3.6 Participants receiving medication for PTSD

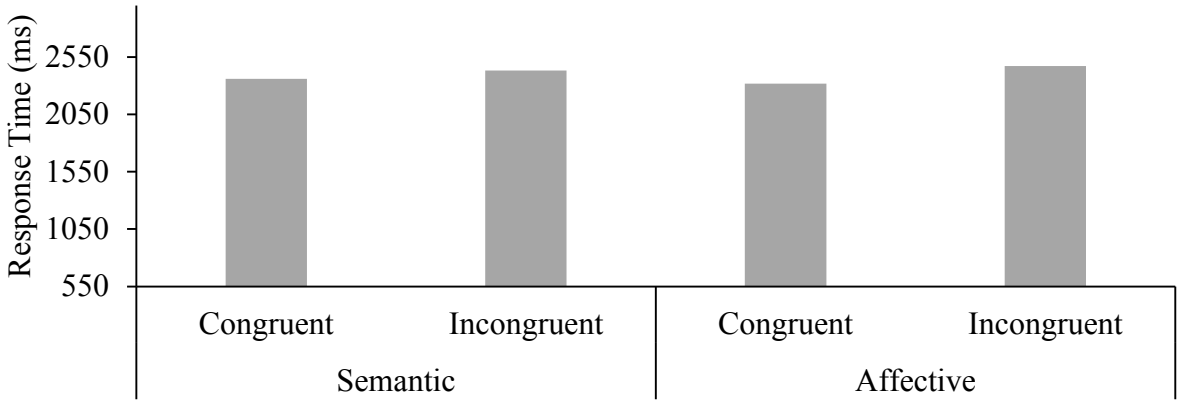

Figure D6. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for a participant receiving medication for relief of symptoms associated with PTSD.

Semantic priming data for the participant receiving medication for PTSD symptoms (see Figure D6) indicates much slower RT as compared to the main analysis. Although the expected pattern was found for semantic trials (semantically congruent trials were 72 ms faster than semantically incongruent trials), affectively congruent trials were also 153 ms faster than affectively incongruent trials.

D.3.7 Participants receiving anti-psychotic medication

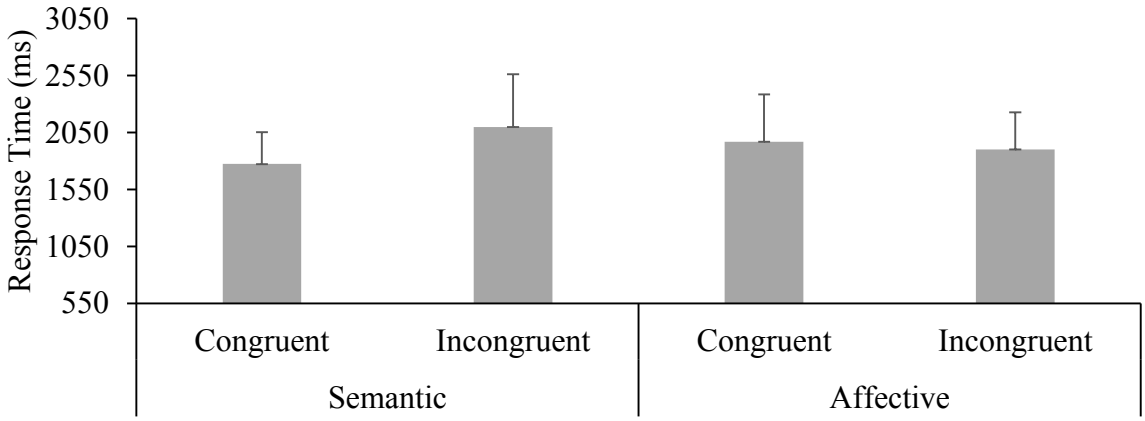

Figure D7. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for participants receiving anti-psychotic medication. Error bars show 2 *SE*.

Three participants in the Semantic Priming task were in receipt of anti-psychotic medication, evidencing slower RT as compared to the main analysis (see Figure D7). A reliable semantic priming effect was found, with RT to semantically congruent trials 323 ms faster than RT to semantically incongruent trials. RT to affectively congruent trials was 67 ms slower than RT to affectively incongruent trials.

D.3.8 Participants receiving ADHD medication

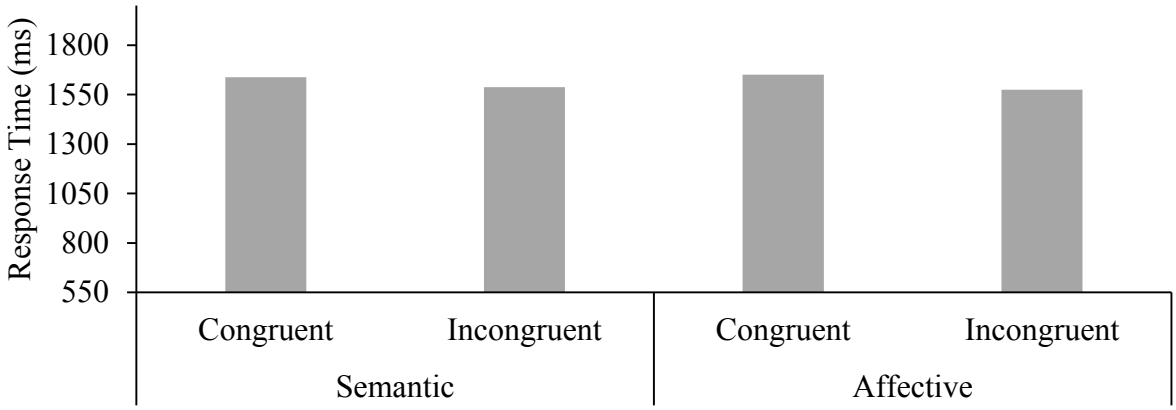

Figure D8. Mean response time, in ms, to semantically congruent and incongruent, and affectively congruent and incongruent trials in the Semantic Priming task for a participant receiving medication for symptoms associated with PTSD.

The participant receiving Ritalin displayed slower RT in the Semantic Priming task (see Figure D8) as compared to the main analysis. There was no evidence of priming for this participant: RT to semantically incongruent trials was 50 ms faster than RT to semantically congruent trials. RT to affectively incongruent trials was also faster, by 75 ms, than RT to affectively congruent trials.