




2017

# Understanding Antisocial Behavior Through Autonomic Arousal, Nutrition, And Non-Invasive Brain Stimulation

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# Understanding Antisocial Behavior Through Autonomic Arousal, Nutrition, And Non-Invasive Brain Stimulation

## **Abstract**

Despite increasing awareness of the contributions of biological sciences in criminology, the extent to which biological variables are incorporated in criminological research and theories remains limited. This dissertation consists of five papers that examine biological factors in conjunction with social environmental and psychological variables to gain a more complete understanding of the etiology of antisocial behavior. Paper 1 examined whether a biological mechanism may help to explain why social environmental factors that are identified in many criminological theories are associated with antisocial behavior. The finding that low heart rate partly mediated the relationship between early social adversity and antisocial behavior in children gives rise to a social neurocriminology perspective whereby social environmental factors influence biology to in turn lead to crime. Paper 2 expanded on this empirical proof of concept by proposing a biopsychosocial model to demonstrate how autonomic arousal can be incorporated into extant criminological theories. Paper 3 employed a biological perspective to explain an important phenomenon in criminology regarding the higher rate of male crime. Using longitudinal mediation analysis, the study is the first to document that lower heart rates in males at age 11 years partly explain their higher levels of offending in adulthood. Findings support the consideration of biological processes in theoretical accounts of the gender gap. Paper 4 examined whether a nutritional factor, vitamin D, confers resilience to childhood antisocial behavior. The finding that meeting vitamin D sufficiency (serum 25-hydroxyvitamin D concentration  $\geq 30$  ng/mL) nullified the social adversity-antisocial behavior relationship documents for the first time, a protective effect of vitamin D on antisocial behavior. Paper 5 examined the neural mechanisms underlying antisocial behavior using transcranial direct current stimulation. A double-blind, placebo-controlled, stratified, randomized trial on healthy adults provided the first experimental evidence that increasing activity in the prefrontal cortex can reduce intentions to commit aggression. This effect was account for, in part, by enhanced perceptions of moral wrongfulness regarding the aggressive acts. Together, these studies have the potential to advance the field of criminology at conceptual and theoretical levels, as well as knowledge on the development of prevention and intervention programs for such behavior.

## **Degree Type**

Dissertation

## **Degree Name**

Doctor of Philosophy (PhD)

## **Graduate Group**

Criminology

## **First Advisor**

Adrian Raine

## **Keywords**

antisocial behavior, biosocial criminology, crime, heart rate, transcranial direct current stimulation, vitamin D

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**Subject Categories**

Criminology | Criminology and Criminal Justice

UNDERSTANDING ANTISOCIAL BEHAVIOR THROUGH AUTONOMIC  
AROUSAL, NUTRITION, AND NON-INVASIVE BRAIN STIMULATION

Olivia Choy

A DISSERTATION

in

Criminology

Presented to the Faculties of the University of Pennsylvania

in

Partial Fulfillment of the Requirements for the

Degree of Doctor of Philosophy

2017

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## ACKNOWLEDGMENT

I am very grateful to all the people who have contributed to my PhD experience in inimitable ways. Most of all, I must thank my supervisor, Adrian Raine, who has been instrumental in every stage of my university experience, for it was in my freshman year that I was first introduced to the idea of a PhD, long before it became clear that I would complete this dissertation. I am immensely thankful for his generous mentorship and support over the past 7 years. I owe so very much of what I know about academia to him.

I also thank Jason Schnittker and Martha Farah. From volunteering to try tDCS to agreeing to go through five papers, I so appreciate their enthusiasm and support, which have served as big sources of motivation for me as the dissertation transitioned from proposals to completed papers. Beyond my dissertation committee, my appreciation goes to the individuals who contributed to versions of these papers in various ways: David Farrington, Peter Venables, Roy Hamilton, Jill Portnoy, Anna Rudo-Hutt, Yu Gao, and Liana Soyfer.

Additionally, the studies in this dissertation were funded, in part, under grants from the Pennsylvania Department of Health (SAP# 4100043366), the Clinical and Translational Research Center, Perelman School of Medicine, University of Pennsylvania (UL1-RR-024134), the National Institute of Mental Health (MH-46435), the Wellcome Trust (UK), and the Leverhulme Trust (UK).

The PhD journey has been made easier with the support of the Criminology department. I especially thank John MacDonald and Charles Loeffler for their guidance and advice, as well as Knakiya Hagans, Caroline Gonzalez Ciccone, and Addie Métivier

for their invaluable help. I would also like to acknowledge with gratitude, past and current members of the Raine Lab and fellow PhD students, who have been a big part of my PhD experience in numerous ways.

Finally, I thank my family whose encouragement to pursue Criminology, which began even before my very first semester at Penn, was truly the first step towards this dissertation.

