Psychopathic personality traits and antisocial behaviours in adults: Behavioural, emotional, and physiological correlates

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Declaration of Authorship

I, Guilherme Welter Wendt, hereby declare that this thesis and the work presented in the following pages in it is entirely my own. Where I have consulted the work of others, this is always clearly stated.

Signed:

Date: 28/08/2018

Abstract

This thesis presents a series of studies that contribute to the literature about psychopathic traits in the general population, addressing physiological, emotional and behavioural correlates. At the level of behaviour, studies within Chapter Three showed positive correlations between bullying and psychopathy, and found better explanatory power for Primary Psychopathy in predicting bullying (Study 1). Additionally, bully–victims were found to present higher psychopathic traits (Study 2). At the physiological level, Chapter Four presents two studies examining cardiovascular functioning at rest and cardiovascular reactivity to stress. Rebellious Nonconformity, Total Psychopathy, and Social Influence showed significant, negative associations with resting heart rate (Study 3). Subsequently, Study 4 reports an association between psychopathic behaviours with threatening physiological responses, showing that participants high on their total levels of psychopathic personality traits and on Machiavellian Egocentricity were marked by a maladaptive pattern of hypothalamic–pituitary–adrenal reactivity to stress. Finally, this thesis aimed to extend the psychophysiological findings to the level of behaviour while also accounting for emotional deficiencies (cf. Chapter Five). Study 5 revealed that secondary variants of psychopathy were significant in predicting perceived stress. Additionally, the associations between psychopathic variants and perceived stress were mediated by specific deficiencies in empathy (e.g., difficulties in identifying and describing feelings). In summary, data presented in this thesis indicated differential associations of psychopathic traits in cardiovascular functioning at rest, and central nervous system reactivity to laboratory–induced stress. Further, that elevated self–report psychopathy was accompanied by higher behavioural and emotional difficulties.

Contents

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Abbreviations

AB – Antisocial behaviours

ACTH – Adrenocorticotropic hormone

ANOVA – Analysis of variance

ANS – Autonomic nervous system

APA – American Psychiatric Association

APD – Antisocial Personality Disorder

APSD – Antisocial Process Screening Device

BAS – Behavioural activation system

BIS – Behavioural inhibition system

BP – Bresuch–Pagan test

BPD – Borderline Personality Disorder

BPSM – Bio–psychosocial model of Challenge and Threat

CD – Conduct disorder

CFI – Comparative fit index

CI – Confidence interval

CNS – Central nervous system

CO – Cardiac output

CP – Conduct problems

CU – Callous–unemotional traits

CVLM – Caudal ventrolateral medulla

DBP – Diastolic blood pressure

DDF – Difficulties describing feelings

DIF – Difficulties identifying feelings

DLPFC – Dorsolateral prefrontal cortex

DSM – Diagnostic and Statistical Manual of Mental Disorders

DV – Dependent variable

DVN – Dorsal vagal motor nucleus

EEG – Electroencephalography

EMG – Electromyography

EOT – Externally oriented thinking

EPR – Event–related potential

FDR – False discovery rate

GSR – Galvanic skin response

HC – Harvey–Collier's test

HiTOP – The Hierarchical Taxonomy of Psychopathology

HP – Heart period

HPA – Hypothalamic pituitary adrenal axis

HR – Heart rate

HRV – Heart rate variability

IBS – Illinois Bullying scale

ICD–10 – International Statistical Classification of Diseases and Related Health Problems 10th Revision

IQ – Intelligence quotient

IRT – Item–Response Theory

IV – Independent variable

LCE – Linear curve estimation

LPA – Latent profile analysis

LRHR – Low resting heart rate

LSRP – Levenson Self–Report Psychopathy scale

LVET – Left–ventricular contractility reactivity

MBP – Mean blood pressure

mPFC – Medial prefrontal cortex

NA – Nucleus ambiguus

NHST – Null hypothesis significance testing

NTS – Nucleus of the solitary tract

PCL–R – Psychopathy Checklist Revised

PCL–SV – Psychopathy Checklist: Screening Version

PCL–YV – Psychopathy Checklist: Youth Version

PEP – Pre–ejection period

PFC – Prefrontal cortex

PNS – Parasympathetic nervous system

PP – Primary psychopathy

PPI – Psychopathic Personality Inventory

PPI–R–40 – Psychopathic Personality Inventory Genetic Derived Form

PSS – Perceived stress scale

RDoC – Research Domain Criteria

RHR – Resting heart rate

RM – Response Modulation

RMHP – Response Modulation Hypothesis of Psychopathy

RMSE – Root mean square error

RMSEA – Root mean square error of approximation

RSA – Respiratory sinus arrhythmia

RVLM – Rostral ventrolateral medulla

SAM – Sympathetic–adrenal medullary axis

SBP – Systolic blood pressure

SCL – Skin conductance levels

SEM – Structural equation modeling

SNS – Sympathetic nervous system

SP – Secondary psychopathy

SRMR – Standardized root mean residual

SRP – Self–Report psychopathy scale

SV – Stroke volume

TAS – Toronto alexithymia scale

TPR – Total peripheral resistance

VC – Ventricular contractility

VIF – Variable inflation factor

YPT – Youth Psychopathic Traits Inventory

Dissemination

Most studies presented in this thesis will be considered for publication. The earlier work (comprehending the studies conducted while I was a 1st and 2nd year PhD Student) has already been sent for consideration at journals specialised in aggression and psychopathy. Hence, studies within Chapter Three possess the following status:

Study 1, Chapter Three. "Adult bullying and primary and secondary psychopathic traits: insights from a community sample" has been published (*Journal of Aggression, Maltreatment & Trauma,* vol. 27, n. 2, 2018).

Study 2, Chapter Three. "Bullying involvement and psychopathic personality traits: disentangling the links amongst college students" has been accepted for publication (*European Journal of Education and Psychology*).

The experimental work has been partially presented at the following conferences:

Study 4, Chapter Four. "How do you respond? Antisocial behaviours are linked to threatening physiological responses" was presented at the *7th Biennial Meeting of the Society for the Scientific Study of Psychopathy* (Belgium, 2017), *24th Biennial Meeting of the International Society for the Study of Behavioural Development* (Lithuania, 2016) and at the *31st International Congress of Psychology* (Japan, 2016).

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CHAPTER ONE – GENERAL INTRODUCTION

Chapter overview

The present thesis is concerned with how traits associated with the various delineations of antisocial behaviours (AB), including psychopathy, might be understood in terms of their psychophysiological, affective, and behavioural manifestations. The purpose of the first two chapters is to provide relevant information regarding how this thesis and its hypotheses were conceptualised. Chapter One commences with an overview of research on AB, examining how this literature has evolved to a consideration of the related traits, rather than the behaviour per se. This first chapter will also discuss the proposed factorial solutions for psychopathy – along with comprising facets – and how these might be useful in understanding specific forms of antisocial/aggressive behaviours, particularly the phenomenon of bullying in adults. This will be followed by literature review on aetiological factors, and – given the focus on psychophysiological correlates – biological influences on psychopathy and AB (Chapter Two).

1.1 Defining antisocial behaviour

The development of AB involves a complex interplay of environmental and biological factors within a given context (Byrd, Loeber, & Pardini, 2014; Fowles, 2018; Frick & Thornton, 2017). Definitions for AB vary significantly between authors and cultures, normally being included on a continuum of severity (Curtis, 2016). The Nuffield Council on Bioethics published a document in 2002 providing a comprehensive list of definitions for AB used by professionals. The publication stated that although criminologists, psychologists, and clinicians differ slightly in their definitions for AB, there seems to exist a consensus that these acts involve the violation of social norms and others' rights. From this basic foundation, many disciplines have dedicated focus on the manifestations of AB. For example, AB is included in the psychological and psychiatric traditions to help in establishing diagnosis for oppositional disorder, conduct disorder, and antisocial personality

disorder (Rutter, Giller, & Hagell, 1998; World Health Organisation, 2015). AB is frequently defined as the combination of acts such as aggression and rule–breaking, which, together with defiant personality traits, form a cluster of relevant indicators used to predict the degree of risk for severe criminal involvement (American Psychiatric Association, 2013; Curtis, 2016; Niv, Tuvblad, Raine, & Baker, 2013). Consequently, another approach focuses rather more in criminal behaviours, in which AB is taken as any act contrary to the law (Miller, 2017; Rutter et al., 1998).

Nonetheless, AB is not always a violation of the law, which stresses to the importance of sociocultural factors in better comprehending these behaviours (Byrd et al., 2014; Curtis, 2016; Fowles, 2018). For both children and adults, antisociality is heterogeneous in its nature, requiring competing models to explain the routes by which one might develop advanced (or severe) AB (Colins, Andershed, Salekin, & Fanti, 2018; Frick & Marsee, 2018). A significant number of developmental life course studies suggested that aetiological differences occur between individuals with chronic and moderate antisocial trajectories (McCuish, Corrado, Lussier, & Hart, 2014). With the intent of clarifying differences in the individual differences in the stability of AB, Moffitt (1993) presented a dual–taxonomy proposition in which temporary versus persistent antisociality was examined. According to these theories, a large group of subjects present themselves with AB for a very short time (normally during adolescence), whereas a rather smaller group displays persistent AB. Differences in temporal stability – coupled with the inconsistencies in situations in which AB are displayed – suggest that deviant behaviours could be sporadic and instrumental for most individuals. For instance, some adolescents might engage in bullying behaviours at school in order to achieve dominance, status, and power over others, but could behave accordingly in other social environments (Hanish, Sallquist, DiDonato, Fabes, & Martin, 2012). For them, AB is inconsistent, occurring for brief moments. Additionally, these adolescents can cease using aggressive strategies at school and at other social contexts when prosocial attitudes are perceived as being more rewarding (Moffitt, 1993).

A related proposition examines the developmental period in which behavioural problems emerge (Frick & Marsee, 2018). While those who start to behave in an antisocial manner during adolescence seem to reflect an exacerbated version of the "adolescent rebellion", those showing AB in childhood tend to extend their difficulties at further developmental phases (Frick & Viding, 2009). This age of onset approach is one that has been used in the Diagnostic and Statistical Manual of Mental Disorders since its fourth edition (APA, 1994, 2013). Research has largely supported the hypothesis that profiles of functioning between those who show an onset of AB during childhood and adolescence would differ, although there is some evidence showing that abnormalities within neurophysiological patterns of emotional processing are present in individuals with conduct problems notwithstanding the age of onset (Fairchild et al., 2011; Passamonti, Fairchild, Goodyer, Hurford, Hagan, Rowe, & Calder, 2010; but see Moffitt, 2018). Further, there is also a common difficulty in reliably reporting the 'true' age of onset of AB, where some children only come to the attention of services during adolescence, but file review or family interview may suggest AB being evident far earlier in the child's life.

Early–onset, or persistent AB is typically marked by stable patterns of AB across situations and contexts, hence denoting a more trait–like disposition. In addition, these individuals present early signs of aggression at home, difficulties in adapting to social norms within the community, becoming eventually problematic at work, and can engage in a variety of types of criminal acts in various contexts (Moffitt, 1993; Patterson, DeBaryshe, & Ramsey, 1989). While these persistent forms of AB are likely to be influenced by neuropsychological differences, the adolescent–limited presentation has its risk factors mostly centred on peer norms and social mimicry (APA, 2013; Moffitt, 1993; Moffitt & Caspi, 2005). This dual–causal taxonomy has clear consequences for prevention and treatment (Frick & Marsee, 2018; Moffitt, Caspi, Rutter, & Silva, 2004; Sullivan, Piquero, & Cullen, 2012; Thornberry, 2004).

It is also important to realise that AB does not develop without interaction with others in the environment. Patterson et al. (1989) proposed a developmental perspective on AB in which family and school conflicts, social rejection, and

deviant peer–relations were included. According to this perspective, AB can be defined as a "developmental trait" (Patterson et al., 1989, p. 329) which origins in the early–life experiences and progresses throughout adulthood. Behaviours displayed during one developmental stage are anticipated to cause feedback from the environment later in life, which would then lead to a series of mutual actions and reactions from the individual and its environment. This perspective assumes that coercive and antisocial behaviours are reinforced by significant figures, such as close friends, relatives, and school staff, which would arguably add a greater resistance to the extinction of these behaviours (Lilienfeld, 2018).

These theoretical models to explain AB have received empirical scrutiny, evident from research on risk factors for AB, including 'mild' presentations to more extreme forms (Golden, Zachar, Lowry, & Tran, 2017; Krueger, Hicks, Patrick, Carlson, Iacono, & McGue, 2002; LeBlanc & Loeber; 1998; Moffitt, 2006; Salekin, 2016). Adrian Raine has spearheaded much of the work searching for biological markers and mechanisms involved in AB. In 1990, Raine and colleagues used electroencephalography (EEG) to attempt to predict later criminal involvement. The investigation showed differences in terms of general low arousal in the pre–frontal cortex (e.g., theta waves in the EEG) in addition to diminished cardiovascular and electrodermal measurements (Raine, Venables, & Williams, 1990). The investigation revealed deficits in arousal at the age of 15 in the cardiovascular and electrodermal systems of participants who were later involved with criminality. This work has given continuity to earlier examinations of biological indicators believed to index psychopaths' tendency for low fear (Lykken, 1957), adding to a consistent understanding that low arousal would partly explain higher involvement with AB (Eysenck, 1977).

Raine's (1988) assertions that some strong genetic influences could partially explain his findings in terms of low arousal at both brain regions and in the autonomic nervous system (ANS) were further supported by large investigations. Two twin studies, combining a total of 4,854 twin pairs (Jaffee, Caspi, Moffitt, & Taylor, 2004; Viding, Blair, Moffitt, & Plomin, 2005), suggested strong genetic risks for AB. In respect to early psychopathic traits, evidence presented by Viding et al. (2005) suggest that children with high levels of callous–

unemotional (CU) traits have higher risks for AB; when the levels of CU traits are low, however, the genetic risk for AB is moderate and under influence of shared environment. This proposes that the AB seen in those with elevated CU traits is driven, in part, by exclusive neurological aetiologies, suggesting to distinct genetic origins in comparison to those with higher AB, but with low CU traits (Gard, Dotterer, & Hyde, 2018; Hyde, Shaw, & Hariri, 2013). It has also been possible to consider the role of environment in a sample of non-related parents and children. Waller et al. (2017c) noted that parenting was a predictor of early CU traits amongst adopted children; specifically, lower positive reinforcement (e.g., warmth) was exclusively linked to callous-unemotional traits at the age of 27 months, suggesting that parenting in early childhood is a potentially important factor in the development of these antisocial traits, independently of passive gene-environment correlations.

More recently, Lewis, Boisvert, Connolly, and Boutwell (2018) examined the co–occurrence between psychopathic personality traits and AB using a behavioural genetic approach in a representative sample of American youths, including 872 monozygotic and dizygotic twins. AB was assessed using questions including physical (e.g., “get into a serious physical fight”), highly violent (e.g., “shot or stabbed someone”), and non–violent forms of antisocial and criminal conduct (e.g., “steal something worth more than $50”), and psychopathic personality traits were measured with items from the Five Factor Model, such as "I am not really interested in others" and "I live my life without much thought for the future" (FFM; Derefinko & Lynam, 2007). Genetic influences were responsible for 39% and 53% of the variance in psychopathic personality traits and criminal/antisocial behaviours, respectively, and non–shared environmental influences accounted for 61% and 47% of the variance in psychopathic personality traits and criminal/antisocial behaviours, respectively. A significant correlation of .29 was found between genetic influences on psychopathic personality traits and AB, and the overlap between these two constructs was largely explained (i.e., 58%) by common additive genetic components (Lewis et al., 2018). Moreover, psychopathy was not linked to criminal/antisocial behaviours when genetic and shared environmental influences were controlled for, suggesting that shared genetic influences are important in elucidating the covariance of criminal/antisocial

behaviours and psychopathic personality traits (Lewis et al., 2018). Another illustrative example comes from a work conducted by Niv et al. (2013), who reported the results from a large twin study examining risk factors for AB. This investigation examined both genetic and environmental influences on AB from childhood (9 to 12 years–old) to early adulthood (19–20 years–old). No sex differences were found in genetic and environmental influences reported. However, aggression and rule–breaking behaviours were under the influence of a common factor (e.g., general AB), for which genetic and environmental influences were shared. Both aggression and rule–breaking also had exclusive genetic and environmental influences. Developmental processes during childhood and adolescence have been assumed to provide an explanation between different forms of AB, yet here they have been reported to originate from a general common factor.

These investigations offer evidence to the idea that AB, including psychopathic personality traits, might fit into a trait–like framework for certain subgroups (Krueger et al., 2002; Lewis et al., 2018; Nelson & Foell, 2018; Viding et al., 2005). Relatedly, Patrick and Hajcak (2016) emphasised the recent claims made by the Research Domain Criteria (RDoC) for more biologically–oriented investigations for better understanding aetiological mechanisms that might explain various problematic behaviours. Indeed, there are robust indicators regarding biological under–arousal, coupled with vulnerabilities to externalising psychopathologies, that might enhance the occurrence of dysfunctional interpersonal styles in children, adolescents, and adults (Forth & Burke, 1998; Lorber, 2004; Margolin & Gordis, 2000; Raine & Dunkin, 1990; Raine & Jones, 1987; Scarpa, Raine, Venables, & Mednick, 1997). However, Jaffee et al. (2004) noted that experiencing maltreatment in infancy and childhood affects antisocial traits even after controlling for the genetic transmission of these traits, which indicates that one avenue for reducing the impact of AB on society would benefit from adopting approaches to prevent child abuse. Bullying is one specific (and particularly problematic) form of AB that some children are exposed to (Hanish et al., 2012), and some diagnosis tools for conduct disorder in children specifically measure excessive levels of bullying and fighting as key criteria (e.g., International Statistical Classification of Diseases and Related Health Problems 10th Revision

[ICD–10]; WHO, 2015). For these reasons, bullying behaviours are clearly important in the study of AB, and will be examined in the next section.

1.1.1 Bullying, and its relation to our understanding of antisocial behaviour

Considering sociocultural variations and the broader extent covered by definitions of "antisocial behaviour", specific, but nested concepts have been used in the scientific literature intending to provide a more narrowed account of various aetiological mechanisms associated to AB (Curtis, 2016; Walters, 2012). One important area related to increased risks for AB is the concomitant progression of callousness and early aggressive behaviours, particularly bullying (Fanti & Kimonis, 2012; Fontaine, Hanscombe, Berg, McCrory, & Viding, 2016; Free, 2017; Muñoz, Qualter, & Padgett, 2011). Indeed, these investigations contribute to a growing investment in exploring the Externalising Spectrum – which contains psychopathic, aggressive, bullying behaviours – within the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017; Patrick, Kramer, Krueger, & Markon, 2013). Accordingly, this section will examine how the manifestation of AB, such as bullying, could possibly be comorbid to psychopathy and, to a broader extent, how both phenomena might originate from a common underlying factor (Hare, 2001; Krueger et al., 2002; Nelson & Foell, 2018).

Bullying is a deliberate, repetitive, and aggressive act that occurs in a relational situation of imbalance of power (Olweus, 1991). Surveys conducted with child and adolescent samples suggest that perpetrators of both direct (e.g., face–to–face aggression, including fighting and arguments) and indirect (e.g., social exclusion, spreading rumours) forms are characterised by increased social and behavioural difficulties (Jolliffe & Farrington, 2011; Smith, Polenik, Nakasita, & Jones, 2012). Pure bullies (i.e., those predominantly perpetrators) use more proactive aggression and score higher in measures of AB (Craig, 1998) and are at risk for developing consistent antisocial features (Copeland, Wolke, Angold, & Costello, 2013; Sourander et al., 2007). Additionally, bullies have been reported to also show behaviours familiar to the literature on psychopathy, such as impulsivity, manipulation, and delinquency (Gini, Albiero, Benelli, & Altoè, 2007; Menesini, Sánchez, Fonzi, Ortega, Costabile, & Lo Feudo, 2003; Warden &

Mackinnon, 2003). These characteristics clearly overlap with the descriptions of three facets of the psychopathic personality, namely: antisocial, interpersonal, and lifestyle (Hare, 2003).

Victims (or targets of bullying), in turn, do not present with a specific, clear–cut profile (Zych, Farrington, Llorent, & Ttofi, 2017). Nonetheless, risk factors for victimisation include the presence of externalising problems, interpersonal deficits, and limited number of friends (Menesini & Salmivalli, 2017). In addition, the chances of becoming a victim are contingent to the goals that perpetrators possess. This means that a very popular individual can be targeted if the bully aims status, for example (Reijntjes, Vermande, Thomaes, Goossens, Olthof, Aleva, & Van der Meulen, 2016).

Rodkin, Espelage, and Hanish (2015) observe that social structures contain members who have more power and prestige, and individual navigation within these structures can result in conflict. Individuals aiming to increase their position in their social system might use proactive forms of aggression to establish their social status, or to diminish the position of others in the hierarchy (Rodkin et al., 2015). However, these disputes invariably lead to environmental feedback, in which AB displayed by bullies might trigger an aggressive response from the victims (i.e., retaliation; Hanish et al., 2012). In this context, dynamics of aggressive/antisocial behaviours shape a profile that, over the years, is expected to result in internalising and externalising difficulties (Dodge, Coie, & Lynam, 2006). Academics working in the field of child and adolescent psychology have been interested in assessing what factors may put individuals at risk for bullying involvement (Arseneault, Milne, Taylor, Adams, Delgado, Caspi, & Moffitt, 2008; Singham et al., 2017). At the turn of the millennium, Sutton and collaborators set important questions for the study of a group of children who were 'hard' in their response to discipline, less responsive to anti–bullying policies and with elevated interpersonal abilities when compared to their victims (Sutton, Smith, & Swettenham, 1999a). Interestingly, this same group of researchers detected that, contrary to prior expectations where bullying was a result of deficiencies in social skills (Crick & Dodge, 1994), this group of children were competent in tasks measuring Theory of Mind (Sutton, Smith, & Swettenham, 1999a, 1999b). So, it is

likely that the common conception of bullies being socially unskilled is not the whole story.

Researchers have started to explore the trajectories of aggression, victimisation and CU traits as risk factors for adult psychopathy (Fontaine, McCrory, Boivin, Moffitt, & Viding, 2011; Fontaine et al., 2016; Free, 2017; Lynam & Gudonis, 2005). Fanti and Kimonis (2012) explored the role of conduct problems (CP) and CU traits in a longitudinal investigation, discovering that those with combination of both CP/CU+ showed greater initial intensity of bullying, and this pattern continued over time. In addition, youth who scored high on narcissistic traits were more involved with victimisation and showed more bully–related behaviour. In addition, high scores on the impulsivity subscale of the Antisocial Process Screening Device–Youth Version (APSD; Frick & Hare, 2001) were related to victimisation (i.e., being a victim). Recent meta–analysis results show that other important areas related to the broad concept of psychopathy, such as cognitive and affective empathy, are compromised amongst bully–victims in relation to non–involved counterparts (Zych, Ttofi, & Farrington, 2016).

Given the robust research findings, it is rather sensible to assume that children and adolescents who have been repeatedly exposed to the same risk factors for AB (e.g., biological vulnerabilities, early–life adversities, trauma) show increasing ratio for graduation into more severe forms of antisocial behaviour such as psychopathy (Barker, Arseneault, Brendgen, Fontaine, & Maughan, 2008; McCuish, Corrado, Hart, & DeLisi, 2015; Portnoy, Chen, & Raine, 2013). Moreover, one important element to detract from the consequences of childhood bullying into early adulthood is rather opposite to psychopathy, that is, the presence of prosocial behaviours (Ttofi, Farrington, & Lösel, 2014). A review of prospective longitudinal studies examined the impact of being a bully (bullying perpetration) and the experience of victimisation later in life, indicating that having adequate social skills, secure attachment to parents, and a circle of intimate friends could mitigate against the impact of these behaviours experienced at school and minimise the occurrence of internalising and externalising problems in adulthood (Ttofi, Bowes, Farrington, & Lösel, 2014). However, as noted by Hare (2001):

> "Many of the characteristics important for inhibiting antisocial and violent behaviour – empathy, close emotional bonds, fear of punishment, guilt – are lacking or seriously deficient in psychopaths. Moreover, their egocentricity, grandiosity, sense of entitlement, impulsivity, general lack of behavioural inhibitions, and need for power and control, constitute what might be described as the perfect prescription for asocial, antisocial, and criminal acts" (Hare, 2011, p. 11).

In summary, contemporary research has begun to explore joint trajectories of bullying and victimisation in explaining persistent AB and psychopathy. The existent literature still has many areas to unfold, such as the applicability of these findings to non–forensic samples, combined to a necessity to uncover the extent in which biological mechanisms could be underpinning these behaviours across different populations (Blair, 2010b; Giovazolias & Malikiosi–Loizos, 2015; Vlachou, Andreou, Botsoglou, & Didaskalou, 2011). Later in this thesis, Chapter Three is dedicated in understanding bullying dynamics in respect to psychopathic personality traits. The remainder of this chapter aims to provide an in–depth analysis of the construct of psychopathy, its variants or subtypes, with special attention to assessment and diagnosis tools. Factorial solutions – along with the core personality traits seen in individuals with high levels of psychopathic personality tendencies – will be discussed.

1.1.2 Psychopathy

Psychopathy is a term that encompasses a series of distinct profile of affect and behaviour, associated with numerous difficulties for individuals and wider society (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; Zimak, Suhr, & Bolinger, 2014). Elevated levels of psychopathic personality traits are closely linked to higher proness to externalising behaviours (Nelson & Foell, 2018), being associated with other relevant types of AB such as disinhibition (Patrick, 2018), poor behavioural control (Hare, 2001), and criminal offending and re–offending (Kiehl & Hoffman, 2011).

Those described as psychopaths intrigue us due to their presentation as being (traditionally) charming and manipulative, and as a consequence of their actions, often shocking or outside of our everyday lives. Cases of famous psychopaths are present since ancient times. DeLisi (2016) cites passages from the Bible in which features such as criminality, pathological lying and rebellious

behaviours are attached to deviant figures. In today's world, meticulous descriptions of those suspected to present with this personality inclination flood the media constantly, inspiring popular books, TV series, and movies (e.g., American Psycho, Misery, Fatal Attraction, the Silence of the Lambs, Trainspotting, We Need to Talk about Kevin). In the scientific realm, a pattern of interest and rigorous inquiry started with Hervey Cleckley in the 1940s, and has continued with the foremost intent of protecting individuals and the society from the acts carried out by psychopaths and those high on psychopathic (LeBreton, Binning, & Adorno, 2006), and related dark traits (Hodson, Hogg, & MacInnis, 2009; Miller & Lynam, 2003; Vize, Lynam, Collison, & Miller, 2018).

It is difficult to track precisely when the psychopathic–related behaviours started to be documented in the specialised medical literature. However, a general idea of the concept underwent more serious consideration during the 17th century, with the proposition of psychopathy as a manifestation of a mental disorder, which alluded to absurd, yet lucid behaviours (*manie sans delire* or moral insanity; Herpertz & Sass, 2000; Pinel, 1962; Skeem, Polaschek, Patrick, & Lilienfeld, 2011). In the early 20th century, a group of subjects who expressed antisocial tendencies began to be labelled – although in an unsystematic way – as sociopathic (Lykken, 2006)[2]. This approach was largely focused on an individual's irresponsibility, erratic (strange) lifestyle, deviance from societies' norms, unusual habits, absurd, and aggressive behaviours. As noted by Lykken (2006), little importance to personality factors as equally relevant in classifying the disorder was given in the early days of psychopathy research. A landmark work, "*The Mask of Sanity*" (Cleckley, 1941), highly influenced contemporary approaches to understand psychopathy. Cleckley's work (1941) described a profile of deficits in impulse control and aggression, but also of charm, grandiosity, and manipulation. More importantly, Cleckley was interested in the underlying aspects of the psychopathy personality, which culminated in him presenting 16 criteria for diagnosing psychopathy. These are displayed in the Table 1.1.

[2] Sociopathy and psychopathy were used interchangeably before Cleckley's work. Sociopathy also denoted that the individual presenting with antisocial behaviours was a product of the society (Vaillant, 1975).

Table 1.1: *Cleckley's criteria for Psychopathy*

Superficial charm	Shallow emotions
Absence of neurotic symptoms	Lack of insight and empathy
Lack of anxiety	Lack of gratitude
Irresponsibility	Absurd behaviour
Pathological lying	No history of genuine suicide attempts
Antisocial behaviour	Disintegrated sex life
Poor judgment and impairments to learn from experience	Failure in having a life plan
Inadequately motivated antisocial behaviour	Pathological egocentricity

Source. Cleckley (1941).

By using Cleckley's 16 item diagnosistic criteria, professionals were able to get a richer picture of the behaviours and other traits associated with psychopathy. The definition of 'psychopaths' included individuals who could not express genuine empathy, to feel guilt or shame and were, therefore, incapable of establishing healthy relationships (Black, 2015; Dadds, Hunter, Hawes, Frost, Vassallo, Bunn, & Merz, 2009; de Waal, 2008; Hare, 2003; Holmqvist, 2008). Added to this constellation of traits were the absence of neurotic symptoms, selfishness, parasitic lifestyle, and, quite often, psychopaths were believed to present with elevated to superior levels of intelligence (cf. Table 1.1; Cleckley, 1941). According to Cleckley, not all of these 16 characteristics needed to be present in an individual to meet the criteria for psychopathy. In Cleckley's clinical profile of the psychopath, presented within the third section of *The Mask of Sanity* (1941), the author argued that more often than not prototypical cases would present with many of his 16 criteria. Nonetheless, in some cases, an individual with firm diagnosis of psychopathy would not fully meet the absence of "nervousness" requirement, as well as could commit and/or attempt suicide.

It was Benjamin Karpman, in 1941, who first offered a distinction between psychopathic subtypes. For Karpman (1941), both primary and secondary psychopaths frequently have a similar behavioural outlook. However, emotional deficiencies were believed to have stronger biological influence for those with dominant primary (idiopathic) traits, whereas secondary psychopathy was understood as form of adaptation in front of early adversities, parental neglect, and abuse (Poythress & Skeem, 2006). Consequently, Karpman (1941) postulated that these secondary variants would result in higher levels of anxiety and other internalising disorders due to an inner neurotic conflict which, interestingly, would be amendable in the psychotherapeutic process.

Cleckley and Karpman were not the only scholars to dedicate significant effort into the realm of psychopathy. Building upon Karpman's seminal work on the distinction between primary and secondary variants, David Lykken (1957, 2006) integrated major contributions of Gray's (1982) theory of personality in his thesis that deficiencies in the Behavioural Inhibition and in the Behavioural Activation (BIS/BAS) systems could underpin specific aetiological mechanisms for psychopathic variants. According to this perspective, while primary psychopathy is marked by a low responsivity within the BIS, secondary psychopathy would be better characterised by a high reactivity within the BAS (Fowles, 1993; Poythress & Skeem, 2006). In accordance to Karpman's work, Porter (1996) likewise hypothesised that primary psychopaths are mostly under influence of biological determinants, while those seen as secondary psychopaths posses with an "acquired" form of the condition. Importantly, these authors (Karpman, 1941; Lykken, 2006; Porter, 1996) have formed a significant basis in which the concept of psychopathy started to be understood in the light of its various casual mechanisms. Skeem, Johansson, Andershed, Kerr, and Louden (2007) used model–based cluster analysis of the PCL–R (Hare, 2003) and measures of trait anxiety to examine Karpman's assertions on the distinctions between primary and secondary variants of psychopathy. As expected, secondary psychopaths showed greater trait anxiety and fewer total scores on the PCL–R. Moreover, these individuals had higher symptoms of borderline personality as well as higher levels of irritability, withdrawal, and poor assertiveness when compared to primary psychopaths.

Although a more extensive discussion on the various factor solutions for psychopathy will be presented shortly (see section 1.2), it is appropriate to remember that Harpur, Hare, and Hakstian (1989) were amongst the first scholars to emphasise the differential associations between psychopathic subdimensions and external variables when using the Hare's Psychopathy Checklist (PCL; Hare, 1980). The study reported on data from more than 1,000 males recruited from prisons and forensic institutions in North America, showing that antisocial features were more strongly linked to a profile labelled as "Chronically unstable and antisocial life–style; social deviance" (or PCL Factor 2; Hare, 1980), while narcissistic, charming features were more robustly associated with the PCL Factor 1, called "Selfish, callous, and remorseless use of others" (Harpur et al., 1989). A

meta–analysis including 95 studies and 15,826 subjects (M_{age} = 29.7 years) revealed a positive relationship between psychopathy and AB, including violent and non–violent offending, recidivism, and institutional infractions (Leistico, Salekin, DeCoster, & Rogers, 2008). Moreover, impulsive and antisocial behavioural facets of the psychopathic personality (Factor 2; Hare, 2003) had stronger relationships with AB (e.g., mean weighted *d* of .57) when compared to the affective/interpersonal facets (Factor 1; Hare, 2003), in which the observed mean weighted *d* was .40 (Leistico et al., 2008).

Developments in both theory and methodological approaches have been discussed over the past decades, and the conceptualisation of psychopathy has been refined, along with an understanding of associated risk factors, its costs and impacts to society, and aetiological mechanisms (Bijttebier & Decoene, 2009; Blair, 2010b; Zimak et al., 2014). With the publication of the third edition of the Diagnostic and Statistical Manual of Mental Disorders, a clear, distinct diagnostic category encompassing psychopathic traits was added into the Personality Disorders Cluster: The Antisocial Personality Disorder (APD) (American Psychiatric Association, 1980). This has been considered an important, but incomplete step towards an accurate diagnostic view of psychopathy (Brazil, van Dongen, Maes, Mars, & Baskin–Sommers, 2016).

To date, DSM has not, arguably, been suitable for diagnosing psychopathy, even though the initial intention was to account for its respective taxon (Vasey, Kotov, Frick, & Loney, 2005). A useful review on the construct of psychopathy in the DSM's five editions was recently published by Crego and Widiger (2015), who noted that hallmark characteristics – such as glibness and superficial charm, as well as lack of empathy – were excluded from the respective manual. This means that the DSM lacks assessment of affective and interpersonal traits of psychopathy, giving more emphasis on behavioural characteristics. The latest edition of the DSM (DSM–V; American Psychiatric Association, 2013) states that, to be diagnosed with APD, a person should exhibit the symptoms and characteristics described in Table 1.2.

Table 1.2: *Diagnostics criteria for Antisocial Personality Disorder (DSM–V)*

A. A pervasive pattern of disregard for and violation of the rights of others, occurring since the age of 15 years, as indicated by three (or more) of the following criteria:

1. Failure to conform to social norms with respect to lawful behaviours, as indicated by repeatedly performing acts that are grounds for arrest.
2. Deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure.
3. Impulsivity or failure to plan ahead.
4. Irritability and aggressiveness, as indicated by repeated physical fights or assaults.
5. Reckless disregard for safety of self or others.
6. Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behaviour or honour financial obligations. –
7. Lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another.

B. The individual is at least age 18 years.
C. There is evidence of conduct disorder with onset before the age of 15 years–old.
D. The occurrence of antisocial behaviour is not exclusively during the course of schizophrenia or bipolar disorder.

Source. Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013, p. 659).

The utility of classification manuals such as the DSM and ICD–10 may be important for the justice system and for offender management, but perhaps less useful for delivering treatment and for creating specific prevention strategies (Blair, 2010b; Ogloff & Lyon, 1998). Regardless of its limitations, which will not be discussed in depth in the current section, the DSM approach does not exclude an individual from presenting with APD and comorbid psychopathy (Basoglu et al., 2008; Crocker, Mueser, Drake, Clark, McHugo, Ackerson, & Alterman, 2005; Kosson, Lorenz, & Newman, 2006). The caution in using the DSM perspective could also be extended to the categorical view of psychopathy (Lynam, 2002). Criticism centres mostly on the notion that this approach is rather arbitrary, giving special emphasis on criminal behaviour and less focus on key personality traits (Hare, Hart, & Harpur, 1991; LeBreton et al., 2006). Nevertheless, the DSM emphasises, since its third edition, about the existence of an 'antisocial route', which usually begins with early disruptive behaviours and continues into adult life, either as a manifestation of antisocial and violent behaviours (1), as well as in terms of exhibition of shallow affect, disregard for others, and consistent exploitation of people (2) (American Psychiatric Association, 1980, 2013; see also Brazil et al., 2016).

Currently, psychopathy may be diagnosed in adults by using the Psychopathic Checklist Revised (PCL–R; Hare, 2003)[3] and its correspondent versions in youth, namely: The Antisocial Process Screening Device (APSD; Frick & Hare, 2001), which is suitable for children and adolescents aged between 6 and 13 years–old, and the Psychopathy Checklist: Youth Version, designed especially for use from early adolescence (~12 years–old) up to the age of 18 (PCL: YV; Forth, Kosson, & Hare, 2003). For more rapid assessments, and with purposes other than obtaining a diagnosis, professionals may use the Psychopathy Checklist: Screening Version (PCL:SV; Hart, Cox, & Hare, 2001), which was formulated based on items from previous PCL versions, and in accordance with relevant clinical literature (Hart et al., 2001).

Psychopathic personality traits and features, however, are a focus of various other measures, which do not propose to provide a clinical diagnosis, but rather examine the traits in a more dimensional way, being better suited to a general community, or non–criminal, sample (Benning, Patrick, Blonigen, Hicks, & Iacono, 2005; Benning, Patrick, Hicks, Blonigen, & Krueger, 2003). Amongst these, the revised Psychopathic Personality Inventory (PPI–R; Lilienfeld & Andrews, 1996) and its newly proposed shorter version examining only 40 items (Eisenbarth, Lilienfeld, & Yarkoni, 2015), the Self–Report Psychopathy scale–II (SRP–II; Forth, Brown, Hart, & Hare, 1996), the Self–Report Psychopathy scale–III (SRP–III; Williams, Nathanson, & Paulhus, 2003; Williams, Paulhus, & Hare, 2007), the Levenson Self–Report Psychopathy scale (LSRP; Levenson, Kiehl, & Fitzpatrick, 1995), and the Youth Psychopathic Traits Inventory (YPT; Andershed, Kerr, Stattin, & Levander, 2002) are widely used. Each of these measures has its strengths and limitations, and it is beyond the scope of this thesis to assess the performance of self–report measures of psychopathic traits.

[3] Each item is assessed in the range of 0 (does not apply at all) to 2 (item applies largely). The total possible score is 40. Cut–off points for diagnosing psychopathy vary, but on average an individual scoring equal or above 30 is believed to be a psychopath (Hare, 2003; but see also Skeem et al., 2011).

1.2 Factor models for psychopathy

1.2.1 A two–factor solution for psychopathy

One major contribution to the field has been made by Robert Hare (Hare, 2003; Hare & Neumann, 2006; Mullins–Nelson, Salekin, & Leistico, 2006). Hare's work resulted in the two–factor structure for psychopathy, including predominant deficits in four main areas (also called "facets"; Hare, 1993, 2001, 2003). Factor 1 corresponds to affective and interpersonal traits (e.g., superficial charm and lack of guilt), whereas Factor 2 accounts for behavioural characteristics (e.g., early behavioural problems and parasitic lifestyle). [4,5] The traits included in the two–factor solution of the Hare's Psychopathy Checklist Revised (PCL–R; Hare, 2003) were divided into four facets. The checklist measures aspects such as superficial charm (facet 1), poor behavioural control and problems in establishing relationships (facet 2), impulsivity (facet 3), and diversified criminal career (as opposed to 'speciality' in one crime; facet 4). The first two facets are believed to correspond to the core traits (or Factor 1), with best discriminant utility in differentiating psychopathy from other personality and developmental disorders (Glenn & Raine, 2014). Facets belonging to Factor 2 (e.g., lifestyle and antisocial) are sometimes labelled as components of 'secondary psychopathy', being uniquely associated to difficulties not always shared by those possessing with dominant 'primary' traits (e.g., internalising problems; Vassileva, Kosson, Abramowitz, & Conrod, 2005). The subdivision into factors was a result of intensive research, albeit more applied settings still attribute psychopathy to prototypical cases (e.g., Factor 1) (see Skeem et al., 2011, for a comprehensive review on this respect). A more in–depth discussion on these diagnostic controversies is beyond the reach of this section, but it is important to advise the reader about the existence of divergent points of view about traits that are indispensable for considering

[4] It is important to distinguish that Hare's work does not include all the 16 criteria coined by Cleckley, but these are still important for characterisation of the construct, especially in terms of the descriptions corresponding to facets 1 (interpersonal) and 2 (affective) (Baskin–Sommers, 2017).

[5] Although three and four–factor models of psychopathy have also been proposed (Johansson, Andershed, Kerr, & Levander, 2002; Weaver, Meyer, Van Nort, & Tristan, 2006), this thesis considers the two–factor approach as it is well–documented in the literature, and it seems to represent the most common phenotype seen (Fowles & Dindo, 2006). However, in the Section 1.3.2 other factorial solutions of the PCL–R will be described.

someone as a psychopath (Karpman, 1941; Lee & Salekin, 2010). Table 1.3 assembles Hare's criteria for the construct.

Table 1.3: *Hare's Psychopathy criteria (PCL–R)*

Factor 1	Facet 1 (interpersonal)	1 Glibness/superficial charm 2. Grandiose sense of self–worth 4. Pathological lying 5. Conning/manipulative
	Facet 2 (affective)	6. Lack of remorse or guilt 7. Shallow affect 8. Callous/lack of empathy 16. Failure to accept responsibility
Factor 2	Facet 3 (lifestyle)	3. Need for stimulation/proneness to boredom 9. Parasitic lifestyle 13. Lack of realistic, long–term goals 14. Impulsivity 15. Irresponsibility
	Facet 4 (antisocial)	10. Poor behavioural control 12. Early behavioural problems 17. Many short–term marital relationships 18. Juvenile delinquency 19. Revocation of conditional release
Extra items		11. Promiscuous sexual behaviour * 2. Criminal versatility *

Source. Hare (2003). *Note.* Asterisk denotes items that do not load into any of the PCL–R factors.

Even though some of the criteria for psychopathy included under Hare's fourth facet have clear links with APD, it is important to emphasise that psychopathy is not the same as APD and is also distinct from sociopathy (Crocker et al., 2005; Hare, 2003; Kosson et al., 2006; Pemment, 2013; Shnaidman, 2016). This distinction is suitable (and necessary) for both the context of the current thesis and to the broad psychopathy/biosocial criminology specialised literatures (Dolan & Coid, 1993; Hare et al., 1991).

1.2.1.1 Interpersonal and affective facets

The differences in affect processing and difficulties with interpersonal relationships help to shape psychopathy as unique within the psychiatric literature, adding specificity in the diagnostic process (Hare, 1993). These two facets contain deficits associated with the construct that are perhaps amongst the most difficult to understand (cf. Table 1.3). It is arguably difficult for neurotypical individuals to empathise with a group of people who appear to have a deficit in

their emotional empathy, which manifests as a lesser, or lack of, emotional responsiveness commensurate with another's emotional state (Eisenberg & Strayer, 1987; Hare, 2003).

The deficits in terms of affective understanding and processing in psychopathy are illustrated in Table 1.3, where empathy impairments receive great attention. Within this facet is also included the characteristic feature of lack of guilt, highly associated with the concept of empathy (Roberts, Strayer, & Denham, 2014). The centrality of empathy for the construct of psychopathy is unquestionable, being a central element to define the condition through history. Cleckley (1941, p. 350), when referring to a prototypical case of psychopathy, commented that: "he has no ability to know how others feel… or to feel subjectively anything comparable about the situation". Including processes such as emotional contagion, identification with others, understanding, prosocial attitudes and perspective taking, empathy is essential for moral development (de Vignemont & Singer, 2006; Eisenberg & Strayer, 1987).

For the context of this literature review, important is the notion that there are many different definitions of empathy as there are views regarding how it develops (Eisenberg & Strayer, 1987; McDonald & Messinger, 2011). Usually, empathy can be divided into two components: cognitive and affective (Anastácio, Vagos, Nobre–Lima, Rijo, & Jolliffe, 2016; Blair, 2005, 2006; Jolliffe & Farrington, 2006). While affective empathy works as a sense of experiencing 'what it feels like' for another individual and allows humans to share emotions, cognitive empathy permits an individual to comprehend what thoughts another person may have on a given moment (Singer, 2006). It is believed that affective empathy expresses more pronouncedly from early childhood, concomitant to the emergence of early dysfunctional traits seen in psychopathy, while the cognitive form usually becomes sophisticated throughout early adolescence (Eisenberg, Cumberland, Guthrie, Murphy, & Shepard, 2005). Warren (2009) reported that empathy scores were significant in partially mediating the relationship between psychopathy and indirect aggression for male and female participants; moreover, affective empathy partially mediated the psychopathy–indirect aggression association for both sexes. However, affective empathy fully mediated the relationship between psychopathic personality traits and indirect aggression for males, suggesting that psychopathy–

related use of this form of aggression was completely due to deficiencies within the affective component of empathy.

Blair, Mitchell, and Blair (2005) proposed the Integrated Emotion Systems (IES) model in which deficiencies in empathy have specific biological causing mechanisms. For the IES (Figure 1.1), dysfunctions within the amygdala are emphasised, receiving much attention of scholars interested in biological mechanisms underpinning psychopathy. As it is unlikely that a single brain region is responsible for the profile of traits and behaviours seen in psychopathy, a model that considers the connectivity between different areas is probable more useful to clarify the brain–based biological influences on psychopathy (Blair, 2010a; da Cunha–Bang et al., 2017; DeLisi, 2016; Glenn, Raine, Yaralian, & Yang, 2010; Korponay et al., 2017, Seara–Cardoso & Viding, 2015).

As shown in Figure 1.1, the arrows marked with the number 1 represent the transfer of sensory input from the sensory cortex (SC) to basolateral (BLA) and central nuclei (CeA) of the amygdala. Arrow 2 illustrates the projections to the brainstem, and arrows with a number 3 comprise the exchange of information to ventromedial frontal cortex (vmFC) and insula. An arrow from the vmFC to the motor cortex (MC), important to generate a motor response, is also exhibited (Figure 1.1). Conditioned stimulus (CS) and unconditioned response (UR) are depicted, thus creating the CS–UR association at "*a*" (CeA), whereas a CS–affect representation occurs at "*b*" (BLA) (Blair, 2006).

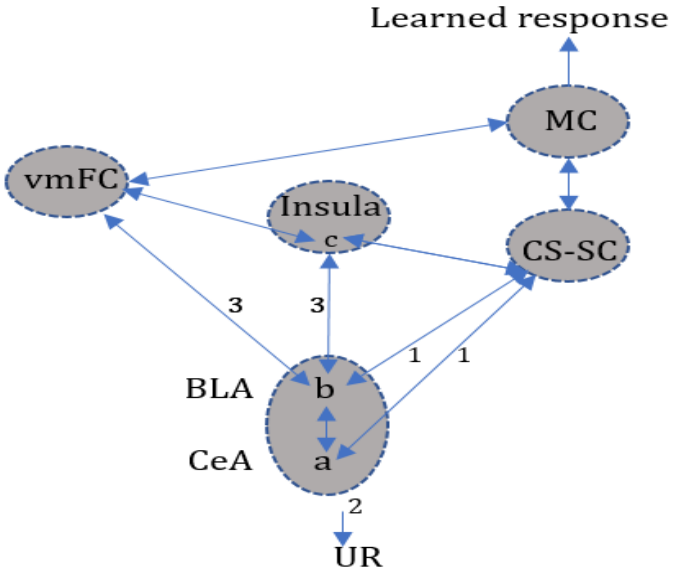

Figure 1.1 The Integrated Emotions System Model (Adapted from Blair, 2006).

According to Baskin–Sommers and Newman (2012), accumulating evidence corroborates the propositions of the IS model, but what is interesting is that psychopaths, as well as those with higher levels of psychopathic traits, can display either hyper– or hypo– reactivity to emotional stimuli within the amygdala (Glenn, Raine, & Schug, 2009; Müller et al., 2003). These results are supportive of brain dysregulations and deficient connectivity, suggesting that divergences in functional activity could reflect white–matter microstructural abnormalities situated in tracts connecting prefrontal regions to the amygdala (Blair, 2010b; Müller et al., 2003; Waller, Dotterer, Murray, Maxwell, & Hyde, 2017a).

There is empirical support to the negative association between affective empathy to psychopathic traits. Evidence for this is available in both adults (Mullins–Nelson et al., 2006; Oliver, Neufeld, Dziobek, & Mitchell, 2016) and child/adolescent research (Jones, Happé, Gilbert, Burnett, & Viding, 2010; Lethbridge, Richardson, Reidy, & Taroyan, 2017; Lui, Barry, & Sacco, 2016), which complement extensive clinical material indicating to a clear profile of empathy deficit – particularly affective empathy – in psychopathy (Cleckley, 1941; Hare, 2001, 2003). In addition to difficulties in understanding and responding to others' emotions, it is well established that psychopaths have difficulties in understanding and attributing emotional valence to their own experiences (Louth, Hare, & Linden, 1998). Problems in describing and identifying feelings are important deficits that co–occur in psychopathy and other 'disorders of empathy', such as alexithymia (Takamatsu & Takai, 2017) and autism (Bird & Viding, 2014). The construct of alexithymia encompasses an inadequacy in experiencing and expressing emotions (Apfel & Sifneos, 1979), and its features include difficulty in labelling feelings, deficits in emotional consciousness, and poor imagination (Takamatsu & Takai, 2017; Wiethaeuper, Balbinotti, Pelisoli, & Barbosa, 2005). Studies have indicated that there is a link between alexithymia and limited empathy (Bird, Silani, Brindley, White, Frith, & Singer, 2010; Grynberg, Luminet, Corneille, Grezes, & Berthoz, 2010; Guttman & Laporte, 2002). Unsurprisingly, correlational studies have linked alexithymia to psychopathy (Lander, Lutz–Zois, Rye, & Goodnight, 2012; Louth et al., 1998), and also psychopathy to low empathy (Dadds et al., 2009). For that reason, elevated traits of alexithymia or the condition per se might play a significant role in dissecting and differentiating how individuals process and

describe their internal states, which would influence affect and behaviours (Sifneos, 1973). In this thesis, an exploration of the predictive value of those deficits in understanding the links between psychopathy and behavioural stress is presented in Chapter Five.

1.2.1.1.1 Developmental considerations relevant to psychopathy

Within the most recent iteration of the DSM–V (American Psychiatric Association, 2013), the definition of Conduct Disorder has been developed to include a specifier to this diagnosis for those who also present with 'limited prosocial emotions', such as reduced empathy and lack of guilt expression. These characteristics clearly overlap with the concept of CU traits, which have been linked to numerous problems comparable with those identified in adult psychopathy (Fontaine et al., 2016; Frick, 2004; Frick, Ray, Thorton, & Kahn, 2014; Kimonis et al., 2008; Lahey, 2014; Moran, Rowe, Flach, Briskman, Ford, Maughan, & Goodman, 2009).

The term 'CU traits' refers to a distinct construct of antisocial behaviour, which is characterised by the absence of emotional responsiveness (e.g., lack of anxiety, deficient empathic response and compromised manifestation of remorse), as well as by persistent, negative acts intended to harm others (Feilhauer, Cima, & Arntz, 2012; Frick, Cornell, Barry, Bodin, & Dane, 2003; Pardini, Lochman, & Frick, 2003). These traits were first considered as a downward extension of psychopathic traits in adults by Paul Frick and colleagues (Frick, O'Brien, Wootton, & McBurnett, 1994), which proposed that shallowness of affect, low to no sense of guilt, deficiencies within affective empathy, and chronic dysfunctional behaviour are key indicators for CU traits. Investigations have demonstrated severe emotional and behavioural difficulties amongst those possessing elevated early psychopathic traits, and there is an emerging picture of a clear cognitive–affective profile that includes clear impairment in the ability to recognise fear stimuli (De Brito et al., 2009; Frick et al., 2003; Jones, Laurens, Herba, Barker, & Viding, 2009; Kotler & McMahon, 2010; Viding, Fontaine, & McCrory, 2012; Viding, Simmonds, Petrides, & Frederickson, 2009).

Not every child with CU traits will show more severe forms of aggression, but elevated CU traits appear to index a specific risk factor for these outcomes (Frick & Viding, 2009). As shown in past research, these individuals have

difficulties in detecting emotions such as fear and sadness, and self–report behavioural data of being themselves fearlessness add to the equation (Klingzell, Fanti, Colins, Frogner, Andershed, & Andershed, 2016; Pardini, Lochman, & Powell, 2007; see also Pisano et al., 2017). These children and adolescents do not appear to respond to typically efforts at socialisation from parents and others in their lives, and do not adequately develop prosocial behaviours because they have difficulties in responding to punishment and distress in others, which may result in deficits in developing components of empathy (Frick & Viding, 2009; Waller, Shaw, & Hyde, 2017b; White & Frick 2010). On the other hand, those children considered CP/CU– do not typically present with proactive aggression. These individuals (with a 'hot type' behavioural profile) show more reactive aggression and their phenotype often displays high levels of anxiety and the presence of remorse is not rare (Frick & Viding, 2009).

