In the eye of the beholder: Psychopathy and pupil response to emotion

A thesis submitted for the degree of Doctor of Philosophy

2016

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Declarations

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Dedication and acknowledgements

Firstly, I would like to express my sincere gratitude to my supervisor Professor Robert Snowden for his encouragement, knowledge and direction throughout my PhD research, as well as giving me the opportunity to pursue my PhD in the first instance. His guidance and feedback were imperative to the progression of my skills as a researcher and to the completion of my thesis.

Many thanks to Professor Nicola Gray for her input and advice into the conception and development of my research.

I greatly appreciate the support of my research laboratory – specifically Dr Katie O'Farrell and Aimee McKinnon – throughout my PhD for their insightful feedback on my research, as well as being a sympathetic ear.

I gratefully acknowledge the support of the Wellcome Trust/NISCHR and the School of Psychology at Cardiff University for the funding received to undertake my PhD.

I want to extend my sincere thanks to the multidisciplinary teams working at Llanarth Court Hospital, Kneesworth House, Ty Catrin and Ty Cwm Rhondda. Their support and encouragement was invaluable in the recruitment of my participants and my PhD research would not have been achievable without their backing. I would also like to thank in particular the psychology team at Llanarth Court Hospital for welcoming me into their group and making data collection a pleasure (and for providing so much cake).

I am indebted to all my friends in (and outside of) Cardiff for providing me with many welcome distractions throughout my PhD. Special thanks to Alex for always letting me win and to Matt for being a fantastic house-mate.

I would like to say a heart-felt thank you to my Mum, Dad, brother Matthew (all the way in Australia) and Grandma for their emotional (and sometimes financial in the case

of my parents!) support during my PhD. They have always been there for me, living every minute of my research, which has given me the belief and fortitude that I needed. The journey has not always been easy, but I am very thankful for their kindness and solace. And finally to Andrea, who has carried the baton of being my emotional crutch throughout the writing-up of my thesis. I am so grateful for your patience and reassurance, as well as your motivational speeches (of which there is one for every occasion) that have kept my spirits high. I could not have completed my PhD without the support of my family and Andrea.

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List of abbreviations

Abbreviation	Meaning	Page
ADFES	Amsterdam Dynamic Facial Expression Set	97
ADI	Arrogant and Deceitful Interpersonal Style	7
ASPD	Antisocial Personality Disorder	12
BLA	Basolateral amygdala	19
CeA	Central amygdala	19
DAAM	Differential Amygdala Activation Model	19
DAE	Deficient Affective Experience	7
EI	Emotional index	78
IADS	International Affective Digitised Sounds	38
IAPS	International Affective Picture System	37
IES	Integrated Emotions Systems	18
IIB	Impulsive and Irresponsible Behavioural Style	7
LSRP	Levenson Primary and Secondary Psychopathy Scale	9
MCMC	Markov Chain Monte Carlo	67
OFC	Orbitofrontal cortex	96
PCL-R	Psychopathy Checklist-Revised	4
PCL:SV	Psychopathy Checklist-Revised: Screening Version	16
PCL:YV	Psychopathy Checklist-Revised: Youth Version	16
PLR	Pupil light reflex	37
PPI/PPI-R	Psychopathic Personality Inventory/ Psychopathic Personality Inventory - Revised	9
RMH	Response Modulation Hypothesis	19
SCR	Skin conductance response	23
SRP	Hare Self Report Psychopathy Scale – fourth edition	9
Tri-PM	Triarchic Psychopathy Measure	11
VIM	Violence Inhibition Mechanism	18
vmPFC	Ventromedial prefrontal cortex	96
WASI	Wechsler Abbreviated Scale of Intelligence	124
WAIS	Wechsler Adult Intelligence Scale	125

Abstract

Psychopathy is a disorder of personality, typically characterised by interpersonal and affective personality traits as well as impulsive and irresponsible behaviour, with the disorder linked to antisocial behaviour. Psychopathy, in particular core interpersonal/affective psychopathy traits, is associated with an emotional impairment, which has been frequently associated to negative stimuli. However, evidence for autonomic responsivity during passive-viewing of emotion has been equivocal, although much previous research has failed to convincingly investigate this question. Therefore, the current thesis developed a pupillometry paradigm to measure autonomic responsivity during passive-viewing of negatively and positively valenced images, static facial expressions, dynamic facial expressions and sound-clips as a function of psychopathy traits. Chapter 2 highlighted the importance of controlling for luminance, contrast and colour for complex visual stimuli due to their constrictive effect on pupil diameter, as well as demonstrating that presentation duration and habituation play little role in moderating emotional modulation of pupil diameter to visual stimuli. Chapter 3 investigated psychopathy within a population of 102 (52 female) undergraduate students, finding that no dimension of the Triarchic Psychopathy Measure was related to pupil responsivity to emotion, although this was likely due to a lack of psychopathy within the sample. Chapter 4 explored psychopathy within 82 male forensic psychiatric patients, observing that the interpersonal/affective dimension of the Psychopathy Checklist-Revised (PCL-R) was selectively associated to early pupil hypo-responsivity to negative stimuli, but not positive stimuli, and this pattern was consistent across images, dynamic facial expressions and sound-clips. The lifestyle/antisocial dimension of the PCL-R was unrelated to pupil responsivity to emotion. These findings implicate that individuals high in interpersonal/affective psychopathy traits have an underlying deficient defensive motivational system that extends to autonomic responsivity during passive-viewing of negative stimuli, as well as suggesting the existence of an attentional/processing

impairment given that autonomic hypo-responsivity emerged only for early pupil reactivity.

1 Chapter 1: General introduction

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ADI	Arrogant and Deceitful Interpersonal Style	7
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1.1 What is psychopathy and why is it important?

Psychopathy is a constellation of personality traits typically characterised by interpersonal (e.g. grandiosity, manipulativeness) and affective personality traits (e.g. callousness, shallow affect, a lack of remorse and empathy), as well as a disinhibited and irresponsible behavioural style (Hare, 2003). The best-estimate of the prevalence of psychopathy in the UK is just under 1% in the general population (Coid, Yang, Ullrich, Roberts, & Hare, 2009). Despite its relatively low prevalence, psychopathy is one of the most important legal constructs as it places a disproportionate economic demand on society, the majority of which is related to forensic costs (Kiehl & Hoffman, 2011). To illustrate, the prevalence of psychopathy increases considerably within offender populations (Coid, Yang, Ullrich, Roberts, Moran, et al., 2009; Kiehl & Hoffman, 2011) and, also, psychopathy is strongly predictive of recidivism (Hart, Kropp, & Hare, 1988), violent recidivism (Rice & Harris, 1997), as well as poor institutional adjustment and treatment outcomes (Leistico, Salekin, DeCoster, & Rogers, 2008; Walters, 2003), which is particularly linked to the antisocial features of the disorder (Leistico et al., 2008; Walters, Knight, Grann, & Dahle, 2008) (this is discussed further in 1.2.2.3 Four-facet model of psychopathy). However, the detrimental impact of psychopathy is not limited to overt antisocial behaviour as there are many individuals high in psychopathy traits within the wider community who disregard or violate societal rules and expectations, but manage to avoid conviction by the criminal justice system (Hare, 1999). To illustrate, Hare (2003) reports the existence of the ‘white collar psychopath’, who is an unreliable, untrustworthy and predatory employee who can succeed within corporate structures often at the expense of clients, patients or the general public such as in the involvement of fraud, scams and a myriad of unethical corporate practices. The impact of psychopathy is particularly concerning given that individuals high in psychopathy are released earlier from prison (Porter, Brinke, & Wilson, 2009) and are perceived within

corporations as charismatic and effective communicators, traits associated with effective leaders (Babiak, Neumann, & Hare, 2010).

1.2 The conceptualisation of psychopathy

1.2.1 Unitary models of psychopathy

The modern conceptualisation of psychopathy derives most directly from the pioneering work of Hervey Cleckley within psychiatric patients and is described in his seminal monograph *The Mask of Sanity* (Cleckley, 1941). Cleckley described psychopaths as presenting as initially well-adjusted and superficially charming, but over time it becomes evident that the individual is characterised by a shallow affective experience, lack of remorse or empathy, an incapacity for love, as well as demonstrating irresponsibility, insincerity, unreliability, lack of planning, poor judgement and an inability to learn from experience. Importantly, Cleckley did not outline individuals high in psychopathy as dangerous, violent or cruel; instead, their antisocial behaviour was seen as a consequence of their dysfunctional personality rather than central to the construct. Cleckley's clinical profile of psychopathy has served as a template from which modern descriptions of psychopathy have evolved.

In contrast to Cleckley's work, Robins (1978, 1996) and McCord and McCord (1964) proposed psychopathy to be associated with a more antisocial personality based on their work with offenders. McCord and McCord (1964) described psychopathy as characterised by a maladaptive and disturbed personality featuring aggression, callousness, parasitic behaviour and impulsivity, as well as recognising the shallow affective experience identified by Cleckley (1941). They noted that serious and varied offending behaviour was common among individuals with psychopathy, but that criminality was not inevitable. In order to address concerns regarding the difficulties in reliably defining psychopathy using Cleckley's personality traits, Lee Robins emphasised behavioural aspects of psychopathy, focusing on longitudinal studies that identified that

children who were delinquent (e.g. lying, stealing, aggressive behaviour) were more likely to continue to exhibit antisocial behaviour into later life (Robins, 1978, 1996). However, although the use of behavioural features improved the reliability of diagnosis, many researchers argued that the validity of the construct was sacrificed as a consequence making the definition under-inclusive and failing to capture interpersonal/affective psychopathy traits in the absence of antisocial behaviour (Lilienfeld, 1994).

Today, the clinical description of psychopathy is dominated by Robert Hare's work and the development of his Psychopathy Checklist-Revised (PCL-R; Hare, 2003), which largely agreed with Cleckley's conceptualisation of interpersonal, affective and behavioural traits, but also placed greater emphasis on antisocial behaviour recognising the work of Lee Robins (Robins, 1978, 1996). The individual is assessed across a 20-item checklist based on a semi-structured interview and a comprehensive file-review to give the individual a total PCL-R score. The PCL-R was originally designed to index psychopathy as a unitary construct and a total cut-off score of 30 was recommended for an individual to be deemed as psychopathic (Hare & Neumann, 2008), although a threshold of 25 is often used within European samples (Cooke & Michie, 1999; Cooke, Michie, Hart, & Clark, 2005). The PCL-R has shown theoretically meaningful correlations with self-report, laboratory and psychophysiological measures (Hare, 2003), as well as being a good predictor of recidivism and violent recidivism across a number of settings and populations (Dolan & Doyle, 2000; Hare, 2003; Hart et al., 1988; Hemphill, Hare, & Wong, 1998; Salekin, Rogers, & Sewell, 1996; Tengström, Grann, Långström, & Kullgren, 2000). The PCL-R has become the most extensively validated measure of psychopathy, although a number of scholars have expressed concerns that the measure's dominance has impeded scientific investigation as the tool has become equated with the construct of psychopathy itself (Skeem & Cooke, 2010; Skeem, Polaschek, Patrick, & Lilienfeld, 2011).

1.2.2 Multi-faceted models of psychopathy

1.2.2.1 Dual pathway model of psychopathy

Traditionally psychopathy has been considered as a unitary construct with a single underlying cause (Neumann, Hare, & Newman, 2007), yet recent models have highlighted the multi-faceted nature of psychopathy proposing intersecting trait dimensions. The dual pathway model of psychopathy emphasises separate temperamental dimensions within psychopathy with distinctive neurobiological underpinnings (Fowles & Dindo, 2006; Patrick & Bernat, 2009). These underlying dispositions relate to trait fearlessness (thought to reflect under-reactivity of the brain's defensive motivational system) and a dysfunction in the regulation of behaviour and emotional responses (thought to represent deficiencies in frontal cortical brain regions leading to impairments in planning and affective/behavioural control). This dual process model is related to the proposal that there are variants of psychopathy with 'primary psychopathy' reflecting an innately fearless high psychopathy individual characterised by low anxiety, whereas 'secondary psychopathy' reflects a more emotionally and behaviourally volatile variant (Karpman, 1941).

Despite the PCL-R originally being intended to index psychopathy as a unitary construct, the measure provides scores across two dimensions: Factor 1, which relates to interpersonal/affective psychopathy traits (e.g. grandiosity, manipulativeness, callousness/lack of empathy, shallow affect and a lack of remorse), whereas Factor 2 reflects the lifestyle/antisocial dimension of psychopathy (e.g. impulsivity, irresponsibility, parasitic lifestyle, juvenile delinquency and criminal versatility). The factors of the PCL-R are largely considered to be theoretically aligned with the underlying temperamental dimensions described in the dual pathway model (Patrick & Bernat, 2009); Factor 1 represents temperamental fearlessness and Factor 2 reflects elevated emotionality and regulatory deficits (Fowles & Dindo, 2006; Lilienfeld, Watts, Francis Smith, Berg, & Latzman, 2015; Patrick, 2007; Patrick & Brislin, 2015). Support for the dual pathway

model has come from a body of literature that identifies that the factors of the PCL-R are related to diverging theoretically meaningful personality correlates that are consistent with the dual pathway model. Factor 1 has been found to be associated with greater fearlessness, emotional stability, positive emotionality, instrumental aggression, behavioural control, as well as reduced empathy and diminished negative emotionality (i.e. depression, fear, anger, anxiety) (Del Gaizo & Falkenbach, 2008; Hicks & Patrick, 2006; Lilienfeld, Watts, et al., 2015; Seara-Cardoso, Dolberg, Neumann, Roiser, & Viding, 2013; Seara-Cardoso, Neumann, Roiser, McCrory, & Viding, 2012; Skeem, Poythress, Edens, Lilienfeld, & Cale, 2003; Yildirim & Derksen, 2015), whereas Factor 2 has been linked to elevated negative emotionality, reactive aggression, disinhibition, impulsivity, greater risk taking and anxiety (Coid, Freestone, & Ullrich, 2012; Drislane, Patrick, & Arsal, 2014; Falkenbach, Poythress, & Creevy, 2008; Falkenbach, Stern, & Creevy, 2014; Hicks, Markon, Patrick, Krueger, & Newman, 2004; Hicks & Patrick, 2006; Hyde, Byrd, Votruba-Drzal, Hariri, & Manuck, 2014; Lyons, 2015; Salekin, Chen, Sellbom, Lester, & MacDougall, 2014; Skeem, Johansson, Andershed, Kerr, & Louden, 2007; Swogger & Kosson, 2007). There is now increasing evidence that the PCL-R factors are related to differing external criterion variables, which is unusual for two components considered to represent a unitary construct highlighting the multi-faceted nature of psychopathy (Lilienfeld, Watts, et al., 2015; Patrick & Bernat, 2009). Moreover, scholars have argued that only Factor 1 is indexing core characteristics inherent to the traditional emotionally deficient high psychopathy construct (Poythress & Hall, 2011; Skeem & Cooke, 2010). In contrast, the inclusion of Factor 2 items leads to the PCL-R identifying a more deviant and maladjusted high psychopathy individual (Yildirim & Derksen, 2015).

1.2.2.2 Three-factor model of psychopathy

Researchers have criticised the construct validity of the PCL-R arguing that antisocial behaviour is a likely consequence of the psychopathy rather than an inherent component

of the disorder (Cooke & Michie, 2001; Cooke, Michie, Hart, & Patrick, 2006; Lilienfeld, Watts, et al., 2015; Skeem & Cooke, 2010). Cooke and Michie (2001) were among the first to contend that the significance of antisocial behaviour within the PCL-R was unclear. Instead, they proposed a 3-factor model of psychopathy after finding that the two-factor model was not an adequate structural fit for the PCL-R data of 2,067 participants from both prison and forensic psychiatric settings. Their model essentially splits Factor 1 of the PCL-R into two distinct dimensions called 'Arrogant and Deceitful Interpersonal Style' (ADI) and 'Deficient Affective Experience' (DAE), whilst a third cluster reflected the Lifestyle items of the PCL-R called 'Impulsive and Irresponsible Behavioural Style' (IIB). They argued that criminality is not essential to the psychopathy and that the PCL-R's emphasis on offending behaviour may reflect the antisocial population that the tool was developed within. Lilienfeld, Watts, et al. (2015) has supported the dissection of Factor 1 into interpersonal and affective facets, as they show differing personality correlates with the interpersonal dimension linked to adaptive functioning, whereas the affective component was tied to antagonism and low negative emotionality.

1.2.2.3 Four-facet model of psychopathy

Hare and Neumann (2005) and Hare (2003) disagreed with the removal of antisocial behaviour from the definition of psychopathy and proposed a four-facet model of psychopathy breaking the two PCL-R factors down further into four components: Facet 1 (Interpersonal), Facet 2 (Affective), Facet 3 (Lifestyle) and Facet 4 (Antisocial). Hare and colleagues maintained that it is important to recognise the dynamic interaction between personality traits associated to psychopathy and antisocial behaviour. They suggest that personality traits should not be viewed as static entities that lead to the development of behaviour, but rather as a reciprocal relationship with the two influencing each other over the course of development (e.g. increased exposure to criminality may cause desensitisation and callousness). Hare (2003) further reported support for the 4-

facet model finding that confirmatory factor analysis demonstrated the model to be an excellent description of psychopathy across male and female offenders, male forensic psychiatric patients and for PCL-R file review alone. There is further evidence that the 4-facet model is an excellent fit for psychopathy data across a variety of contexts and diverse national samples (Hare & Neumann, 2008; Mokros et al., 2011; Neumann et al., 2007; Neumann, Johansson, & Hare, 2013; Vitacco, Neumann, & Jackson, 2005).

Walters et al. (2008) reported evidence indicating that the PCL-R's ability to predict recidivism lies with the inclusion of the antisocial facet, with the interpersonal, affective and lifestyle facets contributing minimally to predicting antisocial behaviour. On the one hand, this reflects the necessity of including the facet to predict criminality for psychopathy. Conversely, it equally questions the role of psychopathy as an important legal construct, as proposed by Hare (2003), given that prior criminality was the best predictor of future criminality rather than the remaining psychopathy facets. However, Blackburn (2007) argued that the antisocial component of the PCL-R taps a broad disposition of criminality that is closely related to antisocial behaviour, rather than simply reflecting the link between previous and future criminal acts. However, the relevance of antisociality to psychopathy continues to be debated (see Skeem & Cooke, 2010 for a review).

1.2.2.4 Self-report measures derived from the PCL-R

Despite the PCL-R's psychometric strengths and wide-spread use, it is time-consuming and not appropriate within non-offender samples due to the need for in-depth historical information. This has led to the development of self-report measures of psychopathy (Lilienfeld & Fowler, 2006). This raises further issues in relation to the validity of self-report to index psychopathy, as well as relating to a wider debate regarding whether psychopathy is measurable across normal personality dimensions. These controversies will be addressed later in this chapter (see 1.2.3 Self-report versus clinically-rated measures of psychopathy and 1.2.4 Psychopathy as a taxonic versus

dimensional construct). Several of these self-report psychopathy tools derive from the PCL-R and, therefore, reflect the tool's structure. One such measure is the Levenson Primary and Secondary Psychopathy Scale (LSRP, Levenson, Kiehl & Fitzpatrick, 1995), which adheres closely to the two-factor model of the PCL-R by indexing the primary and secondary psychopathy dimensions. The construct validity of the LSRP has been demonstrated with both dimensions showing theoretically meaningful personality correlations. LSRP Factor 1 (primary psychopathy) was related to low agreeableness, high narcissistic personality disorder and ratings of prototypical psychopathy, while LSRP Factor 2 (secondary psychopathy) was more strongly related to neuroticism and conscientiousness. A further self-report measure derived from the PCL-R is the Hare Self Report Psychopathy Scale – fourth edition (SRP; Paulhus, Neumann & Hare, In Press), which has gone through several iterations and parallels the four-facet PCL-R model (Williams & Paulhus, 2004). The SRP has shown good construct validity as the interpersonal and affective facets of psychopathy were predominantly associated with low agreeableness, whereas the lifestyle and antisocial facets were associated with negative emotionality, low conscientiousness and low agreeableness (Gaughan, Miller, & Lynam, 2012; Neumann & Pardini, 2014). This indicates that self-report measures derived from the PCL-R show that interpersonal/affective psychopathy traits are tied to an antagonistic nature, while lifestyle/antisocial psychopathy traits are associated to negative emotionality and disinhibition.

1.2.2.5 Psychopathic-personality inventory

The Psychopathic Personality Inventory – Revised (PPI-R), originally the Psychopathic Personality Inventory (PPI), was developed by Lilienfeld and Andrews (1996) using exploratory analysis and multiple revisions to develop the measure. In contrast to the PCL-R, the development of the PPI was not based on the assumption of a unitary higher-order psychopathic construct. The PPI-R is constructed of 154 items after the removal of 33 items from the PPI that were culturally specific and to lower the

reading level of the measure (Lilienfeld & Widows, 2005). Factor analysis of the PPI/PPI-R items (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Benning, Patrick, Hicks, Blonigen, & Krueger, 2003) has shown that the items can be organised into eight unidimensional personality subscales that load onto two higher-order factors: PPI-I (Fearless Dominance) and PPI-II (Impulsive Antisociality). There is a remaining independent subscale called Coldheartedness that fails to load onto either factor. Fearless Dominance is related to the interpersonal/affective features of psychopathy with negative relationships to empathy, anxiety, anxiety-related disorders, somatic concerns, stress reactivity, neuroticism, harm avoidance and negative affect, as well as positive relationships to narcissism, thrill-seeking behaviour, reward-sensitivity and positive affect (Benning, Patrick, Blonigen, et al., 2005; Benning et al., 2003; Benning, Patrick, Salekin, & Leistico, 2005; Falkenbach et al., 2014; Maples et al., 2014; Miller & Lynam, 2012; Patrick, Edens, Poythress, Lilienfeld, & Benning, 2006; Ross, Benning, Patrick, Thompson, & Thurston, 2008). Impulsive Antisociality shows positive correlations with criterion measures reflecting maladjustment and behavioural deviancy. Specifically, Impulsive Antisociality has been positively associated with antisocial behaviour, aggressiveness, irresponsibility, substance abuse, impulsiveness, disinhibition, neuroticism, anxiety, and depression (Benning, Patrick, Blonigen, et al., 2005; Benning et al., 2003; Benning, Patrick, Salekin, et al., 2005; Miller & Lynam, 2012; Patrick et al., 2006; Ross et al., 2008).

The Fearless Dominance scale is also related to positive-adjustment criterion measures such as higher assertiveness, social potency, sociability, social dominance and social achievement (Benning, Patrick, Blonigen, et al., 2005; Benning et al., 2003; Maples et al., 2014). Therefore, the PPI-R's conceptualisation of psychopathy is capturing a more socially adaptive and socially potent definition of psychopathy compared to the PCL-R (and measures derived from it), in line with Cleckley's original positive-adjustment traits (Yildirim & Derksen, 2015). Indeed, Fearless Dominance

shows only modest association to Factor 1 of the PCL-R (Benning, Patrick, Blonigen, et al., 2005; Copestake, Gray, & Snowden, 2011; Malterer, Lilienfeld, Neumann, & Newman, 2010; Marcus, Fulton, & Edens, 2013; Miller & Lynam, 2012; Poythress, Edens, et al., 2010) and the primary psychopathy component of the LSRP (Marcus et al., 2013). Interestingly, Factor 1 of the SRP has been reported to be highly related to Fearless Dominance (Benning, Patrick, Salekin, et al., 2005), although early versions of the SRP have been found to not correspond closely to the PCL-R's two-factor model (Benning, Patrick, Salekin, et al., 2005; Williams & Paulhus, 2004). Further evidence of the divergence between PPI-R and the PCL-R is reflected in the minimal relationship shown between the PPI-R factors. This highlights that the dimensions of the PPI-R capture fundamentally different dispositional dimensions to one another, in contrast to the PCL-R factors that are highly interrelated (Benning et al., 2003; Neumann, Uzieblo, Crombez, & Hare, 2013; Patrick et al., 2006). The existence of positive-adjustment indicators within the PPI-R represents an ongoing debate in the literature as to whether these features are integral to the disorder (Skeem et al., 2011). Hare and Neumann (2010) argued that positive-adjustment traits have little relevance to psychopathy and are a possible consequence of the disorder, whereas other researchers have argued that these traits are central to the disorder (Lilienfeld, Smith, et al., 2015; Lilienfeld, Watts, et al., 2015; Patrick & Drislane, 2015).

1.2.2.6 Triarchic psychopathy model

A further framework that emphasises the importance of the positive-adjustment component of psychopathy is the Triarchic Model of Psychopathy (Patrick, 2010a; Patrick, Fowles, & Krueger, 2009). Patrick and colleagues formulated their triarchic model, and the accompanying Triarchic Psychopathy Measure (Tri-PM) to integrate and reconcile differing historical definitions of personality associated to psychopathy. The triarchic model proposes three distinct phenotypic constructs: Boldness, Meanness and Disinhibition. Boldness refers to the positive-adjustment features of psychopathy,

specifically the nexus of social dominance, low stress reactivity and fearlessness. Boldness is considered to reflect the manifestation of a latent disposition towards fearlessness; that is, the sensitivity of the individual's defensive system towards threat (Patrick & Drislane, 2015). Poy, Segarra, Esteller, Lopez, and Molto (2014) reported that, in relation to the Five-Factor Model of personality (Digman, 1990), Boldness was associated to low neuroticism, high extraversion, high openness to experience, consistent with the theoretical description of this dimension representing a fearless, socially potent, narcissistic and antagonistic pathology. The Triarchic model places an emphasis on Boldness differentiating psychopathy from alternate antisocial disorders, such as antisocial personality disorder, and research has supported this distinction (Venables, Hall, & Patrick, 2014; Wall, Wygant, & Sellbom, 2015), although the recent definition of Antisocial Personality Disorder (ASPD) (American Psychiatric Association, 2013) has shown improvements in capturing the Boldness dimension (Strickland, Drislane, Lucy, Krueger, & Patrick, 2013). Boldness is captured considerably by Fearless Dominance of the PPI-R (Drislane, Patrick, & Arsal, 2014; Esteller, Poy, & Moltó, 2016; Hall et al., 2014; Sellbom & Phillips, 2013), highlighting that both dimensions are tapping the same positive-adjustment features of psychopathy, as well as by Facet 1 (Interpersonal) of the PCL-R (Patrick, 2010b; Patrick et al., 2009; Venables et al., 2014).

Meanness reflects cruelty, lack of empathy, predatory aggression and excitement seeking. Meanness is thought to act as an intersection between the central affective-interpersonal deficits of psychopathy and externalising psychopathology (Patrick & Drislane, 2015), overlapping with both Boldness and Disinhibition (Almeida et al., 2015; Craig, Gray, & Snowden, 2013; Drislane, Patrick, & Arsal, 2014; Drislane, Patrick, Sourander, et al., 2014; Marion et al., 2013; Poy et al., 2014; Sellbom & Phillips, 2013; Stanley, Wygant, & Sellbom, 2013; Venables et al., 2014). Furthermore, in relation to external personality criterion, Meanness has been found to relate to fearlessness and diminished empathy, likewise to Boldness, as well as showing a personality profile

characterised by low Agreeableness and Conscientiousness similar to Disinhibition (Poy et al., 2014; Sellbom & Phillips, 2013; Stanley et al., 2013). Meanness is also positively related to both Factor 1 and Factor 2 of the PCL-R (Venables et al., 2014), as well as both the Fearless Dominance and Impulsive Antisociality dimensions of the PPI-R (Esteller et al., 2016) highlighting that Meanness is tapping both interpersonal/affective and behavioural components of psychopathy.

Disinhibition entails the component of psychopathy characterised by a lack of inhibitory control and difficulties regulating affect leading to impulsive and irresponsible externalising behaviour (Patrick & Drislane, 2015). In support, Disinhibition has been associated with a pattern of increased neuroticism, anxiety, sensation-seeking, reward-sensitivity, and externalising behaviours (Poy et al., 2014; Stanley et al., 2013), as well as being captured extensively by Facet 3 (Lifestyle) of the PCL-R (Hall et al., 2014; Venables et al., 2014) and PPI-R Impulsive Antisociality (Drislane, Patrick, & Arsal, 2014; Esteller et al., 2016; Hall et al., 2014; Patrick et al., 2009; Sellbom & Phillips, 2013) consistent with the dimension's theoretical definition. However, Patrick et al. (2009) argues that Disinhibition is not synonymous with psychopathy; rather psychopathy reflects the combination of Disinhibition alongside dispositional Boldness or Meanness.

1.2.3 Self-report versus clinically-rated measures of psychopathy

The development of self-report measures of psychopathy has led researchers to consider whether this method is a reliable way to index psychopathy. This is especially relevant considering that psychopathy is associated with pathological lying and a lack of insight into their own personality and behaviour (Lilienfeld, Fowler, & Patrick, 2006). However, Lilienfeld and Andrews (1996) highlighted that psychopathy is slightly or even moderately negatively related to social desirability. This suggests that individuals high in psychopathy are reporting accurately for socially unwanted behaviours such as aggression, antisocial activity and poor impulse control, perhaps reflecting that these

individuals have a different view of what is ‘normal’ behaviour. Also, although individuals with psychopathy may lack insight into their own behaviours and, thus, do not give factually correct responses, their responses are revealing of their perception of themselves and their interaction with others (Lilienfeld et al., 2006). Many items on self-reported psychopathy measures are interested in the individual’s beliefs rather than a ‘true’ response. For example, consider the first item of the PPI-R, ‘If I really want to, I can persuade most people of almost anything’. This item is an indicator of the individual’s narcissism rather than their ability to manipulate; hence, the individual’s beliefs about themselves are more relevant than factually accurate responses. Ray et al. (2013) underlined that psychopathy was not associated with a positive response bias in a meta-analysis by examining studies that explored the relationship between the PPI/PPI-R and LSRP to inaccurate response styles (i.e. social desirability/faking good and faking bad). Across both measures, total psychopathy and Impulsive Antisociality/Secondary psychopathy were negatively associated with a socially desirable/faking good response pattern, and even positively related to faking bad response sets suggestive of over-reporting of negative behaviour. Fearless Dominance/Primary psychopathy showed no relationship to inaccurate response styles.

However, the relationship between self-report measures and clinically-rated measures of psychopathy, typically the PCL-R, have shown only modest correlations (Brinkley, Schmitt, Smith, & Newman, 2001; Marcus et al., 2013; Miller & Lynam, 2012; Poythress, Lilienfeld, et al., 2010; Venables et al., 2014; Williams, Nathanson, & Paulhus, 2003; Williams & Paulhus, 2004). However, this does not necessarily undermine the use of self-report measure to index psychopathy, as these associations are consistent with expected moderate correlations for inventories of the same construct across different measurement domains (Campbell & Fiske, 1959). Conversely, at a dimensional level of psychopathy, self-reported inventories tend to be better suited for indexing the lifestyle/antisocial aspects of psychopathy rather than the interpersonal/affective

dimension (Benning, Patrick, Salekin, et al., 2005; Brinkley et al., 2001; Copestake et al., 2011; Lilienfeld, Smith, et al., 2015; Malterer et al., 2010; Marcus et al., 2013; Miller, Gaughan, & Pryor, 2008; Poythress, Lilienfeld, et al., 2010; Venables et al., 2014). Overall, self-report measures are a useful index of psychopathy given that psychopathy is not associated to under-reporting of socially deviant behaviour, yet researchers should be wary that questionnaires may not be tapping interpersonal/affective psychopathy traits as closely as clinically-rated measures.

1.2.4 Psychopathy as a taxonic versus dimensional construct

The emergence of self-report measures of psychopathy additionally relates to a wider debate regarding whether psychopathy is a taxonic (psychopathic or not psychopathic) or dimensional construct (degrees of psychopathy). This discussion is integral to understanding psychopathy as it impacts not only on the empirical understanding of the disorder, but also on legal policy and clinical decision makers (Marcus, John, & Edens, 2004). Lilienfeld (1998) identified several implications arising from the question of whether psychopathy has a categorical or dimensional latent structure. Firstly, this debate affects the hypothesised etiology of the disorder because a categorical approach assumes a single underlying cause to psychopathy, whereas a dimensional approach is likely to have a multifactorial etiology that lead to degrees of psychopathy. Further, if psychopathy is a taxonic construct then research would need to rely on studying psychopathy within forensic or clinical samples where the prevalence of psychopathy is high, unless the base-rate of psychopathy were sufficiently elevated in alternative populations. Finally, a further implication of this debate relates to the generalisability of findings regarding psychopathy. If psychopathy is dimensional, then this indicates that findings from non-clinical and non-offender samples can be extended to more severely affected populations, whereas a taxonic approach indicates that this is not possible.

Harris, Rice, and Quinsey (1994) investigated the latent structure of psychopathy within a sample of 653 offenders in a maximum security institution using the PCL-R, concluding that psychopathy was a taxon. However, a closer examination of their analyses suggests that Factor 2 was taxonic, whereas there was no evidence of taxonicity for Factor 1. Indeed, Skilling, Harris, Rice, and Quinsey (2002) reanalysed their data finding evidence of taxonicity specific to the lifestyle/antisocial items and not for the interpersonal/affective component of psychopathy. However, both studies were criticised for a number of methodological issues, including employing highly skewed predictors and adopting a dichotomous scoring scheme for psychopathy (Edens, Marcus, Lilienfeld, and Poythress Jr, 2006).

Recent research using taxonometric techniques have largely indicated that psychopathy can be considered as a dimensional construct. Marcus, Lilienfeld, Edens, and Poythress (2006) examined the latent structure of psychopathy within a sample of 309 offenders using the PPI. They found no evidence to consider psychopathy as a taxonic construct concluding that psychopathy may be considered as existing on a continuum. Further studies have similarly found that the latent structure of psychopathy is distributed dimensionally within forensic populations using the PCL-R (Edens et al., 2006; Guay, Ruscio, Knight, & Hare, 2007). Walters, Duncan, and Mitchell-Perez (2007) further reported no evidence that psychopathy can be considered as a taxonic construct within a sample of offenders across the four PCL-R facets, and instead exists on a continuum. The same research group replicated this finding using the screening version of the PCL-R (PCL:SV; Hart, Cox & Hare, 1995) within a larger forensic and psychiatric sample of 2,250 adults (Walters, Gray, et al., 2007). Moreover, the dimensionality of psychopathy has also been demonstrated to extend to delinquent adolescents across clinician rated (PCL:YV; Forth, Kosson, Hare, Sevecke & Krischer, 2014) and self-report measures of psychopathy (Edens, Marcus, & Vaughn, 2011; Murrie et al., 2007; Walters, 2014). Overall, the weight of evidence indicates that psychopathy can be best

understood as existing along a continuum reflecting differences in degree rather than kind.

1.3 Psychopathy and emotional processing

Emotions can be seen as a state of readiness for adaptive action reflecting the activity of two opposing systems: the defensive system, related to avoidance behaviour, and the appetitive system, associated with approach behaviour, with the two systems working together to maintain survival (Lang, 1995). Either motivation system can be activated, with the amygdala identified as a key neural structure within this survival network (Lang & Davis, 2006), which in turn activates somatic and autonomic physiological processes intended to ensure the organism's survival (Bradley, Codispoti, Cuthbert, & Lang, 2001).

1.3.1 Theories of emotional processing in psychopathy

There are several contemporary theories that have proposed that psychopathy is associated with emotional processing impairments. The general emotional deficit perspective posits that psychopathy is characterised by a blunted emotional experience that extends across all emotional valences. This hypothesis is consistent with Cleckley (1941)'s description that psychopathy is characterised by a blunted emotional experience that impedes the ability of these individuals to understand the emotional significance of events and the relevance to themselves. An impairment in response to both negative and positive cues would suggest that psychopathy is associated to an insensitivity in both defensive and appetitive motivational systems.

In contrast, several models have contended that psychopathy is associated to a deficit in response to specifically negative stimuli with several distinct variants proposed. The low fear hypothesis proposes that psychopathy is associated to a low-fear temperament caused by a weak defensive motivational system (Fowles, 1980; Lykken, 1957; Lykken, 1995; Lykken, 1996). This perspective identifies that psychopathy is

associated with an impairment in relation to specifically fear. Reduced capacity for experiencing fear would lead to deficits in the ability to recognise fear or empathise with others, as well as predisposing these individuals to antisocial behaviours as they are unable to learn from punishment and are driven to seek rewards. This model does not expect deficits in response to positive stimuli proposing that the appetitive motivational system is intact or even over-active (Fowles, 1980; Lykken, 1995). Similarly, the Violence Inhibition Mechanism (VIM) model (Blair, Jones, Clark, & Smith, 1997; Blair, 1995) describes that psychopathy is associated to a specific negative impairment, but instead focuses on the role of empathy in moral development. Blair describes the existence of an early developmental system called the VIM that is activated by distress cues and generates a negative emotional response in observers resulting in increased autonomic reactivity, attention and activation of the neural threat response system. The VIM is central to the development of moral socialisation of individuals through associating the negative emotional experience triggered by the distress cues of others with the specific behaviour of the individual that caused that harm to others. It is proposed that psychopathy is associated with a deficient VIM leading to an insensitivity to interpersonal distress cues and a failure to learn about moral transgressions. Hence, the VIM model proposes that psychopathy is associated with an impairment in processing fearful and sad stimuli.

The idea that psychopathy is related to a persistent insensitivity to emotion (or specific emotions) has been challenged by perspectives that emphasise wider associative or attentional deficits in psychopathy that incorporate emotional impairments. Blair (2005) developed the VIM model into the Integrated Emotions Systems (IES) model that emphasised the role of the amygdala in affective impairments related to psychopathy. The model proposed that high psychopathy individuals are deficient in the formation of stimulus-reinforcement associations due to amygdala dysfunction, which is central to these representations (Everitt, Cardinal, Parkinson, & Robbins, 2003). That is,

high psychopathy individuals are impaired in their ability to acquire conditioned stimulus associations, as well as the affective and sensory representation of the conditioned stimuli. This reflects a pervasive inability to form associations that extends beyond difficulties forming associations involving moral transgressions as outlined in the VIM model. The IES model assumes that high psychopathy groups are impaired in the formation of both negative and positive stimulus-reinforcement associations, but expects greater impairments to negative associations. This is consistent with research that has highlighted that the amygdala is responsive to both negatively and positively-valenced stimuli (Baxter & Murray, 2002; Everitt et al., 2003), but shows greater reactivity to negative cues in particular (Breiter et al., 1996; Morris et al., 1996).

The Response Modulation Hypothesis (RMH; Newman & Lorenz, 2003) proposes that psychopathy is associated to an impairment in processing emotion due to an early attentional bottleneck which disrupts or delays the concurrent processing of information peripheral to the individual's ongoing goal-directed behaviour. The high psychopathy individual will display affective deficits when an emotion-irrelevant early attentional bottleneck is established inhibiting the processing of later information. As the hypothesised impairment is attentional in nature, the RMH model expects psychopathy to be associated with deficits in further domains (i.e. language/semantic processing, set-shifting) rather than emotional processing alone (Baskin-Sommers, Curtin, & Newman, 2013). Additionally, this model would predict deficits in processing emotion to be universal across emotional valences (i.e. not specific to negative affect) providing that an attentional bottleneck is established,

Moul, Killcross, and Dadds (2012) built upon the RMH and the IES model proposing the differential amygdala activation model (DAAM) to synthesise both attentional and emotion-based accounts of psychopathy. The DAAM emphasises that the belief of unified amygdala function is untenable and, instead, considers the separate roles of the central amygdala (CeA) and the basolateral amygdala (BLA), reflecting the distinction

between regions that are genetically older and those that are more recently evolved. The model indicates that the BLA is involved in the allocation of attention to stimuli within the environment based on their predicted value (Roesch, Calu, Esber, & Schoenbaum, 2010a, 2010b), as well as altering the value of an outcome (e.g. the amount of reward or punishment) (Balleine & Killcross, 2006). The CeA is responsible for encoding the valence of that outcome (e.g. negative or positive) (Balleine & Killcross, 2006). To illustrate, if a button was associated with a reward or punishment, the CeA would form the association whether the button was negative or positive, while the BLA would form the association to the extent of the reward or punishment. Moul et al. (2012) proposed that the central amygdala, comprised of the central and medial nuclei, is responding normally (or at increased levels), while the basolateral amygdala, comprised of the lateral, basal and accessory basal nuclei, is underactive within the high psychopathy individual. Therefore, emotional deficits associated to psychopathy are proposed as a result of a failure to shift attention to emotion-relevant stimulus (e.g. eyes of fearful faces), in line with the RMH, but also due to a failure to accurately encode emotional representations impairing associative learning, consistent with the IES model.

Notably, the DAAM indicates that individuals high in psychopathy can show normal autonomic responses to stimuli; rather, autonomic deficits to emotion for high psychopathy individuals are caused by an impaired ability to shift attention to emotion-relevant features or to encode the value of that stimulus. The model unfortunately fails to explicitly state whether it would expect psychopathy to be associated with impairments across all emotional stimuli or specific to negative stimuli. However, given that the model is centred on amygdala dysfunction, it seems likely that the model would propose psychopathy to be associated with an impairment across both negative and positive stimuli (Baxter & Murray, 2002; Everitt et al., 2003) with a more pronounced deficit to negative cues (Breiter et al., 1996; Morris et al., 1996).

1.3.2 Impaired emotional processing in psychopathy

As mentioned, psychopathy is associated with affective personality traits typically characterised by callousness, shallow affect, a lack of remorse and deficient empathy. A wealth of research has also demonstrated that individuals high in psychopathy show abnormal emotional processing across behavioural tasks, as well as physiological and neural hypo-responsivity to emotion. To illustrate, individuals high in psychopathy across both offender and community populations (using a range of psychopathy measures) have shown an impaired ability to recognise emotions (Bagley, Abramowitz, & Kosson, 2009; Blair, Colledge, Murray, & Mitchell, 2001; Blair et al., 2004; Blair et al., 2002; Bowen, Morgan, Moore, & van Goozen, 2013; Brook & Kosson, 2013; Dawel, O'Kearney, McKone, & Palermo, 2012; Dolan & Fullam, 2006; Hastings, Tangney, & Stuewig, 2008; Montagne et al.; Prado, Treeby, & Crowe, 2015; Snowden, Craig, & Gray, 2013; Stanković, Nešić, Obrenović, Stojanović, & Milošević, 2015), lower affective empathy (Seara-Cardoso et al., 2013; Seara-Cardoso et al., 2012), diminished emotional modulation of behavioural responses (Blair, Richell, et al., 2006; Edalati, Walsh, & Kosson, 2015; Kosson, Lorenz, & Newman, 2006; Mitchell, Richell, Leonard, & Blair, 2006; Williamson, Harpur, & Hare, 1991), reduced physiological anticipatory anxiety (Dindo & Fowles, 2011; Ishikawa, Raine, Lencz, Bihrle, & Lacasse, 2001; Ogloff & Wong, 1990; Wang, Baker, Gao, Raine, & Lozano, 2012), impaired fear conditioning (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; López, Poy, Patrick, & Moltó, 2013), attenuated electro-cortical responses to negative stimuli (Medina, Kirilko, & Grose-Fifer, 2016; Sadeh & Verona, 2012; Venables, Hall, Yancey, & Patrick, 2015), deficient fear-potentiated startle response (Baskin-Sommers et al., 2011, 2013; Benning, Patrick, & Iacono, 2005; Esteller et al., 2016; Herpertz, Werth, et al., 2001; Justus & Finn, 2007; Levenston, Patrick, Bradley, & Lang, 2000; Newman, Curtin, Bertsch, & Baskin-Sommers, 2010; Pastor, Molto, Vila, & Lang, 2003; Patrick, Bradley, & Lang, 1993; Sadeh & Verona, 2012; Vaidyanathan, Hall, Patrick, & Bernat, 2011; Vaidyanathan, Patrick, & Bernat, 2009; Vanman, Mejia, Dawson, Schell, & Raine, 2003; Verona, Bresin,

& Patrick, 2013), attenuated emotional potentiation of corrugator muscle activity (Flor et al., 2002; Patrick et al., 1993; Patrick, Cuthbert, & Lang, 1994), and deficient responsivity within emotion centres of the brain (predominantly the amygdala) (Birbaumer et al., 2005; Contreras-Rodriguez et al., 2014; Decety, Skelly, & Kiehl, 2013; Dolan & Fullam, 2009; Glenn, Raine, & Schug, 2009; Han, Alders, Greening, Neufeld, & Mitchell, 2012; Harenski, Edwards, Harenski, & Kiehl, 2014; Harenski, Kim, & Hamann, 2009; Hyde et al., 2014; Kiehl et al., 2001; Meffert, Gazzola, den Boer, Bartels, & Keysers, 2013; Mier et al., 2014; Pujol et al., 2011; Rilling et al., 2007). Overall, it is apparent that psychopathy is characterised by an impairment in processing emotions that extends across a range of paradigms.

1.3.3 The dimensions of psychopathy and emotional processing

Given the theorised multi-faceted nature of psychopathy (see 1.2.2 Multi-faceted models of psychopathy), research has increasingly explored emotional processing in relation to the interpersonal/affective and lifestyle/antisocial dimensions of psychopathy. Theoretically, it seems credible that the impairments in processing emotion that characterise psychopathy would be driven by the interpersonal/affective dimension given that this component reflects the core emotional dysfunction associated to psychopathy.

The results across behavioural tasks (e.g. recognising emotion, emotional modulation of reaction times) have been equivocal as research has conflictingly demonstrated that emotional deficits are driven by the interpersonal/affective dimension (Blair, Morton, Leonard, & Blair, 2006; Dawel et al., 2012; Lorenz & Newman, 2002), by the lifestyle/antisocial dimension (Brook & Kosson, 2013), or by both dimensions independently (Bagley et al., 2009; Blair, Richell, et al., 2006; Blair et al., 2002), as well as by neither dimension (Book, Quinsey, & Langford, 2007; Dolan & Fullam, 2006; Hastings et al., 2008; Pham & Philippot, 2010).

The literature is more consistent in relation to psychophysiological paradigms with the interpersonal/affective dimension of psychopathy frequently associated to deficient reactivity to negative stimuli with little or no relationship for the lifestyle/antisocial dimensions. To illustrate, the interpersonal/affective dimension (across a range of psychopathy measures) has been uniquely linked to reduced anticipatory anxiety of an aversive noise as measured by skin conductance responses (SCR) (Dindo & Fowles, 2011; Wang et al., 2012), deficient acquisition of fear measured again through SCR (López et al., 2013), attenuated electro-cortical responses to negative stimuli (Medina et al., 2016; Sadeh & Verona, 2012; Venables et al., 2015), and diminished fear-potentiated startle response (Baskin-Sommers et al., 2011; Benning, Patrick, & Iacono, 2005; Esteller et al., 2016; Patrick et al., 1993; Sadeh & Verona, 2012; Sutton, Vitale, & Newman, 2002; Vaidyanathan et al., 2011; Vaidyanathan et al., 2009; Vanman et al., 2003; Verona et al., 2013). Moreover, one of the most consistent findings across the literature is that psychopathy is associated to deficient fear-potentiated startle response that is driven by high interpersonal/affective psychopathy scores.

Neural studies have additionally reported that children with callous/unemotional traits (the childhood precursor to adult psychopathy, Barry et al, 2000) and adults high in interpersonal/affective psychopathy traits show attenuated activity in the amygdala in response to negative stimuli or when processing moral emotions (Carré, Hyde, Neumann, Viding, & Hariri, 2013; Glenn et al., 2009; Gordon, Baird, & End, 2004; Harenski et al., 2014; Harenski, Harenski, Shane, & Kiehl, 2010; Harenski et al., 2009; Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh & Cardinale, 2014; Marsh et al., 2013; Marsh et al., 2008; Viding et al., 2012). However, alternative studies have reported that decreased amygdala reactivity to emotion was linked to both interpersonal/affective and lifestyle/antisocial psychopathy traits (Glenn et al., 2009), neither dimension (Contreras-Rodriguez et al., 2014; Harenski et al., 2010), while one study reported that

the lifestyle/antisocial component of the PCL-R was specifically predictive of diminished amygdala activity (Harenski et al., 2014).

Across the literature, there is a pattern that emotional processing deficits associated to psychopathy are driven primarily by interpersonal/affective psychopathy traits, and this is particularly evident for psychophysiological responses to emotion. However, this unique association has not been demonstrated consistently across tasks, which may reflect the diversity of paradigms and emotional stimuli employed. Notably, the interpersonal/affective dimension of psychopathy has been more frequently linked to impairments in response to negative stimuli rather than positive stimuli, consistent with a dual pathway model that emphasises that the interpersonal/affective dimension of psychopathy reflects an insensitive defensive motivational system. However, much of the research has examined only negative and neutral stimuli, meaning that the findings cannot be interpreted in relation to positive stimuli.

1.3.4 Passive-viewing of emotion in psychopathy

The autonomic nervous system is responsible for the control of involuntary and visceral bodily functions, such as cardiovascular, digestive and respiratory functions. There are two systems involved within the autonomic nervous system: the sympathetic and the parasympathetic systems. The sympathetic nervous system is responsible for mobilising the ‘fight or flight’ response, for example increasing heart rate, sweat responses and breathing, whilst the parasympathetic nervous system, often termed the ‘rest and digest’ system, controls vegetative actions such as digestion, sexual arousal and salivation. The two systems work in compliment to one another. Real life events or symbols of these events such as images or sound-clips that are perceived as emotive can trigger changes in autonomic activity, specifically an increase in sympathetic activity and a decrease in parasympathetic activity, thought to reflect mobilisation for action (Lang & Bradley, 2010). Furthermore, the degree of autonomic responsivity is considered

to be an indicator of the sensitivity of an organism's motivational system, with reactivity to negative cues reflecting defensive motivational activation and reactivity to positive cues reflecting appetitive motivational activation (Lang & Bradley, 2010). Therefore, measuring an individual's autonomic reactivity to negative and positive stimuli can inform understanding of their underlying motivational processes, with lower reactivity indicating a less sensitive defensive motivational system (in response to negative cues) or appetitive motivational system (in response to positive cues) (Fowles, 1980; Levenston et al., 2000).

Despite a pattern that interpersonal/affective psychopathy traits predominantly drive the emotional processing deficits associated to psychopathy, particularly for psychophysiological reactivity to emotion (see 1.3.3 The dimensions of psychopathy and emotional processing), empirical research exploring autonomic responsivity during the passive-viewing of emotion has presented equivocal findings with the dimensions of psychopathy receiving relatively scant attention. For simplicity, the current thesis refers to 'passive-viewing' for the exposure of both visual and auditory stimuli to an individual. Additionally, the literature has also tended to use the terms 'arousal' and 'responsivity' interchangeably; the current thesis will use the term 'responsivity' when describing autonomic reactivity to emotion. The majority of studies have measured SCR and so this autonomic measure will be focused on in the current section.

Several studies have identified that psychopathy is associated with reduced autonomic reactivity to specifically negative stimuli. Benning, Patrick, and Iacono (2005) measured electrodermal responses to negative (e.g. mutilated bodies and disease images), positive (e.g. erotic and adventure images) and neutral images (e.g. household objects) within a male community sample. The positive images were rated as more emotionally arousing by the participants and this was reflected in greater SCRs to positive stimuli across the sample. The Fearless Dominance dimension of the PPI-R was associated with smaller emotional modulation of SCR to negative images (compared to

neutral images), with the Impulsive Antisociality dimension unrelated. Neither dimension was related to SCR to positive images (compared to neutral images). While this initially appears to indicate a deficit specific to negative images, an alternative explanation is that impaired emotional responsivity only occurs at lower levels of emotional arousal as the negative images produced lower SCR magnitudes for the sample (including low psychopathy groups). Therefore, it is difficult to draw firm conclusions regarding the nature of autonomic hypo-responsivity to negative and positive images. Additionally, the Impulsive Antisocial scale was negatively associated with overall response magnitudes to all images (including affectively neutral stimuli) suggesting an overall hypo-responsivity associated to the lifestyle/antisocial component of psychopathy. Blair et al. (1997) and Blair (1999) reported a similar specifically negative deficit for adults high in psychopathy (defined using a PCL-R total threshold of 30) and children with high callous/unemotional traits although this hypo-responsivity was specific to 'distress' images (e.g. crying children/adults) with normal electrodermal responses to 'threatening' (e.g. threatening animals and pointed guns) and neutral images.

Further research has found autonomic hypo-responsivity to the presentation of emotion associated to psychopathy, but this impairment was observed across both negative and positive stimuli. To illustrate, Verona, Patrick, Curtin, Bradley, and Lang (2004) presented 68 male inmates with emotionally arousing (and neutral) sound-clips, consisting of a laughing baby, erotic moan and a crowd cheering for the positive stimuli and a crying baby and people being attacked for the negative stimuli. It was found that offenders scoring high on Factor 1 of the PCL-R (interpersonal/affective) had attenuated SCR for negative and positive sound-clips (compared to neutral sound-clips), as well as reduced overall response magnitudes to all stimuli. However, only three sounds were played to participants (each repeated three times) and so it could be argued that the effects were specific to this small stimuli set.

Bate, Boduszek, Dhingra, and Bale (2014) reported that the primary psychopathy scale of the LSRP within a male and female undergraduate sample was negatively predictive of an 'emotional response' measure (SCR to negative and positive images combined) whilst the secondary psychopathy dimension was positively predictive of this emotional response. However, this effect only occurred for participants lower in intelligence. At high levels of intelligence there was no relationship between primary and secondary psychopathy dimensions and SCR to emotional images. However, as negative and positive images were combined it is difficult to interpret whether autonomic hypo-responsivity was driven by a specific valence or both valences. Additionally, the authors have not defined how the emotional response was calculated meaning that it is not clear whether this emotional response reflects overall response magnitudes to affective stimuli or the difference in SCR between affective and neutral images. Therefore, an overall deficit in electrodermal responses (including to neutral images) cannot be ruled out.

Additionally, Zimak, Suhr, and Bolinger (2014) administered the short-form of the PPI-R to a sample of undergraduate men categorised into high and low psychopathy groups based on thresholds consistent with previous literature. Participants in the high psychopathy group demonstrated lower SCR to both negative and positive images. Again, however, an overall response deficit cannot be ignored as no neutral images were presented to the participants for comparison. Exploratory statistical analysis revealed that neither dimension was related to SCR to negative images (no likewise analysis was run for positive images) although this procedure was only conducted within the high psychopathy group limiting the interpretation (and statistical power) of the analysis.

Levenston et al. (2000) reported evidence that autonomic response deficits in relation to psychopathy exist specifically in response to positive affect. Male offenders were categorised into either high psychopathy (PCL ≥ 30) or low psychopathy groups (PCL-R ≤ 20) and viewed negative (either mutilation, assault or threatening), positive (either

erotic or thrilling) and neutral images. Both groups demonstrated normal increased modulation of SCR for negative and positive images compared to neutral images. However, when the images were broken down into specific sub-types, the high psychopathy group showed diminished emotional modulation of SCR specifically to thrill images (e.g. rollercoasters, ski-jumping, cliff diving) compared to neutral images. The factors of the PCL-R were not explored individually.