## ABSTRACT

### UNDERSTANDING ANTISOCIAL BEHAVIOR THROUGH AUTONOMIC AROUSAL, NUTRITION, AND NON-INVASIVE BRAIN STIMULATION

Olivia Choy

Adrian Raine

Despite increasing awareness of the contributions of biological sciences in criminology, the extent to which biological variables are incorporated in criminological research and theories remains limited. This dissertation consists of five papers that examine biological factors in conjunction with social environmental and psychological variables to gain a more complete understanding of the etiology of antisocial behavior. Paper 1 examined whether a biological mechanism may help to explain *why* social environmental factors that are identified in many criminological theories are associated with antisocial behavior. The finding that low heart rate partly mediated the relationship between early social adversity and antisocial behavior in children gives rise to a social neurocriminology perspective whereby social environmental factors influence biology to in turn lead to crime. Paper 2 expanded on this empirical proof of concept by proposing a biopsychosocial model to demonstrate how autonomic arousal can be incorporated into extant criminological theories. Paper 3 employed a biological perspective to explain an important phenomenon in criminology regarding the higher rate of male crime. Using longitudinal mediation analysis, the study is the first to document that lower heart rates in males at age 11 years partly explain their higher levels of offending in adulthood.

Findings support the consideration of biological processes in theoretical accounts of the gender gap. Paper 4 examined whether a nutritional factor, vitamin D, confers resilience to childhood antisocial behavior. The finding that meeting vitamin D sufficiency (serum 25-hydroxyvitamin D concentration  $\geq 30$  ng/mL) nullified the social adversity-antisocial behavior relationship documents for the first time, a protective effect of vitamin D on antisocial behavior. Paper 5 examined the neural mechanisms underlying antisocial behavior using transcranial direct current stimulation. A double-blind, placebo-controlled, stratified, randomized trial on healthy adults provided the first experimental evidence that increasing activity in the prefrontal cortex can reduce intentions to commit aggression. This effect was account for, in part, by enhanced perceptions of moral wrongfulness regarding the aggressive acts. Together, these studies have the potential to advance the field of criminology at conceptual and theoretical levels, as well as knowledge on the development of prevention and intervention programs for such behavior.



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## GENERAL INTRODUCTION

The field of criminology has historically studied crime and antisocial behavior from a socially oriented perspective (Wilson & Scarpa, 2012). However, the inability of sociological constructs from traditional theories to fully explain antisocial behavior, even when examined simultaneously, suggests that other etiological mechanisms may be in play (e.g., Rebellon et al., 2015). Moreover, research from behavioral genetics has convincingly established that approximately 50% of the variation in antisocial behavior has a biological basis (Burt, 2009). This has led to the acknowledgment that criminological theories that fail to include biological influences may be limited in scope (DeLisi, 2012; Wilson & Scarpa, 2012).

Despite evidence that criminology is increasingly aware of the contributions of biological sciences, several gaps remain. First, many criminological theories still fail to incorporate biological influences (Wright & Boisvert, 2009). There remains a dearth of research addressing the relevance of biological processes to traditional socially oriented theories, as well as developmental and life-course theories of crime. Second, as risk factors are typically the focus of criminological research, less is known about factors that may *protect* individuals in high-risk environments from engaging in antisocial behavior (Hall et al., 2012; Portnoy, Chen, & Raine, 2013). Third, investigations of biological risk factors for antisocial behavior have largely supported correlational conclusions, rather than causal interpretations (Loeber, Byrd, & Farrington, 2015). Furthermore, although recent studies have begun to examine the mechanisms underlying the link between

biological risk factors and antisocial behavior, such mediating processes remain to be well-elucidated (Portnoy et al., 2014b; Sijtsema et al., 2010).

This dissertation consists of five papers that aim to bridge these gaps in the literature. The first limitation is addressed in the first three papers. Paper 1 contributes to the understanding of the etiology of crime by explaining *why* social adversity, a construct in many criminological theories, is associated with antisocial behavior. In contrast to studies that have explored social processes linking environmental factors to antisocial behavior (e.g., Agnew, 1992; Sampson, Raudenbush, & Earls, 1997; Sutherland, 1939), this study examined whether a biological mechanism, heart rate, helps to explain why social risk gives rise to such behavior. Paper 2 built on Paper 1 by providing a review of the empirical literature on the associations between autonomic arousal, measured by heart rate and skin conductance levels, and key psychological and social constructs in developmental and life-course criminological theories. A biopsychosocial framework was proposed to illustrate how a biological factor can be incorporated into extant criminological theories to improve the explanation and prediction of crime and antisocial behavior. Paper 3 employed a biological perspective to explain an important phenomenon in criminology, namely the higher rate of male crime (Heimer, Lauritsen, & Lynch, 2009; Steffensmeier, Zhong, & Ackerman, 2009). Dominant explanations for the gender gap, such as greater exposure of males to delinquent peers (Akers, 2009; Sutherland, 1947), males' heightened exposure to strain (Agnew, 1992; Broidy & Agnew, 1997), and differences in parenting and socialization that contribute to females' better abilities to control impulses (Gottfredson & Hirschi, 1990), have stemmed from sociological perspectives and are limited in their ability to fully explain the gender difference in crime



