Low Heart Rate and Crime: Exploring the Link From an Analytical Perspective

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Declaration

This thesis is the result of my own work and includes nothing which is the outcome of work done in collaboration except as declared in the Preface and specified in the text. It is not substantially the same as any that I have submitted, or, is being concurrently submitted for a degree or diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared in the Preface and specified in the text. I further state that no substantial part of my thesis has already been submitted, or, is being concurrently submitted for any such degree, diploma or other qualification at the University of Cambridge or any other University or similar institution except as declared in the Preface and specified in the text. It does not exceed the prescribed word limit for the Law Degree Committee.

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Title: Low Heart Rate and Crime: Exploring the Link From an Analytical Perspective
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Abstract

Although low resting heart rate is often cited as "the best-replicated biological correlate" of antisocial behaviour, this association remains unexplained. Several theoretical explanations have been proposed throughout the years, but they have been mostly based on unfounded and questionable assumptions. Amongst these, fearlessness and stimulation-seeking hypotheses have gained ground and become well established in the literature, despite the fact that they have been subjected to scarce empirical verifications yielding mixed results. Therefore, employing situational action theory as a theoretical framework, a new explanatory model of the relationship between low heart rate and crime was developed. It was argued that heart rate cannot be a cause of crime, but only a marker of a cause of crime. Specifically, it was hypothesised that a person's crime propensity would fully mediate the link between heart rate and crime. In order to test the crime propensity model and compare its efficiency against the fearlessness and stimulation-seeking hypotheses, the Cambridge adolescent behaviour study was conducted. It set out to explore the mechanism underlying the heart rate-crime association in a sample of 487 adolescents recruited from 14 schools in England. Participants completed self-report measures of stimulation-seeking, fearlessness, crime propensity, and criminal behaviour, and their heart rate was measured at rest and in response to a stressor. Crime involvement was significantly associated only with heart rate reactivity, whereas the link with resting heart rate was non-significant. Out of the three models with fearlessness, stimulation-seeking, and crime propensity as potential mediators, only crime propensity fully mediated the link between heart rate reactivity and crime, rendering the direct effect of heart rate reactivity on crime non-significant. Therefore, the empirical results supported the newly developed crime propensity model and highlighted the benefits of replacing the prevalent risk factor approach in (biosocial) criminology with an analytical approach that aims to explain what causes crime and how, and, equally importantly, what cannot cause crime.

Preface

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Chapter 1:

Low Heart Rate and Antisocial Behaviour: Review of Empirical Evidence

1.1. Biosocial Criminology: Are We in Need of (Another) Paradigm Shift?

Criminology has traditionally relied on social factors in the study of crime, as sociological criminology was the dominating and uncontested paradigm in the 20th century (Cullen, 2009). However, in recent years a substantial body of empirical evidence has drawn attention to the fact that biological factors too have an important role in explaining criminal behaviour. This has paved the way for a new and up-and-coming paradigm – a biosocial criminology. It represents an integrative and multidisciplinary perspective that seeks to incorporate knowledge and methods from various fields such as genetics, neuroscience, endocrinology, and evolutionary psychology, along with findings about environmental factors, into a study of crime and antisocial behaviour (Beaver & Walsh, 2011). Instead of focusing on a pseudo-dichotomy of nature vs nurture, it explores how the interaction between nature and nurture gives rise to antisocial propensity (Eichelberger & Barnes, 2015). Although initially there was a strong resistance against biosocial criminology based mostly on ideological grounds (Wright & Cullen, 2012), that surprisingly still lingers on (e.g., Carrier & Walby, 2014; Fallin, Whooley, & Barker, 2018; Larregue & Rollins, 2019), it has become increasingly difficult to ignore the role of biological factors in criminal behaviour due to a large number of empirical evidence that has accumulated over time. During the past 15 years, there has been a significant increase in published biosocial work transforming such publications from being outliers to appearing often in criminological journals and having dedicated special issues (Barnes & Boutwell, 2015; DeLisi, 2009; Vaske, 2017). A Google Scholar search for the keywords *biosocial criminology* by the end of 2019 gave more than 9000 results, out of which more than 60% has been published in the last 10 years.

As a consequence of the increase in biosocial research in criminology, nowadays there is a well-documented knowledge about which biological factors are related to criminal behaviour, alongside recent efforts to apply these findings to intervention programmes aimed at reducing antisocial behaviour (Bootsman, 2019; Choy, Focquaert, & Raine, 2020; Glenn & McCauley, 2019). The list of biological correlates of crime, aggression, and antisocial behaviour in general is virtually endless, including structural and functional abnormalities of different brain regions (e.g., the prefrontal cortex, amygdala, striatum), neurotransmitters (e.g., serotonin, noradrenalin, dopamine), hormones (e.g., testosterone, thyroid hormones, cortisol), candidate genes (e.g., MAO-A, DAT1, 5-HTTLPR), neurocognitive malfunctioning (e.g., working memory, attention, inhibition), psychophysiology (e.g., indices of cardiovascular, brain wave, and electrodermal activity), prenatal and perinatal factors (e.g., birth complications, maternal nicotine and/or alcohol consumption, malnutrition, heavy metal blood levels; Beaver & Walsh, 2011; DeLisi & Vaughn, 2015; Glenn & Raine, 2014; Ogilvie, Stewart, Chan, & Shum, 2011; Raine, 2002, 2018; Van Der Gronde, Kempes, Van El, Rinne, & Pieters, 2014; Walsh & Beaver, 2009). Biosocial research has advanced criminology by bringing biology, a long-overlooked, but an indispensable part of the criminological puzzle, back into the discussion. It has also undoubtably enriched factual knowledge about antisocial behaviour. However, criminology has not yet fully seized all the opportunities to advance the understanding of crime that the biosocial perspective has to offer.

Findings from biosocial studies could be used to falsify existing criminological theories, contribute to a more nuanced understanding of how individual crime propensity develops, and help guide future research endeavours into a more promising direction. Although there have been some attempts in using biosocial empirical data to falsify certain hypotheses from mainstream criminological theories (e.g., Beaver, Wright, & DeLisi, 2007; Wikström & Treiber, 2007), these have not gained ground in the field. Instead, criminology has continued its trend of adding more theories without falsifying the existing ones (Beaver & Walsh, 2011). For example, leaving aside all the other problems of the general theory of crime (Gottfredson & Hirschi, 1990), its hypotheses that development of self-control is completed by the age of 10 and solely determined by (parental) socialisation stand in stark contrast with genetic, neuroscientific, and neuropsychological literature. There is an overwhelming empirical evidence which shows that the ability to exercise self-control depends on an efficient functioning of executive functions seated in the prefrontal cortex which develop well into young adulthood following a rapid prefrontal brain maturation during adolescence (Beaver et al., 2007; Caballero, Granberg, & Tseng, 2016; Cauffman, Steinberg, & Piquero, 2005; Jackson & Beaver, 2013; Steinberg et al., 2008; Wikström & Treiber, 2007; Yurgelun-Todd, 2007). Likewise, research exploring development of impulsivity (i.e., low self-control) shows that it too follows a trajectory of linear decrease from childhood into the adulthood (Burt, 2020; Burt, Sweeten, & Simons, 2014; Steinberg et al., 2008, 2009). The premise that self-control remains stable after the suggested formative period is clearly refuted by a number of studies showing that self-control is only moderately stable and that there are both intra- and inter-individual differences across the life-span (for a review, please see Burt et al., 2014). Therefore, the idea that one's self-control is fully developed by the age of 10 and unchangeable thereafter is simply untenable.

Biosocial research also refutes the other major hypothesis of the general theory of crime relating to the notion that low self-control is solely caused by ineffective (parental) socialisation practices. Instead, the ability to exercise self-control also depends on the integrity of specific neurocognitive factors, including the above-mentioned executive functions supported by the prefrontal cortex. Neuroscientific research has clearly demonstrated that distinct prefrontal brain areas support different functions indispensable for efficient behavioural inhibition. Specifically, the dorsolateral prefrontal cortex is relevant for the cognitive aspects of self-control such as working memory and attention, whereas the orbitomedial prefrontal cortex underpins the emotional and motivational aspects such as affect control and value-based decision-making (Boes et al., 2011; Damasio, 1994; Ishikawa & Raine, 2003; Schneider & Koenigs, 2017; Szczepanski & Knight, 2014). Indeed, lesion studies and research involving brain stimulation techniques in healthy participants have provided evidence for the causal role of the prefrontal cortex in exhibiting self-control. Namely, it has been reported that patients following the prefrontal damage exhibit impulsive and disinhibited behaviour (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Meyers, Berman, Scheibel, & Hayman, 1992; Schneider & Koenigs, 2017; Szczepanski & Knight, 2014), whereas applying brain stimulation techniques over the prefrontal cortex of healthy participants has been shown to induce changes in exhibiting self-control (Brevet-Aeby, Brunelin, Iceta, Padovan, & Poulet, 2016; Cho et al., 2010; Falcone et al., 2016; Knoch & Fehr, 2007). Finally, behavioural genetics studies have demonstrated a significant genetic contribution to self-control variability at a population level (Beaver et al., 2009; Beaver, Wright, DeLisi, & Vaughn, 2008; Wright & Beaver, 2005).

However, despite all the accumulated empirical evidence disproving specific hypotheses of the general theory of crime, it has not been modified nor discarded; instead, it is still widely used across the field in its original form (e.g., Donner, Marcum, Jennings, Higgins, & Banfield, 2014; Hirtenlehner & Kunz, 2017; Vazsonyi, Jiskrova, Ksinan, & Blatný, 2016; Vazsonyi, Mikuška, & Kelley, 2017). Therefore, as the scientific progress of any discipline is based on falsification of some theories and validation of others (Bernard, 1990), it is hard to argue that criminology has moved forward despite all the novel biosocial data. In that context, the more important question is not whether a theory has accumulated a large amount of empirical support, but if any sound research has falsified it. As seen in the example of the general theory of crime, criminology has not done much in the past decades to advance its scientific progress and falsify its numerous theories despite advances in biosocial research whose findings could contribute to the falsification process.

Apart from falsifying criminological theories, biosocial findings could also be used to delineate causal pathways through which an interaction between biology and environment gives rise to individual tendency to commit crime. This could be achieved through a two-fold process involving reviewing and integrating existing elements of biosocial knowledge, as well as generating new findings by conducting novel, biologically-informed research. Current biosocial literature could be reviewed and parsed through to distinguish factors that could be causally related to individual crime propensity, such as the brain mechanisms underpinning ability to exercise self-control or moral decision-making (e.g., Ishikawa & Raine, 2003; Raine, 2019; Treiber, 2011; Wikström & Treiber, 2007), and set them apart from mere correlates of crime that could only be markers of causes at best, such as low-seated ears or low resting heart rate (Glenn & Raine, 2014; Raine, 2013). As an example of this practice, Burt and Simons (2014) suggested that the well-established link between childhood maltreatment and later antisocial and criminal behaviour (Afifi, Fortier, Sareen, & Taillieu, 2019; Currie & Tekin, 2012; Mersky, Topitzes, & Reynolds, 2012; Smith, Ireland, Thornberry, & Elwyn, 2008; Smith & Thornberry, 1995) could be explained using neurological mechanisms. Namely, they integrated findings showing that childhood adversity is associated with structural and functional alterations in the amygdala (e.g., McCrory et al., 2013; Pechtel, Lyons-Ruth, Anderson, & Teicher, 2014), the prefrontal cortex (e.g., Hart et al., 2018; Van Harmelen et al., 2010), and their inter-connectivity (e.g., Birn, Patriat, Phillips, Germain, & Herringa, 2014; Jedd et al., 2015), which in turn underpin emotion regulation (e.g., Banks, Eddy, Angstadt, Nathan, & Luan Phan, 2007; Golkar et al., 2012; Hare, Tottenham, Davidson, Glover, & Casey, 2005) and impulsivity (e.g., Goya-Maldonado et al., 2010; Kerr et al., 2015), into a potential explanatory mechanism by which childhood maltreatment causes crime through altering brain development and decreasing self-control.

Furthermore, future criminological research investigating individual causes of crime could generate more precise and nuanced explanations if it were guided by biosocial findings about the bi-directional and dynamic interaction between biological and environmental factors. For example, almost 20 years ago a high-impact publication using data from the Dunedin longitudinal study demonstrated differential effects of childhood maltreatment on a person's later antisocial behaviour conditional on the individual's MAO-A genotype[1] (Caspi et al., 2002). Therefore, more of such research could provide invaluable insights into how the same environmental factors can lead to different antisocial outcomes depending on the

[1] Specifically, this refers to a functional polymorphism in the gene encoding monoamine oxidase A enzyme which breaks down neurotransmitters such as serotonin, dopamine, and norepinephrine, and thereby affects brain neurochemistry (Caspi et al., 2002). Since the original finding was published, the effect of this particular gene-environment interaction on antisocial behaviour has been replicated across different studies (for a review, please see Byrd & Manuck, 2014).

underlying biological characteristics of an individual. In a like manner, future criminological research inspired by the latest developments in epigenetics, which refers to any process that changes gene expression without affecting the DNA sequence (Weinhold, 2006), has a potential to inform on how different environmental conditions can lead to different outcomes by affecting gene expression which in turn changes neural biochemistry (Burt & Simons, 2014; Palumbo, Mariotti, Iofrida, & Pellegrini, 2018). Having this in mind, it has been encouraging to see the recent emergence of articles introducing epigenetics into the field of criminology (Burt & Simons, 2014; Palumbo et al., 2018; Walsh & Yun, 2014), including the latest article attempting to integrate epigenetics and micro-geographic perspective within the criminology of place which proposed that living in a crime hot-spot can lead to crime through interaction with genetic factors (Leshem & Weisburd, 2019). In addition, findings regarding neuroplasticity (i.e., the capacity of brain to change and rewire its structure and function in response to experiences throughout life-course; Draganski et al., 2006; Maguire, Woollett, & Spiers, 2006; Münte, Altenmüller, & Jäncke, 2002; Voss, Thomas, Cisneros-Franco, & De Villers-Sidani, 2017) and adult neurogenesis (i.e., the ability of brain to create new neurons throughout life; Kempermann et al., 2018; Ming & Song, 2011; Steiner, Tata, & Frisén, 2019; Zhao, Deng, & Gage, 2008) have opened up exciting new avenues of research that could provide important insights on the fine interaction between nature and nurture, and how it leads to prosocial or antisocial behaviour. Apart from generating a more comprehensive understanding of crime propensity development, this type of research could also inform and inspire novel prevention and intervention programmes (Leshem & Weisburd, 2019; Palumbo et al., 2018; Tremblay, 2015). Overall, it seems that there is much room for improvement in criminology and that the field could benefit more from biosocial paradigm if it were to apply biological findings to both criminological theories and research.

1.1.1. Risk Factor Approach: A Dominating Paradigm

One of the main reasons why biosocial findings have not been sufficiently used to advance our understanding of criminal behaviour is the prevailing risk-factor (i.e., correlational) paradigm, that has plagued not only biosocial criminology, but the entire discipline in general. Instead of an analytical perspective, biosocial criminology generally tends to employ a correlational approach which focuses exclusively on statistical associations between variables, without considering if these variables could be causally related to crime and how (i.e., through which mechanisms; Treiber, 2017a). This approach has led to piling up a large number of (in many cases, causally irrelevant) correlates which obscure the overall understanding of crime rather than advancing it (Wikström, 2006). Furthermore, in some instances when an attempt to explain the mechanism underlying the link between biological factors and crime has been made, scholars use the most convenient criminological theories without taking into consideration the major criticisms these theories face, which renders their explanations dubious (e.g., Armstrong & Boutwell, 2012; Rocque, Posick, & Felix, 2015). This is the consequence of the aforementioned lack of falsification process in criminology, which leaves the discipline in a poor state where one is quickly overwhelmed by an abundance of theories which have one thing in common - they have never been (acknowledged as) refuted despite the enormous volume of research (Bruinsma, 2016). As a result, biosocial criminology has developed a strong evidence base about biological correlates of antisocial behaviour, but distinguishing which few of the many correlates have causal properties and delineating the mechanisms by which they are associated with crime has been lagging behind.

The fundamental problem with the risk-factor approach is that it does not make a difference between causes, markers, correlates, and statistical flukes (Wikström, 2012; Wikström & Bouhana, 2017). A "risk factor" is a variable that correlates with an antisocial outcome, but it does not reveal anything more about the association, including the most crucial matter: whether it is causally related to the outcome or not. A cause will necessarily correlate with the outcome, but a correlation does not automatically imply causation. However, due to two logical fallacies, *post hoc, ergo propter hoc* ("after this, therefore because of this") and *cum hoc, ergo propter hoc* ("with this, therefore because of this"), there is a danger of mistaking a simple correlate for a cause as it is often the case in the (biosocial) literature (Levitin, 2018). Therefore, a risk factor approach has essentially led to hoarding an excessive quantity of correlates without actually determining whether they can cause crime which should be the main concern of any discipline which aims to inform public policy. Moreover, an exclusive focus on a statistical significance of an association between a predictor and an antisocial outcome is not a very informative approach. Namely, in large enough samples even the smallest relationships will be "statistically significant" despite a lack of any real-world importance. In many cases, such risk factors are not even particularly useful predictors as the effect size of their relationship with crime may be very small (Wikström, 2012). For instance, people with a history of head injuries may be more likely to commit crimes than people without such medical record, but still the majority of people who suffer brain injuries will not become criminals. In fact, in an attempt to avoid such overhyped claims and misuses of statistics, more than 800 academics have recently called for a complete abandonment of the concept of statistical significance (Amrhein, Greenland, & Mcshane, 2019).

If criminology does not have a clear understanding of how crime happens and what are the mechanisms of crime causation, it cannot advance nor develop effective prevention and intervention programmes. Designing such programmes based on a risk-factor approach would necessarily involve a lot of wasted resources as it is practically “a shot in the dark” because in the majority of the cases they will be tackling markers of antisocial behaviour that are not the source of crime and hence changing them will not generate the desired outcome. Favouring such an approach of treating symptoms instead of causes of crime would be equivalent to giving a patient with meningitis a paracetamol to treat the fever instead of antibiotics which would tackle the bacterial cause of the disease. Despite this, it is often recommended that reducing such risk factors should be an appropriate policy response. Not only could such advice prove unhelpful, but it could also lead to potentially harmful policy decisions.

On the other hand, one of the main arguments put forward by the proponents of risk-factor approach is that a variable should be valued based on its ability to predict crime, and for that purpose it should not matter whether it is a cause of crime or not (Glenn & Raine, 2014). Even though such argumentation provides a step forward in terms of acknowledgment that correlation does not imply causation, this approach is not particularly useful nor will it help criminology progress as a science. Even if by some unlikely chance we find a set of predictors which could very precisely predict crime occurrence, unless they are the causal factors, we would not be able to use such information to prevent crime which should be the overarching goal. The risk factor approach may predict crime, but it certainly cannot explain it. Moreover, taking this road may unintentionally lead to unforeseen adverse consequences, which are starting to show in algorithms that predict crime and recidivism (Benbouzid, 2019; Brennan, Dieterich, & Ehret, 2009; Hannah-Moffat, 2019; Perry, McInnis, Price, Smith, & Hollywood, 2013). Namely, with the advent of machine-learning, there has been an explosion in application of algorithms in various aspects of human life, including criminal justice system. These algorithms are produced using the risk-factor approach, that is, a machine is fed by historical crime data and then finds patterns related to crime based on correlations and regressions. A number of such predictive algorithms have been created for the purpose of objectively informing decision-making in policing and criminal justice system, such as during pretrial, determining parole, and sentencing. However, recently these algorithms have been a target of serious accusations regarding their effectiveness and fairness, with specific criticism of their criminalisation of poverty and racial minorities (Dressel & Farid, 2018; Fry, 2018; O'Neil, 2016). Many of these criminal risk assessment algorithms have been found to mistakenly label people from low-income and minority communities as having a high risk for reoffending, which is a consequence of the fact that being poor or black correlates with crime, but it certainly does not cause it. These inaccuracies led to a shared statement signed by more than 100 civil rights and community organizations expressing their concerns about the use of algorithms in pretrial risk assessments and their potential worsening of racial inequalities ("The use of pretrial", 2018).

There are many other examples of making wrong decisions in the criminal justice system based on a correlate that is not causally related to crime. For instance, in the United States, a 19-year old man was convicted of statutory rape after having a consensual sex with a 14-year old girl, for which sentencing guidelines recommended seven to 16 months in prison. However, he was sentenced to 18 months in prison as a risk assessment algorithm increased the upper limit to two years of incarceration. Further investigation showed that the algorithm used young age as a risk factor, and had he been 17 years older, paradoxically, the algorithm would have suggested no prison sentence at all (Fry, 2018). Therefore, given the questionable effectiveness of such algorithms and the seriousness of their role in influencing life-altering decisions, it is hard to argue that predicting crime based on mere correlates is as harmless as previously suggested.

As documented in the previous paragraphs, there is a strong need for a shift to the analytical approach that will specifically aim to explain why and how crime happens. As biosocial criminology has accumulated a vast knowledge about correlates of crime, the next step would be to tidy-up and distinguish which few amongst all variables have a potential to be causally related to crime. For each association, it should be determined whether there is a plausible causal process which would explain how a certain biological factor is linked to crime. If a variable has a potential to be a cause of crime, further research should be undertaken to investigate what is its specific role in the underlying mechanism that brings about crime and describe such process as precisely as possible. For example, having minor physical anomalies, such as low-seated ears (Glenn & Raine, 2014; Raine, 1993a, 2002), cannot cause anyone to commit a crime. Similarly, a surgery to correct low-seated ears would not prevent offending either. However, having a dysfunctional dorsolateral prefrontal cortex that causes poor executive functioning could be a potential cause of (the cause of) crime, as it could lead to difficulties in exercising self-control and acting in accordance with one's own morality in situations where there is an external environmental pressure to transgress. Therefore, intervention programs that are based on cognitive nurturing with a goal to establish and potentiate neural pathways and strengthen executive functions have the potential to be successful (Wikström, 2019; Wikström & Treiber, 2019).

The principal aim of the current study is to advocate for a change towards analytical perspective in biosocial criminology by setting an example. Work presented herein will take "the best-replicated biological correlate" of antisocial behaviour (Ortiz & Raine, 2004, p. 154), low heart rate, and critically examine its role in criminal behaviour. Given the supremacy of the correlational approach, it is not at all surprising that there is an abundance of research studies demonstrating the statistical association between low heart rate and antisocial behaviour, while the underlying causal mechanisms remain unknown. Although theoretical explanations of this association have been proposed, they lack rigorous empirical verification and still remain in the realm of speculation (Armstrong, 2011; Raine, 2002). The goal of the present study is to address this research gap and explore the underlying mechanism linking low heart rate and crime by employing the analytical approach and placing this biological marker within a theoretical context.

1.2. Review of the Link Between Low Heart Rate and Antisocial Behaviour

The objective of the current section is to provide an in-depth and up-to-date review of the empirical studies investigating the association between heart rate measured at rest (i.e., resting heart rate) and in response to a stressor (i.e., heart rate reactivity) and different measures of antisocial behaviour. It will be divided into two parts that will shed light onto studies supporting such a link, but also empirical findings refuting it. Finally, there will be an attempt to explain the inconsistencies of the research results.

1.2.1. Empirical Evidence Supporting the Antisocial Behaviour-Heart Rate Link

In the past few decades, a substantial body of research has accumulated demonstrating the negative association between heart rate and multiple types of antisocial behaviour, crime included. Low heart rate has been linked to aggression (Armstrong, Keller, Franklin, & Macmillan, 2009; Jennings, Pardini, & Matthews, 2017; Raine, Venables, & Mednick, 1997), including its reactive and proactive forms (Portnoy et al., 2014; Xu, Raine, Yu, & Krieg, 2014), delinquency (Choy et al., 2015; Gao, Huang, & Li, 2017; Loeber, Pardini, Stouthamer-Loeber, & Raine, 2007; Portnoy et al., 2014), arrest and conviction frequencies (Armstrong, Boisvert, Flores, Symonds, & Gangitano, 2017; Jennings, Piquero, & Farrington, 2013; Koegl, Farrington, & Raine, 2018; Raine, Venables, & Williams, 1990), inclusive of violent and non-violent offending (Choy, Raine, Venables, & Farrington, 2017; Latvala, Kuja-Halkola, Almqvist, Larsson, & Lichtenstein, 2015; Murray et al., 2016; Van de Weijer, de Jong, Bijleveld, Blokland, & Raine, 2017), and a number of specific antisocial behaviours such as lying (Dierckx et al., 2014), academic dishonesty (Portnoy, Legee, Raine, Choy, & Rudo-Hutt, 2019), stalking (Boisvert et al., 2017), and rule-breaking behaviours (Fagan, Zhang, & Gao, 2017; Sijtsema et al., 2010). Furthermore, low heart rate has been found to correlate with a range of personality features of interest to criminology, such as impulsivity (Bennett, Blissett, Carroll, & Ginty, 2014; Bibbey, Ginty, Brindle, Phillips, & Carroll, 2016; Choy et al., 2015; Mathias & Stanford, 2003; Schmidt, Mussel, & Hewig, 2013), callous-unemotional traits (Fagan et al., 2017; Gao et al., 2017; Kavish et al., 2017), psychopathy (Choy et al., 2015; Fanti et al., 2017; Raine, Fung, Portnoy, Choy, & Spring, 2014), and hostility (Jennings et al., 2017). Importantly, these findings have been replicated using different methods of assessing antisocial behaviour including self- and observer-reported measures (Armstrong et al., 2009; Fagan et al., 2017; Portnoy et al., 2014; Raine et al., 1997; Sijtsema et al., 2013), clinical diagnoses of antisocial personality disorder and various disruptive disorders (Fairchild et al., 2008; Popma et al., 2006; Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000; Van Goozen, Matths, Cohen-Kettenis, Buitelaar, & Van England, 2000; Van Goozen et al., 1998), and official criminal records data (Choy et al., 2017; Jennings et al., 2013; Murray et al., 2016; Van de Weijer et al., 2017).

The relationship between low heart rate and antisocial behaviour has been confirmed in different populations, including student, convict, delinquent, clinical, and community samples (De Vries-Bouw et al., 2011; Fagan et al., 2017; Fairchild et al., 2008; Koegl et al., 2018; Osumi, Shimazaki, Imai, Sugiura, & Ohira, 2007). It has also been shown to hold across the life span, from early childhood into adulthood (Armstrong et al., 2009; Jennings et al., 2017; Maliphant, Watkins, & Davies, 2003; Portnoy et al., 2014; Raine et al., 2014, 1997), and in both female and male subjects (Brzozowski, Gillespie, Dixon, & Mitchell, 2018; Jennings et al., 2013; Latvala et al., 2015; Sijtsema et al., 2013). Furthermore, this finding appears to be a cross-cultural phenomenon, as it has been reported in samples from more than a dozen countries across the continents, such as Mauritius, Brazil, China, Canada, and Sweden (Raine, 2015).

The replication across different research designs and involving a range of control variables have further added to the robustness of the evidence underpinning the relationship between low heart rate and antisocial behaviour. This result has been found to hold up after taking into an account a number of possible confounding variables, such as body mass index, smoking, alcohol intake, physical fitness, age, race, gender, socioeconomic status, cognitive ability, self-control, attachment to parents, and peer delinquency, amongst others (Armstrong et al., 2009; Cauffman et al., 2005; Jennings et al., 2013; Latvala et al., 2015). In addition to cross-sectional design, the association between heart rate and criminal offending has been confirmed in longitudinal studies as well. After controlling for the effects of relevant covariates, low resting heart rate measured throughout adolescence, from early puberty to late adolescence, has been associated with violent and non-violent crimes committed later in life, with follow-up periods from just a few years to up to more than three and a half decades (Choy et al., 2017; Latvala et al., 2015; Murray et al., 2016; Raine et al., 1990; Van de Weijer et al., 2017; Wadsworth, 1976). There are also some preliminary findings that resting heart rate measured in early childhood can predict later antisocial behaviour. Data from the Mauritius child health project (Raine et al., 1997) have shown that a low heart rate assessed at age as early as three years can predict parent-rated aggressive behaviour in children eight years later. The most profound contribution of this finding is that it eliminates the effects of many variables with a confounding influence that necessarily or potentially happen later in life, such as smoking, alcohol and/or drug use, athleticism, puberty, and antisocial lifestyle. In general, besides documenting the predictive power of heart rate, prospective studies are also significant because they discard the possibility that a criminal lifestyle could somehow cause low heart rate (Ortiz & Raine, 2004).

Furthermore, low heart rate has been put forward in criminological literature as diagnostically specific to antisocial behaviour (Ortiz & Raine, 2004; Raine, 2002). The reasoning behind this conclusion has been based on findings that children expressing antisocial and disruptive behaviours have lower heart rates compared to typically developing and psychiatric control samples (Dietrich et al., 2007; Fairchild et al., 2008; Maliphant et al., 2003; Popma et al., 2006), and that internalizing psychiatric disorders, such as depression and anxiety disorders, have been found to be, if anything, characterized by high cardiovascular activity (Dierckx et al., 2014; Monk et al., 2001; Schumann, Andrack, & Bär, 2017). This may seem as a very promising development, particularly in light of the fact that psychiatry has put much effort during the recent decades into finding biomarkers of mental disorders, alas without much success. Twenty years ago, the American Psychiatric Association task force for the latest edition of the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association, 2013) set out to include biological measures into diagnostic criteria for mental disorders, and yet their endeavours had not materialised (Kupfer, 2013). The underlying reason was not a lack of potential biomarkers for psychiatric disorders, but their lack of specificity, that is, the fact that the same biomarkers were related to a number of different psychiatric disorders (Venigalla, Mekala, Hassan, Ahmed, Zain, & Dar, 2017). Therefore, an indication of low resting heart rate potentially being a specific indicator featuring only in antisocial and disruptive disorders seemed as a very alluring finding. Indeed, the DSM-5 classified low heart rate among the risk and prognostic factors for conduct disorder, a childhood precursor of antisocial personality disorder, and stressed its diagnostic specificity. A valid and clinically useful biomarker should also be able to predict treatment response, and one study has provided some preliminary findings that this might be the case with low heart rate. Namely, the study examining the success of an intensive cognitive behavioural intervention aimed at reducing aggression and delinquency in a group of children with disruptive behaviour disorder has shown that low heart rate was the only significant predictor of therapy success among several other variables, including cognitive functioning and severity of disruptive symptoms before the intervention (Stadler et al., 2008). The results have clearly demonstrated that a group of children who did not respond to the therapy had a significantly lower resting heart rate. At the end, however, even though heart rate seemed to be the most replicated of all potential biomarkers for conduct problems, it was not included amongst the diagnostic criteria of the DSM-5. The supporting evidence was not strong enough and there was no clear cut-off point for what makes a "low heart rate" hence diminishing its clinical usefulness (Moffitt et al., 2008).

On the other hand, high heart rate has been proposed as a "buffering protective factor" which supposedly protects individuals from engaging in antisocial and criminal behaviour when they are otherwise exposed to "risk factors" (Adjorlolo, 2017; Portnoy, Chen, & Raine, 2013; Sijtsema et al., 2013). There has been some initial evidence providing support that high heart rate, measured either at rest or in response to a stimulus, is associated with lower antisocial behaviour. For example, a study employing a sample of adolescents recruited from residential welfare institutions has shown that participants with high resting heart rate have demonstrated the lowest score on a measure of antisocial behaviour (Lösel & Bender, 1997). Similarly, heart rate reactivity has been found to be heightened in a group of non-criminal men with criminal fathers in comparison to groups of criminal men with criminal fathers, criminal men with non-criminal fathers, and non-criminal men with non-criminal fathers (Brennan et al., 1997). Such findings have been interpreted in the literature as an evidence that high risk individuals may be protected from developing antisocial behaviours by their high heart rate (Adjorlolo, 2017; Wilson & Scarpa, 2013). However, this proposal is problematic as it does not include a mechanism by which a high heart rate could avert a person from exhibiting antisocial behaviour. The cited findings point to an association at best, and do not offer evidence of a causal link whereby increasing heart rate prevents a person from committing antisocial acts.

In a like manner, high heart rate has also been associated with desistance from criminal behaviour. A prospective study has shown that antisocial adolescents who desisted from adult crime had significantly higher resting heart rate than a group of antisocial adolescents who had become criminals by the age of 29 years (Raine, Venables, & Williams, 1995). However, subsequent studies have failed to replicate the finding that high heart rate measured either at rest or in response to stress predicts desistance from antisocial behaviour measured from early adolescence into adulthood (Baker et al., 2009; De Vries-Bouw et al., 2011; Hammerton et al., 2018; Loeber et al., 2007).

1.2.2. Empirical Evidence Refuting the Antisocial Behaviour-Heart Rate Link

Although a review of the literature points to a robust, well-replicated inverse association between heart rate and antisocial behaviour, there have been some reports of inconsistent results that should be acknowledged. Such findings may be broadly categorized into those showing no evidence of the association (e.g., Piquero, Ling, Raine, & Piquero, 2020) and those showing a positive relationship (e.g., Deutz et al., 2019). Studies that belong to the former group are usually associated with certain methodological characteristics such as having a small sample (e.g., Scarpa, Tanaka, & Haden, 2008), utilizing samples of young children or women (e.g., Wilson & Scarpa, 2013), or using psychopathy as a measure of antisocial behaviour (e.g., Kavish, Boisvert, et al., 2019). Studies belonging to the latter group often use clinical samples of children and adolescents (e.g., De Wied, Boxtel, Posthumus, Goudena, & Matthys, 2009).

As to the studies showing no relationship between heart rate and antisocial behaviour, some of them suffer from certain methodological issues, such as having a small sample size which may lead to non-significant results due to low statistical power, especially having in mind that such effects are usually of small magnitude (De Wied, Van Boxtel, Matthys, & Meeus, 2012; Scarpa, Haden, & Tanaka, 2010; Scarpa et al., 2008). Another commonality of a group of studies that have reported non-significant results is measuring antisocial behaviour in early childhood. It seems that heart rate measured at age as early as 14 months, and up to 36 months, does not predict aggressive and externalizing behaviour at ages three and seven years (Dierckx et al., 2014; Van Hulle, Corley, Zahn-Waxler, Kagan, & Hewitt, 2000). Similarly, a study looking into aggressive behaviour of four-year-olds has not found evidence of lower heart rate in aggressive children or in children diagnosed with oppositional defiant disorder or conduct disorder (Posthumus, Böcker, Raaijmakers, Van Engeland, & Matthys, 2009). One possibility is that the relationship between heart rate and antisocial behaviour does not show until preadolescent and adolescent period. However, since aggression is a developmentally common phenomenon in early childhood (Tremblay, 2015), it is also plausible that at this age the link between heart rate and antisocial behaviour cannot shine through because most pre-schoolers exhibit aggressive behaviour. Developmental research shows a trajectory of aggression which involves an increase of this behaviour during early childhood, followed by a decrease from the preschool age to adolescence (Côté, Vaillancourt, LeBlanc, Nagin, & Tremblay, 2006; NICHD Early Child Care Research, 2004). Therefore, it is possible that low heart rate characterizes only a minority of children who do not follow this trajectory and instead persist in aggressive behaviour. Results from the Mauritius child health project (Raine, Venables, et al., 1997) which have shown that heart rate measured at age three years predicted aggressive behaviour at age 11 provide preliminary support for the latter explanation, but further research is necessary to draw firm conclusions.

Another source of non-significant results stems from female samples. Several studies utilizing samples of preadolescent, adolescent, and adult females have failed to replicate the negative association between heart rate and a range of antisocial behaviours, including aggression, rule-breaking behaviour, and callous-unemotional traits (Boisvert et al., 2017; Fagan et al., 2017; Kavish et al., 2017; Sijtsema et al., 2010; Wilson & Scarpa, 2013).

However, two meta-analyses have concluded that this link can be found in female participants as well. A meta-analysis conducted on studies using samples of children and adolescents has found no significant difference in effect sizes for girls and boys (Ortiz & Raine, 2004). Similarly, a subsequent meta-analysis conducted on 115 independent effect sizes obtained from studies including all age groups has concluded that the link between resting heart rate and antisocial behaviour is found in both male and female samples, although the summary effect size calculated from male samples was higher (Portnoy & Farrington, 2015). Out of 18 studies conducted using only female samples, 13 had effect sizes in the expected, negative direction.

Finally, mixed findings have been reported when analysing the relationship between heart rate and psychopathy. Some studies have demonstrated a negative link between heart rate measured either at rest or in response to a stimuli, and psychopathic traits (Choy et al., 2015; Fanti et al., 2017; Gao, Raine, & Schug, 2012; Kyranides, Fanti, Sikki, & Patrick, 2017), whereas others found no evidence of a relationship between these variables (Kavish, Boisvert, et al., 2019; Kavish, Fu, Vaughn, Qian, & Boutwell, 2019; Portnoy et al., 2014). Explanation of inconsistent findings may lie in the fact that there is no clear consensus among researchers about what features psychopathy actually entails, which has led to parallel existence of many different measures of psychopathy that tap into similar but not identical constructs (Copestake, Gray, & Snowden, 2011; Lilienfeld, 1994; Patrick, Fowles, & Krueger, 2009; Salekin, Chen, Sellbom, Lester, & MacDougall, 2014; Williams, Paulhus, & Hare, 2007; Wright, 2009). Particularly, there is a disagreement whether antisocial behaviour represents an inherent part of psychopathy or it is just a consequence of psychopathic personality traits (Cooke, Michie, Hart, & Clark, 2004; Hare & Neumann, 2005, 2010; Skeem & Cooke, 2010). This has propelled some authors to suggest that divergent results could be explained by taking into an account if a measure of psychopathy used in a study included items describing overt antisocial behaviour or not (Kavish, Boisvert, et al., 2019; Kavish, Fu, et al., 2019). They have suggested that the link between psychopathy and heart rate could be explained by inclusion of antisocial behaviour, and once a measure included only personality features of psychopathy, the relationship would disappear. However, although there are some studies confirming this conclusion (Hammerton et al., 2018; Ishikawa, Raine, Lencz, Bihrle, & Lacasse, 2001; Kavish, Boisvert, et al., 2019; Kavish, Fu, et al., 2019), there is also a significant number of studies which have reported a negative link between heart rate and the affective or non-behavioural features of psychopathy (Fagan et al., 2017; Gao et al., 2017; Kavish et al., 2017). Therefore, this explanation does not seem to be fully supported by the empirical data.

Another methodological challenge in psychopathy research is reflected in discrepancies regarding how different studies define psychopathy even if it is measured using the same instrument. Psychopathy Checklist-Revised (Hare, 2003) is one of the most commonly used measures of psychopathy which yields a score on a scale between zero and 40, with a suggested cut-off point of 30 or greater to indicate psychopathy. However, despite the instructions in the instrument manual, different researchers have used different cut-off points to define psychopathy in their studies, often decreasing the critical threshold to mid-20s or lower (Koenigs, 2012). Therefore, the inconsistencies in the results regarding the link between psychopathic traits and heart rate are possibly a consequence of methodological issues characterising the sub-field of psychopathy research.

The most conclusive answer to the question whether there is a relationship between heart rate and psychopathy could be obtained by conducting a meta-analysis. Thus far, two meta-analyses have been reported with conflicting results. Lorber (2004) has concluded there was no evidence of the link between resting heart rate and psychopathy, whereas Portnoy and Farrington (2015) have found a significant negative link with the effect size of $d = -0.19$, $p < .01$. However, Lorber's (2004) meta-analysis suffers from certain methodological problems which could have potentially led to biased conclusions and underestimation of the summary effect size. Firstly, it has included 17 effect sizes which were all calculated from small-scale studies where the number of participants ranged from 20 to 64 per study. Secondly, the bulk of the studies compared psychopaths vs non-psychopaths, with only a few studies looking at the link between continuous variables. Finally, seven studies included into the meta-analysis have been coded as having no effect whatsoever ($d = 0$), despite the fact that these articles had not provided enough details to actually calculate their exact effect sizes. The authors of these studies had just reported a "non-significant" result that Lorber translated into an effect size of zero which is not the same (i.e., an effect size could be higher than zero but still non-significant). On the other hand, Portnoy and Farrington's (2015) meta-analysis has not incorporated the seven questionable non-significant studies into the analysis, and hence their results might have overestimated the strength of the relationship. Therefore, it seems that conclusive results regarding the link between heart rate and psychopathy are not yet available. A potential way forward could lie in conducting a new meta-analysis that would include novel research findings, address the methodological issues in question, and include a measure of psychopathy as a moderating variable.

Besides some studies showing no association between heart rate and antisocial behaviour, findings about positive links represent another type of inconsistent data. Out of the 115 independent effect sizes included in a meta-analysis exploring the link between resting heart rate and antisocial behaviour, 34 were in a positive direction with seven studies having a statistically significant result (Portnoy & Farrington, 2015). However, these findings could be explained by taking a closer look into the sample type and outcome variables used in these studies. A significant number of the studies showing a positive association either included in the above mentioned meta-analysis or conducted at a later date (De Wied et al., 2009; Deutz et al., 2019; Schoorl, Rijn, Wied, Goozen, & Swaab, 2016; Zahn & Kruesi, 1993), have used clinical samples of children diagnosed with disruptive behaviour disorders, or have used such symptoms as an outcome measure of antisocial behaviour. However, children with externalizing problems are far from being a homogenous group. They often suffer from comorbid internalizing problems which represents more of a rule rather than an exception, with a significant number of children diagnosed with disruptive behaviour disorder showing a pattern of high anxiety, impulsivity, and emotional dysregulation (De Wied et al., 2009; Deutz et al., 2019; Dierckx et al., 2014). As internalizing problems, anxiety in particular, have been found to be associated with high heart rate, it is plausible that they represent confounding factor which is the actual driving force of the positive link between heart rate and antisocial behaviour in such samples. Indeed, resting heart rate has been shown to increase with dysregulation, a common factor among externalizing and internalizing symptoms comprised of anxiety, depression, aggression, and attention problems (Deutz et al., 2019). There are also other potentially confounding variables which could perhaps explain the findings of a positive association between heart rate and antisocial behaviour. Specifically, these children are often taking medications and suffer from other comorbid diagnoses associated with high resting heart rate, such as autism spectrum disorder (Bal et al., 2010; Ming, Julu, Brimacombe, Connor, & Daniels, 2005).

Finally, another reason closely related to heterogeneity of children with disruptive behaviours, but that could also explain some of the findings regarding the positive association between heart rate and antisocial behaviour in other samples as well, might lie in the type of aggression measured in such studies. Namely, there are two distinct types of aggression that are suggested to be associated with different physiological features: reactive aggression, a defensive response to a threatening or frustrating incident associated with strong negative emotions, and proactive aggression, an instrumental aggression motivated by an anticipation of reward in absence of strong emotional arousal (Scarpa et al., 2010; Stadler, Poustka, &

Sterzer, 2010). It has been proposed that physiological underarousal is associated with proactive aggression, whereas reactive aggression is characterized by physiological overarousal (Scarpa & Raine, 2000). Indeed, there has been some preliminary evidence supporting this conclusion wherein low and high heart rates were related to proactive and reactive aggression, respectively (Scarpa et al., 2010; Schoorl et al., 2016). Another study has shown that in a sample of more than 300 school children resting heart rate was inversely associated with the both types of aggressive behaviour (Raine et al., 2014). However, once the proactive aggression was controlled for, reactive aggression ceased to be significantly associated with heart rate, whereas the negative association with proactive aggression remained significant even after accounting for the effect of reactive aggression. Therefore, a positive association between heart rate and antisocial behaviour can be explained in the many cases by the use of clinical samples in which it is hard to disentangle the effects of confounding factors, or by measuring antisocial behaviour through reactive aggression.

1.3. Is There a Relationship Between Low Heart Rate and Antisocial Behaviour?

Overall, the review of the literature seems to suggest a robust and well-replicated association between low heart rate and a range of antisocial behaviours, but some inconsistent findings have been reported as well. Therefore, a meta-analysis should provide the most reliable answer to the questions of the strength of the relationship, homogeneity of its effect sizes, and the influence of possible moderating variables.

Thus far there have been several meta-analyses looking at the relationship between heart rate and various antisocial behaviours. The first two meta-analyses have examined studies published up until 2002, with one looking into the link between heart rate measured at rest, in response to a task, and as heart rate reactivity (as a function of change from resting state), and aggression, psychopathy, and conduct problems (Lorber, 2004), whereas the other publication has examined heart rate measured at rest or in response to stress, and antisocial behaviour in samples of children and adolescents (Ortiz & Raine, 2004). Lorber (2004) has concluded that both aggression and conduct problems were significantly related to low resting heart rate with the effect sizes of d = - 0.38 (across 16 studies) and d = - 0.33 (across 13 studies), respectively. Results regarding heart rate measured in response to a task have suggested a different conclusion. Neither aggression nor conduct problems have been found to be significantly related to task heart rate across 14 and eight studies, respectively.

However, the analyses have not differentiated between types of tasks, and therefore stressful, neutral, and orienting tasks have been merged into the same category, which led to substantial heterogeneity of the included effect sizes. Unsurprisingly, valence of a task has emerged as a significant moderating variable in the case of aggression, and a significant negative effect has emerged for the studies that used aversive tasks. This meta-analysis suffers from other limitations as well, such as excluding longitudinal studies, not considering general antisocial behaviour, and coding studies which had reported non-significant results without including a precise effect size as having an effect size of zero.

Next, Ortiz and Raine's (2004) publication has included results from two meta-analyses of effect sizes retrieved from studies with samples of participants younger than 19 years which focused on antisocial behaviour on the whole, bringing together conducts such as aggression, delinquency, violence, and externalizing disorders. The primary meta-analysis has focused only on resting heart rate, whereas the secondary meta-analysis has explored heart rate measured in response to a stressor. Using 45 independent effect sizes from a dataset of 5868 participants, the former meta-analysis has yielded a significant overall effect size of $d = - 0.44$, $p < .001$. In total, 43 effect sizes were negative, whereas there were only two positive effect sizes, both statistically non-significant ($p > .05$). The latter meta-analysis has been on a smaller scale involving less than 600 participants and 10 effect sizes, all of which were negative. Its result has pointed to a strong negative effect of $d = -0.76$, $p < .001$. Furthermore, the relationship between heart rate and general antisocial behaviour seems robust as none of the moderating variables has significantly influenced the effect size, including sex, age, source of participants' recruitment, and the study design (despite an average nine years of follow-up in prospective studies).

Finally, the most recent meta-analysis of the association between resting heart rate and antisocial behaviour has attempted to address the shortcomings of the prior meta-analyses (Portnoy & Farrington, 2015). It included both published and unpublished studies up to 2012, yielding 115 independent effect sizes in total. The resulting overall effect size ranged from $d = - 0.15$, $p < .01$ to $d = - 0.20$, $p < .01$, depending on the type of the model used to calculate these values. Authors have concluded that the relationship was well-replicated and robust, and that it held across age, sex, types of antisocial behaviour, different recruitment sources, and study designs. However, it is striking that the overall effect size is much weaker compared to the ones reported in prior meta-analyses. Furthermore, Ortiz and Raine (2004) reported that out of all the studies included in their analysis, 96% had negative effect sizes, whereas only 70% of the effect sizes in the latest meta-analysis were in the expected direction, with 31% having a statistically significant effect. Portnoy and Farrington (2015) have suggested that this difference could be potentially explained by the “Proteus phenomenon” (Ioannidis & Trikalinos, 2005), which acknowledges a trend wherein once a new observation has been noted, significant findings are more likely to be published, and when they accumulate in a considerable number, the trend reverses, and non-significant or refuting findings become more appealing for publication. Finding of a significant negative association between the year of publication and the strength of its effect size has been put forward as supportive of this explanation. However, the authors have also concluded that the publication bias is the unlikely cause of their overall findings, and therefore there may be other reasons why there have been more non-significant findings in later years, including employing better and more rigorous methodology.

A meticulous review of studies examining the relationship between heart rate and antisocial behaviour reveals that there may be another, overlooked reason that could potentially explain the inconsistent findings. It has been noted that there are discrepancies in the way resting heart rate has been measured across the studies, such that it is not always clear whether a “resting heart rate” had been indeed measured at rest, or in response to a stressor. A number of studies reporting a significant inverse relationship involving resting heart rate have used measurement procedures that participants may have actually experienced as stressful. Several studies using samples of children and adolescents have been conducted in laboratory rooms and/or using equipment that may have been perceived as invasive, such as electrodes, especially if they have been placed below a child’s chest (Raine et al., 1990, 1997; Xu et al., 2014). For example, one of these studies included crying of its three-year-old participants as a control variable (Raine, Venables, et al., 1997). A technician was assessing how much a child cried during the heart rate measurement on a scale from one, whereby the child did not cry, to five, which meant uncontrollable crying. Therefore, it is safe to assume that at least some of the children in the study experienced this assessment as stressful, which means that their heart rate was actually measured in response to a stressor, not at rest. Next, it has been recommended to measure heart rate for at least a full minute in order to obtain an accurate baseline measurement (Cornet, 2015; Kobayashi, 2013; Pickering, 2013; Smith, Duell, & Martin, 2003). A reading resulting from a shorter measurement may be imprecise due to potential changes in the heart rhythm, with a possible error being further amplified by multiplication with an integer (e.g., measuring heart rate for 30 seconds and multiplying it by two to obtain a number of beats per minute). However, many studies have failed to adhere to these basic recommendations (Armstrong & Boutwell, 2012; Armstrong et al., 2009; Raine et al., 2014). Furthermore, several studies have used measures obtained from medical examination files, and such assessments could be experienced as stressful and anxiety-inducing by some people (Koegl et al., 2018; Latvala et al., 2015; Van de Weijer et al., 2017). This is additionally complicated in the cases where the medical examination has been conducted as part of a mandatory army conscription procedure, as there could be other reasons why the resulting heart rate measurement may not represent a valid baseline measure. For example, such examination could be perceived as stressful by some people as it determines whether they would be sent to serve in a military, or some conscripts may even temporarily tamper with their health measures to be freed of the service. It is also questionable how precise are these readings given that they are measured in a high number of people over very short periods of time. One study has noted that sometimes physicians would measure a heart rate for only 15 seconds and then multiply it by four to get a number of beats per minute, which would in that case represent only an approximation of heart rate (Van de Weijer et al., 2017).

Furthermore, a bulk of the studies showing a negative association with antisocial behaviour have measured resting heart rate while participants sat quietly and were instructed to relax (e.g., Brzozowski et al., 2018; Jennings et al., 2013; Xu et al., 2014). However, the main problem with this procedure is that it cannot ensure a standardised experience for all participants. Some people may be bored or sleepy, some may feel anxious in anticipation what will happen next or just by being in an unfamiliar situation, while others may be thinking of something else that could incite various emotions, all of which can influence their heart rate in different ways. Almost 20 years ago, it was recommended in psychophysiological literature that a resting measure of heart rate should be obtained while the participants are engaged in a minimally demanding task such as watching a relaxing video, as it had been proven better at achieving cardiovascular baseline in comparison to resting quietly (Piferi, Kline, Younger, & Lawler, 2000). Interestingly, often studies that have followed this advice and measured resting heart rate while the participants were watching the recommended relaxing video (Coral Sea Dreaming, Small World Music Inc.) have found no associations with antisocial behaviour measures (Colasante & Malti, 2017; Oldenhof et al., 2019; Prätzlich et al., 2019; Van Zonneveld, Platje, De Sonneville, Van Goozen, & Swaab, 2017; Wilson & Scarpa, 2013, 2014). Moreover, studies that have used similar minimally demanding tasks, such as watching other types of relaxing videos, reading a magazine, or completing questionnaires, or that have otherwise put more effort into obtaining a more valid baseline measure of heart rate, such as conducting research at a participant's home or measuring heart rate over a longer period, have also found no associations with different antisocial behaviours (Bimmel, Van Ijzendoorn, Bakermans-Kranenburg, Juffer, & De Geus, 2008; De Vries-Bouw et al., 2011; Galán, Choe, Forbes, & Shaw, 2017; Posthumus et al., 2009). Some of these studies have also included a measure of heart rate during a stressor, and, despite a lack of association with resting heart rate, antisocial behaviours have shown a significant inverse link with the heart rate reactivity (Bimmel et al., 2008; De Vries-Bouw et al., 2011; Van Zonneveld et al., 2017). Having in mind these results, it is possible that the relationship between low heart rate and antisocial behaviour does not apply to resting heart rate, but only to heart rate reactivity. This hypothesis can be tested by conducting an updated meta-analysis which would include the method of obtaining baseline cardiovascular activity as a moderating variable. However, it is crucial that future studies start employing standardised protocols for measuring resting heart rate and heart rate reactivity. Heart rate at rest and cardiac response to stress are two distinct phenomena with different underlying mechanisms and should not be therefore conflated in research if we are to better understand their relationship with antisocial behaviour.

Chapter 2:

How is Low Heart Rate Associated with Antisocial Behaviour? Review of Proposed Explanations

2.1. Review of Proposed Mechanisms Linking Heart Rate and Antisocial Behaviour

Despite the overwhelming number of empirical studies showing a robust, inverse association between heart rate and criminal behaviour, little is known beyond the mere existence of the link due to the predominance of the correlational research paradigm. If there is indeed such a relationship, it is crucial to understand how, that is, through which mechanism, are these variables associated with one another. Although different explanations of the underlying mechanism have been proposed, these hypotheses are mostly underdeveloped, unsubstantiated, based on questionable assumptions, and lack rigorous empirical verification. This chapter aims to list, summarise, and scrutinise existing theoretical explanations provided by different authors across the criminological literature.

2.1.1. Low Arousal and Stimulation-Seeking

A predecessor of the stimulation-seeking hypothesis is the low arousal theory derived from Eysenck's theory of personality and its relation to criminality (Eysenck, 1977, 1997). Eysenck has proposed that certain personality characteristics are more likely to be associated with criminal conduct through their associated biological features. Specifically, he argued that individuals characterised by high extraversion and/or psychoticism are more likely to be engaged in antisocial behaviour as they have low arousal that may predispose them towards criminal behaviour. He has further suggested that low arousal may lead to antisocial behaviour by disrupting orderly development of one's conscience. It has been hypothesised that conscience is a conditioned response learnt through classical conditioning wherein arousal plays an important role. Therefore, his explanation is that low arousal promotes antisocial and criminal behaviour via lack of conscience caused by poor conditioning. Eysenck has also suggested an alternative mechanism through which low arousal may be associated with crime. Namely, he has suggested that individuals with low arousal may seek ways of increasing their arousal, such as engaging in risk-taking and impulsive actions, socializing with too many people, drug abuse, and promiscuous behaviour. The assumption is that these behaviours also increase chances of committing crime, although not necessarily leading to it.

Low arousal theory seems to be underdeveloped due to many vague and/or unfounded assumptions, such as the lack of specific explanation of how low arousal disrupts classical conditioning, why development of “conscience” would be contingent only on classical conditioning, and how the suggested arousal-increasing activities (such as being promiscuous or socializing with many people) bring about crime. In addition, the mechanism involving behaviours aimed at increasing arousal disregards the fact that a risky lifestyle will not have an equal influence on all people, but rather its effect will depend on their personal crime propensities (Svensson & Pauwels, 2010). Another important issue relates to a lack of clear definition of arousal and what measures of the central and/or autonomic nervous system functioning it entails. Arousal has been measured in the literature through very different means including resting electroencephalography, skin conductance, heart rate, and skin potential levels. However, research shows that there are no relations between these different measures or, if found, these inter-correlations are very low (Raine, 2002; Venables, 1987). Therefore, the low arousal theory does not appear to be able to provide a viable explanation of the relationship between heart rate and crime, as it lacks clarity and precision in defining its core concept and the specific mechanisms though which it brings about crime. Although the low arousal theory has been mostly abandoned in its original form, its derivate, a stimulation-seeking hypothesis, is currently one of the dominating theoretical explanations.

According to the stimulation-seeking hypothesis, low level of arousal is experienced as an unpleasant physiological state (Eysenck, 1997; Quay, 1965; Raine et al., 1997). In order to address this problem and increase their arousal to a more optimal level, individuals characterised by low heart rate engage in stimulation-seeking behaviours, which may also include antisocial and criminal acts. Although very popular in the literature, this explanation is based on highly questionable assumptions. For example, an assumption that low heart rate is experienced as unpleasant is unfounded. There is no reason to believe that this might be the case, and if anything, a high heart rate is more likely to be experienced as discomforting. Another highly problematic core assumption is that antisocial behaviour is considered arousing and that it leads to a more comfortable physiological state.

Apart from sketchy assumptions, it is also left unexplained how low arousal drives people to engage in stimulating behaviours. Research has indeed found some support for the positive association between stimulation-seeking personality traits and aggressive and deviant behaviours (Hammerton et al., 2018; Raine, Reynolds, Venables, Mednick, & Farrington, 1998; Vazsonyi & Ksinan, 2017; Wilson & Scarpa, 2014). However, a meta-analysis performed on 43 effect sizes from studies exploring the association of stimulation-seeking and aggression has reported a significant, but small summary effect size ($d = 0.19$, $p < .001$). In addition, the relationship varied significantly across different methodological moderating variables (Wilson & Scarpa, 2011). On the other hand, there have been mixed results with regards to the link between stimulation-seeking and resting heart rate or heart rate reactivity. A few studies have indeed reported a negative association (Hammerton et al., 2018; Sijtsema et al., 2010), but a number of empirical data have failed to detect any significant link between the two (Armstrong & Boutwell, 2012; Breivik, Roth, & Jørgensen, 1998; Kavish, Boisvert, et al., 2019; Portnoy et al., 2014; Wilson & Scarpa, 2013, 2014).

In addition, an overall interpretation of the stimulation-seeking research results has been complicated by the fact that this construct has been often conflated with impulsivity, even though they are independent constructs (Burt et al., 2014; Harden & Tucker-Drob, 2011; Magid, MacLean, & Colder, 2007; Steinberg et al., 2008; Vazsonyi & Ksinan, 2017). Stimulation-seeking has been defined as a personality trait characterised by "seeking of varied, novel, complex, and intense sensations and experiences, and the willingness to take physical, social, legal, and financial risks for the sake of such experience" (Zuckerman, 1994, p. 27). It has been further stressed that taking risks is related to stimulation-seeking, but it is not a core component of the definition. That is to say, stimulation-seeking individuals are prepared to take risks in order to experience wanted sensations, but do not seek risk for its own sake. On the other hand, a concept of impulsivity applies to a deficient self-control or response inhibition that prompts hurried, careless, and unplanned behaviour (Steinberg et al., 2008). As any other behaviour, stimulation-seeking can be carried out impulsively, but certainly not all stimulating behaviours are conducted in such manner. In fact, engaging in stimulating activities can be premediated and planned in advance (e.g., organising an outdoor rock climbing trip). Similarly, not all impulsive behaviour will have a stimulating effect (e.g., ending a friendship abruptly). Therefore, even though these traits may come together in a person, they are in fact distinct and independent factors (Burt et al., 2014). Furthermore, these constructs follow different developmental trajectories and are supported by distinct neural systems (Harden & Tucker-Drob, 2011; Steinberg et al., 2008; Vazsonyi & Ksinan, 2017). Impulsivity is closely associated with the cognitive control system underpinned by the prefrontal cortex functioning and connecting areas of the anterior cingulate cortex (Steinberg, 2007; Steinberg et al., 2008). It tends to decline linearly throughout adolescence and then stabilise in early '20s, which is consistent with findings about developmental trajectory of the prefrontal cortex which matures into early adulthood (Harden & Tucker-Drob, 2011; Steinberg et al., 2008; Vazsonyi & Ksinan, 2017). On the other hand, stimulation-seeking has been associated with the socio-emotional system underpinned by the limbic and paralimbic areas of the brain (Somerville, Jones, & Casey, 2010; Steinberg, 2007; Steinberg et al., 2008). It develops along a curvilinear pattern associated with puberty as it increases until mid-adolescence, after which it remains at the same level or declines (Harden & Tucker-Drob, 2011; Steinberg et al., 2008; Vazsonyi & Ksinan, 2017). Its trajectory is in line with neuroscientific findings showing that subcortical regions associated with motivational, affective, and rewarding stimuli are more responsive at this age in comparison to both children and adult samples (Harden & Tucker-Drob, 2011; Somerville et al., 2010; Steinberg, 2007; Steinberg et al., 2008).

Theoretical conflation of stimulation-seeking and impulsivity has in turn affected operationalisation of these constructs. In many cases, these two personality traits have been either deliberately combined into a single construct (e.g., impulsive sensation-seeking subscale of the Zuckerman-Kuhlman Personality Questionnaire; Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993), or have been intended as a measure of one of the two constructs, but have instead included items tapping into the other construct as well. For example, one of the most widely used measures of stimulation-seeking, Zuckerman's Sensation Seeking Scale-V (Zuckerman, 1994), consists of four subscales, one of which is disinhibition which assesses impulsivity. Similarly, many measures of impulsivity also involve stimulation-seeking items. Exploratory factor analysis conducted on several widely used measures of impulsivity has yielded four factors, one of which was stimulation-seeking (Whiteside & Lynam, 2001).

Therefore, although the stimulation-seeking hypothesis has been increasingly recognised as one of the main explanations of the heart rate – antisocial behaviour link, it does not seem to withstand a meticulous scrutiny. It is based on flimsy and unsupported assumptions and does not appear to offer a viable underlying mechanism connecting heart rate to crime. In addition, interpreting most of the research findings related to stimulation-seeking has been further complicated by its conflation with impulsivity. Therefore, the studies showing the link with antisocial behaviour or heart rate often cannot distinguish between the effects of stimulation-seeking and impulsivity or disinhibition.

2.1.2. Fearlessness

According to the fearlessness hypothesis, low heart rate represents an indicator of low fear (Raine, 1996b; Raine, 1993b). It is assumed that the testing situation in which people's heart rates are being measured are at least mildly stressful. For example, they are often part of a medical examination or conscription testing, they are conducted in unfamiliar settings, or precede unknown experimental tasks, all of which can unintentionally induce anticipatory anxiety. Research from the 1980s about decorated bomb disposal experts and paratrooper veterans has been cited in support of the idea that low heart rate is a marker of fearlessness, as it has shown that these individuals who presumably lack fear are characterised by low heart rates (Cox, Hallam, O'Connor, & Rachman, 1983; McMillan & Rachman, 1987; O'Connor, Hallam, & Rachman, 1985). It is further assumed that fearlessness trait predisposes people to committing crimes as such behaviour requires a lack of fear of the adverse consequences, either in terms of getting hurt or having to face legal consequences (Raine, 1996b; Raine, 1993b; Raine & Liu, 1998). The other proposed mechanism linking fearlessness to crime suggests that a lack of fear adversely affects socialization and development of conscience via poor fear conditioning, which in turn facilitates antisocial behaviour. It is believed that if a child does not fear punishment, this would interfere with the socialization process.

Studies exploring the link between fearlessness and crime have not provided much support for it. For example, data from the Pittsburgh youth study has demonstrated that fear is indeed positively associated with reactive aggression, but there were no significant links with proactive aggression, and violent and non-violent delinquency (Portnoy et al., 2014). Similarly, Mauritius child health project results have shown that, although there was initally a correlation between a behavioural measure of fearlessness at age three and parent-rated measure of aggression at age 11, it was abolished after entering covariates (Raine et al., 1998). Similarly, the idea that resting heart rate is inversely associated with fearlessness has not been supported by data (Portnoy et al., 2019, 2014). In fact, it is important to point out that the very empirical studies about the decorated bomb disposal experts and paratrooper veterans that have been used to inspire and support the fearlessness hypothesis have not been interpreted accurately. Firstly, the results of the statistical analyses have shown that these presumably fearless individuals maintained lower heart rate *reactivity* to stress compared to the control group, but there were no significant differences in *resting* heart rates between the groups (Cox et al., 1983; McMillan & Rachman, 1987; O'Connor et al., 1985). Secondly, the decorated bomb disposal experts have not differed significantly from their non-decorated counterparts on reports of subjective feelings, that is, their subjective reactions to the testing situation have been the same, which is not in line with the fearlessness hypothesis (Cox et al., 1983; O'Connor et al., 1985). Therefore, the cited studies have not found that supposedly fearless individuals actually experience lower levels of fear, but only that their visceral reactions to stress, that is, heart rate responses, were lower. Interpreting these studies as supportive of the fearlessness hypothesis fails to acknowledge the crucial difference between the threat response and conscious experience of fear. These reactions stem from separate neurobiological systems, as the brain network that identifies and responds to threat is different from the system that enables subjective feelings of fear (Hoppenbrouwers, Bulten, & Brazil, 2016; LeDoux, 2013, 2014). Therefore, although the threat response and feeling afraid often go together, they are indeed independent. When built-in bodily threat reactions are put in motion, that does not necessarily mean they also result in feeling afraid.

Similarly to the previous explanations, the fearlessness hypothesis is also founded on questionable assumptions regarding its causal effect on committing crime. The claim that antisocial behaviour requires a lack of fear has not been backed by any evidence, and certainly there are situations where people do feel afraid but commit crimes nevertheless. Furthermore, the explanation is fundamentally probabilistic, as it postulates that those who are more fearful are less likely to engage in antisocial and criminal behaviours. It does not provide a robust mechanism of how fearlessness causes crime, nor it accounts for the individual differences between people in their propensity to commit crimes. More specifically, this reasoning does not consider the fact that not all people are equally motivated and willing to commit crimes. In effect, the deterrent quality of fear is relevant just for a minority of people who are motivated and actively deliberate committing crimes (Wikström, 2008, 2014; Wikström, Tseloni, & Karlis, 2011). The fearlessness hypothesis does not account for this, but rather implies that the deterrence is equally relevant for everyone which is not the case. For example, in many instances when people need more money, the majority will not steal not because they will actively decide to restrain themselves from committing theft due to their fear of consequences, but because it will not even cross their minds as an option. This is not to say that fear will never be a factor in crime causation, as there are indeed instances when individuals who consider committing a crime decide after some deliberation not to follow through due to the deterrent effects of fear. However, for the majority of the people fear will not be a relevant factor as they will not even consider committing crime, and this will also be true in cases of persistent offenders who habitually commit crime in criminogenic settings without putting much thought into it. Therefore, the presumption that people abide by the law due to their fear of consequences oversimplifies complex motivation behind human behaviour, and does not apply to the majority of people for most of the time.

Furthermore, it is assumed that a lack of fear in childhood leads to poor socialization and deficient conscience (Raine, 1993b, 1996). However, there are two main issues with this argument. Firstly, it is not clear from the provided explanation whether this disruption is caused by poor fear conditioning (as mentioned in e.g., Raine, 2002), which is a form of Pavlovian classical conditioning, or a low fear of punishment (as noted in e.g., Raine, 1996a), which is associated with a type of operant conditioning, alongside positive and negative reinforcement (LeDoux, 2014; Skinner, 1965). These are two very different types of learning and cannot be used interchangeably. Secondly, even if a child has poor fear conditioning or does not react to punishment, this will not necessarily lead to inadequate socialization as there are many other processes of learning, such as different types of conditioning, reinforcement techniques, observational learning, trial and error, and following instructions (Domjan, 2015). Therefore, the idea that low fear leads to antisocial behaviour through disrupted socialization is imprecise and dubious.

In sum, the fearlessness hypothesis is characterised by certain inconsistencies and unsupported core assumptions which diminish its usefulness in explaining the link between low heart rate and crime. Moreover, it is not supported by data either, including the empirical studies whose results it has been originally based upon.

2.1.3. Noradrenaline

Raine (1993b, 2002) has suggested that reduced noradrenergic functioning may explain the link between low heart rate and crime. The rationale behind such proposal is that, on one hand, noradrenaline is associated with heart rate, and on the other, it shows a negative link with antisocial behaviour. In support of this hypothesis, a meta-analysis and two empirical studies have been provided as evidence (Raine, 2002).

The meta-analysis involving 32 effect sizes aimed to compare levels of noradrenaline in antisocial populations against that in control groups (Raine, 1993b). However, the results showed that the overall effect size for noradrenaline was non-significant, that is, on average the antisocial groups did not differ from the comparison groups on levels of noradrenaline. It was only when the analysis was restricted to studies measuring noradrenaline from cerebrospinal fluid that a significant negative effect emerged (d = - 0.41, $p < .05$). Furthermore, when the analysis was limited only to studies measuring noradrenaline from urine samples, a significant positive effect size was reported (d = 0.19, $p < .05$). Therefore, the results of the cited meta-analysis are far from conclusive and do not lend the necessary support for the claim that antisocial populations have reduced levels of noradrenaline.

The hypothesis was also backed by two other studies (Rogeness, Javors, Mass, & Macedo, 1990; Rogeness, Cepeda, Macedo, Fisher, & Harris, 1990) which were reported to have "found both reduced heart rate and reduced noradrenaline in conduct disordered children" (Raine, 2002, p. 420). However, the first study (Rogeness et al., 1990a) only compared age, heart rate, systolic, and diastolic blood pressure between two groups of children and adolescents admitted to a psychiatric hospital, those diagnosed with conduct disorder and those diagnosed with other disorders (such as depression, anxiety, and psychosis). Although heart rate was labelled as resting, it was actually measured on the day when the children were admitted to the hospital which was most likely a very stressful event for them. Therefore, it is questionable whether this measure represents a resting heart rate, or rather a heart rate reactivity to stress. Nevertheless, the results showed that children with conduct disorder indeed had lower heart rate in comparison to the other psychiatric patients, but noradrenaline level was not measured, and therefore this study cannot be taken as an evidence to support the hypothesis in question.