As a likely result of the emotional deficits seen in psychopathy even at early stages of human development, individuals can engage in offensive and violent behaviours due to a combination of emotional detachment with a need for stimulation (Dadds et al., 2012; Frick & White, 2008; Herpertz & Sass, 2000). In other words, the 'core' traits attributed to Factor 1 might facilitate the development of problems encompassed within Factor 2 (Gonzalez–Tapia, Obsuth, & Heeds, 2017). Consequently, the next section will consider the lifestyle and antisocial facets of psychopathy. Deficits in these two facets are assumed to be highly correlated with emotional and behavioural difficulties that this thesis aims to explore, hence the adequacy of discussing them within the introductory chapter.

1.2.1.2 Lifestyle and antisocial facets

Even nowadays, the figure of a psychopath is frequently associated with images of serial killers, violent and dangerous individuals, and cannibals, amongst other extreme examples. However, these characteristics alone do not totally encompass the current view of the core components of this form of personality disturbance (Reid, 2017). Although physical violence might be displayed by psychopaths, it is often the case that the 'weapons' chosen by them are more discrete. In some cases,

psychopaths appear perfectly sane. In other words, they are able to deceive or con people and society by wearing a *mask* (Cleckley, 1941).

Diagnostic tools include poor behavioural control, early disruptive problems and juvenile delinquency as potential indicators of psychopathy. These criteria have been comprised into Hare's fourth facet of the PCL–R, and are expected to load within Factor 2 (Hare, 2001, 2003). This same factor includes characteristics such as a necessity for stimulation and proneness to boredom, parasitic lifestyle, problems in establishing long–term goals, as well as impulsivity and irresponsible behaviours (e.g., third facet) (cf. Table 1.3; Hare, 2001, 2003).

It is within Factor 2 that the main violent and aggressive attitudes displayed by those high in psychopathic tendencies are inserted. Hare and Neumann (2006) remembered that some researchers suggested that Cleckley and others have described psychopathy predominantly in terms of personality features, without reference to antisocial aspects. In consequence of this, a view of 'less importance' to antisociality was incorporated into psychopathy assessment. For instance, Glenn and Raine (2014) affirmed that: "the crux of psychopathy is not the display of antisocial behaviour, *per se*, but rather the distinctive personality traits, including emotional deficits that characterise these individuals" (p. 3) are most normally known for.

However, Hare and Neumann (2006) hold the position that "many of the key personality traits considered most relevant to psychopathy are themselves inferred from behaviours that are antisocial, asocial, or otherwise harmful to others" (p. 60). Additionally, broad psychopathological elements of impulsivity and antisocial behaviours have expressive genetic overlap with externalising psychopathic traits (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005; Hare & Neumann, 2006). Certainly, the discussion about whether antisocial features are essential for characterising prototypical psychopaths is important, especially when considering the criminal versatility that psychopathy is assumed to possess (Hemphill, Templeman, Wong, & Hare, 1998; Nelson & Foell, 2018; Ruchensky, Edens, Corker, Donnellan, Witt, & Blonigen, 2017). It is beyond the scope of the current thesis to investigate participants' involvement with crime and severe delinquency. However, when compared to non–psychopaths, those presenting

with clinically significant levels of psychopathic traits engage with crimes at proportions as high as three times the average estimates for the general population; additionally, psychopathy is related to incremented gravidity in recidivism (Asscher, van Vugt, Stams, Deković, Eichelsheim, & Yousfi, 2011; Baskin–Sommers, 2017; Hemphill et al., 1998).

The motives for engagement with crime and aggression amongst those presenting with elevated psychopathic tendencies have resulted in distinct, but complementary theories. For instance, one proposition is that individuals scoring high in psychopathic traits might undertake aggressive behaviours for sensation seeking purposes, overcoming an inherited unpleasant state that they presumably have (Hansel, Johnsen, Thornton, Waage, & Thayer, 2007). An alternative model hypothesises that psychopathy is marked by the absence of fear, which then would explain antisocial behaviours (Patrick, 2001). Propositions also suggest that psychopaths have impaired modulation of responses, which manifests in difficulties in adjusting antisocial behaviours when there is a dominant response pattern in operation, usually towards rewarding stimuli (Newman, Patterson, & Kosson, 1987; see also Smith & Lilienfeld, 2015). Early onset of these dysfunctional biobehavioural dispositions – diminished fear, inherited under aroused ANS and impaired ability to adjust behaviours towards prosocially – can lead to greater involvement with more severe types of antisocial behaviour, which stresses the necessity for accurate understanding into aetiological issues (Hemphill, Hare, & Wong, 1998; McCuish et al., 2015; McCuish et al., 2014; Wendt, Jones, & Arteche, 2017).

1.2.2 Three and four–factor solutions for psychopathy

Although the PCL was originally created containing two–factors, further developments in the measure have been proposed (Hare, 1980; Hare, Neumann, & Mokros, 2018). The facets of the PCL–R (Hare, 2003) described in the previous sections are sometimes organised in distinct factor solutions (Cooke, Michie, & Hart, 2006; Krstic, Neumann, Roy, Robertson, Knight, & Hare, 2017; Neumann, Vitacco, & Mokros, 2016), as it will be shown in the following two sections.

1.2.2.1 A three–factor solution for psychopathy

A three–factor solution has been proposed based on the justification that only 13 out of the 20 PCL–R items are non–redundant (Cooke & Michie, 2001). These three factors, represented in a hierarchical structure with a superordinate construct (i.e., psychopathy), were named as follows: Arrogant and deceitful interpersonal style (comprising the items 1 to 4), deficient affective experience (comprising the items 6 to 8, and 16), and impulsive/irresponsible behavioural style (comprising the items 10, 12, and 17 to 19). Accordingly, these are exactly the same items from Hare's interpersonal, affective, and lifestyle facets (Cooke et al., 2006).

Noticeably, the three–factor solution does not include the fourth facet (e.g., antisocial), as well as excludes the two PCL–R extra items of promiscuous sexual behaviour and criminal versatility. Cooke and his collaborators defend that AB are a consequence of psychopathic traits and not an integral part of the construct, which would justify the decision of excluding items 10 to 12, and 17 to 20 (Cooke et al., 2006). There has been some replication of this factor solution in both adolescent and adult–based studies (Cooke, Michie, Hart, & Clark, 2004; Skeem, Mulvey, & Grisso, 2003), but some concern exists around the view that AB are just a consequence of psychopath's "core" affective traits (Hare & Neumann, 2005, 2010). Moreover, methodological aspects have also raised the possibility that a three–factor solution might not represent well the construct of psychopathy (Hare & Neumann, 2010). In sum, this proposition warrants further research, which might clarify its suitability for use in samples across various contexts (Warren, 2009).

1.2.2.2 A four–factor solution for psychopathy

In 2003, Hare proposed that his original plan for a four–facets, two–factor solution for psychopathy could likewise be represented by a four–factor structure. Hare and Neumann (2008) reported excellent fit indices in the validation studies conducted with adolescents and adults (values for the Tucker Lewis index were .97 and .94, respectively, and the standardized root mean values were .05 for both samples).

The four–factor structure has shown suitability for use in different populations and by using distinct statistical procedures to assess its adequacy. For example, in a study using both latent variable (e.g., structural equation modeling; SEM) and person–centred approaches (e.g., latent profile analysis; LPA) to explore the evidence for a four–factor model in a sample comprised of male sex offenders, Krstic et al. (2017) confirmed the adequate psychometric properties of the PCL–R in this population. Precisely, the 4 factors (e.g.; interpersonal [items 1 to 5], affective [6 to 8, and 16], lifestyle [3, 9, and 13 to 15], and antisocial [10, 12, and 18 to 20]) yielded the following fit parameters: comparative fit index of .90, and a root mean square error of approximation of .06 (traditional confirmatory factor analysis). LPA analyses supported a four–class solution (classification accuracy = .83 to .91; Bayesian Information Criterion = 15774.83) comprising the following groups: prototypic, callous/conning, sociopathic, and general offender. A recent validation of a four–factor proposition for the PCL–R was made available in a study with adult female offenders (Eisenbarth, Krammer, Edwards, Kiehl, & Neumann, 2018). Confirmatory factor analyses, conducted with mean– and variance–adjusted test statistics and using a diagonally weighted least squares estimator with robust standard errors, revealed a comparative fit index of .92 and a root mean square error of approximation of .05 (Eisenbarth et al., 2018).

1.2.3 A summary on psychopathic factorial solutions

There are competing factor solutions for the current dominant tool for diagnosing psychopathy in adults: the PCL–R (Hare, 2003). While the three–factor solution argues that items assessing antisocial tendencies are not an integral part of the construct, the two– and four–factor solutions take into consideration items belonging to the Hare's antisocial facet/factor.

Although there are other specificities regarding factor solutions for psychopathy not covered in the previous sections, it seems reasonable to add that variables beyond statistics and mathematics might as well influence in the decision of choosing certain facets/factors to assess psychopathic traits in different populations. My work follows a narrative which understands that psychopathic personality traits are fundamentally antisocial, notwithstanding the fact that a

psychopathic outlook is not limited to AB (Hare, 2011). As such, the characterological assumptions of both the two– and four–factor solutions (Hare, 2003) guided the selection of self–report measures used in this thesis. The two–factor solution for the PCL–R has shown differential associations between its factors with self–report measures that aim to assess psychopathic personality traits in the general population. Poythress et al. (2010) reported a significant correlation of .25 and .17 between Hare's PCL–R Factors 1 and 2 with PPI Factors 1 and 2 (Lilienfeld & Andrews, 1996), respectively. As for the Levenson's measure (Levenson et al., 1995), the study showed significant associations between Factor 1 with primary (r = .23) and secondary psychopathy (r = .06), and between Factor 2 with primary and secondary psychopathy (r's = .29) (Poythress et al., 2010). Indeed, the differential link between Hare's Factor 1 and Levenson's primary profile is well–documented (cf. Poythress & Skeem, 2006).

Similarly, some scholars noted that the factor structure of the PPI (Lilienfeld & Andrews, 1996) might roughly resembles the two–factor model for psychopathy captured by the PCL–R (Benning et al., 2003, 2005; Eisenbarth et al., 2018). Moreover, early report has linked psychopathic behaviours assessed by the PPI (e.g., Rebellious Nonconformity, Blame Externalisation, and Machiavellian Egocentricity) to direct and indirect aggression amongst adults (Warren, 2009), which strongly encouraged the examination of the association of these traits with bullying behaviours in adults. In addition of having some correspondence to the most widely used diagnostic tool for psychopathy, self–report measures could also posit some advantages. For instance, the use of screening tools might assist in uncovering psychopathic features that are underrepresented in the items that constitute the PCL–R (Hare, 2003; Verschuere et al., 2018). Moreover, studies combining two or more well–validated tools can reduce the risks of biases due to mono–measurement (cf. Skeem & Cooke, 2010).

1.3 Incidence of psychopathy and subclinical psychopathy

Most community–based studies estimate that 1 in 100 individuals are expected to display significant levels of psychopathy, with a higher prevalence in clinical settings (Barnes, 2014; Herpertz & Sass, 2000). The most recent report of the

prevalence of psychopathy in England, Wales and Scotland was .6% (95% CI: .2–1.6; Coid, Yang, Ullrich, Roberts, & Hare, 2009). Statistics for psychopathy incidence are extremely hard to estimate because many individuals displaying elevated interpersonal and emotional traits (e.g., Factor 1) may avoid the legal system, and are, to some extent, 'successful' (Gao & Raine, 2010). Also, data on the incidence of psychopathy is affected by its form of diagnosis, which is relatively costly and time consuming (Barnes, 2014). In American and British correctional settings, 64 to 100% of prisoners meet the American Psychiatric Association criteria for Antisocial Personality Disorder, while for psychopathy the numbers are significantly smaller, but by no means irrelevant (Blackburn & Coid, 1999; Herpertz & Sass, 2000). As commented by Hare (2001), the impact of psychopaths – who are believed to be one in every hundred individuals, yet corresponding to 25% of the incarcerated population – can be very large, which might encourage efforts in terms of prevention and intervention methodologies.

While some prefer a categorical approach to differentiate psychopaths from non–psychopathic criminals, others advocate for a dimensional approach where the psychopathic personality is understood as a continuum of adaptive and maladaptive traits and behaviours (Corr, 2010; Hoppenbrouwers, Bulten, & Brazil, 2016). Even though much of the research on psychopathic individuals has been conducted in forensic settings, there is increasing interest in studying individuals with higher levels of psychopathic traits in the general population (Hodson et al., 2009; LeBreton et al., 2006).

So–called 'subclinical psychopaths' do not differ from prototypical cases based on their types of behaviour and affect. Instead, differences are rather quantitative than qualitative. Therefore, subclinical psychopathy is relatively more common than clinical psychopathy, with estimates around 5 to 15% in the general population (LeBreton et al., 2006). The importance of studying sub–clinical/non–criminal psychopaths and their correspondent associated variables has gained attention over the last decade, mainly because of the amount of symptom variability and recent findings regarding different routes in which someone might progress into further, stable stages of severe psychopathy (Moreira, Almeida,

Pinto, & Fávero, 2014; Pasion, Fernandes, Pereira, & Barbosa, 2017; Skeem et al., 2011).

Wilson, Abramowitz, Vasilev, Bozgunov and Vassileva (2014, p. 4) made an important argument stating that "establishing the validity of psychopathy in community samples is also a necessary step towards utilizing the construct in public health research" internationally. Adequate management of psychopathy is imperative, since its social and economic impacts on society are massive, yet not fully known[6] (Cleckley, 1941; Hare, 2003; Kiehl & Hoffman, 2011). Consequently, in this thesis, when referring to psychopathy, I will be considering the phenomenon in its broadest terms of psychopathic personality traits, rather than in a diagnostic fashion or language. In addition, this thesis adopts the subtypes of primary and secondary variants, which overlap with characterisations of psychopathic Factors 1 and 2, respectively (Poythress & Skeem, 2006). However, it seems premature to state that primary psychopathy is equal to the Factor 1 profile (Patrick, Venables, & Skeem, 2012; Skeem et al., 2011).

1.4 Chapter summary

In this chapter, a summary of research of developmental issues relevant to the study of AB has been presented, integrating theories with the intent of better capturing mechanisms that would explain aggressive and bullying behaviours in the context of high levels of psychopathic personality traits. Psychopathy is likely to be best understood as a collection of personality traits and antisocial behaviours, so this chapter also explored earlier distinctions and propositions for psychopathic subtypes, examining how research has led to current conceptualisations of the construct. Additional relevant themes for studying psychopathy were introduced, such as its forms of clinical diagnosis (along with factor propositions and descriptions of important associated deficits) and its assessment in the general population. Finally, evidence on the prevalence of psychopathy and subclinical psychopathy was discussed. One next step in

[6] Estimates for the total cost attributed to psychopathy are nearly impossible to obtain. However, a proxy of U$$ 460 trillion was estimated in 2009 within the US. This amount includes average indirect costs (police, courts, and prison), but do not account for expenses in psychiatric institutions, treatment for victims, and unknown fraud (Kiehl & Hoffman, 2011).

deepening the effort in understanding psychopathy implicates the exam of its core biological vulnerabilities, which will be accomplished in Chapter Two.

CHAPTER TWO – BIOLOGICAL MECHANISMS IN THE SCIENTIFIC STUDY OF PSYCHOPATHIC PERSONALITY TRAITS, AGGRESSIVE, AND ANTISOCIAL BEHAVIOURS

Chapter overview

The previous chapter presented an overview of developmental factors relevant to understand distinct types of AB, exploring some of the core deficits in affect and behaviour commonly seen in the manifestation of psychopathy. However, it is important to those interested in the aetiology and treatment of these difficulties to understand the relevant potential biological underpinnings. Consequently, the purpose of the current chapter is threefold: (i) to discuss methodology relevant to how psychological science can infer its constructs from physiological indicators; (ii) to examine possible biological mechanisms that might underpin psychopathy and AB, with emphasis on psychophysiological studies; and (iii) to provide an overview of the thesis in terms of its aims and structure.

To accomplish the first goal of this chapter, the next section discusses how psychological constructs can be obtained from physiological indicators, including descriptions of widely–used methods for assessing the ANS. It is believed that these considerations might assist in the interpretation of past psychophysiological studies discussed subsequently (Section 2.3), being equally relevant for contextualising the experiments conducted for this thesis (Chapter Four).

2.1 Inferring psychological constructs from physiological responses

Psychophysiological measures, when taken continuously in the context of an experiment, allow researchers to infer on participants underlying motivations and internal states without having to rely on external questionnaires (Edgar, Keller, Heller, & Miller, 2007). As shown in the Bechara, Damasio, Tranel and Damasios's study (1997), changes in the pattern of physiological responses can also be predictive of conscious awareness. In other words, the body can reveal answers that individuals might not normally disclose themselves, or even states that

individuals are not even aware of. Bodily responses, as such, are clues to emotional states. These procedures also add value in the intent of indexing behaviours without participants' ability of controlling them (e.g., social desirability bias; Blascovich, Vanman, Mendes, & Dickerson, 2011). It is in this context that psychophysiology is introduced, a discipline dedicated in interpreting various biological mechanisms that result in meaningful psychological information. To comprehend how this field has evolved, a brief overview of past empirical and theoretical work – which have culminated in today's most used methods – is of great importance.

2.1.1 Psychophysiological inference: An evolving framework

Research into the physiological correspondences of human emotions and behaviours begun with William James, who affirmed in 1884, that specific emotions experienced by individuals were a direct result of physiological processes. The decades after James's work have been marked by a flourishing of methods across many disciplines, and major advancements in the understanding of the "psycho–physiology" connection were possible (Cannon, 1927; Norman, Berntson, & Cacioppo, 2014; Schachter & Singer, 1962).

The early days of psychophysiological research witnessed innumerable obstacles. Difficulties in comparing the magnitude of emotional experiences – combined with poor synchronisation between physiological apparatus to accurately detect the commencement and the extinction of certain emotions – were amongst the biggest challenges (Norman et al., 2014). In this sense, Ekman, Levenson, and Friesen (1983) hypothesised that undifferentiated responses could be translated into more meaningful emotional experiences when in conjunction with additional physiological indicators. This has been exemplified in their proposition that anger would be more appropriately defined by increase in both HR and skin temperature, while fear would be marked by increased HR but reduced skin temperature (Ekman et al., 1983).

A seminal paper, published in 1990 by Cacioppo and Tassinary, established new avenues for conducting psychophysiological research. In the publication, the

authors introduced what was called the 'identity thesis', which assumes that there are physiological concomitants of psychological phenomena. However, this proposition does not imply that these concomitants will be *one–to–one* in all situations. In other words, researchers should not expect that a single physiological process could be responsible for producing a certain psychological response, or in the other hand, that one psychological event would be linked to one isolated physiological process. Moreover, the identity thesis postulates that not every physiological process necessarily has significance in the psychological domain. As such, individual differences, contextual influences, and the effect of time would be important factors in the specification of a "physiological–psychological" relation (Cacioppo & Tassinary, 1990; Norman et al., 2014).

In contrast, every psychological event has its origins in the physiological level (Cacioppo & Tassinary, 1990). The authors suggested that a *null relation* between one physiological indicator with a certain psychological event supports the exclusion of a physiological variable in a further attempt of identifying biological concomitants of the given psychological event. A subsequent relation that might occur was called *one–to–one*, in which a unique element from the psychological domain was linked to a single element in the physiological level, and vice versa (Cacioppo & Tassinary, 1990). Hence, *one–to–many* relationships occur when one psychological indicator has two or more correspondents in the physiological level (Cacioppo & Tassinary, 1990). These types of relations can be presented in a much simpler fashion way by transforming them into a *one–to–one* relation (Cacioppo & Tassinary, 1990). This is attained by creating a new group of physiological elements to characterise a response pattern in face of a manipulation in the psychological domain. The example already given of Ekman et al.'s (1983) attempt to specify a pattern of physiological changes in HR and in skin temperature to characterise fear could roughly be used as a tentative of combining physiological concomitants and/or physiological outcomes of a psychological event into one new variable (Cacioppo & Tassinary, 1990). The remainder relations described by Cacioppo and Tassinary (1990) are *many–to–one* and *many–to–many*, in which two or more psychological elements are related to one physiological indicator, and when various psychological variables are linked to two or more indicators from the physiological domain, respectively. Both *many–to–one*

and *many–to–many* relations impede the establishment of conclusive patterns of psychological events in face of physiological changes (Cacioppo & Tassinary, 1990). In addition, when *one–to–one* relationships occur solely under certain circumstances (e.g., context–limited), the physiological indicator receives the status of *marker* of the psychological phenomena; but if *one–to–one* relationships have greater generalisation, the suggested term is *invariant* (Beauchaine, 2009; Richter & Slade, 2017). Two others widely used terms in psychophysiological research that were explained by Cacioppo and Tassinary (1990) include *outcomes* and *concomitants. Outcomes* are *one–to–many* relationships between a physiological indicator and psychological events in a limited context, and *concomitants* are *one–to–many* relationships between a physiological indicator and psychological events in various contexts (Richter & Slade, 2017). Importantly, psychophysiological interpretation is reinforced when a *one–to–one* link exists, notwithstanding the fact that not every psychophysiological link could be reduced to a *one–to–one* relation; nevertheless, researchers can progressively break down physiological changes over time, thus creating a new set of indicators (Cacioppo & Tassinary, 1990).

More recently, Richter and Slade (2017) summarised the conditions that must be fulfilled for detecting psychophysiological invariants, markers, concomitants, and outcomes. The authors noted that physiological responses must be specific to extent that they do not alter in face of other psychological event, being equally stable in all contexts. This is very difficult to accomplish in the light of such a diverse existence of contexts, and so researchers might never accomplish this task. In respect to markers, it is necessary to validate any potential physiological measure so it can be demonstrated to be responsive to changes in the correspondent psychological construct in a specific context, providing also that other psychological phenomena have no effect on the marker in question (Richter & Slade, 2017). As for concomitants, these are defined as many psychological events associated with one physiological indicator, occurring in multiple contexts; and an outcome closely resembles a concomitant in terms of its characterisation of a *many–to–one* association but differs in respect of being context–limited (Cacioppo & Tassinary, 1990; Richter & Slade, 2017).

In addition to these psychophysiological processes, contemporary approaches also postulate that scholars should provide evidence for predictive (e.g., the physiological indicator of interest significantly predict a given psychological construct), concurrent (e.g., the physiological measure differentiates groups which are supposedly assumed to diverge in relation to a given psychological indicator), convergent and discriminant validity (e.g., when a physiological indicator is associated [convergent] or not [discriminant] to a set of psychological variables; Richter & Slade, 2017). Consequently, given the theoretical background presented here, the next goal of this chapter will be to describe mechanisms and procedures for acquiring psychologically meaningful data out of physiological indicators.

2.1.2 A psychophysiological approach to the Autonomic Nervous System

It is a function of the central nervous system (CNS) to control most of the physiological processes within the body, which occur mainly by the interconnectivity between the brain, the pituitary system, and the peripheral neuronal and endocrine circuits (Blascovich et al., 2011). The Autonomic Nervous System (ANS), as a part of the peripheral nervous system, assists the body in the balance of internal and external demands (e.g., maintaining homeostasis; Mendes, 2009). This is realised by a complex set of neural mechanisms understood as autonomic regulation (Berntson & Cacioppo, 2007; Janig, 2003). There are several theories that aim to study autonomic regulation, being usually classified by their level of analysis (Kreibig, 2012). At the psychological level of analysis, while the model of componential process (Ellsworth & Scherer, 2003) and the model of cardiovascular appraisal (Blascovich & Tomaka, 1996) deal with the functioning of appraisal modules, the Bio–informational theory of emotional imagery explains the functioning of associative networks (Lang, 1979).

As for theories on the brain level, there are two main categories for analysis: functioning of brain–behavioural systems and functioning of behavioural modes (Kreibig, 2012). The former contains four different models, namely the dual–system model (Lang & Bradley, 2010), the model of behavioural coping (Schneiderman & McCabe, 1989), the polyvagal theory (Porges, 2001), and the

theory of reinforcement sensitivity (Beauchaine, 2001; Gray, 1982; but see Stoeber & Corr, 2017). The latter (e.g., behavioural modes) includes the defensive coping proposition (Bandler, Keay, Floyd, & Price, 2000; Stemmler, 2003) and the predator stage model (Lang, Davis, & Öhman, 2000). Finally, there are two groups of theories that deal with the peripheral levels of physiological inference (Kreibig, 2010, 2012). The first group considers the functional aspects of the ANS, including the already mentioned Cannon's model of undifferentiated sympathetic activation (Cannon, 1927), the model of autonomic space (Berntson, Cacioppo, & Quigley, 1991), and the models of parasympathetic activation and sympathetic versus parasympathetic dominance (Gellhorn, 1970; Vingerhoets, 1985). The second group examines the effects of transmitter substances, such as the catecholamine (Ax, 1953) and receptor–types hypotheses (Stemmler, 2003).

The control of the cardiovascular autonomic functioning involves a synergic action of various brain regions, such as the dorsal motor nucleus (DVN), the nucleus ambiguus (NA), the rostral ventrolateral medulla (RVLM), and the nucleus of the solitary tract (NTS) (Duschek et al., 2009; Thayer & Lane, 2009). Figure 2.1 portrays the model of neurovisceral integration of emotions, a proposition for understanding processes involved with cardiovascular autonomic functioning. According to this model, prefrontal cortical parts of the brain constrain the amygdala, leading to the disinhibition of the central nucleus, which culminates in increasing HR and lowering heart rate variability (HRV) by three routes (Thayer & Lane, 2009). This model implies that elevated vagal tone would result in better emotion regulation and in elevated performance in executive cognitive processing (Laborde, Mosley, & Thayer, 2017).

As shown in the Figure 2.1, the first route occurs when there is activation of neurons situated in the RVLM as a result of decreased inhibition from tonically active neurons in the caudal ventrolateral medulla (CVLM), which would provoke intensification in sympathetic activity. The second route begins with the inhibition of neuronal activity from the solitary nucleus region, causing the inhibition of both the NA and the DVN, in which the product is lowered parasympathetic activity; and, finally, the third route assumes activation of neurons from the RVLM area, resulting in elevated sympathetic responsiveness. Thus, reduced activation of the

prefrontal cortex would lead to disinhibition of the tonically inhibited central nucleus of the amygdala, leading to a concomitant disinhibition of the sympathoexcitatory neurons in the rostral ventrolateral medulla (as explained in route number one) and an inhibition of parasympathoexcitatory neurons (as described in the route number two). Both processes are believed to result in increased HR and decreased HRV (Thayer & Lane, 2009).

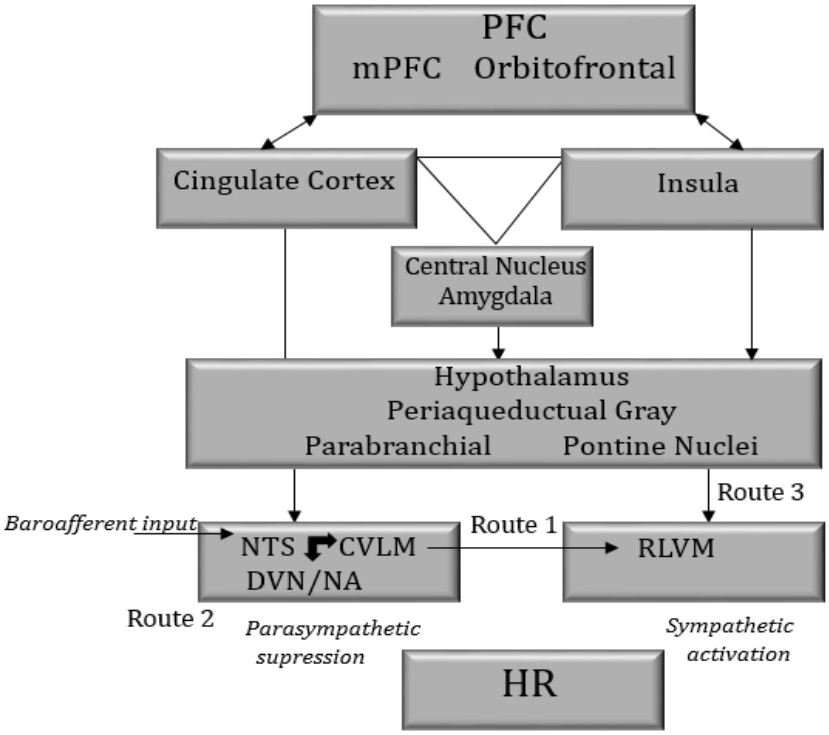

Figure 2.1 – Pathways by which the prefrontal cortex exerts control of heart rate. *Note.* The prefrontal, cingulate, and insula are cybernetically linked to the amygdala. By the stimulation of the central nucleus of the amygdala (CeA), the nucleus of the solitary tract (NTS) is inhibited. This then inhibits the inputs from the caudal ventrolateral medullary (CVLM) to the neurons at the rostral ventrolateral medulla (RVLM). At the same time, this process supresses the vagal motor neurons in the dorsal vagal motor nucleus (DVN) and nucleus ambiguus (NA). This model also postulates to the possibility of the CeA activating the sympathoexcitatory neurons in the RVLM. As such, the cascade influence of the prefrontal cortex would be an increase in HR via the disinhibition of the CeA and a disinhibition of the medullary cardioacceleratory mechanisms. Adapted from Thayer and Lane (2009, p. 84).

Accordingly, autonomic regulation of the cardiovascular system is realised by the functioning of two branches of the ANS: sympathetic and parasympathetic nervous systems (Janig, 2003). Sympathetic activation results in intensifications in cardiac contractility, HR, and vascular tone in consequence of effects on the myocardium, vascular musculature, and on the sinus node. In the other hand, parasympathetic activation predominates at rest and normally constrains the activity within the sinus node, resulting in decreased HR (Duschek, Muckenthaler,

Werner, & Reyes del Paso, 2009; Mccraty & Shaffer, 2015; Thayer & Lane, 2009). Nerves from the parasympathetic nervous system have more rapid effects (e.g., less than a second) in comparison to nerves from the sympathetic branch (e.g., five seconds or more; Nunan, Sandercock, & Brodie, 2010). Moreover, notwithstanding the fact that sympathetic and parasympathetic activation of the ANS have opposing effects, the coordination of these processes is under unitary control (e.g., the hypothalamus; Berntson & Cacioppo, 2007; Janig, 2003). An example of these opposing effects is the baroreceptor cardiac reflex (BCR), in which hemodynamic events (such as high blood pressure) cause a reduction in the cardiac sympathetic tone and an increase in the parasympathetic outflow aiming to reduce HR, ventricular contractility, and cardiac output (Berntson & Cacioppo, 2007). The projections from the baroreceptor afferents into the nucleus tractus solitary is represented in the Figure 2.1. For instance, there are conditions in which hemodynamic and cardiovascular functioning (e.g., blood pressure and HR intensifying at the same time) act against the patterns of the baroreflex reflex and homeostatic regulation (Dembowsky & Seller, 1995). These cases occur mainly when the organism is under stress, being an adaptive response to mobilise metabolic reactions (Duschek et al., 2009). The suppression of the setpoint of the BCR reflex allows complex neural systems to prevail over homeostatic responses (Van Roon, Mulder, Althaus, & Mulder, 2004), resulting in flexible patterns of allostatic regulation (Berntson & Cacioppo, 2007). Considerations of the aspects mentioned so far have invariably directed efforts for more specificity when exploring biological substrates of psychological phenomenon. Indeed, several studies attempted to detect differential patterns of ANS functioning in predicting human emotions and behaviours (Kreibig, 2010; Levenson, 2003). Cacioppo, Berntson Larsen, Poehlmann, and Ito (2000) presented a meta–analysis that aimed to explore whether emotion–specific patterns of autonomic functioning would exist. The study included 22 measures of autonomic responses, and findings were more robust for blood pressure, finger temperature, and HR. In this study, HR acceleration was more prominent for anger, fear and sadness when compared with disgust. Diastolic blood pressure has shown to be elevated in situations evoking anger rather than fear, sadness, and happiness. For emotional valence, subsequent moderated meta–analyses identified that blood volume, cardiac output, diastolic

blood pressure, HR, left ventricular ejection time, pulse transit time, and pre–ejection period were more greatly activated for negative in comparison to positive discrete emotions. The authors concluded that emotions such as anger and fear might exert differential effects of measures of peripheral cardiovascular reactivity, and that physiological indicators of bodily tension, stroke volume, respiration amplitude, and facial temperature are perhaps less reliable in terms of their specific emotional differentiation (Cacioppo et al., 2000).

Kreibig (2010) also conducted a study reviewing 134 experimental investigations of emotional effects on peripheral physiological responses amongst healthy adults. The procedure for selecting autonomic measures respected the criteria of including only protocols in which emotions were primed and individual physiological responses (not composites) were provided in face of emotional reactivity. Physiological responses were then classified into eight positive emotion groups (affection, including love, tenderness, and sympathy; amusement, including humour, mirth, and happiness in response to slapstick comedy; anticipatory pleasure, including appetite and sexual arousal; contentment, including pleasure, serenity, calmness, peacefulness, and relaxation; happiness, joy and/or elation [but excluding happiness in response to slapstick comedy]; pride; and relief and/or safety), six negative emotions (anger, including approach–oriented anger, indignation, withdrawal–oriented anger, anger in defence of other, and anger in self–defence; anxiety, including dental anxiety, performance anxiety, and agitation); disgust, including food–related disgust and disease–related disgust; embarrassment, including social anxiety, social rejection, and shame; fear and/or threat; and sadness, including dejection, depression, and achievement failure), and two groups containing emotions in which a clear valence connotation was impossible to be obtained (e.g., surprise and/or wonder, and suspense).

The study found that measurements of HR were amplified when participants responded to surprise, as well as to both positive (e.g., imaginary anticipatory pleasure, happiness, and joy) and negative emotions (e.g., anger, anxiety, contamination–related disgust, crying sadness embarrassment, and fear/threat). In the other hand, HR reduced in response to acute sadness, affection, contentment, imminent–threat fear, mutilation–related disgust, non–crying

sadness, suspense, and visual anticipatory pleasure (Kreibig, 2010). The T–wave amplitude, used as an index of sympathetic influence on the heart, was diminished when participants reacted to anger and fear but augmented in response to contentment and mutilation–related disgust (Kreibig, 2010). An increase in left–ventricular contractility reactivity (LVET) was only found for contentment, while anger, mutilation–related disgust, fear, and joy were marked by decreased LVET. In respect to indicators of pre–ejection period (PEP), a measure of the time between the left ventricle contracting and the opening of the aortic valve, findings were reduced for anger, contamination– and mutilation–related disgust, embarrassment, and fear, and increased for acute sadness, amusement, and happiness. Reduced blood pressure (systolic, diastolic and mean blood pressure) and amplified pulse transit time were noted for acute sadness reactivity. As for contentment and happiness, the study found a pattern suggestive of diminished blood pressure in response to contentment and an extended pulse transit time for happiness. Fear/threat demonstrated a reduction in total peripheral resistance (TPR), contrary to the remainder emotions analysed in the study in which either no changes (e.g., joy, mutilation–related disgust, and pride) or increases were noted (e.g., amusement, anger, anticipatory sadness, contamination–related disgust, embarrassment, and happiness) (Kreibig, 2010).

As for respiration and electrodermal patterns of ANS reactivity, key differentiations were observed for contamination–related disgust, in which parameters of respiratory timing suggested a pattern of faster breathing followed by bigger expiratory activity and smaller duration in the inspiration process. Electrodermal activity (including nonspecific skin conductance response rate [nSRR], skin conductance level [SCL], and skin conductance response [SCR]) was increased for most emotions (e.g., amusement, anger, anticipatory pleasure, anxiety, contamination–related disgust, and fear and threat), albeit, contentment, relief, and sadness (acute and non–crying sadness) were marked by a decreased pattern of physiological reactivity. Importantly, while the study conducted by Kreibig (2010) concerned patterns of ANS responses to various emotions in healthy individuals, significant work in the field of AB and psychopathy (e.g., Beauchaine, 2015; Marsh, Beauchaine, & Williams, 2008; Muñoz, Kimonis, Frick, & Aucoin, 2013; Nederhof, Marceau, Shirtcliff, Hastings, & Oldehinkel, 2015) have

provided further evidence of physiological functioning in clinical populations, and will be explored in this chapter (e.g., Section 2.2).

2.1.3 Physiological procedures for data acquisition

Aligned with the ideas disseminated by many scholars (Blascovich et al., 2011; Cacioppo et al., 2000; Ekman et al., 1983), researchers have begun to develop multi–method approaches for obtaining and interpreting peripheral physiological responses. These efforts have clearly made important methodological and theoretical contributions to a variety of constructs relevant to psychological science.

In line with the identity thesis proposed by Cacioppo and Tassinary (1990), multi–method protocols in use in contemporary psychophysiological research often combine two or more biological systems. This is believed to provide a richer, in–depth assessment of the physiological correspondents of psychological phenomena. As such, there is a crescent popularity of methods combining responses from both the autonomic and neuroendocrine systems, in which electrodermal, electrocardiogram (ECG), and impedance cardiography procedures are frequently adopted. A brief note on methodological aspects to be taken into consideration for using these procedures will be discussed next.

2.1.3.1 ECG recording

Electrical signals generated at various stages of the heart's cycle can be assessed using ECG procedures. A typical ECG examination consists of several inflections, commonly referred as P, Q, R, S, and T waves. The cardiac cycle commences when an electrical impulse, not detected on the ECG, is generated at the sinoatrial node. As commented by Blascovich et al. (2011), this leads to a depolarization within the heart atria and is usually defined as the P wave. The cycle Q, R, and S illustrates the depolarization of the ventricles, which is followed by a repolarization (e.g., T wave).

There are three common techniques for electrodes placement that result in an upward deflection of the Q–R complex: Lead I, Lead II and Lead III (Stern, Jay, &

Quigley, 2001a). In the first case, electrodes are placed above the right and left wrists, inside the arms. In this case, the positively charged lead is placed on the left arm. A lead II configuration, used in the studies presented in Chapter Four, places electrodes at either on the right arm and left ankle or over the torso. In both cases, the negatively charged lead goes on the left side. Finally, a Lead III configuration places the electrodes on the left wrist and left ankle, and the negatively charged lead is attached at the wrist (Blascovich et al., 2011). In all these cases, special attention is given to the location and condition of the skin where electrodes are applied. Researchers should ensure that the skin is dry and without excessive hair. If that be the case, preparing the area (e.g., shaving) and/or imposing higher pressure to guarantee connectivity (e.g., use of medical tape to secure electrodes) is paramount in order to ensure signal with low levels of noise. Although these procedures account for reducing greatly the presence of noise, special attention should also be given to sampling speed. As such, it is advisable to record ECG at least at 1000 Hz, as 1000 samples per second help to trace all possible inflections in the waveforms (Blascovich et al., 2011). Stern et al. (2001a) noted that ECG recordings allow the estimation of not only HR–related indicators – such as respiratory sinus arrhythmia (RSA, discussed shortly) and interbeat intervals[7] – but are relevant in determining cardiac output, PEP, and stroke volume. To obtain these indicators, researchers should combine impedance cardiography procedures into their experimental paradigms.

2.1.3.2 Impedance cardiography recording

In respect to impedance cardiography, a procedure which estimates the changes in blood flow inside the heart (e.g., stroke volume) and the timing of opening and closing of aortic valves, researchers are normally requested to use either spot or band electrodes (Blascovich et al., 2011). This procedure uses an output of electricity from outer sensors (located at upper and lower back; $mAmps_{range}$ = .1 to 4). The impedance (i.e., resistance) is captured by the inner sensors. An increase in blood flowing inside the thoracic cavity (Z_0 / basal impedance) results in reduction in impedance (Mendes, 2009). The first derivative of the waveform, also known as

[7] Calculated by assessing the time between R waves, usually expressed in milliseconds (Stern et al., 2001a).

Δz/Δt, represents a change in basal impedance over a change in time. This creates a waveform that permits the estimation of the total volume of blood expelled from the organ on a single heart beat (i.e., stroke volume; Blascovich et al., 2011). Moreover, the inclusion of measures obtained using impedance cardiography is important for assessing both sympathetic (e.g., PEP) and parasympathetic influences (e.g., RSA) on the heart (Stern et al., 2001a).

2.1.3.3 Electrodermal recording

Electrodermal activity (EDA) has been one of the most investigated response systems within psychophysiological studies (Dawson, Schell, & Filion, 2007). Notwithstanding the fact that most eccrine sweat glands have the goal of maintaining thermoregulation, Edelberg (1972) noted that both palmar and plantar sweat glands are reactive to psychologically meaningful phenomena. In addition, the EDA activity is mostly under control of the sympathetic branch of the ANS (Dawson et al., 2007; Shields, MacDowell, Fairchild, & Campbell, 1987). The method of measuring skin conductance response uses primarily two procedures: one in which there is an application of an external electrical current on participants' skin, known as the exosomatic method, and another – and most widely used – in which the measurement does not rely on a peripheral application of electrical current but focuses on the electrical signals passing through the skin's surface, known as the endosomatic method (Dawson et al., 2007; Stern, Jay, & Quigley, 2001b). Both procedures for measuring skin conductance levels (SCL) and skin conductance responses (SCR) follow the endosomatic method. Recordings on EDA are taken using two electrodes applied to participants palms (normally at the nondominant hand); moreover, the area in which the electrodes are applied should not be cleaned using alcohol or other substances that might cause abrasion, thus avoiding interference on skin's conductive properties (Dawson et al., 2007). In a similar fashion, there are several packages for the acquisition of data on EDA, but it is important to consider not only the most appropriate recording system, but also other factors such as good–quality electrodes and conducting gel, environmental

factors (including room temperature), and correct application of electrodes on the skin (Dawson et al., 2007)[8].

2.1.4 A summary on psychophysiological inference

Section 2.1 explored the role of cardiovascular tonic activity and reactivity and how they could inform researchers about emotional states. There seems to exist a consensus regarding the use of multi–method approaches in studying psychophysiological mechanisms that could explain human behaviours and emotions (Kreibig, 2010). The literature is relatively abundant in terms of methodological issues that should be considered when planning, conducting, analysing, reporting, and interpreting autonomic and neuroendocrine indicators of human emotional experiences (Berntson et al., 2007; Cacioppo et al., 2000; Edgdar et al., 2007; Jennings et al., 1981; Laborde et al., 2017; Mccraty & Shaffer, 2015; Mendes, 2009; Sherwood et al., 1990).

There are, arguably, other relevant aspects that should be considered when employing either a single– or multi–method investigation. For instance, for years researchers assumed that a functional unity of the sympathoadreno–medullary system (SAM) existed (Kreibig, 2010). However, the SAM is currently understood as being formed by two distinct parts (e.g., neural and hormonal; Folkow, 2000). Consequently, scholars should consider that these constituent parts present with differential functional roles, which should be interpreted accordingly. For instance, while the neuronal branch covers adjustments in cardiovascular responses in face of emotional responding, the hormonal activity alters metabolic indicators and blood coagulability (Folkow, 2000).

In addition to cardiovascular and electrodermal mechanisms, measurements of respiration patterns are of great importance for scholars interested in autonomic indicators of emotional responding (Kreibig, 2010; Reyes del Paso, Langewitz, Mulder, van Roon, & Duschek, 2013). This is partially due to

[8] A more in–depth account of EDA usage in psychophysiological research is beyond the reach of this section, and there are several robust publications containing theoretical and methodological guidelines for implementing EDA analysis, such as Dawson et al.'s (2007) chapter from the third edition of the *Handbook of Psychophysiology*, and Stern et al.'s (2001b) chapter included in the second edition of the *Psychophysiological Recording* book.

the existence of a great interaction of the respiratory and cardiovascular systems (Grossman & Taylor, 2007). RSA, also known as high–frequency HRV, comprises the change in the time intervals between adjacent heartbeats and usually reflects cardiac vagal index (Grossman & Taylor, 2007); however, RSA and cardiac vagal tone may sometimes be dissociated (Shaffer, McCraty, & Zerr, 2014). Therefore, RSA might be an additional variable to be considered when analysing cardiovascular aspects of ANS functioning (Kreibig, 2010; Mccraty & Shaffer, 2015; Stern et al., 2001a), especially when in combination with earlier guidelines published by the Society for Psychophysiological Research (Jennings, Bberg, Hutcheson, Obrist, Porges, & Turpin, 1981; Sherwood, Allen, Fahrenberg, Kelsey, Lovallo, & van Doornen, 1990) and in line with previous work in the field of social and affective neuroscience (Beauchaine, 2009, 2015; Beauchaine, Gatzke–Kopp, & Mead, 2007; Mendes, 2009).

As this thesis focuses largely on psychophysiological research, the next section will therefore cover key biological characteristics that underpin psychopathy and AB, with focus on recent findings from the autonomic, neuroendocrine, and cardiovascular systems. These will be empirically explored in chapter Four, examining particularly resting heart rate (autonomic and cardiovascular nervous systems) and cardiovascular reactivity to stress (combination autonomic, cardiovascular, and neuroendocrine systems).

2.2 Quantitative studies into the biological mechanisms associated with psychopathic personality traits and antisocial behaviours

It is the intent of the current section to further explore how psychophysiological studies have been elucidating aetiological mechanisms and biological correlates of AB and psychopathic tendencies. Table 2.1 summarises results from psychophysiological studies conducted in the past decade in face of psychopathy and antisocial behaviour[9].

[9] Manuscript in preparation (provisory title: Cardiovascular correlates of psychopathy and AB: A decade of research). The search strategy for including studies presented in Table 2.1 comprised the use of the following Boolean terms: "(psychopathy OR psychopathic OR antisocial OR callous) AND (Psychophysiology OR physiology OR physiological OR biological OR biomarker OR Psychophysiological OR cardiovascular)". 1,391 results were obtained from University of Chicago Press Journals, Project MUSE, SpringerLink Open Access,

For clarity, psychophysiological investigations retrieved using the systematic search will be described in relation to two distinct domains of assessments: studies using measurements taken at both rest (Section 2.2.1) and in response to various experimental paradigms (Section 2.2.2). Other relevant factors, such as methodological particularities and gender differences will be examined whenever appropriated.

Dialnet, Scientific Electronic Library Online, SAGE Journals, Dawsonera, PubMed Central, Oxford Journals, National Library of Sweden (SwePub), Directory of Open Access Journals (DOAJ), Taylor & Francis Online, SpringerLink, U.S. Dept. of Education, Wiley Online Library, SciVerse ScienceDirect (Elsevier), Arts & Humanities Citation Index/Science Citation Index Expanded/Social Sciences Citation Index (Web of Science) and MEDLINE/PubMed. After filtering the results published in the past 10 years, 767 potential articles remained. The next step comprised the abstract reading of each result. The criteria for including articles in this review in preparation were: empirical study published in a peer–reviewed journal (i); addressing at least one cardiovascular measure in respect to psychopathy or antisocial behaviour (ii); written in English (iii). Considered these criteria, 33 articles survived.