As previously described, both Benning, Patrick, and Iacono (2005) and Verona et al. (2004) have reported associations between the dimensions of psychopathy and *overall* autonomic hypo-responsivity (as well as specifically emotional impairments). Several further studies have reported that overall psychopathy was related to autonomic hyporesponsivity that was not specific to emotional stimuli. Sutton et al. (2002) explored electrodermal to negative, positive and neutral images within a sample of 172 female offenders using the PCL-R. High psychopathy (PCL-R ≥ 30), intermediate (PCL-R 20 – 30) and low psychopathy groups (PCL-R ≤ 20) were defined using the cut-offs outlined in Hare (1991). There was a main effect of group, but no group by valence interaction indicating that the high psychopathy group showed smaller SCR overall magnitudes compared to the intermediate and low psychopathy groups across all images. Further dimensional analyses revealed that overall psychopathy was predictive of smaller response magnitudes to unpleasant images (neither Factor 1 nor 2 was independently significant), while Factor 1 was negatively predictive of response magnitudes to neutral images. No analyses in relation to the PCL-R factors were reported for pleasant images.

Similarly, Pastor et al. (2003) presented negative, positive and neutral images to offenders categorised into high psychopathy (PCL-R ≥ 27), intermediate (PCL-R 19 - 27), and low psychopathy groups (PCL-R < 19), and measured electrodermal. The high psychopathy and the intermediate groups showed smaller SCR magnitudes than the low psychopathy group, but there was no group by image valence interaction suggesting autonomic hypo-responsivity regardless of emotionality in relation to psychopathy. The

individual correlates of the PCL-R factors were not explored so the conclusions are limited to overall psychopathy.

Additionally, Herpertz, Werth, et al. (2001) reported smaller overall electrodermal response magnitudes in response to negative, positive and neutral images for a male forensic psychiatric group high in psychopathy (defined using the PCL:SV) compared to low psychopathy patients and a control group. However, again, the dimensions of psychopathy were not explored individually. These findings should be treated with caution as the low psychopathy groups consisted of forensic patients diagnosed with Borderline/Emotionally unstable personality disorder, which is associated with hyper-emotionality (Donegan et al., 2003; Herpertz, Dietrich, et al., 2001; Mitchell, Dickens, & Picchioni, 2014; Weinberg, Klonsky, & Hajcak, 2009), as well as a non-offender/non-psychiatric control group who are likely to differ vastly on a range of factors (e.g. previous mental illness, previous substance abuse) although the authors matched for intelligence and education.

Further research has failed to observe that psychopathy was associated with abnormal autonomic response to the presentation of affective stimuli. Patrick et al. (1993) reported that psychopathy in a sexual offender population was unrelated to SCR in response to negative, positive and neutral images. Three psychopathy groups were defined: High psychopathy (PCL-R ≥ 30), intermediate (PCL-R 20 - 30) and low psychopathy groups (PCL-R ≤ 20). There was no difference between groups for electrodermal reactivity to images across valences. The individual correlates of the factors were not explored.

Additionally, Ragsdale, Mitchell, Cassisi, and Bedwell (2013) presented negative (distress and threatening) and neutral images to a male and female university sample measuring psychopathy through the PPI-R. Both PPI-R factors and overall PPI-R scores were unrelated to SCR to emotionally arousing and neutral images. However, in contrast

to Ragsdale et al. (2013), the majority of previous studies that have identified hypo-responsivity as a function of psychopathy within community populations have pre-screened larger pools of participants to include individuals high in psychopathy traits (Benning, Patrick, & Iacono, 2005; Zimak et al., 2014). This enhances the opportunity to identify psychopathy-related effects.

Overall, while there is some evidence that interpersonal/affective psychopathy traits are uniquely associated to autonomic hypo-responsivity during passive-viewing of emotion (Bate et al., 2014; Benning, Patrick, & Iacono, 2005; Verona et al., 2004), much of the empirical literature has not investigated the independent contribution of the psychopathy dimensions. Additionally, the interpretation of much of the previous research is limited by methodological problems, such as limited stimuli sets and not presenting positive or neutral stimuli. It is also notable that both Benning, Patrick, and Iacono (2005) and Verona et al. (2004) measured the difference in autonomic reactivity between affective and neutral stimuli to identify that interpersonal/affective psychopathy traits were related to autonomic hypo-responsivity to emotion. This measure may be more sensitive than indexing overall autonomic response magnitudes for identifying diminished emotional modulation. In addition, the evidence that psychopathy (and its dimensions) is related to diminished overall autonomic responsivity is not convincing with small effect sizes identified (Brook, Brieman, & Kosson, 2013). It would also be useful to explore autonomic responsivity to neutral stimuli directly to ensure that findings of diminished overall autonomic responses are not being driven by decreased reactivity to emotional stimuli. Given the contrasting literature, it would be informative to comprehensively explore autonomic responsivity to negative and positive stimuli as a function of psychopathy to improve the understanding of defensive and appetitive motivational systems within psychopathy.

1.3.5 Evidence for theories of emotional processing in psychopathy

While the majority of studies report that psychopathy is associated to diminished responsiveness to emotional cues, the nature of this impairment has varied. Across the literature, empirical research has reported that psychopathy is associated to an emotional impairment across a range of valences (including positive stimuli) (Bagley et al., 2009; Baskin-Sommers et al., 2013; Bate et al., 2014; Blair, Richell, et al., 2006; Contreras-Rodriguez et al., 2014; Dolan & Fullam, 2006; Hastings et al., 2008; Mitchell et al., 2006; Seara-Cardoso et al., 2012; Verona et al., 2004; Williamson et al., 1991; Zimak et al., 2014), although specific or more severe deficits are identified in response to negative stimuli (particularly fearful stimuli) (Baskin-Sommers et al., 2011; Benning, Patrick, & Iacono, 2005; Birbaumer et al., 2005; Blair et al., 2004; Blair et al., 2002; Dindo & Fowles, 2011; Esteller et al., 2016; Flor et al., 2002; Han et al., 2012; Ishikawa et al., 2001; Justus & Finn, 2007; Kiehl et al., 2001; López et al., 2013; Medina et al., 2016; Montagne et al., 2005; Newman et al., 2010; Ogloff & Wong, 1990; Pastor et al., 2003; Patrick et al., 1993; Patrick et al., 1994; Sadeh & Verona, 2012; Snowden et al., 2013; Stanković et al., 2015; Vaidyanathan et al., 2011; Vaidyanathan et al., 2009; Venables et al., 2015; Wang et al., 2012). This pattern of findings partially supports both a general emotional deficit perspective and a low-fear perspective (Brook et al., 2013). Additionally, there are several studies that have reported that psychopathy is associated with impairments in processing specifically distress cues (fear/sad) (Blair, 1999; Blair et al., 2001; Blair et al., 1997; Bowen et al., 2013; Decety et al., 2013; Dolan & Fullam, 2006; Seara-Cardoso et al., 2013) consistent with the VIM model. However, this diverging pattern of findings across the literature has failed to convincingly corroborate the position of general or specific emotional deficit perspectives (see Brook et al., 2013 for a review). Additionally, while psychopathy has been more frequently associated with impairments in processing and responding to negative stimuli, opposed to positive stimuli, it is important to note that much research has failed to present positive stimuli.

Further, there is evidence that psychopathy is associated with deficits in conditioning reflective of impaired stimulus-reinforcement encoding and consistent with both the IES and the DAAM. To illustrate, individuals high in psychopathy showed deficient aversive conditioning (Birbaumer et al., 2005; Flor et al., 2002; Rilling et al., 2007) and impaired passive avoidance learning (Newman & Kosson, 1986). However, the IES has been challenged by research that has found that attention moderates the association between psychopathy and impairments in processing emotion consistent with the RMH.

Children with callous/unemotional traits show deficits in the recognition of fearful facial expression due to a failure to pay attention to the eyes, yet this impairment can be alleviated by explicitly directing the children's attention towards the eyes (Dadds, El Masry, Wimalaweera, & Guastella, 2008; Dadds et al., 2006). Similarly, Newman et al. (2010) demonstrated that offenders high in psychopathy showed deficient fear-potentiated startle response while categorising coloured letter stimuli when asked to respond to threat-irrelevant aspects of the stimuli (e.g. the case of the letter), but showed normal fear-potentiated startle under conditions where they categorised the threat-relevant aspects of the stimuli (e.g. the colour of the letter). This suggests that psychopathy is not related to a persistent insensitivity to emotion (or specific emotions), but rather affective deficits occur as results of an attentional impairment. Baskin-Sommers et al. (2011) similarly found that affective deficits in relation to psychopathy could be moderated by varying the focus of attention, but only if the threat cues were presented after alternative goal-directed focus was established. That is, high psychopathy individuals showed normal aversive conditioning, reflected in augmented fear-potentiated startle responses, if the threat-relevant information (i.e. the colour of the letter) was presented early, regardless of their attentional focus. They interpreted this as evidence of an early attentional bottleneck that blocks the processing of secondary information consistent with the RMH.

Further evidence for the role of attention in psychopathy comes from research that indicates that individuals high in psychopathy show attenuated early negative-potentiated startle responses, but comparable responses to individuals low in psychopathy at later stages reflecting delayed processing (Levenston et al., 2000; Sutton et al., 2002). Studies have also reported that psychopathy is related to attenuated startle reflex modulation to negative images, but only for images that require greater perceptual demands suggesting that this attentional bottleneck can be overcome through lowering the perceptual processing demands of a task (Baskin-Sommers et al., 2013; Sadeh & Verona, 2012). These studies indicate that psychopathy is associated to impairments in responding to emotion, but this is as a result of higher-order attention-emotion interaction. Notably these studies only identified deficits to negative stimuli in relation to psychopathy, although again the majority failed to present positive stimuli.

The DAAM similarly predicted that psychopathy was related to emotional insensitivity through an attentional impairment, but rather than an attentional bottleneck, they proposed that psychopathy was associated with a deficient ability to reflexively shift attention to salient emotional features caused by an under-active BLA (see 1.3.1 Theories of emotional processing). Moul et al. (2012) interpreted the above-described research (affective deficits associated to psychopathy can be alleviated by directing attention towards the emotion) as evidence of a top-down mechanism overcoming impairments in the BLA-related reflexive gaze shift. Additionally, in support of the DAAM, Boccardi et al. (2011) reported that high psychopathy offenders (PCL-R scores were 21 – 40) showed smaller BLA volume and increased CeA volume compared to matched control participants. Further support for this model comes from animal studies where lesions of the BLA leads to an impaired startle response to a conditioned stimulus (Walker, Paschall, & Davis, 2005), the cessation of conditioned fear responses (McDannald & Galarce, 2011), an inability to alter their initial representations of outcome values (Hatfield, Han, Conley, Gallagher, & Holland, 1996), as well as producing

behavioural changes associated to psychopathy such as impulsivity (Winstanley, Theobald, Cardinal, & Robbins, 2004). However, Moul et al. (2012) identified that the predictions of the DAAM have yet to be tested thoroughly, although advances in technical resolution and specificity should make it possible to isolate the functional contributions of the specific sub-nuclei of the amygdala.

Overall, the results have failed to convincingly substantiate any of the perspectives of emotional processing for psychopathy, with each model receiving partial support. The findings suggest that, although psychopathy has been associated to impairments to both negative and positive stimuli, psychopathy has been more frequently associated to an insensitivity to negative cues, particularly fear in support of low-fear models. Additionally, recent studies have recognised that psychopathy may be associated to deficits in processing emotion through an attentional impairment, recognising the contribution of the RMH and DAAM that do not assume a universal insensitivity to emotion (or specific emotions). In particular, I suggest that the DAAM offers the greatest potential as a model to unify both the emotional and attentional deficits observed for individuals high in psychopathy traits, given that the perspective focuses on amygdala dysfunction (specifically the BLA) offering an explanation for greater deficits to negative stimuli as well as the role of attention.

1.4 Pupillometry

Most studies of psychopathy have measured autonomic responses by indexing electrodermal or cardiovascular reactivity, but pupillometry offers an objective measure of autonomic responsivity that is fast, easy to administer and non-invasive (no wires are attached to the participant). The predominant influence on pupil diameter is luminance, but increasingly researchers have recognised that the pupil also responds to psychological factors. Just and Carpenter (1993) suggested that the pupil can index the intensity with which the cognitive system is operating as pupil size was found to increase

with increasing cognitive load (Hess & Polt, 1964; Kahneman & Beatty, 1966). Additionally, the pupil is responsive to emotion with stimuli perceived to have emotional content leading to greater pupil diameter compared to emotionally neutral stimuli (discussed later in 1.4.2 The influence of emotion on the pupil).

1.4.1 Physiology of the pupil

The pupil is a hole located in the centre of the iris. It can vary in size from 1.5 mm in bright light to 9 mm in darkness (Lowenstein & Loewenfeld, 1962) from an average of 3 mm (Wyatt, 1995). Changes in pupillary size occur spontaneously and are impossible to suppress at will, whether it is provoked by external stimuli or mental events (Loewenfeld, 1993). It has a contractile structure consisting of two muscles groups controlled by opposing divisions of the autonomic nervous system: a circular group called the sphincter (or constrictor) muscle, which is innervated by the parasympathetic nervous system, and the radially-arranged dilator muscle group that is activated by the sympathetic nervous system. The dynamic 'push-pull' between the activities of these two iris muscles determines pupil diameter. Hence, pupil dilation can occur through both excitation of the sympathetic division or inhibition of the parasympathetic system, with the opposing activity leading to pupil constriction (Bradley, Miccoli, Escrig, & Lang, 2008; Steinhauer, Siegle, Condray, & Pless, 2004; White & Depue, 1999).

The locus coeruleus is a subcortical structure that acts as the major nucleus for the release of norepinephrine (also called noradrenaline), a catecholamine with a central role in response to stress. The locus coeruleus plays a central role in arousal and is responsible for autonomic regulation through both sympathetic and parasympathetic outflow (Szabadi, 2012). It contributes to changes in pupil diameter through two pathways, both involving norepinephrine functioning (White & Depue, 1999). The first route, mediated through posterior hypothalamic nuclei, is reflective of sympathetic activity and leads to phasic pupil dilation through norepinephrine acting upon alpha-1

postsynaptic receptors located directly on the dilator muscle group (Loewenfeld, 1993). The second route to pupil dilation is mediated by inhibition of the Edinger-Westphal nucleus (EWN), located in the midbrain, which is the parasympathetic pre-ganglionic nucleus. Even in the absence of stimulation the EWN sends tonic innervation signals to the sphincter muscles leading to pupil constriction, but this ongoing constrictive signal can be interrupted through cortico-limbic pathways sensitive to a variety of cognitive and emotion processes leading to pupil dilation (White & Depue, 1999). Indeed, pupil diameter is closely related to the activation of locus coeruleus (Alnæs et al., 2014; Koss, 1986; Rajkowski, Kubiak, & Aston-Jones, 1993) and, as such, can be considered as an indicator of ongoing autonomic responsivity.

1.4.2 The influence of emotion on the pupil

Hess and Polt (1960)'s influential study was the first to employ pupillometry to study emotional influences by showing male and female adult participants images of nudes, babies and a neutral landscape while measuring pupil size. It was found that the pupil was larger in response to positive images, but smaller in response to negative images. A second study was conducted by Libby, Lacey, and Lacey (1973) where they showed 34 male participants 30 images (across negative, positive and neutral valences) and measured participants' pupil diameter in response. Their findings were a little confusing as, although they found that increased pupil dilation was associated with 'attention-grabbing' images that loaded onto emotionality, they also found that pupil dilation was greatest to neutral pictures compared to negative and positive images. Aboyoun and Dabbs (1998) presented pictures of nude and clothed males and females to a mixed-gender undergraduate population. In contrast to Hess and Polt (1960) it was found that nude images led to greater pupil diameter compared to clothed images regardless of gender (of the participant or the image). However, these early studies were limited by methodological problems, such as small sample sizes, few images and unclear luminance control.

A seminal study by Bradley et al. (2008) assessed the effects of subjective image valence and arousal on pupillary responses using a well-validated large set of images selected from the International Affective Picture System (IAPS; Lang, Bradley & Cuthbert, 2008). They presented 27 university students with 96 images consisting of negative, positive and neutral pictures for six seconds. They matched the negative and positive images for subjective arousal (based on the accompanying normative ratings of the stimuli). They found that pupil diameter, following the pupil light reflex (PLR), was larger to emotionally arousing images regardless of whether these were negative or positive. These basic findings have now been replicated on many occasions in response to emotionally arousing images (Arriaga et al., 2015; Babiker, Faye, Prehn, & Malik, 2015; Bradley & Lang, 2015; Geangu, Hauf, Bhardwaj, & Bentz, 2011; Henderson, Bradley, & Lang, 2014; Snowden et al., 2016; Van Steenbergen, Band, & Hommel, 2011), static and morphed facial expressions (Burkhouse, Siegle, Woody, Kudinova, & Gibb, 2015; Prehn, Schlagenhauf, et al., 2013), as well as video-clips depicting affect (Geangu et al., 2011; Rieger & Savin-Williams, 2012; Rosa, Esteves, & Arriaga, 2015). Additionally, the magnitude of the PLR has also been found to be reduced (i.e. larger pupil diameter) in response to emotional stimuli (Henderson et al., 2014). Therefore, pupil responsivity is considered to index emotional arousal rather than the hedonic valence of stimuli (Bradley et al., 2008).

Research has also explored pupil diameter to non-visual stimuli, which is useful as it removes visual influence on the pupil. Dabbs (1997) played four sound-clips (sexual, aggressive and two neutral) lasting 30-seconds to an undergraduate sample while their pupil responses were measured. The sexual sound-clips led to the largest pupil diameter, with no difference between the remaining non-sexual stimuli. However, the generalisability of these findings to each valence may be limited considering that only one sound was presented per category. Partala and Surakka (2003) improved on this study by exploring the effect of sound-clip emotionality using a large sample of well-

validated sound-clips from the International Affective Digitised Sounds (IADS; Bradley & Lang, 2007). Thirty-one community participants were presented with 30 sound-clips (10 negative, 10 positive and 10 neutral) with the emotional sounds matched for subjective arousal according to the accompanying normative ratings. Each sound-clip lasted for six seconds. The results paralleled those of Bradley et al. (2008) as larger pupil dilation was observed to emotionally arousing sound-clips regardless of hedonic valence compared to neutral sound-clips. However, it is notable that neither Dabbs (1997) nor Partala and Surakka (2003) matched across sound-clips for decibel levels and, therefore, the results may simply reflect differences in perceived sound-levels (Liao, Kidani, Yoneya, Kashino, & Furukawa, 2016). Despite stimuli difficulties, there is evidence that emotional sound-clips lead to increased pupil diameter compared to neutral stimuli likewise to the above-reported visual stimuli.

1.4.3 The sympathetic and parasympathetic contribution to pupil reactivity to emotion

Pupil diameter is considered to reflect the ongoing activity of the sympathetic and parasympathetic nervous system. At the onset of an image or video-clip there is typically a pupillary light reflex (PLR) that is considered to be a response to the visual attributes of a stimulus (Barbur, 2004); the specific causes of the PLR are discussed in the next chapter (see 2 Chapter 2: Visual and psychological parameters that can affect pupil diameter). It is largely accepted that the parasympathetic system is primarily responsible for the PLR in response to increases in light flux (Lowenstein & Loewenfeld, 1950). One method to explore this is through the topical administration of pharmacological agents that diffuse through the cornea to the iris either blocking dilatory or constrictor muscles, thus isolating either the sympathetic or parasympathetic contribution. Steinhauer, Condray, and Pless (2015) found that tropicamide, which blocks the sphincter muscles, abolished the PLR completely, while dapiprazole, which blocks the dilator muscles, did not affect the PLR, indicating that the parasympathetic system is primarily responsible

for the PLR. Henderson et al. (2014) demonstrated that the magnitude of the PLR could be attenuated through the introduction of emotion. However, it is not clear whether this effect occurred through an increase in sympathetic activity or a reduction in parasympathetic activity (or a combination of both), as no study has explored the role of the autonomic nervous system on the PLR to more complex visual stimuli to the author's knowledge.

Most studies that are interested in measuring pupil reactivity to emotive visual stimuli index pupil diameter following the PLR, and research indicates that pupil reactivity over this period reflects sympathetic activation. Bradley et al. (2008) reported that increases in pupil diameter to emotional images measured from 2 - 6 seconds (during a six second presentation) were positively related to SCR, a sympathetically-mediated process (Wallin, 1981), while showing no relationship to heart-rate, mediated primarily by parasympathetic activity (Berntson, Boysen, Bauer, & Torello, 1989). Bradley et al. (2008) concluded that pupillary changes in response to emotion reflect emotional arousal associated with increased sympathetic activity. Although not directly related to emotional changes in pupil diameter, Lowenstein and Loewenfeld (1950) reported that the recovery of the pupil following a PLR takes place in two stages predominantly reflecting sympathetic contribution: the initial redilation which is rapid and reflects both SNS activation and PNS withdrawal, while the later phase reflect predominantly SNS. Indeed, Smith and Smith (1999) found that redilation following the PLR to light pulses primarily reflects sympathetic activation by studying individual with bilateral Horner's Syndrome, which is a disorder where the individual's sympathetic trunk is damaged. It was found that pupil reactivity following the PLR, specifically the time taken to reach 75% of pre-PLR pupil size, was highly predictive of these individuals as they showed slower recovery times. This suggests that pupil diameter following the PLR is predominantly influenced by sympathetic activation.

However, despite this evidence, the current thesis is careful to not make any claims regarding the underlying contribution of the autonomic nervous system to pupil diameter, given the uncertainty of the mechanisms that underlie emotional modulation of the PLR. Rather, the primary concern of this thesis is indexing emotional modulation through changes in pupil diameter and to apply this paradigm to explore psychopathy, as has been applied to investigate alternative clinical disorders, such as depression and autism spectrum disorders (Burkhouse et al., 2015; Jin, Steding, & Webb, 2015; Kuchinke, Schneider, Kotz, & Jacobs, 2011; Nuske, Vivanti, Hudry, & Dissanayake, 2014; Sepeta et al., 2012; Siegle, Steinhauer, Friedman, Thompson, & Thase, 2011; Steidtmann, Ingram, & Siegle, 2010).

1.5 Chapter outline

Psychopathy has been frequently associated to a deficit in processing emotion, particularly negatively valenced stimuli, which has been more typically linked to the interpersonal/affective dimension of psychopathy. However, many of these studies failed to present positive stimuli limiting the conclusion drawn. Empirical evidence for autonomic responsivity during passive-viewing of emotion has been equivocal in relation to psychopathy, although much previous research has failed to convincingly investigate this question. The present thesis aimed to correct these issues to explore autonomic responsivity to a comprehensive set of emotional (negative and positive) images, static facial expressions, dynamic facial expressions and sound-clips as a function of psychopathy. This would inform the understanding of the sensitivity of the underlying defensive and appetitive motivational systems within psychopathy (Fowles, 1980; Levenston et al., 2000). Additionally, the current thesis adopted a multi-faceted approach to explore the independent contribution of the underlying dimensions of psychopathy. Autonomic responsivity to emotion was measured through changes in pupil diameter, given that pupillometry is advantageous as a fast, relatively cheap and non-intrusive measure of autonomic responsivity. Additionally, pupillometry has not been previously

applied to explore psychopathy, so the current research represented a novel application of the paradigm.

Chapter 2 describes how several visual and psychological factors were examined to explore their influence on pupil diameter, in order to aid the development of a paradigm capable of accurately measuring emotional modulation of the pupil. Chapter 3 then explored pupil responsivity to the passive-viewing of negative and positive stimuli across visual and auditory domains as a function of psychopathy within an undergraduate population. Chapter 4 similarly investigated pupil responsivity to the passive-viewing of emotional stimuli as a function of psychopathy, but within a forensic psychiatric sample where levels of psychopathy were greater. Chapter 5 then discusses the practical and theoretical implications of the current findings.

2 Chapter 2: Visual and psychological parameters that can affect pupil diameter

Abbreviation	Meaning	Page
MCMC	Markov Chain Monte Carlo	67
PLR	Pupil light reflex	43

2.1 Introduction

Chapter 2 focused on the development of a paradigm capable of accurately measuring emotional modulation of the pupil that could be later applied to investigate autonomic responsivity to emotion in relation to psychopathy. The pupil increases in size in response to emotional stimuli compared to neutral stimuli (see Chapter 1, Pupillometry and emotion). In response to visual stimuli, this emotional modulation causes larger pupil diameter during the pupil light reflex (PLR) (i.e. an attenuated PLR) (Henderson et al., 2014) and then larger pupil diameter following the PLR as the pupil dilates/recovers (Bradley et al., 2008). The current thesis was interested to index pupil diameter to visual stimuli after the PLR as this is thought to represent emotional arousal in response to the stimuli (Bradley et al., 2008). Despite this index, it was important to consider visual influences that may moderate the magnitude of the PLR as this has a clear and direct influence over subsequent pupil diameter. To date, the majority of studies exploring moderators of the PLR have employed simple images (e.g. sinusoidal gratings or a single patch of light), but these findings may not extend to more complex visual images (e.g. natural scenes). Therefore, Experiment 1 - 3 examined physical characteristics of visual images (luminance, contrast and colour respectively) that may impact upon the PLR. Only visual stimuli was explored as auditory stimuli does not trigger a PLR. We intended to use dynamic facial stimuli in future research, but I focused on complex visual images given the large potential differences in luminance between stimuli. Furthermore, it was important to consider factors that may moderate the magnitude of emotional modulation of pupil diameter following the PLR; Experiment 4 and 5 explored how differing presentation methods may moderate emotional modulation of the pupil, specifically the duration of image presentation and whether pupil responses habituate to emotion.

The main determinant of pupil diameter is the amount of light entering the eye with increasing light leading to greater constriction. This change in pupil diameter to ambient light is relatively slow, and is termed the steady-state component, where pupil size is a

proportional reflection of the summation of all luminance signals over a large spatial area. However, previous research employing simple visual stimuli has demonstrated that the magnitude of the PLR is not solely moderated by luminance as a transient pupil constriction has been observed in response to flashes of darkness, isoluminant stimuli with increased contrast, or isoluminant colour flashes (Barbur, 2004; Clynes, 1961; Kohn & Clynes, 1969; Young, Han, & Wu, 1993). Therefore, a secondary transient process has been proposed that is responsive to the degree of change for local visual signals leading to pupil constriction even in the absence of luminance changes (Barbur, 2004; Clynes, 1961; Kohn & Clynes, 1969; Young et al., 1993). Barbur, Harlow, and Sahraie (1992) suggested that this transient rate-sensitive mechanism leads to pupil constriction through abrupt changes in neural input to the visual cortex causing a disruption of the inhibitory signal to the pupillometry nucleus, which allows for greater parasympathetic innervation of the iris sphincter muscle. Therefore, when a stimulus is viewed it will excite both the steady-state and the transient processes, with the exact characteristics of the stimuli governing the contributions of both systems. However, as mentioned, little research has directly considered whether the physical characteristics of more complex visual stimuli show similar effects on the pupil as seen to simple visual stimuli. An understanding of these influences was imperative if this thesis was to isolate changes in pupil diameter that are specific to emotion in future experiments. Therefore, Experiment 1 – 3 explored the influence of image luminance, contrast and colour respectively on the PLR for visual images. Experiment 4 and 5 considered how differing presentation methods may moderate emotional modulation of the pupil, specifically the duration of image presentation and whether pupil responses habituate to emotion.

2.2 General methods

2.2.1 Participants

All participants were recruited from Cardiff University; specific sample sizes and characteristics are reported within each experiment. Participants were asked to not wear bifocal or varifocal glasses on the day of testing as well as being requested to not consume caffeine or smoke 60-minutes prior to testing as this can influence the pupil response (Erdem et al., 2015; Wilhelm, Stuiber, Lüdtke & Wilhelm). All participants gave written informed consent to participate in the experimental procedures and were debriefed fully to the research aims. All experimental procedures were given ethical approval by Cardiff University.

2.2.2 Materials and design

The natural images reported across Experiment 1 – 5 were selected from a subset of images from the IAPS. The IAPS are accompanied by normative ratings for arousal and valence; these ratings were used throughout the thesis. Each image was rated from 1 – 9 on a Likert scale for subjective valence and arousal with higher ratings indicating more pleasant images and more arousing images respectively. Throughout the thesis, valence and arousal ratings are reported for images selected from the IAPS based on these normative ratings.

Images were manipulated using Adobe Photoshop Elements 12.0. Luminance was manipulated along the luminance scale 0 – 255 (the higher values indicating brighter stimuli). Image contrast was taken to be the standard deviation of all pixel luminance (Moulden, Kingdom, & Gatley, 1990) and was manipulated along a scale from 0 - 120 (higher values indicate greater contrast). The specific luminance and contrast values are reported for each experiment as there are slight variations. The target stimulus was preceded by a fixation screen presented for 2000 ms; this was a plain grey slide displaying a fixation cross composed of alternating light and dark grey pixels. A blank grey recovery screen was presented after the target image for 5000 ms to allow pupil diameter to return to baseline although a software error meant that

no data was recorded for the last 200 – 300 ms throughout each experiment. The luminance of the fixation and recovery screen were matched to the target image/s presented and the specific luminance is reported for each experiment. See Appendix 3 for a schematic illustration of the trial structure, which was used through the subsequent experiments.

2.2.3 Pupil data acquisition and cleaning

A Tobii X2-60 Hz eye tracker recorded pupil data throughout each task which allowed free movement of the head during the task. The hardware consisted of an eye-tracking device located below the computer monitor that captured eye movements by illuminating the pupil via an infrared light source and using two image sensors to record the reflection patterns. The eye tracker was calibrated to each participant's eyes before each task using a 5-point calibration screen. Pictures were displayed on a 48.30 cm display monitor with a resolution of 1920 x 1080 and each participant was seated 57 cm from the screen. The experiment took place in a dim, sound-proof experimental room at Cardiff University.

Data was cleaned and analysed using Matlab (MathWorks, version 8.5). I removed any pupil diameter increase or decrease of 0.0375 cm within one data reading (over a period of approximately 16.67 ms) as these are thought to be an artefact (Partala & Surakka, 2003). I also deleted the first data point that followed missing data to avoid abnormal readings. Data for each pupil was smoothed using a low-pass Savitzky-Golay filter (Savitzky & Golay, 1964) for a span of 5 readings (over an approximate period of 83.35 ms).

Pupil size was determined by calculating the mean diameter across both eyes. Initial pupil diameter for each trial was calculated over the period 200 ms prior to stimulus-onset (Leknes et al., 2012). For every trial, initial pupil diameter was subtracted from subsequent pupil size to establish baseline-corrected pupil diameter. Then mean baseline-corrected pupil diameter was calculated over time windows for each task; the

specific time windows and subsequent analyses are described in each experiment's method.

Trials were omitted if there was less than 50 % data across image presentation. In relation to Experiment 4 and 5 where emotion was introduced, participant means were only calculated for each image valence if there was valid data for at least 50 % of trials for that given valence. Participants were excluded if they recorded less than 50 % valid data across all trials during stimulus presentation. Participant means were identified as an outlier and removed if their data for a given valence was outside the interval defined as three times the interquartile range (Tukey, 1977). The dependent measures across experiments did not violate the assumption of normality as measured by Kolmogorov-Smirnov tests. Within each experiment, I applied a Greenhouse-Geisser correction where Mauchly's Test of Sphericity was violated and this is reported.

2.2.4 Effect sizes

The thesis describes a variety of statistical analyses and has reported effect sizes throughout to give an indication of the magnitude of the effect of an independent variable on a dependent variable (Lakens, 2013).

For correlational analyses, I reported the strength of associations using Pearson's *r* and for multiple linear regression analyses I reported standardised beta (β) for each association to assess the unique association.

I also assessed the impact of stimuli emotionality by running a number of analyses of variance (ANOVA). When conducting an ANOVA, eta squared (η^2) is one of the most widely-used effect sizes in research (Lakens, 2013). Eta squared represents the proportion of variability of the dependent measure that the independent variable accounts for, and can be interpreted in terms of percentage given that the entire effects of all portions of a model including error will total 1.00 (Levine & Hullett, 2002). Partial

eta squared (η_p^2) can be reported for ANOVAs where more than one independent variable is used to isolate the unique variance associated with that variable or to aid comparability of effect sizes across studies with similar design (Keppel, 1991; Olejnik & Algina, 2003). Therefore, the current research reports eta squared for one-way repeated measures ANOVAs and partial eta squared for ANOVAs with more than one independent variable.

Cohen's *d* (Cohen, 1977) is typically used to detail the standardised mean difference of an effect for comparison tests, which was reported for between-subjects comparisons. For repeated measures comparisons, Cohen's d_{av} (Cumming, 2013) aids comparability of effect sizes across experimental designs as it controls for the correlation between repeated measures (Lakens, 2013). However, the formula used to calculate Cohen's *d* gives a biased estimate of the population effect size (Hedges & Olkin, 1985), which can be corrected by applying Hedges' *g*. Therefore, I calculated Cohen's *d* for between-subjects comparisons and Cohen's d_{av} for repeated measures comparisons and applied Hedge's correction as recommended and described in Lakens (2013).

2.3 Experiment 1: Image luminance

As described previously, a transient pupil constriction has been observed in response to flashes of darkness (Clynes, 1961). This is explained by the idea that pupil diameter during the PLR is considered to be moderated by two processes, one responsive to the luminance of a stimuli that determines the steady-state size of the pupil and a secondary transient process that is reactive to the degree of change in local visual signals (Barbur, 2004). However, little research has examined whether the same pattern exists within complex visual images.

The present study was interested to examine whether changes in overall image luminance (relative to the preceding slide) for a complex visual image moderate the PLR likewise to simple stimuli. A single image was manipulated to be brighter, darker or to

hold equal luminance to the preceding slide and participant's pupil diameter was measured in response. Consistent with changes to the pupil observed in response to the luminance of simple stimuli, I expected that brighter images would lead to a greater PLR (smaller pupil diameter), but that the equal luminance image would also lead to a (smaller) PLR reflecting the secondary transient process responsive to local increases in light flux.

2.3.1 Method

Five participants (3 female) took part from Cardiff University aged 24 – 51 (M = 31.00, SD = 11.29). I selected a single neutral image (no. 2514) from the IAPS as it was subjectively rated as neutral and low-arousing (valence = 5.19, arousal = 3.5). The image was converted to grey-scale, and the luminance was manipulated to display five luminance variations (0, 63.75, 127.5, 191 and 255; see General methods for further information regarding the scale). I matched contrast for the 63.75, 127.5, 191 image variations to a value of 50 (it was not possible to match contrast for images with a luminance of 0 and 255 as these were plain black and white images). The fixation and recovery slide were manipulated to an overall luminance value matching the median image of 127.5. Each image was presented randomly on five occasions for 1000 ms, which was expected to be sufficient to explore the full PLR (Henderson et al., 2014). To quantify the magnitude of the PLR, I calculated mean baseline-corrected pupil diameter from 500 – 1000 ms post-image onset and ran a repeated measures ANOVA with luminance as an independent variable over 5 levels. Two-tailed significance values are reported throughout the chapter for all effects for simplicity.

2.3.2 Results

As can be seen in Figure 2.1, there was a pupil constriction at image onset with a latency of approximately 300 ms, which, unsurprisingly, increased in magnitude with greater image luminance. The brightest image (luminance value of 255) led to a large

constriction with a nadir of -1.35 mm, followed by sharp pupil dilation after image offset that slowed until it approached baseline size. A smaller constriction was observed for the image set at a luminance of 191.25 before pupil recovery that began prior to image-offset. Despite overall luminance being constant with the preceding fixation slide, images set at a luminance of 127.5 led to pupil constriction before recovery back to baseline pupil diameter beginning before image-offset. Despite the image set at a luminance of 63.75 showing an overall darker luminance compared to the preceding fixation slide, a slight constriction was initially observed before a rapid dilation that continued for 100 ms after stimulus offset, followed by a larger secondary constriction and subsequent recovery. Furthermore, the darkest slide (luminance value of 0) led to pupil dilation with a latency of 400 ms reaching a peak of 0.5 mm. However when this dark stimulus was removed there was a constriction of the pupil and recovery back to baseline pupil diameter. A repeated measures ANOVA (over five levels of luminance) revealed a main effect of luminance, F (4, 16) = 100.25, $p < .001$, $\eta^2 = .96$, with brighter images leading to significantly larger PLR (or smaller pupil diameter; $p < .05$), although the difference between the two darkest images was not significant ($p = .05$).

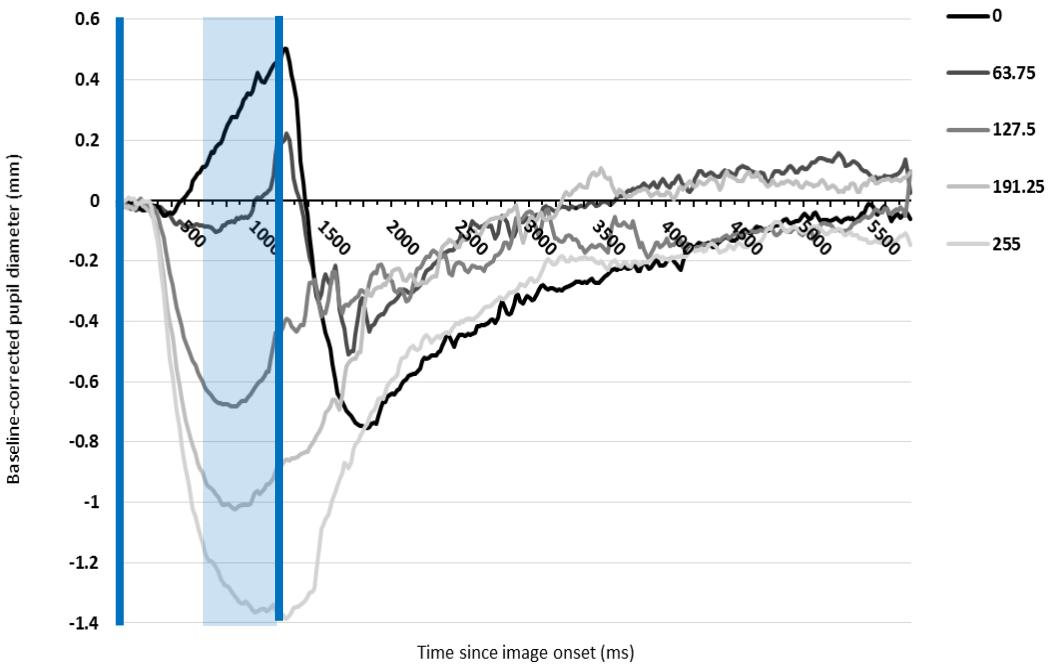


Figure 2.1 Baseline-correct pupil diameter in response to a neutral image of varying luminance. Blue bars indicate image-onset and offset and blue shading indicates the data analysis window (N = 5).

2.3.3 Discussion

As predicted, the results indicated that the PLR was increased for brighter natural images consistent with research using simpler stimuli (Barbur, 2004; Barbur, Wolf, & Lennie, 1998). This constriction response had a latency of approximately 300 ms consistent with Barbur et al. (1998). The constriction reached a nadir between 800 – 1000 ms and showed pupil recovery prior to or from image offset. The darker images showed a slower latency, with a dilation occurring later than constriction responses. A further response latency was evident at image offset for the darker images where the pupil continued to dilate for 100 – 200 ms after image-offset. The results indicate a process (the steady-state component) that is responsive to the overall luminance signals of an image consistent with research using simple stimuli.

As expected, the images demonstrated that luminance is not the only contributor to the PLR. Firstly, darker images led to a slight constriction prior to pupil dilation (possibly

accounting for the slower dilation responses), which would not have happened based on luminance alone. Moreover, a clear PLR was observed for images of matching overall luminance to the preceding slide, which would not be expected according to the overall summation of luminance signals. As mentioned, previous work using stimuli has similarly observed paradoxical pupil constrictions in response to flashes of darkness (Clynes, 1961), as well as to isoluminant increased contrast gratings (Barbur, 2004), suggesting a transient process that is sensitive to the degree of change in local visual signals leading to pupil constriction. The current experiment has provided evidence that the same processes may exist in response to more complex images. The following experiment explored this process through manipulating image contrast (the difference between the dark and the light parts of an image), while overall luminance was held constant.

2.4 Experiment 2: Image contrast

Experiment 1 highlighted that the pupil is not solely responding to the overall brightness of complex images. Indeed, Barbur (2004) previously demonstrated that the pupil is sensitive to changes in stimuli contrast, defined as the differences between the darkest and lightest parts of the image, for simple grating stimuli with larger pupil constriction seen for greater contrast. Therefore, despite an absence in a change of overall luminance, a constriction is observed, which is thought to reflect the transient component of the PLR. However, this has not been explored within natural images. The Michelson contrast expression (Michelson, 1927) has been typically used to measure contrast for simple patterns (e.g. gratings); this equation reflects the difference between the lightest and darkest parts of the grating divided by the sum of the lightest and darkest parts. However, definitions of image contrast are more difficult for natural images due to their greater degree of complexity. Moulden et al. (1990) reviewed various metrics for contrast, finding that the standard deviation of the luminance provides a useful metric for measuring contrast with images of random dot patterns. Hence, standard deviation of luminance was adopted as the index of image contrast and pupil diameter was measured

while participants viewed neutral images manipulated over seven contrast variations. I expected that greater contrast would cause greater PLR consistent with data for simple grating stimuli (Barbur, 2004).

2.4.1 Method

The same five participants as Experiment 1 took part in this experiment. Six neutral images were selected[1] from the IAPS that represented neutral subjective valence (M = 5.20, SD = 1.15) and low subjective arousal (M = 3.04, SD = 1.92). I converted the images to grey-scale and equalised across images for luminance (value of 95). Contrast was manipulated across images to the mean contrast of the images (value of 66), which acted as 100 % contrast stimuli. I created a further 6 contrast variations by manipulating these images to create images with contrasts of 50 %, 25 %, 12.5 %, 6.25 %, 3.13 % and 1.56 % of the original 100 % image. Luminance was kept constant across all images. Therefore, six images at seven contrast variations were presented to participants for 1000 ms each. Likewise to Experiment 1, I calculated mean baseline-corrected pupil diameter from 500 – 1000 ms post-image onset to quantify the magnitude of the PLR and ran a repeated measures ANOVA with contrast as an independent variable over 7 levels.

[1] Images selected were 2191, 2214, 1357, 2514, 7004 and 7080.

2.4.2 Results

As can be seen in Figure 2.2, the PLR begins at 300 ms post-image onset with the magnitude of the reflex ranging from approximately -0.05 to -0.80 mm depending on image contrast. The pupil constriction was initially rapid but slows through image presentation and for all images, apart from the 100 % image contrast, the pupil has begun to recover before image-offset with recovery back to baseline pupil size. It is evident that image contrast had a clear effect on the PLR with higher contrast leading to greater constriction magnitude.

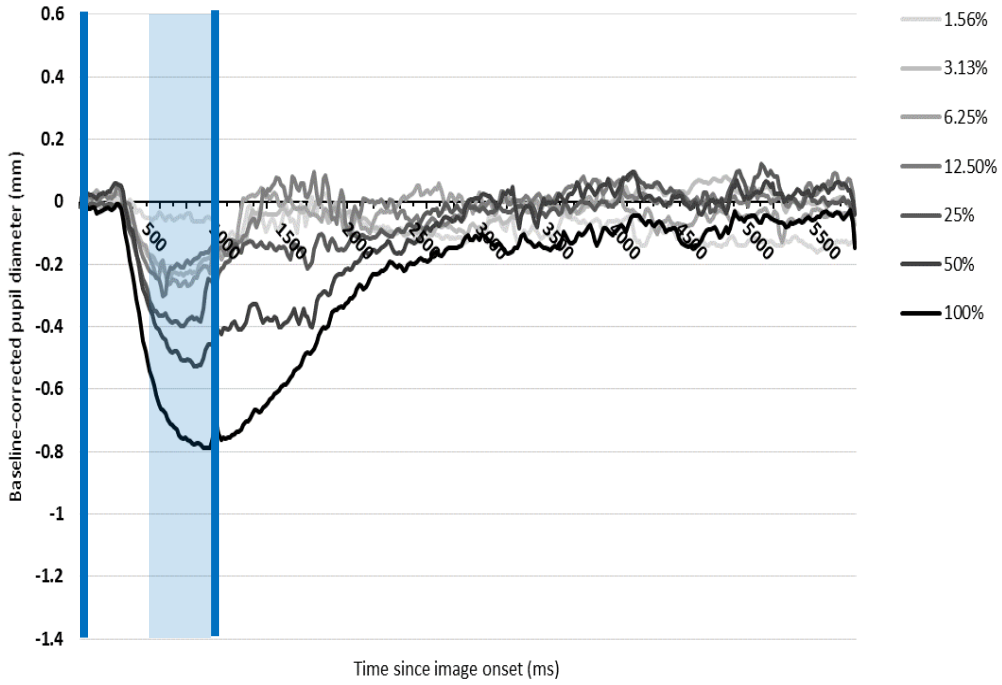


Figure 2.2 Baseline-correct pupil diameter in response to neutral images of varying contrast. Blue bars indicate image-onset and offset, and blue shading indicates the data analysis window (N = 5).

To quantify this, Figure 2.3 plots the response amplitude as a function of contrast calculated using the minimum baseline-corrected pupil diameter (maximum point of constriction) during image presentation. Images of the lowest contrast produced little PLR with the first noticeable pupil constriction occurring at 3.13 % contrast and with a gradual increase in PLR until 12.5 % contrast. The PLR then magnified (smaller pupil diameter) with increasing contrast in a linear manner with no hint of response saturation. A repeated measures ANOVA (over seven levels of contrast) identified a main effect of contrast, *F* (6,

24) = 18.59, $p < .001$, $\eta^2 = .82$. Planned comparisons revealed that differences between each contrast step were not significant (likely reflecting a lack of power, $p > .08$), apart from the 1.56 to 3.13 % contrast ($p = .04$) and the 50 to 100% contrast ($p = .04$), but significantly larger PLR (smaller pupil size) was observed in response to 22.5 %, 50 % and 100 %

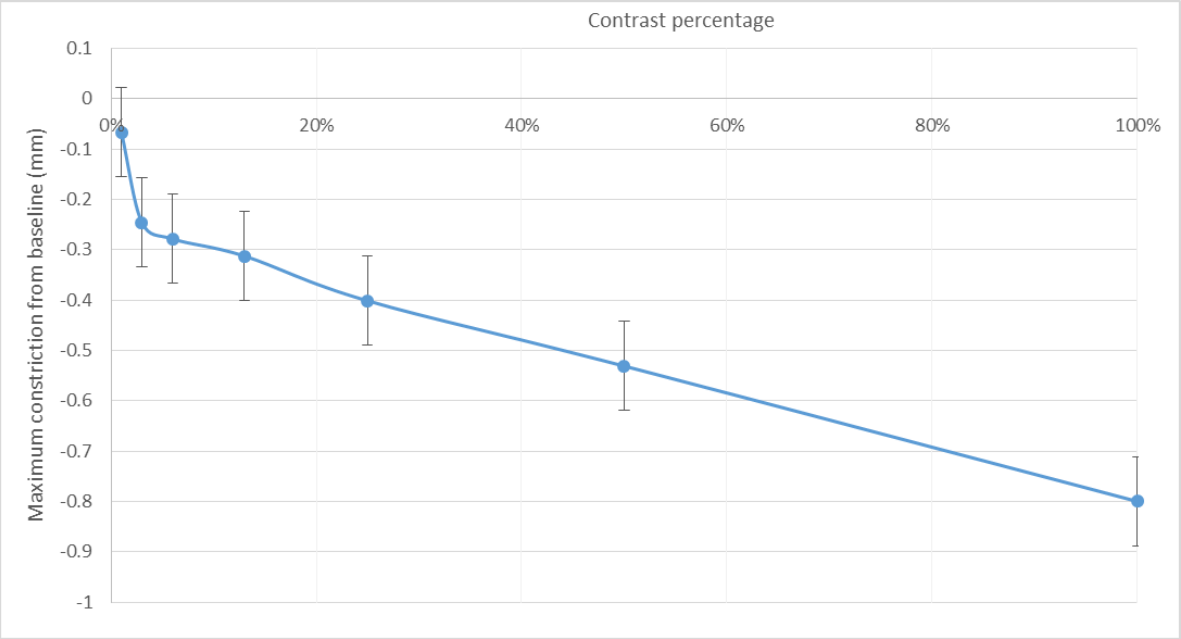


Figure 2.3 Baseline-corrected minimum pupil diameter (±1 SE) as a function of image contrast (N = 5).

contrast ($p < .05$) compared to lower image contrast.

2.4.3 Discussion

In line with prediction, the results clearly show that the contrast of natural images affected pupil diameter and was consistent with research using simple stimuli (Barbur, 2004). The magnitude of the PLR increased as image contrast increased. This finding can be linked back to the two underlying processes that contribute to pupil diameter in response to stimuli, with a transient process responding to the degree of change in local visual signals, before the steady-state process exerts a greater effect. Greater contrast leads to greater change in local light flux (despite overall luminance remaining the same) and, therefore, greater pupil constriction. Barbur et al. (1992) argued that abrupt changes in neural input to the visual cortex cause a disruption of the inhibitory effect to the

pupillometry nucleus, which allows for greater parasympathetic innervation of the iris sphincter muscle leading to the observed transient pupil constriction. It can be speculated that greater contrast causes greater disruption of the inhibitory effect leading to larger parasympathetic influence and the augmented PLR.

It was apparent that contrast had a significant effect on pupil diameter and, therefore, in future experiments image contrast was matched across valence; I continued to use the standard deviation of luminance to measure contrast (Moulden et al., 1990). It is acknowledged that there may be limitations to this approach given differences in local spatial structure within natural images that can affect the pupil (Binda, Pereverzeva, & Murray, 2013), but this method maintained the meaning of the image which was the primary focus of the thesis.

2.5 Experiment 3. Image colour

Changes in stimulus colour have been shown to lead to an altered PLR independent of light flux as colour saturation increases (Barbur et al., 1992; Barbur et al., 1998; Gamlin, Zhang, Harlow, & Barbur, 1998; Young et al., 1993). Barbur (2004) demonstrated that, despite comparable pupil response amplitude, the onset of isoluminant chromatic gratings led to longer pupil latency as well as an absence of the constriction seen at stimulus offset for the achromatic stimulus. However, again, this research has been conducted using simple stimuli and, therefore, the present experiment was interested to know whether chromaticity of natural images affects the PLR in the same manner. Chromatic images were compared to their achromatic equivalent and the PLR was measured in response. I hypothesised that the PLR would be of an equivalent magnitude across chromatic and achromatic images, but with a delayed latency for chromatic images consistent with data using simple stimuli.

2.5.1 Method

The same five participants recruited for Experiment 1 and 2 took part in this experiment. The same six neutral images that were used in Experiment 2 were presented again. I used the original colour images from the IAPS and also created a grey-scale version of each image. I manipulated all chromatic and achromatic images to match for luminance (value of 95) and contrast (value of 71). Each chromatic and achromatic image was presented five times for 1000 ms. The image continued to be preceded and followed by the grey fixation and recovery screen; these were luminance and contrast matched to the target images. The PLR was again quantified as mean baseline-corrected pupil diameter over 500 – 1000 ms post-image onset and a *t*-test was conducted between PLR to chromatic versus achromatic images.

2.5.2 Results

Figure 2.4 demonstrates that isoluminant chromatic and achromatic natural images have differing effects on the PLR. It is noticeable that the latency of the pupil constriction occurred identically at around 250 – 300 ms post-image onset regardless of chromaticity, with greater constriction seen for the colour images (approximately -1.00 mm) at around 900ms, compared to a smaller constriction (approximately -0.70 mm) reaching a nadir at 800ms for grey-scale images. The pupil initially recovers rapidly, particularly for the smaller pupil observed in response to the chromatic images, until approximately 2000 ms, where recovery slows for both stimuli until it reaches baseline pupil diameter at around 5000 ms. There was a small secondary constriction for both stimuli at image-offset that appears equal across chromaticity. A two-tailed within-subjects t-test revealed that the PLR was significantly larger (smaller pupil diameter) to chromatic images compared to achromatic images, $t\ (4) = 11.55$, $p < .001$, $g_{av} = 1.01$.

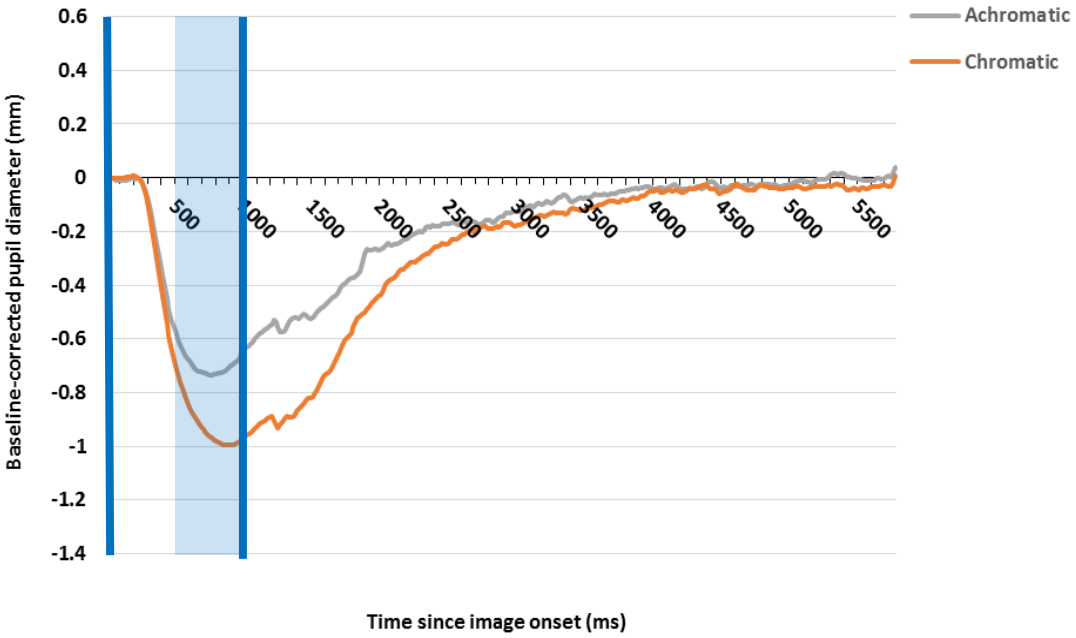


Figure 2.4 Baseline corrected pupil size change in response to colour or grey-scale emotionally neutral images. Blue bars indicate image-onset and offset, and blue shading indicates the data analysis window (N = 5).