The second study (Rogeness et al., 1990b) used a sample of boys admitted to the psychiatric hospital who were split into groups with high and low dopamine-β-hydroxylase (DβH) activity, which is an enzyme responsible for converting dopamine into noradrenaline. The groups were then compared with regards to psychiatric diagnoses, symptoms, and catecholamine levels. The results showed that indeed the low DβH group had more diagnosis of under-socialised conduct disorder and lower levels of several noradrenaline metabolites. However, this study cannot be taken as a proof in favour of the noradrenaline hypothesis. Firstly, the study did not include a measure of heart rate. Secondly, the ratios which were utilised as a proxy for noradrenaline function had not been previously verified as valid measures. Thirdly, the study did not test for an association between noradrenaline and symptoms of conduct disorder irrespective of the levels of DβH activity. Therefore, the evidence presented as supportive of the noradrenaline hypothesis does not seem to hold up after scrutiny. Moreover, Raine did not offer a mechanism that would explain how reduced noradrenaline accounts for the link between low heart rate and crime. Overall, the noradrenaline hypothesis seems to have little to offer to the explanation of the heart rate-crime relationship in its current version.

2.1.4. Right Hemisphere Functioning

It has been proposed that the right hemisphere functioning may underlie both low heart rate and antisocial behaviour (Ortiz & Raine, 2004; Raine, 2002). The right hemisphere has been reported as dominant in regulating autonomic functions, along with findings of low heart rate accompanying reduced right hemisphere functioning. On the other hand, it has also been argued that poor functioning of the right hemisphere had been found in antisocial and violent individuals. The underlying mechanism connecting such brain deficits with criminal behaviour has been based on the approach-withdrawal model of the affect lateralization (Davidson, 1984, 1987, 1992). According to the model, the left anterior brain regions are associated with positive emotions and approach-related motivation, whereas the right anterior functioning is linked to the aversive emotions and withdrawal motivation. Therefore, it has been suggested that poor right hemisphere functioning is associated with low heart rate and deficient withdrawal system which would make people less cautions about potentially risky situations that could lead to antisocial behaviour.

However, the right hemisphere hypothesis too has failed to provide a viable explanation of the relationship between heart rate and crime. The approach-withdrawal model is not widely accepted in neuroscience and it remains at a level of speculation (Gainotti, 2019b, 2019a; Shankman & Klein, 2003). In fact, it has been subjected to much criticism, both on a theoretical and empirical level. The aspect of a model wherein it equates positive emotions with approach and negative emotions with withdrawal has been considered overly simplistic. For example, anger is a negative emotion, but it is related to the approach response and it has been associated with the left hemisphere activation which is not in line with the model's predictions (Gainotti, 2019a; Russell & Barrett, 1999). On the empirical level, it has been argued that, on one hand, the majority of the studies supportive of the approach-withdrawal model suffer from certain methodological issues, and on the other, there have been many findings from experimental and clinical studies which cannot be explained by the model (Gainotti, 2019a, 2019b; Killgore & Yurgelun-Todd, 2007). Therefore, the main mechanism used in the right hemisphere hypothesis is actually an assumption. Furthermore, even if the approach-withdrawal model would be somehow proven to be true, the right hemisphere hypothesis still does not provide a clear explanation how a deficient withdrawal system leads to criminal behaviour.

Moreover, the two core assumptions of the hypothesis, that heart rate is controlled by the right hemisphere, and that antisocial individuals are characterized by decreased right hemisphere functioning, are also problematic. Although it has been indeed suggested in the past that the right hemisphere is dominant for the control of autonomic responses, heart rate included, the evidence seems to suggest that such view oversimplifies in fact a more complex model of regulation involving both parasympathetic and sympathetic responses which are drawing upon both hemispheres (Beissner, Meissner, Bär, & Napadow, 2013; Ghchime et al., 2016; Hagemann, Waldstein, & Thayer, 2003; Oppenheimer, Gelb, Girvin, & Hachinski, 1992; Wittling, Block, Genzel, & Schweiger, 1998). Similarly, there is ample evidence that antisocial behaviour and aggression are associated with brain deficits scattered across both hemispheres (Perach-Barzilay et al., 2013; Raine, Buchsbaum, & LaCasse, 1997; Yang & Raine, 2009). Therefore, the right hemisphere hypothesis seems unable to provide a viable underlying causal explanation of the relationship between low heart rate and antisocial behaviour.

2.1.5. Vagal Tone

Another proposed explanation of the link between low heart rate and crime is based on an increased vagal tone, which refers to the activity of the vagus nerve, a component of the parasympathetic nervous system which innervates the cardiac sinoatrial node (Raine, 1993b, 2002). It had been suggested that high vagal tone causes low heart rate which was in turn a marker of passive coping reaction to a stressor. However, in the light of subsequent empirical data showing that antisocial behaviour was in contrast associated with a reduced vagal functioning, it was later hypothesized that low heart rate might be a result of the sympathetic underarousal given that all else being equal low vagal response would result in high heart rate (Raine, 2002). Therefore, the vagal tone hypothesis is flimsy and is neither supported by the empirical data, nor it provides a mechanism by which the vagal tone is associated with antisocial behaviour.

However, recently there has been an attempt to revive the increased vagal activity hypothesis (Brzozowski et al., 2018; Gillespie, Brzozowski, & Mitchell, 2018). On this occasion, a high heart rate variability, presumed to be a measure of vagal tone, and affiliated increases in the prefrontal executive functioning and inhibition have been put forward as an underlying mechanism of the association between low heart rate and crime. In sum, it has been hypothesised that antisocial individuals with low heart rate paradoxically have increased levels of inhibition and self-control. Presumed that such an account is correct, it has been suggested that low heart rate is in fact associated with proactive aggression, which, as it is premeditated and calculated, requires a high self-control. However, empirical data does not seem to support this hypothesis. Firstly, if anything, low heart rate seems to be associated with increased impulsivity, lower inhibition, and poorer cognitive ability (Bennett et al., 2014; Bibbey et al., 2016; Choy et al., 2015; Gao, Borlam, & Zhang, 2015; Ginty, Phillips, Der, Deary, & Carroll, 2011; Mathias & Stanford, 2003; Schmidt et al., 2013). Secondly, low heart rate is associated with a range of antisocial behaviours, and not just premeditated crime and proactive aggression (see sections 1.2. and 1.3.). Finally, a study conducted with a specific aim to test the hypothesis by which a pattern of low resting heart rate and high heart rate variability is associated only with the proactive type of aggression only partly supported this prediction (Brzozowski et al., 2018). In a sample of 92 female university students, perpetrators of interpersonal violence indeed exhibited lower resting heart rate and higher heart rate variability, as well as higher aggressive behaviour. However, contrary to the hypothesis, the relationship between resting heart rate and proactive aggression was non-significant when reactive aggression was accounted for, and vice versa. Therefore, these findings seem to suggest that resting heart rate is associated with a general aggressive behaviour, and not solely with proactive aggression as the increased vagal tone hypothesis predicts. Overall, this review seems to indicate that this is an improbable description of the relationship between low heart rate and antisocial behaviour.

2.1.6. Lower Limbic Hypoactivity and Autonomic Feedback

Armstrong has suggested two explanations to account for the negative link between heart rate and antisocial behaviour (Armstrong, 2011). According to one model, the link is spurious with both variables being caused by a third factor, and the other model proposes that low heart rate is in fact a cause of crime.

2.1.6.1. Lower limbic hypoactivity.

The first hypothesis states that both low resting heart rate and certain cognitive and affective features associated with antisocial behaviour are caused by a hypoactivity in the limbic areas, namely the amygdala, insular cortex, and anterior cingulate cortex (Armstrong, 2011). Specifically, Armstrong argued that these areas are associated with heart rate on one hand, and on the other hand with both antisocial behaviour and a set of characteristics that, if deficient, may contribute to insensitivity to negative consequences for others, such as empathy for pain, fear conditioning, and emotion recognition.

The rationale for the hypothesis was based on the central autonomic network, an internal regulation system in charge of the central control of autonomic activity, including the heart rate, as a function of maintaining homeostasis and responding to stress (Benarroch, 1993, 1997; Thayer, Hansen, Saus-Rose, & Johnsen, 2009). It includes a system of interconnected brain areas that range from the prefrontal cortex, through the limbic system, down to the brain stem, including the orbitomedial prefrontal cortex, insula, the central nucleus of the amygdala, hypothalamus, and the ventrolateral medulla. These areas control heart rate by sending inputs to the sinoatrial node, the heart’s natural pacemaker, through efferent neurons of the autonomic nervous system. Out of all the areas comprising the central autonomic network, Armstrong chose the amygdala, insular cortex, and anterior cingulate cortex, as key to understanding the low heart rate – antisocial behaviour link. He argued that empirical data had showed these regions were associated with both heart rate on one hand, and antisocial behaviour, psychopathy, and callous-unemotional traits, on the other hand. Furthermore, as hypoactivity of these central autonomic network structures had been linked to deficits in empathy, recognition of emotional expressions, experiencing one’s own emotions, fear conditioning, and sensitivity to another’s pain, Armstrong argued that the resulting insensitivity to the negative consequences for others was the underlying mechanism linking these affected brain areas to crime.

In comparison to the previous hypotheses, Armstrong’s (2011) proposition is overall better developed, more detailed, and attempts to integrate research findings from different fields which is necessary if we seek to explain biosocial findings. However, his arguments have several major weaknesses. Firstly, a lack of sensitivity to other people’s pain is not a general cause of crime, and hence it cannot be a mechanism explaining the link between the brain, heart rate, and antisocial behaviour. There are many different types of antisocial behaviours that do not involve hurting other people, and the lack of sensitivity to other’s pain is not a good explanatory mechanism even solely for the interpersonal antisocial behaviours (e.g., if a person is indeed insensitive to other people's pain it does not mean he or she will be motivated and consider inflicting pain on others). Secondly, the rationale for excluding the prefrontal areas of the central autonomic network from the set of brain structures whose functioning may underlie the heart rate-crime link is unconvincing. Despite acknowledging that the prefrontal cortex is associated with both heart rate and antisocial behaviour, Armstrong (2011) argued it did not underlie the association between these variables for the following reasons:

1. The prefrontal cortex was not associated with fear conditioning
2. The prefrontal cortex was not part of the neural overlap between experiencing one's own pain and observing pain in others
3. The prefrontal cortex was not related to some of the features of psychopathy, such as a lack of concern for others
4. Some studies had showed that psychopathic individuals may actually have increased prefrontal functioning
5. Although dysfunction of the medial prefrontal cortex was associated with impulsive antisocial behaviour, it did not lead to the instrumental type which is "the hallmark of the psychopath" (Armstrong, 2011, p. 55)

Putting aside the debate, although an important one, about the difference between fear conditioning and threat conditioning (see Hoppenbrouwers et al., 2016; LeDoux, 2013, 2014) which was not acknowledged in the paper, the presented arguments do not make a case for excluding the prefrontal cortex, especially its orbitomedial[2] area, as a potential neural underpinning linking low heart rate and antisocial behaviour. With regards to the first argument, this region of the prefrontal cortex has been in fact found to have an important role in fear/threat conditioning (Alvarez, Biggs, Chen, Pine, & Grillon, 2008; Birbaumer et al., 2005; Hoppenbrouwers et al., 2016; Hugdahl, 1998). Even more importantly, it is not at all clear why involvement in fear conditioning and experience of pain are the necessary features

[2] The terms orbitofrontal cortex and ventromedial prefrontal cortex have not been used consistently across the literature. They have been used interchangeably (e.g., Lough & Hodges, 2002; Zelazo & Müller, 2002), to refer to distinct brain regions (e.g., Bouret & Richmond, 2010; Suchy, 2009), or, in some cases, the orbitofrontal cortex has been considered as a part of the ventromedial prefrontal cortex (e.g., Rosenbloom et al., 2012; Treiber, 2011). Therefore, it has been decided to use the term orbitomedial prefrontal cortex throughout this work as a compromise solution to label the orbital and medial regions of the prefrontal cortex. This term therefore allows to encompass research findings relating to both orbitofrontal and ventromedial prefrontal cortices.

the brain structure must have to be eligible as a confounding factor explaining the heart rate-antisocial behaviour link. Not only has the orbitomedial prefrontal cortex been associated with the cognitive and affective features emphasized by Armstrong, such as fear conditioning, empathy (Bzdok et al., 2012; Masten, Morelli, & Eisenberger, 2011; Rankin et al., 2006), and emotion recognition (Adolphs, 2002; Heberlein, Padon, Gillihan, Farah, & Fellows, 2008; Hornak et al., 2003), but it has also been related to the variables even more causally relevant to the criminal behaviour, such as decision-making (Bechara, 2004; Damasio, 1994; Wallis, 2007), moral reasoning (Boccia et al., 2017; Ciaramelli, Muccioli, Làdavas, & Di Pellegrino, 2007; Fumagalli & Priori, 2012; Koenigs et al., 2007; Raine & Yang, 2006), emotion regulation (Etkin, Büchel, & Gross, 2015; Hiser & Koenigs, 2018; Motzkin, Philippi, Wolf, Baskaya, & Koenigs, 2015; Quirk & Beer, 2006), guilt (Krajbich, Adolphs, Tranel, Denburg, & Camerer, 2009; Mclatchie, Giner-Sorolla, & Derbyshire, 2016; Wagner, N'Diaye, Ethofer, & Vuilleumier, 2011), and behavioural and cognitive inhibition (Horn, Dolan, Elliott, Deakin, & Woodruff, 2003; Hornberger et al., 2011; Szatkowska, Szymańska, Bojarski, & Grabowska, 2007).

As with regards to the last three arguments, it seems that the author erroneously treated antisocial behaviour as identical to psychopathy. Even if the purpose was indeed to explain the link between heart rate and psychopathy, these arguments are misleading. Regarding the third argument, although one of the reviews cited in support of it (Blair, 2007a) stated that the core functional impairments of psychopathy were more likely to be associated with the amygdala damage, rather than the orbitofrontal cortex impairments, the overall conclusion of the presented (supposedly) supportive reviews was that the disruption in integrated functioning of both the amygdala and the orbitomedial prefrontal cortex led to psychopathy (Blair, 2007a, 2007b). In addition, there is ample empirical evidence showing that psychopathy is indeed associated with structural and functional deficits in this specific brain area (Koenigs, 2012; Poppa & Bechara, 2015).

As to the fourth argument, some studies have indeed found an increased prefrontal functioning in psychopathic individuals (Glenn & Raine, 2008), but this finding applies only to the dorsolateral prefrontal cortex which is not a part of the central autonomic network, and hence it is irrelevant for the discussion. Finally, the fifth argument is erroneous, as psychopathy has been associated with both reactive and instrumental types of aggression (Blais, Solodukhin, & Forth, 2014).

Most importantly, besides being inaccurate, the last three arguments are also immaterial. Namely, the goal of the hypothesis was to explain the link between heart rate and antisocial behaviour, whereas these arguments refer to psychopathy instead. Although antisocial behaviour is arguably a part of psychopathy and these two phenomena are related, it would be a mistake to treat them as identical (Hare, 1996; Leistico, Salekin, DeCoster, & Rogers, 2008; Venables, Hall, & Patrick, 2014). In addition, criminality per se is not even a component of the psychopathy construct, although it might be associated with the psychopathic personality traits (Hare & Neumann, 2010). Therefore, the presented hypothesis has been a step forward in the overall discussion about factors that may explain the relationship between heart rate and crime. However, due to imprecise description of the underlying causal mechanisms and flawed choice of potential neural confounders, it does not offer a reasonable explanation of the relationship of interest.

2.1.6.2. Autonomic feedback.

This hypothesis proposes that low heart rate causes antisocial behaviour through the feedback regarding the peripheral bodily state that afferent neurons carry to the brain stem, limbic system, and the prefrontal cortex where it is then applied to the emotional and cognitive processes that influence behaviour (Armstrong, 2011). Specifically, the model outlines that the low heart rate causes crime through a lack of concern for others due to the inability to understand how one's behaviour affects them. Failure to anticipate how would other people feel was attributed to a missing link between autonomic arousal that would normally accompany personal negative emotions and the signs of these emotions in others. It was further suggested that the reduced autonomic feedback might stem from the deficiencies in three potential sources: the amygdala, anterior cingulate cortex, and insular cortex, as areas responsible for generating autonomic arousal; afferent neurons of the autonomic nervous system which carry the feedback from periphery to the higher cortical structures; brain stem and cortical structures responsible for the central representation of autonomic feedback.

In a subsequent article (Armstrong & Boutwell, 2012), the hypothesis was further specified such that the relationship between low heart rate and crime was suggested to be explained by a reduced autonomic feedback to the insular cortex, where the central representation of bodily state is taking place. As the insular cortex had been associated with empathy for pain and fear conditioning, it was argued that reduced arousal may lead to a reduction of anticipated unpleasant autonomic states which would follow the encounter with pain in others or an aversive stimulus. In sum, the low heart rate may cause an inability to understand how one’s behaviour may influence other people and therefore lead to antisocial behaviour.

The argumentation in favour of the causal effect of low heart rate does not seem compelling. Firstly, Armstrong (along with Boutwell in the subsequent article) constantly refers throughout both papers to the “reduced feedback”, when in fact there is only evidence that the heart rate is reduced, not the autonomic feedback. Secondly, the hypothesis is based on many unfounded assumptions. Although physiological arousal is indeed an important part of emotional experience, it is overly simplistic to equate autonomic bodily reactions with conscious feelings (LeDoux, 2013; LeDoux & Hofmann, 2018; LeDoux & Pine, 2016). There are many studies showing that an aversive stimulus can induce a physiological arousal in people without them experiencing negative emotions or even being aware of the stimulus itself (LeDoux, 2014, LeDoux & Pine, 2016). Similarly, individuals can significantly differ in their autonomic responses to stressful tasks without showing differences in emotional responses, that is, they would consciously experience the task as equally stressful (Bennett et al., 2014; Cox et al., 1983; Fairchild et al., 2008; O’Connor et al., 1985). Therefore, the underlying notion that physiological arousal causes emotions (and therefore behaviour) is too reductionistic. In addition, it completely disregards the role of cognitive processing in emotional experiences. Emotions arise from a cognitive evaluation of a situation that a person is a part of, wherein their autonomic arousal represents only one of many informational inputs that are being processed (LeDoux & Hofmann, 2018). Therefore, autonomic feedback only contributes to subjective feelings, but does not determine them. In addition, research does seem supportive of the role of the insular cortex in central representation of autonomic state, but there is no definitive proof for the notion that it in turn generates emotions (LeDoux & Hofmann, 2018).

Similarly, the notion that people cannot understand how their antisocial actions affect others without experiencing attendant autonomic arousal is completely unfounded. Even if some people do not have an emotional reaction to the fact that their behaviour may result in negative consequences for others, there is no reason to believe that they cannot understand this on a cognitive level. Put differently, they may not care whether their antisocial actions hurt other people, but they would still be able to understand that such behaviour has adverse consequences for them (Cima, Tonnaer, & Hauser, 2010).

Therefore, this hypothesis seems untenable, as its two core assumptions, that physiological arousal causes emotions and hence behaviour, and that people cannot understand consequences of their behaviour without experiencing physiological arousal/emotions, are simply incorrect.

2.2. Empirical Testing of the Proposed Theoretical Hypotheses

Research regarding the link between low heart rate and antisocial behaviour has been predominantly focused on replicating the results of their association across different methodological designs. Unfortunately, there has been little effort to empirically verify the potential theoretical explanations accounting for the existence of this relationship. Out of all proposed mechanisms, only the stimulation-seeking and fearlessness hypotheses have gained ground in the field and are often presented as potential explanations in the literature, but without a strong empirical backing. Thus far, there have been only five studies exploring which personality trait mediates the link between low heart rate and crime. Out of these, four studies have concluded it was the stimulation-seeking that accounted for the link (Hammerton et al., 2018; Portnoy et al., 2019, 2014; Sijtsema et al., 2010), and one study reported in favour of the fearlessness hypothesis (Armstrong & Boutwell, 2012). However, certain methodological concerns and the actual statistical results make these findings unconvincing.

The first study (Sijtsema et al., 2010), which reported that stimulation-seeking and not fearlessness explained the link between low heart rate and rule-breaking behaviour, used an inappropriate measure as a proxy for fearlessness. Specifically, it used a measure of behavioural inhibition even though such an instrument would be more suitable for an assessment of self-control. Furthermore, no mediation effects were found in a female subsample. In a male subsample, out of six models testing the mediation effects of stimulation-seeking and fearlessness measured at three different time points (at ages 11, 13.5, and 16), only one model yielded statistically significant results, showing that stimulation-seeking partially mediated the link between heart rate and rule-breaking behaviour at age 16.

The subsequent study aimed to compare the mediation effects of sensation-seeking and fearlessness in a relationship between antisocial behaviour and heart rate measured at rest and in response to a social stress task and a cognitive task (Portnoy et al., 2014). Both tasks failed to elicit autonomic responses and therefore all heart rate measures were combined into a single latent variable. The study concluded that the only significant mediator was stimulation-seeking. However, stimulation-seeking was not associated with heart rate, and it explained only 36% and 27% of the total effect[3] of heart rate on aggression and non-violent delinquency, respectively. The indirect effect of heart rate on aggression rendered the direct effect non-significant, but in case of the model predicting non-violent delinquency, the direct effect of heart rate remained significant. Furthermore, the measure of stimulation-seeking used in the study has been criticised for including impulsivity items and therefore does not represent a valid measure (Steinberg et al., 2008).

Thus far, there has been only one study which concluded that its results were supportive of the fearlessness and not the stimulation-seeking model of the relationship between low heart rate and antisocial behaviour (Armstrong & Boutwell, 2012). In this study, participants were given three scenarios describing situations in which a person committed a crime, namely an assault, drunk driving, or a theft, after which they were asked to assess the likelihood they would commit each of the crimes as the protagonists in the scenarios did. Given that the mediators were considered in light of the rational choice theory, tendency to underestimate costs and overvalue benefits of antisocial behaviour were used as proxy measures for fearlessness and stimulation-seeking, respectively. Specifically, fearlessness was measured using questions in which participants assessed the likelihood of getting arrested and convicted should they commit the crime and the extent to which the arrest and conviction would cause problems to them, as well as whether they would experience guilt or shame should they commit the crime and the extent these emotions would impose a problem for them. Stimulation-seeking was assessed by asking participants to assess how much fun would it be to commit the crimes as described in the scenarios. The results showed that low resting heart[4] was associated with lower perceived likelihood of arrest and conviction aggregated across all scenarios, but not with the tendency to anticipate committing the described crimes as fun. The anticipated guilt and shame significantly mediated the link between low heart rate and likelihood to commit the assault as described in the scenario. On the basis of these results, the authors concluded that the study was supportive of the fearlessness hypothesis.

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[3]This is not to suggest a causal effect of heart rate on antisocial behaviour. Phrases such as "total effect of heart rate on antisocial behaviour" used here and throughout the thesis should be considered solely in the context of describing statistical relationships within a mediation model.

[4] Resting heart rate was not used as a continuous variable in the analyses. Instead, the authors created two groups of participants based on their heart rate and used this binary variable in the further analyses. Low resting heart rate consisted of participants who had an average resting heart rate under one standard deviation below the sample's mean (n = 26), and the remainder of the participants were allocated to the other group (n = 125; Armstrong & Boutwell, 2012).

However, certain features of the study design inherently limit the conclusions drawn by the authors. Firstly, the choice of the theoretical framework the study was based upon is arguable, as the rational choice theory has been heavily criticised from both theoretical and empirical perspectives (Kahneman, 2011; Kroneberg, Heintze, & Mehlkop, 2010; Treiber, 2017a; Wikström & Treiber, 2016). Secondly, the validity of the instruments used to measure the mediators, especially fearlessness, is highly questionable. There has been no evidence offered to support the assumption that fearless individuals tend to underestimate the costs of their actions. Fearlessness refers to a lack of emotional response to an aversive situation, which does not necessarily imply that a fearless person is not cognitively aware of the potential consequences associated with committing a crime. The assumption that the anticipation of low guilt and shame reflects fearlessness is also highly problematic. Finally, generalizability of the findings is arguable as they were based on a relatively small sample of undergraduate students attending criminology courses (N = 151). Therefore, this study does not seem to provide a significant support for the fearlessness hypothesis given the methodological concerns outlined here. Furthermore, the statistically significant associations between low heart rate and anticipated costs of committing crimes were not replicated across all scenarios and measures of cost.

Finally, two recent studies have also explored the role of other potential mediators, namely self-control and callous-unemotional traits (Hammerton et al., 2018; Portnoy et al., 2019). One study explored fearlessness, stimulation-seeking, and self-control as potential mediators of the relationship between heart rate and antisocial behaviour (Portnoy et al., 2019). The results showed that both stimulation-seeking and self-control, but not fearlessness, mediated the link. However, the study assessed only academic dishonesty as an outcome variable, a very niche type of antisocial behaviour. Furthermore, the sample included a small number of undergraduate students (N = 137) and the mediation analyses were conducted only in a female subsample (n = 90). In addition, the measure of stimulation-seeking was inadequate as it has been criticised for including impulsivity items (Steinberg et al., 2008). Another notable finding from this study was that the resting heart rate showed a significant positive association only with self-control, whereas it was unrelated to stimulation-seeking and fearlessness.

The final study explored the role of stimulation-seeking and callous-unemotional traits as potential mediators of the relationship between resting heart rate and antisocial behaviour in a large sample of more than 4000 people (Hammerton et al., 2018). Lower resting heart rate at age 12 was not associated with callous-unemotional traits at age 14, but the effect of heart rate on antisocial behaviour in mid-adolescence was significantly mediated through stimulation-seeking. However, the indirect effect of stimulation-seeking on the relationship between heart rate in childhood and antisocial behaviour in early adulthood was not statistically significant.

As demonstrated throughout this chapter, none of the existing proposals have been able to provide a viable explanation of the relationship between low heart rate and antisocial behaviour. In addition, there has been little attempt to empirically verify these theoretical explanations, as the scarce empirical research primarily targeted stimulation-seeking and fearlessness hypotheses as the most prominent models in the literature. The limited data available thus far have yielded mixed results, which is unsurprising given the theoretical drawbacks of these hypotheses. Overall, these findings seem to favour the stimulation-seeking theory, but they are far from conclusive as they either only partly explain the relationship between heart rate and antisocial behaviour, or have certain methodological characteristics which make their interpretation ambiguous. Therefore, this review of theoretical and empirical aspects of the proposed explanations suggests that there is a need to develop a better model of the relationship between heart rate and crime. In the next chapter, this association will be critically assessed and placed within the context of a criminological theory which offers a satisfactory model of crime causation. This will represent the foundation of a new model of the relationship between low heart rate and crime which will offer a viable and testable alternative to the previous attempts to explain this phenomena.

Chapter 3:

Remodelling the Heart Rate – Crime Relationship Using Situational Action Theory

3.1. Low Heart Rate: A Cause or Correlate of Crime?

One of the most important features of any heart rate-crime relationship model is whether it positions heart rate as a cause or merely a correlate of crime. In some hypotheses, low heart rate is considered as a marker of a presumed cause of crime. For example, the fearlessness hypothesis views heart rate as an indicator of a factor that leads to crime. Specifically, heart rate is a marker of a lack of fear which facilitates antisocial and criminal behaviours. On the other hand, some hypotheses have proposed that low heart rate itself causes crime through different mechanisms. For instance, in the stimulation-seeking and autonomic feedback hypotheses, low heart rate causes crime by inducing a need for thrill-seeking behaviour and an inability to understand negative consequences that antisocial behaviour can cause for others, respectively.

Correct specification of whether the low heart rate represents a marker or a cause of crime is not only important for theoretical purposes, but also because it has profound implications for designing intervention techniques to tackle antisocial behaviour. For example, one of the proposed clinical implications of the possibility that low heart rate causes crime is that underaroused children with conduct disorder could benefit from a stimulant treatment as it has been found to reduce antisocial behaviour and increase heart rate (Ortiz & Raine, 2004). Similarly, it has been proposed that a biofeedback technique which aims to increase heart rate could be used as an intervention strategy to reduce aggression (Raine, 1996b; Raine & Liu, 1998). With regards to the stimulation-seeking hypothesis, some authors have suggested that an intervention programme addressing antisocial behaviour in individuals with low heart rate should focus on teaching them prosocial methods to increase their arousal and satisfy the need for stimulation without engaging in illegal acts (Hammerton et al., 2018; Portnoy et al., 2014). It is hence necessary to correctly specify the relationship between heart rate and crime, in order to avoid promoting inaccurate intervention and prevention programmes. If an intervention method is based on wrong assumptions, at best it can be ineffective and waste financial resources and time. However, consequences may be more severe, especially if proposed interventions involve use of unnecessary and potentially harmful medications. Therefore, it is essential that an explanatory model correctly specifies whether heart rate is a cause or just a correlate of antisocial behaviour.

It has been suggested before (Wikström, 2014), and it will be further argued here, that low heart rate cannot be a cause of crime. At best, it can only be a marker of a cause. If something causes crime, then its manipulation should always lead to predicted changes in crime as well. In addition to establishing causation through experimental procedures, a theoretical analysis should be able to provide an explanation of such a link, that is, to outline a plausible underlying mechanism (Wikström, 2012). If low heart rate indeed causes crime, then when someone's heart rate decreases (due to good fitness levels, medication, mindfulness, etc.), it should result in a higher individual tendency towards committing crime. From everyday experience, we know this is not the case. Professional athletes in general have a low resting heart rate due to the enlarged and hence more efficient hearts (Oakley, 2001; Serra-Grima, Puig, Doñate, Gich, & Ramon, 2008), but there is no evidence that they are more likely to engage in antisocial and criminal behaviours. Similarly, hypnosis and meditation have been found to decrease heart rate (De La Cruz et al., 2019), but there have been no reports that such practices lead to criminal behaviour. Moreover, some studies have actually suggested that practising meditation could reduce antisocial behaviour and criminal recidivism (Bleick & Abrams, 1987; Himelstein, 2011; Jones, Clayborne, Grant, & Rutherford, 2003; Orme-Johnson, 2011).

Furthermore, alcohol has been a known correlate of antisocial behaviour and research has repeatedly shown that a significant number of offenders had been intoxicated at the time of committing crime (Felson & Staff, 2011; Martin, 2001; Pajevic, Batinic, & Stevanovic, 2017). Experimental manipulation has also confirmed that increased alcohol intake is followed by a higher aggressive behaviour in both women and men (Duke, Giancola, Morris, Holt, & Gunn, 2011). However, alcohol also stimulates the sympathetic nervous system activity and increases heart rate levels which is quite the opposite of what the idea of low heart rate as a cause of crime would predict (Brunner et al., 2017; Sagawa et al., 2011; Spaak et al., 2008).

Similarly, use of medication which causes heart rate to decrease, such as beta blockers, should also result in an increased antisocial behaviour as a side-effect amongst the medicated patients. Not only this is not the case, but if anything beta blockers have been found to reduce aggressive behaviour (Haspel, 1995; Silver et al., 1999). A similar argument would apply to certain medical conditions associated with low heart rate as well. For example, hyperthyroidism is an endocrinological disorder caused by excessive production of thyroid hormone which also includes symptoms of rapid heart rate (World Health Organization, 1992). However, increased thyroid hormone levels have been associated with higher criminal recidivism and aggression, even though it also increases resting heart rate (Evrensel, Ünsalver, & Özşahin, 2016; Stalenheim, 2004).

In sum, there are no experimental data showing that change in heart rate leads to a change in personal propensity to commit crimes, nor there is a viable theoretical model that could offer a mechanism through which heart rate causes crime. Therefore, there is a strong case that heart rate should be explored only as a marker, and not the cause, of antisocial and criminal behaviour.

3.2. Choosing a Theoretical Framework: Why Situational Action Theory?

In order to interpret research findings and propose a model that would provide a viable explanation of the link between heart rate and crime, we need the guidance of a well-developed and empirically supported theoretical framework which offers a precise description of causal processes leading to crime. However, criminology offers an abundance of theories given equal attention yet which cannot possibly all be valid. Thirty years ago, it was noted that "despite the enormous volume of quantitative research, there has been no substantial progress in falsifying the criminology theories that existed 20 years ago" (Bernard, 1990, p. 347). Fast forward decades later and criminology is still a fragmented discipline which has not made significant scientific progress as it has neither advanced in negative learning, that is, falsifying theories and rejecting those which are not viable, nor in positive learning, that is, accumulating supportive knowledge in context of another theory to a point where it is no longer questioned and becomes a generally accepted theoretical framework (Bruinsma, 2016; Weisburd & Piquero, 2008). Criminological theories are characterised by several major problems that have hindered the emergence of a common theoretical framework which would explain crime and offer much needed means of separating causal from correlational relationships.

Firstly, there is not even an agreement about what criminology aims to explain, simply put, what crime actually is (Wikström, 2010, 2012, 2017; Wikström, Oberwittler, Treiber, & Hardie, 2012). The starting point of any theory should be a clear statement about what is the object of explanation. Without this prerequisite, it is hard to imagine how a theory would be able to explain why crime happens and what the relevant causal factors are. The surprising lack of a generally accepted definition of crime is a consequence of the fact that criminological theories have rarely found it necessary to include a clear definition of crime. Few of those which have put effort into defining their object of interest have offered a plausible definition. As an illustrative example, the general theory of crime defines it as "acts of force and fraud undertaken in the pursuit of self-interest" (Gottfredson & Hirschi, 1990, p. 15), which is not an acceptable definition of crime for several reasons. Firstly, a motivation for committing crime should not be incorporated into the definition of crime (i.e., it confuses explanation and definition). Secondly, as the authors argue that all human action is conducted in self-interest, this does not represent a distinguishing feature of criminal behaviour. Next, it is hard to argue that *all* crimes are conducted in self-interest (e.g., taking part in a fight to defend a friend, aiding and abetting). Finally, there are other crimes which do not involve elements of fraud and force (e.g., drug possession/trafficking, probation violation, solicitation, drunk driving; Wikström & Treiber, 2007). It is hard to imagine how a theory would explain any phenomenon without firstly defining what it aims to explain.

Secondly, most criminological theories do not address the question of how crime happens, that is, by which causal processes (Wikström et al., 2012; Wikström, 2010). If a theory does not specify an action mechanism that brings about crime, it cannot answer what causes crime and how to prevent it. Therefore, it is not surprising that most theories are fundamentally probabilistic. In essence, they list factors that increase the likelihood of crime occurrence without actually explaining what moves people to engage in breaking the law (Treiber, 2017a). Even though the majority of criminological theories have not outlined the process which explains how crime happens, several prominent theories have adopted a rational choice perspective as their chosen action mechanism (Treiber, 2017a). However, this comes with its own problems. Specifically, such an approach deals only with the process of choice in a way which disregards the fact that people do not make a choice between all objectively possible options, but only between those which they personally perceive. Therefore, understanding how people come to see their action options and when a crime becomes an action alternative are more fundamental questions to the explanation of crime as the relevance of choice process depends on it. Furthermore, decision-making is not an unidimensional process, but rather consists of two distinct systems referred to as rational choice and habitual choice (Evans & Frankish, 2009; Kahneman, 2011; Treiber, 2017a). Rational choice theory focuses only on the former, whereas the latter process, which is automatic, fast, and used more frequently in everyday life, is completely neglected. Finally, the rational choice approach is based on the assumption that people's actions are driven solely by self-interest, which represents a gross oversimplification of human motivation (for details, see Treiber, 2017a; Wikström & Treiber, 2016). Therefore, criminological theories mostly do not specify action mechanism that brings about crime, and those that do focus only on one aspect of decision-making which can indeed explain crime in some instances, but only occasionally and under specific conditions, and hence cannot represent a general action mechanism.

Thirdly, in general, criminological theories aim to explain one of two core empirical findings; that the distribution of crime in a population is highly skewed such that a small number of people commit majority of crimes, and that there are crime hotspots (i.e., crimes are concentrated in space and time; Wikström, Mann, & Hardie, 2018). Therefore, there is a division in criminology between person-oriented theories that aim to explain the former and the environment-oriented theories which focus on the latter (Wikström et al., 2012; Wikström, 2010). Person-oriented theories aim to explain why some people are more likely to engage in criminal activities than others, but fail to acknowledge the necessity of certain environmental features to trigger the criminogenic characteristics of a person (e.g., Gottfredson & Hirschi, 1990). On the other hand, the environment-oriented theories focus on why some settings are more criminogenic than others, but neglect the role of an individual and relevant personal factors (e.g., Sampson, 2006). However, every action is a result of an interaction between a person and a setting, and neither approach can explain crime by focusing on only one side of the coin. As crime happens only when certain people come into contact with certain settings, there is a need to integrate the knowledge from both types of theories.

Next, criminological theories usually lack a precise delineation between direct and indirect causes of crime. Many theories operate on very different levels of explanation, and therefore it is necessary to bridge micro and macro levels of explanation by making a theoretical system which will feature a clear distinction between a situational level of explanation where the interplay between personal and environmental factors directly causes crime, and a developmental level which involves wider social factors and individual life history features that affect crime indirectly, through their effects on the direct causes of crime (i.e., the causes of the causes). The latter cannot account for what causes crime, but they can help explain critical questions such as how people acquire different crime propensities, why some settings become more criminogenic than others, and how different people come to be exposed to different environments (Wikström, 2006; Wikström et al., 2012). Therefore, indirect causal factors are indispensable to understanding the emergence, development, and change in the direct causes of crime. In order to understand how crime happens and how to prevent it, it is necessary to develop insights about both direct and indirect causal factors situated at an appropriate level of explanation.

Finally, empirical testing remains a sore spot for criminological theory. A majority of theories have never been subjected to empirical verification (Bruinsma, 2016). Very often it is impossible to falsify them as they are described in vague terms from which precise predictions cannot be derived, with even the key concepts defined ambiguously (e.g., as in routine activity theory). If a falsification attempt has been made, it is rare that theories are tested using studies specifically designed for that purpose which raises a question of how valid the measures are of their core concepts (Bruinsma, 2016). Given the level of fragmentation in criminology and disinterest in falsification, it is not surprising that most theories do not have a good track record of explaining crime. An analysis of studies aiming to test criminological theories published between 1968 and 2005 has shown that most of the variance in crime remains unexplained, with a quarter of the studies explaining less than 20% of variation (Weisburd & Piquero, 2008). Such a low explanatory power was found despite the fact that many studies artificially inflated R^2 values through model overspecification, statistical manipulation[5], and inclusion of control variables. However, even more troubling is that it seems that there had not been any improvement in explaining crime over the course of almost 40 years. The finding that theories have often left around 80% to 90% of variance in crime unexplained, could be interpreted as another call for criminology to integrate its dispersed knowledge.

Given the overall state of criminological theory and research, the frequent confusion of causes and correlates is not surprising. It is hard to disentangle which few of all the hundreds of crime correlates have causal properties without relying on a precise analytical framework which delineates a complete system of direct causal factors and how they bring about crime. However, there is a theory that can offer a productive analytical framework for understanding the relationship between heart rate and crime. Namely, situational action theory is a general theory of crime causation which has been specifically designed to address all of the above-mentioned problems in criminology (Wikström, 2006, 2010; Wikström et al., 2012). It offers a viable definition of crime; it is centred around an action mechanism which integrates personal and environmental causal factors; it makes a distinction between direct and indirect causes of crime. Furthermore, this theory has not only integrated the person- and environment-oriented perspectives in criminology, but it has also incorporated insights from other disciplines, including neuroscience and biology, which is particularly important if we

[5] An example of statistical manipulation would be adding prior crime involvement in a model, even though past crime could predict future crime, and hence increase R^2, but would not add anything to its explanation.

are to understand the findings from biosocial research (Treiber, 2013, 2017; Wikström & Treiber, 2007).

In addition, as falsification was one of the main concerns in the development of situational action theory, it offers a detailed and testable framework where all the concepts and their relationships are precisely defined so that specific predictions can be derived (Wikström & Treiber, 2016). The theory was developed alongside a large longitudinal study, Peterborough adolescent and young adult development study (PADS+), which was specifically designed to test its core premises (Wikström et al., 2012). The study has followed a random sample of 716 young people from Peterborough, which represented a third of that age cohort, since 2003 when they were 12 years old. In the first wave, participants' parents were interviewed to collect data about the family's social situation at the time and retrospective data about participants' childhood. Participants were then interviewed in the next eight waves, starting with the first wave in adolescence (at age 13), and having the latest wave in their early adulthood (at age 24). The study is characterised by high retention rates (97% up to 2010; 91% up to 2015), innovative methodology (specifically the use of the space-time budget, see Wikström, Ceccato, Hardie, & Treiber, 2010; Wikström et al., 2012), and excellence in data quality (questionnaire data were collected in interviewer-led small groups of four students; psychometric tests and space-time budget were conducted in one-to-one sessions; data were supplemented with information from the official sources, including criminal justice agencies). Data referred to participants' personal characteristics, environmental features, and their interaction (i.e., participant's exposure to settings with specific characteristics), and were collected from different sources using many types of instruments. Figure 3.1. depicts an overview of methodology used in PADS+. The results of the subsequent analyses involving PADS+ data have offered a firm support for the theory (Wikström, 2009, 2014; Wikström et al., 2018, 2012).

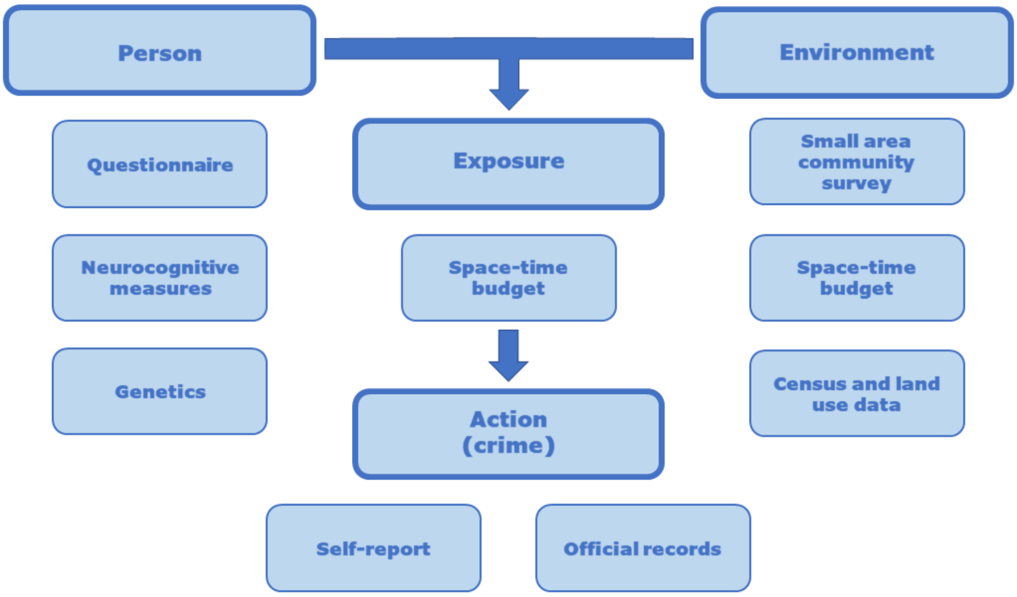

Figure 3.1. Overview of Peterborough adolescent and young adult development study methodology. Adapted from "Social disadvantage and crime: A criminological puzzle" by P. O. H. Wikström and K. Treiber, 2016, *American Behavioral Scientist, 60(10)*, p. 1238.

Furthermore, the theory has received empirical support from other studies conducted either independently or as full replications across several countries (Hirtenlehner & Hardie, 2016; Hirtenlehner & Kunz, 2016; Pauwels, Svensson, & Hirtenlehner, 2018; Serrano-Maíllo, 2018; Svensson, Pauwels, & Weerman, 2010; Wikström & Svensson, 2010). In addition to explaining different types of crimes, from shoplifting to terrorism, it has also proven useful for understanding other criminological phenomena, such as radicalization, political extremism, gender gap in crime, cross-national differences in violence, and the paradoxical relationship between social disadvantage and crime (Hirtenlehner & Treiber, 2017; Perry, Wikström, & Roman, 2018; Schils & Pauwels, 2014, 2016; Wikström & Bouhana, 2017; Wikström & Svensson, 2008; Wikström & Treiber, 2016b). Therefore, situational action theory promises to offer a suitable theoretical framework through which the relationship between low heart rate and criminal behaviour can be explained.

3.3. Analysing Crime: Situational Action Theory

Situational action theory (Wikström, 2006, 2010, 2012, 2014, 2019b; Wikström et al., 2012; Wikström & Treiber, 2007, 2016a) is a dynamic (i.e., emphasises the situational interaction between people and settings) and mechanism-based theory of crime causation which aims to provide an explanation for all types of crimes. Although there has been some scepticism regarding whether a general theory is possible at all in criminology given the stark differences between various crimes (Wilson & Herrnstein, 1985), situational action theory has provided a rather elegant solution to this issue. Namely, the theory defines crime as actions that breach moral rules of conduct defined in law, wherein the concept of moral rules refers to rules regarding what is right or wrong to do, or not to do, in particular circumstances (Wikström, 2006, 2010, 2014). Crime is therefore analysed as a moral action, not a specific content. It represents a part of a wider category of moral rule breaches which refer to both formal and informal rules established by different sources (e.g., parliament, family, gangs, different clubs, societies, and associations). Law is one of many sets of moral rules that can guide people's actions and it covers various acts with little in common. In addition, crimes also differ across countries, jurisdictions, and time (e.g., cannabis possession for personal use has been decriminalised in the Netherlands since the 1970s, but it had been criminalised beforehand, and is still criminalised in the majority of other countries; Graham, 2001; Reuter, 2010). The only thing that all crimes in all places and at all times have in common is that they are acts of breaking rules stated in law. By focusing on what all crimes have in common (i.e., the process of rule-breaking), instead of what makes them different (i.e., the content of different crimes), situational action theory is able to explain any type of crime. Therefore, explaining why crime happens means explaining why people follow and breach rules of conduct, not why they do specific acts (e.g., steal a phone, sell drugs, or assault someone). The major advantage of having a general theoretical framework is that it can be applied to any type of crime, from shoplifting to genocide, as the causal process is always the same (Hirtenlehner & Treiber, 2017; Schils & Pauwels, 2014, 2016; Wikström, 2019b; Wikström & Treiber, 2009a; Wikström et al., 2011).

3.3.1. The Core Framework

Crime is an outcome of a perception-choice process which results from the interaction between a person's crime propensity and exposure to criminogenic places (Wikström, 2010, 2012, 2014; Wikström et al., 2012; Wikström & Treiber, 2017). It will happen once a person sees crime as an option and then chooses to act upon it either automatically or after some deliberation in a particular situation. People vary in their crime propensity, that is, tendency to perceive and choose crime as an action alternative in a response to a motivation. When in the same setting, a person with high crime propensity is more likely to commit a crime than a person with low crime propensity. In addition, the same individual can have different propensities for different crimes. For example, they may have a high tendency to steal, but a low propensity for violence. However, even a very high crime propensity is not enough to cause crime on its own. People are indeed the sources of their actions, but the causes of criminal acts are always situational, in the sense that they happen at the point of convergence between a person and environment (Wikström, 2012, 2014, 2017, 2019b; Wikström & Treiber, 2016a). Crime propensity needs to be triggered by exposure to a criminogenic setting; and criminogenic exposure is only significant in an interaction with a person with adequate crime propensity. People with high crime propensity will not commit crimes in every single setting, but only when exposed to those with a certain level of criminogenic features; and people with low propensity are unlikely to commit crimes even when they take part in a highly criminogenic environment. In certain situations, crime propensity will be a more influential factor, and at other times it will be the criminogenic exposure, but there will always be at least a minimal interaction between the two. Therefore, crime is never a result of either personal or environmental features, but it is always a consequence of their interaction.

The perception-choice process provides an analytical framework to determine which factors can have the capacity to cause and, in a like manner, prevent crime. The causes of crime are only those variables that can directly influence a person to see and choose an act of crime as an action alternative in a specific situation (Wikström, 2006, 2012, 2017; Wikström et al., 2012). Therefore, only individual crime propensity and criminogenic features of settings can be considered as causes of crime. Crime propensity depends on an individual's morality and ability to exercise self-control. A setting is as criminogenic as its (perceived) moral norms and their enforcement (monitoring and sanctioning of moral norm violations) encourage, or at least do not discourage, criminal activity. This is not to say that the influence of higher-level social factors (e.g., inequality), and developmental processes and life history of an individual (e.g., neurocognitive factors) is not relevant. These factors can affect the perception-choice process indirectly through their effect on crime propensity and exposure to criminogenic settings, and therefore are best analysed as the "causes of the causes" (Wikström, 2014, 2019a; Wikström et al., 2012; Wikström & Treiber, 2007, 2009b). In the subsequent paragraphs, only features of situational action theory related to individual factors will be explored in more detail as it is expected that the heart rate link to crime is through personal crime propensity.

3.3.2. Crime Propensity

An individual's crime propensity is comprised of their moral rules and attached moral emotions, and the ability to exercise self-control (Wikström, 2004, 2010, 2014; Wikström et al., 2012, 2011; Wikström & Svensson, 2008, 2010; Wikström & Treiber, 2007). The more one's personal moral rules match the rules of law, the lower will be their tendency to commit crime. However, it is not only important what one thinks is right and wrong to do in particular circumstances (i.e., what moral rules they hold), but also how much one cares about adhering to such norms. If one holds a moral rule but would not feel bad if transgressing, then such moral rule could be easily breached if an appropriate environmental inducement appeared. Therefore, the strength of moral rules depends on the intensity of accompanying moral emotions of guilt and shame. The more one would feel bad in front of oneself (guilty) or others (ashamed) in case of breaching a moral rule one holds, the less likely it will be that the person will violate the norm in question.

Although morality represents the core of crime propensity, the ability to exercise self-control is important in situations when criminogenic features of a setting strongly encourage breaking a moral rule a person holds (Hirtenlehner & Hardie, 2016; Wikström et al., 2012; Wikström & Svensson, 2010; Wikström & Treiber, 2007). The stronger a person's ability to exercise self-control, the more likely it will be that the person will follow their moral rules even in situations when there is a strong external incitement to do the contrary. The ability to resist environmental pressure to act against one's moral norm mainly depends on one's executive functions, but it can be also temporarily diminished by certain factors such as drug or alcohol intoxication, strong emotions, or prolonged period of stress. The term "executive functions" has been loosely applied across the literature to a number of top-down cognitive functions supported by the prefrontal cortex, such as inhibition, selective attention, working memory, interference control, and abstract reasoning (Diamond, 2013; Wikström & Treiber, 2007). In situational action theory, executive capabilities have been specifically defined as a set of cognitive functions that enable a person to create and apply internal representations to decision-making process, where the concept of internal representations refer to an informational framework composed of an integration between a person's internal knowledge stemming from past experiences and external sensory information coming from the environment (Wikström & Treiber, 2007).

3.3.3. Crime as Situational Action: The Perception-Choice Process

A perception-choice process is always initiated by motivation. There are two types of crime-related motivators: a temptation, which is a result of interaction between one's desires (or commitments) and opportunities to satisfy a desire (or fulfil a commitment), and a provocation, which happens when an external interference makes one angry or annoyed with its perceived source or a substitute (Wikström, 2004, 2014; Wikström et. al., 2012; Wikström & Treiber, 2007, 2009a, 2016a). As a general rule, temptations are followed by positive emotions, whereas provocations are associated with negative emotions. However, motivation is necessary to trigger the causal process, but it is not a sufficient factor in crime causation as it cannot explain why crime happens. If a person is not motivated to commit a crime, it will certainly not happen. A person needs to be motivated to commit a crime in the first place in order for it to occur, but the motivation itself can only be a reason for an act, not its cause. There are no particular motives that always cause a specific crime or that are unique to a specific crime (e.g., aggravated assault can happen for many different reasons), and there are usually different ways to satisfy the same motive with crime being only one of the possible means (e.g., assaulting a person who spilt a beer over us is only one of many possible reactions to such a situation).

Appropriate motivation will set the ground for crime, but it will only be realised if a person sees crime as an action alternative and then chooses to act upon it. What a person sees as a viable response to a particular motivation depends on an interaction between their personal morality and perceived moral norms of a place in which the person takes part. This interaction creates a moral filter which determines what action alternatives a person will perceive (Wikström, 2010, 2014, 2019b; Wikström et al., 2012). If an actor's personal morality and perceived moral norms of their environment both discourage breaking the law when reacting to a particular temptation or provocation, then it is unlikely they will see crime as an option. However, if moral aspects of both an individual and their setting encourage breaking the rule of conduct, then the person will most probably see crime as a viable response to a particular motivation. This relationship is referred to in situational action theory as a principle of moral correspondence (Wikström, 2010, 2014, 2019b; Wikström et al., 2012; Wikström & Treiber, 2009a).

If a person does not even see crime as a viable course of action in response to a particular motivation, then crime will not happen and the process of choice will be irrelevant for such an outcome (Wikström, 2014, 2019b; Wikström et al., 2012). Perception of response options precedes and determines the relevance of the process of choice, and it is, therefore, more fundamental to the crime causation. In fact, the main reason why the majority of people do not commit crimes in response to temptations and provocations they encounter is that they do not perceive crime as an option in the first place, and not because they actively choose not to commit a crime or because they refrain themselves from breaking the law (Wikström, 2004, 2006; Wikström et al., 2012, 2011). If, however, a person sees crime as an action alternative, whether crime will happen or not will depend on the process of choice. Integrating and building up on the knowledge base from criminology, behavioural sciences, and neuroscience, situational action theory proposes that there are two types of choice processes: a habitual choice and a rational deliberate choice (Treiber, 2011; Wikström, 2004, 2014 2019b; Wikström et al., 2012). When people act out of habit, they perceive only one action alternative and automatically choose to carry it out without any deliberation (Treiber, 2011; Wikström, 2014, 2019b; Wikström et al., 2012; Wikström & Treiber, 2007, 2016a). Essentially, a person automatically reacts to an external cue which triggers previously learned associations through repeated exposure to the same or similar circumstances. Therefore, a habitual choice is most likely to happen in familiar circumstances when there is a correspondence between personal moral rules and perceived moral norms of the setting. It is also possible that intense emotions or high stress nudge a person into reacting in such a way, regardless of the familiarity of a setting. As such, habitual choice is always oriented towards the past as it relies on previous experience to guide present actions. In essence, it is a behavioural shortcut based on an association between a past action and a subsequent outcome. Therefore, it is highly time-efficient, mostly unconscious, and does not require cognitive effort. However, this comes at a cost of a lack of reflection which can sometime lead people to choose actions they would not consider in their best interest had they thought about it beforehand. Habits can be broken by anything that could drive a person to refocus their attention and consider other action alternatives, such as appearance of a strong deterrent in a setting or a change in person’s morality (Wikström & Treiber, 2009).

In case of rational deliberation, an individual perceives several action responses to a particular motivation, where at least one includes breaking a rule of law (Treiber, 2011; Wikström, 2014, 2019b; Wikström et al., 2012; Wikström & Treiber, 2007, 2016a). Deliberation refers to weighing potential costs and benefits of the perceived options in order to choose the one which would satisfy the particular motivation in the best way (as the actor comes to see it) under the given circumstances. As an individual has to predict how each option would unfold, it is future-oriented and requires more time and cognitive power, but it allows a more comprehensive analysis of the situation. People will most likely deliberate in unfamiliar or uncertain situations and when there is a conflict between personal moral rules and perceived rules of a surrounding setting. Whether crime will eventually happen or not will depend on the outcome of deliberation.

3.3.4. The Principle of the Conditional Relevance of Controls

Self-control and deterrence are relevant factors in crime causation only when people deliberate over several action alternatives to a particular motivation, where at least one option includes breaking the law (Hirtenlehner & Hardie, 2016; Wikström, 2012, 2014; Wikström et al., 2012, 2011). If a person does not even see crime as an option, or chooses crime habitually as the only perceived course of action, then both internal and external controls will be irrelevant as there is nothing to control for. If a person deliberates, which type of control will be relevant depends mainly on the nature of a conflict between personal moral rules and perceived moral rules of a setting. Self-control is relevant in situations when there is an external pressure on a person to break a moral rule they personally hold (e.g., a peer pressure to shoplift when that conflicts with the person’s moral rules). Deterrence is relevant in situations where a person’s moral rules favour breaking the law, but the features of a setting discourage doing so by creating fear of consequences (e.g., a person is deterred from the intent to shoplift after seeing security guards). Therefore, self-control enables people to adhere to their own personal rules, whereas deterrence compels people to follow the moral rules of a setting.

Self-control refers to a success in following one’s own personal rules despite the external pressure to transgress (Treiber, 2011; Wikström, 2010, 2014; Wikström et al., 2012; Wikström & Svensson, 2010; Wikström & Treiber, 2007). As a success or failure to exhibit self-control arises from a situation in which an individual responds to a particular setting, self-control is a situational factor and not a personality trait. Ability to exercise self-control will depend on the outcome of the interaction between one’s executive functions (and other more temporary personal factors such as intoxication, intense emotions, or stress) and their environment. In different places and circumstances, an individual will exhibit diverse levels of self-control, and therefore it does not depend only on personal characteristics. Similarly, deterrence is a result of a person’s perception of deterrent qualities of surrounding environment, namely monitoring of its moral rules and sanctions for their transgressing (Wikström, 2010, 2014; Wikström et al., 2012, 2011). If a person sees monitoring to be efficient and sanctions to be harsh in a particular setting, then the person could be deterred from choosing crime as an option (Wikström, 2007). There is also a link between external and internal controls such that executive functions determine how sensitive a person is to deterrent cues in an environment (i.e., how strong a deterrent cue must be to become a factor in deliberation; Wikström & Treiber, 2007). For example, if in the same setting, a person with a more efficient executive functioning will be able to process more environmental cues and take into account their significance when deliberating, whereas a person with poor executive functions may be more susceptible to provocations or frictions, and overlook some deterrent indicators, especially subtle ones. People with deficient executive functions may also fail to comprehend how certain environmental features could affect the course of action they want to take, or to take into consideration the risks of various negative consequences, legal and other, when deliberating. Therefore, there should be a negative association between one's executive functions and the threshold that strength of an environmental deterrent cue has to pass in order for it to affect deliberation (Wikström, 2008; Wikström & Treiber, 2007).

The functioning of the complete framework centred around the perception-choice process suggests that perception and personal morality are more fundamental to crime causation than the process of choice and ability to exercise self-control, respectively (Treiber, 2017a; Wikström, 2004, 2014, 2019b; Wikström, Oberwittler, Treiber, & Hardie, 2017; Wikström & Svensson, 2010). Namely, perception of action alternatives precedes the process of choice and determines its relevance (i.e., if a person does not see crime as an option, the process of choice will be of no relevance in explaining why the person did not break the law). Similarly, even though both personal morality and ability to exercise self-control determine crime propensity, the former comes as the principal individual crime cause as it determines the outcome of a perception process through its role in the moral filter. As the ability to exercise self-control is only relevant to crime causation through its influence in the process of choice, its effect in crime causation is dependent on personal morality (or more specifically, the interaction between personal morality and moral context of a setting; Wikström & Svensson, 2010; Wikström & Treiber, 2007). If a person does not see crime as an action alternative to a particular motivation, the ability to exercise self-control will not be a relevant factor as there will be nothing to control. The ability to exercise self-control will also be an insignificant factor in the process of choice when a person decides to abide by or break the law out of habit. In that case, the setting nudges a person into a certain behaviour and they see only one potent action alternative that is executed automatically. Therefore, there is no room for self-control in such a scenario. Finally, if a person's moral norms do not conflict with breaking the law (i.e., they do not think it is wrong to commit a particular crime in specific circumstances), again self-control will not be a relevant factor in crime causation as there is nothing to control for. The only relevant control in such a case is deterrence, as certain features of a setting (such as effective monitoring or severe sanctions) may deter a person from committing a crime even though their own personal morals would not prevent them from doing so. Therefore, personal morality is always a significant factor in crime causation as it affects perception of action alternatives in response to a temptation or provocation. The ability to exercise self-control will have a role in crime causation only in instances where a person is externally encouraged to break their moral norms and actively deliberates whether to do so. In sum, even though a personal crime propensity is determined by both the ability to exercise self-control and personal morality, the latter is more fundamental factor in crime causation. This is not to say that the ability to exercise self-control is not important, quite the contrary; it can be a decisive factor whether a person will commit a crime or not, but only in specific circumstances. The complete perception-choice process is presented graphically in Figure 3.2.

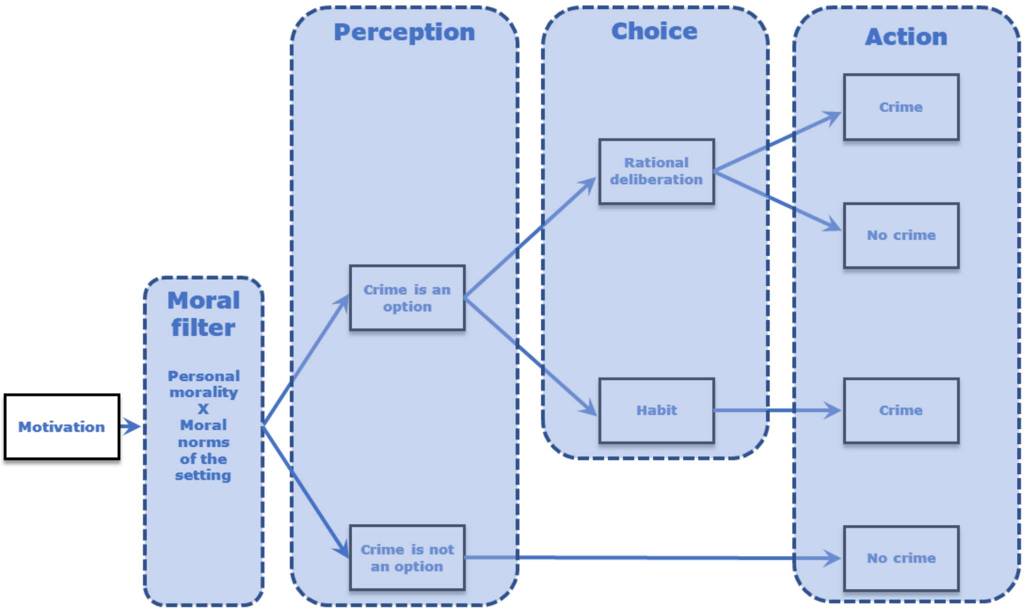

Figure 3.2. Perception-choice process. Adapted from "Breaking Rules: The Social and Situational Dynamics of Young People's Urban Crime" by P. O. H. Wikström, D. Oberwittler, K. Treiber, and B. Hardie, 2012, Oxford: Oxford University Press, p. 29.

3.4. Crime Propensity as a Mediator Between Heart Rate and Crime

Situational action theory has been chosen as a theoretical framework to explain the link between low heart rate and crime for two main reasons. Firstly, it has addressed the problems that had plagued criminological theory for decades. It has offered solutions to all major issues in criminological theory (see section 3.2.): the lack of a suitable definition of crime (crime as *acts of breaking moral rules of conduct*); the need for an action mechanism which explains how crime happens (*the perception-choice process*); division between person-oriented and environment-oriented theories (crime as a result of *an interaction between an individual's crime propensity and their exposure to criminogenic settings*); confusion caused by theories explaining crimes on very different levels of explanation (integrating micro and macro levels of explanation though the concepts of *causes of crime and causes of the causes*); falsifiability and testability (all key concepts are precisely defined and there have been a number of studies specifically designed to test the theory's assertions; Treiber, 2017b; Wikström, 2006, 2010, 2017, 2019b; Wikström et al., 2012). Secondly, the theory has been put under heavy empirical scrutiny which has overall provided supportive results thus far. Its core hypothesis have been confirmed not only by PADS+ (Haar & Wikström, 2010; Hardie, 2019; Treiber, 2014; Trivedi-Bateman, 2019; Wikström, 2009, 2014; Wikström et al., 2010, 2018, 2011; Wikström & Treiber, 2016b), which was specifically designed to test situational action theory, but by a number of other studies across different countries as well (Antonaccio, Botchkovar, & Hughes, 2017; Craig, 2019; Hirtenlehner & Hardie, 2016; Hirtenlehner & Kunz, 2016; Hirtenlehner, Pauwels, & Mesko, 2014, 2015; Hirtenlehner & Treiber, 2017; Kokkalera, Marshall, & Marshall, 2020; Pauwels, 2018; Serrano-Maíllo, 2018; Svensson et al., 2010; Wikström & Svensson, 2008, 2010). Therefore, there are strong arguments to believe that situational action theory will offer appropriate means to explain the link between low heart rate and crime, and place this empirical correlate within a theoretical framework.

Although low heart rate has been established in biosocial criminology as one of the best replicated correlates of crime for more than a decade, a viable explanation for such a relationship is still lacking. Considering this association from the perspective of situational action theory as a general theory of crime should offer a promising way forward. According to its analytical framework, and in line with the analysis at the beginning of this chapter (see section 3.1.), heart rate cannot be a cause of crime as having a slow heart rate cannot move people to commit crime (Wikström, 2010, 2014). On the other hand, heart rate does indeed show an inverse link with antisocial behaviour, and therefore a possible explanation for this relationship is that it is associated with a cause of crime, and thereby, crime itself.

As heart rate represents a biological variable, it is more reasonable to expect that it is a correlate of causes of crime related to person and not a setting. Therefore, it is hypothesised that low heart rate correlates with high crime propensity which, in turn, mediates the link between heart rate and crime. Low heart rate may be an indicator of a blunted emotional response, and it is therefore expected that moral emotions will explain the bulk of the relationship between heart rate and crime. This is also in line with the theory as it has established morality as the fundamental cause that moves people to break law (or adhere to it). However, it is also feasible that heart rate is associated with the ability to exercise self-control, which would then explain the remaining variance of the link. Therefore, it is proposed that heart rate cannot be a cause of crime, and that it is only a correlate of crime propensity. It is hypothesised that so far elusive relationship between heart rate and crime can be fully explained by the mediating effect of personal morality and ability to exercise self-control.

However, in order to provide a comprehensive and detailed explanation of the link between low heart rate and crime, it is necessary to go beyond the mediating effects of this relationship, and explain how heart rate is associated with crime propensity. Low heart rate cannot be a cause of the causes of crime as there is no viable explanation of how it could cause people to have weak morality or poor ability to exercise self-control (or indeed, to be exposed to criminogenic settings). Given that the certain brain regions located in the prefrontal cortex and the limbic system have causal influence on both heart rate and personal crime propensity, it is proposed that low heart rate is a marker of a central dysfunction which is a “cause of the cause” of crime. Specifically, it is hypothesised that deficient functioning of the orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala may lead to low heart rate and engagement in criminal behaviour through its effect on crime propensity. Therefore, the model suggests that the link between heart rate and crime is spurious and caused by the same causal factor. Although the current study will primarily focus on assessing the mediating effects of crime propensity on the relationship between heart rate and crime, the theoretical support for the neurocognitive aspects of the model and a preliminary test of the full model will be presented in detail in Chapter 6.

Apart from theoretical feasibility, the newly proposed model of the underlying mechanism linking heart rate to crime can be indirectly supported by the readily available empirical findings. A significant number of studies have already demonstrated the association between impulsivity (i.e., failure to exercise self-control), measured either via questionnaires or behavioural tasks, and low heart rate measured at rest and in response to stress (Allen, Hogan, & Laird, 2009; Bennett et al., 2014; Bibbey et al., 2016; Mathias & Stanford, 2003; Schmidt et al., 2013). Furthermore, due to the conflation of impulsivity and stimulation-seeking measures (see section 2.1.1.), it is a reasonable possibility that some of the positive results reported by the studies which concluded that impulsive stimulation-seeking mediated the link between heart rate and antisocial behaviour is due to the impulsivity items (Portnoy et al., 2019, 2014). Finally, a recent study examined for the first time self-control as a possible mediator of the link between resting heart rate and antisocial behaviour (although measured through academic dishonesty, a very niche type of antisocial behaviour; Portnoy et al., 2019). The results showed that the relationship was indeed significantly mediated by self-control. Furthermore, only self-control exhibited a significant association with resting heart rate, whereas the other two mediators, fearlessness and stimulation-seeking, did not. Overall, these studies have been supportive of the positive link between heart rate and the ability to exercise self-control.

As for the other component of crime propensity, thus far empirical studies have not shown much interest for the association between heart rate and personal morality. However, a study examining the link between heart rate and crime from the perspective of the rational choice theory has paradoxically provided support for the mediating effect of moral emotions (Armstrong & Boutwell, 2012). Namely, the study has explored the links between resting heart rate, perceived costs and benefits of committing an assault as described in a given scenario, and a likelihood of committing the assault, wherein one of the measures of costs was anticipated shame/guilt should a person commit the assault. The results have shown that the participants with low heart rate were less likely to anticipate feeling guilty or ashamed should they commit the assault as described in the scenario. Moreover, anticipated moral emotions mediated the link between resting heart rate and the intent to commit the assault. Therefore, these results are clearly supportive of the proposition that moral emotions may represent a mediating factor in the relationship between heart rate and antisocial behaviour.

In general, there has been some indirect empirical support for the model in which the link between low heart rate and crime is mediated by weak personal morality and low ability to exercise self-control. However, in order to put this model under a proper empirical scrutiny, it is necessary to conduct a new study specifically designed to test its hypotheses. For this reason, the Cambridge adolescent behaviour study, which will be introduced in the next chapter, has been set up with aim to directly test the new model of explanation of the link between low heart rate and crime.

Chapter 4:

Cambridge Adolescent Behaviour Study: Aims and Methodology

4.1. Aims and Objectives of the Study

The Cambridge adolescent behaviour study was designed with the aim to test the new model of the relationship between low heart rate and crime with criminal propensity as a mediating variable, and to compare it with other, well-established models. Although many potential explanations have been put forward, only the stimulation-seeking and fearlessness hypotheses have become widely accepted in the literature and are often cited as variables that are likely to account for the relationship between heart rate and crime, either independently or jointly (e.g., Latvala et al., 2015; Portnoy & Farrington, 2015; Scarpa et al., 2008). However, neither has received sufficient empirical backing due to a limited number of studies which have yielded inconclusive results (see section 2.2.). Therefore, the main objective of this study is to test and compare the three proposed models of the link between low heart rate and crime, with crime propensity, stimulation-seeking, and fearlessness as mediating variables.