Table 2.1: *Summary of psychophysiological studies conducted in past decade on psychopathy and AB*

Study	Country	Sample characteristics/ Setting	Domain assessed	Psychological/behavioural measures and tasks used	Biological measurement	Main findings
Allen et al. (2009)	United States	101 college students, being 46 males (M_{age} = 19.2, *SD* = 1.4)	Impulsivity	Barratt Impulsiveness Scale, Ego–Undercontrol Scale. Task: Simple reaction time task and speech task	Diastolic blood pressure, heart rate, systolic blood pressure	Impulsivity was positively linked with systolic blood pressure during rest and negatively linked with HR reactivity
Armstrong et al. (2009)	United States	105 college students, being 58 males (M_{age} = 21.43, *SD* = 5.7)	Antisocial behaviours and aggression	Self–Control Scale and Self–Report measures of antisocial behaviour, delinquent peers, and attachment to parents from National Youth Survey	Heart rate	After controlling for age, race, gender, BMI, and physical fitness, participants with LRHR had higher levels of severe AB in comparison to those with average or high resting HR
Brenner and Beauchaine (2011)	United States	206 children, being 72 girls (M_{age} = 9.9, *SD* = 1.5)	Conduct disorder/Conduct Problems	Child Behaviour Checklist, Child Symptom Inventory, Customary Drinking and Drug Use Record. Task: Monetary–incentive task	Pre–ejection period and pre–ejection period reactivity	Resting PEP was negatively linked with CD. Diminished PEP reactivity to a monetary–incentive task predicted alcohol use, and there was an interaction of conduct problems with anxiety and depression in predicting the likelihood for using marijuana
Casey et al. (2013)	United States	61 male prisoners (M_{age} = 41, *SD* = 11)	Psychopathy	Eysenck Personality Questionnaire, Psychopathy Checklist – Revised. Task: International Affective Picture System	Heart rate	Participants scoring above 30 in the PCL–R raised HR when viewing negative pictures, which was not detected for those with scores below 30 (Cohen's *d* = .49, *p* = .01). Significant negative associations between the PCL–R Factor 1 (*r* = – .31) and total scores of psychopathy (*r* = –.29) were detected for the HR 'Experience Index'
Crozier et al. (2008)	United States	585 adolescents aged between 16 to 18 years old, being 52% males	Antisocial behaviour	Adolescent Behaviour Questionnaire, Child Behaviour Checklist, Youth Self–Report, Reactive–Proactive Aggression Questionnaire. Task: Social information processing	Resting heart rate, heart rate reactivity	LRHR was directly linked to AB for males only. Nonetheless, high HRR and high RHR had indirect effects on AB via deviant patterns of social information processing for both males and females
De Vries–Bouw et al. (2011)	Holland	68 male adolescents who were attending a programme for delinquency (M_{age} = 13.9, *SD* = .8)	Antisocial behaviour (juvenile delinquency)	Self–reported Negative Affect. Task: Public speaking task	Heart rate, heart rate variability	Attenuated HR reactivity to stress predicted the length of time until future reoffending and a stronger HRV reactivity predicted higher rates of these behaviours

De Wied et al. (2009)	Holland	44 boys aged between 8 and 12 years, being 22 healthy controls	Disruptive behaviour disorders	Child Behaviour Checklist, Diagnostic Interview Schedule for Children, Teacher's Report Form, Wechsler Intelligence Scale for Children–Revised (Vocabulary and Block Design). Task: Viewing of emotional clips	Electromyographic, heart rate	Corrugator muscle reactivity to anger and sadness was significantly smaller for boys with disruptive behaviours in comparison to controls. These disruptive participants also had significantly less cardiac deceleration during sadness
De Wied et al. (2012)	Holland	63 boys aged between 12 and 15 years, being 17 with Disruptive behaviour disorders/low CU and 14 Disruptive behaviour disorders/high CU	Callous–unemotional traits, Disruptive behaviour disorders	Antisocial Process Screening Device, Child Behaviour Checklist, Diagnostic Interview Schedule for Children, Teacher's Report Form, Wechsler Intelligence Scale for Children–Revised (Vocabulary and Block Design). Task: Viewing of emotional clips	Electromyographic (facial), heart rate	Participants with disruptive behaviours with elevated CU traits had diminished levels of empathic sadness in comparison to controls, which was apparent at both self–reporting and physiological measurements (e.g., HR reactivity and corrugator muscle reactivity)
Gao et al. (2012)	United States	138 males (M_{age} = 35.72, *SD* = 8.61) recruited from the community	Psychopathy	Psychopathy Checklist – Revised, Wechsler Intelligence Scale – III (Similarities, Arithmetic, Digit Symbol and Picture Completion) plus comprehensive criminal search and gathering of collateral data from ten different sources. Task: Emotion–inducing task	Heart rate, skin conductance	Psychopathic individuals showed lower HR at baseline and at other time points in comparison to a control group. In addition, psychopaths with and without impairments in recognising body sensations did not differ on HR, which suggested a mismatch between autonomic functioning and body sensations
Gatzke–Kopp et al. (2015)	United States	240 children, being 105 as a CP group (M_{age} = 6.00, *SD* = .41)	Conduct problems	Strengths and Difficulties Questionnaire. Task: Go/No–Go	Electroencephalogram, heart rate, skin conductance	Kindergarten children with and without CP were compared in respect to autonomic reactivity and P3b brain responses. Groups did not differ in skin conductance. However, higher increase in HR was detected amongst those with CP while performing a frustration task. This same group of children had a pronounced decrement in the P3b amplitude in comparison with their responses at rewarding tasks
Jennings et al. (2013)	United Kingdom	386 males. No information is given regarding mean age	Conviction frequency, violence	No standard psychological questionnaire has been used, but authors refer to questions regarding impulsivity, restlessness, substance use, and environmental risk factors	Heart rate (resting)	RHR in males at the age of 18 was a significant predictor in all models forecasting the total number of offending and violent offending at the age of 50. In sum, those with higher RHR gathered less criminal convictions up to the age of 50

Kyranides et al. (2016)	Cyprus	2.444 adolescents (M_{age} = 15.96, *SD* = .89), being 45% males completed questionnaires at T1. 88 (M_{age} = 19.92, *SD* = .99), being 50% males who took part in the experiment (T2)	Psychopathy (CU traits, aggression, CD)	Adult Self–Report Inventory–4, Antisocial Process Screening Device, Buss and Perry Aggression Questionnaire, Inventory of Callous–Unemotional Traits, Emotion Regulation Questionnaire, State–Trait Anxiety Inventory, Triarchic Psychopathy Measure. Task: Affective ranking after viewing erotic, violent, and neutral videos	Electromyography, heart rate, skin conductance, startle response	Resting HR was only linked to impulsivity during the first phase of the study (adolescence; *r* = – .22), but it was associated to all dimensions of the Triarchic Psychopathy Measure (*r*'s$_{range}$= – .28 to . –21) during young adulthood. HR reactivity to violent videos was associated to boldness (*r* = – .24) in young adults, and HR reactivity to erotic scenes was linked to impulsivity in adolescents (*r* = .30). While no significant associations were detected for skin conductance, results for startle potentiation reactivity between neutral versus violent videos were significant for narcissism (*r* = – .28) and CU traits (*r* = – .34) amongst adolescents, and for meanness in young adults (*r* = – .26)
Lobbestael and Arntz (2010)	Holland	147 adults with BPD (*n* = 45; M_{age} = 33.82, *SD* = 7.83), ASPD (*n* = 21; M_{age} = 30.29; *SD* = 7.79), patients with cluster C personality disorder (*n* = 46; M_{age} = 35.80, *SD* = 9.32) and typical developing controls (*n* = 35; M_{age} = 36.91, *SD* = 11.84)	Antisocial personality disorder	Interview for Traumatic Events in Childhood, Profile of Mood States, Psychopathy Checklist–revised, Schema Mode Inventory, Structured Clinical Interview for DSM–IV Axis I and II disorders. Task: Seeing neutral, abuse related, and positive; implicit association test	Heart rate, Galvanic Skin Response (level and reactivity), Facial EMG, Systolic blood pressure (SBP) and diastolic blood pressure (DBP)	There were no significant differences between participants with Borderline Personality Disorder in comparison with those with a diagnosis of Antisocial Personality Disorder in respect to baseline levels of DBP, HR, and SCL. During baseline, borderline patients had, however, diminished levels of SBP when compared to a control group, coupled with lower SC responses in comparison to antisocial and normal participants. Participants with antisocial personality showed elevated SCL in response to stress in comparison to patients with cluster C personality disorder. Facial EMG at baseline revealed diminished frowning levels amongst antisocial individuals when compared to the other groups
Marsh et al. (2008)	United States	54 boys, being 31 with DBD (M_{age} = 9.8, *SD* = 1.4) and 23 controls (M_{age} = 10.5, *SD* = 1.5)	Disruptive behaviour disorders	Child Behaviour Checklist, Child Symptom Inventory. Task: Sad emotion induction	Cardiac pre–ejection period, skin conductance level, respiratory sinus arrhythmia	Typical developing boys showed diminished SCL, extended PEP, and increased RSA in response to sad facial expressions. On the other hand, boys with CP did not change their RSA reactivity in response to these same expressions

Mills–Koonce et al. (2015)	United States	870 participants took part at the age of 6 (wave one) and 15 months (wave two). Groups were formed by CP/CU+ (n = 51/50), CP (n = 54/43) and comparison (n = 765/678) at waves one and two, respectively	Callous–unemotional traits, Conduct problems	Disruptive Behaviour Disorder Rating Scale, Inventory of Callous–Unemotional Traits. Task: Fear challenge task	Heart period, respiratory sinus arrhythmia, salivary cortisol	No differences were detected between groups of children at the age of 6 months. Nonetheless, at 15 months of age, those with a combination of CU traits and CP showed higher cortisol levels prior to a fear challenge task (Cohen's d_{range} = .41 to .49), as well as displayed lower levels of HP and RSA compared to those belonging to the other groups (CP only and children without CP). Albeit there were no differences between groups in respect to cortisol reactivity, children with CU traits and CP had higher overall cortisol levels in comparison to the other groups (Cohen's d_{range} = .47 to .57)
Muñoz and Anastassiou – Hadjicharalambous (2011)	United States	38 preschool children, aged between 4.5 to 5.5 years. 22 were boys	Disinhibition, impulsivity	Children's behaviour questionnaire, Child Behaviour Checklist. Tasks: Card playing task, circle–drawing task, Peabody picture vocabulary task, passive avoidance task, reward and punishment and only punishment (go/no–go)	Cardiac pre–ejection period, heart period, respiratory sinus arrhythmia	Even though no links between baseline physiological activity and externalising problems were detected, the study showed a positive association between uninhibited temperament with heart period (r = .36). Moreover, impulsivity was linked to extended PEP at baseline (r = .41). In respect to physiological reactivity, PEP differences were noted between uninhibited and inhibited children. As such, those with inhibited behaviours presented with smaller PEP values during the first minute but prolonged PEP at the second minute. On the other hand, uninhibited children started the task with longer level of PEP but shortened it at the second minute
Muñoz et al. (2013)	United States	85 detained boys (M_{age}= 15.5, SD = 1.28)	Aggression, Callous–Unemotional Traits, Psychopathy (Narcissism and impulsivity)	Antisocial Process Screening Device, Inventory of Callous–Unemotional Traits, Peer Conflict Scale. Task: The dot–probe task, the Competitive Reaction Time Task.	Heart rate, respiratory sinus arrhythmia, skin conductance	The study found that narcissism was not significantly linked to measures of autonomic reactivity. However, significant differences were noted in the highly provocative scenario used to evaluate aggression, precisely when participants with a combination of high sympathetic and parasympathetic activity and elevated narcissism showed higher levels of aggression when compared to those low on narcissism but with a similar psychophysiological profile
Nederhof et al. (2015)	Holland	715 adolescents were tested at ages of 16.3 and 19.1, being 50.9% female	Internalising and externalising problems	Child Behaviour Checklist, Youth Self–Report. Task: Groningen Social Stress Task	Heart rate, cardiac pre–ejection period, respiratory sinus arrhythmia, salivary cortisol	Elevated HR reactivity was a significant predictor of internalising problems only for boys. For girls, low HR reactivity was linked to externalising problems. As for the other biological indicators, the study showed that reduced RSA, diminished PEP reactivity, and small cortisol levels were important in predicting externalising problems for boys (and not for girls)

Osumi et al. (2007)	Japan	32 college students aged between 18 and 22 years, being 50% males	Psychopathy	Primary and Secondary Psychopathy Scales, Positive and Negative Affect Schedule Scales. Task: International Affective Picture System and watching an Unpleasant movie	Heart rate, skin temperature	HR escalation after exposition to a short clip of a murder scene was 3.4 bpm for those high on psychopathy and of 8.8 bpm amongst those scoring low. Skin temperature declined less amongst secondary psychopaths in comparison to those presenting with dominant primary traits
Posthumus et al. (2009)	Holland	225 preschool children, (M_{age} = 4.3, *SD* = .25), being 64.5% boys	Conduct disorder/Conduct Problems	Child Behaviour Checklist, Diagnostic Interview Schedule for Children IV—Parent version, Wechsler Preschool and Primary Scales of Intelligence–Revised	Heart rate and heart rate reactivity, skin conductance levels and reactivity	Children with elevated levels of aggression had lower SC levels and SC reactivity when compared to children with low aggression. Significant differences were also noted for children with Oppositional Defiant Disorder or Conduct Disorder, who showed lower SC levels in comparison to children with Attention Deficit Hyperactivity Disorder and children with comorbid Attention Deficit Hyperactivity Disorder + Oppositional Defiant Disorder or Conduct Disorder
Raine et al. (2014)	Hong Kong	334 children and adolescents (M_{age} = 13.22, *SD* = 1.19). Most of participants were males (58.4%)	Psychopathy	Antisocial Process Screening Device, The Reactive and Proactive Aggression Questionnaire	Resting heart rate	Significant correlations were identified between RHR and reactive (*r* = –.15) and proactive aggression (*r* = –.18), as well as with impulsivity, narcissism, and total score of the Antisocial Process Screening Device (*r's* = – .17, – .15, and – .15, respectively)
Rothemund et al. (2012)	Germany	22 males, being 11 psychopaths (M_{age} = 31, *SD* = 6.4) and 11 typical developing controls (*M* = 28, *SD* = 6.7)	Psychopathy	Anxiety Disorders Interview Schedule, PCL–R, PCL–SV, Positive and Negative Affect Schedule, Sensation Seeking Scale, State Trait Anxiety Inventory. Task: Aversive differential conditioning experiment	EMG, ERP, heart rate, skin conductance reactivity	Psychopaths demonstrated deficits in presenting with differential startle responses, lack of increased corrugator activity, and lack of increased SCR when compared to healthy controls
Scarpa et al. (2008)	United States	40 children between 7 and 13 years–old (M_{age} = 9.8, *SD* = 1.81). 57.5% of participants were boys	Reactive and proactive aggression	Anxiety Disorders Interview Schedule for DSM–IV, Children's Report of Exposure to Violence, Revised Parent Rating Scale for Reactive and Proactive Aggression	Heart rate, heart rate variability	Community violence victimisation predicted higher levels of proactive aggression only in participants with LRHR (β = – .78). In addition, witnessing community violence predicted elevated involvement with reactive aggression in children with high levels of resting HRV (β = .90). These results were robust after controlling for other types of community violence exposure and comorbid psychiatric diagnosis

Scarpa et al. (2010)	United States	68 children aged between 6 and 13 years–old (M_{age} = 9.64, *SD* = 2.08). 62% of participants were boys	Reactive and proactive aggression	Anxiety Disorders Interview Schedule for DSM–IV, Child Version, Parent Interview Schedule, Child Behaviour Checklist, Revised Parent Rating Scale for Reactive and Proactive Aggression	Resting heart rate, skin conductance, heart rate variability	HRV predicted both proactive (β = .43) and reactive (β = – .47) aggression. SC level was also a significant predictor of proactive (β = .46) and reactive (β = – .41). There was no significant predictive role of resting HR in explaining proactive and reactive aggression
Serafim et al. (2009)	Brazil	110 males, aged 18 years–old or more	Psychopathy	Psychopathy Checklist–Revised, State–Trait Anxiety Inventory. Task: International Affective Picture System	Heart rate	Negative associations between the PCL–R Factor 1 and HR were detected while psychopaths viewed neutral (*r* = –.64), pleasant (*r* = – .53), and unpleasant images (*r* = – .66). Non–psychopaths murderers showed an increase in HR when presented with pleasant images while participants who committed murder did not show variations in HR
Sijtsema et al. (2010)	Holland	2230 children were assessed at T1 (M_{age} = 11.09, *SD* = .56), 2149 at T2 (M_{age} =13.56, = .53), and 1816 at T3 (M_{age} = 16.27, *SD* = .73)	Antisocial behaviour	Behavioural Inhibition System/ Behavioural Activation System (BIS/BAS) scales, Early Adolescent Temperament Questionnaire Revised, Revised Neuroticism–Extroversion–Openness Personality–Inventory (NEO–PI–R), Youth Self–Report	Heart rate	HR at age 11 predicted rule breaking behaviours at age 16 only for boys (β = – .07). For boys (β = – .07) and girls (β = – .06), HR longitudinally predicted fun seeking (BIS/BAS). Moreover, HR at age 11 predicted adventurism (NEO–PI–R) at age 16 for boys (β = – .16) and girls (β = – .09)
Sijtsema et al. (2011)	Holland	119 participants, aged between 9 to 16 years (M_{age} = 12.47, *SD* = 1.96). 100% girls	Relational and physical aggression	Children's Rejection Sensitivity Questionnaire, Children's Social Behaviour Scale–Teacher Report. Task: Adapted Cyberball	Heart rate, respiratory sinus arrhythmia, skin conductance	HR and SC reactivity were linked to (*r*'s = – .22 and – .19) and significantly predicted (β's = – .20 and – .17, respectively) relational aggression
Sijtsema et al. (2013)	Holland	2230 children were assessed at T1 (M_{age} = 11.09, *SD* = .56), 2149 at T2 (M_{age} = 13.56, *SD* = .53)	Antisocial behaviour	Antisocial Behaviour Questionnaire, Peer Nomination for Bullying	Heart rate	The study reported that HR was correlated to affiliation with bullies (*r* = – .11) and with antisocial behaviour (*r* = – .10). Moreover, moderation analyses revealed that those who were affiliated to peers less inclined to bully others, HR measurement was not linked to AB. In the other hand, increased affiliation with bullies resulted in an association between HR and AB (β = –.028). Amongst boys only, mediation analyses showed a partial indirect effect of –.013 between HR and AB via affiliation with bullies

Sijtsema et al. (2015)	Holland	2230 children were assessed at T1 (M_{age} = 11.09, *SD* = .56), 2149 at T2 (M_{age} = 13.56, = .53), and 1816 at T3 (M_{age} = 16.27, *SD* = .73)	Antisocial behaviour	Antisocial Behaviour Questionnaire. Task: Groningen Social Stress Task	Cardiac pre–ejection period, respiratory sinus arrhythmia	Antisocial behaviour was linked to PEP while participants were performing a speech (*r* = .08) and to RSA at rest, while giving a speech, and during recovery (*r*'s = .08, .17 and .09, respectively). Simple slope analyses revealed that experiencing severe adversities was associated with antisocial behaviour only amongst boys with blunted PEP difference between resting and task phases (β = .17). Moreover, moderation analyses showed that boys with diminished RSA difference score between rest and task and reporting more adversities were more likely to display antisocial behaviours in comparison to boys with amplified RSA difference score between rest and task phases
Stanger et al. (2012)	United Kingdom	66 team sports athletes aged between 18 and 25 years (M_{age} = 19.95, *SD* = 1.61). 60.6% were males	Antisocial behaviour, psychopathy	Interpersonal Reactivity Index, Moral Disengagement in Sport Scale – Short, Prosocial and Antisocial Behaviour in Sport Scale, Self–Report Psychopathy scale III. Task: Picture viewing (20 unpleasant, 20 neutrals, and 20 pleasant)	Electro cutaneous startle blink, evoked potentials, heart rate, skin conductance	Psychopathy (and not antisocial behaviour) was correlated to both blink magnitude (*r* = – .39) and changes in HR (*r* = .31). There were no significant associations between psychopathy and antisocial behaviours with SC and P300 amplitude in response to unpleasant pictures
Sylvers et al. (2008)	United States	120 college students (41% males) from the ages of 18 to 21	Antisocial personality disorder, Narcissistic personality disorder	Short Coolidge axis II inventory, Structured Clinical Interview for DSM–IV Axis II. Task: Count–down, The International Affective Picture System	Cardiac pre–ejection period, respiratory sinus arrhythmia, skin conductance	For the count–down task, the antisocial personality features significantly predicted PEP reactivity only when measured in according to DSM's structured interview (β = .22), which was also noted in the regression models explaining SC levels reactivity (β = .37). Both narcissistic personality features as measured by the Short Coolidge Axis II inventory and DSM's tool did not predict changes in reactivity in the levels of SC. As for the task using the International Affective Picture System, regression models showed that antisocial personality features (DSM) did not predict RSA reactivity while participants were presented with slides of happiness, sadness, and fear

Sylvers et al. (2010)	United States	100 college students (50% males). No information was given about age	Antisocial personality disorder	Aggression Questionnaire, Psychopathic Personality Inventory–Short Form, Structured Clinical Interview for DSM–IV Axis II. Task: Count–down, The International Affective Picture System	Cardiac pre–ejection period, respiratory sinus arrhythmia, skin conductance	Antisocial personality disorder predicted (β = – .44) electrodermal hypo reactivity for males only during a passive coping task. However, those antisocial traits were significant in predicting RSA hyper reactivity amongst females (β = .50) and also in explaining PEP hyper reactivity (β = – .35) in males while viewing threatening slides. Finally, while viewing images of others in distressful situations, APD explained RSA hyper reactivity solely for females (β = .34)
Wang et al. (2012)	United States	1219 children (M_{age} = 9.6, *SD* = .58), being 51.1% girls	Psychopathy	Childhood Psychopathy Scale. Task: Count–down	Heart rate, skin conductance	While HR acceleration was positively linked to the callousness–disinhibition domain of childhood psychopathy for boys (r = .15) and girls (r = .15), non–specific SC responses were only correlated to the manipulative/deceitfulness domain in boys (r = – .21)

Notes. AB: antisocial behaviour; APD: antisocial personality disorder; BAS: behavioural Activation System; BIS: behavioural Inhibition System; CP: conduct problems; DBP: diastolic blood pressure; EPR: event–related potential; EMG: electromyography; GSR: galvanic skin response; HR: heart rate; HP: heart period; HRV: heart rate variability; LRHR: low resting heart rate; NEO–PI–R: revised Neuroticism–Extroversion–Openness Personality– Inventory; PEP: Cardiac pre–ejection period; RSA: respiratory sinus arrhythmia; SBP: systolic blood pressure; SC: skin conductance; SCL: skin conductance levels; SCR: skin conductance reactivity.

As can be inferred from the studies contained within Table 2.1, there seems to exist a particular emphasis into psychophysiological examinations amongst children and adolescents. Also, there is a large number of studies focusing on incarcerated individuals.

2.2.1 Studies examining ANS functioning at rest

HR at rest is assumed to reflect tonic levels of ANS activity and homeostatic regulation (Porges, Doussard–Roosevelt, Portales, & Greenspan, 1996). LRHR has been suggested to be a biological indicator of trait–fearlessness (Crozier, Dodge, Fontaine, Lansford, Bates, Pettit, & Levenson, 2008). Direct support for this is available from investigations conducted with young children, indicating that uninhibited temperament is linked to LRHR (Muñoz and Anastassiou–Hadjicharalambous, 2011; Scarpa et al., 1997). Arguably, the absence of fear in response to punishment might shape learning and socialisation, leading, over the years, to stable patterns of interpersonal functioning (Crozier et al., 2008). Not surprisingly, most studies conducted over the last decade (Table 2.1) in which the ANS was investigated at rest included measurements of HR and its components, such as HRV and heart period (HP).

Following past research which proposed that AB and early traits of psychopathy can be manifested in a life–course trajectory (hence suggesting strong influence of biological mechanisms), Mills–Koonce, Wagner, Willoughby, Stifter, Blair and Granger (2015) found important differences in biological mechanisms amongst individuals with only 15 months of age. This birth cohort investigation revealed distinct aetiological factors for AB, showing that those with a combination of CU traits and CP presented with higher baseline cortisol levels, as well as displayed lower levels of HP (which is inversely related to HR) and RSA compared to those belonging to the other groups (i.e., CP only and children without CP). In an investigation with young children (M_{age} = 5.23, SD = .27), Muñoz and Anastassiou–Hadjicharalambous (2011) reported similar results, in which uninhibited temperament was associated with lower HP (r = .36, p < .05), which denotes lower HR.

More recently, Kyranides, Fanti, Sikki and Patrick (2016) confirmed the negative links between resting HR with impulsivity in adolescents, providing further support to the proposition of RHR as an index of fearlessness amongst typical developing adolescents. RHR was correlated to impulsivity only during adolescence (r = – .22), being nonetheless associated to all dimensions of the Triarchic Psychopathy Measure during young adulthood (r's$_{range}$ = – .28 to . –21). The study reported also on negative associations between RHR with both disinhibition and meanness amongst Cyprian adults. In a study with male adults with and without elevated psychopathic personality traits recruited from the community, Gao, Raine, and Schug (2012) found lower HR at baseline and at other time points amongst those high (M = 28.03) in comparison to those scoring low (M = 18.77) on the PCL–R (Hare, 2003). Similarly, Armstrong, Keller, Franklin, and Macmillan (2009) reported that, after controlling for age, race, gender, body mass index, and physical fitness, college students with LRHR had higher levels of severe AB in comparison to those with average or high RHR.

Advances in contemporary research made possible a better understanding of complex processes involving individuals and the environment, shifting dichotomies towards an integrated biosocial approach (Beauchaine & Thayer, 2015; DeLisi, Wright, Beaver, & Vaughn, 2011; Hare & Neumann, 2006). As an example of such unified perspective, Sijtsema et al. (2013) reported that lower levels of HR were indirectly associated with AB in pre– and early adolescent boys via association with bullies. These findings were interpreted in the light of the sensation seeking theory of AB, which states that there is a tendency amongst those displaying biological features of low arousal to seek violent and aggressive activities (Hansel et al., 2007; Raine, 2002). Analogous patterns have been identified amongst children and adolescents in Asia, whereas HR has been reported to interact with social and environmental factors (Raine, Fung, Portnoy, Choy, & Spring, 2014). In this investigation, significant, negative, and small associations were identified between HR and reactive and proactive aggression, as well with impulsivity and narcissism. Interestingly, LRHR was associated with greater levels of reactive aggression only at high intensity (and not low) of social adversities (Raine et al., 2014). Notably, an important message from this study

includes the consideration of factors that go beyond specific, biologically based domains to address environmental levels surrounding individuals.

Studies with typical developing children and young adults provide evidence of the associations between HR and AB longitudinally. For instance, HR in boys at the age of 11 predicted rule–breaking behaviours at the age of 16, being associated with more aggression at the same age (Sijtsema, Veenstra, Lindenberg, van Roon, Verhulst, Ormel, & Riese, 2010). Jennings, Piquero and Farrington (2013) reported that RHR in males at the age of 18 was a significant predictor in all models forecasting the total number of crimes committed and violent offending at the age of 50. In sum, those with higher RHR gathered less criminal convictions up to the age of 50. Together, these studies exploring cardiovascular mechanisms at rest are important to the extent that they elucidate biobehavioural mechanisms that could explain AB, psychopathy, and maladjustment. For instance, low levels of sympathetic arousal can prone individuals to engage in risky activities (Raine, 2002) and to be less responsive to parenting and school efforts (Muñoz & Anastassiou–Hadjicharalambous, 2011).

Researchers have begun to disentangle ANS reactivity to more closely examine its associations with AB and psychopathy. Indeed, De Vries–Bouw, Popma, Vermeiren, Doreleijers, Van De Ven and Jansen (2011) affirmed that strong HRV is capable of predicting higher rates of recidivism amongst adolescents with antisocial tendencies and criminal records. By definition, the time between consecutive R peaks[10] in the waveform is conceptualised as HRV, being a well–known measure of the activation of the parasympathetic nervous system (PNS; Beauchaine, 2015; Marsh et al., 2008; Thayer & Lane, 2009).

Scarpa, Haden, and Tanaka (2010) found that HRV significantly predicted both proactive (β = .43) and reactive (β = – .47) aggression in children (M_{age} = 9.64), which indicated that, rather than general associations with ANS over– or under–arousal, different forms of aggression can be differentially associated with ANS functioning. Albeit there were no significant findings for HR, reactive aggression was associated with reductions in both parasympathetic influences on

[10] ECG recordings are composed of inflections reflecting slopes in the waveform. The basic waves are P, Q, R, S, and T. The R wave represents the electrical stimulus passing through the main portion of the ventricular walls and is the first upward inflection after the P wave, which makes it easier to identify.

the heart (i.e., decreased HRV) as well as sympathetic activity on the eccrine glands (i.e., decreased SC). As for proactive forms of aggression, a distinct pattern was found (i.e., increased HRV and increased SC). In an earlier report, Scarpa, Tanaka, and Chiara Haden (2008) explored the moderation role of resting HR and HRV between reactive and proactive aggression and exposure to violence in children. Amongst participants with LRHR, there was an association between proactive aggression and previous experiences of victimisation; however, those with high resting HR demonstrated fewer problems related to proactive aggression. Additionally, witnessing community violence positively correlated with reactive aggression amongst those high on resting HRV but with reduced reactive aggression in those with low levels of HRV. This investigation suggested that the environment might have particular effects in the development and maintenance of reactive aggression, probably due to the fact that witnessing violence exerts influence on individual's attribution of intention (e.g., higher expectations of violence would prone people to react on a defensive manner; Scarpa et al., 2008). It could also be the case that community violence might aggravate predispositions towards antisocial behaviours (Liu, 2004). Finally, Lobbestael and Arntz (2010) examined physiological responses between participants with Borderline Personality Disorder (BPD) and with APD. The results indicated that these personality disorders did not present statistically significant differences in baseline levels of HR, SCL, and diastolic blood pressure. One possible reason given for not being significant differences between groups in autonomic mechanisms usually linked to AB was related to the small number of participants (n = 21) with complete data on APD.

2.2.2 Studies examining ANS reactivity

As seen in studies examining ANS activity at rest, investigations on biological mechanisms associated with psychopathy and AB have been conducted to explore how individuals might differ in their physiological functioning when in response to a variety of emotional stimuli (e.g., Casey, Rogers, Burns, & Yiend, 2013; Osumi, Shimazaki, Imai, Sugiura, & Ohira, 2007; Serafim, Barros, Valim, & Gorenstein, 2009) and when reacting to stressors (e.g., Casey et al., 2013; Lobbestael & Arntz,

2010). In respect to HR reactivity, which indexes phasic ANS activity (i.e., emotion regulation; Porges et al., 1996), the polyvagal theory (Porges, 2001) assumes that two branches of the vagus nerve induce the heart when individuals are faced with emotional and threatening situations: (1) the dorsal motor nucleus branch, involved in suppressing metabolic demands when the organism is in danger, and (2) the phylogenetically newer branch, which emerges from the nucleus ambiguous and is arranged to determine the fight or flight responses to emotional stimuli (De Vries–Bouw et al., 2011). Accordingly, higher vagal tone is assumed to result in overall better social functioning (Laborde et al., 2017), while reduced vagal tone is linked to more inter– and externalising difficulties, including psychopathy and AB (Beauchaine, 2001).

In an investigation in Japan, Osumi et al. (2007) noted that HR increase after the exposition to a short clip of a murder scene was 3.4 beats per minute for those high on psychopathy and of 8.8 beats per minute amongst those with low scores, which made explicit that high psychopathy is not only associated with low levels of cardiovascular functioning at rest, but also when participants react to distressing content. In a similar fashion, Serafim et al. (2009), in a study conducted in Brazil, reported negative, significant associations amongst PCL–R Factor 1 with HR while psychopaths viewed neutral (r = –.64), pleasant (r = – .53), and unpleasant images (r = – .66). Murderer–psychopaths did not show heart rate variations according to the type of images, albeit non–psychopaths murderers displayed an increase in HR when presented with pleasant images, which was interpreted as an indicator of affective deficits for those with a diagnosis of psychopathy. Likewise, HR reactivity to violent videos has been previously associated to boldness (r = – .24) in young adults (Kyranides et al., 2016), which seems to reinforce the low fear disposition assumed to be important in the biological constitution of individuals high on psychopathic personality traits. In summary, these studies indicate that some patterns of neurobiological functioning – especially HR – seem to underlie psychopathic tendencies, being highly replicable across various cultures, settings, and age groups.

One further study examined the physiological responses of violent offenders while viewing positive and negative images that were accompanied by

emotion regulation instructions (Casey et al., 2013). Significant negative associations between Hare's (2003) PCL–R Factor 1 (r = – .31) and total score of psychopathy (r = –.29) and the heart rate 'Experience Index' (i.e., participants were requested to 'imagine themselves' in the picture) were detected for negative images. In addition, Factor 1 significantly predicted (β = – .51) the heart rate 'Experience Index' for negative pictures, suggesting a unique pattern of emotional reaction to contents that would generally be experienced in a negative manner. In this study, those with scores above the cut–off point of 30 (PCL–R; Hare, 2003) raised HR when viewing negative pictures (e.g., attacks, fires, guns), which was not detected for those with scores below 30. One possible interpretation to the findings presented by Casey et al. (2013) is that severe psychopaths presented with cardiac speeding compatible with pleasant experiences while viewing images supposed to elicit unpleasant experiences. For them, negative stimuli were interpreted as a reward (Lang, Lang, Bradley, & Cuthbert, 1999). Data reported by Casey et al. (2013) reinforced that differences in behaviour, interpersonal functioning and environmental circumstances might interact in different forms with psychopathic tendencies. Importantly, these findings suggest that these individuals might not form a homogeneous group. Indeed, evidence that some individuals with AB possess a tonic level of physiological hyperarousal, which is triggered in face of environmental factors, has been also reported amongst adolescents (Crozier et al., 2008). In this study, while high RHR and high RHV had indirect effects on AB via deviant patterns of social information processing for both male and female adolescents, LRHR was directly linked to AB for males only (Crozier et al., 2008).

Nederhof et al. (2015) explored measures of the hypothalamic pituitary adrenal axis (HPA; e.g., cortisol), ANS (e.g., HR), parasympathetic nervous system (e.g., RSA), and sympathetic nervous system (e.g., PEP). Physiological reactivity was recorded in response to a social stressor task, which involved giving a speech that would be judged and rated based on participants' performance. High HR reactivity predicted internalising problems in boys, whereas low HR reactivity was longitudinally linked to externalising problems amongst girls. Additionally, diminished RSA, blunted PEP reactivity, and low cortisol levels predicted future externalising problems in boys only, picturing a profile marked by

parasympathetic withdrawal, sympathetic activation and high HPA–reactivity. PEP differences between uninhibited and inhibited children were also identified when searching for manuscripts published in the last 10 years. For instance, participants with inhibited behaviours presented with smaller PEP values during the first minute of an interview with a novel experimenter but prolonged PEP at the second minute. On the other hand, uninhibited children started the task with longer level of PEP but shortened it at the second minute (Muñoz & Anastassiou–Hadjicharalambous, 2011). Analyses of HP and RSA reactivity during the interview were conducted, but no significant differences were noted between uninhibited and inhibited children

In a study with detained boys, Muñoz et al. (2013) investigated physiological reactivity to provocation and to emotional content (i.e., distress, positive, and neutral contents). The researchers were particularly interested in the role of psychopathy–linked narcissism, and included measures of both branches of the ANS (e.g., sympathetic and parasympathetic). Cluster analysis was carried out to profile these boys in terms of emotional reactivity, yielding 3 groups: (one) "coactivators", characterised by increased sympathetic and parasympathetic activity, (two) "sympathetic activators", comprising those who displayed increased sympathetic activity and slightly increase in parasympathetic activity, and (three) "low activators", aggregating boys who showed small changes in both branches. Further analyses revealed significant differences between groups in both laboratory–assessment of aggression and in self–report measures (Muñoz et al., 2013). Significant differences in the levels of aggression were detected in the highly provocative scenario. Here, those in group one (coactivators) and high on narcissism differed when compared to those in the same group, but low on narcissism (groups were formed by median split of the narcissism subscale of the Antisocial Process Screening Device/APSD; Frick & Hare, 2001). Similarly, boys high on narcissism reported greater proactive aggression than those low on narcissism. These findings suggested that narcissism was associated to elevated self–report use of "cold–blooded" aggression amongst those with physiological hypo–responsive type, whereas the group hyper–reactive showed defensive behaviours during the laboratory task (Muñoz et al., 2013).

In adults, narcissistic and antisocial personality features have been investigated in relation to patterns of physiological reactivity. Antisocial features – as measured in accordance to the DSM–IV's criteria – were not significant in predicting RSA reactivity to emotions such as happiness, sadness, and fear (Sylvers, Brubaker, Alden, Brennan, & Lilienfeld, 2008). However, these features significantly predicted PEP reactivity to a countdown task (β = .22) and were equally significant in explaining SCL reactivity (β = .37). Narcissistic personality features did not predict changes in reactivity in SCL, but were associated to RSA decreases and PEP shortening whilst participants viewed happy slides.

Marsh et al. (2008) reported that sad facial expressions were associated with reduced sympathetic (lower skin conductance levels and extended pre–ejection period [PEP][11]) and increased parasympathetic reactivity (higher RSA) amongst typical developing boys. However, those with CP displayed a different pattern of less correspondence between emotion induction of sadness and autonomic reactivity (no changes in RSA). In adults, evidence suggested parasympathetic nervous system dysfunctions in women and sympathetic nervous system dysfunctions amongst males (Sylvers, Brennan, Lilienfeld, & Alden, 2010). Features of APD were related with electrodermal (EDA) hypo reactivity in males during a passive coping task, while APD characteristics were associated with RSA hyper reactivity amongst females and with PEP hyper reactivity in males while viewing threatening slides. Finally, while viewing images of others in distressful situations, APD associated with RSA hyper reactivity only for females (Sylvers et al., 2010).

Despite an increment in the studies examining biological correlates of psychopathy and AB, Sijtsema, Shoulberg and Murray–Close (2011) argued that there is less attention to the role of gender in previous psychophysiological research. One of the reasons for this might be that the majority of studies have focused on physical forms of aggression. To provide a more nuanced account of this, the researchers examined whether physiological reactivity to social exclusion could be linked to relational aggression. The study asked 119 girls (M_{age} = 12.47) to play a 3–min ball–throwing game after baseline recordings were acquired. During

[11] Pre–ejection period (PEP) is a measure of the time from the left ventricle contracting (Q point) to the opening of the aortic valve.

the game, participants were excluded after receiving the ball twice and, during the rest of the game, the other players throw the ball between themselves. The results showed that HR and SC reactivity were negatively linked to relational aggression. Moreover, both HR and SC reactivity significantly predicted this form of aggressive behaviour.

The role of culture has also been taken into consideration when examining autonomic and neuroendocrine patterns of physiological reactivity. Lovallo (2013) illustrated that disrupted parenting and adversity experienced during early–years was linked to abnormal physiological functioning, as well as were associated with behavioural impulsivity. Specifically, data from this cohort study with more than 400 adults showed diminished cortisol and low HR reactivity amongst those with previous exposure to adversity. There were also significant detrimental effects of these experiences on participant's cognitive (working memory and general intelligence) and affective abilities (high neuroticism, more antisocial traits [PPI Factor 2; Lilienfeld & Andrews, 1996], and elevated depressive symptoms), which led the authors to suggest a model for the impact of adversity on the prefrontal cortex and part of the limbic system (Lovallo, 2013). This model postulates that early stress and adversity influence key brain regions responsible for affect regulation and coping, which would prone individuals towards impulsivity and other inter– and externalising difficulties.

Another study revealed that experiencing severe adversities was associated with AB only amongst boys with blunted PEP difference between resting and task phases (β = .17; Sijtsema, Van Roon, Groot, & Riese, 2015). Moreover, moderation analyses showed that boys with a combination of diminished RSA reactivity between rest and task phases and reporting more adversities were more likely to display AB in comparison to boys with amplified RSA reactivity (Sijtsema et al., 2015). Wang, Baker, Gao, Raine, & Lozano (2012) argued that although HR acceleration has been found to be positively linked to the callousness–disinhibition domain of childhood psychopathy for both boys (r = .15) and girls (r = .15), non–specific SC responses were only correlated to the manipulative/deceitfulness domain in boys (r = – .21). Together, these investigations reinforced the role of gender in ANS reactivity patterns and its differential links with AB and

psychopathy, suggesting that differences between sexes could guide more tailored therapeutic strategies (Sijtsema et al., 2015; Wang et al., 2012).

In addition, Herpertz, Vloet, Mueller, Domes, Willmes and Herpertz–Dahlmann (2007) identified autonomic abnormalities in fathers and their respective sons, suggesting a pattern of transmission of antisocial behaviour within families. In this study, 44 boys possessing with early–onset of CD and their biological fathers, along with and 36 healthy controls were assessed in terms of HR and SCL. The fathers of boys with CD showed less reactivity in SCL to emotional stimuli and more aggressive behaviour than did the fathers of controls. There were significant, moderate, and positive correlations between fathers and sons with CD for measures of HR and SCL.

2.3 The aims of this thesis

This thesis aims to investigate behavioural, emotional, and physiological aspects of psychopathic personality traits and antisocial behaviours amongst adults recruited from the community. These first two introductory chapters have explored the incremented validity of emotional and behavioural difficulties that could underpin the relationship between psychopathy[12] and aggression (i.e., bullying perpetration and victimisation), summarising key deficits in psychopathy, particularly in from psychophysiological research. As shown so far, there has been emergent attention on the neurobiological factors that may explain antisocial behaviours, and strong evidence of a general low arousal, poor frontal brain functioning, as well hormonal and genetic correspondences to adult psychopathy and child behavioural and emotional problems (Blair, 2007a, 2007b; Blair, Meffert, Hwang, & White, 2018; Cornet, 2015; Debowska, Boduszek, Hyland, & Goodson, 2014; Rilling, Glenn, Jairam, Pagnoni, Goldsmith, Elfenbein, & Lilienfeld, 2007; Sebastian, De Brito, McCrory, Hyde, Lockwood, Cecil, & Viding, 2016).

Research also indicates that psychopathic personality traits are associated with abnormal physiological reactivity to emotional stimuli, as well as could play an important role in determining physiological responses to stress (Jones et al.,

[12] In this thesis, the use of the word 'psychopath' refers to those possessing also with elevated levels of psychopathic personality traits, not necessarily the formal diagnosis of the condition.

2009a; Lopez–Duran, Olson, Hajal, Felt, & Vazquez, 2009; Walker, Papilloud, Huzard, & Sandi, 2016). On the other hand, characteristics such as high skin conductance, elevated levels of resting heart rate (RHR), and high intelligence (e.g., IQ) could serve as mitigating aspects against risk factors often associated with antisocial behaviour (Portnoy et al., 2013). Nonetheless, biological influences on psychopathy and antisocial behaviours are still poorly understood (Anderson et al., 2017; DeLisi et al., 2011; Griffiths & Jalava, 2017; Hare, 2001). By detecting biological mechanisms underpinning psychopathology, professionals may develop strategies for early diagnosis, build theory, and create more tailored intervention programmes (Portnoy et al., 2013). It is within this framework that this thesis is included, aiming to explore a series of correlates of psychopathic personality traits and antisocial behaviours in adults. As noted earlier, a distinction between psychopathic subtypes was proposed decades ago, in which secondary variants were assumed to be associated with internalising difficulties, while primary psychopathy was influenced by biological features supposed to index greater immunity to environmental influences, lack of fear, and a more limited capacity for empathy (Karpman, 1941; Porter, 1996; Poythress & Skeem, 2006). Following this, the behavioural, physiological, and emotional domains examined in this thesis take into consideration not only the overall total scores of psychopathic personality traits, but also the variants and factors of the construct.

2.3.1 Behavioural domain

This thesis' first aim is to advance in the understanding of the manifestation of aggressive behaviours (bullying) amongst adults in the context of high levels of psychopathic tendencies. As argued in Chapter One, psychopathic personality traits have repeatedly been associated with aggression, being bullying an important predictor of later criminal involvement (Barker et al., 2008; Bender & Losel, 2011; Fontaine et al., 2016; Piquero, Farrington, Fontaine, Vincent, Coid, & Ullrich, 2012). The extent in which both phenomena are linked in typical developing adults, however, still warrants research.

2.3.1.1 Study 1 – Adult bullying and primary and secondary psychopathic traits: Insights from a community sample

Important aspects in bullying perpetration research include the examination of psychological processes (i.e., the motives used to bully others) and concurrent correlates (i.e., personality characteristics and environmental factors; Rodkin et al., 2015). In agreement with the latter, Study 1 was designed to investigate the relationship between psychopathic personality traits and bullying behaviours, to examine the predictive role of psychopathy and gender in explaining various types of bullying involvement (i.e., perpetration, fighting, and victimisation), and to explore the mediation effect of bullying victimisation on the relationship between both primary and secondary psychopathic variants with bullying perpetrating behaviour.

Based on the contents presented in Section 1.1.1, which introduced a number of important investigations linking both bullying and psychopathy to elevated emotional and behavioural difficulties, Study 1 hypothesises that these two constructs would be positively associated with each other (Fanti & Kimonis, 2012, 2013; Warren, 2009). Another prior prediction of Study 1 is that psychopathic personality traits would predict bullying behaviours, which has been reported in the youth and adult literature (Baughman, Dearing, Giammarco, & Vernon, 2012; Fanti, Frick, & Georgiou, 2009b; Muñoz et al., 2011; Warren, 2009). Moreover, past research linked psychopathy to greater severity of instrumental aggression, remarking that the co–occurrence of victimisation and high psychopathy increased the use of proactive forms of aggression (Barker et al., 2008). Following this, a final prediction of the first study is that a positive indirect effect for the relationship between psychopathic variants and bullying perpetration as mediated by victimisation should emerge. These aims and hypotheses are examined in the investigation "Adult bullying and primary and secondary psychopathic traits: Insights from a community sample", presented in the next chapter.

2.3.1.2 Study 2 – Bullying involvement and psychopathic personality traits: Disentangling the links amongst college students

Section 1.1.1 argued that children with a combination of behavioural problems and early psychopathic personality traits not only engage more in bullying behaviours, but also at a greater intensity; in addition, they tend to replicate these behaviours over time (cf. Fanti & Kimonis, 2012; van Geel, Toprak, Goemans, Zwaanswijk, & Vedder, 2016). A robust literature has been built upon findings stressing that although CU traits and CP certainly matter for comprehending bullying dynamics, additional psychopathy–linked constructs might as well be important (i.e., narcissism and impulsivity; van Geel et al., 2016). While the former has shown to increase the involvement with both bullying perpetration and bullying victimisation, the latter has been linked to higher chances of becoming a target of bullies (i.e., bullying victimisation). Levels of machiavellianism were also noted to be elevated amongst children who fall into a bully–victim category (Sutton & Keogh, 2000).

Clearly, past evidence suggested so far that children, but not adults, are in serious risk of being victimised by others when presenting high levels of psychopathic personality traits (Fontaine et al., 2016). The work in this thesis comes from the position that the same might be true in adults. The inherent stability of personality traits should lead to a capability of replicating these findings in young adults, coupled with the fact that early work has established a bad prognosis for psychopathic traits (Lilienfeld, 2018; Ribeiro da Silva, Rijo, & Salekin, 2015). As such, questions remain as to whether bullying perpetration and bullying victimisation are linked to psychopathic personality traits in adults, and as to whether are there differences in groups of individuals with differential involvement with bullying dynamics. Accordingly, the second cross–sectional study of this thesis ("Bullying involvement and psychopathic personality traits: Disentangling the links amongst college students", presented in Chapter Three) was designed to answer to these questions.

This second study assumes that a more detailed account on the co–occurrence of bullying and various psychopathic personality traits in adults can be achieved by comparing the levels of those traits in respect to participants' roles in

bullying. Hypotheses were formulated in which higher involvement with bullying would correlate with elevated levels of psychopathic personality traits. Ragatz, Anderson, Fremouw, and Schwartz (2011) reported that concurrent bullying perpetration and victimisation resulted in overall higher levels psychopathic personality traits. Thus, it has been predicted that bullying–victims would have significant differences in their scores on a self–report measure of psychopathic personality traits when compared to adults who were categorised as being only victims, solely bullies, or non–involved (Fanti & Kimonis, 2013; Sutton & Keogh, 2000).

2.3.2 Physiological domain

Hare proposed that biology accounts for as much as the variance [of psychopathy] of environmental factors combined, including early trauma and exposure to violence (Hare, 2001). Indeed, Viding et al. (2005) noticed that CP are more heritable for those high in CU traits than for those with low psychopathic traits (CU–), indicating to an innate vulnerability for expression of antisocial behaviours and suggesting that risk genes might be different for CU+ and CU– (Sadeh et al., 2010; Viding, Jones, Paul, Moffitt, & Plomin, 2008). In addition of presenting moderate to strong genetic influence, psychopathic traits have been linked to functional and structural brain differences, particularly in respect to emotion–processing (Anderson et al., 2017; De Brito et al., 2011; Fontaine, Rijsdijk, McCrory, & Viding, 2010; Griffiths & Jalava, 2017; Korponay et al., 2017; Jones et al., 2009a; Jones et al., 2009b; Marsh et al., 2013; Raine, Reynolds, Venables, & Mednick, 1997a; Viding et al., 2012).

Subsequently, the second aim of this thesis concerns an examination of psychophysiological aspects of aggressive behaviours, bullying, and psychopathic personality traits. Evidence currently available in both clinical and community samples is convincing enough that there seems to exist a "biological division" between psychopaths and non–psychopaths (Hare, 2001). As emphasised within Section 1.1.2, early reports show a robust association between basal autonomic functioning with antisocial behaviour and psychopathy, coupled with convincing indication of neuroendocrine abnormalities in this population (DeLisi et al., 2011;

De Vries–Bouw et al., 2011; Hansel et al., 2007; Muñoz et al., 2013; Pasion et al., 2017; Raine, 2002; Sylvers et al., 2010). Nonetheless, less is known in respect to the validity of these biological indicators of low arousal and neuroendocrine abnormalities amongst individuals possessing with distinct degrees of psychopathic personality traits (Skeem et al., 2011; Wilson et al., 2014).

2.3.2.1 Study 3 – Resting heart rate and psychopathic personality traits in adults

Addictive effects of biological concomitants and psychosocial factors in discriminating antisocial individuals from typical developing controls were reported in previous research. Interestingly, Raine (2001) noted that autonomic deficits – including HR – added incremented validity (R^2 = 76.7%) above and beyond psychosocial risk factors alone (R^2 = 41.3%) (e.g., social economic status, parents' involvement with crime, history of abuse). HR is an autonomic biological indicator that is especially relevant to the present work as it is under influence of brain regions previously linked to psychopathy (e.g., the amygdala; Blair, 2005, 2017). Several studies examined HR while individuals were in resting conditions and in response to various emotion–eliciting paradigms (Crozier et al., 2008). However, much of the previous psychophysiological work in psychopathy and antisocial tendencies has been carried out with clinical, child–based samples (cf. Table 2.1; Hansen et al., 2007; Wilson et al., 2014; see also Skeem et al., 2011). Consequently, it is important to explore and replicate the validity of these findings into different groups, which could have theoretical and practical implications for the understanding of aetiological pathways and correlates of these behaviours throughout developmental stages.

The third study in this thesis – and first psychophysiological investigation – is presented in Chapter Four ("Resting heart rate and psychopathic personality traits in adults"). It explores the links possibly existing between a consecrated indicator of antisocial behaviour (e.g., RHR) with facets of psychopathy, as well as explores the predictors of such biological indicator. The rationale for this study is based on a burgeoning line of research, which has consistently revealed that psychopathic traits are robustly associated with LRHR (Portnoy & Farrington, 2015; Sijtsema et al., 2010). The relationship between LRHR has also been

established with different forms of AB, such as rule breaking (Sijtsema et al., 2010), criminality (Jennings et al., 2013), and impulsivity (Kyranides et al., 2016). Consequently, it has been hypothesised for Study 3 that higher psychopathic personality traits would be negatively correlated with measurements of HR at rest. This study further predicts that regression models should yield significant results when psychopathic personality traits are included as predictors, principally those assumed to index lack of fear (i.e., fearlessness).

2.3.2.2 Study 4 – How do you respond? Antisocial behaviours are linked to threatening physiological responses

Barrett (2006) stated that it is difficult to propose unique and invariant autonomic substrates of emotional experiences, but defended that a more comprehensive view could be obtained using protocols assessing general conditions of "threat and challenge" (p. 41). Guided by The Bio–Psychosocial model of Challenge and Threat (BPSM; Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996), the fourth study examines whether psychopathic personality traits might be useful in differentiating physiological reactivity to stress. Physiological responses of challenge and threat are usually described using a bipolar continuum, in which negative values would indicate that individuals' resources are not sufficient to overcome the demands of a given task (i.e., threat appraisal; Seery, 2011).

It has been hypothesised that psychopathy would not be linked to an increased response to basic threats, but suggestions that this could be true in situations involving frustration were made (Blair, 2006; 2010b). Indeed, past research revealed patterns of either hypo– or hyper–reactivity to stress (Vaughn, DeLisi, & Matto, 2014). For example, contrary to the well–known hypo–reactive group of psychopaths (e.g., Factor 1), there seems to exist a variant of psychopathy highly reactive to minor stress, which may dispose individuals to impulsivity and aggression (Crozier et al., 2008; Johnson, Mikolajewski, Shirtcliff, Eckel, & Taylor, 2015). Furthermore, meta–analytical results have yielded that early psychopathic traits, along with narcissism and impulsivity, were related to more aggressive behaviour and bullying (van Geel et al., 2016). This, in turn, has been associated with abnormal stress reactivity, but the replicability of these findings amongst

typical developing adults is less known (Hamilton, Newman, Delville, & Delville, 2008; Lopez–Duran et al., 2009; Walker et al., 2016).

In face of past evidence, Study 4 predicted that psychopathic personality traits would be inversely associated with challenge, in which negative correlations between psychopathic personality traits with challenge and threat reactivity indexes would emerge. In addition, following previous studies in which physiological hyper reactivity has been associated with antisocial behaviours (Hamilton et al., 2008; Lopez–Duran et al., 2009), it was also predicted that bullying behaviours should be negatively correlated with challenge and threat reactivity indexes.

2.3.3 Emotional domain

This thesis' third aim is to dissect sub putative deficits in emotional functioning to comprehend the manifestation of psychopathic traits in adults. As discussed in Section 1.2.1.1, it is believed that psychopathic traits might be linked to higher difficulties in understanding own emotional experiences (Louth et al., 1998; Takamatsu & Takai, 2017). Alexithymia is a term that encompasses these difficulties, including difficulties such as labelling feelings and poor consciousness; higher traits of alexithymia could be significant in differentiating how individuals process and describe their internal states, thus influencing behaviours (Sifneos, 1973).

2.3.3.1 Study 5 – The role of emotional deficits in understanding the associations between psychopathic personality traits and self–reported stress in adults

This fifth study investigates not only the associations between perceived stress and psychopathy (giving continuity to experimental data reported in Study 4), but also the individual impact of each of the psychopathic variants on stress, including direct and indirect links between primary and secondary psychopathy on stress via dimensions of alexithymia. In addition, this study investigates which variants of psychopathy could predict self–reported stress in adults.

It has been anticipated that positive and significant associations between perceived stress and psychopathic personality traits would emerge (Boozer, Forte,

& Harris, 2005), albeit traits resembling self–centred impulsivity aspects (e.g., Factor 2 or secondary variants) should be more strongly linked to self–report perceived stress as they index the instable, impulsive and anxious phenotype (Karpman, 1941; Lykken, 1957). The second hypothesis predicts that secondary (and not primary) psychopathy should then significantly predict total levels of stress. Finally, alexithymia dimensions were expected to mediate the relationship between perceived stress and primary and secondary psychopathy. However, bigger effects were expected to occur for the secondary variant (cf. Booze et al., 2005).

The last chapter (Chapter Six) provides a general summary of this thesis, analysing the contributions of each one of the five studies presented, their meaning and implications for the comprehension of emotional, behavioural, and physiological correlates of the psychopathic personality. Chapter Six also provides some discussion and insight into the process of the research contained in this thesis, with the intent of establishing an agenda for future research. Importantly, this thesis embeds the recommendation of Patrick and colleagues into multivariate correlational studies in combination to experimental investigations to link theoretical predictions with behavioural and physiological findings (Patrick, Venables, Yancey, Hicks, Nelson, & Kramer, 2013). Additionally, the STROBE statement for cross–sectional data was followed whenever applicable (von Elm, Altman, Egger, Pocock, Gøtzsche, & Vandenbroucke, 2007). Similarly, ethical aspects were carefully debated at each stage of this thesis, and relevant information on this is presented at the methodological sections of each empirical chapter.

CHAPTER THREE – PSYCHOPATHIC PERSONALITY TRAITS, AGGRESSIVE, AND ANTISOCIAL BEHAVIOURS IN ADULTS

Chapter overview

The literature reviewed in the previous chapters indicated that there is an emergent, yet robust, understanding that prolonged involvement with bullying can explain and predict unique trajectories into antisocial behaviour and psychopathy. As such, an association between callous–unemotional (CU traits), or psychopathic personality traits and bullying has been demonstrated consistently in children and adolescents (van Geel et al., 2016). Reports also have highlighted the role of traits such as narcissism and impulsivity as being equally important in understanding bullying behaviours (Fanti & Kimonis, 2012, 2013; van Geel et al., 2016).

Recent work has shown that bullying remains an important issue in adulthood as well (Baughman et al., 2012; Warren, 2009), but much more has to be studied about the nature of this association in adults. Reasons for this could include, but are not limited to the stability in dynamics of aggression and victimisation from infancy to adult life (i); incremental and persistent necessity for specific groups, presenting with emotional and behavioural difficulties, to gain and maintain power over others (ii); inability of bullies to adequately incorporate and develop prosocial behaviours beyond school grounds (iii), amongst others (Kapoor, Alynkia, & Jadahv, 2016; Loeber & Hay, 1997; Lynam, 1997). It is the purpose of this chapter to extend the discussion on the core emotional and behavioural deficits accompanied by high psychopathic personality traits, with a special focus on the links between psychopathy with aggressive behaviours and bullying. Firstly, this chapter presents theoretical material from both bullying and psychopathy literature, examining the similarities between these constructs as well as their impacts on individual's emotional and behavioural problems. Subsequently, it reports on two studies that have been carried out on the associations between bullying and psychopathic personality traits. The findings from these investigations are then discussed, alongside with their limitations, implications, and directions for further research.

3.1 Aggressive behaviour, bullying and psychopathy

Bullying is usually described as an intentional and aggressive act, carried out by a group or an individual in a situation of power imbalance (Olweus, 1991). The majority of the literature regarding bullying is published with samples composed of children and adolescents. In adults, although there is a significant number of papers on workplace bullying, the research is rather more limited (Bender & Losel, 2011; Chen & Huang, 2015; Ortiz–León, Jaimes–Medrano, Tafoya–Ramos, Mujica–Amaya, Olmedo–Canchola, & Carrasco–Rojas, 2014; Warren, 2009).

One of the most important elements for the characterisation of bullying is the manifestation of an imbalance of power, typifying this phenomenon as abusive, cruel, and unfair (Nansel, Overpeck, Pilla, Ruan, Simons–Morton, & Scheidt, 2001; Rigby & Smith, 2011). However, importantly for this thesis is certainly the idea that some bullies can be described as cold, Machiavellian, and calculating, and have been reported to not express emotional empathy towards their victim(s), which has a clear overlap with what is already known about psychopathic traits (Frick et al., 2003; Juvonen & Graham, 2014; Kawabata, Crick, & Hamaguchi, 2013; Kimonis et al., 2011; Nansel et al., 2001).