(Bell, 2009; Booth et al., 2008; Rebellon et al., 2015; Svensson, 2003). Given long-standing evidence that resting heart rate levels differ by gender (Ostchega, Porter, Hughes, Dillon, & Nwankwo, 2011), a low heart rate was examined as a partial explanation for why males are more criminal than females in a sample from Mauritius.

Regarding the second research gap, Paper 4 sought to contribute to the literature on protective factors for antisocial behavior by examining the role of a prohormone, vitamin D, on antisocial behavior in children. Vitamin D is involved in the regulation of brain development and has a neuroprotective function, which may help to protect against the damaging neurobiological effects of stress from chronic social adversity (Kalueff & Tuohimaa, 2007; Mackay-Sim, Féron, Eyles, Burne, & McGrath, 2004; Groves, McGrath, & Burne, 2014). Thus, the study aimed to contribute to the limited scientific foundation for the notion that youth in high-risk environments can be protected from engaging in antisocial behavior by testing whether vitamin D confers resilience to such behavior.

Paper 5 aimed to address the third limitation by adopting an experimental design to draw a *causal* inference about the neural basis of antisocial behavior in healthy adults. In a double-blind, placebo-controlled, stratified, randomized trial, the causal role of the prefrontal cortex on antisocial behavior was investigated using transcranial direct current stimulation. Additionally, the mechanism underlying the relationship between prefrontal functioning and intentions to commit aggression was assessed to add to our mechanistic understanding of the etiology of antisocial behavior. Together, the five papers employ a multidisciplinary approach, examining biological factors in conjunction with

psychological and social environmental variables, to gain a more complete understanding of the processes leading to antisocial behavior and how such behavior can be reduced.

**PAPER 1. THE MEDIATING ROLE OF HEART RATE ON THE SOCIAL ADVERSITY-ANTISOCIAL BEHAVIOR RELATIONSHIP: A SOCIAL NEUROCRIMINOLOGY PERSPECTIVE**

**Abstract**

Although social environmental factors are featured in criminological theories and documented to constitute significant criminogenic forces, little research has investigated biological processes that may help explain *why* social risk gives rise to antisocial behavior. This study tests the hypothesis that the social adversity-antisocial behavior relationship is partly mediated by a biological mechanism, low heart rate. 18 indicators of social adversity and heart rate measured at rest and in anticipation of a speech stressor were assessed alongside nine measures of antisocial behavior including delinquency (Youth Self-Report [YSR] and Child Behavior Checklist [CBCL]), conduct disorder (Conduct Disorder and Oppositional Defiant Disorder Questionnaire), and child psychopathy (Antisocial Process Screening Device [APSD]) in a community sample of 388 children aged 11 to 12 years. PROCESS was used to test mediation models. Low heart rate was a partial mediator of the adversity-antisocial behavior relationship, explaining 20.35% and 15.40% of the effect of social adversity on delinquency and overall antisocial behavior, respectively. Findings are, to the authors' knowledge, one of the first to establish any biological risk factor as a mediator of the social adversity-antisocial behavior relationship and suggest that social processes alter autonomic functioning in a way to predispose to antisocial behavior. While not definitive, results give rise to a social neurocriminology theory that argues that the social environment

influences biological risk factors in a way to predispose to antisocial and criminal behavior.

## **Background**

The role of environmental factors has long been acknowledged in the development of antisocial behavior, a construct that captures the violations of rules and the rights of others common to delinquency, criminality, aggression, conduct disorder, and psychopathy (Glenn & Yang, 2012; Lorber, 2004; Ogilvie, Stewart, Chan, & Shum, 2011; Stoff, Breiling, & Maser, 1997; Thornberry & Krohn, 2000). Studies have referred to a combination of environmental risk factors for antisocial behavior generally as “social adversity” (Moffitt, 1990; Moffitt & Caspi, 2001; Raine et al., 2005). Factors including bad home environments (Sutherland & Cressey, 1970; Wilson & Petersilia, 2011), urbanization (Flango & Sherbenou, 1976; Horney, Tolan, & Weisburd, 2012), and negative familial conditions early in life have been claimed to constitute significant criminogenic forces (Loeber & Farrington, 2000; Loeber, Farrington, Stouthamer-Loeber, Moffitt, & Caspi, 1998; Welsh, 2012). As such, the construct of social adversity has been featured prominently in many criminological theories, including strain models (Agnew, 1992; Cloward & Ohlin, 1960; Cohen, 1955; Merton, 1938), social control theory (Hirschi, 1969), cultural deviance theories (Shaw & McKay, 1942; Sutherland, 1947), and life-course theories (Sampson & Laub, 2005) and constitutes an important construct in the field of criminology.