Apart from this principal goal, the study aimed to address the methodological issues featuring in a number of prior empirical studies on the subject of the relationship between heart rate and antisocial behaviour identified in the previous chapters. Firstly, as noted in Chapter 1 (see section 1.3.), heart rate has often been discussed as having been measured in resting state, when it was actually measured under circumstances which were likely to trigger anticipatory anxiety (e.g., Latvala et al., 2015; Raine, Venables, et al., 1997). Furthermore, a significant portion of studies have measured resting heart rate in unstandardised conditions by instructing participants to sit still and relax, which people tend to experience differently (e.g., Armstrong et al., 2009; Portnoy et al., 2014). Therefore, very often it is unclear whether a study included a measure of heart rate assessed in resting state or under the influence of stress or some other emotion or condition, despite being labelled as a resting heart rate. The importance of this matter is further highlighted by the observation that many of the non-significant results pertaining to the relationship between heart rate and antisocial behaviour have been found in studies which utilized a standardised protocol for inducing baseline heart rate (e.g., Prätzlich et al., 2019; Wilson & Scarpa, 2014). Some of these studies have also included a stress-inducing protocol, and their findings have demonstrated that only heart rate reactivity inversely correlated with antisocial behaviour, whereas a properly measured resting heart rate did not (e.g., Bimmel et al., 2008; Van Zonneveld et al., 2017). In addition, it has been recommended that a heart rate should be measured for at least a minute in order to obtain a reliable baseline measure, which has been violated in many studies (e.g., Armstrong & Boutwell, 2012; Raine et al., 2014). Given these potential threats to the validity of the heart rate measure, it is currently uncertain whether antisocial behaviour correlates with both resting heart rate and heart rate reactivity, or just the latter. Therefore, in order to clarify this essential methodological ambiguity, the current study will use reliable measures of both resting heart rate and heart rate reactivity to stress, each measured for the recommended duration in response to standardised protocols for inducing resting and stressful conditions, respectively. These measures will be then examined individually in relation to involvement in criminal behaviour.

Secondly, as explained in Chapter 2 (see section 2.1.1.), stimulation-seeking is often conflated with impulsivity (i.e., a failure to exhibit self-control), which is then reflected in their flawed operationalisation. Namely, even though they are independent constructs with different developmental trajectories and neural underpinnings, many stimulation-seeking measures contain items tapping into impulsivity, and vice versa (e.g., Burt et al., 2014; Steinberg et al., 2008; Vazsonyi & Ksinan, 2017). This conflation has also been present in research exploring mediation effects of the relationship between low heart rate and antisocial behaviour. Out of four studies that have been supportive of the stimulation-seeking hypothesis, two (Portnoy et al., 2019, 2014) have used an impulsive sensation-seeking subscale of a personality questionnaire (Zuckerman et al., 1993). As the name indicates, this instrument directly taps into both impulsivity and stimulation-seeking, wherein many items refer to a lack of planning and tendency to act without thinking (Zuckerman et al., 1993). Conclusions stemming from such studies are therefore inherently limited as it is impossible to disentangle unique relations between stimulation-seeking and impulsivity, on one hand, and heart rate and antisocial behaviour, on the other, due to the conflation of the first two constructs. The other two studies have not used appropriate measures of stimulation-seeking either. One study (Hammerton et al., 2018) used only the intensity subscale of the Arnett's Inventory of Sensation-seeking (Arnett, 1994), and therefore disregarded other, equally important aspects of stimulation-seeking referring to novelty and complexity of stimulating experiences (Ferrando & Chico, 2001; Zuckerman, 1994). Furthermore, the intensity subscale is chiefly related to the disinhibition subscale of the Zuckerman's sensation-seeking scale (SSS-V; Zuckerman, 1994) which includes impulsivity items (Ferrando & Chico, 2001). In addition, the Arnett's Inventory of Sensation-Seeking suffers from significant methodological flaws, specifically low internal reliability and weak factor structure (Arnett, 1994; Ferrando & Chico, 2001). The fourth study (Sijtsema et al., 2010) did not use a stimulation-seeking instrument, but instead utilised the adventurism facet from the Revised Neuroticism-Extroversion-Openness Personality Inventory (Costa & McCrae, 1992), which is also characterised by a low reliability. Therefore, as these studies have assessed stimulation-seeking through instruments of questionable validity, it is not clear whether obtained mediating effects indeed stem from the thrill seeking behaviour or items tapping into other constructs. In order to address this research question, the aim of this study was to test the stimulation-seeking hypothesis using a reliable and valid measure which does not include impulsivity items.

Thirdly, the study aimed to introduce some clarity when defining the outcome variable. Namely, heart rate has been explored in relation to a number of antisocial variables, including both personality traits (e.g.., psychopathy, impulsivity, callous-unemotional traits; e.g., Bibbey et al., 2016; Choy et al., 2015; Fanti et al., 2017; Kavish et al., 2017) and behaviours anywhere on a spectrum ranging from minor misbehaviours within legal limits to the most serious crimes (e.g., academic dishonesty, hostility, lying, aggression, drug offences, convictions for violent and non-violent crimes; e.g., Choy et al., 2017; Dierckx et al., 2014; Jennings et al., 2013; Portnoy et al., 2019, 2014). Studies frequently use vaguely defined concepts, such as antisocial behaviour, delinquency, externalising, and disruptive behaviours, where there is no clear consensus on what they encompass or how to measure them. Operationalisation of such variables often involves a mixture of items tapping into various criminal and legal behaviours. As an illustrative example, one of the widely used measures of aggression in research regarding heart rate (e.g., Brzozowski et al., 2018; Portnoy et al., 2014; Raine et al., 2014), the Reactive-Proactive Questionnaire, contains items with very distinct legal connotations, such as “Used force to obtain money or things from others” (i.e., robbery) and “Yelled at others when they have annoyed you” (i.e., misbehaviour) (Raine et al., 2006, p. 170). The usefulness of this approach depends on the goals of the study. However, one of the aims of the Cambridge adolescent behaviour study was to establish whether heart rate correlates with criminal behaviour specifically. Measuring crime as defined by situational action theory allows for an assessment of various criminal acts, but it excludes “antisocial behaviours” which involve morally dubious, but not necessarily illegal acts. Apart from conceptual clarity, using a clearly defined variable has a benefit of enabling a precise operationalisation and measurement.

Finally, the study intended to improve the operationalisation of crime by introducing items measuring cyber-crime, which has been largely neglected in criminology despite its importance. During the last two decades, there has been a significant increase in the frequency of cyber-crimes followed by a reduction in numbers of more traditional types of crimes, leading to a conclusion that the overall crime rate has not declined, as it often appeared, but rather changed its form (Office for National Statistics, 2015; Weulen Kranenbarg, Holt, & van Gelder, 2019). In 2015, when the Office for National Statistics added for the first time items pertaining to cyber-crime to its Crime Survey for England and Wales, the overall crime rate doubled. The rate of traditional crimes showed a fall of eight percent to an estimated 6.5 million offences (compared with the estimate of seven million for the previous year), whereas cyber-crime accounted for 5.1 million online fraud offences and 2.5 million computer misuse offences, leading to a total of 14.1 million offences. Determining the exact costs associated with cyber-crime is a particularly challenging task. Therefore, it is not surprising that the estimated annual costs to the United Kingdom government range from £1.2 million to £3 billion per year (Home Office, 2018). According to the Detica and Cabinet Office research report, estimated cyber-crime costs to the United Kingdom are £27 billion per year, with the lion's share (£21 billion) affecting its business sector (Detica, 2011). Cyber-crime has also emerged as one of the top priorities tackled by the national security strategies across different countries, often emerging as a more serious threat than organised crime and fraud (Wall & Williams, 2013).

Despite the relatively high prevalence of cyber-crime and associated hefty costs, criminology has been lagging behind in addressing this topic from both theoretical and empirical perspectives. For example, a search for articles mentioning cyber-crime in *Criminology*, the field's flagship journal, has returned only six hits. Out of these, only two articles have actually explored cyber-crime related topics, while the other four were false positives.[6] Indeed, such a problem is also evident in biosocial criminology, where none of the aforementioned articles in the previous chapters exploring the link between heart rate and antisocial behaviour has included measures which included cyber-crime items. Therefore, this study set a goal to explore the link between low heart rate and crime which was considered on a broader scope and included not only different traditional crimes, but cyber-crimes as well.

[6] The search was conducted in September 2019 using Wiley Online Library. Two articles reported about research regarding a cyber-crime topic, with one exploring means of deterring unauthorised access to computers, and the other investigating the link between self-control and cyber victimisation (amongst other types of victimisation) in a meta-analysis (Maimon, Alper, Sobesto, & Cukier, 2014; Pratt, Turanovic, Fox, & Wright, 2014). Other four articles were false hits as they have not explored a topic related to cyber-crime (Button & Worthen, 2017; Holtfreter, Reisig, & Pratt, 2008; Maimon, Antonaccio, & French, 2012; Michalowski & Carlson, 1999).

4.2. Study Hypotheses

Before an attempt to uncover the underlying process linking low heart rate to crime, it is first necessary to establish the exact nature of this relationship. Namely, in Chapter 1 and the previous section, a concern was raised that some of the findings of a negative relationship between low heart rate and crime may have been wrongly attributed to resting heart rate instead of heart rate reactivity. The concern was further substantiated by the non-significant results obtained in the studies that have used a valid measure of resting heart rate (i.e., heart rate measured in response to a standardised protocol for inducing resting state). Therefore, it is hypothesised that after obtaining valid measures of both resting heart rate and heart rate reactivity to stress crime will demonstrate a significant inverse association only with the latter variable.

After determining which of the heart rate measures are associated with crime, the next step is to explain this result. Crime propensity, stimulation-seeking, and fearlessness models of the underlying mechanism state that the relationship between low heart rate and criminal behaviour is mediated by increased crime propensity (i.e., conceptualised as weak morality and poor ability to exercise self-control), high stimulation-seeking, and low fear, respectively. Drawing from the review of theory and empirical findings regarding the three models presented throughout Chapters 2 and 3, it is expected that only crime propensity will fully mediate this association. As to its individual components, it is anticipated that all three variables will significantly mediate the relationship, wherein moral rules and moral emotions will demonstrate significantly stronger mediation effects in comparison to the ability to exercise self-control. It is further hypothesised that fear will be a non-significant mediator of the relationship between heart rate and crime, whereas stimulation-seeking will exhibit only partial mediation due to the confounding effects of impulsivity. Namely, the rationale for the latter hypothesis is based on the fact that stimulation-seeking and impulsivity are distinct but correlated constructs (Ellingson, Verges, Littlefield, Martin, & Slutske, 2013; Magid et al., 2007; Quinn & Harden, 2013; Steinberg et al., 2008), and that impulsivity is expected to be a significant mediator as per crime propensity hypothesis. It is therefore further anticipated that stimulation-seeking will exhibit non-significant mediating effect once the ability to exercise self-control is added to the mediation model testing the stimulation-seeking hypothesis.

4.3. Cambridge Adolescent Behaviour Study: Methods

The Cambridge adolescent behaviour study was specifically designed to bring to light the underlying factor connecting low heart rate to crime, and to ascertain which of the three proposed mediating models accounts for this relationship. The research proposal was reviewed and approved by the Institute of Criminology's Ethics Committee in May 2018, and the study itself was conducted from September 2018 to January 2019. It involved a sample of 487[7] Year 12 students recruited from 14 schools located across four East of England counties, namely Bedfordshire, Hertfordshire, Norfolk, and Suffolk. The schools were randomly selected from the official register of schools produced by the Department for Education[8]. In order to target only mainstream schools in the chosen areas, the list was firstly filtered across four categories: county (Bedfordshire, Hertfordshire, Norfolk, and Suffolk), students' age (Year 12 students), type of establishment (all types of schools except independent and special schools), and students' gender (only mixed gender schools, excluding boys- and girls-only schools). The refined list consisted of 141 schools out of which 80 were randomly selected to be invited to take part in the research. Letters of invitations were sent by post to the headteachers at the beginning of the school year, followed by e-mail reminders two weeks later in cases of no reply, and telephone calls as the last resort. Out of all contacted schools, 16 replied affirmatively to the invitations to take part in the study. The final sample consisted of 14 schools, as two schools had to drop out due to technical difficulties in accommodating research at their schools (specifically, lack of staff and a suitable room with computers). Six schools were located in Hertfordshire, three schools each in Suffolk and Norfolk, and two remaining schools were based in Bedfordshire. This distribution of schools across the counties was proportionate to the number of schools in these areas. Out of 141 schools eligible for the study, 64 were located in Hertfordshire, 30 in Norfolk, 25 in Suffolk, and only 22 in Bedfordshire. Therefore, the sample of schools followed the general trend of school allocation across these East of England counties.

According to the research proposal, in each school 50 Year 12 students would be randomly selected from the school register to be invited to participate in the study. This requirement was largely met, except in one case where a school could not implement random

[7] The total sample consisted of 493 participants, but six cases were omitted from the analyses as they clearly contained untruthful answers found across the questionnaires (e.g., reporting carrying bombs or shotguns to school, answering affirmatively to every crime question and reporting random frequencies for each crime).

[8] Retrieved on 1st August 2018 from https://www.gov.uk/government/publications/schools-in-england.

selection of students. Instead, all of their students who had taken Psychology and/or Sociology as their A-levels participated in the study (n = 32). They represented a third of the entire cohort and, according to the school, did not differ from the other students in terms of demographic characteristics and academic ability. Descriptive statistics confirmed that this group of students performed on all key variables within the range of scores set by other schools, and therefore were retained in the sample. The number of recruited students from each school ranged from 14 to 50, with an average of 35 participants per school (M = 34.79, SD = 10.43). Two schools had small sixth forms with just 17 and 27 Year 12 students in total (with 14 and 22 students attending the school on the day of the testing, respectively), and in one school testing was conducted on an open day when 21 selected students could not attend the testing. Across all the schools, only seven students declined to take part in the research. The pre-selected students who were not in the school on the day of the testing were not replaced by other students. Follow-up of the pre-selected students who did not take part in the research was unattainable as the schools could not accommodate another testing session due to the impact it had on school resources, specifically in terms of time taken out of lessons and very limited staff and room availability.

After the headteacher's approval of the study, arrangements were made with the school staff about technicalities of implementing the research protocol during the lesson hours. Selected students were usually divided into two groups to take part in the research, with a few instances when there was only one group. The size of the groups ranged from 12 to 32 participants (M = 20.29, SD = 4.97). At each school, the study was implemented in a computer room with internet access and with a researcher and a teacher present. The researcher began the session by explaining the research process which consisted of two parts, after which the students had the opportunity to ask questions. Following that, students opened an online platform on allocated computers which began with an informed consent form. If a student agreed to take part in the study after reading the information sheet, the platform would then proceed to the set of questionnaires for them to fill in. They firstly completed a few introductory demographic questions, followed by a set of neurocognitive tasks, after which they filled in self-report questionnaires regarding their morality, crime involvement, self-control, fearfulness, stimulation-seeking, and health and lifestyle. Completing the tasks and questionnaires presented on the online platform usually took about 45 minutes to an hour. The second part of the research process involved measuring heart rate. This was completed in groups of four students in the same computer room, but in a separate area with four tablets. Each student was seated in front of their tablet and they were given headphones and heart rate monitors to put on their upper arms. After a short introduction by the researcher, the students proceeded to watch a two-minute long relaxing video, followed by a stressful task which lasted for three minutes. After the last task had finished, the students received a debriefing note which contained the explanation of the stress task. Their heart rates were continuously monitored throughout both tasks. As participation in the study was completely anonymous, each student was given a unique identifying code so that their answers on the online platform could be matched with their heart rate measures. After all the students had completed both parts of the study, the researcher addressed the group and debriefed participants regarding the stress task and answered any questions that students may have had.

4.3.1. Participants

The sample included 487 16- and 17-year-old students attending Year 12. This particular age was chosen as the most suitable given the age-crime curve phenomenon, the observation that crime rate sharply increases in early adolescence up to a peak in mid to late adolescence, after which it decreases (Farrington, 1986; Loeber et al., 2015; Shulman, Steinberg, & Piquero, 2013). Academics have not yet reached a consensus about what causes this occurrence, but the correlation between age and crime is widely accepted as robust. When expressed graphically across the life-span, it takes an asymmetrical, positively skewed bell shape, indicating that there is an increase in the proportion of offenders throughout adolescence, which after reaching a peak, starts to drop off in late adolescence or early adulthood (Loeber & Farrington, 2014). As a general rule, the age-crime curve is unimodal with a peak between the ages of 14 and 17, after which it subsides (Wikström et al., 2012). There are some differences in findings across the studies regarding peak time of offending, as it changes depending on the type of crime (e.g., property crime peaks earlier than violent offences) and the method of assessing crime (e.g., self-reported crimes peak earlier than official crime records), but the overall trend remains the same (Farrington, 1986; Loeber, 2012; Moffitt, Caspi, Rutter, & Silva, 2001; Shulman et al., 2013; Wikström, 1990). According to the PADS+ data, offender prevalence peaked at the age of 13 to 14 years followed by a downturn, whereas the overall crime rate peaked at age 15 to 16 years old (Wikström et al., 2012). Therefore, a decision to recruit Year 12 students was made primarily because this age fits within the age range when crime engagement peaks. Alternatively, the study could have targeted Year 10 and 11 students, but the sample would likely have been much smaller as the students are involved in heavy preparations during this period for the General Certificate of Secondary Education examination being sat at the end of Year 11.

The sample contained a slightly higher proportion of female students, with 273 (56%) female students and 214 (44%) male students. As the study was conducted during the first term of Year 12, the majority of the participants were aged 16 at the time of the testing (n = 379, 78%), and the remainder had turned 17. In total, 29 students (6%) reported receiving free school meals. The information regarding their ethnicity and parents' education is presented in Table 4.1.

Table 4.1.

Frequencies of Ethnicity and Parents' Educational Level

Variable	Frequency	Percentage
Ethnicity		
White	400	82.1
Asian	46	9.4
Black	11	2.3
Mixed	22	4.5
Other	8	1.6
Total	487	100
Mothers' education		
Not completed compulsory school	27	5.5
Completed compulsory school (GCSE)	190	39.0
Completed A-levels	122	25.1
Completed University degree	148	30.4
Total	487	100
Fathers' education		
Not completed compulsory school	39	8.0
Completed compulsory school (GCSE)	185	38.0
Completed A-levels	115	23.6
Completed University degree	146	30.0
Total	485	99.6

Note. Mothers' education = the highest completed level of education by mothers; Fathers' education = the highest completed level of education by fathers; GCSE = General Certificate of Secondary Education.

4.3.2. Measures

The online platform containing the questionnaires began with an informed consent form, and if consent was given, the participants were presented with a short demographics questionnaire (see Appendix 1). Afterwards, participants completed two neurocognitive tasks, namely working memory and emotion recognition tasks. Finally, they completed self-report questionnaires tapping into different constructs as follows: moral rules, crime, shame, guilt, ability to exercise self-control, fearfulness, stimulation-seeking, and health and lifestyle. Table 4.2. contains the descriptive statistics for all measures used, and further details regarding each measure are presented throughout the following paragraphs. Reported descriptive statistics were calculated using IBM SPSS Statistics for Windows, Version 26.0, and the graphs were produced in R version 3.5.3 (R Core Team, 2019) using the packages ggplot2 3.1.0 (Wickham, 2016).

Table 4.2.

Descriptive Statistics of the Measures Used in the Cambridge Adolescent Behaviour Study

Variable	Mean	Median	SD	Min	Max	Range	*n*	α
Working memory	8.13	8.00	2.58	0	14	0-14	484	NA
Emotion recognition	31.12	32.00	4.46	7	40	0-40	484	.72
Crime frequency	14.24	1.00	59.59	0	795	0-3445	487	.48
Crime versatility	1.33	1.00	1.54	0	7	0-14	487	.60
Moral rules	3.10	3.11	0.38	1.00	3.95	1.00-4.00	487	.84
Shame	2.50	2.56	0.45	1.00	3.00	1.00-3.00	487	.88
Guilt	2.34	2.44	0.43	1.00	3.00	1.00-3.00	487	.85
AtE self-control	2.73	2.75	0.48	1.38	4.00	1.00-4.00	486	.68
Fearfulness	2.86	2.75	0.75	1.00	4.75	1.00-5.00	486	.81
Stimulation-seeking	3.34	3.36	0.71	1.00	5.00	1.00-5.00	485	.85

Note. SD = Standard deviation; α = Cronbach's alpha; Min = Minimal score obtained in the sample; Max = Maximal score obtained in the sample; Range = Minimum and maximum scores of the scales; *n* = size of the subsample used to calculate descriptive statistics for each variable; AtE self-control = Ability to exercise self-control.

4.3.2.1. Working memory.

Working memory refers to a neural system responsible for maintenance of information in an active state in a temporary storage and manipulation of these mental representations (Baddeley, 1998; Baddeley, 1992; Levy & Goldman-Rakic, 2000). These basic cognitive processes are necessary for higher-order functions, such as reasoning, planning, learning, and language comprehension. The prefrontal cortex represents the neural seat for the working memory functions, specifically its dorsolateral region (Barbey, Koenigs, & Grafman, 2013; Curtis & D'Esposito, 2003; D'Esposito, Postle, & Rypma, 2000; Levy & Goldman-Rakic, 2000).

In this study, working memory was assessed using a version of the Digit Span Backwards task adapted for computer use. It represents a common method of assessing working memory (Ramsay & Reynolds, 1995), which is often used as a subtest in many neuropsychological batteries, including Wechsler Adult Intelligence Scale (Wechsler, 2008). It taps into both maintenance and manipulation of information, and it relies heavily on the dorsolateral prefrontal cortex as shown in neuroimaging, repetitive transcranial magnetic stimulation, and human lesion studies (Aleman & Van'T Wout, 2008; Barbey et al., 2013; Gerton et al., 2004; Hoshi et al., 2000). Participants were presented with a sequence of numbers on a computer screen with an instruction to repeat them in a reverse order from the initial presentation. All numbers were typed in black font against a white background. Each number in a sequence was presented for one second, but the participants had unlimited time to provide an answer. Before the task started, participants firstly engaged in a practise trial consisting of two sequences of numbers to make sure they understood the instructions. The task consisted of seven trials, each involving two sequences of numbers of equal length. In the first trial, participants were presented with two different sequences consisting of two numbers. If answered correctly on at least one of the two sequences, they were presented with the next trial involving two different sequences consisting of three numbers. The sequences increased progressively for one number in every following trial as long as the participant correctly repeated at least one of the two sequences. After participant failed to repeat the numbers in a reverse order to both sequences in a trial, the task terminated. For each correctly answered sequence participants would be awarded one point, and therefore their total score could range from zero to 14 as there were seven trials each consisting of two sequences of numbers. The average capacity of participants' working memory was about five units (M = 4.74, SD = 1.49), which is in line with the results of prior studies involving the same age group (Grégoire & Van der Linden, 1997; Hooper, Luciana, Conklin, & Yarger, 2004). As can be seen in histogram presented in Figure 4.1, scores were relatively normally distributed. The sequences of numbers used in this study are listed in the Appendix 2, whereas the task's descriptive statistics are to be found in Table 4.2.

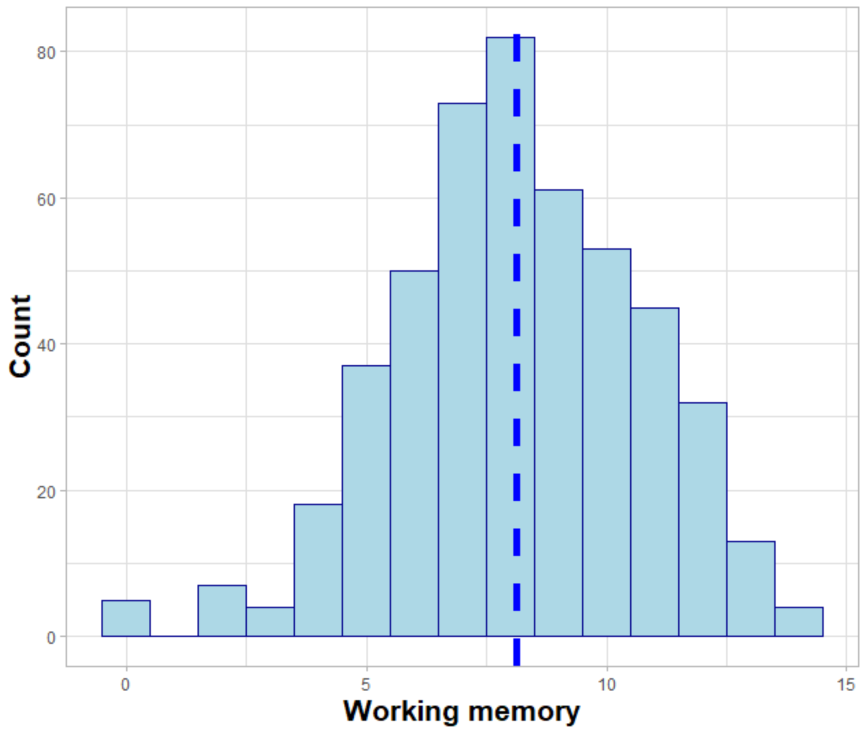

Figure 4.1. Frequency distribution of the Digit Span Backwards task. Dashed line represents the mean value of working memory (*n* = 484).

4.3.2.2. Emotion recognition.

Ability to recognise emotional expressions in other people was assessed with the Facial Emotion Recognition Task (Wolf, Philippi, Motzkin, Baskaya, & Koenigs, 2014). Each trial began with a fixation cross presented for 4 ± 1 seconds, after which participants were shown a photograph of a person's face with an emotional expression for three seconds. Participants then had an unlimited time to choose the emotion shown from a list of six emotions. Stimuli consisted of photographs of ten actors and ten actresses, each showing two different emotions, which were chosen from the Karolinska directed emotional faces set (Lundqvist, Flykt, & Ohman, 1998). The photographs were converted to greyscale, cropped to include only faces, matched for size, and presented so that the tip of the nose would always appear at the same spot where the fixation cross had appeared beforehand. There were 40 photos in total, with seven photos each of anger, sadness, happiness, fear, and disgust, and five photos showing neutral expressions. Stimuli were presented in a random order. Participants received one point for each correctly recognised emotion, and could therefore achieve a sum score between zero and 40. The histogram presented in Figure 4.2. suggests a somewhat negatively skewed distribution. A relatively high success rate in the Facial

Emotion Recognition Task is in line with other studies measuring the ability to recognise emotion using either the same (Wolf et al., 2014) or a different instrument (Lawrence, Campbell, & Skuse, 2015; Pajevic, Vukosavljevic-Gvozden, Stevanovic, & Neumann, 2018; Voyer, Russell, & McKenna, 2002; Wright, Riedel, Sechrest, Lane, & Smith, 2018). Further descriptive statistics about the emotion recognition task are presented in Table 4.2.

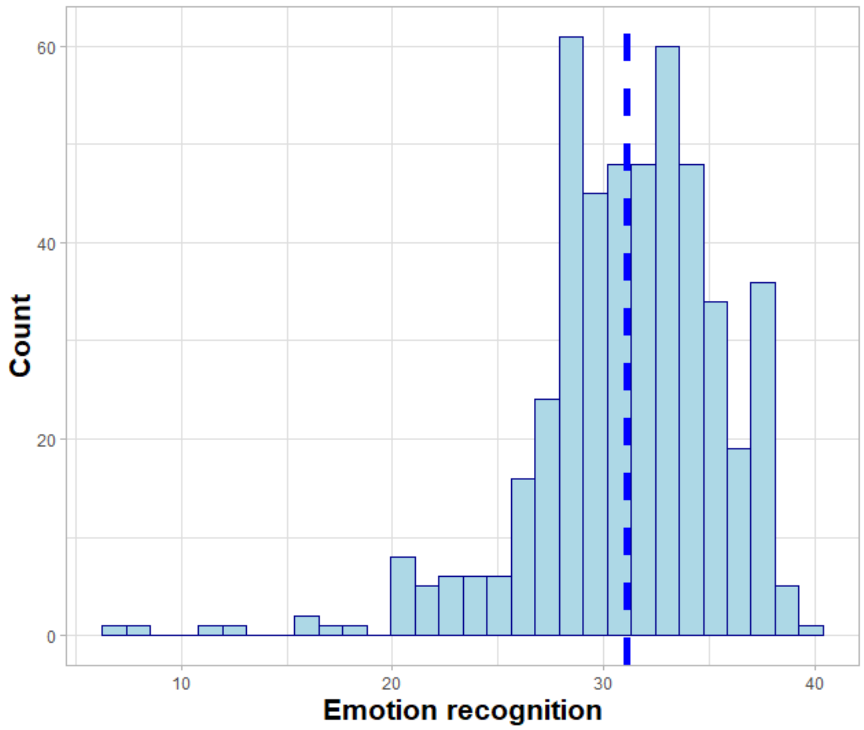

Figure 4.2. Frequency distribution of the Facial Emotion Recognition Task. Dashed line represents the mean value of emotion recognition (*n* = 484).

4.3.2.3. Crime.

Engagement in criminal behaviour was assessed using an adapted and extended PADS+ crime questionnaire (Wikström et al., 2012). The original PADS+ questionnaire explored 10 different crimes: shoplifting, theft from a person, theft from a car, theft of a car, residential burglary, non-residential burglary, robbery, assault, vandalism, and arson. In addition to the original items, three new items[9] were added which tapped into some of the most common types of cyber-crime: unauthorised access (to a computer system or an online

[9] The items were developed in collaboration with Dr Alice Hutchings, University Lecturer in the Security Group at the Computer Laboratory, University of Cambridge.

account), malware[10] (its distribution or development), and denial of service attack[11]. These acts are criminal in the United Kingdom under the Computer Misuse Act from 1990. Therefore, the crime scale measured 13 different types of crimes in total.

As PADS+ was a longitudinal study, its questions regarding criminal engagement referred to a one year period. However, as the Cambridge adolescent behaviour study is a cross-sectional study, all the questions were rephrased to cover longer periods. Specifically, participants were firstly asked whether they had committed each of the crimes since they had started secondary school (hence the questions covered a five year period). If answered affirmatively, they were presented with an additional question which inferred how many times had they committed the crime in question during the same period. Two total crime scores were then calculated for each participant, one for crime versatility based on answers to the first question, and the other for crime frequency based on answers to the second question. They each measured the same 13 crimes, with an exception that crime versatility score included an additional item regarding carrying weapons[12] and therefore tapped into 14 different crimes.

Crime can be measured using frequency or versatility scores, with each having their own specific advantages and disadvantages. Crime frequency, measured as a sum of numbers of times a participant had committed each crime, represents a more nuanced measure as it distinguishes between people who had engaged in crime once, a few times, or who are frequent offenders. On the other hand, it may be difficult for some people to provide a correct number of times they had committed crime since the question applies to a relatively long period of time, especially if they had offended often. Crime versatility refers to a number of types of crimes an individual had engaged in, regardless of their individual frequency. Versatility scores in general show higher reliability and are less skewed than frequency scores, but on the other hand they are less precise as they fail to make a crucial difference between people who have committed a crime once and those who offend regularly (Hirtenlehner & Kunz, 2016). For example, a crime frequency score would be more

[10] Malicious software, or malware, refers to computer programs used for breaking into, or compromising, computer systems (The Crown Prosecution Service, 2020).

[11] Denial of service attack is a cyber-attack in which the perpetrator seeks to make a machine or network resource unavailable to its intended users by disrupting services of a host using incoming traffic originating from many different sources flooding the victim (The Crown Prosecution Service, 2020).

[12] In the original PADS+ data, items measuring carrying knives or other weapons were not part of the crime questionnaire, but another set of questions. The question tapping into the frequency of carrying weapons had a four point Likert-type response scale with "always", "most times", "sometimes", and "not very often" as answer options. Therefore, it was not possible to add carrying weapons to the total crime frequency score due to the different response scale used in comparison to other crime frequency items.

informative than a crime versatility score for a person who frequently commits only one type of crime (e.g., if they had committed 15 assaults, their crime frequency score would be 15, whereas their crime versatility score would be one). In such a case, crime frequency would accurately describe the level of crime involvement of that particular person, whereas the crime versatility score would undervalue the extent of their criminal behaviour.

In crime frequency questions, participants were asked to enter a number of times they had committed the crime in question since they had started secondary school. This method is more informative than simply using a multiple choice format based on categorical answers which merges very different values into the same category. However, it can also create a problem with outliers as no restrictions were placed on possible answer values. Indeed, in several cases participants gave answers which were clearly imprecise crime frequencies (e.g., some participants reported they had committed a particular crime a million times, 100 000 times, 999 times, etc.). Therefore, these values had to be dealt with either by capping the values or excluding these participants from the analyses. In case of each participant who reported an unusual crime frequency, their answers to all questionnaires were carefully examined. As a result, information obtained from six participants were omitted from the analyses as they gave dubious answers throughout the questionnaires. For participants whose answers looked legitimate, their crime frequency was capped in cases of extreme values. According to the PADS+ data, between the ages of 12 and 16, a subsample of most frequent offenders committed on average one crime per week. Drawing from these results, any extreme crime frequency value was capped to 265, which is the number of crimes the participants would have committed had they committed one crime per week between the start of their secondary school until the time of the testing. Twelve values were capped in total, wherein five values were capped for vandalism, three for shoplifting, two for assault, and one value each for robbery and malware use. These capped values belonged to nine participants in total, two female and seven male students.

Both crime frequency and, to a lesser extent, crime variability, were positively skewed. The piling up of low scores in the crime distribution is a common finding in criminological studies (e.g., Bushway, Paternoster, & Brame, 2003; Grasmick, Tittle, Bursik, & Arneklev, 1993; Moffitt, Krueger, Caspi, & Fagan, 2000; Vazsonyi, Clifford Wittekind, Belliston, & Van Loh, 2004; Wikström et al., 2012), and is expected given that crime is a relatively rare phenomena. Therefore, both variables were log transformed in order to bring them closer to a normal distribution. Crime variables were expressed through log transformed values in all subsequent analyses, except when calculating basic descriptive statistics, the results of which are shown in Table 4.2. For the ease of presentation, only analyses wherein crime was measured using log transformed crime frequency scores will be reported here. However, all the analyses were replicated using log transformed crime versatility scores and will be reported in case there were significant differences in results. Otherwise, they will be omitted to avoid unnecessary repetitions. Distribution of crime frequency before and after log transformation is presented in Figure 4.3. Crime questionnaire is presented in Appendix 3.

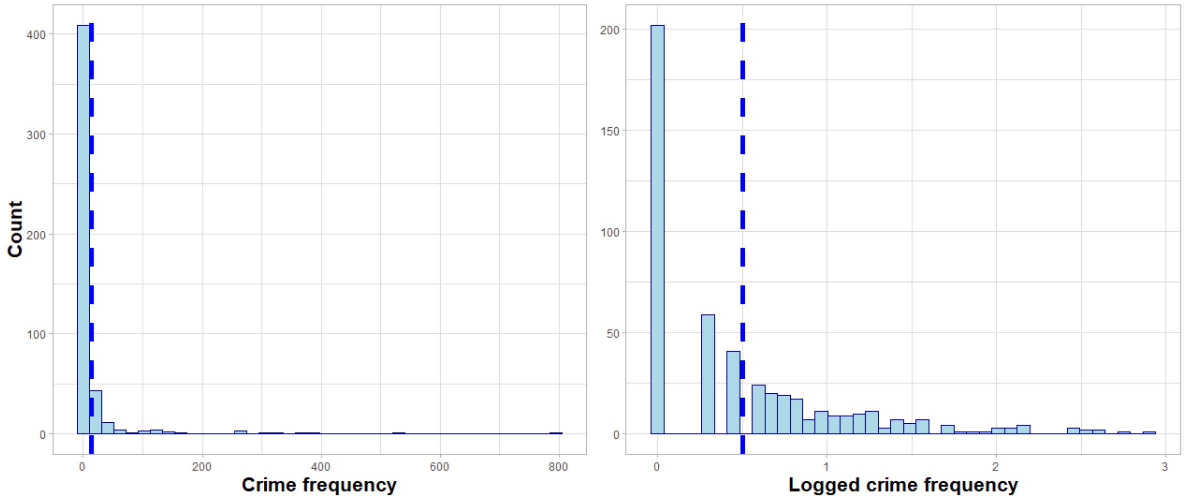

Figure 4.3. Frequency distribution of crime frequency. Figure on the left denotes the histogram of untransformed crime frequency scores. Figure on the right denotes the histogram of log transformed crime frequency scores. Dashed lines represent the mean values of crime frequency and logged crime frequency on the left and right histograms, respectively ($N = 487$).

In total, more than a half of the participants ($n = 288$, 59%) reported having committed at least one crime since they had started secondary school, which is slightly lower than what was found in the PADS+ sample (70%; Wikström et al., 2012). About 23% ($n = 111$) committed only one type of crime, whereas more than a third ($n = 177$, 36%) committed two or more types of crime. The most versatile offenders ($n = 4$, 1%) engaged in seven out of 14 listed crimes. Amongst those who reported criminal behaviour ($n = 285$[13], 59%), the crime frequency ranged from one to 795 crimes per person ($M = 24.33$, $Mdn = 4.00$[14], $SD = 76.36$). About 12% ($n = 59$) of the entire sample committed a crime only

[13] In total, 288 participants responded positively to at least one crime versatility question. Out of those, two reported carrying weapons as their only crime which was not followed up by a frequency question (i.e., how many times they had done it), and one participant did not respond to a crime frequency question that followed after he reported having committed a crime. Therefore, using a crime frequency variable 285 participants reported committing at least one crime, whereas according to the crime versatility variable this number is 288.

[14] Taking into account the skewed nature of crime data, the median was included as an additional measure of central tendency as it is relatively unaffected by outliers (Field, 2018).

once. The most common types of self-reported crimes were assault (*n* = 129, 27%), unauthorised access to computer material (*n* = 125, 26%), theft from a person (*n* = 87, 18%), vandalism (*n* = 86, 18%), and arson (*n* = 85, 18%). None of the students responded positively to the questions whether they had ever committed residential burglary and theft from a car. A similar pattern of the most prevalent offences was found in the PADS+ data as well, but their prevalence rates were higher. The most common types of crimes in PADS+ were assault (56%), vandalism (38%), arson (36%), shoplifting (34%), and theft from person (25%; Wikström et al., 2012).

Descriptive statistics for all[15] types of crime are presented in Table 4.3. Due to the positively skewed nature of crime (e.g., Falk et al., 2014; Wikström et al., 2012), the central tendency was described using two measures, mean and median. Taking into account both measures enables a more balanced description of participants' crime involvement as they have specific advantages and disadvantages. The mean is calculated using every score, and is therefore also driven by extreme values, whereas the median is relatively unaffected by outliers but at a cost of discarding most of the scores (Field, 2018).

The variable *carrying a weapon* reported in Table 4.3. requires a special note given the lack of its descriptive statistics. Information regarding its prevalence was calculated using data from the crime versatility question enquiring whether participants had ever carried a knife or other weapons when they went to school or outdoors. Participants who responded affirmatively were asked a frequency related follow-up question which measured how often they carried a weapon (*n* = 12, 3%). However, participants responded to this question by choosing one of four possible descriptive answers, namely "always", "most times", "sometimes", and "not very often". Therefore, descriptive statistics regarding frequency of carrying weapons could not be calculated as in case with other types of crime. Out of 12 participants who reported carrying weapons, eight replied with "not very often", and two each responded with "most times" and "sometimes".

[15] Except for theft from a car and residential burglary, as none of the students reported affirmatively to the questions tapping into these crimes.

Table 4.3.

Descriptive Statistics of Self-Reported Crime

Crime	Prevalence	*M*	*Mdn*	*SD*	Min	Max
Shoplifting	77 (15.8%)	16.03	3.00	52.06	1	265
Non-residential burglary	3 (0.6%)	5.33	3.00	5.86	1	12
Theft from a person	87 (17.9%)	5.79	2.00	12.82	1	100
Theft of a car	1 (0.2%)	1.00	1.00	NA	1	1
Vandalism	86 (17.7%)	21.83	2.00	62.89	1	265
Arson	85 (17.5%)	6.71	3.00	12.67	1	69
Robbery	21 (4.3%)	20.52	4.00	58.05	1	265
Assault	129 (26.5%)	8.71	2.00	34.92	1	265
Carrying a weapon	12 (2.5%)	NA	NA	NA	NA	NA
Unauthorised access	125 (25.7%)	6.62	2.00	16.29	1	100
Malware	8 (1.6%)	52.33	7.50	104.83	1	265
Denial of service	12 (2.5%)	3.00	1.00	6.00	1	22

Note. M = Mean; *Mdn* = Median; *SD* = Standard deviation; Min = Minimal reported value of crime frequency; Max = Maximal reported value of crime frequency.
Prevalence of crime was calculated using the crime versatility data from the full sample (*N* = 487). The remainder of descriptive statistics refers to crime frequency data from the offender sub-samples. Specifically, *M, Mdn, SD,* Min, and Max were all calculated using crime frequency data from sub-samples of participants who reported they had committed a crime for which the descriptive statistics are reported. The number of participants in each sub-sample ranged from *n* = 1 for theft of a car to *n* = 129 for assault.

As cyber-crime items were new and exploratory, they were followed up by additional questions. Participants who reported having accessed another person's computer or an online account without permission were asked what type of material they had accessed and by what means (*n* = 125, 26%). Most often participants accessed another person's social networking account (*n* = 65, 13%), e-mail (*n* = 47, 10%), and computer files, such as photos, videos, and documents (*n* = 33, 7%). In a few instances, participants would access paid services, such as school account (*n* = 5, 1%), Netflix or Spotify account (*n* = 3, 1%), and games account (*n* = 2, < 1%). In only one case a participant reported accessing another person's bank account. Most often participants would gain unauthorised access to an account by using non-sophisticated measures such as guessing a password (*n* = 48, 10%), entering an account after the owner had forgotten to log out (*n* = 42, 9%), or watching the owner enter their log-in details (*n* = 30, 6%). Only three participants had used a more advanced methods involving a brute force attack (*n* = 2, < 1%) and keylogger (*n* = 1, < 1%).[16]

Participants who reported having used malware were asked about the type of malware they usually used (*n* = 8, 2%). The participants reported using different malicious software, including spyware (*n* = 4, < 1%), viruses (*n* = 3, < 1%), worms (*n* = 2, < 1%), spam programs (*n* = 1, < 1%), and malware specialised to disrupt Android mobile operating system (*n* = 1, < 1%). None of the participants reported using ransomware software or Trojans.

Finally, participants who admitted to deploying a denial of service attack were asked about their motivation behind these acts (*n* = 12, 3%). Seven participants reported having committed these attacks for fun, three said it was a retaliatory measure, two had done it due to social or political reasons (i.e., hacktivism), and four reported other reasons such as to test programs or "show authority". None of the participants reported having committed a denial of service attack to win in an online game or for financial gain.

4.3.2.4. Moral rules.

Personal moral rules were assessed using an extended version of the PADS+ generalised morality scale (Wikström et al., 2012). The original scale included 16 questions tapping into different acts of breaking rules, both legal and illegal, which differed by the level of seriousness. As with the crime scale, the morality scale was enriched by adding three new items tapping into personal morals regarding committing three cyber-crimes, namely unauthorised access, distribution or development of malware, and deployment of denial of service attack. Each item asked participants to assess how wrong it would be for someone their age to do a specific act. They gave their answers on a four point Likert-type response scale where answers ranged from "not wrong at all" to "very wrong". The total score was calculated as a mean across all scores, and could be in range between one, indicating a low

[16] Brute force attack is a more sophisticated mean of guessing password, whereby the attacker consecutively generates a large number of possible passwords until the correct one is identified. Keylogger is a surveillance program which records the keys struck on a keyboard, and thereby provides a mean of accessing password details once the owner types it in.

morality, and four, indicating high morality. Descriptive statistics of the moral rules scale are presented in Table 4.2., whereas the distribution of scores is showed on a histogram depicted in Figure 4.4.

Figure 4.4. Frequency distribution of moral rules. Dashed line represents the mean value of moral rules (*N* = 487).

The moral rules scale and distribution of participants' answers to each item are presented in Table 4.4. On average, participants showed a relatively high morality (*M* = 3.10, *SD* = 0.38), which is slightly higher in comparison to the PADS+ finding for the same age group (*M* = 1.83, *SD* = 0.65; wherein the range of scores was between zero and three; Wikström et al., 2012). There was a very similar pattern of results regarding the average "wrongfulness" rankings across the items in the Cambridge adolescent behaviour study and PADS+. Acts perceived as most wrong on average by both samples were using a weapon or force to get money or things from another person, breaking into a building to steal something, smashing streetlights for fun, spraying graffiti on a house wall, and stealing a CD from a shop. Acts perceived as the most morally permissible by both groups were getting drunk with friends, skipping homework, and stealing a pencil. Committing selected cyber-crimes was described on average as wrong to very wrong.

Table 4.4.

Frequencies of Answers on Moral Rules Scale

How wrong it is for someone your age to…	Not wrong at all (=1)	A little wrong (=2)	Wrong (=3)	Very wrong (=4)	*M*	*SD*
Steal a pencil from a classmate	62 (13%)	324 (67%)	85 (18%)	16 (3%)	2.11	0.65
Skip doing homework for school	45 (9%)	260 (53%)	160 (33%)	22 (5%)	2.33	0.70
Ride a bike through a red light	31 (6%)	90 (19%)	208 (43%)	158 (32%)	3.01	0.88
Go skateboarding in a place where skateboarding is not allowed	41 (8%)	193 (40%)	200 (41%)	53 (11%)	2.54	0.80
Hit another young person who make a rude comment	51 (11%)	129 (27%)	188 (39%)	119 (24%)	2.77	0.94
Lie, disobey, or talk back to teachers	21 (4%)	112 (23%)	245 (50%)	109 (22%)	2.91	0.79
Get drunk with friends on a Friday evening	268 (55%)	134 (28%)	52 (11%)	33 (7%)	1.69	0.91
Smoke cigarettes	64 (13%)	120 (25%)	117 (24%)	186 (38%)	2.87	1.07
Skip school without an excuse	14 (3%)	113 (23%)	243 (50%)	117 (24%)	2.95	0.77
Tease a classmate because of the way he or she dresses	10 (2%)	38 (8%)	139 (29%)	300 (62%)	3.50	0.73
Smash a street light for fun	5 (1%)	11 (2%)	97 (20%)	374 (77%)	3.72	0.55
Paint graffiti on a house wall	4 (1%)	14 (3%)	116 (24%)	353 (73%)	3.68	0.57
Steal a CD from a shop	3 (1%)	19 (4%)	174 (36%)	291 (60%)	3.55	0.60
Smoke cannabis	72 (15%)	126 (26%)	118 (24%)	171 (35%)	2.80	1.08

How wrong it is for someone your age to…	Not wrong at all (=1)	A little wrong (=2)	Wrong (=3)	Very wrong (=4)	*M*	*SD*
Break into or try to break into a building to steal something	2 (0%)	3 (1%)	62 (13%)	420 (86%)	3.85	0.41
Use a weapon or force to get money or things from another young person	2 (0%)	1 (0%)	19 (4%)	465 (96%)	3.94	0.29
Access someone else's computer, e-mail, or an online account without their permission	6 (1%)	28 (6%)	179 (37%)	273 (56%)	3.48	0.66
Develop or distribute malware or other harmful software to deliberately damage or infect another person's computer	5 (1%)	13 (3%)	96 (20%)	373 (77%)	3.72	0.56
Use an online attack to disrupt an online service or an individual's network connection	4 (1%)	52 (11%)	158 (32%)	273 (56%)	3.44	0.71

Note. M = Mean; *SD* = Standard deviation.
Distribution of scores was calculated using the full sample (*N* = 487).

4.3.2.5. Moral emotions: Shame and guilt.

Tendency to experience shame and guilt after a wrongdoing were assessed using extended versions of the shame and guilt scales used in PADS+ (Wikström et al., 2012). The shame and guilt scales utilised in this study had nine items each, after three cyber-crime items were added to each of the original six item measures. Table 4.2. contains the descriptive statistics pertaining to the moral emotions scales.

The shame scale measured how much the participants would feel ashamed in front of others if caught shoplifting, breaking into a car, or accessing someone's computer without permission. There were three questions for each offence measuring how much the participants would feel ashamed in front of their best friends, teachers, and parents. Participants gave their answers on a three point Likert-type response scale, involving "no, not at all", "yes, a little", and "yes, very much" response options. The total score was calculated as a mean of all items, and could range from one, indicating low tendency to experience shame, to three, indicating high tendency to experience shame. The distribution of scores was negatively skewed to some extent as can be seen in Figure 4.5.

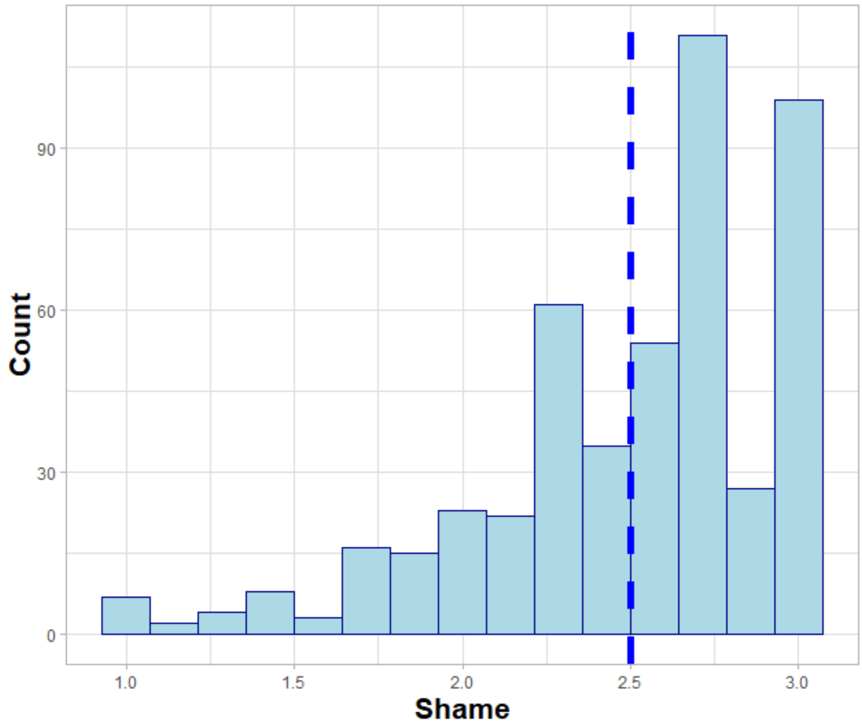

Figure 4.5. Frequency distribution of tendency to experience shame. Dashed line represents the mean value of shame (*N* = 487).

The shame scale and the distribution of participants' answers to each item are presented in Table 4.5. Participants on average reported relatively strong tendency to experience shame (*M* = 2.50, *SD* = 0.45) should they break certain rules, which is similar to the results obtained in PADS+ (*M* = 56.5, *SD* = 13.4, wherein the maximum value was 72; Trivedi-Bateman, 2004). As can be seen, participants' experience of shame differs as a function of who found out about their wrongdoings. There was a general trend amongst participants to feel least ashamed if caught by their best friends, more ashamed if caught by their teacher, and most ashamed if caught by their parents for committing the same crime. Overall, participants anticipated feeling least ashamed if their best friends learnt they accessed another person's computer or an online account without a permission. They reported they would feel most ashamed if they shoplifted or broke into a car and their parents found out about it.

Table 4.5.

Frequencies of Answers on Shame Scale

Items	No, not at all (=1)	Yes, a little (=2)	Yes, very much (=3)	*M*	*SD*
If you were caught shoplifting and…					
.. your best friends found out about it, would you feel ashamed?	89 (18%)	193 (40%)	205 (42%)	2.24	0.74
…your teachers found out about it, would you feel ashamed?	39 (8%)	133 (27%)	315 (65%)	2.57	0.64
…your parents found out about it, would you feel ashamed?	13 (3%)	52 (11%)	422 (87%)	2.84	0.43
If you were caught breaking into a car and…					
.. your best friends found out about it, would you feel ashamed?	54 (11%)	180 (37%)	253 (52%)	2.41	0.68
…your teachers found out about it, would you feel ashamed?	31 (6%)	109 (22%)	347 (71%)	2.65	0.60
…your parents found out about it, would you feel ashamed?	11 (2%)	35 (7%)	441 (91%)	2.88	0.39
If you were caught accessing a machine and…[17]					
.. your best friends found out about it, would you feel ashamed?	117 (24%)	207 (43%)	163 (34%)	2.09	0.75
…your teachers found out about it, would you feel ashamed?	66 (14%)	177 (36%)	244 (50%)	2.37	0.71
…your parents found out about it, would you feel ashamed?	56 (12%)	147 (30%)	284 (58%)	2.47	0.69

Note. *M* = Mean; *SD* = Standard deviation.
Distribution of scores was calculated using the full sample (*N* = 487).

The guilt scale assessed how much the participants would feel guilty had they broken certain moral rules. The original six item scale was enriched for three new cyber-crime items tapping into unauthorised access, development or distribution of malware, and deployment of

[17]This full sentence was: “If you were caught accessing someone else's computer, email or an online account without their permission, and…”.

a denial of service attack. Participants gave their answers on the same three point Likert-type response scale as in the case of the shame scale, and the total score was calculated in the same manner, with low scores indicating low tendency to experience guilt and high scores indicating high tendency to experience guilt. The histogram of scores on the guilt scale shows a somewhat negatively skewed distribution as presented in Figure 4.6.

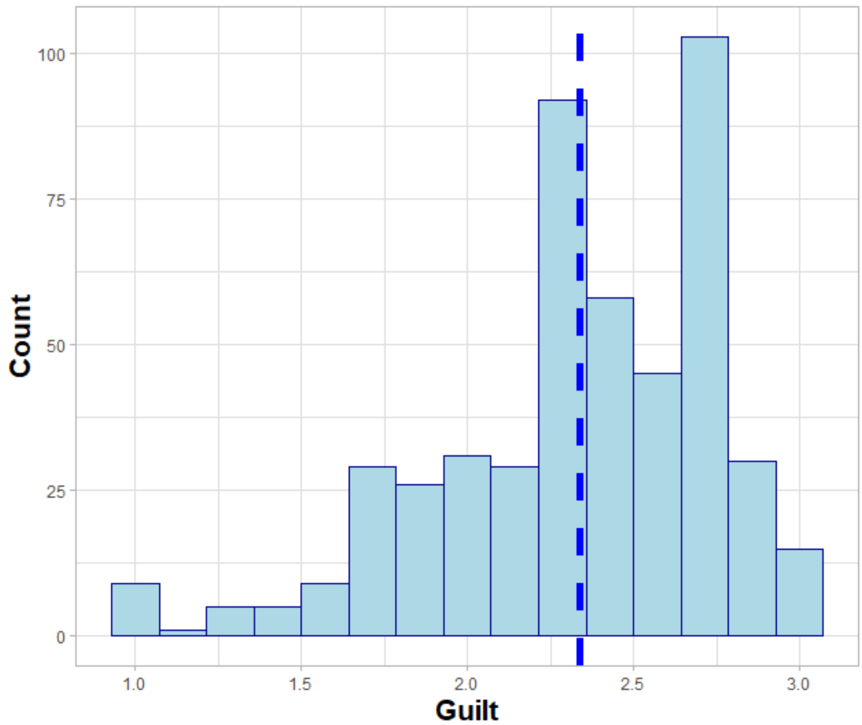

Figure 4.6. Frequency distribution of tendency to experience guilt. Dashed line represents the mean value of guilt (*N* = 487).

The guilt scale and the distribution of participants' answers to each item are presented in Table 4.6. Participants on average reported relatively strong tendency to experience guilt (*M* = 2.34, *SD* = 0.43) should they break certain rules, which is similar to the results obtained in PADS+ (*M* = 50.7, *SD* = 13.2, wherein the maximum value was 72; Trivedi-Bateman, 2004). Regarding their answers on the guilt scale, participants would feel least guilty if they cheated on a test or hit another person who made a rude remark. They would feel most guilty if they broke into a car, teased another student so they started to cry, or deliberately damaged or infected another person's computer with malware.

Moral emotions were somewhat negatively skewed in both the Cambridge adolescent behaviour study and PADS+, with shame exhibiting more pronounced skew in the distribution of scores. Furthermore, in both studies participants reported lower average tendency to experience guilt in comparison to the average tendency to experience shame.

Table 4.6.

Frequencies of Answers on Guilt Scale

Items	No, not at all (=1)	Yes, a little (=2)	Yes, very much (=3)	*M*	*SD*
Would you feel guilty if you…					
…did something your parents have told you absolutely not to do?	56 (12%)	279 (53%)	152 (31%)	2.20	0.62
…cheated on a test?	152 (31%)	250 (51%)	85 (18%)	1.86	0.69
…teased another student so they started to cry?	28 (6%)	109 (22%)	350 (72%)	2.66	0.58
…stole something from a shop?	41 (8%)	163 (34%)	283 (58%)	2.50	0.65
…hit another student who made a rude remark to you?	220 (45%)	192 (39%)	75 (15%)	1.70	0.72
…broke into a car and stole something?	11 (2%)	72 (15%)	404 (83%)	2.81	0.45
…accessed someone else's computer, email or an online account without their permission?	55 (11%)	232 (48%)	200 (41%)	2.30	0.66
…developed or distributed malware to deliberately damage or infect another person's computer?	31 (6%)	123 (25%)	333 (68%)	2.62	0.60
…used an online attack to disrupt an online service or an individual's network connection?	66 (14%)	165 (34%)	256 (53%)	2.39	0.71

Note. *M* = Mean; *SD* = Standard deviation.
Distribution of scores was calculated using the full sample (*N* = 487).

4.3.2.6. Ability to exercise self-control.

The ability to exercise self-control was assessed using a version of the low self-control scale (Grasmick et al., 1993) modified to fit the definition of poor ability to exercise self-control as defined by situational action theory (Wikström et al., 2012). The original low self-control scale aims to measure self-control as described in the general theory of crime

(Gottfredson & Hirschi, 1990). It contains 24 items tapping into six components of low self-control: impulsivity, preference for simple tasks, risk-seeking, preference for physical activity, self-centredness, and volatile temper (i.e., low frustration tolerance; Grasmick et al., 1993).

However, according to situational action theory, self-control is a situational concept which is the product of an interaction between a person's ability to exercise self-control and their environment (Wikström & Treiber, 2007). That is, self-control refers to a behaviour, something that a person does (or does not do) in a specific setting, and not what they are. The scale was therefore modified to capture self-control as the ability to manage conflicting rule-guidance and to uphold personal morality in the face of pressure to do otherwise. It captures the general need to exercise self-control by asking about experiences of exhibiting self-control generally. This was achieved by selecting some of the fitting items from the original scale and developing some new items. The modified scale disregards self-centredness and preference for physical activity as they are not relevant to situational action theory's definition of self-control, and taps only into impulsivity, risk-taking, and orientation towards the future.

The modified scale consists of eight statements describing poor ability to exercise self-control. Participants were asked to state how well each item described them by using a four point Likert-type response scale ranging from "strongly agree" to "strongly disagree". The total score was measured as a mean across all items, and it could range between one, indicating a poor ability to exercise self-control, and four, indicating strong ability to exercise self-control. The scores were relatively normally distributed as shown in the histogram presented in Figure 4.7. The mean value of the total sample (M = 2.73, SD = 0.48) was in line with the findings from PADS+ (M = 1.32, SD = 0.37; wherein the range of scores was between zero and three, with high score indicating poor ability to exercise self-control; Wikström et al., 2012). The descriptive statistics of the scale are shown in Table 4.2.

Figure 4.7. Frequency distribution of ability to exercise self-control. Dashed line represents the mean value of the ability to exercise self-control (*n* = 486).

The distribution of the participants' answers to each item of the ability to exercise self-control scale is presented in Table 4.7. The pattern of mean scores across the items is relatively similar to the results found in PADS+ for the same age group (Wikström et al., 2012).

Table 4.7.

Frequencies of Answers on Ability to Exercise Self-Control Scale

Item	Strongly agree (=1)	Mostly agree (=2)	Mostly disagree (=3)	Strongly disagree (=4)	*M*	*SD*
When I am really angry, other people better stay away from me	44 (9%)	169 (35%)	187 (38%)	86 (18%)	2.65	0.87
I often act on the spur of the moment without stopping to think	60 (12%)	156 (32%)	210 (43%)	60 (12%)	2.56	0.86
I sometimes find it exciting to do things that may be dangerous	67 (14%)	189 (39%)	156 (32%)	74 (15%)	2.49	0.91
I don't devote much thought and effort preparing for the future	23 (5%)	110 (23%)	181 (37%)	172 (35%)	3.03	0.88
Sometimes I will take a risk just for the fun of it	56 (12%)	184 (38%)	176 (36%)	70 (14%)	2.53	0.88
I often try to avoid things that I know will be difficult	56 (12%)	175 (36%)	206 (42%)	49 (10%)	2.51	0.83
I never think about what will happen to me in the future	18 (4%)	40 (8%)	176 (36%)	252 (52%)	3.36	0.79
I lose my temper pretty easily	54 (11%)	137 (28%)	177 (36%)	118 (24%)	2.74	0.95

Note. *M* = Mean; *SD* = Standard deviation.
Distribution of scores was calculated using *n* = 486.

4.3.2.7. Crime propensity.

As mentioned in Chapter 3, a person's crime propensity consists of personal morality and the ability to exercise self-control. In PADS+ the composite measure for crime propensity was calculated by adding standardised scores of the scales measuring moral rules and ability to exercise self-control, wherein the former was previously rescaled so that high scores reflected low morality (Wikström et al., 2012). However, given the importance of the moral emotions for the explanation of the relationship between heart rate and crime as explained in Chapter 3 (see section 3.4.), the crime propensity score was calculated using the total scores of moral rules, moral emotions, and the ability to exercise self-control. The crime propensity score was therefore calculated in a similar manner as to that in PADS+, with the addition of the moral emotions measure. A step by step procedure of calculating crime propensity score is presented below.

Firstly, moral rules, moral emotions, and ability to exercise self-control were rescaled so that high scores represented weak personal morality and poor ability to exercise self-control. Secondly, z-scores were calculated for each of the rescaled variables. Finally, these three z-scores were summed to create a composite measure of crime propensity. High score represented high crime propensity, that is, low ability to exercise self-control and weak personal morality, both in terms of moral rules and moral emotions. As can be seen in Figure 4.8., the resulting crime propensity variable had a relatively normal distribution, which is in line with the results from PADS+ (Wikström et al., 2012).

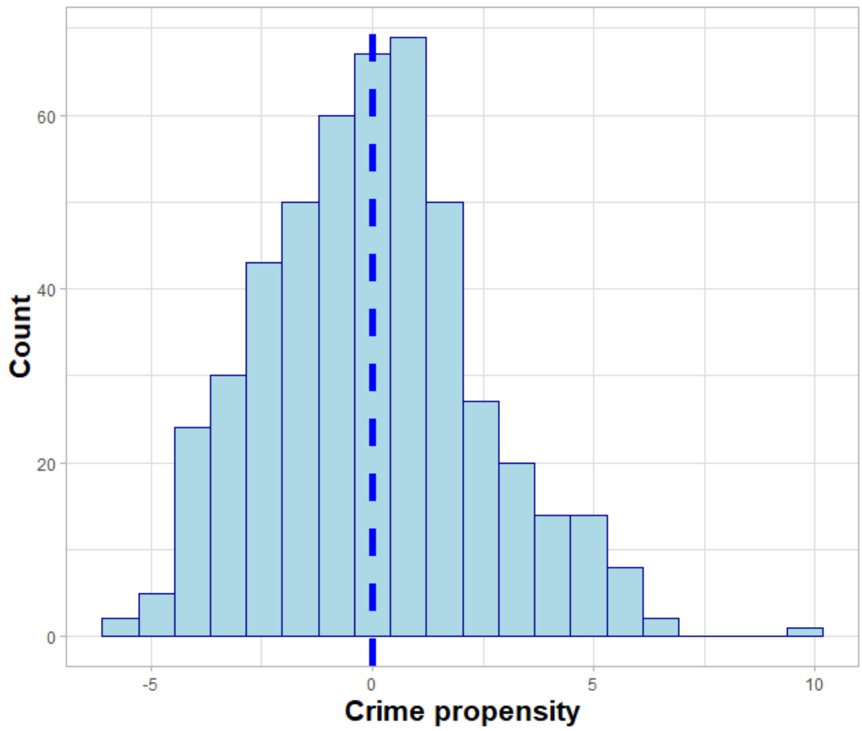

Figure 4.8. Frequency distribution of crime propensity. Dashed line represents the mean value of crime propensity (*n* = 486).

4.3.2.8. Fearfulness.

The tendency to experience fear was measured using a fearfulness facet of the emotionality factor from the HEXACO Personality Inventory Revised (Lee & Ashton, 2006). The inventory measures a six dimensional model of personality structure named HEXACO[18], a widely used framework to capture variations in human personality which has consistently shown considerable advantages over other models (Ashton & Lee, 2007, 2008, 2018; Ashton, Lee, & de Vries, 2014; Lee & Ashton, 2014). The fearfulness facet measures tendency to experience fear in different situations. It consists of eight statements which participants were asked to respond how much they agreed with. Answers were given using a five point Likert-type response scale where the provided options ranged from "strongly disagree" to "strongly agree". The total score was computed as a mean across all items, after reverse-keyed items were recoded. It could range between values of one and five. Participants who score low on the scale tend to feel little fear and are relatively insensitive to physical harm, whereas those who have high scores are fearful and tend to avoid situations that could be potentially

[18] H stands for Honesty–Humility, E for Emotionality, X for Extraversion, A for Agreeableness, C for Conscientiousness, and O for Openness to Experience (Ashton & Lee, 2001, 2007)

dangerous (Ashton et al., 2014). The histogram in Figure 4.9. shows a relatively normal distribution of fearfulness scores, and the relevant descriptive statistics are presented in Table 4.2.

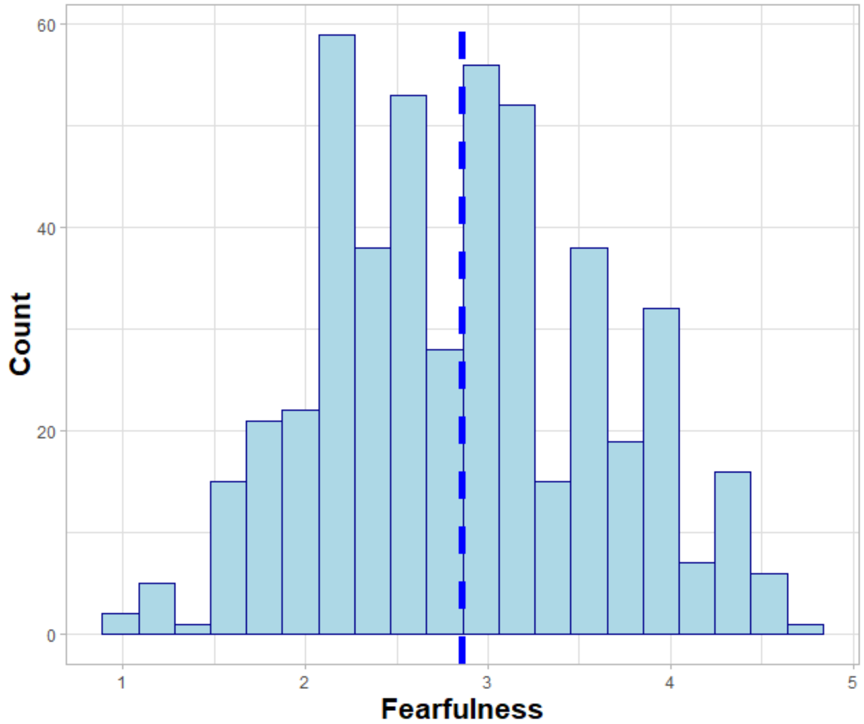

Figure 4.9. Frequency distribution of fearfulness. Dashed line represents the mean value of fearfulness (*n* = 486).

On average, participants were relatively neutral to fearfulness items, not showing extreme fearfulness nor fearlessness (*M* = 2.75, *SD* = 0.75). This result is in line with the normative descriptive statistics found in a college student sample (*M* = 3.00, *SD* = .76, *N* = 887), which were sent along with the instrument by the authors of the inventory. The distribution of participants' responses to each fearfulness item is presented in Table 4.8.

Table 4.8.

Frequencies of Answers on Fearfulness Scale

Item	Strongly disagree (=1)	Disagree (=2)	Neutral (=3)	Agree (=4)	Strongly agree (=5)	*M*	*SD*
I would feel afraid if I had to travel in bad weather conditions	94 (19%)	114 (23%)	124 (26%)	122 (25%)	32 (7%)	2.76	1.21
Item removed for copyright reasons. Copyright holders are Michael C. Ashton and Kibeom Lee.	26 (5%)	120 (25%)	126 (26%)	157 (32%)	57 (12%)	2.80	1.10
Item removed for copyright reasons. Copyright holders are Michael C. Ashton and Kibeom Lee.	9 (2%)	58 (12%)	69 (14%)	195 (40%)	155 (32%)	2.12	1.05
I don't mind doing jobs that involve dangerous work	35 (7%)	141 (29%)	130 (27%)	128 (26%)	52 (11%)	2.96	1.13
Item removed for copyright reasons. Copyright holders are Michael C. Ashton and Kibeom Lee.	61 (13%)	137 (28%)	179 (37%)	88 (18%)	21 (4%)	3.27	1.03
Item removed for copyright reasons. Copyright holders are Michael C. Ashton and Kibeom Lee.	105 (22%)	156 (32%)	85 (18%)	88 (18%)	52 (11%)	2.64	1.29
When it comes to physical danger, I am very fearful	34 (7%)	153 (31%)	131 (27%)	124 (26%)	44 (9%)	2.98	1.10
Even in an emergency I wouldn't feel like panicking	85 (18%)	170 (35%)	99 (20%)	96 (20%)	36 (7%)	3.35	1.19

Note. M = Mean score; SD = Standard deviation. Distribution of scores was calculated using *n* = 486.