2.5.3 Discussion

Against expectations, the present results for natural images differed as chromatic images compared to the same achromatic image led to a greater PLR with similar latency, but a delayed nadir, as well as showing a secondary small constriction after image offset. This suggests that the two elements of increased luminance contrast (from the preceding slide) and colour combine to cause a larger PLR. Young et al. (1993) suggested that the pupil is indeed responsive to colour and luminance alone, but argued against the existence of specific colour and luminance visual pathways. Rather, the PLR to luminance and colour occur through the same underlying mechanisms. The amplified PLR observed in response to chromatic compared to achromatic images may reflect simply an additional visual input that causes greater disruption of the inhibitory signal to the pupillometry nucleus causing augmented transient pupil constriction (see 2.4.3 Discussion for a further explanation of this process). Regardless, this experiment highlighted that colour within natural images affects pupil diameter and, therefore, visual

stimuli in future experiments will be presented in grey-scale (where necessary) removing chromaticity as a confounding variable.

2.6 Experiment 4: Duration

It is evident that the physical characteristics of natural images affect the PLR, but it was important to consider several factors that may influence the impact of emotion on pupil diameter following the PLR; Experiment 4 examined image presentation duration, while Experiment 5 investigated whether pupil responses to emotion habituate with repeated presentation. Reducing the presentation duration of visual cues is practically advantageous as it reduces the length of each trial, which can help reduce participant fatigue or allow for a greater number of trials to improve the signal-to-noise ratio; however, the image has to be presented for long enough to overcome the PLR and to give the participant the opportunity to process the image.

Previous experiments that have identified elevated pupil diameter to affective images have largely presented stimuli for several seconds (Bradley et al., 2008; Ferrari et al., 2016; Henderson et al., 2014; Hess & Polt, 1960; Jin et al., 2015; Lemaire, Aguillon-Hernandez, Bonnet-Brilhault, Martineau, & El-Hage, 2014). However, Codispoti, Mazzetti, and Bradley (2009) demonstrated that affective images led to larger electrodermal responses regardless of presentation duration, and this was evident even at images shown for just 25 ms. Moreover, there was little evidence that emotional reactivity increased with greater exposure. Similarly, Pessoa, Japee, Sturman, and Ungerleider (2006) identified that individuals could reliably detect fearful cues after 67 ms of exposure and this was reflected in elevated amygdala responses, although this effect was for facial expressions, which are often thought of as a unique social and biological cue (Hariri, Tessitore, Mattay, Fera, & Weinberger, 2002). Regardless, there is evidence that very brief presentations of emotion can cause increases in physiological responsivity meaning longer presentations of visual affect may be redundant. Indeed,

Van Steenbergen et al. (2011) reported greater pupil diameter to negative and positive images compared to neutral images when participants were exposed to the stimulus for just 500 ms. However, the presentation of emotive images was part of an attentional narrowing task and the analysis-window spanned participants performing pro/anti-saccadic eye movements, which is problematic given that increased cognitive load or the recruitment of executive control processes affects pupil diameter (Cohen, Moyal, & Henik, 2015).

In the current experiment, participants passively viewed negative and neutral images across four different durations: 100, 300, 1000 and 3000 ms. I expected that negative images would lead to larger pupil diameter compared to neutral images, but that there would be no interaction between image valence and presentation duration consistent with research that identified elevated autonomic responsivity to emotion regardless of exposure duration (Codispoti et al., 2009). Each stimulus was presented for 100 and 300 ms to explore whether emotional modulation of the pupil was evident under conditions where no saccades could be initiated (Gilchrist, 2011; Miles & Kawano, 1987; Sumner, 2011) to rule out the possibility of differential viewing patterns as an explanation for a potential lack of emotional modulation. Images were presented for 1000 ms consistent with the previous experiments, and 3000 ms to examine participant's affective pupil response following a complete PLR.

2.6.1 Methods

Twenty-two participants (17 female) were recruited from Cardiff University aged 18 – 51 (M = 25.91, SD = 8.06) based on power calculations (G*Power; Faul, Erdfelder, Lang & Buchner, 2007) calculated for a repeated measures ANOVA with 80 % power (α = .05) to detect a medium effect size of f^2 = 0.15 (Cohen, 1992) consistent with previous studies exploring pupillometry to emotion (Bradley et al., 2008; Henderson et al., 2014;

Partala & Surakka, 2003)[2]. Twenty images[3] were selected from the IAPS, specifically 10 negative (mean valence/arousal = 2.91, 6.20) and 10 neutral images (mean valence/arousal = 5.17, 3.10) were chosen. The negative and neutral images differed significantly for subjective valence (*p*s < .001), and the negative images were rated as more subjectively arousing than the neutral images ($p < .001$). In accordance with Experiment 1 – 3, all images were converted to grey-scale. Each image was matched to the stimuli set's median luminance (value of 95) and contrast was matched across valence categories (M = 52.83, SD = 18.15). Each image was randomly presented at each of four durations: 100, 300, 1000, and 3000 ms. A recovery slide followed the target image for 5000 ms like previous experiments. To quantify pupil response, I calculated the mean baseline-corrected pupil diameter for the time-window 1000 - 2000 ms post image onset for both negative and neutral stimuli for each participant. This time window was chosen as it avoids the initial pupil constriction that occurs in response to visual stimulation (reaching maximum constriction between 500 – 1000 ms). A repeated measures ANOVA was run between emotional content (negative, neutral) and presentation duration (100, 300, 1000, 3000 ms). For the current experiment, 85.10 % of total data was valid during the analysis response window.

2.6.2 Results

Figure 2.5 demonstrates that the pupil was larger when viewing negative images than during the neutral images and this occurred regardless of presentation duration. A PLR with a latency of 300 ms was observed at all durations with a comparable magnitude for 300, 1000 and 3000 ms presentation (between approximately -0.50 and -0.60 mm), which included attenuated constriction in response to the negative images. The PLR to images presented for 100 ms was smaller and was equivalent across negative and

[2] Experiment 5 used a similar power calculation.
[3] IAPs images used were: Neutral: 2036, 2190, 2214, 2383, 2393, 2514, 2745.1, 2850, 2870, 5731; Negative: 1301, 1304, 1525, 5973, 6231, 6250, 6242, 6263, 6370, 9901.

neutral images. Across all stimuli, the pupil then rapidly increased in diameter until approximately 1500 ms post image-onset where recovery slowed before reaching baseline pupil diameter with faster recovery for shorter presentation. Negative stimuli continued to lead to larger pupil diameter as the pupil recovered back to baseline pupil diameter following the PLR across 300, 1000 and 3000 ms durations. Following 100 ms presentation lengths, negative modulation of pupil diameter occurred at approximately 1300 ms post-image onset.

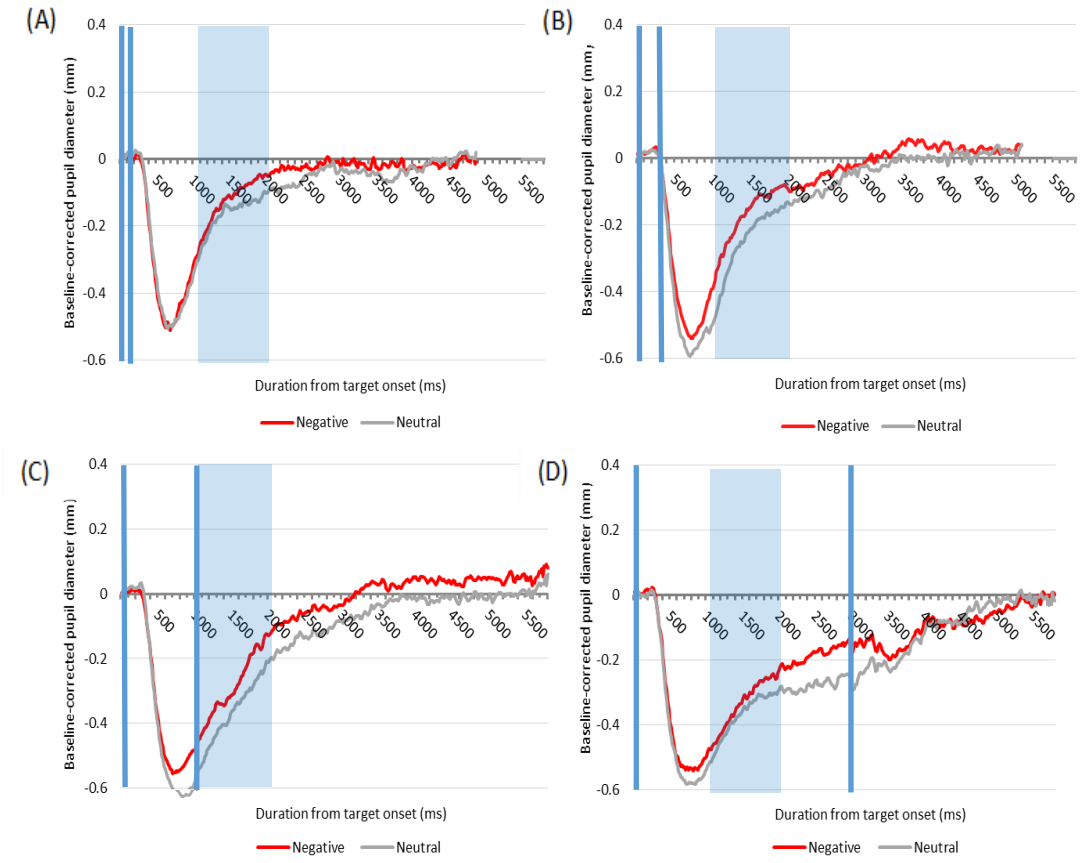


Figure 2.5 Baseline-corrected pupil size change in response to negative and neutral images presented over varying presentation duration. Images were presented for (A) 100 ms, (B) 300 ms, (C) 1000 ms and (D) 3000 ms. Blue bars indicate image-onset and offset and blue shading indicates the data analysis window (N = 22).

Mean baseline-corrected pupil diameter was calculated over 1000 – 2000 ms post-image onset and is presented in Figure 2.6 A 2 x 4 repeated measures ANOVA, with factors of emotion (negative and neutral) and presentation duration (100, 300, 1000 and 3000 ms) examined pupil diameter over this analysis window revealing a main effect of image emotionality, F (1, 21) = 17.30, p < .001, η_p^2 = .45, and stimulus duration using a Greenhouse-Geisser correction, F (1.41, 29.55) = 17.91, p < .001, η_p^2 = .46, with no interaction, F (3, 63) = 0.86, p = .46, η_p^2 = .04. The main effect of duration was further tested via post-hoc comparisons (Bonferroni-corrected α = .008 [.05/6], calculated based on six comparisons across the four presentation durations). This showed that the pupil diameter was larger at 100 than at 300 ms, t (21) = 4.23, p < .001, g_{av} = 0.53, and at 300 than at 1000 ms, t (21) = 4.02, p = .001, g_{av} = 0.82, but there was no significant difference between 1000 and 3000 ms, t (21) = 0.99, p = .33, g_{av} = 0.09.

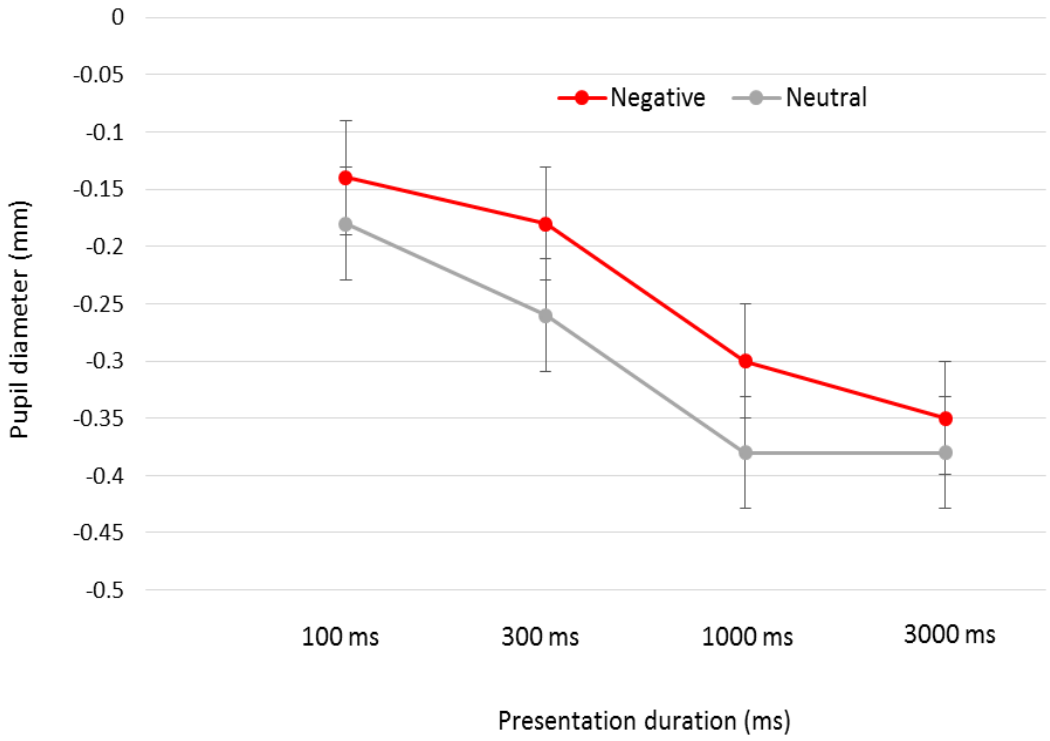


Figure 2.6 Mean baseline-correct pupil diameter in response to negative and neutral images over four presentation durations. The error bars represent ± standard error of the mean.

2.6.3 Discussion

This experiment examined whether varying the presentation duration affected negative modulation of pupil diameter. As expected, negative images led to larger pupil diameter than neutral images, and there was no interaction between emotion and presentation duration. This indicates that negative images led to increased pupil diameter regardless of presentation duration consistent with Codispoti et al. (2009). Crucially, this suggests that presenting emotional stimuli for longer durations is not necessary when examining emotional modulation of pupil diameter for negative versus neutral images. Additionally, it was observed that pupil diameter increased with shorter presentation duration (100 > 300 > 1000 ms) separate to emotion. This may be due to the early removal of the constrictive influence that accompanies elevated image contrast (see Experiment 2). However, the primary interest for the current thesis was that negative images continued to lead to larger pupil diameter than neutral images regardless of presentation duration.

2.7 Experiment 5: Habituation

It is important to understand whether changes in pupil diameter in response to emotion lessen over repeated presentation of emotive visual stimuli, as future experiments will present multiple affective images. Electrodermal and cardiovascular responses have been previously shown to habituate to emotion over repeated exposure, but, importantly, electrodermal increases to affect could be reinstated by introducing novel stimuli (Bradley, Lang, & Cuthbert, 1993). Previous research has demonstrated that the PLR habituates as previously presented images (either presented consecutively or distributed) caused smaller PLRs than novel images, with the effect greatest for consecutive images (Bradley & Lang, 2015; Ferrari et al., 2016; Kafkas & Montaldi, 2015;

Naber, Frässle, Rutishauser, & Einhäuser, 2013; Võ et al., 2008). Importantly, this effect was not specific to emotional images.

Two recent studies have investigated whether emotional modulation of pupil diameter lessens with repeated affective presentation. Bradley and Lang (2015) presented violent, erotic and neutral images during a free-viewing paradigm either once, or repeatedly (either consecutively or distributed). They found that violent and erotic images led to larger pupil diameter compared to neutral images for novel images and these differences continued to be found for repeated presentation (consecutive or distributed). However, overall pupil response magnitudes decreased in response to consecutive erotic images compared to novel or distributed presentation indicating habituation to consecutive presentation for these images. Violent images led to the same pupil response magnitudes regardless of novel or repeated (consecutive or distributed) presentation. The authors found elevated pupil response to novel erotic images compared to novel violent stimuli, and suggested that the habituation observed for pupil response to erotic images primarily reflects greater initial arousal. Additionally, Ferrari et al. (2016) conducted a similar experiment presenting negative, positive (including erotic images) and neutral images either once or repeated consecutively. The authors found that emotional images (composed of both negative and positive images) led to larger pupil diameter than neutral images when images were novel, but that this affective advantage disappeared with repeated consecutive presentation suggesting habituation of pupil responses to emotion for massed presentations. However, as the authors analysed their data by grouping negative and positive images together, the results may have been driven by habituation to specifically erotic images as found by Bradley and Lang (2015). Overall, there is evidence that pupil responses to emotionally arousing stimuli do habituate with repeated presentation, but this may be specific to consecutive presentations and to highly arousing erotic stimuli.

The current experiment explored whether emotional modulation of pupil diameter attenuated with repeated presentation of negative images, as well as presenting neutral images for control stimuli. Images were presented once randomly within a block, and there were four blocks. The current experiment was also interested to observe whether emotional modulation of pupil diameter attenuated across increasing trials within each block, reflecting habituation to negative images as an entirety. Firstly, I expected that negative images would continue to lead to larger pupil diameter compared to neutral images over repeated presentation, likewise to Bradley and Lang (2015). I also predicted that pupil response to negative images would not diminish over increasing trials within each block, as Bradley and Lang (2015) identified that this process only occurred to erotic images which I did not employ due to the paradigm's intended later clinical application. However, I predicted that pupil diameter would increase with repeated presentations and this effect would be across both negative and neutral images reflecting habituation of the PLR (Bradley & Lang, 2015; Ferrari et al., 2016; Kafkas & Montaldi, 2015; Naber et al., 2013; Võ et al., 2008).

2.7.1 Methods

Twenty-two participants (15 female) were recruited from Cardiff University aged 18 – 51 (M = 24.27, SD = 6.90). The image set was identical to the stimuli used in Experiment 4 (10 negative and 10 neutral images) and were presented randomly to participants in a block of 20 images for 2000 ms. I presented participants with four blocks, with a gap of three minutes between blocks 2 and 3 to reduce participant fatigue. Blocks 1 to 2 and blocks 3 to 4 ran consecutively with no gap.

To quantify each participant's pupil response to negative and neutral images, I again calculated mean baseline-corrected pupil size over 1000 - 2000 ms post stimulus onset for both negative and neutral images. A 2 x 10 x 4 repeated measures ANOVA was run with factors of emotional content (negative, neutral), trial position within the block

(1 - 10) and block (1 - 4). Missing values were replaced with the mean value calculated using a Markov Chain Monte Carlo (MCMC) multiple imputation method[4]. Twenty imputations were calculated (Graham, Olchowski, & Gilreath, 2007). For the current experiment, 87.52 % of total data was defined as missing during the analysis response window.

2.7.2 Results

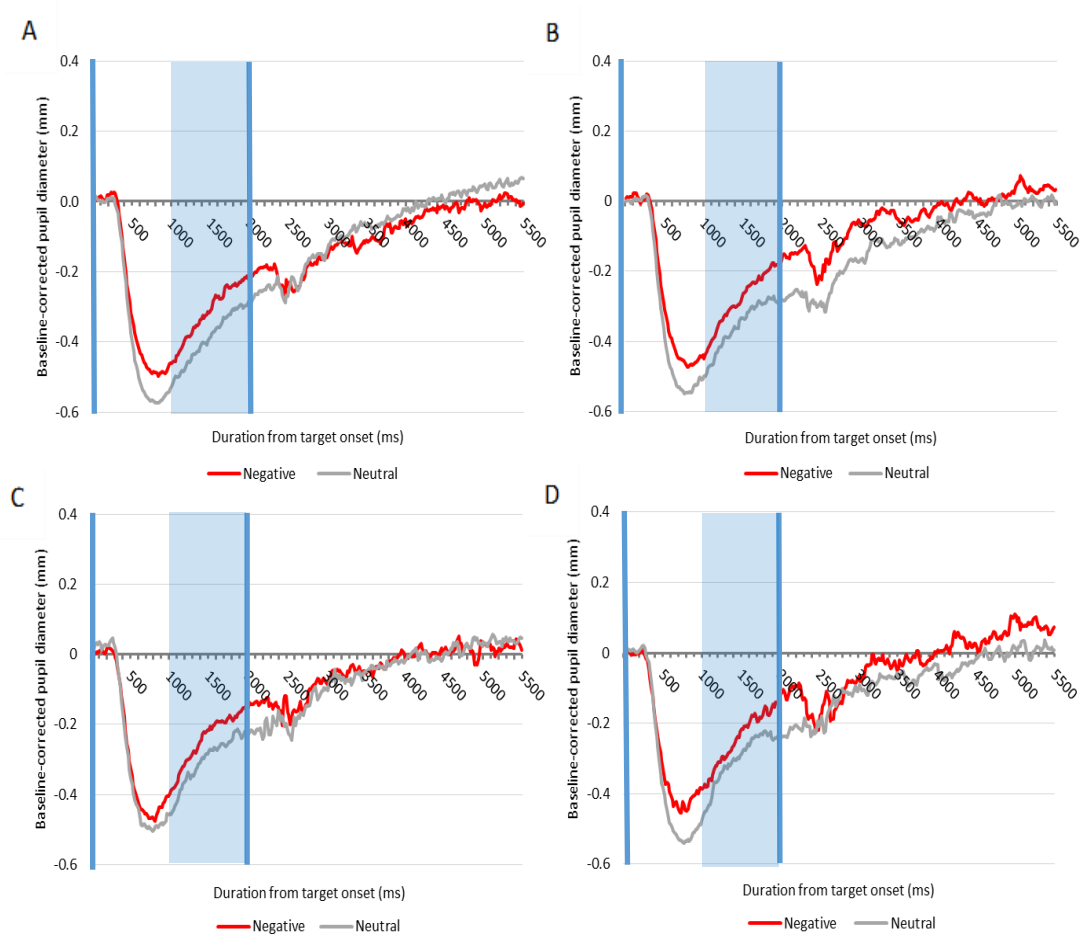


Figure 2.7 Baseline-corrected pupil size change in response to negative and neutral images presented repeatedly over 4 blocks (A – D indicates block 1 – 4). Blue bars indicate image-onset and offset, and blue shading indicates the data analysis window (N = 22).

[4] The MCMC method was chosen to take into account within-participant and across-participant variability in pupil diameter when estimating the missing values, with the only disadvantage to the procedure being a more lengthy and complicated process that produces larger data set (Rubin, 2004).

Participants viewed the same negative and neutral images four times, and it is evident from Figure 2.7 that a similar response pattern occurred across each block with negative images continuing to lead to larger pupil diameter across repeated presentation. Once again a PLR was observed with a latency of 300 ms which reached a nadir of -0.45 for negative images and -0.55 for neutral images. A rapid increase in pupil diameter was then evident until about 1500 ms post-image onset, where pupil recovery slowed until it reached baseline size, and even showed a tendency to exceed initial diameter.

Mean baseline-corrected pupil diameter was calculated over 1000 – 2000 ms post-image onset (see Figure 2.8) and entered into a repeated measures ANOVA with factors of emotion (negative and neutral), trial position (from trial 1 to 10) and block (block 1, 2, 3 and 4). The repeated measures ANOVA showed that the pupil was more dilated during the negative images than during the neutral images, F (1, 21) = 23.79, p < .001, η_p^2 = .53), and that there was a main effect of image block, F (3, 63) = 4.69, p = .01, η_p^2 = .18,

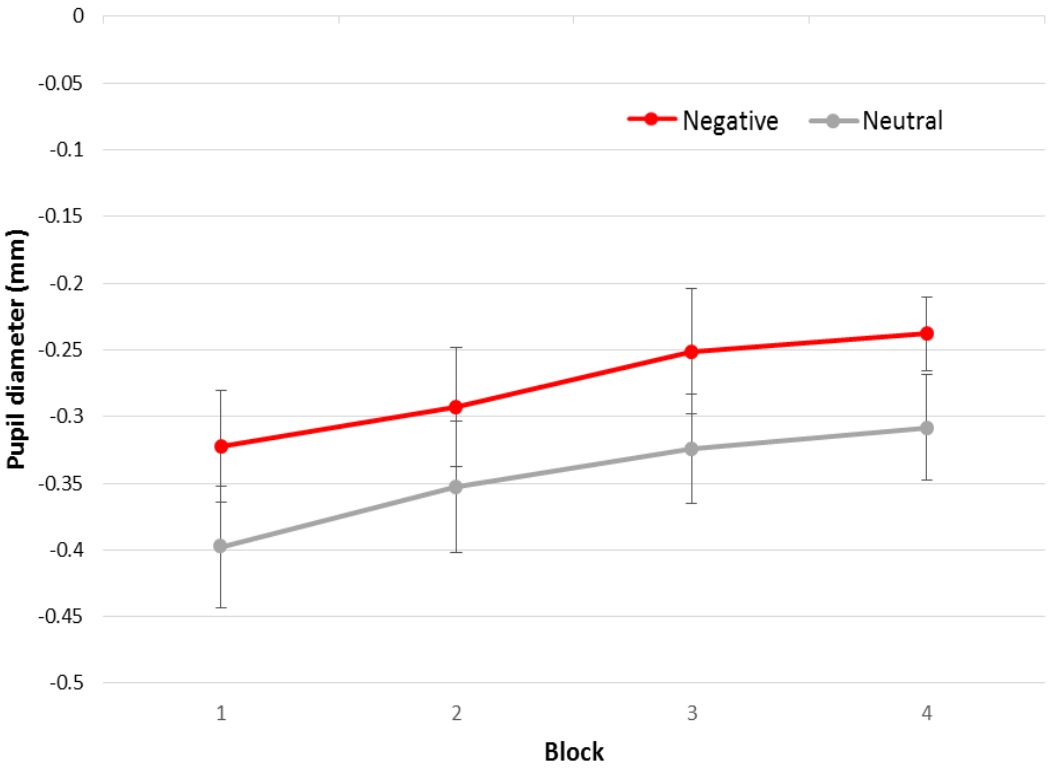


Figure 2.8 Mean baseline-correct pupil diameter in response to negative and neutral images over four presentation blocks. The error bars represent ± standard error of the mean.

with increasing pupil diameter (or smaller PLR) across blocks. There was no main effect of trial position within block using a Greenhouse-Geisser correction, F (4.72, 99.05) = 0.67, p = .74, η_p^2 = .04. Moreover, the main focus was that there might be a reduction in the magnitude of emotional modulation across or within blocks demonstrating habituation of affective reactivity. However, the interaction between emotional content and block was not significant, F (3, 63) = .08, p = .97, η_p^2 = .004, and neither was the interaction between emotion and trial position using a Greenhouse-Geisser correction, F (5.77, 121.15) = 1.28, p = .27, η_p^2 = .06. The two-way interaction between trial and block was also not significant, F (27, 567) = 1.40, p = .09, η_p^2 = .06, nor was the three-way interaction between trial, emotional content and block using a Greenhouse-Geisser correction, F (11.61, 243.87) = 1.14, p = .33, η_p^2 = .05.

2.7.3 Discussion

Experiment 5 was interested to know whether emotional modulation of pupil diameter would habituate across repeated presentation. As predicted, negative images continued to lead to elevated pupil diameter compared to neutral images across blocks, and also there was no evidence of diminished pupil diameter to negative images across increasing trials within blocks. Importantly, the results indicate no evidence of habituation for pupil diameter changes in response to emotion. As expected, the PLR appeared to habituate across all images.

Most psychophysiological measures are thought to habituate to repeated presentations. As discussed previously, electrodermal and cardiovascular responses have been previously shown to habituate to emotion over repeated exposure (Bradley et al., 1993). However, the startle response continued to differentiate between negative, positive and neutral images despite repeated presentations suggesting that some psychophysiological measures do not habituate or at least show different rates of habituation. The pupil may show an elevated threshold for response habituation as there

is evidence of attenuated responses over repeated presentations, but only for highly arousing erotic stimuli that is particularly arousing when first viewed and when repeated presentation was consecutive (Bradley & Lang, 2015). However, the current finding that emotional modulation of the pupil did not diminish over repeated presentation is promising for research interested to explore pupil responsivity to emotion, as conducted in Chapter 3 and 4.

2.8 General Discussion

This chapter has explored various considerations that can affect the magnitude of the PLR (Experiment 1 – 3), as well as factors that may play a role when exploring emotional modulation of pupil diameter (Experiment 4 – 5) in response to natural scenes. Although the current thesis is primarily interested in pupil diameter following the PLR, it is important to consider influences on this constriction as it directly affects subsequent pupil diameter. Experiment 1 reported that luminance has a clear effect on the PLR to natural images (with greater constriction for brighter images), but that luminance was not the sole influence of constriction magnitude as isoluminant stimuli and even slightly darker stimuli (to the preceding slide) led to a pupil constriction. It is proposed that the current results are consistent with the processes identified using simple stimuli; that is, a steady-state component sensitive to overall luminance, as well as a secondary transient rate-sensitive process that is responsive to the degree of change in local visual signals.

Experiment 2 explored this further, finding that greater image contrast led to a larger PLR, paralleling research using simple visual cues. It is speculated that these findings reflect the activity of the transient rate-sensitive component and are consistent with Barbur et al (1992)'s assertion that abrupt changes in stimuli interrupt the inhibitory influence exerted by the visual cortex leading to greater parasympathetic activation and, in turn, greater pupil constriction.

Furthermore, the data in Experiment 3 showed that isoluminant and contrast-matched chromatic natural images led to a larger PLR than their achromatic equivalent. This experiment highlighted that colour affects pupil diameter in response to complex images, although the underlying mechanism for this additional constrictive chromatic influence is not clear. Overall, Experiment 1 – 3 underlined the importance of carefully controlling for the luminance, contrast and colour of visual stimuli when measuring pupil diameter.

Experiment 4 revealed no effect of image duration on emotional modulation of the pupil. Negative images led to larger pupil diameter compared to neutral images regardless of presentation duration. Therefore, the following chapters presented images for shorter periods compared to previous influential pupillometry research (Bradley et al., 2008) in order to reduce participant fatigue.

Experiment 5 examined whether emotional modulation of the pupil response would habituate across repeated presentation of negative images. No evidence was found that emotional modulation of pupil diameter to negative images habituated with repeated presentation, although there was evidence for an attenuated PLR with repeated presentation regardless of valence. Hence, it seems likely that affective stimuli would continue to produce increased pupil diameter (compared to neutral stimuli) over repeated affective exposure.

It is important to note that the results for Experiment 4 and 5 are not certain to generalise to auditory stimuli, as presented in future chapters, although it has been shown that auditory stimuli are largely tapping into a comparable emotional response to visual stimuli (Bradley & Lang, 2000).

Overall, this chapter has highlighted the importance of controlling for physical features of visual stimuli when measuring pupil diameter, as well as demonstrating that

presentation duration and habituation play little role in moderating pupil response to affective visual stimuli (at least with distributed presentation of non-erotic images).

3 Chapter 3: Psychopathy and pupil response to emotion within a community sample

Abbreviation	Meaning	Page
ADFES	Amsterdam Dynamic Facial Expression Set	97
EI	Emotional index	78
IADS	International Affective Digitised Sounds	104
IAPS	International Affective Picture System	83
OFC	Orbitofrontal cortex	96
PCL-R	Psychopathy Checklist-Revised	96
PLR	Pupil light reflex	78
PPI-R	Psychopathic Personality Inventory - Revised	111
SCR	Skin conductance response	74
Tri-PM	Triarchic Psychopathy Measure	74
vmPFC	Ventromedial prefrontal cortex	96

Chapter 2 indicated the importance of controlling for physical features of visual stimuli when measuring pupil diameter, as well as demonstrating that presentation duration and habituation play little role in moderating pupil response to affective visual stimuli. Chapter 3 applied these findings to use pupillometry to explore whether psychopathy traits were associated with autonomic hypo-responsivity during passive-viewing of negative and positive stimuli across visual and auditory domains. This was explored within an undergraduate community sample. An increasing number of studies have investigated psychopathy within normal community samples given the development of a number of self-report measures of psychopathy (see 1.2.2 Multi-faceted models of psychopathy) as well as psychopathy being increasingly understood as a dimensional construct (see 1.2.4 Psychopathy as a taxonic versus dimensional construct). In the current chapter, psychopathy traits were measured using the Triarchic Psychopathy Measure (Tri-PM), given that the triarchic model was explicitly developed from previous psychopathy literature conceptualising an underlying genotypic disposition towards fearlessness, as well as recognising the adaptive features of psychopathy that are likely to be relevant in a non-offender sample (Patrick et al., 2009).

Experiment 6 measured pupil diameter in response to affective images. Affective images have been employed many times in previous experiments to measure autonomic responsivity (typically using skin conductance response, SCR) as a function of psychopathy (see 1.3.4 Passive-viewing of emotion in psychopathy), but no previous study has employed pupillometry. However, the depiction of emotion in such images involves the participant to process complex scenes that are difficult to truly match across physical properties.

Images of facial expressions have also been frequently used to demonstrate deficits in the processing of emotional stimuli in psychopaths (Dawel et al., 2012). Facial expressions are thought to represent a unique social and emotional stimuli (Fridlund, 2014) that is processed by a highly specialised system (Posamentier & Abdi, 2003).

Images of facial expressions are also advantageous for pupillometry research as the stimuli depicting the different valences are highly similar in terms of luminance and contrast. A number of studies have measured pupil diameter to static or morphed facial affect for adults (Duque, Sanchez, & Vazquez, 2014; Kret, Roelofs, Stekelenburg, & de Gelder, 2013; Prehn, Kazzer, et al., 2013) and children (Burkhouse et al., 2015; Farzin, Rivera, & Hessl, 2009; Geangu et al., 2011; Sepeta et al., 2012), although many present only a limited range of facial expressions, with some studies not displaying a neutral expression for comparison. Experiment 7 examined pupil diameter to facial affect by presenting images of fearful, happy, disgusted, angry and sad faces, as well as an emotionally neutral expression for comparison. Surprise was not explored due to doubts regarding the dissociation between the emotion of surprise and facial displays (Reisenzein, Bördgen, Holtbernd, & Matz, 2006)

While static facial expressions have often been used to investigate the processing of emotional stimuli, they are an impoverished version of real emotional expression where the display of affect is dynamic. Experiment 8 presented video-clips of facial affect (dynamic facial expressions) as they are thought to be more ecologically valid than static expressions because they represent the dynamic, complex and subtle expression of real-life emotion more accurately (Recio, Sommer, & Schacht, 2011; Rymarczyk, Biele, Grabowska, & Majczynski, 2011; Sato, Fujimura, & Suzuki, 2008; Simons, Detenber, Roedema, & Reiss, 1999; Weyers, Mühlberger, Hefele, & Pauli, 2006). In addition, to the author's knowledge, no study has previously examined pupil response to video-clips of facial affect in adults.

Finally, Experiment 9 measured pupil diameter to affective sound-clips; this was advantageous as it removes any visual influence on the pupil, yet few studies have examined pupil reactivity to affective and neutral sound-clips (Dabbs, 1997; Jin et al., 2015; Partala & Surakka, 2003). Verona et al. (2004), to the author's knowledge, is the

only previous study to measure autonomic responses (using SCR) to emotional sound-clips in relation to psychopathy, but no previous study has employed pupillometry.

3.1 General methods

3.1.1 Participants

One-hundred and two participants were recruited (52 female) with a mean age of 21.08 (S.D. = 3.57) from the School of Psychology at Cardiff University and took part in each of the four experiments. Participant sample size was based on an *apriori* power calculation (G*Power 3.1; Faul, Erdfelder, Lang & Buchner, 2007) for a linear multiple regression including the three Tri-PM predictor variables (Boldness, Meanness and Disinhibition) with 90% power (α = .05) to detect a medium effect size, f^2 = 0.15 (Cohen, 1992), consistent with previous research exploring the relationship between psychopathy and autonomic hypo-responsivity using electrodermal responses (Lorber, 2004). The recommended sample size was 99 participants. All participants had normal or corrected-to-normal vision. Participants were requested to not consume caffeine or smoke 60-minutes prior to testing. Participants were either paid money or given research credits as part of their psychology undergraduate course.

All experimental procedures were given ethical approval by the Ethical Committee of the School of Psychology, Cardiff University. All participants gave written informed consent to participate in the experimental procedures, and were fully debriefed at the end of the session.

3.1.2 Design

Each stimuli type (affective images, static facial expressions, dynamic facial expressions and affective sound-clips) was presented as an individual experiment and

every participant took part in each experiment in the same order (as listed above). Examples of the stimuli presented , as well as a schematic illustration of the structure of each trial, are detailed in Appendix 3. The task design was similar to that described in Chapter 2: A fixation screen (for 2000 ms), stimuli (presentation duration varied between stimuli type) and then recovery screen (presentation duration varied between stimuli). The fixation screen was a grey slide displaying a fixation cross composed of alternating light and dark grey pixels, while the recovery screen was a grey slide, both identical to Chapter 2. The fixation and recovery screen were matched to the target stimuli for overall luminance during visual stimuli tasks. Stimulus presentation order was randomised for each task. All stimuli were manipulated using Adobe Photoshop Elements 12. As described in Chapter 2, luminance was manipulated along the luminance scale of 0 – 255 (higher values indicating brighter stimulus), while image contrast (standard deviation of all pixel luminance) was altered along a scale from 0 - 120 (higher values indicate greater contrast). Participants were read these instructions prior to each task, “You are now going to be presented with some images/video-clips/sound-clips. Your task is to pay attention to them, and keep your eyes on the screen while keeping your head as still as you can”.

3.1.3 Triarchic psychopathy measure

The Tri-PM is a self-report measure of psychopathy constructed of 58 statements to which the participant responds ‘False’, ‘Mostly False’, ‘Mostly True’ or ‘True’. Each item was scored from 0 – 3 (higher scores indicate higher psychopathy traits) giving participants a score along the dimensions of Boldness (maximum score = 57), Meanness (maximum score = 57) and Disinhibition (maximum score = 60). Each participant completed the Tri-PM in between the second (static facial expressions) and third task (dynamic facial expressions).

3.1.4 Data acquisition and analyses

Stimuli was displayed using the same equipment as described in Chapter 2 (see 2.2.3 Pupil data acquisition and cleaning). The same data acquisition and cleaning process was used as reported in the previous chapter (see 2.2.3 Pupil data acquisition and cleaning).

I calculated initial pupil diameter as reported in the previous chapter and used this measure to established baseline-corrected pupil diameter (see Chapter 2, General methods, Pupil data acquisition).

To quantify pupil diameter in response to stimuli, I calculated mean baseline-corrected pupil diameter over time windows for each valence within each task; the specific time-windows are described in more detail in each experiment's method. For the visual tasks, these time-windows were defined following the pupil light reflex (PLR). Repeated measures ANOVAs were run entering stimuli valence as a within-subjects variable to assess the effect of stimuli emotionality on pupil diameter, and planned comparison *t*-tests were conducted between pupil diameter to each affective and neutral stimuli.

To examine psychopathy effects on pupil diameter changes in response to emotion, I subtracted mean neutral baseline-corrected pupil diameter from mean baseline-corrected pupil diameter for each emotional stimuli (over the specific time-windows that will be later described in each task). This identified emotional modulation (or potentiation) of pupil diameter that was specific to the particular emotion in comparison to neutral stimuli across each task. This is consistent with measures employed by Verona et al. (2004) and Benning, Patrick, and Iacono (2005) to index skin conductance response (SCR) to emotional stimuli. This measure was termed as 'Emotional Index' (EI) for clarity throughout the thesis, with the specific valence (or emotion) that was compared to neutral stimuli reported (e.g. $EI_{Negative}$). Later references to 'emotional modulation' in relation to the current experiments are referring to this EI. The relationship between Boldness,

Meanness and Disinhibition and each EI was explored by conducting Pearson's zero-order/partial correlations, as well as multiple linear regressions to assess the unique contribution of each Tri-PM subscale.

I also examined whether psychopathy was associated with overall pupil responsivity to neutral stimuli, as individual differences in autonomic responsivity (separate to emotion) may influence the degree of emotional modulation shown. That is, diminished autonomic responsivity to neutral stimuli may reflect a restricted range of pupillary movement that could result in reduced pupil dilation in response to emotion. I wanted to ensure that psychopathy was not related to abnormal emotional modulation of pupil diameter simply through diminished pupil responsivity in general. Therefore, I explored the relationship between each Tri-PM subscale and baseline-corrected pupil diameter to neutral stimuli for each task using Pearson's zero-order correlations, as well as multiple linear regressions.

I tested that the data met the assumptions of linear regression (linear relationship, lack of multicollinearity, no autocorrelation, homoscedasticity and multivariate normality). Linearity was established through visual inspection of plots between predicted residuals and observed residuals. There was little evidence of multicollinearity between Tri-PM scores, as I observed a variance inflation factor of $1.22 < 1.73$ (across tasks) indicating this assumption was not violated (Craney & Surles, 2002). Auto-correlation was assessed using the Durbin-Watson's d test finding that the residuals were not linearly auto-correlated ($1.58 < d < 2.30$). To test homoscedasticity, I observed that the error terms along the regression line appeared equal with no obvious pattern emerging suggesting that the assumption was not violated. Finally, I explored multivariate normality within the data using boxplots, histograms and normal probability plot and I also conducted the Kolmogorov-Smirnov test. The majority of the pupil measures were normally distributed, although several did violate this assumption and so the data for

these measures was log10 transformed for statistical analyses[5]. The Kolmogorov-Smirnov test also showed that Meanness and Disinhibition violated the assumption of multivariate normality with a positive skew, and so both variables were transformed using a log10 transformation for statistical analyses to meet the assumption of multivariate normality. Meaningful untransformed data is reported for means and standard deviations. There were no differences in results using log transformed data consistent with propositions that deviations from a normal distribution have little effect on results within large sample sizes (> 30 participants) (Ghasemi & Zahediasl, 2012; Öztuna, Elhan, & Tüccar, 2006).

Trials were omitted if there was less than 50 % data for the selected time-window. Participant means were only calculated for each valence if at least 50 % of trials held valid data for the given valence. Participant means for a given valence were identified as an outlier and removed if their data for a given valence was outside the interval defined as three times the interquartile range (Tukey, 1977). Participants were excluded if they recorded less than 50 % valid data across all trials during stimulus presentation.

Two-tailed significance values were reported for all effects for simplicity.

3.1.5 Confounding variables

Confounding variables were considered to be variables that related to both factors of interest (Meinert, 2012); that is, the Tri-PM subscales and the pupillary measures. Male participants compared to female participants showed higher levels of Boldness, t (100) = 4.45, p < .001, d = 0.88, Meanness, r (79.55) = 4.88, p < .001, d = 1.09, and Disinhibition, r (100) = 2.05, p = .04, d = 0.41, and so it was important to explore the interaction between participant gender and psychopathy in relation to the pupillary

[5] The measures that were log10 transformed were pupil diameter to neutral static and dynamic facial expressions, as well EI_{Happy}, $EI_{Disgust}$, $EI_{Angry,}$ EI_{Sad} for the static facial expressions and EI_{Happy} for the dynamic facial expressions.

measures (see Table 0.5 in Appendix 1 for Tri-PM scores across gender). I conducted hierarchical regression analyses with the dichotomous variable gender (0 = male, 1 = female) and the centred Tri-PM subscales entered at the first step, and the interactions between gender and each subscale entered at the second step (Aguinis, 2004). The predicted variable was each pupillary measure that was described previously to explore psychopathy effects (EIs and pupil diameter to neutral stimuli). I adjusted the alpha level to be more conservative as I made no specific gender predictions in relation to psychopathy (Bonferroni-corrected α = .017 [.05/3], calculated based on the three Tri-PM subscale x participant gender interactions). I did not control for participant age in subsequent analyses as zero-order correlations revealed that age was unrelated to any of the Tri-PM subscales (Boldness, r[102] = .07, p = .47; Meanness, r[102] = -.04, p = .67; Disinhibition, r[102] = -.04, p = .68).

As the pupil can constrict to a minimum diameter of around 1.5 mm and can constrict to a maximum diameter of around 9 mm, I considered that individual differences in initial pupil diameter may limit the range of possible pupillary movement potentially influencing the degree of emotional modulation of pupil diameter. For example, for the sound-clips, an individual with a large initial pupil (i.e. close to the maximum 9 mm) cannot show the same degree of pupil dilation as an individual with a small initial pupil diameter (i.e. close to the minimum 1.5 mm). However, the subsequent results remained constant for all pupillary measures across tasks when controlling for individual differences in initial pupil diameter, suggesting that absolute initial pupil diameter did not affect pupil responsivity. Therefore, I did not consider initial pupil diameter further.

3.2 Experiment 6: Affective images

Images depicting emotion have been repeatedly used in emotion research demonstrating that affective compared to neutral images lead to increased physiological responsivity across electrodermal (Bernat, Patrick, Benning, & Tellegen, 2006; Bradley,

Codispoti, Cuthbert, et al., 2001; Codispoti & De Cesarei, 2007; Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Pastor et al., 2003), cardiovascular (Aue, Flykt, & Scherer, 2007; Bradley, Codispoti, Cuthbert, et al., 2001; Codispoti & De Cesarei, 2007; Lang, Greenwald, Bradley, & Hamm, 1993), respiratory (Ritz, George, & Dahme, 2000), electrocortical (Cuthbert et al., 2000; Schupp et al., 2004), and pupillary activity (Aboyoun & Dabbs, 1998; Bradley et al., 2008; Chae et al., 2008; Cohen et al., 2015; Lemaire et al., 2014; Steinhauer, Boller, Zubin, & Pearlman, 1983). Psychopathy research has used affective images to explore autonomic responsivity to the passive-viewing of emotion stimuli in both offender and community samples and, as previously described the presence and nature of this impairment has not been consistent (see 1.3.4 Passive-viewing of emotion in psychopathy).

Participants viewed affective (negative and positive) and neutral images, while their pupil diameter was measured in response. Given that pupil responsivity is thought to indicate emotional arousal (Bradley et al., 2008), the current predictions reflected the accompanying normative arousal ratings. I expected larger pupil diameter to the emotional images compared to the neutral images, with negative images showing greater pupil size than positive images.

I made several predictions in relation to psychopathy. Firstly, I predicted that Boldness and Meanness would be selectively associated with diminished emotional modulation of pupil diameter, indicating autonomic hypo-responsivity, to *specifically* negative images, with no relationship expected for Disinhibition. That is, I predicted that Boldness and Meanness would be negatively associated to EI to negative images, with no association for Disinhibition, and none of the subscales were hypothesised to be associated to EI for positive images. This reflects that the interpersonal/affective dimension of psychopathy has been linked to an impairment in processing emotional cues that has, intermittently, included autonomic hypo-responsivity to emotion, while the lifestyle/antisocial component of psychopathy has been largely unrelated to autonomic

responsivity to emotion (see 1.3.3 The dimensions of psychopathy and emotional processing and 1.3.4 Passive-viewing of emotion in psychopathy). Further, I hypothesised autonomic hypo-responsivity to specifically negative images, as psychopathy has been theoretically and empirically linked to a greater impairment to negative stimuli compared to positive stimuli (as described in 1.3 Psychopathy and emotional processing), and Boldness and Meanness have been theoretically conceptualised as the phenotypical expression of fearlessness (Patrick et al., 2009). I also predicted that all Tri-PM subscales would be unrelated to pupil responsivity to neutral images reflecting normal pupil response magnitudes separate to emotion. Psychopathy has not been convincingly linked to diminished overall autonomic response magnitudes, with small effect sizes within the studies that have identified an association (Brook et al., 2013) (see 1.3.4 Passive-viewing of emotion in psychopathy). Additionally, much of this research has failed to directly explore autonomic responses to neutral stimuli, raising the possibility that overall diminished responses were driven by smaller responses to emotional stimuli rather than a global impairment. Indeed, startle response paradigms have identified that interpersonal/affective or lifestyle/antisocial psychopathy traits are unrelated to startle response magnitudes to neutral stimuli (Esteller et al., 2016; Sutton et al., 2002).

3.2.1 Method

Thirty images were selected from the International Affective Picture System (IAPS) consisting of 10 negative images (mean valence/arousal = 2.94, 6.53), 10 positive images (mean valence/arousal = 7.87, 4.74) and 10 neutral images (mean valence/arousal = 5.18, 2.90)[6]. Images were selected based on Barke, Stahl, and Kroner-Herwig (2012) who categorised the IAPS into fearful, happy and neutral stimuli,

[6] IAPS images selected were: Negative: 1301, 1304, 1525, 1930, 2811, 6260, 6250, 6263, 6370, 6510; Positive: 1440, 1441, 1460, 1463, 1710, 1721, 1750, 2070, 4641, 8380; Neutral: 2036, 7009, 7010, 7020, 7042, 7045, 7052, 7150, 7179, 7205.

as I wanted to select affective images that were unambiguously negative or positive while the paradigm was continuing to be developed. Also, I did not include erotic images given that I later wanted to apply the paradigm to offender samples that included sexual offenders and was concerned that erotic stimuli may introduce further difficulties, especially given that erotic images can lead to disproportionately large autonomic responses within normal healthy populations (Weinberg & Hajcak, 2010). The stimuli list differed significantly for valence (*p*s < .001). Both negative and positive images were subjectively rated as more arousing than neutral images, with greater arousal ratings for negative images (all *p*s < .001). Images were presented in grey-scale, and overall luminance was manipulated to the mean luminance value of the sample (95; see 3.1.2 Design), while luminance contrast was matched across valence categories (M = 52.83, SD = 18.15) so that there was no difference between valences (*p* = .82). Each image was presented for 2000 ms because I wanted a brief presentation that allowed the assessment of initial emotional reaction to the stimulus, while still gathering sufficient data following the PLR. Overall, each trial lasted for 9000 ms.

3.2.1.1 Data analysis

To examine emotional modulation of the pupil following the PLR, I calculated mean baseline-corrected pupil dilation over 1000 – 2000 ms post-image onset. A repeated measures ANOVA was run to assess whether image emotionality significantly affected pupil diameter.

Several measures were calculated to explore the relationship between psychopathy and pupil response to the images: EI (the difference between mean baseline-corrected pupil diameter in response to negative or positive images compared to neutral images calculated over 1000 – 2000 ms post image-onset) and pupil diameter to neutral stimuli (taken to be mean baseline-corrected pupil diameter to neutral stimuli over the same time-window). I ran zero-order/partial correlations and multiple linear regressions to

assess the relationship between Tri-PM subscales and each EI, as well as pupil diameter to neutral images.

I also explored participant gender and psychopathy interactions on pupil responses by conducting hierarchical linear regression analyses including participant gender and the three centred Tri-PM subscales at the first step, and the three gender x Tri-PM subscales interactions at the second step. The predicted variable was each EI and baseline-corrected pupil diameter to neutral stimuli respectively.

Five participants were excluded from the sample for containing too much missing data leaving a total sample of 97 participants (47 female). Spearman-Brown split-half reliability checks revealed good internal consistency for pupil diameter over 1000 – 2000 ms post-image onset (r = .90). Across this sample, 92.55 % of total data during image presentation was defined as valid.

3.2.2 Results

Table 3.1 shows the mean values of the Tri-PM subscales, and the internal reliability was high for all three Tri-PM subscales. The present study found that across the total sample Meanness was positively related to both Boldness, r (102) = .43, p < .001, and Disinhibition, r (102) = .53, p < .001, with no relationship evidenced between Boldness and Disinhibition, r (102) = .14, p = .18.

Table 3.1 Mean scores, standard deviation, range and internal consistency for the Tri-PM subscales

	Mean (SD)	Range	ICC
Boldness	29.44 (8.73)	7 – 50	.86
Meanness	12.60 (8.52)	1 – 46	.91
Disinhibition	14.18 (7.50)	2 - 39	.85

ICC, internal consistency measured by Cronbach's α

3.2.2.1 Manipulation check

As can be seen in Figure 3.1, there was a PLR with an approximate latency of 300 ms and a nadir of -0.45 mm occurring at around 800 ms post-image onset. The pupil then increased in size until the offset of the stimulus at 2000 ms post-image onset, where there was a smaller secondary constriction again with a latency of around 300 ms following image-offset. A transient rapid increase in pupil dilation was then observed until 3000 ms before a slower recovery reaching baseline pupil diameter at around 5000 – 6000 ms. It is evident from Figure 3.1 that pupil diameter was larger to negative images following the PLR compared to positive and neutral images. Across the sample of 97

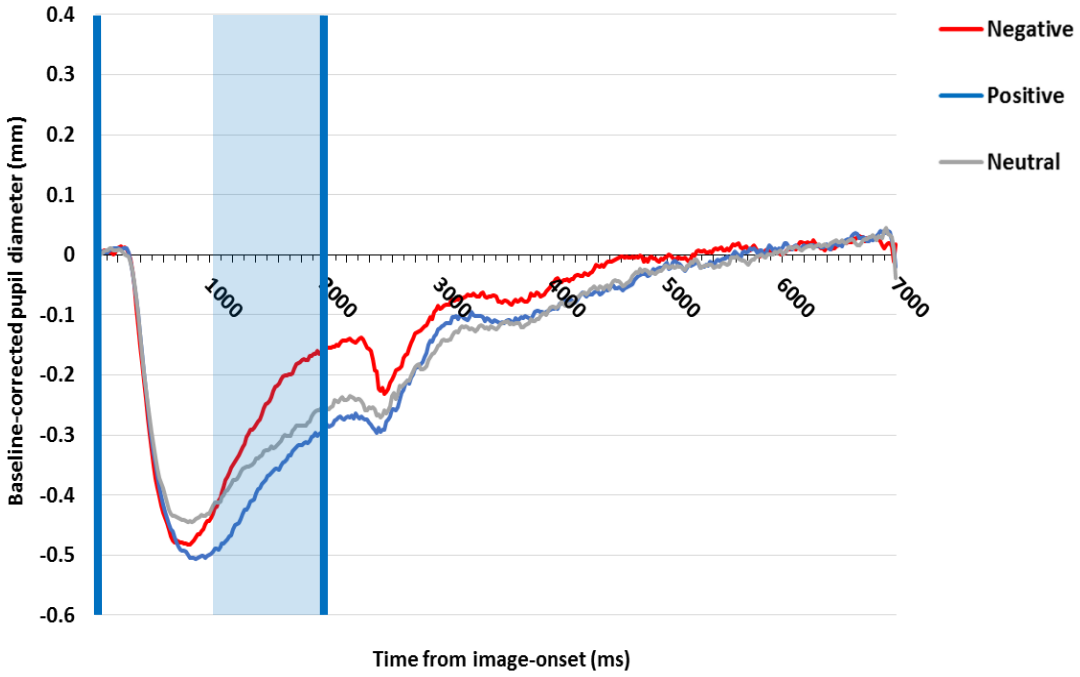


Figure 3.1 Baseline corrected pupil diameter change in response to negative, positive and neutral images. The blue bars indicate image onset and offset. Blue shading specifies the time window used for analyses (N = 97).

participants, a repeated measures ANOVA with the factor of valence (negative, positive and neutral) revealed that there was a main effect of emotion on pupil diameter over the analysis window, F (2, 192) = 23.18, $p < .001$, $\eta^2 = .19$, with greater pupil size to negative images compared to both positive, t (96) = 6.82, $p < .001$, $g_{av} = 0.41$, and neutral images t (96) = 3.58, $p = .001$, $g_{av} = 0.24$. Interestingly, neutral images led to greater pupil diameter compared to positive images, t (96) = -3.21 $p = .002$, $g_{av} = 0.21$.