Antisocial behavior can begin early in life (Tremblay, 2000) and early antisocial behavior has potentially serious implications for society, as it places individuals at increased risk for offending throughout the life course (Lanctot, Cernkovich, & Giordano, 2007; Loeber & Farrington, 2000; Piquero, Carriaga, Diamond, Kazemian, & Farrington, 2012; Piquero, Farrington, Welsh, Tremblay, & Jennings, 2009). Forms of antisocial

behavior including delinquency, conduct disorder, and child psychopathy are significant precursors to future offending (DeLisi, 2009; Dyck, Campbell, Schmidt, & Wershler, 2013; Fergusson & Horwood, 1995; Fergusson, Horwood, & Ridder, 2005; Hart & Hare, 1997; Lynam, Caspi, Moffitt, Loeber, & Stouthamer-Loeber, 2007; Lynam, Miller, Vachon, Loeber, & Stouthamer-Loeber, 2009; Robins, 1991). Conduct disorder is characterized by “a repetitive and persistent pattern of behavior in which the basic rights of others and major age-appropriate societal norms or rules are violated” (American Psychiatric Association, 2013), while child psychopathy is assessed in the APSD by callous-unemotional, narcissistic, and impulsive traits, factors that reflect the emotional, cognitive, and behavioral dimensions of the construct, respectively (Kotler & McMahon, 2005). Though less commonly included in criminological research, conduct disorder and child psychopathy deserve cross-disciplinary attention as they occur with greater frequency among cases defined as delinquent by criminologists. In addition, psychopaths are much more likely to be classified as career criminals, a construct of central importance in criminology (Blumstein, Cohen, Roth, & Visher, 1986; Vaughn & DeLisi, 2008).

However, there is a gap in the social adversity-antisocial behavior literature regarding the mechanisms, namely, the intervening variables (Bennett & George, 1997; Reskin, 2003), which underlie this relationship. The hypothesized processes vary according to different criminological theories (Tanner-Smith, Wilson, & Lipsey, 2013). Proposed mechanisms linking environmental factors to antisocial behavior include collective efficacy (Sampson, Raudenbush, & Earls, 1997), strain from inconsistent means and goals (Agnew, 1992), socialization to attitudes tolerant of antisocial behavior

(Akers, Krohn, Lanza-Kaduce, & Radosevich, 1979; Sutherland, 1939), self-reported violence and hyperactivity (Theobald, Farrington, & Piquero, 2013), quality of local services (Ellen & Turner, 1997), parent health (Kjellstrand & Eddy, 2011), parenting processes (Carson, Sullivan, Cochran, & Lersch, 2008; Thornberry, Freeman-Gallan, & Lovegrove, 2009), and labeling and stigma processes (Murray, Loeber, & Pardini, 2012). In stark contrast to this rich social literature, only limited research has been conducted on biological mediating mechanisms. Although a body of literature exists on biosocial *moderating* effects with respect to antisocial behavior (Beaver, Barnes, Boutwell, & Cooper, 2009; Boutwell & Beaver, 2008; Raine, 2002) and on environmental influences on biological functioning per se (Del Giudice, Ellis, & Shirtcliff, 2011; Ellis & Boyce, 2008), very few studies have formally tested *mediation* models to delineate biological processes that can help explain *why* social risk gives rise to antisocial behavior. In one study, testosterone levels were documented to mediate the association between adverse neighborhoods and antisocial behavior (Tarter et al., 2009), while another found that cortisol levels did not significantly mediate the link between childhood traumatic experiences and aggression (Cima, Smeets, & Jelicic, 2008). However, these studies have several limitations including being conducted solely on males, having small sample sizes, and failing to consider other contextual risk factors. To our knowledge, these are the only two empirical studies ever conducted on biological mediation and none have emanated from the discipline of criminology.

Recently, a more thorough multilevel understanding of antisocial behavior that includes interactional and bidirectional relationships between social and biological risk factors has been encouraged within the discipline of criminology (Barnes, Boutwell,