4.3.2.9. Stimulation-Seeking.

Stimulation-seeking was measured with items drawn from the widely used impulsive sensation-seeking scale from the Zuckerman-Kuhlman Personality Questionnaire (Zuckerman et al., 1993). This scale consists of 19 items that tap into both impulsivity and stimulation-seeking. In the interest of addressing the problem of conflation of these two distinct concepts in the research of the relationship between low heart rate and crime, this study used only 11 items which measured stimulation-seeking, omitting eight impulsivity items. Each item consisted of a statement to which participants responded how well it described them. In the original version, respondents would give an answer using a dichotomous scale which includes only “true” and “false” options. However, in order to maximise variance across the scale and avoid technical issues resulting from a forced-choice format, the response scale was modified into a five point Likert-type response format wherein the answers ranged from “strongly disagree” to “strongly agree” (Anastasi, 1988; Whiteside & Lynam, 2001). The total score was calculated as a mean score across all items, and could take a value between one, which indicates a low stimulation-seeking tendency, and five, which indicates a strong stimulation-seeking tendency. As seen in Figure 4.10., the distribution of scores has a bell-shaped form indicating a normal distribution. Descriptive statistics can be found in Table 4.2. along with other measures.

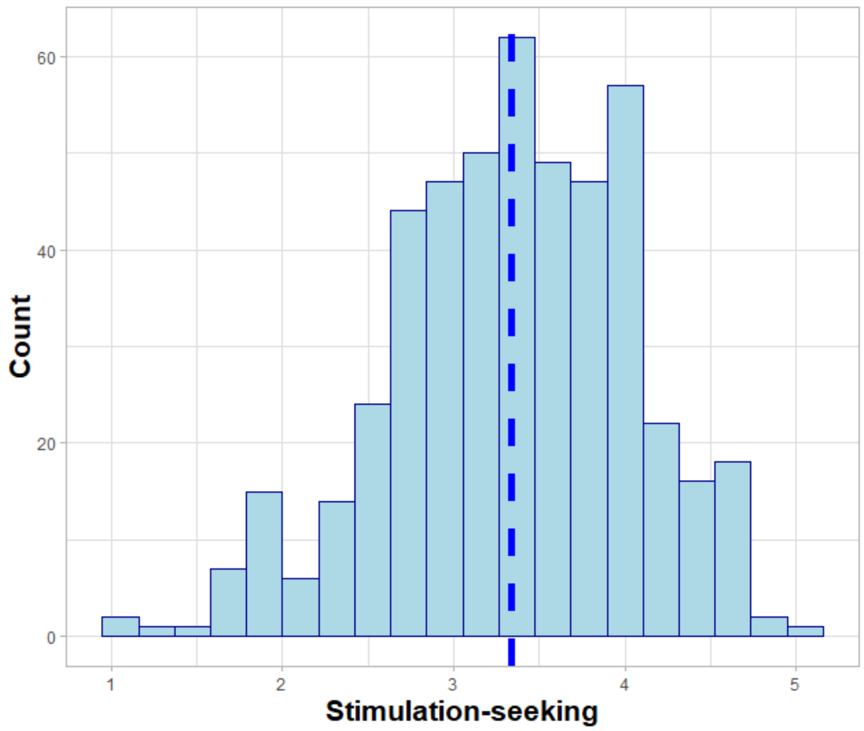

Figure 4.10. Frequency distribution of stimulation-seeking. Dashed line represents the mean value of stimulation-seeking (n = 485).

On average, participants scored in the upper half of the response distribution (M = 3.34, SD = 0.71), indicating a somewhat higher stimulation-seeking. This result was expected given that a heightened stimulation-seeking during adolescence is a well-established finding (Collado, Felton, MacPherson, & Lejuez, 2014; Kong et al., 2013; Shulman, Harden, Chein, & Steinberg, 2015; Steinberg, 2005, 2010). This is also in line with other research findings measuring stimulation-seeking using the same instrument in this specific age group (Kong et al., 2013; Steinberg, 2010; Steinberg et al., 2008, 2018), although direct comparisons are not as straightforward due to the use of force-response choice in the original scale. Overall distribution of responses to each item of the scale is presented in Table 4.9.

Table 4.9.

Frequencies of Answers on Stimulation-Seeking Scale

Item	Strongly disagree (=1)	Disagree (=2)	Neutral (=3)	Agree (=4)	Strongly agree (=5)	*M*	*SD*
I like to have new and exciting experiences and sensations even if they are a little frightening	6 (1%)	30 (6%)	67 (14%)	258 (53%)	124 (26%)	3.96	0.87
I would like to take off on a trip with no preplanned or definite routes or timetable	56 (12%)	103 (21%)	67 (14%)	138 (28%)	121 (25%)	3.34	1.36
I like doing things just for the thrill of it	13 (3%)	60 (12%)	119 (24%)	207 (43%)	86 (18%)	3.60	1.00
I tend to change interests frequently	21 (4%)	151 (31%)	97 (20%)	170 (35%)	46 (9%)	3.14	1.09
I sometimes like to do things that are a little frightening	9 (2%)	67 (14%)	101 (21%)	255 (52%)	53 (11%)	3.57	0.92
I'll try anything once	28 (6%)	90 (19%)	132 (27%)	160 (33%)	75 (15%)	3.34	1.12
I would like the kind of life where one is on the move and travelling a lot, with lots of change and excitement	46 (9%)	99 (20%)	114 (23%)	144 (30%)	82 (17%)	3.24	1.23

Item	Strongly disagree (=1)	Disagree (=2)	Neutral (=3)	Agree (=4)	Strongly agree (=5)	*M*	*SD*
I sometimes do “crazy” things just for fun	29 (6%)	130 (27%)	132 (27%)	145 (30%)	49 (10%)	3.11	1.10
I like to explore a strange city or section of town by myself, even if it means getting lost	57 (12%)	143 (29%)	96 (20%)	114 (23%)	75 (15%)	3.01	1.27
I prefer friends who are excitingly unpredictable	18 (4%)	77 (16%)	197 (41%)	135 (28%)	58 (12%)	3.28	0.99
I like “wild” uninhibited parties	67 (14%)	89 (18%)	136 (28%)	114 (23%)	79 (16%)	3.10	1.27

Note. M = Mean score; SD = Standard deviation.
Distribution of scores was calculated using *n* = 485.

4.3.2.10. Lifestyle and health covariates.

In order to take into account effects of possible cofounding variables on the relationship between heart rate and crime as recommended in the literature (Armstrong et al., 2009; Murray et al., 2016; Portnoy et al., 2014; Prätzlich et al., 2019), the following covariates were examined: sex, race, socioeconomic status (measured using parental education and receiving free school meals as proxy measures), physical fitness, body mass index, smoking, caffeine intake, drug use, and alcohol consumption. Sex, race, and socioeconomic status variables were measured using the demographics questionnaire (see Appendix 1), whereas the remainder of the covariates were measured using a lifestyle and health questionnaire presented in Appendix 4.

Level of physical fitness was assessed using a scale which measured how many times during a typical week participants engaged in mild, moderate, and vigorous exercise for longer than 25 minutes (Armstrong et al., 2009). For each category, participants would respond using a five point Likert-type response scale where answers ranged from “never” to “10 times or more”. The final score was calculated by adding together weighted scores of mild, moderate, and vigorous exercise by a factor of 1, 2, and 3, respectively. It could take a value within a range between zero and 24, wherein higher values indicate more frequent engagement in physical activity. Participants on average scored in the lower third of the spectrum (*M* = 6.59, *SD* = 4.86). In total, about 65% of the participants had a score between zero and seven points, 29% between eight and 15, and only 5% scored 16 or higher. The distribution of participants' responses to each item is represented in Table 4.10.

Table 4.10.

Frequencies of Answers on Physical Fitness Scale

During a typical week, how many times do you do the following exercises for more than 25 minutes	Never (=0)	1-3 times (=1)	4-6 times (=2)	7-9 times (=3)	10 or more times (=4)	*M*	*SD*
Mild exercise that requires minimal effort where you could easily sing while doing the activity, such as yoga, fishing, bowling, golf, and leisure walking	97 (20%)	177 (36%)	112 (23%)	53 (11%)	43 (9%)	1.52	1.19
Moderate exercise that is not exhausting where you could easily carry on a conversation while doing the activity, such as jogging, non-competitive sports, leisure dancing, and leisure swimming	129 (27%)	233 (48%)	89 (18%)	14 (3%)	17 (4%)	1.08	0.94
Vigorous exercise where you become winded or too out of breath to carry on a conversation while doing the activity, such as running, competitive sports games (soccer, football, basketball, etc.), energetic dancing, and swimming laps	189 (39%)	183 (38%)	59 (12%)	37 (8%)	14 (3%)	0.97	1.04

Note. *M* = Mean; *SD* = Standard deviation.
Distribution of scores was calculated using *n* = 482.

As for the body mass index, the participants were asked to report their current height and weight. This information was used to calculate the index as weight divided by the square of the height expressed in units of kg/m2. On average participants had a normal weight (M = 21.49, SD = 3.43), with around 16% of the participants being underweight and 11% being overweight or obese.

Smoking was assessed using a one item measure asking how many cigarettes a person smoked during a typical day. The answers were presented on a six point Likert-type response scale ranging from “none/I do not smoke” to “more than 20”, where higher scores indicate higher frequency of smoking. A big majority of the participants did not smoke (n = 453, 93%), whereas those who smoked would usually have five or less cigarettes per day (n = 18, 4%), and less than a dozen would smoke more frequently (n = 11, 2%).

The participants were also asked to report their caffeine consumption 12 hours prior to the testing. About 44% (n = 213) did not take any caffeinated drink, 50% (n = 241) had between one and three drinks, and about 6% (n = 28) had more than three drinks.

Drug use was assessed with a two item scale used in PADS+ (Wikström et al., 2012). If participants responded affirmatively to the question enquiring about whether they had ever tried any drugs, they would be then asked about the frequency of use for each of the 15 listed drugs. In total, less than a third of the participants reported they had tried drugs (n = 130, 27%). When a follow-up question enquired about specific types of substances used, cannabis emerged as the by far most popular choice whereby 25% (n = 123) of the participants had tried it at least one, followed by ecstasy (n = 19, 4%), ketamine (n = 9, 2%), cocaine (n = 8, 2%), amphetamines (n = 7, 1%), and tranquilisers (n = 5, 1%).

Alcohol use was measured with the Alcohol Use Disorders Identification Test – Consumption (AUDIT-C), a widely used, standardised three item screening tool for active alcohol abuse and dependence (Bush, Kivlahan, McDonell, Fihn, & Bradley, 1998). On each question about their drinking habits participants gave an answer on a five point response scale. Each response was awarded a point between zero and four. The total score was calculated as a sum of scores on all three items, and therefore it could be in a range between zero and 12, whereby higher scores indicate increased risk drinking. On average, participants scored three points (M = 3.07, SD = 2.77), and about a third of the sample (n = 154, 32%) scored five or above which was a cut-off point signifying increased risk drinking. The distribution of participants’ answers to each item is presented in Table 4.11.

Table 4.11.
Frequencies of Answers on AUDIT-C

Items	Scoring system						
	0	**1**	**2**	**3**	**4**	***M***	***SD***
How often do you have a drink containing alcohol?	**Never** 129 (27%)	**Monthly or less** 170 (35%)	**2-4 times per month** 135 (28%)	**2-3 times per week** 39 (8%)	**4+ times per week** 9 (2%)	1.23	0.99
How many units of alcohol do you drink on a typical day when you are drinking?	**1-2** 283 (58%)	**3-4** 34 (7%)	**5-6** 56 (12%)	**7-9** 70 (14%)	**10+** 39 (8%)	1.06	1.42
How often have you had 6 or more units if female, or 8 or more if male, on a single occasion in the last year?	**Never** 222 (46%)	**Less than monthly** 165 (34%)	**Monthly** 78 (16%)	**Weekly** 15 (3%)	**Daily or almost daily** 2 (0%)	0.78	0.86

Note. *M* = Mean; *SD* = Standard deviation.
Distribution of scores was calculated using *n* = 482.

4.3.2.11. Heart rate measures.

Heart rate was measured using Polar® OH1 optical heart rate monitors. These consist of an optical heart rate sensor attached to an adjustable elastic strap that participants wore snugly around the upper arm of their non-dominant hand. The sensor tracks heart rate continuously using photoplethysmography, a new technology which optically measures the volumetric changes of blood vessels under the skin. It consists of six light-emitting diodes that shine light onto the skin and a photodiode that measures the amount of light reflected back into it and uses this information to calculate heart rate readings. Research has shown that OH1 monitor provides a valid measure of heart rate which is in excellent agreement with the results stemming from electrocardiography, the gold standard for measuring heart rate (Hermand, Cassirame, Ennequin, & Hue, 2019; Hettiarachchi, Hanoun, Nahavandi, & Nahavandi, 2019; Schubert, Clark, & De La Rosa, 2018).

As soon as the participants were seated in front of the tablets, they immediately put on the heart rate monitors before listening to the instructions. They therefore wore the monitors for a few minutes before the actual heart rate measurement commenced, which allowed the participants to adjust to wearing the strap in order to get the most accurate measurements for the research. The protocols for inducing baseline and stress conditions were presented using Lenovo Miix 320-10ICR tablets with a 10.1" display and Vogek on-ear headphones. After listening to the instructions, the participants were firstly exposed to the protocol for inducing baseline heart rate. They watched a relaxing aquatic video with calming music ("Coral Sea Dreaming', Small World Music, Inc.) for 125 seconds. This method has been recommended as superior for achieving a baseline level of heart rate compared to the traditional method of resting quietly, as this minimally demanding task controls for the confounding effects of anxiety, anticipation, or reminiscence (Cornet, 2015; Piferi et al., 2000). Average resting heart rate was 77 beats per minute (bpm; $M = 77.15$, SD = 12.10). Male participants had a lower resting heart rate ($M = 74.43$, $SD = 12.40$) than female participants ($M = 79.28$, $SD = 11.43$), which is in line with the well-replicated finding about sex differences in resting heart rate (Aladin et al., 2014; Valentini & Parati, 2009).

After the relaxing video was finished, participants were presented with a short instructive video which explained the stress task that followed. The Montreal Imaging Stress Task (MIST; Dedovic et al., 2005) was used as a stress inducing protocol, as its experimental condition has been found to significantly increase physiologic, endocrinologic, and self-reported indicators of stress reactions as compared with rest and control test conditions (Gossett et al., 2018; Voellmin et al., 2015; Wheelock et al., 2016). It is a standardised computer-based task which presents a series of challenging mental arithmetic problems that a participant has to solve within a limited time period, with an additional element of social evaluative threat. As can be seen in Figure 4.11., the computer program consists of a window showing an arithmetic task, a response dial with 10 numbers from zero to nine, a text box which generates feedback on the submitted answer ("correct" or "incorrect"), a time bar indicating how much time is left to submit an answer, and a performance line with two indicators, one of the participant's success rate and the other of the average performance of the entire sample.

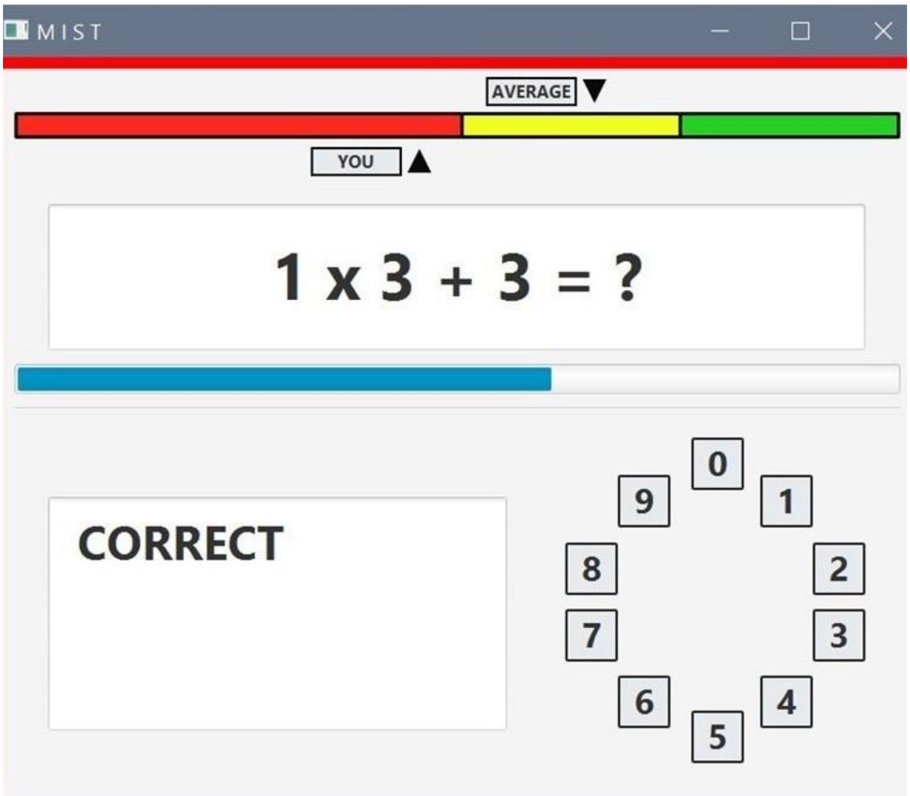

Figure 4.11. Graphical user interface of the Montreal Imaging Stress Task. On the top of the page there is a performance bar with two indicators, followed by a field presenting the arithmetic task, and a time limit bar below. On the bottom left, there is a feedback field, and on the bottom right, there is a rotary where participants entered their response.

The MIST has three conditions: rest, control, and experimental. The first two are control conditions of relevance to neuroimaging studies, and only the last condition induces stress and was therefore relevant to this study. The experimental condition was administered to each participant for 180 seconds. The participants were required to solve and submit a response to each arithmetic task within the imposed time limit. The underlying algorithm created an arithmetic problem using up to four numbers ranging from zero to 99 and up to four basic operations (addition, subtraction, multiplication, and division). The resulting problem always had a one-digit solution so that participants could answer with a single stroke on the touch-screen display. There were five difficulty levels of the arithmetic tasks depending on the number of elements in the task, size of the integers, and types and number of the operations used (e.g., an addition of two one-digit integers, a multiplication of two one-digit integers followed by a subtraction of a two-digit integer). The program recorded participants' average response time and success rate, and then used this information to adjust the enforced time limit for the subsequent tasks. If a participant correctly solved three arithmetic problems in a row, the time limit for the following tasks would be reduced to 10% less than the average response rate for the previous three tasks. On the other hand, if a participant failed to solve three arithmetic problems in a row, the time limit for the subsequent tasks would be increased by 10%. Therefore, no matter the mathematical ability of the participants, their success rate was forced to be roughly in a range from 20% to 45%.

In addition to elicited failure, this task induced stress by using a social evaluation element as well. Specifically, the performance bar shown to the participants contained two indicators, one for the participants' individual success rate, and the other supposedly indicating average performance of the entire sample. However, the participants' personal performance indicator usually appeared in the red area of the bar, signifying a poor outcome, whereas the indicator showing their peers' success appeared in the green or yellow area, indicating a high performance. According to the original protocol (Dedovic et al., 2005), between the experimental trials the researcher tells each participant about their individual performance and reminds them that the average success rate for the entire sample is 80% to 90% successfully solved tasks, and that their performance must be close or equal to the average if their data are to be used in the study. In addition, the participants are informed that the entire research team is following their performance in an adjacent room, after which the researcher leaves the room and another experimental trial commences. However, due to the technical difficulties of implementing verbal instructions between the tasks in the current study, analogous instructions were given to the participants at the beginning of the task. Participants were presented with an instruction video explaining the task wherein they were informed that their peers had solved about 80% to 90% of the tasks correctly, and that they were expected to do the same. They were told it was very important to perform the calculations accurately so that their performance indicator would stay in the green area of the bar as otherwise their data could not be used in the study. Finally, the participants were told that the study leader was following the experiment remotely to check if everyone's performance was within the green area. However, due to the underlying algorithm adjusting the difficulty of the tasks tailor-made for each participant, they would not be able to perform at the required level irrespective of their math skills and effort. The exact wording of the instructions is shown in Appendix 5. After the experiment ended, each participant was given a debriefing note explaining the nature of the task.

The average heart rate during the stress task was 85 bpm (*M* = 85.33, *SD* = 13.38). Heart rate reactivity was calculated as resting heart rate subtracted from the stress heart rate. On average, the stress task increased heart rate eight bpm (*M* = 8.19, *SD* = 7.22), which is higher than in other similar studies (Portnoy et al., 2014). Heart rate reactivity ranged from -11 to 45 bpm, where a negative sign indicates that a participant's heart rate was lower during the stress task compared to the baseline measure, and vice versa for a positive sign. In total, there were 39 participants (8%) whose stress heart rate was lower than their resting heart rate, which is in line with findings from other relevant studies (De Vries-Bouw et al., 2011; Sijtsema et al., 2013). Resting heart rate and heart rate reactivity were both fairly normally distributed, as can be seen in Figure 4.12. A paired-samples t-test was conducted in order to test whether the stress task significantly increased participants' heart rates. The difference between the means of resting heart rate and stress heart rate, - 8.19, BCa[19] 95% CI [- 8.84, - 7.57], was statistically significant, t (481) = - 24.91, $p < .001$, with a medium to large effect size of d = 0.68.

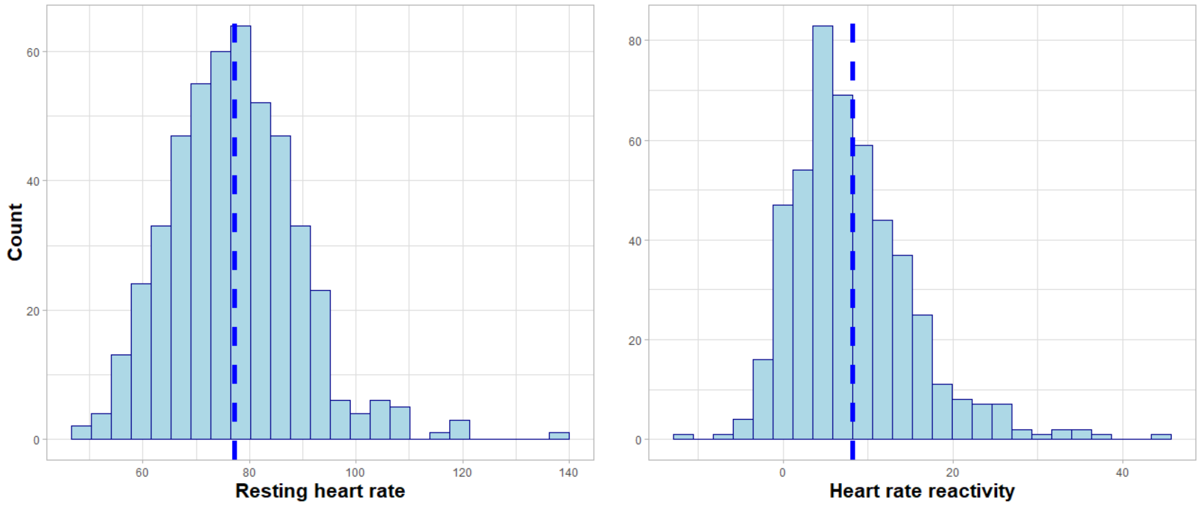

Figure 4.12. Frequency distribution of heart rate measures. Dashed lines in the left and right histogram represent the mean value of resting heart rate (*n* = 483) and heart rate reactivity (*n* = 482), respectively.

[19] Bootstrap results were based on 5000 bootstrap samples.

Chapter 5:

Findings and Discussion: Uncovering the Relationship Between Low Heart Rate and Crime

5.1. Is There a Relationship Between Resting Heart Rate and Crime?

Before advancing to the task of explaining the relationship between low heart rate and crime, it is firstly necessary to determine exactly which heart rate measure is associated with crime, the baseline or heart rate reactivity. Although low resting heart rate is considered as one of the best replicated correlates of antisocial behaviour (Ortiz & Raine, 2004; Portnoy & Farrington, 2015), a careful examination of methodology used to measure heart rate across the studies raises doubts whether the inverse relationship applies to resting heart rate or heart rate reactivity, or possibly both. In the following paragraphs, studies using different methods of assessing heart rate will be scrutinised in detail and implications of their results in relation to antisocial behaviour will be discussed. Next, the statistical results of the Cambridge adolescent behaviour study regarding the link between heart rate measures and crime will be presented. Finally, a discussion about these findings and their ability to offer new insight into the nature of this relationship will follow.

5.1.1. Different Methods of Measuring Heart Rate and Their Implications

There is an abundance of studies showing a significant negative link between resting heart rate and different types of antisocial behaviour that have accumulated over the years (see section 1.2.1.). However, labelling heart rate measures as resting is dubious in many studies, and therefore it is unclear whether these results indeed refer to a resting heart rate, or heart rate reactivity instead. A large number of studies have utilised an unstandardised protocol to measure “resting” heart rate wherein they instructed a person to sit still, relax, and keep their eyes open or closed (e.g., Brzozowski et al., 2018; Choy et al., 2017; Gao et al., 2017; Jennings et al., 2013; Raine et al., 1990). The main issue with the silent resting is that it cannot ensure that participants spend this time in the same manner, as individual differences between them affect the way they implement such instructions (Piferi et al., 2000). Some people may be anxious due to unfamiliar research environment or anticipation of what comes next, some may be thinking about different things that could affect their autonomic measures, others may be bored or sleepy, and so forth. Therefore, it is difficult to determine whether heart rate measure obtained using such protocol represents a resting heart rate, or heart rate in reaction to stress or some other condition.

A highly cited study published in *Criminology* in 2014 (Portnoy et al., 2014) can serve as an illustration of the problems that arise from using an unstandardised protocol to obtain baseline measure and the overlooked issue of correctly labelling heart rate measures. Namely, the authors measured heart rate continuously during different tasks in the following order: a first resting period (three minutes), a social stressor task (four minutes), a cognitive task (eight minutes), and a final resting period (three minutes). In both resting periods, participants were instructed to sit still and close their eyes. The results showed that the average heart rates were very similar across all four tasks[20], and therefore it was decided to use these observed variables to generate a latent variable which was then applied in all subsequent analyses. However, this raises a question of what the latent heart rate variable actually represented and how to interpret these results. Either heart rate during resting periods did not tap into resting heart rate, but rather heart rate reactivity to stress, or the subsequent stress task did not elicit a stress response, but instead represented a resting heart rate. Therefore, without answering these questions it is hard to draw firm conclusions from the study's findings as it is unclear whether they refer to resting or stress heart rate.

Another potential problem affecting some of the studies using silent resting instructions is a short duration of cardiac measurement. Namely, even though it is recommended to measure heart rate continuously for at least a full minute to get a reliable baseline measure (Cornet, 2015; Pickering, 2013; Smith et al., 2003), some studies have used a heart rate measure obtained in a matter of seconds (Armstrong & Boutwell, 2012; Armstrong et al., 2009; Raine et al., 2014). For example, a frequently used CONTEC CMS 50D finger pulse oximeter generates a result after only one to two seconds following its application (according to Armstrong & Boutwell, 2012). Measuring heart rate for a very short period in a novel situation right after an unfamiliar researcher has applied a heart rate monitor to a participant would hardly result in a resting heart rate. Instead, it could be described better as a heart rate in reaction to such a situation. Other irregularities have been noticed as well. For example, in one of the studies which used the above-mentioned finger pulse oximeter, heart rate has been measured twice, once after the participants were seated and again after five minutes had elapsed (Armstrong et al., 2009). However, instead of calculating an average of those two measures, the authors have used just the first measurement in the subsequent analyses as it showed a stronger association with antisocial behaviour. This is a

[20] First resting period: M = 68.28, SD = 10.24. Cognitive task: M = 68.21, SD = 10.06. Stress task: M = 67.97, SD = 10.06. Final resting period: M = 65.96, SD = 10.13 (Portnoy et al., 2014)

questionable decision for two principle reasons. Firstly, it is unadvisable practice to change research plans after taking into account which result suits your agenda better, and secondly, it would be more reasonable to assume that the second measure represented a better approximation of resting heart rate as it was taken after the participants had more time to acclimate to the situation.

Furthermore, several studies have reported a significant negative link between resting heart rate and offending using data from military conscription and prison health examination files (Koegl et al., 2018; Latvala et al., 2015; Van de Weijer et al., 2017). However, it is arguable whether heart rate measured during medical examination for conscription purposes or prison transfers could be indeed considered as a true baseline measure. Medical examination is perceived as stressful by many people, especially if decision regarding their conscription depends on it. In addition, the precision of heart rate measurements in these situations is not a priority and usually an approximation is enough (Van de Weijer et al., 2017). Finally, some people may even tamper temporarily with their health in order to avoid joining the army. Indeed, a study which used data from medical examinations conducted in a prison reported that their sample had a higher average heart rate than what was usually found in other studies involving participants of similar age (Koegl et al., 2018).

In recent years, however, there has been an increasing number of studies which used a standardised protocol for measuring resting heart rate developed to eliminate individual differences amongst participants and decrease the variability of their experiences during rest (Piferi et al., 2000). This is normally achieved by engaging participants in a minimally demanding task, such as watching a relaxing video followed by soothing music. Although this has not yet become a standard research practice, many studies that have measured heart rate in this manner have failed to find a link between resting heart rate and different antisocial measures (Colasante & Malti, 2017; Oldenhof et al., 2019; Prätzlich et al., 2019; Wilson & Scarpa, 2014). Similarly, studies which have engaged their participants in other minimally demanding tasks whilst measuring their resting heart rate have also reported a non-significant link with antisocial behaviours (e.g., Bimmel et al., 2008; De Vries-Bouw et al., 2011). For instance, a study which has not found significant associations between resting heart rate measured in boys at age 12 (N = 160) and antisocial and violent behaviour measured cross-sectionally and prospectively, measured heart rate continuously for five minutes while the participants were reading a magazine which ensured a valid measure of resting heart rate (Galán et al., 2017).

On the other hand, although there have been fewer studies that have measured heart rate reactivity to stress, these studies seem to point to a stronger relationship with antisocial behaviours in comparison to the results pertaining only to the resting heart rate (Choy et al., 2015; Ortiz & Raine, 2004). Furthermore, the results of some criminological studies which have recorded both resting heart rate and heart rate reactivity to stress, have found that only the latter measure showed significant negative relationship with various antisocial behaviours (Bimmel et al., 2008; De Vries-Bouw et al., 2011; Fairchild et al., 2008).

Therefore, before trying to explain why there is a link between low heart rate and crime, it is essential to first establish under which circumstances heart rate correlates with criminal behaviour. There are some indications that it is in fact heart rate reactivity that is inversely associated with antisocial behaviours, and not resting heart rate, but more research is needed to draw firm conclusions. It is necessary to devise new studies which would employ valid measures of both resting heart rate and heart rate reactivity that rely on appropriate standardised protocols for inducing resting and stress conditions, respectively. The Cambridge adolescent behaviour study was designed to address this research question, amongst others, and its results are presented in the following section.

5.1.2. Cambridge Adolescent Behaviour Study: Heart Rate and Crime

The hypotheses regarding the associations between heart rate measures and criminal behaviour were tested using Pearson product-moment correlation coefficients. The significance of a correlation coefficient was established using its *p* value and 95% confidence interval calculated using bias corrected and accelerated bootstrap method (Efron & Tibshirani, 1993). As the crime variables were positively skewed, the bootstrapped confidence intervals are important to consider when determining significance because they are robust and unaffected by the scores distribution (Field, 2018). They were derived from 5000 bootstrap samples and are reported in square brackets. A correlation was regarded as significant if its *p* value was lower than the standard threshold of .05 and its confidence interval did not include or cross zero. All correlations were calculated in IBM SPSS Statistics, Version 26.0 and graphs were produced in R version 3.5.3 (R Core Team, 2019) using the packages ggplot2 3.1.0 (Wickham, 2016).

Both resting heart rate and heart rate reactivity were normally distributed, and crime frequency was log transformed in order to abate the positive skew. The results were in line with the hypotheses outlined in the previous chapter (see section 4.2). There was no significant correlation between crime frequency and resting heart rate, r = - .06, 95% BCa CI [- .15, .04], p = .204, whereas heart rate reactivity did exhibit significant negative association with crime frequency, r = - .14, 95% BCa CI [- .21, - .05], p = .003. The former was calculated using a sample of n = 483, due to four missing values in resting heart rate, and the latter was based on a sample of n = 482 as five values were missing in the heart rate reactivity variable. The link between crime and heart rate reactivity is visually presented in Figure 5.1. The scatterplot shows a trend wherein as the crime frequency increases, the heart rate reactivity decreases.

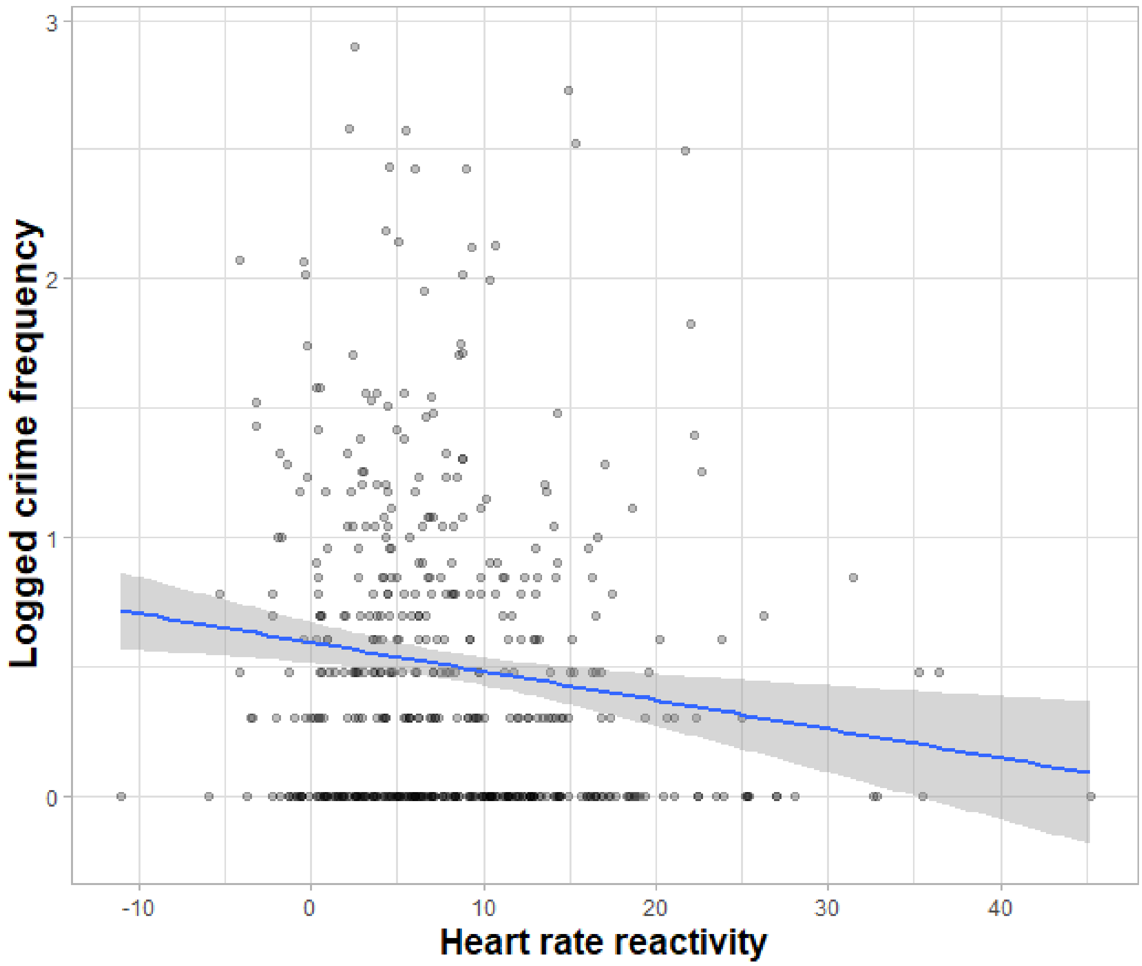

Figure 5.1. Scatterplot of the relationship between crime and heart rate reactivity. The blue line represents a linear regression line, while the surrounding grey area covers the 95% confidence interval (n = 482).

In order to explore how robust the relationship was between blunted heart rate reactivity and criminal behaviour, a series of partial correlations were run to account for the effect of potential confounding variables. Firstly, correlations between each control variable and both crime frequency and heart rate reactivity were examined. Only control variables which were significantly correlated with both heart rate reactivity and crime were retained for later use in the partial correlations. The following control variables were examined: sex, race (white vs non-white), parental education[21], whether a participant received free school meals, physical fitness, smoking, alcohol consumption, caffeine intake, drug use, and body mass index. As sex, race, and free school meals were discrete dichotomous variables, their correlations with heart rate reactivity and crime were calculated using point-biserial correlation coefficients. Correlations involving parental education were derived using a Kendall's tau[22] statistic as it is an ordinal variable. The links of the remainder of control variables with heart rate reactivity and crime were examined using Pearson's product-moment correlation.

Out of all control variables, only sex and alcohol consumption showed significant associations with both heart rate reactivity and crime frequency. Sex significantly correlated with criminal behaviour, $r = -.30$, 95% BCa CI [- 0.37, -0.22], $p < .001$, and heart rate reactivity, $r = .12$, 95% BCa CI [.03, .21], $p = .008$, such that, compared to female participants, males exhibited lower heart rate reactivity and higher criminal involvement. Alcohol consumption was also significantly associated with heart rate reactivity, $r = -.13$, 95% BCa CI [-.21, - .04], $p = .008$, and crime, $r = .27$, 95% BCa CI [.16, .37], $p < .001$. All the other control variables either correlated with only one of the two variables, or were not associated with either (see Appendix 6 for the results). Finally, the unique relationship between heart rate reactivity and crime was examined using partial correlations which accounted for the two control variables. The association seemed robust as it remained significant when controlling for either sex ($r = -.11$, 95% BCa CI [- .18, - .03], $p = .021$) or alcohol consumption ($r = -.11$, 95% BCa CI [- .19, - .02], $p = .019$). Therefore, the link between blunted heart rate reactivity and crime did not appear to be caused by confounding effects of relevant demographic or health factors.

[21] Participants were asked about their mothers' and fathers' highest completed level of education, and the parental education variable was calculated as a sum of standardised values (z-scores) of these variables.

[22] Kendall's tau was used instead of Spearman's rho as it has been found to provide a better estimate of the correlation in the population (Field, 2018; Howell, 2012).

5.1.3. Is Crime Associated with Resting Heart Rate or Heart Rate Reactivity?

By addressing the methodological limitations of prior research, the Cambridge adolescent behaviour study sought to determine which heart rate measure was significantly associated with involvement in crime. The results obtained using valid measures of both resting heart rate and heart rate reactivity clearly supported the hypotheses outlined in Chapter 4 (see section 4.1.), whereby only the latter measure was found to be significantly associated with criminal behaviour.

Furthermore, the robustness of this relationship was examined by exploring how it was affected by inclusion of control variables suggested as relevant in the literature (e.g., Armstrong et al., 2009; Jennings et al., 2013; Portnoy et al., 2014; Prätzlich et al., 2019; Valentini & Parati, 2009). The study included a range of possible confounding variables, out of which only sex and alcohol use correlated with both heart rate reactivity and crime. Both of these control variables somewhat attenuated the strength of the relationship between low heart rate reactivity and crime, but it still remained significant. This is not a surprising finding given that a number of studies have already demonstrated the robustness of the heart rate – antisocial behaviour link in the presence of different covariates, such as sex, age, race, physical fitness, body mass index, socioeconomic status, general cognitive ability (IQ), and alcohol intake (e.g.., Armstrong et al., 2009; Jennings et al., 2013; Latvala et al., 2015; Murray et al., 2016).

However, special attention should be given to the potential confounding effect of cigarette smoking, as it has been recently put forward as a particularly important covariate in criminological research pertaining to physiological measures (Prätzlich et al., 2019). The underlying reasoning was that smoking was associated with criminal behaviour and that it also affected brain and autonomic nervous system functioning. Indeed, the results of the study by Prätzlich and colleagues (2019) have shown that the correlations between resting measures of autonomic nervous system and antisocial behaviour ceased to be significant after taking into account the effects of age and smoking. This did not apply, though, to the resting heart rate measure as it was not significantly associated with antisocial behaviour to begin with[23]. Therefore, the authors concluded that smoking, best measured as a continuous

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[23] This study used a valid procedure to obtain a resting heart rate measure. Namely, the electrodes measuring heart rate were firstly applied to the participant's body after which they were given 10 minutes to acclimate to the experimental procedure. Only after the adjustment period had elapsed, the participants were asked to watch a five minute long aquatic video that was also used in the Cambridge adolescent behaviour study

variable, should always be controlled for in studies exploring the links between autonomic nervous system functions and antisocial behaviour.

At first such reasoning might seem legitimate, since smoking is indeed associated with both antisocial behaviour and heart rate. However, taking a closer look at these relationships demonstrates that smoking cannot explain the link between low heart rate and increased antisocial behaviour. Specifically, smoking indeed tends to be positively associated with antisocial behaviour (Jennings et al., 2013; Prätzlich et al., 2019; Talati, Keyes, & Hasin, 2016; Weiss, Nguyen, Trung, Ngo, & Lau, 2019), but it also causes heart rate to increase (Al-Safi, 2005; Cryer, Haymond, Santiago, & Shah, 1976; Gidding et al., 1995; Linneberg et al., 2015; Papathanasiou et al., 2013). As antisocial individuals tend to have lower heart rates, smoking can hardly account for such a connection as it is associated with higher heart rate. Therefore, it is not surprising that in a number of studies the link between low heart rate and various antisocial behaviours has remained significant despite being controlled for smoking (e.g., Jennings et al., 2013; Koegl et al., 2018; Murray et al., 2016), including the results from the Cambridge adolescent behaviour study. This is another illustration of the need to take an analytical approach in criminology, instead of blindly relying on statistical associations between variables without giving them much thought.

In sum, the results of the Cambridge adolescent behaviour study seem to suggest that it is actually heart rate reactivity to stress, and not resting heart rate, that is associated with various criminal behaviours. Importantly, this relationship proved robust as it stayed significant despite taking into account the relevant covariates. As the nature of the relationship between heart rate and crime was unravelled, the next step was to explain how heart rate reactivity is associated with crime. The following section will therefore review the results from the Cambridge adolescent behaviour study pertaining to the two well-established explanations of this relationship, namely the fearlessness and stimulation-seeking hypotheses, and the newly developed crime propensity model based on situational action theory.

(Prätzlich et al., 2018). This finding provides further support for the hypothesis that resting heart rate is unrelated to antisocial and criminal behaviour.

5.2. How is Heart Rate Reactivity Associated with Crime?

This section will begin by outlining the data analysis plan for testing the fearlessness, stimulation-seeking, and crime propensity hypotheses. After the analysis plan is described in detail, the results from the Cambridge adolescent behaviour study regarding the bivariate relationships between each mediator on one hand and heart rate reactivity and crime on the other will be reported. Finally, the statistical results regarding mediation effects will be shown for each hypothesis, followed by a discussion in light of the relevant literature review to find the answer to the main research question: How is heart rate associated with crime?

5.2.1. Data Analysis Plan

Before testing the mediation effects, the bivariate relationships between each mediator on one hand and crime and heart rate reactivity on the other will be assessed. These relationships will be established using Pearson product-moment correlation coefficients. The coefficients will be reported along with their bias corrected and accelerated 95% confidence intervals calculated from 5000 bootstrap samples.

The mediation effects of fearlessness, stimulation-seeking, and crime propensity will be assessed using structural equation modelling, specifically path analysis (Schumacker & Lomax, 2016). Before running the mediation analyses, each model will be tested for the violation of multicollinearity. This will be assessed by examining the correlations between the predictors in the model and the multicollinearity statistics, specifically tolerance statistics and variance inflation factor (VIF). A lack of multicollinearity will be demonstrated if there are no correlations higher than .80, and the tolerance statistics are above the threshold of .10 and the VIF values are below 10.0 (Field, 2018; Kline, 2016). Initially, three mediation models will be run to test the mediation effects of each of the proposed mediators with heart rate reactivity and crime as the predictor and outcome variables, respectively. If stimulation-seeking and crime propensity prove to be significant mediators, this will be followed-up with additional mediation models. In case of the stimulation-seeking hypothesis, a multiple mediation model with the stimulation-seeking and the ability to exercise self-control as mediators will be run in order to control for the effect of impulsivity. As for the crime propensity hypothesis, a multiple mediation model with moral rules, moral emotions, and ability to exercise self-control as simultaneous mediators will be tested to establish individual effects of each crime propensity component. Residual variances of mediators will be allowed to covary as recommended in the literature on multiple mediation (Preacher & Hayes, 2008).

Significance of path values will be determined by taking into account both their *p* values and 95% confidence interval. There will be no mediation if indirect effect proves non-significant (i.e., $p > .05$ and/or 95% confidence interval crosses or includes a zero). Partial mediation will be established if both direct and indirect effects of heart rate reactivity on crime are significant (i.e., $p < .05$ and 95% confidence interval does not cross or include a zero). Finally, a full mediation will be declared if indirect effect of heart rate reactivity on crime through a mediator is significant, and the direct effect is non-significant.

For each mediation model the parameter estimates will be derived using maximum likelihood method with robust, bootstrapped standard errors calculated from 5000 bootstrap samples due to violation of multivariate normality. For each path value and indirect effect, robust bias corrected and accelerated 95% confidence intervals using 5000 samples will be calculated. Across all variables used in these models, the number of missing values ranged from none in crime frequency to five in heart rate reactivity. Missing data will be imputed using the full information maximum likelihood method. All path analysis models will be produced in R version 3.5.3 (R Core Team, 2019) using the package lavaan 0.6-3 (Rosseel, 2012).

5.2.2. The Link Between Heart Rate Reactivity and Mediators

Heart rate reactivity was significantly associated with stimulation-seeking and crime propensity, but not with fearfulness, $r = .06$, 95% BCa CI [-.04, .14], $p = .230$. Stimulation-seeking was negatively correlated with heart rate reactivity, $r = -.13$, 95% BCa CI [-.22, -.04], $p = .004$. As for the crime propensity, heart rate reactivity was significantly associated with both the composite score, $r = -.15$, 95% BCa CI [- .24, - .07], $p = .001$, and each of its elements. Specifically, guilt exhibited the strongest correlation, $r = .14$, 95% BCa CI [.05, .22], $p = .003$, followed by moral rules, $r = .13$, 95% BCa CI [.03, .22], $p = .005$, shame, $r = .11$, 95% BCa CI [.02, .19], $p = .017$, and the ability to exercise self-control, $r = .11$, 95% BCa CI [.02, .20], $p = .017$. Therefore, the lower the participants' heart rate reactivity, the higher was their tendency towards stimulation-seeking and crime propensity. On the other hand, as the heart rate reactivity increased, so did the moral rules, ability to exercise self-control, and moral emotions. The scatterplots of the relationships between heart rate reactivity on one hand, and crime propensity and stimulation-seeking on the other are represented in Figure 5.2.

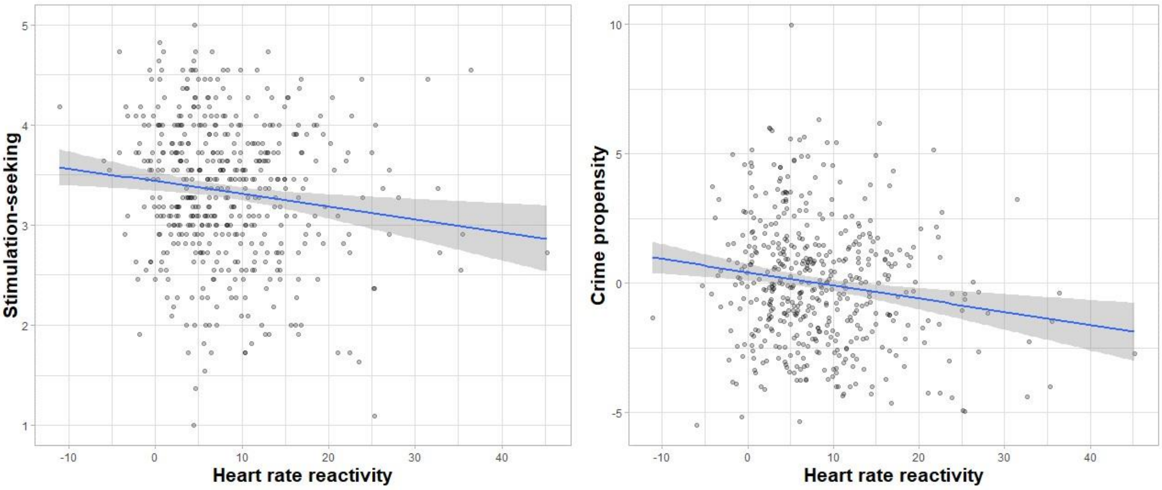

Figure 5.2. Scatterplot of the statistically significant relationships between heart rate reactivity and mediating variables. The scatterplot on the left represents the relationship between stimulation-seeking and heart rate reactivity (n = 480). The scatterplot on the right represents the relationship between crime propensity and heart rate reactivity (n = 481). The blue lines represent linear regression lines, while the surrounding grey area covers the 95% confidence interval.

5.2.3. The Link Between Crime and Mediating Variables

The results of the analyses pertaining to the associations between criminal involvement and the mediating variables were all statistically significant. Crime propensity showed the strongest link, r = .56, 95% BCa CI [.48, .63], p < .001, as it shared about a third (31%) of the variability in crime. This relationship is visually presented in Figure 5.3. Out of its constituent elements, guilt had the strongest correlation with criminal involvement, r = - .51, 95% BCa CI [- .58, - .43], p < .001, followed by moral rules, r = - .47, 95% BCa CI [- .56, - .38], p < .001, shame, r = - .42, 95% BCa CI [- .50, - .33], p < .001, and the ability to exercise self-control, r = - .41, 95% BCa CI [- .48, - .34], p < .001. The direction of correlations indicates that criminal behaviour increases as the crime propensity increases, or as the ability to exercise self-control, moral emotions, and moral rules decrease.

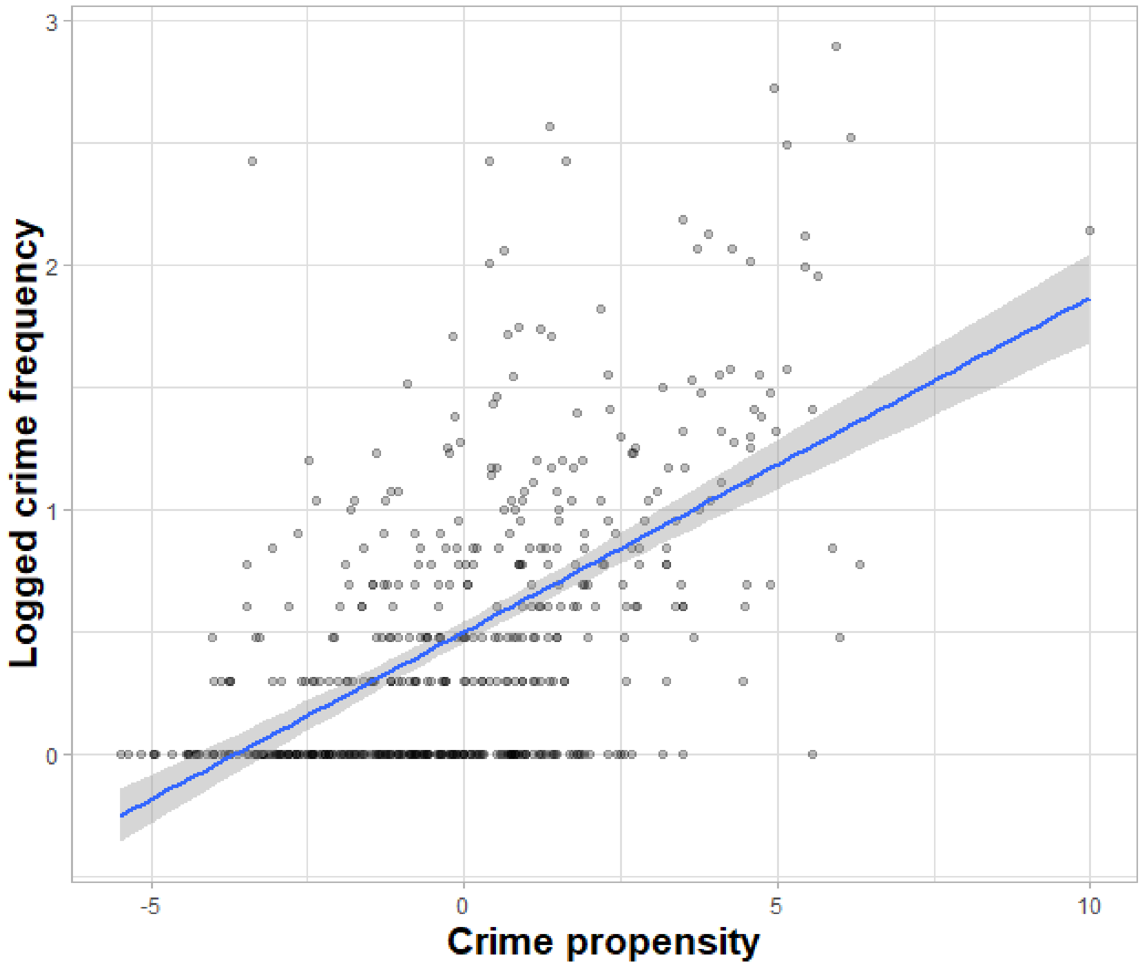

Figure 5.3. Scatterplot of the relationship between crime and crime propensity. The blue line represents a linear regression line, while the surrounding grey area covers the 95% confidence interval (*n* = 486).

As for the other two potential mediators, fearfulness was negatively associated with crime, $r = -.32$, 95% BCa CI [- .39, - .24], $p < .001$, whereas stimulation-seeking showed a positive link, $r = .27$, 95% BCa CI [.18, .35], $p < .001$. The former is graphically presented in Figure 5.4., whereas the latter is depicted in Figure 5.5. Therefore, crime frequency increased as the tendency to experience fear decreased or stimulation-seeking increased.

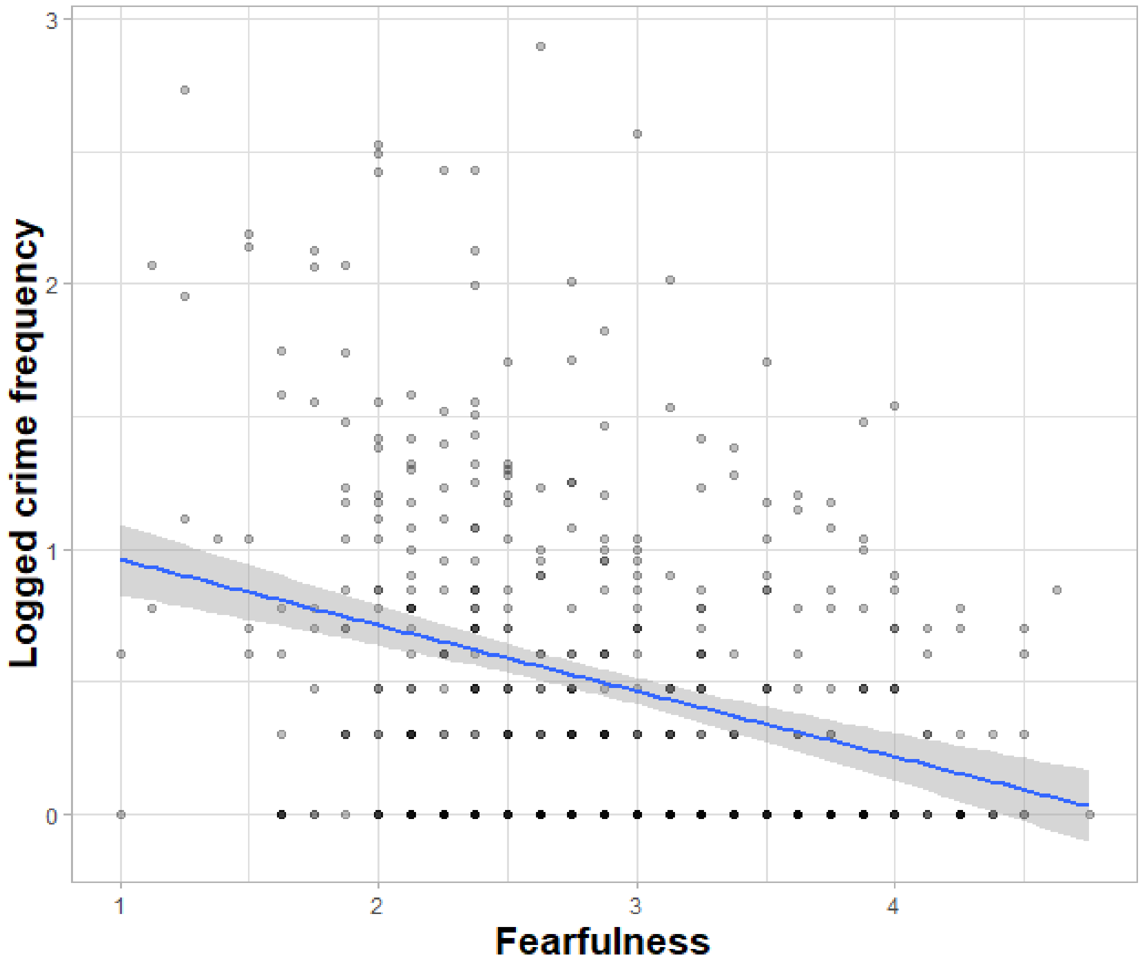

Figure 5.4. Scatterplot of the relationship between crime and fearfulness. The blue line represents a linear regression line, while the surrounding grey area covers the 95% confidence interval (n = 486).

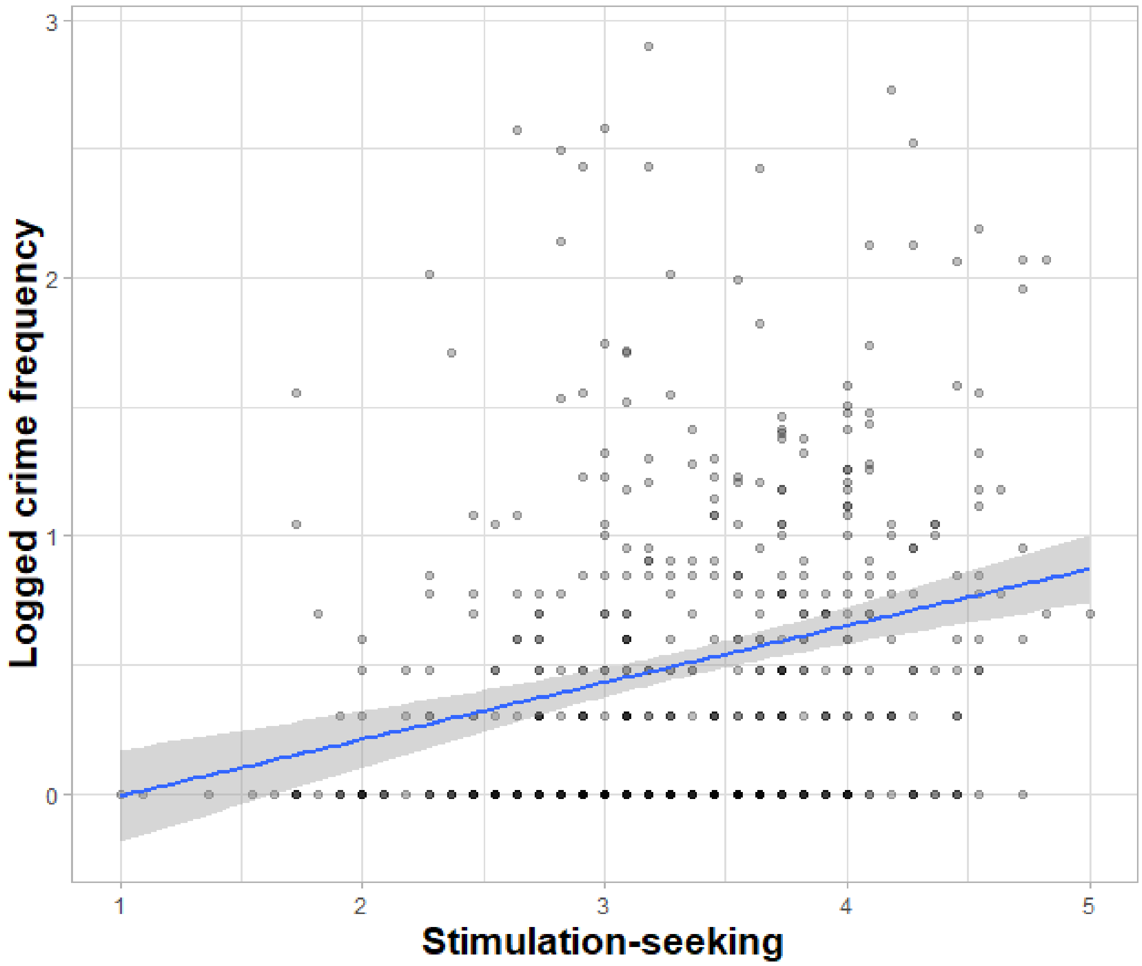

Figure 5.5. Scatterplot of the relationship between crime and stimulation-seeking. The blue line represents a linear regression line, while the surrounding grey area covers the 95% confidence interval (n = 485).

5.2.4. Testing Mediation Models: The Fearlessness Hypothesis

Firstly, the fearlessness hypothesis, which assumes that tendency to experience fear mediates the link between low heart rate reactivity and crime, was tested. Multicollinearity was of no concern in this model as the correlation coefficient between the independent variables, heart rate reactivity and fearlessness, was not significant, r = .05, p = .234, and all tolerance statistics were well above the threshold of .10 and all VIF values were well below 10.0.

The fearlessness hypothesis was not supported by the data. Although the tendency to experience fear significantly predicted criminal behaviour in a negative direction, b = - 0.248, 95% BCa CI [- 0.317, - 0.179], p < .001, it was not predicted by heart rate reactivity, *b* = 0.006, 95% BCa CI [- 0.004, 0.015], *p* = .208, and the indirect effect of heart rate reactivity on crime through fearfulness was non-significant, *b* = - 0.001, 95% BCa CI [- 0.004, 0.001], *p* = .215. After accounting for the effect of tendency to experience fear, the direct effect of heart rate reactivity on criminal involvement remained significant, *b* = - 0.010, 95% BCa CI [- 0.016, - 0.003], *p* = .003. The mediation model showing standardised path coefficients is presented in Figure 5.6.

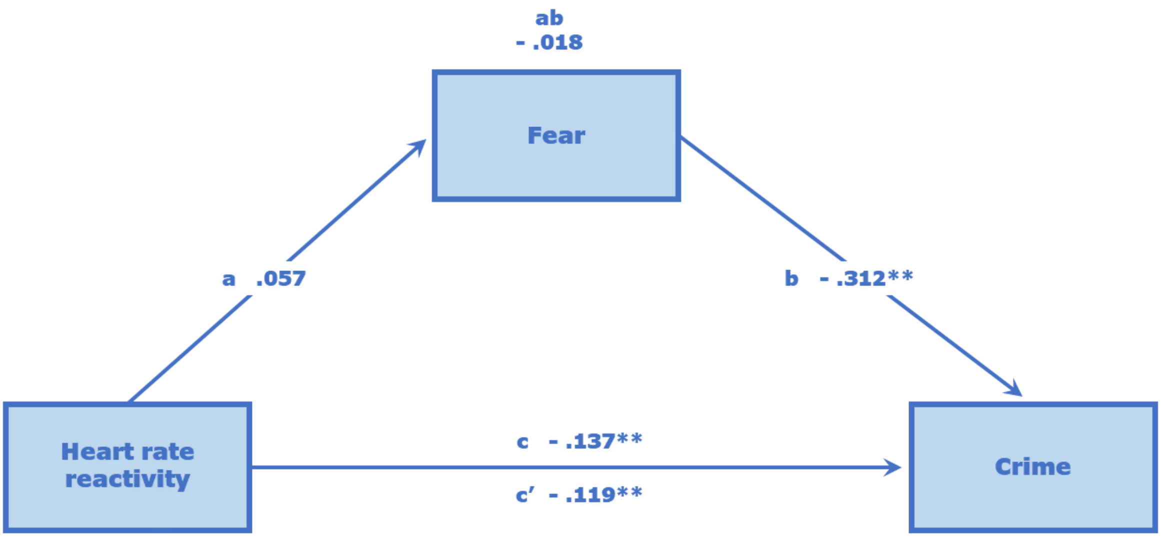

Figure 5.6. Mediation model of the fearlessness hypothesis. Standardised parameter estimates are shown for each path: a, b, c', c, and ab (*N* = 487). Paths c and c' refer to the total effect and direct effect of heart rate reactivity on crime, respectively. Path ab refers to the indirect effect.
**p* < .05. ***p* < .01.

The failure of the fearlessness hypothesis to explain the relationship between low heart rate and crime is not surprising given its theoretical drawbacks explained in detail in Chapter 2 (see section 2.1.2.) The assumption that low heart rate in a testing situation equals to a lack of fear conflates visceral threat responses, heart rate reaction included, and conscious experience of fear, which are in fact independent constructs arising in different brain regions (Hoppenbrouwers et al., 2016; LeDoux, 2013, 2014). Furthermore, the notion that fearlessness facilitates involvement in crime is based on the unspoken assumption that all people are equally motivated to commit crimes, but the ones that are fearful do not act on it as they are afraid of the consequences. This proposition is clearly not in line with reality. It is indeed true that in some situations a fear of consequences can deter a person who is already motivated and deliberates about committing crime, but fearlessness on its own cannot explain why crime happens.

As expected given its theoretical problems, the fearlessness hypothesis has not received an unequivocal empirical support, even though it still dominates the literature as one of the probable explanations of the link between low heart rate and crime (e.g., Choy et al., 2017; Koegl et al., 2018; Portnoy & Farrington, 2015). Out of four studies testing the fearlessness hypothesis, only one has interpreted its results as supportive of it (Armstrong & Boutwell, 2012; Portnoy et al., 2019, 2014; Sijtsema et al., 2010). However, this conclusion is doubtful once one takes a closer look into the study's methodological decisions (Armstrong & Boutwell, 2012). As the fearlessness hypothesis has been viewed through the lens of the rational choice theory, it has been assumed that individuals who lack fear tend to underestimate costs associated with committing a crime, namely the likelihood of being arrested and convicted, the extent to which that would cause them problems, and anticipation of feeling guilty and/or ashamed should they commit the crime as described in a given scenario. The results have shown that anticipated guilt and shame mediated the link between low heart rate and an intent to commit the assault as described in one of the scenarios, which has been interpreted in support of the fearlessness hypothesis. However, such an interpretation is far-fetched as moral emotions of shame and guilt are different from fear, and cannot be used interchangeably. Moreover, measuring fearlessness through reduced likelihood of sanctions represents a contentious practice. It could be argued that, in fact, a fearless person is someone who decides to commit a crime despite the high likelihood of getting arrested and convicted, and the attached problems that go with it. Fearlessness refers to a lack of emotional response to potentially adverse consequences of an action, not necessarily a disability to anticipate them (McMillan & Rachman, 1988). Therefore, the only study which has been interpreted in favour of the fearlessness hypothesis actually does not provide evidence in support of it. In fact, these results are instead better interpreted as supportive of the crime propensity hypothesis, as the anticipated shame and guilt following a crime refer to one of its core components.

The other studies which have explored fearlessness as a mediator of the relationship between low heart rate and antisocial behaviour have shown that this hypothesis is not supported by the data (Portnoy et al., 2019, 2014; Sijtsema et al., 2010). However, one study operationalised a lack of fear through behavioural inhibition and impulsivity (Sijtsema et al., 2010), which is highly problematic, whereas another predicted academic dishonesty, and therefore it is questionable how much can these results be generalised to criminal behaviour (Portnoy et al., 2019). Nevertheless, the remaining study explored mediating effects of state fear in relation to an effect of heart rate on non-violent delinquency and aggression, examined in separate models (Portnoy et al., 2014). Both models pointed to a non-significant indirect effect of state fear, and therefore the results did not support the fearlessness hypothesis.

In comparison with the study by Portnoy and colleagues (2014), the Cambridge adolescent behaviour study utilised trait fear as a potential mediation variable. However, the results led to the same conclusion. Namely, although trait fear negatively predicted involvement in crime, it was not associated with heart rate reactivity and the indirect effect was not significant (see Figure 5.6.). Therefore, as both state and trait fear have failed to mediate the link between low heart rate and antisocial behaviours, it is time for the literature to acknowledge the results which have falsified the fearlessness hypothesis and consider different explanations.