Previous studies have examined the role of psychopathic traits in bullying behaviours across various age–ranges (Fanti, Brookmeyer, Henrich, & Kuperminc, 2009a; Fanti & Kimonis, 2012; Gacono & Hughes, 2004; Gumpel, 2014; Golmaryami, 2013; Muñoz et al., 2011; Thornton, Frick, Crapanzano, & Terranova, 2013; van Geel et al., 2016; Viding et al., 2009). Child and adolescent studies have indicated that the early onset of psychopathic personality traits – also known as callous–unemotional or CU traits – are consistently associated with incidences of direct bullying (Crapanzano, Frick, Childs, & Terranova, 2011; Golmaryami, 2013; van Geel et al., 2016; Viding et al., 2009). Direct bullying refers to situations where bullies typically need to confront their victims face–to–face. One possible explanation is that deficits in affective empathy offer a mediating role between the presence of elevated CU traits and direct bullying behaviours, where elevated CU traits are associated with poorer affective empathy (but not cognitive) (Jones et al., 2010).

In addition, data from children with psychopathic tendencies indicate that this group is more likely to experience peer rejection, but what is interesting is that peer rejection has relatively little impact on their social self–concept (Warren, Jones, & Frederickson, 2015). Precisely, a positive correlation was found between social exclusion with CU traits (r = .31) amongst children with social, emotional and behavioural difficulties, receiving special education services. However, this group of pupils did not display significant associations between CU traits to social acceptance and overall self–concept (Warren et al., 2015). This is by some means expected, as psychopaths have been identified as unconscientiousness and more prone to exhibit self–enhancement features (Gustafson & Ritzer, 1995), often displaying with a shortage of insight (Lilienfeld & Fowler, 2006).

The combination of these dysfunctional personality traits exemplifies the lack of concern and the inevitable use of manipulation and power over victims, displayed by bullies who also present themselves with elements of grandiosity (Juvonen & Graham, 2014). Interestingly, Orue, Calvete and Gamez–Guadix (2016) identified that grandiosity and impulsivity are important predictors of disruptive, overt–reactive behaviours, in a longitudinal study with adolescents. Fanti and Kimonis (2013) added that specific dimensions of psychopathic traits in children and adolescents are incremental in explaining variance in the occurrence of bullying beyond the manifestation of conduct problems alone. These findings combined seem to suggest that psychopathic personality traits may play an important role in the initiation and perpetuation of peer aggression and bullying, highlighting the role of fearlessness and indifference towards the other's emotions, as critical for understanding the shared features of bullying and psychopathy (Fite, Raine, Stouthamer–Loeber, Loeber, & Pardini, 2010).

Research has also shown differences in the role of early psychopathic traits in bullying behaviour across gender, whereas boys tend to display greater severity of physical aggression (Fanti & Kimonis, 2012; Thornton et al., 2013). These findings are in line with previous work on bullying and seem to not vary amongst cultures (for a meta–analytic review, see Archer, 2004). In the psychopathy literature, it is often the case that male do engage in more violent acts when compared to female counterparts. However, the studies previously cited have been

carried out with child and adolescent samples and there is very little work extending these concepts into adulthood (Warren, 2009).

3.1.1 Bullying and psychopathy in young adults

Bullying has been previously linked to self–report measures of psychopathy amongst typical developing adults (Williams et al., 2003), and research has shown that both bullies (i.e., perpetrators of bullying) and psychopaths share a common positive view about using hostility, manipulation and aggression as problem–solving techniques (Nielsen, Skogstad, Matthiesen, Glasø, Aasland, Notelaers, & Einarsen, 2009; Warren, 2009). Experiences of bullying have a predictive power in understanding aggression (Juvonen & Graham, 2014), antisocial behaviour (Bender & Losel, 2011) and delinquency in adults (Barker et al., 2008).

Bullying is never a pleasant experience. As such, research has suggested that experiencing bullying in adulthood could increase the risk for alcohol–related problems (Rospenda, Richman, Wolff, & Burke, 2013), suicide (Sinyor, Schaffer, & Cheung, 2014), stress (Qamar, Khan, & Kiani, 2015), and might likewise lead to deficits in individuals' perception of quality of life (Chen & Huang, 2015). It has been identified that 2 to 14.3% of adults are exposed to workplace bullying in Norway (Nielsen et al., 2009), while up to 50% of North American workers are estimated to experience at least one episode of bullying per week (Lutgen–Sandvik, Tracy, & Alberts, 2007). A large study conducted with American college students (N = 1,135) showed that nearly 40% of participants said that they agree to some extent that bullying is a part of growing up. Moreover, 14.3% agreed that ignoring the aggressor is enough for preventing it from happening again. 6.4% were victims, 24.4% revealed that they had bullied others, and 18.1% were classified as bully–victims (Garland, Policastro, Richards, & Miller, 2017). However, reports of the prevalence of bullying in adults are sensitive to external factors (e.g., cultural and methodological), coupled with the fact that bullying could be manifested in more sophisticated ways in different stages of human development.

A recent review of studies on interventions to reduce workplace bullying indicated that in some circumstances the outcome is either ineffective or has

resulted in an increase of this type of behaviour, quite likely due to employees' increased awareness (Escartin, 2016). Additionally, psychopaths found in schools and universities could bully others to achieve their goals of gaining power and domination. Therefore, it seems sensible to look at this phenomenon and its consequences along the lifespan, bearing in mind not merely which aspects are associated with negative outcomes but likewise what can be done to prevent its occurrence (Garland et al., 2017; Ortiz–León et al., 2014).

3.2 Study 1 – Adult bullying and primary and secondary psychopathic traits: Insights from a community sample[13]

The research presented so far indicates that psychopathy and bullying are two types of problematic behaviours associated with undesirable consequences for the individual as well as at social levels (Barker et al., 2008; Bender & Losel, 2011). Those with higher levels of aggression and who are bullied often display a great degree of frustration and a hostile attributional bias when dealing with ambiguity (Crick & Dodge, 1994), as well as use retaliation strategies as a problem–solving technique in social situations (Troop–Gordon & Asher, 2005), which entangles reoccurring cycles of aggression and victimisation (Rodkin et al., 2015). With no surprise, child and adolescent data indicated to a positive association between CU traits and premeditated aggression (or instrumental); moreover, CU traits were assumed to result in proactive aggression when combined with experiences of victimisation (Barker et al., 2008; Fanti et al., 2009b; Fontaine et al., 2016).

Following up on these data, it seems sensible to propose that psychopathic traits could be connected to bullying perpetrating behaviours via a mediating effect of victimisation. However, even the relationship between bullying and AB – including psychopathy – is not often researched in adulthood. Study 1 was designed in attention to this matter, and fits within one of the broad contributions of this thesis which is to explore behavioural difficulties associated with psychopathic personality traits, in the hope that these clarifications could bring insight into both risk and protective factors of AB. Hence, the current study aims to

[13] This manuscript has been accepted for publication in the Journal of Aggression, Maltreatment, and Trauma by Taylor and Francis Group, LLC, www.tandfonline.com.

investigate the relationship between psychopathy and bullying behaviours in a sample composed by adults, equally exploring the predictive role of gender and psychopathic variants in explaining various types of bullying involvement. Another aim of this study is to take the first look at the experiences of victimisation, as it mediates the links between primary and secondary psychopathic personality traits in adults and bullying perpetrating behaviour.

3.2.1 Hypotheses

In line with previous research (e.g., Fanti & Kimonis, 2012; 2013), this study hypothesises that psychopathic personality traits would be positively associated with aggressive behaviour towards others (e.g., fighting and bullying others). It also hypothesises that psychopathic personality traits would positively predict bullying perpetration (cf. Muñoz et al., 2011). Because previous work linked CU traits to severe types of aggressive behaviour, particularly in its instrumental form, combined with data showing that the co–occurrence of psychopathic traits and experiences of victimisation leads to a greater involvement with proactive aggression, it has been hypothesised that the relationship between psychopathic personality traits and bullying perpetration would be mediated by participants' levels of self–reported victimisation. Specifically, predictions were made in respect of a positive indirect effect for the relationship between psychopathic variants and bullying perpetration as mediated by victimisation, in which higher levels of psychopathy would be connected to more victimisation, which would associate to bullying perpetration (Barker et al., 2008; Blair, 2007b; Fanti et al., 2009b; Glenn & Raine, 2009; Rodkin et al., 2015; Troop–Gordon & Asher, 2005).

3.2.2 Method

3.2.2.1 Participants and design

The sample in this cross–sectional study comprised 233 young adults (M_{age} = 25.6 years, *SD* = 5.6 years). The majority of participants (83%) were female and regularly enrolled as students at university level. This study used regression analyses to examine the predictive value of psychopathic personality traits in

predicting bullying in adults. Additional mediation models were used to explore the specific role that experiences of victimisation could have in the relationship between the psychopathic variants (i.e., primary and secondary), with proactive aggression (i.e., bullying others). Further details on these procedures are described in the Section 3.2.2.4 (Data analysis).

3.2.2.2 Procedures

The study received ethical approval from the Goldsmiths Psychology Research Ethics Committee. Participants were recruited online and by using the Research Participation Scheme at two universities in the United Kingdom. Prior to completing the questionnaires, participants were presented with an outline of the study, and were asked to provide consent for participation. Measures were presented in the following order: Illinois Bullying Scale (Espelage & Holt, 2001) and Levenson Primary and Secondary Psychopathy Scales (Levenson et al., 1995). Descriptions for both measures are presented in the Section 3.2.2.3. Most of the participants completed the survey online (e.g., following an electronic link that directed to the study's webpage) or by using an Apple iPad during an in–lab visit, taking approximately 25 minutes. When requested, participants completed the survey using pen and paper.

3.2.2.3 Measures

A demographic questionnaire including questions about gender, age, field of study and the university where the student is enrolled was used. A space for extra comments was also added in this brief questionnaire. To facilitate participant's maximum ease in their reports on bullying and psychopathy experiences, demographic questions were reduced to a minimum as possible. Hence, to assess psychopathy, the Levenson Primary and Secondary Psychopathy Scales were administered (LPSP; Levenson et al., 1995) and to measure bullying behaviours in adults, the Illinois Bullying Scale was used (IBS; Espelage & Holt, 2001).

3.2.2.3.1 Illinois Bullying Scale (Espelage & Holt, 2001)

The IBS, an 18–item measure, is designed to assess the frequency of bullying behaviour in its direct and indirect forms, having also a subscale for explicit aggression (e.g. fighting). It is completed using a 5–point scale ranging from never (0) to seven or more times (4). Three subscales comprise the IBS, namely the bullying subscale (perpetrator), the fighting subscale and, lastly, the victimisation subscale (Appendix A).

Participants are instructed to complete the IBS bearing in mind their experiences over the past month. "Other students made fun of me", "I started arguments or conflicts", and "I got into physical fights" are example questions of the victimisation, bullying, and fighting subscales. Past research with university students (Kapoor et al., 2016) and adults (Hoetger, Hazen, & Brank, 2015) have shown adequate psychometric properties for the IBS (α_{range} = .86 – .90). In the current investigation, Cronbach's alphas were α =.73 for the perpetrator scale, α =.74 for the victimisation subscale, α =.80 for fighting subscale, and α =.89 for the total scale. Exploratory factor analysis was performed to confirm the psychometric properties of the Illinois Bullying Scale in the current study as the measure has not been previously used in the context of psychopathy research. Results of the principal component analysis, with oblique rotation, assured a three–factor structure (i.e., bullying, fighting, and victimisation), explaining 50.1% of the variance. All items loaded >.3. Kaiser–Meyer–Olkin Measure of Sampling Adequacy test yielded acceptable results (.746), as well as with the Bartlett's sphericity test ($X^2(153) = 1.67$; $p < .001$) (Damásio, 2012).

3.2.2.3.2 Levenson Primary and Secondary Psychopathy Scales (Levenson et al., 1995)

The LPSP is a 26–item questionnaire designed in a 4–point scale, in which score 1 means disagree strongly and score 4 means agree strongly. It assesses two domains related to psychopathy in adulthood (i.e. primary and secondary psychopathy), being widely used amongst non–clinical samples. Several studies have demonstrated the adequacy of the LPSP in terms of psychometric properties in community samples (Gummelt, Anestis, & Carbonell, 2012; Hauck–Filho & Teixeira, 2014; Salekin, Chen, Sellbom, Lester, & MacDougall, 2014). The scales

were constructed to correspond to Hare's PCL–R factors I and II (Lilienfeld & Fowler, 2006; Salekin et al., 2014). "I let others worry about higher values; my main concern is with the bottom line" and "I don't plan anything very far in advance", are items that assess primary and secondary psychopathy, respectively (Appendix B). Cronbach's alphas were α = .80 for primary psychopathy scale, α = .66 for secondary psychopathy and α =.83 for the total scale.

3.2.2.4 Data analysis

Data were analysed using SPSS 22.0 and JASP version 0.80 software (Love et al., 2016). Data were first checked for normal distribution, followed by the inspection of the outliers. The means and standard deviations were obtained for all the instruments to provide a characterisation of the sample. Two–tailed correlations and multiple linear regressions were used to analyse associations and predictors. To minimise issues due to multiple comparisons (i.e., committing the Type I error) the Benjamini and Hochberg's (1995) suggested method for false discovery rate (FDR) was employed using an electronic calculator (SDM Project Web, 2017). For linear regression, data were transformed using Log^{10} method since normal distribution was not achieved. Mediation analyses were performed using regression procedures and the significance of the indirect effect was tested using bootstrapping with 1000 samples (Baron & Kenny, 1986; Hayes 2013; Jose, 2013). *G**Power version 3.1.9.2 was used to confirm that for all analyses the number of participants was sufficient enough for securing 95% of power and α = .05 or less.

3.3 Results

3.3.1 Descriptive results

Table 3.1 assembles the means and standard deviations for bullying and psychopathy in respect to the participants' gender. Means for primary and secondary psychopathy were interestingly high for a community sample. Gillespie (2014), in a study with violent offenders in the U.K., found similar means for primary and secondary psychopathy of 29.9 (*SD* = 8.6) and 23.1 (*SD* = 4.9) respectively.

For bullying, there was a significant difference between males and females in the perpetrator behaviour, which is in accordance with previous findings within similar contexts (e.g., Baughman et al., 2012) and seems to replicate an overall tendency for males to engage with aggression (Loeber & Hay, 1997).

Table 3.1: *Means and standard deviations for bullying and psychopathy (raw scores)*

Variable	***M***		***SD***		***T***	**Cohen's d (effect size)**
	Male	**Female**	**Male**	**Female**		
Primary psychopathy	29.56	28.57	7.29	6.56	.82	–
Secondary psychopathy	19.87	19.72	4.37	4.14	.20	–
Total psychopathy	49.42	48.29	9.67	9.37	.72	–
Bullying perpetrator	2.58	1.53	2.93	2.07	2.80*	.41 (.20)
Bullying victimisation	1.18	.87	1.77	1.85	1.02	–
Bullying fighting	.47	.30	1.21	.98	.61	–
Total bullying	4.22	2.70	4.88	3.89	2.24	–

Note. * $p < .05$.

3.3.2 Correlational analyses

As shown in Table 3.2, numerous correlations (Pearson; raw scores) were evident between psychopathic personality traits and bullying behaviours. Interestingly, the IBS total score correlated more strongly with LPSP total score (r = .45, p < .001), albeit the links between total bullying (e.g., IBS total) with psychopathic variants were also of moderate strength (r = .41, p < .01, and r = .35, p < .001, for primary and secondary psychopathy, respectively). All IBS' subscales correlated also with LPSP primary and secondary psychopathy. Analyses were also performed with the variable gender controlled, but no major effects were identified (Table 3.2). Moreover, the Benjamini and Hochberg's (1995) suggested method for false discovery rate revealed that all correlations kept statistical significance when examined the effect of multiple comparisons.

Table 3.2: *Correlations between bullying and psychopathy*

		1	2	3	4	5	6	7
1 Primary psychopathy	*r*	–	**.46**	**.92**	**.41**	**.25**	**.29**	**.41**
	p	–	**< .001**	**< .001**	**< .001**	**< .001**	**< .001**	**< .001**
	Upper 95% CI	–	.56	.93	.51	.36	.40	.51
	Lower 95% CI	–	.35	.89	.30	.12	.17	.30
2 Secondary psychopathy	*r*		–	**.77**	**.38**	**.18**	**.22**	**.35**
	p		–	**< .001**	**< .001**	**.005**	**< .001**	**< .001**
	Upper 95% CI		–	.82	.49	.30	.34	.46
	Lower 95% CI		–	.71	.27	.05	.10	.23
3 Total psychopathy	*r*			–	**.46**	**.26**	**.30**	**.45**
	p			–	**< .001**	**< .001**	**< .001**	**< .001**
	Upper 95% CI			–	.56	.37	.42	.54
	Lower 95% CI			–	.36	.13	.18	.34
4 Bullying perpetration behaviour	*r*				–	**.39**	**.38**	**.82**
	p				–	**< .001**	**< .001**	**< .001**
	Upper 95% CI				–	.49	.49	.86
	Lower 95% CI				–	.28	.27	.78
5 Victimisation	*r*					–	**.58**	**.81**
	p					–	**< .001**	**< .001**
	Upper 95% CI					–	.66	.85
	Lower 95% CI					–	.49	.76
6 Fighting	*r*						–	**.72**
	p						–	**< .001**
	Upper 95% CI						–	.78
	Lower 95% CI						–	.65
7 Total bullying	*r*							—

3.3.3 Regression analyses

Regression models were performed to predict bullying involvement (i.e., IBS total score, and subscales of bullying, fighting, and victimisation). The predictors used were gender, primary and secondary psychopathy (Table 3.3). As it can be seen in Table 3, primary psychopathy predicted involvement in all forms of bullying behaviours amongst adults. Additionally, primary psychopathy solely predicted fighting and victimisation, accounting for nearly 10% of the explained variance. Gender and secondary psychopathic traits had incremental effects on the influence of primary traits in predicting perpetrator forms, and contributed in explaining the total involvement with bullying (e.g. the combination of perpetrator/bullying, victimisation, and fighting).

Table 3.3: *Multiple linear regressions examining bullying predictors*

	Perpetrator			Victim			Fighting			Bullying total		
	B	*SE*	*β*	*B*	*SE*	*β*	*B*	*SE*	*B*	*B*	*SE*	*β*
Primary psychopathy	.85	.21	.26*	.70	.19	.25*	.56	.13	.30*	1.24	.26	.31*
Secondary psychopathy	.77	.23	.21*	.14	.21	.04	.08	.14	.04	.66	.29	.15*
Gender	–11	.04	–.14*	–.05	.04	–.08	–.02	.02	–.04	–.12	.06	–.13*
Adjusted R^2	19.3			.08			.09			18.4		
Model fit	$F_{(3,232)} = 19.54$			$F_{(3,232)} = 7.71$			$F_{(3,232)} = 9.38$			$F_{(3,232)} = 18.40$		
	$p < .001$			$p < .001$			$p < .001$			$p < .001$		

Note. * $p < .05$. Durbin–Watson's values have been used to analyse residuals. In this study, the values were appropriated (2.104, 1.975, 1.880, and 2.037 for the perpetrator, victim, fighting and bullying total models, respectively); Root mean square error (RMSE) has been used as an additional metric for model performance and the values were also acceptable (.279, .255, .173, and .346 for the perpetrator, victim, fighting and bullying total models, respectively). Data have been Log^{10} transformed and regression constants were included in all models.

3.3.4 Mediation analyses

To test for mediation effects, several assumptions were tested (cf. Baron & Kenny, 1986; Hayes & Cai, 2007; MacKinnon, Lockwood, & Williams, 2004) in two models predicting bullying/perpetrator: the first having primary psychopathy as a predictor, and the second one having secondary psychopathy as a predictor.

3.3.4.1 Model 1 – Primary psychopathy as independent variable

The first assumption of mediation is that the mediator should predict the dependent variable (bullying/perpetrator form). The results are in line with this requirement ($R^2 = .15$, $\beta = .39$, $p < .001$). In addition, the independent variable (IV) should also predict the mediator. This assumption was confirmed ($R^2 = .16$, $\beta = .41$, $p < .001$). The final condition assumes that when IV and the mediator are included together in the model, the relationship between IV and the dependent variable (DV) declines and the variance explained increases (Jose, 2013). This condition was also supported. Beta's value decreased to .33 and R^2 increased to .25 (Sobel's $z = 3.201$, $p = .001$; 95% CI for the bootstrapped unstandardized indirect effect

ranged from .007 to .051)[14], with .18 indirect to the total ratio effect size. Hence, partial mediation occurred and 18% of the total effect of primary psychopathy on bullying (perpetration) goes through experiences of victimisation (Figure 3.1).

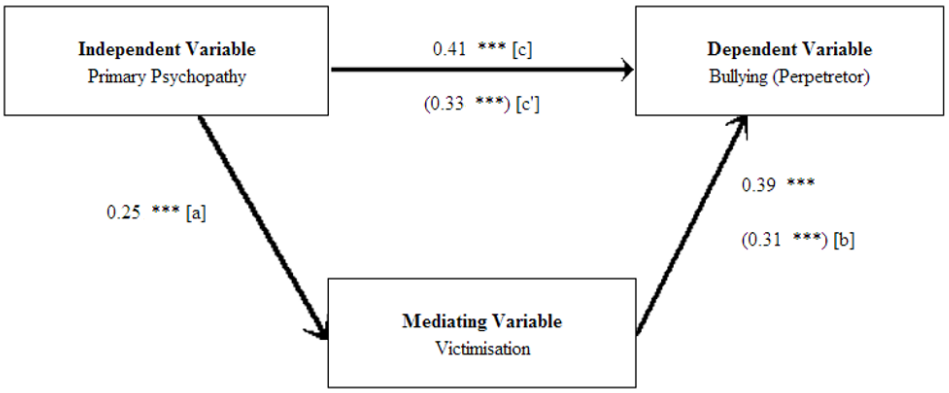

Figure 3.1 – Mediation role of victimisation between primary psychopathy and the perpetrator of bullying.

Note. The values in parentheses are beta weights and the other values correspond to correlations (Pearson).

3.3.4.2 Model 2 – Secondary psychopathy as independent variable

The first assumption of the mediator predicting the DV was achieved (R^2 = .15, β = .39, p < .001). In addition, the IV should also predict the mediator, which was confirmed (R^2 = .14, β = .38, p < .001). The final condition of the mediation effect requires a reduction in the relationship between IV and DV when the mediator variable is included. Here, beta's value decreased to .32 and R^2 increased to .25 (Sobel's z= 2.59, p =.009, with .15 indirect to the ratio effect size; 95% CI for the bootstrapped unstandardized indirect effect ranged from .002 to .079). As with the previous model, partial mediation was detected. Here, 15% of the total effect of secondary psychopathy on bullying (perpetration) goes through the experiences of victimisation (Figure 3.2).

[14] The Bresuch–Pagan test (BP) indicated the existence of heteroskedasticity (BP $_{(3)}$ = 98.49, p < .001), which informed the decision of using heteroskedasticity–consistent standard error estimators for obtaining the 95% CI for the bootstrapped unstandardized indirect effect.

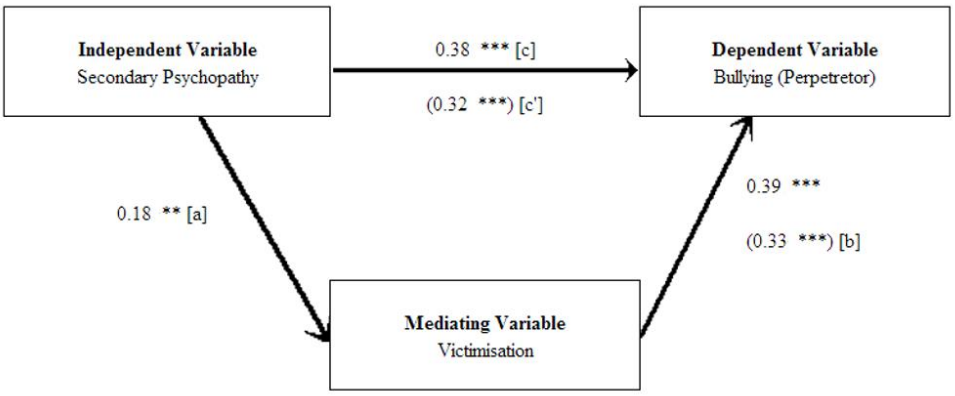

Figure 3.2 – Mediation role of victimisation between secondary psychopathy and the perpetrator of bullying.

Note. The values in parentheses are beta weights and the other values correspond to correlations (Pearson).

3.4 Discussion

This study aimed to comprehend the relationship between primary and secondary psychopathy variants and bullying behaviours in adults, and how far gender and psychopathic variants predict bullying. Additionally, it sought to test for further mediation effects of the experiences of victimisation in the association between bullying and psychopathy. The first hypothesis for this study was that psychopathic personality traits would be positively associated and would positively predict bullying behaviours. Based on data from child and adolescent studies, it was also postulated that the relationship between psychopathic traits and the perpetration of bullying would be mediated by the effects of victimisation (Barker et al., 2008; Fanti et al., 2009).

This study showed that bullying behaviours are associated with psychopathic traits in adults, the relationships between primary psychopathy and bullying behaviours being the strongest. The magnitude of the correlations detected in this study was bigger than previously reported in studies with children and adolescents (r = .27; Van Geel et al., 2016). This could simply reflect a greater autonomy experienced by young adults in regard to expression of aggressive behaviour, combined with less structured (and consequently less contingent) routines. Also, this could reflect the lack of awareness by schools, universities and institutions in terms of bullying dynamics relevant to adult life, which would reflect in diminished intents in preventing and combating adult bullying.

No previous studies were identified in the literature investigating bullying behaviours explicitly and psychopathic personality traits using the LSRP (Levenson et al., 1995), which impedes direct comparisons. However, in accordance with the results and in line with the study's predictions, Coyne and Thomas (2008) found positive relationships between self–reported primary and secondary psychopathy – as measured by the LSRP – with direct and indirect aggression in a study with British college students. Additionally, total psychopathy as measured by the Short D–3 (Paulhus & Williams, 2002) has shown a positive correlation with total bullying in adults (r = .55; Baughman et al., 2012).

Warren (2009) and Williams et al. (2003) also reported positive correlations between direct and indirect bullying with psychopathic personality traits as measured by the PPI–R (Lilienfeld & Widows, 2005) and the SRP–II (Williams et al., 2003), respectively. To the best of my knowledge, these are the only published studies investigating bullying behaviours in adult life in relation with psychopathic personality traits, which is rather surprising giving the relatively abundant evidence that both phenomena impact severely on individual's quality of life (Barker et al., 2008; Bender & Losel, 2011; Chen & Huang, 2015).

In addition, the hypothesis that psychopathy (both primary and secondary) would be a significant predictor of the bullying perpetrator behaviour was supported. These same predictors were also significant in explaining total involvement with bullying; primary psychopathy displayed, however, better explanatory power (e.g., β = .31 (primary psychopathy) versus β = .15 (secondary psychopathy). Consequently, findings do suggest that components of cold–blooded psychopathy could be driving the engagement of this sample with aggressive behaviours (Levenson et al., 1995; Lykken, 2006). Moreover, primary variant alone explained 10% of the experiences of victimisation and fighting, which is different from the pattern detected in a non–forensic sample of adults in the United Kingdom; in this study, secondary traits, not primary, predicted direct and indirect aggression (Warren, 2009). Nonetheless, discrepancies between studies could reflect the use of distinct methods for assessing bullying versus direct and indirect aggression protocols.

For Baron and Kenny (1986), a mediation relationship occurs when one variable influences a dependent variable indirectly through its links with a third mediating variable. Analyses of mediation suggested the role of bullying victimisation in mediating the relationship between psychopathic variants and bullying perpetration. Importantly, the models tested here only partially mediated these relationships, thus indicating that other variables may also increase participants' engagement with bullying perpetration. Arguably, this may be comprehended as dynamics of aggression are under influence of several factors, including temperamental dispositions, environmental contexts, amongst others (Resnick, Bearman, Blum, Bauman, Harris, Jones, & Udry, 1997; Vassallo, Edwards, Renda, & Olsson, 2014). Nonetheless, the significant and positive indirect effects for victimisation on the relationship between primary and secondary psychopathic variants and bullying perpetration have some overlap to prior findings showing that psychopathic features would increase the ratio of victimisation (Fontaine et al., 2016), thus leading to instrumental aggression in adolescents (Fanti et al., 2009) and adults (Glenn & Raine, 2009). Moreover, mediation analyses between psychopathic personality traits (PPI–R; Lilienfeld & Widows, 2005) and indirect aggression were also conducted in another study carried out in a sample of British adults (Warren, 2009). However, the proposed mediator for Warren (2009) was empathy, thus direct comparison is rather difficult to be made.

Positive, but small, correlations between fighting and all aspects of psychopathy, alongside with positive, moderate correlations between bullying (perpetrator) and psychopathic traits were detected. Hence, these participants could present with the absence of fear, one of the "core" components of antisocial behaviour (Jones et al., 2009a; Warren, 2009; Witt, Donnellan, Blonigen, Krueger, & Conger, 2009). These results support the understanding of the failure of individuals with elevated psychopathic traits to respect others' rights, often resulting in aggression and maladjustment (Edens, Poythress, Lilienfeld, Patrick, & Test, 2008; Lykken, 2006). A similar pattern of violation of social norms through the use of physical violence is often detected amongst children involved with systematic episodes of peer aggression (Juvonen & Graham, 2014). Hence, the data reported in this study provides continuity with the conclusions drawn in investigations with the youth population. Relevant to remember that early

behavioural problems, such as bullying, along with juvenile delinquency are diagnostic categories for the 'gold standard' measure of psychopathy (PCL–R; Hare, 2003; cf. Table 1.3).

It was also identified that males showed higher means on IBS total score and in the subscales of fighting, victimisation, and bullying. These findings are in line with bullying literature involving children and adolescents (Nansel et al., 2001) as well with adult samples (Archer, 2004), but could also reflect an unequal proportion of males and females in the current investigation. Nonetheless, in the study about bullying and the dark–triad personality traits carried out by Baughman et al. (2012), males also scored higher than females on bullying involvement. This is consistent with current knowledge regarding more (explicit) aggressive behaviours amongst adult males (Juvonen & Graham, 2014). Fanti et al. (2009) noted that boys had higher risk (β = – .31[15]) for exhibiting bullying behaviour. Moreover, demographic factors such as maternal education were negatively related to bullying (β = –.15), and victimisation was a statistically strong predictor of bullying behaviour (β = .52). Hierarchical models showed also that callous (β = .12) and uncaring (β = .18) subscales of the ICU (Frick, 2004) predicted bullying behaviour above and beyond the effects of victimisation and maternal education.

Although it was not possible to determine whether participants in this study had been involved with bullying since their school years, the results suggest that bullying does occur in adults, and, as similarly seen in children, may be associated with CU traits and with other psychopathic characteristics, such as narcissism and impulsivity (Crapanzano et al., 2011; van Geel et al., 2016; Viding et al., 2009). In addition, one may agree that children with a combination of psychopathic traits and externalising problems, such as bullying, tend to show a worse prognosis if compared with their typical developing peers. This combination is especially important once psychopathy gets worse over time for both males and females which culminates with less responsiveness to interventions (Ribeiro da Silva et al., 2015).

[15] In this study, gender was coded as 1 for boys and 2 for girls (Fanti et al., 2009).

As shown in past research, victims and perpetrators of bullying can be penalised on their abilities to regulate emotions and affective states due to aggression and victimisation experiences (Mahady–Wilton, Craig, & Pepler, 2000). As a child progresses into further developmental stages, the phenomena of 'aging out' may explain the short lives of some forms of aggressive behaviours and their replacement by more sophisticated, planned, and cunning types of conduct (Juvonen & Graham, 2014).

Van Geel et al.'s (2016) meta–analysis results showed an effect of age in the strength of the relationship between narcissistic and impulsive aspects of psychopathy with bullying, suggestive of a greater severity as the individual gets older. Even though I did not measure these psychopathic domains, findings from Study 1 seem to be congruent to those reported by the authors. Hence, it is plausible to consider that children frequently involved with bullying are at risk to perpetuate this pattern of disruptive behaviours into further developmental stages, which might contribute towards 'cycles of violence'. For instance, evidence from longitudinal research suggest that, amongst children aged seven, high levels of psychopathic traits predicted future victimisation (at the age of 14) (Fontaine et al., 2016).

At present, there is very little published work on psychopathy and the experience of victimisation. One previous work by Fanti and Kimonis (2012) reports such an association in young adolescents, and suggests that impulsivity and narcissism are likely to contribute towards an individuals' actual and perceived victimisation. Similarly, Fontaine et al. (2016) indicated that children with early psychopathic traits could benefit from bullying prevention programmes, mainly because this group is at higher risk for becoming targeted during early and mid–adolescence. My investigation was not able to examine this premise in any more detail other than the possible mediation effect, and it was not the initial scope of this study to examine rates of victimisation in adults, but this is clearly an interesting avenue for future investigation.

It is relevant to note that cross–sectional designs can account for multiple ways of defining the relationships for mediation analyses, and variables in these scenarios can exert mutual effects (e.g., each variable can act as a mediator on the

links between the remainder variables; Judd & Sadler, 2008; Roe, 2012). More recent perspectives of the procedure of "reverse mediation" (e.g., Lemmer & Gollwitzer, 2017) indicate that the process of comparing alternative models on an "ad hoc" basis, in which each variable is tested (e.g., X–> M–> Y versus X –> Y –> M), can likewise induce to error. In most cases, a most firm decision on the causal ordering of the independent variables, the mediators, and the dependent variables would require experimental control and/or temporality (Wiedermann & von Eye, 2015). In this sense, the results section of this study should be taken in the light of its limitations and in the perspective that alternatives models might likewise hold true (Lemmer & Gollwitzer, 2017).

An additional – and important remark – is that in the case of cross–sectional data (used in this study), the conditions of both isolation and direction in mediation analyses can be considered only theoretically. This occurs due to statistical difficulties for assessing direction, coupled with the fact that reverse causation can be discarded only after thoughtful theoretical examination (Wiedermann & von Eye, 2015). In this perspective, it is important to examine the "timing" of the variables[16], without forgetting that these conclusions can be complex and, in most cases, controversial (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). A related approach to examine a mediation proposition is to test the indirect effect of the independent variable via the mediator on the dependent variable against zero (Lemmer & Gollwitzer, 2017). Albeit the Sobel's test is frequently used in psychological research, novel (and more robust) methods have been suggested. Several authors have included resampling methods – including the bias–corrected bootstrap method – as the procedure of choice (Hayes, 2013; MacKinnon et al., 2002; MacKinnon et al., 2004; Pieters, 2017). As such, future studies examining and/or trying to replicate the findings reported in this chapter should consider these methodological implications.

Further work could benefit from trying to corroborate findings here presented by addressing these phenomena beyond the cross–sectional approach. A wealthy amount of research with child and adolescent samples might inspire longitudinal examinations of bullying dynamics and its links with psychopathy.

[16] According to Tate (2015), values of the predictor must exist before the values of the mediator, and the values of the mediator(s) must exist in time before the values of the outcome.

Another limitation of this study that could be taken into account in future research is regarding the use of self–report measures, especially for psychopathy. Extending the age range covered, inclusion of covariates, as well as comprising an equal proportion of males and females would certainly contribute to obtain a clearer view into the nature of the relationship between bullying and psychopathic personality traits.

Again, the adoption of robust designs in combination with a strong methodological assessment should inform the casual paths and, therefore, could be of use in explicitly detecting risk and protective factors (Coolican, 2014). Nonetheless, given the relatively early stages of bullying research in adulthood, the use of qualitative approach in gathering and analysing data could bring resourceful information on peculiar dynamics of aggression and victimisation, and consequently supporting with action points that could be implemented in intervention and prevention programmes in schools and universities.

3.5 Study 2 – Bullying involvement and psychopathic personality traits: Disentangling the links amongst college students

In Study 1, interesting findings were presented in respect to the association between bullying behaviours with primary and secondary variants of the psychopathic personality. The second study of this thesis, 'Bullying involvement and psychopathic personality traits: disentangling the links amongst college students'[17], was designed with the intent to further examine the role of psychopathy in bullying dynamics, specifically via participants' involvement with these behaviours. Precisely, the current investigation proposes to compare psychopathic personality traits in relation to participants' roles in bullying, giving continuity to previous work conducted with children. Claims for detailed, sophisticated measurements of bullying were made by Sutton and Keogh (2000) when suggesting methodological guidelines for investigations exploring differential associations between bullying and psychopathy–linked constructs, such as machiavellianism. The authors argued that larger sample sizes could help

[17] Manuscript accepted for publication in the *European Journal of Education and Psychology*.

in the comprehension of important differences in psychopathic personality traits for bullies, victims, and bully/victims. For these scholars, behaviourally manipulative styles were expected to domain youth who bully, while pessimistic styles and poor trust in others were assumed to characterise attitudinal levels of those victimised (Sutton & Keogh, 2000). Hence, for the purpose of this study, measuring a variety of psychopathic personality traits was preferred over methods restricted to psychopathic variants, such as primary and secondary. This was done in order to further examine the links possibly existing between psychopathic traits and bullying behaviours. Amongst the self–report measures of psychopathy designed for use in non–clinical samples, the Psychopathic Personality Inventory Genetic Derived form is a recently developed tool, capturing the core personality traits related to this condition (PPI–R–40; Eisenbarth et al., 2015). However, Ruchensky et al. (2017) highlighted that, although promising, it is imperative to perform further examinations on the psychometric properties of the PPI–R–40 in other samples and to explore its associations to external correlates. It is allusive to highlight that it is not rare to examine the predictive value of psychopathic measures by exploring their associations with aggressive behaviours (Warren, 2009) and bullying (Williams et al., 2007). Thus, this study aims to also investigate the utility of the PPI–R–40 in exploring its associations to negative outcomes in a community sample of college students. Therefore, correlations between PPI–R–40 subscales will be investigated regarding bullying perpetration and bullying victimisation.

3.5.1 Hypotheses

Perpetrators of bullying and individuals high on psychopathic traits have more risk for experiencing aggression and victimisation (Fontaine et al., 2016). Following on this, and aligned to the results from the previous study, Study 2 predicts that there would be significant, positive associations between self–reported bullying and psychopathic personality traits. Moreover, child and adolescent data showed that the co–occurrence of bullying and victimisation (i.e., bully–victims) resulted in higher levels of self–report measures of psychopathy (Ragatz et al., 2011). Consequently, the key prediction for this study is that bullying–victims would

show elevated psychopathic traits when compared to those belonging to the other bullying groups (Fanti & Kimonis, 2013; Sutton & Keogh, 2000).

3.5.2 Method

3.5.2.1 Participants and design

The sample in this cross–sectional study comprised 273 college students (M_{age} = 25.5, *SD* = 6.1). The study received ethical approval from the Goldsmiths Psychology and London South Bank University Research ethics committees.

3.5.2.2 Procedures

Participants were recruited via a Research Participation Scheme at two universities in the U.K. Prior to completing the questionnaires, participants were presented with an outline of the study, which included information on data protection and privacy, and they were asked to provide consent for participation. Participants received a debriefing for this study, along with the appropriate number of credits for their participation.

3.5.2.3 Measures

3.5.2.3.1 Psychopathic Personality Inventory Genetic Derived form (PPI–R–40; Eisenbarth et al., 2015);

The PPI–R is a self–report questionnaire arranged on a 4–point Likert scale, assessing a variety of domains associated to antisocial behaviours and psychopathy. The measure is assumed to capture the impulsive aspects of psychopathy, as well as its affective and interpersonal components (Benning et al., 2003; Neumann, Malterer, & Newman, 2008)[18]. Recently, an alternative 40–items solution was presented by Eisenbarth et al. (2015), and these items were analysed for this study. Overall, adequate results of internal consistency were obtained (α_{range} = .60 – .79).

[18] The authors of this proposed shorter version noted that measurements of other constructs associated with psychopathy - combining externa criteria such as interviews and physiological data - are needed establish a robust affinity of the PPI–R–40 with the original PPI–R (Eisenbarth et al., 2015).

3.5.2.3.2 Illinois Bullying Scale (IBS; Espelage & Holt, 2001);

This measure was described in section 3.2.2.3.2. For study 2, alphas were again satisfactory (α_{range} = .73 – .81).

3.5.2.4 Data analysis

To respond to the research aims, the data analytic strategy differs from Study 1 mainly due to its focus in comparing levels of psychopathic personality traits in relation to bullying groups. For instance, previous research showed unique patterns of emotional and behavioural problems amongst bullies, bully–victims, pure victims and non–involved (Craig, 1998; Copeland et al., 2013; Sourander et al., 2007). Hence, in Study 2, 4 groups were created, namely: non–involved, pure victims, pure bullies, and bully–victims. These groups were also used in an investigation which compared psychopathic personality traits amongst children who were pure victims, pure bullies, bully–victims and non–involved in bullying behaviours (Sutton & Keogh, 2000). For the behaviours of bullying others, the IBS's subscales of bullying and fighting were combined into one composite ('bullying'). As bullying requires repetition, participants were coded into one category only when reported '2 or more times' in the incidence of bullying others or being victimised in the past 30 days. Those who reported 2 or more times of engagement in concomitant bullying of others and of being a victim were grouped as 'bully–victims'.

As in Study 1, correlations (Pearson) were used to better explore the links between bullying and psychopathy, however here exploring specifically the dimensions captured by the PPI–R–40. Likewise, the Benjamini and Hochberg's (1995) suggested method for false discovery rate (FDR) was employed to these correlational analyses. For group analyses, ANOVA was used to compare non–involved, pure victims, pure bullies, and bully–victims in regard to psychopathic traits (Coolican, 2014). In studies involving unequal cell sizes, the assumptions for conducting ANOVA's must be carefully examined (in particular, those related to violations of normality and equality of variances; Haslam & McGarty, 2014). As

such, both the classical Levene's test and more robust procedures were conducted, particularly the Brown–Forsythe test (Boos & Brownie, 2004; Brown & Forsythe, 1974; Wang et al., 2017)[19].

3.6 Results

3.6.1 Descriptive results

Around half (50.9%; *n* = 139) of participants reported not having been involved with any behaviour related to bullying over the past month. The remainder, 49.1%, of participants reported that they had been involved in some sort of bullying, namely: 25.3% (*n* = 69) reporting having bullied someone two or more times and were not themselves victims in the last month; 4.0% (*n* = 11) were victims exclusively; 19.8% were bullies/victims (*n* = 54).

3.6.2 Inferential analyses

In order to present a complete picture of the links between bullying and the psychopathic personality, a full correlation matrix is presented in Table 3.4. 95% confidence intervals were provided due to the multiple comparisons. Another reason for fully reporting correlational findings is because this type of procedure is important and informative for potential future inclusion in meta–analytical studies (Schmidt & Hunter, 2014). However, a text–description will be followed, highlighting those associations most relevant to the current work. When using the Benjamini and Hochberg's (1995) method for false discovery rate, all the significant associations reported in Table 3.4 kept their significance below *p* .05.

In respect to the requirements for comparing means using ANOVA's, the dependent variables (PPI–R–40 subscales and factors) were normally distributed (e.g., Kurtosis ranging from –1.08 to –.17, and skewness between –.25 to .72; Hair, Black, Babin, & Anderson, 2010). Levene's test calculations revealed no significant

[19] Wang et al. (2017) reported a simulation study which examined the performance of methods for assessing the assumptions for homogeneity of variance in one–way ANOVA's. Several criteria were taken in consideration in the analyses, including the number of groups, patterns of sample sizes in each group, and patterns of population variances. In terms of Type I error rates, better performance was found for the Levene's test with square deviations and for the Brown–Forsythe test. For statistical power, all the examined procedures returned similar results, except for the Levene's test with square deviations which had less power than the O'Brien test, Ramsey test, and Brown–Forsythe test (Wang et al., 2017).

violations of the homogeneity of variances (all *p*'s > .05; Haslam & McGarty, 2014), which encouraged the conduction of analysis of variance. Table 3.5 displays the results of multiple 4 (bullying roles) x 1 (psychopathy subscales) ANOVA's that were run to detect possible differences between bullying roles regarding PPI–R–40 subscales, factors, and total scores. The significant effects were submitted to further processing due to unequal cell sizes (Wang et al., 2017). As such, *p* values depicted in the Table 3.5 are according to the Brown–Forsythe test.

Table 3.4: *Correlations between psychopathic personality traits with bullying and victimisation*

		1	2	3	4	5	6	7	8	9	10	11	12	13
1 Blame externalisation	*r*	–	.05	**.15**	**.36**	**.31**	–.08	**–.23**	**.32**	–.05	**.74**	**–.12**	**.28**	**.35**
	p	–	.34	**.01**	**< .001**	**< .001**	.15	**< .001**	**< .001**	.42	**< .001**	**.04**	**< .001**	**< .001**
	Upper 95% CI	–	.18	.27	.47	.42	.03	–.11	.43	.07	.79	–.00	.39	.45
	Lower 95% CI	–	–.06	.03	.25	.19	–.21	–.34	.21	–.17	.68	–.24	.16	.24
2 Carefree Nonplanfulness	*r*		–	**.13**	.11	**.22**	–.03	–.02	**.38**	.05	**.53**	**.26**	**.15**	.05
	p		–	**.03**	.07	**< .001**	.60	.64	**< .001**	.43	**< .001**	**< .001**	**.01**	.38
	Upper 95% CI		–	.25	.23	.33	.09	.09	.48	.17	.62	.37	.27	.17
	Lower 95% CI		–	.01	–.01	.10	–.15	–.15	.27	–.07	.44	.14	.02	–.06
3 Fearlessness	*r*			–	**.25**	**.47**	**.20**	**.18**	**.67**	**.72**	**.26**	.10	**.17**	**.16**
	p			–	**< .001**	**< .001**	**.001**	**.003**	**< .001**	**< .001**	**< .001**	.10	**.007**	**.009**
	Upper 95% CI			–	.36	.56	.31	.30	.74	.77	.38	.22	.28	.28
	Lower 95% CI			–	.13	.37	.07	.06	.60	.65	.15	–.02	.04	.04
4 Machiavellian Egocentricity	*r*				–	**.43**	**.21**	**–.12**	**.51**	**.17**	**.73**	**.20**	**.33**	**.14**
	p				–	**< .001**	**< .001**	**.04**	**< .001**	**.004**	**< .001**	**< .001**	**< .001**	**.02**
	Upper 95% CI				–	.52	.32	–.00	.59	.29	.78	.32	.44	.26
	Lower 95% CI				–	.32	.09	–.24	.41	.05	.66	.08	.22	.02
5 Rebellious Nonconformity	*r*					–	**.27**	**.16**	**.74**	**.44**	**.47**	**.17**	**.30**	**.19**
	p					–	**< .001**	**.01**	**< .001**	**< .001**	**< .001**	**.005**	**< .001**	**.002**
	Upper 95% CI					–	.38	.27	.79	.54	.56	.29	.41	.31

Table 3.4: *Correlations between psychopathic personality traits with bullying and victimisation*

		1	2	3	4	5	6	7	8	9	10	11	12	13
	Lower 95% CI						–	.15	.03	.68	.34	.37	.05	.18	.07
6 Social Influence	*r*							–	**.38**	**.51**	**.70**	.04	.10	**.17**	–.01
	p							–	**< .001**	**< .001**	**< .001**	.54	.10	**.005**	.83
	Upper 95% CI							–	.48	.59	.76	.16	.22	.29	.11
	Lower 95% CI							–	.27	.41	.63	–.08	–.02	.05	–.13
7 Stress Immunity	*r*								–	**.42**	**.69**	**–.20**	**.20**	–.03	–.03
	p								–	**< .001**	**< .001**	**.001**	**.001**	.60	.60
	Upper 95% CI								–	.52	.75	–.08	.32	.09	.09
	Lower 95% CI								–	.31	.62	–.31	.08	–.15	–.15
8 Total Psychopathy	*r*									–	**.77**	**.59**	**.43**	**.36**	**.20**
	p									–	**< .001**	**< .001**	**< .001**	**< .001**	**.001**
	Upper 95% CI									–	.82	.67	.52	.46	.32
	Lower 95% CI									–	.72	.51	.32	.25	.08
9 Fearlessness factor	*r*										–	.08	**.18**	**.15**	.07
	p										–	.19	**.003**	**.01**	.25
	Upper 95% CI										–	.20	.30	.27	.19
	Lower 95% CI										–	–.04	.06	.03	–.05
10 Self–Centred Impulsivity factor	*r*											–	**.15**	**.38**	**.28**
	p											–	**.01**	**< .001**	**< .001**

Table 3.4: *Correlations between psychopathic personality traits with bullying and victimisation*

		1	2	3	4	5	6	7	8	9	10	11	12	13
	Upper 95% CI										–	.27	.48	.39
	Lower 95% CI										–	.03	.27	.17
11 Coldheartedness factor	*r*											–	.08	–.08
	p											–	.16	.17
	Upper 95% CI											–	.20	.03
	Lower 95% CI											–	–.03	–.20
12 Bullying behaviour	*r*												–	**.50**
	p												–	**< .001**
	Upper 95% CI												–	.58
	Lower 95% CI												–	.40
13 Victimisation	*r*													–

Table 3.5: *Comparing the means for psychopathic personality traits and bullying roles*

Groups		M	*SD*	*SE*	Lower 95% CI	Upper 95% CI	Z (*p*)	n^2	Post–hoc
Blame Externalisation	Non–involved (1)	9.13	2.85	.24	8.65	9.62	9.72 (.001)	.10	4>3>1*
	Pure victim (2)	11.18	3.86	1.16	8.58	13.78			
	Pure bullies (3)	10.56	3.20	.40	9.75	11.36			
	Bully–Victims (4)	11.69	2.92	.43	10.81	12.57			
	Total	10.03	3.15	.19	9.64	10.42			
Carefree Nonplanfulness	Non–involved (1)	8.49	2.48	.21	8.06	8.91	1.73 (.161)	.02	–
	Pure victim (2)	9.00	2.68	.80	7.20	10.80			
	Pure bullies (3)	9.27	2.83	.35	8.56	9.98			
	Bully–Victims (4)	9.22	2.61	.39	8.44	10.01			
	Total	8.83	2.62	.16	8.51	9.16			
Fearlessness	Non–involved (1)	10.79	4.03	.34	10.10	11.48	1.87 (.135)	.02	–
	Pure victim (2)	11.64	4.65	1.40	8.51	14.76			
	Pure bullies (3)	11.17	4.12	.51	10.14	12.21			
	Bully–Victims (4)	12.42	3.77	.56	11.29	13.56			
	Total	11.21	4.06	.25	10.71	11.72			
Machiavellian Egocentricity	Non–involved (1)	9.37	2.45	.21	8.95	9.78	9.12 (.001)	.10	4>3>1*
	Pure victim (2)	9.36	3.04	.91	7.32	11.41			
	Pure bullies (3)	10.62	2.84	.35	9.90	11.33			
	Bully–Victims (4)	11.60	2.91	.43	10.72	12.48			
	Total	10.08	2.79	.17	9.73	10.42			
Rebellious Nonconformity	Non–involved (1)	9.61	2.78	.24	9.14	10.09	5.80 (.001)	.06	4>1*
	Pure victim (2)	9.82	3.18	.96	7.68	11.96			
	Pure bullies (3)	10.48	2.90	.36	9.75	11.21			
	Bully–Victims (4)	11.60	2.84	.42	10.74	12.46			

Table 3.5: *Comparing the means for psychopathic personality traits and bullying roles*

	Total	10.19	2.92	.18	9.83	10.55			
Social Influence	Non–involved (1)	12.75	3.16	.27	12.21	13.29	2.86 (.06)	.03	–
	Pure victim (2)	11.36	3.64	1.09	8.92	13.81			
	Pure bullies (3)	13.65	2.78	.35	12.95	14.35			
	Bully–Victims (4)	13.62	3.02	.45	12.71	14.53			
	Total	13.07	3.10	.19	12.68	13.45			
Stress Immunity	Non–involved (1)	12.13	3.11	.26	11.60	12.67	.29 (.833)	.00	–
	Pure victim (2)	12.18	3.76	1.13	9.65	14.71			
	Pure bullies (3)	11.83	3.13	.39	11.03	12.62			
	Bully–Victims (4)	11.71	2.85	.42	10.85	12.57			
	Total	11.98	3.09	.19	11.60	12.37			
PPI–R–40 Total	Non–involved (1)	82.19	11.53	.99	80 .22	84.17	8.90 (.001)	.09	4>3>1*
	Pure victim (2)	82.73	13.92	4.19	73.37	92.08			
	Pure bullies (3)	87.44	11.95	1.50	84.43	90.45			
	Bully–Victims (4)	91.89	10.80	1.61	88.64	95.14			
	Total	85.25	12.15	.76	83.74	86.75			
Fearlessness Factor	Non–involved (1)	35.67	7.26	.62	34.43	36.91	1.06 (.363)	.01	–
	Pure victim (2)	35.18	9.87	2.97	28.55	41.82			
	Pure bullies (3)	36.65	7.08	.89	34.87	38.44			
	Bully–Victims (4)	37.76	6.82	1.01	35.71	39.80			
	Total	36.26	7.27	.45	35.36	37.17			
Coldheartedness factor	Non–involved (1)	8.96	2.54	.22	8.52	9.39	1.47 (.222)	.01	–
	Pure victim (2)	7.73	2.10	.63	6.32	9.14			
	Pure bullies (3)	9.32	2.62	.33	8.66	9.98			
	Bully–Victims (4)	9.42	3.08	.46	8.50	10.35			
	Total	9.08	2.65	.16	8.75	9.40			

Table 3.5: *Comparing the means for psychopathic personality traits and bullying roles*

Self–Centred Impulsivity Factor	Non–involved (1)	26.99	5.24	.45	26.09	27.88	14.07 (.001)	.14	4>3>1*
	Pure victim (2)	29.55	5.95	1.79	25.54	33.55			
	Pure bullies (3)	30.44	5.89	.74	28.96	31.93			
	Bully–Victims (4)	32.51	4.98	.74	31.01	34.01			
	Total	28.94	5.80	.36	28.22	29.66			

Note. * $p < .05$. As noted in the Section 3.5.2.4, investigations comparing groups with unequal cell sizes using ANOVA's must employ powerful methods for assessing any possible violation in the homogeneity of variances (Wang et al., 2017). Consequently, a subsequent inspection of the results from the Brown–Forsythe test was performed, and the results indicated that, with the exception of Social Influence (old p value = .04; new p value = .06), all other statistically significant differences survived this analysis (p values ranging from <.001 to .002).