Beaver, Gibson, & Wright, 2014; Burt & Simons, 2014). Moreover, with the emergence of a public health approach to crime and violence that involves using knowledge of risk factors to inform the development of interventions, there is an increasing need to move beyond a surface-level description of risk factor associations to provide a deeper understanding of relationships (Welsh, Braga, & Sullivan, 2012; Wikström, 2006). Guided by these directions in criminology, a clearer understanding of the mechanisms linking environmental contextual factors to antisocial behavior is sought by employing what we term a “social neurocriminology” perspective – social influences on biology and the brain that in turn give rise to crime. Such an approach is not only consistent with the views of classic criminologists who claim that biological factors are significant to antisocial behavior only to the extent that they are related to environmental conditions (Sutherland & Cressey, 1970), but also answers calls for more integrative empirical research efforts in biosocial criminology (Burt & Simons, 2014). While neurocriminology has been defined as the application of neuroscience techniques to understand the origins of crime (Raine, 2013), with implications for prevention, prediction, and punishment (Glenn & Raine, 2014), social neurocriminology in contrast aims to bring social processes centrally into the equation in a manner that is consistent with criminologists strongly opposed to behavioral genetic studies on crime (Burt & Simons, 2014) as well as those who have strongly defended basic neuroscience research (Barnes et al., 2014) . It is within this new social neuroscience theoretical framework that the current study is embedded.

### **Heart Rate and Antisocial Behavior**



One putative biological mechanism that may help explain the adversity-antisocial behavior relationship is low heart rate. Low heart rate is arguably the best replicated biological correlate of antisocial behavior in children and adolescents (Ortiz & Raine, 2004). A meta-analysis revealed that low heart rates at rest ( $d = -.44$ ) and during stress ( $d = -.76$ ) were associated with antisocial behavior in children (Ortiz & Raine, 2004). The heart rate-antisocial behavior relationship has been observed across different species of animals, cultures, and genders, independent of potential confounds such as height, weight, and physical exercise, exclusive of other psychiatric conditions, and in prospective longitudinal studies (Cauffman, Steinberg, & Piquero, 2005; Cherkovich & Tatoyan, 1973; Eisermann, 1992; Farrington, 1997; Jennings, Piquero, & Farrington, 2013; Lorber, 2004; Moffitt, Caspi, Rutter, & Silva, 2001; Ortiz & Raine, 2004; Raine, 2002).

Within the heart rate-antisocial behavior literature, mixed results have been obtained regarding the underlying mechanism and the generalizability of the association between heart rate and subtypes of antisocial behavior such as child psychopathy. Mixed support has been found for the stimulation-seeking theory and the fearlessness theory (Armstrong & Boutwell, 2012; Armstrong, Keller, Franklin, & MacMillan, 2009; Portnoy et al., 2014b; Quay, 1965; Raine, 2002; Sijtsema et al., 2010; Venables, 1987). The stimulation-seeking theory posits that underarousal inclines individuals to seek stimulation through antisocial behavior. Fearlessness theory claims that reduced arousal during stress indexes a low fear in individuals who are then more likely to engage in antisocial behaviors that require a degree of fearlessness to execute. Additionally, studies on heart rate and psychopathy have yielded mixed results. In contrast to findings by

Baker et al. (2009) and Raine, Fung, Portnoy, Choy, and Spring (2014), Portnoy et al. (2014b) found no association between heart rate and psychopathy scores in a Western adolescent male sample. However, to date, no studies of heart rate have been conducted on a Western sample using the APSD, which is the most extensively used and validated measure of psychopathic-like behavior in children and adolescents. Despite these gaps, the robustness of the heart rate-antisocial behavior relationship renders this cardiovascular measure a particularly useful biological factor to incorporate into traditional criminological research.

### **Social Adversity and Biological Functioning**

An expanding body of research exists on biology and individuals' environments (Boyce, Barr, & Zeltzer, 1992; Champagne & Curley, 2005; Gottman & Katz, 1989; Henry & Stephens, 1977). Environmental risk factors such as lower socioeconomic status and child neglect have been associated with changes in the functioning of physiological stress response systems (Ellis & Boyce, 2008; Glaser, 2000; Hackman & Farah, 2009). One study found that boys from broken homes before the age of 4 (death of a parent, parents' divorce or separation, or long separation away from home) had lower pulse rates at age 11 compared to those from intact homes (Wadsworth, 1976). Lower pulse rates for boys were also related to larger family size. These findings suggest that early social adversity may lower heart rate later in life. Other studies have documented that individuals who have experienced aversive life events exhibit reduced heart rate in response to an unpleasant, stressful stimulus compared to those who have not experienced such events (Hagenaars, Stins, & Roelofs, 2012). In addition, environmental

risk factors such as poverty, unemployment, public assistance, higher costs of renting relative to income, and low property values have been linked to reduced brain serotonergic responsivity, which has been documented to co-occur with lower heart rates during stress (Manuck et al., 2005; Williams et al., 2001). In contrast, some studies have found that children exposed to risk factors such as disruptive parenting and marital conflict exhibit higher heart rate levels (Propper & Holochwost, 2013).