5.2.5. Testing Mediation Models: The Stimulation-Seeking Hypothesis

After discarding the fearlessness hypothesis, another well-established proposal in the literature, the stimulation-seeking hypothesis, was tested. The assumption of multicollinearity was not violated in the model. There was no extreme collinearity as the correlation between the predictors, heart rate reactivity and stimulation-seeking, was $r = -.13$, $p = .004$. A lack of multicollinearity was further confirmed by the collinearity statistics, as all tolerance statistics were well above the threshold of .10 and all VIF values were well below 10.0. The stimulation-seeking hypothesis was partially supported by the data. Specifically, stimulation-seeking was significantly associated with both heart rate reactivity, $b = -0.013$, 95% BCa CI [- 0.022, - 0.003], $p = .007$, and crime, $b = 0.208$, 95% BCa CI [0.138, 0.278], $p < .001$. It significantly mediated the link between heart rate reactivity and crime frequency, $b = -0.003$, 95% BCa CI [- 0.005, - 0.001], $p = .017$. However, despite its presence as a mediator in the model, direct effect of heart rate reactivity on crime frequency remained significant, $b = -0.009$, 95% BCa CI [- 0.015, - 0.002], $p = .009$. The results of the path analysis with standardised coefficients are presented in Figure 5.7.

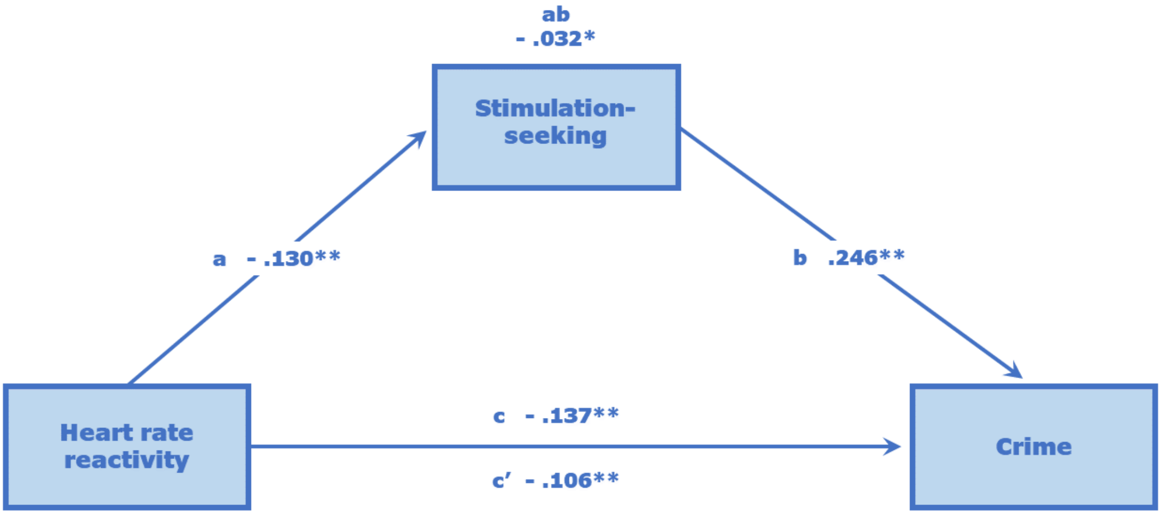

Figure 5.7. Mediation model of the stimulation-seeking hypothesis. Standardised parameter estimates are shown for each path: a, b, c', c, and ab (*N* = 487). Paths c and c' refer to the total effect and direct effect of heart rate reactivity on crime, respectively. Path ab refers to the indirect effect.
*p < .05. **p < .01.

In order to test the hypothesis that the mediation effect of stimulation-seeking is confounded by impulsivity, a multiple mediation model with stimulation-seeking and the ability to exercise self-control as mediators was run. An examination of correlations between heart rate reactivity, stimulation-seeking, and ability to exercise self-control showed no extreme collinearity as the largest correlation was r = - .42, p < .001, found between stimulation-seeking and the ability to exercise self-control. A lack of multicollinearity was further confirmed by the collinearity statistics, as all tolerance statistics were well above the threshold of .10 and all VIF values were well below 10.0. There was a statistically significant effect of heart rate reactivity on both stimulation seeking, b = - 0.013, 95% BCa CI [- 0.022, - 0.003], p = .007, and the ability to exercise self-control, b = 0.007, 95% BCa CI [0.001, 0.013], p = .013. In turn, both stimulation-seeking, b = 0.081, 95% BCa CI [0.003, 0.155], p = .033, and the ability to exercise self-control, b = - 0.448, 95% BCa CI [- 0.559, - 0.334], p < .001, significantly predicted crime frequency. However, once both variables were assessed simultaneously in the mediation model, only the indirect effect of the ability to exercise self-control was significant, b = - 0.003, 95% BCa CI [- 0.006, - 0.001], p = .017. Stimulation-seeking, on the other hand, ceased to be a significant mediating variable, *b* = - 0.001, 95% BCa CI [- 0.003, 0.000], *p* = .114. A sum of the indirect effects of both variables explained 38% of the total association between low heart rate reactivity and criminal behaviour. However, the direct effect of heart rate reactivity on criminal behaviour remained significant despite the presence of both mediators, *b* = - 0.007, 95% BCa CI [- 0.013, - 0.001], *p* = .024, signifying that the relationship was not fully explained by either self-control or stimulation-seeking and that another, unaccounted variable played an important role in the mediation. The results of the path analysis with standardised coefficients are presented in Figure 5.8.

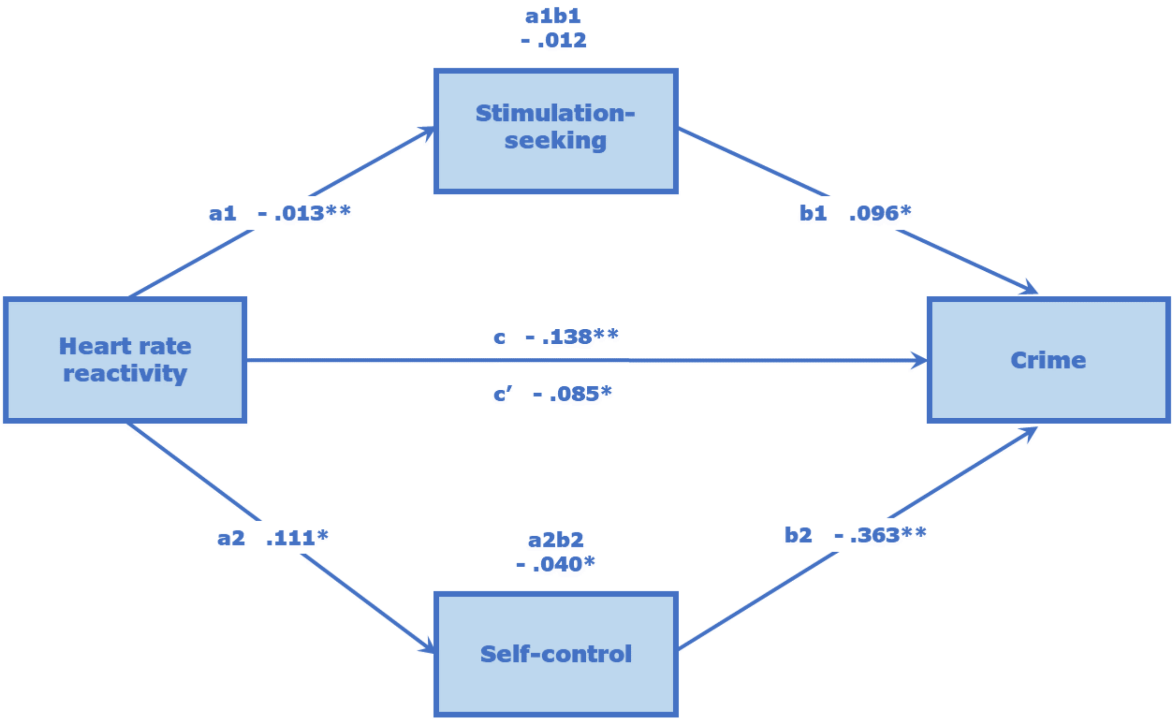

Figure 5.8. Multiple mediation model involving stimulation-seeking and the ability to exercise self-control. Standardised parameter estimates are shown for each path: a1, a2, b1, b2, a1b1, a2b2, c', and c (*N* = 487). Paths c and c' refer to the total effect and direct effect of heart rate reactivity on crime, respectively. Paths $a_n b_n$ refer to the indirect effects. Mediator residuals were allowed to covary in the model, but this was not shown graphically for the ease of presentation.
*p < .05. **p < .01.

The review of the stimulation-seeking hypothesis presented in Chapter 2 (see section 2.1.1.) highlighted both theoretical and methodological weaknesses of this explanation. The main theoretical problems with the stimulation-seeking hypothesis are the unfounded core assumptions that low heart rate causes discomfort, and that committing crime is arousing and leads to a more pleasant physiological state. In addition, it falsely positions low heart rate as a cause of crime, as it states that low arousal drives people to commit crime in order to increase their physiological arousal. Unlike the fearlessness hypothesis, the stimulation-seeking hypothesis has received some empirical support. Namely, out of five relevant studies, four have found a significant indirect effect of heart rate on antisocial behaviours going through stimulation-seeking (Hammerton et al., 2018; Portnoy et al., 2019, 2014; Sijtsema et al., 2010). However, a closer inspection of these studies show that their support for the stimulation-seeking hypothesis is not as solid as it may look initially. Firstly, the choice of instruments to measure stimulation-seeking in these studies is somewhat arguable. Specifically, two studies have used a measure which conflates stimulation-seeking and impulsivity, and therefore it is impossible to unambiguously interpret the results and disentangle the effects of these two independent constructs (Portnoy et al., 2019, 2014). Another study measured stimulation-seeking through related, but not identical constructs such as the high-intensity pleasure/surgency, behavioural activation system, and adventurism (Sijtsema et al., 2010). The final study used an instrument specifically designed to measure stimulation-seeking, but included only one of its subscales and disregarded other, equally important aspects of this construct (Hammerton et al., 2018).

Even if the methodological issues affecting these studies are put aside, overall their statistical results are inconclusive. Some studies have assessed the indirect effect of stimulation-seeking using a few models which differed by the employed instruments, time points when the measurements were taken, or have simply used separate models for each sex. However, the results have been inconsistent even within the same studies as the significant mediating effects have been found in some but not in other models (Hammerton et al., 2018; Sijtsema et al., 2010). Moreover, even if the focus is only on the models which have found a significant mediating effect of stimulation-seeking, their results are unconvincing. In some studies, although a significant indirect effect has been reported, the direct effect of heart rate on antisocial behaviour has still remained significant despite the presence of stimulation-seeking as a mediator in the model[24] (Portnoy et al., 2019, 2014; Sijtsema et al., 2010).

[24] In a study by Portnoy and colleagues (2014), there were two mediation models, one predicted aggression, and the other non-violent delinquency. In both models, the indirect effect of stimulation-seeking was significant, but in the former the direct effect of heart rate was non-significant, and in the latter the direct effect of heart rate remained significant.

Finally, even the mediation model which has yielded a significant mediating effect of stimulation-seeking, rendering the direct effect of heart rate non-significant, has explained only just above a third of the relationship between heart rate and aggression, therefore leaving more than 60% of the variance unaccounted for (Portnoy, et al., 2014).

As the empirical support for the stimulation-seeking hypothesis has been somewhat equivocal, the Cambridge adolescent behaviour study was designed in order to address the limitations of the prior research. Firstly, stimulation-seeking was assessed using a valid instrument specifically designed for that purpose which excluded impulsivity items that would hinder subsequent interpretation of the findings. The results showed that although the mediating effect of stimulation-seeking was found to be significant, the direct effect of heart rate reactivity on crime also remained significant despite the presence of the mediator in the model. In fact, stimulation-seeking explained less than a quarter (23%) of the relationship between heart rate reactivity and crime. Therefore, the empirical support for this hypothesis was limited by the fact that stimulation-seeking only partially mediated the relationship between heart rate reactivity and criminal behaviour.

Secondly, the Cambridge adolescent behaviour study included a measure of the ability to exercise self-control in order to test the hypothesis that the partial mediation of stimulation-seeking is due to its correlation with impulsivity. There has been only one study beforehand exploring mediating effects of both stimulation-seeking and self-control in the context of the relationship between low heart rate and antisocial behaviour (Portnoy et al., 2019). Their results showed that, when analysed separately, both stimulation-seeking and self-control had significant mediation effects, but only the latter variable rendered the direct effect of heart rate non-significant[25]. Once these variables were entered simultaneously in a mediation model, their indirect effects did not differ significantly from one another, but besides that information, the authors did not provide any further details about the results of this particular model. In addition, the conclusions that can be drawn from this study are seriously limited by its methodological characteristics. Namely, the instrument used to measure stimulation-seeking included impulsivity items; the sample consisted of undergraduate psychology students and the mediation effects were tested using a small subsample of female participants (n = 90); the independent variable was academic

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[25] In the model with self-control as a mediating variable, the direct effect of resting heart rate on academic dishonesty had a *p*-value higher than .05, but lower than .10. Unfortunately, the authors did not report bootstrapped confidence intervals, nor standardised coefficients, and therefore it is hard to draw any other conclusions than that the direct effect was non-significant in terms of its *p*-value being above the traditional threshold of .05.

dishonesty, a niche antisocial behaviour. However, the Cambridge adolescent behaviour study did not have these limitations and its results have provided a firm support for the hypothesis that the mediating effects of stimulation-seeking are due to its conflation with impulsivity.

Taking stock of the review of relevant theoretical and empirical literature, as well as the results presented here, there does not seem to be enough support for the stimulation-seeking hypothesis. Moreover, there are reasons to believe that the results which have been considered supportive may have been confounded by the unaccounted effect of impulsivity. Although results from the Cambridge adolescent behaviour study back this claim, more research is needed for a more definitive answer. Therefore, it is important that future studies use a valid measure of stimulation-seeking, untainted by impulsivity items, and account for the effects of self-control.

5.2.6. Testing Mediation Models: The Crime Propensity Hypothesis

As shown in Chapter 2 and this section, the two most prominent explanations, the stimulation-seeking and fearlessness hypotheses, seem unfit to explain the relationship between low heart rate and antisocial behaviour, crime included. Therefore, there was a need for a new model that would be grounded in a well-developed and empirically supported theory. In response, a novel model based on situational action theory was developed and presented throughout Chapter 3. It was proposed that the central dysfunction of the certain brain regions causes both low heart rate reactivity and increased crime propensity that in turn leads to crime. It was therefore expected that the relationship between heart rate and criminal behaviour will be fully explained through the mediating effects of crime propensity. In the next paragraphs, the crime propensity hypothesis will be tested using the data from the Cambridge adolescent behaviour study and the findings will be reported, followed by a discussion of their relevance.

Before running the mediation model of the link between heart rate reactivity and crime with crime propensity as a mediator, it was established that there was no violation of the assumption of multicollinearity. There was no extreme collinearity as the correlation between the predictors, heart rate reactivity and crime propensity, was $r = -.15$, $p = .001$. The lack of multicollinearity was further confirmed by the collinearity statistics, as all tolerance statistics were well above the threshold of .10 and all VIF values were well below 10.0. The crime propensity hypothesis was fully supported by the data. Heart rate reactivity negatively predicted crime propensity, *b* = - 0.053, 95% BCa CI [- 0.083, - 0.023], *p* < .001 , which in turn positively predicted frequency of criminal involvement, *b* = 0.137, 95% BCa CI [0.116, 0.158], *p* < .001, yielding a significant indirect effect size, *b* = - 0.007, 95% BCa CI [- 0.012, - 0.003], *p* < .001. Inclusion of crime propensity as a mediator rendered the direct effect of heart rate reactivity on crime non-significant, *b* = - 0.004, 95% BCa CI [-0.010, 0.001], *p* = .145. The results of the path analysis with the standardised path coefficients are visually presented in Figure 5.9.

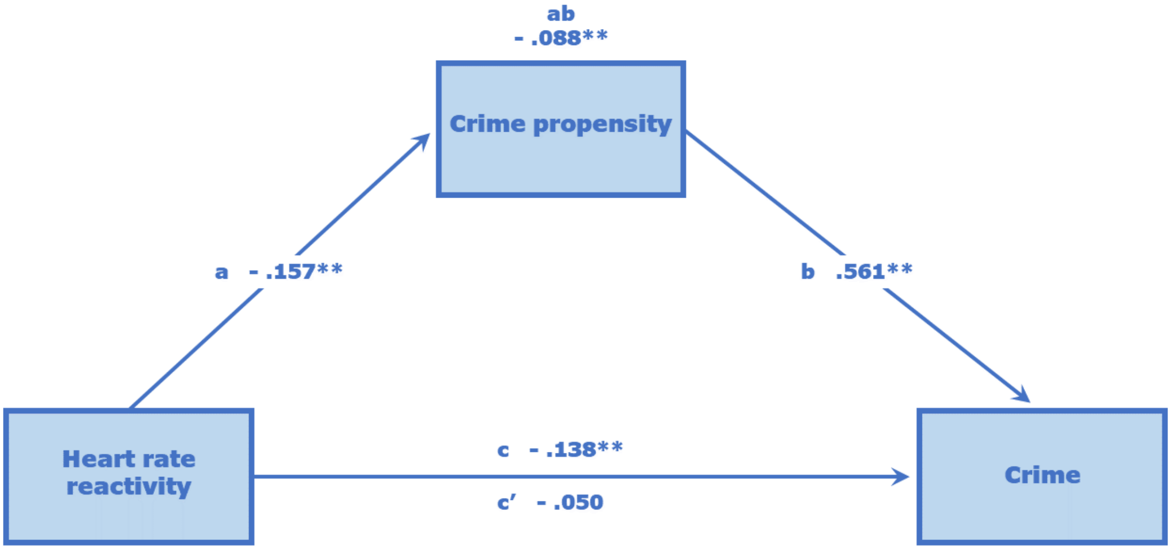

Figure 5.9. Mediation model of the crime propensity hypothesis. Standardised parameter estimates are shown for each path: a, b, c', c, and ab (*N* = 487). Paths c and c' refer to the total effect and direct effect of heart rate reactivity on crime, respectively. Path ab refers to the indirect effect.
*p < .05. **p < .01.

As crime propensity explained the relationship between low heart rate reactivity and criminal behaviour, the next step was to tease apart specific indirect effects that go through its components. Therefore, another multiple mediation model with the same specifications as the one before was run in order to test the mediation effects of moral rules, moral emotions[26], and the ability to exercise self-control. The assumption of multicollinearity was not violated in the model. An examination of correlations between heart rate reactivity, moral rules, moral

[26] Moral emotions were calculated as a sum of standardised scores from the guilt and shame scales.

emotions, and ability to exercise self-control showed no extreme collinearity as the largest correlation was $r = .61$, $p < .001$, found between moral rules and moral emotions. A lack of multicollinearity was further confirmed by the collinearity statistics, as all tolerance statistics were well above the threshold of .10 and all VIF values were well below 10.0. All three indirect mediation effects going through moral rules, $b = - 0.002$, 95% BCa CI [- 0.005, - 0.001], $p = .041$, moral emotions, $b = - 0.003$, 95% BCa CI [-0.006, -0.001], $p = .007$, and the ability to exercise self-control, $b = - 0.002$, 95% BCa CI [-0.004, - 0.001], $p = .035$, were statistically significant. Direct effect of heart rate reactivity on criminal involvement was rendered non-significant, $b = - .004$, 95% BCa CI [- 0.010, 0.001], $p = .152$. In addition, three contrasts were specified in order to test whether there were significant differences between the sizes of the indirect effects. All results were non-significant, signifying that indirect effects of moral rules and moral emotions, $b = 0.001$, 95% BCa CI [- 0.002, 0.004], $p = .460$, moral rules and the ability to exercise self-control, $b = .000$, 95% BCa CI [- 0.003, 0.002], $p = .763$, and moral emotions and the ability to exercise self-control, $b = - 0.002$, 95% BCa CI [-0.005, 0.001], $p = .240$, did not differ from each other. Therefore, after accounting for the shared variance between the mediators, each crime propensity component uniquely mediated the relationship between low heart rate reactivity and criminal behaviour, rendering the direct effect of heart rate reactivity on criminal behaviour non-significant. The results of the multiple mediation involving the standardised path values are presented in Figure 5.10.

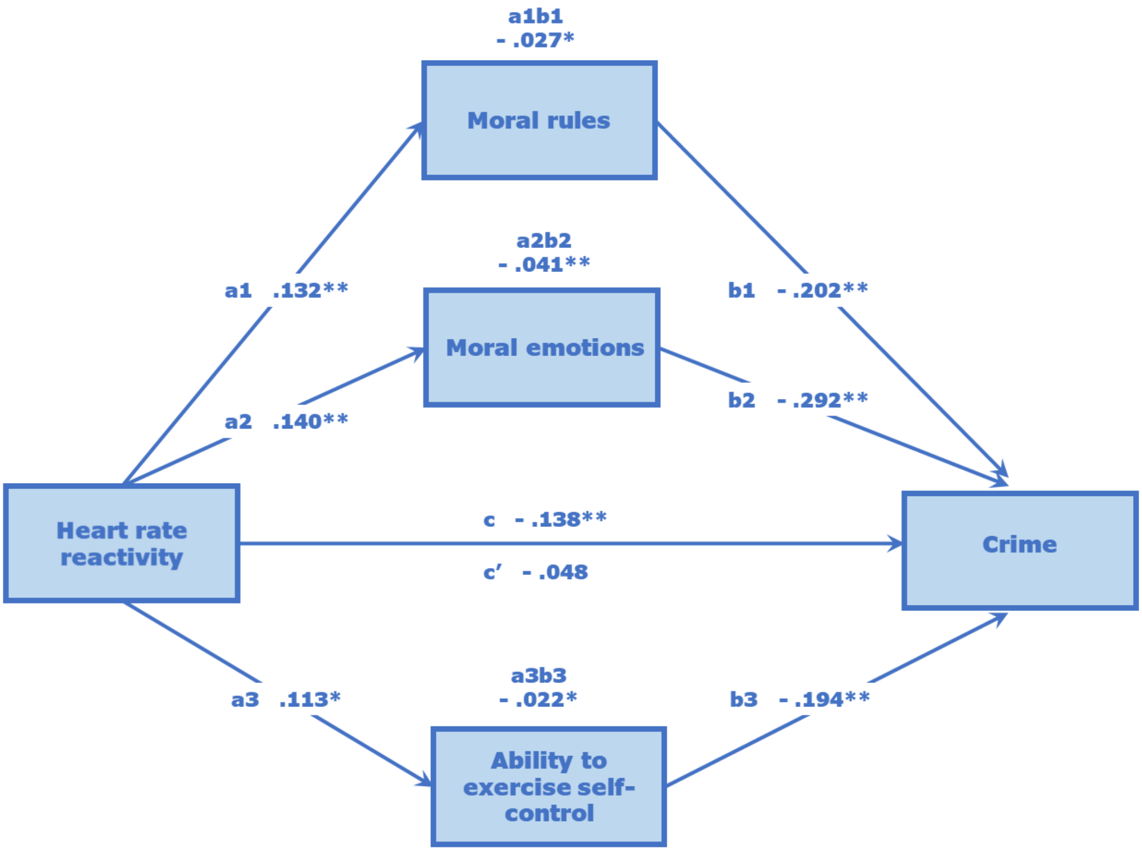

Figure 5.10. Multiple mediation model of the crime propensity hypothesis. Standardised parameter estimates are shown for each path: a1, a2, a3, b1, b2, b3, a1b1, a2b2, a3b3, c', and c (*N* = 487). Paths c and c' refer to the total effect and direct effect of heart rate reactivity on crime, respectively. Paths $a_n b_n$ refer to the indirect effects. Mediator residuals were allowed to covary in the model, but this was not shown graphically for the ease of presentation.
*p < .05. **p < .01.

As this is a newly developed theory, until now there was only indirect empirical support for its premises. One of the studies which tested the stimulation-seeking and fearlessness hypotheses has paradoxically provided empirical support for the crime propensity model (Armstrong & Boutwell, 2012). Namely, as previously mentioned, this study measured fearlessness through a reduced anticipation of guilt and shame should one commit an assault as described in a given scenario. The results showed that participants with low resting[27] heart rate were less likely to anticipate guilt and shame and more likely to endorse committing the assault. Furthermore, anticipated moral emotions were confirmed as a mediating factor of the link between low resting heart rate and an intent to commit the

[27] Although it is arguable whether this heart rate measure tapped into resting heart rate.

assault. Therefore, as moral emotions of guilt and shame are a core component of crime propensity, these results provide support for the crime propensity model. Similarly, the only study which had beforehand explored self-control, the other fundamental component of crime propensity, as a potential mediator between low heart rate and antisocial behaviour, had confirmed its relevance (Portnoy et al., 2019).

Until now, support for the crime propensity model has been only circumstantial as, apart from the fact that these studies have not been specifically designed to test this hypothesis, their conclusions are somewhat limited by their methodology. Therefore, the Cambridge adolescent behaviour study was conducted in order to specifically test the crime propensity model and address the methodological limitations of the prior studies. Its results showed unequivocal support for the crime propensity model. Not only did this variable significantly mediate the link between low heart rate reactivity and crime, it also rendered the direct effect of heart rate reactivity non-significant. In comparison, crime propensity explained 64% of the total effect of heart rate reactivity on crime, whereas the stimulation-seeking model accounted for only 23%. Similarly, the stimulation-seeking model from a previous study interpreted as supportive of the stimulation-seeking hypothesis, explained only 36% of the total effect of heart rate on aggression, and 27% of the total effect of heart rate on non-violent delinquency (Portnoy et al., 2014). Therefore, the crime propensity model is not only rooted in a well-developed theoretical framework, but it has also received strong empirical support that has far surpassed evidence for any of the previous models.

It was also hypothesized that both morality and the ability to exercise self-control will mediate the relationship between heart rate and crime, but that the former would have a stronger effect. Previous research has shown that the link between low heart rate and various antisocial behaviours remained significant despite controlling for the effects of self-control or impulsivity (Armstrong et al., 2009; Boisvert et al., 2017; Cauffman et al., 2005; Jennings et al., 2013). Therefore, there had been reasons to believe that the low ability to exercise self-control would not be able to fully mediate the link between heart rate and crime, although the partial mediation was still possible. On the other hand, there had been empirical reasons to believe that moral emotions could fully mediate the relationship in question (Armstrong & Boutwell, 2012). Apart from empirical reasons, the fact that morality is considered more fundamental to engagement in crime compared to the ability to exercise self-control was another reason for this prediction (e.g., Wikström et al., 2012; Wikström & Svensson, 2010). However, data from the Cambridge adolescent behaviour study did not confirm this specific hypothesis. Namely, once the shared variance amongst the mediators was controlled for, all three components of crime propensity uniquely mediated the link between low heart rate reactivity and crime, and no significant differences between the effect sizes of the indirect effects emerged. Therefore, moral rules, moral emotions, and the ability to exercise self-control seem all equally important in explaining the link between heart rate and criminal behaviour.

5.3. Limitations of the Cambridge Adolescent Behaviour Study

The Cambridge adolescent behaviour study provided a much needed advancement in biosocial criminology by focusing on causes of crime and potential explanations of how certain criminological phenomena happen, instead of fixating on correlational relationships and risk-factors as it is usually the case in such research. It also introduced cyber-crime items into a measure of criminal behaviour, which has been long overlooked in criminology despite its importance. The study addressed the methodological limitations of the prior research by using valid instruments for measuring key constructs, clearly defining the independent variable, and employing a varied sample. As a result, its results led to straightforward and unambiguous conclusions regarding the underlying mechanism explaining the link between low heart rate and crime. However, this study is not without limitations and they will be listed in the subsequent paragraphs.

Firstly, there is a question of how representative the sample of Year 12 students is given that the acceptance rate of schools to take part in the research was 18%. The response rate was within the expected range based on prior projects involving schools in England, especially given that the study warranted considerable concessions from the participating schools. However, despite the small, albeit realistic acceptance rate, it is believed the study tapped into a varied sample. As explained in Chapter 4 (section 4.3.), the number of schools recruited across Bedfordshire, Hertfordshire, Suffolk, and Norfolk, mimics the ratio of the total number of schools in these counties. Furthermore, all schools were mixed-gender and non-selective. The sample included different types of schools (academy sponsor led mainstream, academy converter mainstream, foundation, and free schools) located in cities, towns, and rural areas. Participating schools also differed in the quality of the service they provided, given that the sample consisted of schools rated as outstanding, good, require improvement, or inadequate, that is, across all grades of the Ofsted scale. In sum, the final sample included a number of very different schools which enabled the study to have participants from diverse backgrounds, but its representativeness of the Year 12 students across the East of England cannot be verified.

Secondly, weight and height which were used to calculate body mass index, a covariate in the analyses assessing the relationship between low heart rate and crime, were assessed using a self-report measure. Unfortunately, it was unfeasible to physically measure participants' height and weight during the testing sessions at schools, and the self-report measures were the only alternative. Therefore, the accuracy of these data remains uncertain. However, it is not expected that the type of measurement would change the conclusions in any significant way for two reasons. Firstly, despite some evidence of over- or under-estimation, research seems to suggest that self-reported weight and height measurements in samples of young people are fairly accurate and can be used as an alternative to calculate valid body mass index when objective measurements are not available (Bowring et al., 2012; Nikolaou, Hankey, & Lean, 2017; Olfert et al., 2018; Quick et al., 2015). Secondly, already a number of prior studies have rejected the hypothesis that the link between low heart rate and antisocial behaviours is somehow affected by body mass, height, or weight (Armstrong et al., 2009; Jennings et al., 2013; Latvala et al., 2015; Murray et al., 2016; Raine, Venables, et al., 1997). Therefore, despite the fact that body measurements were taken using a self-report measure, there are strong reasons to believe that the results would not change had more objective measurements been used.

Thirdly, the fearfulness scale used to measure the tendency to experience fear has a significant number of items tapping specifically into fear of injury and physical pain. Namely, out of eight items, six tap into fear of physical harm, with four doing so explicitly, and two implicitly. However, this instrument was still either broader in scope or more relevant in comparison to the measures used in most of the previous studies testing the fearlessness hypothesis. For example, tendency to experience fear has been measured using state fear, behavioural inhibition, and costs associated with committing crime (such as low anticipated likelihood of sanctions and weak anticipated guilt and shame; Armstrong & Boutwell, 2012; Portnoy et al., 2014; Sijtsema et al., 2010). As a precaution, the analyses were repeated using a fearfulness measure reduced to include only the two items tapping into a general tendency to experience fear[28]. The results showed that fearfulness remained a non-significant mediator of the relationship between low heart rate reactivity and crime, and

[28] These items were [Item removed for copyright reasons. Copyright holders are Michael C. Ashton and Kibeom Lee.] and "Even in an emergency I wouldn't feel like panicking".

therefore there would be no difference in conclusions (see results in Appendix 7). This outcome is also in line with the only other study which tested the fearlessness hypothesis using an appropriate measure of trait fearlessness tapping into its different aspects (Portnoy et al., 2019), namely the fearlessness subscale of the Psychopathic Personality Inventory (Lilienfeld & Andrews, 1996). Nevertheless, future studies may benefit from using a wide-ranging instrument of tendency to experience fear that would tap into different aspects of fear, involving both physical and social harm.

Finally, even though the study explained the mediating effects of the relationship between low heart rate reactivity and crime, the underlying mechanism is not yet fully understood. It was hypothesised and then empirically confirmed using data from the Cambridge adolescent behaviour study that low heart rate reactivity correlates with high crime propensity, which in turn explains the relationship between the former variable and criminal behaviour. However, the explanation of the link between heart rate and crime propensity was only briefly mentioned when it was argued that it was a spurious relationship as both variables were caused by the same dysfunction of a specific brain network. In order to offer a comprehensive explanation of the mechanism by which heart rate is associated with crime, it is necessary to provide further details about the neurocognitive aspects of the crime propensity hypothesis. Therefore, the next chapter will offer a theoretical answer to the question of why there is an association between blunted heart rate reactivity and tendency to see and choose crime as an action alternative in response to a temptation or provocation, along with the results of preliminary empirical tests of the full crime propensity hypothesis.

Chapter 6:

What Next? Explaining the Link Between Heart Rate Reactivity and Crime Propensity

6.1. Heart Rate Is Not a Cause of Crime: Understanding the Link With Crime Propensity

As outlined in the introductory chapters, the primary aim of this study was to explain the link between heart rate and crime. This goal was successfully achieved by introducing a model well-grounded in theory which was able to fully explain the empirical link between low heart rate reactivity and engagement in criminal behaviour. However, in order to provide a comprehensive description of the underlying mechanism, it is necessary to move beyond mediation effects and expand the model to account for the relationship between the independent variable and the mediator. We learnt that crime propensity entirely mediates the effect of heart rate reactivity on crime, but now the final missing part of the puzzle is to be addressed – why is heart rate reactivity associated with crime propensity?

As explained in Chapter 3 (section 3.1.), heart rate cannot be causally related to either crime or crime propensity. Having a blunted heart rate reactivity cannot move anyone to perceive and choose crime as an action response to a particular temptation or friction. Even though this relationship is only correlational, its origin still ought to be explained. The fact that both heart rate and criminal propensity are centrally underpinned by functions of certain brain regions gives rise to a possibility that their correlation is spurious and stems from the mutual causal factor. Namely, like other physiological functions, heart rate is centrally controlled by a specific neurological network which makes adjustments in line with the requirements of the internal and external environments (e.g., Benarroch, 1993, 1997; Thayer & Lane, 2009). Similarly, theoretical premises of situational action theory and empirical findings from neurocriminology show that neurocognitive functions are part of the causal process leading to criminal behaviour through their influence on crime propensity and the perception-choice process (e.g., Raine, 2019; Treiber, 2011, 2014, 2017; Wikström & Treiber, 2007). Therefore, it is possible that a central dysfunction of the overlapping brain regions causes both blunted heart rate reactivity and deficits in morality, ability to exercise self-control, and decision-making which make individuals prone to committing crime.

In order to pinpoint the specific neural localisation that could underlie the link between low heart rate reactivity and crime propensity, a review of relevant findings from neuroscience, physiology, psychology, and criminology has been conducted. Convergence of insights across these fields has yielded the following brain areas as mutual to heart rate, the perception-choice process (specifically, crime propensity and decision-making), and criminal behaviour: the orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala.

Guided by situational action theory and informed by findings from neuroscience and psychology, the proposed model of the link between heart rate and crime is as follows: The dysfunction of all or some parts of the above-mentioned fronto-temporal regions leads to impairments in moral emotions, weak behavioural inhibition (i.e., poor ability to exercise self-control), and deficient habitual decision-making, which in turn affect the perception-choice process and thus lead to criminal behaviour in certain situations. As the same brain regions regulate heart rate reactivity to stress, it is proposed that the relationship between heart rate and crime propensity is confounded by the shared underlying central dysfunction of the lower fronto-temporal regions. The subsequent paragraphs will outline theoretical and empirical support for the premises this hypothesis is based upon. Firstly, the evidence for the central control of heart rate will be presented, followed by an overview of neural underpinnings of antisocial behaviour, crime propensity, and decision-making. This section will be concluded by a summary of the extended version of the crime propensity model.

6.1.1. The Central Control of Heart Rate

Heart rate is directly controlled by the sinoatrial node, a natural pacemaker located within the heart which constantly generates action potentials (i.e., electrical impulses) that cause cardiac muscle to contract. The activity of the sinoatrial node is in turn determined by the sympathetic and parasympathetic branches of the autonomic nervous system which have antagonistic effects on the heart rate (Bear, Connors, & Paradiso, 2016). The former releases norepinephrine, a neurotransmitter which has an excitatory influence on the sinoatrial node leading to heart rate acceleration, and the latter inhibits the firing of the pacemaker through discharge of acetylcholine resulting in heart rate deceleration (Appelhans & Luecken, 2006; Gordan, Gwathmey, & Xie, 2015; Thayer & Lane, 2009). Finally, the activity of the autonomic nervous system, and therefore heart rate by proxy, is regulated at the central level by an interconnected system of brain areas known as the central autonomic network (briefly mentioned in section 2.1.6.1.). Therefore, heart rate is controlled by the centres at different tiers of hierarchy with a network of brain regions regulating the whole system at the top level.

The central autonomic network consists of the following regions located across the cortical, limbic, and brainstem areas: the medial and insular prefrontal cortices, the central nucleus of the amygdala and the bed nucleus of the stria terminalis, the hypothalamus, the periaqueductal grey matter, the parabrachial nucleus, the nucleus of the solitary tract, and the ventrolateral medulla (Benarroch, 1993). This distributed network of interconnected brain regions receives and integrates visceral and sensory inputs regarding body functions and external environment, respectively, and in turn regulates physiological arousal in response to these internal and external demands. In particular, the medial prefrontal cortex, insula, and amygdala are proposed as responsible for higher-order processing of the visceral and sensory information and initiating a physiological response which is then carried out by the lower brain regions and the autonomic nervous system.

Although the central autonomic network was originally based on findings from the animal experimental studies, it has since accumulated a compelling evidence base stemming from research involving human participants. A meta-analysis conducted on 43 neuroimaging studies aiming to delineate brain areas involved in autonomic processing has confirmed the activation of the areas belonging to the central autonomic network (Beissner et al., 2013). Namely, the orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala, amongst others, were found to be commonly activated across the studies. The results of a meta-analysis focusing on functional neuroimaging studies exploring patterns of neural activation associated specifically with heart rate too has provided support for the involvement of the above-mentioned brain regions (Ruiz Vargas, Sörös, Shoemaker, & Hachinski, 2016). In addition, a recent study has also revealed a significant negative association between functional connectivity between regions of the central autonomic network and heart rate (De La Cruz et al., 2019).

Furthermore, a number of lesion studies have confirmed the causal role of the fronto-temporal regions in regulating autonomic arousal (Bechara, Damasio, Damasio, & Lee, 1999; Critchley et al., 2003; De Morree, Rutten, Szabo, Sitskoorn, & Kop, 2016; Schneider & Koenigs, 2017). Specifically, patients showed blunted autonomic reactivity and/or bradycardia following damage to the orbitomedial prefrontal cortex, anterior cingulate cortex, insula[29], or amygdala. In addition, although fewer in number compared to the animal studies, experiments in humans have also demonstrated that electrical stimulation of the orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala leads to changes in heart rate (Benarroch, 1993; Inman et al., 2018; Oppenheimer et al., 1992; Parvizi, Rangarajan, Shirer, Desai, & Greicius, 2013). Such findings from the lesion studies and stimulation experiments are important due to their causal implications, as neuroimaging studies can only

[29] Structural deficits of insular cortex have been associated with a range of disturbances in cardiac regulation, including arrythmia, bradycardia, and tachycardia (Christensen et al., 2005; De Morree et al., 2016; Nagai, Hoshide, & Kario, 2010; Seeck et al., 2003).

reveal relationships at a correlational level. In sum, there seems to be a significant empirical support for the involvement of the orbitomedial prefrontal cortex, insular cortex, anterior cingulate cortex, and amygdala (amongst others) in the central regulation of heart rate.

6.1.2. Neural Underpinnings of Antisocial Behaviour

In recent years, biosocial criminology has amassed a large body of evidence demonstrating neurological and neurocognitive impairments associated with antisocial behaviour and crime. An attempt to identify neurological underpinnings of criminal behaviour quickly leaves one overwhelmed by a large number of different brain regions and cognitive processes that have been associated over the years with different types of antisocial behaviours. However, as it is the case in general with human behaviour and personality traits, the neurocognitive deficits related to antisocial behaviours are mainly located in the fronto-temporal areas involving the prefrontal cortex and the limbic system (Coccaro, Sripada, Yanowitch, & Phan, 2011; Raine, 2018). According to the neuroimaging studies, brain areas that are most commonly found to be structurally and functionally impaired in antisocial individuals are the dorsolateral prefrontal cortex, orbitomedial prefrontal cortex, anterior cingulate cortex, and the amygdala (Boes, Tranel, Anderson, & Nopoulos, 2008; Chester, Lynam, Milich, & DeWall, 2017; Coccaro et al., 2018; Glenn & Raine, 2011; Glenn, Yang, & Raine, 2012; Raine & Yang, 2006; Yang, Glenn, & Raine, 2008; Yang & Raine, 2009). A growing body of evidence from neuroimaging studies has indicated that the role of the insula too merits a serious consideration when delineating neural underpinnings of antisocial behaviour (Fairchild et al., 2011; Krokoszinski, Westenberger, & Hosser, 2018; Noordermeer, Luman, & Oosterlaan, 2016; Repple et al., 2017; Sterzer, Stadler, Poustka, & Kleinschmidt, 2007).

Building on the cross-sectional research, emerging prospective studies have generated some preliminary indications that neural measures obtained using functional and structural brain imaging techniques are also useful in predicting future offending (Aharoni et al., 2013; Aharoni et al., 2014; Kiehl et al., 2018; Steele et al., 2015). Namely, low brain activity in the anterior cingulate cortex during error processing in a behavioural inhibition task has significantly predicted subsequent rearrest rates in adult offenders during a follow-up period of up to four years (Aharoni et al., 2013, 2014; Steele et al., 2015). Similarly, a recent study has shown that a model based on structural imaging data from an offender sample involving reduced grey matter volume in the anterior temporal lobes, amygdala, and the orbitofrontal cortex was able to predict reoffending in an another sample of offenders (Kiehl et al., 2018). Although overall predictive models of crime are mostly incomplete, leaving a bulk of variance unexplained, the fact that neural factors have been found to significantly contribute to the prediction of offending over other traditional variables lends support to their importance in understanding antisocial behaviour.

In general, neuroimaging studies have provided a robust evidence base demonstrating that antisocial behaviour is associated with structural and functional deficiencies of the fronto-temporal brain regions**.** On the downside, they cannot answer whether identified neural dysfunctions represent causes or correlates of crime due to the nature of their data (Adolphs, 2016). However, empirical findings coming from the lesion studies, patients with neurodegenerative diseases, and recently emerging experiments employing different brain stimulation techniques can provide crucial insight into a potentially causal character of these links. Indeed, they have supported the notion that neural factors have an important role in the causal chain leading to antisocial behaviours. There are a number of studies documenting cases in which previously socially well adapted individuals developed antisocial behaviour after suffering damage to the orbitomedial prefrontal cortex (Bramham, Morris, Hornak, Bullock, & Polkey, 2009; Meyers et al., 1992). These patients often develop clinical picture known in the literature as “pseudo-psychopathy” (Blumer & Benson, 1975) or “acquired sociopathy” (Damasio, 1994), which reflects personality changes similar to the profile of antisocial personality disorder. It seems that particularly severe antisocial behaviour happens in cases when there is a congenital malformation of the orbitomedial prefrontal cortex, or when its damage occurs early in life (Anderson et al., 1999; Boes et al., 2011). Given the difficulty of recruiting neurological patients with specific focal lesions, a recent study involving 40 patients with brain lesions represents a particularly valuable source of insight into neural underpinnings of crime (Darby, Horn, Cushman, & Fox, 2018). The authors have firstly identified 17 patients with documented temporal link of the brain lesions preceding the onset of criminal behaviour. There was no specific brain region that was affected in all cases, which stresses the complexity of pinpointing neural localization subserving a specific function or behaviour. Identified lesions were scattered throughout the fronto-temporal region, with the orbitomedial prefrontal cortex being the most frequently affected region, followed by the amygdala and the anterior lateral temporal lobe. However, lesion network mapping has revealed that even though spatially dispersed, all lesions belonged to a brain network of functionally connected areas consisting of the orbitomedial prefrontal cortex and the anterior temporal lobes. Finally, these results have been replicated in a sample of 23 patients with a tentative temporal link between neural damage and crime. The replication analysis showed that all lesions were functionally connected to the orbitofrontal cortex, and most were linked to the anterior temporal lobe, amygdala, ventromedial prefrontal cortex, and nucleus accumbens.

Patients with certain neurological diseases represent another valuable source of insight into the causal effects of brain dysfunctions. As the name already suggests, (behaviour variant) frontotemporal dementia represents a neurodegenerative disorder characterised by atrophy of specific frontal and temporal brain areas, particularly the orbitofrontal, anterior cingulate, and anterior insular cortices (Liljegren et al., 2015; Rabinovici & Miller, 2010; Seeley, Crawford, Zhou, Miller, & Greicius, 2009). The resulting core symptoms are apathy, behavioural disinhibition, lack of empathy, and violation of social and moral rules, including engaging in antisocial behaviours, whilst memory and visuospatial ability remain mostly spared (Bang, Spina, & Miller, 2015; Mendez, 2006, 2009; Mendez, Anderson, & Shapira, 2005; Rosenbloom, Schmahmann, & Price, 2012). As the orbitomedial prefrontal cortex is more affected by the neurodegenerative process than the dorsolateral prefrontal cortex (Mendez, 2009; Rosenbloom et al., 2012), these patients show diminished emotional concern for others and impaired emotional moral reasoning, whilst retaining intact moral knowledge and awareness that their antisocial behaviour is wrong (Mendez, Anderson, et al., 2005; Mendez, Chen, Shapira, & Miller, 2005). Research has shown that patients with frontotemporal dementia have significantly higher prevalence of antisocial behaviours compared to the patients with Alzheimer’s disease which affects different brain regions[30] (Diehl-Schmid, Perneczky, Koch, Nedopil, & Kurz, 2013; Hornberger et al., 2011; Liljegren et al., 2015; Miller, Darby, Benson, Cummings, & Miller, 1997). Crime specifically has been found to follow the onset of frontotemporal dementia in about half of the patients (Diehl-Schmid et al., 2013; Mendez, Chen, et al., 2005). Therefore, this body of research seems to suggest that antisocial behaviour is caused by structural impairments of the fronto-temporal regions. Similarly, a study involving patients suffering from pharmacoresistant epilepsy affecting the orbitomedial prefrontal and anterior cingulate cortices revealed that these patients fulfilled diagnostic criteria for antisocial behaviour disorder, with many of them having committed crimes (Trebuchon, Bartolomei, McGonigal, Laguitton, & Chauvel, 2013).

[30] Frontotemporal dementia affects the anterior fronto-temporal regions, whereas Alzheimer’s disease mostly targets the hippocampal and posterior temporal-parietal regions (Liljegren et al., 2015; Miller et al., 1997).

Crucially, this study provided a further proof for the causal role of the affected brain regions in criminal behaviour since antisocial behaviour disappeared in all patients after seizure control had been reestablished.

Finally, recently emerging studies using non-invasive brain stimulation techniques, which induce changes in cortical excitability, have generated experimental data in support of the causal role of the prefrontal cortex in antisocial behaviour. These studies are usually conducted in a form of randomised control trials in which participants are randomly assigned to either an experimental group which receives real stimulation or a control group which receives sham stimulation in order to control for the placebo effects, after which these groups are compared on an outcome variable. Research employing transcranial direct current stimulation to enhance neural activity in the dorsolateral prefrontal cortex in healthy non-offender adults has shown that this intervention is able to reduce aggression (Choy, Raine, & Hamilton, 2018; Dambacher et al., 2015). Namely, the experimental groups which received real stimulation have exhibited reduced proactive behavioural aggression, reported lower likelihood to commit physical and sexual assaults, and judged these assaults as more wrong compared to the sham control groups. These results have been replicated in a sample of male violent offenders in whom experimentally induced increase in the dorsolateral prefrontal cortex excitability has resulted in reduced self-reported aggression (Molero-Chamizo, Martín Riquel, Moriana, Nitsche, & Rivera-Urbina, 2019). Alternatively, there have been also findings that inhibitory stimulation over the dorsolateral prefrontal cortex leads to increased aggressive behaviour (Perach-Barzilay et al., 2013). Specifically, it seems that inhibitory effects of the transcranial magnetic stimulation over the left dorsolateral prefrontal cortex cause a greater increase in aggression compared to the disruption of the right dorsolateral prefrontal functioning.

Converging findings from the neuroimaging studies, patients with neurodegenerative diseases or brain lesions, and experimental brain stimulation studies leads to a conclusion that criminal behaviour is related to the impairments of the dorsolateral prefrontal cortex, orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala, amongst others. This applies to deficiencies related to both structure and activity of these areas. However, only neurocognitive studies can elucidate what specific functions underpinned by these brain areas are deficient in individuals who exhibit antisocial behaviour. Executive functions represent an “umbrella term” used in cognitive psychology to describe a number of cognitive processes that enable purposeful and goal-oriented behaviour (Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011). It has been suggested that there are three core executive functions, namely inhibitory control, working memory, and cognitive flexibility, from which higher-order functions, involving reasoning, planning, and problem-solving, are constructed (Diamond, 2013; Hofmann, Schmeichel, & Baddeley, 2012). However, there is no widely accepted finite list of cognitive processes comprising executive functions which can sometimes cause confusion as a number of different variables are used in their operationalisation across the literature (Ogilvie et al., 2011; Salthouse, 2005; Wikström & Treiber, 2007).

Executive functions have been historically associated with the frontal lobe functioning, but updated overview of neuroimaging and lesion studies suggests that these should not be used synonymously as performance on the executive function tasks seems to be subserved by a broad network of cortical and subcortical brain regions (Alvarez & Emory, 2006; Bettcher et al., 2016; Ogilvie et al., 2011; Zelazo & Müller, 2002). Overall, research shows that common measures of executive functions are sensitive, but not specific to impairment of the prefrontal cortex, and that they draw heavily on other brain regions as well. Equating executive deficiencies with frontal dysfunction might be related to the fact that executive functions were traditionally viewed as rational processes assessed using abstract tasks lacking affective aspects (Zelazo & Carlson, 2012). However, due to the influx of findings stressing the role of emotions and habitual processes in decision-making, the scope of the processes subsumed under executive functions have expanded and they have been recently split into the hot and cool types. Hot executive functions refer to top-down cognitive processes which are characterised by emotional and motivational components (De Brito, Viding, Kumari, Blackwood, & Hodgins, 2013; Zelazo & Carlson, 2012; Zelazo & Müller, 2002). They are assessed with tasks tapping into processes such as response reversal, affective decision-making, and emotional processing (De Brito et al., 2013; Noordermeer et al., 2016). On the other hand, cool executive functions represent top-down cognitive processes that are elicited by abstract and affectively neutral problems (De Brito et al., 2013; Zelazo & Carlson, 2012). They involve functions such as working memory, response inhibition, and attentional flexibility (De Brito et al., 2013; Noordermeer et al., 2016). These two types of executive functions are underpinned by different functional-anatomical neural networks and seem to follow a different developmental trajectory with hot executive functions showing a somewhat delayed development (Gläscher et al., 2012; Zelazo & Carlson, 2012). Hot executive functions are supported by the orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala, whereas cool executive functions rely mainly on the dorsolateral prefrontal cortex (De Brito et al., 2013; Noordermeer et al., 2016; Zelazo

& Müller, 2002). Such neuroanatomical foundation of hot and cool executive functions is in line with their differential relations with the affective and motivational aspects of cognitive processes. Namely, unlike the dorsolateral prefrontal cortex, the orbitomedial prefrontal cortex has strong bilateral connections to the amygdala and other limbic areas crucially associated with emotions and motivation, whereby it is in the perfect position to integrate cognitive and affective information necessary for the hot executive tasks (De Brito et al., 2013; Zelazo & Müller, 2002).

In line with the conclusions of the previously reviewed neuroimaging studies, neuropsychological research has confirmed deficient executive functioning amongst antisocial individuals (Baliousis, Duggan, McCarthy, Huband, & Völlm, 2019; De Brito et al., 2013; Meijers, Harte, Jonker, & Meynen, 2015; Morgan & Lilienfeld, 2000; Noordermeer et al., 2016; Ogilvie et al., 2011; Syngelaki, Moore, Savage, Fairchild, & Van Goozen, 2009). A meta-analysis involving 126 studies has shown a robust association between antisocial behaviour and impaired executive functions that applies to different antisocial groups and executive tasks (Ogilvie et al., 2011). The overall mean effect size was in the medium range ($d = .44$, $p < .001$), with criminality having the largest effect size across different antisocial groups ($d = .61$, $p < .001$). A recent review of studies employing prison population has confirmed that adult offenders tend to perform significantly worse on a number of executive functions compared to non-offender control samples (Meijers et al., 2015). Furthermore, a study which differentiated between the types of executive functions has demonstrated that, compared to a non-offender group, violent offenders exhibited poor performance in tasks assessing both hot and cool executive functions (De Brito et al., 2013). Similarly, a meta-analysis and systematic review exploring neuroimaging findings involving individuals with oppositional defiant disorder/conduct disorder have found that they tend to have smaller volume and lower brain activity in areas underpinning hot executive functions, and to a smaller extent, cool executive functions (Noordermeer et al., 2016).

In sum, converging evidence from neuroscience, neurology, and neuropsychology seems to suggest that antisocial behaviour is associated with dysfunctions in the dorsolateral prefrontal cortex subserving cool executive functions on one hand and the lower fronto-temporal regions involving the orbitomedial prefrontal cortex, anterior cingulate cortex[31],

[31] It is important to note that the anterior cingulate cortex is structurally and functionally heterogenous, and its functions are somewhat more nuanced than presented here (Margulies et al., 2007). Namely, there seems to be a functional division between the rostral and caudal areas of the anterior cingulate cortex wherein the former represents an affective subregion connected with the limbic system and the latter is a cognitive subregion interacting with the dorsolateral prefrontal cortex (Etkin, Egner, & Kalisch, 2011; Margulies et al., 2007). It has

insula, and amygdala, supporting hot executive functions on the other hand. Juxtaposing these areas against the areas identified in the previous section as involved in the central control of heart rate points to a remarkable similarity. Namely, the brain regions underpinning hot executive functions belong to the neural network which regulates heart rate. However, the dorsolateral prefrontal cortex which seems to be heavily involved in different antisocial behaviours is not part of the central autonomic network, and therefore is not expected to be relevant for the explanation of the link between heart rate and crime.

6.1.3. Neural Underpinnings of the Underlying Causal Mechanism

As demonstrated in the previous two sections of this chapter, there is a considerable overlap between neural regions involved in antisocial behaviour on one hand and heart rate control on the other. However, the question remains whether the identified lower fronto-temporal regions are also involved in the mechanisms leading to criminal behaviour as proposed by situational action theory. Therefore, this section will outline neural bases of the personal aspects of the perception-choice process, namely morality, the ability to exercise self-control, and decision-making.

6.1.3.1. Neural basis of crime propensity.

According to situational action theory, a person's crime propensity fundamentally depends on their morality, that is, what one thinks is right or wrong to do in a particular situation and the extent to which one cares about adhering to such norms. Moral rules and moral emotions are therefore inherently intertwined, as moral emotions regulate the strength of one's moral rules. For example, if one thinks it is wrong to steal, but would not feel particularly guilty or ashamed of stealing, then that person is more likely to perceive stealing as an option in a criminogenic setting compared to someone with strong moral emotions. As

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been further suggested that the caudal and rostral regions distinctively support cool and hot executive functions, respectively (Gläscher et al., 2012; Petrovic & Castellanos, 2016). However, it has been suggested recently that both subregions underpin emotional processing, but instead they make different contributions, wherein the rostral area is involved in regulating limbic outputs and the caudal area is involved in emotion evaluation and expression (Etkin et al., 2011).

moral rules are fundamentally a matter of content, this review of the neural underpinnings of morality will instead focus on moral emotions and moral decision-making.

Although there is an abundance of research exploring the neural roots of basic emotions (e.g., anger, fear), only lately there has been an increased interest in understanding moral emotions from a neurobiological perspective (Bastin, Harrison, Davey, Moll, & Whittle, 2016). Moral emotions, such as guilt and shame, represent complex emotions that involve emotional processing, social cognition, and self-referential processes (Bastin et al., 2016; Gifuni, Kendal, & Jollant, 2017; Mclatchie et al., 2016; Roth, Kaffenberger, Herwig, & Brühl, 2014). As self-conscious emotions, they are cognitively demanding and require evaluations of one’s behaviour, but also inferring about other people’s mental states and their view of one’s behaviour (Beer, Heerey, Keltner, Scabini, & Knight, 2003). It is therefore not surprising that neuroimaging studies involving healthy adult participants show that moral emotions engage a distributed network of the frontal, temporal, parietal, and limbic regions. Shame and guilt are normally found to recruit circuits typically involved in affective processing, such as the orbitomedial prefrontal cortex, anterior cingulate cortex, insular cortex, and amygdala, as well as the circuits involved in self-referential processes and social cognition, namely the dorsomedial prefrontal cortex, dorsolateral and ventrolateral prefrontal cortices, posterior cingulate cortex, and precuneus (Bastin et al., 2016; Blair & Fowler, 2008; Fourie, Thomas, Amodio, Warton, & Meintjes, 2014; Gifuni et al., 2017; Gilead, Katzir, Eyal, & Liberman, 2016; Mclatchie et al., 2016; Michl et al., 2014; Molenberghs et al., 2015; Roth et al., 2014; Shin et al., 2000; Ty, Mitchell, & Finger, 2017).

The integrity of the orbitomedial prefrontal cortex seems to be particularly important for enabling a person to experience guilt (Wagner et al., 2011). Indeed, research investigating emotional functioning in patients with focal lesions to the orbitomedial prefrontal cortex has shown that they exhibit severely diminished guilt, whereas they remain perfectly capable of experiencing other emotions such as anger and envy (Koenigs et al., 2007; Krajbich et al., 2009). Such findings have been explained by a theoretical distinction between primary and secondary emotions (Damasio, 1994). Primary emotions, such as fear, anger, and surprise, are considered innate and they emerge early on in life, whereas secondary emotions, such as shame and guilt, occur at later stages as they are built on the basis of the primary emotions. These emotions also have different neuroanatomical foundations, wherein the primary emotions are chiefly supported by the amygdala and the anterior cingulate cortex, whereas the secondary emotions mainly depend on the functioning of the orbitomedial prefrontal cortex. Hence patients with damage to their limbic system are characterised by a more noticeable emotional blunting associated with the impairments in both types of emotions, while patients with the orbitomedial prefrontal cortex lesions may at first look emotionally unimpaired due to spared primary emotions and more subtle nature of deficits affecting moral emotions (Damasio, 1994).

Moral emotions are closely related to moral judgement as any deficit in experiencing shame or guilt is also reflected in non-optimal functioning of moral reasoning. It is therefore not surprising that moral decision-making also shares a similar neural basis consisting of the fronto-limbic areas centred around the orbitomedial prefrontal cortex. According to the functional imaging research, the regions that are most frequently recruited during various moral reasoning tasks are the orbitomedial and dorsolateral prefrontal cortices, insula, anterior and posterior cingulate cortices, precuneus, amygdala, and temporoparietal junction (Blair & Fowler, 2008; Boccia et al., 2017; Eres, Louis, & Molenberghs, 2018; Fede & Kiehl, 2020; Marazziti, Baroni, Landi, Ceresoli, & Dell'Osso, 2013; Mendez, 2009; Raine, 2019; Raine & Yang, 2006; Rosenbloom et al., 2012). As in the case of moral emotions, the orbitomedial prefrontal cortex with its connections to the limbic system has been considered as critical for the development and effective functioning of moral reasoning. Namely, research shows that patients with lesions to the orbitomedial prefrontal cortex that occurred early in ontogenesis have both deficient knowledge about moral rules and immature moral reasoning (Anderson et al., 1999; Boes et al., 2011; Mendez, 2009; Taber-Thomas et al., 2014). Their moral development does not evolve beyond the pre-conventional stage characterised by abnormally egocentric reasoning guided solely by self-interest. On the other hand, patients whose lesions to the orbitomedial prefrontal cortex happened later in life have intact moral knowledge, but their moral reasoning is still impaired (Koenigs et al., 2007; Rosenbloom et al., 2012; Schneider & Koenigs, 2017; Taber-Thomas et al., 2014; Young et al., 2010). They do not endorse immature self-serving rule violations like the early-onset patients, but they do show abnormally utilitarian moral judgment, that is, they endorse harmful actions that lead to aggregate welfare (Greene, 2007). When faced with hypothetical moral dilemmas, the adult-onset patients tend to focus on maximizing the overall benefit even when that involves committing emotionally aversive actions which usually repels healthy participants (e.g., killing one person to save five).

Findings from patients with frontotemporal dementia, who have structurally compromised orbitomedial part of the prefrontal cortex, too are in line with the conclusion regarding the importance of this brain region in moral reasoning. Their knowledge about moral rules and impersonal, rational moral reasoning are largely unaffected, but their emotional moral judgement is remarkably utilitarian (Mendez, 2006; Mendez, Anderson, et al., 2005; Mendez, Chen, et al., 2005). Furthermore, research shows that brain stimulation applied over the medial prefrontal cortex in healthy participants induces changes in moral reasoning by increasing utilitarian responses to certain moral dilemmas (Riva, Manfrinati, Sacchi, Pisoni, & Romero Lauro, 2019). Therefore, there is a substantial evidence base demonstrating that the orbitomedial prefrontal cortex along with its connections to the limbic and paralimbic areas, such as the anterior cingulate cortex and amygdala, are crucial for experiencing moral emotions and applying them in affective moral reasoning.

Converging results from the lesion and brain imaging studies seems to suggest that the orbitomedial prefrontal cortex has an integrative role in moral judgement (Greene, 2007; Shenhav & Greene, 2014). The amygdala is suggested to have a “bottom-up” influence, whereby it provides affective assessment of the situation and sends automatic emotional response to the orbitomedial prefrontal cortex. On the other hand, the dorsolateral prefrontal cortex, the seat of the cool executive functions, supports utilitarian reasoning by conducting aggregate cost-benefit analysis. The role of the anterior cingulate cortex is to detect and recruit a response to a conflict between the negative emotions produced by the amygdala and the overall benefits of the harmful action calculated by the dorsolateral prefrontal cortex. Finally, the orbitomedial prefrontal cortex generates an “all things considered” judgement by integrating the amygdala’s emotional aversiveness and the dorsolateral prefrontal cortex’s utilitarian calculation (Greene, 2007; Shenhav & Greene, 2014).

Even though morality is the fundamental component of crime propensity, the ability to exercise self-control is also important in crime causation under specific circumstances, namely whilst making a deliberate choice in a state of conflict between a person’s morality and an external pressure to transgress. An individual’s ability to exercise self-control depends mainly on the effectiveness of their executive functions to recognise a conflict between their motivation and morality, inhibit responses while deliberating, and finally choose an action that is in line with their morality (Wikström & Treiber, 2007). In other words, it fundamentally depends on the integrity of cool executive functions underpinned by the dorsolateral prefrontal cortex, such as working memory, cognitive control, and response inhibition (Beaver et al., 2007; Wikström & Treiber, 2007). These functions support deliberate decision-making by activating, organising, maintaining, and integrating relevant information into internal representations within one’s virtual cognitive space, whilst also facilitating inhibition of emotional responses (Treiber, 2011, 2014; Wikström & Treiber, 2007).

In addition to the dorsolateral area, the orbitomedial part of the prefrontal cortex is also involved in creation, maintenance, and evaluation of the internal representations that support decision-making and hence exercising self-control (Wikström & Treiber, 2007). If these internal representations are deficient, then a person's behaviour will be driven by external cues which would likely result in impulsive actions. In addition, the orbitomedial prefrontal cortex has an important role in modulating emotional responses. Namely, due to its strong bilateral connections with limbic system that is heavily involved in emotional processing and generating responses, the orbitomedial prefrontal cortex is in the optimal position to exhibit top-down control and suppress emotional reactions in order to support self-control (Beaver et al., 2007; Treiber, 2011; Wikström & Treiber, 2007).

Indeed, research exploring the neural basis of self-control shows its dependence on the structural integrity and optimal functioning of the prefrontal cortex. Empirical evidence demonstrates that the dorsolateral prefrontal cortex is responsible for various functions underpinning the ability to exercise self-control such as working memory, attention, planning, and regulation of impulsive behaviour (Cho et al., 2010, 2012; Szczepanski & Knight, 2014). Similarly, research supports the critical role of the orbitomedial prefrontal cortex in underpinning emotion regulation, impulse control, information integration, adequate frustration tolerance, and other processes fundamental for exerting self-control (Anderson et al., 1999; Boes et al., 2011; Gupta, Koscik, Bechara, & Tranel, 2011; Koenigs et al., 2007; Liljegren et al., 2015; Mendez, Chen, et al., 2005; Meyers et al., 1992; Schneider & Koenigs, 2017; Szczepanski & Knight, 2014; Trebuchon et al., 2013). Overall, it seems that the ability to exercise self-control is supported by both cool, non-affective and hot, affective executive functions underpinned by the dorsolateral and orbitomedial prefrontal cortices, respectively.

6.1.3.2. Neural basis of decision-making.

Besides establishing neural underpinnings of crime propensity, it is also important to outline brain regions supporting the decision-making processes which are relevant to the process of choice, which were briefly touched upon in the previous section on the ability to exercise self-control. After a person perceives crime as an option in a particular situation, whether or not they will pursue such an action will depend on the choice they make (see section 3.3.3.). Two types of choices as outlined by situational action theory, habitual choice and rational deliberate choice, are in line with the two types of decision-making defined by the dual-process theory in cognitive psychology, Type 1 and Type 2 processing, respectively (Evans, 2008; Evans & Stanovich, 2013; Kahneman, 2003, 2011; Stanovich & West, 2000).

Rational deliberation is based on Type 2 processing which is distinguished by cognitive decoupling and dependency on working memory (Evans & Stanovich, 2013). Underpinned by working memory faculties, cognitive decoupling of secondary representations[32] enables mental simulations which are the basis of hypothetical thinking and abstract reasoning (Evans, 2003; Stanovich, 2009; Stanovich & Toplak, 2012). As this process is computationally demanding, it is constrained by the capacity and efficiency of a person's working memory. Due to such heavy loading on limited working memory capacities, this type of processing tends to be slow, deliberative, sequential, conscious, abstract, controlled, effortful, low capacity, and associated with general intelligence (Evans, 2008; Evans, 2003; Evans & Stanovich, 2013). As it is highly dependent on cool executive functions, Type 2 processing is primarily neuroanatomically supported by the dorsolateral prefrontal cortex (Treiber, 2011, 2014; Wikström & Treiber, 2007).

Habitual choice is underpinned by Type 1 processing which is defined by its autonomy and a lack of dependency on working memory (Evans & Stanovich, 2013; Stanovich & Toplak, 2012). Namely, it does not require intervention from the higher-order cognitive systems, but it is rather executed automatically when a triggering stimulus is present. Unlike Type 2 processing, it puts minimal load on working memory and does not involve effortful attention. As such, Type 1 processing tends to be fast, automatic, parallel, unconscious, intuitive, high capacity, contextualised, and uncorrelated with general intelligence (Evans, 2008; Evans & Stanovich, 2013; Stanovich & Toplak, 2012). It involves innate behaviours and experience-based knowledge acquired by associative learning and conditioning (Evans, 2003; Evans & Stanovich, 2013). In addition to facilitating habitual choice, Type 1 processing also has an important role in the perception of action alternatives through its influence on the moral filter (Treiber, 2011, 2014). Neuroanatomically it has been associated with the orbitomedial prefrontal cortex and its affective neural network involving the limbic regions (Treiber, 2011, 2014; Wikström & Treiber, 2007).

The somatic marker hypothesis (Bechara, Damasio, & Damasio, 2003; Damasio, 1994, 1995, 1996; Damasio, Tranel, & Damasio, 1990) provides an explanation of the

[32] Primary representation is an image of the world as it is and it has direct links with one's response to it, whereas a secondary representation is a copy of the primary one which is decoupled from the world and used in simulations (Leslie, 1987; Perner, 1991). There is a need for two separate representations so that the representation of the world as it is does not get mixed with simulated representations of potential responses and their outcomes (Stanovich, 2009; Stanovich & Toplak, 2012).

mechanism through which a neural network centred in the orbitomedial prefrontal cortex supports habitual choice and Type 1 processing. After a series of empirical studies showing that the patients with focal lesions to the orbitomedial prefrontal cortex have severe deficits in experiencing emotions, generating autonomic arousal to socially meaningful stimuli, and making advantageous decisions in personal and social realms (e.g., Bechara, Tranel, Damasio, & Damasio, 1996; Damasio, 1995; Damasio et al., 1990), Damasio and colleagues have concluded that emotions and their attached somatic expressions have an important role in the process of decision-making. They have hypothesised that the orbitomedial prefrontal cortex is responsible for making and storing the experience-based associations between certain classes of situations and emotions that have arisen in such situations. As emotions express themselves primarily, although not exclusively, through changes in the state of the body, these emotional changes have been referred to as somatic markers. These learnt associations are dispositional such that once a person encounters a situation that was in the past linked with a specific emotional reaction, the orbitomedial prefrontal cortex automatically reactivates the appropriate somatic marker. The main function of the somatic markers is to aid the decision-making process by labelling outcomes of possible courses of actions as positive or negative based on the prior experience. In other words, they serve as qualifying signals by automatically rejecting unfavourable choices and endorsing advantageous choices. Indeed, research shows that without the ability to generate and reactivate somatic markers as a fast and automatic guiding system, patients with lesions to the orbitomedial prefrontal cortex are governed solely by immediate prospects and remain oblivious to the future consequences, positive and negative alike (Bechara, Tranel, & Damasio, 2000; Bechara, Damasio, Damasio, & Anderson, 1994).

Due to their biasing role in decision-making, somatic markers facilitate rational deliberation by constricting the number of courses of action a person would consider in a given situation. Otherwise, without somatic markers to quickly and automatically qualify potential responses as favourable or unfavourable, all options would be viewed as equal and that would result in having too many response-outcome pairs to assess in a cost-benefit analysis within a reasonable time period. On the other hand, if the reactivated somatic marker is particularly strong, it will suppress all other options leading to an automatic choice. Therefore, due to the associative and dispositional nature of the somatic markers, they represent a foundation of what situational action theory refers to as habitual choice. Finally, by signalling anticipated shame and guilt following the choice of crime as an action response, somatic markers also have a role in the perception process through its influence on the moral filter. In sum, somatic markers represent a mechanism underlying habitual choice, but they also facilitate the moral filter functioning and rational decision-making (Treiber, 2011, 2014).