With the exception of Carefree Non–Planfulness, Fearlessness, Stress Immunity and Social Influence, all other psychopathic domains measured by the PPI–R–40 were positively correlated with victimisation (r_{range} = .07 – .35). This means that as more participants were victimised, the more they reported levels of Blame Externalisation, Machiavellianism, Rebellious Nonconformity, Self–Centred Impulsivity factor and total psychopathy (i.e., PPI–R–40 total score). On the other hand, Carefree Non–Planfulness, Fearlessness, and Stress Immunity were the dimensions of psychopathy which did not reach significant levels of association with the perpetration of bullying. Hence, higher self–reporting of bullying others was linked with higher levels of Blame Externalisation, Machiavellianism, Rebellious Nonconformity, Self–Centred Impulsivity factor, Social Influence, and total psychopathy (r_{range} = .08 – .38; Table 3.4).

Analyses of variance (ANOVA) demonstrated significant differences between participants' involvement with bullying for Blame Externalisation, Machiavellian Egocentricity, Rebellious Nonconformity, Total Psychopathy and Self–Centred Impulsivity factor (n^2_{range} = .03 – 14; Table 3.5). Bonferroni post–hoc tests revealed that, for Blame Externalisation and Machiavellian Egocentricity, participants uninvolved in bullying differed significantly from pure–bullies and from bully–victims in these subscales. Same pattern was observed for PPI–R–40 total score and for the Self–Centred Impulsivity factor. For Rebellious Nonconformity,

differences were statistically significantly different between non–involved and bully–victims only.

3.7 Discussion

This study sought to compare the levels of psychopathic traits in regard to participants' roles in bullying. In addition, possible associations were also examined between bullying, victimisation and psychopathic personality traits as measured by the PPI–R–40. In line with the predictions, bully–victims scored higher on all domains assessed by the PPI–R–40 with the exception of Carefree Non–Planfulness, Stress Immunity and Social Influence, in which pure bullies scored slightly above bully–victims. These results speak to those presented by Ragatz et al. (2011) who found that bully–victims presented significantly higher scores for psychopathy, as well as for criminal thoughts, proactive aggression, and criminal offenses than those who neither were bullied nor perpetrated bullying acts. In addition, those who were bully–victims were more prone for reactive aggression than perpetrators or victims alone. Proactive aggression can be characterised as occurring in a pre–arranged and insensitive manner towards the victim(s), while reactive aggression occurs as an impulsive act in response to provocation (Dodge, 1991; Ragatz et al., 2011).

Regarding the prediction that participants would differ in terms of psychopathic personality traits according to bullying roles, interesting results emerged, specifically for Blame Externalisation, Machiavellian Egocentricity, Rebellious Nonconformity, Social Influence, Self–Centred Impulsivity factor and total psychopathy (Table 3.5). Post–hoc analyses revealed differences in scores between uninvolved, pure–bullies and bully–victims for Blame Externalisation, Machiavellian Egocentricity, Self–Centred Impulsivity and PPI–R–40 total score. In these cases, bully–victims scored significantly higher. According to Losey (2011), those psychopathic traits are linked with negative emotions, such as resentment and even retaliatory behaviours. Additionally, Blame Externalisation was the strongest variable associated with juvenile law–breaking in previous research (DeLisi, Angton, Vaughn, Trulson, Caudill, & Beaver, 2014). Thus, some victims of bullying might display aggressive behaviour as retaliation for the aggression

suffered. In this way, victimisation can be a triggering factor for bullying others (DeCamp & Newby, 2015). This suggests an explanatory hypothesis to the phenomenon found in this study that nearly a fifth of the respondents (19.8%) were both perpetrators and victims of bullying. With effect, DeCamp and Newby (2015) reported that victims of bullying are at special risk for aggressive behaviour because of a previous history of victimisation and violence exposure. However, the authors emphasised that this issue has not yet been fully clarified, possibly because the vast amount of research on bullying has considered only its manifestation in youth. Thus, this study has revealed that adults can also be perpetrators and victims of bullying, and has shown that specific facets of psychopathic personality play an important part on this manifestation. These results, when combined with previous work showing that youth high on psychopathic traits are longitudinally at higher risk for being victimised (Fontaine et al., 2016) are suggestive of pervasive, stable, and deleterious effects of psychopathic personality traits on individuals' social relations.

When considering that bullying is a relational phenomenon with the imbalance of power being one of the key criteria (Rodkin et al., 2015), differences detected in this study between pure bullies and bully–victims in comparison to uninvolved and pure victims, combined with correlational findings, seem to have theoretical reasoning and confirm past reports. For instance, previous work has shown a trend towards violence and delinquency behaviours amongst pure–bullies and bully–victims (Carbone–Lopez, Esbensen, & Brick, 2010; DeCamp & Newby, 2015; Higgins, Khey, Dawson–Edwards, & Marcum, 2012). Supporting these findings, the results evaluated in this study suggested that concomitant perpetrators and victims of bullying tended to violate social rules (i.e., Rebellious Nonconformity) and to equally not take responsibilities for their acts (i.e., Blame Externalisation), whereas pure bullies were more likely to exert power over others (i.e., Social Influence). For example, these people tended to score higher for questions like 'I do not care about following the rules' and 'I make my own rules as I go along' of the PPI–R–40.

Machiavellian Egocentricity was more strongly related to the bullying behaviour than to victimisation (cf. Table 3.4). The literature reveals that pure

bullies tend to be more manipulative, insensitive, and less empathic than pure victims (Sutton et al., 1999a). This suggests a greater tendency for psychopathic behaviours amongst those engaged exclusively in bullying others.

Machiavellianism, in addition, has been also associated to successful psychopathy (Brankley & Rule, 2014), a variation in the presentation of the disorder commonly seen amongst college students (Warren, 2009). In turn, Blame Externalisation correlated positively with behaviours of bullying others, but had a stronger relationship with being a victim, indicating that people who were victims of bullying tended to blame others more often. Although direct comparisons with previous studies are not possible due to conceptual and methodological issues, these correlational results are congruent to those presented by Warren (2009), in which Machiavellian Egocentricity, Blame Externalisation and Rebellious Nonconformity were positively linked with indirect aggression. Sutton and Keogh (2000) reported higher levels of machiavellianism (as measured using the Kiddie Mach scale [KMscale]; Christie & Geis, 1970) in children who were predominantly bullies (M_{KMscale} = 60) and bully–victims (M_{KMscale} = 61.7) when compared to non–involved youth (M_{KMscale} = 48.40).

One hypothesis that might be raised is that perhaps the overlap between bullying and psychopathy is related to specific traits that usually load into PPI's analogous Factor 2 (Self–Centred Impulsivity), comprising the subscales of Blame Externalisation, Machiavellian Egocentricity, and Rebellious Nonconformity. Even though data here presented cannot firmly state this, future work could explore the overlap between both phenomena, once identifying common features could result into convergent, more effective interventions.

It shall be noted, however, that it is not only psychopathy, but also other types of personality disorders, that may prone individuals to display behaviours of non–conformity to social norms, such as narcissist personality disorder, borderline personality disorder, and paranoid personality disorder (Cleckley, 1941; Koeppel, Boutwell, & Barnes, 2015; McMains & Mullins, 2014). Therefore, caution is warranted in interpreting these results, although programmes aiming to reduce the overall impact of bullying behaviours on individuals and communities should consider all these nuances seriously. In the same direction, the results present

numerous limitations due to use of cross–sectional design, which impedes the establishment of casual relationships, and by relying on self–report measures. Moreover, analyses of group differences were exploratory and, given unequal cell sizes, must be interpreted with caution (Sutton & Keogh, 2000). Additionally, possible immediate applications of these findings are limited to settings with similar cultural and demographical characteristics.

Given that bully–victims show a number of important differences when compared with bullies, it is not at all surprising to find that bully–victims are distinguishable from victims in their psychopathic traits (Ragatz et al., 2011). Therefore, data evaluated in this study suggested that specific traits of the psychopathic personality – especially Machiavellian Egocentricity, Blame Externalisation and Rebellious Nonconformity – are important in the comprehension of the bully–victim relationship. These findings are coherent with those detected in child and adolescent investigations. For instance, a recent meta–analysis including more than 40.000 participants found positive links between domains of youth psychopathy and bullying behaviours. Interestingly, impulsivity and narcissism were largely associated with bullying amongst older adolescents, whereas no age influences were detected for the links between CU traits and bullying (van Geel et al., 2016).

3.8 Chapter summary

Developmental science has made important efforts towards the comprehension of the stability of bullying dynamics, especially in the perpetrator form. It is estimated that around 10% of severely aggressive children will continue to intimidate others as they 'age out' (Juvonen & Graham, 2014). Due to limited investigation on potential factors associated with adult bullying, much is yet to be realised. Together, studies 1 and 2 emphasise the role of psychopathic traits and bullying behaviours as reciprocal risk factors for emotional and behavioural difficulties, which place them as important issues. Hence, the results resonate with Warren's assertion, that when stated "it is arguable that psychopathy may play a role in predicting involvement in bullying" (Warren, 2009, p. 245). A possible

explanation for this might be that the combination of affective and interpersonal characteristics puts individuals at risk to bully others and, in turn, to become the targets of systematic episodes of aggression (Fontaine et al., 2016). Another explanation could be partly explained by genetic influences. For instance, behavioural genetic investigations have shown that the heritability of the behaviour of bullying others was around 61% (Vlachou et al., 2011), whereas for conduct problems a recent report indicated a genetic influence of 73% (Pingault, Rijsdijk, Zheng, Plomin, & Viding, 2015). In regard to victimisation, one understanding is that, as seen in children and adolescents, those with higher psychopathic traits might experience social exclusion and could eventually become the target of aggressive acts due to an inability to conform to social norms, to respect others, and to display prosocial behaviours (Fontaine et al., 2016).

Nonetheless, the consequences of being involved in bullying in all forms can have long–lasting effects (Baughman et al., 2012; Juvonen & Graham, 2014). Thus, psychopathic traits and bullying involvement may be possible a cause of aggressive behaviour experienced during childhood and adolescence, suggesting a developmental pattern of aggression from childhood to young adulthood (DiLalla & Bersted, 2015). In face of this, results have the potential to support targeted interventions in educational settings. In this sense, schools, universities and colleges might aim to develop preventive programmes to attenuate the consequences of adult bullying. Interventions may be especially important to break the cycle of violence in which participants could have been involved in for a long time. Many individuals high on psychopathic traits see universities, colleges and institutions as "stepping points" for success (Gao & Raine, 2010). In addition, some types of bullying behaviours displayed in adulthood may be classified under criminal laws and, therefore, have conceivable application in forensic realms (Limber & Small, 2003; Wolke & Lereya, 2015; Zapf, Escartín, Einarsen, Hoel, & Vartia, 2011).

Nevertheless, even though aggressive behaviour, bullying and psychopathy are frequently associated together, it is important to highlight that they do not associate as a unitary construct. This distinction has theoretical and possibly practical meaning since reports on bullying prevention and intervention tend to

return satisfactory outcomes, which is not often the case for programmes targeting the effects of psychopathic personality traits (Ribeiro da Silva et al., 2015; Sutton et al., 1999a, 1999b). Although these studies present limitations due to the cross–sectional design, these are amongst the first investigations to examine the role of psychopathic personality traits in adult bullying, and provide continuity from the report on child and adolescent data. The magnitude of the correlations between bullying and both psychopathic variants detected in study 1 was bigger than the average reported (r = .27; van Geel et al., 2016) in studies with children and adolescents, which could inform about greater severity of these behaviours in adult life. While statistical analyses were adjusted for the sex ratio, in the current investigations it was not possible to obtain equal proportion of males and females. Another limitation is that these results are partially true for English speaking individuals, predominantly from the U. K. Hence, future work might benefit from including a large number of males, as well presenting data from different countries. Additionally, some biological and contextual mechanisms underpinning both phenomena might also play an important role in both eliciting and maintaining high levels of aggression and antisocial behaviour, which is certainly a meritorious avenue for future research. Within the current thesis, these will be explored more deeply in Chapter Six, which investigates precisely the role of neuroendocrine reactivity as a proxy for biological vulnerability to aggression, bullying, and psychopathic tendencies.

CHAPTER FOUR – PSYCHOPHYSIOLOGICAL INVESTIGATIONS ON PSYCHOPATHIC PERSONALITY TRAITS, AGGRESSIVE, AND ANTISOCIAL BEHAVIOURS IN ADULTS

Chapter overview

In the previous chapters, an emphasis was given to emotional and behavioural characteristics associated with psychopathy and with psychopathic personality traits. The interest in biological processes underpinning antisociality, although inherent for this thesis, is also shared amongst many scholars in the field of developmental psychopathology, aggression, biosocial criminology, and psychophysiology of AB (Hare, 2001; Raine, 2001). Therefore, investigations in these areas have proliferated, conducted with the intent of identifying possible biological processes that might help in the accurate identification of psychological disorders and their subsequent management (Portnoy & Farrington, 2015). As discussed in Chapter Two, there are dozens of recent publications looking for various biological mechanisms associated with AB and psychopathy, the most dominant method being psychophysiological research, conducted with child and adolescent samples (De Vries–Bouw et al., 2011; Gatzke–Kopp et al., 2015; Kyranides et al., 2016; Posthumus et al., 2009; Raine et al., 2014; Scarpa et al., 2010; Sijtsema et al., 2015), and studies with adults (Casey et al., 2013; Gao et al., 2012; Lobbestael & Arntz, 2010; Rothemund et al., 2012; Sylvers et al., 2010). Amongst these, reports on the links between RHR and psychopathy are numerous, but much more has to be studied in respect to the nature of this association in adults. Consequently, one of the purposes of this chapter is to conduct psychophysiological investigations on heart rate at rest, examining its associations with psychopathic personality traits amongst typical developing adults (Study 3). Following up on these correlational procedures, an additional goal of this third study is to perform exploratory analyses about the predictive role of psychopathic tendencies in explaining measurements of RHR in this population.

As evidenced in the section "Inferring psychological constructs from physiological responses", presented in Chapter Two, there have been claims for

assessing patterns of autonomic regulation rather than relying on single responses (Stemmler, 2003). These claims rest support on the organisation of the CNS, arranged to result in integrated physiological functioning (Kreibig, 2010). Accordingly, personality features have no exclusive, invariant biological correspondents. In other words, autonomic tonic activity and reactivity follow conditions of challenge and threat (Barrett, 2006). One potential next step would be to examine not only biobehavioural mechanisms of arousal at rest, but to investigate reactivity recordings from autonomic and neuroendocrine systems to gain a richer understanding of mechanisms of physiological regulation (Blascovich & Tomaka, 1996).

Undoubtedly, stressful situations are ingredients of daily life for many individuals (Crum, Akinola, Martin, & Fath, 2017). Consequently, this fourth study proposes a method of understanding physiological responses to stress, examining the relations with psychopathic traits and with aggressive/bullying behaviours. This will be achieved by exploring the simultaneous activation of the hypothalamic pituitary (HPA) and sympathetic–adrenal medullary (SAM) systems in response to laboratory–induced stress. Another aim of this study includes an investigation of variables that might predict patterns of physiological reactivity in adults. By the end of this chapter, a discussion of data collected at rest and in response to stress is presented.

4.1 Study 3 – Resting heart rate and psychopathic personality traits in adults

As shown in Chapter Two, RHR is influenced by both sympathetic and parasympathetic branches of the ANS. Amongst individuals in good physical health, HR is determined by the net effect of the actions between the sympathetic (causing acceleration) and parasympathetic (causing deceleration) divisions (Nagai, Hoshide, & Kario, 2010; Thayer & Lane, 2009). Although both branches are tonically active at rest, parasympathetic effects seem to prevail (Mccraty & Shaffer, 2015).

Low resting heart rate (LRHR) is considered for some scholars as being one of the strongest biological risk factors for psychopathy identified so far (Cornet,

2015; Raine, Venables, & Mednick, 1997b). Several reports have indicated that lower RHR was associated to high levels of psychopathic traits as well as to different forms of AB (Portnoy, Raine, Chen, Pardini, Loeber, & Jennings, 2014; see also: Lorber, 2004; Ortiz & Raine, 2004). A meta–analysis – comprising a total of 95 studies published between 1997 and 2001 – showed that low arousal at rest, as evidenced by measurements of RHR, was negatively associated with general aggression and with the presence of conduct problems in children and adolescents (*d*'s = − .38 and − .33, respectively; Lorber, 2004). A more recent meta–analysis, including data from adults and reporting on 115 independent effect sizes, yielded significant fixed effects ranging from *d* = − .11 (aggression) to –. 35 (violence); Psychopathy was also significantly linked to LRHR, with a fixed effect of *d* = –.19 (Portnoy & Farrington, 2015). One of the most accepted hypothesis for understanding physiological differences within the ANS is that high psychopathic individuals share a common – and largely inherited – disposition for low arousal, which is perceived as unpleasant and enhances the engagement with AB (Popma, Vermeiren, Jansen, & Doreleijers, 2007). Support for this hypothesis was encountered in a prospective longitudinal study in the United Kingdom that found that LRHR at age 18 resulted in more criminal convictions at the age of 50 (Jennings et al., 2013). In addition, HR was significantly higher amongst non–violent males in comparison to violent ones at the age of 18. Confounder variables – such as body mass index, sports habits, and use of tobacco were examined – and results demonstrated that they did not diminish the relationship between LRHR and high incidence of crime (Jennings et al., 2013). Cross–sectional evidence provide support for an association between LRHR and severe delinquency. In a study with college students, participants belonging to the group with low RHR (< 66 beats per minute; bpm) reported higher involvement with severe AB when compared to those with average (67 to 94 bpm) and high RHR (> 95 bpm) (Armstrong et al., 2009). These results were robust even after controlling for variables that may affect RHR, such as body mass index, age, and gender. Moreover, there are indications for diminished cardiovascular reactivity to distress in high psychopathic individuals. In a study in Japan, HR increase after the exposition to negative stimuli (e.g., short clip of a murder scene) was 3.4 bpm for those high on psychopathy and of 8.8 bpm amongst those scoring low (Osumi et al.,

2007). Serafim et al. (2009) examined the role of HR in a sample of psychopathic murderers and non–psychopathic murderers using the International Affective Picture System (Lang et al., 1999). Negative, significant associations amongst PCL–R Factor 1 with HR were detected while psychopaths viewed neutral (r = –.64), pleasant (r = – .53), and unpleasant images (r = – .66). Murderer–psychopaths did not show HR variations according to the type of images, albeit non–psychopaths murderer displayed an increase in HR when presented with pleasant images, which was interpreted as an indicator of affective deficits for those with a diagnosis of psychopathy (e.g., PCL–R > 30; Hare, 2003; Patrick, Cuthbert, & Lang, 1994; Serafim et al., 2009; Verona, Patrick, Curtin, Bradley, & Lang, 2004).

Despite the significant amount of work into the links between RHR and AB, and between HR reactivity and AB, research is warranted in this area for numerous reasons. Firstly, the majority of available data is from incarcerated individuals, mostly males (Portnoy & Farrington, 2015). Secondly, many studies have measured HR using unsystematic (diversified) methodology. Thirdly, inconsistencies in choosing reliable measures of psychopathy are common, which is a cause of concern in terms of convergent and predictive validity (Richter & Slade, 2017). Finally, there still exists some misunderstanding and limited knowledge about the reasons why those high on psychopathic traits and/or high on AB are prone to display low basal levels of autonomic arousal, and precisely which domains/dominant traits of psychopathy are mostly linked to biological low arousal. For instance, not every report indicates significant results, and many of them do not agree in the direction of the relationship (i.e. some of the work indicates that the more psychopathic the individual, the lesser would be the RHR levels, while others indicate positive association between RHR and psychopathy; Loeber, 2004; see also Portnoy & Farrington, 2015). Given the necessity for more clarification on the nature of the differences on RHR and psychopathy, the central aim of this study is to investigate whether RHR is linked to psychopathic personality traits amongst typical developing adults. A related, exploratory aim is to investigate which psychopathic dimensions would be significant in predicting RHR.

4.1.1 Hypotheses

Considering that LRHR is assumed to be the most robust biological risk factor for psychopathy (Baker, Tuvblad, Reynolds, Zheng, Lozano, & Raine, 2009; Portnoy & Farrington, 2015; Raine, 2002; Serafim et al., 2009), the first hypothesis is that significant, negative associations will be detected between psychopathic personality traits and RHR.

The fearlessness theory assumes that low arousal is linked to unpleasant physiological states, so AB have a function of increasing these levels to a more pleasant degree (Lykken, 1957; Raine, 2005; Raine et al., 2014; Zuckerman & Need, 1979). Indeed, earlier propositions for distinctions in psychopathy suggested that Factor 1 is under greater biological influences that index lack of fear (Karpman, 1941; Porter, 1996; Poythress & Skeem, 2006). Data collected with incarcerated individuals showed that Hare's PCL Factor 1 (Hare, 2003) was significant in negatively predicting RHR (Hansen et al., 2007). Following this, another hypothesis states that sub putative domains of the psychopathic personality will predict differences in autonomic functioning at rest, in which traits more closely resembling fearlessness features should significantly explain RHR.

4.2 Method

4.2.1 Participants and design

This cross–sectional study involved 101 adults (M_{age} = 24.7, SD = 7.8; 85 females). The investigation received ethical approval from the Goldsmiths Psychology and London South Bank University research ethics committees. Participants were recruited via a Research Participation Scheme at these two universities. Prior to physiological data acquisition, the participants were presented with an outline of the study, and were asked to provide consent for participation. All participants received both a study debriefing via electronic email and verbally as well. When requested, course credits were granted for students' attendance in the current investigation.

4.2.2 Measures

4.2.2.1 Psychopathic Personality Inventory Genetic Derived form (PPI–R–40; Eisenbarth et al., 2015);

Information regarding this measure was given within Section 3.5.2.3.1. Reliability tests indicated acceptable results in the current study (α_{total} = .72; α's = .71, .69, and .67 for Self–Centred Impulsivity, Fearlessness, and Coldheartedness factors, respectively). For the subscales, alphas were also calculated (α's = .70, .68, 71, .58, .53, .63 and .63 for Blame Externalisation, Carefree Non–Planfulness, Fearlessness, Machiavellian Egocentricity, Rebellious Nonconformity, Social Influence, and Stress Immunity, respectively).

4.2.2.2 Levenson Primary and Secondary Psychopathy scales (LPSP; Levenson et al., 1995);

The LPSP is a 26–item questionnaire designed in a 4–point scale in which 1 indicates 'disagree strongly' and 4 indicates 'agree strongly'. It assesses different domains related to psychopathy in adulthood (i.e. primary and secondary psychopathy). Reliability tests indicated acceptable results in the current study (α_{total} = .72, $\alpha_{\text{primary psychopathy}}$ = .67, and $\alpha_{\text{secondary psychopathy}}$ = .60). More detailed information regarding this measure was given within Section 3.2.2.3.2, and the differential associations between the LPSP with other types of assessment of psychopathy has been provided (e.g., Section 1.2.3).

4.2.3 Psychophysiological procedures for data acquisition and analysis

Electrocardiography and impedance cardiography measures were obtained continuously using a Standard II Lead configuration via a BIOPAC ECG100C amplifier (Biopac Systems, Inc., Goleta, CA, USA) in order to provide basal transthoracic impedance (ZO), and its first derivative (dZ/Dt), which allows the calculation of heart rate (Sherwood et al., 1990). On average, data collection recording started approximately 10 minutes after arrival at the laboratory. Data were collected, cleaned and submitted to analysis using AcqKnowledge 4.2 (Biopac Systems). Each file recording was inspected with the visualisation function of the

AcqKnowledge software. The purpose of this step was to check if there was any artefact due to movements of participants during data collection (Blascovich et al., 2011). Waveform areas containing artifacts were then deleted or smoothed automatically so damaged data was interpolated with estimates from adjacent regions of the wave.

For statistical analysis, resting results are the mean product of the final two minutes of the physiological recording. Pearson correlational analyses were run to address the first aim of this study. To avoid issues with multiple comparisons, the Benjamini and Hochberg's (1995) suggested method for false discovery rate (FDR) was employed using an electronic calculator (SDM Project Web, 2017). For examining the predictors of RHR (e.g., the second aim of this study), regression models were tested using the Stepwise method, after inspecting if data satisfied the assumptions for this type of analysis.

4.3 Results

4.3.1 Correlational analyses

Table 4.1 presents the Pearson correlations between resting heart rate and psychopathy measures. Except for secondary psychopathy, all correlations were in the expected direction. Two–tailed significant results were obtained for Rebellious Nonconformity, Social Influence, Stress Immunity, Total Psychopathy (PPI–R–40) and for Factor 1 (Fearlessness) (r_{range} = – .22 to – .28, p_{range} = .004 – .03) and are flagged in bold. When FDR correction is applied (Benjamini and Hochberg's method), only Rebellious Nonconformity, Total Psychopathy and Social Influence retained significance (new *p* values = .03, .03 and .04, respectively). Supplementary Tables 1 and 2 (Appendix G) report on correlational data separately for male and female participants.

Table 4.1: *Full correlation matrix of the links between HR and Psychopathy*

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1 Resting heart rate	*r*	–	−.13	.08	−.05	−.001	−.12	−.01	−.16	**−.28**	**−.26**	**−.22**	**−.28**	**−.23**	−.15	−.05
	p	–	.18	.43	.58	.99	.22	.87	.11	**.004**	**.009**	**.027**	**.005**	**.02**	.14	.60
	Upper 95% CI	–	.06	.27	.14	.19	.07	.18	.04	−.09	−.06	−.02	−.08	−.03	.05	.14
	Lower 95% CI	–	−.32	−.12	−.25	−.20	−.31	−.21	−.34	−.45	−.43	−.40	−.45	−.41	−.33	−.24
2 Primary psychopathy	*r*		–	.37	.88	.07	−.10	.22	.48	.16	−.08	−.10	.12	.05	.23	.05
	p		–	< .001	< .001	.46	.29	.02	< .001	.11	.44	.30	.23	.60	.02	.63
	Upper 95% CI		–	.52	.92	.26	.09	.40	.61	.34	.12	.09	.31	.25	.41	.24
	Lower 95% CI		–	.19	.84	−.12	−.29	.03	.31	−.04	−.27	−.29	−.07	−.14	.04	−.15
3 Secondary psychopathy	*r*			–	.75	.47	.27	.02	.28	.18	−.30	−.41	.09	−.31	.58	−.08
	p			–	< .001	< .001	.006	.80	.005	.06	.002	< .001	.35	.002	< .001	.39
	Upper 95% CI			–	.83	.61	.44	.22	.45	.36	−.11	−.24	.28	−.12	.69	.11
	Lower 95% CI			–	.65	.30	.08	−.17	.09	−.01	−.47	−.56	−.10	−.47	.43	−.28
4 Total psychopathy (Levenson)	*r*				–	.28	.06	.17	.47	.20	−.20	−.28	.13	−.11	.45	−.008
	p				–	.004	.55	.08	< .001	.04	.04	.005	.19	.25	< .001	.93
	Upper 95% CI				–	.45	.25	.35	.61	.38	−.01	−.09	.32	.08	.59	.19
	Lower 95% CI				–	.09	−.14	−.02	.31	.008	−.38	−.45	−.06	−.30	.28	−.20
5 Blame Externalisation	*r*					–	−.08	.15	.24	.29	−.20	−.28	.23	−.12	.70	−.29
	p					–	.43	.12	.01	.003	.04	.004	.02	.21	< .001	.003
	Upper 95% CI					–	.12	.34	.42	.46	−.005	−.09	.41	.07	.79	−.10
	Lower 95% CI					–	−.27	−.04	.05	.10	−.38	−.45	.04	−.31	.59	−.46
6 Carefree Non–Planfulness	*r*						–	.04	−.05	.18	.04	.06	.43	.07	.45	.38
	p						–	.67	.62	.06	.70	.54	< .001	.48	< .001	< .001
	Upper 95% CI						–	.24	.15	.36	.23	.25	.58	.26	.60	.54
	Lower 95% CI						–	−.15	−.24	−.01	−.16	−.13	.25	−.13	.28	.20
7 Fearlessness	*r*							–	.19	.36	.07	.08	.59	.67	.21	−.03
	p							–	.06	< .001	.44	.43	< .001	< .001	.03	.72
	Upper 95% CI							–	.37	.52	.27	.27	.71	.76	.39	.16
	Lower 95% CI							–	−.01	.18	−.12	−.12	.45	.54	.02	−.23
8 Machiavellian Egocentricity	*r*								–	.38	.10	−.13	.38	.11	.63	.08
	p								–	< .001	.30	.20	< .001	.29	< .001	.42
	Upper 95% CI								–	.53	.29	.07	.54	.30	.74	.27
	Lower 95% CI								–	.20	−.09	−.31	.20	−.09	.50	−.12
9 Rebellious Nonconformity	*r*									–	.25	.11	.70	.38	.47	−.02
	p									–	.01	.25	< .001	< .001	< .001	.80

Table 4.1: *Full correlation matrix of the links between HR and Psychopathy*

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
	Upper 95% CI									–	.42	.30	.79	.54	.61	.17
	Lower 95% CI									–	.05	– .08	.59	.20	.30	– .22
10 Social Influence	*r*										–	.37	.49	.67	– .05	.20
	p										–	< .001	< .001	< .001	.61	.05
	Upper 95% CI										–	.52	.62	.77	.14	.38
	Lower 95% CI										–	.18	.33	.55	– .24	.002
11 Stress Immunity	*r*											–	.38	.65	– .21	.21
	p											–	< .001	< .001	.04	.03
	Upper 95% CI											–	.54	.75	– .01	.39
	Lower 95% CI											–	.20	.53	– .39	.02
12 Total psychopathy (PPI–R–40)	*r*												–	.75	.56	.35
	p												–	< .001	< .001	< .001
	Upper 95% CI												–	.82	.68	.51
	Lower 95% CI												–	.65	.42	.17
13 PPI–R–40 F1 Fearlessness	*r*													–	.01	.16
	p													–	.89	.10
	Upper 95% CI													–	.21	.35
	Lower 95% CI													–	– .18	– .03
14 PPI–R–40 F2 Self–Centred Impulsivity	*r*														–	.06
	p														–	.54
	Upper 95% CI														–	.25
	Lower 95% CI														–	– .13
15 PPI–R–40 F3 Coldheartedness	*r*															–
	p															–
	Upper 95% CI															–
	Lower 95% CI															–

4.3.2 Regression analyses

A series of assumptions were checked before conducting regression analyses considering RHR as the outcome variable. First, predictors were eligible for inclusion in regression models after inspecting the results of FDR. Hence, subsequent regression models would only include the subscales of Social Influence and Rebellious Nonconformity, as well as the total score on the PPI–R–40. Second, homoscedastic requirements were examined for the relationships between the predictors and RHR using the Bresuch–Pagan test (BP) in *R*. Results were satisfactory for models examining the predictive role of both the PPI–R–40 subscales ($BP_{(1)} = 2.84$, $p = .24$) and the total score of the same measure ($BP_{(1)} = .11$, $p = .73$). Third, the Harvey–Collier's test (HC) confirmed the assumption of linearity ($HC_{(96)} = 1.06$, $p = .28$ and $HC_{(96)} = .74$, $p = .45$ for models included in Tables 4.2 and 4.3, respectively). Finally, to inspect potential problems with multicollinearity, variable inflation factors (VIF's) were calculated, coupled with inspection of skewness and kurtosis, which did not indicate further problems for conducting regression analyses (Hair et al., 2010).

Table 4.2: *PPI–R–40 subscales predicting HR at rest*

Models		Unstandardized	SE	Standardized	*t*	*p*	Model Fit (R^2_{Adj})
1	Intercept	96.81	5.11		18.91	< .001	$F_{(1,98)} = 8.48$,
	Rebellious Nonconformity	–1.33	.45	–.28	–2.91	.004	$p = .004$ (7%)
2	Intercept	106.09	6.77		15.66	< .001	
	Rebellious Nonconformity	–1.09	.46	–.23	–2.35	.02	$F_{(1,97)} = 6.47$,
	Social Influence	–.87	.42	–.20	–2.04	.04	$p = .002$ (10%)

Note. Durbin–Watson's values have been used to analyse residuals. In this study, the values were appropriated (2.926 and 1.928 for models 1 and 2, respectively); Collinearity values were acceptable (VIF 1.07 for both predictors retained using the Stepwise method). Skewness values were – .37 (Social Influence) and –.01(Rebellious Nonconformity), and kurtosis values were – .06 (Social Influence) and –.66 (Rebellious Nonconformity).

Table 4.3: *PPI–R–40 total score predicting HR at rest*

Model		Unstandardized	SE	Standardized	*t*	*p*	Model Fit (R^2_{Adj})
1	Intercept	112.60	10.71		10.51	< .001	$F_{(1,98)} = 8.08$,
	Total psychopathy	–.34	.12	–.27	–2.84	.005	$p = .005$ (6.7%)

Note. Durbin–Watson's value has been used to analyse residuals, and values were appropriated (e.g., 1.968); Collinearity values (VIF 1.00) did not indicate the presence of multicollinearity. Skewness value was – .22 and kurtosis was – .46.

As can be noted from the regression analyses taken the PPI–R–40 subscales (Table 4.2), the solution with better explanatory power seems to be Model 2, capable of explaining 10% of the variance of RHR. In addition, the total score on the PPI–R–40 was included as a single predictor of RHR, yielding significant results and explaining nearly 7% of the variance (Table 4.3). Regression analyses exploring the predictive role of the LPSP (Levenson et al., 1995) and the factors of PPI–R–40 were not conducted since correlational results indicated that the associations between these measures with RHR did not reach significance, or, when significance was obtained, FDR analyses indicated that new *p* values would be above .05 when the effect of multiple comparisons is taken into account. Moreover, as shown in Supplementary Tables 3 and 4 (Appendix G), gender does not seem to significantly predict RHR in this sample.

4.4 Discussion

Cumulative evidence indicates that LRHR is a risk factor solely for antisocial behaviour, not being linked to other mental disorders (Raine, 2002). The links existent between AB and HR are robust even after some covariates – such as age and gender – are controlled; Moreover, it applies to multiple forms of AB – including aggression and psychopathy, and is stable for both clinical and community samples, as well as in cross–sectional and longitudinal studies (Ortiz & Raine, 2004; Portnoy et al., 2014; see also Portnoy & Farrington, 2015). However, it is interesting that around a third of the effect sizes retrieved by the latest meta–analysis in this matter were not in the expected direction (Portnoy & Farrington, 2015), which signals that the evidence (as abundant as it might be) regarding HR and AB still warrants further examination. Consequently, this study explored the associations of RHR with psychopathic personality traits, equally examining the predictors of this biological indicator. Results are discussed in turn.

4.4.1 Associations between RHR and psychopathic personality traits

In respect to this study's first hypothesis, results depicted in Table 4.1 corroborated the prediction of detecting significant, negative associations between psychopathic personality traits and RHR, and are in accordance with the majority of findings already published (Portnoy et al., 2014; see also Portnoy & Farrington, 2015). All correlations (significant or not) were in the expected direction, with the exception of Levenson's Secondary Psychopathy, which showed a positive, non–significant, and very weak correlation with RHR.

Correlational data showed negative associations between RHR with Total Psychopathy and for Factor 1 (Fearlessness). Likewise, the subscales of Social Influence and Stress Immunity – that load into the PPI–R–40 Factor 1 – were negatively associated with RHR. Moreover, the subscale of Rebellious Nonconformity was linked with diminished RHR. After correcting for multiple comparisons, statistical significance was maintained for the correlations between RHR and Rebellious Nonconformity, Total Psychopathy, and Social Influence (p's < .05, r's$_{range}$ = – .28 to – .26; Cohen's d = – .56). These significant correlations could represent some risk for involvement with criminal activities (Jennings et al., 2013), meaning that the higher the participant–reported traits associated with the construct of psychopathy, the lower their RHR.

Importantly, biological risks factors alone cannot fully account for engagement with complex behaviours such as crime (Popma et al., 2007). However, data reported here suggests that, as seen in many studies with community and forensic samples, psychopathic features seem to be associated with diminished basal functioning of the ANS, which is then linked to augmented risk for crime and for displaying severe forms of antisocial behaviour (Ortiz & Raine, 2004; Portnoy & Farrington, 2015). A firmer understanding on casual roles of RHR in explaining adverse outcomes is beyond the reach of this study. However – although exact comparisons to previous studies are not possible –, the effect size detected for the correlations presented in Table 4.1 (d = – .56) was similar to the one reported in a meta–analysis containing 5,868 children and adolescents, in which the overall effect size for the relationship between AB and RHR

was −.44 (Ortiz & Raine, 2004). In addition, age, gender, sample characteristics (e.g., community or clinical), study design (concurrent or prospective), and even the method of measuring RHR (sophisticated versus simple recording) did not significantly moderate the strength of the effect reported by Ortiz and Raine (2004). Lorber (2004) also indicated to smaller effect sizes when compared to those reported in this study (Cohen's d_{range} = −.38 to – .33 for aggression and CP, respectively). However, these two previous meta–analyses focused on investigations conducted with children and adolescents, which impedes direct comparisons. As for adults, the overall effect size for the relationship between RHR and AB reported on the latest meta–analysis on this matter was d = – .15, and between RHR and psychopathy was d = – .19 (Portnoy & Farrington, 2015). Hence, bigger effect sizes were found amongst participants who took part in Study 3. Possible reasons for these differences might include the low number of publications that have gathered data from adults (i.e., 33 versus 80 child/adolescent studies), and also the fact that few studies assessed psychopathy (i.e., 22 versus 109 on other behavioural problems; Portnoy & Farrington, 2015).

4.4.2 Psychopathic personality traits predicting RHR

Raine (2005, 2013) proposed the role of fearlessness as central to understanding autonomic hypo arousal seen in psychopathy and other forms of antisocial behaviour. This is closely linked to this study's second aim, in which sub putative domains of the psychopathic personality were assumed to more strongly predict RHR. Significant betas were detected for Rebellious Nonconformity (β = –.23) and Social Influence (β = –.20), and these two predictors were capable of explaining 10% of the variance of RHR (cf. Table 4.2). These results supported only partly the second hypothesis, which stated that fearlessness features would significantly predict RHR. Factor 1 (Fearlessness Dominance) did not keep statistical significance when the effect of multiple comparisons was accounted for, and was excluded from regression analyses.

However, one of the Factor 1 constituent subscales did associate and did predict RHR (Social Influence), along with Rebellious Nonconformity. Regression models have some overlap to a previous study in which the interpersonal facet of psychopathy significantly predicted RHR (β = – .51; Hansen et al., 2007). However, the study was conducted in a sample of male inmates. Studies including the PPI–R–40 and its predictive role in levels of RHR were not located in the literature, which impedes comparisons to findings presented in here.

One important consideration incudes the mechanisms by which the fearlessness proposition can be manifested in terms levels RHR in individuals attending to a laboratory testing session. As suggested by Portnoy et al. (2014), even when the measurement of RHR is performed in a completely stress–free environment, it is often the case that expectations of "future testing" or imminent assessment would be perceived as anxiogenic. In normal conditions, individuals would show signals of arousal (Portnoy et al., 2014). Those high in psychopathic traits, however, are believed to present with lack of or much diminished fear in response to these events (Hare, 1971). RHR, in this scenario, would be a proxy for the absence of fear experienced by those with elevated psychopathy.

4.4.3 Implications and limitations

One critic that was addressed to previous work relies on the fact that cardiovascular responses indicative of the ANS functioning were measured unsystematically (Lorber, 2004; Ortiz & Raine, 2004; Raine et al., 2014). Therefore, this study used a robust method for assessing RHR continuously. Another limitation from previous investigations is related to the use of specific measurement of psychopathic traits (Ortiz & Raine, 2004; Portnoy & Farrington, 2015). Hence, both Levenson Primary and Secondary Psychopathy scales (LPSP; Levenson et al., 1995) and the Psychopathic Personality Inventory Genetic Derived form (PPI–R–40; Eisenbarth et al., 2015) were used in the hope that this could provide further predictive and convergent validity of findings (Rilling et al., 2007).

Results presented in this study could be meaningful in, at least, three ways. Firstly, this non–criminal and non–forensic sample has reported levels of secondary psychopathy (*M* = 21.82) comparable to sexual (*M* = 19.2) and violent offenders (*M* = 23.1) (Gillespie, 2014). In addition, the means found for primary psychopathy (*M* = 31.97) were also higher than those reported by Gillespie (2014) for sexual (*M* = 26.5) and violent offenders in the U.K. (*M* = 29.9). For example, elevated scores in secondary psychopathic traits can result in more reactive aggression (Warren, 2009) and adult bullying (Wendt & Bartoli, 2018), mood and anxiety disorders (Skeem et al., 2011), whereas high scores in the primary variant can prone individuals in engaging with proactive aggression (Warren, 2009), bullying (Wendt & Bartoli, 2018), and thrill–seeking activities (including risk for substance abuse; Brazil et al., 2016; Skeem et al., 2011). In respect to these differences in levels of psychopathic personality traits between participants of Study 3 with those reported by Gillespie (2014), one compelling explanation is that, amongst clinical populations, self–report measures are more likely to be biased (Lilienfeld & Fowler, 2006). Indeed, Gillespie (2014) argued that issues around deception and socially desirable response patterns can be particularly challenging when studying offending participants. Indeed, in–depth assessments of traits such as pathological lying, and conning/manipulative tendencies require extensive clinical training, not being, therefore, well captured by self–report methods (Gillespie, 2014; Hare, 2003).

Secondly, the negative associations found between RHR and psychopathic personality traits would indicate that participants are at higher risks for future offending (Douglas, Vincent, & Edens, 2006; Jennings et al., 2013; Lee & Salekin, 2010; Ortiz & Raine, 2004). Nonetheless, it is possible that Type II error could have occurred in the correlational analyses as a result of the application of FDR, so some caution is warranted in interpreting this second implication (Rothman, 1990). Thirdly, if future work corroborates results presented here, heart rate assessments may be included in a physical work–up for improved diagnosis and more refined therapeutic approaches (Ortiz & Raine, 2004; Portnoy et al., 2014). Notably, behavioural interventions that tap onto the theoretical explanations of the origins of the low RHR–psychopathy

relationship – such as the absence of fear, social dominance, and need for stimulation – might prescribe more tailored actions to increase efficacy in treatment for those particularly high on characteristics linked to Factor 1 (Portnoy et al., 2014; Raine, 2002). It is believed that individual differences in personality dimensions are partly explained by patterns of ANS functioning (Stadler, Grasmann, Fegert, Holtmann, Poustka, & Schmeck, 2008). As such, to develop preventive and treatment programmes for personality disorders, it is imperative to comprehend differential associations between certain traits with RHR (Hansen et al., 2007). For example, Stadler et al. (2008) noted that LRHR significantly moderated treatment outcomes for children with disruptive behaviour. In this investigation, participants with significant lowers levels of RHR at the beginning of the programme did not show improvements in their problematic behaviours in response to cognitive-behaviour therapy. Interestingly, children with high levels of RHR did show significant progress, such as stronger decline in aggression and delinquent behaviour. For Stadler et al. (2008), those with LRHR might be less responsive to standard cognitive–behavioural interventions, albeit pharmacological strategies and biofeedback techniques might aid in designing best treatment options.

It might be equally interesting to further test if the evidence here presented in respect to there being significant links between low arousal with Rebellious Nonconformity, Social Influence and Total Psychopathy could be also detected at other biological domains, including the brain and hormonal levels. Future research is needed to clarify how low RHR interact with brain and other biological processes in explaining behaviour. Differences in aetiogical aspects of ANS functioning in respect to traits belonging to Factor 2 (which was positively linked with RHR, albeit at a non–significant level) also warrants further research and replication. Here, it would be interesting to test if characteristics that load within this factor are indeed less meaningful in the understanding of low RHR as a risk factor for psychopathic symptoms, or if, perhaps, these domains are mediated/moderated by other relevant variables not included in the present investigation.

Finally, the current study must be considered in the light of its limitations. For instance, data were collected with an unequal proportion of males and females, although physiological analyses were adjusted for the sex, age, and body mass index of the participants. This could have implications for the generalisation of findings, most precisely in terms of levels of self–reported psychopathic features in females (Sevecke, Lehmkuhl, & Krischer, 2009; Skeem et al., 2011; Verona & Vitale, 2006; but see also Schmeelk, Sylvers, & Lilienfeld, 2008). Also, a low alpha was found for Rebellious Nonconformity, which would normally require some caution in interpreting data in respect to this subscale. However, the PPI–R–40 was abbreviated using techniques that overtly aim to eliminate redundant variance. In this context, values indicating to a low internal consistency are desirable (Eisenbarth et al., 2015). In addition, the discussion of findings here presented was based on data gathered from participants recruited from London universities. As such, possible efforts in replicating these results using criminal samples in diverse contexts could return a rather different picture.

4.5 Study 4 – How do you respond? Antisocial behaviours are linked to threatening physiological responses

Clearly, comprehending the associations between baseline measurements of the ANS with psychopathic personality traits is important. However, physiological recordings – combining autonomic and neuroendocrine responses – are especially relevant to inform how individuals regulate their responses to stress (Kreibig, 2010; Stemmler, 2003). Indeed, no invariant biological correspondent has been identified for personality traits; rather, patterns of autonomic reactivity follow general conditions of challenge and threat (Barrett, 2006). The goal of this fourth study was to explore the simultaneous activation of the HPA and SAM systems in response to laboratory–induced stress and its relationships with psychopathic personality traits and bullying.

Accumulating research advocates that psychopathy is a multidimensional construct, with differential associations with psychopathologies and distinct biological

underpinning mechanisms (Kendler, Aggen, & Patrick, 2012). Bullying, in turn, is a type of AB which is intentional and repetitive. Individuals involved with bullying show numerous difficulties, including higher use of proactive and reactive aggression and are a greater risk for developing more consistent antisocial tendencies (Craig, 1998; Copeland et al., 2013; Jolliffe & Farrington, 2011; Olweus, 1991; Smith et al., 2012). Prior research yielded important findings showing a pattern of endocrine and psychophysiological hypo–arousal within the HPA and SAM systems in individuals at risk for AB and in established offenders (Nederhof et al., 2015; Susman, 2006; Thompson et al., 2014). Amongst people identified as being psychopathic, differences in these systems have been observed only inconsistently, and appear to be linked to particular sub–dimensions of the construct. These apparently oppositional findings may be explained by taking a more nuanced approach to the concept of psychopathy. Some evidence exists which supports the idea different dimensions of psychopathy are linked to different patterns of physiological reactivity. For instance, HPA hypo activity to stress is commonly seen in children with CU traits, while hyper activity has been detected among youth with severe levels of AB and CP, without elevated CU traits (Lopez–Duran et al., 2009; Walker et al., 2016). In adults, research on the role of psychopathic traits in determining HPA reactivity to stress is scarce (Glenn, Remmel, Raine, Schug, Gao, & Granger, 2015).

Studies focusing on neuroendocrine aspects of bullying behaviours have returned inconsistent results. One promising explanation to these findings rely on the fact that most investigations on bullying take place when participants are still developing (including the development of the HPA axis; Platje et al., 2013). Moreover, other researchers argue that methodological aspects might account for divergent findings, including disparities in measuring the HPA and its correlates (e.g., basal levels of cortisol, levels of cortisol after exposition to stimuli [recovery levels]). Nevertheless, there are some data supporting the understanding that children involved with bullying victimisation present with a pattern of psychophysiological reactivity to stressors marked by blunted cortisol awakening response (Knack, Jensen–Campbell, & Baum, 2011). Research also posits that aggressive behaviours in

children can be linked to low basal HPA activity but enhanced reactivity to minor stress (Lopez–Duran et al., 2009; McBurnett, Lahey, Rathouz, & Loeber, 2000). However, very little is known in respect to the associations between antisocial and bullying behaviours with neuroendocrine responses to stress after the maturity of the HPA axis. Arguably, a clear comprehension of the role of neuroendocrine mechanisms in bullying behaviours can perhaps be obtained by measuring the effects of the HPA axis using more direct procedures, such as real time recording of various physiological indicators assumed to be associated with stress responsive systems.

4.5.1 Stress reactivity and psychophysiological indicators of Challenge and Threat

The HPA axis is the main neuroendocrine system responsive to stress, connecting the CNS to the hormonal system. Its functions include helping the body in the process of adapting to demands and responding to threat, as well as in maintaining homeostasis after challenging situations (Herman & Cullinan, 1997). Responses to stress are mediated by 'super–fast' systems (e.g., the ANS), involving also slower (e.g., immune, neuroendocrine, and enteric systems) and 'super slower' processes (e.g., oxidative stress; Mendes & Park, 2014). Limbic regions, including the prefrontal cortex, the hippocampus, and the amygdala, are presumed to be involved in the processing of psychological stress (Herman & Cullinan, 1997). In physiological terms, stress responses begin when the hypothalamus secretes the corticotropin–releasing hormone, which culminates with the release of adrenocorticotropic hormone (ACTH) from the pituitary (Kudielka & Kirschbaum, 2005). In reaction to this, ACTH causes the secretion of glucocorticoids from the adrenal cortex, with cortisol being the principal product amongst humans. In acute stress response, the availability of energy comes as a result of the secretion of epinephrine and cortisol (Schneiderman, Ironson, & Siegel, 2005). Subsequently, there is an increase in blood pressure via two hemodynamic patterns: amplified cardiac output (CO), resulting in higher HR and stroke volume (SV), known as the myocardial mechanism; and via vasculature constriction or vascular mechanism (Schneiderman et al., 2005). Dickerson and Kemeny's (2004) meta–analysis reviewed 208 stress studies in order to understand

the conditions capable of eliciting HPA reactivity in laboratories, concluding that motivated–performance tasks provoked robust physiological reactions, even when controlling for methodological matters and stressor characteristics.

The Bio–Psychosocial model of Challenge and Threat (BPSM; Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996) is a dominant, well supported model describing the processes underpinning stress appraisals and the subsequent cognitive, physiological, and behavioural responses (Rith–Najarian, McLaughlin, Sheridan, & Nock, 2014). The BPSM argues that psychological appraisal processes are responsible for physiological responses to stressors when individuals become engaged in a goal–relevant task, resulting in two states – *challenge* and *threat* (Seery, 2011). A challenge state occurs when the evaluation of the resources can exceed the demands given to a certain individual during a goal–oriented task (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996). Challenge states are linked with enough coping strategies. On the other hand, a threat state is associated with negative affective states, poor focus, disorganised emotions, and a marked conflict between approach and avoidance behaviours (Blascovich & Mendes, 2000). Research has shown that whereas challenged individuals are more likely to present better performance in cognitive (Mendes, Blascovich, Lickel, & Hunter, 2002) and physical activities (Moore, Vine, Wilson, & Freeman, 2014), those displaying with predominantly threatening appraisals tend to deliver a worse performance (Allen, Frings, & Hunter, 2012; Blascovich & Mendes, 2000; Mendes, Blascovich, Major, & Seery, 2001). This occurs since vascular resistance (or TPR) intermediates with oxygenated blood transmission to the brain and its peripheral tissues (McLaughlin, Sheridan, Alves, & Mendes, 2014).

A considerable amount of research (e.g., > 30 studies) validated the cardiovascular responses as biological indicators of challenge and threat (Allen et al., 2012; Frings, Hurst, Cleveland, Blascovich, & Abrams, 2012). Higher heart HR and LVET from baseline to task phases are indicatives of participant's *engagement* with a given task. Additionally, those who experience a challenge motivational state are expected to display increases in CO and decreases in TPR; whereas threat is linked

with little change in CO and little change or even increased TPR (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996; Frings et al., 2012).

Challenge and threat appraisals have physiological effects on the body and are assumed to be at opposite ends in a bipolar *continuum*. While the heightening effect on HR and CO seen in both challenge and threat appraisals are coordinated mainly by the SAM activity, a key role in distinguishing threat (e.g., high TPR) is played by the HPA when it inhibits the release of epinephrine, blunting the SAM vasodilatory effects (Seery, 2011). Figure 4.1 illustrates the hemodynamic responses within the BPSM.