This apparent contradiction may be explained by the fact that acute (short term) stress elevates autonomic functions, while chronic (long term, sustained) stress dampens down autonomic functioning. Social adversity can influence heart rate by affecting the stress response system, which involves the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenocortical (HPA) axis. In response to a stressor, the sympathetic-adrenal-medullary (SAM) system of the SNS secretes adrenaline as part of the body's fight or flight response, while activation of the HPA axis leads to the release of stress hormones such as cortisol from the adrenal gland, resulting in increased heart rate (Gunnar, 2007). The parasympathetic arm of the autonomic nervous system reverses the effects of SAM activity and provides input to regions of the brain such as the amygdala that reduce reactivity to threat stimuli. However, long-term chronic exposure to stress, as when individuals experience social adversity early in life, can impose a biological burden on the physiological systems involved in coping and adaptation (McEwen, 1998). The high allostatic load can result in alterations in autonomic nervous system regulation and make it difficult for the body to respond adequately to stressors (McEwen, 1998). One proposed mechanism is the alteration of amygdala activity (Susman, 2006). Impaired amygdala functioning results in attenuation of the SNS and

HPA axis reactions to stress (Susman, 2006; Wentworth et al., 2013). While activation of the amygdala and stress response systems have an important adaptive function, downregulation of the stress system and autonomic functioning enables individuals to regulate the stress system so as not to continually evoke a chronic cardiovascular response to threatening situations and undergo excessive energy expenditure. Thus, social adversity can affect autonomic functioning by disrupting brain architecture.

### **Current Study**

This study examines the biological pathway leading to antisocial behavior specifically in children given that interventions for antisocial behavior are likely to be more effective at younger ages (Junger, Feder, & Côté, 2007). Since (a) social adversity is associated with antisocial behavior, (b) heart rate is associated with antisocial behavior, and (c) environmental processes can influence biological functioning, this study examines whether low heart rate may play some role in the relationship between social adversity and antisocial behavior. Based on a social neurocriminological approach, we hypothesize that social adversity experienced early in life predisposes to reduced heart rate (the mediator) measured at age 11 and 12 years which in turn predisposes to antisocial behavior.

Mediation effects for a total antisocial behavior score are examined in addition to the individual dimensions of delinquency, conduct disorder, and child psychopathy, as well as their subscales. The benefits to utilizing the overall behavioral measure as well as exploring specific dimensions within the larger construct of child antisocial behavior have been noted by other researchers (Piquero et al., 2009). It enables us to examine the

totality of the effects, while the disaggregation of the nonunitary constructs may be beneficial for theoretical development and the consideration of interventions (Piquero et al., 2009; Sullivan, Childs, & O'Connell, 2010). Additionally, parent-reported and child-reported ratings of antisocial behavior are analyzed together and separately.

## **Methods**

### **Subjects**

Data were collected on a sample of 454 children aged 11 and 12 years (mean age = 11.45 years,  $SD = .50$ ) recruited from within the city of Philadelphia and contiguous suburbs. Recruitment methods in the geographic sampling area included targeted mailing, flyers in recreation centers, libraries, health clinics, local stores, charter schools, and other community centers, advertisements, personal referrals, and enrollment through health care providers. Subjects with a diagnosis of psychiatric disorder, mental retardation, or a pervasive developmental disorder were excluded from the study. Eight subjects were deemed ineligible or withdrew after data collection. Additional information about subject recruitment and exclusionary criteria can be found in Liu et al. (2013) and Richmond, Cheney, Soyfer, Kimmel, & Raine (2013).

Complete data were obtained for 388 subjects (190 males and 198 females). Of the 388 participants, 12.4% ( $n = 48$ ) were White, 80.4% were African American ( $n = 312$ ), and 7.0% ( $n = 27$ ) were Asian, Native American, Hispanic, or multiracial. One participant declined to respond. No significant differences were observed in general antisocial behavior, conduct disorder scores, child psychopathy scores, age, sex, or race between individuals who were included in the analyses and those who were not ( $p > .05$ ).

The caregiver of each subject also participated in the study, serving as an informant for the child's behavior. Caregiver participation primarily involved the biological mothers (91.5%). Written informed consent and assent were obtained from both parents and children, and the study protocols were approved by the Institutional Review Board of the University of Pennsylvania and the Philadelphia Department of Health.

### **Antisocial Measures**

**Delinquency.** Subjects and their caregivers completed the YSR and its supplement, the CBCL, respectively (Achenbach & Rescorla, 2001). The scales included items regarding problem behaviors based on the preceding 6 months. Each problem behavior item was answered based on the degree it characterizes the child: 0 (not true), 1 (somewhat or sometimes true), or 2 (very or often true). Scores for the 32 items that make up the rule-breaking behavior and aggressive behavior syndromes were summed to obtain an overall scale we referred to as delinquency. The use of these two syndromes in the analysis of childhood antisocial behavior has been made by other researchers (Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007). The delinquency dimension includes behaviors that are more overt and that are considered "acting out," such as vandalism, truancy, lying, stealing, and destroying property (see Appendix A for full details; Lowe, 1998).