Although the orbitomedial prefrontal cortex is most commonly associated with the somatic markers, their unhindered functioning also depends on the integrity of other cortical and subcortical regions, such as the amygdala and insula, and the peripheral nervous system (Bechara, Tranel, & Damasio, 2000; Damasio, 1996; Poppa & Bechara, 2018). Namely, the amygdala is crucial for triggering autonomic reactions to primary inducers, that is, stimuli that unconditionally or through learning induce appetitive or aversive emotions. The orbitomedial prefrontal cortex on the other hand produces autonomic reactions to secondary inducers that represent a memory or a thought of a primary inducer (Bechara et al., 2003; Gupta et al., 2011). Therefore, an intact functioning of the amygdala is a prerequisite for the orbitomedial prefrontal cortex to develop appropriate triggering of somatic reactions to the secondary inducers, after which reactions to these stimuli are less dependent on the amygdala. The orbitomedial prefrontal cortex is necessary to link the aspects of a situation and the emotional reactions induced by the amygdala in relation to a specific outcome by associative learning, but it is also crucial in reactivating these somatic markers once the stored aspect of a situation is encountered. The activation of the somatic markers can be accomplished via "body loop", when the amygdala triggers somatic changes which are then relayed back to the insula, or via "as if loop", when the somatosensory signal goes straight to the insula, without eliciting the somatic changes (Damasio, 1995, 1996). Indeed, research has confirmed that focal lesions to both the amygdala and the orbitomedial prefrontal cortex cause inability to generate anticipatory somatic markers and make advantageous choices (Bechara et al., 1999).

In sum, literature reviewed here seems to suggest that the rational deliberate choice depends on Type 2 processing underpinned mainly by the dorsolateral prefrontal cortex. On the other hand, perception of action alternatives (i.e., moral filter) and habitual choice are supported by Type 1 processing and the somatic markers system, which rely neuroanatomically on the lower fronto-temporal neural network involving the orbitomedial prefrontal cortex, insula, and amygdala.

6.1.4. The Final Piece of the Puzzle: Explaining the Link Between Heart Rate Reactivity and Crime Propensity

Neurocognitive factors have been incorporated in situational action theory as “causes of the causes” as they have indirect causal effect on criminal behaviour through their influence on the perception-choice process (Treiber, 2014). As the review of the empirical studies conducted in this chapter clearly demonstrates, the theoretical framework of crime causation outlined in situational action theory is consistent with current knowledge about how brain networks support morality, self-control, and decision-making. Summing-up different lines of research, it seems there are two functionally and anatomically different neural networks of relevance to crime causation. Specifically, there is a neural network seated in the dorsolateral prefrontal cortex which is crucial for the optimal functioning of cool executive functions which in turn support the ability to exercise self-control and rational deliberate decision-making. The other relevant neural network consists of the middle and lower parts of the prefrontal cortex along with their strong connections to the limbic system, with the orbitomedial prefrontal cortex and the amygdala representing its central nodes. This network is fundamental for hot executive functions which are involved in crime propensity, both morality and to a lesser degree the ability to exercise self-control, and habitual decision-making.

Review of the research findings regarding the localization of the central control of heart rate has converged at a distributed network consisting of the cortical, limbic, and brainstem areas. Juxtaposing brain areas involved in the autonomic regulation on the hand, and causes of crime, on the other hand, has revealed a common network of the fronto-temporal areas, involving the orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala. Importantly, the network centred in the dorsolateral prefrontal cortex is a point of divergence, as it is heavily implicated in crime causation but it is does not belong to the central autonomic network. Therefore, drawing upon research findings from a number of different fields (e.g., neuroscience, neurology, physiology, criminology, cognitive psychology), it is hypothesised that the link between heart rate reactivity and crime propensity is due to their shared neural underpinnings. It is suggested here that the dysfunction of the above-mentioned fronto-temporal areas causes both blunted heart rate reactivity and impairments in morality, ability to exercise self-control, and habitual decision-making that in turn lead to criminal behaviour under certain circumstances (i.e., dependent on the interaction with criminogenic exposure). Therefore, low heart rate reactivity to stress could be understood as a marker of a non-optimal functioning of the affective neural network spanning from the orbitomedial prefrontal cortex, through the anterior cingulate and insular cortices, down to the amygdala, which in turn causes increased crime propensity.

In addition, the suggested model wherein low heart rate reactivity to stress represents a marker of the impairments localised in the lower fronto-limbic areas also has the potential to explain other empirical findings related to blunted autonomic reactivity. For example, some studies have found that low heart rate reactivity to threat cues in women, but not their resting heart rate, is associated with retrospective and prospective sexual victimization (Soler-Baillo, Marx, & Sloan, 2005; Waldron, Wilson, Patriquin, & Scarpa, 2015). These results have been explained by an underlying disability to recognise and respond to threat stimuli which would otherwise help them avoid dangerous situations. Indeed, this interpretation is in line with the proposed model given that effective detection, processing, and response to threat depend on the integrity of the aforementioned fronto-limbic circuitry (Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Fisher et al., 2011; Ginty, Gianaros, Derbyshire, Phillips, & Carroll, 2013; Hu, 2018; Isenberg et al., 1999; Öhman, 2005). A finding that low heart rate measured in 18-year-old men during a conscription assessment is associated with higher likelihood of having unintentional injures later in life can also be potentially explained along the same lines (Latvala et al., 2015).

Finally, the model presented above is in line with recent developments in the fields of behavioural medicine and physiology. Namely, since the late 1970s it has been considered that exaggerated heart rate reactions to stress are associated with the development of cardiovascular diseases as outlined by the reactivity hypothesis (Obrist, 1976, 1981). However, recent evidence has shown that blunted autonomic reaction to acute stress is not benign nor protective as it was initially thought, but rather it is associated with adverse health and behaviour outcomes, such as addictions, impulsivity, depression, bulimia, obesity, lower cognitive ability, and poor self-reported health (Bibbey et al., 2016; Carroll, Ginty, Whittaker, Lovallo, & de Rooij, 2017; Carroll, Lovallo, & Phillips, 2009; Gao et al., 2015; Ginty et al., 2013, 2011). To account for these findings, it has been suggested that blunted physiological reactivity to stress may be a peripheral marker of sub-optimal functioning of the fronto-limbic areas that support motivated behaviour, emotion regulation, and purposeful behaviour (Bibbey et al., 2016; Carroll et al., 2017, 2009; Ginty et al., 2013; Lovallo, 2013). Specifically, the central motivational dysregulation model proposes that the dysfunctions of the prefrontal cortex and the limbic system cause reduced stress reactivity, impulsive and short-term oriented cognitive processing, and unstable affect regulation, which in turn lead to impulsive behaviours associated with poor health outcomes (Lovallo, 2013).

The reviewed support for the outlined model of the relationship between blunted heart rate reactivity and crime is only circumstantial. Although predictions deduced from this model are straightforward and falsifiable, it was not possible to fully test this model in the Cambridge adolescent behaviour study as it was unfeasible to obtain the necessary neuroimaging data. However, the study included some neurocognitive measures to enable preliminary testing of the complete crime propensity model which is presented in the next section.

6.2. Testing the Complete Crime Propensity Model

This chapter will be concluded by presenting the results of the preliminary test of the extended crime propensity model which incorporates the neurocognitive causes of the crime propensity into the explanation of the relationship between heart rate reactivity and crime. Firstly, specific hypotheses stemming from the proposed model will be outlined, followed by a methods section. Next, the results of the statistical analyses will be presented. The chapter will be brought to an end by a discussion of the results.

6.2.1. Study Hypotheses

The analyses that will be presented in the following sections were conducted with aim to test the complete crime propensity model of the relationship between heart rate and crime as outlined in this chapter. Briefly, the model states that the subtle perturbations specific to the lower fronto-temporal regions supporting hot executive functions cause both low heart rate reactivity and increased crime propensity, that is, low morality and weak ability to exercise self-control. It is further hypothesised that high crime propensity leads to criminal behaviour, whereas the low heart rate reactivity is not part of this causal chain. In order to document the specificity of the lower fronto-temporal areas and hot executive functions, this study also included a test of a control model wherein the dysfunction of the dorsolateral prefrontal cortex and its associated cool executive functions is used instead of the measure of the fronto-temporal functioning. It is hypothesised that the dysfunction of the dorsolateral prefrontal cortex leads to increased crime propensity, and therefore crime itself, but that it does not cause blunted heart rate reactivity.

Therefore, it is expected that a task tapping into hot executive functions will be on one hand negatively associated with crime propensity and criminal behaviour, and on the other hand positively associated with heart rate reactivity. As for the control model, it is predicted that a task tapping into cool executive functions will be negatively associated with criminal propensity and crime, but that it will not show a significant association with heart rate reactivity. It is further expected that the former model involving hot executive functions will fit the data better. As for the bivariate relationships regarding individual crime propensity components, it is expected that the ability to exercise self-control will be positively associated with both cool and hot executive functions, whereas guilt and shame will demonstrate positive relationships only with hot executive function and will be unrelated to cool executive function.

6.2.2. Methods

As the following analyses were conducted using data from the Cambridge adolescent behaviour study, all relevant information concerning methodology used is contained in the Chapter 4. Only the neurocognitive measures will be briefly commented on here, namely the working memory and emotion recognition tasks which were used to tap into the functioning of the dorsolateral prefrontal cortex and the lower fronto-temporal regions centred around the orbitomedial prefrontal cortex and the amygdala, respectively.

Digit Span Backwards task (see section 4.3.2.1.) measures verbal working memory which represents a core cool executive function. It involves both maintenance and manipulation of information in the short-term memory, and fundamentally depends on the integrity of the dorsolateral prefrontal cortex (De Brito et al., 2013; Diamond, 2013; Szczepanski & Knight, 2014). Working memory has been specifically associated with the functioning of the dorsolateral areas of the prefrontal cortex, as patients with damage to the orbitomedial regions have in general intact cool executive functions (Bechara, Tranel, Damasio, & Damasio, 1996; Damasio et al., 1990; Saver & Damasio, 1991).

Functioning of the lower fronto-temporal areas was measured using the emotion recognition task (see section 4.3.2.2.) indexing hot executive functioning (McDonald, 2013; Zimmerman, Ownsworth, O’Donovan, Roberts, & Gullo, 2016). Accurate recognition of facial expressions of emotions depends on a distributed network of the fronto-temporal regions, particularly the orbitomedial prefrontal cortex and the amygdala (Adolphs, 2002; Mendez, 2006; Yang et al., 2007). A number of lesion studies have documented that following damage to the orbitomedial prefrontal cortex, these patients experience severe deficits in recognising a range of different emotions from facial cues (Hornak et al., 2003; Hornak, Rolls, & Wade, 1996; Rosenbloom et al., 2012; Schneider & Koenigs, 2017; Szczepanski & Knight, 2014). It is important to note that specifically the orbitomedial areas of the prefrontal cortex are fundamental for the general ability to recognise emotions, as the damage to the dorsolateral regions of the prefrontal cortex has not been associated with these deficits (Heberlein et al., 2008; Hornak et al., 2003). Historically, the amygdala was associated primarily with fear recognition, but it is now widely accepted that it has an important role in general emotion processing as it has been found that its damage leads to inability to recognise a range of emotions (Adolphs, 2002; Ashwin, Chapman, Colle, & Baron-Cohen, 2006; Yang et al., 2007).

6.2.3. Statistical Analyses

The bivariate relationships between the neurocognitive variables, crime propensity, heart rate reactivity, and crime were assessed in the same manner as in the previous analyses (see section 5.2.2.). Two models involving different neurocognitive variables as the causes of crime propensity and heart rate reactivity were specified. In the first model, hot executive function was the exogenous variable which predicted crime propensity and heart rate reactivity, with the former in turn predicting criminal behaviour. All variables were observed, with the exception of crime propensity which was a latent variable manifested by guilt, shame, and the ability to exercise self-control. These three observed variables were previously reversed for their use in the structural equation modelling such that high crime propensity reflected weak moral emotions and poor ability to exercise self-control. Residual variances of guilt and shame were allowed to correlate, as it was believed that being moral emotions they had something in common that was not captured by crime propensity. By default, the factor loading of guilt was fixed to one in order to fix the scale of the crime propensity. The second model was identical except that the exogenous variable was cool executive function. Parameter estimates were derived using maximum likelihood method with robust, bootstrapped standard errors calculated from 5000 bootstrap samples due to some deviations from normality in the data. Robust bias-corrected and accelerated 95% confidence intervals using 5000 samples were produced for each path coefficient value. There were no missing values for shame, guilt, and crime, whereas there was one missing value for the ability to exercise self-control, three each for hot and cool executive functions, and five for heart rate reactivity. Missing data were estimated using the full information maximum likelihood method.

The significance of each model will be assessed using three criteria (Schumacker & Lomax, 2016). Firstly, the chi-square test as a measure of global fit should be non-significant. Secondly, individual path values specified by the model should be significant. Finally, the direction and the size of the parameter values should be theoretically meaningful. In line with Kline's (2016) suggestions, the following fit statistics were reported in addition to the chi-square: root mean square error of approximation (RMSEA) along with its 90% confidence interval, comparative fit index (CFI), and standardised root mean square residual (SRMR). RMSEA values below .08 and .06 represent acceptable and good fit, respectively; CFI values above .90 and .95 represent acceptable and good fit, respectively; SRMR values below .06 represent good fit (Brown, 2015). Finally, Bayesian information criterion (BIC) will be used to compare which model fits the data better. Lower values indicate better fitting model.

6.2.4. Results

As predicted, emotion recognition which measured the lower fronto-temporal functioning showed significant correlations in the expected direction with all the other variables. Namely, there was a significant negative correlation with criminal behaviour, $r = -.20$, 95% BCa CI [- .33, - .07], $p < .001$, whereas it was positively correlated with heart rate reactivity, $r = .10$, 95% BCa CI [.03, .18], $p = .024$, shame, $r = .11$, 95% BCa CI [.01, .22], $p = .015$, guilt, $r = .16$, 95% BCa CI [.06, .26], $p < .001$, and the ability to exercise self-control, $r = .14$, 95% BCa CI [.05, .22], $p = .003$. In line with the hypotheses, working memory which tapped into the dorsolateral prefrontal functioning was significantly correlated with the ability to exercise self-control, $r = .12$, 95% BCa CI [.03, .22], $p = .007$, whereas there were no significant correlations with shame, $r = .03$, 95% BCa CI [- .06, .13], $p = .474$, guilt, $r = .09$, 95% BCa CI [- .01, .18], $p = .06$, and heart rate reactivity, $r = .01$, 95% BCa CI [- .09, .11], $p = .807$. However, contrary to the predictions, working memory was not significantly associated with involvement in crime, r = - .07, 95% BCa CI [- .16, .02], p = .138.

The measurement model consisted of one latent variable defined by three indicators, and therefore the model fit could not be evaluated. However, factor loadings were statistically significant (p < .001) and their standardised values ranged between .62 for the ability to exercise self-control to .75 for guilt.

The model assessing hot executive function as the cause of the cause was evaluated first. There was no violation of the assumption of multicollinearity. An examination of the correlations between emotion recognition, heart rate reactivity, shame, guilt, and ability to exercise self-control showed no extreme multicollinearity as the largest correlation was r = .71, p < .001, found between shame and guilt. The lack of multicollinearity was further confirmed by the collinearity statistics, as all tolerance statistics were well above the threshold of .10 and all VIF values were well below 10.0. The model fit the data well as the chi-square was non-significant, all path values were significant and in the expected directions, and the model fit indices showed a good fit of the data to the hypothesised structure. The model fit indices were as follows: χ^2 (8, N = 487) = 12.70, p = .123; RMSEA = .04, 90% CI [.00, .07]; CFI = .99; SRMR = .04. Emotion recognition significantly predicted both crime propensity, b = - 0.02, 95% BCa CI [- 0.03, - 0.01], p < .001, and heart rate reactivity, b = 0.17, 95% BCa CI [0.05, 0.29], p = .009, in the expected directions. In turn, crime propensity significantly predicted criminal behaviour, b = 1.30, 95% BCa CI [1.05, 1.60], p < .001. The graph illustrating this structural model with the standardised path coefficients is presented in Figure 6.1.

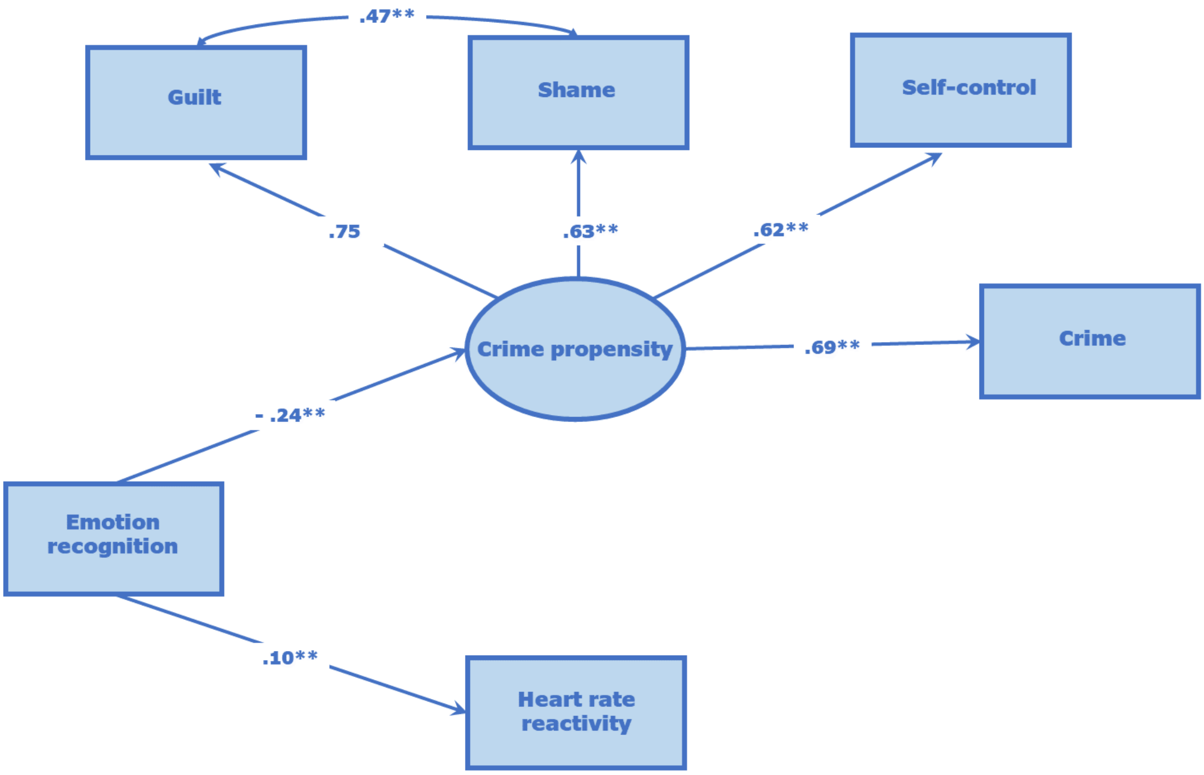

Figure 6.1. Fronto-temporal structural model. Standardised parameter estimates are shown for each path (N = 487).
*p < .05. **p < .01.

The control model assessing cool executive function as the cause of the cause was evaluated next. As in the case of the previous model, there was no violation of the assumption of multicollinearity. An examination of correlations between working memory, heart rate reactivity, shame, guilt, and ability to exercise self-control showed no extreme multicollinearity as the largest correlation was the one found between shame and guilt. The lack of multicollinearity was further confirmed by the collinearity statistics, as all tolerance statistics were well above the threshold of .10 and all VIF values were well below 10.0. The analysis showed that the structural model with the dorsolateral prefrontal functioning did not fit the data adequately. The chi-square was significant χ^2 (8, N = 487) = 16.52, p = .036, implying that the observed and the implied theoretical variance-covariance matrices were significantly different. Other model fit indices were as follows: RMSEA = .05, 90% CI [.01, .08]; CFI = .99; SRMR = .05. As predicted, working memory did not predict changes in heart rate reactivity, b = 0.03, 95% BCa CI [- 0.25, 0.32], p = .828, and increased crime propensity did significantly predict higher crime involvement, b = 1.27, 95% BCa CI [1.04, 1.55],

p < .001. However, working memory predicted crime propensity only marginally, b = - 0.01, 95% BCa CI [- 0.03, 0.00], p = .054. The graph illustrating this structural model with the standardised path coefficients is presented in Figure 6.2. Finally, BIC values showed that the fronto-temporal model (BIC = 5393.201) provided a better fit in comparison to the dorsolateral prefrontal cortex model (BIC = 5418.639).

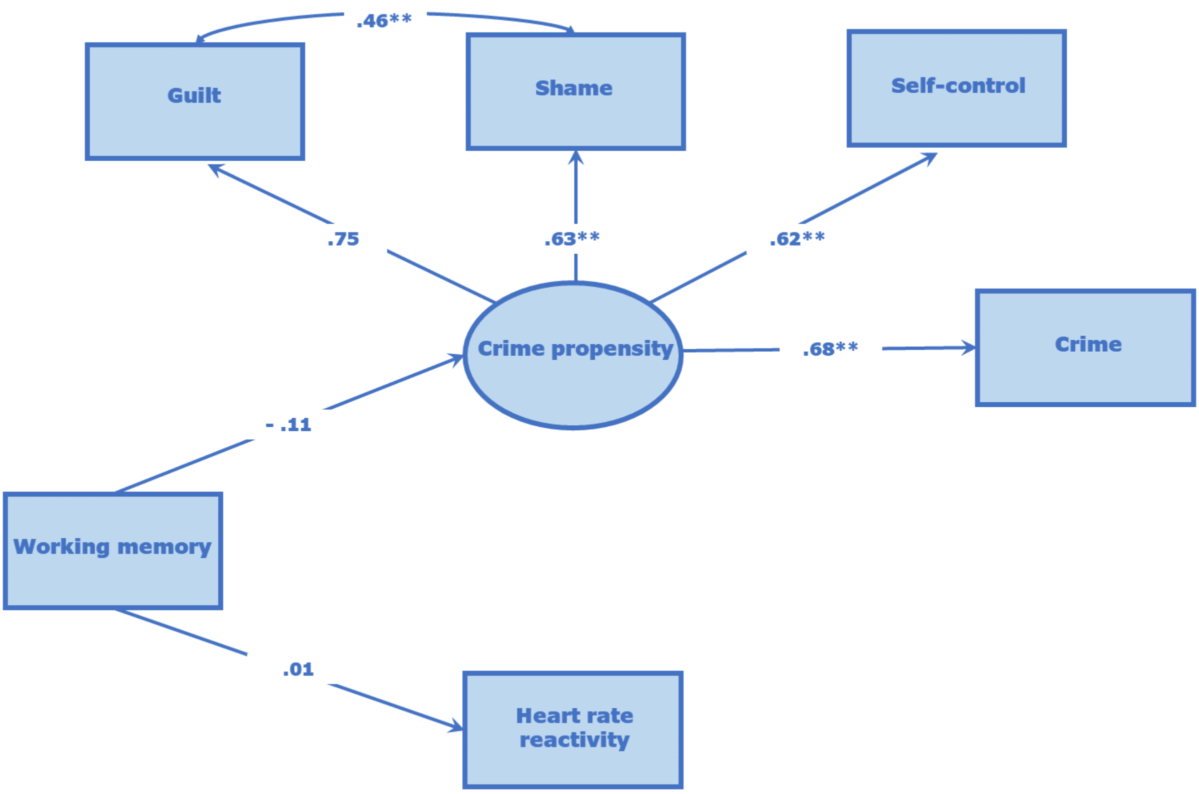

Figure 6.2. Dorsolateral prefrontal cortex structural model. Standardised parameter estimates are shown for each path (N = 487).
*p < .05. **p < .01.

6.2.5. Discussion

Overall, the results seem to confirm the hypotheses outlined at the beginning of this section. Most importantly, the complete explanatory model of the relationship between heart rate and crime, which incorporated neurocognitive factors, received empirical backing. Data from the Cambridge adolescent behaviour study supported the explanation wherein blunted heart rate reactivity on one hand and high crime propensity and in turn criminal behaviour itself on the other hand originate from the shared impairments of hot executive functions. The subtle dysfunctions of the fronto-temporal areas centred around the orbitomedial prefrontal cortex and the amygdala seem to cause diminished moral emotions, poor ability to exercise self-control, and blunted heart rate reactivity to acute stress. Therefore, the inverse association between heart rate reactivity and crime is likely spurious and confounded by the common causal factor. Equally importantly, the results demonstrated specificity of the common cause given that the underlying neural dysfunction did not seem to be global, but instead confined to the lower prefrontal and limbic areas supporting the hot executive functions. Specifically, the model wherein cool executive functioning of the dorsolateral prefrontal cortex was tested as the common source of both low heart rate reactivity and increased crime propensity was not supported by the existing data. Therefore, the fronto-temporal model was supported by both the theoretical review laid out in the first half of this chapter and the empirical results that followed in the second half.

Looking at the results concerning the bivariate relationships, specific hypotheses referring to the functioning of the lower fronto-temporal regions were all supported by the data. Namely, as predicted, poor hot executive functioning was associated with increased criminal behaviour, diminished moral emotions, and low ability to exercise self-control. Indeed, these results are also in line with a number of prior studies which have demonstrated impaired ability to recognise emotional expressions among antisocial populations (Bowen, Morgan, Moore, & Van Goozen, 2014; Dawel, O'Kearney, McKone, & Palermo, 2012; Hoaken, Allaby, & Earle, 2007; Marsh & Blair, 2008). As for the dorsolateral prefrontal cortex functioning, all hypotheses involving individual crime propensity variables were empirically endorsed. Involvement of the cool executive functions in supporting the ability to exhibit self-control, as outlined in situational action theory, was confirmed (Treiber, 2014; Wikström & Treiber, 2007). It is also important to note the aggregate pattern of results relating to the ability to exercise self-control. Namely, although biosocial criminology tends to associate self-control with purely cognitive executive processes (e.g., Beaver et al., 2007; Cauffman et al., 2005; Wikström & Treiber, 2007), the theoretical review and results outlined in this chapter suggest that self-control involves top-down control of both affective and cognitive processes. Therefore, it seems reasonable to revise the mainstream understanding of the ability to exercise self-control by including more explicitly not only cool, but hot executive functions as well (Buckholtz, 2015). Finally, the expected inverse relationship between cool executive functions and criminal behaviour was not supported by the data. The lack of a significant association is somewhat unexpected, but it is not utterly surprising given the overall weak links between neurocognitive factors and crime due to a number of mediating processes happening in between (Treiber, 2014). In addition, these results confirm the more important role of habitual processes in criminal decision-making in comparison to rational deliberation as suggested by situational action theory (Treiber, 2014).

Although the empirical results presented here provide clear support for the fronto-temporal model of the link between blunted heart rate reactivity and engagement in crime, they should be interpreted in light of the study's limitations. Firstly, the collected data did not include structural or functional neuroimaging measurements pertaining to the brain regions of interest which would help strengthen the overall conclusions. Secondly, both hot and cool executive functioning were each assessed using a single instrument. Given the inherent difficulties of designing tasks that would tap exclusively into hot or cold executive processes and specific neural regions (Zelazo & Müller, 2002), it is necessary to replicate these results using different neurocognitive measures. Therefore, the obtained results should be interpreted as providing only preliminary support for the fronto-temporal model stressing the common cause underlying both heart rate reactivity and crime propensity. Future studies are strongly encouraged to test the extended crime propensity model using neuroimaging data and a battery of neurocognitive tasks which would enable drawing more robust conclusions.

Chapter 7:

The Importance of Delineating Causality: Key Findings

The final chapter will be concise and to the point wherein the main contributions of the Cambridge adolescent behaviour study will be recapitulated. Firstly, the need to embrace an analytical approach in biosocial criminology and, indeed, the whole field in general, was explained and emphasised in Chapter 1. In the past years, biosocial criminology has come a long way from being an outlier to representing “a new paradigm capable of guiding criminology in the 21st century” (Cullen, 2009, p. xvii). It has brought (back) biology and all the attached advancements in science and technology into the field of criminology, and stressed the importance of the interaction between biological and environmental factors in understanding criminal behaviour. As any theory which aims to explain crime while ignoring the human body is incomplete, biosocial criminology has indeed advanced the field by introducing a missing piece of the criminological puzzle. However, if the biosocial paradigm truly aims to revolutionise and guide criminology in this century, it needs to move from the risk-factor approach of piling up hundreds of correlates of crime without trying to understand how these numerous factors are actually associated with crime. Instead, it is necessary to sort out which few of many correlates are causally related to crime, and by what means. The overarching goal of this study was to contribute to making such a paradigm shift in biosocial criminology by taking one of the most prominent correlates of crime, low heart rate, and establishing its relation to crime.

Secondly, a thorough review of the research literature on the connection between heart rate and a range of antisocial behaviours, including crime, was conducted throughout Chapter 1. This led to a conclusion that this link is indeed robust as suggested in many prior reviews and meta-analyses. However, this meticulous analysis of the empirical studies also resulted in a completely new insight regarding the fundamental nature of the relationship of interest. Namely, the conclusions of many studies which have reported a negative association between resting heart rate and antisocial behaviour proved questionable given their methodological characteristics. In many cases, these studies have either measured resting heart rate in unstandardised conditions, which different participants could have experienced differently, or in potentially stressful circumstances. Therefore, there is a strong possibility that such studies have in fact measured heart rate reactivity under the false label of resting heart rate. Furthermore, the studies which have measured resting heart rate under standardised conditions for eliciting baseline level have very often failed to find a significant relationship with antisocial behaviours. On the other hand, studies which have measured heart rate in response to stress, although fewer in numbers, have usually reported finding significant results not only with antisocial behaviours, but with a range of other outcomes, such as threat detection, addiction, and impulsivity. This observation gave rise to a hypothesis that antisocial behaviour is associated with blunted heart rate reactivity, whereas it is unrelated to resting heart rate.

Thirdly, Chapter 2 contains a comprehensive overview of the proposed theoretical explanations of the inverse link between heart rate and antisocial behaviours. Some of these explanations are more established, like the fearlessness and stimulation-seeking hypotheses, whereas others have been only briefly mentioned in the literature and are less known, such as the autonomic feedback and right hemisphere functioning hypotheses. All proposals were subjected to theoretical evaluation in order to test their usefulness in providing a sound explanation of the heart rate – antisocial behaviour association. However, the result of this review led to a conclusion that not a single theoretical explanation offered a viable solution which could withstand a detailed scrutiny. In general, these hypotheses have been based upon unfounded and/or questionable assumptions. In addition, few empirical studies which have been conducted in order to test some of these explanations, primarily the fearlessness and stimulation-seeking hypotheses, were reviewed as well. In line with theoretical evaluation, the research findings were unconvincing having in mind identified methodological issues and the actual statistical results.

Fourthly, after pinpointing the problems plaguing the existing theoretical explanations of the empirical connection between low heart rate and antisocial behaviour, Chapter 3 followed up with outlining a new, viable model that would be able to account for this phenomenon. Employing situational action theory as a theoretical framework of the analysis, it was argued that heart rate cannot be a cause of crime, and that instead it was a marker of a cause of crime. As heart rate represents a personal characteristic, it was suggested that its association with criminal behaviour could be explained through crime propensity. Specifically, it was hypothesised that low heart reactivity correlates with weak morality and poor ability to exercise self-control which comprise high crime propensity that in turn leads to criminal behaviour in certain situations. Therefore, the crime propensity model predicted that the relationship between heart rate and crime would be fully explained by the mediating effect of crime propensity.

Next, Chapters 4 and 5 presented the Cambridge adolescent behaviour study and its results, respectively. This study was specifically designed to address the research questions that were raised in the prior chapters. Its principal goal was to test the new model of the heart rate – crime association outlined in Chapter 3, and to compare it with two well-established, yet unlikely explanations, the fearlessness and stimulation-seeking hypotheses. In addition, the study aimed to address other important uncertainties that were identified in the preceding review. Primarily, it was of paramount importance to establish whether criminal behaviour was associated with heart rate reactivity to stress, resting heart rate, or both. This was dealt with by employing standardised protocols of measuring heart rate under resting and stressful conditions. Next, both fearlessness and stimulation-seeking concepts were measured using valid instruments which has not always been the case in the prior studies. This made it possible to have a pure measure of stimulation-seeking and avoid its conflations with impulsivity, an issue that has plagued much of the previous research. At last, an outcome measure assessed criminal behaviour specifically, and therefore avoided the usual pitfalls of using a loosely defined concept of antisocial behaviour. In addition, the instrument used to measure criminal behaviour also included items that tapped into cyber-crime which has been strikingly lacking in the criminological research.

The results have firstly established the exact nature of the link between low heart rate and crime. Namely, it was found that crime was inversely associated only with heart rate reactivity to stress, and was unrelated to resting heart rate. However, the most profound finding from the Cambridge adolescent behaviour study was that the newly developed crime propensity model fully accounted for the link between heart rate reactivity and engagement in crime. As for the other two models, the fearlessness hypothesis did not mediate the link at all, whereas the stimulation-seeking exhibited only partial mediation. In fact, the latter ceased to be a significant mediator once the ability to exercise self-control, a construct distinct from stimulation-seeking, but related to it, was accounted for in the model. Therefore, the empirical results did not support the most prominent explanations of the link between heart rate and antisocial behaviour. Moreover, the results gave rise to a hypothesis that the prior findings pertaining to the significant mediation effects of stimulation-seeking could be due to its conflation with impulsivity. The study also provided an important insight into the cyber-crime activity of English youth which is a completely new finding.

Finally, the complete crime propensity model which aimed to include an explanation of the link between crime propensity and heart rate reactivity was outlined in detail throughout Chapter 6. Given that heart rate cannot be a cause of crime propensity, it was argued that their relationship is spurious and that it could be explained by their links to a common, third factor. By converging evidence from neuroscience, criminology, and cognitive psychology, a theoretical model was proposed wherein both increased crime propensity and blunted heart rate reactivity resulted from the same neurocognitive dysfunction viewed as “the cause of the cause”. Specifically, it was argued that the subtle perturbations in the lower fronto-temporal regions involving the orbitomedial prefrontal cortex, anterior cingulate cortex, insula, and amygdala, caused diminished moral emotions and poor ability to exercise self-control, which in turn led to criminal behaviour in certain situations. The same neural dysfunction was proposed to cause blunted heart rate reactivity to stress. In addition to a review of empirical findings and theoretical work supporting this model, it was also empirically tested using data from the Cambridge adolescent behaviour study. In the analyses, two models were compared with regards to their fit to the data. The first model involved a measure of hot executive functions which tapped into the functioning of the lower fronto-temporal regions as the common cause of both heart rate reactivity and crime propensity, whereas the other model employed a measure of cool executive functions underpinned by the dorsolateral prefrontal cortex, a designated control brain region. The results provided empirical support for the first model whereby the dysfunction of the lower fronto-temporal regions leads to both blunted heart rate reactivity and increased crime propensity that in turn causes criminal behaviour. As predicted, the alternative, control model was not supported by the empirical results.

Therefore, by outlining a clear and precise theoretical mechanism underlying the relationship between low heart rate and crime, and providing the empirical results to support it, this study has contributed towards tidying up the knowledge base in biosocial criminology and disentangling correlates from the causes of crime in an effort to bring the analytical paradigm into (biosocial) criminology.

List of References

Adjorlolo, S. (2017). Biological protective factors of criminal offending: Implications for risk assessment research and practice. *The Journal of Forensic Psychiatry & Psychology, 28*(1), 108–132. https://doi.org/10.1080/14789949.2016.1261175

Adolphs, R. (2002). Neural systems for recognizing emotion. *Current Opinion in Neurobiology*, *12*, 169–177. https://doi.org/10.1016/S0959-4388(02)00301-X

Adolphs, R. (2016). Human lesion studies in the 21st Century. *Neuron*, *90*, 1151–1153. https://doi.org/10.1016/j.neuron.2016.05.014

Afifi, T. O., Fortier, J., Sareen, J., & Taillieu, T. (2019). Associations of harsh physical punishment and child maltreatment in childhood with antisocial behaviors in adulthood. *JAMA Network Open*, *2*(1), 1–10. https://doi.org/10.1001/jamanetworkopen.2018.7374

Aharoni, E., Vincent, G. M., Harenski, C. L., Calhoun, V. D., Sinnott-Armstrong, W., Gazzaniga, M. S., & Kiehl, K. A. (2013). Neuroprediction of future rearrest. *Proceedings of the National Academy of Sciences*, *110*(15), 6223–6228. https://doi.org/10.1073/pnas.1219302110

Aharoni, Eyal, Mallett, J., Vincent, G. M., Harenski, C. L., Calhoun, V. D., Sinnott-Armstrong, W., … Kiehl, K. A. (2014). Predictive accuracy in the neuroprediction of rearrest. *Social Neuroscience*, *9*(4), 332–336. https://doi.org/10.1080/17470919.2014.907201

Al-Safi, S. A. (2005). Does smoking affect blood pressure and heart rate? *European Journal of Cardiovascular Nursing*, *4*(4), 286–289. https://doi.org/10.1016/j.ejcnurse.2005.03.004

Aladin, A. I., Whelton, S. P., Al-Mallah, M. H., Blaha, M. J., Keteyian, S. J., Juraschek, S. P., … Michos, E. D. (2014). Relation of resting heart rate to risk for all-cause mortality by gender after considering exercise capacity (the Henry Ford exercise testing project). *American Journal of Cardiology, 114*(11), 1701–1706. https://doi.org/10.1016/j.amjcard.2014.08.042

Aleman, A., & Van’T Wout, M. (2008). Repetitive transcranial magnetic stimulation over the right dorsolateral prefrontal cortex disrupts digit span task performance. *Neuropsychobiology, 57*, 44–48. https://doi.org/10.1159/000129666

Allen, M. T., Hogan, A. M., & Laird, L. K. (2009). The relationships of impulsivity and cardiovascular responses : The role of gender and task type. *International Journal of Psychophysiology, 73*(3), 369–376. https://doi.org/10.1016/j.ijpsycho.2009.05.014

Alvarez, J. A., & Emory, E. (2006). Executive function and the frontal lobes: A meta-analytic review. *Neuropsychology Review*, *16*, 17–42. https://doi.org/10.1007/s11065-006-9002-x

Alvarez, R. P., Biggs, A., Chen, G., Pine, D. S., & Grillon, C. (2008). Contextual fear conditioning in humans: cortical-hippocampal and amygdala contributions. *The Journal of Neuroscience*, *28*(24), 6211–6219. https://doi.org/10.1523/JNEUROSCI.1246-08.2008

American Psychiatric Association. (2013). Disruptive, impulse-control, and conduct disorders. In *Diagnostic and statistical manual of mental disorders (5th ed.)*. Washington, DC: Author. https://doi.org/10.1176/appi.books.9780890425596.dsm15

Amrhein, V., Greenland, S., & Mcshane, B. (2019). Retire statistical significance. *Nature*, *567*, 305–307. https://doi.org/10.1038/d41586-019-00857-9

Anastasi, A. (1988). *Psychological testing* (6th ed). New York: Macmillan.

Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature Neuroscience*, *2*(11), 1032–1037. https://doi.org/10.1038/14833

Antonaccio, O., Botchkovar, E. V., & Hughes, L. A. (2017). Ecological determinants of situated choice in situational action theory: Does neighborhood matter? *Journal of Research in Crime and Delinquency*, *54*(2), 208–243. https://doi.org/10.1177/0022427816678908

Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, *10*(3), 229–240. https://doi.org/10.1037/1089-2680.10.3.229

Armstrong, T. A. (2011). The relationship between low resting heart rate and antisocial behavior: Correlation or causation? In K. M. Beaver & A. Walsh (Eds.), *The Ashgate Research Companion to Biosocial Theories of Crime* (pp. 45–68). Farnham, Surrey: Ashgate Publishing Limited.

Armstrong, T. A., Boisvert, D., Flores, S., Symonds, M., & Gangitano, D. (2017). Heart rate, serotonin transporter linked polymorphic region (5-HTTLPR) genotype, and violence in an incarcerated sample. *Journal of Criminal Justice*, *51*, 1–8. https://doi.org/10.1016/j.jcrimjus.2017.05.012

Armstrong, T. A., & Boutwell, B. B. (2012). Low resting heart rate and rational choice: Integrating biological correlates of crime in criminological theories. *Journal of Criminal Justice*, *40*(1), 31–39. https://doi.org/10.1016/j.jcrimjus.2011.11.001

Armstrong, T. A., Keller, S., Franklin, T. W., & Macmillan, S. N. (2009). Low resting heart rate and antisocial behavior: A brief review of evidence and preliminary results from a new test. *Criminal Justice and Behavior*, *36*(11), 1125–1140. https://doi.org/10.1177/0093854809342855

Arnett, J. (1994). Sensation seeking: A new conceptualization and a new scale. *Personality and Individual Differences*, *16*(2), 289–296. https://doi.org/10.1016/0191-8869(94)90165-1

Ashton, M. C., & Lee, K. (2001). A theoretical basis for the major dimensions of personality. *European Journal of Personality*, *15*, 327–353. https://doi.org/10.1002/per.417

Ashton, M. C., & Lee, K. (2007). Empirical, theoretical, and practical advantages of the HEXACO model of personality structure. *Personality and Social Psychology Review*, *11*(2), 150–166. https://doi.org/10.1177/1088868306294907

Ashton, M. C., & Lee, K. (2008). The prediction of Honesty-Humility-related criteria by the HEXACO and Five-Factor Models of personality. *Journal of Research in Personality*, *42*(5), 1216–1228. https://doi.org/10.1016/j.jrp.2008.03.006

Ashton, M. C., & Lee, K. (2018). How well do Big Five measures capture HEXACO scale variance? *Journal of Personality Assessment*. *101*(6), 567-573. https://doi.org/10.1080/00223891.2018.1448986

Ashton, M. C., Lee, K., & De Vries, R. E. (2014). The HEXACO Honesty-Humility, Agreeableness, and Emotionality factors: A review of research and theory. *Personality and Social Psychology Review*, *18*(2), 139–152. https://doi.org/10.1177/1088868314523838

Ashwin, C., Chapman, E., Colle, L., & Baron-Cohen, S. (2006). Impaired recognition of negative basic emotions in autism: A test of the amygdala theory. *Social Neuroscience*, *1*(3–4), 349–363. https://doi.org/10.1080/17470910601040772

Baddeley, A. D. (1992). Working memory. *Science*, 255(5044), 556–559. https://doi.org/10.1126/science.1736359

Baddeley, A. D. (1998). Recent developments in working memory. *Current Opinion in Neurobiology*, *8*(2), 234–238. https://doi.org/10.1016/S0959-4388(98)80145-1

Baker, L. A., Tuvblad, C., Reynolds, C., Zheng, M., Lozano, D. I., & Raine, A. (2009). Resting heart rate and the development of antisocial behavior from age 9 to 14: Genetic and environmental influences. *Development and Psychopathology*, *21*(3), 939–960. https://doi.org/10.1017/S0954579409000509

Bal, E., Harden, E., Lamb, D., Van Hecke, A. V., Denver, J. W., & Porges, S. W. (2010).

Emotion recognition in children with autism spectrum disorders: Relations to eye gaze and autonomic state. *Journal of Autism and Developmental Disorders*, *40*(3), 358–370. https://doi.org/10.1007/s10803-009-0884-3

Baliousis, M., Duggan, C., McCarthy, L., Huband, N., & Völlm, B. (2019). Executive function, attention, and memory deficits in antisocial personality disorder and psychopathy. *Psychiatry Research*, *278*, 151–161. https://doi.org/10.1016/j.psychres.2019.05.046

Bang, J., Spina, S., & Miller, B. L. (2015). Frontotemporal dementia. *The Lancet*, *386*(10004), 1672–1682. https://doi.org/10.1016/S0140-6736(15)00461-4

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Luan Phan, K. (2007). Amygdala-frontal connectivity during emotion regulation. *Social Cognitive and Affective Neuroscience*, *2*(4), 303–312. https://doi.org/10.1093/scan/nsm029

Barbey, A. K., Koenigs, M., & Grafman, J. (2013). Dorsolateral prefrontal contributions to human working memory. *Cortex*, *49*(5), 1195–1205. https://doi.org/10.1016/j.cortex.2012.05.022

Barnes, J. C., & Boutwell, B. B. (Eds.). (2015). Biosocial criminology [Special issue]. *Criminal Justice Studies*, *28*(1).

Bastin, C., Harrison, B. J., Davey, C. G., Moll, J., & Whittle, S. (2016). Feelings of shame, embarrassment and guilt and their neural correlates: A systematic review. *Neuroscience and Biobehavioral Reviews*, *71*, 455–471. https://doi.org/10.1016/j.neubiorev.2016.09.019

Bear, M.F., Connors, B.W., & Paradiso, M.A. (2016). *Neuroscience: Exploring the brain* (4th ed.). Philadelphia: Wolters Kluwer.

Beaver, K. M., & Walsh, A. (2011). *The Ashgate research companion to biosocial theories of crime*. Farnham, Surrey: Ashgate Publishing Limited. https://doi.org/10.4324/9781315612768.ch10

Beaver, K. M., Eagle Schutt, J., Boutwell, B. B., Ratchford, M., Roberts, K., & Barnes, J. C. (2009). Genetic and environmental influences on levels of self-control and delinquent peer affiliation results from a longitudinal sample of adolescent twins. *Criminal Justice and Behavior*, *36*(1), 41–60. https://doi.org/10.1177/0093854808326992

Beaver, K. M., Wright, J. P., & DeLisi, M. (2007). Self-control as an executive function: Reformulating Gottfredson and Hirschi’s parental socialization thesis. *Criminal Justice and Behavior*, *34*(10), 1345–1361.

Beaver, K. M., Wright, J. P., DeLisi, M., & Vaughn, M. G. (2008). Genetic influences on the stability of low self-control: Results from a longitudinal sample of twins. *Journal of Criminal Justice*, *36*(6), 478–485. https://doi.org/10.1016/j.jcrimjus.2008.09.006

Beaver, K. M, & Walsh, A. (2011). Biosocial Criminology. In K. M. Beaver & A. Walsh (Eds.), *The Ashgate Research Companion to Biosocial Theories of Crime* (pp. 3–16). Farnham, Surrey: Ashgate.

Bechara, A., Tranel, D., & Damasio, H. (2000). Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain*, *123*, 2189–2202. https://doi.org/10.1093/brain/123.11.2189

Bechara, A. (2004). The role of emotion in decision-making: Evidence from neurological patients with orbitofrontal damage. *Brain and Cognition*, *55*(1), 30–40. https://doi.org/10.1016/j.bandc.2003.04.001

Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, *50*, 7–15. https://doi.org/10.1016/0010-0277(94)90018-3

Bechara, A., Damasio, H., & Damasio, A. R. (2003). Role of the amygdala in decision-making. *Annals of the New York Academy of Sciences*, *985*(1), 356–369. https://doi.org/10.1111/j.1749-6632.2003.tb07094.x

Bechara, A., Damasio, H., Damasio, A. R., & Lee, G. P. (1999). Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *The Journal of Neuroscience*, *19*(13), 5473–5481. https://doi.org/0270-6474/99/19135473-09$05.00/0

Bechara, A., Tranel, D., Damasio, H., & Damasio, A. R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cerebral Cortex*, *6*, 215–225. https://doi.org/10.1093/cercor/6.2.215

Beer, J. S., Heerey, E. A., Keltner, D., Scabini, D., & Knight, R. T. (2003). The regulatory function of self-conscious emotion: Insights from patients with orbitofrontal damage. *Journal of Personality and Social Psychology*, *85*(4), 594–604. https://doi.org/10.1037/0022-3514.85.4.594

Beissner, F., Meissner, K., Bär, K. J., & Napadow, V. (2013). The autonomic brain: An activation likelihood estimation meta-analysis for central processing of autonomic function. *Journal of Neuroscience, 33*(25), 10503–10511. https://doi.org/10.1523/JNEUROSCI.1103-13.2013

Benarroch, E. E. (1993). The central autonomic network: Functional organization, dysfunction, and perspective. *Mayo Clinic Proceedings*, *68*(10), 988–1001.

https://doi.org/10.1016/S0025-6196(12)62272-1

Benarroch, E. E. (1997). The central autonomic network. In P. A. Low (Ed.), *Clinical Autonomic Disorders* (2nd ed., pp. 17–23). Philadelphia: Lippincott-Raven.

Benbouzid, B. (2019). To predict and to manage. Predictive policing in the United States. *Big Data and Society*, 1–13. https://doi.org/10.1177/2053951719861703

Bennett, C., Blissett, J., Carroll, D., & Ginty, A. T. (2014). Rated and measured impulsivity in children is associated with diminished cardiac reactions to acute psychological stress. *Biological Psychology*, *102*, 68–72. https://doi.org/10.1016/j.biopsycho.2014.07.009

Bernard, T. J. (1990). Twenty years of testing theories: What have we learned and why? *Journal of Research in Crime and Delinquency*, *27*(4), 325–347. https://doi.org/10.1177/0022427890027004002

Bettcher, B. M., Mungas, D., Patel, N., Elofson, J., Dutt, S., Wynn, M., … Kramer, J. H. (2016). Neuroanatomical substrates of executive functions: Beyond prefrontal structures. *Neuropsychologia*, *85*, 100–109. https://doi.org/10.1016/j.neuropsychologia.2016.03.001

Bibbey, A., Ginty, A. T., Brindle, R. C., Phillips, A. C., & Carroll, D. (2016). Blunted cardiac stress reactors exhibit relatively high levels of behavioural impulsivity. *Physiology & Behavior*, *159*, 40–44. https://doi.org/10.1016/j.physbeh.2016.03.011

Bimmel, N., Van Ijzendoorn, M. H., Bakermans-Kranenburg, M. J., Juffer, F., & De Geus, E. J. C. (2008). Problem behavior and heart rate reactivity in adopted adolescents: Longitudinal and concurrent relations. *Journal of Research on Adolescence*, *18*(2), 201–214. https://doi.org/10.1111/j.1532-7795.2008.00557.x

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

Birn, R. M., Patriat, R., Phillips, M. L., Germain, A., & Herringa, R. J. (2014). Childhood maltreatment and combat posttraumatic stress differentially predict fear-related fronto-subcortical connectivity. *Depression and Anxiety*, *31*, 880–892. https://doi.org/10.1002/da.22291

Blair, R. J. R., & Fowler, K. (2008). Moral emotions and moral reasoning from the perspective of affective cognitive neuroscience: A selective review. *European Journal of Developmental Science*, *2*(3), 303–323. https://doi.org/10.3233/DEV-2008-2308

Blair, R. J. R. (2007a). Dysfunctions of medial and lateral orbitofrontal cortex in psychopathy. *Annals of the New York Academy of Sciences*, *1121*, 461–479. https://doi.org/10.1196/annals.1401.017

Blair, R. J. R. (2007b). The amygdala and ventromedial prefrontal cortex in morality and psychopathy. *Trends in Cognitive Sciences*, *11*(9), 387–392. https://doi.org/10.1016/j.tics.2007.07.003

Blais, J., Solodukhin, E., & Forth, A. E. (2014). A meta-analysis exploring the relationship between psychopathy and instrumental versus reactive violence. *Criminal Justice and Behavior*, *41*(7), 797–821. https://doi.org/10.1177/0093854813519629

Bleick, C. R., & Abrams, A. I. (1987). The transcendental meditation program and criminal recidivism in California. *Journal of Criminal Justice*, *15*(3), 211–230. https://doi.org/10.1016/0047-2352(87)90045-6

Blumer, D., & Benson, D. F. (1975). Personality changes with frontal lobe lesions. In D. Blumer & D. F. Benson (Eds.), *Psychiatric aspects of neurological disease* (pp. 151–170). Grune and Stratton: Grune and Stratton.

Boccia, M., Dacquino, C., Piccardi, L., Cordellieri, P., Guariglia, C., Ferlazzo, F., … Giannini, A. M. (2017). Neural foundation of human moral reasoning: An ALE meta-analysis about the role of personal perspective. *Brain Imaging and Behavior*, *11*(1), 278–292. https://doi.org/10.1007/s11682-016-9505-x

Boes, A. D., Grafft, A. H., Joshi, C., Chuang, N. A., Nopoulos, P., & Anderson, S. W. (2011). Behavioral effects of congenital ventromedial prefrontal cortex malformation. *BMC Neurology*, *11*(151), 1–11. https://doi.org/10.1186/1471-2377-11-151

Boes, A. D., Tranel, D., Anderson, S. W., & Nopoulos, P. (2008). Right anterior cingulate: A neuroanatomical correlate of aggression and defiance in boys. *Behavioral Neuroscience*, *122*(3), 677–684. https://doi.org/10.1037/0735-7044.122.3.677

Boisvert, D., Wells, J., Armstrong, T., Lewis, R. H., Woeckener, M., & Nobles, M. R. (2017). Low resting heart rate and stalking perpetration. *Journal of Interpersonal Violence*, 1–26. https://doi.org/10.1177/0886260517698823

Bootsman, F. (2019). Neurobiological intervention and prediction of treatment outcome in the juvenile criminal justice system. *Journal of Criminal Justice, 85*, 1-10. https://doi.org/https://doi.org/10.1016/j.jcrimjus.2018.05.001

Bouret, S., & Richmond, B. J. (2010). Ventromedial and orbital prefrontal neurons differentially encode internally and externally driven motivational values in monkeys. *Journal of Neuroscience*, *30*(25), 8591–8601. https://doi.org/10.1523/JNEUROSCI.0049-10.2010

Bowen, K. L., Morgan, J. E., Moore, S. C., & Van Goozen, S. H. M. (2014). Young offenders' emotion recognition dysfunction across emotion intensities: Explaining variation using psychopathic traits, conduct disorder and offense severity. *Journal of Psychopathology and Behavioral Assessment, 36,* 60–73. https://doi.org/10.1007/s10862-013-9368-z

Bowring, A. L., Peeters, A., Freak-Poli, R., Lim, M. S. C., Gouillou, M., & Hellard, M. (2012). Measuring the accuracy of self-reported height and weight in a community-based sample of young people. *BMC Medical Research Methodology*, *12*(1), 1471–2288. https://doi.org/10.1186/1471-2288-12-175

Bramham, J., Morris, R. G., Hornak, J., Bullock, P., & Polkey, C. E. (2009). Social and emotional functioning following bilateral and unilateral neurosurgical prefrontal cortex lesions. *Journal of Neuropsychology*, *3*(Pt 1), 125–143. https://doi.org/10.1348/174866408X293994

Breivik, G., Roth, W. T., & Jørgensen, P. E. (1998). Personality, psychological states and heart rate in novice and expert parachutists. *Personality and Individual Differences*, *25*(2), 365–380. https://doi.org/10.1016/S0191-8869(98)00058-0

Brennan, P. A., Raine, A., Schulsinger, F., Kirkegaard-Sorensen, L., Knop, J., Hutchings, B., … Mednick, S. A. (1997). Psychophysiological protective factors for male subjects at high risk for criminal behavior. *American Journal of Psychiatry*, *154*(6), 853–855. https://doi.org/10.1176/ajp.154.6.853

Brennan, T., Dieterich, W., & Ehret, B. (2009). Evaluating the predictive validity of the Compas risk and needs assessment system. *Criminal Justice and Behavior*, *36*(1), 21–40. https://doi.org/10.1177/0093854808326545

Brevet-Aeby, C., Brunelin, J., Iceta, S., Padovan, C., & Poulet, E. (2016). Prefrontal cortex and impulsivity: Interest of noninvasive brain stimulation. *Neuroscience and Biobehavioral Reviews*, *71*, 112–134. https://doi.org/10.1016/j.neubiorev.2016.08.028

Brown, T. A. (2015). *Confirmatory factor analysis for applied research* (2nd ed.). London: The Guilford Press.

Bruinsma, G. (2016). Proliferation of crime causation theories in an era of fragmentation: Reflections on the current state of criminological theory. *European Journal of Criminology*, *13*(6), 659–676. https://doi.org/10.1177/1477370816667884

Brunner, S., Herbel, R., Drobesch, C., Peters, A., Massberg, S., Kääb, S., & Sinner, M. F. (2017). Alcohol consumption, sinus tachycardia, and cardiac arrhythmias at the Munich Octoberfest: Results from the Munich Beer Related Electrocardiogram Workup Study

(MunichBREW). *European Heart Journal*, *38*(27), 2100-2106. https://doi.org/10.1093/eurheartj/ehx156

Brzozowski, A., Gillespie, S. M., Dixon, L., & Mitchell, I. J. (2018). Cardiac autonomic function and psychological characteristics of heterosexual female perpetrators of intimate partner physical aggression. *Journal of Interpersonal Violence*. https://doi.org/10.1177/0886260518775748

Burt, C. H. (2020). Self-control and crime: Beyond Gottfredson & Hirschi's theory. *Annual Review of Criminology*, *3,* 43-73. https://doi.org/10.1146/annurev-criminol-011419-041344

Buckholtz, J. W. (2015). Social norms, self-control, and the value of antisocial behavior. *Current Opinion in Behavioral Sciences*, *3,* 122-129. https://doi.org/10.1016/j.cobeha.2015.03.004

Burt, C. H., & Simons, R. L. (2014). Pulling back the curtain on heritability studies: Biosocial criminology in the postgenomic era. *Criminology*, *52*(2), 223–262. https://doi.org/10.1111/1745-9125.12050

Burt, C. H., Sweeten, G., & Simons, R. L. (2014). Self-control through emerging adulthood: Instability, multidimensionality, and criminological significance. *Criminology*, *52*(3), 450–487. https://doi.org/10.1111/1745-9125.12045

Bush, K., Kivlahan, D. R., McDonell, M. B., Fihn, S. D., & Bradley, K. A. (1998). The AUDIT alcohol consumption questions (AUDIT-C): An effective brief screening test for problem drinking. *Archives of Internal Medicine*, *158*(16), 1789–1795. https://doi.org/10.1001/archinte.158.16.1789

Bushway, S. D., Paternoster, R., & Brame, R. (2003). Examining the prevalence of criminal desistance. *Criminology*, *41*(2), 423–448. https://doi.org/10.1111/j.1745-9125.2003.tb00993.x

Button, D. M., & Worthen, M. G. F. (2017). Applying a general strain theory framework to understand school weapon carrying among LGBQ and heterosexual youth. *Criminology*, *55*(4), 806–832. https://doi.org/10.1111/1745-9125.12151

Byrd, A. L., & Manuck, S. B. (2014). MAOA, childhood maltreatment, and antisocial behavior: Meta-analysis of a gene-environment interaction. *Biological Psychiatry, 75*(1), 9–17. https://doi.org/10.1016/j.biopsych.2013.05.004

Bzdok, D., Schilbach, L., Vogeley, K., Schneider, K., Laird, A. R., Langner, R., & Eickhoff, S. B. (2012). Parsing the neural correlates of moral cognition: ALE meta-analysis on morality, theory of mind, and empathy. *Brain Structure and Function*, *217*(4), 783–796.

https://doi.org/10.1007/s00429-012-0380-y

Caballero, A., Granberg, R., & Tseng, K. Y. (2016). Mechanisms contributing to prefrontal cortex maturation during adolescence. *Neuroscience and Biobehavioral Reviews*, *70*, 4–12. https://doi.org/10.1016/j.neubiorev.2016.05.013

Carrier, N., & Walby, K. (2014). Ptolemizing Lombroso: The pseudo-revolution of biosocial criminology. *Journal of Theoretical and Philosophical Criminology*, *6*(1), 1–45.

Carroll, D., Ginty, A. T., Whittaker, A. C., Lovallo, W. R., & de Rooij, S. R. (2017). The behavioural, cognitive, and neural corollaries of blunted cardiovascular and cortisol reactions to acute psychological stress. *Neuroscience and Biobehavioral Reviews*, *77*, 74–86. https://doi.org/10.1016/j.neubiorev.2017.02.025

Carroll, D., Lovallo, W. R., & Phillips, A. C. (2009). Are large physiological reactions to acute psychological stress always bad for health? *Social and Personality Psychology Compass*, *3*(5), 725–743. https://doi.org/10.1111/j.1751-9004.2009.00205.x

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., … Fuster, V. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, *297*(5582), 851–854. https://doi.org/10.1126/science.1072290

Cauffman, E., Steinberg, L., & Piquero, A. R. (2005). Psychological, neuropsychological and physiological correlates of serious antisocial behavior in adolescence: The role of self-control. *Criminology*, *43*(1), 133–176. https://doi.org/10.1111/j.0011-1348.2005.00005.x

Chester, D. S., Lynam, D. R., Milich, R., & DeWall, C. N. (2017). Physical aggressiveness and gray matter deficits in ventromedial prefrontal cortex. *Cortex*, *97*, 17–22. https://doi.org/10.1016/j.cortex.2017.09.024

Cho, S. S., Ko, J. H., Pellecchia, G., Van Eimeren, T., Cilia, R., & Strafella, A. P. (2010). Continuous theta burst stimulation of right dorsolateral prefrontal cortex induces changes in impulsivity level. *Brain Stimulation*, *3*(3), 170–176. https://doi.org/10.1016/j.brs.2009.10.002

Cho, S. S., Pellecchia, G., Ko, J. H., Ray, N., Obeso, I., Houle, S., & Strafella, A. P. (2012). Effect of continuous theta burst stimulation of the right dorsolateral prefrontal cortex on cerebral blood flow changes during decision making. *Brain Stimulation*, *5*(2), 116–123. https://doi.org/10.1016/j.brs.2012.03.007

Choy, O., Focquaert, F., & Raine, A. (2020). Benign biological interventions to reduce offending. *Neuroethics*, *13*, 29–41. https://doi.org/10.1007/s12152-018-9360-0

Choy, O., Raine, A., & Hamilton, R. H. (2018). Stimulation of the prefrontal cortex reduces intentions to commit aggression: A randomized, double-blind, placebo-controlled, stratified, parallel-group trial. *Journal of Neuroscience*, *38*(29), 6505–6512. https://doi.org/10.1523/JNEUROSCI.3317-17.2018

Choy, O., Raine, A., Portnoy, J., Rudo-Hutt, A., Gao, Y., & Soyfer, L. (2015). The mediating role of heart rate on the social adversity-antisocial behavior relationship: A social neurocriminology perspective. *Journal of Research in Crime and Delinquency*, *52*(3), 303–341. https://doi.org/10.1177/0022427814565905

Choy, O., Raine, A., Venables, P. H., & Farrington, D. P. (2017). Explaining the gender gap in crime: The role of heart rate. *Criminology*, *55*(2), 465–487. https://doi.org/10.1111/1745-9125.12138

Christensen, H., Boysen, G., Christensen, A. F., & Johannesen, H. H. (2005). Insular lesions, ECG abnormalities, and in outcome in acute stroke. *Journal of Neurology, Neurosurgery and Psychiatry*, *76*(2), 269–271. https://doi.org/10.1136/jnnp.2004.037531

Ciaramelli, E., Muccioli, M., Làdavas, E., & Di Pellegrino, G. (2007). Selective deficit in personal moral judgment following damage to ventromedial prefrontal cortex. *Social Cognitive and Affective Neuroscience*, *2*(2), 84–92. https://doi.org/10.1093/scan/nsm001

Cima, M., Tonnaer, F., & Hauser, M. D. (2010). Psychopaths know right from wrong but don’t care. *Social Cognitive and Affective Neuroscience*, *5*(1), 59–67. https://doi.org/10.1093/scan/nsp051

Coccaro, E. F., Cremers, H., Fanning, J., Nosal, E., Lee, R., Keedy, S., & Jacobson, K. C. (2018). Reduced frontal grey matter, life history of aggression, and underlying genetic influence. *Psychiatry Research - Neuroimaging*, *271*, 126–134. https://doi.org/10.1016/j.pscychresns.2017.11.007

Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry*, *62*(2), 168–178. https://doi.org/10.1016/j.biopsych.2006.08.024

Coccaro, E. F., Sripada, C. S., Yanowitch, R. N., & Phan, K. L. (2011). Corticolimbic function in impulsive aggressive behavior. *Biological Psychiatry*, *69*(12), 1153–1159. https://doi.org/10.1016/j.biopsych.2011.02.032

Colasante, T., & Malti, T. (2017). Resting heart rate, guilt, and sympathy: A developmental psychophysiological study of physical aggression. *Psychophysiology*, *54*(1), 1770–1781. https://doi.org/10.1111/psyp.12915

Collado, A., Felton, J. W., MacPherson, L., & Lejuez, C. W. (2014). Longitudinal trajectories of sensation seeking, risk taking propensity, and impulsivity across early to middle adolescence. *Addictive Behaviors*, *39*(11), 1580–1588. https://doi.org/10.1016/j.addbeh.2014.01.024

Cooke, D. J., Michie, C., Hart, S. D., & Clark, D. A. (2004). Reconstructing psychopathy: Clarifying the significance of antisocial and socially deviant behavior in the diagnosis of psychopathic personality disorder. *Journal of Personality Disorders, 18*(4), 337–357. https://doi.org/10.1521/pedi.2004.18.4.337

Copestake, S., Gray, N. S., & Snowden, R. J. (2011). A comparison of a self-report measure of psychopathy with the psychopathy checklist-revised in a UK sample of offenders. *Journal of Forensic Psychiatry and Psychology*, *22*(2), 169–182. https://doi.org/10.1080/14789949.2010.545134

Cornet, L. J. M. (2015). Using basic neurobiological measures in criminological research. *Crime Science*, *4*(7), 1–16. https://doi.org/10.1186/s40163-015-0018-5

Costa, P. T., & McCrae, R. R. (1992). *Revised NEO Personality Inventory (NEO PI-R) and the Five Factor Inventory (NEO-FFI): Professional manual*. Odessa, FL: Psychological Assessment Resources. https://doi.org/10.1016/S0191-8869(03)00118-1

Côté, S., Vaillancourt, T., LeBlanc, J. C., Nagin, D. S., & Tremblay, R. E. (2006). The development of physical aggression from toddlerhood to pre-adolescence: A nation wide longitudinal study of Canadian children. *Journal of Abnormal Child Psychology*, *34*(1), 71–85. https://doi.org/10.1007/s10802-005-9001-z

Cox, D., Hallam, R., O'Connor, K., & Rachman, S. (1983). An experimental analysis of fearlessness and courage. *British Journal of Psychology*, *74*, 107–117. https://doi.org/10.1111/j.2044-8295.1983.tb01847.x

Craig, J. M. (2019). Extending situational action theory to white-collar crime. *Deviant Behavior*, *40*(2), 171–186. https://doi.org/10.1080/01639625.2017.1420444

Critchley, H. D., Mathias, C. J., Josephs, O., O'Doherty, J., Zanini, S., Dewar, B. K., … Dolan, R. J. (2003). Human cingulate cortex and autonomic control: Converging neuroimaging and clinical evidence. *Brain*, *126*(10), 2139–2152. https://doi.org/10.1093/brain/awg216

Cryer, P. E., Haymond, M. W., Santiago, J. V., & Shah, S. D. (1976). Norepinephrine and epinephrine release and adrenergic mediation of smoking-associated hemodynamic and metabolic events. *New England Journal of Medicine*, *295*(11), 573–577. https://doi.org/10.1056/NEJM197609092951101

Cullen, F. T. (2009). Foreword. In *Biosocial criminology: New directions in theory and research* (pp. xv–xvii). New York, NY: Routledge.

Currie, J., & Tekin, E. (2012). Understanding the cycle: Childhood maltreatment and future crime. *Journal of Human Resources*, *47*(2), 509–549. https://doi.org/10.3368/jhr.47.2.509

Curtis, C. E., & D'Esposito, M. (2003). Persistent activity in the prefrontal cortex during working memory. *Trends in Cognitive Sciences*, *7*(9), 415–423. https://doi.org/10.1016/S1364-6613(03)00197-9

D'Esposito, M., Postle, B. R., & Rypma, B. (2000). Prefrontal cortical contributions to working memory: Evidence from event-related fMRI studies. *Experimental Brain Research*, *133*(1), 3–11. https://doi.org/10.1007/s002210000395

Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Putnam.

Damasio, A. R. (1995). On Some Functions of the Human Prefrontal Cortex. *Annals of the New York Academy of Sciences*, *769*, 241–251.

Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *351*(1346), 1413–1420. https://doi.org/10.1098/rstb.1996.0125

Damasio, A. R., Tranel, D., & Damasio, H. (1990). Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. *Behavioural Brain Research*, *41*(2), 81–94. https://doi.org/10.1016/0166-4328(90)90144-4

Dambacher, F., Schuhmann, T., Lobbestael, J., Arntz, A., Brugman, S., & Sack, A. T. (2015). Reducing proactive aggression through non-invasive brain stimulation. *Social Cognitive and Affective Neuroscience*, *10*(10), 1303–1309. https://doi.org/10.1093/scan/nsv018

Darby, R. R., Horn, A., Cushman, F., & Fox, M. D. (2018). Lesion network localization of criminal behavior. *Proceedings of the National Academy of Sciences of the United States of America*, *115*(3), 601–606. https://doi.org/10.1073/pnas.1706587115

Davidson, R. J. (1984). Affect, cognition and hemispheric specialization. In C. E. Izard, J. Kagan, & R. Zajonc (Eds.), *Emotion, cognition and behavior* (pp. 320–365). New York: Cambridge University Press.

Davidson, R. J. (1987). Cerebral asymmetry and the nature of emotion: Implications for the study of individual differences and psychopathology. In R. Takahashi, P. Flor-Henry, J. Gruzelier, & S. Niwa (Eds.), *Cerebral dynamics, laterality and psychopatholoogy* (pp. 71–83). New York: Elsevier.