3.2.2.2 Psychopathy

3.2.2.2.1 Emotional modulation of pupil diameter

I examined whether Tri-PM scores were related to $EI_{Negative}$ and $EI_{Positive}$ (see Table 3.2). Zero-order correlation showed that Boldness, Meanness and Disinhibition were unrelated to $EI_{Negative}$ or $EI_{Positive}$, and multiple linear regressions revealed that the three Tri-PM subscales did not uniquely predict either EI.

Table 3.2 Summary of zero-order correlations and multiple linear regressions run between Triarchic-Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) with each emotional index (EI: emotion minus neutral) for pupil diameter to images.

	Boldness			Meanness			Disinhibition			
	r	*t*	*β*	*r*	*t*	*β*	*r*	*t*	*β*	R^2
$EI_{Negative}$	-.02	-0.17	-.02	-.03	0.17	.02	-.07	-0.66	-.08	.01
$EI_{Positive}$	.02	-0.05	-.01	.05	0.53	.07	-.001	-0.32	-.04	.003

* $p < .05$ (two-tailed test)
β, standardised beta
Correlational analyses, degrees of freedom = 97
Multiple linear regression analyses, degrees of freedom = 3, 93

3.2.2.2.2 Pupil diameter to neutral images

The relationship between Tri-PM subscales and pupil diameter to neutral images was explored across 97 participants using zero-order correlations and multiple linear regressions. All the Tri-PM subscales were unrelated to pupil diameter to neutral images (Boldness, r [97] = .07, p = .51; Meanness, r [97] = -.06, p = .53, Disinhibition, r [97] = .001, p = .99) and a multiple linear regression revealed that Boldness, t (93) = 1.05, p = .30, β = .12, Meanness, t (93) = -1.11, p = .27, β = -.15, and Disinhibition, t (93) = 0.53, p = .60, β = .07, did not uniquely predict pupil diameter to neutral images.

3.2.2.3 Gender analysis

To explore whether there was an interaction between psychopathy and gender predicting each of the pupillary measures, hierarchical multiple linear regressions were run predicting each EI and pupil diameter to neutral images (see Table 0.1 in Appendix 1). A Bonferroni-corrected alpha level was adopted based on the addition of three gender by Tri-PM interactive predictors ($\alpha = .017$). No significant effects of gender or interactions between Tri-PM subscales and gender were found across the pupillary measures, apart from male participants showed larger pupil diameter to neutral images.

3.2.3 Discussion

The present results demonstrated that the subscales of the Tri-PM were unrelated to negative and positive EI in response to images, indicating that psychopathy traits within the current community sample were unrelated to emotional modulation of pupil diameter in response to images. This is contrary to the hypothesis that Boldness and Meanness would be associated with diminished emotional modulation of the pupil to specifically negative images. The current data also showed that the Tri-PM subscales were not associated with pupil diameter to neutral images, thought to indicate normal autonomic reactivity in general, which was predicted. The results were consistent across male and female participants.

Across all participants, larger pupil diameter was observed to negative images as expected, but positive images failed to lead to increased pupil size compared to neutral images contrary to predictions.

3.3 Experiment 7: Static facial expressions

Facial expressions of emotion lead to greater neural activation in the amygdala and increased electrodermal reactivity compared to affective scenes (Hariri et al., 2002). Hariri and colleagues suggest that this may be due to the unique social and biological importance that facial stimuli holds. Indeed, facial affect is considered to characterise

both the individual's emotional response and social communication to observers (Fridlund, 2014), and the relative ease and speed with which complex facial expressions are interpreted indicates a unique stimuli processed by a highly specialised system (Posamentier & Abdi, 2003). This highlights the importance of using facial expressions to explore affective autonomic reactivity.

Affective faces in relation to neutral faces lead to elevated neural activity (Breiter et al., 1996; Hariri, Bookheimer, & Mazziotta, 2000; Hariri et al., 2002; Morris et al., 1996; Whalen et al., 1998) and greater autonomic activity (Fusar-Poli, Landi, & O'Connor, 2009; Jönsson & Sonnby-Borgström, 2003; Williams et al., 2007), which includes pupil dilation (Burkhouse, Siegle, & Gibb, 2014; Farzin et al., 2009; Geangu et al., 2011; Prehn, Kazzer, et al., 2013). Furthermore, negative faces, particularly angry and fearful expressions, compared to positive faces lead to an attentional bias (Fox et al., 2000; Hunnius, de Wit, Vrins, & von Hofsten, 2011; Öhman, Lundqvist, & Esteves, 2001), greater amygdala activation (Breiter et al., 1996; Morris et al., 1996), and elevated electrocortical activity (Leppänen, Kauppinen, Peltola, & Hietanen, 2007). Increased orienting and physiological responsivity to negative faces may further highlight the biological significance of facial expressions as there is an adaptive value in mobilising defensive motivational systems in response to likely signals of danger, such as fearful or angry facial expressions, whereas the motivational significance of happy faces is more ambiguous.

Psychopathy has been associated with poorer recognition of facial affect (see 1.3.2 Impaired emotional processing in psychopathy) with greater impairments for negative emotions (Brook et al., 2013; Dawel et al., 2012). Moreover, Dawel et al. (2012) reported evidence that the interpersonal/affective dimension of psychopathy was specifically associated with an impairment for processing fearful faces, although a lack of relevant studies means that deficits for other emotions cannot be ruled out. Yet little research has examined whether these impairments associated to psychopathy extend to autonomic

responsivity to facial expressions. Blair et al. (1997) reported that high psychopathy adults demonstrated diminished electrodermal responses to distress stimuli that was composed of predominantly distressed faces compared to control participants, although it is not certain that this finding reflects responsivity to facial affect specifically as non-facial stimuli was also presented. To the author's knowledge, no other study has investigated autonomic responsivity during passive-viewing of facial affect, although research has found that the interpersonal/affective dimension of psychopathy is associated to an abnormal neural response pattern to images of facial affect within limbic regions (Carré et al., 2013; Contreras-Rodriguez et al., 2014; Gordon et al., 2004; Han et al., 2012; Jones et al., 2009; Marsh et al., 2008).

For the current study, participants were presented with negative, positive and neutral static facial expressions and pupil diameter was measured in response as a function of Tri-PM scores. Specifically, participants viewed images of fearful, happy, disgusted, angry and sad facial expressions as the current experiment was interested to explore autonomic responses as a function of psychopathy to a range of negative expressions, as well as positive expressions. Individuals high in psychopathy have demonstrated reduced recognition for each facial expression previously (Brook et al., 2013; Dawel et al., 2012).

I hypothesised that emotional facial expressions would lead to larger pupil diameter than neutral facial expressions. I also expected that negative facial expressions would lead to larger pupil diameter than positive facial expressions, consistent with previously described research that has highlighted greater physiological responsivity to negative faces (Breiter et al., 1996; Morris et al., 1996).

My psychopathy predictions paralleled the previous experiment; I expected that Boldness and Meanness would be selectively associated with diminished negative modulation of pupil diameter in response to facial expressions, reflecting autonomic

hypo-responsivity, with no relationship for Disinhibition. Specifically, I predicted that Boldness and Meanness would be negatively related to EI for fearful, disgusted, angry and sad facial expressions, with no effect for Disinhibition across these facial expressions. None of the subscales were expected to be associated to EI for happy facial expressions. I again predicted no relationship between Tri-PM subscales and pupil diameter to neutral faces indicating normal overall autonomic responsivity.

3.3.1 Method

Images of posed facial expression images were selected from the Radboud Faces Database (Langner et al., 2010) consisting of four male and four female actors pulling facial expressions to fit the emotional categories of fear, happiness, neutral, disgust, anger and sadness (48 images in total)[7]. Each actor demonstrated each facial expression and so luminance, contrast and colour were highly similar across valence. Therefore, the original images were presented (i.e. not manipulated). All actors were presented facing forwards and with direct gaze. Likewise to the previous experiment, the static faces were presented for 2000 ms.

3.3.1.1 Data analyses

The data analyses was similar to Experiment 6: Affective images. I calculated mean baseline-corrected pupil diameter over 1000 – 2000 ms post-image onset to investigate emotional modulation. Repeated measures ANOVAs were run to assess whether facial expression significantly affected pupil diameter over this analysis window. Two pupillary measures were calculated to explore pupil response to the images: EI (the difference between mean baseline-corrected pupil diameter in response to fearful, happy, disgusted, angry, or sad faces compared to neutral expressions calculated over 1000 – 2000 ms post image-onset) and pupil diameter to neutral faces (taken to be mean

[7] Actors selected from the Radboud Faces Database were: 01, 02, 03, 04, 05, 07, 09 and 12.

baseline-corrected pupil diameter over the same time-window). I ran zero-order correlations and multiple linear regressions to assess the relationship between Tri-PM subscales and both pupillary measures respectively. I also ran hierarchical linear regressions exploring participant gender and Tri-PM interactions in relation to psychopathy for each pupillary measure (see General Methods, Data analysis).

Seven participants were excluded from the sample for containing too much missing data leaving a total sample of 95 participants (48 female). Spearman-Brown split-half reliability checks revealed good internal consistency for pupil diameter over 1000 – 2000 ms post-image onset (r = .98). Across this sample, 91.04 % of total data during image presentation was defined as valid.

3.3.2 Results

3.3.2.1 Manipulation check

As can be seen in Figure 3.2, the facial expressions caused a PLR similar to the affective images with an approximate latency of 300 ms post-image onset and a maximum constriction of around -0.50 mm which occurred at around 800 ms. The pupil then increased rapidly in size slowing towards image-offset, with a suggestion of a secondary constriction 300 ms after image offset. A fast increase in pupil diameter was then observed until around 3000 ms where the pupil slowly recovered back to baseline pupil diameter at around 4000 ms, before actually exceeding baseline size for the remainder of the trial. Across 95 participants, a repeated measures ANOVA with a factor

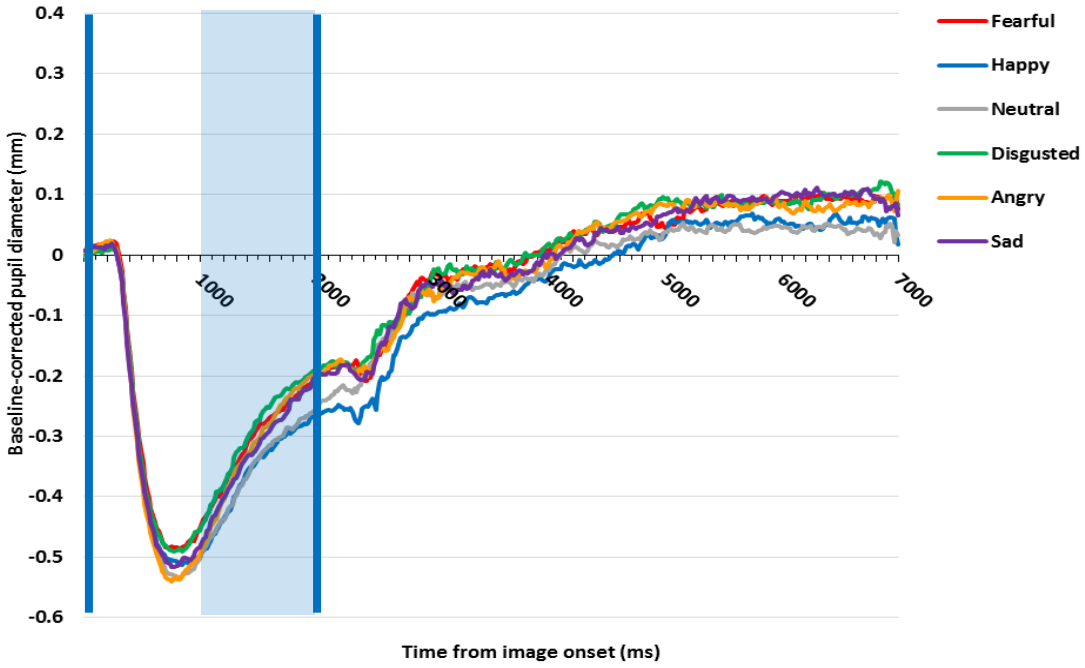


Figure 3.2 Baseline corrected pupil diameter change in response to static fearful, happy, neutral, disgusted, angry and sad facial expressions. The blue bars indicate image onset and offset. Blue shading specifies the time window used for analyses (N = 95).

of emotion (fearful, happy, neutral, disgusted, angry and sad) revealed that there was a main effect of emotion on pupil diameter following the PLR using a Greenhouse-Geisser correction, F (4.48, 421.32) = 4.16, p < .01, η^2 = .04. Planned comparisons revealed that fearful, t (94) = 2.86, p = .01, g_{av} = 0.16, disgusted, t (94) = 3.08, p < .01, g_{av} = 0.19, and angry, t (94) = 2.20, p = .03, g_{av} = 0.13, facial expressions led to significantly greater pupil diameter than neutral faces. Sad expressions, t (94) = 1.83, p = .07, g_{av} = 0.11, and happy

expressions, t (94) = -0.02, p = .98, g_{av} = 0.00, did not lead to significantly larger pupil diameter compared to neutral faces.

3.3.2.2 Psychopathy

3.3.2.2.1 Emotional modulation of pupil diameter

It was explored whether Tri-PM scores were associated with each EI for images of facial affect (see Table 3.3). Zero-order correlations revealed that Boldness, Meanness and Disinhibition were all unrelated to any EI, and multiple linear regressions showed that none of the subscales uniquely predicted the EI for each facial expression.

Table 3.3 Summary of zero-order correlations and multiple linear regressions run between Triarchic-Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) with each emotional index (EI: emotion minus neutral) for pupil diameter to static facial expressions.

	Boldness			Meanness			Disinhibition			
	r	*t*	*β*	*r*	*t*	*β*	*r*	*t*	*β*	R^2
$EI_{Fearful}$	.03	0.10	.01	.00	0.96	.13	-.17	-1.98	-.24	.04
EI_{Happy}	.07	0.18	.02	.11	0.96	.13	.01	-0.51	-.06	.02
$EI_{Disgusted}$	-.04	-0.83	-.10	.05	1.17	.16	-.05	-1.00	-.12	.02
EI_{Angry}	-.01	0.08	.01	-.06	-0.35	-.05	-.05	-0.16	-.02	.00
EI_{Sad}	.03	0.28	.03	-.00	-0.01	-.00	-.03	-0.25	-.03	.00

* $p < .05$ (two-tailed test)
β, standardised beta
Correlational analyses, degrees of freedom = 95
Multiple linear regression analyses, degrees of freedom = 3, 91

3.3.2.2.2 Pupil diameter to neutral static faces

Relationships were explored across the 95 participants using zero-order correlations between Tri-PM subscales and pupil diameter to neutral faces. Zero-order correlations revealed that Boldness, r (95) = -.05, p = .65, Meanness, r (95) = -.03, p = .79, and

Disinhibition, r (95) = .07, p = .54, were unrelated to pupil diameter to neutral facial expressions. A multiple linear regression further revealed that none of the Tri-PM subscales uniquely predicted pupil diameter to neutral faces (Boldness, t [91] = -0.27, p = .79, β = -.03; Meanness, t [91] = -0.52, p = .61, β = -.07; Disinhibition, t [91] = 0.86, p = .39, β = .11).

3.3.2.3 Gender analysis

To investigate whether gender played a role in relation to psychopathy effects, hierarchical multiple linear regressions were run predicting each EI, as well as pupil diameter to neutral faces (see Table 0.2 in Appendix 1). A Bonferroni-corrected alpha level (α = .017) was adopted. There were no significant effects of gender or interactions between Tri-PM subscales and gender across the pupillary measures.

3.3.3 Discussion

Like Experiment 1, the current experiment failed to observe that Tri-PM dimensions were associated with each EI in response to images of facial expressions, suggesting that psychopathy traits within the current community sample were unrelated to emotional modulation of pupil diameter in response to images of facial affect. This contradicts the hypothesis that Boldness and Meanness would be related to diminished emotional modulation of the pupil to specifically negative facial expressions. It was again observed that the Tri-PM subscales were not associated with pupil diameter to neutral facial expressions, thought to be an indicator of general autonomic reactivity, as predicted. The results were consistent across male and female participants.

Across the sample, pupil diameter was larger in response to negative facial expressions as expected, yet happy faces failed to lead to increased pupil diameter compared to neutral faces contrary to predictions.

3.4 Experiment 8: Dynamic facial expressions

Images of facial affect lack ecological validity as real-life facial expressions are dynamic, complex and often subtle. Therefore, research has increasingly utilised dynamic facial expressions that are more representative of real life facial affect. Indeed, studies have found that dynamic facial expressions compared to static expressions lead to improved recognition (Ambadar, Schooler, & Cohn, 2005; Harwood, Hall, & Shinkfield, 1999; Recio et al., 2011; Uono, Sato, & Toichi, 2010), greater autonomic responsivity (Recio et al., 2011; Rymarczyk et al., 2011; Sato et al., 2008; Simons et al., 1999; Weyers et al., 2006) and increased recruitment of more extensive emotion-specific neural networks (Kessler et al., 2011; Kilts, Egan, Gideon, Ely, & Hoffman, 2003; Sato, Kochiyama, Yoshikawa, Naito, & Matsumura, 2004; Trautmann, Fehr, & Herrmann, 2009) suggestive of increased validity.

Psychopathy has been associated to impairments in response to dynamic facial expressions more frequently than to static facial expressions (Brook et al., 2013). To illustrate, psychopathy has been associated with poorer recognition of negative and positive dynamic facial affect (Blair et al., 2001; Blair et al., 2004; Dadds et al., 2008; Dolan & Fullam, 2006; Hastings et al., 2008; Montagne et al., 2005; Pham & Philippot, 2010).

To the author's knowledge, no study has examined the autonomic responsivity during passive-viewing of dynamic facial expressions as a function of psychopathy. However, Decety, Skelly, Yoder, and Kiehl (2014) reported that offenders high in psychopathy, as measured by the Psychopathy Checklist-Revised (PCL-R), showed attenuated neural activation in orbitofrontal (OFC) and ventromedial prefrontal cortex (vmPFC) to dynamic negative and positive facial expressions. These are regions that are involved in affective processing and reciprocally connect to the amygdala, a connection that has reduced structural integrity in individuals high in interpersonal/affective traits (Wolf et al., 2015). However, Decety et al (2014) reported that individuals high in psychopathy displayed normal (and at times even elevated) amygdala reactivity to the dynamic facial stimuli.

Also, no clear association emerged between diminished neural activity in OFC/vmPFC regions and either Factor 1 (interpersonal/affective) or Factor 2 (lifestyle/antisocial) as measured by the PCL-R. However, more research is needed to understand whether psychopathy is associated to autonomic hypo-responsivity in response to dynamic facial expressions.

Previous research utilising dynamic facial stimuli has predominantly used morphed stimuli, where images of neutral faces transition through to a target emotional facial expressions. However, Experiment 8 presented participants with video-clips of facial affect as these are considered to be more realistic than morphed facial expressions, as well as having a clear neutral target facial expression for comparison. Participants viewed negative, positive and neutral dynamic facial expressions, and it was investigated whether participant's Tri-PM scores were associated with pupil diameter in response to dynamic facial affect. The same facial expressions as the previous experiment were presented (fear, happiness, neutral, disgust, anger and sadness), and, therefore, my hypotheses were identical to those made previously.

3.4.1 Method

Forty-eight colour video-clips were selected from the Amsterdam Dynamic Facial Expression Set (ADFES; van der Schalk, Hawk, Fishcer & Doosje, 2011) comprised of four male and four female actors pulling facial expressions to fit the emotional categories of fear, happiness, neutral, disgust, anger and sadness[8]. The videos were presented for 4000 ms and they depicted an actor displaying a neutral face before changing into the target expression at approximately 1300 -1400 ms post video-clip onset. Screenshots were taken from the end of each video-clip and luminance, contrast and colour were

[8] Actors selected from the Amsterdam Dynamic Facial Expression Set were: F01, F02, F03, F05, M02, M03, M04 and M08.

similar across emotions. Therefore, all videos were presented in colour. All actors were presented facing forwards and with direct gaze.

3.4.1.1 Data analysis

The pupillary measures calculated were identical to that described in the previous experiment, although mean baseline-corrected pupil diameter was calculated over an early (2000 – 3000 ms post video-clip onset) and late analysis window (3000 – 4000 ms post video-clip onset) in order to assess the pattern of pupillary response over time. I ran repeated measures ANOVAs to assess whether dynamic facial expressions significantly influenced pupil diameter over the early and late time-window. To assess the association between Tri-PM psychopathy traits and pupil response, I calculated EIs (the difference between mean baseline-corrected pupil diameter in response to fearful, happy, disgusted, angry or sad expressions compared to neutral expressions) over both early and late time-windows, as well as pupil diameter to neutral faces over both time-windows. I ran correlations and multiple linear regressions examining the relationship between Tri-PM subscales and each EI across the early and late analysis windows and pupil diameter to neutral dynamic facial affect. I ran hierarchical linear regressions exploring participant gender and Tri-PM interactions in relation to psychopathy for each pupillary measure (see General Methods, Data analysis).

Ten participants were excluded from the sample for containing too much missing data leaving a total sample of 92 (49 female) participants. Spearman-Brown split-half reliability checks revealed good internal consistency for early (r = .93) and late pupil diameter (.91). Across this sample, 88.72 % of total data during image presentation was defined as valid.

3.4.2 Results

3.4.2.1 Manipulation check

As can be seen in Figure 3.3, the video-clips led to an initial constriction with a latency of 300 ms after video-clip onset and a nadir of around -0.175mm occurring at 700 ms, before the pupil dilated sharply until 1800 ms to approximately 0.08 mm above baseline pupil diameter. At this point, pupil dilation slowed, but continued to increase until video-clip offset at 4000 ms, where there was a delay of 300 ms before a secondary rapid pupil constriction that fell below baseline pupil diameter. The pupil then recovered back to, and actually exceeded, baseline pupil diameter, although Figure 3.3 does not display the full recovery period. It is evident that pupil diameter was larger in response to negative dynamic facial expressions (fearful, disgusted, angry and sad) compared to the neutral and happy facial expressions from 1800 ms post-stimuli onset, which is 300 – 400 ms after the faces begin to express the target emotion. Across 92 participants, repeated measures ANOVAs with the factor of emotion (fearful, happy, neutral, disgusted, angry and sad) revealed that there was no effect of facial emotionality on pupil diameter over the early analysis window using a Greenhouse-Geisser correction, F (4.24, 386.16) = 2.21, p = .06, η^2 = .02, although this approached significance, but that there was a significant main effect of facial affect during the later analysis window using a Greenhouse-Geisser correction, F (4.29, 390.30) = 2.51, p = .04, η^2 = .03. Planned comparisons revealed that there was no difference in pupil diameter between affective and neutral faces during the early time-window (ts [91] 0.47 < 1.71, ps > .09, g_{av} < 0.16), but during the later time window, disgusted, t (91) = 2.16, p = .03, g_{av} = 0.17, angry, t (91) = 2.15, p = .03, g_{av} = 0.18, and sad facial expressions, t (91) = 2.70, p = .01, g_{av} = 0.24, led to significantly greater pupil response than neutral faces. Fearful, t (91) = 1.36, p = .18, g_{av} = 0.14, and happy facial expressions, t (91) = -0.11, p = .91, g_{av} = 0.01, showed comparable pupil diameter to neutral faces.

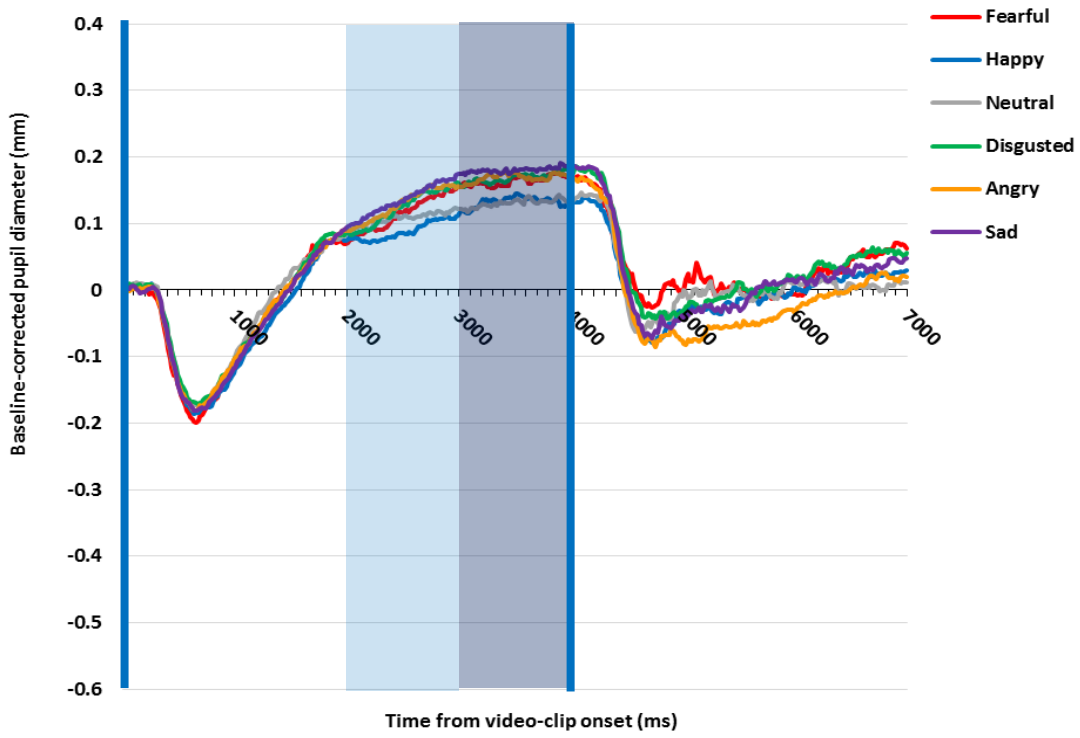


Figure 3.3 Baseline corrected pupil diameter change in response to dynamic fearful, happy, neutral, disgusted, angry and sad facial expressions. The blue bars indicate video-clip onset and offset. Light blue shading specifies the early time window and the dark blue shading indicates the late time-window used for analyses (N = 92).

3.4.2.2 Psychopathy

3.4.2.2.1 Emotional modulation of pupil diameter

Zero-order correlations showed that Boldness, Meanness and Disinhibition were all unrelated to any of the EIs, and multiple linear regressions revealed that the three Tri-PM subscales did not uniquely predict the EIs for dynamic facial affect (see Table 3.4).

Table 3.4 Summary of zero-order correlations and multiple linear regressions run between Triarchic-Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) with each emotional index (EI: emotion minus neutral) for pupil diameter to dynamic facial expressions.

		Boldness			Meanness			Disinhibition			
		r	*t*	*β*	*r*	*t*	*β*	*r*	*t*	*β*	R^2
Early	$EI_{fearful}$	.01	0.18	.02	-.03	-0.05	-.01	-.07	-0.51	-.06	.01

	EI_{Happy}	.11	0.65	.08	.09	0.83	.11	-.05	-0.98	-.12	.03
	$EI_{Disgusted}$	.08	0.42	.05	.07	0.59	.08	-.02	-0.52	-.06	.01
	EI_{Angry}	-.03	-1.09	-.13	.15	1.82	.25	.02	-0.69	-.09	.04
	EI_{Sad}	.06	0.24	.03	.09	0.49	.07	.05	0.09	.01	.01
Late	$EI_{Fearful}$	-.01	0.14	.02	-.05	-0.21	-.03	-.07	-0.47	-.06	.01
	EI_{Happy}	.10	0.83	.10	.05	0.14	.02	-.01	-0.29	-.04	.01
	$EI_{Disgusted}$	.03	0.40	.05	-.02	-0.30	-.04	-.01	0.02	.00	.00
	EI_{Angry}	-.05	-0.84	-.10	.07	0.90	.13	.03	-0.20	-.03	.01
	EI_{Sad}	.06	0.51	.06	.03	0.02	.00	.01	0.02	.00	.00

* $p < .05$ (two-tailed test)
β, standardised beta
Correlational analyses, degrees of freedom = 92
Multiple linear regression analyses, degrees of freedom = 3, 88

3.4.2.2.2 Pupil diameter to neutral dynamic faces

The relationship between Tri-PM subscales and pupil diameter to neutral dynamic stimuli was explored across 92 participants using zero-order correlations and multiple linear regressions. Zero-order correlations showed that Boldness, Meanness, and Disinhibition were not associated with pupil diameter to neutral facial expressions over the early (Boldness, r [92] = -.02, p = .86; Meanness, r [92] = -.06, p = .59; Disinhibition, r [92] = -.01, p = .89) or late time-window (Boldness, r [92] = -.02, p = .83; Meanness, r [92] = -.05, p = .63; Disinhibition, r [92] = -.03, p = .81). Furthermore, none of the Tri-PM subscales were uniquely predictive of pupil diameter to neutral dynamic faces for either time-window (early time-window: Boldness, t [87] = -0.33, p = .74, β = -.04; Meanness, t [87] = -0.95, p = .35, β = -.13; Disinhibition, t [87] = -0.11, p = .92, β = .01: late time-window: Boldness, t [87] = -0.46, p = .64, β = -.06; Meanness, t [87] = -0.84, p = .40, β = -.12; Disinhibition, t [87] = -0.08, p = .94, β = .01).

3.4.2.3 Gender analysis

Hierarchical multiple linear regressions were run to explore the interaction between participant gender and psychopathy for the pupillary measures during the dynamic facial expressions, like the previous experiment (Bonferroni-corrected $\alpha = .017$). As can be seen in Table 0.3 in Appendix 1, there were no significant effects of gender or interactions between Tri-PM subscales and gender on pupillary response, apart from female participants showing greater $EI_{Disgusted}$ over both time-windows.

3.4.3 Discussion

Tri-PM dimensions were again unrelated to each EI in response to dynamic facial expressions, suggesting that psychopathy traits within the current community sample were unrelated to emotional modulation of pupil diameter for dynamic facial affect. This is in contrast to the hypothesis that Boldness and Meanness would be associated to diminished emotional modulation of the pupil to specifically negative facial expressions. The findings paralleled those found for static facial expressions in the previous experiment. As predicted, the Tri-PM subscales showed no association with pupil diameter to neutral images, thought to indicate normal general autonomic responsivity. The results were consistent across male and female participants, apart from female participants showing greater emotional modulation in response to disgusted facial expressions.

Emotional modulation across the sample paralleled the results for the static facial expressions. Negative dynamic expressions caused elevated pupil diameter, as hypothesised, but positive faces failed to lead to increased pupil diameter compared to neutral dynamic faces, contrary to expectations.

3.5 Experiment 9: Affective sound-clips

The auditory modality has received relatively less attention than the visual modality in psychopathy research, despite the belief that auditory cues are largely tapping into a

comparable emotional response (Bradley & Lang, 2000). Research indicates that affective compared to neutral auditory stimuli generates greater amygdala responses (Fecteau, Belin, Joanette, & Armony, 2007; Klinge, Röder, & Büchel, 2010), increased electrodermal responses (Bradley & Lang, 2000; Verona et al., 2004) and larger pupil diameter (Gingras, Marin, Puig-Waldmüller, & Fitch, 2015; Partala & Surakka, 2003). Moreover, auditory stimuli are particularly advantageous when measuring changes in pupil diameter, as this method removes the confounding PLR elicited by visual stimuli allowing the isolation of changes in pupil diameter in response to emotion.

Psychopathy is associated to impairments in processing aural emotional cues likewise to visual affective cues. For example, high psychopathy individuals have been shown to be significantly worse at recognising vocal affect and emotional sentences compared to control participants (Bagley et al., 2009; Blair, Budhani, Colledge, & Scott, 2005; Blair et al., 2002; Stevens, Charman, & Blair, 2001). Dawel et al. (2012) reviewed the literature and reported that psychopathy was associated to a deficit for vocal affect across negative and positive emotions, although the largest effect sizes were found for fearful stimuli. In relation to passive-viewing of emotion, as described previously, Verona et al. (2004) reported that inmates high in interpersonal/affective psychopathy traits according to the PCL-R showed attenuated electrodermal responses to both negative and positive sound-clips (compared to responses to neutral sound-clips). However, only three sounds were played to participants (each repeated three times) and so it could be argued that the effects were specific to this small stimuli set. No other study, to the author's knowledge, has explored autonomic responses to the passive-viewing of affective auditory stimuli in relation to psychopathy.

In the current experiment, participants were presented with negative, positive and neutral sound-clips and participant's pupil diameter was measured in response. Like Experiment 6, the predictions for pupil diameter in response to emotion reflected the accompanying normative arousal ratings, as the pupil is thought to indicate emotional

arousal (Bradley et al., 2008). I expected larger pupil diameter to the negative sound-clips compared to both positive and neutral sound-clips, but no difference in pupil diameter was predicted in response to positive and neutral sound-clips as they were rated equally for arousal.

My psychopathy predictions were similar to the previous experiments. I expected that Boldness and Meanness would be selectively associated with diminished negative modulation of pupil diameter, reflecting autonomic hypo-responsivity, with no relationship for Disinhibition; that is, Boldness and Meanness would be negatively related to EI for negative sound-clips in response to sound-clips, with Disinhibition unrelated to this measure. None of the subscales would be associated to EI for positive sound-clips. I also predicted no relationship between any Tri-PM subscale and the pupil diameter to neutral sound-clips, thought to indicate normal general autonomic responsivity.

3.5.1 Method

I selected 30 sound-clips from the International Affective Digitalised Sounds (IADS)[9] consisting of 10 negative sound-clips (mean valence/arousal = 2.66, 7.25), 10 positive sound-clips (mean valence/arousal = 7.40, 5.52) and 10 neutral sound-clips (mean valence/arousal = 4.90, 5.18). I selected affective sound-clips that were classified as fearful or happy based on Stevenson and James (2008), and neutral sound-clips based on normative valence ratings between 4 and 6 (the middle values on the scale). Like the affective images employed in Experiment 6, I selected sound-clips that were unambiguously negative or positive while the paradigm was continuing to be developed. Again, I did not include erotic stimuli (see 3.2.1 Method for an explanation). Each emotion was significantly different for valence (*p*s < .001). The negative sound-clips had greater arousal ratings compared to both the positive and neutral sound-clips (*p*s < .001), with

[9] IADS sound-clips selected were: Negative: 106, 276, 277, 286, 291, 424, 625, 699, 711, 712; Positive: 110, 151, 220, 226, 230, 353, 810, 811, 815, 820; Neutral: 114, 120, 246, 320, 364, 368, 410, 425, 701, 723.

no difference between positive and neutral sound-clips (p = .33). Sound-clips were matched across emotional stimuli for maximum and average root mean square decibel level *(p*s > .05), and played to all participants at a comfortable set volume through headphones. The sound-clips were presented for 6000 ms and presentation order was randomised. A grey fixation slide was presented throughout the whole task (the same employed in Experiment 6). There was an extended inter-trial interval of 10,000 ms following the sound-clips as preliminary pilot work revealed that a longer period was necessary for the pupil to return to baseline diameter.

3.5.1.1 Data analysis

Mean baseline-corrected pupil diameter was calculated across an early (500 – 1500 ms post sound-clip onset), and a late analysis window (2500 – 3500 post sound-clip onset). The early time-window was selected as this was the period over which emotional modulation begins to occur and the late time-window was selected as this was the period where emotional modulation was largest. Repeated measures ANOVAs were conducted to assess emotion modulation of pupil diameter to sound-clips over the early and late analysis windows.

I calculated EIs (the difference between mean baseline-corrected pupil diameter in response to negative/positive versus neutral sound-clips respectively) and pupil diameter to neutral sound-clips across the early and late analysis windows. Zero-order and multiple linear regressions were run to assess the relationship between Tri-PM subscales and the pupillary measures across both analysis windows. I ran hierarchical linear regressions exploring participant gender and Tri-PM interactions in relation to psychopathy for each pupillary measure (see General Methods, Data analysis).

I removed five participants due to excessive missing data leaving a sample of 98 participants (49 female). Spearman-Brown split-half reliability checks were run revealing moderate internal reliability for early (r = .57) and late pupil diameter (.71) compared to

the previous visual task. This seems likely to be due to the absence of the reliable PLR (Bar, Boettger, Till, Dolicek, & Sauer, 2005; Fotiou, Fountoulakis, Goulas, Alexopoulos, & Palikaras, 2000), which subsequently affects pupil diameter over the analyses windows. I was unable to find internal consistency estimates for pupil responses to emotion within the literature, but there is evidence that galvanic skin response shows only modest test-retest reliability in response to negative emotion (Arena, Blanchard, Andrasik, Cotch, & Myers, 1983; Waters, Williamson, Bernard, Blouin, & Faulstich, 1987). Across this sample, 85.36 % of total data during sound-clip presentation was defined as valid.

3.5.2 Results

3.5.2.1 Manipulation check

Figure 3.4 demonstrates the changes in pupil diameter from baseline across sound-clip presentation. In response to the sound-clips, the pupil began to dilate steadily with a latency of around 300 ms with emotional modulation occurring from approximately 500 ms. Pupil dilation slowed reaching a maximum pupil diameter of 0.175 – 0.3 mm between 2000 – 4000 ms depending on the sound-clip valence with a later peak for the negative sound-clips. The pupil then showed a gradual reduction in diameter although the pupil remained markedly dilated compared to baseline levels at sound-clip offset. The pupil continued to reduce in size but remained above baseline pupil diameter at 1000 ms

following sound-clip offset, although the graph does not display the full recovery period, which continued for a further 7000 ms.

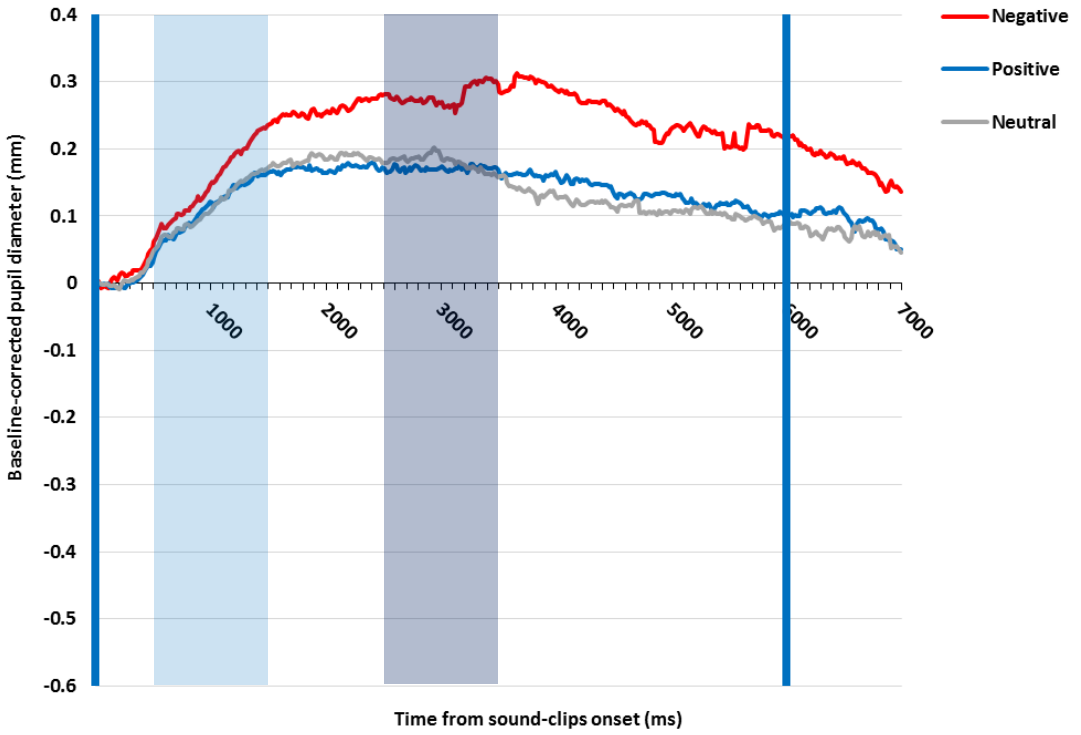


Figure 3.4 Baseline corrected pupil diameter change in response to negative, positive and neutral sound-clips. The blue bars indicate sound-clip onset and offset. Light blue shading specifies the early time window and the dark blue shading indicates the late time-window used for analyses (N = 97).

Across 97 participants, repeated measures ANOVAs with the factor of emotion (negative, positive and neutral) revealed that there was a significant effect of sound-clip emotionality over the early, F (2, 192) = 5.45, $p < .01$, $\eta^2 = .05$, and late time window, F (2, 192) = 22.77, $p < .001$, $\eta^2 = .19$, which was driven by greater pupil diameter to negative sound-clips compared to positive (early, t [96] = 2.93, $p < .01$, $g_{av} = 0.32$; late, t [97] = 5.91, $p < .001$, $g_{av} = 0.60$) and neutral sound-clips (early, t [96] = 3.05, $p < .01$, $g_{av} = 0.36$; late, t [96] = 5.59, $p < .001$, $g_{av} = 0.65$). There was no difference between positive and neutral sound-clips at either time-window (early, t [96] = 0.15, $p = .88$, $g_{av} = 0.02$; late, t [96] = 0.04, $p = .97$, $g_{av} = 0.00$).

3.5.2.2 Psychopathy

3.5.2.2.1 Emotional modulation of pupil diameter

Zero-order correlations and multiple linear regression analyses were run between Tri-PM subscales and $EI_{Negative}$ and $EI_{Positive}$ for sound-clips (see Table 3.5). Tri-PM scores were unrelated to EI for either negative or positive sound-clips and failed to uniquely predict emotional modulation of pupil diameter.

Table 3.5 Summary of zero-order correlations and multiple linear regressions run between Triarchic-Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) with each emotional index (EI: emotion minus neutral) for pupil diameter to sound-clips.

		Boldness			Meanness			Disinhibition			
		r	*t*	*β*	*r*	*t*	*β*	*r*	*t*	*β*	R^2
Early	$EI_{Negative}$	.09	0.85	.10	.02	0.11	.02	-.05	-0.59	-.07	.01
	$EI_{Positive}$	.03	0.62	.07	-.05	-0.88	-.12	.01	0.53	.07	.01
Late	$EI_{Negative}$	-.03	-0.34	-.04	-.03	0.40	.05	-.11	-1.06	-.13	.01
	$EI_{Positive}$	-.03	0.30	.03	-.15	-1.02	-.14	-.12	-0.43	-.05	.03

* $p < .05$ (two-tailed test)
β, standardised beta
Correlational analyses, degrees of freedom = 97
Multiple linear regression analyses, degrees of freedom = 3, 93

3.5.2.2.2 Pupil diameter to neutral sound-clips

Zero-order correlations demonstrated across 97 participants that across the early and late time-windows neither Boldness (early, r [97] = .01, p = .95; late, r [97] = .05, p = .66), Meanness (early, r [97] = .04, p = .73; late, r [95] = .15, p = .15) nor Disinhibition (early, r [97] = .04, p = .69; late, r [97] = .04, p = 70) were related to pupil diameter to neutral sound-clips. Also, none of the subscales were uniquely predictive of pupil

diameter to neutral sound-clips over the early (Boldness, t [93] = -06, p = .95, β = -.01; Meanness, t [93] = 0.18, p = .86, β = .02; Disinhibition, t [93] = 0.24, p = .81, β = .03) or late time-window (Boldness, t [93] = -24, p = .81, β = -.03; Meanness, t [93] = 1.41, p = .16, β = .19; Disinhibition, t [93] = -0.46, p = .65, β = -.06.

3.5.2.3 Gender analysis

Hierarchical multiple linear regressions were run to explore the interaction between participant gender and psychopathy traits predicting EI and pupil diameter to neutral sound-clips over both time-windows (Bonferroni-corrected α = .017). As can be seen in Table 0.4 in Appendix 1, there were no effects of gender or interactions between Tri-PM subscales and gender on pupillary responses.

3.5.3 Discussion

The present results again indicated that the Tri-PM subscales within the current undergraduate sample were unrelated to negative and positive EI in response to sound-clips, suggesting that psychopathy traits within the current community sample were not associated with abnormal emotional modulation of pupil diameter to sound-clips. This is in contrast to the expectation that Boldness and Meanness would be related to diminished emotional modulation of the pupil to specifically negative stimuli. As predicted, it was observed that there was no relationship between the Tri-PM subscales and pupil diameter in response to neutral sound-clips, thought to reflect normal overall autonomic responsivity. The results were consistent across male and female participants.

Across all participants, the pattern of emotional modulation confirmed the predictions. Negative sound-clips led to larger pupil diameter than both positive and neutral sound-clips, with no difference in pupil size in response to positive and neutral sound-clips.

3.6 General experimental discussion

Chapter 3 extended previous emotion and psychopathy research by exploring whether psychopathy traits in an undergraduate community sample were associated with autonomic hypo-responsivity to the passive-viewing of negative and positive images, static facial expressions, dynamic facial expressions and sound-clips. However, the data indicated that psychopathy traits, as measured by the Tri-PM, were unrelated to pupil responsivity to emotional stimuli. Tri-PM scores were also unrelated to general physiological responsivity as measured by pupil diameter to neutral stimuli. Across the sample, pupil size was greater to negative stimuli than neutral stimuli, but positive stimuli was not associated with greater pupil diameter across all four tasks.

3.6.1 Psychopathy and emotional processing

Boldness and Meanness, indicative of interpersonal/affective psychopathy traits, were unrelated to autonomic responsivity to emotional images, static facial expressions, dynamic facial expressions and sound-clips as measured by changes in pupil diameter. This is in contrast to studies that identified that the interpersonal/affective dimension of psychopathy was related to hypo-responsivity to emotion measured through changes in skin conductance response (SCR) (Bate et al., 2014; Benning, Patrick, & Iacono, 2005; Verona et al., 2004). It could be argued that this discrepancy is a result of sample gender as the majority of these studies recruited male participants only, whereas the present sample consisted of both male and female participants; indeed, previous studies that have recruited female participants (Ragsdale et al., 2013; Sutton et al., 2002) have also failed to demonstrate an association between psychopathy and hypo-responsivity to emotional stimuli. Previous research has reported that psychopathy is more prevalent in males (Coid, Yang, Ullrich, Roberts, & Hare, 2009; Coid, Yang, Ullrich, Roberts, Moran, et al., 2009) and there are reported gender differences in the conceptualisation of psychopathy (Forouzan & Cooke, 2005), which may account for specific-gender effects. Yet, the current chapter found no interaction between participant gender and Tri-PM

scores in predicting emotional modulation of pupil diameter, suggesting that psychopathy was unrelated to emotional responsivity across both male and female participants.

Emotional deficits in relation to psychopathy are typically thought of as difficult to detect (Wilson, Juodis, & Porter, 2011), with particularly small effect sizes in relation to autonomic reactivity (Brook et al., 2013) and this seems particularly pertinent within community samples where levels of psychopathy are lower than offender or clinical samples. To overcome this challenge in community populations, researchers have pre-screened larger samples to include high-psychopathy individuals. Both Benning, Patrick, and Iacono (2005) and Zimak et al. (2014) increased the representation of psychopathy within their community sample by pre-selecting high psychopathy individuals and reported that psychopathy was associated to attenuated SCRs to affective images. In contrast, Ragsdale et al. (2013) failed to screen community participants for high/low psychopathy traits and found that Psychopathic Personality Inventory - Revised (PPI-R) total and factor scores were unrelated to electrodermal responses to affective images. The present chapter similarly did not screen for high psychopathy individuals, which may have limited the representation of psychopathy within the sample and, therefore, restricted the present study's capabilities of detecting affective deficits as a function of psychopathy. In support of this explanation, the participant sample in the current chapter showed reduced Boldness and Meanness scores compared to the majority of previous samples (Almeida et al., 2015; Snowden et al., 2013; Stanley et al., 2013; Vieira et al., 2014; Vieira et al., 2015). Alternatively, Esteller et al. (2016) reported comparable Tri-PM subscales to the present study and identified that Boldness was predictive of deficient threat-potentiated startle responses, although it is important to recognise that impaired fear-potentiated startle responses is one of the most consistently identified findings within the psychopathy literature in contrast to autonomic hypo-responsivity to passive-viewing of emotion (Brook et al., 2013). Therefore, researchers may need to explore the extreme end of the psychopathy spectrum to identify autonomic hypo-

responsivity to emotion. Indeed, Coid and Yang (2008) reported that, within a large British community sample, psychopathy was a dimensional construct until a threshold where there was a dramatic increase in social and behavioural difficulties, and it could be argued that the same threshold may exist for affective deficits.

3.6.2 Emotional stimuli and pupil diameter

Looking across the tasks, negative images, static facial expressions, dynamic facial expressions and sound-clips led to larger pupil diameter compared to neutral stimuli, whereas the positive stimuli failed to produce significant dilation (across all experiments). This is consistent with previous psychophysiological studies that identified that negative affect caused elevated autonomic responses compared to positive affect (not including erotic stimuli) typically unless stimuli were matched for subjective arousal (Bradley, Codispoti, Cuthbert, et al., 2001; Bradley, Codispoti, Sabatinelli, & Lang, 2001; Most, Smith, Cooter, Levy, & Zald, 2007; Sarlo, Palomba, Buodo, Minghetti, & Stegagno, 2005; Weinberg & Hajcak, 2010). Indeed, there is evidence that the amygdala, which is central to the generation of autonomic reactivity to emotion (including pupil responses) (Applegate, Kapp, Underwood, & McNall, 1983; Bechara, Tranel, Damasio, & Adolphs, 1995; Furmark, Fischer, Wik, Larsson, & Fredrikson, 1997; Gloor, 1997; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007; Ursin & Kaada, 1960; Williams et al., 2001), demonstrates preferential responsivity to negative compared to positive affect (Adolphs, Tranel, Damasio, & Damasio, 1994, 1995; Adolphs et al., 1999; Breiter et al., 1996; Calder, 1996; Canli, Sivers, Whitfield, Gotlib, & Gabrieli, 2002; Hamann et al., 1996; Somerville, Kim, Johnstone, Alexander, & Whalen, 2004). Further, as the amygdala has been proposed as an encoder of the representation of value (Morrison & Salzman, 2010), this suggests that positive affect may fail to hold the same motivational importance as negative affect.

Previously, it has been suggested that pupil diameter reflects the degree of emotional arousal (Bradley et al., 2008). Consistent with this, previous studies that have identified elevated pupil size to both negative and positive images (Bradley et al., 2008; Dietz, Bradley, Okun, & Bowers, 2011; Jin et al., 2015) and sound-clips (Partala & Surakka, 2003) have employed stimuli matched for subjective arousal ratings. However, Experiment 6 observed comparable pupil diameter in response to positive and neutral images despite positive images being more subjectively arousing. This suggests that emotional arousal, as indexed by pupil diameter, and subjective arousal are not equivalent. Consistent with this idea, Henderson et al. (2014) found that erotic images led to greater pupil diameter than violent images despite both images sets being matched for subjective arousal ratings. Additionally, Weinberg and Hajcak (2010) used electromyography to highlight several discrepancies between self-reported arousal and physiological responses to emotion, arguing that physiological reactivity is determined not only by perceived arousal to an affective stimulus, but also by the motivational significance of that stimulus. For example, they found that erotic images led to brain potentials disproportionately larger than those elicited by affiliative (e.g. cuddly animals, smiling faces) and exciting images (e.g. exciting sports) despite similar subjective arousal. They suggest that exciting and affiliative images do not convey survival-relevant information and fail to trigger motivational systems. This may explain the failure to observe an autonomic advantage to the positive compared to the neutral images in Chapter 6 as solely affiliative positive images were employed. Furthermore, it has been found that substantial increases in SCR only occur in response to the most arousing images (Bradley, Codispoti, Cuthbert, et al., 2001) suggesting that there is a threshold for motivation activation, which the current positive images may not have surpassed.

Positive and neutral affect for both static and dynamic facial stimuli led to comparable pupil diameter, despite the expectation that happy faces would induce greater autonomic responsivity than neutral expressions. This mirrors the neural reactivity of the amygdala

to facial expressions, as this region has been found to be more responsive to negative expressions compared to positive faces (Morris et al., 1996). Research indicates that happy faces are less reliant on the amygdala as patients with bilateral amygdala lesions show deficits in recognising negative faces, but no deficits in the recognition of happy faces (Adolphs et al., 1994; Adolphs et al., 1999; Calder, 1996). The amygdala may be less central to the processing of happy faces as they do not hold motivational relevance to the individual (Morrison & Salzman, 2010) in comparison to fearful or angry faces that may signal immediate danger. An additional explanation is that neutral faces can be evaluated as negative due to an implicit social expectation for a positive facial expression (Lee et al., 2008; Thomas et al., 2001). This is particularly pertinent in response to dynamic faces where a lack of movement conveys negative emotion (Wallbott & Ricci-Bitti, 1993). It could be speculated that this perceived negativity could have caused a degree of autonomic responsivity that led to comparable pupil diameter between positive and neutral facial affect.

3.6.3 Considerations and next steps

There were several aspect of the current experimental design that were modified for Chapter 4. Firstly, Chapter 4 was concerned to increase the levels of psychopathy within the sample and, therefore, applied the current paradigm to a forensic psychiatric population where there were higher levels of psychopathy than typically found in community populations. Furthermore, in the present chapter, subjective arousal was not matched between negative and positive images/sound-clips because the current chapter wanted to select stimuli that were unambiguously negative or positive, while the paradigm was being developed. However, it was now felt that the paradigm was effectively indexing autonomic responses to emotion, and so negative and positive images/sound-clips were matched for subjective arousal ratings for a fairer comparison in the following chapter. However, erotic stimuli were still not presented, which is frequently used to match positive to negative stimuli.

Neutral images led to an unexpected attenuated PLR response in Experiment 6 (see Figure 3.1), despite image luminance, contrast and colour being matched across images. It is not immediately apparent why this reduced constriction occurred, but it is speculated that differences in local luminance and contrast may have led to changes in pupil diameter as has been shown in simple stimuli previously (Binda, Pereverzeva, & Murray, 2014; Ukai, 1985; Wang, Boehnke, Itti, & Munoz, 2014 2014). Therefore, the neutral images for the following chapter were changed to stimuli more similar to those employed by Bradley et al. (2008).

Additionally, the current chapter demonstrated a similar pattern of emotional modulation across static and dynamic facial expressions, but pupil diameter to dynamic facial expressions was elevated, considered to reflect greater arousal. This perhaps reflects the dynamic facial stimuli's greater ecological validity. Given time considerations, it was sensible to assess pupil reactivity to dynamic faces only within the forensic psychiatric sample. Also, it was decided to reduce the range of facial expressions presented from six to four in order to keep the dynamic faces task as brief as possible.

In addition, this chapter highlighted that men showed greater psychopathy traits, which is a typical finding in the literature within both offender and community populations (Coid, Yang, Ullrich, Roberts, & Hare, 2009; Coid, Yang, Ullrich, Roberts, Moran, et al., 2009). Therefore, only male participants were recruited in the following chapter.