**Conduct disorder.** Subjects and their caregivers also completed the Conduct Disorder and Oppositional Defiant Disorder Questionnaire, a 24-item scale developed to assess conduct disorder and oppositional defiant disorder in terms of the *Diagnostic and*

*Statistical Manual of Mental Disorders-IV-TR* symptoms (Raine, 2008). The questionnaire asks about the occurrence of behaviors in the past year based on a 3-point scale: 0 (never), 1 (sometimes), or 2 (often). Items used to assess conduct disorder include how often a subject has “bullied or threatened someone,” “started a physical fight,” or “stolen things or shoplifted” (see Appendix A). The responses for 15 items were summed to calculate the total conduct disorder score. As the conduct disorder items can be further divided into seven aggressive and eight nonaggressive items, the aggression and nonaggression subfactors of conduct disorder were also analyzed.

**Child psychopathy.** Psychopathic personality traits in children were assessed by subjects and their caregivers using the APSD (Frick & Hare, 2001), which is particularly appropriate for use in a community sample (Marsee, Silverthorn, & Frick, 2005). The scale comprises 20 items answered on a three-point scale: 0 (not at all true), 1 (sometimes true), or 2 (definitely true). It is scored to derive an overall psychopathy score and can be distinguished into subcategories consisting of callous-unemotional, narcissistic, and impulsive traits (see Appendix A).

**General antisocial behavior.** Total delinquency, conduct disorder, and child psychopathy scores were standardized by transforming each value into Z scores. The standardized scores were subsequently summed to obtain a general measure of antisocial behavior.

## **Social Adversity**

Demographic information obtained by research assistants during an interview with caregivers was summarized and coded into 18 items. The items reflect early childhood adversity that is associated with long-term stress. Example items included parental education, parents' employment status, teenage pregnancy, living accommodation status, family size, parental supervision, whether the child is living with both biological parents or was separated from their mother between 6 months and 2.5 years of age, parents' physical and mental illness, and whether parents had been arrested, detained, or imprisoned (see Appendix B for full details).

The process of a face validity approach in combining theoretically connected, albeit uncorrelated variables into an overall construct has been adopted by other researchers and in other realms of research (Moffitt, 1990; Moffitt & Caspi, 2001; Raine et al., 2005). For example, the autonomic indicators of heart rate and skin conductance levels are recognized aspects of the same overall construct of physiological arousal despite having low correlations (Lazarus, Speisman, & Mordkoff, 1963). Similarly, despite a lack of association between individual birth complications such as breech delivery and placenta previa, variables have been combined into a composite birth complication score (Berendes, Weiss, Deutschberger, & Jackson, 1965; Kandel & Mednick, 1991). Recently, a study on individuals from low-income Philadelphia neighborhoods has suggested that current assessments of adverse childhood experiences may not adequately encompass the breadth of adversity experienced by low-income urban children and future research should consider a broader range of social environmental factors including single-parent homes, parental divorce and separation, and death and illness of family members (Wade, Shea, Rubin, & Wood, 2014). Thus, this



study aims to capture a better understanding of the impact of childhood adversity on antisocial behavior by broadening the range of experiences assessed and thus enhance content validity.

Responses to the demographic items were recoded into dichotomous variables: 0 (not adverse) or 1 (socially adverse) based on the respective frequencies for responses to each item. Thus, for each adversity indicator, one point was given. A total social adversity score for each subject was calculated by summing their scores according to their responses on the 18 items. Higher scores on the index reflected greater social adversity. For this scale only, any missing responses were dealt with by a simple imputation procedure to predict social adversity scores for cases with missing values.

### **Procedures for Rest and Stress Conditions**

During the 2-minute rest phase, the child was left in the laboratory assessment room and directed to sit still without moving their hand while looking at an “X” on the computer screen placed 1 meter in front of them. Following the rest phase, an experimenter entered the lab assessment room and a 4-minute social stressor task was administered. The stress task is similar to the speech task in the Trier Social Stress Test, which is considered the most widely used psychosocial stress protocol in laboratory studies (Kirschbaum, 2010; Kirschbaum, Pirke, & Hellhammer, 1993). A video recorder was set up in the room to increase the level of stress experienced. Subjects were then instructed to face the computer monitor for 2 minutes thinking about the worst or most stressful event that had ever happened to them before having to deliver a 2-minute speech in front of the camera, describing the event to the researcher. They were notified of a

countdown from 120 to 0 that flashed on the screen counting down their speech duration and that they would be evaluated on the quality of their speech. When the countdown reached 0, the children described the event to the researcher for 2 minutes, including what the experience was and how they reacted to it. Stress-inducing prompts urging the subjects to continue the story were occasionally given if they paused during the speech task.