Davidson, R. J. (1992). Anterior cerebral asymmetry and the nature of emotion. *Brain and Cognition*, *20*(1), 125–151. https://doi.org/10.1016/0278-2626(92)90065-T

Dawel, A., O'Kearney, R., McKone, E., & Palermo, R. (2012). Not just fear and sadness: Meta-analytic evidence of pervasive emotion recognition deficits for facial and vocal expressions in psychopathy. *Neuroscience and Biobehavioral Reviews*, *36*(10), 2288–2304. https://doi.org/10.1016/j.neubiorev.2012.08.006

De Brito, S. A., Viding, E., Kumari, V., Blackwood, N., & Hodgins, S. (2013). Cool and hot executive function impairments in violent offenders with antisocial personality disorder with and without psychopathy. *PLOS ONE*, *8*(6), 1–12. https://doi.org/10.1371/journal.pone.0065566

De La Cruz, F., Schumann, A., Köhler, S., Reichenbach, J. R., Wagner, G., & Bär, K. J. (2019). The relationship between heart rate and functional connectivity of brain regions involved in autonomic control. *NeuroImage*, *196*, 318–328. https://doi.org/10.1016/j.neuroimage.2019.04.014

De Morree, H. M., Rutten, G. J., Szabo, B. M., Sitskoorn, M. M., & Kop, W. J. (2016). Effects of insula resection on autonomic nervous system activity. *Journal of Neurosurgical Anesthesiology*, *28*(2), 153–158. https://doi.org/10.1097/ANA.0000000000000207

De Vries-Bouw, M., Popma, A., Vermeiren, R., Doreleijers, T. A. H., Van De Ven, P. M., & Jansen, L. M. C. (2011). The predictive value of low heart rate and heart rate variability during stress for reoffending in delinquent male adolescents. *Psychophysiology*, *48*(11), 1597–1604. https://doi.org/10.1111/j.1469-8986.2011.01233.x

De Wied, M., Boxtel, A. Van, Posthumus, J. A., Goudena, P. P., & Matthys, W. (2009). Facial EMG and heart rate responses to emotion-inducing film clips in boys with disruptive behavior disorders. *Psychophysiology*, *46*(5), 996–1004. https://doi.org/10.1111/j.1469-8986.2009.00851.x

De Wied, M., Van Boxtel, A., Matthys, W., & Meeus, W. (2012). Verbal, facial and autonomic responses to empathy-eliciting film clips by disruptive male adolescents with high versus low callous-unemotional traits. *Journal of Abnormal Child Psychology*, *40*(2), 211–223. https://doi.org/10.1007/s10802-011-9557-8

Dedovic, K., Renwick, R., Mahani, N. K., Engert, V., Lupien, S. J., & Pruessner, J. C. (2005). The Montreal Imaging Stress Task: Using functional imaging to investigate the effects of perceiving and processing psychosocial stress in the human brain. *Journal of Psychiatry and Neuroscience*, *30*(5), 319–325.

DeLisi, M. (2009). Special issue on biosocial criminology [Special issue]. *Criminal Justice and Behavior, 36*(11).

DeLisi, M. & Vaughn, M. G. (2015). *The Routledge international handbook of biosocial criminology*. Abingdon, England: Routledge.

Detica. (2011). *The cost of cyber crime.* Retrieved from https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/60942/THE-COST-OF-CYBER-CRIME-SUMMARY-FINAL.pdf

Deutz, M. H. F., Woltering, S., Vossen, H. G. M., Deković, M., van Baar, A. L., & Prinzie, P. (2019). Underlying psychophysiology of dysregulation: Resting heart rate and heart rate reactivity in relation to childhood dysregulation. *Journal of the American Academy of Child and Adolescent Psychiatry*, *58*(6), 589–599. https://doi.org/10.1016/j.jaac.2018.09.434

Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, *64*(1), 135–168. https://doi.org/10.1146/annurev-psych-113011-143750

Diehl-Schmid, J., Perneczky, R., Koch, J., Nedopil, N., & Kurz, A. (2013). Guilty by suspicion? Criminal behavior in frontotemporal lobar degeneration. *Cognitive and Behavioral Neurology*, *26*(2), 73–77. https://doi.org/10.1097/WNN.0b013e31829cff11

Dierckx, B., Kok, R., Tulen, J. H. M., Jaddoe, V. W., Hofman, A., Verhulst, F. C., … Tiemeier, H. (2014). A prospective study of heart rate and externalising behaviours in young children. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *55*(4), 402–410. https://doi.org/10.1111/jcpp.12175

Dietrich, A., Riese, H., Sondeijker, F. E. P. L., Greaves-Lord, K., Van Roon, A. M., Ormel, J., … Rosmalen, J. G. M. (2007). Externalizing and internalizing problems in relation to autonomic function: A population-based study in preadolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *46*(3), 378–386. https://doi.org/10.1097/CHI.0b013e31802b91ea

Domjan, M. (2015). *The essentials of learning and behavior* (7th ed.). Belmont, CA: Wadsworth Publishing. https://doi.org/10.1037/0000057-000

Donner, C. M., Marcum, C. D., Jennings, W. G., Higgins, G. E., & Banfield, J. (2014). Low self-control and cybercrime: Exploring the utility of the general theory of crime beyond digital piracy. *Computers in Human Behavior*, *34*, 165–172. https://doi.org/10.1016/j.chb.2014.01.040

Draganski, B., Gaser, C., Kempermann, G., Kühn, H. G., Winkler, J., Büchel, C., & May, A. (2006). Temporal and spatial dynamics of brain structure changes during extensive learning. *The Journal of Neuroscience*, *26*(23), 6314–6317. https://doi.org/10.1523/JNEUROSCI.4628-05.2006

Dressel, J., & Farid, H. (2018). The accuracy, fairness, and limits of predicting recidivism. *Science Advances*, *4*(1), 1-5. https://doi.org/10.1126/sciadv.aao5580

Duke, A. A., Giancola, P. R., Morris, D. H., Holt, J. C. D., & Gunn, R. L. (2011). Alcohol dose and aggression: Another reason why drinking more is a bad idea. *Journal of Studies on Alcohol and Drugs*, *72*(1), 34–43. https://doi.org/10.15288/jsad.2011.72.34

Efron, B., & Tibshirani, R. J. (1993). *An introduction to the bootstrap*. London: Chapman & Hall. https://doi.org/10.1007/978-1-4899-4541-9

Eichelberger, R., & Barnes, J. C. (2015). Biosocial criminology. In W. G. Jennings (Ed.), *The encyclopedia of crime & punishment* (pp. 1–8). New York: John Wiley & Sons Inc. https://doi.org/10.1002/9781118519639.wbecp185

Eisinga, R., Grotenhuis, M. te, & Pelzer, B. (2013). The reliability of a two-item scale: Pearson, Cronbach, or Spearman-Brown? *International Journal of Public Health*, *58*(4), 637–642. https://doi.org/https://doi.org/10.1007/s00038-012-0416-3

Ellingson, J. M., Verges, A., Littlefield, A. K., Martin, N. G., & Slutske, W. S. (2013). Are bottom-up and top-down traits in dual-systems models of risky behavior genetically distinct? *Behavior Genetics*, *43*(6), 480–490. https://doi.org/10.1007/s10519-013-9615-9

Eres, R., Louis, W. R., & Molenberghs, P. (2018). Common and distinct neural networks involved in fMRI studies investigating morality: An ALE meta-analysis. *Social Neuroscience*, *13*(4), 384–398. https://doi.org/10.1080/17470919.2017.1357657

Etkin, A., Büchel, C., & Gross, J. J. (2015). The neural bases of emotion regulation. *Nature Reviews Neuroscience*, *16*, 693–700. https://doi.org/10.1038/nrn4044

Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences*, *15*(2), 85–93. https://doi.org/10.1016/j.tics.2010.11.004

Evans, J. S. B. T., & Frankish, K. (2009). *In two minds: Dual processes and beyond*. New York, NY: Oxford University Press. https://doi.org/10.1093/acprof:oso/9780199230167.001.0001

Evans, J. S. B. T. (2008). Dual-processing accounts of reasoning, judgment, and social cognition. *Annual Review of Psychology*, *59*, 255–278. https://doi.org/10.1146/annurev.psych.59.103006.093629

Evans, J. S. B.T. (2003). In two minds: Dual-process accounts of reasoning. *Trends in Cognitive Sciences*, *7*(10), 454–459. https://doi.org/10.1016/j.tics.2003.08.012

Evans, J. S. B.T., & Stanovich, K. E. (2013). Dual-process theories of higher cognition: Advancing the debate. *Perspectives on Psychological Science*, *8*(3), 223–241.

https://doi.org/10.1177/1745691612460685

Evrensel, A., Ünsalver, B. Ö., & Özşahin, A. (2016). The relationship between aggression and serum thyroid hormone level in individuals diagnosed with antisocial personality disorder. *Archives of Neuropsychiatry*, *53*(2), 120–125. https://doi.org/10.5152/npa.2015.9895

Eysenck, H. J. (1977). *Crime and personality*. London, England: Routledge & Kegan Paul Ltd. https://doi.org/10.1177/002581727904700104

Eysenck, H. J. (1997). Personality and the biosocial model of anti-social and criminal behaviour. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 21–38). New York, NY: Plenum. https://doi.org/10.1007/978-1-4757-4648-8_2

Fagan, S. E., Zhang, W., & Gao, Y. (2017). Social adversity and antisocial behavior: Mediating effects of autonomic nervous system activity. *Journal of Abnormal Child Psychology*, *45*(8), 1553–1564. https://doi.org/10.1007/s10802-017-0262-0

Fairchild, G., Passamonti, L., Hurford, G., Hagan, C. C., Von Dem Hagen, E. A. H., Van Goozen, S. H. M., … Calder, A. J. (2011). Brain structure abnormalities in early-onset and adolescent-onset conduct disorder. *American Journal of Psychiatry*, *168*(6), 624–633. https://doi.org/10.1176/appi.ajp.2010.10081184

Fairchild, G., van Goozen, S. H. M., Stollery, S. J., Brown, J., Gardiner, J., Herbert, J., & Goodyer, I. M. (2008). Cortisol diurnal rhythm and stress reactivity in male adolescents with early-onset or adolescence-onset conduct disorder. *Biological Psychiatry*, *64*(7), 599–606. https://doi.org/10.1016/j.biopsych.2008.05.022

Falcone, M., Bernardo, L., Ashare, R. L., Hamilton, R., Faseyitan, O., McKee, S. A., … Lerman, C. (2016). Transcranial direct current brain stimulation increases ability to resist smoking. *Brain Stimulation*, *9*(2), 191–196. https://doi.org/10.1016/j.brs.2015.10.004

Falk, Ö., Wallinius, M., Lundström, S., Frisell, T., Anckarsäter, H., & Kerekes, N. (2014). The 1 % of the population accountable for 63 % of all violent crime convictions. *Social Psychiatry and Psychiatric Epidemiology*, *49*(4), 559–571. https://doi.org/10.1007/s00127-013-0783-y

Fanti, K. A., Kyranides, M. N., Georgiou, G., Petridou, M., Colins, O. F., Tuvblad, C., & Andershed, H. (2017). Callous-unemotional, impulsive-irresponsible, and grandiose-manipulative traits: Distinct associations with heart rate, skin conductance, and startle responses to violent and erotic scenes. *Psychophysiology*, *54*(5), 663–672.

https://doi.org/10.1111/psyp.12837

Farrington, D. P. (1986). Age and crime. *Crime and Justice: An Annual Review of Research*, *7*, 189–250. https://doi.org/10.1086/449114

Fede, S. J., & Kiehl, K. A. (2020). Meta-analysis of the moral brain: Patterns of neural engagement assessed using multilevel kernel density analysis. *Brain Imaging and Behavior*, *14*(2), 534-547. https://doi.org/10.1007/s11682-019-00035-5

Felson, R. B., & Staff, J. (2011). The effects of alcohol intoxication on violent versus other offending. *Criminal Justice and Behavior*, *37*(12), 1343–1360. https://doi.org/10.1177/0093854810382003

Ferrando, P. J., & Chico, E. (2001). The construct of sensation seeking as measured by Zuckerman's SSS-V and Arnett's AISS: A structural equation model. *Personality and Individual Differences*, *31*(7), 1121–1133. https://doi.org/10.1016/S0191-8869(00)00208-7

Field, A. (2018). *Discovering statistics using IBM SPSS statistics* (5th ed.). London, England: SAGE Publications Ltd.

Fisher, P. M., Price, J. C., Meltzer, C. C., Moses-Kolko, E. L., Becker, C., Berga, S. L., & Hariri, A. R. (2011). Medial prefrontal cortex serotonin 1A and 2A receptor binding interacts to predict threat-related amygdala reactivity. *Biology of Mood & Anxiety Disorders*, *1*(2), 1–11. https://doi.org/10.1186/2045-5380-1-2

Fourie, M. M., Thomas, K. G. F., Amodio, D. M., Warton, C. M. R., & Meintjes, E. M. (2014). Neural correlates of experienced moral emotion: An fMRI investigation of emotion in response to prejudice feedback. *Social Neuroscience*, *9*(2), 203–218. https://doi.org/10.1080/17470919.2013.878750

Fry, H. (2018). *Hello world: How to be human in the age of the machine*. New York, NY: Doubleday.

Fumagalli, M., & Priori, A. (2012). Functional and clinical neuroanatomy of morality. *Brain*, 135(7), 2006–2021. https://doi.org/10.1093/brain/awr334

Gainotti, G. (2019a). Emotions and the right hemisphere: Can new data clarify old models? *The Neuroscientist*, *25*(3), 258–270. https://doi.org/10.1177/1073858418785342

Gainotti, G. (2019b). The role of the right hemisphere in emotional and behavioral disorders of patients with frontotemporal lobar degeneration: An updated review. *Frontiers in Aging Neuroscience*, *11*, 1–16. https://doi.org/10.3389/fnagi.2019.00055

Galán, C. A., Choe, D. E., Forbes, E. E., & Shaw, D. S. (2017). Interactions between empathy and resting heart rate in early adolescence predict violent behavior in late adolescence and early adulthood. *Journal of Child Psychology and Psychiatry*, *58*(12), 1370–1380. https://doi.org/10.1111/jcpp.12771

Gao, Y., Borlam, D., & Zhang, W. (2015). The association between heart rate reactivity and fluid intelligence in children. *Biological Psychology, 107*, 69–75. https://doi.org/10.1016/j.biopsycho.2015.03.006

Gao, Y., Huang, Y., & Li, X. (2017). Interaction between prenatal maternal stress and autonomic arousal in predicting conduct problems and psychopathic traits in children. *Journal of Psychopathology and Behavioral Assessment*, *39*(1), 1–14. https://doi.org/10.1007/s10862-016-9556-8

Gao, Y., Raine, A., & Schug, R. A. (2012). Somatic aphasia: Mismatch of body sensations with autonomic stress reactivity in psychopathy. *Biological Psychology, 90*(3), 228–233. https://doi.org/10.1016/j.biopsycho.2012.03.015

Gerton, B. K., Brown, T. T., Meyer-Lindenberg, A., Kohn, P., Holt, J. L., Olsen, R. K., & Berman, K. F. (2004). Shared and distinct neurophysiological components of the digits forward and backward tasks as revealed by functional neuroimaging. *Neuropsychologia*, *42*(13), 1781–1787. https://doi.org/10.1016/j.neuropsychologia.2004.04.023

Ghchime, R., Benjelloun, H., Kiai, H., Belaidi, H., Lahjouji, F., & Ouazzani, R. (2016). Cerebral hemispheric lateralization associated with hippocampal sclerosis may affect interictal cardiovascular autonomic functions in temporal lobe epilepsy. *Epilepsy Research and Treatment*, 1–7. https://doi.org/10.1155/2016/7417540

Gidding, S. S., Xie, X., Liu, K., Manolio, T., Flack, J. M., & Gardin, J. M. (1995). Cardiac function in smokers and nonsmokers: The CARDIA study. *Journal of the American College of Cardiology*, *26*(1), 211–216. https://doi.org/10.1016/0735-1097(95)00118-J

Gifuni, A. J., Kendal, A., & Jollant, F. (2017). Neural mapping of guilt: A quantitative meta-analysis of functional imaging studies. *Brain Imaging and Behavior*, *11*(4), 1164–1178. https://doi.org/10.1007/s11682-016-9606-6

Gilead, M., Katzir, M., Eyal, T., & Liberman, N. (2016). Neural correlates of processing “self-conscious” vs. “basic” emotions. *Neuropsychologia*, *81*, 207–218. https://doi.org/10.1016/j.neuropsychologia.2015.12.009

Gillespie, S. M., Brzozowski, A., & Mitchell, I. J. (2018). Self-regulation and aggressive antisocial behaviour: Insights from amygdala-prefrontal and heart-brain interactions. *Psychology, Crime and Law*, *24*(3), 243–257. https://doi.org/10.1080/1068316X.2017.1414816

Ginty, A. T., Gianaros, P. J., Derbyshire, S. W. G., Phillips, A. C., & Carroll, D. (2013).

Blunted cardiac stress reactivity relates to neural hypoactivation. *Psychophysiology*, *50*(3), 219–229. https://doi.org/10.1111/psyp.12017

Ginty, A. T., Phillips, A. C., Der, G., Deary, I. J., & Carroll, D. (2011). Heart rate reactivity is associated with future cognitive ability and cognitive change in a large community sample. *International Journal of Psychophysiology, 82*(2), 167–174. https://doi.org/10.1016/j.ijpsycho.2011.08.004

Gläscher, J., Adolphs, R., Damasio, H., Bechara, A., Rudrauf, D., Calamia, M., … Tranel, D. (2012). Lesion mapping of cognitive control and value-based decision making in the prefrontal cortex. *Proceedings of the National Academy of Sciences of the United States of America*, *109*(36), 14681–14686. https://doi.org/10.1073/pnas.1206608109

Glenn, A. L., & McCauley, K. E. (2019). How biosocial research can improve interventions for antisocial behavior. *Journal of Contemporary Criminal Justice*, *35*(1), 103 –119. https://doi.org/https://doi.org/10.1177/1043986218810608

Glenn, A. L., & Raine, A. (2008). The neurobiology of psychopathy. *Psychiatric Clinics of North America*, *31*(3), 463–475. https://doi.org/10.1016/j.psc.2008.03.004

Glenn, A. L., & Raine, A. (2011). Antisocial personality disorders. In J. Decety & J. T. Cacioppo (Eds.), *The Oxford handbook of social neuroscience* (pp. 885–894). Oxford, England: Oxford University Press. https://doi.org/10.1093/oxfordhb/9780195342161.013.00580.1016/j.psc.2008.03.004

Glenn, A. L., & Raine, A. (2014). Neurocriminology: Implications for the punishment, prediction and prevention of criminal behaviour. *Nature Reviews Neuroscience*, *15*, 54–63. https://doi.org/10.1038/nrn3640

Glenn, A. L., Yang, Y., & Raine, A. (2012). Neuroimaging in psychopathy and antisocial personality disorder: Functional significance and a neurodevelopmental hypothesis. In J. R. Simpson (Ed.), *Neuroimaging in forensic psychiatry: From the clinic to the courtroom* (pp. 81–98). Chichester, England: John Wiley & Sons, Ltd. https://doi.org/10.1002/9781119968900.ch5

Golkar, A., Lonsdorf, T. B., Olsson, A., Lindstrom, K. M., Berrebi, J., Fransson, P., … Öhman, A. (2012). Distinct contributions of the dorsolateral prefrontal and orbitofrontal cortex during emotion regulation. *PLoS ONE*, *7*(11), 1–9. https://doi.org/10.1371/journal.pone.0048107

Gordan, R., Gwathmey, J. K., & Xie, L.-H. (2015). Autonomic and endocrine control of cardiovascular function. *World Journal of Cardiology*, *7*(4), 204–214. https://doi.org/10.4330/wjc.v7.i4.204

Gossett, E. W., Wheelock, M. D., Goodman, A. M., Orem, T. R., Harnett, N. G., Wood, K. H., … Knight, D. C. (2018). Anticipatory stress associated with functional magnetic resonance imaging: Implications for psychosocial stress research. *International Journal of Psychophysiology*, *125*, 35–41. https://doi.org/10.1016/j.ijpsycho.2018.02.005

Gottfredson, M. R., & Hirschi, T. (1990). *A general theory of crime*. Stanford, CA: Stanford University Press.

Goya-Maldonado, R., Walther, S., Simon, J., Stippich, C., Weisbrod, M., & Kaiser, S. (2010). Motor impulsivity and the ventrolateral prefrontal cortex. *Psychiatry Research - Neuroimaging*, *183*(1), 89–91. https://doi.org/10.1016/j.pscychresns.2010.04.006

Graham, D. (2001). Decriminalization of marijuana: An analysis of the laws in the United States and the Netherlands and suggestions for reform. *Loyola of Los Angeles International and Comparative Law Review*, *23*(2), 297–328.

Grasmick, H. G., Tittle, C. R., Bursik, R. J., & Arneklev, B. J. (1993). Testing the core empirical implications of Gottfredson and Hirschi's general theory of crime. *Journal of Research in Crime and Delinquency*, *30*(1), 5–29. https://doi.org/10.1177/0022427893030001002

Greene, J. D. (2007). Why are VMPFC patients more utilitarian? A dual-process theory of moral judgment explains. *Trends in Cognitive Sciences*, *11*(8), 322–323. https://doi.org/10.1016/j.tics.2007.06.004

Grégoire, J., & Van der Linden, M. (1997). Effect of age on forward and backward digit spans. *Aging, Neuropsychology, and Cognition*, *4*(2), 140–149. https://doi.org/10.1080/13825589708256642

Gupta, R., Koscik, T. R., Bechara, A., & Tranel, D. (2011). The amygdala and decision-making. *Neuropsychologia*, *49*(4), 760–766. https://doi.org/10.1016/j.neuropsychologia.2010.09.029

Haar, D. H., & Wikström, P.-O. H. (2010). Crime propensity, criminogenic exposure and violent scenario responses: Testing situational action theory in regression and Rasch models. *European Journal of Applied Mathematics*, *21*(4-5), 307–323. https://doi.org/10.1017/S0956792510000161

Hagemann, D., Waldstein, S. R., & Thayer, J. F. (2003). Central and autonomic nervous system integration in emotion. *Brain and Cognition*, *52*, 79–87. https://doi.org/10.1016/S0278-2626(03)00011-3

Hammerton, G., Heron, J., Mahedy, L., Maughan, B., Hickman, M., & Murray, J. (2018). Low resting heart rate, sensation seeking and the course of antisocial behaviour across adolescence and young adulthood. *Psychological Medicine*, *48*(13), 2194–2201. https://doi.org/10.1017/S0033291717003683

Hannah-Moffat, K. (2019). Algorithmic risk governance: Big data analytics, race and information activism in criminal justice debates. *Theoretical Criminology*, *23*(4), 453–470. https://doi.org/10.1177/1362480618763582

Harden, K. P., & Tucker-Drob, E. M. (2011). Individual differences in the development of sensation seeking and impulsivity during adolescence: Further evidence for a dual systems model. *Developmental Psychology*, *47*(3), 739–746. https://doi.org/10.1037/a0023279

Hardie, B. (2019). Why monitoring doesn't always matter: The interaction of personal propensity with physical and psychological parental presence in a situational explanation of adolescent offending. *Deviant Behavior*, 1–24. https://doi.org/10.1080/01639625.2019.1673924

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

Hare, R. D., & Neumann, C. S. (2005). Structural models of psychopathy. *Current Psychiatry Reports*, *7*, 57–64. https://doi.org/10.1007/s11920-005-0026-3

Hare, R. D., & Neumann, C. S. (2010). The role of antisociality in the psychopathy construct: Comment on Skeem and Cooke (2010). *Psychological Assessment*, *22*(2), 446–454. https://doi.org/10.1037/a0013635

Hare, R. D. (1996). Psychopathy and antisocial personality disorder: A case of diagnostic confusion. *Psychiatric Times*, *13*(2), 39–40. https://doi.org/10168/54831

Hare, T. A., Tottenham, N., Davidson, M. C., Glover, G. H., & Casey, B. J. (2005). Contributions of amygdala and striatal activity in emotion regulation. *Biological Psychiatry*, *57*(6), 624–632. https://doi.org/10.1016/j.biopsych.2004.12.038

Hart, H., Lim, L., Mehta, M. A., Simmons, A., Mirza, K. A. H., & Rubia, K. (2018). Altered fear processing in adolescents with a history of severe childhood maltreatment: An fMRI study. *Psychological Medicine*, *48*(7), 1092–1101. https://doi.org/10.1017/S0033291716003585

Haspel, T. (1995). Beta-blockers and the treatment of aggression. *Harvard Review of Psychiatry*, *2*(5), 274–281. https://doi.org/10.3109/10673229509017146

Heberlein, A. S., Padon, A. A, Gillihan, S. J., Farah, M. J., & Fellows, L. K. (2008). Ventromedial frontal lobe plays a critical role in facial emotion recognition. *Journal of Cognitive Neuroscience*, *20*(4), 721–733. https://doi.org/10.1162/jocn.2008.20049

Hermand, E., Cassirame, J., Ennequin, G., & Hue, O. (2019). Validation of a photoplethysmographic heart rate monitor: Polar OH1. *International Journal of Sports Medicine*, *40*(7), 462–467. https://doi.org/10.1055/a-0875-4033

Hettiarachchi, I. T., Hanoun, S., Nahavandi, D., & Nahavandi, S. (2019). Validation of Polar OH1 optical heart rate sensor for moderate and high intensity physical activities. *PLoS ONE*, *14*(5), 1–13. https://doi.org/https://doi.org/10.1371/journal.pone.0217288

Himelstein, S. (2011). Meditation research: The state of the art in correctional settings. *International Journal of Offender Therapy and Comparative Criminology*, *55*(4), 646–661. https://doi.org/10.1177/0306624X10364485

Hirtenlehner, H., & Hardie, B. (2016). On the conditional relevance of controls: An application of situational action theory to shoplifting. *Deviant Behavior*, *37*(3), 315–331. https://doi.org/10.1080/01639625.2015.1026764

Hirtenlehner, H., & Kunz, F. (2016). The interaction between self-control and morality in crime causation among older adults. *European Journal of Criminology*, *13*(3), 393–409. https://doi.org/10.1177/1477370815623567

Hirtenlehner, H., & Kunz, F. (2017). Can self-control theory explain offending in late adulthood? Evidence from Germany. *Journal of Criminal Justice*, *48*, 37–47. https://doi.org/10.1016/j.jcrimjus.2016.12.001

Hirtenlehner, H., Pauwels, L. J. R., & Mesko, G. (2014). Is the effect of perceived deterrence on juvenile offending contingent on the level of self-control? Results from three countries. *British Journal of Criminology*, *54*(1), 128–150. https://doi.org/10.1093/bjc/azt053

Hirtenlehner, H., Pauwels, L., & Mesko, G. (2015). Is the criminogenic effect of exposure to peer delinquency dependent on the ability to exercise self-control? Results from three countries. *Journal of Criminal Justice*, *43*(6), 532–543. https://doi.org/10.1016/j.jcrimjus.2015.05.006

Hirtenlehner, H., & Treiber, K. (2017). Can situational action theory explain the gender gap in adolescent shoplifting? Results from Austria. *International Criminal Justice Review*, *27*(3), 165-187. https://doi.org/10.1177/1057567717690199

Hiser, J., & Koenigs, M. (2018). The multifaceted role of ventromedial prefrontal cortex in emotion, decision-making, social cognition, and psychopathology. *Biological Psychiatry*, *83*(8), 638–647. https://doi.org/10.1016/j.biopsych.2017.10.030

Hoaken, P. N. S., Allaby, D. B., & Earle, J. (2007). Executive cognitive functioning and the recognition of facial expressions of emotion in incarcerated violent offenders, non-violent offenders, and controls. *Aggressive Behavior*, *33*(5), 412–421. https://doi.org/10.1002/ab.20194

Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences, 16*(3), 174–180. https://doi.org/10.1016/j.tics.2012.01.006

Holtfreter, K., Reisig, M. D., & Pratt, T. C. (2008). Low self-control, routine activities, and fraud victimization. *Criminology, 46*(1), 189–220. https://doi.org/10.1111/j.1745-9125.2008.00101.x

Home Office. (2018). *Understanding the costs of cyber crime: A report of key findings from the Costs of Cyber Crime Working Group*. Retrieved from https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/674046/understanding-costs-of-cyber-crime-horr96.pdf

Hooper, C. J., Luciana, M., Conklin, H. M., & Yarger, R. S. (2004). Adolescents' performance on the Iowa gambling task: Implications for the development of decision making and ventromedial prefrontal cortex. *Developmental Psychology, 40*(6), 1148–1158. https://doi.org/10.1037/0012-1649.40.6.1148

Hoppenbrouwers, S. S., Bulten, B. H., & Brazil, I. A. (2016). Parsing fear: A reassessment of the evidence for fear deficits in psychopathy. *Psychological Bulletin*, *142*(6), 573–600. https://doi.org/10.1037/bul0000040

Horn, N. R., Dolan, M., Elliott, R., Deakin, J. F. W., & Woodruff, P. W. R. (2003). Response inhibition and impulsivity: An fMRI study. *Neuropsychologia*, *41*(14), 1959–1966. https://doi.org/10.1016/S0028-3932(03)00077-0

Hornak, J., Bramham, J., Rolls, E. T., Morris, R. G., O'Doherty, J., Bullock, P. R., & Polkey, C. E. (2003). Changes in emotion after circumscribed surgical lesions of the orbitofrontal and cingulate cortices. *Brain*, *126*(7), 1691–1712. https://doi.org/10.1093/brain/awg168

Hornak, J., Rolls, E. T., & Wade, D. (1996). Face and voice expression identification in patients with emotional and behavioural changes following ventral frontal lobe damage. *Neuropsychologia*, *34*(4), 247–261. https://doi.org/10.1016/0028-3932(95)00106-9

Hornberger, M., Geng, J., Hodges, J. R., Andersson, J., Jenkinson, M., Smith, S., … Gorno-Tempini, M. (2011). Convergent grey and white matter evidence of orbitofrontal cortex changes related to disinhibition in behavioural variant frontotemporal dementia. *Brain, 134*(9), 2502–2512. https://doi.org/10.1093/brain/awr173

Hoshi, Y., Oda, I., Wada, Y., Ito, Y., Yamashita, Y., Oda, M., … Tamura, M. (2000).

Visuospatial imagery is a fruitful strategy for the digit span backward task: A study with near-infrared optical tomography. *Cognitive Brain Research*, *9*(3), 339–342. https://doi.org/10.1016/S0926-6410(00)00006-9

Howell, D. C. (2012). *Statistical methods for psychology* (8th ed.). Belmont, CA: Wadsworth.

Hu, K. (2018). Neural activity to threat in ventromedial prefrontal cortex correlates with individual differences in anxiety and reward processing. *Neuropsychologia*, *117*, 566–573. https://doi.org/10.1016/j.neuropsychologia.2018.07.004

Hugdahl, K. (1998). Cortical control of human classical conditioning: Autonomic and positron emission tomography data. *Psychophysiology*, *35*(2), 170–178.

Inman, C. S., Bijanki, K. R., Bass, D. I., Gross, R. E., Hamann, S., & Willie, J. T. (2018). Human amygdala stimulation effects on emotion physiology and emotional experience. *Neuropsychologia*. https://doi.org/10.1016/j.neuropsychologia.2018.03.019

Ioannidis, J. P. A., & Trikalinos, T. A. (2005). Early extreme contradictory estimates may appear in published research: The Proteus phenomenon in molecular genetics research and randomized trials. *Journal of Clinical Epidemiology*, *58*(6), 543-549. https://doi.org/10.1016/j.jclinepi.2004.10.019

Isenberg, N., Silbersweig, D., Engelien, A., Emmerich, S., Malavade, K., Beattie, B., … Stern, E. (1999). Linguistic threat activates the human amygdala. *Proceedings of the National Academy of Sciences of the United States of America*, *96*(18), 10456–10459. https://doi.org/10.1073/pnas.96.18.10456

Ishikawa, S. S., & Raine, A. (2003). Prefrontal deficits and antisocial behavior: A causal model. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 277–304). New York, NY: Guilford.

Ishikawa, S. S., Raine, A., Lencz, T., Bihrle, S., & Lacasse, L. (2001). Autonomic stress reactivity and executive functions in successful and unsuccessful criminal psychopaths from the community. *Journal of Abnormal Psychology*, *110*(3), 423–432. https://doi.org/10.1037/0021-843X.110.3.423

Jackson, D. B., & Beaver, K. M. (2013). The influence of neuropsychological deficits in early childhood on low self-control and misconduct through early adolescence. *Journal of Criminal Justice*, *41*(4), 243–251. https://doi.org/10.1016/j.jcrimjus.2013.05.002

Jedd, K., Hunt, R. H., Cicchetti, D., Hunt, E., Cowell, R. A., Rogosch, F. A., … Thomas, K. M. (2015). Long-term consequences of childhood maltreatment: Altered amygdala functional connectivity. *Development and Psychopathology*, *27*(4 Pt 2), 1577–1589. https://doi.org/10.1017/S0954579415000954

Jennings, J. R., Pardini, D. A., & Matthews, K. A. (2017). Heart rate, health, and hurtful behavior. *Psychophysiology*, *54*, 399–408. https://doi.org/10.1111/psyp.12802

Jennings, Wesley G., Piquero, A. R., & Farrington, D. P. (2013). Does resting heart rate at age 18 distinguish general and violent offending up to age 50? Findings from the Cambridge Study in Delinquent Development. *Journal of Criminal Justice*, *41*(4), 213–219. https://doi.org/10.1016/j.jcrimjus.2013.05.003

Jones, C., Clayborne, M., Grant, J. D., & Rutherford, G. (2003). Attacking crime at its source: Consciousness-based education in the prevention of violence and antisocial behavior. *Journal of Offender Rehabilitation*, *36*(1–4), 229–255. https://doi.org/10.1300/J076v36n01_11

Kahneman, D. (2011). *Thinking, fast and slow*. New York, NY: Farrar, Straus and Giroux.

Kavish, N., Boisvert, D., Wells, J., Lewis, R., Cooke, E., Woeckener, M., & Armstrong, T. (2019). On the associations between indicators of resting arousal levels, physiological reactivity, sensation seeking, and psychopathic traits. *Personality and Individual Differences*, *141*, 218–225. https://doi.org/10.1016/j.paid.2019.01.013

Kavish, N., Fu, Q. J., Vaughn, M. G., Qian, Z., & Boutwell, B. B. (2019). Resting heart rate and psychopathy revisited: Findings from the Add Health Survey. *International Journal of Offender Therapy and Comparative Criminology*, *63*(4), 543–557. https://doi.org/10.1177/0306624X18806748

Kavish, N., Vaughn, M. G., Cho, E., Barth, A., Boutwell, B., Vaughn, S., … Martinez, L. (2017). Physiological arousal and juvenile psychopathy: Is low resting heart rate associated with affective dimensions? *Psychiatric Quarterly*, *88*(1), 103–114. https://doi.org/10.1007/s11126-016-9437-z

Kempermann, G., Gage, F. H., Aigner, L., Song, H., Curtis, M. A., Thuret, S., … Frisén, J. (2018). Human adult neurogenesis: Evidence and remaining questions. *Cell Stem Cell*, *23*(1), 25–30. https://doi.org/10.1016/j.stem.2018.04.004

Kerr, K. L., Avery, J. A., Barcalow, J. C., Moseman, S. E., Bodurka, J., Bellgowan, P. S. F., & Simmons, W. K. (2015). Trait impulsivity is related to ventral ACC and amygdala activity during primary reward anticipation. *Social Cognitive and Affective Neuroscience*, *10*(1), 36–42. https://doi.org/10.1093/scan/nsu023

Kiehl, K. A., Anderson, N. E., Aharoni, E., Maurer, J. M., Harenski, K. A., Rao, V., … Steele, V. R. (2018). Age of gray matters: Neuroprediction of recidivism. *NeuroImage: Clinical*, *19*, 813–823. https://doi.org/10.1016/j.nicl.2018.05.036

Killgore, W. D. S., & Yurgelun-Todd, D. A. (2007). The right-hemisphere and valence hypotheses: Could the both be right (and sometimes left)? *Social Cognitive and Affective Neuroscience*, *2*(3), 240–250. https://doi.org/10.1093/scan/nsm020

Kline, R. B. (2016). *Principles and practice of structural equation modeling* (4th ed.). New York, NY: The Guilford Press.

Knoch, D., & Fehr, E. (2007). Resisting the power of temptations: The right prefrontal cortex and self-control. *Annals of the New York Academy of Sciences*, *1104*, 123–134. https://doi.org/10.1196/annals.1390.004

Kobayashi, H. (2013). Effect of measurement duration on accuracy of pulse-counting. *Ergonomics*, *56*(12), 1940–1944. https://doi.org/10.1080/00140139.2013.840743

Koegl, C. J., Farrington, D. P., & Raine, A. (2018). The relationship between low resting heart rate, systolic blood pressure and antisocial behavior in incarcerated males. *Journal of Criminal Justice*, *55*, 88–95. https://doi.org/10.1016/j.jcrimjus.2018.02.004

Koenigs, M. (2012). The role of prefrontal cortex in psychopathy. *Reviews in the Neurosciences*, *23*(3), 253–262. https://doi.org/10.1515/revneuro-2012-0036

Koenigs, M., Young, L., Adolphs, R., Tranel, D., Cushman, F., Hauser, M., & Damasio, A. R. (2007). Damage to the prefrontal cortex increases utilitarian moral judgements. *Nature*, *446*, 908–911. https://doi.org/10.1038/nature05631

Kokkalera, S. S., Marshall, I. H., & Marshall, C. E. (2020). How exceptional is India? A test of situational action theory. *Asian Journal of Criminology*. https://doi.org/10.1007/s11417-020-09312-5

Kong, G., Smith, A. E., McMahon, T. J., Cavallo, D. A., Schepis, T. S., Desai, R. A., … Krishnan-Sarin, S. (2013). Pubertal status, sensation-seeking, impulsivity, and substance use in high school-aged boys and girls. *Journal of Addiction Medicine*, *7*(2), 116–121. https://doi.org/10.1097/ADM.0b013e31828230ca

Krajbich, I., Adolphs, R., Tranel, D., Denburg, N. L., & Camerer, C. F. (2009). Economic games quantify diminished sense of guilt in patients with damage to the prefrontal cortex. *Journal of Neuroscience*, *29*(7), 2188–2192. https://doi.org/10.1523/JNEUROSCI.5086-08.2009

Krokoszinski, L., Westenberger, A., & Hosser, D. (2018). Emotional responsiveness in convicted fraudsters: A study on baseline activation of the anterior insula and its influence on moral decision-making. *Journal of Forensic Psychiatry and Psychology*, *29*(4), 527–543. https://doi.org/10.1080/14789949.2018.1425470

Kroneberg, C., Heintze, I., & Mehlkop, G. (2010). The interplay of moral norms and instrumental incentives in crime causation. *Criminology*, *48*(1), 259–294.

https://doi.org/10.1111/j.1745-9125.2010.00187.x

Kupfer, D. (2013). The DSM-5 - an interview with David Kupfer. *BMC Medicine, 11*(203). https://doi.org/doi:10.1186/1741-7015-11-203

Kyranides, M. N., Fanti, K. A., Sikki, M., & Patrick, C. J. (2017). Triarchic dimensions of psychopathy in young adulthood: Associations with clinical and physiological measures after accounting for adolescent psychopathic traits. *Personality Disorders: Theory, Research, and Treatment*, *8*(2), 140–149. https://doi.org/10.1037/per0000193

Larregue, J., & Rollins, O. (2019). Biosocial criminology and the mismeasure of race. *Ethnic and Racial Studies*, *42*(12), 1990-2007. https://doi.org/https://www.tandfonline.com/action/showCitFormats?doi=10.1080/01419870.2018.1527938

Latvala, A., Kuja-Halkola, R., Almqvist, C., Larsson, H., & Lichtenstein, P. (2015). A longitudinal study of resting heart rate and violent criminality in more than 700000 men. *JAMA Psychiatry*, *72*(10), 971–978. https://doi.org/10.1001/jamapsychiatry.2015.1165

Lawrence, K., Campbell, R., & Skuse, D. (2015). Age, gender, and puberty influence the development of facial emotion recognition. *Frontiers in Psychology*, *6*(761), 1–14. https://doi.org/10.3389/fpsyg.2015.00761

LeDoux, J. E. (2013). The slippery slope of fear. *Trends in Cognitive Sciences*, *17*(4), 155–156. https://doi.org/10.1016/j.tics.2013.02.004

LeDoux, J. E. (2014). Coming to terms with fear. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(8), 2871–2878. https://doi.org/10.1073/pnas.1400335111

LeDoux, J. E., & Hofmann, S. G. (2018). The subjective experience of emotion: A fearful view. *Current Opinion in Behavioral Sciences*, *19*, 67–72. https://doi.org/10.1016/j.cobeha.2017.09.011

LeDoux, J. E., & Pine, D. S. (2016). Using neuroscience to help understand fear and anxiety: A two-system framework. *American Journal of Psychiatry*, *173*(11), 1083–1093. https://doi.org/10.1176/appi.ajp.2016.16030353

Lee, K., & Ashton, M. C. (2006). Further assessment of the HEXACO personality inventory: Two new facet scales and an observer report form. *Psychological Assessment*, *18*(2), 182–191. https://doi.org/10.1037/1040-3590.18.2.182

Lee, K., & Ashton, M. C. (2014). The Dark Triad, the Big Five, and the HEXACO model. *Personality and Individual Differences*, *67*, 2–5. https://doi.org/10.1016/j.paid.2014.01.048

Leistico, A. M. R., Salekin, R. T., DeCoster, J., & Rogers, R. (2008). A large-scale meta-analysis relating the hare measures of psychopathy to antisocial conduct. *Law and Human Behavior*, *32*(1), 28–45. https://doi.org/10.1007/s10979-007-9096-6

Leshem, R., & Weisburd, D. (2019). Epigenetics and hot spots of crime: Rethinking the relationship between genetics and criminal behavior. *Journal of Contemporary Criminal Justice*, *35*(2), 186–204. https://doi.org/10.1177/1043986219828924

Leslie, A. M. (1987). Pretense and representation: The origins of "Theory of Mind." *Psychological Review*, *94*(4), 412–426. https://doi.org/10.1037/0033-295X.94.4.412

Levitin, D. J. (2018). *A field guide to lies and statistics: A neuroscientist on how to make sense of a complex world*. London, England: Penguin.

Levy, R., & Goldman-Rakic, P. S. (2000). Segregation of working memory functions within the dorsolateral prefrontal cortex. *Experimental Brain Research*, *133*(1), 23–32. https://doi.org/10.1007/s002210000397

Lilienfeld, S. O. (1994). Conceptual problems in the assessment of psychopathy. *Clinical Psychology Review*, *14*(1), 17–38. https://doi.org/10.1016/0272-7358(94)90046-9

Lilienfeld, S. O., & Andrews, B. P. (1996). Development and preliminary validation of a self-report measure of psychopathic personality traits in noncriminal populations. *Journal of Personality Assessment*, *66*(3), 488–524. https://doi.org/10.1207/s15327752jpa6603_3

Liljegren, M., Naasan, G., Temlett, J., Perry, D. C., Rankin, K. P., Merrilees, J., … Miller, B. L. (2015). Criminal behavior in frontotemporal dementia and Alzheimer disease. *JAMA Neurology*, *72*(3), 295–300. https://doi.org/10.1001/jamaneurol.2014.3781

Linneberg, A., Jacobsen, R. K., Skaaby, T., Taylor, A. E., Fluharty, M. E., Jeppesen, J. L., … Husemoen, L. L. N. (2015). Effect of smoking on blood pressure and resting heart rate: A Mendelian randomization meta-analysis in the CARTA consortium. *Circulation: Cardiovascular Genetics*, *8*(6), 832–841. https://doi.org/10.1161/CIRCGENETICS.115.001225

Loeber, R. (2012). Does the study of the age-crime curve have a future? In R. Loeber & B. C. Welsh (Eds.), *The future of criminology* (pp. 11-19). New York, NY: Oxford University Press. https://doi.org/10.1093/acprof:oso/9780199917938.003.0002

Loeber, R., & Farrington, D. P. (2014). Age-crime curve. In G. Bruinsma & D. Weisburd (Eds.), *Encyclopedia of criminology and criminal justice* (pp. 12–18). New York: Springer. https://doi.org/10.1007/978-1-4614-5690-2_474

Loeber, R., Farrington, D. P., Hipwell, A. E., Stepp, S. D., Pardini, D., & Ahonen, L. (2015). Constancy and change in the prevalence and frequency of offending when based on longitudinal self-reports or official records: Comparisons by gender, race, and crime type. *Journal of Developmental and Life-Course Criminology*, *1*(2), 150–168. https://doi.org/10.1007/s40865-015-0010-5

Loeber, R., Pardini, D. A., Stouthamer-Loeber, M., & Raine, A. (2007). Do cognitive, physiological, and psychosocial risk and promotive factors predict desistance from delinquency in males? *Development and Psychopathology*, *19*(3), 867–887. https://doi.org/10.1017/s0954579407000429

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

Lösel, F., & Bender, D. (1997). Heart rate and psychosocial correlates of antisocial behavior in high-risk adolescents. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 321–324). New York, NY: Springer.

Lough, S., & Hodges, J. R. (2002). Measuring and modifying abnormal social cognition in frontal variant frontotemporal dementia. *Journal of Psychosomatic Research*, *53*(2), 639–646. https://doi.org/10.1016/S0022-3999(02)00433-6

Lovallo, W. R. (2013). Early life adversity reduces stress reactivity and enhances impulsive behavior: Implications for health behaviors. *International Journal of Psychophysiology*, *90*(1), 8–16. https://doi.org/10.1016/j.ijpsycho.2012.10.006

Lundqvist, D., Flykt, A., & Ohman, A. (1998). The Karolinska directed emotional faces (KDEF). *CD ROM from Department of Clinical Neuroscience, Psychology Section, Karolinska Institutet*. https://doi.org/10.1017/S0048577299971664

Magid, V., MacLean, M. G., & Colder, C. R. (2007). Differentiating between sensation seeking and impulsivity through their mediated relations with alcohol use and problems. *Addictive Behaviors*, *32*(10), 2046–2061. https://doi.org/10.1016/j.addbeh.2007.01.015

Maguire, E. A., Woollett, K., & Spiers, H. J. (2006). London taxi drivers and bus drivers: A structural MRI and neuropsychological analysis. *Hippocampus*, *16*, 1091–1101. https://doi.org/10.1002/hipo.20233

Maimon, D., Alper, M., Sobesto, B., & Cukier, M. (2014). Restrictive deterrent effects of a warning banner in an attacked computer system. *Criminology*, *52*(1), 33–59. https://doi.org/10.1111/1745-9125.12028

Maimon, D., Antonaccio, O., & French, M. T. (2012). Severe sanctions, easy choice? Investigating the role of school sanctions in preventing adolescent violent offending. *Criminology*, *50*(2), 495–524. https://doi.org/10.1111/j.1745-9125.2011.00268.x

Maliphant, R., Watkins, C., & Davies, J. G. V. (2003). Disruptive behaviour in non-referred mainstream school children, aged seven to nine: A psychophysiological contribution. *Educational Psychology*, *23*(4), 437–455. https://doi.org/10.1080/01443410303209

Marazziti, D., Baroni, S., Landi, P., Ceresoli, D., & Dell'Osso, L. (2013). The neurobiology of moral sense: Facts or hypotheses? *Annals of General Psychiatry*, *12*(6), 1–12. https://doi.org/10.1186/1744-859X-12-6

Margulies, D. S., Kelly, A. M. C., Uddin, L. Q., Biswal, B. B., Castellanos, F. X., & Milham, M. P. (2007). Mapping the functional connectivity of anterior cingulate cortex. *NeuroImage*, *37*(2), 579–588. https://doi.org/10.1016/j.neuroimage.2007.05.019

Marsh, A. A., & Blair, R. J. R. (2008). Deficits in facial affect recognition among antisocial populations: A meta-analysis. *Neuroscience and Biobehavioral Reviews*, *32*(3), 454–465. https://doi.org/10.1016/j.neubiorev.2007.08.003

Martin, S. E. (2001). The links between alcohol, crime and the criminal justice system: Explanations, evidence and interventions. *The American Journal on Addictions*, *10*(2), 136–158. https://doi.org/10.4324/9780203503508

Masten, C. L., Morelli, S. A., & Eisenberger, N. I. (2011). An fMRI investigation of empathy for "social pain" and subsequent prosocial behavior. *NeuroImage*, *55*(1), 381–388. https://doi.org/10.1016/j.neuroimage.2010.11.060

Mathias, C. W., & Stanford, M. S. (2003). Impulsiveness and arousal: Heart rate under conditions of rest and challenge in healthy males. *Personality and Individual Differences*, *35*, 355–371. https://doi.org/10.1016/S0191-8869(02)00195-2

McCrory, E. J., De Brito, S. A., Kelly, P. A., Bird, G., Sebastian, C. L., Mechelli, A., … Viding, E. (2013). Amygdala activation in maltreated children during pre-attentive emotional processing. *The British Journal of Psychiatry*, *202*(4), 269–276. https://doi.org/10.1192/bjp.bp.112.116624

McDonald, S. (2013). Impairments in social cognition following severe traumatic brain injury. *Journal of the International Neuropsychological Society*, *19*(3), 231–246. https://doi.org/10.1017/S1355617712001506

Mclatchie, N., Giner-Sorolla, R., & Derbyshire, S. W. G. (2016). 'Imagined guilt' vs 'recollected guilt': Implications for fMRI. *Social Cognitive and Affective Neuroscience*, *11*(5), 703–711. https://doi.org/10.1093/scan/nsw001

McMillan, T. M., & Rachman, S. (1987). Fearlessness and courage: A laboratory study of paratrooper veterans of the Falklands War. *British Journal of Psychology*, *78*, 375–383. https://doi.org/10.1111/j.2044-8295.1987.tb02255.x

McMillan, T. M., & Rachman, S. J. (1988). Fearlessness and courage in paratroopers undergoing training. *Personality and Individual Differences*, *9*(2), 373–378. https://doi.org/10.1016/0191-8869(88)90100-6

Meijers, J., Harte, J. M., Jonker, F. A., & Meynen, G. (2015). Prison brain? Executive dysfunction in prisoners. *Frontiers in Psychology*, *6*(43), 1–6. https://doi.org/10.3389/fpsyg.2015.00043

Mendez, M. F. (2006). What frontotemporal dementia reveals about the neurobiological basis of morality. *Medical Hypotheses*, *67*(2), 411–418. https://doi.org/10.1016/j.mehy.2006.01.048

Mendez, M. F. (2009). The neurobiology of moral behavior: Review and neuropsychiatric implications. *CNS Spectrums*, *14*(11), 608–620. https://doi.org/10.1017/S1092852900023853

Mendez, M. F., Anderson, E., & Shapira, J. S. (2005). An investigation of moral judgement in frontotemporal dementia. *Cognitive and Behavioral Neurology*, *18*(4), 193–197. https://doi.org/10.1097/01.wnn.0000191292.17964.bb

Mendez, M. F., Chen, A. K., Shapira, J. S., & Miller, B. L. (2005). Acquired sociopathy and frontotemporal dementia. *Dementia and Geriatric Cognitive Disorders*, *20*(2-3), 99–104. https://doi.org/10.1159/000086474

Mersky, J. P., Topitzes, J., & Reynolds, A. J. (2012). Unsafe at any age: Linking childhood and adolescent maltreatment to delinquency and crime. *Journal of Research in Crime and Delinquency*, *49*(2), 295–318. https://doi.org/10.1177/0022427811415284

Meyers, C. A., Berman, S. A., Scheibel, R. S., & Hayman, A. (1992). Case report: Acquired antisocial personality disorder associated with unilateral left orbital frontal lobe damage. *Journal of Psychiatry & Neuroscience*, *17*(3), 121–125.

Michalowski, R. J., & Carlson, S. M. (1999). Unemployment, imprisonment, and social structures of accumulation: Historical contingency in the Rusche-Kirkchheimer hypothesis. *Criminology*, *37*(2), 217–250. https://doi.org/10.1111/j.1745-9125.1999.tb00485.x

Michl, P., Meindl, T., Meister, F., Born, C., Engel, R. R., Reiser, M., & Hennig-Fast, K. (2014). Neurobiological underpinnings of shame and guilt: A pilot fMRI study. *Social Cognitive and Affective Neuroscience*, *9*(2), 150–157. https://doi.org/10.1093/scan/nss114

Miller, B. L., Darby, A., Benson, D. F., Cummings, J. L., & Miller, M. H. (1997). Aggressive, socially disruptive and antisocial behaviour associated with fronto-temporal dementia. *British Journal of Psychiatry*, *170*(2), 150–155. https://doi.org/10.1192/bjp.170.2.150

Ming, G. L., & Song, H. (2011). Adult neurogenesis in the mammalian brain: Significant answers and significant questions. *Neuron*, *70*(4), 687–702. https://doi.org/10.1016/j.neuron.2011.05.001.Adult

Ming, X., Julu, P. O. O., Brimacombe, M., Connor, S., & Daniels, M. L. (2005). Reduced cardiac parasympathetic activity in children with autism. *Brain and Development*, *27*(7), 509–516. https://doi.org/10.1016/j.braindev.2005.01.003

Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. A. (2001). *Sex differences in antisocial behaviour: Conduct disorder, delinquency, and violence in the Dunedin Longitudinal Study*. Cambridge, England: Cambridge University Press.

Moffitt, T. E., Arseneault, L., Jaffee, S. R., Kim-Cohen, J., Koenen, K. C., Odgers, C. L., … Viding, E. (2008). Research Review: DSM-V conduct disorder: Research needs for an evidence base. *Journal of Child Psychology and Psychiatry*, *49*(1), 3–33. https://doi.org/doi:10.1111/j.1469-7610.2007.01823.x

Moffitt, T. E., Krueger, R. F., Caspi, A., & Fagan, J. (2000). Partner abuse and general crime: How are they the same? How are they different? *Criminology*, *38*(1), 199–232. https://doi.org/10.1111/j.1745-9125.2000.tb00888.x

Molenberghs, P., Ogilvie, C., Louis, W. R., Decety, J., Bagnall, J., & Bain, P. G. (2015). The neural correlates of justified and unjustified killing: An fMRI study. *Social Cognitive and Affective Neuroscience*, *10*(10), 1397–1404. https://doi.org/10.1093/scan/nsv027

Molero-Chamizo, A., Martín Riquel, R., Moriana, J. A., Nitsche, M. A., & Rivera-Urbina, G. N. (2019). Bilateral prefrontal cortex anodal tDCS effects on self-reported aggressiveness in imprisoned violent offenders. *Neuroscience*, *397*, 31–40. https://doi.org/10.1016/j.neuroscience.2018.11.018

Monk, C., Kovelenko, P., Ellman, L. M., Sloan, R. P., Bagiella, E., Gorman, J. M., & Pine, D. S. (2001). Enhanced stress reactivity in paediatric anxiety disorders: Implications for future cardiovascular health. *International Journal of Neuropsychopharmacology*, *4*(2), 199–206. https://doi.org/10.1017/S146114570100236X

Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review*, *20*(1), 113-36. https://doi.org/10.1016/S0272-7358(98)00096-8

Motzkin, J. C., Philippi, C. L., Wolf, R. C., Baskaya, M. K., & Koenigs, M. (2015). Ventromedial prefrontal cortex is critical for the regulation of amygdala activity in humans. *Biological Psychiatry*, *77*(3), 276–284. https://doi.org/10.1016/j.biopsych.2014.02.014

Münte, T. F., Altenmüller, E., & Jäncke, L. (2002). The musician's brain as a model of neuroplasticity. *Nature Reviews Neuroscience*, *3*, 473–478. https://doi.org/10.1038/nrn843

Murray, J., Hallal, P. C., Mielke, G. I., Raine, A., Wehrmeister, F. C., Anselmi, L., & Barros, F. C. (2016). Low resting heart rate is associated with violence in late adolescence: A prospective birth cohort study in Brazil. *International Journal of Epidemiology*, *45*(2), 491–500. https://doi.org/10.1093/ije/dyv340

Nagai, M., Hoshide, S., & Kario, K. (2010). The insular cortex and cardiovascular system: A new insight into the brain-heart axis. *Journal of the American Society of Hypertension*, *4*(4), 174–182. https://doi.org/10.1016/j.jash.2010.05.001

NICHD Early Child Care Research. (2004). Trajectories of physical aggression from toddlerhood to middle childhood: Predictors, correlates, and outcomes. *Monographs of the Society for Research in Child Development*, *69*(4), 1–129. https://doi.org/10.1111/j.0037-976X.2004.00312.x

Nikolaou, C. K., Hankey, C. R., & Lean, M. E. J. (2017). Accuracy of on-line self-reported weights and heights by young adults. *European Journal of Public Health*, *27*(5), 898–903. https://doi.org/10.1093/eurpub/ckx077

Noordermeer, S. D. S., Luman, M., & Oosterlaan, J. (2016). A systematic review and meta-analysis of neuroimaging in oppositional defiant disorder (ODD) and conduct disorder (CD) taking attention-deficit hyperactivity disorder (ADHD) into account. *Neuropsychology Review*, 26(1), 44–72. https://doi.org/10.1007/s11065-015-9315-8

O'Connor, K., Hallam, R., & Rachman, S. (1985). Fearlessness and courage: A replication experiment. *British Journal of Psychology*, *76*(2), 187–197. https://doi.org/10.1111/j.2044-8295.1985.tb01942.x

O'Neil, C. (2016). *Weapons of math destruction: How big data increases inequality and threatens democracy*. London, England: Penguin.

Oakley, D. (2001). General cardiology: The athlete's heart. *Heart*, *86*(6), 722–726. https://doi.org/10.1136/heart.86.6.722

Obrist, P. A. (1976). The cardiovascular-behavioral interaction - As it appears today. *Psychophysiology*, *13*(2), 95–107. https://doi.org/10.1111/j.1469-8986.1976.tb00081.x

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A Perspective*. New York, NY: Plenum Press.

Office for National Statistics. (2015). Improving crime statistics in England and Wales. Retrieved from http://webarchive.nationalarchives.gov.uk/20160105160709/http://www.ons.gov.uk/ons/rel/crime-stats/crime-statistics/year-ending-june-2015/sty-fraud.html

Ogilvie, J. M., Stewart, A. L., Chan, R. C. K., & Shum, D. H. K. (2011). Neuropsychological measures of executive function and antisocial behavior: A meta-analysis. *Criminology*, *49*(4), 1063–1107. https://doi.org/10.1111/j.1745-9125.2011.00252.x

Öhman, A. (2005). The role of the amygdala in human fear: Automatic detection of threat. *Psychoneuroendocrinology*, *30*(10), 953–958. https://doi.org/10.1016/j.psyneuen.2005.03.019

Oldenhof, H., Prätzlich, M., Ackermann, K., Baker, R., Batchelor, M., Baumann, S., … Popma, A. (2019). Baseline autonomic nervous system activity in female children and adolescents with conduct disorder: Psychophysiological findings from the FemNAT-CD study. *Journal of Criminal Justice*, *65*, 1-10. https://doi.org/10.1016/j.jcrimjus.2018.05.011

Olfert, M. D., Barr, M. L., Charlier, C. M., Famodu, O. A., Zhou, W., Mathews, A. E., … Colby, S. E. (2018). Self-reported vs. measured height, weight, and BMI in young adults. *International Journal of Environmental Research and Public Health*, *15*(10), 1–9. https://doi.org/10.3390/ijerph15102216

Oppenheimer, S. M., Gelb, A., Girvin, J. P., & Hachinski, V. C. (1992). Cardiovascular effects of human insular cortex stimulation. *Neurology*, *42*(9), 1727–1732. https://doi.org/10.1212/wnl.42.9.1727

Orme-Johnson, D. W. (2011). The use of meditation in corrections. *International Journal of Offender Therapy and Comparative Criminology*, *55*(4), 662–664. https://doi.org/10.1177/0306624X10371803

Ortiz, J., & Raine, A. (2004). Heart rate level and antisocial behavior in children and adolescents: A meta-analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, *43*(2), 154–162. https://doi.org/10.1097/00004583-200402000-00010

Osumi, T., Shimazaki, H., Imai, A., Sugiura, Y., & Ohira, H. (2007). Psychopathic traits and cardiovascular responses to emotional stimuli. *Personality and Individual Differences*, *42*(7), 1391–1402. https://doi.org/10.1016/j.paid.2006.10.016

Pajevic, M., Batinic, B., & Stevanovic, N. (2017). Subtypes of homicide offenders based on psychopathic traits. *International Journal of Law and Psychiatry*, *55*, 45–53.

https://doi.org/10.1016/j.ijlp.2017.10.007

Pajevic, M., Vukosavljevic-Gvozden, T., Stevanovic, N., & Neumann, C. S. (2018). The relationship between the Dark Tetrad and a two-dimensional view of empathy. *Personality and Individual Differences*, *123*, 125–130. https://doi.org/10.1016/j.paid.2017.11.009

Palumbo, S., Mariotti, V., Iofrida, C., & Pellegrini, S. (2018). Genes and aggressive behavior: Epigenetic mechanisms underlying individual susceptibility to aversive environments. *Frontiers in Behavioral Neuroscience*, *12*(117), 1–9. https://doi.org/10.3389/fnbeh.2018.00117

Papathanasiou, G., Georgakopoulos, D., Papageorgiou, E., Zerva, E., Michalis, L., Kalfakakou, V., & Evangelou, A. (2013). Effects of smoking on heart rate at rest and during exercise, and on heart rate recovery, in young adults. *Hellenic Journal of Cardiology*, *54*(3), 168–177.

Parvizi, J., Rangarajan, V., Shirer, W. R., Desai, N., & Greicius, M. D. (2013). The will to persevere induced by electrical stimulation of the human cingulate gyrus. *Neuron*, *80*(6), 1359–1367. https://doi.org/10.1016/j.neuron.2013.10.057

Patrick, C. J., Fowles, D. C., & Krueger, R. F. (2009). Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. *Development and Psychopathology*, *21*(3), 913–938. https://doi.org/10.1017/S0954579409000492

Pauwels, L. J. R. (2018). Analysing the perception–choice process in situational action theory. A randomized scenario study. *European Journal of Criminology*, *15*(1), 130–147. https://doi.org/10.1177/1477370817732195

Pauwels, L. J. R., Svensson, R., & Hirtenlehner, H. (2018). Testing situational action theory: A narrative review of studies published between 2006 and 2015. *European Journal of Criminology*, *15*(1), 32–55. https://doi.org/10.1177/1477370817732185

Pechtel, P., Lyons-Ruth, K., Anderson, C. M., & Teicher, M. H. (2014). Sensitive periods of amygdala development: The role of maltreatment in preadolescence. *NeuroImage*, *97*, 236–244. https://doi.org/10.1016/j.neuroimage.2014.04.025

Perach-Barzilay, N., Tauber, A., Klein, E., Chistyakov, A., Ne’eman, R., & Shamay-Tsoory, S. G. (2013). Asymmetry in the dorsolateral prefrontal cortex and aggressive behavior: A continuous theta-burst magnetic stimulation study. *Social Neuroscience*, *8*(2), 178–188. https://doi.org/10.1080/17470919.2012.720602

Perner, J. (1991). *Understanding the representational mind*. Cambridge, MA: MIT Press.

Perry, G., Wikström, P.-O. H., & Roman, G. D. (2018). Differentiating right-wing extremism from potential for violent extremism: The role of criminogenic exposure. *International Journal of Developmental Sciences*, *12*(1–2), 103–113. https://doi.org/10.3233/DEV-170240

Perry, W., McInnis, B., Price, C., Smith, S., & Hollywood, J. (2013). *Predictive policing: The role of crime forecasting in law enforcement operations*. RAND Corporation. https://doi.org/10.7249/rr233

Petrovic, P., & Castellanos, F. X. (2016). Top-down dysregulation—from ADHD to emotional instability. *Frontiers in Behavioral Neuroscience*, *10*(70), 1–25. https://doi.org/10.3389/fnbeh.2016.00070

Pickering, D. (2013). How to measure the pulse. *Community Eye Health Journal*, *26*(82), 37.

Piferi, R. L., Kline, K. A., Younger, J., & Lawler, K. A. (2000). An alternative approach for achieving cardiovascular baseline: Viewing an aquatic video. *International Journal of Psychophysiology*, *37*(2), 207–217. https://doi.org/10.1016/S0167-8760(00)00102-1

Piquero, N. L., Ling, S., Raine, A., & Piquero, A. R. (2020). Heart rate fails to predict white collar crime. *American Journal of Criminal Justice*, *45*, 349–362. https://doi.org/10.1007/s12103-019-09503-6

Popma, A., Jansen, L. M. C., Vermeiren, R., Steiner, H., Raine, A., Van Goozen, S. H. M., … Doreleijers, T. A. H. (2006). Hypothalamus pituitary adrenal axis and autonomic activity during stress in delinquent male adolescents and controls. *Psychoneuroendocrinology*, *31*(8), 948–957. https://doi.org/10.1016/j.psyneuen.2006.05.005

Poppa, T., & Bechara, A. (2015). A neural perspective of immoral behavior and psychopathy. *AJOB Neuroscience*, *6*(3), 15–24. https://doi.org/10.1080/21507740.2015.1047052

Poppa, T., & Bechara, A. (2018). The somatic marker hypothesis: Revisiting the role of the ‘body-loop’ in decision-making. *Current Opinion in Behavioral Sciences*, *19*, 61–66. https://doi.org/10.1016/j.cobeha.2017.10.007

Portnoy, J., Chen, F. R., & Raine, A. (2013). Biological protective factors for antisocial and criminal behavior. *Journal of Criminal Justice*, *41*(5), 292–299. https://doi.org/10.1016/j.jcrimjus.2013.06.018

Portnoy, J., & Farrington, D. P. (2015). Resting heart rate and antisocial behavior: An updated systematic review and meta-analysis. *Aggression and Violent Behavior*, *22*, 33–45. https://doi.org/10.1016/j.avb.2015.02.004

Portnoy, J., Legee, K., Raine, A., Choy, O., & Rudo-Hutt, A. S. (2019). Biosocial risk factors for academic dishonesty: Testing a new mediation model in young adults. *Journal of*

Contemporary Criminal Justice, *35*(1), 21-35. https://doi.org/10.1177/1043986218810590

Portnoy, J., Raine, A., Chen, F. R., Pardini, D., Loeber, R., & Jennings, J. R. (2014). Heart rate and antisocial behavior: The mediating role of impulsive sensation seeking. *Criminology, 52*(2), 292–311. https://doi.org/10.1111/1745-9125.12038

Posthumus, J. A., Böcker, K. B. E., Raaijmakers, M. A. J., Van Engeland, H., & Matthys, W. (2009). Heart rate and skin conductance in four-year-old children with aggressive behavior. *Biological Psychology*, *82*(2), 164–168. https://doi.org/10.1016/j.biopsycho.2009.07.003

Pratt, T. C., Turanovic, J. J., Fox, K. A., & Wright, K. A. (2014). Self-control and victimization: A meta-analysis. *Criminology, 52*(1), 87–116. https://doi.org/10.1111/1745-9125.12030

Prätzlich, M., Oldenhof, H., Steppan, M., Ackermann, K., Baker, R., Batchelor, M., … Stadler, C. (2019). Resting autonomic nervous system activity is unrelated to antisocial behaviour dimensions in adolescents: Cross-sectional findings from a European multi-centre study. *Journal of Criminal Justice*, *65,* 1-11. https://doi.org/10.1016/j.jcrimjus.2018.01.004

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*(3), 879–891. https://doi.org/10.3758/BRM.40.3.879

Quay, H. C. (1965). Psychopathic personality as pathological stimulation-seeking. *The American Journal of Psychiatry*, *122*(2), 180–183. https://doi.org/10.1176/ajp.122.2.180

Quick, V., Byrd-Bredbenner, C., Shoff, S., White, A. A., Lohse, B., Horacek, T., … Greene, G. (2015). Concordance of self-report and measured height and weight of college students. *Journal of Nutrition Education and Behavior*, *47*(1), 94–98. https://doi.org/10.1016/j.jneb.2014.08.012

Quinn, P. D., & Harden, K. P. (2013). Differential changes in impulsivity and sensation seeking and the escalation of substance use from adolescence to early adulthood. *Development and Psychopathology*, *25*(1), 223–239. https://doi.org/10.1017/S0954579412000284

Quirk, G. J., & Beer, J. S. (2006). Prefrontal involvement in the regulation of emotion: Convergence of rat and human studies. *Current Opinion in Neurobiology, 16*(6), 723–727. https://doi.org/10.1016/j.conb.2006.07.004

R Core Team (2019). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL https://www.R-project.org/.

Rabinovici, G. D., & Miller, B. L. (2010). Frontotemporal lobar degeneration: Epidemiology, pathophysiology, diagnosis and management. *CNS Drugs*, *24*(5), 375–398. https://doi.org/10.2165/11533100-000000000-00000

Raine, A. (1993a). Other biological factors: Head injury, pregnancy, and birth complications, physical appearance, hormones, diet, and lead. In *The psychopathology of crime: Criminal behavior as a clinical disorder* (pp. 191–214). San Diego, CA: Academic Press.