Overall, the current chapter has demonstrated that each of the Tri-PM subscales within a community sample were unrelated to emotional modulation of pupil diameter in response to emotional images, static facial expressions, dynamic facial expressions and emotional sound-clips. Furthermore, it was found that the Tri-PM dimensions were not associated with general autonomic responsivity, as measured by pupil diameter to neutral sound-clips. It was suggested that the undergraduate sample contained an under-representation of psychopathy traits, which may have limited the ability to detect

autonomic hypo-responsivity associated to psychopathy. Therefore, the following chapter repeated the current experimental design (with the above-discussed improvements) within a forensic psychiatric sample where there would be higher levels of psychopathy.

4 Chapter 4: Psychopathy and pupil response to emotion within a forensic psychiatric sample

Abbreviation	Meaning	Page
DAAM	Differential Amygdala Activation Model	150
EI	Emotional index	128
IADS	International Affective Digitised Sounds	139
IAPS	International Affective Picture System	128
PCL-R	Psychopathy Checklist-Revised	118
PLR	Pupil light reflex	130
RMH	Response Modulation Hypothesis	150
WASI	Wechsler Abbreviated Scale of Intelligence	124
WAIS	Wechsler Adult Intelligence Scale	125

Chapter 3 failed to find that psychopathy within a community population was associated to deficient autonomic responding to emotion as measured by changes in pupil diameter, although this may have been due to a lack of psychopathy within the community sample. Indeed, most empirical research relating to psychopathy has been undertaken within forensic samples where the levels of psychopathy are higher. Further, offenders are accompanied by large amounts of collateral file information that allows for examination of psychopathy using clinical rating (see 1.2.3 Self-report versus clinically-rated measures of psychopathy). Therefore, Chapter 4 repeated the experimental design from Chapter 3 within a male forensic psychiatric sample where there would be greater levels of psychopathy. There were several further minor improvements to the experimental design (see 3.6.3 Considerations and next steps). Therefore, participants passively viewed emotional (negative and positive) and neutral images, dynamic facial expressions, and sound-clips. The Psychopathy Checklist-Revised (PCL-R) was conducted for each participant as this is the most extensively validated and widely-used assessment of psychopathy (Hare, 2003). Psychopathy was explored within a 2-factor model given the increasing body of research that indicates the distinctive nature of the psychopathy dimensions (see 1.2.2.1 Dual pathway model of psychopathy) with emotional impairments primarily linked to Factor 1 representing interpersonal/affective psychopathy traits (see 1.3.3 The dimensions of psychopathy and emotional processing).

4.1 General methods

4.1.1 Participants

Eighty-two male participants were recruited from low and medium secure forensic psychiatric hospitals in South Wales and Cambridgeshire. Table 4.1 describes the participant sample characteristics. Participant sample size was based on an *apriori* power calculation for a linear multiple regression including two predictor variables (Factor

1 and Factor 2) with 90% power (α = .05) to detect a medium effect size (f^2 = 0.15). The recommended sample size was 88 participants, which was not quite reached due to time and practical constraints.

Participants were eligible to take part in the study if they had the ability to give informed consent, were not currently psychotic and had an IQ above 70. The responsible clinician for each patient made this judgement. IQ was also assessed as part of the research procedure (see 4.1.4 Wechsler Abbreviated Scale of Intelligence). All participants were free from any documented history of head injury (defined as a loss of consciousness for more than one hour, Kiehl et al, 2001), spoke English as their first language and had normal/corrected-to-normal vision. There was a diversity of index offences across the sample (see Table 4.1). Furthermore, the majority of the sample were white (see Table 4.1). Participants gave written informed consent to participate in the experimental procedures and for the research team to access their hospital patient file, and they were paid for their participation. All participants were debriefed fully to the research aims. All experimental procedures were given NHS ethical approval (REC reference: 14/SC/1198).

Medication has been shown to have an effect on autonomic activity disrupting the balance of sympathetic and parasympathetic regulation. For example, anti-psychotic medication, such as clozapine, have been linked to autonomic dysregulation (Agelink et al., 2001; Cohen, Loewenthal, Matar, & Kotler, 2001; Iwamoto et al., 2012) although this is limited to cardiac function. Anti-anxiety medication seek to target the subjective and physical symptoms of stress and anxiety through sedative properties. Therefore, it is not surprising that anti-anxiety medications, such as lorazepam, have been reported to attenuate electrodermal responses to negative stimuli (Agelink, Majewski, Andrich, & Mueck-Weymann, 2002; Siepmann et al., 2007). In addition, anti-depressant medication have been linked to autonomic dysregulation measured through skin conductance levels and heart-rate variability (Kemp et al., 2010; Siepmann, Grossmann, Mück-Weymann, &

Kirch, 2003; Zimmermann-Viehoff, Kuehl, Danker-Hopfe, Whooley, & Otte, 2014), although there is an indication that cardiac autonomic dysfunction may be specific to the tricyclic form of anti-depressants (Kemp et al., 2010; Zimmermann-Viehoff et al., 2014). Therefore, it is important to consider medication dosage within the sample and explore any potential effects on pupil response. This is discussed further in the paragraph below and in 4.1.6 Confounding variables).

Within the sample, I recorded whether participants were taking anti-psychotic, anti-anxiety and anti-depressant medication with 32.90 % of the sample being medication-free. Only active metabolites were recorded based on the maximum half-life window. Across the sample, 61.00 % of the participants were taking anti-psychotic medication (atypical only = 46.34 %; typical only = 10.98 %; both atypical and typical = 3.66 %), 26.80% of the sample were taking benzodiazepines, while 20.70% of participants were taking anti-depressant medication. I converted each medication type into standardised units using equivalent dosages in order to assess the influence of medication (discussed later in 4.1.6 Confounding variables). Dosages of anti-psychotic medication were converted into standard units of chlorpromazine according to equivalent dosages described by Andreasen, Pressler, Nopoulos, Miller, and Ho (2010)[10]. I quantified anti-anxiety medication into standardised valium units according to equivalent dosages (Ashton, 2002; "Benzodiazepine equivalence table," 2007)[11]. Moreover, anti-depressant

[10] I quantified flupenthixol decanoate, amisulpride, zuclopenthixol and zuclopenthixol decanoate into standard chlorpromazine units based on Bazire (2014) and Danivas and Venkatasubramanian (2013) as these anti-psychotic medication were not included in Andreasen et al. (2010).

[11] Two patients were taking buspirone, which I was unable to convert to standardised units due to differing mechanism of action compared to benzodiazepines (Loane & Politis, 2012). However, I left the participants within the sample as buspirone is not associated with side-effects of sedation or cognitive and psychomotor impairment unlike benzodiazepines.

medication was measured by converting dosages into standardised fluoxetine units according to equivalent dosages identified by Hayasaka et al. (2015)[12].

Participant's previous alcohol and substance use was categorised from their collateral patient file information with 68.30/64.70 % of participants showing previous alcohol/substance abuse prior to admission (20.70 % showed no history of alcohol/substance abuse). Random drugs tests carried out by the hospitals during the research period were negative for all cases.

I also examined mental health diagnoses within the sample with 48.80 % diagnosed with schizophrenia, schizotypal and delusional disorders, 9.80 % with a mood disorder, 7.30 % with a neurotic, stress-related and somatoform disorder, as well as 63.40 % with a personality disorder (see Table 4.1 for a more detailed breakdown). However, 26.80 % of the participants had comorbid mental health diagnoses, the majority of which was a combination of personality disorder and another mental health diagnosis (see Table 0.2 in Appendix 2 for further information).

I additionally explored PCL-R total and factor scores as a function of previous alcohol/substance use, as well as mental health diagnoses (see Table 0.1, 0.2 and 0.3 in Appendix 2). Table 0.1 showed that participants who have previously abused alcohol or substances showed higher Factor 2 scores compared to individuals without a history of alcohol or substance abuse. Additionally, individuals with a personality disorder showed higher PCL-R total and factor scores compared to other diagnoses, which was unsurprisingly linked to a diagnosis of dissocial personality disorder (see Tables 0.2 and 0.3).

Table 4.1 Summary of participant sample characteristics

[12] No study has explored equivalent dosage to date for citalopram (Hayasaka et al., 2015), which three of the patients were taking and so I used an equivalent dosage of 30 mg (in relation to 40 mg of fluoxetine), which was based on the median of the recommended target dosage range.

	Mean	SD	Range
Age	38.55	12.84	21 - 75
IQ	89.31	12.48	70 - 115
PCL-R total	19.29	8.35	1.10 – 37
Factor 1	7.00	4.47	0 – 16
Factor 2	10.70	4.48	0 – 20
Medication			
Standardised chlorpromazine dosage (mg)	197.17	218.57	0 – 839.00
Standardised valium dosage (mg)	2.84	6.76	0 – 40.00
Standardised fluoxetine dosage (mg)	8.66	19.59	0 – 81.20
Highest educational qualification achieved [a b]	*n*		
None	38		
NVQ Level 1	11		
GCSE	19		
AS/A-Level	7		
Bachelor's degree	4		
Postgraduate degree	3		
Index offence	*n*		
Arson	6		
Assault and grievous bodily harm [b]	26		
Breach of order	5		
Criminal damage	1		
Drug offences	2		
Kidnapping/false imprisonment	1		
Murder and manslaughter [c]	6		
Possession of a weapon	4		
Sexual offenses	23		
Theft	8		
Previous alcohol/substance abuse	*n*		
Alcohol abuse	56		
Substance abuse	53		
Mental health diagnoses	*n*		
Schizophrenia, schizotypal and delusional disorders			
Schizophrenia	29		
Persistent delusional disorder	4		
Schizoaffective disorder	3		
Schizotypal personality	2		
Unspecified nonorganic psychosis	2		

Mood disorders			
Bipolar affective disorder	2		
Depressive disorder	4		
Mania with psychotic symptoms	1		
Unspecified mood disorder	1		
Neurotic, stress-related and somatoform disorders			
Obsessive compulsive disorder	3		
Post-traumatic stress disorder	4		
Personality disorder			
Emotionally unstable personality disorder	19		
Dissocial personality disorder	37		
Mixed personality disorder	5		
Other specific personality disorders	1		
Paranoid personality disorder	4		
Schizoid personality disorder	2		
Ethnicity	*n*		
White	77		
Black	4		
Asian	1		

[a] Older qualifications were categorised equivalent to their contemporary level
[b] Including threats
[c] Including attempted murder

4.1.2 Design

The experimental procedure was identical to the previous chapter apart from the absence of the task presenting static facial expressions (see 3.1 General methods). Examples of the stimuli presented, as well as a schematic illustration of the structure of each trial, are detailed in Appendix 3. Participant completed questionnaires after the dynamic faces task, but these are not reported in the current thesis.

4.1.3 Psychopathy Checklist-Revised

Psychopathy was measured using the PCL-R. Across the sample, 31.70 % of participants had previously been assessed using the PCL-R by trained clinical/forensic psychologists within the last 5 years. The remaining 68.30 % of participants were

assessed on the PCL-R through a collateral review of patient file information following the research session. The rater (D.B.) was supervised and extensively trained in the use of the PCL-R by Professor Robert Snowden and is validated as a reliable assessor with total PCL-R ratings falling within one standard error of measurement compared to official (Darkstone) target ratings (inter-class correlation = .87). It is important to recognise that there are limitations to conducting the PCL-R by file-review alone, although considerable research indicates that reliable and valid PCL-R ratings can be made on the basis of file information alone (Hare, 2003). Grann, Langstrom, Tengstrom, and Stalenheim (1998) demonstrated that PCL-R scores from file review alone were highly correlated (.88) with scores obtained from file review and semi-structured interview. Moreover, the current sample showed PCL-R total and factor scores in line with previous research conducted in forensic psychiatric samples (Hare, 2003):

Hare (2003) reported that PCL-R total scores show high inter-rater reliability (standard procedures = .88) and internal consistency (standard procedures =.81, file-review only = .89) within forensic psychiatric patients. File-reviews typically only involve one rater and so no estimate was calculated for interrater reliability although data from Firestone, Bradford, Greenberg, and Larose (1998) and Harris, Rice, and Cormier (1991) indicate high interrater agreement. Additionally, within forensic psychiatric patients both Factor 1 and Factor 2 demonstrate good interrater reliability (Factor 1 = .79; Factor 2 = .87) and internal consistency (Factor 1 = .79; Factor 2 = .73) for standard procedures and high internal consistency for file-review alone (Factor 1 = .80; Factor 2 = .82) (Hare, 2003). Again, data was not available for inter-rater reliability for the PCL-R factors based on file-review alone given that these typically involve only one rater.

4.1.4 Wechsler Abbreviated Scale of Intelligence

Intelligence was measured using the two-subtest form of the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999), which is a shortened version of the

Wechsler Adult Intelligence Scale (WAIS-III; Wechsler, 1997) that gives an estimate of full-scale IQ. Scores on the shortened WASI have been found to correlate with the WAIS-III (Axelrod, 2002; Wechsler, 1999; Zhu, Tulsky, & Leyva, 1999). Twenty-six participants had previously completed the WAIS-III or WAIS-IV (Wechsler, 2014) and so this score was recorded from their patient notes as a more comprehensive assessment of intelligence. IQ scores were similar across WASI (M = 90.14, SD = 12.88) and WAIS-III/WAIS-IV (M = 87.50, SD = 11.60) with no difference between scores, t (80) = 0.89, p = .38, d = 0.20.

4.1.5 Data acquisition and analyses

Data acquisition and cleaning was identical to that described in Chapter 2 (see 2.2.3 Pupil data acquisition and cleaning). Also, the pupillary measures calculated were identical to that described in Chapter 3 (see 3.1.4 Data acquisition and analyses). The experiment took place within a dim and quiet room within each hospital. Zero-order/partial correlations and multiple linear regressions were run exploring the relationship between PCL-R total, Factor 1 and Factor 2, with EIs for each valence (or emotion), as well as pupil diameter to neutral stimuli for each task.

The assumptions of linear regression (linear relationship, lack of multicollinearity, no autocorrelation, homoscedasticity and multivariate normality) were not violated across each variable. Linearity was established through visual inspection of plots between predicted residuals and observed residuals. There was little evidence of multicollinearity between Factor 1 and Factor 2. The two factors were positively correlated (see 4.2.2 Results), but I observed a variance inflation factor of 1.24 < 1.26 (across tasks) indicating this assumption was not violated (Craney & Surles, 2002). Auto-correlation was assessed using the Durbin-Watson's d test finding that the residuals were not linearly auto-correlated (1.70 < d < 2.36). To test homoscedasticity, I observed that the error terms along the regression line appeared equal with no obvious pattern emerging

suggesting that the assumption was not violated. Finally, I explored multivariate normality within the data using boxplots, histograms and normal probability plot, which visually demonstrated normally distributed data. I also conducted the Kolmogorov-Smirnov test, which showed that the majority of the pupil measures were normally distributed (*p*s > .05). However, several of the pupillary measures did violate the Kolmogorov-Smirnov test and these variables were log10-transformed[13]. Meaningful untransformed data is reported for means and standard deviations. There were no differences in results using log transformed data. The PCL-R scores did not violate the assumption of autocorrelation and showed normal distribution. Two-tailed significance values are reported for all effects for simplicity.

4.1.6 Confounding variables

Confounding variables were again considered to be variables that related to *both* PCL-R scores (whether this was total score or either factor) and the respective pupillary measures (Meinert, 2012). Confounding variables were controlled for within subsequent statistical analysis; the specific analyses are reported within each experiment's method.

I found that chlorpromazine dosage was negatively associated with PCL-R total, r (82) = -.28, p = .01, and Factor 1, r (82) = -.36, p = .001, and valium dosage was positively associated with Factor 2, r (82) = .27, p = .01, as well as a number of the pupillary measures. Therefore, I controlled for chlorpromazine and valium dosage respectively where these medications were related to pupillary measures also (see each experiment's method for the specific analyses). Fluoxetine dosage was unrelated to PCL-R scores (*p*s > .44) and was not controlled for across any reported analyses. Similarly, the centred

[13] The measures that were log10 transformed were pupil diameter to neutral images and $EI_{Positive}$ for the images, EI_{Fear} (early and late time-window) and EI_{Happy} (late time-window) for the dynamic facial expressions, as well as $EI_{Negative}$ (late time-window) for the sound-clips.

interactions between medications were also unrelated to PCL-R scores (*p*s > .14) and so were not controlled for in future analyses.

Participant age was positively related to Factor 1, r (82) = .29, p = .01, but showed no association to any pupillary measures across tasks (*p*s > .09) and so was not controlled for in subsequent statistical analysis.

Lastly, IQ was negatively related to Factor 2 only, r (82) = -.26, p = .02, and a single pupillary measure from the dynamic faces task. There is a debate about the validity of treating IQ as a covariate within experimental designs as intelligence reflects the culmination of an individual's genetics, biology, cognition, education and experiences (Dennis et al., 2009). However, as there was only a single pupillary measure that related to IQ, I have reported the analysis with and without IQ controlled for.

The subsequent findings across pupillary measures were not altered by controlling for individual differences in initial pupil diameter and, therefore, I did not consider initial pupil diameter further (see 3.1.5 Confounding variables for a further explanation).

4.2 Experiment 10: Affective images

Participants were presented with emotional (negative and positive) and neutral images and their pupil diameter was measured in response as a function of PCL-R scores. The affective images were matched across negative and positive images for subjective arousal. Given that pupil response is thought of as a measure of emotional arousal (Bradley et al., 2008), this allowed pupil responsivity to be compared across negative and positive stimuli in relation to psychopathy more equally. In addition, the neutral images were changed from the previous chapter to neutral stimuli more similar to those employed by Bradley et al. (2008).

I expected that negative and positive images would lead to larger pupil diameter compared to neutral images reflecting greater emotional arousal (Bradley et al., 2008; Henderson et al., 2014).

I made several predictions in relation to psychopathy that paralleled those made in the previous chapter considering that Factor 1 of the PCL-R indexes the interpersonal/affective dimension of psychopathy. I predicted that Factor 1 would be selectively associated with diminished emotional modulation of pupil diameter, reflecting autonomic hypo-responsivity, in response to specifically negative images, with no relationship expected for Factor 2. That is, I predicted that Factor 1 would be negatively related to emotional index (EI) for negative images with no association for Factor 2, and neither factor was hypothesised to be associated to EI for positive images. Total PCL-R scores were expected to be associated to diminished emotional modulation of pupil diameter to negative images, as a consequence of Factor 1. Finally, I predicted that PCL-R scores would be unrelated to pupil responsivity to neutral images reflecting normal overall autonomic responsivity for psychopathy.

4.2.1 Method:

Thirty images were selected from the International Affective Picture System (IAPS), specifically 10 negative (mean valence/arousal based on IAPs normative ratings = 2.91, 6.20), 10 positive (mean valence/arousal = 7.60, 5.89) and 10 neutral images (mean valence/arousal = 5.17, 3.10) were chosen[14]. No erotic images were used. Each emotion differed significantly for subjective valence (*p*s < .001). There was no difference between negative and positive images for subjective arousal, t (18) = 1.34, p = .20, g_{av} = 0.58, with both showing greater subjective arousal than neutral images (negative: t [18] = 16.59, p

[14] Images selected were: Negative: 1301, 1304, 1525, 5973, 6231, 6250, 6242, 6263, 6370, 9901; Positive: 1710, 2347, 4599, 4641, 7330, 8200, 8370, 8380, 8470, 8490; Neutral: 2036, 2190, 2214, 2383, 2393, 2514, 2745.1, 2850, 2870, 5731. These images were selected based on O'Farrell, Gray, and Snowden (In preparation).

< .001, g_{av} = 7.20; positive: *t* [18] = 13.47, *ps* < .001, g_{av} = 5.92). All images were presented in grey-scale and manipulated to the median luminance value of 95 for the sample (see 2.2.2 Materials and design for scale). Contrast (M = 61.60, SD = 7.31) was manipulated to match across valence categories (*p* = .14). Pictures were displayed on a 39.60 cm laptop display monitor with a screen resolution of 1920 x 1080 and each participant was sat 57 cm from the screen.

Experimental design was identical to that described in the previous chapter (see 3.2.1 Method).

4.2.1.1 Data analyses

Data analysis was identical to the previous affective images task (see Chapter 3, Experiment 1: Affective images, Data analysis), except PCL-R total, Factor 1 and Factor 2 were entered into associative and predictive models.

Spearman-Brown split-half reliability checks revealed good internal consistency for pupil diameter over 1000 – 2000 ms post-image onset (*r* = .96). Three participants were excluded from the sample for containing too much missing data leaving a sample of 79 participants. The percentage of possible valid data collected during stimulus presentation was 92.51 %.

4.2.1.2 Confounding variables

I described previously that a number of variables were related to PCL-R scores (see 4.1.6 Confounding variables). Chlorpromazine was inversely associated with PCL-R total and Factor 1, while valium dosage was positively related to Factor 2. Chlorpromazine dosage was also positively associated with pupil diameter in response to neutral images (chlorpromazine, *r* [79] = .31, *p* = .01). Therefore, in subsequent statistical analyses examining the effect of psychopathy on the pupil diameter to neutral images, I controlled for chlorpromazine dosage in relation to PCL-R scores. Chlorpromazine and valium

dosage were unrelated to any further pupillary measures (ps > .09) and were, therefore, not included in statistical analyses for these pupillary indexes.

4.2.2 Results

Table 4.1 shows the mean values for total PCL-R scores and factor scores. Factor 1 was positively correlated to Factor 2, r (82) = .44, p < .001.

4.2.2.1 Manipulation check

As can be seen in Figure 4.1, there was a pupil light reflex (PLR) at image onset with a latency of approximately 300 ms that reached a nadir around -0.30 mm at around 1000 ms. Following this PLR, pupil diameter steadily increased over the remaining image presentation and after image-offset for a further 600 ms, where there was a sharp increase in pupil diameter back to baseline pupil size. Over the remaining recovery period, pupil diameter appears to reach a constant pupil size slightly above baseline pupil diameter.

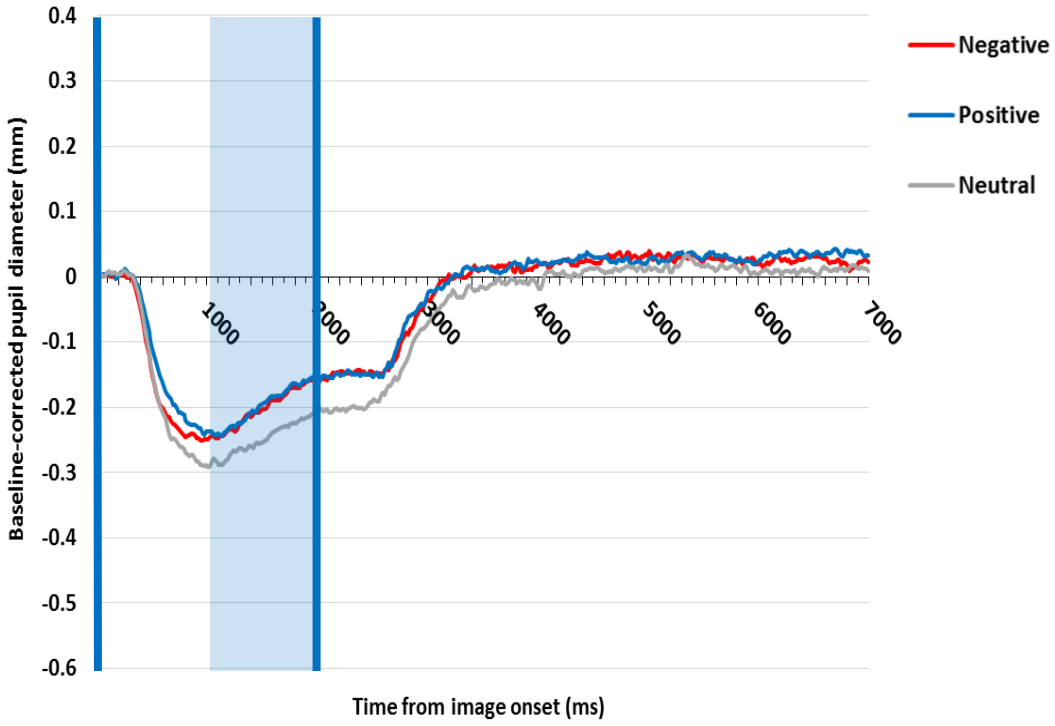


Figure 4.1 Baseline-corrected pupil diameter in response to unpleasant, pleasant and neutral images. The blue bars indicate image onset and offset. Blue shading specifies the time window used for analyses (N = 79).

Across 79 participants, a repeated measures ANOVA with the factor of emotion (negative, positive and neutral) over the analysis window (1000 – 2000 ms post image onset) showed a significant effect of image emotionality, F (2, 156) = 15.66, $p < .001$, $\eta^2 = .17$, with planned comparison t-tests identifying larger pupil diameter observed in response to negative and positive images compared to neutral images (negative, t [78] = 4.23, $p < .001$, $g_{av} = 0.24$; positive, t [78] = 5.46, $p < .001$, $g_{av} = 0.27$).

4.2.2.2 Psychopathy

4.2.2.2.1 Emotional modulation of pupil diameter

PCL-R scores were examined in relation to each EI (see Table 4.2). Zero-order correlations revealed that total PCL-R scores were not related to either $EI_{Negative}$ or $EI_{Positive}$. However, when examining the specific psychopathy factors, Factor 1 was negatively correlated with $EI_{Negative}$, whereas Factor 2 was unrelated to $EI_{Negative}$. Furthermore, a multiple linear regression revealed that Factor 1 uniquely predicted $EI_{Negative}$ with Factor 2 continuing to show no relationship. Both factors were unrelated to $EI_{Positive}$, and failed to uniquely predict this measure[15].

Table 4.2 Summary of zero-order correlations and multiple linear regressions run between Psychopathy-Checklist Revised (PCL-R) total and factor scores with each emotional index (EI: emotion minus neutral) for pupil diameter to images.

	PCL-R total	Factor 1 (Interpersonal/Affective)			Factor 2 (Lifestyle/Antisocial		
	r	*r*	*t*	*β*	*r*	*t*	*β*
$EI_{Negative}$	-.17	-.23*	-2.06	-.26*	-.06	0.44	.06
$EI_{Positive}$	-.06	-.04	0.44	.06	-.02	-0.51	-.01

* $p < .05$ (two-tailed).
β, standardised beta.
Correlational analyses, degrees of freedom = 79

[15] As an additional check, the results did not alter when medication dosage (chlorpromazine, valium and fluoxetine) was controlled for; notably, Factor 1 continued to negatively predict $EI_{Negative}$, t (73) = -2.43, $p = .02$, $\beta = -.32$.

Multiple linear regression analyses, degrees of freedom = 2, 76

4.2.2.2.2 Pupil diameter to neutral images

The relationship between PCL-R scores and pupil diameter to neutral images was explored within 79 participants using partial correlations and a multiple linear regression controlling for chlorpromazine. Neither PCL-R total, r (76) = .21, p = .07, Factor 1, r (76) = .19, p = .10, nor Factor 2, r (76) = .17, p = .15, were significantly related to pupil diameter to neutral images. A multiple linear regression revealed that the unique variance associated with each factor failed to predict pupil diameter in response to neutral images (Factor 1, t [75] = 1.13, p = .26, β = .15; Factor 2, t [75] = 0.82, p = .41, β = .10).

4.2.3 Discussion

As expected, Factor 1 was negatively associated to and uniquely predictive of EI to negative images, while Factor 2 showed no relationship. Neither factor was related to EI for positive images, as predicted. This suggests that the interpersonal/affective dimension of psychopathy is selectively associated to autonomic hypo-responsivity to specifically negative images. Against predictions, total PCL-R was not associated to EI to negative images (or to positive images as expected) indicating that overall psychopathy was unrelated to autonomic hypo-responsivity to emotion. PCL-R scores were unrelated to pupil diameter to neutral images, as predicted, indicating that psychopathy was not associated with abnormal overall responses to images.

Additionally, negative and positive images led to similarly larger pupil diameter than neutral images as hypothesised.

4.3 Experiment 11: Dynamic facial expressions

Participants viewed video-clips of negative (fearful and angry), happy and neutral facial expressions and measured pupil diameter in response as a function of PCL-R

scores. Fearful facial expressions were presented given the consistent evidence that implicates impairments for processing fear within psychopathy. Happy expressions were presented to examine autonomic responsivity to positive facial affect as a function of psychopathy. Neutral expressions were presented as a comparison to measure emotional modulation. Lastly, angry faces were presented given that this expression most likely signals an immediate threat of danger and interpersonal/affective psychopathy traits are thought to be reflective of temperamental fearlessness (Fowles & Dindo, 2009; Patrick, 2010a; Patrick & Bernat, 2009).

I predicted that negative facial expressions would lead to larger pupil diameter than both positive and neutral facial expressions, with no difference in pupil size between positive and neutral faces, in line with the data observed for facial expressions in Chapter 3 (see 3.3 Experiment 7: Static facial expressions and 3.4 Experiment 8: Dynamic facial expressions).

The predictions in relation to psychopathy paralleled those made for the previous experiment. I expected that Factor 1 would be selectively associated with diminished emotional modulation of pupil diameter, reflecting autonomic hypo-responsivity, in response to negative facial expressions, with no relationship for Factor 2. That is, I predicted that Factor 1 would be negatively related to EI for fearful and angry facial expressions with no association for Factor 2, and neither factor was hypothesised to be associated to EI for happy facial expressions. Total PCL-R scores were again expected to be associated to diminished negative modulation of pupil diameter as a consequence of Factor 1. Finally, I predicted that PCL-R scores would be unrelated to pupil diameter to neutral facial expressions, reflecting normal overall autonomic responsivity.

4.3.1 Method

The same video-clips of dynamic facial expressions from Experiment 8 from the previous chapter (see 3.4.1 Method) were presented to the participants, although only

for the emotional categories of fear, happiness, neutral, and anger. Screenshots were taken from the end of each video-clip and facial expression valence showed similar luminance, contrast and colour values with this reduced stimuli sample.

Experimental design was identical to dynamic faces task in the previous chapter (see Chapter 3, Experiment 3: Dynamic facial expressions, Design).

4.3.1.1 Data analyses

Data analyses was identical to Experiment 8 from the previous chapter (see 3.4.1 Method) apart from PCL-R total, Factor 1 and Factor 2 were entered into associative and predictive models. Mean baseline-corrected pupil diameter was calculated across an early (2000 – 3000 ms post video-clip onset), and a late analysis window (3000 – 4000 post video-clip onset).

Spearman-Brown split-half reliability checks were run identifying good internal consistency for early (r = .88) and late pupil diameter (.87). Valid data was collected for 78 participants in response to the dynamic facial expressions as four participant's datasets were removed for containing too much missing data. For the remaining participants, the percentage of possible valid data collected during stimulus presentation was 88.44 %.

4.3.1.2 Confounding variables

As reported previously, chlorpromazine dosage was inversely correlated with PCL-R total and Factor 1 scores, and valium was positively associated to Factor 2 (see 'confounding variables' in general method). Yet, chlorpromazine dosage was unrelated to any of the pupillary measures during the dynamic faces task (ps > .16) and was, therefore, not controlled for across analyses. Valium dosage was not associated with pupil diameter to neutral faces (ps > .09), but did evidence a positive association to EI_{Fear} over the early time-window, r (78) = .22, p = .053, and EI_{Angry} over the early and late time-windows, rs (78) = .34 and .36, ps = .002 and .001. Although, valium was unrelated to

the remaining EIs (*p*s > .15), I decided to control for valium dosage across all EI analyses to protect against Type I errors.

Lastly, participant IQ was inversely related to Factor 2 and also showed a negative relationship to EI_{Happy} during the early time-window, r (78) = -.23, p = .045. I have reported the analysis between these two variable with and without IQ entered as a covariate given the debate regarding whether IQ can be treated as a confounding variable (see 4.1.6 Confounding variables).

4.3.2 Results

4.3.2.1 Manipulation check

As can be seen in Figure 4.2, a PLR was observed after video-clip onset after approximately 400 ms that reached its nadir at 1000 ms. The time-course of the PLR was similar to that observed to the affective images although the magnitude to the dynamic faces was smaller reaching a constriction diameter of only -0.08 mm. Pupil diameter then increased steadily, with emotional modulation of the pupil occurring at around 1700 ms post video-clip onset and pupil diameter reaching baseline size at around 2000 ms (for the affective faces). The pupil then remained constant in size until 4400 ms after facial expression onset (400 ms after video-clip offset). At this point, a secondary smaller constriction was observed that reached a nadir of approximately 0.07 mm at 4800 ms (800 ms after video-clip offset) before recovery back to baseline pupil size at 6000 ms. Across 78 participants, there was a small degree of emotional modulation early on after the PLR, but repeated measures ANOVAs with a factor of emotion (fear, happy, neutral and angry) revealed that there was no significant main effect of facial emotion on pupil diameter at the early, F (3, 231) = 2.07, p = .11, η^2 = .03, or late time window, F (3, 231) = 1.29, p = .28, η^2 = .02. Planned comparison between pupil diameter to affective and neutral expressions revealed that pupil diameter was larger over the early time-window in response to fearful, t (77) = 2.24, p = .03, g_{av} = 0.20,

and angry faces, t (77) = 2.11, p = .04, g_{av} = 0.21, compared to neutral faces respectively with no difference between happy and neutral expression, t (77) = 1.14, p = .26, g_{av} = 0.11. Over the late time-window, there was no difference in pupil diameter between fearful, t (77) = 1.45, p = .15, g_{av} = 0.14, happy, t (77) = 0.74, p = .46, g_{av} = 0.07, or angry faces, t (77) = 1.89, p = .06, g_{av} = 0.17, compared to neutral expressions.

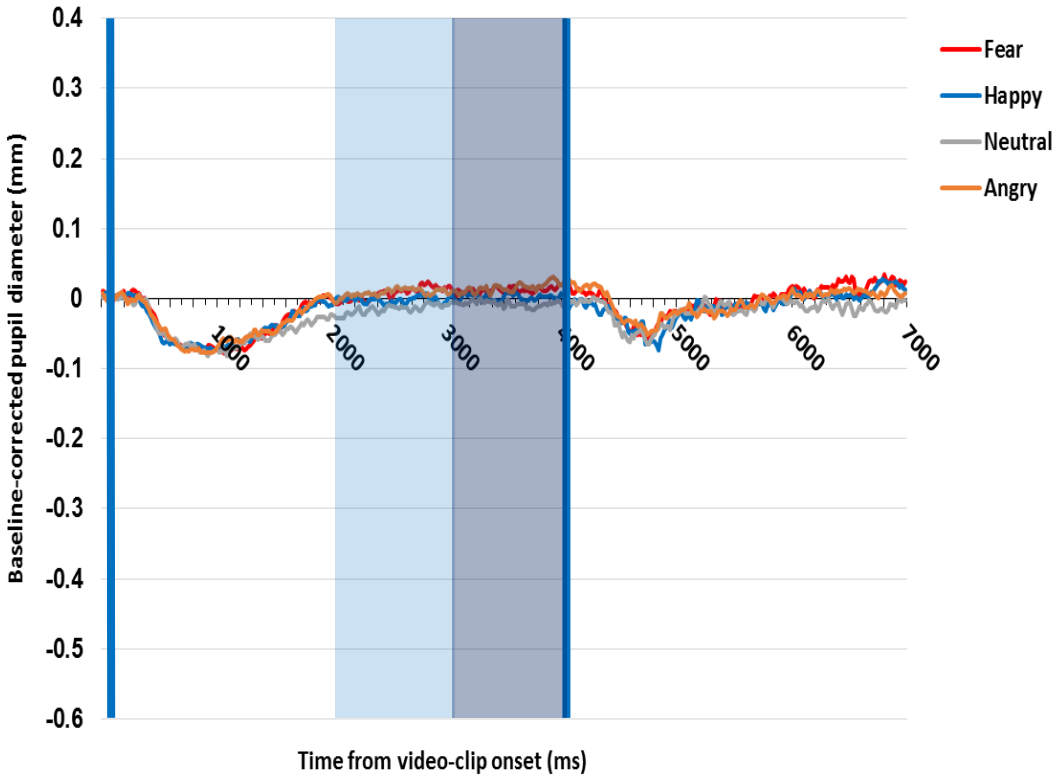


Figure 4.2 Baseline-corrected pupil diameter in response to fearful, happy, neutral and angry dynamic facial expression. The blue bars indicate video-clip onset and offset. Light blue shading specifies the early time window and the dark blue shading indicates the late time-window used for analyses (N = 78).

4.3.2.2 Psychopathy

4.3.2.2.1 Emotional modulation of pupil diameter

Partial correlations and multiple linear regressions were run between PCL-R scores (controlling for valium dosage) and EIs in response to the dynamic faces across the early and late time-windows (see Table 4.3). Correlations revealed that PCL-R total was unrelated to each EI apart from a positive association with EI_{Happy} over the late time-window. Factor 1 was negatively associated with EI_{Angry} across the early time-window

and positively associated with EI_{Happy} across the late time-window, with no further correlations. Factor 2 showed no association with any EI. A multiple linear regression revealed Factor 1 as a unique negative predictor of both EI_{Fear} and EI_{Angry} over the early time-window, as well as a positive predictor of EI_{Happy} across the late time-window[16]. Factor 1 did not predict the remaining EIs. The regression analyses showed that Factor 2 remained unrelated to each EI, although a positive predictive trend was identified for EI_{Fear} over the early time-window (p = .06).[17]

Table 4.3 Summary of zero-order correlations and multiple linear regressions run between Psychopathy-Checklist Revised (PCL-R) total and factor scores with each emotional index (EI: emotion minus neutral) for pupil diameter to dynamic facial expressions.

		PCL-R total	Factor 1 (Interpersonal/Affective)			Factor 2 (Lifestyle/Antisocial		
Time window		*r*	*r*	*t*	*β*	*r*	*t*	*β*
Early	EI_{Fear}	-.04	-.14	-2.00	-.25*	.12	1.91	.24
	EI_{Happy}	.18	.17	1.14	.15	.13	0.46	.06
	EI_{Angry}	-.15	-.26*	-2.72	-.32*	.01	1.37	.17
Late	EI_{Fear}	-01	.001	-0.37	-.05	.08	0.82	.11
	EI_{Happy}	.28*	.35*	2.99	.37*	.14	-0.25	-.03
	EI_{Angry}	-.10	-.12	-1.07	-.13	-.03	0.28	.04

* $p < .05$ (two-tailed).
β, standardised beta.
Correlational analyses, degrees of freedom = 78
Multiple linear regression analyses, degrees of freedom = 2, 75

[16] As an additional check, the results remained the same in response to facial expressions when all medication dosage (chlorpromazine, valium and fluoxetine) were controlled for; notably, Factor 1 continued to predict EI_{Fear} and EI_{Angry} over the early time-window, and positively predict EI_{Happy} over the late time-window (ps < .03).
[17] Factor 2 remained unrelated to and did not predict EI_{Happy} over the early time-window when participant IQ was controlled for (ps > .50).

4.3.2.2.2 Pupil diameter to neutral dynamic facial expressions

Zero-order correlations indicated that across 78 participants PCL-R total was unrelated to pupil diameter to neutral faces over either the early and late time-windows (early, r [78] = -.09, p = .41; late, r [78] = -.15, p = .18) as was Factor 1 (early, r [78] = -.03, p = .83; late, r [78] = -.11, p = .33) and Factor 2 (early, r [78] = -.14, p = .23; late, r [78] = -.15, p = .18). Moreover, neither Factor 1 (early, t [75] = 0.35, p = .73, β = -.05; late, t [75] = -0.44, p = .66, β = -.06), nor Factor 2 (early, t [75] = -1.23, p = .22, β = -.16; late, t [75] = -1.00, p = .32, β = -.13), uniquely predicted pupil diameter to neutral faces over either time-window.

4.3.3 Discussion

Factor 1 was negatively predictive of EI for fearful and angry facial expressions (although the associative relationship was not significant for fearful faces), as hypothesised, while Factor 2 showed no relationship, although this effect was specific to the early time-window. This suggests that the interpersonal/affective dimension of psychopathy is selectively associated to a pattern of autonomic hypo-responsivity to specifically negative facial expressions, but this is limited to early responses. Interestingly, Factor 1 was positively related to EI for happy facial expressions, during the late time-window, while Factor 2 was unrelated over either time-window. This suggests that the interpersonal/affective dimension of psychopathy is associated to autonomic hyper-responsivity to happy facial expressions, but this is limited to late responses. Contrary to predictions, total PCL-R scores were not associated to EI for either fearful or angry facial expressions, but was positively related to EI for happy facial expressions during the late time-window (driven by Factor 1). As expected, PCL-R scores were unrelated to pupil diameter to neutral stimuli indicating normal overall autonomic responsivity.

Limited emotional modulation of the pupil was observed in response to the dynamic faces, although negative facial expressions led to increased pupil diameter compared to neutral facial expressions over the early time-window.

4.4 Experiment 12: Affective sound-clips

Participants were presented with emotional (negative and positive) and neutral sound-clips and their pupil diameter was measured in response as a function of PCL-R scores. Negative and positive sound-clips were matched for subjective arousal so that autonomic arousal could be explored across valences more equally as a function of psychopathy. I now expected increased pupil diameter in response to both negative and positive sound-clips in comparison to neutral sound-clips, reflecting increased emotional arousal, with no difference in pupil size between negative and positive sound-clips.

In relation to psychopathy, I again predicted that Factor 1 would be selectively associated with diminished emotional modulation of pupil diameter, reflecting autonomic hypo-responsivity, in response to negative sound-clips, with no relationship for Factor 2. That is, I predicted that Factor 1 would be negatively related to EI for negative sound-clips with no association for Factor 2, and neither factor was hypothesised to be associated to EI for positive sound-clips. Total PCL-R scores were expected to be associated to diminished negative modulation of pupil diameter, as a consequence of Factor 1. Finally, I predicted that PCL-R scores would be unrelated to pupil responsivity to neutral sound-clips, reflecting normal overall autonomic responsivity.

4.4.1 Method

Thirty sound-clips were selected from the International Affective Digitised Sounds (IADS) (Bradley & Lang, 2007) consisting of 10 negative sound-clips (mean valence/arousal = 2.87, 7.09), 10 positive sound-clips (mean valence/arousal = 7.17,

6.76) and 10 neutral sound-clips (mean valence/arousal = 5.06, 5.05)[18]. No erotic stimuli were used. All emotions differed significantly for subjective valence (*p*s < .001). There was no difference between negative and positive sound-clips for subjective arousal, *t* (18) = 1.50, *p* = .15, with both affective sound-clips rated as more arousing than neutral sound-clips (*ps* < .001). Sound-clips did not differ across negative, positive and neutral sound-clips for average and maximum root mean square decibel level *(p*s > .05). Sound-clips were played to participants at a comfortable set volume through headphones.

Experimental design was identical to that described in Experiment 9 from the previous chapter (see 3.5.1 Method in the previous chapter).

4.4.1.1 Data analyses

Data analyses were identical to that described in Experiment 9 from the previous chapter (see 3.5.1.1 Data analysis), except PCL-R total, Factor 1 and Factor 2 were entered into associative and predictive models. Mean baseline-corrected pupil diameter was calculated across an early (500 – 1500 ms post sound-clip onset), and a late analysis window (2500 – 3500 post sound-clip onset).

Spearman-Brown split-half reliability checks revealed good internal consistency for early (.67) and late pupil diameter (.75) which was used to calculate the emotional differences. Five participants were excluded from this task for containing too much missing data leaving a sample size of 77. The percentage of possible valid data collected during the entirety of sound-clip presentation was 87.70 %.

4.4.1.2 Confounding variables

It was previously reported that chlorpromazine dosage was inversely related to PCL-R total and Factor 1 scores, while valium dosage was positively correlated with Factor 2

[18] IADS sound-clips selected were: Negative: 106, 115, 310, 424, 600, 625, 626, 712, 714, 732; Positive: 220, 311, 352, 353, 360, 365, 415, 808, 815, 817; Neutral: 120, 152, 170, 246, 361, 364, 368, 425, 500, 701.

scores (see 4.1.6 Confounding variables). Chlorpromazine dosage was unrelated to EIs across both time-windows (*p*s > .23), but did show a negative association with pupil diameter to neutral sound-clips over the early, r (75) = -.28, p = .02, and late time-window, r (77) = -.28, p = .01. Valium dosage showed a negative association to only $EI_{Positive}$ during the late time window, r (72) = -.26, p = .03, with no relationship to the remaining EIs (*p*s > .17) or to pupil diameter to neutral sound-clips (p > .38). Chlorpromazine dosage was controlled for when examining pupil diameter to neutral sound-clips and valium dosage was controlled for across all EIs to protect against Type I errors.

4.4.2 Results

4.4.2.1 Manipulation check

As can be seen in Figure 4.3, the sound-clips led to a pupil dilation with a latency from stimulus-onset of around 400 ms. Emotional modulation began to emerge from approximately 1300 post sound-clip onset with pupil diameter reaching a peak between 1.25 – 1.90 mm around 2500 - 3500 ms with the affective sound-clips showing larger and later maximum dilation. The pupil then showed a gradual reduction in diameter although the pupil remains markedly dilated compared to baseline levels at sound-clip offset. The pupil remained approximately 0.10 mm above baseline pupil size although the graph does not display the full recovery period, which continued for 8000 ms following sound-clip offset.

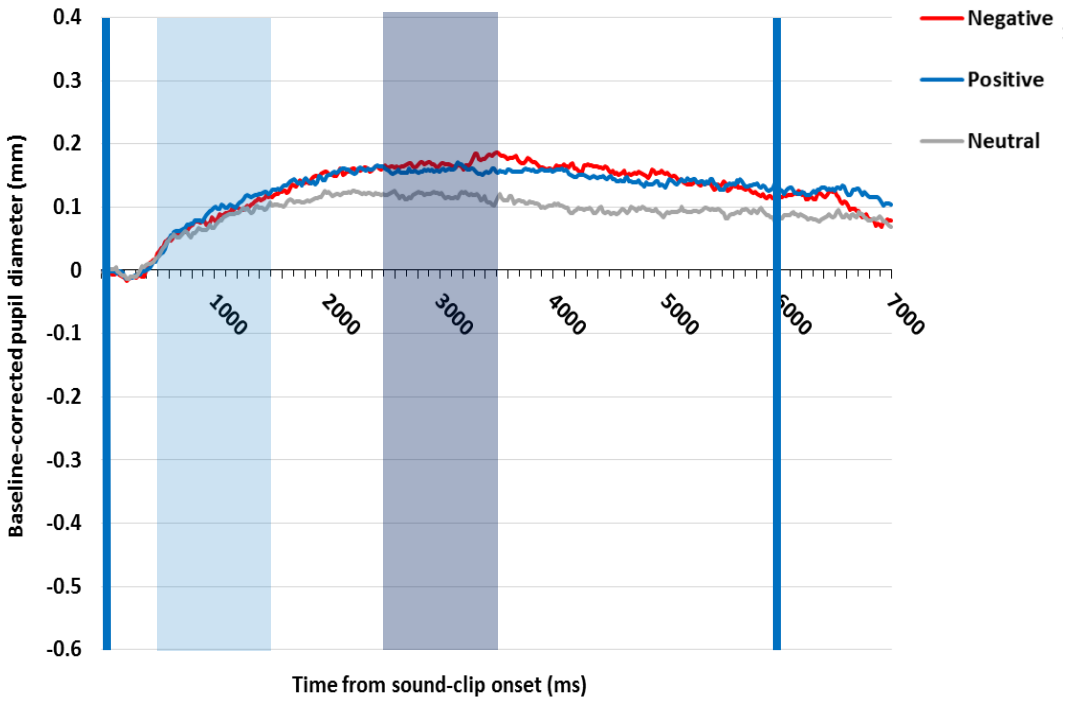


Figure 4.3 Baseline-corrected pupil diameter in response to unpleasant, pleasant and neutral sound-clips. The blue bars indicate sound-clip onset and offset. Light blue shading specifies the early time window and the dark blue shading indicates the late time-window used for analyses (N = 77).

Across a sample of 77 participants, although mean pupil diameter data was missing across positive and neutral sound-clips for two further participants, repeated measures ANOVAs with a factor of emotion (negative, positive and neutral) confirmed a significant main effect of emotion across the early, F (2, 146) = 3.44, p = .04, η^2 = .05, and late time window, F (2, 146) = 9.89, p < .001, η^2 = .12. Pupil diameter was larger in response to negative than neutral sound-clips over the late analysis window, t (76) = 4.32, p < .001, g_{av} = 0.44, but the difference did not reach significance for the early time window, t (74) = 1.67, p = .10, g_{av} = 0.16. Positive sounds produced greater dilation than neutral sounds over both analyses windows (early, t [73] = 2.51, p = .01, g_{av} = 0.28; late, t [73] = 3.45, p = .001, g_{av} = 0.36).

4.4.2.2 Psychopathy

4.4.2.2.1 Emotional modulation of pupil diameter

Partial correlations and multiple linear regressions were run between PCL-R scores (controlling for valium dosage) and EIs in response to the sound-clips, and this was explored across both the early and late time-windows (see Table 4.4). Zero-order correlations showed that PCL-R total was unrelated to $EI_{Negative}$ or $EI_{Positive}$ for the early time-window. Factor 1 showed a negative relationship to $EI_{Negative}$ during the early time-window, while Factor 2 showed no association. However, a multiple linear regression revealed that neither PCL-R factors uniquely predicted $EI_{Negative}$, although Factor 1 demonstrated a negative predictive relationship that approached significance, t (71) = -1.85, p = .07, β = -.24. PCL-R total and factor scores were unrelated and neither PCL-R factor predicted $EI_{Negative}$ or $EI_{Positive}$ over the late time-window[19].

Table 4.4 Summary of zero-order correlations and multiple linear regressions run between Psychopathy-Checklist Revised (PCL-R) total and factor scores with each emotional index (EI: emotion minus neutral) for pupil diameter to sound-clips.

		PCL-R total	Factor 1 (Interpersonal/Affective)			Factor 2 (Lifestyle/Antisocial		
Time window		*r*	*r*	*t*	*β*	*r*	*t*	*β*
Early	$EI_{Negative}$	-.23	-.26*	-1.85	-.24	-.14	-0.07	-.01
	$EI_{Positive}$	-.10	-.07	-0.32	-.04	-.11	-0.88	-.12
Late	$EI_{Negative}$	-.06	-.02	0.09	.01	-.06	-0.05	-.01
	$EI_{Positive}$	.06	.06	0.30	.04	.06	0.30	.04

* $p < .05$ (two-tailed).
β, standardised beta.
Correlational analyses, degrees of freedom = 75
Multiple linear regression analyses, degrees of freedom = 2, 72

[19] As an additional check, the analyses were conducted again controlling for all medication dosage (chlorpromazine, valium and fluoxetine). The pattern of the results remained the same, although now Factor 1 was negatively related to $EI_{Negative}$ over the early time-window at a trend level of significance (p = .07, two tailed).

4.4.2.2.2 Pupil diameter to neutral sound-clips

Across 75 participants (as two participants from the subsample of 77 participant were missing mean pupil diameter data in response to neutral sound-clips), PCL-R scores were explored in relation to pupil diameter to neutral sound-clips across the early and late time windows (controlling for chlorpromazine dosage). Partial correlations demonstrated that neither PCL-R total, (early, r [72] = .04, p = .73; late, r [72] = -.02, p = .89), Factor 1, (early, r [72] = -.05, p = .65; late, r [72] = -.07, p = .58), nor Factor 2, (early, r [72] = .12, p = .29; late, r [72] = .04, p = .75), were related to pupil diameter in response to neutral sound-clip across either time-window. A multiple linear regression showed neither Factor 1 (early, t [71] = -1.07, p = .29, β = -.14; late, t [73] = -0.68, p = .50, β = -.09), nor Factor 2 (early, t [71] = 1.44, p = .16, β = .18; late, t [73] = 0.58, p = .56, β = .07), uniquely predicted pupil diameter to neutral faces over either time-window.

4.4.3 Discussion

Factor 1 was negatively associated to EI for negative sound-clips as expected although this was specific to the early time-window (and the unique predictive relationship failed to surpass two-tailed statistical significance), while Factor 2 showed no relationship. Neither factor was related to EI for positive sound-clips, as hypothesised. This suggests that the interpersonal/affective dimension of psychopathy is associated to autonomic hypo-responsivity to specifically negative sound-clips, but this is limited to early responses. Against predictions, total PCL-R scores were unrelated to EI for negative sound-clips (as well as positive sound-clips as expected) suggesting that overall psychopathy was not associated to autonomic hypo-responsivity to emotion. There was no association between PCL-R scores to pupil responsivity to neutral sound-clips as expected, indicating that psychopathy was not associated with abnormal overall autonomic responsivity to images.

Additionally, negative and positive sound-clips led to increased pupil diameter compared to neutral sound-clips.

4.5 General experimental discussion

The present study explored whether psychopathy within a male forensic psychiatric population was associated with abnormal pupil reactivity to emotion. The data indicated that interpersonal/affective psychopathy traits, indexed by Factor 1 of the PCL-R, was selectively associated to a pattern of diminished early pupil responsivity to specifically negative stimuli and this pattern was consistent across images, dynamic facial expressions and sound-clips. Interpersonal/affective psychopathy traits were unrelated to pupil responsivity to positive stimuli, apart from a selective association to increased late pupil responsivity to happy facial expressions (compared to neutral faces). Lifestyle/antisocial psychopathy traits, indexed by Factor 2 of the PCL-R, was unrelated to pupil responsivity to emotional stimuli. Also, psychopathy scores were not related to overall pupil responses to neutral stimuli throughout any task, indicating that psychopathy was associated to normal autonomic responses in the absence of emotion.

4.5.1 Hypo-responsivity to negative stimuli was related to interpersonal/affective psychopathy traits

The current data provides support for a growing body of literature that emphasises the distinct nature of the psychopathy factors, highlighting the importance to explore psychopathy as a multi-faceted construct especially given that overall psychopathy within the current study masked effects that were specific to the interpersonal/affective dimension.

The interpersonal/affective component was predictive of attenuated autonomic reactivity to emotionally arousing negative stimuli across images, dynamic facial expressions and sound-clips, consistent with dual pathway models of psychopathy that emphasises that the interpersonal/affective dimension of psychopathy represents an

underlying deficient defensive motivational system (Fowles & Dindo, 2009; Patrick, 1994; Patrick & Bernat, 2009). Psychopathy was not explored at a facet level and, hence, cannot offer any conclusion in relation to either the three or four-facet models. The interpersonal/affective dimension is increasingly understood as the core component of psychopathy that is related to dispositional fearlessness and a lack of anxiety (Lilienfeld, Watts, et al., 2015; Skeem et al., 2003; Yildirim & Derksen, 2015), as well as impaired physiological responses to negative stimuli (Benning, Patrick, & Iacono, 2005; Decety et al., 2013; Dolan & Fullam, 2009; Esteller et al., 2016; Harenski et al., 2010; Lorenz & Newman, 2002; Medina et al., 2016; Patrick et al., 1993; Sadeh & Verona, 2012; Vaidyanathan et al., 2011; Venables et al., 2015; Verona et al., 2013; Verona et al., 2004).