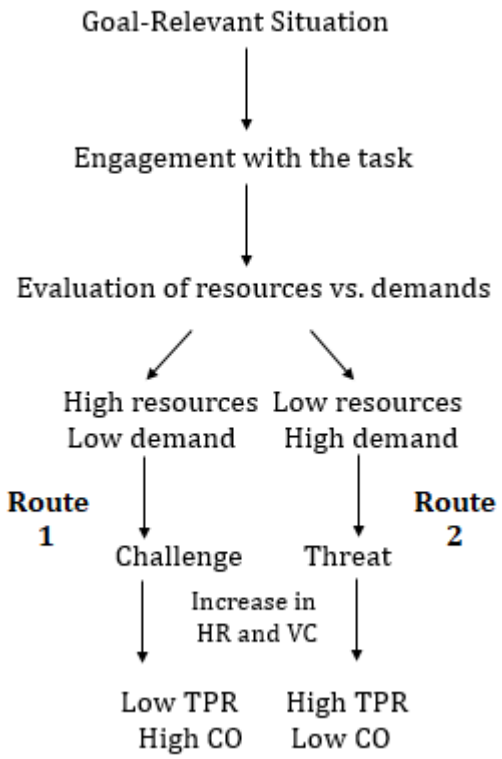

Figure 4.1 – Overview of the BPSM, in which higher HR and LVET reactivity scores from baseline to task phases are indicative of a participant's engagement with a given task. Additionally, route number 1 explains that those who experience a challenging motivational state would display increases in CO and decreases in TPR. Finally, route number 2 comprises the assumptions for those responding in a threatening manner, assumed to show little change in CO and little change or even increased TPR (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996; Frings et al., 2012). Figure adapted from Seery (2011).
Note. CO, cardiac output. HR, heart rate. TPR, total peripheral resistance. VC, ventricular contractility

In order to facilitate interpretation of the effects of evoked states of challenge and threat within the body, a composite score is used. First, values of CO reactivity are

calculated by subtracting baseline CO from values exhibited at other timepoints, thus yielding Z scores for CO reactivity. Then, values of TPR reactivity are computed by subtracting values from baseline SVR from values exhibited at other timepoints, thus creating Z scores for TPR reactivity. Finally, indexes for challenge and threat (C&T) can be obtained by subtracting TPR Z scores from the CO Z scores, for each comparison timepoint. Positive values would indicate that a given participant displayed relatively challenge, whereas negative values would index a threatening appraisal. Further details on this are given within Section 4.6.4.

4.5.2 Psychophysiology of the HPA and SAM systems in relation to AB, aggression, and psychopathy

The HPA axis is regulated by internal mechanisms such that any prolonged exposure to stressors can lead to dysregulation; in other words, the two extremes – either hyper or hypo–responsiveness – within this system have been linked to psychopathology (Hostinar & Gunnar, 2013). For aggressive phenotypes, a U–shaped function has been proposed to explain differences in the functioning of the HPA axis (Walker et al., 2016). In the light of the psychopathy literature, Davis (1992) has pointed out the role of the amygdala in prompting cardiovascular reactivity to stress. Moreover, the joint coordination of physiological responses from the HPA and SAM systems is assumed to be under direct control of the limbic system, in which the amygdala plays an important role (Herman & Cullinan, 1997). Needless to say, this specific brain region has been widely linked to severe levels of AB and psychopathy (Blair, 2007a).

Studies linking challenge and threat appraisals directly to AB and psychopathic personality traits are, to the best of my knowledge, non–existent. Nonetheless, the overall results from Chida and Hamer's (2008) meta–analysis found that positive psychological states or traits were significantly linked with reduced HPA axis reactivity (r = –.14; 95% CI = – .25, .02; p = .015). More recently, Allen et al. (2012) reported that physiological responses indicative of threat appraisals (i.e., HPA activation and increase in TPR) were negatively associated with openness and agreeableness personality traits in adults. Another report has established associations

between physiological threat responses to negative affect (Mendes et al., 2001). At the behavioural level, aberrant HPA axis reactivity to stress has been associated with abnormal levels of aggression in both humans and animals (Walker et al., 2016). Data from non–experimental research has shown that a positive link between threat appraisal with neuroticism exists, equally demonstrating to a negative correlation between extroversion and threat (Mak, Blewitt, & Heaven, 2004). Interestingly, low agreeableness was related to the Factor 1 of psychopathy (i.e., selfishness, callousness, and interpersonal manipulation), whereas the Factor 2 (impulsivity, instability, and social deviance) was again linked with low agreeableness, less conscientiousness, and high neuroticism in adolescents (Lynam, Caspi, Moffitt, Raine, Loeber, & Stouthamer–Loeber, 2005). Glenn, Raine, Schug, Gao, and Granger (2011) postulated that the HPA axis could be under active in psychopaths, and based on the final–product (e.g., testosterone) of the hypothalamic–pituitary–gonadal axis (HPG), the authors proposed a link between this axis with AB and aggression. What is unclear, however, is if the authors considered psychopathy as a unitary and homogeneous concept. More recently, Thompson et al. (2014) assumed that deficits in both HPA and HPG axes could indicate additional risk factors for psychopathy. Interestingly, both extremes of under or over reactivity could result in maladjustment. Therefore, psychopaths – and those high on psychopathic traits – display a distinct response to stress when compared to typical developing peers.

Abnormal HPA hyper reactivity to minor stress has been linked to aggressive tendencies in children (Lopez–Duran et al., 2009; Walker et al., 2016). In addition, there has been experimental indication that children involved with bullying victimisation present with a pattern of psychophysiological reactivity to stressors marked by blunted cortisol awakening response (also known as cortisol mobilisation response; Knack et al., 2011). One possible explanation for this is that repetitive victimisation is highly stressful, which would alter important neuroendocrine processes within the body over time. With an altered set–point within the HPA, these subjects are believed to present with a physiological deficit, impairing their ability to effectively cope with threats (Knack et al., 2011; Vaillancourt, Hymel, & McDougall,

2013). In respect to neuroendocrine aspects of bullying perpetration, research is far less common (Arseneault, 2017), and investigations on psychophysiological aspects of adult bullying is rare.

4.5.3 Hypotheses

Following previous evidence for associations between physiological threat with negative affect, this study hypothesises that psychopathic traits would be inversely linked with challenge. In other words, a negative correlation between psychopathic personality traits with challenge and threat reactivity indexes was predicted (Chida & Hamer, 2008; Mak et al., 2004; Mendes et al., 2001). Moreover, since HPA hyper reactivity has been previously linked to aggression (Hamilton et al., 2008; Lopez–Duran et al., 2009), for this study it was also foreseen that bullying behaviours should be negatively correlated with challenge and threat reactivity indexes.

4.6 Method

4.6.1 Participants and design

Seventy–five participants were recruited from London Universities. Sixty–eight participants (83.3% female, M_{age} = 24.57 years, SD = 2.73) completed this experimental study with random allocation of participants. Complete data from the remaining seven participants was not properly collected due to equipment malfunction (e.g., sensors detaching during the study leading to poor quality / absent signals or automatic recalibrations of the blood pressure monitoring equipment occurring mid–study). Sample size has been calculated based on a power analysis using specific software (e.g., G*Power version 3.1; Faul, Erdfelder, Lang, & Buchner, 2007). Power analyses calculations were set for a two–tail bivariate correlation with 95% power (α = .05). In order to detect a moderate effect size, a total number of 44 participants would be required, which is consistent with recent work examining the relationship between psychopathic personality and autonomic hypo–responsivity in similar contexts (e.g., non–forensic samples; Burley, Gray, & Snowden, 2017).

4.6.2 Self–Report Measures

4.6.2.1 Psychopathic Personality Inventory Genetic Derived form (PPI–R–40; Eisenbarth et al., 2015)

Information regarding this measure was given within Section 3.5.2.3.1. Reliability tests indicated acceptable results in the current study (α_{total} = .72; α's = .65, .71, and .67 for Self–Centred Impulsivity, Fearlessness, and Coldheartedness factors, respectively). For the subscales, alphas were also calculated (α's = .71, .63, 74, .71, .44, .69 and .62 for Blame Externalisation, Carefree Non–Planfulness, Fearlessness, Machiavellian Egocentricity, Rebellious Nonconformity, Social Influence, and Stress Immunity, respectively).

4.6.2.2 Illinois Bullying Scale (IBS; Espelage & Holt, 2001)

This 18–item measure is designed to assess the frequency of bullying experiences over the last 30 days, and items are disposed on a 5–point Likert scale. For the current study, all the subscales were used, which showed acceptable indices of internal consistency (α = .84 for the overall scale, α = .87 for victimisation; α =.74 for the bully (perpetrator), and α = .78 for the fighting subscale). Self–report bullying was here used due to the fact that participants did not belong to the same classes. Otherwise, peer–nominations would have been incorporated in addition to self–reporting research, mainly because it best captures bullying dynamics. Nonetheless, self–report is the preferable method for obtaining subjective views on bullying, and are believed to assess relatively stable individual differences (cf. Jimerson, Swearer, & Espelage, 2009; Olweus, 2009). The three Illinois Bullying Scales (i.e., bully, fighting, and victimisation) are included as an Appendix (A).

4.6.3 Laboratory task

Participants were randomly assigned to a condition of higher–demand (threat) or lower–demand (challenge). The following instructions were given to all participants after physiological baseline recording was completed: "The next activity will require

you to try to find 15 words in two word–search tasks. Words can appear horizontally and vertically. The first task will be only an exercise; however, if you perform well (5 or more words found) or really well (10 or more words found out of 15) on the second, harder task you will receive a treat. 10 or more words equal a full chocolate bar, and between 5 and 9 words allow you to have half of a chocolate bar. You will be timed during the second task (300 seconds). For each task, you will be also required to rate the difficulty (ranging from (0) *very easy* to (7) *very difficult*)" (cf. Frings, Eskisan, Spada, & Albery, 2015).

During the priming phase, participants allocated in the threatening condition received a word–search task containing only 3 words to be found (Appendix F, page 2), whereas those in the challenging scenario received a task containing 15 words to be found (Appendix F, page 1). Arguably, participants in the threatening version of the prime would not qualify for a reward if the succeeding task was indeed harder. All participants completed the same word–search task in the task phase (i.e., containing 15 words; Appendix F, page 3).

4.6.4 Psychophysiological procedures for data acquisition and analysis

To obtain the cardiovascular measures that differentiate challenge and threat states (e.g., CO, HR, PEP, TPR, and ventricular contractility [VC]) three physiological techniques were used: electrocardiography, impedance cardiography, and blood pressure measurement (Blascovich et al., 2011; Frings et al., 2012). These are structured experimental procedures widely validated within the BPSM framework (cf. Frings et al., 2015).

Electrocardiography measures were obtained continuously using a Standard Lead II configuration, via a BIOPAC ECG100C amplifier, with ground lead provided by the impedance measures. Impedance cardiography data was recorded using a BIOPAC NICO100C amplifier at 100Hz via electrodes in order to provide basal transthoracic impedance (ZO) and its first derivative (dZ/Dt) which allow the calculation CO, HR, and PEP (Biopac Systems, Inc., Goleta, CA, USA). Continuous blood pressure had been

taken also at 1 KHz, by using a CNSsytems CNAP monitor, connected to a BIOPAC DA100c amplifier.

All measures were taken continuously (online) for an overall of fifteen minutes, 5 of which constituted the baseline (T1), 5 during practice/priming of participants into challenge or threat conditions (T2), and, finally, 5 minutes for the task phase (T3). All recordings commenced after obtaining written consent from participants. On average, data collection for baseline results started 8–10 minutes after arrival at the laboratory. Data were collected, inspected, cleaned, and submitted to offline analyses using AcqKnowledge version 4.2 software (Biopac Systems) and in accordance with psychophysiological procedures, especially those suggested by Blascovich et al. (2011). Measures obtained via impedance cardiography were reduced to produce interbeat interval files after visual inspection of cardiographic waveforms, which consists of scanning the data for atypical points (artifacts) at adjacent areas and interpolating the file to retain interbeat intervals series.

For all measures, reactivity scores are calculated. To aid interpretation and to account for the interdependent nature of these measures, a single score was calculated reflecting a bipolar dimension of threat – challenge for each possible reactivity comparison: (a) between baseline and prime; (b) between baseline and task; and (c) between prime and task (Frings et al., 2012). For creating the bipolar dimensions of threat – challenge between baseline x prime and for baseline x task, I subtracted the last 2 minutes of resting baseline levels from the first 2 minutes in the levels exhibited during motivated performance situations (prime and task). For the last index of threat – challenge used in the current study (prime x baseline), I subtracted the last 2 minutes of physiological recordings during the prime condition from the first 2 minutes in the task. Challenge and threat indexes (C&T) for each reactivity comparison are based on *z* scores and were calculated as follows:

$$C\&T_{index} = ZCO\ reactivity - ZTPR\ reactivity^{20}$$

[20] Total peripheral resistance reactivity (TPR) was calculated by subtracting the last 2 minutes of baseline systemic vascular resistance (SVR) from the first 2 minutes in the levels exhibited at prime (baseline x prime reactivity), and

Paired *t* tests were used to check for participants' engagement with the task. According to the BPSM (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996; Frings et al., 2012), significant changes in HR, or in HR and LVET between baseline and reactivity recordings are indicative of a successful mobilisation of physiological responses on a given experiment. To attend the first aim of the study, correlations (Pearson) were used. These were firstly conducted with physiological data collapsing across groups, followed by subsequent examinations of the effects of experimental conditions. In attention to multiple comparisons, correlational data was further examined using the method developed by Benjamini and Hochberg (1995) using an electronic calculator (SDM Project Web, 2017). Finally, exploratory analyses on the predictors of challenge and threat reactivity indexes were conducted using the stepwise method.

4.7 Results

4.7.1 Manipulation check

To check for task engagement, a comparison (paired *t* test) of HR was undertaken between the baseline condition and the practice/prime phase ($t_{(67)} = 3.07$, $p = .003$), and between the baseline condition and the task phase ($t_{(67)} = 6.37$, $p < .001$). In addition, paired *t* tests revealed significant differences for LVET scores from baseline to task phase ($t_{(67)} = 2.38$, $p = .02$). These results showing increase in HR and LVET indicate that participants were engaged in the tasks, which is one of the requirements for proceeding with further analyses using the BPSM approach (Blascovich et al., 2011; Frings et al., 2012). No further differences were noted in the reactivity scores for MPB, PEP, SVR and CO. The means and standard deviations of all the cardiovascular measures are depicted in Table 4.4.

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between the last 2 mininutes of SRV at baseline from the first two minutes of SVR during task (baseline x task reactivity). For the last reactivity (prime x task) I subtracted the last 2 minutes of physiological recordings of SVR at prime from the first 2 minutes during the task.

Table 4.4: *Means and standard deviations (SD) for cardiac indexes by phase*

	HR		MBP		PEP	
Baseline	82.98	(12.61)	95.11	(10.72)	.0148	(.1043)
Prime	85.64	(13.81)	94.87	(11.10)	.1299	(.0971)
Task	88.44	(14.26)	95.06	(11.75)	.1262	(.0815)
	LVET		**SVR**		**CO**	
Baseline	.3335	(.0527)	1731	(763.5)	5.477	(4.640)
Prime	.3275	(.0536)	1800	(705.1)	5.469	(5.044)
Task	.3237	(.0513)	1752	(708.7)	5.600	(4.743)

Note. CO: cardiac output. HR: heart rate. LVET: left ventricular contractility. MBP: mean blood pressure. PEP: pre–ejection period. SVR: systemic vascular resistance.

Tables 4.5 and 4.6 summarise descriptive and inferential statistics between the groups during the experimental tasks, respectively. As expected, participants in the challenging condition found more words during priming, and evaluated this task as less difficult. Interestingly, those primed in the threatening condition had a poorer performance in the second task, which was the same for all participants ($t_{(65)} = 2.04$, $p = .045$; Table 4.6). This indicates the effect of threatening priming on performance.

Table 4.5: *Descriptive for task performance and perceived difficulty*

	Condition	N	Mean	SD	SE
Words found (prime)	Challenge	34	12.91	2.32	.39
	Threat	33	1.93	.78	.13
Difficulty (prime)	Challenge	34	3.20	1.17	.20
	Threat	33	5.63	.92	.16
Words found (task)	Challenge	34	10.91	3.14	.54
	Threat	33	9.36	3.06	.53
Difficulty (task)	Challenge	34	3.97	1.60	.27
	Threat	33	3.69	1.23	.21

Table 4.6: *T and Welch's t tests for the laboratory task*

							95% Confidence Interval	
	t	df	*p*	Mean Difference	SE Difference	Cohen's d	Lower	Upper
Words found (prime)	25.99	40.67	< .001 [b]	10.97	.422	50.60	10.11	11.82
Difficulty (prime)	9.37	65.00	< .001 [a]	–2.43	.258	–18.46	–2.94	–1.91
Words found (task)	2.04	65.00	.045 [a]	1.54	.758	4.02	.03	3.06
Difficulty (task)	.78	61.87	.438 [b]	.27	.349	1.53	–.42	.97

Note. [a] Student's T–Test; [b] Welch's T–Test.

4.7.2 Reactivity to stress

Patterns of HR, TPR and CO reactivity are shown in Table 4.7, alongside with variances and ranges associated with challenge and threat indexes. Variance between phases of the challenge and threat indexes above and below 0 indicated that some participants were challenged (e.g., experienced vasodilation) and some threatened (e.g., vasodilatation was countered by vasoconstriction) during the study's phases. The first phenomenon is believed to occur as result of SAM activation, while the latter is due to HPA activation (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996; Frings et al., 2012).

Table 4.7: *Total peripheral resistance, heart rate, cardiac output reactivity, and challenge and threat index by comparison*

Index	Reactivity comparison	Range	*M* (SD)
HR	Baseline x prime	–18.94 – 35.31	2.66 (7.14)
	Baseline x task	–14.47 – 23.66	5.45 (7.05)
	Prime x task	–35.23 – 23.77	2.79 (6.81)
TPR	Baseline x prime	–2813 – 1782	69.01 (597.3)
	Baseline x task	–3151 – 1809	20.66 (607.7)
	Prime x task	–791 – 1220	–48.35 (284.65)
CO	Baseline x prime	–3.99 – 3.60	–.01 (1.19)
	Baseline x task	–3.56 – 3.82	.12 (1.05)
	Prime x task	–2.64 – 2.10	.13 (.67)
Challenge/threat	Baseline x prime	–4.46 – 6.42	.00 (1.83)
reactivity index	Baseline x task	–5.25 – 7.22	.00 (1.85)
	Prime x task	–6.17 – 4.52	.00 (1.77)

Note. CO: cardiac output. HR: heart rate. TPR: total peripheral resistance.

4.7.3 Physiological reactivity and AB: Total sample

In this section Pearson's correlations (two–tailed) between challenge and threat indexes with bullying (Table 4.8) and psychopathy (Tables 4.9 and 4.10) are presented. Separate tables were created to aid interpretation and to respond to predictions (cf. Section 4.5.3). It was decided to present a full correlation matrix once this is recommended for potential inclusion in meta–analyses in the future, and it allows for a richer comprehension of data presented (Schmidt & Hunter, 2014). Bivariate correlations showed that C&T reactivity index between prime and task phases was negatively associated with bully (perpetration) behaviour (r = –.24, p = .04), total psychopathy (r = –.29, p = .01) and with Machiavellian Egocentricity (r = –

.25, p = .03). The association between threat appraisal with physical aggression (IBS fighting subscale) was not significant (r = –.22, p = .06) (Table 4.8). Machiavellian Egocentricity was also linked with C&T index between baseline and task phases (r = –.31, p = .01) (Table 4.9). Total psychopathy was associated with C&T index between prime and task phases (r = –.29, p = .01; Table 4.10). In summary, greater challenge was linked with less self–reported bullying (perpetrator), Machiavellian Egocentricity, and total psychopathy. A significant tendency was also detected between C&T reactivity indexes and physical bullying (fighting) and the subscale of Carefree Nonplanfulness (p's = .06), as well as for the Fearlessness factor of psychopathy (p = .07). When analyses were controlled for the effect of gender, these same associations did not change either in strength (e.g., r values) or significance levels.

Table 4.8: *Correlational analyses (bullying and C&T)*

		1	**2**	**3**	**4**	**5**	**6**	**7**
1 Total bullying	*r*	–	.79 ***	.71 ***	.78 ***	–.01	–.10	–.17
	p	–	< .001	< .001	< .001	.90	.38	.16
	Upper 95% CI	–	.87	.81	.86	.22	.13	.06
	Lower 95% CI	–	.69	.57	.67	–.25	–.33	–.39
2 Victimisation	*r*		–	.23	.55 ***	–.11	–.09	.03
	p		–	.05	< .001	.35	.42	.75
	Upper 95% CI		–	.45	.70	.12	.14	.27
	Lower 95% CI		–	–.00	.36	–.34	–.32	–.20
3 Bully	*r*			–	.36 **	.10	–.01	**–.24** *
	p			–	.003	.38	.90	**.04**
	Upper 95% CI			–	.55	.33	.22	–.004
	Lower 95% CI			–	.13	–.13	–.25	–.45
4 Fighting	*r*				–	–.02	–.15	–.22
	p				–	.82	.21	.06
	Upper 95% CI				–	.21	.09	.01
	Lower 95% CI				–	–.26	–.37	–.44
5 Baseline x Prime Reactivity	*r*					–	.86 ***	–.32 **
	p					–	< .001	.007
	Upper 95% CI					–	.91	–.09
	Lower 95% CI					–	.79	–.52
6 Baseline x Task Reactivity	*r*						–	.18
	p						–	.12
	Upper 95% CI						–	.40
	Lower 95% CI						–	–.05
7 Prime x Task Reactivity	*r*							–

Notes. * p < .05, ** p < .01, *** p < .001.

Table 4.9: *Correlational analyses (psychopathy subscales and C&T)*

		1	2	3	4	5	6	7	8	9	10
1 Blame Externalisation	*r*	—	−.11	.00	.23 *	.22	−.30 *	−.21	−.14	−.11	.08
	p	—	.34	.99	.05	.07	.01	.07	.25	.36	.48
	Upper 95% CI	—	.12	.23	.45	.43	−.07	.02	.10	.13	.31
	Lower 95% CI	—	−.34	−.23	.00	−.02	−.50	−.43	−.36	−.34	−.15
2 Carefree Non−Planfulness	*r*		—	.12	.02	.19	−.05	.12	.17	.06	−.21
	p		—	.30	.81	.10	.67	.29	.15	.61	.07
	Upper 95% CI		—	.35	.26	.41	.18	.35	.39	.29	.02
	Lower 95% CI		—	−.11	−.21	−.04	−.28	−.11	−.06	−.17	−.43
3 Fearlessness	*r*			—	.09	.24 *	.06	.13	.03	−.01	−.09
	p			—	.42	.04	.58	.28	.81	.88	.43
	Upper 95% CI			—	.32	.45	.30	.35	.26	.22	.14
	Lower 95% CI			—	−.14	.005	−.17	−.11	−.21	−.25	−.32
4 Machiavellian Egocentricity	*r*				—	.35 **	.13	−.03	−.16	**−.31** **	**−.25** *
	p				—	.003	.28	.75	.17	**.01**	**.03**
	Upper 95% CI				—	.54	.36	.20	.07	−.07	−.01
	Lower 95% CI				—	.12	−.10	−.27	−.39	−.51	−.46
5 Rebellious Nonconformity	*r*					—	.23	.21	−.01	−.10	−.15
	p					—	.06	.08	.91	.41	.19
	Upper 95% CI					—	.44	.42	.22	.14	.08
	Lower 95% CI					—	−.01	−.02	−.25	−.33	−.38
6 Social Influence	*r*						—	.37 **	.07	−.02	−.21
	p						—	.002	.53	.82	.08
	Upper 95% CI						—	.55	.30	.21	.02
	Lower 95% CI						—	.14	−.16	−.26	−.42
7 Stress Immunity	*r*							—	.03	−.04	−.14
	p							—	.78	.71	.24
	Upper 95% CI							—	.26	.19	.09
	Lower 95% CI							—	−.20	−.28	−.36
8 Baseline x Prime Reactivity	*r*								—	.86 ***	−.32 **
	p								—	< .001	.007
	Upper 95% CI								—	.91	−.09
	Lower 95% CI								—	.79	−.52
9 Baseline x Task Reactivity	*r*									—	.18
	p									—	.12
	Upper 95% CI									—	.40
	Lower 95% CI									—	−.05
10 Prime x Task Reactivity	*r*										—

Table 4.10: *Correlational analyses (total psychopathy and C&T)*

		1	2	3	4	5	6	7
1 Total psychopathy (PPI–R–40)	*r*	—	.74***	.51***	.37**	.01	–.15	**–.29***
	p	—	< .001	< .001	.002	.96	.21	**.01**
	Upper 95% CI	—	.83	.66	.56	.243	.08	–.06
	Lower 95% CI	—	.61	.31	.14	–.23	–.37	–.50
2 Fearlessness Dominance factor	*r*		—	–.05	.11	.06	–.04	–.21
	p		—	.67	.33	.58	.73	.07
	Upper 95% CI		—	.18	.34	.30	.19	.02
	Lower 95% CI		—	–.28	–.12	–.17	–.27	–.43
3 Self–centred impulsivity factor	*r*			—	.09	–.08	–.19	–.17
	p			—	.44	.51	.12	.14
	Upper 95% CI			—	.32	.16	.05	.06
	Lower 95% CI			—	–.14	–.31	–.41	–.39
4 Coldheartedness factor	*r*				—	–.03	–.14	–.22
	p				—	.78	.24	.06
	Upper 95% CI				—	.20	.10	.01
	Lower 95% CI				—	–.27	–.36	–.43
5 Baseline x Prime Reactivity	*r*					—	.86***	–.32**
	p					—	< .001	.007
	Upper 95% CI					—	.91	–.09
	Lower 95% CI					—	.79	–.52
6 Baseline x Task Reactivity	*r*						—	.18
	p						—	.12
	Upper 95% CI						—	.40
	Lower 95% CI						—	–.05
7 Prime x Task Reactivity	*r*							—

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

A method for FDR was applied to the analyses presented in Tables 4.8 to 4.10. According to this procedure, the associations that retained significance were those detected for Machiavellian Egocentricity with the reactivity index between baseline and task phases (new *p* value = .03), and between the total score on the PPI–R–40 and the reactivity index between prime and task phases (new *p* value = .02).

4.7.4 Physiological reactivity and AB: Examining the effects of priming conditions and gender

In order to obtain a more detailed account of the single effects of challenge and threat priming conditions, subsequent analyses were conducted separately for those who received either a threatening (Supplementary Tables 5 to 7) or a challenging experimental allocation (Supplementary Tables 8 to 10). These tables are displayed in the Appendix G. As for the threatening condition, Supplementary Table 5 showed a positive and moderate link between the reactivity index for baseline and task phases with bullying victimisation (r = .38, p = .03). Still on this condition, a negative link between the subscale of Carefree Non–Planfulness with the prime x task reactivity was noted (r = – 36, p = .04; Supplementary Table 6). The Benjamini and Hochberg's method (1995) would indicate that these associations are not significant when accounted for the effects of multiple comparisons (p's > .05).

Results for the challenge condition would suggest a significant association between psychopathy and physiological reactivity. For instance, Supplementary Table 10 contains negative and moderate links between the reactivity index between prime and task phases with Fearlessness Dominance (r = –. 34, p = .04), and Coldheartedness (r = –.35, p = .04) and total psychopathy (r = – .35, p = .04); however, when accounted for the effects of multiple comparisons, new p values were above .05 (e.g., p = .08).

To obtain a more detailed account of the effects of challenge and threat in respect to participants' gender, subsequent analyses were conducted separately for male (Supplementary Tables 11 to 13) and female participants (Supplementary

Tables 14 to 16). These tables are displayed in the Appendix G. There were no significant associations between the physiological reactivity indexes with both bullying and psychopathic personality traits amongst males. Nonetheless, for the group of women only, there was a significant association between total psychopathy with the reactivity index between prime and task phases (r = –.36, p = .007, corrected p value; cf. Supplementary Table 16).

4.7.5 Predictors of physiological reactivity indexes

Several assumptions were checked before running regression analyses. Homoscedastic requirements were analysed in *R* for the relationships between the predictors which survived analyses of FDR (that said, between Machiavellian Egocentricity with the baseline x task reactivity index, and between the total score on the PPI–R–40 and the prime x task reactivity index). The Harvey–Collier's test indicated linearity for indexes between baseline x task ($HC_{(64)}$ = .23, p = .81) and prime x task ($HC_{(65)}$ = .45, p = .64) phases. Moreover, non–significant results for Bresuch–Pagan test (BP) indicated the absence of problems regarding heteroskedasticity for the reactivity between baseline x task ($BP_{(2)}$ = 7.53, p = .06) and for the model predicting the reactivity between prime x task ($BP_{(1)}$ = .01, p = .99) (Berry & Feldman, 1985).

Using the total sample, linear regression analyses were run to predict the reactivity indexes from psychopathic personality traits. The model for prime x task reactivity showed that total psychopathy (PPI–R–40) significantly predicted this index (β = –.29, p = .013) ($F_{(1,67)}$ = 6.45, p = .013, R^2_{Adj} = .07). Moreover, a significant predictive role of Machiavellian Egocentricity (β = –.31, p = .01) ($F_{(1,67)}$ = 7.06, p = .01, R^2_{Adj} = .08) was noted for the reactivity index between baseline and task phases. Subsequent regression models were not examined for reactivity indexes according to prime conditions since the analyses reported in the section 4.7.4 suggested that, when accounted for the effects of multiple comparisons, new p values were above .05. In addition, Supplementary Table 17 contains results from hierarchical regression models that included gender in the first step, revealing that gender was not a significant predictor of the reactivity indexes between baseline and task phases, and also between prime and task phases.

4.8 Discussion

Problematic behaviours seen in psychopathy and in other forms of antisocial, aggressive behaviours can reflect developmental processes, in which pre–existing tendencies and vulnerabilities interact and mature over time (Loeber & Hay, 1997; McLaughlin et al., 2014). As for physiological reactivity amongst individuals with high levels of psychopathic personality traits, there is an understanding that some mechanisms linked to stress–responsive systems could potentially be altered due to genetic and environmental factors (Hamilton et al., 2008; McLaughlin et al., 2014; Schneiderman et al., 2005; Van Voorhees & Scarpa, 2004).

This study considered related risks–factors for developing emotional and behavioural difficulties associated to antisocial behaviours, such psychopathic tendencies, involvement with bullying and aggression (Hansel et al., 2007; McCuish et al., 2015; Wendt et al., 2017). Following previous work showing that physiological threat has been associated with self–reported negative affect (Chida & Hamer, 2008; Mak et al., 2004; Mendes et al., 2001), this study hypothesised that psychopathic personality traits – as more closely linked to negative than to positive affect – would be inversely associated with challenge. Moreover, since patterns of threat appraisals have been linked to aggression (Hamilton et al., 2008; Lopez–Duran et al., 2009), this study also predicted that bullying behaviours would be negatively correlated with challenge and threat reactivity indexes. These hypotheses will be discussed in turn.

4.8.1 Physiological reactivity and psychopathy

Research in social and experimental psychology commonly analyses the effects of task conditions in respect to different phases within a given study (Blascovich, 2014; Rohleder, Beulen, Chen, Wolf, & Kirschbaum, 2007; Scheepers & Eemers, 2005; Van Zuure & Muris, 1993). Reactivity indexes used in this study would suggest that psychopathic personality traits affect physiological responses to stress at different moments. For instance, correlational analyses (corrected for multiple comparisons) indicated that Machiavellian Egocentricity was negatively linked with the reactivity index between baseline x task phases. Total psychopathy, in turn, was negatively associated to the reactivity index between prime x task

phases. These two reactivity indexes designate physiological changes that occurred after the conclusion of the priming condition. Indeed, there were no significant associations between psychopathic personality traits with physiological reactivity in the period comprehending the recording of the last two minutes of baseline and the first two minutes of the prime phase.

Regression analyses suggested that total psychopathy (β = –.29, p = .013; R^2_{Adj} = .07) and Machiavellian Egocentricity (β = –.31, p = .01; R^2_{Adj} = .08) negatively predicted physiological reactivity between prime x task, and between baseline x task phases, respectively. Simply put, individuals high on total psychopathy and in traits resembling Machiavellian Egocentricity evaluated the experimental demands as exceeding their resources or abilities to cope. Hence, components indexing lack of empathy and emotional detachment for achieving one's own desires were accompanied by poor cardiovascular efficiency, higher negative affect and hormonal responses, and less cognitive capacity (Crum et al., 2017; Kassam, Koslov, & Mendes, 2009). These threatening responses indicate to abnormal reactivity within the HPA axis. The supposed origins for dysregulations in these stress responsive systems are still under investigation, but some scholars argue that when the organism is under prolonged exposure to stressful events, an impairment in the hippocampus diminishes the capacity for controlling the release stress–related hormones (Jacobson & Sapolsky, 1991; Pelkey, Chittajallu, Craig, Tricoire, Wester, & McBain, 2017).

Even though it is potentially difficult to compare the findings presented so far with different studies due to the methodological uniqueness of the BPSM approach, these results build upon previous theoretical models for psychopathy. The associations between psychopathic personality traits with threat appraisal add evidence to Blair's (2006; 2010b) postulations about the connection of psychopathic tendencies with hyper physiological reactivity to frustration. What could have happened to the participants of this study is that, when confronted with the possibility of not receiving the expected reward (e.g., chocolate bar), the threat circuitry was activated via top–down regulation from the frontal cortex, which is assumed to have a cascade effect on the amygdala and hypothalamus (Blair, 2004; 2010b).

The hypothalamus, along with the amygdala, is a region closely implicated in individual responses to environmental stress (Blair et al., 2005). The threat circuitry, in simple terms, is initially activated within the amygdala, which in turn connects to the hypothalamus. The hypothalamus then activates the pituitary gland to release ACTH. ACTH then enters the bloodstream via the adrenal gland, releasing cortisol. Cortisol, then, attaches to specific receptors at hippocampal regions (Blair et al., 2005). Moreover, stress can result in permanent damage of the dendrites of the hippocampus (McEwen, Gould, & Sakai, 1992). In this respect, it would then be sensible to speculate about environmental factors accounting for certain biological deficiencies seen in psychopathy (Newman, 1998). There has been also support that environmental stress might indirectly produce dendritic arborization within the amygdala (basolateral nucleus; Vyas, MitraShankaranarayana–Rao, & Chattarji, 2002), as well as can modulate hormonal responses to threat (Bremner & Vermetten, 2001; Charney, 2003). Considering that the amygdala constitutes the threat circuitry, these changes would implicate in augmented risk for antisocial behaviours (Blair et al., 2005)[21].

4.8.2 Physiological reactivity and bullying behaviours

In terms of the hypothesis around the associations between physiological reactivity with aggression and bullying, findings were less clear. This investigation predicted that bullying behaviours would be associated negatively with reactivity indexes of challenge and threat. Before performing corrections due to multiple comparisons, the behaviour of bullying others (i.e., bullying perpetration) was negatively associated with the reactivity index between prime x task phases. This means that the more participants have reported that they bully others, the less

[21] According to the model of neurovisceral integration of emotions, the prefrontal cortex (e.g., orbitofrontal cortex and medial prefrontal cortex) constrains the amygdala, which in turn could result in abnormalities in terms of physiological and behavioural responses (Thayer & Lane, 2009). Clearly, there is a need for better understanding of the top–down regulation of prefrontal cortex that results in amygdala attenuation. One promising mechanism argues that 'biased' information could be communicated from the prefrontal cortex to other areas to promote the most adaptive, automatic response to emotional stimuli (Davidson, 2002). The source of this bias might have its roots in a type of somatic marker (Damasio, 1996). In psychopathy, it is likely that the somatic marker system proposed by Damasio is deficient. Evidence for this is mostly apparent for those high in psychopathic traits who have difficulties inhibiting dissocial tendencies regardless of their inappropriateness within the context (hence avoiding associated consequences). Differences are also reflected in inferior performance in tasks that require attentional shifts, which resembles performance found in patients with orbitofrontal lesions (Blair, Colledge, & Mitchell, 2001; Brower, 2001; Raine & Yang, 2006).

challenge they experienced during the laboratory task. Although these findings did not retain statistical significance when a FDR method is used, they provide some support to early examinations of psychophysiological reactivity amongst children and adolescents with severe involvement with AB, but not elevated CU traits (Crozier et al., 2008; Kimonis, Fanti, Goulter, & Hall, 2017; Lopez–Duran et al., 2009; Walker et al., 2016). Nonetheless, previous work examining physiological reactivity to stress and its associations to AB and bullying focused on changes in HR and HRV (Crozier et al., 2008) or cortisol (Gonzalez–Cabrera, Calvete, León–Mejía, Pérez–Sancho, & Peinado, 2017; Knack et al., 2011; Lopez–Duran et al., 2009). Additionally, past research was limited to samples of children (Lopez–Duran et al., 2009), mixed adolescents (Gonzalez–Cabrera et al., 2017; Knack et al., 2011), and adolescent boys (Crozier et al., 2008). As such, future studies, both in children and adults, might perhaps clarify the psychophysiological profile of individuals with different degrees of involvement with bullying. In addition, some of the relationships included in the supplementary tables are worth commenting on. Prior to correcting for multiple comparisons, an unexpected finding was noted for bullying victimisation and physiological reactivity. Although this thesis' hypotheses did not include any prior prediction for victimisation, the positive correlation between this form of bullying involvement with physiological reactivity would suggest that experiencing victimisation could be linked to a more efficient mobilisation of physiological resources in face of motivated–performance tasks. Even though it was beyond the scope of this investigation to further explore how information processing might interact with biobehavioural mechanisms to explain problematic behaviours, there is evidence that involvement bullying might affect individual's ability to process information (Mahady–Wilton et al., 2000; Pincham, Bryce, & Fearon, 2015). Certainly, clarification into these issues are much needed, which encourage future research on the intersection of cognition, culture and biology.

4.8.3 Implications and limitations

One direct implication of the study of mechanisms underpinning reactivity to stress is related to prevention of important societal problems, such as violence and

aggression (Krueger et al., 2002). In turn, there are claims in the literature for clearer explanations about patterns of neuroendocrine and psychophysiological responses linked to the threat circuitry amongst those presenting with elevated levels of externalising traits (Kotov et al., 2017) and psychopathy (Blair, 2010b). This is much needed given the negative impact of psychopathic and antisocial behaviours, and considering the high co–occurrence of both phenomena. Indeed, psychopathic features commonly labelled as secondary psychopathy are strongly (r = .97) associated with the latent externalising spectrum of adult psychopathology, defined by the covariance of symptoms of AB, disinhibitory tendencies, and substance use and abuse (Krueger et al., 2002).

This study has also some theoretical implications. Hamilton, Hiatt–Racer, and Newman (2015) suggested that dysfunctions seen in the context of psychopathy could occur as consequence of distinct neural functionalities, which interact with cognitive and affective processing. Data seem to provide further consideration to the Response Modulation Hypothesis of Psychopathy (Gorenstein & Newman, 1980; but see Newman & Baskin–Sommers, 2011). From baseline to task phases, participants higher on psychopathic personality traits of Machiavellian Egocentricity showed reactivity indexes compatible to threat. Hence, it is assumed that elevated scores on this specific subscale of the PPI–R–40 (Eisenbarth et al., 2015) have detrimental effects on participant's ability to shift attention once they become focused on rewarding outcomes (Smith & Lilienfeld, 2015). This study has also some overlap with the BIS/BAS motivational systems literature, in which psychopathic variants are believed to predict behaviour (e.g., primary traits would be marked by lower inhibition, while secondary variant would reflect a strong sensitivity to reward; Carver & White, 1994). However, instead of using self–report measures to infer about participant's physiological processes, the BPSM uses a robust procedure for assessing biological indicators of approach and avoidance that predict behaviour and emotions (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996; Frings et al., 2012; McLaughlin et al., 2014; Seery, 2011).

Even though the BPSM accounts for cognitive processes underlying participant's approach to a given goal–relevant task, these were not explored. As

such, research into basic psychological processes amongst individuals with various degrees of psychopathic personality traits might further clarify biobehavioural mechanisms involved in stress response for this population. As the laboratory setting is rather controlled, participants might have experienced feelings of being assessed and judged. However, in forensic and, to some extent, organisational and educational settings, individuals do go through periodic episodes of assessment. What data suggested is that those with high levels of psychopathy could perceive these events as threatening. Cumulative evidence paired these appraisals with negative affect, poor focus, and prejudicated performance (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996).

One strong aspect of this study refers to the measurement of neuroendocrine responses in real time, as opposed to methods assessing levels of stress reactivity using saliva sampling and blood testing (Hamilton et al., 2008; Mendes & Park, 2014; McLaughlin et al., 2014). Further work might particularly benefit from adding extra measures of psychopathic personality traits and diagnosis tools, such as the PCL–R (Hare, 2003) and interpersonal assessment of the condition (Kosson, Steuerwald Forth, & Kirkhart, 1997), combined with other sources of biological data in order to advance the field[22]. Nonetheless, the generalisation of these findings is limited, predominantly due to the fact of using a non–probabilistic sample of adults recruited from the community. Another important limitation is related to the unequal proportion of males to females. Even though the BPSM has methodological procedures for accounting for gender effects at the physiological level, it is often difficult to perform post–hoc controlling for psychological cofounders. Certainly, a promising avenue for research could include diversified recruiting procedures so that demographical bias might be avoided. However, and perhaps much important for the time being, future work could particularly focus on strategies for preventing and or treating those abnormal physiological responses amongst individuals at risk. Certainly, the effect of pharmacological drugs could potentially give further insight on this matter, as well

[22] In respect to this topic, it is important to stress the recent claims made by the Research Domain Criteria (RDoc) for more biologically–oriented investigations for advancing the scientific study of psychopathy (cf. Patrick & Hajcak, 2016), as well as the growing interest on the Externalising Spectrum – containing psychopathy and aggressive behaviours such as bullying – within an Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017; Patrick et al., 2013).

as prevention and intervention programmes that aim to target impaired integration of attentional and affectional processes (Baskin–Sommers, Curtin, & Newman, 2015; Hamilton et al., 2015; Newman, 1998).

4.9 Chapter summary

In summary, findings reported in the third study could help in better understanding specific traits that might strongly affect autonomic hypo– or hyper–arousal, adding to emergent conceptualisations of psychopathy as a heterogeneous (Hauck–Filho, Teixeira, & Dias, 2012; Portnoy & Farrington, 2015; Thompson, Ramos, & Willet, 2014), multidimensional phenomenon (Brazil et al., 2016; Neumann, Hare, & Pardini, 2015). In addition, results might be particularly useful in future programmes aiming to provide treatment for subgroups of individuals with high levels of psychopathy personality traits but with differential levels of arousal within the ANS (Hansen et al., 2007; Stadler et al., 2008). Considering that the evidence regarding low arousal within the cardiovascular system is robust and highly replicable, perhaps one potential (and important) step might include studies that directly manipulate levels of arousal and track these effects across time.

Findings from the fourth study provided continuity to early reports in which physiological reactivity was explored amongst children and adolescence (Crozier et al., 2008; Lopez–Duran et al., 2009; McLaughlin et al., 2014). However, no previous studies were located combining measures of externalising problems – such as bullying and psychopathic personality traits – in the study of the threat circuitry. Thus, data from Study 4 seem to suggest to an altered functioning of the HPA axis, which has been previously linked with more aggression, less empathic behaviours, and higher levels of psychopathy (Chida & Hamer, 2008; Hamilton et al., 2008; McLaughlin et al., 2014; Sandi & Haller, 2015).

In combination, results of Studies 3 and 4 have the potential to become informative for model specifications for further research aiming to investigate autonomic and neuroendocrine biological differences amongst psychopaths and people high in psychopathic personality traits, a promising area for research (Blair, 2015; Dalgeish, 2004; Patrick et al., 2013; Venables et al., 2017). Another question

that remains unanswered regards the validity of psychopathic personality traits in predicting stress beyond the physiological level. Hence, it is the purpose of the next chapter to explore whether these findings can be replicated at the behavioural level, incorporating an examination of empathic deficiencies (in particular affective empathy) in the psychopathy–stress association.

CHAPTER FIVE – THE ROLE OF EMOTIONAL DEFICITS IN UNDERSTANDING THE ASSOCIATIONS BETWEEN PSYCHOPATHIC PERSONALITY TRAITS AND SELF–REPORTED STRESS

Chapter overview

The previous chapter discussed some characteristics by which individuals with high levels of psychopathic personality traits could differ from those presenting with low levels of psychopathy in terms of their physiological basal functioning and in their physiological reactivity to stress. The data indicated there seems to exist two major ways in which psychopathic features could explain different patterns of stress reactivity: 1) A hypo–responsiveness pattern is dominant amongst those characterised as fearless (or equivalent to Hare's PCL–R Factor 1; Hare, 2003); and 2) another variant of psychopathy is likely to be marked by hyper–reactivity to stress (Johnson et al., 2015). Amongst these cases (also known as secondary or type–II) are individuals who show less propensity to be immune to stressors (Patrick, 1995; Verona, Patrick, & Joiner, 2001).

Study 3 suggested evidence towards autonomic hypo arousal while at rest for individuals high in traits under Factor 1, whereas Study 4 showed that total psychopathic personality scores and the subscale of Machiavellian Egocentricity were linked with physiological threat responses (where those higher in psychopathy showed enhanced HPA reactivity). Together, these findings are in line with a pattern of autonomic hypo arousal, combined with a highly reactive neuroendocrine system (Marsh et al., 2008; Mills–Koonce et al., 2015; Nederhof et al., 2015) and are indicative of these responsive systems acting in opposition to each other to achieve allostasis in psychopathic variants. A remaining question is whether this pattern could be replicated at the behavioural level. Consequently, it is the purpose of this chapter to further investigate the associations between psychopathic personality traits and levels of perceived stress. Considering that a particular profile of emotional deficits is essential in differentiating psychopathy from other personality disorders (Blair, 1999), this work also aims to explore whether participant's understanding of their own emotions could potentially

mediate the relationships between primary and secondary psychopathic traits and levels of perceived stress.

5.1 Study 5 – The role of emotional deficits in understanding the associations between psychopathic personality traits and self–reported stress in adults

The role of biological and behavioural indicators of stress response have gained substantial importance in studies within the field of biosocial criminology in recent years (Barker & Meehan, 2018; Johnson et al., 2015). On the face of the psychopathic literature, two interesting models for understanding the links between stress and these personality traits exist. One model suggests that psychopathy is marked by a distinct profile of emotional deficits (Blair, 1999), while another proposes a higher defensive reaction–threshold in individuals with elevated psychopathic traits (Patrick, 2001). While the latter assumes that a very intense emotional valence is necessary to mobilise an individual high in psychopathy traits, the former posits that those individuals are biologically deficient in effectively processing emotional content. These models provide useful information regarding lack of emotional responsivity to stressful situations, which, amongst other indicators, is more apparent in psychopaths' inability to display startle potentiation when dealing with distress (Blair, 1999; Patrick, 2001). These emotional deficits are also reflected in little or no changes in skin conductance reactivity to emotional stimuli (Herpertz et al., 2007; Marsh et al., 2008), smaller changes in HR in response to distress (Lorber, 2004; Osumi et al., 2007; Serafim et al., 2009), and diminished amygdala reactivity to fear stimuli (e.g., Dotterer, Hyde, Swartz, Hariri, & Williamson, 2017; McCloskey, Phan, & Coccaro, 2005; Völlm, Richardson, McKie, Elliott, Dolan, & Deakin, 2007).

Although experimental evidence showing physiological deficits in stress reactivity and recovery are relatively abundant (e.g., Beauchaine et al., 2007; Mulder, 2011; Nederhof et al., 2015; Sijtsema et al., 2015), few studies have been conducted examining objectively the associations between psychopathy and levels of perceived stress. Previous work explored the associations of psychopathic personality traits to post–traumatic stress (Barker & Meehan, 2018; Pham, 2012) and between antisocial personality disorder and perceived stress (Goldstein,

Dawson, Smith, & Grant, 2012). Past research also linked dimensions commonly seen in psychopathy (i.e. narcissism) to elevated self–report perceived stress (Besser & Zeigler–Hill, 2011; Sommer, Kirkland, Newman, Estrella, & Andreassi, 2009). A moderate, positive correlation between perceived stress and negative affect amongst adults was detected by Watson (1988), who also reported a weak, negative association between stress and positive affect. Interestingly, Silver et al. (2011) found that psychopathy and perceived stress are amongst significant (positive) predictors of concomitant violence offending and victimisation. In addition, Crozier et al. (2008) assessed the evidence for the fear deficit hypothesis and the hyper–responsiveness to minor stressors seen in individuals presenting with antisocial tendencies by using a social processing paradigm. The experiment involved viewing videotaped vignettes followed by listening to narrated stories. The social processing paradigm stimuli contained ambiguous provocation, aggressive response to provocations, and nonaggressive response to provocations. In both approaches (e.g., video and audio), participants were asked to imagine themselves as the protagonist in the hypothetical event, and questions were followed to assess how they processed the socially relevant information presented in the stimuli (e.g., "How would you want this situation to turn out?"; Croziet et al., 2008, p. 7).

Responses were transformed so higher scores indicated positive evaluation of aggressive reactions, which was used as a proxy of social information processing deficits in the study. Researchers discovered that children and adolescents with low levels of autonomic arousal (e.g., heart rate) displayed more engagement with antisocial behaviours without the mediation effect of social information deficiencies. This pattern was called "trait–like", and those individuals were more likely to present with fearlessness, manipulation and retaliation behaviours. On the other hand, this study found another pattern of high autonomic reactivity to stressors, in which deviant social information processing mediated the links between antisocial behaviours and heart rate measurements at rest and in response to provocations (Crozier et al., 2008). This investigation forms the basis of the hypothesis that cognitive psychological processes might be indirectly driving individuals with high levels of antisocial behaviours to respond to minor provocations in a maladaptive manner. Consequently, it might be worthwhile to

further understand if other emotional deficits also offer increment utility in the comprehension of stress in psychopathy. Amongst these, alexithymia is certainly a strong candidate. As Takamatsu and Takai (2017) noted, the construct of alexithymia has shown particular relevance in the scientific study of psychopathy as both conditions entail deficiencies in empathic response. Meaning 'no words for emotion', alexithymia denotes a very specific limitation in individual's affective empathy (Bird & Viding, 2014; Oakley, Brewer, Bird, & Catmur, 2016; Takamatsu & Takai, 2017), being negatively linked with empathy (Bird et al., 2010; Grynberg et al., 2010) and positively with psychopathy (Lander et al., 2012; Louth et al., 1998). Those who present with social information deficits in favour of use of aggression to solve ambiguous provocations are arguably less empathic, particularly in respect to the affective component of empathy (Bird & Viding, 2014; Blair, 1999; Crozier et al., 2008; Takamatsu & Takai, 2017).

Considering the literature reviewed above, this study was designed (i) to investigate whether there are associations between perceived stress and psychopathy, and, if significant, are these results in line with psychophysiological findings, presented both in this thesis and with the broad psychopathy literature? This study also sought to explore which variants of psychopathy could potentially predict self–report stress in adults. As suggested by Blair (1999), emotional deficits could mediate behavioural responses seen in psychopathy; hence, this study will also be examining (ii) the predictive validity of psychopathy on understanding perceived stress and (iii) the possible mediator role of emotional deficits, in particular alexithymia, in the relationship between stress and psychopathic variants.

5.1.1 Hypotheses

Based on previous research and congruent with physiological data obtained via experimental design (Chapter Four), the following hypotheses were set forth. Firstly, it was foreseen that there would be positive associations between perceived stress and psychopathic personality traits. Specifically, traits belonging predominantly to Factor 2 (e.g., self–centred impulsivity or secondary variants) will be more robustly linked to self–report perceived stress than those essentially belonging to Factor 1 (interpersonal and affective traits or primary variants).

Empirical data indicates that immunity to stress loads mostly into the primary variant of psychopathy (Benning et al., 2003; Eisenbarth et al., 2015; Neumann et al., 2008). From this assumption stems the second hypothesis, which foresees that secondary (and not primary) psychopathic traits should predict total levels of stress. Additionally, given that psychopathic personality traits can be comorbid with alexithymia, mainly in terms of emotional deficits (Apfel & Sifneos, 1979; Cairncross, Veselka, Schermer, & Vernon, 2013; Grieve & Mahar, 2010; Grynberg et al., 2010; Guttman & Laporte, 2002; Lander et al., 2012), alexithymia dimensions would be a significant mediator in the relationship between perceived stress and primary and secondary psychopathy. However, larger effects were expected to occur for the secondary variant (Benning et al., 2003; Boozer et al., 2005).

5.2 Method

5.2.1 Participants, procedures, and design

This cross–sectional study involved 264 adults (M_{age} = 21.7, SD = 6.92). Levels of perceived stress were set as the dependent variable, and psychopathic variants – along with alexithymia dimensions – as independent variables. For this study, a percentage of 70.1 was accounted for females. *G*Power (version 3.1.9.2) was used to confirm that for all analyses the number of participants was sufficiently enough for securing 95% of power and α = .05 or less (Cohen, 1988; Faul et al., 2007). Participants responded to the questionnaires online using Goldsmiths University Qualitrics account from July 2016 to April 2017. Measures were presented after obtaining formal consent from participants, which were recruited on the basis of being aged 18 years–old or more, fluent in English, and regularly enrolled as university students. In addition, prior to completing the measures, participants were reassured about their rights to abandon the study at any time, as well as being informed about data privacy and anonymity issues. Course credits were granted as a form of retribution for participation.