Raine, A. (1993b). *The psychopathology of crime: Criminal behavior as a clinical disorder*. San Diego: Academic Press. https://doi.org/10.1016/C2009-0-03301-7

Raine, A. (1996a). Autonomic nervous system activity and violence. In D. M. Stoff & R. B. Cairns (Eds.), *Aggression and violence: Genetic, neurobiological, and biosocial perspectives* (pp. 145–168). Mahwah, NJ: Lawrence Erlbaum Associates Publishers.

Raine, A. (1996b). Autonomic nervous system factors underlying disinhibited, antisocial, and violent behavior: Biosocial perspectives and treatment implications. *Annals of the New York Academy of Sciences*, *794*(1), 46–59. https://doi.org/10.1111/j.1749-6632.1996.tb32508.x

Raine, A. (2002). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggresive behavior in children. *Journal of Child Psychology and Psychiatry*, *43*(4), 417-434. https://doi.org/10.1111/1469-7610.00034

Raine, A. (2013). *The anatomy of violence : The biological roots of crime*. London, England: Allen Lane.

Raine, A. (2015). Low resting heart rate as an unequivocal risk factor for both the perpetration of and exposure to violence. *JAMA Psychiatry*, *72*(10), 962–964. https://doi.org/10.1001/jamapsychiatry.2015.1178.Conflict

Raine, A. (2018). Antisocial personality as a neurodevelopmental disorder. *Annual Review of Clinical Psychology*, *14*, 259–289. https://doi.org/10.1146/annurev-clinpsy-050817-084819

Raine, A. (2019). The neuromoral theory of antisocial, violent, and psychopathic behavior. *Psychiatry Research*, *277*, 64–69. https://doi.org/10.1016/j.psychres.2018.11.025

Raine, A., Buchsbaum, M., & LaCasse, L. (1997). Brain abnormalities in murderers indicated by positron emission tomography. *Biological Psychiatry*, *42*, 495–508. https://doi.org/10.1016/S0006-3223(96)00362-9

Raine, A., Dodge, K., Loeber, R., Gatzke-Kopp, L., Lynam, D., Reynolds, C., … Liu, J. (2006). The reactive-proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. *Aggressive Behavior*, *32*(2), 159–171. https://doi.org/10.1002/ab.20115

Raine, A., Fung, A. L. C., Portnoy, J., Choy, O., & Spring, V. L. (2014). Low heart rate as a risk factor for child and adolescent proactive aggressive and impulsive psychopathic behavior. *Aggressive Behavior*, *40*(4), 290–299. https://doi.org/10.1002/ab.21523

Raine, A., Lencz, T., Bihrle, S., LaCasse, L., & Colletti, P. (2000). Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Archives of General Psychiatry*, *57*(2), 119–127. https://doi.org/10.1001/archpsyc.57.2.119

Raine, A., & Liu, J. H. (1998). Biological predispositions to violence and their implications for biosocial treatment and prevention. *Psychology, Crime and Law*, *4*(2), 107–125. https://doi.org/10.1080/10683169808401752

Raine, A., Reynolds, C., Venables, P. H., Mednick, S. A., & Farrington, D. P. (1998). Fearlessness, stimulation-seeking, and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. *Archives of General Psychiatry*, *55*(8), 745–751. https://doi.org/10.1001/archpsyc.55.8.745

Raine, A, Venables, P. H., & Williams, M. (1990). Relationships between central and autonomic measures of arousal at age 15 years and criminality at age 24 years. Archives of General Psychiatry, 47(11), 1003–1007. https://doi.org/10.1001/archpsyc.1990.01810230019003

Raine, A., Venables, P. H., & Williams, M. (1995). High autonomic arousal and electrodermal orienting at age 15 years as protective factors against criminal behavior at age 29 years. *American Journal of Psychiatry*, *152*(11), 1595–1600. https://doi.org/10.1176/ajp.152.11.1595

Raine, A., Venables, P., & Mednick, S. (1997). Low resting heart rate at age 3 years predisposes to aggression at age 11 years: Evidence from the Mauritius Child Health Project. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*(10), 1457–1464. https://doi.org/10.1097/00004583-199710000-00029

Raine, A., & Yang, Y. (2006). Neural foundations to moral reasoning and antisocial behavior. *Social Cognitive and Affective Neuroscience*, *1*(3), 203–213. https://doi.org/10.1093/scan/nsl033

Ramsay, M. C., & Reynolds, C. R. (1995). Separate digits tests: A brief history, a literature review, and a reexamination of the factor structure of the test of memory and learning (TOMAL). *Neuropsychology Review*, *5*(3), 151–171. https://doi.org/10.1007/BF02214760

Rankin, K. P., Gorno-Tempini, M. L., Allison, S. C., Stanley, C. M., Glenn, S., Weiner, M. W., & Miller, B. L. (2006). Structural anatomy of empathy in neurodegenerative disease. *Brain*, *129*(11), 2945–2956. https://doi.org/10.1093/brain/awl254

Repple, J., Pawliczek, C. M., Voss, B., Siegel, S., Schneider, F., Kohn, N., & Habel, U. (2017). From provocation to aggression: The neural network. *BMC Neuroscience*, *18*(73), 1–9. https://doi.org/10.1186/s12868-017-0390-z

Reuter, P. H. (2010). *Marijuana legalization: What can be learned from other countries?* Baltimore: RAND Corporation. Retrieved from https://www.rand.org/content/dam/rand/pubs/working_papers/2010/RAND_WR771.pdf

Riva, P., Manfrinati, A., Sacchi, S., Pisoni, A., & Romero Lauro, L. J. (2019). Selective changes in moral judgment by noninvasive brain stimulation of the medial prefrontal cortex. *Cognitive, Affective and Behavioral Neuroscience*, *19*, 797–810. https://doi.org/10.3758/s13415-018-00664-1

Rocque, M., Posick, C., & Felix, S. (2015). The role of the brain in urban violent offending: Integrating biology with structural theories of 'the streets.' *Criminal Justice Studies*, *28*(1), 84–103. https://doi.org/10.1080/1478601X.2014.1000006

Rogeness, Graham A., Cepeda, C., Macedo, C. A., Fisher, C., & Harris, W. R. (1990a). Differences in heart rate and blood pressure in children with conduct disorder, major depression, and separation anxiety. *Psychiatry Research*, *33*(2), 199–206. https://doi.org/10.1016/0165-1781(90)90074-F

Rogeness, G.A., Javors, M. A., Mass, J. W., & Macedo, C. A. (1990b). Catecholamines and diagnoses in children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*(2), 234–241. https://doi.org/10.1097/00004583-199003000-00012

Rosenbloom, M. H., Schmahmann, J. D., & Price, B. H. (2012). The functional neuroanatomy of decision-making. *Journal of Neuropsychiatry and Clinical Neurosciences*, *24*(3), 266–277. https://doi.org/10.1176/appi.neuropsych.11060139

Rosseel, Y. (2012). Lavaan: An R package for structural equation modeling. *Journal of Statistical Software*, *48*(2), 1–36. https://doi.org/10.18637/jss.v048.i02

Roth, L., Kaffenberger, T., Herwig, U., & Brühl, A. B. (2014). Brain activation associated with pride and shame. *Neuropsychobiology*, *69*(2), 95–106. https://doi.org/10.1159/000358090

Ruiz Vargas, E., Sörös, P., Shoemaker, J. K., & Hachinski, V. (2016). Human cerebral circuitry related to cardiac control: A neuroimaging meta-analysis. *Annals of Neurology*, *79*, 709–716. https://doi.org/10.1002/ana.24642

Russell, J., & Barrett, L. (1999). Core affect, prototypical emotional episodes, and other things called emotion: Dissecting the elephant. *Journal of Personality and Social Psychology*, *76*(5), 805–819. https://doi.org/10.1037/0022-3514.76.5.805

Sagawa, Y., Kondo, H., Matsubuchi, N., Takemura, T., Kanayama, H., Kaneko, Y., … Shimizu, T. (2011). Alcohol has a dose-related effect on parasympathetic nerve activity during sleep. *Alcoholism: Clinical and Experimental Research*, *35*(11), 2093–2100. https://doi.org/10.1111/j.1530-0277.2011.01558.x

Salekin, R. T., Chen, D. R., Sellbom, M., Lester, W. S., & MacDougall, E. (2014). Examining the factor structure and convergent and discriminant validity of the Levenson Self-Report Psychopathy Scale: Is the two-factor model the best fitting model? *Personality Disorders: Theory, Research, and Treatment*, *5*(3), 289–304. https://doi.org/10.1037/per0000073

Salthouse, T. A. (2005). Relations between cognitive abilities and measures of executive functioning. *Neuropsychology*, *19*(4), 532–545. https://doi.org/10.1037/0894-4105.19.4.532

Sampson, R. J. (2006). Collective efficacy theory: Lessons learned and directions for future inquiry. In F. T. Cullen, J. P. Wright, & K. R. Blevins (Eds.), *Advances in criminological theory: Vol. 15. Taking stock: The status of criminological theory* (pp. 149–167). Abingdon, England: Routledge. https://doi.org/10.4324/9781315130620-6

Saver, J. L., & Damasio, A. R. (1991). Preserved access and processing of social knowledge in a patient with acquired sociopathy due to ventromedial frontal damage. *Neuropsychologia*, *29*(12), 1241–1249. https://doi.org/10.1016/0028-3932(91)90037-9

Scarpa, A., Haden, S. C., & Tanaka, A. (2010). Being hot-tempered: Autonomic, emotional, and behavioral distinctions between childhood reactive and proactive aggression. *Biological Psychology*, *84*(3), 488–496. https://doi.org/10.1016/j.biopsycho.2009.11.006

Scarpa, A., & Raine, A. (2000). Violence associated with anger and impulsivity. In J. C. Borod (Ed.), *The neuropsychology of emotion* (pp. 320–339). New York, NY: Oxford University Press.

Scarpa, A., Tanaka, A., & Haden, S. C. (2008). Biosocial bases of reactive and proactive aggression: The roles of community violence exposure and heart rate. *Journal of Community Psychology*, *36*(8), 969–988. https://doi.org/10.1002/jcop

Schils, N., & Pauwels, L. J. R. (2014). Explaining violent extremism for subgroups by gender and immigrant background, using SAT as a framework. *Journal of Strategic Security*, *7*(3), 22–47. https://doi.org/10.5038/1944-0472.7.3.2

Schils, N., & Pauwels, L. J. R. (2016). Political violence and the mediating role of violent extremist propensities. *Journal of Strategic Security*, *9*(2), 70–91. https://doi.org/10.5038/1944-0472.9.2.1491

Schmidt, B., Mussel, P., & Hewig, J. (2013). I'm too calm - Let's take a risk! On the impact of state and trait arousal on risk taking. *Psychophysiology*, *50*(5), 498–503. https://doi.org/10.1111/psyp.12032

Schneider, B., & Koenigs, M. (2017). Human lesion studies of ventromedial prefrontal cortex. *Neuropsychologia*, *107*, 84–93. https://doi.org/10.1016/j.neuropsychologia.2017.09.035

Schoorl, J., Rijn, S. Van, Wied, M. De, Goozen, S. H. M. Van, & Swaab, H. (2016). Variability in emotional/behavioral problems in boys with oppositional defiant disorder or conduct disorder: The role of arousal. *European Child & Adolescent Psychiatry*, *25*(8), 821–830. https://doi.org/10.1007/s00787-015-0790-5

Schubert, M., Clark, A., & De La Rosa, A. (2018). The Polar® OH1 optical heart rate sensor is valid during moderate-vigorous exercise. *Sports Medicine International Open*, *2*(3), E67–E70. https://doi.org/10.1055/a-0631-0920

Schumacker, R. E., & Lomax, R. G. (2016). *A beginner's guide to structural equation modeling* (4th ed.). New York, NY: Routledge.

Schumann, A., Andrack, C., & Bär, K. J. (2017). Differences of sympathetic and parasympathetic modulation in major depression. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *79*(Pt B), 324–331. https://doi.org/10.1016/j.pnpbp.2017.07.009

Seeck, M., Zaim, S., Chaves-Vischer, V., Blanke, O., Maeder-Ingvar, M., Weissert, M., & Roulet, E. (2003). Ictal bradycardia in a young child with focal cortical dysplasia in the right insular cortex. *European Journal of Paediatric Neurology*, *7*(4), 177–181. https://doi.org/10.1016/S1090-3798(03)00051-5

Seeley, W. W., Crawford, R. K., Zhou, J., Miller, B. L., & Greicius, M. D. (2009). Neurodegenerative diseases target large-scale human brain networks. *Neuron*, *62*(1), 42–52. https://doi.org/10.1016/j.neuron.2009.03.024

Serra-Grima, R., Puig, T., Doñate, M., Gich, I., & Ramon, J. (2008). Long-term follow-up of bradycardia in elite athletes. *International Journal of Sports Medicine*, *29*(11), 934–937.

https://doi.org/10.1055/s-2008-1038602

Serrano-Maíllo, A. (2018). Crime contemplation and self-control: A test of situational action theory's hypothesis about their interaction in crime causation. *European Journal of Criminology*, *15*(1), 93–110. https://doi.org/10.1177/1477370817732193

Shankman, S. A., & Klein, D. N. (2003). The relation between depression and anxiety: An evaluation of the tripartite, approach-withdrawal and valence-arousal models. *Clinical Psychology Review*, *23*(4), 605–637. https://doi.org/10.1016/S0272-7358(03)00038-2

Shenhav, A., & Greene, J. D. (2014). Integrative moral judgment: Dissociating the roles of the amygdala and ventromedial prefrontal cortex. *Journal of Neuroscience*, *34*(13), 4741–4749. https://doi.org/10.1523/JNEUROSCI.3390-13.2014

Shin, L. M., Dougherty, D. D., Orr, S. P., Pitman, R. K., Lasko, M., MacKlin, M. L., … Rauch, S. L. (2000). Activation of anterior paralimbic structures during guilt-related script-driven imagery. *Biological Psychiatry*, *48*(1), 43–50. https://doi.org/10.1016/S0006-3223(00)00251-1

Shulman, E. P., Harden, K. P., Chein, J. M., & Steinberg, L. (2015). Sex differences in the developmental trajectories of impulse control and sensation-seeking from early adolescence to early adulthood. *Journal of Youth and Adolescence*, *44*(1), 1–17. https://doi.org/10.1007/s10964-014-0116-9

Shulman, E. P., Steinberg, L. D., & Piquero, A. R. (2013). The age-crime curve in adolescence and early adulthood is not due to age differences in economic status. *Journal of Youth and Adolescence*, *42*, 848–860. https://doi.org/10.1007/s10964-013-9950-4

Sijtsema, J. J., Veenstra, R., Lindenberg, S., Van Roon, A. M., Verhulst, F. C., Ormel, J., & Riese, H. (2013). Heart rate and antisocial behavior: Mediation and moderation by affiliation with bullies. The TRAILS study. *Journal of Adolescent Health*, *52*(1), 102–107. https://doi.org/10.1016/j.jadohealth.2012.04.018

Sijtsema, J. J, Veenstra, R., Lindenberg, S., van Roon, A. M., Verhulst, F. C., Ormel, J., & Riese, H. (2010). Mediation of sensation seeking and behavioral inhibition on the relationship between heart rate and antisocial behavior. The TRAILS study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *49*(5), 493–502. https://doi.org/10.1016/j.jaac.2010.02.005

Sijtsema, J. Jurrit, Nederhof, E., Veenstra, R., Ormel, J., Oldehinkel, A. J., & Ellis, B. J. (2013). Effects of family cohesion and heart rate reactivity on aggressive/rule-breaking behavior and prosocial behavior in adolescence: The Tracking Adolescents' Individual

Lives Survey study. *Development and Psychopathology*, *25*(3), 699–712. https://doi.org/10.1017/S0954579413000114

Silver, J. M., Yudofsky, S. C., Slater, J. A., Kugelmass Gold, R., Katz Stryer, B. L., Williams, D. T., … Endicott, J. (1999). Propranolol treatment of chronically hospitalized aggressive patients. *Journal of Neuropsychiatry and Clinical Neurosciences*, *11*(3), 328–335. https://doi.org/10.1176/jnp.11.3.328

Skeem, J. L., & Cooke, D. J. (2010). Is criminal behavior a central component of psychopathy? Conceptual directions for resolving the debate. *Psychological Assessment*, *22*(2), 433–445. https://doi.org/10.1037/a0008512

Skinner, B. F. (1965). *Science and human behavior*. New York, NY: Free Press.

Smith, C. A., Ireland, T. O., Thornberry, T. P., & Elwyn, L. (2008). Childhood maltreatment and antisocial behavior: Comparison of self-reported and substantiated maltreatment. *American Journal of Orthopsychiatry*, *78*(2), 173–186. https://doi.org/10.1037/0002-9432.78.2.173

Smith, C., & Thornberry, T. P. (1995). The relationship between childhood maltreatment and adolescent involvement in delinquency. *Criminology*, *33*(4), 451–482. https://doi.org/10.1111/j.1745-9125.1995.tb01186.x

Smith, S. F., Duell, D. J., & Martin, B. C. (2003). *Clinical nursing skills: Basic to advanced skills* (6th ed.). Upper Saddle River, NJ: Prentice Hall.

Soler-Baillo, J. M., Marx, B. P., & Sloan, D. M. (2005). The psychophysiological correlates of risk recognition among victims and non-victims of sexual assault. *Behaviour Research and Therapy*, *43*(2), 169–181. https://doi.org/10.1016/j.brat.2004.01.004

Somerville, L. H., Jones, R. M., & Casey, B. J. (2010). A time of change: Behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. *Brain and Cognition*, *72*(1), 124–133. https://doi.org/10.1016/j.bandc.2009.07.003

Spaak, J., Merlocco, A. C., Soleas, G. J., Tomlinson, G., Morris, B. L., Picton, P., … Floras, J. S. (2008). Dose-related effects of red wine and alcohol on hemodynamics, sympathetic nerve activity, and arterial diameter. *American Journal of Physiology - Heart and Circulatory Physiology*, *294*(2), H605–H612. https://doi.org/10.1152/ajpheart.01162.2007

Stadler, C., Grasmann, D., Fegert, J. M., Holtmann, M., Poustka, F., & Schmeck, K. (2008). Heart rate and treatment effect in children with disruptive behavior disorders. *Child Psychiatry and Human Development*, *39*(3), 299–309. https://doi.org/10.1007/s10578-007-0089-y

Stadler, C., Poustka, F., & Sterzer, P. (2010). The heterogeneity of disruptive behavior disorders - implications for neurobiological research and treatment. *Frontiers in Psychiatry*, *1*(21), 1–14. https://doi.org/10.3389/fpsyt.2010.00021

Stalenheim, E. G. (2004). Long-term validity of biological markers of psychopathy and criminal recidivism: Follow-up 6-8 years after forensic psychiatric investigation. *Psychiatry Research*, *121*(3), 281–291. https://doi.org/10.1016/j.psychres.2003.07.002

Stanovich, K. E., & West, R. F. (2000). Individual differences in reasoning: Implications for the rationality debate? *The Behavioral and Brain Sciences*, *23*(5), 645–665. https://doi.org/10.1017/S0140525X00003435

Stanovich, K. E. (2009). Distinguishing the reflective, algorithmic, and autonomous minds: Is it time for a tri-process theory? In J. S. B. T. Evans & K. Frankish (Eds.), *In two minds: Dual processes and beyond* (pp. 55–88). New York, NY: Oxford University Press. https://doi.org/https://psycnet.apa.org/doi/10.1093/acprof:oso/9780199230167.003.0003

Stanovich, K. E., & Toplak, M. E. (2012). Defining features versus incidental correlates of Type 1 and Type 2 processing. *Mind and Society*, *11*, 3–13. https://doi.org/10.1007/s11299-011-0093-6

Steele, V. R., Claus, E. D., Aharoni, E., Vincent, G. M., Calhoun, V. D., & Kiehl, K. A. (2015). Multimodal imaging measures predict rearrest. *Frontiers in Human Neuroscience*, *9*(425), 1–13. https://doi.org/10.3389/fnhum.2015.00425

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences*, *9*(2), 69–74. https://doi.org/10.1016/j.tics.2004.12.005

Steinberg, L. (2007). Risk taking in adolescence: New perspectives from brain and behavioral science. *Current Directions in Psychological Science*, *16*(2), 55–59. https://doi.org/10.1111/j.1467-8721.2007.00475.x

Steinberg, L. (2010). A dual systems model of adolescent risk-taking. *Developmental Psychobiology*, *52*(3), 216–224. https://doi.org/10.1002/dev.20445

Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology*, *44*(6), 1764–1778. https://doi.org/10.1037/a0012955

Steinberg, L., Graham, S., O'Brien, L., Woolard, J., Cauffman, E., & Banich, M. (2009). Age differences in future orientation and delay discounting. *Child Development*, *80*(1), 28–44. https://doi.org/10.1111/j.1467-8624.2008.01244.x

Steinberg, L., Icenogle, G., Shulman, E. P., Breiner, K., Chein, J., Bacchini, D., … Takash,

H. M. S. (2018). Around the world, adolescence is a time of heightened sensation seeking and immature self-regulation. *Developmental Science*, *21*(2), 1–13. https://doi.org/10.1111/desc.12532

Steiner, E., Tata, M., & Frisén, J. (2019). A fresh look at adult neurogenesis. *Nature Medicine*, *25*, 542–543. https://doi.org/10.1038/s41591-019-0408-4

Sterzer, P., Stadler, C., Poustka, F., & Kleinschmidt, A. (2007). A structural neural deficit in adolescents with conduct disorder and its association with lack of empathy. *NeuroImage*, *37*(1), 335–342. https://doi.org/10.1016/j.neuroimage.2007.04.043

Suchy, Y. (2009). Executive functioning: Overview, assessment, and research issues for non-neuropsychologists. *Annals of Behavioral Medicine*, *37*(2), 106–116. https://doi.org/10.1007/s12160-009-9097-4

Svensson, R., & Pauwels, L. (2010). Is a risky lifestyle always “risky”? The interaction between individual propensity and lifestyle risk in adolescent offending: A test in two urban samples. *Crime and Delinquency*, *56*(4), 608–626. https://doi.org/10.1177/0011128708324290

Svensson, R., Pauwels, L., & Weerman, F. M. (2010). Does the effect of self-control on adolescent offending vary by level of morality? A test in three countries. *Criminal Justice and Behavior*, *37*(6), 732–743. https://doi.org/10.4324/9781315094908

Syngelaki, E. M., Moore, S. C., Savage, J. C., Fairchild, G., & Van Goozen, S. H. M. (2009). Executive functioning and risky decision making in young male offenders. *Criminal Justice and Behavior*, *36*(11), 1213–1227. https://doi.org/10.1177/0093854809343095

Szatkowska, I., Szymańska, O., Bojarski, P., & Grabowska, A. (2007). Cognitive inhibition in patients with medial orbitofrontal damage. *Experimental Brain Research*, *181*(1), 109–115. https://doi.org/10.1007/s00221-007-0906-3

Szczepanski, S. M., & Knight, R. T. (2014). Insights into human behavior from lesions to the prefrontal cortex. *Neuron*, 83(5), 1002-1018. https://doi.org/10.1016/j.neuron.2014.08.011

Taber-Thomas, B. C., Asp, E. W., Koenigs, M., Sutterer, M., Anderson, S. W., & Tranel, D. (2014). Arrested development: Early prefrontal lesions impair the maturation of moral judgement. *Brain*, *137*(4), 1254–1261. https://doi.org/10.1093/brain/awt377

Talati, A., Keyes, K. M., & Hasin, D. S. (2016). Changing relationships between smoking and psychiatric disorders across twentieth century birth cohorts: Clinical and research implications. *Molecular Psychiatry*, *21*(4), 464–471. https://doi.org/10.1038/mp.2015.224

Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart rate variability, prefrontal neural function, and cognitive performance: The neurovisceral integration perspective on self-regulation, adaptation, and health. *Annals of Behavioral Medicine, 37*(2), 141–153. https://doi.org/10.1007/s12160-009-9101-z

Thayer, J. F., & Lane, R. D. (2009). Claude Bernard and the heart-brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience and Biobehavioral Reviews*, *33*(2), 81–88. https://doi.org/10.1016/j.neubiorev.2008.08.004

The use of pretrial "risk assessment" instruments: A shared statement of civil rights concerns. (2018). Retrieved from http://civilrightsdocs.info/pdf/criminal-justice/Pretrial-Risk-Assessment-Full.pdf

Trebuchon, A., Bartolomei, F., McGonigal, A., Laguitton, V., & Chauvel, P. (2013). Reversible antisocial behavior in ventromedial prefrontal lobe epilepsy. *Epilepsy and Behavior*, *29*(2), 367–373. https://doi.org/10.1016/j.yebeh.2013.08.007

Treiber, K. (2011). The neuroscientific basis of situational action theory. In K. M. Beaver & A. Walsh (Eds.), *The Ashgate research companion to biosocial theories of crime* (pp. 213–247). Farnham, England: Ashgate Publishing Limited.

Treiber, K. (2014). A neuropsychological test of criminal decision-making: Regional prefrontal influences in a dual process model. In J.-L. Van Gelder, H. Elffers, D. Reynald, & D. Nagin (Eds.), *Affect and cognition in criminal decision making* (pp. 193–220). Abingdon, England: Routledge.

Treiber, K. (2017a). Biosocial criminology and models of criminal decision making. In W. Bernasco, J. L. Van Gelder, & H. Elffers (Eds.), The Oxford handbook of offender decision making (pp. 87–120). Oxford, England: Oxford University Press.

Treiber, K. (2017b). Situational action theory and PADS+: Theoretical and methodological advances in the study of life-course criminology. In A. Blokland & V. Van der Geest (Eds.), *International handbook of life course criminology* (pp. 50–73). Abingdon, England: Routledge.

Tremblay, R. E. (2015). Developmental origins of chronic physical aggression: An international perspective on using singletons, twins and epigenetics. *European Journal of Criminology*, *12*(5), 551–561. https://doi.org/10.1177/1477370815600617

Trivedi-Bateman, N. (2019). The combined roles of moral emotion and moral rules in explaining acts of violence using a situational action theory perspective. *Journal of Interpersonal Violence*, 1–26. https://doi.org/10.1177/0886260519852634

Ty, A., Mitchell, D. G. V, & Finger, E. (2017). Making amends: Neural systems supporting donation decisions prompting guilt and restitution. *Personality and Individual Differences*, *107*, 28–36. https://doi.org/10.1016/j.paid.2016.11.004

Valentini, M., & Parati, G. (2009). Variables influencing heart rate. *Progress in Cardiovascular Diseases*, *52*(1), 11–19. https://doi.org/10.1016/j.pcad.2009.05.004

Van de Weijer, S., de Jong, R., Bijleveld, C., Blokland, A., & Raine, A. (2017). The role of heart rate levels in the intergenerational transmission of crime. *Societies*, *7*(3), 23. https://doi.org/10.3390/soc7030023

Van Der Gronde, T., Kempes, M., Van El, C., Rinne, T., & Pieters, T. (2014). Neurobiological correlates in forensic assessment: A systematic review. *PLoS ONE*, *9*(10), 1–15. https://doi.org/10.1371/journal.pone.0110672

Van Goozen, S. H. M., Matths, W., Cohen-Kettenis, P. T., Buitelaar, J. K., & Van England, H. (2000). Hypothalamic-pituitary-adrenal axis and autonomic nervous system activity in disruptive children and matched controls. *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*(11), 1438–1445. https://doi.org/10.1097/00004583-200011000-00019

Van Goozen, S. H. M., Matthys, W., Cohen-Kettenis, P. T., Gispen-De Wied, C., Wiegant, V. M., & Van Engeland, H. (1998). Salivary cortisol and cardiovascular activity during stress in oppositional-defiant disorder boys and normal controls. *Biological Psychiatry*, *43*(7), 531–539. https://doi.org/10.1016/S0006-3223(97)00253-9

Van Harmelen, A. L., Van Tol, M. J., Van Der Wee, N. J. A., Veltman, D. J., Aleman, A., Spinhoven, P., … Elzinga, B. M. (2010). Reduced medial prefrontal cortex volume in adults reporting childhood emotional maltreatment. *Biological Psychiatry*, *68*(9), 832–838. https://doi.org/10.1016/j.biopsych.2010.06.011

Van Hulle, C. A, Corley, R., Zahn-Waxler, C., Kagan, J., & Hewitt, J. K. (2000). Early childhood heart rate does not predict externalizing behavior problems at age 7 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*(10), 1238–1244. https://doi.org/10.1097/00004583-200010000-00010

Van Zonneveld, L., Platje, E., de Sonneville, L., van Goozen, S., & Swaab, H. (2017). Affective empathy, cognitive empathy and social attention in children at high risk of criminal behaviour. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *58*(8), 913–921. https://doi.org/10.1111/jcpp.12724

Vaske, J. C. (Ed.). (2017). Using biosocial criminology to improve criminal justice outcomes [Special issue]. *Criminal Justice and Behavior*, 44(8), 1050-1072. https://doi.org/10.1177/0093854817716484

Vazsonyi, A. T., Clifford Wittekind, J. E., Belliston, L. M., & Van Loh, T. D. (2004). Extending the general theory of crime to “The East:” Low self-control in Japanese late adolescents. *Journal of Quantitative Criminology*, *20*(3), 189–216. https://doi.org/10.1023/B:JOQC.0000037731.28786.e3

Vazsonyi, A. T., Jiskrova, G. K., Ksinan, A. J., & Blatný, M. (2016). An empirical test of self-control theory in Roma adolescents. *Journal of Criminal Justice*, *44*, 66–76. https://doi.org/10.1016/j.jcrimjus.2015.12.004

Vazsonyi, A. T., & Ksinan, A. J. (2017). Understanding deviance through the dual systems model: Converging evidence for criminology and developmental sciences. *Personality and Individual Differences*, *111*, 58–64. https://doi.org/10.1016/j.paid.2017.01.030

Vazsonyi, A. T., Mikuška, J., & Kelley, E. L. (2017). It’s time: A meta-analysis on the self-control-deviance link. *Journal of Criminal Justice*, *48*, 48–63. https://doi.org/10.1016/j.jcrimjus.2016.10.001

Venables, N. C., Hall, J. R., & Patrick, C. J. (2014). Differentiating psychopathy from antisocial personality disorder: A triarchic model perspective. *Psychological Medicine*, *44*, 1005–1013. https://doi.org/10.1017/S003329171300161X

Venables, P. (1987). Autonomic nervous system factors in criminal behavior. In S. Mednick, T. Moffitt, & S. Stack (Eds.), *The causes of crime: New biological approaches* (pp. 110–136). Cambridge, England: Cambridge University Press. https://doi.org/doi:10.1017/CBO9780511753282.009

Venigalla, H., Mekala, M. H., Hassan, M., Ahmed, R., Zain, H., Dar, S., Veliz, S. S. (2017). An update on biomarkers in psychiatric disorders - Are we aware, do we use in our clinical practice? *Mental Health in Family Medicine*, *13*, 471–479.

Voellmin, A., Winzeler, K., Hug, E., Wilhelm, F. H., Schaefer, V., Gaab, J., … Bader, K. (2015). Blunted endocrine and cardiovascular reactivity in young healthy women reporting a history of childhood adversity. *Psychoneuroendocrinology*, *51*, 58–67. https://doi.org/10.1016/j.psyneuen.2014.09.008

Voss, P., Thomas, M. E., Cisneros-Franco, J. M., & De Villers-Sidani, É. (2017). Dynamic brains and the changing rules of neuroplasticity: Implications for learning and recovery. *Frontiers in Psychology*, *8*(1657), 1–11. https://doi.org/10.3389/fpsyg.2017.01657

Voyer, D., Russell, A., & McKenna, J. (2002). On the reliability of laterality effects in a dichotic emotion recognition task. *Journal of Clinical and Experimental Neuropsychology*, *24*(5), 605–614. https://doi.org/10.1076/jcen.24.5.605.1007

Wadsworth, M. E. J. (1976). Delinquency, pulse rates and early emotional deprivation.

British Journal of Criminology, *16*(3), 245–256. https://doi.org/10.1093/oxfordjournals.bjc.a046738

Wagner, U., N'Diaye, K., Ethofer, T., & Vuilleumier, P. (2011). Guilt-specific processing in the prefrontal cortex. *Cerebral Cortex, 21*(11), 2461–2470. https://doi.org/10.1093/cercor/bhr016

Waldron, J. C., Wilson, L. C., Patriquin, M. A., & Scarpa, A. (2015). Sexual victimization history, depression, and task physiology as predictors of sexual revictimization: Results from a 6-month prospective pilot study. *Journal of Interpersonal Violence*, *30*(4), 622–639. https://doi.org/10.1177/0886260514535258

Wall, D. S., & Williams, M. L. (2013). Policing cybercrime: Networked and social media technologies and the challenges for policing. *Policing and Society*, *23*(4), 409–412. https://doi.org/10.1080/10439463.2013.780222

Wallis, J. D. (2007). Orbitofrontal cortex and its contribution to decision-making. *Annual Review of Neuroscience*, *30*, 31–56. https://doi.org/10.1146/annurev.neuro.30.051606.094334

Walsh, A., & Beaver, K. M. (2009). *Biosocial criminology: New directions in theory and research*. New York, NY: Routledge. https://doi.org/10.4324/9780203929919

Walsh, A., & Yun, I. (2014). Epigenetics and allostasis: Implications for criminology. *Criminal Justice Review*, *39*(4), 411–431. https://doi.org/10.1177/0734016814530148

Wechsler, D. (2008). *Wechsler adult intelligence scale — fourth edition: Technical and interpretive manual*. San Antonio, TX: Pearson Assessment.

Weinhold, B. (2006). Epigenetics: The science of change. *Environmental Health Perspectives*, *114*(3), A161–A167. https://doi.org/10.1289/ehp.114-a160

Weisburd, D., & Piquero, A. R. (2008). How well do criminologists explain crime? Statistical modeling in published studies. *Crime and Justice: Review of Research*, *37*(1), 453–502. https://doi.org/10.1086/524284

Weiss, B., Nguyen, T., Trung, L., Ngo, V., & Lau, A. (2019). Tobacco smoking and antisocial deviance among Vietnamese, Vietnamese-American, and European-American adolescents. *Journal of Abnormal Child Psychology*, *47*(1), 59–69. https://doi.org/10.1007/s10802-018-0416-8

Weulen Kranenbarg, M., Holt, T. J., & van Gelder, J. L. (2019). Offending and victimization in the digital age: Comparing correlates of cybercrime and traditional offending-only, victimization-only and the victimization-offending overlap. *Deviant Behavior*, *40*(1), 40–55. https://doi.org/10.1080/01639625.2017.1411030

Wheelock, M. D., Harnett, N. G., Wood, K. H., Orem, T. R., Granger, D. A., Mrug, S., & Knight, D. C. (2016). Prefrontal cortex activity is associated with biobehavioral components of the stress response. *Frontiers in Human Neuroscience, 10*(583), 1–12. https://doi.org/10.3389/fnhum.2016.00583

Whiteside, S. P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, *30*, 669–689. https://doi.org/10.1016/S0191-8869(00)00064-7

Wickham, H. (2016). *ggplot2: Elegant graphics for data analysis*. https://doi.org/10.1093/bioinformatics/btr406

Wikström, P.-O. H. (1990). Age and crime in a Stockholm cohort. *Journal of Quantitative Criminology*, *6*(6), 1990. https://doi.org/10.1007/BF01065290

Wikström, P.-O. H. (2004). Crime as alternative: Towards a cross-level situational action theory of crime causation. In J. McCord (Ed.), *Beyond empiricism: Institutions and intentions in the study of crime* (pp. 1–37). New Brunswick, NJ: Transaction.

Wikström, P.-O. H. (2006). Individuals, settings, and acts of crime: Situational mechanisms and the explanation of crime. In P. -O. H. Wikström & R. Sampson (Eds.), *The explanation of crime: Context, mechanisms, and development* (pp. 61–107). Cambridge, England: Cambridge University Press. https://doi.org/10.1017/CBO9780511489341.004

Wikström, P.-O. H. (2008). Deterrence and deterrence experiences: Preventing crime through the threat of punishment. In S. Shoran, O. Beck, & M. Kett (Eds.), *International handbook of penology and criminal justice* (pp. 345–378). Boca Raton, FL: CRC Press.

Wikström, P.-O. H. (2009). Crime propensity, criminogenic exposure and crime involvement in early to mid adolescence. *Monatsschrift Für Kriminologie Und Strafrechtsreform*, *92*(2–3), 253–266. https://doi.org/10.1515/mks-2009-922-312

Wikström, P.-O. H. (2010). Explaining crime as moral actions. In S. Hitlin & S. Vaisey (Eds.), *Handbook of the sociology of morality* (pp. 211–239). New York, NY: Springer. https://doi.org/10.1007/978-1-4419-6896-8_12

Wikström, P.-O. H. (2012). Does everything matter? Addressing the problem of causation and explanation in the study of crime. In J. M. McGloin, C. J. Sullivan, & L. W. Kennedy (Eds.), *When crime appears: The role of emergence*. Abingdon, England: Routledge.

Wikström, P.-O. H. (2014). Why crime happens: A situational action theory. In G. Manzo (Ed.), *Analytical sociology: Actions and networks* (pp. 74–94). Hoboken, NJ: Wiley-Blackwell. https://doi.org/10.1002/9781118762707.ch03

Wikström, P.-O. H. (2017). Character, circumstances, and the causes of crime: Towards an analytical criminology. In A. Liebling, S. Maruna, & L. McAra (Eds.), *The Oxford handbook of criminology*. Oxford, England: Oxford University Press. https://doi.org/10.1093/he/9780198719441.003.0023

Wikström, P.-O. H. (2019a). Explaining crime and criminal careers: The DEA model of situational action theory. *Journal of Developmental and Life-Course Criminology*. https://doi.org/https://doi.org/10.1007/s40865-019-00116-5

Wikström, P.-O. H. (2019b). Situational action theory: A general, dynamic and mechanism-based theory of crime and its causes (2nd ed.). In M. D. Krohn, N. Hendrix, G. Penly Hall, & A. J. Lizotte (Eds.), *Handbook on crime and deviance* (pp. 259–281). Charn, Switzerland: Springer. https://doi.org/10.1007/978-3-030-20779-3_14

Wikström, P.-O. H., & Bouhana, N. (2017). Analyzing radicalization and terrorism: A situational action theory. In G. LaFree & J. D. Freilich (Eds.), *The handbook of the criminology of terrorism* (pp. 175–186). Chichester, England: John Wiley & Sons, Inc.

Wikström, P.-O. H., Ceccato, V., Hardie, B., & Treiber, K. (2010). Activity fields and the dynamics of crime: Advancing knowledge about the role of the environment in crime causation. *Journal of Quantitative Criminology*, *26*(1), 55–87. https://doi.org/10.1007/s10940-009-9083-9

Wikström, P.-O. H., Mann, R. P., & Hardie, B. (2018). Young people's differential vulnerability to criminogenic exposure: Bridging the gap between people- and place-oriented approaches in the study of crime causation. *European Journal of Criminology*, *15*(1), 10–31. https://doi.org/10.1177/1477370817732477

Wikström, P.-O. H., Oberwittler, D., Treiber, K., & Hardie, B. (2012). *Breaking rules: The social and situational dynamics of young people's urban crime*. Oxford, England: Oxford University Press.

Wikström, P.-O. H., Oberwittler, D., Treiber, K., & Hardie, B. (2017). Situational action theory. In T. R. McGee & P. Mazerolle (Eds.), *Developmental and life-course criminological theories* (pp. 125–170). London, England: Routledge https://doi.org/10.4324/9781315094908

Wikström, P.-O. H., & Svensson, R. (2008). Why are English youths more violent than Swedish youths?: A comparative study of the role of crime propensity, lifestyles and their interactions in two cities. *European Journal of Criminology*, *5*(3), 309–330. https://doi.org/10.1177/1477370808090835

Wikström, P.-O. H., & Svensson, R. (2010). When does self-control matter? The interaction between morality and self-control in crime causation. *European Journal of Criminology*, *7*(5), 395–410. https://doi.org/10.1177/1477370810372132

Wikström, P.-O. H., & Treiber, K. (2007). The role of self-control in crime causation: Beyond Gottfredson and Hirschi's general theory of crime. *European Journal of Criminology, 4*(2), 237–264. https://doi.org/10.1177/1477370807074858

Wikström, P.-O. H., & Treiber, K. (2009a). Violence as situational action. *International Journal of Conflict and Violence*, *3*(1), 75–96. https://doi.org/10.4119/UNIBI/ijcv.49

Wikström, P.-O. H., & Treiber, K. (2009b). What drives persistent offending? The neglected and unexplored role of the social environment. In J. Savage (Ed.), *The Development of Persistent Criminality* (pp. 389–420). Oxford, England: Oxford University Press. https://doi.org/10.1093/acprof:oso/9780195310313.003.0019

Wikström, P.-O. H., & Treiber, K. (2016a). Situational theory: The importance of interactions and action mechanisms in the explanation of crime. In A. R. Piquero (Ed.), *The handbook of criminological theory* (pp. 415–444). Chichester, England: John Wiley & Sons, Inc.

Wikström, P.-O. H., & Treiber, K. (2016b). Social disadvantage and crime: A criminological puzzle. *American Behavioral Scientist*, *60*(10), 1232–1259. https://doi.org/10.1177/0002764216643134

Wikström, P.-O. H., & Treiber, K. (2017). Beyond risk factors: An analytical approach to crime prevention. In B. Teasdale & M. S. Bradley (Eds.), *Preventing crime and violence* (pp. 73–87). Cham, Switzerland: Springer.

Wikström, P.-O. H., & Treiber, K. (2019). The dynamics of change: Criminogenic interactions and life-course patterns in crime. In D. P. Farrington, L. Kazemian, & A. R. Piquero (Eds.), *The Oxford handbook of developmental and life-course criminology* (pp. 272–294). Oxford, England: Oxford University Press.

Wikström, P.-O. H., Tseloni, A., & Karlis, D. (2011). Do people comply with the law because they fear getting caught? *European Journal of Criminology*, *8*(5), 401–420. https://doi.org/10.1177/1477370811416415

Williams, K. M., Paulhus, D. L., & Hare, R. D. (2007). Capturing the four-factor structure of psychopathy in college students via self-report. *Journal of Personality Assessment*, *88*(2), 205–219. https://doi.org/10.1080/00223890701268074

Wilson, J. Q., & Herrnstein, R. J. (1985). *Crime and human nature*. New York, NY: Touchstone Books.

Wilson, L. C., & Scarpa, A. (2011). The link between sensation seeking and aggression: A meta-analytic review. *Aggressive Behavior*, *37*(1), 81–90. https://doi.org/10.1002/ab.20369

Wilson, L. C., & Scarpa, A. (2013). Baseline heart rate, sensation seeking, and aggression in young adult women: A two-sample examination. *Aggressive Behavior, 39*(4), 280–289. https://doi.org/10.1002/ab.21477

Wilson, L. C., & Scarpa, A. (2014). Aggressive behavior: An alternative model of resting heart rate and sensation seeking. *Aggressive Behavior*, *40*(1), 91–98. https://doi.org/10.1002/ab.21504

Wittling, W., Block, A., Genzel, S., & Schweiger, E. (1998). Hemisphere asymmetry in parasympathetic control of the heart. *Neuropsychologia*, *36*(5), 461–468. https://doi.org/10.1016/S0028-3932(97)00129-2

Wolf, R. C., Philippi, C. L., Motzkin, J. C., Baskaya, M. K., & Koenigs, M. (2014). Ventromedial prefrontal cortex mediates visual attention during facial emotion recognition. *Brain*, *137*(Pt 6), 1772–1780. https://doi.org/10.1093/brain/awu063

World Health Organization. (1992). The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines. In *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva, Switzerland: World Health Organization.

Wright, E. M. (2009). The measurement of psychopathy: Dimensional and taxometric approaches. *International Journal of Offender Therapy and Comparative Criminology*, *53*(4), 464–481. https://doi.org/10.1177/0306624X08319416

Wright, J. P., & Beaver, K. M. (2005). Do parents matter in creating self-control in their children? A genetically informed test of Gottfredson and Hirschi's theory of low self-control. *Criminology*, *43*(4), 1169–1202. https://doi.org/10.1111/j.1745-9125.2005.00036.x

Wright, J. P., & Cullen, F. T. (2012). The future of biosocial criminology: Beyond scholars' professional ideology. *Journal of Contemporary Criminal Justice*, *28*(3), 237–253. https://doi.org/10.1177/1043986212450216

Wright, R., Riedel, R., Sechrest, L., Lane, R. D., & Smith, R. (2018). Sex differences in emotion recognition ability: The mediating role of trait emotional awareness. *Motivation and Emotion*, *42*, 149–160. https://doi.org/10.1007/s11031-017-9648-0

Xu, Y., Raine, A., Yu, L., & Krieg, A. (2014). Resting heart rate, vagal tone, and reactive and proactive aggression in Chinese children. *Journal of Abnormal Child Psychology*, *42*(3), 501–514. https://doi.org/10.1007/s10802-013-9792-2

Yang, T. T., Simmons, A. N., Matthews, S. C., Tapert, S. F., Bischoff-Grethe, A., Frank, G. K. W., … Paulus, M. P. (2007). Increased amygdala activation is related to heart rate during emotion processing in adolescent subjects. *Neuroscience Letters, 428*(2–3), 109–114. https://doi.org/10.1016/j.neulet.2007.09.039

Yang, Y., Glenn, A. L., & Raine, A. (2008). Brain abnormalities in antisocial individuals: Implications for the law. *Behavioral Sciences and the Law*, *26*(1), 65-83. https://doi.org/10.1002/bsl.788

Yang, Y., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta-analysis. *Psychiatry Research - Neuroimaging*, *174*(2), 81–88. https://doi.org/10.1016/j.pscychresns.2009.03.012

Young, L., Bechara, A., Tranel, D., Damasio, H., Hauser, M., & Damasio, A. R. (2010). Damage to ventromedial prefrontal cortex impairs judgment of harmful intent. *Neuron, 65*(6), 845–851. https://doi.org/10.1016/j.neuron.2010.03.003

Yurgelun-Todd, D. (2007). Emotional and cognitive changes during adolescence. *Current Opinion in Neurobiology*, *17*(2), 251–257. https://doi.org/10.1016/j.conb.2007.03.009

Zahn, T. P., & Kruesi, M. J. (1993). Autonomic activity in boys with disruptive behavior disorders. *Psychophysiology, 30*(6), 605–614. https://doi.org/10.1111/j.1469-8986.1993.tb02086.x

Zelazo, P. D., & Carlson, S. M. (2012). Hot and cool executive function in childhood and adolescence: Development and plasticity. *Child Development Perspectives*, *6*(4), 354–360. https://doi.org/10.1111/j.1750-8606.2012.00246.x

Zelazo, P. D., & Müller, U. (2002). Executive function in typical and atypical development. In U. Goswami (Ed.), *Blackwell handbook of childhood cognitive development*. Malden, MA: Blackwell Publisher Ltd. https://doi.org/10.1002/9781444325485.ch22

Zhao, C., Deng, W., & Gage, F. H. (2008). Mechanisms and functional implications of adult neurogenesis. *Cell*, *132*(4), 645–660. https://doi.org/10.1016/j.cell.2008.01.033

Zimmerman, D. L., Ownsworth, T., O'Donovan, A., Roberts, J., & Gullo, M. J. (2016). Independence of hot and cold executive function deficits in high-functioning adults with autism spectrum disorder. *Frontiers in Human Neuroscience, 10*(24), 1–14. https://doi.org/10.3389/fnhum.2016.00024

Zuckerman, M. (1994). *Behavioral expressions and biosocial bases of sensation seeking*. Cambridge, England: Cambridge University Press.

Zuckerman, M., Kuhlman, D. M., Joireman, J., Teta, P., & Kraft, M. (1993). A comparison of three structural models for personality: The Big Three, the Big Five, and the Alternative

Five. *Journal of Personality and Social Psychology*, *65*(4), 757–768. https://doi.org/10.1037/0022-3514.65.4.757

Appendices

Appendix 1. Demographics Questionnaire

1. What is your sex?

Response options: Male; Female.

2. How old are you?

Response options: 16; 17; Other (type-in answer option).

3. What is your ethnicity?

Response options: White; Asian/Asian British; Black/Black British; Mixed/Multiple ethnic groups; Other ethnic group.

4. What is the highest level of education completed by your mother?

Response options: Not completed compulsory school; Completed compulsory school (GCSEs); A-levels; University degree.

5. What is the highest level of education completed by your father?

Response options: Not completed compulsory school; Completed compulsory school (GCSEs); A-levels; University degree.

6. Are you receiving free school meals?

Response options: Yes; No.

Appendix 2. Working Memory

Instructions: "You will be presented with a series of numbers and your goal is to repeat the numbers in the reverse order as they were presented. For example, if you are presented with numbers 3 and 5, respectively, the correct answer would be 53. This is a measure of your memory span, and therefore it is not allowed to write down the numbers. The goal of the task is to try to remember and reverse the order of numbers using only your mind, without using any external help (such as pencil and paper). Each number will be presented for one second, but you will have unlimited time to submit an answer."

Repeat the numbers in the reverse order as they appeared:

Trial 1: 2 4

5 8

Trial 2: 6 2 9

4 1 5

Trial 3: 3 2 7 9

4 9 6 8

Trial 4: 1 5 2 8 6

6 1 8 4 3

Trial 5: 5 3 9 4 1 8

7 2 4 8 5 6

Trial 6: 8 1 2 9 3 6 5

4 7 3 9 1 2 8

Trial 7: 9 4 3 7 6 2 5 8

7 2 8 1 9 6 5 3

Appendix 3. Crime Questionnaire

Participants were firstly asked whether they had committed a crime since they had started secondary school (crime versatility). If answered negatively, they would move to the next question. If answered affirmatively, they would be asked how many times had they committed the crime (crime frequency). In the interest of clarity, crime frequency and crime versatility questions will be presented independently.

3.1. Crime versatility.

Response options: “Yes” (=1), “No” (=0).

1. Since you started secondary school, have you stolen anything from a shop (for example, a CD, clothes, cosmetics, or any other things)?
2. Since you started secondary school, have you broken into someone’s house or flat to steal something?

3. Since you started secondary school, have you broken into a non-residential building to steal something (for example, broken into a shop, school, warehouse, office)?
4. Since you started secondary school, have you broken into a car to steal something?
5. Since you started secondary school, have you stolen anything from another person (for example, money, a mobile telephone, a bicycle, a wallet or a purse, a hand-bag, jewellery, a watch)? *Not counting events in which you broke into a car, house or non-residential building or shop-lifted
6. Since you started secondary school, have you stolen a car?
7. Since you started secondary school, have you damaged or destroyed things not belonging to you for fun or because you were bored or angry (for example, smashed windows or street lights, scratched the paint off cars, sprayed graffiti on a wall, damaged a bicycle)?
8. Since you started secondary school, have you set fire to something you were not supposed to set fire to (for example, started a fire in a school, started a fire in an empty building, set fire to a house, started a fire in a playground, started a fire in a wood)?
9. Since you started secondary school, have you used a weapon, hit or threatened to hurt someone, to take money or other things from them?
10. Since you started secondary school, have you beaten up or hit someone (for example, punched, stabbed, kicked, or head-butted someone)? *Not counting events when you took money or other things from someone
11. When you are out by yourself or with your friends do you ever carry a knife or other weapon?[33] *Do not count when you are at home or in school

Follow up questions:

11a. How often do you carry a knife or other weapon when you are out by yourself or with your friends? (response options: “Always”, “Most times”, “Sometimes”, “Not very often”)

11b. When you carry a weapon, what kind of weapon do you usually carry? (response options: “Knife”, “Other, please say what ____”)

12. When you are at school do you ever carry a knife or other weapon?

Follow up questions:

12a. How often do you carry a knife or other weapon when you are at school? (response options: “Always”, “Most times”, “Sometimes”, “Not very often”)

[33] The answers from the questions regarding carrying knifes or other weapons when out and at school were merged into a single variable.

12b. When you carry a weapon in school, what kind of weapon do you usually carry? (response options: “Knife”, “Other, please say what ____”)

13. Since you started secondary school, have you accessed someone else's computer, email or an online account without their permission?

Follow up questions:

13a. What type of computer material have you accessed without the owner's permission? (response options: “Files stored on a hard drive or cloud storage (for example, photos, videos, and documents)”, “Social networking account”, “Email account”, “Bank account”, “Other, please say what _____”)

13b. How do you usually obtain access to someone else's computer or an online account? (response options: “Accessing someone else's computer that is not password protected”, “Using the computer or an online account after their owner had forgotten to log out”, “Watching the person enter their details and then using their username and password to log in”, “Guessing password”, “Malware, please say what type”, “Other, please say what _____”)

14. Since you started secondary school, have you developed or distributed malware (for example, a computer virus) or other harmful software to deliberately damage or infect another person’s computer?

Follow up questions:

14a. What type of malware do you usually use? (response options: “Viruses”, “Worms”, “Trojans”, “Ransomware”, “Spyware”, “Other, please say what _____”)

15. Since you started secondary school, have you used a denial of service (DoS) attack (including “booter” or “stresser” services) to disrupt an online service or an individual’s internet connection?

Follow up questions:

15a. Why do you usually initiate a DoS attack? (response options: “For fun”, “To retaliate”, “To win in an online game by knocking the other player offline”, “For social or political reasons (hacktivism)”, “For financial gain”, “Other, please say what _____”)

3.2. Crime frequency.

Response option: type-in answer (participants were required to enter a number describing how many times they had committed each crime)

1. How many times have you stolen something from a shop?
2. How many times have you broken into someone’s house or flat to steal something?
3. How many times have you broken into a non-residential building to steal something?
4. How many times have you broken into a car to steal something?
5. How many times have you stolen something from another person?
6. How many times have you stolen a car?
7. How many times have you damaged or destroyed something not belonging to you?
8. How many times have you set fire to something you were not supposed to?
9. How many times have you used a weapon, hit or threatened to hurt someone, to take money or other things from them?
10. How many times have you beaten up or hit someone?

    Follow up questions:

    10a. Did you use a weapon the last time you beat up someone? (response options: “Yes”, “No”)

    10b. What sort of weapon did you use? (response options: “Knife”, “Other sharp instrument (for example, a broken bottle or razor)”, “Blunt instrument (for example, a piece of wood, iron bar or cosh)”, “Air gun”, “Firearm”)
11. How many times have you accessed someone else's computer or an online account without permission?
12. How many times have you developed or distributed malware or other harmful software to deliberately damage or infect other person’s computer?
13. How many times have you used a DoS attack to disrupt an online service or an individual’s internet connection?

Appendix 4. Lifestyle and Health Questionnaire

1. During a typical week, how many times do you do the following exercises for more than 25 minutes:

    A. Mild exercise that requires minimal effort where you could easily sing while doing the activity, such as yoga, fishing, bowling, golf, and leisure walking
    Response options: "Never" (=0), "1-3" (=1), "4-6" (=2), "7-9" (=3), "10 times or more" (=4).

    B. Moderate exercise that is not exhausting where you could easily carry on a conversation while doing the activity, such as jogging, non-competitive sports, leisure dancing, and leisure swimming
    Response options: "Never" (=0), "1-3" (=1), "4-6" (=2), "7-9" (=3), "10 times or more" (=4).

    C. Vigorous exercise where you become winded or too out of breath to carry on a conversation while doing the activity, such as running, competitive sports games (soccer, football, basketball, etc.), energetic dancing, and swimming laps
    Response options: "Never" (=0), "1-3" (=1), "4-6" (=2), "7-9" (=3), "10 times or more" (=4).

2. How many cigarettes do you smoke on an average day?
    Response options: "None / I do not smoke" (=0), "1-5" (=1), "6-10" (=2), "11-15" (=3), "16-20" (=4), "more than 20" (=5).

3. How often do you have a drink containing alcohol?
    Response options: "Never" (=0), "Monthly or less" (=1), "2-4 times a month" (=2), "2-3 times a week" (=3), "4 or more times per week" (=4).

4. How many units of alcohol do you drink on a typical day when you are drinking?

Response options: “1-2” (=0), “3-4” (=1), “5-6” (=2), “7-9” (=3), “10 or more” (=4)[34].

5. How often have you had 6 or more units of alcohol on a single occasion in the last year?[35] / How often have you had 8 or more units of alcohol on a single occasion in the last year?[36]

Response options: “Never” (=0), “Less than monthly” (=1), “Monthly” (=2), “Weekly” (=3), “Daily or almost daily” (=4).

6. How many caffeine-containing drinks (e.g., coffee, tea [excluding fruit and herbal teas, such as camomile or mint tea], coke, energy drinks) have you consumed in the past 12 hours?

Response options: “0” (=0), “1” (=1), “2” (=2), “3” (=3), “4” (=4), “5” (=5), “more than 5” (=6).

7. Have you ever tried drugs (cannabis, amphetamines, ecstasy, heroin, cocaine, crack, LSD, GBH, crystal meth, tranquilizers, magic mushrooms, or anabolic steroids)?

Response options: “Yes” (=1), “No” (=0).

8. For the drugs you have tried, please state how many times you have used them since you started secondary school.[37]

Listed drugs: “Cannabis (also called marijuana, grass, hash, dope, pot, weed, blow, skunk, ganja, spliff, wacky backy)”, “Amphetamines (also called speed, whizz, sulph, billy, sulphate, whites, base)”, “Ecstasy (also called E, pills, adam, doves, mitsubishis, rolexs, dolphins, XTC)”, “Heroin (also called smack, brown, dragon, gear, H, horse, junk, skag)”, “Cocaine (also called powder, C, charlie, coke, snow, percy, white, toot)”, “Crack (also called rocks, stone, wash, freebase)”, “LSD (also called acid, tabs, trips, dots, flash, smilies, rainbows)”, “Crystal methamphetamine (also called

[34] This question was not presented to the participants who responded with “never” to the previous question.

[35] This question was presented to female participants only

[36] This question was presented to male participants only

[37] This questions was presented only if a participant response affirmatively to the previous question

crystal meth, ice, crank, glass)", "Methadone (also called linctus, physeptone)", "GHB (also called GBH, Georgie Home Boy, liquid ecstasy, liquid Z, liquid G, fantasy)", "DXM, DM (cough suppressant)", "Tranquilizers (also called diazepam,valium, vallies, downers, roofies, benzos, temazepam, king kong pills, jellies, tams, mazzies, beans, eggs, rugby balls, ruggers, terminators, vitamin T)", "Magic mushrooms (also called shrooms, magics, liberties)", "Ketamine (also called green, K, super K)", "Anabolic steroids (also called roids)"

Response options: "Once or twice" (=1), "A few times (3-5 times)" (=2), "Many times (6-10 times)" (=3), "Very many times (11-50 times)" (=4), "Very, very many times (50+ times)" (=5).

9. Have you ever sold drugs (cannabis, amphetamines, ecstasy, heroin, cocaine, crack, LSD, GBH, crystal meth, tranquilizers, magic mushrooms, or anabolic steroids)?

Response options: "Yes" (=1), "No" (=0).

10. Please enter how many times you have sold different drugs.[38]

Listed drugs: "Cannabis (also called marijuana, grass, hash, dope, pot, weed, blow, skunk, ganja, spliff, wacky backy)", "Amphetamines (also called speed, whizz, sulph, billy, sulphate, whites, base)", "Ecstasy (also called E, pills, adam, doves, mitsubishis, rolexs, dolphins, XTC)", "Heroin (also called smack, brown, dragon, gear, H, horse, junk, skag)", "Cocaine (also called powder, C, charlie, coke, snow, percy, white, toot)", "Crack (also called rocks, stone, wash, freebase)", "LSD (also called acid, tabs, trips, dots, flash, smilies, rainbows)", "Crystal methamphetamine (also called crystal meth, ice, crank, glass)", "Methadone (also called linctus, physeptone)", "GHB (also called GBH, Georgie Home Boy, liquid ecstasy, liquid Z, liquid G, fantasy)", "DXM, DM (cough suppressant)", "Tranquilizers (also called diazepam,valium, vallies, downers, roofies, benzos, temazepam, king kong pills, jellies, tams, mazzies, beans, eggs, rugby balls, ruggers, terminators, vitamin T)", "Magic mushrooms (also called shrooms, magics, liberties)", "Ketamine (also called green, K, super K)", "Anabolic steroids (also called roids)"

Response option: type-in answer (participants are required to enter a number describing how many times had they sold each type of drugs).

[38] This question was presented only if a participant responded affirmatively to the previous question

11. What is your height?

Response option: type-in answer (participants are required to enter a number describing their height).

12. What is your weight?

Response option: type-in answer (participants are required to enter a number describing their weight).

Appendix 5. Instructions for the Montreal Imaging Stress Task

"Now that the video has finished, please listen carefully to the instructions for the mental arithmetic task that will follow. You will be working through a computer program which will display a series of mental arithmetic tasks for you to solve. Your goal is to solve each of these tasks correctly within the time limit. Prior participants in this study have performed well and solved 80% to 90% of the tasks correctly. Here is a screenshot of the program for you to see what it will look like. Here is where the arithmetic task will be displayed. Here is where you submit a response by pressing the appropriate number. Remember that this is a touch-screen, so the fastest way to submit an answer is by selecting a response using your finger. For example, two times two is four, and four plus four is eight, so the correct answer in this case would be eight. The program will always create tasks for which the solution is a number between zero and nine. As such, you only need to press a number once to select your response. Here is where the elapsed time will be shown. The progress bar moves from left to right and indicates how much time you have left to solve the task. Here is where the performance bar will be shown. The green part of the bar indicates good performance, the yellow part is marginal performance, and the red part signifies poor performance. There are also two performance indicators. You, the one below the bar, shows your own performance, and average, the one above the bar, shows an average performance of all the participants in this study. After you submit a response, you will then receive a feedback on your submitted response here. If you submit a correct answer, "correct" will appear in this text-box. However, if you submit a wrong answer, or you run out of time, "incorrect" will appear in this feedback box. It is very important to perform these calculations accurately. You must solve at least 80% of the tasks correctly, because otherwise we will not be able to use your data in this study. The required threshold is indicated by the green area of the performance bar. In order for the test to work, your performance must be in the green range of the bar. The study leader will monitor the whole experiment remotely to check if everyone's performance is within the green area. Now that you understand these instructions, you may proceed to the arithmetic task. Please begin the task now by pressing the continue button as shown on this screenshot."

Appendix 6. Correlations Between Control Variables, Heart Rate, and Crime

Free school meals correlated only with crime, $r = .12$, 95% BCa CI [.03, .21], $p = .009$, such that participants who received free school meals committed more crimes. Race only correlated with crime, $r = .13$, 95% BCa CI [.03, .22], $p = .006$, such that compared to non-white participants, white participants reported committing fewer criminal offences. Parental education did not correlate with either variable. Physical fitness correlated only with crime, $r = .18$, 95% BCa CI [.07, .29], $p < .001$. Smoking correlated only with crime, $r = .23$, 95% BCa CI [.12, 33], $p < .000$. Caffeine consumption prior to testing correlated only with crime, $r = .13$, 95% BCa CI [.04, .22], $p = .007$. Drug use correlated only with crime, $r = .38$, 95% BCa CI [.29, .51], $p < .001$. Body mass index correlated with neither of the variables.

Appendix 7. Fearlessness Mediation Model Using Modified Scale

Path analysis exploring mediation effects of fearfulness in context of the relationship between heart rate reactivity and crime was run in R version 3.5.3 (R Core Team, 2019) using the package lavaan 0.6-3 (Rosseel, 2012), whereas the histogram was produced using package ggplot2 3.1.0 (Wickham, 2016). Fearfulness was measured as a mean of two items, namely [Item removed for copyright reasons. Copyright holders are Michael C. Ashton and Kibeom Lee.] and "Even in an emergency I wouldn't feel like panicking". Internal consistency was measured using Spearman-Brown coefficient as it was found to be superior in comparison to Cronbach's alpha for assessing reliability of two-item subscales (Eisinga, Grotenhuis, & Pelzer, 2013). The resulting value of .51 indicated a low reliability, just above the acceptable level of .50. Histogram of the modified fearfulness indicated a relatively normal distribution (see Figure A7.1). Crime frequency was log transformed to alleviate the positive skew.

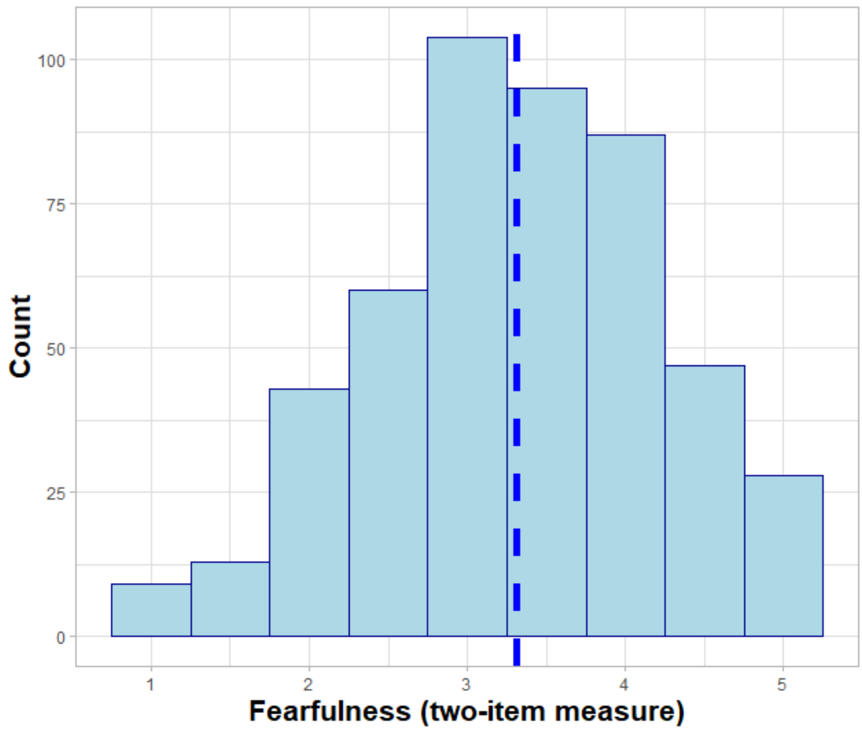

Figure A7.1. Frequency distribution of modified fearfulness. Dashed line represents the mean value of fearfulness (n = 486).

The assumption of multicollinearity was not violated in the model. There was no extreme collinearity as the correlation between the predictors, heart rate reactivity and modified fearfulness, was r = .11, p = .015. The lack of multicollinearity was further confirmed by collinearity statistics, as all tolerance statistics were well above the threshold of .10 and all variance inflation factor values were well below 10.0 (Kline, 2016).

Parameter estimates were derived using maximum likelihood method with robust, bootstrapped standard errors calculated from 5000 bootstrap samples due to violation of multivariate normality. For each path value and indirect effect, robust bias corrected and accelerated 95% confidence intervals using 5000 samples were produced. Across all variables used in these models, the number of missing values ranged from none in crime frequency to five in heart rate reactivity. Missing data were estimated using the full information maximum likelihood method.

There was a statistically significant effect of heart rate reactivity on modified fearfulness, b = 0.014, 95% BCa CI [0.003, 0.026], p = .016, which in turn significantly predicted crime, b = - 0.132, 95% BCa CI [- 0.196, - 0.066], p < .001. Direct effect of heart rate reactivity on crime was statistically significant, b = - 0.009, 95% BCa CI [- 0.016, - 0.002], p = .007, while the indirect effect through modified fearfulness was not statistically significant, *b* = - 0.002, 95% BCa CI [- 0.004, 0.000], *p* = .038. The results involving the standardised coefficients are presented in Figure A7.2.

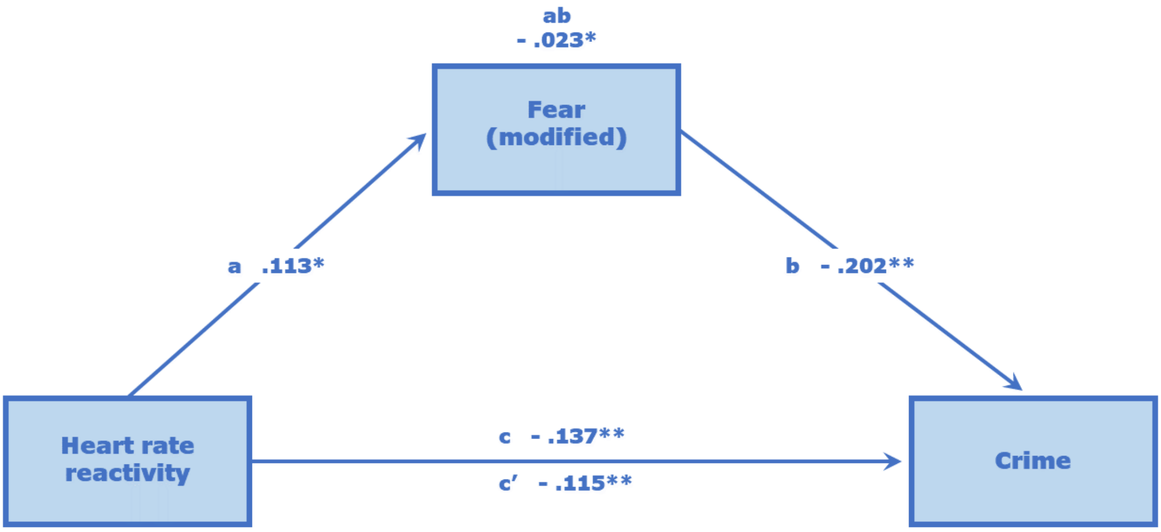

Figure A7.2. Mediation model of modified fearlessness. Standardised parameter estimates are shown for each path: a, b, c’, c, and ab (*n* = 487). Paths c and c’ refer to the total effect and direct effect of heart rate reactivity on crime, respectively. Path ab refers to the indirect effect. *p < .05. **p < .01.