It was reported previously that while psychopathy has been associated to impairments to both negative and positive stimuli, psychopathy, and the interpersonal/affective dimension specifically, has been more frequently associated to a deficit to negative emotion (see 1.3 Psychopathy and emotional processing). However, many of these studies failed to present positive stimuli, limiting the conclusions drawn. The current findings illustrate that interpersonal/affective psychopathy traits are selectively associated to hypo-responsivity to *specifically* negative stimuli across a comprehensive set of negative and positive images, facial expressions and sound-clips. This is contrary to a general emotional deficit perspective and supportive of models that emphasise a specifically negative impairment in relation to psychopathy. Also, the negative stimuli can be considered as predominantly threatening, as the fearful stimuli depicted pointed guns, snarling animals, and attack-sounds, and angry facial expressions that seem likely to convey a threat to the observer. Therefore, the findings most closely support a low-fear hypothesis reflecting an insensitive defensive motivational system, further supporting a dual pathway model of psychopathy that highlights that the interpersonal/affective dimension of psychopathy represents an

underlying deficient defensive motivational system. Additionally, given that hypo-responsivity to negative stimuli was only evident over early time-windows, this indicates a further more complex impairment for individuals high in interpersonal/affective psychopathy traits; this will be discussed shortly (see 4.5.2 Autonomic hypo-responsivity was specific to early reactivity).

The sensitivity of the defensive motivational system is tied closely to amygdala activity (Lang & Bradley, 2010) and, indeed, the underlying neural mechanism of the emotional deficiency that characterises individuals high in interpersonal/affective psychopathy has been attributed to a genetically determined attenuation of amygdala reactivity (Blair, 2006; Blair, 2013; Kiehl, 2006). In support, as previously described, children with callous/unemotional traits and adults high in interpersonal/affective psychopathy traits show attenuated activity in the amygdala in response to negative stimuli or when processing moral emotions (see 1.3.3 The dimensions of psychopathy and emotional processing) as well as structural abnormalities in the amygdala (Vieira et al., 2015; Wolf et al., 2015). Therefore, the emotional processing deficits of individuals high in interpersonal/affective psychopathy traits may reflect an impaired or insensitive amygdala.

Amygdala dysfunction may account for the specific impairments observed in the present chapter to negative stimuli, given that the processing of negative emotion (particularly fear) is more intrinsically linked to amygdala function compared to positive emotions (Adolphs et al., 1994; Adolphs et al., 1999; Calder, 1996; Morris et al., 1996) with the amygdala central to the generation of functional responses to both fear and anger cues (Adams, Gordon, Baird, Ambady, & Kleck, 2003; Scott et al., 1997; Whalen et al., 2001). However, neural networks including the amygdala are responsive to positive emotions (Burgdorf & Panksepp, 2006; Hamann & Mao, 2002). This suggests that high psychopathy individuals will exhibit impairments for positive stimuli, but deficits will be more severe to negative stimuli (and hence more readily identified), which fits with

the general pattern of previous findings in the psychopathy literature (see 1.3.5 Evidence for theories of emotional processing in psychopathy).

Conversely, late autonomic hyper-responsivity was observed in response to happy dynamic facial expressions in relation to the interpersonal/affective dimension of psychopathy. The functioning of the appetitive system within individuals high in psychopathy is not clear with a general emotional deficit perspective arguing for a deficient appetitive system, while alternative perspectives propose no deficit or even an over-sensitivity in reward systems (Fowles, 1980). Indeed, several studies have indicated that interpersonal/affective psychopathy traits are positively associated with self-reported behavioural activation system, which is an indication of reward sensitivity (Falkenbach et al., 2014; Poythress, Edens, et al., 2010). One possible etiological mechanism was proposed by Yildirim and Derksen (2015) who reported that emotionally deficient high psychopathy individuals are characterised by increased testosterone alongside under-active hypothalamic pituitary adrenal (HPA) axis activity leading to an enhancement in dopamine-mediated reward sensitivity and a dampening of amygdala driven threat reactivity. This highlights that individuals high in interpersonal/affective psychopathy deficits may show elevated responsivity to positive cues alongside diminished fear responsivity, consistent with the current findings. However, in the current study, hyper-responsivity to happy facial expressions was observed only during the late time-window suggesting a further complex process, likewise to the autonomic hypo-responsivity observed to negative stimuli.

Moreover, autonomic hyper-responsivity was not observed in response to positive images or sound-clips perhaps highlighting the unique motivational significance of facial stimuli. Alternatively, this specific findings may be due to the use of dynamic faces opposed to static emotional stimuli, which causes recruitment of different emotion-specific neural networks (Kessler et al., 2011; Kilts et al., 2003; Sato et al., 2004; Trautmann et al., 2009). In support, Decety et al. (2014) compared high psychopathy

individuals to control participants for neural responses to dynamic facial expressions finding that the high psychopathy group demonstrated increased amygdala responses to dynamic happy facial expressions consistent with the current results. However, in contrast to the current results that found this relationship was specific to interpersonal/affective psychopathy traits, Decety and colleagues reported no unique relationship with either PCL-R factor. Therefore, it is clear more research is needed to understand autonomic responsivity to positive stimuli within individuals high in interpersonal/affective psychopathy traits.

The current chapter showed that lifestyle/antisocial psychopathy traits were unrelated to autonomic reactivity to emotionally arousing stimuli, in contrast to the interpersonal/affective dimension of psychopathy. This finding is consistent with previous empirical literature that typically describes the lifestyle/antisocial dimension to be unrelated to the emotional processing deficits that characterise psychopathy (see 1.3.3 The dimensions of psychopathy and emotional processing). The dual-pathway model proposed that the lifestyle/antisocial component of psychopathy is thought to reflect impairments in the ability to regulate affect and behaviour (Patrick & Bernat, 2009) and this dimension has been associated to dispositional traits of greater emotionality and anxiety (Coid et al., 2012; Drislane, Patrick, & Arsal, 2014; Falkenbach et al., 2008; Falkenbach et al., 2014; Hicks et al., 2004; Hicks & Patrick, 2006; Hyde et al., 2014; Lyons, 2015; Salekin et al., 2014; Skeem et al., 2007; Swogger & Kosson, 2007). However, the current chapter highlights that lifestyle/antisocial psychopathy traits were related to a pattern of normal autonomic responsivity to emotion rather than increased sensitivity to emotion. Therefore, the current findings further highlight the importance to consider psychopathy as a multi-faceted construct given the diverging, and possibly opposing (Hicks & Patrick, 2006), correlates of the interpersonal/affective and lifestyle/antisocial dimensions.

4.5.2 Autonomic hypo-responsivity was specific to early reactivity

Interestingly, autonomic hypo-responsivity in relation to interpersonal/affective psychopathy traits was identified for specifically *early* responses to negative stimuli (i.e. under 2000 ms from the onset of emotional stimuli). Interpersonal/affective psychopathy traits were not associated with pupil hypo-responsivity to negative stimuli over later time-windows suggesting that hypo-responsivity to negative stimuli interacts with an early attentional/processing impairment. As described previously (see 1.3.1 Theories of emotional processing in psychopathy), the Response Modulation Hypothesis (RMH) proposes that high psychopathy individuals display an early attentional bottleneck that disrupts or delays the concurrent processing of information peripheral to the individual's ongoing goal-directed behaviour. It could be argued that the present results are consistent with an attentional bottleneck for individuals high in interpersonal/affective psychopathy traits that constrained the processing of early affective information leading to impaired autonomic responsivity. It is conceivable that within these individuals the emotional stimuli was complex enough to lead to a delay in processing and/or a failure to initially attend to the relevant emotional cue attenuating early autonomic responses to emotion. However, with prolonged exposure, these individuals were able to process the emotional cues leading to increasingly normal autonomic responsivity over presentation.

While the RMH appears promising in explaining why autonomic deficits in psychopathy were observed specifically during the earlier time windows of the pupil response, this model cannot account for why impairments were specific to negative stimuli given that the RMH assumes a non-specific emotional deficit that is dependent on attention. Alternatively, it was previously speculated that the Differential Amygdala Activation Model (DAAM) would hypothesise attentional impairments in response to both negative and positive stimuli, but with more severe deficits in response to negative cues, as the model is centred on amygdala dysfunction (specifically the basolateral amygdala) (see 1.3.1 Theories of emotional processing in psychopathy). This could account for the

present findings, suggesting that deficits to positive stimuli may need even more restricted attention or weaker stimuli to be apparent in the data. I therefore suggest that the current data fits best with the DAAM. However, despite this, the DAAM fails to report the individual role of the interpersonal/affective dimension of psychopathy, instead suggesting that this attentional impairment is universal across psychopathy. Given that the current findings of autonomic hypo-responsivity to negative stimuli were specific to the interpersonal/affective dimension of psychopathy, the DAAM cannot offer a complete explanation of the current results.

The explanation for this specific result may reflect a divergence in the hemispheric dynamics of the amygdala in relation to the dimensions of psychopathy. Hecht (2011) proposed that the interpersonal/affective component of psychopathy is characterised by hypo-responsivity of the right hemisphere, while the lifestyle/antisocial component is related to hyper-responsivity of the left hemisphere. In support, empirical research has identified that adults and children high in interpersonal/affective psychopathy traits have shown underactivity of the amygdala in response to negative stimuli that is specific to the right hemisphere (Gordon et al., 2004; Harenski et al., 2014; Jones et al., 2009; Marsh & Cardinale, 2014; Viding et al., 2012). Research has reported lateralised amygdala functionality, with the right hemisphere responsible for an initial, fast and reflexive shift to implicit and salient emotion that leads to physiological arousal, followed by a more prolonged cognitive evaluation of the emotional stimuli subserved by the left amygdala (Costafreda, Brammer, David, & Fu, 2008; Gläscher & Adolphs, 2003; Hardee, Thompson, & Puce, 2008; Markowitsch, 1999; Morris, Öhman, & Dolan, 1999; Sergerie, Chochol, & Armony, 2008; Wright et al., 2001). Additionally, Lanteaume et al. (2007) reported that electrical stimulation of the right amygdala led to the experience of negative emotions, particularly for fear and sadness, accompanied by increased SCR, but not positive emotions. In contrast, left hemisphere stimulation led to the experience of both negative and positive emotions. Therefore, the current findings for individuals high in

interpersonal/affective psychopathy traits may reflect a hypo-responsive right amygdala causing early autonomic hypo-responsivity to specifically negative stimuli, before later recruitment of the unaffected left hemisphere capacities resulting in normal autonomic activity over time.

4.5.3 Emotion and pupil response across tasks

Following on from the previous chapter, the stimuli was altered to ensure that the negative/positive images and sound-clips were matched for subjective arousal ratings. Indeed, negative/positive images and sound-clips led to comparable elevated pupil diameter suggesting that pupil response to emotion is reflective of emotional arousal regardless of valence (Bradley et al., 2008; Partala & Surakka, 2003).

Planned comparisons revealed greater pupil diameter to negative facial expressions compared to neutral expressions over the early time-window, but there was no difference in pupil diameter in response to happy and neutral facial expressions, likewise to the previous chapter. This is consistent with previous research that indicates greater physiological responsivity towards negative facial expressions compared to positive facial expressions (Breiter et al., 1996; Leppänen et al., 2007; Morris et al., 1996). As previously described, this may reflect the greater motivational significance of negative facial expressions (compared to positive faces) as likely signals of danger. It was also interesting to note that negative facial expressions led to larger pupil diameter only during the early pupil response window, in contrast to the previous chapter where emotional modulation was largest over the late time-window. From observing the pupil waveforms, it was apparent that general responsivity, as well as the emotional modulatory effect on the pupil, was limited in comparison to the community sample in Chapter 3. The forensic patient sample demonstrated an attenuated PLR followed by reduced pupil diameter in response to the dynamic faces, which appears to have limited the range of emotional modulation. Furthermore, this highlights that the patient sample

demonstrated a lack of responsivity to all stimuli across tasks characterised by attenuated changes in pupil diameter.

It is speculated that this overall hypo-responsivity reflects the vast differences between the community undergraduate sample within Chapter 3 and the forensic psychiatric sample within the current chapter. To illustrate, the forensic psychiatric population were conceivably less educated, exposed to greater trauma, had lower IQs, older, male only, taking greater amounts of medication and abused alcohol/substance to a greater extent. Although I have made attempts to control or rule out many of these factors as artefacts within the latter sample, which are discussed shortly, these factors serve to highlights the vast differences between the populations that may have led to differences in overall physiological responsivity. Also, many of the patients were diagnosed with mental health illness including schizophrenia, which has been previously associated with attenuated autonomic activity (Fujibayashi et al., 2009; Zahn, Carpenter, & McGlashan, 1981a; Zahn et al., 1997). However, it can be argued that this did not affect the current findings as there is evidence that impaired autonomic activity is specific for individuals who are psychotically unwell (Toichi et al., 1999; Zahn, Carpenter, & McGlashan, 1981b) and the current chapter ensured to only recruit participants if they were not currently psychotic.

4.5.4 Considerations

It is noted that there are limitations to the current experiment as there are several characteristics inherent to the research population that may have influenced pupil responses. Firstly, there were a variety of mental health diagnoses within the sample that can result in complex disturbances to emotional processing and behaviour. For example, as illustrated above, psychoses can lead to attenuated autonomic activity, although only participants who were not currently psychotic were recruited. Research has proposed that schizophrenia is associated with reduced neural activity to emotion in the amygdala, particularly in relation to negative stimuli (Anticevic et al., 2010; Li, Chan,

McAlonan, & Gong, 2009; Taylor et al., 2012). Anticevic et al. (2010) also reported that deficient amygdala activation is only present for patients with schizophrenia for emotional-neutral contrasts, predominantly for negative stimuli as few studies have explored responsivity to positive stimuli. Therefore, if patients with high interpersonal/affective psychopathy traits were also diagnosed with schizophrenia, then this may offer an alternate explanation to the current findings. However, as can be seen in Table 0.2 in Appendix 2, Factor 1 scores (as well as the other PCL-R scores) were relatively lower in patients diagnosed with schizophrenia contradicting this idea. Hence, it can be argued that the current psychopathy findings may be under-estimated if the patients with schizophrenia (who had low Factor 1 scores) did indeed show attenuated autonomic responsivity for negative-neutral contrasts.

Many of the individuals in the current sample had a diagnoses of a personality disorder (see Table 0.3 for a detailed breakdown of personality disorder diagnosis). This is relevant as a personality disorder is characterised by a disruption to the way these individuals think and feel, such as their emotional processing. There is high degree of overlap across dissocial personality disorder and psychopathy, such as callous, lack of remorse and failure to accept responsibility, and indeed Table 0.3 indicates that it was the individuals diagnosed with dissocial personality disorder within the current sample that showed high PCL-R scores. However, other personality disorders have been associated to disturbances in emotional processing that may have confounded the current data. To illustrate, emotionally unstable personality disorder is characterised by rapid changes in mood and marked affective reactivity that is considered to reflect an underlying emotional disturbance. However, Herpertz, Werth, et al. (2001) found that offenders diagnosed with borderline personality disorder (emotionally unstable personality disorder based on the Diagnostic and Statistical Manual of Mental Disorders) showed normal autonomic responses to emotion while individuals high in psychopathy evidence hypo-responsivity. Similarly, Kuo and Linehan (2009) reported that individuals

with borderline personality disorder did not display heightened reactivity to emotionally evocative stimuli compared to matched controls. This suggests that the increased emotional responsivity associated to emotional unstable personality disorders does not extend to autonomic reactivity, in contrast to individuals high in psychopathy, likely having little effect on pupil reactivity to emotion. However, there is little research that focuses on the effects of alternative personality disorders (e.g. paranoid, mixed) for autonomic reactivity to emotion, although few participants held these diagnoses within the current sample.

Alternatively, individuals with depression show greater pupil response to emotional stimuli than healthy controls (Burkhouse et al., 2017; Burkhouse et al., 2014; Burkhouse et al., 2015; Siegle, Granholm, Ingram, & Matt, 2001; Siegle, Steinhauer, Carter, Ramel, & Thase, 2003). However, as can be seen in Table 0.2, only eight patients within the current sample were diagnosed with a mood disorder, and largely these patients did not show abnormal PCL-R Factor 1 scores suggesting relatively little influence of mood disorder diagnoses on the current findings. However, four of these patients had further co-occurring diagnoses meaning that it is difficult to draw conclusions regarding the impact of a mood disorder on autonomic reactivity in the current research.

However, more complex interactions may exist between mental health diagnoses that impact on emotional functioning and it would be useful to replicate the current psychopathy findings within alternative populations with lower psychopathology.

Additionally, participant's previous substance/alcohol abuse was not controlled for, which may have affected pupil response. Psychopathy has previously been associated with historical substance abuse, although this has been found in relation to the lifestyle/antisocial component of psychopathy with the interpersonal/affective dimension being unrelated or even a protective factor (Schulz, Murphy, & Verona, 2016; Smith &

Newman, 1990; Walsh, Allen, & Kosson, 2007). Therefore, historical substance/alcohol abuse seem unlikely to have affected the results observed for specifically interpersonal/affective psychopathy traits. In support, patients were categorised into previous substance/alcohol use based on their historical information (see Table 0.1 in Appendix 2) and it is evident that individuals who have a history of misusing substances/alcohol show elevated Factor 2 scores, but similar Factor 1 scores to individuals who have not abused substances/alcohol.

Interpersonal/affective psychopathy traits continued to be associated with autonomic hypo-responsivity to negative stimuli regardless of the inclusion of psychotropic medication across each task, suggesting that drug action was unrelated to this effect. However, it is useful to consider the method that was used to control for psychotropic medication. Specifically, psychotropic medication dosage was controlled for if the active metabolite was within the maximum half-life window. This quantified the dosage that the participant had taken over a longer period (usually within the last 24 hours), but this cannot inform regarding the exact amount of metabolite active during the research session. A more specific measure of psychiatric medication dosage would be useful. However, even if the specific amount of active metabolite could be quantified accurately it would be difficult to determine the specific influence of the medication, given that each individual is affected by psychotropic medication to differing degrees. Furthermore, while the interaction between anti-psychotic, anti-anxiety and anti-depressant medication was explored, it was not possible to isolate the interaction between specific medication types. Overall, extensive efforts were made to control for the effects of psychotropic medication to protect the validity of the findings, but inevitably the effects of psychotropic medication cannot be completely precluded as more complex interactions may exist.

The predictive relationship between interpersonal/affective psychopathy traits and autonomic hypo-responsivity was weaker for negative sound-clips compared to images and dynamic facial expressions, failing to surpass conservative two-tailed statistical

significance. However, it was considered justified to continue to discuss the unique relationship as the trend direction was consistent with the remaining tasks and the associative negative relationship between the interpersonal/affective dimension and early pupil responsivity to negative sound-clips surpassed significance. The weaker effect may simply reflect the lower reliability associated to pupil responsivity to emotion, which was previously attributed to the absence of the reliable PLR (see 3.5.1 Method).

The present chapter identified that interpersonal/affective psychopathy traits selectively predicted early autonomic hypo-responsivity during passive-viewing of negative images, dynamic facial expressions and sound-clips, using pupillometry. These findings add to the body of literature that implicates that the emotional processing deficits that characterise psychopathy are driven by specifically interpersonal/affective psychopathy traits reflective of an underlying deficient defensive motivational system.

5 Chapter 5: General discussion

Abbreviation	Meaning	Page
PCL-R	Psychopathy Checklist-Revised	159
PLR	Pupil light reflex	168
SCR	Skin conductance response	160
Tri-PM	Triarchic Psychopathy Measure	159

Psychopathy has been frequently associated to a deficit in processing emotion, particularly negatively valenced stimuli, which has been linked to the interpersonal/affective dimension of psychopathy. However, evidence for autonomic responsivity during passive-viewing of emotion is equivocal in relation to psychopathy, although the interpretation of much of this research is limited by failures to explore the independent contribution of the dimensions of psychopathy, as well as methodological problems. The present thesis aimed to correct these issues by exploring pupil responsivity to a comprehensive set of emotional (negative and positive) and neutral images, facial expressions and sound-clips as a function of the dimensions of psychopathy. To the author's knowledge, pupillometry has not been applied previously to explore psychopathy despite the paradigm's advantage as a fast, relatively cheap and non-intrusive measure of autonomic responsivity to emotion.

No dimension of psychopathy was related to pupil responsivity to passive viewing of emotion within an undergraduate population, as measured by the Triarchic Psychopathy Measure (Tri-PM), although this was argued to be due to a lack of psychopathy within the sample. However, within a forensic psychiatric population, where the levels of psychopathy were higher, three principal findings emerged. First, the interpersonal/affective dimension of psychopathy, as measured by the Psychopathy Checklist-Revised (PCL-R), was selectively associated to diminished pupil responsivity to emotion across images, facial expressions and sound-clips. Second, this association was specific to negative stimuli (across each stimuli domain), with the dimensions of psychopathy unrelated to positive stimuli. Finally, this association was specific to early pupil responses to negative stimuli.

5.1 Theoretical implications

The current findings fit with a growing body of research that indicates that the interpersonal/affective component of psychopathy is selectively associated to an

emotional processing deficit (see 1.3 Psychopathy and emotional processing), and demonstrates that this impairment extends to autonomic responsivity during passive-viewing. This finding relates most closely to Benning, Patrick, and Iacono (2005) who reported a similar specific negative impairment as Fearless Dominance within a community population was associated to smaller increases in skin conductance response (SCR) to specifically negative (compare to neutral) images. However, Benning, Patrick, and Iacono (2005) reported that positive images led to larger SCRs than negative images across the sample and so their results could alternatively reflect that psychopathy is associated to an impairment to emotional stimuli that only emerges at lower (subtler) levels of emotional arousal. However, the present thesis was able to demonstrate that autonomic hypo-responsivity in relation to interpersonal/affective psychopathy traits was specific to negative stimuli, as both negative and positive stimuli led to equivalent increases in pupil diameter. The consistency of the current findings across visual and auditory stimuli domains was particularly promising. However, the current results require replication given that a number of studies found a differing pattern of autonomic responsivity across psychopathy (see 1.3.4 Passive-viewing of emotion in psychopathy).

This thesis provides further evidence of the necessity to investigate psychopathy as a multi-faceted construct. Further, the findings support a dual pathway model that considers that the interpersonal/affective features of psychopathy reflect a fearless temperament caused by an insensitivity in the brain's defensive motivational system. This is consistent with the wealth of research that indicates that interpersonal/affective psychopathy traits are associated to deficient fear-potentiated startle response (see 1.3.3 The dimensions of psychopathy and emotional processing), an indicator of defensive motivational action (Lang, Bradley, & Cuthbert, 1997). This impaired reactivity in the core defensive system is considered to reflect a deficient amygdala, which will be discussed later.

A possible implication of an insensitive defensive motivation system is that individuals high in interpersonal/affective psychopathy traits will approach situations that others would quickly withdraw from, as they do not experience the same fears and apprehensions inhibiting behaviour. This may lead these individuals to engage in risky and antisocial acts, consistent with research showing that the interpersonal/affective dimension of psychopathy is associated to low harm avoidance (Benning et al., 2003; Benning, Patrick, Salekin, et al., 2005). A further consequence of a deficient defensive system is that an insensitivity to negative cues, which typically serve as a social reinforcer, may lead to further impairments in moral socialisation and empathetic learning (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006). Individuals high in interpersonal/affective psychopathy traits will be indifferent to the distress and fear of others and, therefore, will not learn to associate their actions to this negative outcome (a fearful facial expression). This will lead to behaviours characterised by a lack of affect and little concern for others, such as instrumental aggression (Blais, Solodukhin, & Forth, 2014; Forth & Flight, 2007; Glenn & Raine, 2009; Reidy, Zeichner, Miller, & Martinez, 2007; Vitacco, Neumann, Caldwell, Leistico, & Van Rybroek, 2006; Walsh, Swogger, & Kosson, 2009). Additionally, an insensitivity to negative emotionality may explain the inverse association between interpersonal/affective psychopathy traits and internalising disorders (i.e. depression, anxiety) (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005).

The current findings also relate to the idea of an emotionally deficient variant of psychopathy (primary psychopathy) and an emotionally reactive variant (secondary psychopathy), reflected in the divergence of the interpersonal/affective and lifestyle/antisocial dimensions of psychopathy. Specifically, the current thesis provides evidence for the emotionally deficient primary psychopathy variant. As well as being related to differing (and, at times, opposing) personality traits, it has been proposed that these variants of psychopathy diverge in their aetiology. Primary psychopathy is predominantly attributable to a genetic component, and secondary psychopathy is

developed through greater influence of an adverse environment disturbing emotional development (Blackburn, Logan, Donnelly, & Renwick, 2008; Karpman, 1941; Mealey, 1995; Porter, 1996). In support, research investigating emotional deficiencies and personality traits associated to primary psychopathy (e.g. fearlessness, callous/unemotional traits, low negative emotionality) have largely indicated a significant genetic influence (Baker et al., 2009; Bezdjian, Raine, Baker, & Lynam, 2011; Blair, 2006; Blonigen, Carlson, Krueger, & Patrick, 2003; da Silva, Rijo, & Salekin, 2012; Fontaine, Rijsdijk, McCrory, & Viding, 2010; Hicks et al., 2012; Larsson, Andershed, & Lichtenstein, 2006; Viding, Blair, Moffitt, & Plomin, 2005; Viding, Jones, Paul, Moffitt, & Plomin, 2008; Yildirim & Derksen, 2013), while secondary psychopathy, and the impulsive antisocial behaviour that characterises secondary psychopathy, has been predominantly linked to adverse environmental influences (Brook et al., 2010; Graham, Kimonis, Wasserman, & Kline, 2012; Hicks et al., 2012; Poythress, Skeem, & Lilienfeld, 2006; Viding et al., 2008).

However, primary and secondary psychopathy are not solely determined by genetic or environmental contributions. Primary psychopathy has been associated to physical and emotional neglect (Beaver, Vaughn, DeLisi, Barnes, & Boutwell, 2012; Kimonis, Fanti, Isoma, & Donoghue, 2013), suggesting an absence of protective factors may also be related to an emotional deficit alongside genetic influences. Additionally, Yildirim and Derksen (2015) proposed a role for genetics in the development of secondary psychopathy, as genotypes associated to dispositional traits central to secondary psychopathy (e.g. negative emotionality, hostility and impulsivity) only resulted in antisocial behaviour when exposed to social stressors (e.g. neglect), with socially adaptive behaviours evident in positive environments (Bakermans-Kranenburg, Van IJzendoorn, Pijlman, Mesman, & Juffer, 2008; Bakermans-Kranenburg & Van IJzendoorn, 2006; Belsky & Beaver, 2011; Belsky et al., 2009; Cicchetti, Rogosch, & Thibodeau, 2012; Propper, Willoughby, Halpern, Carbone, & Cox, 2007; Reif et al., 2007;

Sheese, Voelker, Rothbart, & Posner, 2007). Therefore, genetic factors exert an influence on secondary psychopathy, but this effect is mediated through an adverse environment.

As previously described, the emotional deficiency that characterises individuals high in interpersonal/affective psychopathy traits, which relates closely to primary psychopathy, has been attributed to a genetically determined attenuation of amygdala reactivity (Blair, 2005, 2006). In contrast, the emotional volatility and antisociality that characterises secondary psychopathy may be linked to impairments in the frontocortical regions that mediate planning, inhibition and emotional regulation (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Blair, 2007, 2010; Blair & Cipolotti, 2000; Buckholtz et al., 2008; Carlson, Thái, & McLarnon, 2009; Heritage & Benning, 2013; Yang & Raine, 2009; Yildirim & Derksen, 2013, 2015). A deficient amygdala for individuals high in interpersonal/affective psychopathy traits may account for specific (or at least more severe) impairments in response to negative stimuli, given that the amygdala is more responsive to negative compared to positive emotion (Adolphs et al., 1994; Adolphs et al., 1999; Calder, 1996; Morris et al., 1996).

However, this genetically-mediated hypo-responsivity to emotion may be specific to the right amygdala given that only *early* autonomic hypo-responsivity to negative stimuli was observed. The right amygdala is influential in the early and reflexive shift of attention to emotion, as well as the autonomic experience of negative emotion (see 4.5.2 Autonomic hypo-responsivity was specific to early reactivity). Therefore, the current findings of early autonomic hypo-reactivity to negative stimuli may be explained by individuals high in interpersonal/affective traits being characterised by a genetically determined attenuation in the reactivity of specifically the right amygdala. Future neurobiological research should attempt to delineate these effects to understand the genetic risk pathway that lead to the emotional deficit that characterises individuals high in the interpersonal/affective component of psychopathy. Regardless of the exact

underlying neural mechanism, the current thesis highlights that interpersonal/affective psychopathy traits are not associated to a persistent impairment in response to negative stimuli and suggests that attentional/processing factors play a role. Future research should continue to explore the interaction between emotional and attentional deficits in psychopathy.

The current thesis identified several notable findings in relation to the effect of emotional stimuli on the pupil (separate to psychopathy). Firstly, it was found that when negative and positive images or sound-clips were matched for subjective arousal then the pupil showed comparable pupil dilation compared to neutral stimuli, consistent with previous research that matched these normative arousal ratings (Bradley et al., 2008; Dietz et al., 2011; Jin et al., 2015; Partala & Surakka, 2003). This is consistent with the idea that pupil diameter reflects emotional arousal regardless of valence (Bradley et al., 2008). However, the association between subjective arousal and pupil dilation is not straight-forward given that positive images led to comparable pupil diameter to neutral images in the undergraduate population in Chapter 3, despite the positive images being rated as more subjectively arousing. This was consistent with Henderson et al. (2014) who reported that erotic images led to greater pupil dilation compared to violent images despite comparable subjective arousal ratings. It was proposed that this divergence between subjective arousal and pupil dilation may reflect Weinberg and Hajcak (2010)'s contention that autonomic arousal reflects not only perceived arousal to an affective stimulus, but also the motivational significance of that stimulus. The positive images of cuddly animals and smiling faces that the undergraduate population viewed did not convey survival-relevant information and, therefore, failed to trigger motivational systems. Additionally, it was observed across both Chapter 3 and Chapter 4 that negative facial expression led to larger pupil dilation than positive facial expressions, which was considered to reflect the greater motivational significance of negative facial expressions (compared to positive faces) as likely signals of threat.

5.2 Practical implications

Pupillometry holds advantages over alternative measures of autonomic responsivity, such as SCR, as it is fast, relatively cheap and non-intrusive. Therefore, measuring changes in pupil diameter offers a useful and advantageous paradigm to measure autonomic responsivity to emotion that has been applied in the current thesis to identify individuals high in interpersonal/affective psychopathy traits across a range of visual and auditory stimuli. This paradigm has obvious practical application in the identification of individuals with high scores on interpersonal/affective dimension within institutional settings where specific treatments can be targeted at the underlying core affective impairment. Psychopathy has been associated to poor treatment outcome in adults for interventions typically successful for reducing recidivism amongst high-risk offenders (Polaschek & Daly, 2013) and within children high in callous/unemotional traits for parent/parent-child based interventions that are successful in improving outcomes for children with emotional and behavioural problems (Hawes, Price, & Dadds, 2014). However, more recent interventions have been developed specifically for children with callous/unemotional traits targeting core deficits in emotional processing that are showing good outcomes (Datyner, Kimonis, Hunt, & Armstrong, 2016; Hawes et al., 2014; Kimonis & Armstrong, 2012); one technique is to redirect the child's attention towards salient stimuli, such as the eyes of facial expressions, a technique that leads to improvements in empathy and emotion recognition for children with callous/unemotional traits up to six months later (Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012; Dadds et al., 2006). If similar treatments are developed within adult populations, or if the findings from the current thesis translate downwards to children with callous/unemotional traits, then the current pupil response paradigm can be a useful technique to objectively identify individuals that would benefit from these emotion-specific interventions. A further clinical application of the current paradigm is that it could be employed as an objective indicator of treatment success for interventions targeting emotional processing. The

paradigm could be administered at the beginning of treatment and then throughout to assess for increased autonomic responsivity to emotion.

5.3 Directions for future research

As mentioned in the previous section, it would be useful to explore whether pupil hypo-responsivity to negative stimuli is similarly related to the interpersonal/affective features of psychopathy within children, typically indexed as callous/unemotional traits (Frick, 2009). I have described research throughout this thesis that has indicated that children and adolescent high in both callous/unemotional traits demonstrate a similar lack of responsivity to negative stimuli across behavioural, psychophysiological and neural measures suggesting that this fearless temperament is evident relatively early in the developmental lifespan.

It would also be useful to manipulate the focus of attention to explore whether individuals high in interpersonal/affective psychopathy traits show typical pupil responsivity to negative stimuli once they are directed towards the emotional features of a particular stimulus. Attentional theories of psychopathy, such as the Response Modulation Hypothesis and the Differential Amygdala Activation Hypothesis, would suggest that this pattern of findings reflects a top-down mechanism overcoming an attentional bottleneck or deficient gaze shift leading to normal processing of emotional stimuli. This would provide more convincing support that the current findings of early autonomic hypo-responsivity are as a result of an emotional-attentional impairment within individuals high in interpersonal/affective psychopathy traits.

5.4 Considerations

It was previously concluded that psychopathy was unrelated to autonomic hypo-responsivity to emotion within Chapter 3 due to a lack of psychopathy traits within the undergraduate sample (see 3.6.3 Considerations and next steps). Alternatively, it could be argued that psychopathy was unrelated to autonomic hypo-responsivity as the more

socially adaptive psychopathy construct captured by the Tri-PM is not associated to deficient emotional processing, in contrast to the maladjusted high psychopathy individual indexed by the Psychopathy Checklist-Revised (PCL-R). However, contrary to this, Esteller et al. (2016) reported that Boldness of the Tri-PM was associated to deficient potentiation of startle responses to threatening images within a sample of undergraduate students. Additionally, Gao and Raine (2010) proposed that while 'successful' high psychopathy individuals (defined as those without criminal convictions) show normal or enhanced neurobiological functioning across a number of domains that are deficient in 'unsuccessful' high psychopathy individuals, they continue to show emotional impairments including autonomic hypo-responsivity to emotion. Therefore, it seems more likely that psychopathy was unrelated to autonomic hypo-responsivity to emotion within the undergraduate sample in Chapter 3 as a result of low levels of psychopathy.

Several characteristics of the forensic psychiatric sample were previously outlined that may have affected pupil responses to emotional stimuli in Chapter 4, including mental health diagnoses and psychotropic medication (see 4.5.4 Considerations). Considerable efforts have been described to examine the influence of these factors, although it has been acknowledged that further complex interactions cannot be ruled out. Alternatively, this may highlight the strength of the current findings given that they were identified within such a complex population.

Across both Chapter 3 and Chapter 4, a limitation of the dynamic facial task is that the neutral stimuli contained less movement compared to the affective facial expressions. It could be argued any impairments in relation to psychopathy in response to emotional facial expressions may reflect an impairment in motion-responsive networks like the superior temporal sulcus (Peelen, Atkinson, & Vuilleumier, 2010; Schultz & Pilz, 2009), which has been shown to be underactive in high psychopathy individuals in response to sad and pained dynamic facial expressions (Decety et al., 2014). However,

this account does not explain why autonomic hypo-responsivity in relation to interpersonal/affective psychopathy traits was not observed for happy facial expressions as well, which showed increased facial movement compared to neutral faces. Therefore, it seems likely that the autonomic hypo-responsivity in response to dynamic negative facial expressions was not as a result of deficits in motion-sensitive neural regions.

The current thesis was not concerned with identifying the underlying mechanisms of autonomic action for changes in pupil diameter in response to emotion, but rather aimed to take advantage of the pupillometry paradigm to explore psychopathy. However, it is important to recognise that the exact underlying autonomic systems responsible for pupil diameter to emotion are not certain. Increases in autonomic activity in response to emotion are typically considered to reflect sympathetic activity (Lang & Bradley, 2010). Therefore, pupil responses to auditory emotional stimuli, in the absence of visual input, are likely to be a direct measure of sympathetic nervous system activity. However, complex visual stimuli is confounded by an initial pupil light reflex (PLR) at stimuli onset that Chapter 2 suggested was as a result of increased luminance, contrast or colour as a consequence of a transient rate-sensitive mechanism response to the degree of change for local visual signals (see 2.4 Experiment 2: Image contrast). Previous research indicates that this PLR is mediated by parasympathetic processes (see 1.4.2 The influence of emotion on the pupil). It can, therefore, be speculated that baseline-corrected pupil diameter after the PLR may incorporate both parasympathetic activity (from the PLR) and sympathetic activity (the dilation to the emotional value of the visual stimulus). Alternatively, Bradley et al. (2008) reported that pupil diameter to emotional images following the PLR was correlated to electrodermal reactivity, but not cardiovascular reactivity, which they interpreted as evidence that pupil diameter after the PLR is a measure of sympathetic nervous system activity. This finding may reflect that Bradley and colleagues measured pupil diameter over 2 - 6 seconds after stimulus onset, considerably later than the PLR which completes at around 1-second and was, therefore,

relatively distanced from its parasympathetic influence. It would be useful for future research to isolate the contribution of the sympathetic and parasympathetic autonomic nervous system for changes in the pupil diameter in response to emotion, as has been done for cognitive processes using pharmacological agents (Steinhauer et al., 2015).

It was noted previously that the negative stimuli in the current thesis could be largely considered as threatening, and therefore the results most closely support the low-fear hypothesis. However, this also means that the interpretation of the current findings are limited to threatening negative stimuli, although previous studies have identified that individuals high in interpersonal/affective psychopathy traits have shown autonomic hypo-responsivity to non-threatening negative stimuli (e.g. distress and mutilation) (Benning, Patrick, & Iacono, 2005; Blair, 1999; Blair et al., 1997; Verona et al., 2004). Future research should include specific sub-categories of threatening and non-threatening stimuli to systematically explore defensive reactivity in psychopathy across negative stimuli.

5.5 Conclusion

In conclusion, the current thesis found that the interpersonal/affective dimension of psychopathy within a forensic psychiatric sample was selectively associated to early autonomic hypo-responsivity to the passive-viewing of negative visual and auditory stimuli as measured by changes in pupil diameter. The lifestyle/antisocial dimension was unrelated to autonomic reactivity to emotion. The current findings are consistent with a dual pathway model of psychopathy that emphasises that the interpersonal/affective dimension of psychopathy reflect an underlying deficient defensive motivational system. The interpersonal/affective dimension of psychopathy was unrelated to autonomic responsivity to positive stimuli, and even showed late hyper-responsivity to happy dynamic facial expressions, suggesting that the appetitive motivational system is functioning normally (or even over-active) within individuals high in

interpersonal/affective psychopathy traits. Interestingly, autonomic hypo-responsivity in relation to psychopathy emerged for specifically early reactivity to negative stimuli considered to reflect the existence of an attentional/processing impairment. It was proposed that the current findings reflect that the interpersonal/affective dimension of psychopathy is characterised by a genetically determined attenuation of amygdala reactivity that may be specific to the right amygdala, a region influential in the early and reflexive shift of attention to emotion, as well as the autonomic experience of negative emotion. The same findings were not observed within an undergraduate population where no dimension of psychopathy was related to pupil responsivity to emotion considered as a result of a lack of psychopathy within the sample. The current pupillometry paradigm has real-world application as it can be used as a fast, relatively cheap and non-intrusive measure to identify individuals high in interpersonal/affective psychopathy traits and also as an indicator of emotion-specific intervention outcome.

References

Aboyoun, D. C., & Dabbs, J. N. (1998). The Hess pupil dilation findings: Sex or novelty? *Social Behavior and Personality, 26*(4), 415-419.

Adams, R. B., Gordon, H. L., Baird, A. A., Ambady, N., & Kleck, R. E. (2003). Effects of gaze on amygdala sensitivity to anger and fear faces. *Science, 300*(5625), 1536-1536.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. R. (1994). Impaired recognition of emotion in facial expressions following bilateral damage to the human amygdala. *Nature, 372*(6507), 669-672.

Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. R. (1995). Fear and the human amygdala. *The Journal of neuroscience, 15*(9), 5879-5891.

Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., . . . Damasio, A. R. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia, 37*(10), 1111-1117.

Agelink, M. W., Majewski, T., Wurthmann, C., Lukas, K., Ullrich, H., Linka, T., & Klieser, E. (2001). Effects of newer atypical antipsychotics on autonomic neurocardiac function: a comparison between amisulpride, olanzapine, sertindole, and clozapine. *Journal of clinical psychopharmacology, 21*(1), 8-13.

Agelink, M. W., Majewski, T. B., Andrich, J., & Mueck-Weymann, M. (2002). Short-term effects of intravenous benzodiazepines on autonomic neurocardiac regulation in humans: a comparison between midazolam, diazepam, and lorazepam. *Critical care medicine, 30*(5), 997-1006.

Aguinis, H. (2004). *Regression analysis for categorical moderators*. New York: Guilford Press.

Almeida, P. R., Seixas, M. J., Ferreira-Santos, F., Vieira, J. B., Paiva, T. O., Moreira, P. S., & Costa, P. (2015). Empathic, moral and antisocial outcomes associated with distinct components of psychopathy in healthy individuals: a Triarchic model approach. *Personality and Individual Differences, 85*, 205-211.

Alnæs, D., Sneve, M. H., Espeseth, T., Endestad, T., van de Pavert, S. H. P., & Laeng, B. (2014). Pupil size signals mental effort deployed during multiple object tracking and predicts brain activity in the dorsal attention network and the locus coeruleus. *Journal of Vision, 14*(4), 1.

Ambadar, Z., Schooler, J. W., & Cohn, J. F. (2005). Deciphering the enigmatic face the importance of facial dynamics in interpreting subtle facial expressions. *Psychological Science, 16*(5), 403-410.

Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature neuroscience, 2*(11), 1032-1037.

Andreasen, N. C., Pressler, M., Nopoulos, P., Miller, D., & Ho, B.-C. (2010). Antipsychotic dose equivalents and dose-years: a standardized method for comparing exposure to different drugs. *Biological Psychiatry, 67*(3), 255-262.

Anticevic, A., Van Snellenberg, J. X., Cohen, R. E., Repovs, G., Dowd, E. C., & Barch, D. M. (2010). Amygdala recruitment in schizophrenia in response to aversive emotional material: a meta-analysis of neuroimaging studies. *Schizophrenia bulletin, 38*(3), 608 - 621.

Applegate, C. D., Kapp, B. S., Underwood, M. D., & McNall, C. L. (1983). Autonomic and somatomotor effects of amygdala central N. stimulation in awake rabbits. *Physiology & Behavior, 31*(3), 353-360.

Arena, J. G., Blanchard, E. B., Andrasik, F., Cotch, P. A., & Myers, P. E. (1983). Reliability of psychophysiological assessment. *Behaviour Research and Therapy, 21*(4), 447-460.

Arriaga, P., Adrião, J., Madeira, F., Cavaleiro, I., Maia e Silva, A., Barahona, I., & Esteves, F. (2015). A “dry eye” for victims of violence: Effects of playing a violent video game on pupillary dilation to victims and on aggressive behavior. *Psychology of Violence, 5*(2), 199 - 208.

Ashton, C. H. (2002). *Benzodiazepines: How they work and how to withdraw (The Ashton manual)*: http://www.benzo.org.uk/manual/ accessed July 2016.

Association, A. P. (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*: American Psychiatric Publishing.

Aue, T., Flykt, A., & Scherer, K. R. (2007). First evidence for differential and sequential efferent effects of stimulus relevance and goal conduciveness appraisal. *Biol Psychol, 74*(3), 347-357.

Axelrod, B. N. (2002). Validity of the Wechsler abbreviated scale of intelligence and other very short forms of estimating intellectual functioning. *Assessment, 9*(1), 17-23.

Babiak, P., Neumann, C. S., & Hare, R. D. (2010). Corporate psychopathy: Talking the walk. *Behavioral Sciences & the Law, 28*(2), 174-193.

Babiker, A., Faye, I., Prehn, K., & Malik, A. (2015). Machine learning to differentiate between positive and negative emotions using pupil diameter. *Frontiers in Psychology, 6*(1921), 1 - 10.

Bagley, A. D., Abramowitz, C. S., & Kosson, D. S. (2009). Vocal affect recognition and psychopathy: Converging findings across traditional and cluster analytic approaches to assessing the construct. *Journal of Abnormal Psychology, 118*(2), 388.

Baker, L. A., Tuvblad, C., Reynolds, C., Zheng, M., Lozano, D. I., & Raine, A. (2009). Resting heart rate and the development of antisocial behavior from age 9 to 14: Genetic and environmental influences. *Development and Psychopathology, 21*(03), 939-960.

Bakermans-Kranenburg, M. J., Van IJzendoorn, M. H., Pijlman, F. T., Mesman, J., & Juffer, F. (2008). Experimental evidence for differential susceptibility: dopamine D4 receptor polymorphism (DRD4 VNTR) moderates intervention effects on toddlers' externalizing behavior in a randomized controlled trial. *Developmental Psychology, 44*(1), 293 - 300.

Bakermans-Kranenburg, M. J., & Van IJzendoorn, M. H. (2006). Gene-environment interaction of the dopamine D4 receptor (DRD4) and observed maternal insensitivity predicting externalizing behavior in preschoolers. *Developmental Psychobiology, 48*(5), 406-409.

Balleine, B. W., & Killcross, S. (2006). Parallel incentive processing: an integrated view of amygdala function. *Trends in Neurosciences, 29*(5), 272-279.

Bar, K. J., Boettger, M. K., Till, S., Dolicek, J., & Sauer, H. (2005). Lateralization of pupillary light reflex parameters. *Clinical Neurophysiology, 116*(4), 790-798. doi:10.1016/j.clinph.2004.11.007

Barbur, J. (2004). Learning from the pupil - studies of basic mechanisms and clincal applications. *The Visual Neurosciences, 1*, 641-656.

Barbur, J., Harlow, A., & Sahraie, A. (1992). Pupillary responses to stimulus structure, colour and movement. *Ophthalmic and Physiological Optics, 12*(2), 137-141.

Barbur, J., Wolf, J., & Lennie, P. (1998). Visual processing levels revealed by response latencies to changes in different visual attributes. *Proceedings of the Royal Society B-Biological Sciences, 265*(1412), 2321-2325.

Barke, A., Stahl, J., & Kroner-Herwig, B. (2012). Identifying a subset of fear-evoking pictures from the IAPS on the basis of dimensional and categorical ratings for a German sample. *J Behav Ther Exp Psychiatry, 43*, 565-572. doi:10.1016/j.jbtep.2011.07.006

Barry, C. T., Frick, P. J., DeShazo, T. M., McCoy, M., Ellis, M., & Loney, B. R. (2000). The importance of callous–unemotional traits for extending the concept of psychopathy to children. *Journal of Abnormal Psychology, 109*(2), 335.

Baskin-Sommers, A. R., Curtin, J. J., & Newman, J. P. (2011). Specifying the attentional selection that moderates the fearlessness of psychopathic offenders. *Psychological Science, 22*(2), 226 -234.

Baskin-Sommers, A. R., Curtin, J. J., & Newman, J. P. (2013). Emotion-modulated startle in psychopathy: clarifying familiar effects. *Journal of Abnormal Psychology, 122*(2), 458.

Bate, C., Boduszek, D., Dhingra, K., & Bale, C. (2014). Psychopathy, intelligence and emotional responding in a non-forensic sample: an experimental investigation. *The Journal of Forensic Psychiatry & Psychology, 25*(5), 600-612.

Baxter, M. G., & Murray, E. A. (2002). The amygdala and reward. *Nature Reviews Neuroscience, 3*(7), 563-573.

Bazire, S. (2014). *Psychotropic Drug Directory 2014: The Professionals' Pocket Handbook and Aide Memoire*. Dorsington: Lloyd-Reinhold Communications.

Beaver, K. M., Vaughn, M. G., DeLisi, M., Barnes, J., & Boutwell, B. B. (2012). The neuropsychological underpinnings to psychopathic personality traits in a nationally representative and longitudinal sample. *Psychiatric Quarterly, 83*(2), 145-159.

Bechara, A., Tranel, D., Damasio, H., & Adolphs, R. (1995). Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science, 269*(5227), 1115.

Belsky, J., & Beaver, K. M. (2011). Cumulative-genetic plasticity, parenting and adolescent self-regulation. *Journal of Child Psychology and Psychiatry, 52*(5), 619-626.

Belsky, J., Jonassaint, C., Pluess, M., Stanton, M., Brummett, B., & Williams, R. (2009). Vulnerability genes or plasticity genes. *Molecular psychiatry, 14*(8), 746-754.

Benning, S. D., Patrick, C. J., Blonigen, D. M., Hicks, B. M., & Iacono, W. G. (2005). Estimating facets of psychopathy from normal personality traits a step toward community epidemiological investigations. *Assessment, 12*(1), 3-18.

Benning, S. D., Patrick, C. J., Hicks, B. M., Blonigen, D. M., & Krueger, R. F. (2003). Factor structure of the psychopathic personality inventory: validity and implications for clinical assessment. *Psychological Assessment, 15*(3), 340-350.

Benning, S. D., Patrick, C. J., & Iacono, W. G. (2005). Psychopathy, startle blink modulation, and electrodermal reactivity in twin men. *Psychophysiology, 42*(6), 753-762.

Benning, S. D., Patrick, C. J., Salekin, R. T., & Leistico, A.-M. R. (2005). Convergent and Discriminant Validity of Psychopathy Factors Assessed Via Self-Report A Comparison of Three Instruments. *Assessment, 12*(3), 270-289.

Benzodiazepine equivalence table. (2007). *Retrieved from* http://www.benzo.org.uk/bzequiv.htm

Bernat, E., Patrick, C. J., Benning, S. D., & Tellegen, A. (2006). Effects of picture content and intensity on affective physiological response. *Psychophysiology, 43*(1), 93-103.

Berntson, G. G., Boysen, S. T., Bauer, H. R., & Torello, M. S. (1989). Conspecific screams and laughter: Cardiac and behavioral reactions of infant chimpanzees. *Developmental Psychobiology, 22*(8), 771-787.

Bezdjian, S., Raine, A., Baker, L., & Lynam, D. (2011). Psychopathic personality in children: genetic and environmental contributions. *Psychological Medicine, 41*(03), 589-600.

Binda, P., Pereverzeva, M., & Murray, S. O. (2013). Attention to bright surfaces enhances the pupillary light reflex. *The Journal of neuroscience, 33*(5), 2199-2204.

Binda, P., Pereverzeva, M., & Murray, S. O. (2014). Pupil size reflects the focus of feature-based attention. *Journal of Neurophysiology, 112*(12), 3046-3052.

Birbaumer, N., Viet, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., & Flor, H. (2005). Deficient fear conditioning in psychopathy - A functional magnetic resonance imaging study. *Archives of General Psychiatry, 62*(7), 799-805. doi:10.1001/archpsyc.62.7.799

Blackburn, R. (2007). Personality disorder and antisocial deviance: Comments on the debate on the structure of the psychopathy checklist-revised. *Journal of Personality Disorders, 21*(2), -159142.

Blackburn, R., Logan, C., Donnelly, J. P., & Renwick, S. J. (2008). Identifying psychopathic subtypes: Combining an empirical personality classification of offenders with the Psychopathy Checklist-Revised. *Journal of Personality Disorders, 22*(6), 604-622.

Blair, K., Morton, J., Leonard, A., & Blair, R. (2006). Impaired decision-making on the basis of both reward and punishment information in individuals with psychopathy. *Personality and Individual Differences, 41*(1), 155-165.

Blair, K., Richell, R., Mitchell, D., Leonard, A., Morton, J., & Blair, R. (2006). They know the words, but not the music: Affective and semantic priming in individuals with psychopathy. *Biol Psychol, 73*(2), 114-123.

Blair, R. (1999). Responsiveness to distress cues in the child with psychopathic tendencies. *Personality and Individual Differences, 27*(1), 135-145.

Blair, R. (2005). Applying a cognitive neuroscience perspective to the disorder of psychopathy. *Development and Psychopathology, 17*(03), 865-891.

Blair, R. (2006). The emergence of psychopathy: Implications for the neuropsychological approach to developmental disorders. *Cognition, 101*(2), 414-442.

Blair, R. (2007). Dysfunctions of medial and lateral orbitofrontal cortex in psychopathy. *Annals of the New York Academy of Sciences, 1121*(1), 461-479.

Blair, R. (2010). Neuroimaging of psychopathy and antisocial behavior: a targeted review. *Current psychiatry reports, 12*(1), 76-82.

Blair, R. (2013). Psychopathy: cognitive and neural dysfunction. *Dialogues in clinical neuroscience, 15*(2), 181-190.

Blair, R., Budhani, S., Colledge, E., & Scott, S. (2005). Deafness to fear in boys with psychopathic tendencies. *Journal of Child Psychology and Psychiatry, 46*(3), 327-336.

Blair, R., Colledge, E., Murray, L., & Mitchell, D. G. V. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology, 29*(6), 491-498. doi:10.1023/a:1012225108281

Blair, R., Jones, L., Clark, F., & Smith, M. (1997). The psychopathic individual: a lack of responsiveness to distress cues? *Psychophysiology, 34*(2), 192-198.

Blair, R., Mitchell, D. G. V., Peschardt, K. S., Colledge, E., Leonard, R. A., Shine, J. H., . . . Perrett, D. I. (2004). Reduced sensitivity to others' fearful expressions in psychopathic individuals. *Personality and Individual Differences, 37*(6), 1111-1122. doi:10.1016/j.paid.2003.10.008

Blair, R., Mitchell, D. G. V., Richell, R. A., Kelly, S., Leonard, A., Newman, C., & Scott, S. K. (2002). Turning a deaf ear to fear: Impaired recognition of vocal affect in psychopathic individuals. *Journal of Abnormal Psychology, 111*(4), 682-686. doi:10.1037//0021-843x.111.4.682

Blair, R. J., & Cipolotti, L. (2000). Impaired social response reversal. *Brain, 123*(6), 1122-1141.

Blair, R. J. R. (1995). A cognitive developmental approach to morality: Investigating the psychopath. *Cognition, 57*(1), 1-29.

Blair, R. J. R., Peschardt, K., Budhani, S., Mitchell, D., & Pine, D. (2006). The development of psychopathy. *Journal of Child Psychology and Psychiatry, 47*(3-4), 262-276.

Blais, J., Solodukhin, E., & Forth, A. E. (2014). A meta-analysis exploring the relationship between psychopathy and instrumental versus reactive violence. *Criminal Justice and Behavior, 41*(7), 797-821.

Blonigen, D. M., Carlson, S. R., Krueger, R. F., & Patrick, C. J. (2003). A twin study of self-reported psychopathic personality traits. *Personality and Individual Differences, 35*(1), 179-197.

Blonigen, D. M., Hicks, B. M., Krueger, R. F., Patrick, C. J., & Iacono, W. G. (2005). Psychopathic personality traits: Heritability and genetic overlap with internalizing and externalizing psychopathology. *Psychological Medicine, 35*(5), 637-648.