5.2.2 Measures

5.2.2.1 Levenson Primary and Secondary Psychopathy Scales (LPSP; Levenson et al., 1995)

This measure has been previously described (Section 3.2.2.3.2). In the current study, Cronbach's alphas were adequate (α = .86 for the total scale, α = .84 for the primary psychopathy scale and α = .70 for the scale measuring secondary psychopathy).

5.2.2.2 Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983)

The Perceived Stress Scale (PSS) is a widely used instrument for measuring the perception of stress amongst community samples. It is a 10–item measure arranged on a 5–point scale varying from never (0) to very often (4). The PSS requires participants to rate the frequency in which they experienced stressful situations in the past month (Cohen et al., 1983). Research has shown high internal consistency and evidence of predictive and construct validity for the PSS in the realm of psychopathy (Fite et al., 2010; Loeber, Farrington, Stouthamer–Loeber, & Van Kammen, 1998; Neumann & Pardini, 2014). Importantly, the PSS has shown to cover more strongly the negative dimensions of stress[23], which is demonstrated in studies showing moderate to strong positive associations with negative affect (Denovan, Dagnall, Dhingra, & Grogan, 2017) and positive and moderate links with depression (Chang, 1998). In this study the internal consistency was good (α = .86) and the measure is presented in Appendix E.

5.2.2.3 Toronto Alexithymia Scale (TAS–20; Bagby, Parker, & Taylor, 1994)

This is the most widely used measure of alexithymia and items are displayed in a 5–point Likert scale ranging from "Strongly disagree" (1) to "Completely agree" (5). Normally, the TAS–20 is divided into a 3–factor structure: difficulties identifying feelings (DIF; 7 items); difficulties describing feelings (DDF; 5 items);

[23] As opposed to the 'so–called' beneficial stress, optimal stress or resilience to stress (Koerber, Rouse, Stanyar, & Pelletier, 2017; Minois, 2000; Yerkes & Dodson, 1908). The PSS is negatively correlated with measures of resilience (Abolghasemi & Varaniyab, 2010) and quality of life (Chang, 1998). Mitigating factors against the negative consequences of stress are largely unknown, albeit research has linked strong social support, psychological well–being and other environmental factors are important in promoting resilience to stress (Ozbay, Johnson, Dimoulas, Morgan, Charney, & Southwick, 2007).

and externally oriented thinking (EOT; 8 items). Cronbach's alpha for the current study were α = .80, .70, .80 and .63 for the global scale, DIF, DDF, and EOT, respectively. Low values for the EOT Cronbach's alpha are recurrent in the literature (Loas, Speranza, Pham–Scottez, Perez–Diaz, & Corcos, 2012; Meganck, Markey, & Vanheule, 2012; Rieffe, Oosterveld, & Terwogt, 2006; Zimmermann, Quartier, Bernard, Salamin, & Maggiori, 2007; see also: Loas, Braun, Delhaye, & Linkowski, 2017). The TAS–20 is presented in Appendix D.

5.2.3 Data analysis

Firstly, uni– and multivariate normality assumptions tests (e.g., linearity, normality, multicollinearity, and homoscedasticity) were checked and will be fully reported in the section 5.3.4. Secondly, to respond to the first aim, this chapter will report on correlations (Pearson) between self–report perceived stress with primary and secondary psychopathy. Additionally, psychopathic variants will be entered in a regression model in order to predict behavioural stress in this sample (aim 2). To account for possible effects of emotional deficits in explaining the links between self–report stress and psychopathic traits, a series of mediation analyses will be run independently for each model (e.g., primary and secondary variants). Warren (2009) has commented that structural equation models might add additional insight mediation analyses once the effects of shared variances can be taken in consideration, which supports the decision to explore the individual impact of each variant of psychopathy on stress, including direct and indirect links between primary and secondary psychopathy via dimensions of alexithymia (aim 3). Specific methodological procedures for best responding to these 3 aims will be described within the corresponding sections.

5.3 Results

5.3.1 Correlational analyses

Table 5.1 presents the full correlation (Pearson) matrix between perceived stress and psychopathy variants. The association between perceived stress with secondary psychopathy was over twice the size of the correlation detected for primary psychopathy. According to the 95% confidence interval (CI), a coefficient

of correlation of .30 to .53 exists between secondary psychopathy and self–reported stress, which falls into the moderate strength classification (cf. Mukaka, 2012).

Table 5.1: *Correlations between psychopathy and perceived stress*

		1	2	3
1 Primary psychopathy	*r*	–	.54 ***	.19 **
	p	–	< .001	.007
	Upper 95% CI	–	.62	.31
	Lower 95% CI	–	.43	.05
2 Secondary psychopathy	*r*		–	.42 ***
	p		–	< .001
	Upper 95% CI		–	.53
	Lower 95% CI		–	.30
3 Perceived stress	*r*			–

Note. * p < .05, ** p < .01, *** p < .001

5.3.2 Regression analyses

Regression results are depicted in Table 5.2. Secondary, but not primary, psychopathic personality traits significantly and positively predicted overall self–reported perceived stress, explaining 17.3% of the variance.

Table 5.2: *Predictors of perceived stress*

	Unstandardized	Standard Error	Standardized	t	p
Intercept	18.35	2.30		7.98	< .001
Primary psychopathy	–.05	.07	–.05	–.71	.47
Secondary psychopathy	.67	.11	.45	5.96	< .001

Note. Collinearity values were acceptable (VIF 1.439 for both predictors). The model survived analyses of residuals (Durbin–Watson = of 1.813).

5.3.3 Mediation analyses

Several assumptions were checked and confirmed before examining mediation effects of alexithymia' dimensions in predicting perceived stress (Baron & Kenny, 1986; MacKinnon et al., 2002). Although primary psychopathy has not directly predicted stress, the use of mediation analyses procedures could reveal indirect

paths (cf. Warren, 2009). Consequently, in the mediation models tested, both primary and secondary psychopathic traits were set as independent variables (IV).

5.3.3.1 Mediation model 1 – Difficulties describing feelings

Mediation requires that the mediator predicts the dependent variable (DV; perceived stress). Results are in line with this requirement (R^2 = .14, β = .37, p < .001). In addition, the independent variables (IV) should also predict the mediator. This assumption was confirmed for primary (R^2 = .05, β = .24, p < .001) and secondary psychopathy (R^2 = .18, β = .43, p < .001). Another requirement is that when IV's and the mediator are included together, the relationship between IV and the dependent variable (DV) declines and the variance explained increases (Jose, 2013). This condition was again supported for both models. Beta's value decreased to .09 and R^2 increased to .14 in the primary psychopathy model (Sobel's z= 3, p = .002, with .46 indirect to ratio effect size; the bootstrapped unstandardized indirect effects was significant (.08 [95% CI .03 to .15]). (Figure 5.1). For secondary psychopathy, beta decreased to .31, and there was an increase in the variance explained (R^2 = 21.8), with .38 indirect to ratio effect size (Sobel's z= 3.10, p = .001; the bootstrapped unstandardized indirect effect was significant (.15 [95% CI .05 to .26]) (Figure 5.2).

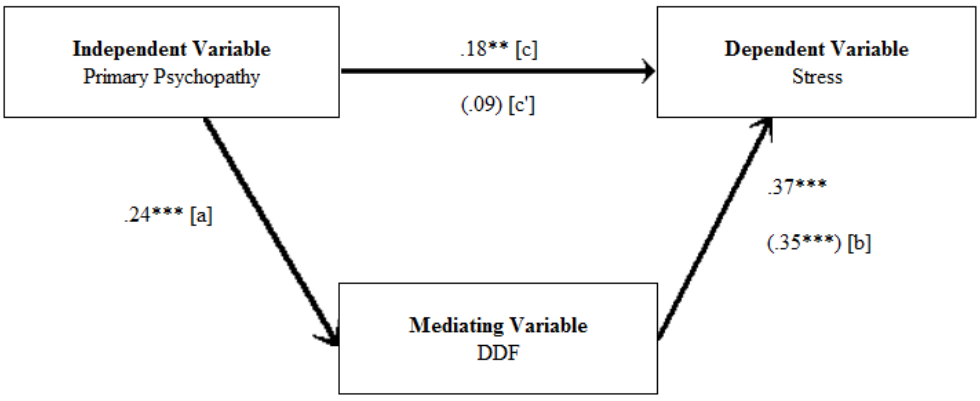

Figure 5.1 – Mediation role of DDF between primary psychopathy and perceived stress.
Note. The values in parentheses are beta weights and the other values correspond to correlations (Pearson).

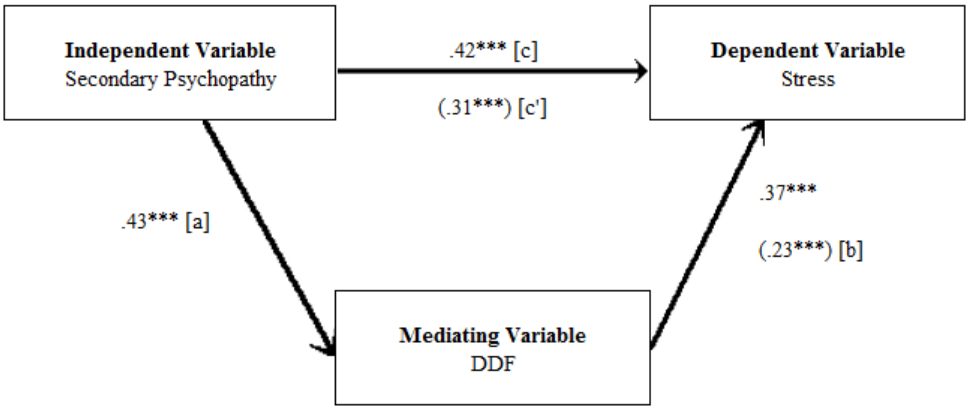

Figure 5.2 – Mediation role of DDF between secondary psychopathy and perceived stress.
Note. The values in parentheses are beta weights and the other values correspond to correlations (Pearson).

5.3.3.2 Mediation model 2 – Externally oriented thinking

Sobel's test for mediation effects of this dimension of alexithymia in the relationship between primary and secondary psychopathy with perceived stress returned non–significant results ($p > .05$). Moreover, bootstrapped unstandardized indirect effects were non–significant (– .01 [95% CI – .08 to .05] and – .03 [95% CI – .10 to .02] for the models taking primary and secondary psychopathy as predictors, respectively).

5.3.3.3 Mediation model 3 – Difficulties identifying feelings

Results confirmed the assumption of the mediator predicting the DV ($R^2 = .29$, $\beta = .54$, $p < .001$). In addition, the IV's also predicted the mediator for primary ($R^2 = .02$, $\beta = .24$, $p = .02$) and secondary psychopathy ($R^2 = .12$, $\beta = .36$, $p < .001$) models. Regarding the last requirement, beta's value decreased to .09 and R^2 increased to .29 in the primary psychopathy model (Sobel's $z = 5.12$, $p = .006$, with .44 indirect to ratio effect size; the bootstrapped unstandardized indirect effect (.08) was significant [95% CI .01 to .16]). (Figure 5.3). For secondary psychopathy, there was an increase in the variance explained ($R^2 = .34$) and in the value of beta (.38), with .38 indirect to ratio effect size (Sobel's $z = 5$, $p < .001$; the bootstrapped unstandardized indirect effect (.23) was significant [95% CI .14 to .35) (Figure 5.4).

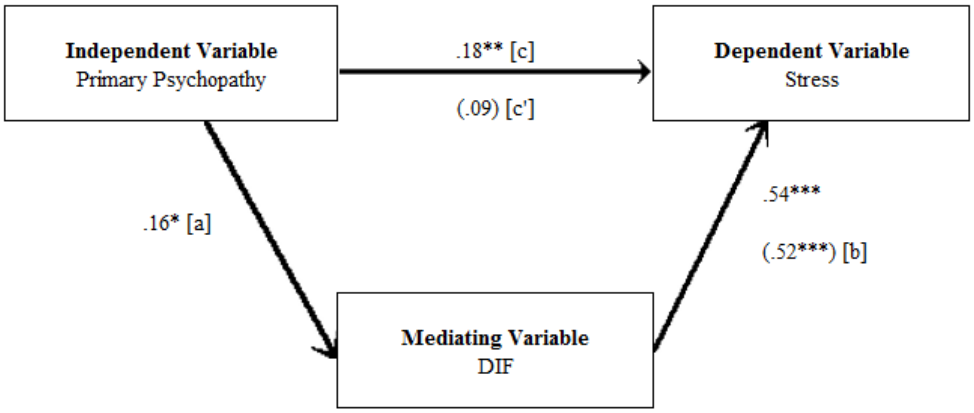

Figure 5.3 – Mediation role of DIF between primary psychopathy and perceived stress.
Note. The values in parentheses are beta weights and the other values correspond to correlations (Pearson).

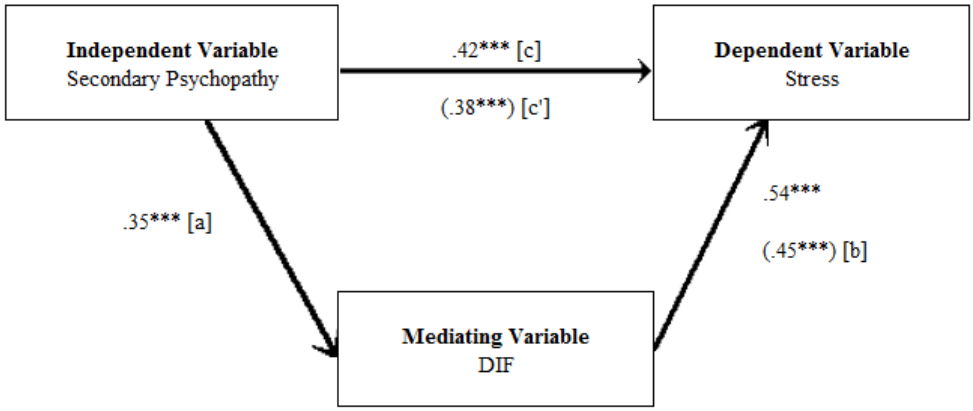

Figure 5.4 – Mediation role of DIF between secondary psychopathy and perceived stress.
Note. The values in parentheses are beta weights and the other values correspond to correlations (Pearson).

5.3.4 Structural model

Warren (2009) used a combination of mediation analyses and structural equation models to examine both direct and indirect associations between psychopathy measures with aggression in college students as mediated by specific deficiencies in empathy. Her rationale was based on the understanding that this technique would provide additional insight to separate mediation analyses since the effects of shared variances can be accounted for. Following this, Study 5 used structural equation model to investigate the individual impact of each psychopathic variant on stress, including direct and indirect links between primary and secondary psychopathy with stress via DIF and DDF dimensions of alexithymia. Firstly, homoscedastic requirements were inspected for the relationships between IV's

and DV's using scatter plots and the Bresuch–Pagan test (BP) in the *R* environment. Non–significant results are desired and indicative of the absence of problems regarding heteroskedasticity, which is an indication of constancy in errors' variance (Berry & Feldman, 1985). For this study, results for this test were satisfactory ($BP_{(4)} = 4.72$, $p = .31$). Secondly, curve estimations for all the relationships in the models were run to determine that they were sufficiently linear to be tested using a covariance–based structural equation model (Kline, 2015). Results for the linear curve estimation (LCE) in comparison to logarithmic, inverse, quadratic, cubic, power, logistic, compound, S, growth, and exponential curves are presented in Table 5.3. Harvey–Collier's Test also indicated linearity ($HC_{(258)} = 1.22$, $p = .22$). Thirdly, variable Inflation Factors (VIF's) for each IV were calculated using multivariate regression procedures aiming to detect potential problems with multicollinearity, and results for skewness and kurtosis were finally inspected to ensure data assumed normal distribution (Hair et al., 2010; Mardia, 1970; Moshagena & Erdfelderb, 2016).

Table 5.3: *Descriptive statistics for uni– and multivariate assumptions*

	PP	SP	DIF	DDF	ST
Mean	30.47	22.14	19.4	15.06	31.65
SD	7.20	4.57	4.64	4.26	6.80
Minimum	17	13	9	5	14
Maximum	54	34	29	25	50
Skewness	.38	.26	.18	–.13	.01
Kurtosis	–.35	–.68	–.77	–.48	–.24
VIF	1.41	1.67	1.44	1.54	–
LCE R^2	.03	17.9	30.1	14.5	–
LCE Z (*p*)	9.67 (.002)	57.13 (<.001)	112.65 (<.001)	44.50 (<.001)	–

Notes. DDF: Difficulties describing feelings; DIF: Difficulties identifying feelings; LCE: Linear curve estimation; PP: Primary psychopathy; SP: Secondary psychopathy; ST: Stress total; VIF: Variable inflation factor.

Results depicted in Table 5.3 supported the assumption of normality uni– and multivariate, which encouraged the testing of competing models (Hair et al., 2010). To account for the effects of both predictors and mediators on the independent variable, structural models were built and statistical procedures used the maximum likelihood estimation method using the following fit indices: Comparative Fit Index (CFI), Standardized Root Mean Residual (SRMR), and Root Mean Square Error of Approximation (RMSEA).

The first model tested included only DIF and DDF dimensions of alexithymia once these were the significant mediators of the relationship between stress to primary and secondary psychopathy variants. This model revealed very poor fit (χ^2 (1, N = 264) = 59.32, p < .001, CFI = .83, SRMR = 10.62, RMSEA = .47). Analyses of modification indices suggested that the path between primary psychopathy and DDF, albeit significant, was from a very small magnitude (R^2 = .05). Hence, a second model was tested with this path removed and constraining the errors of the mediators, which yielded very good fit indices (χ^2 (1, N = 264) = .69, p = .72, CFI = .99, SRMR = .004, RMSEA = .00) (Figure 5.5).

Corroborating the findings from the regression analyses presented within this chapter, the structural model kept only the predictive value of secondary psychopathy as significant in the relationships to the DV via the mediators. This model showed increased explained variance (36%) in comparison to the multiple regression model depicted previously (e.g., Section 5.4.2).

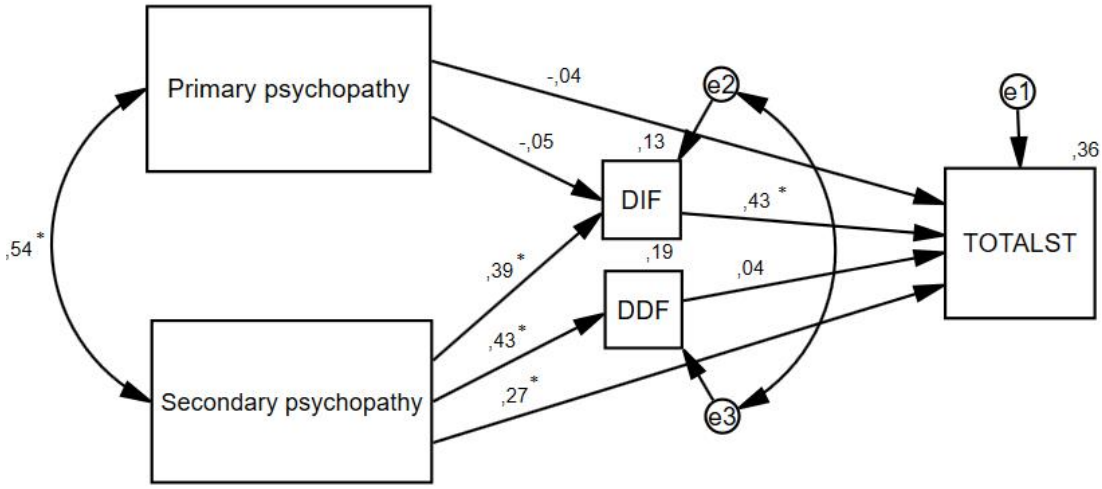

Figure 5.5 – Structural model of the relationship between psychopathy, alexithymia, and perceived stress.
Notes. Values marked with an asterisk are significant at p = .001. DDF: Difficulties describing feelings; DIF: Difficulties identifying feelings; TOTALST: Total scores on the Perceived Stress Scale.

5.4 Discussion

The purpose of this study was to test whether the associations between psychopathy and physiological stress responses, as detected in Study 4, would also be observable at the behavioural level. To reiterate, there were three specific

hypotheses for the current investigation. Firstly, that positive correlations between perceived stress and psychopathic personality traits would be detected. The second hypothesis proposed that the associations between stress and psychopathy traits would be stronger for the secondary variant of psychopathy compared to the primary type (Karpman, 1941; Lykken, 1957; Skeem et al., 2011). Both hypotheses were confirmed. In regards to differential associations between primary and secondary variants, results are aligned with the description of a specific variant of psychopathy in which maladjustment comes because of the combination of elevated emotionality with an overactive behavioural activation system, which could drive individuals into sensation seeking, antisocial behaviours (BAS; Fowles, 1993; Hicks, Markon, Patrick, Krueger, & Newman, 2004; Johnson et al., 2015; Newman, MacCoon, Vaughn, & Sadeh, 2005; Pham, 2012).

However, it could likewise be the case that these individuals also present with a very active behavioural inhibition system (BIS; Carver & White, 1994; Wallace, Malterer, & Newman, 2009). The combination of negative affect and trait anxiety resulted in an overactive BIS could then lead to hyper–vigilance, elevated stress, and threatening attentional bias (Gray, 1982; Watson, 1988). Interestingly, the links between overactive BIS to secondary variant of psychopathy were documented in the literature (Newman et al., 2005), as well as heightened BAS reactivity has been linked to this same variant of the psychopathic personality (Wallace et al., 2009).

The third hypothesis stated that secondary psychopathic personality traits would predict behavioural stress. Results depicted in Table 5.2 and in Figure 5.5 confirmed this prediction, and could be understood as imbalances in both BIS as BAS systems (Gray, 1982), as well as reflect the instable, impulsive and anxious phenotype seen in secondary psychopathy (Karpman, 1941; Lykken, 1957; Skeem et al., 2011). These findings have some similarities to those detected in a mixed sample of college students and workers in the U.S (Boozer et al., 2005). However, in this investigation, levels of self–reported stress were set as predictors of Machiavellianism and not the outcome of interest. Interestingly, stress was a significant, positive predictor of Machiavellian traits (β = .16) (Boozer et al., 2005).

A fourth hypothesis was that alexithymia dimensions would mediate the relationship between perceived stress and psychopathic personality traits. The

results partially supported this hypothesis. There were no mediation effects between EOT on the relationship between both types of psychopathy with stress. However, DIF and DDF were significant partial mediators in the models taken primary and secondary psychopathy as IV. Thus, 46% and 38% of the total effect of primary and secondary psychopathy on perceived stress go through difficulties describing feelings, respectively. Additionally, 44% and 38% of total effects of primary and secondary psychopathy on perceived stress go through difficulties identifying feelings, respectively. Regarding the EOT subscales, two further hypotheses can be raised. The first one is related to its psychometric properties. In this study, a relatively low alpha (.63) was calculated for this subscale. Thus, participants might have encountered difficulties in understanding the items comprised for this subscale (Iacobucci & Duhachek, 2003). Another interpretation could be that those participants have not consistently considered other's perspectives, which is commonly seen in both variants of psychopathy (i.e. primary and secondary; Lander et al., 2012). The partial mediation effect of both domains of alexithymia (identifying and describing feelings) on the relationships between stress and psychopathic variants are in line with emotional deficits seen in individuals high in psychopathic traits (especially empathic deficits; Takamatsu, & Takai, 2017), accounting for expressive explained variance.

The structural model explained more than a third in the variance of behavioural stress when taking into account psychopathic variants and the shared variance of DIF and DDF alexithymic dimensions. This model would suggest that secondary psychopathy has direct and indirect effects on self–reported perceived stress that were mediated by DIF. Although this percentage is moderately high, there remains 64% of the variance in the model not explained by the predictors, which encourages further research. Singh, Arteche, and Holder (2011) and Goerlich–Dobre, Bruce, Martens, Aleman, and Hooker (2014) have stressed that although much symptomatology (e.g., lack of insight, emotional and empathic deficits, and poor understanding of others) is shared between psychopaths and those with high levels of alexithymia, these conditions differ in several aspects. Importantly, primary and secondary psychopathy are believed to have distinct aetiologies, which could explain differential associations to behavioural stress, DIF

and DDF dimensions (Blonigen et al., 2005; Cairncross et al. 2013; Lander et al., 2012; Skeem et al., 2011).

Patrick et al. (2013) proposed a method in which experimental and multivariate cross–sectional studies provide feedback to each other in order to build a theory. As shown in Chapter Four, an enhanced CNS reactivity to laboratory induced stress was associated with higher levels of psychopathy personality traits. This study added that emotional deficits mediate the relationship between stress and psychopathic personality traits. Consequently, clinical work aiming to attenuate the negative consequences of both perceived stress and psychopathy could target these specific deficits in identifying and describing feelings, as well as promoting some level of emotional attachment training (Herpertz & Sass, 2000). Strategies might also be designed differently for those possessing with dominant traits in one or the other variant of psychopathy investigated in this chapter (Skeem et al., 2011; see also Gonzalez–Tapia et al., 2017).

Preventive research might particularly benefit from screening for these deficiencies during early stages of human development, which has shown to result in better outcomes when compared to strategies implemented later in life (Ribeiro da Silva et al., 2015). Efforts in reducing environmental stressors believed to elicit neuroendocrine responses should also be considered in any preventive attempt (Rebellon, Barnes, & Agnew, 2015; Sandi & Haller, 2015). In the same direction, previous research has shown that changes that occur in brain systems due to stress could be transferred into generations; consequently, stress stands out as a key regulator of well–being within societies (Sandi & Haller, 2015).

This study could also have implications to the legal and health systems once individuals high on perceived stress are more likely to perform violent offending (Silver et al., 2011). These individuals tend to show a stable bond with negative affect, which, in turn, can lead to physical suffering and somatic pain (Watson, 1988). Comorbidity with Axis I conditions occur in elevated percentages amongst those scoring high on psychopathic personality traits and amongst established psychopaths, mainly in terms of substance use disorders and somatoform disorder (Assadi, Noroozian, Pakravannejad, Yahyazadeh, Aghayan, Shariat, & Fazel, 2006) and mood and anxiety disorders (Fowles & Dindo, 2006). In this sense, an enhanced perception of stress amongst those with high levels of psychopathic

traits could interact with a proneness to seek medical services and individual care at schools, universities and organisations. It may also be interesting to follow the hypothesis that those seeking for specialised attention could do so with the intent of causing harm and performing fraudulent acts (Cleckley, 1941).

Several limitations to this study should be addressed. For instance, the use of self–report measures could have bias in terms of social desirability and impression management (Smeding, Dompnier, & Darnon, 2017). This could translate into less accuracy in responses in comparison to those obtained via structured interviews for psychopathy, collateral and interpersonal measures, in which at least two different points of view are contrasted in order to get a clearer assessment of the individual (Hare & Neumann, 2006).

Additionally, the data presented here was collected at one time point only, so potential casual relationships could not be explored any further. As in the procedures for examining mediation analyses used in a previous study of this thesis (e.g., Study 1; Chapter Three), the interpretation of findings in this respect should be considered in the light of the possibility that alternative models might occur in cross–sectional research. Psychopathic variants – along with the TAS–20 subscales and the PSS scale – might likewise be included as mediator variables (Judd & Sadler, 2008; Roe, 2012). Several methods have been proposed to support the decision of selecting the most appropriated model for these analyses, including reverse mediation (Lemmer & Gollwitzer, 2017), theoretical examination, temporality, and experimental control (Wiedermann & von Eye, 2015). One suggested method includes a series of examinations as to whether a predictor does exist before the values of the mediator(s), followed by an examination if the values of the mediator(s) would occur before the values of the dependent variable (Tate, 2015). Other supported methods test the indirect effect of the independent variable on the dependent variable via the mediator against zero (Hayes, 2013). In this respect, resampling methods – including the bias–corrected bootstrap method – have been considered more robust when compared to other methods, such as the Sobel's test (Lemmer & Gollwitzer, 2017; MacKinnon et al., 2004; Pieters, 2017).

As with previous chapters, an unequal proportion of male and female participants was obtained, which means that results could be potentially skewed in this sense. With these limitations in mind, further research about behavioural

processes associated with perceived stress could certainly obtain clearer insights by adopting more robust methods for data collection. Amongst these, the use of longitudinal procedures certainly is desirable once it facilitates the establishment of casual links between dependent and independent variables (Kraemer, Yesavage, Taylor, & Kupfer, 2000). The inclusion of a diversified range of participants (e.g., clinical, forensic) from various cultural backgrounds would again add value to the scientific study of behavioural stress and its links with antisocial behaviours and psychopathic personality traits.

Combined with psychophysiological data presented in Chapter Four, the results from the current study seem to indicate a group of individuals at special risk for emotional, behavioural, and health problems (van Dessel et al., 2014; Watson & Pennebaker, 1989). Although the independent sample investigated in the current study signalled that both primary and secondary traits are linked to more levels of self–reported stress, it is the latter which seems to be important in comprehending the stress–psychopathy route.

CHAPTER SIX – GENERAL DISCUSSION OF THE THESIS

Chapter overview

Much of the scientific knowledge on psychopathy is based on research with prototypical cases, mostly of white ethnicity and from North America (Casey et al., 2013; Wilson et al., 2014). It is believed that restricting the scientific study of psychopathy only to those selected cases has deleterious effects at many societal levels (Hauck–Filho et al., 2012; LeBreton et al., 2006). More broadly, the descriptions for antisocial behaviours presented within Section 1.1 indicate that various scientific realms conceptualise the construct of antisocial behaviour somewhat differently; however, there seems to exist a high correlation between clinical, forensic, and psychological measurements of these traits (Nuffield Council on Bioethics, 2002). As argued throughout this thesis, a more nuanced perspective on the validity of psychopathic personality traits in community samples is vital for including assessments of aggressive, antisocial, and psychopathic behaviours in the realm of public health (Wilson et al., 2014). The studies presented in this thesis sought to advance the knowledge in, at least, three ways: investigating behavioural, emotional, and physiological aspects of the psychopathic personality amongst adults recruited from the community.

Limitations of studies were presented and discussed within individual chapters. To avoid repetition, the current chapter aims to present a summary of findings, dedicating special focus to the implications and future directions of the empirical data, especially in terms of bullying/aggression research, biosocial criminology implications as well as regards to new directions for the scientific study of psychopathy. By taking into account both methodological and theoretical challenges implicated in the scientific study of aggressive, antisocial and psychopathic behaviours, this final chapter aims to guide a tentative agenda for future research. These reflections might also be relevant for other scholars with some degree of interest in the issues here covered, particularly the externalising spectrum and its biobehavioural concomitants (Kotov et al., 2017; Krueger & South, 2009).

6.1 Key findings: Psychopathic personality traits and bullying

Two important issues associated with research focusing on bullying usually include an exam of psychological processes (i.e., motives used to bully others) and concurrent correlates (i.e., personality characteristics and environmental factors; Rodkin et al., 2015). As discussed in Section 1.1 and throughout Chapter Three, experiencing both bullying perpetration and bullying victimisation can potentialize the negative consequences at emotional and behavioural levels. For example, Zych et al. (2016) indicated that bully–victims have important deficits in cognitive and affective components of empathy when compared to non–involved individuals, which taps into key areas also compromised in psychopathy. Subsequently, this thesis first explored the nature of the associations between psychopathy with aggression and victimisation seen throughout early developmental stages (e.g., childhood and adolescence), and discussed how these deficits are still playing an important role in perpetuating adult bullying. Although results presented in Chapter Three need further replication, an interesting finding was that the correlations between bullying and both primary and secondary psychopathy variants were bigger than the average previously reported in studies with children and adolescents (van Geel et al., 2016). Subsequent analyses showed that psychopathy (both primary and secondary) and gender (male) significantly predicted bullying perpetration. These same predictors also explained the combined involvement with bullying (i.e., the total score of the measure of bullying), albeit primary psychopathy showed higher betas. Another finding was that bullying victimisation played a significant mediation role on the relationship between psychopathic variants and bullying perpetrating. Continuing the consideration of victimisation, Study 2 revealed that most psychopathic characteristics, as measured by the PPI–R–40, were positively linked with this consequence of bullying, except Carefree Non–Planfulness, Fearlessness, Stress Immunity and Social Influence. The behaviour of bullying others was positively correlated with Blame Externalisation, Machiavellian Egocentricity, Rebellious Nonconformity, Self–Centred Impulsivity factor, Social Influence, and total psychopathy (r_{range} = .08 – .38). As such, it is clearly important to consider the role of psychopathic personality traits in programmes aiming to deter the consequences of bullying and vice versa. Given the relatively few studies that

examined directly the relationship between adult bullying and psychopathy traits and variants, it is also important to continue researching these two problematic behaviours.

6.2 Key findings: Psychophysiological profile

As reported in Chapter Two, specific abnormalities in brain regions have been identified in both structure and function, which could explain psychopath's vulnerability for involvement with violence and broad criminality (Blair, 2005; Raine, 2002; Reidy, Krusemark, Kosson, Kearns, Smith–Darden, & Kiehl, 2017). Nonetheless, even though research into biological mechanisms of the psychopathic taxon are relatively abundant (cf. Chapter Two), there has been little attention to physiological mechanisms underpinning putative sub–domains of the psychopathic personality. As such, it is likely to be useful to further dissect the psychopathic taxon into factors, variants, or subtypes (Brazil et al., 2016; Kimonis et al., 2017; Skeem et al., 2011; Vassileva et al., 2005). In attention to this, Study 3 investigated which facets of the psychopathic personality were statistically associated with RHR. Results from this study indicated that Social Influence, Rebellious Nonconformity, and Stress Immunity were correlated with low RHR. Additionally, PPI–R–40 total score and Factor 1 (Fearlessness) were negatively linked to RHR, a measure previously connected to the fearlessness dimension of psychopathy (Remmel & Glenn, 2015). When corrections due to multiple comparisons were undertaken, Rebellious Nonconformity, Social Influence and the PPI–R–40 total score kept statistical significance. Especially regarding RHR, two models have received much emphasis in the realm of psychopathy: fearlessness and sensation seeking theories (Lykken, 1957; Raine et al., 2014; Zuckerman & Need, 1979; see also Raine, 2002). However, both propositions warrant further empirical validation (Raine et al., 2014). The correlational data presented in study 3 adds evidence to the fearlessness theory of the psychopathic personality as the Social Influence subscale loads into the PPI–R–40 Factor 1 (Fearlessness Dominance; Eisenbarth et al., 2015). Notably, before applying corrections for multiple comparisons, the Fearlessness Dominance factor showed negative, but small, associations with RHR.

Without doubt, the links between cardiovascular functioning at rest with psychopathy (e.g., Gao et al., 2012) and AB (e.g., Armstrong et al., 2009; Crozier et al., 2008; Jennings et al., 2013; Sijtsema et al., 2010) seem to be important. One next question that certainly stands out regards the origin of these differences. As some scholars defend, low RHR must be a highly inherited feature (95% IC = .65 to .82; Boosma & Plomin, 1986), which would explain the existence of significant associations between this measure and antisocial features at early age (e.g., 3 years–old), coupled with the robust predictive validity of low RHR in explaining negative, violent, and criminal outcomes at latter developmental stages (Jennings et al., 2013; Ortiz & Raine, 2004). In the case of the debate as whether LRHR should be considered a biomarker for psychopathic traits, the use of multi–method investigations can help in the clarification of remaining issues. For instance, criticism to the acceptance of LRHR as an inherited feature rests support in the fact that not every report has been able to replicate the finding (Casey et al., 2013; Loeber, 2004; Portnoy & Farrington, 2015). Regardless of these discrepancies in the literature on biological mechanisms underpinning psychopathic and antisocial behaviours, investigations are important to offer robust information for professionals, with an ultimate goal of assisting better approaches in prevention and treatment (Vaughn et al., 2014).

Studies linking stress reactions with personality in humans have focused primarily on its associations with aggression and anxiety, examining specifically the final product of stress response (cortisol) (Hamilton et al., 2008; Lopez–Duran et al., 2009; Mulder, 2011; McLaughlin et al., 2014). Moreover, much of the data concerning stress reactivity and AB come from animal research (Sandi & Haller, 2015; Walker et al., 2016). Efforts in detecting abnormalities in stress–responsive systems are needed in order to design interventions (Blair, 2010b). It is believed that investigations using laboratory induced stress – in which specificities in autonomic and neuroendocrine processes are examined – could inform about broader deficits that may explain participant's behaviour in the outer world (Crum et al., 2017). As such, Study 4 provided a continuation of previous work that used physiological measures to investigate stress response in face of antisocial features,

including bullying and psychopathic tendencies amongst typical developing adults (Crozier et al., 2008; Hamilton et al., 2008; Newman, 2014).

Given that previous research has shown that Factor 1 is meaningful in predicting blunted stress reactivity (i.e., cortisol levels) amongst those with clinical levels of psychopathy (Johnson et al., 2015), Study 4 tested whether this finding could be replicated using sophisticated procedures, such as the online measurement of the HPA and SAM systems in response to challenge and threat appraisals in typical developing adults. Challenge and threat are evoked states of approach and avoidance in face of cognitive, social, or psychological stressful situations (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996; Seery, 2011). Specifically, the second investigation reported in Chapter Four used the BPSM (Blascovich & Mendes, 2000; Blascovich & Tomaka, 1996) to make unique predictions about underlying physiological processes in individuals high on psychopathic personality traits and with distinct degrees of participation with bullying. The intent of considering both bullying and psychopathic phenomena seemed important as they can originate from, or can be highly influenced from specific biological pathways (Patrick & Hajcak, 2016; Ribeiro da Silva et al., 2015); coupled to the fact that no previous work has so far tested the biological mechanisms underpinning both phenomena together in adults.

Initially, results showed that high levels of psychopathic personality traits, as well as high levels of self–reported aggression and bullying, appeared to be associated with threatening physiological responses, in which a relatively higher CO and a lower TPR reactivity indicated to a greater challenge or a lesser threat (Blascovich & Mendes, 2000). Further analyses of the effects of multiple comparisons supported the association only for psychopathic traits of Machiavellian Egocentricity and for the total score of psychopathy. Regression models also showed that these two variables predicted physiological threat reactivity (β = –.29, p = .013, R^2_{Adj} = .07 for total psychopathy, and β = –.31, p = .01, R^2_{Adj} = .08 for Machiavellian Egocentricity). These findings, when combined, suggested to hyper–reactivity to stress in adults recruited from the community and were fairly consistent with expectations that an enhanced HPA activity can be associated with aggression, bullying, and negative affect (Chida & Hamer, 2008;

Hamilton et al., 2008; Walker et al., 2016). Results from the fourth study indicate that high levels of psychopathic traits might enhance an approach to goal–oriented situations characterised by a threatening, hostile predisposition (Kendler et al., 2012; Loeber & Hay, 1997). Threatening appraisals denote dysregulated emotional responsivity (Fairchild, van Goozen, Stollery, & Goodyer, 2008; Pincham et al., 2015) and – as shown in Chapter Four – could result in maladaptive reactivity patterns. Importantly, these results not only corroborate and build upon previous work within the broad area of AB and aggression, but also indicate the direction of the associations between threatening approach with elevated levels of Machiavellian Egocentricity and total self–reported psychopathy. Although this finding was believed to exist, it has not been empirically reported previously (see Blair, 2010b).

Importantly, both experimental studies used a newly developed approach to measure psychopathic tendencies for community samples (e.g., the PPI–R–40; Eisenbarth et al., 2015). For this reason, results from studies 3 and 4, particularly in respect to the associations between physiological data with psychopathic personality traits, must be considered with caution. Benning et al. (2003) – when analysing the factor structure of the PPI – noted that items from the PCL–R Factor 1 (Hare, 2003) such as "grandiose sense of self–worth" and "failure to accept responsibility" should resemble items from the PPI assessing Machiavellian Egocentricity and Blame Externalisation. In psychometric terms, however, these traits did not load into PPI–R–40 Factor 1 (Fearless Dominance; Eisenbarth et al., 2015). Therefore, there is a clear necessity of further examination of whether items from this recently developed measure are perhaps tapping into characteristics from both psychopathy factors. Indeed, studies examining particularly the validity of shorter versions of psychopathy measures, such as the PPI–R–40, are needed for further clarifications about competing factor structures. Moreover, the possibility that sample characteristics might differentially affect the psychometric properties of psychopathic measures, including the PPI and the PPI–R, has been proposed recently (Ruchensky et al., 2017). Regardless of these limitations, important considerations were possible in relation to theories that attempt to explain biological mechanisms underpinning psychopathic, antisocial behaviours. In summary, while Study 3 has provided further support to the

fearlessness proposition of psychopathy, Study 4 added empirical evidence to the response modulation hypothesis for psychopathy and, more directly, to the structural and functional architecture of the threat circuitry amongst individuals with elevated psychopathic traits (Blair et al., 2005; Blair, 2010b).

6.3 Key findings: Psychopathic personality and emotional correlates

When taken in combination, physiological findings presented in Chapter Four suggested that those with higher levels of psychopathic traits displayed low arousal (Study 3) – in which higher levels of psychopathic personality traits were related to low RHR – and increased haemodynamic reactivity to laboratory–induced stress (Study 4). The final empirical study included in this thesis sought to further examine the role of psychopathic personality traits in the understanding of stress at the behavioural level, examining, specifically, the role of emotional deficiencies (Chapter Five). Findings of this study not only were aligned to physiological data presented in Chapter Four, but were also convergent to evolutionary theories which suggested that unemotionally seen in secondary psychopathy might be a result of consecutive social adversities, which serves the functions of survival and allostasis (e.g., maintenance of dysregulated behaviours) (Ellis, Del Giudice, & Shirtcliff, 2013; Gobin, Reddy, Zlotnick, & Johnson, 2015; Ribeiro da Silva et al., 2015).

Perhaps one of the most interesting findings presented in Chapter Five was the mediating role of the difficulties in identifying and in describing feelings on the relationship between stress and psychopathic variants, which accounted for significant variance (e.g., moderate levels). In the context of the current thesis, this last study contributed in the understanding of behavioural, emotional and physiological correlates of the psychopathic personality in typical developing adults. Also, this study proposed some avenues by which professionals could act in order to prevent broad problematic behaviours linked to stress responses in humans (Assadi et al., 2006; Silver et al., 2011).

6.4 Studying groups at risk for emotional and behavioural difficulties: Insights into psychopathy–bullying research

This thesis works from the understanding of psychopathy as a heterogenic and multidimensional disorder; in other words, there exists substantial variance in the behavioural, physiological and emotional symptoms displayed by those high on psychopathic personality traits (Brazil et al., 2016; Hauck–Filho et al., 2012; Skeem et al., 2011; Thompson et al., 2014). For decades, researchers believed that the key element in conceptualising psychopathy was confined to the affective, and not the antisocial, element (Patrick et al., 2012; Skeem et al., 2011). More systematic approaches combining cumulative evidence have begun to reshape this diagnostic assertion for psychopathy, in which aggressive behaviour and antisociality do matter (Neumann et al., 2015). As such, this form of personality disorder is marked not only by absence of empathy and fear, being also linked with elevated involvement with crime and aggression (Lilienfeld & Andrews, 1996; Miller, Hyatt, Maples–Keller, Carter, & Lynam, 2016).

Recently, Lilienfeld (2018) listed a few recommendations for studies focusing on developmental factors of AB and psychopathy, including the necessity of linking both psychopathy with the personality literature, the importance of examining the unique variance of findings from psychopathy measures in comparison to other disorders, and to avoid reliance on total psychopathy scores. By building connections with well–supported models of AB based on learning theory, such as coercive model mentioned within chapter one (Section 1.1) (Patterson et al., 1989), researchers might provide answers to unresolved issues of why children and adolescents persist in disruptive behaviours into later developmental stages regardless of the consequences of their acts (Lilienfeld, 2018; Moffitt & Caspi, 2005).

One other important type of aggressive, antisocial behaviour is bullying. Causing great physical, emotional and/or financial hardship to its victims, it refers to a systematic and unfair relationship between two or more people where a disparity of power is displayed (Menesini & Salmivalli, 2017; Pepler, Jiang, Craig, & Connolly, 2008). Bullies often show a lack of concern and a deficiency of empathy toward their victims (although not always), displaying impulsive behaviour, need

for domination, and selfishness (Gini et al., 2007; Holl, Kirsch, Rohlf, Krahé, & Elsner, 2017; Menesini et al., 2003; van Hazebroek, Olthof, & Goossens, 2017; Warden & Mackinnon, 2003). Victims of this process, in turn, are more likely to present depressive symptoms, low self–esteem and poorer academic outcomes (Rigby, 1999, 2007), poorer mental and physical health, suicidal ideation and suicide attempts (Espelage & Holt, 2001; Nansel et al., 2001; Sigurdson, Wallander, & Sund, 2014; Wolke & Lereya, 2015), and delinquent behaviour (Barker et al., 2008). Finally, bully–victims present themselves with difficulties common to both bullies and victims, which place this group at greater risk for emotional and behavioural difficulties (Arseneault et al., 2008; Barker et al., 2008; Nansel et al., 2001; Singham et al., 2017). Farmer, Petrin, Robertson, Fraser, Hall, Day, and Dadisman (2010) presented a framework of "two social worlds" involved in bullying dynamics. Bullies that are socially integrated might take advantage of AB with the expectation of controlling others, while bullies that are socially marginalised could use AB in response to environmental factors (Farmer et al., 2010). These assertions, albeit deserving further empirical confirmation, can be of use for professional working directly with individuals affected/involved with bullying behaviours. Indeed, just as for psychopathy, bullying is a worldwide problem, causing concern for parents, schools, and students (Lutgen et al., 2007). Explicitly amongst studies conducted with youth, reports indicate very high prevalence rates of up to almost 90% for experience of being a perpetrator of bullying and up to 98% as a victim (Modecki, Minchin, Harbaugh, Guerra, & Runions, 2014). Scholars have detected many risk and protective factors for being involved with bullying, being some of them contradictory (e.g., deficits in social skills) (Berger & Caravita, 2016; Guy, Lee, & Wolke, 2017; Holl et al., 2017; Ttofi, Farrington, Piquero, Lösel, DeLisi, & Murray, 2016; Zych et al., 2017). However, the precise role played by psychopathic personality traits, as well as the explained variance attributed to these traits in bullying is yet to be fully realised. The prevalence of both bullying and psychopathic traits may vary across studies due to numerous reasons, such as sociocultural and methodological aspects. Hence, what could be interpreted as a non–aggressive behaviour in one culture might well be considered bullying in another, which is equally applicable to psychopathy (Cooke, 1998; Hubbard et al., 2002). Here, notable avenues for further research might

benefit from using cross–cultural methods in order to conceive a more robust picture into determinants of these problematic behaviours (Sullivan & Kosson, 2006). Additionally, procedures for assessing both processes vary immensely in terms of estimation methods and frequency covered (e.g., over the past month, over the past year). Hence, research in the future should also take these factors into account, with attention to the need for specific tools for assessment, such as those possessing very good psychometric properties and appropriate cultural sensitivity.

Even though there are many similar characteristics between what is defined as 'bullying' behaviours and psychopathic personality (e.g., lack of empathy, shallow affect and callousness), some conceptual points can help to distinguish between common and specific variances to each phenomenon. As shown in the literature sections of this thesis, both bullies and psychopaths aim for power and often target a fragile victim or group of victims to obtain it. In psychopathy, the perpetration of negative acts occurs quite often as a matter of 'business' (e.g., without emotionally–driven motives; Hare, 2001). In bullying, however, it is often the case that retaliation is the force underneath reactive aggression, at least amongst children and adolescents (Dodge, 1991; Losey, 2011; Ragatz et al., 2011). Frequently, the course of bullying dynamics shows a tendency to extinction during late adolescence, while this appears to not decrease substantially for psychopathic individuals (Baskin–Sommers, 2017; Camodeca, Goossens, Terwogt, & Schuengel, 2002; Hare, 2003; Menesini & Salmivalli, 2017). Given this, adult bullying, a much less explored context of bullying, may occur more frequently as a function of elevated levels of psychopathic traits. Future investigations might provide specific insights into how the use of proactive and reactive forms of aggression is stable amongst those involved with bullying behaviours in combination with higher levels of psychopathic personality traits. These findings would certainly be of high value for those who work with education and workplace bullying (e.g., teachers, school nurses and human resources staff) (Giovazolias & Malikiosi–Loizos, 2015). It is also the case that targets of bullying are sometimes very close to the aggressors (e.g., a classmate or one–time friend) (Mishna, Wiener, & Pepler, 2008). The extent in which these affective bonds are also present in the psychopath–

target relationship is yet to be realised. Deeper studies into these considerations are certainly valuable for expanding the knowledge about both processes, being equally important for clinical and educational settings. Ribeiro da Silva et al. (2015), when exploring past research into the evolutionary basis for psychopathy and antisocial behaviours, proposed that interventions should focus on systems believed to be highly affected in psychopaths and those high in psychopathic personality traits, such as the threat (e.g., reactivity to stress) and the soothing systems. Clearly, future work evaluating the outcomes of such interventions could add unique value to the field.

In addition, there is a clear need for more precise information in respect to genetic influences in both bullying and psychopathy. DeLisi et al. (2011) noted that genetic influences in externalising disorders are associated with a variety of phenotypes, rather than there being a specific and identifiable gene or combination of genes underpinning a specific behaviour. It is not, therefore, unreasonable to suggest different genetic influences underlying the different subgroups of antisocial individuals (Blonigen et al., 2005; Sadeh et al., 2010; Waldman & Rhee, 2006; Viding et al., 2005). For instance, behavioural genetic investigations have shown that inherited influences for behaviours of bullying others and conduct problems were 61% and 73%, respectively (Pingault et al., 2015; Vlachou et al., 2011). In this sense, studies that address specifically the overlap of bullying behaviours and psychopathic traits can provide a more detailed account on these influences.

6.5 Implications within the scientific study of psychopathy and biosocial criminology

Currently, biosocial issues underlying antisociality, including its predictors and determinants, have received much attention in the research agenda. Amongst these predictors, psychopathic traits often play an important role (Kiehl & Hoffman, 2011). Based on the material presented in Chapter One, it is compelling to infer that much of the current view on psychopathy is derived from Cleckley's (1941) and Hare's (2013) extensive work, which defined the construct as a

condition essentially characterised by poor affect, emotional detachment, self–seeking behaviour and use of aggression (Frick & Viding, 2009; Glenn & Raine, 2014; Lynam & Gudonis, 2005; Newman, 1998; Patrick, 2001; Pemment, 2013). Nonetheless, no consensus has been achieved thus far in respect to the precise symptomology for psychopathy, and no professional body has published delimited criteria for it (Skeem et al., 2011).

Biosocial criminology is a scientific area which incorporates knowledge from realms others than sociology and criminology to better capture social problems, such as psychopathy and antisocial behaviours (Rebellon et al., 2015). On one hand, there is an emerging understanding that environmental factors interact with the central and peripheral nervous systems, as well as with the endocrine system, in order to influence behaviours (Rebellon et al., 2015). On the other hand, there is also an understanding that biological and environmental influences do not provide a unique, final answer. As such, the influences of one dimension over another may put an individual at risk for develop psychopathic tendencies (O' Farrell, 2016; Salekin, 2006).

According to the biosocial criminology perspective, no biological mechanism on its own can explain antisociality. For instance, meta–analytic studies suggest that additive genetic influences could account for up to 49% of the variance of psychopathic traits, whereas non–shared environmental influences are assumed to account for the remaining 51% (e.g.; environmental factors, for instance, can account cases of children within the same family receiving varied parenting, belonging to different classes at school and having different friends; Rhee & Waldman, 2002; Waldman & Rhee, 2006). As shown in past research, individuals possessing solely with biological or social risk factors exclusively have presented with low involvement with AB; nonetheless, the concomitant presence of both social and biological factors resulted in elevated involvement with AB (Popma et al., 2007). Clearly, a better comprehension of psychopathy might come by the systematic study of various casual mechanisms (DeLisi & Piquero, 2011; Glenn & Raine, 2013). Thus, the necessity of more research is evident, whether biological–oriented or not, to enrich the actual understanding of the psychopathic personality in different groups and contexts. There has been a rapid expansion in

the use of psychophysiological methods intending to clarify aetiological mechanisms for a variety of psychological events. Importantly, physiological responses can provide an index of a given psychological phenomena, but not a direct representation of affect and emotions (Richter & Slade, 2017). This is consistent with Cacioppo and Tassinary's (1990) model, in which the occurrence of a given psychological event might lead to changes in physiological responses, although the reverse not always holds true. Particularly regarding cardiovascular responses, this thesis provided further evidence in relation of a negative association between levels of psychopathic traits and RHR. Here, it would be certainly interesting to replicate the findings using diversified groups (cf. Anderson & Maxwell, 2016). For instance, data suggested significant associations between the PPI–R–40 total score and its subscales of Rebellious Nonconformity and Social Influence with RHR, likewise indicating that these variables were significant in predicting low arousal. The remaining question is whether this would likewise be true when forensic samples of adults are investigated, and these findings should certainly be explored in both clinical and community samples of children and adolescents. Naturally, further replication is also necessary within the context of adults recruited from the community, which was the case of the current thesis. In addition, the question of whether differential patterns of physiological responding exist in face of emotions within an experiment in comparison to the occurrence in everyday life demands for more applied research (Janig, 2003; Stemmler, 2003). Future studies are also needed in order to further understand the link between relatively higher arousal for those traits typically labelled under the 'secondary' variant. In short, the extent in which low RHR is a robust risk factor across different operationalisations of psychopathy warrants further examination (Smith & Lilienfeld, 2015).

Moreover, there still is necessity in knowing how cardiac functions are linked to the brain and – to some extent – are linked to specific regions of the brain that are also highly affected in the context of psychopathic personality traits (Remmel & Glenn, 2015)[24]. It would be useful to measure concomitantly the

[24] Although the reciprocal actions and reactions of two most important organs of the body (brain and heart) have been proposed many years ago (Darwin, 1872/1965), much more is to yet to be realised, especially regarding how various systems interact in order to influence behaviour and

cardiovascular autonomic function and brain activity in future attempts in getting clearer insights (DeWall & Chester, 2015)[25]. In addition, in the face of increase interest of multi–methods for data analysis (Blascovich et al., 2011; Cacioppo et al., 2000), additional mechanisms of the ANS and other biological systems could be entered together into more complex psychophysiological models. It is often the case that researchers do possess these data, which would only require further statistical processing and modelling specification.

Within biosocial criminology, reactivity to stress is understood as an important marker of fear conditioning, and informs on individuals' social feedback dysregulation (Johnson et al., 2015). With effect, the study of physiological and behavioural indicators of stress could offer precise insights into aetiological mechanisms and on the course of psychopathic traits, which could be then transferred into more accurate procedures for psychological prevention and broader mental health strategies (Johnson et al., 2015; Rebellon et al., 2015), including potential use of pharmacological therapies (Lee, 2015). Additionally, Blair (2010b) noted that the threat system in psychopathy remains largely unexplored amongst humans. The BPSM framework have validated cardiovascular responses to stress as biological markers of challenge and threat using robust, replicable procedures (Allen et al., 2012; Frings et al., 2012). Therefore, future work could try to corroborate the findings here presented using samples with early onset of psychopathic traits (i.e., children with CU traits) as well as amongst

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emotions. Cannon, back in 1927, criticised the causal view of biological components in determining emotion; instead, his view was one that understood the action of the ANS as a concomitant of emotional experiences. Evidence for this was apparent from studies conducted by Schachter and Singer (1962), who showed that a same physiological element (e.g., adrenaline) could result in the experience of varied emotions. In this sense, situational cues (such as cognitive priming) were believed to exert an important role in determining emotional responses. Subsequent developments into psychophysiological investigations, as evidenced in Chapter Two, were capable of providing nuanced approaches to various biological systems and, consequently, were important to clarify the role of biology in influencing behaviours.

[25] Gianaros, Van der Veen, and Jennings (2004) hypothesised that the brain systems responsible for coordination of cardiac autonomic activity are the medial–prefrontal, anterior cingulate, and insular regions of the cortex. In the subcortical regions, the authors cited the amygdala, cerebellum, hypothalamus, solitary tract nucleus, nucleus ambiguous, dorsal motor nucleus, and rostral ventrolateral medulla. Some other physiological mechanisms that could largely account for the relation between LRHR and AB include increased vagal tone, decreased noradrenergic tone, and reduced right–hemisphere functioning (Crozier et al., 2008; Raine, 2002). Data has so far indicated limited support for the hypotheses around decreased noradrenergic tone and reduced right–hemispheric functioning, yet in respect to the vagal tone proposition the evidence is relatively robust (Mezzacappa et al., 1997; Raine, 2002).

those with confirmed diagnosis of psychopathy. Again, follow–up, genetically–informed, and longitudinal studies exploring these physiological mechanisms could help in better understanding biopsychosocial pathways that influence in the manifestation of antisocial behaviours and psychopathology across lifespan (Colins et al., 2018; Curtis, 2016; Hostinar & Gunnar, 2013; Meyer & Hamel, 2014).

Other important issue that deserves attention is in respect to the intense debate in the scientific study of psychopathy as whether the unitarian taxon, as coined by Cleckley (1941) and largely expanded by Hare (2003), is suitable for the operationalisation of psychopathic features in the general population. As Skeem et al. (2011, p. 115) affirms "if in theory and research psychopathy is a diversely defined disorder", which implies distinct perspectives aetiology, in "most clinical and legal contexts psychopathy is instead construed and assessed as if it were a single thing". These discrepancies are certainly important and might implicate distinct policy implications. However, as shown in Chapters Two and Four, studies have indicated that subcategories of adults do not differentiate themselves on psychophysiological tonic activity and reactivity based on psychopathy total scores; instead, significant group differences can be obtained by the absence (type I or primary psychopathy) or presence of internalising psychopathologies (type II or secondary; Serafim et al., 2009; Yildirim & Derksen, 2013). Following this, there seems to exist a clear necessity of better understanding the patterns of abnormal ANS functioning by including in today's research agenda the so–called 'spectrum approach' (as opposed to the categorical approach). One way of doing so is by exploring the phenomena as it manifests itself in various samples across the world (Hoppenbrouwers et al., 2016; Pasion et al., 2017). When a clear understanding of a myriad of correlates of psychopathic, aggressive, and antisocial behaviours is achieved, perhaps a more effective response from society will be possible, in which prevention and intervention programmes will be marked by greater success.

6.6 Concluding remarks

As a psychologist with previous experiences with themes such as school violence and bullying, I opted for conducting my doctoral training by exploring the

underlying mechanisms and possible convergence between both bullying and psychopathic behaviours. One question that has always been intriguing to me refers to the reasons why some individuals are more prone to bully others and present with psychopathic tendencies, while others – albeit sharing the same environment – behave differently? Following this, why would some respond better to psychological and educational interventions, while others are irresponsive, defying, and callous? What are the answers that could possibly respond to these questions and, more importantly, how they can be translated into school and community policies? Certainly, I am amongst many more who share these very same questions, regardless of whether within the academic context or not. Persuasively, Sutton et al. (1999a) have proposed that more research was needed to disentangle normal bullies from more 'hard–core' ones. This last category, for instance, has shown to be resistant to a variety of efforts of behavioural correction. Equally important was the finding of higher performance in Theory of Mind tasks, particularly related to social cognition, amongst this subgroup of perpetrators of bullying behaviours (Sutton et al., 1999a, 1999b). Building up on these previous work, the main findings of this thesis provide support for this theorisation that bullying behaviours are linked to psychopathic tendencies amongst typical developing adults. Moreover, the present thesis suggests that investigations into correlates of antisocial and aggressive behaviours might consider specific difficulties presented by bully–victims. This thesis also consolidates the validity of measurements of biological low arousal in respect to psychopathic tendencies, specifically those of Rebellious Nonconformity, Total Psychopathy, and Social Influence. Moreover, experimental data suggested that features denoting lack of emotional attachment and lack of guilt may indicate augmented risk for poor cardiovascular efficiency, higher negative affect, and poor cognitive capacity (Crum et al., 2017; Kassam et al., 2009). Finally, the present thesis suggests that emotional deficits regarding difficulties in identifying feelings can mediate the association between behavioural stress and psychopathic personality traits.

For years, research into psychopathy focused in severe, prototypical cases. However, recent studies have emphasised to the importance of studying the phenomena as it is presented below clinically significant levels (Gao & Raine,

2010). Modern approaches to the psychopathic personality have considered it as a heterogenic disorder, caused by multiple factors, and still very distant from having clear–cut determinants (Brazil et al., 2016; DeLisi & Piquero, 2011; Skeem et al., 2011). The current thesis argues that the concomitant occurrence of high psychopathy, genetic and biological vulnerabilities and experiences of bullying, aggression and victimisation could delineate a subgroup of individuals at serious risk for reoffending, crime involvement, and psychopathy (Barker et al., 2008; De Brito et al., 2011; Fontaine et al., 2010; Koeppel et al., 2015; Marsh et al., 2013; Raine et al., 1997a, 1997b; Rebellon et al., 2015; Viding et al., 2012). As such, some bullies present a set of purposefully cruel actions, absence of emotional empathy responsiveness towards their victims, which have clear similarities to behaviours typically labelled as 'psychopathic' (Frick et al., 2003; Juvonen & Graham, 2014; Kimonis, Skeem, Cauffman, & Dmitrieva, 2011). The results presented in this thesis can be incorporated into policies aiming to prevent and treat psychopathic behaviours. For instance, the correlates presented in this thesis were differentially meaningful for two variants of psychopathy: primary (or Factor 1; marked by emotional detachment and less anxious) and secondary (or Factor 2; a variant with more pronounced antisociality and internalising problems). This last category, for example, may benefit in particular of early treatment, such as those promoting compassion (e.g.; Compassion–Focused Therapy; Ribeiro da Silva et al., 2015) and intensive parental intervention (Haas, Waschbusch, Pelham, King, Andrade, & Carrey, 2011; Frick, Kimonis, Dandreaux, & Farrell, 2003). In terms of theory and research, this thesis builds upon previous work showing that the presence of high levels of psychopathic personality traits designates a subgroup of antisocial individuals at greater risk for serious emotional and behavioural difficulties (Barnes, 2014; Koeppel et al., 2015; Kotov et al., 2017; Wendt & Bartoli, 2018).

Appendices

Appendix A – Illinois Bullying Scale

		Never	1 or 2 Times	3 or 4 Times	5 or 6 Times	7 or more Times
1.	I upset other students for the fun of it.	a	b	c	d	e
2.	In a group I teased other students.	a	b	c	d	e
3.	I fought students I could easily beat.	a	b	c	d	e
4.	Other students picked on me.	a	b	c	d	e
5.	Other students made fun of me.	a	b	c	d	e
6.	Other students called me names.	a	b	c	d	e
7.	I got hit and pushed by other students.	a	b	c	d	e
8.	I helped harass other students.	a	b	c	d	e
9.	I teased other students.	a	b	c	d	e
10.	I got in a physical fight.	a	b	c	d	e
11.	I threatened to hurt or hit another student.	a	b	c	d	e
12.	I got into a physical fight because I was angry.	a	b	c	d	e
13.	I hit back when someone hit me first.	a	b	c	d	e
14.	I was mean to someone when I was angry.	a	b	c	d	e
15.	I spread rumors about other students.	a	b	c	d	e
16.	I started (instigated) arguments or conflicts.	a	b	c	d	e
17.	I encouraged people to fight.	a	b	c	d	e
18.	I excluded other students from my clique of friends.	a	b	c	d	e

Appendix B – Levenson Primary and Secondary Psychopathy scales

LSRP

The purpose of this questionnaire is to look at the different thoughts and behaviours of individuals. In order to do this, I would like you to read each statement, and rate your answer on a scale of 1 to 4. 1= disagree strongly and 4 = agree strongly. Please tick one of the following four columns, indicating your answer.

	1 Disagree Strongly	**2 Disagree Somewhat**	**3 Agree Somewhat**	**4 Agree Strongly**
Love is overrated				
I am often bored.				
Success is based on survival of the fittest: I am not concerned about losers.				
I often admire a really clever scam.				
I find that I am able to pursue one goal for a long time.				
For me, what's right is whatever I can get away with.				
I tell other people what they want to hear so that they will do what I want them to do.				
I don't plan anything very far in advance.				
My main purpose in life is getting as many goodies as I can.				
I enjoy manipulating other people's feelings.				
When I get frustrated, I often "let off steam" by blowing my top.				
In today's world, I feel justified in doing anything I can get away with to succeed.				
Cheating is not justified because it is unfair to others.				
I have been in a lot of shouting matches with other people.				
I let others worry about higher values; my main concern is with the bottom line.				
Making a lot of money is my most important goal.				
I make a point of trying not to hurt others in pursuit of my goals.				
I quickly lose interest in tasks I start.				

Looking out for myself is my top priority.				
I would be upset if I my success came at someone else's expense.				
I feel bad if my words or actions cause someone else to feel emotional pain.				
Most of my problems are due to the fact that other people just don't understand me.				
Even if I were trying very hard to sell something, I wouldn't lie about it.				
Before I do anything; I carefully consider the possible consequences.				
People who are stupid enough to get ripped off usually deserve it.				
I find myself in the same kinds of trouble, time after time.				

Appendix C – Psychopathy Personality Inventory – Genetic Derived form

This measure has not been included in the thesis in attention to copyright.

The selection of the twenty–item Toronto Alexithymia Scale is available from the following publication:

Bagby, R. M., Parker, J. D., & Taylor, G. J. (1994). The twenty–item Toronto Alexithymia Scale—I. Item selection and cross–validation of the factor structure. *Journal of Psychosomatic Research, 38*(1), 23–32. doi: 10.1016/0022–3999(94)90005–1

Appendix E – Perceived Stress Scale

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate by circling how often you felt or thought a certain way

	Never (1)	Almost Never (2)	Sometimes (3)	Fairly Often (4)	Very Often (5)
In the last month, how often have you been upset because of something that happened unexpectedly? (1)	❍	❍	❍	❍	❍
In the last month, how often have you felt that you were unable to control the important things in your life? (2)	❍	❍	❍	❍	❍
In the last month, how often have you felt nervous and "stressed"? (3)	❍	❍	❍	❍	❍
In the last month, how often have you felt confident about your ability to handle your personal problems? (4)	❍	❍	❍	❍	❍
In the last month, how often have you felt that things were going your way? (5)	❍	❍	❍	❍	❍
In the last month, how often have you found that you could not cope with all the things that you had to do? (6)	❍	❍	❍	❍	❍
In the last	❍	❍	❍	❍	❍

month, how often have you been able to control irritations in your life? (7)					
In the last month, how often have you felt that you were on top of things? (8)	❍	❍	❍	❍	❍
In the last month, how often have you been angered because of things that were outside of your control? (9)	❍	❍	❍	❍	❍
In the last month, how often have you felt difficulties were piling up so high that you could not overcome them? (10)	❍	❍	❍	❍	❍

Word Search (low demand, during prime phase)

Try and find the fifteen words listed below: Words can appear vertically or horizontally. Remember – this is the **first** of **two** word searches and allows you to practice. If in the second word search you find five words you will win a half bar of chocolate. If you find ten you will get a full bar!

S	A	F	B	T	E	W	S	H	E	L	V	I	N	G	B	A	D	E	B
H	S	F	V	N	L	S	B	G	O	O	S	B	Q	O	T	E	V	N	M
O	E	H	D	L	T	Q	J	R	T	G	D	G	H	A	M	P	E	R	G
E	E	F	O	R	A	N	G	E	S	O	S	O	H	E	L	L	A	M	Q
L	A	D	N	Z	S	F	H	A	R	L	F	Y	F	E	B	W	B	Q	B
A	P	S	K	D	A	M	D	D	E	F	Q	F	I	R	O	Y	O	B	R
C	O	A	E	E	P	Q	S	E	W	G	M	D	N	O	S	T	S	Y	E
E	U	G	Y	H	A	E	M	O	N	K	E	Y	E	H	S	F	R	T	A
R	Y	H	W	E	X	I	A	A	R	H	M	A	H	W	A	B	C	T	D
H	T	F	G	G	W	M	X	B	T	D	G	L	A	S	S	E	S	R	S
B	R	T	X	R	S	V	E	T	E	S	M	V	I	O	L	O	M	E	T
A	A	S	Q	Y	C	L	O	U	D	Y	E	E	B	E	I	Q	U	T	I
W	S	A	V	U	W	R	U	E	R	R	F	A	A	D	G	J	E	A	C
E	P	A	P	E	R	S	A	R	E	G	B	F	D	D	E	D	F	H	K
D	A	H	B	S	D	P	D	I	R	H	O	T	F	R	E	R	S	E	S
E	P	E	E	P	W	L	F	Q	E	E	S	G	G	E	R	E	G	D	K
T	E	V	F	O	P	D	G	Q	E	D	S	S	E	A	R	A	S	E	I
Y	D	B	E	O	B	Z	B	R	O	B	W	V	A	M	O	D	E	S	S
I	F	H	S	N	M	A	N	B	L	O	U	S	E	E	B	E	Y	A	D
V	S	U	E	S	A	A	K	S	E	S	L	E	G	B	I	S	Q	U	E

Target words:

Monkey	Blouse	Hamper	Boss	Axe
Oranges	Papers	Donkey	Spoons	**Shoelace**
Cloudy	Dream	Glasses	Breadsticks	Shelving

Word Search (high demand, during prime phase)

Try and find the fifteen words listed below: Words can appear vertically or horizontally. Remember – this is the **first** of **two** word searches. If in the second word search you find five words you will win a half bar of chocolate. If you find ten you will get a full bar!

S	A	F	B	T	E	W	S	A	E	M	Q	I	N	G	B	A	D	E	B
H	S	F	V	N	L	S	B	G	O	O	S	B	Q	O	T	E	V	N	M
O	E	H	Z	L	T	Q	J	R	T	G	D	G	C	A	M	P	U	R	G
W	E	F	O	W	A	N	X	E	S	O	S	O	H	E	L	L	A	M	Q
T	A	D	N	Z	S	F	H	A	R	L	F	Y	F	E	B	W	B	Q	B
A	P	S	A	D	A	M	D	D	E	F	Q	F	I	R	Q	Y	O	B	R
C	O	A	E	E	P	Q	S	E	W	G	M	D	N	O	S	T	S	Y	E
Q	U	G	Y	H	A	E	M	O	N	K	E	Y	E	H	S	F	R	T	A
R	Y	H	W	E	X	I	A	A	R	H	M	A	H	W	A	B	C	T	D
H	T	F	G	G	W	M	X	B	T	D	G	L	E	A	M	E	S	R	E
B	R	T	X	R	S	V	M	T	E	S	M	V	I	O	L	O	M	E	C
A	A	S	Q	Y	C	L	O	U	D	Y	E	E	B	E	I	Q	U	T	K
W	S	A	V	U	W	R	U	E	R	R	F	A	A	D	G	J	E	A	C
E	R	A	P	Q	R	S	A	R	E	G	Q	F	D	D	E	D	F	H	K
D	A	H	B	S	D	P	D	I	R	H	O	T	F	R	E	R	S	E	S
E	P	E	E	P	W	L	F	Q	E	E	S	G	G	E	R	E	G	D	K
T	E	V	F	O	P	D	G	Q	E	D	A	S	E	A	R	A	S	E	I
Y	D	B	E	L	B	Z	B	R	O	B	W	V	A	M	O	D	E	S	S
I	F	H	S	A	M	A	N	B	L	E	S	S	E	E	B	E	Y	A	D
V	S	U	E	N	A	A	K	S	E	S	L	E	G	B	I	S	Q	U	E

Target words:

Monkey	Blouse	Hamper	Boss	Axe
Oranges	Papers	Donkey	Spoons	**Shoelace**
Cloudy	Dream	Glasses	Breadsticks	Shelving

Word Search (same to all participants, and was given after the prime phase)

On the next page is the second word search. Try and find the fifteen words listed at the bottom of the page: Words can appear vertically or horizontally.

Remember – this is the **second** of **two** word searches. If in this word search you find five words you will win a half bar of chocolate. If you find ten you will get a full bar!

Do not turn this page until asked!

S	A	F	B	T	E	W	S	A	E	M	Q	I	N	G	B	A	D	E	B
H	S	F	V	N	L	S	B	G	O	B	R	O	A	D	T	E	V	L	M
O	E	H	Z	E	B	R	A	R	T	G	D	G	C	A	M	P	U	O	G
W	E	F	O	W	A	N	X	E	S	O	S	O	H	R	L	L	A	O	Q
T	A	D	N	Z	S	F	H	A	R	L	F	Y	F	I	B	W	B	M	B
A	P	S	A	D	H	M	D	D	E	F	Q	F	I	C	Q	Y	O	B	R
C	O	A	E	E	I	Q	S	E	W	G	M	D	N	E	S	T	S	Y	E
Q	U	X	Y	H	F	E	P	I	L	E	E	U	E	H	S	F	R	T	A
R	Y	E	W	E	T	I	A	A	R	H	M	A	H	W	A	L	C	T	D
O	T	F	G	G	W	M	X	B	T	D	G	L	E	A	M	O	S	R	E
P	R	T	X	R	S	V	M	T	E	S	M	V	I	O	L	W	M	E	C
E	A	S	Q	Y	S	Z	O	M	B	I	E	E	B	S	I	E	U	T	K
W	F	A	V	U	W	R	U	E	R	R	F	A	A	H	G	R	E	A	C
E	A	A	P	Q	R	S	A	R	E	G	Q	F	D	O	E	D	F	H	K
D	N	H	B	U	D	P	D	I	R	H	O	T	F	E	E	R	S	E	S
E	S	E	E	I	W	L	F	Q	E	E	S	G	G	A	R	E	G	D	K
T	E	V	F	E	P	D	G	Q	E	D	A	S	E	M	R	A	S	E	I
Y	D	B	E	T	B	Z	B	R	O	B	W	V	A	R	O	D	E	S	S
I	F	H	S	A	M	A	N	B	L	E	S	S	E	E	B	E	Y	A	D
V	S	U	E	N	A	A	K	S	E	S	L	E	G	B	I	S	Q	U	E

Target words:

Axe	Shoe	Broad	Bless	Rope
Pile	Zebra	Rice	Shift	Quiet
Zombie	Fans	Flower	Rode	Loom

Correlations displayed in these supplementary tables were not corrected for multiple comparisons. The Benjamini and Hochberg's method was used, and the results of this procedure were discussed in Chapter Four.

Supplementary Table 1: *Full correlation matrix of the links between HR and Psychopathy (males; n =14)*

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1 Resting heart rate	*r*	—	.11	.01	.12	-.18	.09	.40	.17	-.29	-.32	-.47	-.15	-.21	-.17	-.25
	p	—	.71	.96	.66	.53	.74	.15	.56	.30	.26	.08	.60	.47	.54	.39
2 Primary psychopathy	*r*		—	-.52	.59	-.32	-.68	.38	-.03	.43	-.04	.11	.12	-.29	.09	-.43
	p		—	.05	.02	.26	.01	.18	.89	.12	.86	.70	.67	.31	.75	.12
3 Secondary psychopathy	*r*			—	.37	.27	.43	-.56	-.12	-.14	-.05	-.37	-.39	-.13	-.47	.39
	p			—	.18	.34	.12	.03	.67	.62	.86	.19	.16	.64	.09	.15
4 Total psychopathy (Levenson)	*r*				—	-.09	-.34	-.12	-.15	.33	-.10	-.22	-.23	-.44	-.34	-.09
	p				—	.74	.23	.68	.59	.24	.73	.43	.42	.11	.23	.75
5 Blame Externalisation	*r*					—	-.10	-.62	-.21	.09	.46	-.26	-.13	.23	-.34	.71
	p					—	.71	.02	.46	.75	.09	.36	.64	.42	.23	.01
6 Carefree Non–Planfulness	*r*						—	-.07	.25	-.41	-.04	.01	.28	.47	.34	.35
	p						—	.80	.38	.15	.88	.97	.32	.08	.23	.21
7 Coldheartedness	*r*							—	-.05	.08	-.27	.35	.37	.03	.32	-.44
	p							—	.86	.77	.34	.21	.18	.89	.25	.10
8 Fearlessness	*r*								—	.14	.06	-.28	-.06	.38	.55	.10
	p								—	.62	.82	.32	.81	.16	.04	.71
9 Machiavellian Egocentricity	*r*									—	.43	-.08	.03	.29	.08	.40
	p									—	.12	.77	.89	.30	.77	.15
10 Rebellious Nonconformity	*r*										—	.02	.01	.62	.06	.57
	p										—	.92	.98	.01	.83	.03
11 Social Influence	*r*											—	.44	.18	.48	-.23
	p											—	.11	.52	.07	.42
12 Stress Immunity	*r*												—	.53	.70	.11
	p												—	.04	.01	.68
13 Total psychopathy (PPI–R–40)	*r*													—	.65	.67
	p													—	.01	.01
14 PPI–R–40 F1 Fearlessness	*r*														—	.03
	p														—	.90
15 PPI–R–40 F2 Self–Centred Impulsivity	*r*															—

Supplementary Table 2: *Full correlation matrix of the links between HR and Psychopathy (females; n =87)*

		1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**
1 Resting heart rate	*r*	—	-.16	.08	-.07	.03	-.15	-.10	-.03	-.12	-.21	**-.25**	**-.21**	-.26	-.21	-.11
	p	—	.13	.44	.50	.77	.16	.35	.78	.27	**.05**	**.01**	**.04**	**.01**	**.05**	.28
2 Primary psychopathy	*r*		—	.47	.91	.11	-.03	.01	.26	.49	-.10	.18	-.13	.16	.05	.30
	p		—	< .001	< .001	.27	.76	.87	.01	< .001	.34	.08	.21	.13	.64	.01
3 Secondary psychopathy	*r*			—	.79	.50	.25	-.02	.05	.35	-.29	.23	-.41	.14	-.29	.61
	p			—	< .001	< .001	.02	.81	.62	< .001	.005	.02	< .001	.18	.007	< .001
4 Total psychopathy (Levenson)	*r*				—	.31	.09	-.00	.20	.50	-.21	.24	-.28	.18	-.10	.49
	p				—	.003	.37	.99	.06	< .001	.05	.02	.007	.09	.35	< .001
5 Blame Externalisation	*r*					—	-.07	-.25	.20	.26	-.22	.26	-.31	.23	-.11	.71
	p					—	.47	.01	.05	.01	.04	.01	.003	.03	.28	< .001
6 Carefree Non–Planfulness	*r*						—	.44	-.001	-.002	.01	.21	.008	.42	.009	.46
	p						—	< .001	.99	.98	.90	.05	.94	< .001	.93	< .001
7 Coldheartedness	*r*							—	-.04	.07	.17	-.01	.18	.38	.13	.10
	p							—	.68	.53	.12	.91	.08	< .001	.22	.34
8 Fearlessness	*r*								—	.18	.08	.39	.08	.62	.68	.21
	p								—	.09	.42	< .001	.43	< .001	< .001	.04
9 Machiavellian Egocentricity	*r*									—	.08	.35	-.18	.36	.06	.65
	p									—	.45	< .001	.08	< .001	.53	< .001
10 Rebellious Nonconformity	*r*										—	.222	.32	.45	.66	-.08
	p										—	.04	.002	< .001	< .001	.42
11 Social Influence	*r*											—	.09	.69	.38	.44
	p											—	.36	< .001	< .001	< .001
12 Stress Immunity	*r*												—	.33	.64	-.28
	p												—	.002	< .001	.01
13 Total psychopathy (PPI–R–40)	*r*													—	.73	.54
	p													—	< .001	< .001
14 PPI–R–40 F1 Fearlessness	*r*														—	-.03
	p														—	.76
15 PPI–R–40 F2 Self–Centred Impulsivity	*r*															—

Supplementary Table 3: *Hierarchical linear regression examining psychopathic subscales in predicting RHR*

	RHR				
	B	**(*SE*)**	**β (*p*)**	**R^2_{Adj}**	**Δ R^2**
Block 1					
Gender	4.13	(3.74)	.11 (.25)		
Fit statistics		$F_{(1,99)} = 1.33$, $p = .25$		.003	.01
Block 2					
Gender	.08	(3.80)	.002 (.98)		
Rebellious Nonconformity	-1.01	(.47)	-.23 (.002)		
Social Influence	-.86	(.44)	-.20 (.054)		
Fit statistics		$F_{(3,67)} = 3.95$, $p = .02$		.08	.10
		Durbin Watson = 2.15			

Supplementary Table 4: *Hierarchical linear regression examining psychopathy total scores in predicting RHR*

	RHR				
	B	**(*SE*)**	**β (*p*)**	**R^2_{Adj}**	**Δ R^2**
Block 1					
Gender	4.13	(3.74)	.11 (.25)		
Fit statistics		$F_{(1,99)} = 1.33$, $p = .25$		.002	.01
Block 2					
Gender	1.03	(3.85)	-.02 (.78)		
PPI–R–40 total score	-.33	(.12)	-.26 (.01)		
Fit statistics		$F_{(2,99)} = 4.04$, $p = .02$		.05	.06
			Durbin Watson = 2.45		

Supplementary Table 5: *Correlational analyses (bullying and C&T; threatening priming condition; n = 33)*

		1	2	3	4	5	6	7
1 Total bullying	*r*	–						
	p	–						
	Upper 95% CI	–						
	Lower 95% CI	–						
2 Victimisation	*r*	.56	–					
	p	**< .001**	–					
	Upper 95% CI	.76	–					
	Lower 95% CI	.27	–					
3 Bully	*r*	**.85**	.17	–				
	p	**< .001**	.35	–				
	Upper 95% CI	.92	.48	–				
	Lower 95% CI	.71	–.19	–				
4 Fighting	*r*	**.64**	–.04	**.51**	–			
	p	**< .001**	.82	**.001**	–			
	Upper 95% CI	.81	.31	.73	–			
	Lower 95% CI	.39	–.38	.20	–			
5 Base x Prime C&T	*r*	.23	**.38**	.11	–.05	–		
	p	.20	**.03**	.56	.80	–		
	Upper 95% CI	.53	.64	.43	.30	–		
	Lower 95% CI	–.12	.04	–.25	–.38	–		
6 Base x Task C&T	*r*	.08	.24	.01	–.10	**.91**	–	
	p	.65	.18	.95	.57	**< .001**	–	
	Upper 95% CI	.41	.54	.35	.25	.95	–	
	Lower 95% CI	–.27	–.12	–.33	–.43	.82	–	
7 Prime x Task C&T	*r*	–.34	–.33	–.21	–.15	–.22	.20	–
	p	.06	.06	.24	.42	.21	.25	–
	Upper 95% CI	.01	.01	.14	.21	.13	.51	–
	Lower 95% CI	–.61	–.61	–.52	–.47	–.53	–.15	–

Supplementary Table 6: *Correlational analyses (psychopathy subscales and C&T; threatening priming condition; n = 33)*

		1	2	3	4	5	6	7	8	9	10
1 Blame Externalisation	*r*	–									
	p	–									
	Upper 95% CI	–									
	Lower 95% CI	–									
2 Carefree Non–Planfulness	*r*	–.11	–								
	p	.53	–								
	Upper 95% CI	.24	–								
	Lower 95% CI	–.44	–								
3 Fearlessness	*r*	–.13	.19	–							
	p	.48	.29	–							
	Upper 95% CI	.23	.50	–							
	Lower 95% CI	–.45	–.16	–							
4 Machiavellian Egocentricity	*r*	**.34**	.04	–.13	–						
	p	**.05**	.81	.49	–						
	Upper 95% CI	.61	.38	.23	–						
	Lower 95% CI	–.00	–.30	–.45	–						
5 Rebellious Nonconformity	*r*	.04	.30	.03	.19	–					
	p	.82	.09	.86	.29	–					
	Upper 95% CI	.38	.58	.37	.50	–					
	Lower 95% CI	–.31	–.05	–.32	–.16	–					
6 Social Influence	*r*	–.22	–.05	.10	.16	**.35**	–				
	p	.23	.79	.58	.37	**.05**	–				
	Upper 95% CI	.14	.30	.43	.48	.62	–				
	Lower 95% CI	–.52	–.39	–.25	–.19	.00	–				
7 Stress Immunity	*r*	–.16	.18	**.37**	.11	.31	**.41**	–			
	p	.36	.30	**.03**	.54	.08	**.02**	–			
	Upper 95% CI	.19	.50	.63	.44	.59	.66	–			
	Lower 95% CI	–.48	–.17	.03	–.24	–.04	.08	–			
8 Base x Prime C&T	*r*	.09	.14	.08	–.20	.08	–.06	–.08	–		
	p	.64	.43	.67	.27	.67	.74	.67	–		
	Upper 95% CI	.42	.46	.41	.15	.41	.29	.27	–		
	Lower 95% CI	–.27	–.21	–.27	–.51	–.27	–.39	–.41	–		
9 Base x Task C&T	*r*	.08	–.02	.10	–.30	–.04	–.11	–.13	**.91**	–	
	p	.67	.93	.58	.09	.83	.53	.46	**< .001**	–	
	Upper 95% CI	.41	.33	.43	.05	.31	.24	.22	.95	–	
	Lower 95% CI	–.27	–.36	–.25	–.58	–.38	–.44	–.46	.82	–	
10 Prime x Task C&T	*r*	.00	**–.36**	.06	–.21	–.25	–.13	–.10	–.22	.20	–
	p	.99	**.04**	.72	.24	.17	.47	.56	.21	.25	–
	Upper 95% CI	.35	–.02	.40	.14	.11	.22	.25	.13	.51	–
	Lower 95% CI	–.34	–.63	–.28	–.52	–.54	–.45	–.43	–.53	–.15	–

Supplementary Table 7: *Correlational analyses (total psychopathy and C&T; threatening priming condition; n = 33)*

		1	2	3	4	5	6	7
1 Total psychopathy (PPI–R–40)	*r*	–						
	p	–						
	Upper 95% CI	–						
	Lower 95% CI	–						
2 Fearlessness Dominance factor	*r*	.75	–					
	p	**< .001**	–					
	Upper 95% CI	.87	–					
	Lower 95% CI	.55	–					
3 Self–centred impulsivity factor	*r*	.50	–.02	–				
	p	**<.001**	.92	–				
	Upper 95% CI	.72	.33	–				
	Lower 95% CI	.19	–.36	–				
4 Coldheartedness factor	*r*	.26	–.15	.17	–			
	p	.14	.42	.33	–			
	Upper 95% CI	.55	.21	.49	–			
	Lower 95% CI	–.09	–.47	–.18	–			
5 Base x Prime C&T	*r*	.02	–.01	.02	–.00	–		
	p	.90	.93	.89	.98	–		
	Upper 95% CI	.36	.33	.37	.34	–		
	Lower 95% CI	–.32	–.36	–.32	–.35	–		
6 Base x Task C&T	*r*	–.08	–.05	–.12	–.01	**.91**	–	
	p	.67	.79	.52	.94	**< .001**	–	
	Upper 95% CI	.27	.30	.24	.33	.95	–	
	Lower 95% CI	–.41	–.39	–.44	–.35	.82	–	
7 Prime x Task C&T	*r*	–.21	–.06	–.30	–.04	–.22	.20	–
	p	.24	.73	.09	.84	.21	.25	–
	Upper 95% CI	.14	.29	.05	.31	.13	.51	–
	Lower 95% CI	–.52	–.40	–.58	–.37	–.53	–.15	–

Supplementary Table 8: *Correlational analyses (bullying and C&T; challenging priming condition; n = 35*

		1	2	3	4	5	6	7
1 Total bullying	*r*	–						
	p	–						
	Upper 95% CI	–						
	Lower 95% CI	–						
2 Victimisation	*r*	.81	–					
	p	< .001	–					
	Upper 95% CI	.90	–					
	Lower 95% CI	.66	–					
3 Bully	*r*	.65	.18	–				
	p	**< .001**	.31	–				
	Upper 95% CI	.81	.48	–				
	Lower 95% CI	.41	–.17	–				
4 Fighting	*r*	**.80**	**.62**	.28	–			
	p	**< .001**	**< .001**	.11	–			
	Upper 95% CI	.89	.79	.56	–			
	Lower 95% CI	.63	.36	–.06	–			
5 Base x Prime C&T	*r*	–.13	–.32	.10	–.04	–		
	p	.45	.06	.56	.82	–		
	Upper 95% CI	.21	.01	.42	.30	–		
	Lower 95% CI	–.45	–.59	–.24	–.37	–		
6 Base x Task C&T	*r*	–.23	–.27	–.05	–.21	**.83**	–	
	p	.18	.12	.78	.22	**.001**	–	
	Upper 95% CI	.11	.07	.29	.13	.91	–	
	Lower 95% CI	–.52	–.55	–.38	–.51	.69	–	
7 Prime x Task C&T	*r*	–.14	.15	–.26	–.26	**–.39**	.18	–
	p	.43	.39	.13	.13	**.02**	.29	–
	Upper 95% CI	.21	.46	.08	.08	–.06	.49	–
	Lower 95% CI	–.45	–.19	–.55	–.55	–.64	–.16	–

Supplementary Table 9: *Correlational analyses (psychopathy subscales and C&T; challenging priming condition; n = 35)*

			1	2	3	4	5	6	7	8	9	10
1 Blame Externalisation	*r*	–										
	p	–										
	Upper 95% CI	–										
	Lower 95% CI	–										
2 Carefree Non–Planfulness	*r*	–.08	–									
	p	.66	–									
	Upper 95% CI	.26	–									
	Lower 95% CI	–.40	–									
3 Fearlessness	*r*	.05	.13	–								
	p	.75	.47	–								
	Upper 95% CI	.38	.44	–								
	Lower 95% CI	–.28	–.22	–								
4 Machiavellian Egocentricity	*r*	.20	–.03	**.35**	–							
	p	.25	.85	**.04**	–							
	Upper 95% CI	.50	.30	.61	–							
	Lower 95% CI	–.14	–.36	.02	–							
5 Rebellious Nonconformity	*r*	.31	.15	.38	.50	–						
	p	.07	.39	.02	**.001**	–						
	Upper 95% CI	.58	.46	.63	.72	–						
	Lower 95% CI	–.03	–.19	.05	.20	–						
6 Social Influence	*r*	–.36	–.07	.05	.10	.16	–					
	p	.03	.69	.76	.56	.35	–					
	Upper 95% CI	–.03	.27	.38	.42	.47	–					
	Lower 95% CI	–.62	–.39	–.29	–.24	–.18	–					
7 Stress Immunity	*r*	–.27	.10	–.09	–.18	.14	**.34**	–				
	p	.11	.57	.62	.30	.42	**.04**	–				
	Upper 95% CI	.07	.42	.25	.16	.45	.61	–				
	Lower 95% CI	–.56	–.24	–.41	–.48	–.20	.01	–				
8 Base x Prime C&T	*r*	–.31	.24	–.02	–.13	–.08	.18	.12	–			
	p	.07	.17	.91	.46	.66	.29	.48	–			
	Upper 95% CI	.03	.53	.32	.21	.26	.49	.44	–			
	Lower 95% CI	–.58	–.10	–.35	–.44	–.40	–.16	–.22	–			
9 Base x Task C&T	*r*	–.27	.17	–.14	–.32	–.15	.05	.03	**.83**	–		
	p	.12	.32	.43	.06	.39	.78	.84	**.001**	–		
	Upper 95% CI	.07	.48	.21	.01	.19	.38	.36	.91	–		
	Lower 95% CI	–.55	–.17	–.45	–.59	–.46	–.29	–.30	.69	–		
10 Prime x Task C&T	*r*	.13	–.13	–.20	–.30	–.11	–.26	–.17	**–.39**	.18	–	
	p	.45	.46	.26	.08	.52	.13	.33	**.02**	.29	–	
	Upper 95% CI	.45	.21	.15	.04	.23	.08	.17	–.06	.49	–	
	Lower 95% CI	–.21	–.44	–.50	–.58	–.43	–.55	–.48	–.64	–.16	–	

Supplementary Table 10: *Correlational analyses (total psychopathy and C&T; challenging priming condition; n = 35)*

		1	2	3	4	5	6	7
1 Total psychopathy (PPI–R–40)	*r*	–						
	p	–						
	Upper 95% CI	–						
	Lower 95% CI	–						
2 Fearlessness Dominance factor	*r*	.75	–					
	p	< .001	–					
	Upper 95% CI	.86	–					
	Lower 95% CI	.55	–					
3 Self–centred impulsivity factor	*r*	.53	–.07	–				
	p	**.001**	.67	–				
	Upper 95% CI	.73	.27	–				
	Lower 95% CI	.24	–.40	–				
4 Coldheartedness factor	*r*	**.47**	**.39**	.01	–			
	p	**.001**	**.02**	.95	–			
	Upper 95% CI	.69	.64	.34	–			
	Lower 95% CI	.16	.07	–.32	–			
5 Base x Prime C&T	*r*	–.01	.14	–.17	–.05	–		
	p	.95	.42	.33	.80	–		
	Upper 95% CI	.32	.45	.17	.29	–		
	Lower 95% CI	–.34	–.20	–.48	–.37	–		
6 Base x Task C&T	*r*	–.22	–.04	–.26	–.25	.83	–	
	p	.20	.80	.14	.15	< .001	–	
	Upper 95% CI	.12	.29	.08	.09	.91	–	
	Lower 95% CI	–.52	–.37	–.54	–.54	.69	–	
7 Prime x Task C&T	*r*	**–.35**	**–.34**	–.10	**–.35**	**–.39**	.18	–
	p	**.04**	**.05**	.57	**.04**	**.02**	.29	–
	Upper 95% CI	–.02	–.00	.24	–.02	–.06	.49	–
	Lower 95% CI	–.61	–.60	–.42	–.61	–.64	–.16	–

Supplementary Table 11: *Correlational analyses (bullying and C&T; males; n = 11)*

		1	2	3	4	5	6	7
1 Total bullying	*r*	—	**.95**	**.69**	**.93**	-.08	-.24	-.21
	p	—	**< .001**	**.02**	**< .001**	.82	.48	.53
	Upper 95% CI	—	.98	.91	.98	.55	.42	.44
	Lower 95% CI	—	.82	.16	.75	-.65	-.73	-.72
2 Victimisation	*r*		—	.45	.98	-.09	-.20	-.11
	p		—	.16	< .001	.78	.57	.73
	Upper 95% CI		—	.82	.99	.53	.46	.52
	Lower 95% CI		—	-.21	.91	-.66	-.71	-.67
3 Bully	*r*			—	.39	-.001	-.26	-.37
	p			—	.23	.99	.44	.26
	Upper 95% CI			—	.80	.60	.40	.23
	Lower 95% CI			—	-.27	-.60	-.74	-.75
4 Fighting	*r*				—	-.09	-.17	-.07
	p				—	.77	.61	.82
	Upper 95% CI				—	.53	.47	.58
	Lower 95% CI				—	-.66	-.70	-.67
5 Base x Prime C&T	*r*					—	.86	-.62
	p					—	< .001	.05
	Upper 95% CI					—	.96	-.003
	Lower 95% CI					—	.54	-.88
6 Base x Task C&T	*r*						—	-.11
	p						—	.74
	Upper 95% CI						—	.52
	Lower 95% CI						—	-.66
7 Prime x Task C&T	*r*							—
	p							—
	Upper 95% CI							—
	Lower 95% CI							—

Supplementary Table 12: *Correlational analyses (psychopathy subscales and C&T; males; n = 11)*

		1	2	3	4	5	6	7	8	9	10
1 Blame Externalisation	*r*	—	-.13	-.18	.32	**.70**	-.20	-.17	-.13	-.31	-.23
	p	—	.70	.59	.33	**.016**	.55	.61	.71	.34	.48
	Upper 95% CI	—	.51	.47	.77	.91	.45	.47	.51	.35	.42
	Lower 95% CI	—	-.68	-.70	-.34	.18	-.71	-.70	-.67	-.77	-.73
2 Carefree Non–Planfulness	*r*		—	.30	-.49	-.028	.03	.47	.37	.32	-.21
	p		—	.37	.12	.93	.92	.14	.26	.34	.52
	Upper 95% CI		—	.76	.15	.58	.62	.83	.79	.77	.44
	Lower 95% CI		—	-.37	-.84	-.62	-.57	-.17	-.29	-.34	-.72
3 Fearlessness	*r*			—	-.12	-.06	-.32	-.11	.19	.18	-.11
	p			—	.72	.86	.34	.74	.56	.60	.73
	Upper 95% CI			—	.51	.56	.35	.52	.71	.70	.52
	Lower 95% CI			—	-.67	-.63	-.77	-.67	-.46	-.47	-.67
4 Machiavellian Egocentricity	*r*				—	.27	-.16	-.18	-.01	.07	.16
	p				—	.42	.64	.59	.97	.84	.64
	Upper 95% CI				—	.75	.49	.47	.59	.64	.69
	Lower 95% CI				—	-.39	-.69	-.70	-.61	-.55	-.49
5 Rebellious Nonconformity	*r*					—	-.04	.001	.04	.03	-.04
	p					—	.89	.99	.90	.92	.91
	Upper 95% CI					—	.57	.60	.62	.62	.57
	Lower 95% CI					—	-.63	-.60	-.57	-.58	-.62
6 Social Influence	*r*						—	.51	-.19	-.21	.05
	p						—	.11	.58	.53	.87
	Upper 95% CI						—	.85	.46	.44	.63
	Lower 95% CI						—	-.13	-.71	-.72	-.56
7 Stress Immunity	*r*							—	.19	.41	.31
	p							—	.60	.21	.36
	Upper 95% CI							—	.70	.81	.76
	Lower 95% CI							—	-.47	-.25	-.36
8 Base x Prime C&T	*r*								—	.86	-.60
	p								—	< .001	.05
	Upper 95% CI								—	.96	-.003
	Lower 95% CI								—	.54	-.88
9 Base x Task C&T	*r*									—	-.11
	p									—	.73
	Upper 95% CI									—	.52
	Lower 95% CI									—	-.67
10 Prime x Task C&T	*r*										—
	p										—
	Upper 95% CI										—
	Lower 95% CI										—

Supplementary Table 13: *Correlational analyses (total psychopathy and C&T; males; n = 11)*

		1	2	3	4	5	6	7
1 Total psychopathy (PPI–R–40)	*r*	—	**.61**	**.69**	.01	.11	.16	.056
	p	—	**.04**	**.02**	.97	.75	.63	.87
	Upper 95% CI	—	.88	.91	.60	.66	.68	.63
	Lower 95% CI	—	.01	.15	-.59	-.52	-.48	-.56
2 Fearlessness Dominance factor	*r*		—	-.01	.31	.15	.25	.10
	p		—	.98	.35	.65	.45	.76
	Upper 95% CI		—	.59	.77	.69	.74	.66
	Lower 95% CI		—	-.60	-.35	-.49	-.41	-.53
3 Self–centred impulsivity factor	*r*			—	-.49	.16	.02	-.26
	p			—	.13	.63	.95	.44
	Upper 95% CI			—	.16	.69	.61	.40
	Lower 95% CI			—	-.84	-.48	-.58	-.74
4 Coldheartedness factor	*r*				—	-.22	.10	.59
	p				—	.52	.76	.05
	Upper 95% CI				—	.44	.66	.88
	Lower 95% CI				—	-.72	-.53	-.01
5 Base x Prime C&T	*r*					—	.86	-.60
	p					—	< .001	.05
	Upper 95% CI					—	.96	-.003
	Lower 95% CI					—	.54	-.88
6 Base x Task C&T	*r*						—	-.11
	p						—	.74
	Upper 95% CI						—	.52
	Lower 95% CI						—	-.67
7 Prime x Task C&T	*r*							—
	p							—
	Upper 95% CI							—
	Lower 95% CI							—

Supplementary Table 14: *Correlational analyses (bullying and C&T; females; n = 57)*

		1	2	3	4	5	6	7
1 Total bullying	*r*	—	**.76**	**.72**	**.74**	-.005	-.09	-.17
	p	—	**< .001**	**< .001**	**< .001**	.97	.48	.21
	Upper 95% CI	—	.85	.83	.84	.25	.17	.09
	Lower 95% CI	—	.63	.57	.60	-.26	-.34	-.41
2 Victimisation	*r*		—	.20	.46	-.11	-.086	.06
	p		—	.12	< .001	.40	.52	.65
	Upper 95% CI		—	.44	.64	.15	.18	.32
	Lower 95% CI		—	-.05	.22	-.36	-.34	-.20
3 Bully	*r*			—	.36	.12	.003	-.23
	p			—	.006	.38	.98	.08
	Upper 95% CI			—	.56	.37	.26	.03
	Lower 95% CI			—	.10	-.14	-.26	-.46
4 Fighting	*r*				—	-.02	-.15	-.26
	p				—	.89	.25	.05
	Upper 95% CI				—	.24	.11	.004
	Lower 95% CI				—	-.28	-.40	-.48
5 Base x Prime C&T	Pearson's r					—	.87	-.31
	p-value					—	< .001	.02
	Upper 95% CI					—	.92	-.05
	Lower 95% CI					—	.79	-.52
6 Base x Task C&T	*r*						—	.20
	p						—	.13
	Upper 95% CI						—	.44
	Lower 95% CI						—	-.06
7 Prime x Task C&T	*r*							—
	p							—
	Upper 95% CI							—
	Lower 95% CI							—

Supplementary Table 15: *Correlational analyses (psychopathy subscales and C&T; females; n = 57)*

		1	2	3	4	5	6	7	8	9	10
1 Blame Externalisation	*r*	—	-.10	.04	.23	.14	**-.32**	-.22	-.14	-.09	.13
	p	—	.44	.77	.08	.30	**.01**	.10	.29	.50	.34
	Upper 95% CI	—	.16	.30	.46	.39	-.06	.05	.12	.17	.37
	Lower 95% CI	—	-.35	-.22	-.030	-.12	-.54	-.45	-.39	-.34	-.14
2 Carefree Non–Planfulness	*r*		—	.08	.12	.23	-.13	.01	.15	.02	-.23
	p		—	.57	.37	.08	.35	.93	.26	.86	.07
	Upper 95% CI		—	.33	.37	.46	.14	.27	.39	.28	.03
	Lower 95% CI		—	-.19	-.14	-.03	-.37	-.25	-.11	-.24	-.46
3 Fearlessness	*r*			—	.13	.30	.11	.17	.009	-.04	-.01
	p			—	.34	.02	.40	.19	.94	.76	.47
	Upper 95% CI			—	.38	.52	.36	.42	.27	.22	.17
	Lower 95% CI			—	-.13	.04	-.15	-.09	-.25	-.30	-.35
4 Machiavellian Egocentricity	*r*				—	.36	.15	-.03	-.18	-.35	-.29
	p				—	.006	.27	.81	.16	.008	.02
	Upper 95% CI				—	.56	.39	.23	.07	-.01	-.04
	Lower 95% CI				—	.11	-.12	-.29	-.42	-.56	-.51
5 Rebellious Nonconformity	*r*					—	.21	.22	-.03	-.14	-.19
	p					—	.12	.10	.81	.30	.15
	Upper 95% CI					—	.44	.45	.23	.13	.07
	Lower 95% CI					—	-.05	-.04	-.29	-.38	-.43
6 Social Influence	*r*						—	.31	.09	-.04	-.27
	p						—	.02	.52	.74	.04
	Upper 95% CI						—	.52	.34	.22	-.01
	Lower 95% CI						—	.05	-.17	-.30	-.49
7 Stress Immunity	*r*							—	.007	-.11	-.21
	p							—	.96	.42	.11
	Upper 95% CI							—	.26	.16	.05
	Lower 95% CI							—	-.25	-.36	-.45
8 Base x Prime C&T	*r*								—	.87	-.31
	p								—	< .001	.02
	Upper 95% CI								—	.92	-.05
	Lower 95% CI								—	.79	-.52
9 Base x Task C&T	*r*									—	.20
	p									—	.13
	Upper 95% CI									—	.44
	Lower 95% CI									—	-.06
10 Prime x Task C&T	*r*										—
	p										—
	Upper 95% CI										—
	Lower 95% CI										—

Supplementary Table 16: *Correlational analyses (total psychopathy and C&T; females; n = 57)*

		1	2	3	4	5	6	7
1 Total psychopathy (PPI–R–40)	*r*	—	**.73**	**.49**	**.41**	-.02	-.21	**-.36**
	p	—	**< .001**	**< .001**	**.001**	.88	.11	**.005**
	Upper 95% CI	—	.84	.67	.61	.24	.05	-.11
	Lower 95% CI	—	.59	.27	.17	-.28	-.45	-.57
2 Fearlessness Dominance factor	*r*		—	-.080	.07	.05	-.09	-.27
	p		—	.56	.58	.71	.51	.04
	Upper 95% CI		—	.18	.33	.30	.17	-.01
	Lower 95% CI		—	-.33	-.19	-.21	-.34	-.50
3 Self–centred impulsivity factor	*r*			—	.17	-.11	-.22	-.17
	p			—	.19	.42	.11	.19
	Upper 95% CI			—	.42	.15	.05	.09
	Lower 95% CI			—	-.090	-.36	-.45	-.41
4 Coldheartedness factor	*r*				—	-.02	-.17	-.31
	p				—	.88	.20	.02
	Upper 95% CI				—	.24	.09	-.05
	Lower 95% CI				—	-.28	-.41	-.52
5 Base x Prime C&T	*r*					—	.87	-.30
	p					—	< .001	.02
	Upper 95% CI					—	.92	-.05
	Lower 95% CI					—	.79	-.52
6 Base x Task C&T	*r*						—	.20
	p						—	.13
	Upper 95% CI						—	.44
	Lower 95% CI						—	-.06
7 Prime x Task C&T	*r*							—
	p							—
	Upper 95% CI							—
	Lower 95% CI							—

Supplementary Table 17: *Hierarchical linear regression to examine predictors of physiological reactivity*

	Baseline x task reactivity index					**Prime x task reactivity index**				
	B	**(*SE*)**	**β (*p*)**	**R^2_{Adj}**	**Δ R^2**	**B**	**(*SE*)**	**β (*p*)**	**R^2_{Adj}**	**Δ R^2**
Block 1										
Gender	-.37	(0.61)	-.07 (.54)			-.22	(.58)	-.04 (.70)		
Fit statistics	$F_{(1,67)}$ =.77, p = .54			-.09	.06	$F_{(1,67)}$ = .14, p = .70			-.01	.002
Block 2										
Gender	-.49	(.58)	-.09 (.40)			-.70	(.58)	-.14 (.24)		
Machiavellian Egocentricity	-.23	(.88)	-.31 (.01)			-	-	-		
PPI–R–40 total score	-	-	-			-.06	(.21)	-.34 (.007)		
Fit statistics	$F_{(2,67)}$ = 3.87, p = .02			.07	.10	$F_{(2,67)}$ = 3.95, p = .02			.08	0.10
	Durbin Watson = 2.45					Durbin Watson = 2.14				

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