Boccardi, M., Frisoni, G. B., Hare, R. D., Cavedo, E., Najt, P., Pievani, M., . . . Tiihonen, J. (2011). Cortex and amygdala morphology in psychopathy. *Psychiatry Research-Neuroimaging, 193*(2), 85-92. doi:10.1016/j.pscychresns.2010.12.013

Book, A. S., Quinsey, V. L., & Langford, D. (2007). Psychopathy and the perception of affect and vulnerability. *Criminal Justice and Behavior, 34*(4), 531-544.

Bowen, K. L., Morgan, J. E., Moore, S. C., & van Goozen, S. H. M. (2013). Young offenders' emotion recognition dysfunction across emotion intensities: Explaining variation using psychopathic traits, conduct disorder and offense severity. *Journal of Psychopathology and Behavioral Assessment, 36*(1), 60-73. doi:10.1007/s10862-013-9368-z

Bradley, M. M., Codispoti, M., Cuthbert, B. N., & Lang, P. J. (2001). Emotion and Motivation I: Defensive and Appetitive Reactions in Picture Processing. *Emotion, 1*(3), 276-298. doi:10.1037//1528-3542.1.3.276

Bradley, M. M., Codispoti, M., Sabatinelli, D., & Lang, P. J. (2001). Emotion and motivation II: Sex Differences in Picture Processing. *Emotion, 1*(3), 300 - 319.

Bradley, M. M., & Lang, P. J. (2000). Affective reactions to acoustic stimuli. *Psychophysiology, 37*(2), 204-215. doi:10.1017/s0048577200990012

Bradley, M. M., & Lang, P. J. (2007). *The International Affective Digitized Sounds (2nd Edition; IADS-2): Affective ratings of sounds and instruction manual. Technical report B-3.* University of Florida, Gainesville, Fl.

Bradley, M. M., & Lang, P. J. (2015). Memory, emotion, and pupil diameter: Repetition of natural scenes. *Psychophysiology, 52*(9), 1186-1193.

Bradley, M. M., Lang, P. J., & Cuthbert, B. N. (1993). Emotion, novelty, and the startle reflex: habituation in humans. *Behavioral neuroscience, 107*(6), 970-980.

Bradley, M. M., Miccoli, L., Escrig, M., & Lang, P. J. (2008). The pupil as a measure of emotional arousal and autonomic activation. *Psychophysiology, 45*(4), 602-607. doi:10.1111/j.1469-8986.2008.00654.x

Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., . . . Rosen, B. R. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron, 17*(5), 875-887.

Brinkley, C. A., Schmitt, W. A., Smith, S. S., & Newman, J. P. (2001). Construct validation of a self-report psychopathy scale: does Levenson's self-report psychopathy scale measure the same constructs as Hare's psychopathy checklist-revised? *Personality and Individual Differences, 31*(7), 1021-1038.

Brook, M., Brieman, C. L., & Kosson, D. S. (2013). Emotion processing in Psychopathy Checklist - assessed psychopathy: A review of the literature. *Clinical Psychology Review, 33*(8), 979-995. doi:10.1016/j.cpr.2013.07.008

Brook, M., & Kosson, D. S. (2013). Impaired cognitive empathy in criminal psychopathy: Evidence from a laboratory measure of empathic accuracy. *Journal of Abnormal Psychology, 122*(1), 156 - 166.

Brook, M., Panizzon, M. S., Kosson, D. S., Sullivan, E. A., Lyons, M. J., Franz, C. E., . . . Kremen, W. S. (2010). Psychopathic personality traits in middle-aged male twins: a behavior genetic investigation. *Journal of Personality Disorders, 24*(4), 473-486.

Buckholtz, J., Callicott, J., Kolachana, B., Hariri, A., Goldberg, T., Genderson, M., . . . Meyer-Lindenberg, A. (2008). Genetic variation in MAOA modulates ventromedial prefrontal circuitry mediating individual differences in human personality. *Molecular psychiatry, 13*(3), 313-324.

Burgdorf, J., & Panksepp, J. (2006). The neurobiology of positive emotions. *Neuroscience & Biobehavioral Reviews, 30*(2), 173-187.

Burkhouse, K. L., Owens, M., Feurer, C., Sosoo, E., Kudinova, A., & Gibb, B. E. (2017). Increased Neural and Pupillary Reactivity to Emotional Faces in Adolescents with Current and Remitted Major Depressive Disorder. *Social Cognitive and Affective Neuroscience*. doi:10.1093/scan/nsw184

Burkhouse, K. L., Siegle, G. J., & Gibb, B. E. (2014). Pupillary reactivity to emotional stimuli in children of depressed and anxious mothers. *Journal of Child Psychology and Psychiatry, 55*(9), 1009-1016.

Burkhouse, K. L., Siegle, G. J., Woody, M. L., Kudinova, A. Y., & Gibb, B. E. (2015). Pupillary reactivity to sad stimuli as a biomarker of depression risk: Evidence from a prospective study of children. *Journal of Abnormal Psychology, 124*(3), 498-506.

Calder, A. J. (1996). Facial emotion recognition after bilateral amygdala damage: differentially severe impairment of fear. *Cognitive Neuropsychology, 13*(5), 699-745.

Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait-multimethod matrix. *Psychological Bulletin, 56*(2), 81-105.

Canli, T., Sivers, H., Whitfield, S. L., Gotlib, I. H., & Gabrieli, J. D. (2002). Amygdala response to happy faces as a function of extraversion. *Science, 296*(5576), 2191.

Carlson, S. R., Thái, S., & McLarnon, M. E. (2009). Visual P3 amplitude and self-reported psychopathic personality traits: Frontal reduction is associated with self-centered impulsivity. *Psychophysiology, 46*(1), 100-113.

Carré, J. M., Hyde, L. W., Neumann, C. S., Viding, E., & Hariri, A. R. (2013). The neural signatures of distinct psychopathic traits. *Social Neuroscience, 8*(2), 122-135.

Chae, Y., Lee, J. C., Park, K. M., Kang, O. S., Park, H. J., & Lee, H. (2008). Subjective and autonomic responses to smoking-related visual cues. *Journal of Physiological Sciences, 58*(2), 139-145. doi:10.2170/physiolsci.RP014207

Cicchetti, D., Rogosch, F. A., & Thibodeau, E. L. (2012). The effects of child maltreatment on early signs of antisocial behavior: Genetic moderation by tryptophan hydroxylase, serotonin transporter, and monoamine oxidase A genes. *Development and Psychopathology, 24*(03), 907-928.

Cleckley, H. (1941). *The mask of sanity; an attempt to reinterpret the so-called psychopathic personality*. Oxford, England: Mosby.

Clynes, M. (1961). Unidirectional rate sensitivity: A biocybernetic law of reflex and humoral systems as physiologic channels of control and communication. *Annals of the New York Academy of Sciences, 92*(3), 946-969.

Codispoti, M., & De Cesarei, A. (2007). Arousal and attention: Picture size and emotional reactions. *Psychophysiology, 44*(5), 680-686.

Codispoti, M., Mazzetti, M., & Bradley, M. M. (2009). Unmasking emotion: Exposure duration and emotional engagement. *Psychophysiology, 46*(4), 731-738.

Cohen, H., Loewenthal, U., Matar, M., & Kotler, M. (2001). Association of autonomic dysfunction and clozapine. *The British Journal of Psychiatry, 179*(2), 167-171.

Cohen, J. (1977). Statistical power analysis for the behavioral sciences (revised ed.): New York: Academic Press.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155-159. doi:10.1037/0033-2909.112.1.155

Cohen, N., Moyal, N., & Henik, A. (2015). Executive control suppresses pupillary responses to aversive stimuli. *Biol Psychol, 112*, 1-11.

Coid, J., Freestone, M., & Ullrich, S. (2012). Subtypes of psychopathy in the British household population: findings from the national household survey of psychiatric morbidity. *Social psychiatry and psychiatric epidemiology, 47*(6), 879-891.

Coid, J., & Yang, M. (2008). The distribution of psychopathy among a household population: categorical or dimensional? *Social psychiatry and psychiatric epidemiology, 43*(10), 773-781.

Coid, J., Yang, M., Ullrich, S., Roberts, A., & Hare, R. D. (2009). Prevalence and correlates of psychopathic traits in the household population of Great Britain. *International journal of law and psychiatry, 32*(2), 65-73.

Coid, J., Yang, M., Ullrich, S., Roberts, A., Moran, P., Bebbington, P., . . . Lewis, G. (2009). Psychopathy among prisoners in England and Wales. *International journal of law and psychiatry, 32*(3), 134-141.

Contreras-Rodriguez, O., Pujol, J., Batalla, I., Harrison, B. J., Bosque, J., Ibern-Regas, I., . . . Cardoner, N. (2014). Disrupted neural processing of emotional faces in psychopathy. *Social Cognitive and Affective Neuroscience, 9*(4), 505-512. doi:10.1093/scan/nst014

Cooke, D. J., & Michie, C. (1999). Psychopathy across cultures: North America and Scotland compared. *Journal of Abnormal Psychology, 108*(1), 58-68.

Cooke, D. J., & Michie, C. (2001). Refining the construct of psychopathy: towards a hierarchical model. *Psychological Assessment, 13*(2), 171-188.

Cooke, D. J., Michie, C., Hart, S. D., & Clark, D. (2005). Assessing psychopathy in the UK: concerns about cross-cultural generalisability. *The British Journal of Psychiatry, 186*(4), 335-341.

Cooke, D. J., Michie, C., Hart, S. D., & Patrick, C. (2006). Facets of clinical psychopathy. *Handbook of psychopathy*, 91-106.

Copestake, S., Gray, N. S., & Snowden, R. J. (2011). A comparison of a self-report measure of psychopathy with the psychopathy checklist-revised in a UK sample of offenders. *Journal of Forensic Psychiatry & Psychology, 22*(2), 169-182.

Costafreda, S. G., Brammer, M. J., David, A. S., & Fu, C. H. (2008). Predictors of amygdala activation during the processing of emotional stimuli: a meta-analysis of 385 PET and fMRI studies. *Brain research reviews, 58*(1), 57-70.

Craig, R. L., Gray, N. S., & Snowden, R. J. (2013). Recalled parental bonding, current attachment, and the triarchic conceptualisation of psychopathy. *Personality and Individual Differences, 55*(4), 345-350.

Craney, T. A., & Surles, J. G. (2002). Model-dependent variance inflation factor cutoff values. *Quality Engineering, 14*(3), 391-403.

Cumming, G. (2013). *Understanding the new statistics: Effect sizes, confidence intervals, and meta-analysis*. New York: Routledge.

Cuthbert, B. N., Schupp, H. T., Bradley, M. M., Birbaumer, N., & Lang, P. J. (2000). Brain potentials in affective picture processing: covariation with autonomic arousal and affective report. *Biol Psychol, 52*(2), 95-111. doi:http://dx.doi.org/10.1016/S0301-0511(99)00044-7

da Silva, D. R., Rijo, D., & Salekin, R. T. (2012). Child and adolescent psychopathy: A state-of-the-art reflection on the construct and etiological theories. *Journal of Criminal Justice, 40*(4), 269-277.

Dabbs, J. M. (1997). Testosterone and pupillary response to auditory sexual stimuli. *Physiology & Behavior, 62*(4), 909-912. doi:10.1016/s0031-9384(97)00268-0

Dadds, M. R., Cauchi, A. J., Wimalaweera, S., Hawes, D. J., & Brennan, J. (2012). Outcomes, moderators, and mediators of empathic-emotion recognition training for complex conduct problems in childhood. *Psychiatry research, 199*(3), 201-207.

Dadds, M. R., El Masry, Y., Wimalaweera, S., & Guastella, A. J. (2008). Reduced eye gaze explains "Fear Blindness" in childhood psychopathic traits. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*(4), 455-463. doi:10.1097/CHI.0b013e31816407f1

Dadds, M. R., Perry, Y., Hawes, D. J., Merz, S., Riddell, A. C., Haines, D. J., . . . Abeygunawardane, A. I. (2006). Attention to the eyes and fear-recognition deficits in child psychopathy. *British Journal of Psychiatry, 189*, 280-281. doi:10.1192/bjp.bp.105.018150

Danivas, V., & Venkatasubramanian, G. (2013). Current perspectives on chlorpromazine equivalents: Comparing apples and oranges! *Indian journal of psychiatry, 55*(2), 207-208.

Datyner, A., Kimonis, E. R., Hunt, E., & Armstrong, K. (2016). Using a Novel Emotional Skills Module to Enhance Empathic Responding for a Child With Conduct Disorder With Limited Prosocial Emotions. *Clinical Case Studies, 15*(1), 35-52.

Dawel, A., O'Kearney, R., McKone, E., & Palermo, R. (2012). Not just fear and sadness: meta-analytic evidence of pervasive emotion recognition deficits for facial and vocal expressions in psychopathy. *Neuroscience & Biobehavioral Reviews, 36*(10), 2288-2304.

Decety, J., Skelly, L., Yoder, K. J., & Kiehl, K. A. (2014). Neural processing of dynamic emotional facial expressions in psychopaths. *Social Neuroscience, 9*(1), 36-49.

Decety, J., Skelly, L. R., & Kiehl, K. A. (2013). Brain response to empathy-eliciting scenarios involving pain in incarcerated individuals with psychopathy. *JAMA psychiatry, 70*(6), 638-645.

Del Gaizo, A. L., & Falkenbach, D. M. (2008). Primary and secondary psychopathic-traits and their relationship to perception and experience of emotion. *Personality and Individual Differences, 45*(3), 206-212.

Dennis, M., Francis, D. J., Cirino, P. T., Schachar, R., Barnes, M. A., & Fletcher, J. M. (2009). Why IQ is not a covariate in cognitive studies of neurodevelopmental disorders. *Journal of the International Neuropsychological Society, 15*(03), 331-343.

Dietz, J., Bradley, M. M., Okun, M., & Bowers, D. (2011). Emotion and ocular responses in Parkinson's disease. *Neuropsychologia, 49*(12), 3247-3253. doi:10.1016/j.neuropsychologia.2011.07.029

Digman, J. M. (1990). Personality structure: Emergence of the five-factor model. *Annual review of psychology, 41*(1), 417-440.

Dindo, L., & Fowles, D. (2011). Dual temperamental risk factors for psychopathic personality: evidence from self-report and skin conductance. *Journal of Personality and Social Psychology, 100*(3), 557-566.

Dolan, M., & Doyle, M. (2000). Violence risk prediction. *The British Journal of Psychiatry, 177*(4), 303-311.

Dolan, M. C., & Fullam, R. S. (2006). Face affect recognition deficits in personality-disordered offenders: Association with psychopathy. *Psychological Medicine, 36*(11), 1563-1569. doi:10.1017/s0033291706008634

Dolan, M. C., & Fullam, R. S. (2009). Psychopathy and functional magnetic resonance imaging blood oxygenation level-dependent responses to emotional faces in violent patients with schizophrenia. *Biological Psychiatry, 66*(6), 570-577.

Donegan, N. H., Sanislow, C. A., Blumberg, H. P., Fulbright, R. K., Lacadie, C., Skudlarski, P., . . . Wexler, B. E. (2003). Amygdala hyperreactivity in borderline personality disorder: Implications for emotional dysregulation. *Biological Psychiatry, 54*(11), 1284-1293. doi:10.1016/s0006-3223(03)00636-x

Drislane, L. E., Patrick, C. J., & Arsal, G. (2014). Clarifying the content coverage of differing psychopathy inventories through reference to the Triarchic Psychopathy Measure. *Psychological Assessment, 26*(2), 350-362.

Drislane, L. E., Patrick, C. J., Sourander, A., Sillanmäki, L., Aggen, S. H., Elonheimo, H., . . . Kendler, K. S. (2014). Distinct variants of extreme psychopathic individuals in society at large: Evidence from a population-based sample. *Personality Disorders: Theory, research, and treatment, 5*(2), 154-163.

Duque, A., Sanchez, A., & Vazquez, C. (2014). Gaze-fixation and pupil dilation in the processing of emotional faces: The role of rumination. *Cognition and Emotion, 28*(8), 1347-1366.

Edalati, H., Walsh, Z., & Kosson, D. S. (2015). Attentional Bias in Psychopathy An Examination of the Emotional Dot-Probe Task in Male Jail Inmates. *International Journal of Offender Therapy and Comparative Criminology, 60*(11), 1344-1357.

Edens, J. F., Marcus, D. K., Lilienfeld, S. O., & Poythress Jr, N. G. (2006). Psychopathic, not psychopath: taxometric evidence for the dimensional structure of psychopathy. *Journal of Abnormal Psychology, 115*(1), 131-144.

Edens, J. F., Marcus, D. K., & Vaughn, M. G. (2011). Exploring the taxometric status of psychopathy among youthful offenders: is there a juvenile psychopath taxon? *Law and human behavior, 35*(1), 13-24.

Esteller, À., Poy, R., & Moltó, J. (2016). Deficient aversive-potentiated startle and the Triarchic model of psychopathy: the role of boldness. *Biol Psychol, 117*, 131-140.

Everitt, B. J., Cardinal, R. N., Parkinson, J. A., & Robbins, T. W. (2003). Appetitive behavior. *Annals of the New York Academy of Sciences, 985*(1), 233-250.

Falkenbach, D., Poythress, N., & Creevy, C. (2008). The exploration of subclinical psychopathic subtypes and the relationship with types of aggression. *Personality and Individual Differences, 44*(4), 821-832.

Falkenbach, D. M., Stern, S. B., & Creevy, C. (2014). Psychopathy variants: Empirical evidence supporting a subtyping model in a community sample. *Personality Disorders: Theory, research, and treatment, 5*(1), 10-19.

Farzin, F., Rivera, S. M., & Hessl, D. (2009). Brief report: Visual processing of faces in individuals with fragile X syndrome: An eye tracking study. *Journal of autism and developmental disorders, 39*(6), 946-952.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*(2), 175-191.

Fecteau, S., Belin, P., Joanette, Y., & Armony, J. L. (2007). Amygdala responses to nonlinguistic emotional vocalizations. *Neuroimage, 36*(2), 480-487.

Ferrari, V., De Cesarei, A., Mastria, S., Lugli, L., Baroni, G., Nicoletti, R., & Codispoti, M. (2016). Novelty and emotion: Pupillary and cortical responses during viewing of natural scenes. *Biol Psychol, 113*, 75-82.

Firestone, P., Bradford, J. M., Greenberg, D. M., & Larose, M. R. (1998). Homicidal sex offenders: Psychological, phallometric, and diagnostic features. *Journal of the American Academy of Psychiatry and the Law Online, 26*(4), 537-552.

Flor, H., Birbaumer, N., Hermann, C., Ziegler, S., & Patrick, C. J. (2002). Aversive Pavlovian conditioning in psychopaths: Peripheral and central correlates. *Psychophysiology, 39*(4), 505-518. doi:10.1017/s0048577202394046

Fontaine, N. M., Rijsdijk, F. V., McCrory, E. J., & Viding, E. (2010). Etiology of different developmental trajectories of callous-unemotional traits. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(7), 656-664.

Forouzan, E., & Cooke, D. J. (2005). Figuring out la femme fatale: Conceptual and assessment issues concerning psychopathy in females. *Behavioral Sciences & the Law, 23*(6), 765-778.

Forth, A. E., & Flight, J. I. (2007). Instrumentally violent youth: The roles of psychopathic traits, empathy, and attachment. *Criminal Justice and Behavior, 34*(6), 739-751.

Forth, A. E., Kosson, D. S., Hare, R. D., Sevecke, K., & Krischer, M. K. (2014). *Hare Psychopathy checklist, youth version: PCL: YV*: Hogrefe.

Fotiou, F., Fountoulakis, K., Goulas, A., Alexopoulos, L., & Palikaras, A. (2000). Automated standardized pupillometry with optical method for purposes of clinical practice and research. *Clinical Physiology, 20*(5), 336-347.

Fowles, D. C. (1980). The 3 arousal model - implications of Gray's 2-factor learning-theory for heart-rate, electrodermal activity, and psychopathy. *Psychophysiology, 17*(2), 87-104. doi:10.1111/j.1469-8986.1980.tb00117.x

Fowles, D. C., & Dindo, L. (2006). A dual-deficit model of psychopathy. *Handbook of psychopathy*, 14-34.

Fowles, D. C., & Dindo, L. (2009). Temperament and psychopathy: A dual-pathway model. *Current directions in psychological science, 18*(3), 179-183.

Fox, E., Lester, V., Russo, R., Bowles, R., Pichler, A., & Dutton, K. (2000). Facial expressions of emotion: Are angry faces detected more efficiently? *Cognition & Emotion, 14*(1), 61-92.

Frick, P. J. (2009). Extending the construct of psychopathy to youth: Implications for understanding, diagnosing, and treating antisocial children and adolescents. *The Canadian Journal of Psychiatry, 54*(12), 803-812.

Fridlund, A. J. (2014). *Human facial expression: An evolutionary view*. San Diego, California: Academic Press, Inc.

Fujibayashi, M., Matsumoto, T., Kishida, I., Kimura, T., Ishii, C., Ishii, N., & Moritani, T. (2009). Autonomic nervous system activity and psychiatric severity in schizophrenia. *Psychiatry and clinical neurosciences, 63*(4), 538-545.

Furmark, T., Fischer, H., Wik, G., Larsson, M., & Fredrikson, M. (1997). The amygdala and individual differences in human fear conditioning. *Neuroreport, 8*(18), 3957-3960.

Fusar-Poli, P., Landi, P., & O'Connor, C. (2009). Neurophysiological response to emotional faces with increasing intensity of fear: A skin conductance response study. *Journal of Clinical Neuroscience, 16*(7), 981-982.

Gamlin, P. D. R., Zhang, H. Y., Harlow, A., & Barbur, J. L. (1998). Pupil responses to stimulus color, structure and light flux increments in the rhesus monkey. *Vision Research, 38*(21), 3353-3358. doi:10.1016/s0042-6989(98)00096-0

Gao, Y., & Raine, A. (2010). Successful and unsuccessful psychopaths: A neurobiological model. *Behavioral Sciences & the Law, 28*(2), 194-210.

Gaughan, E. T., Miller, J. D., & Lynam, D. R. (2012). Examining the utility of general models of personality in the study of psychopathy: A comparison of the HEXACO-PI-R and NEO PI-R. *Journal of Personality Disorders, 26*(4), 513-523.

Geangu, E., Hauf, P., Bhardwaj, R., & Bentz, W. (2011). Infant Pupil Diameter Changes in Response to Others' Positive and Negative Emotions. *PLoS ONE, 6*(11).

Ghasemi, A., & Zahediasl, S. (2012). Normality tests for statistical analysis: a guide for non-statisticians. *International journal of endocrinology and metabolism, 10*(2), 486-489.

Gilchrist, I. D. (2011). Saccades. In S.P Liversedge, I.D. Gilchrist, & S. Everling (Eds.), *The Oxford handbook of eye movements* (pp. 85 - 94). Oxford, UK: Oxford University Press.

Gingras, B., Marin, M. M., Puig-Waldmüller, E., & Fitch, W. (2015). The Eye is Listening: Music-Induced Arousal and Individual Differences Predict Pupillary Responses. *Frontiers in Human Neuroscience, 9*(619), 1-12.

Gläscher, J., & Adolphs, R. (2003). Processing of the arousal of subliminal and supraliminal emotional stimuli by the human amygdala. *The Journal of neuroscience, 23*(32), 10274-10282.

Glenn, A. L., & Raine, A. (2009). Psychopathy and instrumental aggression: Evolutionary, neurobiological, and legal perspectives. *International journal of law and psychiatry, 32*(4), 253-258.

Glenn, A. L., Raine, A., & Schug, R. A. (2009). The neural correlates of moral decision-making in psychopathy. *Molecular psychiatry, 14*(5-6), 1-10.

Gloor, P. (1997). *The temporal lobe and limbic system*: Oxford University Press, USA.

Gordon, H. L., Baird, A. A., & End, A. (2004). Functional differences among those high and low on a trait measure of psychopathy. *Biological Psychiatry, 56*(7), 516-521.

Graham, J. W., Olchowski, A. E., & Gilreath, T. D. (2007). How many imputations are really needed? Some practical clarifications of multiple imputation theory. *Prevention Science, 8*(3), 206-213.

Graham, N., Kimonis, E. R., Wasserman, A. L., & Kline, S. M. (2012). Associations among childhood abuse and psychopathy facets in male sexual offenders. *Personality Disorders: Theory, research, and treatment, 3*(1), 66-75.

Grann, M., Langstrom, N., Tengstrom, A., & Stalenheim, E. G. (1998). Reliability of file-based retrospective ratings of psychopathy with the PCL-R. *Journal of Personality Assessment, 70*(3), 416-426.

Guay, J.-P., Ruscio, J., Knight, R. A., & Hare, R. D. (2007). A taxometric analysis of the latent structure of psychopathy: evidence for dimensionality. *Journal of Abnormal Psychology, 116*(4), 701 -716.

Hall, J. R., Drislane, L. E., Patrick, C. J., Morano, M., Lilienfeld, S. O., & Poythress, N. G. (2014). Development and validation of Triarchic construct scales from the psychopathic personality inventory. *Psychological Assessment, 26*(2), 447-461.

Hamann, S., & Mao, H. (2002). Positive and negative emotional verbal stimuli elicit activity in the left amygdala. *Neuroreport, 13*(1), 15-19.

Hamann, S. B., Stefanacci, L., Squire, L. R., Adolphs, R., Tranel, D., Damasio, H., & Damasio, A. (1996). Recognizing facial emotion. *Nature, 379*(6565), 497.

Han, T., Alders, G. L., Greening, S. G., Neufeld, R. W., & Mitchell, D. G. (2012). Do fearful eyes activate empathy-related brain regions in individuals with callous traits? *Social Cognitive and Affective Neuroscience, 7*, 958 - 968.

Hardee, J. E., Thompson, J. C., & Puce, A. (2008). The left amygdala knows fear: Laterality in the amygdala response to fearful eyes. *Social Cognitive and Affective Neuroscience, 3*(1), 47-54.

Hare, R. D. (1991). *The Hare psychopathy checklist-revised: Manual*: Multi-Health Systems, Incorporated.

Hare, R. D. (1999). *Without conscience: The disturbing world of the psychopaths among us*: Guilford Press.

Hare, R. D. (2003). *The Hare Psychopathy Checklist – Revised (PCL-R) (2nd edition)*. Toronto: Multi-Health Systems.

Hare, R. D., & Neumann, C. S. (2005). Structural models of psychopathy. *Current psychiatry reports, 7*(1), 57-64.

Hare, R. D., & Neumann, C. S. (2008). Psychopathy as a clinical and empirical construct. *Annu. Rev. Clin. Psychol., 4*, 217-246.

Hare, R. D., & Neumann, C. S. (2010). Psychopathy: Assessment and forensic implications. In L. Malatesti & J. McMillan (Eds.), *Responsibility and psychopathy: Interfacing law, psychiatry and philosophy* (pp. 93-123). New York: Oxford University Press.

Harenski, C. L., Edwards, B. G., Harenski, K. A., & Kiehl, K. A. (2014). Neural correlates of moral and non-moral emotion in female psychopathy. *Frontiers in Human Neuroscience, 8*, 741.

Harenski, C. L., Harenski, K. A., Shane, M. S., & Kiehl, K. A. (2010). Aberrant neural processing of moral violations in criminal psychopaths. *Journal of Abnormal Psychology, 119*(4), 863-874.

Harenski, C. L., Kim, S. H., & Hamann, S. (2009). Neuroticism and psychopathy predict brain activation during moral and nonmoral emotion regulation. *Cognitive, Affective, & Behavioral Neuroscience, 9*(1), 1-15.

Hariri, A. R., Bookheimer, S. Y., & Mazziotta, J. C. (2000). Modulating emotional responses: effects of a neocortical network on the limbic system. *Neuroreport, 11*(1), 43-48.

Hariri, A. R., Tessitore, A., Mattay, V. S., Fera, F., & Weinberger, D. R. (2002). The amygdala response to emotional stimuli: a comparison of faces and scenes. *Neuroimage, 17*(1), 317-323.

Harris, G. T., Rice, M. E., & Cormier, C. A. (1991). Psychopathy and violent recidivism. *Law and human behavior, 15*(6), 625-637.

Harris, G. T., Rice, M. E., & Quinsey, V. L. (1994). Psychopathy as a taxon: evidence that psychopaths are a discrete class. *Journal of consulting and clinical psychology, 62*(2), 387-397.

Hart, S. D., Cox, D. N., & Hare, R. D. (1995). *The Hare psychopathy checklist: Screening version (PCL: SV)*: MSH-Multi-Health Systems, Incorporated.

Hart, S. D., Kropp, P. R., & Hare, R. D. (1988). Performance of male psychopaths following conditional release from prison. *Journal of consulting and clinical psychology, 56*(2), 227-232.

Harwood, N. K., Hall, L. J., & Shinkfield, A. J. (1999). Recognition of facial emotional expressions from moving and static displays by individuals with mental retardation. *American Journal on Mental Retardation, 104*(3), 270-278.

Hastings, M. E., Tangney, J. P., & Stuewig, J. (2008). Psychopathy and identification of facial expressions of emotion. *Personality and Individual Differences, 44*(7), 1474-1483. doi:10.1016/j.paid.2008.01.004

Hatfield, T., Han, J.-S., Conley, M., Gallagher, M., & Holland, P. (1996). Neurotoxic lesions of basolateral, but not central, amygdala interfere with Pavlovian second-order conditioning and reinforcer devaluation effects. *The Journal of neuroscience, 16*(16), 5256-5265.

Hawes, D. J., Price, M. J., & Dadds, M. R. (2014). Callous-unemotional traits and the treatment of conduct problems in childhood and adolescence: A comprehensive review. *Clinical child and family psychology review, 17*(3), 248-267.

Hayasaka, Y., Purgato, M., Magni, L. R., Ogawa, Y., Takeshima, N., Cipriani, A., . . . Furukawa, T. A. (2015). Dose equivalents of antidepressants: Evidence-based recommendations from randomized controlled trials. *Journal of affective disorders, 180*, 179-184.

Hecht, D. (2011). An inter-hemispheric imbalance in the psychopath's brain. *Personality and Individual Differences, 51*(1), 3-10.

Hedges, L., & Olkin, I. (1985). Statistical models for meta-analysis. *New York: Academic Press. Hedges, LV, & Pigott, TD (2001). The power of statistical tests in meta-analysis. Psychological Methods, 6*, 203-217.

Hemphill, J. F., Hare, R. D., & Wong, S. (1998). Psychopathy and recidivism: A review. *Legal and Criminological Psychology, 3*(1), 139-170.

Henderson, R. R., Bradley, M. M., & Lang, P. J. (2014). Modulation of the initial light reflex during affective picture viewing. *Psychophysiology, 51*(9), 815-818.

Heritage, A. J., & Benning, S. D. (2013). Impulsivity and response modulation deficits in psychopathy: Evidence from the ERN and N1. *Journal of Abnormal Psychology, 122*(1), 215-222.

Herpertz, S. C., Dietrich, T. M., Wenning, B., Krings, T., Erberich, S. G., Willmes, K., . . . Sass, H. (2001). Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biological Psychiatry, 50*(4), 292-298. doi:10.1016/s0006-3223(01)01075-7

Herpertz, S. C., Werth, U., Lukas, G., Qunaibi, M., Schuerkens, A., Kunert, H. J., . . . Sass, H. (2001). Emotion in criminal offenders with psychopathy and borderline personality disorder. *Archives of General Psychiatry, 58*(8), 737-745. doi:10.1001/archpsyc.58.8.737

Hess, E. H., & Polt, J. M. (1960). Pupil size as related to interest value of visual stimuli. *Science, 132*(3423), 349-350. doi:10.1126/science.132.3423.349

Hess, E. H., & Polt, J. M. (1964). Pupil size in relation to mental activity during simple problem-solving *Science, 143*(3611), 1190-1192. doi:10.1126/science.143.3611.1190

Hicks, B. M., Carlson, M. D., Blonigen, D. M., Patrick, C. J., Iacono, W. G., & MGue, M. (2012). Psychopathic personality traits and environmental contexts: Differential correlates, gender differences, and genetic mediation. *Personality Disorders: Theory, research, and treatment, 3*(3), 209-227.

Hicks, B. M., Markon, K. E., Patrick, C. J., Krueger, R. F., & Newman, J. P. (2004). Identifying psychopathy subtypes on the basis of personality structure. *Psychological Assessment, 16*(3), 276-288.

Hicks, B. M., & Patrick, C. J. (2006). Psychopathy and negative emotionality: analyses of suppressor effects reveal distinct relations with emotional distress, fearfulness, and anger-hostility. *Journal of Abnormal Psychology, 115*(2), 276-287.

Hunnius, S., de Wit, T. C., Vrins, S., & von Hofsten, C. (2011). Facing threat: Infants' and adults' visual scanning of faces with neutral, happy, sad, angry, and fearful emotional expressions. *Cognition and Emotion, 25*(2), 193-205.

Hyde, L. W., Byrd, A. L., Votruba-Drzal, E., Hariri, A. R., & Manuck, S. B. (2014). Amygdala reactivity and negative emotionality: Divergent correlates of antisocial personality and psychopathy traits in a community sample. *Journal of Abnormal Psychology, 123*(1), 214-224. doi:10.1037/a0035467

Ishikawa, S. S., Raine, A., Lencz, T., Bihrle, S., & Lacasse, L. (2001). Autonomic stress reactivity and executive functions in successful and unsuccessful criminal psychopaths from the community. *Journal of Abnormal Psychology, 110*(3), 423 - 432.

Iwamoto, Y., Kawanishi, C., Kishida, I., Furuno, T., Fujibayashi, M., Ishii, C., . . . Hirayasu, Y. (2012). Dose-dependent effect of antipsychotic drugs on autonomic nervous system activity in schizophrenia. *BMC psychiatry, 12*(1), 199.

Jin, A. B., Steding, L. H., & Webb, A. K. (2015). Reduced emotional and cardiovascular reactivity to emotionally evocative stimuli in major depressive disorder. *International Journal of Psychophysiology, 97*(1), 66-74.

Jones, A. P., Laurens, K. R., Herba, C. M., Barker, G. J., & Viding, E. (2009). Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. *American Journal of Psychiatry, 166*(1), 95-102.

Jönsson, P., & Sonnby-Borgström, M. (2003). The effects of pictures of emotional faces on tonic and phasic autonomic cardiac control in women and men. *Biol Psychol, 62*(2), 157-173.

Just, M. A., & Carpenter, P. A. (1993). The intensity dimension of thought - pupillometric indexes of sentence processing. *Canadian Journal of Experimental Psychology, 47*(2), 310-339. doi:10.1037/h0078820

Justus, A. N., & Finn, P. R. (2007). Startle modulation in non-incarcerated men and women with psychopathic traits. *Personality and Individual Differences, 43*(8), 2057-2071.

Kafkas, A., & Montaldi, D. (2015). The pupillary response discriminates between subjective and objective familiarity and novelty. *Psychophysiology, 52*(10), 1305-1316.

Kahneman, D., & Beatty, J. (1966). Pupil diameter and load on memory. *Science, 154*(3756), 1583-1585. doi:10.1126/science.154.3756.1583

Karpman, B. (1941). On the need of separating psychopathy into two distinct clinical types: the symptomatic and the idiopathic. *Journal of Criminal Psychopathology, 3*, 112-137.

Kemp, A. H., Quintana, D. S., Gray, M. A., Felmingham, K. L., Brown, K., & Gatt, J. M. (2010). Impact of depression and antidepressant treatment on heart rate variability: a review and meta-analysis. *Biological Psychiatry, 67*(11), 1067-1074.

Keppel, G. (1991). *Design and analysis: A researcher's handbook*: Prentice-Hall, Inc.

Kessler, H., Doyen-Waldecker, C., Hofer, C., Hoffmann, H., Traue, H. C., & Abler, B. (2011). Neural correlates of the perception of dynamic versus static facial expressions of emotion. *Psychosocial Medicine, 8*, 1-8.

Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: evidence for paralimbic system dysfunction. *Psychiatry research, 142*(2), 107-128.

Kiehl, K. A., & Hoffman, M. B. (2011). The criminal psychopath: history, neuroscience, treatment, and economics. *Jurimetrics, 51*, 355-397.

Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., & Liddle, P. F. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Biological Psychiatry, 50*(9), 677-684. doi:10.1016/s0006-3223(01)01222-7

Kilts, C. D., Egan, G., Gideon, D. A., Ely, T. D., & Hoffman, J. M. (2003). Dissociable neural pathways are involved in the recognition of emotion in static and dynamic facial expressions. *Neuroimage, 18*(1), 156-168.

Kimonis, E. R., & Armstrong, K. (2012). Adapting parent–child interaction therapy to treat severe conduct problems with callous-unemotional traits a case study. *Clinical Case Studies, 11*(3), 234-252.

Kimonis, E. R., Fanti, K. A., Isoma, Z., & Donoghue, K. (2013). Maltreatment profiles among incarcerated boys with callous-unemotional traits. *Child maltreatment, 18*(2), 108-121.

Klinge, C., Röder, B., & Büchel, C. (2010). Increased amygdala activation to emotional auditory stimuli in the blind. *Brain, 133*(6), 1729-1736.

Kohn, M., & Clynes, M. (1969). Color dynamics of the pupil. *Annals of the New York Academy of Sciences, 156*(2), 931-950.

Koss, M. C. (1986). Pupillary dilation as an index of central-nervous-system alpha-2-adrenoceptor activation. *Journal of Pharmacological Methods, 15*(1), 1-19. doi:10.1016/0160-5402(86)90002-1

Kosson, D. S., Lorenz, A. R., & Newman, J. P. (2006). Effects of comorbid psychopathy on criminal offending and emotion processing in male offenders with antisocial personality disorder. *Journal of Abnormal Psychology, 115*(4), 798 -806.

Kret, M. E., Roelofs, K., Stekelenburg, J. J., & de Gelder, B. (2013). Emotional signals from faces, bodies and scenes influence observers' face expressions, fixations and pupil-size. *Frontiers in Human Neuroscience, 7*(810), 1-9. doi:10.3389/fnhum.2013.00810

Kuchinke, L., Schneider, D., Kotz, S. A., & Jacobs, A. M. (2011). Spontaneous but not explicit processing of positive sentences impaired in Asperger's syndrome: Pupillometric evidence. *Neuropsychologia, 49*(3), 331-338.

Kuo, J. R., & Linehan, M. M. (2009). Disentangling emotion processes in borderline personality disorder: Physiological and self-reported assessment of biological vulnerability, baseline intensity, and reactivity to emotionally evocative stimuli. *Journal of Abnormal Psychology, 118*(3), 531.

Lakens, D. (2013). Calculating and reporting effect sizes to facilitate cumulative science: A practical primer for t-tests and ANOVAs. *Frontiers in Psychology, 4*(863), 1-12.

Lang, P. J. (1995). The emotion probe: studies of motivation and attention. *American psychologist, 50*(5), 372-335.

Lang, P. J., & Bradley, M. M. (2010). Emotion and the motivational brain. *Biol Psychol, 84*(3), 437-450. doi:10.1016/j.biopsycho.2009.10.007

Lang, P. J., Bradley, M. M., & Cuthbert, B. (2008). *International affective picture system (IAPS): Affective ratings of pictures and instruction manual. Technical Report A-8.* University of Florida, Gainesville, FL.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1997). Motivated attention: Affect, activation, and action. In P. J. Lang, R. F. Simons, & M. Balaban (Eds.), *Attention and orienting: Sensory and motivational processes* (pp. 97-135). Mahwah, NJ: Lawrence Erlbaum Associates.

Lang, P. J., & Davis, M. (2006). Emotion, motivation, and the brain: reflex foundations in animal and human research. *Progress in brain research, 156*, 3-29.

Lang, P. J., Greenwald, M. K., Bradley, M. M., & Hamm, A. O. (1993). Looking at pictures: Affective, facial, visceral, and behavioral reactions. *Psychophysiology, 30*, 261-261.

Langner, O., Dotsch, R., Bijlstra, G., Wigboldus, D. H. J., Hawk, S. T., & van Knippenberg, A. (2010). Presentation and validation of the Radboud Faces Database. *Cognition & Emotion, 24*(8), 1377-1388. doi:10.1080/02699930903485076

Lanteaume, L., Khalfa, S., Régis, J., Marquis, P., Chauvel, P., & Bartolomei, F. (2007). Emotion induction after direct intracerebral stimulations of human amygdala. *Cerebral Cortex, 17*(6), 1307-1313.

Larsson, H., Andershed, H., & Lichtenstein, P. (2006). A genetic factor explains most of the variation in the psychopathic personality. *Journal of Abnormal Psychology, 115*(2), 221.

Lee, B.-T., Seok, J.-H., Lee, B.-C., Cho, S. W., Yoon, B.-J., Lee, K.-U., . . . Ham, B.-J. (2008). Neural correlates of affective processing in response to sad and angry facial stimuli in patients

with major depressive disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 32*(3), 778-785.

Leistico, A.-M. R., Salekin, R. T., DeCoster, J., & Rogers, R. (2008). A large-scale meta-analysis relating the hare measures of psychopathy to antisocial conduct. *Law and human behavior, 32*(1), 28-45.

Leknes, S., Wessberg, J., Ellingsen, D.-M., Chelnokova, O., Olausson, H., & Laeng, B. (2012). Oxytocin enhances pupil dilation and sensitivity to 'hidden' emotional expressions. *Social Cognitive and Affective Neuroscience, 8*(7), 741-749nss062.

Lemaire, M., Aguillon-Hernandez, N., Bonnet-Brilhault, F., Martineau, J., & El-Hage, W. (2014). Subjective and physiological emotional response in euthymic bipolar patients: A pilot study. *Psychiatry research, 220*(1), 294-301.

Leppänen, J. M., Kauppinen, P., Peltola, M. J., & Hietanen, J. K. (2007). Differential electrocortical responses to increasing intensities of fearful and happy emotional expressions. *Brain research, 1166*, 103-109.

Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing psychopathic attributes in a noninstitutionalized population. *Journal of Personality and Social Psychology, 68*(1), 151-158.

Levenston, G. K., Patrick, C. J., Bradley, M. M., & Lang, P. J. (2000). The psychopath as observer: Emotion and attention in picture processing. *Journal of Abnormal Psychology, 109*(3), 373-385. doi:10.1037//0021-843x.109.3.373

Levine, T. R., & Hullett, C. R. (2002). Eta squared, partial eta squared, and misreporting of effect size in communication research. *Human Communication Research, 28*(4), 612-625.

Li, H., Chan, R. C., McAlonan, G. M., & Gong, Q.-y. (2009). Facial emotion processing in schizophrenia: a meta-analysis of functional neuroimaging data. *Schizophrenia bulletin, 36*(5), 1029-1039.

Liao, H.-I., Kidani, S., Yoneya, M., Kashino, M., & Furukawa, S. (2016). Correspondences among pupillary dilation response, subjective salience of sounds, and loudness. *Psychonomic bulletin & review, 23*(2), 412-425.

Libby, W. L., Lacey, B. C., & Lacey, J. I. (1973). Pupillary and cardiac activity during visual attention. *Psychophysiology, 10*(3), 270-294. doi:10.1111/j.1469-8986.1973.tb00526.x

Lilienfeld, S., & Widows, M. (2005). PPI-R professional manual. *Lutz, FL: Psychological Assessment Resources*.

Lilienfeld, S. O. (1994). Conceptual problems in the assessment of psychopathy. *Clinical Psychology Review, 14*(1), 17-38.

Lilienfeld, S. O. (1998). Methodological advances and developments in the assessment of psychopathy. *Behaviour Research and Therapy, 36*(1), 99-125.

Lilienfeld, S. O., & Andrews, B. P. (1996). Development and preliminary validation of a self-report measure of psychopathic personality traits in noncriminal population. *Journal of Personality Assessment, 66*(3), 488-524.

Lilienfeld, S. O., & Fowler, K. A. (2006). The self-report assessment of psychopathy. In C. J. Patrick (Ed.), *Handbook of psychopathy* (pp. 107-132). London: The Guildford Press.

Lilienfeld, S. O., Fowler, K. A., & Patrick, C. (2006). The self-report assessment of psychopathy. *Handbook of psychopathy*, 107-132.

Lilienfeld, S. O., Smith, S. F., Sauvigné, K. C., Patrick, C. J., Drislane, L. E., Latzman, R. D., & Krueger, R. F. (2015). Is Boldness Relevant to Psychopathic Personality? Meta-Analytic Relations With Non-Psychopathy Checklist-Based Measures of Psychopathy.

Lilienfeld, S. O., Watts, A. L., Francis Smith, S., Berg, J. M., & Latzman, R. D. (2015). Psychopathy deconstructed and reconstructed: Identifying and assembling the Personality building blocks of Cleckley's chimera. *Journal of Personality, 83*(6), 593-610.

Loane, C., & Politis, M. (2012). Buspirone: What is it all about? *Brain research, 1461*, 111-118.

Loewenfeld, I. (1993). *The pupil: anatomy, physiology and clinical applications*. Detroit: Wayne State University Press.

López, R., Poy, R., Patrick, C. J., & Moltó, J. (2013). Deficient fear conditioning and self-reported psychopathy: The role of fearless dominance. *Psychophysiology, 50*(2), 210-218.

Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta-analysis. *Psychological Bulletin, 130*(4), 531-552. doi:10.1037/0033-2909.130.4.531

Lorenz, A. R., & Newman, J. P. (2002). Deficient response modulation and emotion processing in low-anxious Caucasian psychopathic offenders: results from a lexical decision task. *Emotion, 2*(2), 91-104.

Lowenstein, O., & Loewenfeld, I. (1962). The pupil. In H. Davson (Ed.), *The eye: Muscular mechanisms* (Vol. 3). New York: Academic Press.

Lowenstein, O., & Loewenfeld, I. E. (1950). Mutual role of sympathetic and parasympathetic in shaping of the pupillary reflex to light: pupillographic studies. *Archives of Neurology & Psychiatry, 64*(3), 341-377.

Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *Journal of Abnormal and Social Psychology, 55*(1), 6-10. doi:10.1037/h0047232

Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Lykken, D. T. (1996). Psychopathy, sociopathy, and crime. *Society, 34*(1), 29-38.

Lyons, M. (2015). Risk anything! Secondary, rather than primary psychopathy, is associated with diverse risk-taking in evolutionarily relevant domains. *Evolutionary Behavioral Sciences, 9*(3), 197-203.

Malterer, M. B., Lilienfeld, S. O., Neumann, C. S., & Newman, J. P. (2010). Concurrent validity of the Psychopathic Personality Inventory with offender and community samples. *Assessment, 17*(1), 3 - 15.

Maples, J. L., Miller, J. D., Fortune, E., MacKillop, J., Campbell, W. K., Lynam, D. R., . . . Goodie, A. S. (2014). An examination of the correlates of fearless dominance and self-centered impulsivity among high-frequency gamblers. *Journal of Personality Disorders, 28*(3), 379-393.

Marcus, D. K., Fulton, J. J., & Edens, J. F. (2013). The two-factor model of psychopathic personality: Evidence from the Psychopathic Personality Inventory. *Personality Disorders: Theory, research, and treatment, 4*(1), 67-76.

Marcus, D. K., John, S. L., & Edens, J. F. (2004). A taxometric analysis of psychopathic personality. *Journal of Abnormal Psychology, 113*(4), 626-635.

Marcus, D. K., Lilienfeld, S. O., Edens, J. F., & Poythress, N. G. (2006). Is antisocial personality disorder continuous or categorical? A taxometric analysis. *Psychological Medicine, 36*(11), 1571-1581.

Marion, B. E., Sellbom, M., Salekin, R. T., Toomey, J. A., Kucharski, L. T., & Duncan, S. (2013). An examination of the association between psychopathy and dissimulation using the MMPI-2-RF validity scales. *Law and human behavior, 37*(4), 219-230.

Markowitsch, H. J. (1999). Differential contribution of right and left amygdala to affective information processing. *Behavioural neurology, 11*(4), 233-244.

Marsh, A. A., & Cardinale, E. M. (2014). When psychopathy impairs moral judgments: neural responses during judgments about causing fear. *Social Cognitive and Affective Neuroscience, 9*(1), 3-11.

Marsh, A. A., Finger, E. C., Fowler, K. A., Adalio, C. J., Jurkowitz, I. T., Schechter, J. C., . . . Blair, R. (2013). Empathic responsiveness in amygdala and anterior cingulate cortex in youths with psychopathic traits. *Journal of Child Psychology and Psychiatry, 54*(8), 900-910.

Marsh, A. A., Finger, E. C., Mitchell, D. G., Reid, M. E., Sims, C., Kosson, D. S., . . . Blair, R. (2008). Reduced amygdala response to fearful expressions in children and adolescents with

callous-unemotional traits and disruptive behavior disorders. *American Journal of Psychiatry, 165*(6), 712-720.

McCord, W., & McCord, J. (1964). *The psychopath: An essay on the criminal mind*. New York: Van Nostrand Reinhold.

McDannald, M. A., & Galarce, E. M. (2011). Measuring Pavlovian fear with conditioned freezing and conditioned suppression reveals different roles for the basolateral amygdala. *Brain research, 1374*, 82-89.

Mealey, L. (1995). Primary sociopathy (psychopathy) is a type, secondary is not. *Behavioral and Brain Sciences, 18*(03), 579-599.

Medina, A. L., Kirilko, E., & Grose-Fifer, J. (2016). Emotional processing and psychopathic traits in male college students: an event-related potential study. *International Journal of Psychophysiology, 106*, 39-49.

Meffert, H., Gazzola, V., den Boer, J. A., Bartels, A. A., & Keysers, C. (2013). Reduced spontaneous but relatively normal deliberate vicarious representations in psychopathy. *Brain, 136*(8), 2550-2562.

Meinert, C. L. (2012). *Clinical trials: Design, conduct and analysis*. New York: Oxford University Press.

Michelson, A. A. (1927). *Studies in optics*. Chicaog, IL: University of Chicago Press.

Mier, D., Haddad, L., Diers, K., Dressing, H., Meyer-Lindenberg, A., & Kirsch, P. (2014). Reduced embodied simulation in psychopathy. *The World Journal of Biological Psychiatry, 15*(6), 479-487.

Miles, F., & Kawano, K. (1987). Visual stabilization of the eyes. *Trends in Neurosciences, 10*, 153-158.

Miller, J. D., Gaughan, E. T., & Pryor, L. R. (2008). The Levenson Self-Report Psychopathy Scale an examination of the personality traits and disorders associated with the LSRP factors. *Assessment, 15*(4), 450-463.

Miller, J. D., & Lynam, D. R. (2012). An examination of the Psychopathic Personality Inventory's nomological network: a meta-analytic review. *Personality Disorders: Theory, research, and treatment, 3*(3), 305-326.

Mitchell, A. E., Dickens, G. L., & Picchioni, M. M. (2014). Facial emotion processing in borderline personality disorder: A systematic review and meta-analysis. *Neuropsychology Review, 24*(2), 166-184. doi:10.1007/s11065-014-9254-9

Mitchell, D. G., Richell, R. A., Leonard, A., & Blair, R. J. R. (2006). Emotion at the expense of cognition: psychopathic individuals outperform controls on an operant response task. *Journal of Abnormal Psychology, 115*(3), 559 -566.

Mokros, A., Neumann, C. S., Stadtland, C., Osterheider, M., Nedopil, N., & Hare, R. D. (2011). Assessing measurement invariance of PCL-R assessments from file reviews of North American and German offenders. *International journal of law and psychiatry, 34*(1), 56-63.

Montagne, B., van Honk, J., Kessels, R. P., Frigerio, E., Burt, M., van Zandvoort, M. J., . . . de Haan, E. H. (2005). Reduced efficiency in recognising fear in subjects scoring high on psychopathic personality characteristics. *Personality and Individual Differences, 38*(1), 5-11.

Morris, J., Frith, C., Perrett, D., Rowland, D., Young, A., Calder, A., & Dolan, R. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature, 383*, 31.

Morris, J. S., Öhman, A., & Dolan, R. J. (1999). A subcortical pathway to the right amygdala mediating “unseen” fear. *Proceedings of the National Academy of Sciences, 96*(4), 1680-1685.

Morrison, S. E., & Salzman, C. D. (2010). Re-valuing the amygdala. *Current opinion in neurobiology, 20*(2), 221-230.

Most, S. B., Smith, S. D., Cooter, A. B., Levy, B. N., & Zald, D. H. (2007). The naked truth: Positive, arousing distractors impair rapid target perception. *Cognition and Emotion, 21*(5), 964-981.

Moul, C., Killcross, S., & Dadds, M. R. (2012). A Model of Differential Amygdala Activation in Psychopathy. *Psychological Review, 119*(4), 789-806. doi:10.1037/a0029342

Moulden, B., Kingdom, F., & Gatley, L. F. (1990). The standard deviation of luminance as a metric for contrast in random-dot images. *Perception, 19*(1), 79-101.

Murrie, D. C., Marcus, D. K., Douglas, K. S., Lee, Z., Salekin, R. T., & Vincent, G. (2007). Youth with psychopathy features are not a discrete class: A taxometric analysis. *Journal of Child Psychology and Psychiatry, 48*(7), 714-723.

Naber, M., Frässle, S., Rutishauser, U., & Einhäuser, W. (2013). Pupil size signals novelty and predicts later retrieval success for declarative memories of natural scenes. *Journal of Vision, 13*(2), 1-20.

Neumann, C. S., Hare, R. D., & Newman, J. P. (2007). The super-ordinate nature of the Psychopathy Checklist-Revised. *Journal of Personality Disorders, 21*(2), 102-117.

Neumann, C. S., Johansson, P. T., & Hare, R. D. (2013). The Psychopathy Checklist-Revised (PCL-R), low anxiety, and fearlessness: A structural equation modeling analysis. *Personality Disorders: Theory, research, and treatment, 4*(2), 129-137.

Neumann, C. S., & Pardini, D. (2014). Factor structure and construct validity of the Self-Report Psychopathy (SRP) Scale and the Youth Psychopathic Traits Inventory (YPI) in young men. *Journal of Personality Disorders, 28*(3), 419-433.

Neumann, C. S., Uzieblo, K., Crombez, G., & Hare, R. D. (2013). Understanding the Psychopathic Personality Inventory (PPI) in terms of the unidimensionality, orthogonality, and construct validity of PPI-I and-II. *Personality Disorders, 4*(1), 77-79.

Newman, J. P., Curtin, J. J., Bertsch, J. D., & Baskin-Sommers, A. R. (2010). Attention moderates the fearlessness of psychopathic offenders. *Biological Psychiatry, 67*(1), 66-70. doi:10.1016/j.biopsych.2009.07.035

Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology, 95*(3), 252-256.

Nuske, H. J., Vivanti, G., Hudry, K., & Dissanayake, C. (2014). Pupillometry reveals reduced unconscious emotional reactivity in autism. *Biol Psychol, 101*, 24-35.

O'Farrell, K. R., Gray, N. S., & Snowden, R. J. (In preparation). Abnormalities of pupil response in relation to two measures of psychopathy.

Ogloff, J. R. P., & Wong, S. (1990). Electrodermal and cardiovascular evidence of a coping response in psychopaths. *Criminal Justice and Behavior, 17*(2), 231-245.

Öhman, A., Lundqvist, D., & Esteves, F. (2001). The face in the crowd revisited: A threat advantage with schematic stimuli. *Journal of Personality and Social Psychology, 80*(3), 381-396.

Olejnik, S., & Algina, J. (2003). Generalized eta and omega squared statistics: measures of effect size for some common research designs. *Psychological methods, 8*(4), 434-447.

Öztuna, D., Elhan, A. H., & Tüccar, E. (2006). Investigation of four different normality tests in terms of type 1 error rate and power under different distributions. *Turkish Journal of Medical Sciences, 36*(3), 171-176.

Partala, T., & Surakka, V. (2003). Pupil size variation as an indication of affective processing. *International Journal of Human-Computer Studies, 59*(1-2), 185-198. doi:10.1016/s1071-5819(03)00017-x

Pastor, M. C., Molto, J., Vila, J., & Lang, P. J. (2003). Startle reflex modulation, affective ratings and autonomic reactivity in incarcerated Spanish psychopaths. *Psychophysiology, 40*(6), 934-938. doi:10.1111/1469-8986.00111

Patrick, C. J. (1994). EMOTION AND PSYCHOPATHY - STARTLING NEW INSIGHTS. *Psychophysiology, 31*(4), 319-330. doi:10.1111/j.1469-8986.1994.tb02440.x

Patrick, C. J. (2007). Getting to the heart of psychopathy. In H. Hugues & J. C. Yuille (Eds.), *The psychopath: Theory, research, and practice* (pp. 207-252). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Patrick, C. J. (2010a). Conceptualizing the psychopathic personality: Disinhibited, bold,… Or just plain mean? In R. T. Salekin & D. R. Lynam (Eds.), *Handbook of child and adolescent psychopathy* (pp. 15-48). New York: Guilford Press.

Patrick, C. J. (2010b). *Operationalizing the triarchic conceptualisation of psychopathy: Preliminary description of brief scales for assessment of boldness, meanness and disinhibition*. Retrieved from https://www.phenxtoolkit.org/toolkit_content/supplemental_info/psychiatric/measures/Triarchic_Psychopathy_Measure_Manual.pdf

Patrick, C. J., & Bernat, E. M. (2009). Neurobiology of psychopathy: A two-process theory. In G. G. Bernston & J. T. Cacioppo (Eds.), *Handbook of neuroscience for the behavioural sciences* (pp. 1110 - 1131). New York, NY: Wiley.

Patrick, C. J., Bradley, M. M., & Lang, P. J. (1993). Emotion in the criminal psychopath - startle reflex modulation. *Journal of Abnormal Psychology, 102*(1), 82-92. doi:10.1037//0021-843x.102.1.82

Patrick, C. J., & Brislin, S. J. (2015). Antisocial Personality Disorder/Psychopathy. *The Encyclopedia of Clinical Psychology*, 1-10.

Patrick, C. J., Cuthbert, B. N., & Lang, P. J. (1994). Emotion in the criminal psychopath: Fear image processing. *Journal of Abnormal Psychology, 103*(3), 523-534.

Patrick, C. J., & Drislane, L. E. (2015). Triarchic model of psychopathy: Origins, operationalizations, and observed linkages with personality and general psychopathology. *Journal of Personality, 83*(6), 627-643.

Patrick, C. J., Edens, J. F., Poythress, N. G., Lilienfeld, S. O., & Benning, S. D. (2006). Construct validity of the psychopathic personality inventory two-factor model with offenders. *Psychological Assessment, 18*(2), 204-208.

Patrick, C. J., Fowles, D. C., & Krueger, R. F. (2009). Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. *Development and Psychopathology, 21*(3), 913-938. doi:10.1017/s0954579409000492

Paulhus, D., Neumann, C., & Hare, R. (In Press). Manual for the Self-Report Psychopathy scale 4th edition. *Toronto: Multi-Health Systems*.

Peelen, M. V., Atkinson, A. P., & Vuilleumier, P. (2010). Supramodal representations of perceived emotions in the human brain. *The Journal of neuroscience, 30*(30), 10127-10134.

Pessoa, L., Japee, S., Sturman, D., & Ungerleider, L. G. (2006). Target visibility and visual awareness modulate amygdala responses to fearful faces. *Cerebral Cortex, 16*(3), 366-375. doi:10.1093/cercorbhi115

Pham, T. H., & Philippot, P. (2010). Decoding of facial expression of emotion in criminal psychopaths. *Journal of Personality Disorders, 24*(4), 445-459.

Polaschek, D. L., & Daly, T. E. (2013). Treatment and psychopathy in forensic settings. *Aggression and Violent Behavior, 18*(5), 592-603.

Porter, S. (1996). Without conscience or without active conscience? The etiology of psychopathy revisited. *Aggression and Violent Behavior, 1*(2), 179-189.

Porter, S., Brinke, L., & Wilson, K. (2009). Crime profiles and conditional release performance of psychopathic and non-psychopathic sexual offenders. *Legal and Criminological Psychology, 14*(1), 109-118.

Posamentier, M. T., & Abdi, H. (2003). Processing faces and facial expressions. *Neuropsychology Review, 13*(3), 113-143.

Poy, R., Segarra, P., Esteller, A., Lopez, R., & Molto, J. (2014). FFM description of the triarchic conceptualization of psychopathy in men and women. *Psychological Assessment, 26*(1), 69-76. doi:10.1037/a0034642

Poythress, N. G., Edens, J. F., Skeem, J. L., Lilienfeld, S. O., Douglas, K. S., Frick, P. J., . . . Wang, T. (2010). Identifying subtypes among offenders with antisocial personality disorder: a cluster-analytic study. *Journal of Abnormal Psychology, 119*(2), 389-400.

Poythress, N. G., & Hall, J. R. (2011). Psychopathy and impulsivity reconsidered. *Aggression and Violent Behavior, 16*(2), 120-134.

Poythress, N. G., Lilienfeld, S. O., Skeem, J. L., Douglas, K. S., Edens, J. F., Epstein, M., & Patrick, C. J. (2010). Using the PCL-R to help estimate the validity of two self-report measures of psychopathy with offenders. *Assessment, 17*(2), 206-219.

Poythress, N. G., Skeem, J. L., & Lilienfeld, S. O. (2006). Associations among early abuse, dissociation, and psychopathy in an offender sample. *Journal of Abnormal Psychology, 115*(2), 288-297.

Prado, C. E., Treeby, M. S., & Crowe, S. F. (2015). Examining relationships between facial emotion recognition, self-control, and psychopathic traits in a non-clinical sample. *Personality and Individual Differences, 80*, 22-27.

Prehn, K., Kazzer, P., Lischke, A., Heinrichs, M., Herpertz, S. C., & Domes, G. (2013). Effects of intranasal oxytocin on pupil dilation indicate increased salience of socioaffective stimuli. *Psychophysiology, 50*(6), 528-537.

Prehn, K., Schlagenhauf, F., Schulze, L., Berger, C., Vohs, K., Fleischer, M., . . . Herpertz, S. C. (2013). Neural correlates of risk taking in violent criminal offenders characterized by emotional hypo-and hyper-reactivity. *Social Neuroscience, 8*(2), 136-147.

Propper, C., Willoughby, M., Halpern, C., Carbone, M., & Cox, M. (2007). Parenting quality, DRD4, and the prediction of externalizing and internalizing behaviors in early childhood. *Developmental Psychobiology, 49*(6), 619-632.

Pujol, J., Batalla, I., Contreras-Rodríguez, O., Harrison, B. J., Pera, V., Hernández-Ribas, R., . . . Deus, J. (2011). Breakdown in the brain network subserving moral judgment in criminal psychopathy. *Social Cognitive and Affective Neuroscience, 7*(8), 917 -923.

Ragsdale, K. A., Mitchell, J. C., Cassisi, J. E., & Bedwell, J. S. (2013). Comorbidity of schizotypy and psychopathy: Skin conductance to affective pictures. *Psychiatry research, 210*(3), 1000-1007.

Rajkowski, J., Kubiak, P., & Aston-Jones, G. (1993). *Correlations between locus coeruleus (LC) neural activity, pupil diameter and behavior in monkey support a role of LC in attention.* Paper presented at the Society for neuroscience abstracts.

Ray, J. V., Hall, J., Rivera-Hudson, N., Poythress, N. G., Lilienfeld, S. O., & Morano, M. (2013). The relation between self-reported psychopathic traits and distorted response styles: A meta-analytic review. *Personality Disorders: Theory, research, and treatment, 4*(1), 1-14.

Recio, G., Sommer, W., & Schacht, A. (2011). Electrophysiological correlates of perceiving and evaluating static and dynamic facial emotional expressions. *Brain research, 1376*, 66-75.

Reidy, D. E., Zeichner, A., Miller, J. D., & Martinez, M. A. (2007). Psychopathy and aggression: Examining the role of psychopathy factors in predicting laboratory aggression under hostile and instrumental conditions. *Journal of Research in Personality, 41*(6), 1244-1251.

Reif, A., Rösler, M., Freitag, C. M., Schneider, M., Eujen, A., Kissling, C., . . . Thome, J. (2007). Nature and nurture predispose to violent behavior: serotonergic genes and adverse childhood environment. *Neuropsychopharmacology, 32*(11), 2375-2383.

Reisenzein, R., Bördgen, S., Holtbernd, T., & Matz, D. (2006). Evidence for strong dissociation between emotion and facial displays: The case of surprise. *Journal of Personality and Social Psychology, 91*(2), 295.

Rice, M. E., & Harris, G. T. (1997). Cross-validation and extension of the Violence Risk Appraisal Guide for child molesters and rapists. *Law and human behavior, 21*(2), 231-241.

Rieger, G., & Savin-Williams, R. C. (2012). The eyes have it: Sex and sexual orientation differences in pupil dilation patterns. *PLoS ONE, 7*(8), 1-10.

Rilling, J. K., Glenn, A. L., Jairam, M. R., Pagnoni, G., Goldsmith, D. R., Elfenbein, H. A., & Lilienfeld, S. O. (2007). Neural correlates of social cooperation and non-cooperation as a function of psychopathy. *Biological Psychiatry, 61*(11), 1260-1271.

Ritz, T., George, C., & Dahme, B. (2000). Respiratory resistance during emotional stimulation: evidence for a nonspecific effect of experienced arousal? *Biol Psychol, 52*(2), 143-160.

Robins, L. N. (1978). Sturdy childhood predictors of adult antisocial behaviour: Replications from longitudinal studies. *Psychological Medicine, 8*(04), 611-622.

Robins, L. N. (1996). Deviant children grown up. *European child & adolescent psychiatry, 5*, 44-46.

Roesch, M. R., Calu, D. J., Esber, G. R., & Schoenbaum, G. (2010a). All that glitters… dissociating attention and outcome expectancy from prediction errors signals. *Journal of Neurophysiology, 104*(2), 587-595.

Roesch, M. R., Calu, D. J., Esber, G. R., & Schoenbaum, G. (2010b). Neural correlates of variations in event processing during learning in basolateral amygdala. *The Journal of neuroscience, 30*(7), 2464-2471.

Rosa, P. J., Esteves, F., & Arriaga, P. (2015). Beyond traditional clinical measurements for screening fears and phobias. *IEEE Transactions on Instrumentation and Measurement, 64*(12), 3396-3404.

Ross, S. R., Benning, S. D., Patrick, C. J., Thompson, A., & Thurston, A. (2008). Factors of the Psychopathic Personality Inventory: Criterion-related validity and relationship to the BIS/BAS and five-factor models of personality. *Assessment, 16*(1), 71-87.

Rubin, D. B. (2004). *Multiple imputation for nonresponse in surveys*. Hoboken, NJ: John Wiley & Sons.

Rymarczyk, K., Biele, C., Grabowska, A., & Majczynski, H. (2011). EMG activity in response to static and dynamic facial expressions. *International Journal of Psychophysiology, 79*(2), 330-333.

Sadeh, N., & Verona, E. (2012). Visual complexity attenuates emotional processing in psychopathy: Implications for fear-potentiated startle deficits. *Cognitive, Affective, & Behavioral Neuroscience, 12*(2), 346-360.

Salekin, R. T., Chen, D. R., Sellbom, M., Lester, W. S., & MacDougall, E. (2014). Examining the factor structure and convergent and discriminant validity of the Levenson Self-Report Psychopathy Scale: Is the two-factor model the best fitting model? *Personality Disorders: Theory, research, and treatment, 5*(3), 289-304.

Salekin, R. T., Rogers, R., & Sewell, K. W. (1996). A review and meta-analysis of the Psychopathy Checklist and Psychopathy Checklist-Revised: Predictive validity of dangerousness. *Clinical Psychology: Science and Practice, 3*(3), 203-215.

Sarlo, M., Palomba, D., Buodo, G., Minghetti, R., & Stegagno, L. (2005). Blood pressure changes highlight gender differences in emotional reactivity to arousing pictures. *Biol Psychol, 70*(3), 188-196.

Sato, W., Fujimura, T., & Suzuki, N. (2008). Enhanced facial EMG activity in response to dynamic facial expressions. *International Journal of Psychophysiology, 70*(1), 70-74.

Sato, W., Kochiyama, T., Yoshikawa, S., Naito, E., & Matsumura, M. (2004). Enhanced neural activity in response to dynamic facial expressions of emotion: an fMRI study. *Cognitive Brain Research, 20*(1), 81-91.

Savitzky, A., & Golay, M. J. (1964). Smoothing and differentiation of data by simplified least squares procedures. *Analytical chemistry, 36*(8), 1627-1639.

Schultz, J., & Pilz, K. S. (2009). Natural facial motion enhances cortical responses to faces. *Experimental Brain Research, 194*(3), 465-475.

Schulz, N., Murphy, B., & Verona, E. (2016). Gender differences in psychopathy links to drug use. *Law and human behavior, 40*(2), 159-168.

Schupp, H., Cuthbert, B., Bradley, M., Hillman, C., Hamm, A., & Lang, P. J. (2004). Brain processes in emotional perception: Motivated attention. *Cognition and Emotion, 18*(5), 593-611.

Scott, S. K., Young, A. W., Calder, A. J., Hellawell, D. J., Aggleton, J. P., & Johnsons, M. (1997). Impaired auditory recognition of fear and anger following bilateral amygdala lesions. *Nature, 385*(6613), 254-257.

Seara-Cardoso, A., Dolberg, H., Neumann, C., Roiser, J. P., & Viding, E. (2013). Empathy, morality and psychopathic traits in women. *Personality and Individual Differences, 55*(3), 328-333.

Seara-Cardoso, A., Neumann, C., Roiser, J., McCrory, E., & Viding, E. (2012). Investigating associations between empathy, morality and psychopathic personality traits in the general population. *Personality and Individual Differences, 52*(1), 67-71.

Sellbom, M., & Phillips, T. R. (2013). An examination of the triarchic conceptualization of psychopathy in incarcerated and nonincarcerated samples. *Journal of Abnormal Psychology, 122*(1), 208-214. doi:10.1037/a0029306

Sepeta, L., Tsuchiya, N., Davies, M. S., Sigman, M., Bookheimer, S. Y., & Dapretto, M. (2012). Abnormal social reward processing in autism as indexed by pupillary responses to happy faces. *Journal of Neurodevelopmental Disorders, 4*(1), 17. doi:10.1186/1866-1955-4-17

Sergerie, K., Chochol, C., & Armony, J. L. (2008). The role of the amygdala in emotional processing: a quantitative meta-analysis of functional neuroimaging studies. *Neuroscience & Biobehavioral Reviews, 32*(4), 811-830.

Sheese, B. E., Voelker, P. M., Rothbart, M. K., & Posner, M. I. (2007). Parenting quality interacts with genetic variation in dopamine receptor D4 to influence temperament in early childhood. *Development and Psychopathology, 19*(04), 1039-1046.

Siegle, G. J., Granholm, E., Ingram, R. E., & Matt, G. E. (2001). Pupillary and reaction time measures of sustained processing of negative information in depression. *Biological Psychiatry, 49*(7), 624-636.

Siegle, G. J., Steinhauer, S. R., Carter, C. S., Ramel, W., & Thase, M. E. (2003). Do the seconds turn into hours? Relationships between sustained pupil dilation in response to emotional information and self-reported rumination. *Cognitive Therapy and Research, 27*(3), 365-382.

Siegle, G. J., Steinhauer, S. R., Friedman, E. S., Thompson, W. S., & Thase, M. E. (2011). Remission prognosis for cognitive therapy for recurrent depression using the pupil: utility and neural correlates. *Biological Psychiatry, 69*(8), 726-733.

Siegle, G. J., Thompson, W., Carter, C. S., Steinhauer, S. R., & Thase, M. E. (2007). Increased amygdala and decreased dorsolateral prefrontal BOLD responses in unipolar depression: Related and independent features. *Biological Psychiatry, 61*(2), 198-209. doi:http://dx.doi.org/10.1016/j.biopsych.2006.05.048

Siepmann, M., Grossmann, J., Mück-Weymann, M., & Kirch, W. (2003). Effects of sertraline on autonomic and cognitive functions in healthy volunteers. *Psychopharmacology, 168*(3), 293-298.

Siepmann, M., Heine, B., Kluge, A., Ziemssen, T., Mück-Weymann, M., & Kirch, W. (2007). The effects of lorazepam on skin conductance responses to aversive stimuli in healthy subjects. *Clinical Autonomic Research, 17*(3), 160.

Simons, R. F., Detenber, B. H., Roedema, T. M., & Reiss, J. E. (1999). Emotion processing in three systems: The medium and the message. *Psychophysiology, 36*(05), 619-627.

Skeem, J., Johansson, P., Andershed, H., Kerr, M., & Louden, J. E. (2007). Two subtypes of psychopathic violent offenders that parallel primary and secondary variants. *Journal of Abnormal Psychology, 116*(2), 395-409.

Skeem, J. L., & Cooke, D. J. (2010). Is criminal behavior a central component of psychopathy? Conceptual directions for resolving the debate. *Psychological Assessment, 22*(2), 433-445.

Skeem, J. L., Polaschek, D. L. L., Patrick, C. J., & Lilienfeld, S. O. (2011). Psychopathic personality: Bridging the gap between scientific evidence and public policy. *Psychological Science in the Public Interest, 12*(3), 95-162. doi:10.1177/1529100611426706

Skeem, J. L., Poythress, N., Edens, J. F., Lilienfeld, S. O., & Cale, E. M. (2003). Psychopathic personality or personalities? Exploring potential variants of psychopathy and their implications for risk assessment. *Aggression and Violent Behavior, 8*(5), 513-546.

Skilling, T. A., Harris, G. T., Rice, M. E., & Quinsey, V. L. (2002). Identifying persistently antisocial offenders using the Hare Psychopathy Checklist and DSM antisocial personality disorder criteria. *Psychological Assessment, 14*(1), 27-38.

Smith, S. A., & Smith, S. (1999). Bilateral Horner's syndrome: detection and occurrence. *Journal of Neurology, Neurosurgery & Psychiatry, 66*(1), 48-51.

Smith, S. S., & Newman, J. P. (1990). Alcohol and drug abuse-dependence disorders in psychopathic and nonpsychopathic criminal offenders. *Journal of Abnormal Psychology, 99*(4), 430-439.

Snowden, R. J., Craig, R., & Gray, N. S. (2013). Detection and recognition of emotional expressions: Effects of traits of personality disorder and gender. *Personality and Individual Differences, 54*(2), 158-163. doi:10.1016/j.paid.2012.08.007

Snowden, R. J., O'Farrell, K. R., Burley, D., Erichsen, J. T., Newton, N. V., & Gray, N. S. (2016). The pupil's response to affective pictures: Role of image duration, habituation, and viewing mode. *Psychophysiology, 53*(8), 1217-1223. doi:10.1111/psyp.12668

Somerville, L. H., Kim, H., Johnstone, T., Alexander, A. L., & Whalen, P. J. (2004). Human amygdala responses during presentation of happy and neutral faces: correlations with state anxiety. *Biological Psychiatry, 55*(9), 897-903.

Stanković, M., Nešić, M., Obrenović, J., Stojanović, D., & Milošević, V. (2015). Recognition of facial expressions of emotions in criminal and non-criminal psychopaths: Valence-specific hypothesis. *Personality and Individual Differences, 82*, 242-247.

Stanley, J. H., Wygant, D. B., & Sellbom, M. (2013). Elaborating on the construct validity of the triarchic psychopathy measure in a criminal offender sample. *Journal of Personality Assessment, 95*(4), 343-350.

Steidtmann, D., Ingram, R. E., & Siegle, G. J. (2010). Pupil response to negative emotional information in individuals at risk for depression. *Cognition & Emotion, 24*(3), 480-496. doi:10.1080/02699930902738897

Steinhauer, S. R., Boller, F., Zubin, J., & Pearlman, S. (1983). Pupillary dilation to emotional visual-stimuli revisited. *Psychophysiology, 20*(4), 472-472.

Steinhauer, S. R., Condray, R., & Pless, M. L. (2015). Pharmacological isolation of cognitive components influencing the pupillary light reflex. *Journal of Ophthalmology, 2015*, 179542.

Steinhauer, S. R., Siegle, G. J., Condray, R., & Pless, M. (2004). Sympathetic and parasympathetic innervation of pupillary dilation during sustained processing. *Int J Psychophysiol, 52*(1), 77-86. doi:10.1016/j.ijpsycho.2003.12.005

Stevens, D., Charman, T., & Blair, R. (2001). Recognition of emotion in facial expressions and vocal tones in children with psychopathic tendencies. *The Journal of genetic psychology, 162*(2), 201-211.

Stevenson, R. A., & James, T. W. (2008). Affective auditory stimuli: Characterization of the International Affective Digitized Sounds (IADS) by discrete emotional categories. *Behavior Research Methods, 40*(1), 315-321. doi:10.3758/brm.40.1.315

Strickland, C. M., Drislane, L. E., Lucy, M., Krueger, R. F., & Patrick, C. J. (2013). Characterizing psychopathy using DSM-5 personality traits. *Assessment, 20*(3), 327-338. doi:10.1177/1073191113486691

Sumner, P. (2011). Determinants of saccade latency. In S. Liversedge, I. Gilchrist, & S. Everling (Eds.), *The Oxford handbook of eye movements* (pp. 413-424). New York: Oxford University Press.

Sutton, S. K., Vitale, J. E., & Newman, J. P. (2002). Emotion among women with psychopathy during picture perception. *Journal of Abnormal Psychology, 111*(4), 610-619. doi:10.1037/0021-843X.111.4.610

Swogger, M. T., & Kosson, D. S. (2007). Identifying subtypes of criminal psychopaths: A replication and extension. *Criminal Justice and Behavior, 34*(8), 953-970.

Szabadi, E. (2012). Modulation of physiological reflexes by pain: Role of the locus coeruleus. *Frontiers in integrative neuroscience, 6*, 94.

Taylor, S. F., Kang, J., Brege, I. S., Tso, I. F., Hosanagar, A., & Johnson, T. D. (2012). Meta-analysis of functional neuroimaging studies of emotion perception and experience in schizophrenia. *Biological Psychiatry, 71*(2), 136-145.

Tengström, A., Grann, M., Långström, N., & Kullgren, G. (2000). Psychopathy (PCL-R) as a predictor of violent recidivism among criminal offenders with schizophrenia. *Law and human behavior, 24*(1), 45-58.

Thomas, K. M., Drevets, W. C., Whalen, P. J., Eccard, C. H., Dahl, R. E., Ryan, N. D., & Casey, B. (2001). Amygdala response to facial expressions in children and adults. *Biological Psychiatry, 49*(4), 309-316.

Toichi, M., Kubota, Y., Murai, T., Kamio, Y., Sakihama, M., Toriuchi, T., . . . Miyoshi, K. (1999). The influence of psychotic states on the autonomic nervous system in schizophrenia. *International Journal of Psychophysiology, 31*(2), 147-154.

Trautmann, S. A., Fehr, T., & Herrmann, M. (2009). Emotions in motion: dynamic compared to static facial expressions of disgust and happiness reveal more widespread emotion-specific activations. *Brain research, 1284*, 100-115.

Tukey, J. W. (1977). *Exploratory data analysis*. Reading, MA: Addison-Wesley.

Ukai, K. (1985). Spatial pattern as a stimulus to the pupillary system. *JOSA A, 2*(7), 1094-1100.

Uono, S., Sato, W., & Toichi, M. (2010). Brief report: representational momentum for dynamic facial expressions in pervasive developmental disorder. *Journal of autism and developmental disorders, 40*(3), 371-377.

Ursin, H., & Kaada, B. R. (1960). Functional localization within the amygdaloid complex in the cat. *Electroencephalography and clinical neurophysiology, 12*(1), 1-20.

Vaidyanathan, U., Hall, J. R., Patrick, C. J., & Bernat, E. M. (2011). Clarifying the role of defensive reactivity deficits in psychopathy and antisocial personality using startle reflex methodology. *Journal of Abnormal Psychology, 120*(1), 253-258. doi:10.1037/a0021224

Vaidyanathan, U., Patrick, C. J., & Bernat, E. M. (2009). Startle reflex potentiation during aversive picture viewing as an indicator of trait fear. *Psychophysiology, 46*(1), 75-85.

van der Schalk, J., Hawk, S. T., Fischer, A. H., & Doosje, B. (2011). Moving faces, looking places: Validation of the Amsterdam Dynamic Facial Expression Set (ADFES). *Emotion, 11*(4), 907-920. doi:10.1037/a0023853

Van Steenbergen, H., Band, G. P., & Hommel, B. (2011). Threat but not arousal narrows attention: evidence from pupil dilation and saccade control. *Cognitive and affective control, 2*(281), 1 - 5.

Vanman, E. J., Mejia, V. Y., Dawson, M. E., Schell, A. M., & Raine, A. (2003). Modification of the startle reflex in a community sample: do one or two dimensions of psychopathy underlie emotional processing? *Personality and Individual Differences, 35*(8), 2007-2021.

Venables, N., Hall, J., & Patrick, C. (2014). Differentiating psychopathy from antisocial personality disorder: a triarchic model perspective. *Psychological Medicine, 44*(05), 1005-1013.

Venables, N. C., Hall, J. R., Yancey, J. R., & Patrick, C. J. (2015). Factors of psychopathy and electrocortical response to emotional pictures: Further evidence for a two-process theory. *Journal of Abnormal Psychology, 124*(2), 319-328.

Verona, E., Bresin, K., & Patrick, C. J. (2013). Revisiting psychopathy in women: Cleckley/Hare conceptions and affective response. *Journal of Abnormal Psychology, 122*(4), 1088-1093.

Verona, E., Patrick, C. J., Curtin, J. J., Bradley, M. M., & Lang, P. J. (2004). Psychopathy and physiological response to emotionally evocative sounds. *Journal of Abnormal Psychology, 113*(1), 99-108. doi:10.1037/0021-843x.113.1.99

Viding, E., Blair, R. J. R., Moffitt, T. E., & Plomin, R. (2005). Evidence for substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry, 46*(6), 592-597.

Viding, E., Jones, A. P., Paul, J. F., Moffitt, T. E., & Plomin, R. (2008). Heritability of antisocial behaviour at 9: do callous-unemotional traits matter? *Developmental science, 11*(1), 17-22.

Viding, E., Sebastian, C. L., Dadds, M. R., Lockwood, P. L., Cecil, C. A., De Brito, S. A., & McCrory, E. J. (2012). Amygdala response to preattentive masked fear in children with conduct problems: the role of callous-unemotional traits. *American Journal of Psychiatry, 169*(10), 1109-1116.

Vieira, J. B., Almeida, P. R., Ferreira-Santos, F., Barbosa, F., Marques-Teixeira, J., & Marsh, A. A. (2014). Distinct neural activation patterns underlie economic decisions in high and low psychopathy scorers. *Social Cognitive and Affective Neuroscience, 9*(8), 1099-1107.

Vieira, J. B., Ferreira-Santos, F., Almeida, P. R., Barbosa, F., Marques-Teixeira, J., & Marsh, A. A. (2015). Psychopathic traits are associated with cortical and subcortical volume alterations in healthy individuals. *Social Cognitive and Affective Neuroscience, 10*(12), 1693-1704.

Vitacco, M. J., Neumann, C. S., Caldwell, M. F., Leistico, A.-M., & Van Rybroek, G. J. (2006). Testing factor models of the Psychopathy Checklist: Youth Version and their association with instrumental aggression. *Journal of Personality Assessment, 87*(1), 74-83.

Vitacco, M. J., Neumann, C. S., & Jackson, R. L. (2005). Testing a four-factor model of psychopathy and its association with ethnicity, gender, intelligence, and violence. *Journal of consulting and clinical psychology, 73*(3), 466-476.

Võ, M. L. H., Jacobs, A. M., Kuchinke, L., Hofmann, M., Conrad, M., Schacht, A., & Hutzler, F. (2008). The coupling of emotion and cognition in the eye: Introducing the pupil old/new effect. *Psychophysiology, 45*(1), 130-140.

Walker, D. L., Paschall, G. Y., & Davis, M. (2005). Glutamate receptor antagonist infusions into the basolateral and medial amygdala reveal differential contributions to olfactory vs. context fear conditioning and expression. *Learning & memory, 12*(2), 120-129.

Wall, T. D., Wygant, D. B., & Sellbom, M. (2015). Boldness explains a key difference between psychopathy and antisocial personality disorder. *Psychiatry, Psychology and Law, 22*(1), 94-105.

Wallbott, H. G., & Ricci-Bitti, P. (1993). Decoders' processing of emotional facial expression—a top-down or bottom-up mechanism? *European Journal of Social Psychology, 23*(4), 427-443.

Wallin, B. G. (1981). Sympathetic nerve activity underlying electrodermal and cardiovascular reactions in man. *Psychophysiology, 18*(4), 470-476.

Walsh, Z., Allen, L. C., & Kosson, D. S. (2007). Beyond social deviance: Substance use disorders and the dimensions of psychopathy. *Journal of Personality Disorders, 21*(3), 273-288. doi:10.1521/pedi.2007.21.3.273

Walsh, Z., Swogger, M. T., & Kosson, D. S. (2009). Psychopathy and instrumental violence: Facet level relationships. *Journal of Personality Disorders, 23*(4), 416-424.

Walters, G. D. (2003). Predicting institutional adjustment and recidivism with the psychopathy checklist factor scores: A meta-analysis. *Law and human behavior, 27*(5), 541-558.

Walters, G. D. (2014). The latent structure of psychopathy in male adjudicated delinquents: A cross-domain taxometric analysis. *Personality Disorders: Theory, research, and treatment, 5*(4), 348-355.

Walters, G. D., Duncan, S. A., & Mitchell-Perez, K. (2007). The latent structure of psychopathy a taxometric investigation of the Psychopathy Checklist–Revised in a heterogeneous sample of male prison inmates. *Assessment, 14*(3), 270-278.

Walters, G. D., Gray, N. S., Jackson, R. L., Sewell, K. W., Rogers, R., Taylor, J., & Snowden, R. J. (2007). A taxometric analysis of the Psychopathy Checklist: Screening Version (PCL: SV): Further evidence of dimensionality. *Psychological Assessment, 19*(3), 330.

Walters, G. D., Knight, R. A., Grann, M., & Dahle, K.-P. (2008). Incremental validity of the Psychopathy Checklist facet scores: Predicting release outcome in six samples. *Journal of Abnormal Psychology, 117*(2), 396-405.

Wang, C.-A., Boehnke, S. E., Itti, L., & Munoz, D. P. (2014). Transient pupil response is modulated by contrast-based saliency. *The Journal of neuroscience, 34*(2), 408-417.

Wang, P., Baker, L. A., Gao, Y., Raine, A., & Lozano, D. I. (2012). Psychopathic traits and physiological responses to aversive stimuli in children aged 9–11 years. *Journal of Abnormal Child Psychology, 40*(5), 759-769.

Waters, W. F., Williamson, D. A., Bernard, B. A., Blouin, D. C., & Faulstich, M. E. (1987). Test-retest reliability of psychophysiological assessment. *Behaviour Research and Therapy, 25*(3), 213-221.

Wechsler, D. (1997). *Wechsler adult intelligence scale: Administration and scoring manual, 3rd edition*. San Antonio, TX: Psychological Corporation.

Wechsler, D. (1999). *Wechsler abbreviated scale of intelligence (WASI)*. San Antonio, TX: Psychological Corporation.

Wechsler, D. (2014). Wechsler Adult Intelligence Scale–Fourth Edition (WAIS–IV).

Weinberg, A., & Hajcak, G. (2010). Beyond good and evil: the time-course of neural activity elicited by specific picture content. *Emotion, 10*(6), 767-782.

Weinberg, A., Klonsky, E. D., & Hajcak, G. (2009). Autonomic impairment in Borderline Personality Disorder: A laboratory investigation. *Brain and Cognition, 71*(3), 279-286. doi:10.1016/j.bandc.2009.07.014

Weyers, P., Mühlberger, A., Hefele, C., & Pauli, P. (2006). Electromyographic responses to static and dynamic avatar emotional facial expressions. *Psychophysiology, 43*(5), 450-453.

Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *The Journal of neuroscience, 18*(1), 411-418.

Whalen, P. J., Shin, L. M., McInerney, S. C., Fischer, H., Wright, C. I., & Rauch, S. L. (2001). A functional MRI study of human amygdala responses to facial expressions of fear versus anger. *Emotion, 1*(1), 70-83.

White, T. L., & Depue, R. A. (1999). Differential association of traits of fear and anxiety with norepinephrine and dark-induced pupil reactivity. *Journal of Personality and Social Psychology, 77*(4), 863-877. doi:10.1037/0022-3514.77.4.863

Williams, K. M., Nathanson, C., & Paulhus, D. L. (2003). *Structure and validity of the self-report psychopathy scale-III in normal populations.* Paper presented at the 111th annual convention of the American Psychological Association.

Williams, K. M., & Paulhus, D. L. (2004). Factor structure of the Self-Report Psychopathy scale (SRP-II) in non-forensic samples. *Personality and Individual Differences, 37*(4), 765-778.

Williams, L. L. M., Das, P., Liddell, B. J., Olivieri, G., Peduto, A. S., David, A. S., . . . Harris, A. W. (2007). Fronto-limbic and autonomic disjunctions to negative emotion distinguish schizophrenia subtypes. *Psychiatry Research: Neuroimaging, 155*(1), 29-44.

Williams, L. M., Phillips, M. L., Brammer, M. J., Skerrett, D., Lagopoulos, J., Rennie, C., . . . Peduto, A. (2001). Arousal dissociates amygdala and hippocampal fear responses: evidence from simultaneous fMRI and skin conductance recording. *Neuroimage, 14*(5), 1070-1079.

Williamson, S., Harpur, T. J., & Hare, R. D. (1991). Abnormal processing of affective words by psychopaths. *Psychophysiology, 28*(3), 260-273.

Wilson, K., Juodis, M., & Porter, S. (2011). Fear and loathing in psychopaths: A meta-analytic investigation of the facial affect recognition deficit. *Criminal Justice and Behavior, 38*(7), 659-668.

Winstanley, C. A., Theobald, D. E., Cardinal, R. N., & Robbins, T. W. (2004). Contrasting roles of basolateral amygdala and orbitofrontal cortex in impulsive choice. *The Journal of neuroscience, 24*(20), 4718-4722.

Wolf, R. C., Pujara, M. S., Motzkin, J. C., Newman, J. P., Kiehl, K. A., Decety, J., . . . Koenigs, M. (2015). Interpersonal traits of psychopathy linked to reduced integrity of the uncinate fasciculus. *Human brain mapping, 36*(10), 4202-4209.

Wright, C. I., Fischer, H., Whalen, P. J., McInerney, S. C., Shin, L. M., & Rauch, S. L. (2001). Differential prefrontal cortex and amygdala habituation to repeatedly presented emotional stimuli. *Neuroreport, 12*(2), 379-383.

Wyatt, H. J. (1995). The form of the human pupil. *Vision Research, 35*(14), 2021-2036.

Yang, Y. L., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta-analysis. *Psychiatry Research-Neuroimaging, 174*(2), 81-88. doi:10.1016/j.pscychresns.2009.03.012

Yildirim, B. O., & Derksen, J. J. (2013). Systematic review, structural analysis, and new theoretical perspectives on the role of serotonin and associated genes in the etiology of psychopathy and sociopathy. *Neuroscience & Biobehavioral Reviews, 37*(7), 1254-1296.

Yildirim, B. O., & Derksen, J. J. (2015). Clarifying the heterogeneity in psychopathic samples: towards a new continuum of primary and secondary psychopathy. *Aggression and Violent Behavior, 24*, 9-41.

Young, R. S., Han, B.-C., & Wu, P.-Y. (1993). Transient and sustained components of the pupillary responses evoked by luminance and color. *Vision Research, 33*(4), 437-446.

Zahn, T. P., Carpenter, W. T., & McGlashan, T. H. (1981a). Autonomic nervous system activity in acute schizophrenia: I. Method and comparison with normal controls. *Archives of General Psychiatry, 38*(3), 251-258.

Zahn, T. P., Carpenter, W. T., & McGlashan, T. H. (1981b). Autonomic nervous system activity in acute schizophrenia: II. Relationships to short-term prognosis and clinical state. *Archives of General Psychiatry, 38*(3), 260-266.

Zahn, T. P., Jacobsen, L. K., Gordon, C. T., McKenna, K., Frazier, J. A., & Rapoport, J. L. (1997). Autonomic nervous system markers of psychopathology in childhood-onset schizophrenia. *Archives of General Psychiatry, 54*(10), 904-912.

Zhu, J., Tulsky, D., & Leyva, L. (1999). Using WASI in conjunction with the WAIS-III. *Archives of Clinical Neuropsychology, 14*(8), 724-725.

Zimak, E. H., Suhr, J., & Bolinger, E. M. (2014). Psychophysiological and neuropsychological characteristics of non-incarcerated adult males with higher levels of psychopathic personality traits. *Journal of Psychopathology and Behavioral Assessment, 36*(4), 542-554.

Zimmermann-Viehoff, F., Kuehl, L., Danker-Hopfe, H., Whooley, M., & Otte, C. (2014). Antidepressants, autonomic function and mortality in patients with coronary heart disease: data from the Heart and Soul Study. *Psychological Medicine, 44*(14), 2975-2984.

Appendices

Appendix 1

Table 0.1 Summary of hierarchical regressions exploring gender as a moderating variable between Triarchic Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) and each emotional index (EI; emotion minus neutral) for pupil diameter to images, and pupil diameter to neutral images. Gender was dummy coded (males = 0, females = 1). Subscales were abbreviated to B (Boldness), M (Meanness) and D (Disinhibition) to fit.

		Gender	B	M	D	B x gender	M x gender	D x gender		
		β	*β*	*β*	*β*	*β*	*β*	*β*	R^2	ΔR^2
	Step									
$EI_{Negative}$	1	.21	.03	.09	-.08					
	2	.16	.004	.28	-.17	.09	-.36^{+}	.17	.09	.05
$EI_{Positive}$	1	-.07	-.02	.05	-.04					
	2	-.10	-.05	.20	-.17	.09	-.28	.21	.04	.03
Neutral images	1	-.28*	.05	-.24	.07					
	2	-.25+	.20	-.36^{+}	.05	-.23	.24	-.04	.12	.04

* $p < .017$, Bonferroni-corrected α level
$^{+}$ $p < .05$,
β, standardised beta,
R^2, variance,
ΔR^2, change in variance

Table 0.2 Summary of hierarchical regressions exploring gender as a moderating variable between Triarchic Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) and each emotional index (EI; emotion minus neutral) for pupil diameter to static facial expressions, and pupil diameter to neutral static facial expressions. Gender was dummy coded (males = 0, females = 1). Subscales were abbreviated to B (Boldness), M (Meanness) and D (Disinhibition) to fit.

		Gender	B	M	D	B x gender	M x gender	D x gender		
	Step	*β*	*β*	*β*	*β*	*β*	*β*	*β*	R^2	ΔR^2
$EI_{Fearful}$	1	.03	.02	.14	-.24					
	2	.05	-.01	.08	-.24	.02	.12	-.02	.05	.04
EI_{Happy}	1	.12	.05	.17	-.07					
	2	.15	.07	.09	-.07	-.04	.19	-.02	.04	.03
$EI_{Disgusted}$	1	.09	-.07	.19	-.13					
	2	.09	-.07	.22	-.22	.01	-.05	.14	.03	.05
EI_{Angry}	1	.15	.05	.004	-.02					
	2	.16	-.05	-.05	.01	.11	.12	-.05	.04	.01
EI_{Sad}	1	.09	.06	.03	-.03					
	2	.08	.04	.05	-.03	.02	-.04	.001	.01	.02
Neutral faces	1	$-.26^{+}$	-.10	-.16	.11					
	2	$-.26^{+}$	-.17	-.17	.17	.10	.01	-.07	.06	.01

* $p < .017$, Bonferroni-corrected α level

$^{+}p < .05$,

β, standardised beta,

R^2, variance,

ΔR^2, change in variance

Table 0.3 Summary of hierarchical regressions exploring gender as a moderating variable between Triarchic Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) and each emotional index (EI; emotion minus neutral) for pupil diameter to dynamic facial expressions, and pupil diameter to neutral dynamic facial expressions. Gender was dummy coded (males = 0, females = 1). Gender was dummy coded (males = 0, females = 1). Subscales were abbreviated to B (Boldness), M (Meanness) and D (Disinhibition) to fit.

			Gender	B	M	D	B x gender	M x gender	D x gender		
		Step	*β*	*β*	*β*	*β*	*β*	*β*	*β*	R^2	ΔR^2
Early	$EI_{Fearful}$	1	.09	.05	.02	-.06					
		2	.11	-.05	.01	-.11	.13	.03	.08	.03	.01
	EI_{Happy}	1	-.01	.08	.11	-.12					
		2	.03	.12	-.07	-.15	-.11	.39+	-.04	.10	.07
	$EI_{Disgusted}$	1	.29*	.12	.17	-.05					
		2	.29+	.03	.24	-.15	.18	-.15	.19	.10	.02
	EI_{Angry}	1	.24+	-.07	.32+	-.07					
		2	.24+	-.23	.29	.02	.18	.06	-.11	.10	.02
	EI_{Sad}	1	.15	.07	.11	.02					
		2	.17	-.06	.14	-.12	.21	-.05	.24	.07	.04
Late	$EI_{Fearful}$	1	.12	.05	.01	-.05					
		2	.12	-.03	.02	-.08	.11	-.02	.06	.02	.01
	EI_{Happy}	1	.08	.12	.04	-.03					
		2	.11	.23	-.16	-.003	-.22	.42+	-.15	.08	.07
	$EI_{Disgusted}$	1	.31****	.13	.05	.02					
		2	.30+	.10	.12	-.01	.06	-.14	.07	.08	.01
	EI_{Angry}	1	.24	-.04	.20	-.01					
		2	.24	-.13	.15	.09	.08	.10	-.15	.07	.01
	EI_{Sad}	1	.16	.10	.05	.01					
		2	.17	.08	.04	-.04	.04	.02	.08	.03	.01
Neutral faces		1	-.20	-.04	-.13	.01					
		2	-.21	.08	-.11	-.03	-.15	-.06	.05	.05	.01

* $p < .017$, Bonferroni-corrected α level

+ $p < .05$,

β, standardised beta,

R^2, variance,

ΔR^2, change in variance

Table 0.4 Summary of hierarchical regressions exploring gender as a moderating variable between Triarchic Psychopathy Measure subscales (Boldness, Meanness and Disinhibition) and each emotional index (EI; emotion minus neutral) for pupil diameter to sound-clips, and pupil diameter to neutral images. Gender was dummy coded (males = 0, females = 1). Subscales were abbreviated to B (Boldness), M (Meanness) and D (Disinhibition) to fit.

			Gender	B	M	D	B x gender	M x gender	D x gender		
		Step	*β*	*β*	*β*	*β*	*β*	*β*	*β*	R^2	ΔR^2
Early	$EI_{Negative}$	1	-.03	.09	.01	-.07					
		2	-.02	.001	.05	-.17	.15	-.06	.17	.02	.004
	$EI_{Negative}$	1	.01	.07	-.12	.07					
		2	.01	.15	-.16	.15	-.12	.07	-.14	.02	.01
Late	$EI_{Negative}$	1	.15	-.001	.10	-.13					
		2	.13	-.12	.21	-.19	.19	-.20	.12	.05	.02
	$EI_{Negative}$	1	.13	.07	-.09	-.06					
		2	.10	.11	-.06	.03	-.06	-.09	-.11	.06	.02
Early	Neutral sound-clips	1	.08	.01	.05	.03					
		2	.06	-.06	.16	-.05	.13	-.19	.15	.03	.02
Late	Neutral sound-clips	1	.08	-.01	.12	-.06					
		2	.06	-.10	.28	-.08	.14	-.14	.06	.04	.01

* $p < .017$, Bonferroni-corrected α level

$^{+}p < .05$,

β, standardised beta,

R^2, variance,

ΔR^2, change in variance

Table 0.5 Mean scores, standard deviation and range across gender for the Tri-PM subscales

	Males		Females	
	Mean (SD)	Range	Mean (SD)	Range
Boldness	33.04 (8.08)	18 - 50	25.98 (7.95)	7 - 43
Meanness	16.42 (9.36)	3 – 46	8.92 (5.61)	1 - 24
Disinhibition	15.71 (7.92)	2 - 32	12.71 (6.84)	4 - 39

Appendix 2

Table 0.1 Summary of alcohol/substance abuse in relation to PCL-R scores

		PCL-R Total		PCL-R Factor 1		PCL-R Factor 2	
	n	Mean	SD	Mean	SD	Mean	SD
Alcohol abuse							
Yes	56	19.67	8.39	6.77	4.32	11.21	4.64
No	26	18.47	8.34	7.49	4.83	9.60	3.96
Substance abuse							
Yes	53	19.94	8.65	7.60	4.26	11.67	4.36
No	29	18.10	7.76	8.53	4.42	8.93	4.20

Table 0.2 Summary of mental health diagnoses in relation to PCL-R scores

		PCL-R Total		PCL-R Factor 1		PCL-R Factor 2	
	n	Mean	SD	Mean	SD	Mean	SD
1	23	12.23	6.53	3.64	2.57	7.74	4.77
2	4	16.75	4.74	7.90	2.74	7.33	3.40
3	-	-	-	-	-	-	-
4	32	23.60	6.62	8.77	4.39	12.81	3.09
1 and 2	1	16.00	-	1.00	-	12.00	-
1 and 4	13	21.35	7.91	8.10	4.08	11.77	4.90
2 and 4	2	27.40	13.58	12.55	4.88	11.85	7.28
3 and 4	4	17.90	8.21	5.58	6.14	10.00	2.01
1, 2 and 4	1	16.00	-	4.00	-	12.00	-
1, 3 and 4	1	30.00	-	13.00	-	14.00	-

1 = Schizophrenia, schizotypal and delusional disorders
2 = Mood disorders
3 = Neurotic, stress-related and somatoform disorders
4 = Personality disorder
One participant was yet to receive a formal mental health diagnosis

Table 0.3 Summary of specific personality disorder diagnosis in relation to PCL-R scores

		PCL-R Total		PCL-R Factor 1		PCL-R Factor 2	
	n	Mean	SD	Mean	SD	Mean	SD
1	-	-	-	-	-	-	-
2	1	21.10	-	11.40	-	8.00	-
3	24	26.50	4.76	10.20	3.35	14.21	2.29
4	7	15.96	9.71	4.57	4.79	9.99	4.80
5	1	10.50	-	7.00	-	3.10	-
6	5	22.56	4.82	8.82	3.77	11.98	3.21
1 and 3	2	22.45	4.88	12.00	1.41	7.60	3.68
1 and 4	1	10.50	-	0.00	-	11.10	-
2 and 3	1	19.00	-	10.00	-	5.00	-
3 and 4	10	19.85	6.45	5.99	4.25	12.25	2.14
1, 3 and 4	1	37.00	-	16.00	-	17.00	-

1 = Paranoid personality disorder
2 = Schizoid unstable personality disorder
3 = Dissocial personality disorder
4 = Emotionally unstable personality disorder
5 = Other specific personality disorder
6 = Mixed personality disorder

Appendix 3

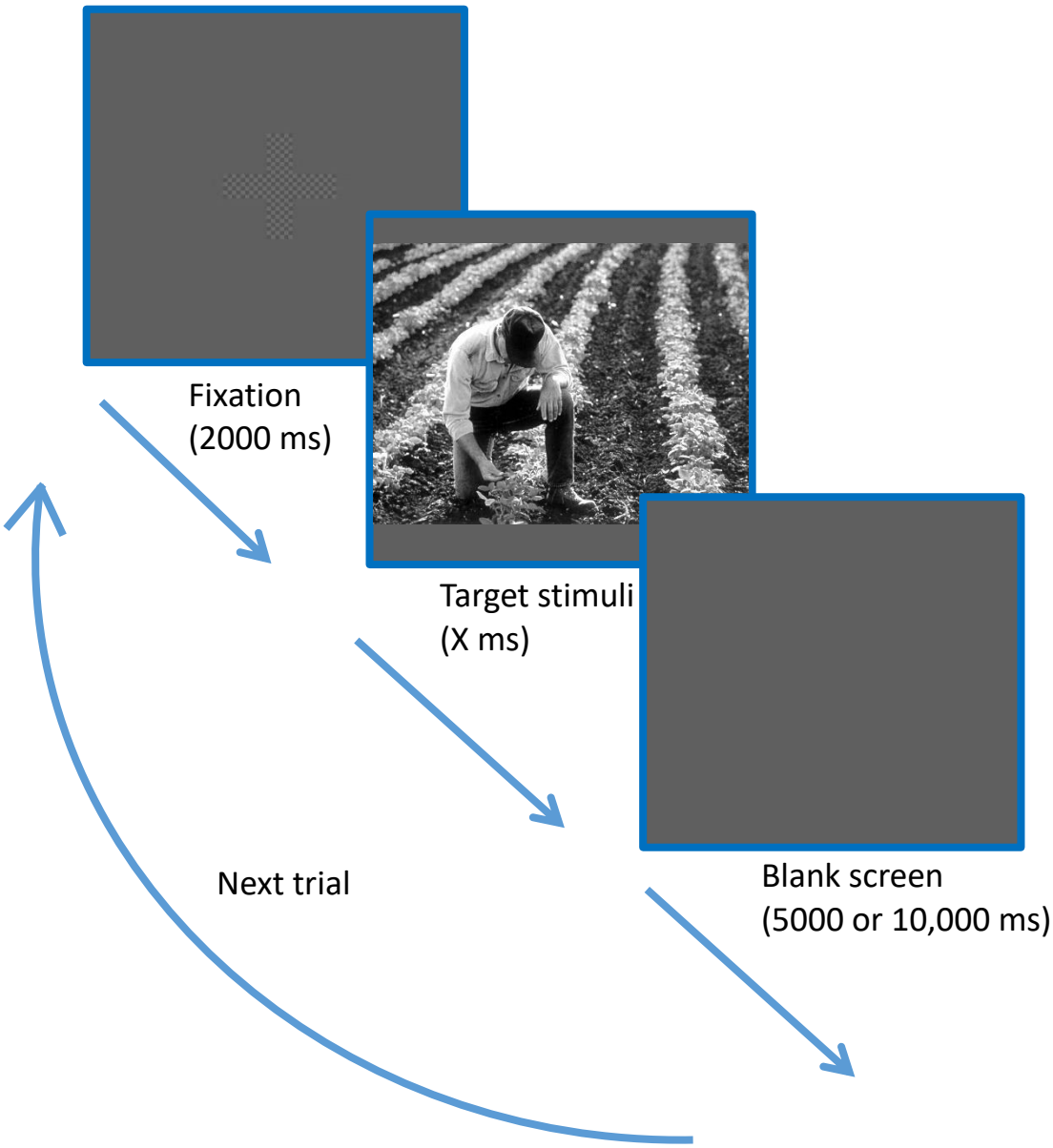
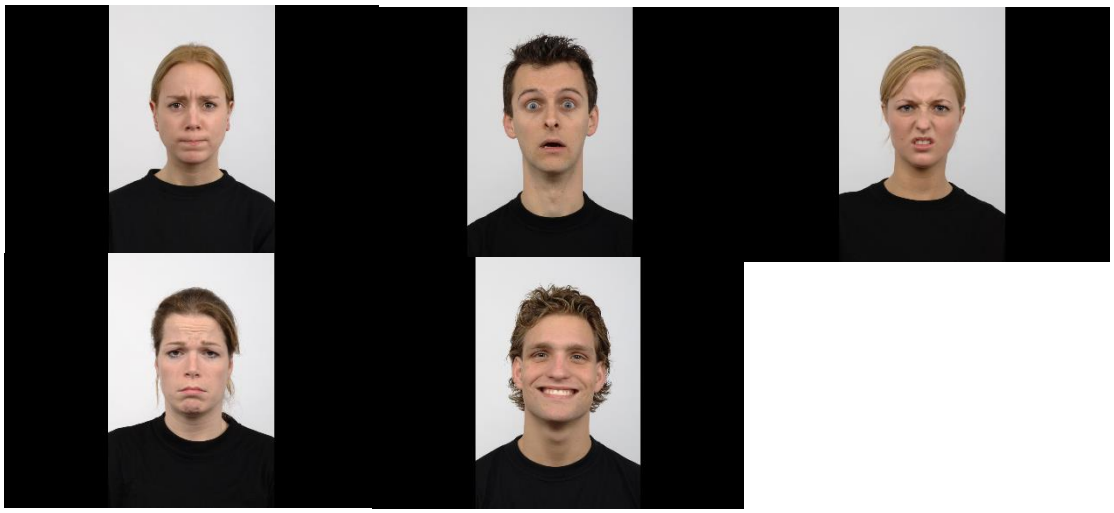


Figure 0.1. Schematic illustration of the structure of a trial. This structure was consistent across experiments.

Examples of images used in Chapter 3 and 4:

Examples of static facial expressions used in Chapter 3:



Screenshot examples of dynamic facial expressions used in Chapter 3 and 4:

Publication List

Snowden, R. J., O'Farrell, K. R., Burley, D., Erichsen, J. T., Newton, N. V., & Gray, N. S. (2016). The pupil's response to affective pictures: Role of image duration, habituation, and viewing mode. *Psychophysiology, 53*(8), 1217-1223. doi:10.1111/psyp.12668

This publication describes the work from Chapter 2 investigating pupil responses to complex images in relation to visual characteristics of the stimuli, as well as the role of presentation duration and repeated presentation of the visual stimuli upon emotional modulation of pupil diameter.

Burley, D. T., Gray, N. S., & Snowden, R. J. (2017). As Far as the Eye Can See: Relationship between Psychopathic Traits and Pupil Response to Affective Stimuli. *PLoS ONE, 12*(1), e0167436. doi:10.1371/journal.pone.0167436

This publication details the work from Chapter 3 exploring pupil responses to emotion in relation to psychopathic traits within a community undergraduate population.