### **Heart Rate Recording and Scoring**

Heart rate data were acquired using a Biopac MP150 data acquisition system, with the signal amplified using a Biopac 100C biopotential amplifier. Data were recorded using a bandpass of 0.5–35 Hz and a 60-Hz notch filter, with the signal digitized at 1,000 Hz. Biopac silver/silver chloride adhesive disposable electrodes were attached to each child on the left and right ribs at the level of the heart during the rest phase and social stressor task. This recording site was chosen to help reduce motion artifact. Skin areas in contact with the electrodes were cleaned with NuPrep abrasive skin prepping paste to lower skin impedance below 10 k $\Omega$  to enhance signal-to-noise ratio and ensure the quality and accuracy of data collection (Farinha, Kellogg, Dickinson, & Davison, 2006). Biopac isotonic recording gel was used as the electrolyte medium and impedance was monitored using a UFI Checktrode impedance meter (Morro Bay, CA).

Data were analyzed using *AcqKnowledge* version 4.1 software (Biopac Systems, Inc., Goleta, CA) and *AcqKnowledge* analytic tools were used to manually clean the data for artifacts. Heart rate levels in beats/minute were calculated using custom scripts in MATLAB. Average heart rate levels were obtained for the resting task, preparation phase

of the stress task, and speech task, with each measure based on a 2-minute sample. Heart rate during the speech preparation period was the focus of analyses for the stress measure as this quiescent task period has minimal movement artifacts. Furthermore, the later speech portion has been found in speech tasks to produce erratic respiratory patterns that impact cardiovascular recordings (Beda, Jandre, Phillips, Giannella-Neto, & Simpson, 2007).

### **Statistical Analyses**

Although zero-order associations are no longer required to exist between the independent, mediating, and dependent variables to establish mediation (Baron & Kenny, 1986; Zhao, Lynch, & Chen, 2010), Pearson correlations were conducted to examine the bivariate associations between heart rate, the antisocial behavior constructs, and social adversity as part of a preliminary analysis for completeness. Using the PROCESS macro, a bootstrapping approach was adopted to test the significance of indirect effects by taking 10,000 bootstrapped samples from the original data set (Hayes, 2013). The bootstrapping procedure is more rigorous and powerful than the Sobel test in that it provides better estimates of standard errors to identify any mediation effect and makes fewer assumptions about the shape of the sampling distribution of the indirect effect (Hayes, 2013; Sobel, 1982). Bootstrapping has also been recommended over the Sobel test, as it has higher power while maintaining reasonable control over the Type I error rate (Preacher & Hayes, 2008).

Additionally, Lin's (1989) concordance correlation coefficients were calculated to assess cross-informant agreement. A two-step regression was also conducted to assess the

extent of mediation by determining the reduction in variance in antisocial behavior explained by social adversity after controlling for heart rate. Statistical analyses were conducted using SPSS version 21.0 (IBM Corp, 2012).

### **Multiple Informants**

Prior studies have found that levels of agreement between caregivers and children reports of antisocial behavior are low to moderate, ranging from .10 to .45 (Achenbach et al., 1987; Baker et al., 2007; Loeber, Green, Lahey, & Stouthamer-Loeber, 1991; Shakoor et al., 2011). In light of similar findings in the present study (Lin's concordance correlation,  $R_c = .17$  to  $.38$ ) and Sullivan and McGloin's (2014) review encouraging the use of different sources of measurement in criminological studies, a more holistic picture is provided by using a composite of the parent and child ratings for all antisocial behavior measures in the analyses. For the Conduct Disorder and Oppositional Defiant Disorder Questionnaire and APSD, the higher of the two ratings from the two informants were taken for each item. This method of combining information from multiple informants has been adopted and recommended by other research groups, as it is deemed more encompassing (Barry, Frick, & Killian, 2003; Frick & Hare, 2001).

## **Results**

### **Heart Rate-Social Adversity-Antisocial Behavior Interrelationships**

**Combined parent and child ratings.** Lower heart rate was associated with higher scores for eight of the nine combined parent-reported and child-reported antisocial behavior measures (Table 1.1). Heart rate was not associated with the non-aggressive

aspect of conduct disorder. In addition, correlational analyses of the combined parent and child ratings indicated that greater social adversity was associated with lower heart rate and higher antisocial behavior scores.

**Parent ratings.** The heart rate-antisocial behavior and social adversity-antisocial behavior relationships were stronger for parent-reported data compared to child self-reported data (see Appendix C for full details). Similar to the results from combined parent and child ratings, heart rate was associated with eight of the nine parent-reported antisocial behavior measures. Among the subscales of conduct disorder and child psychopathy, it is notable that heart rate at rest and during stress was most strongly associated with the impulsivity dimension of psychopathy.

**Child ratings.** For child-reported data, heart rate was only marginally associated with the overall measure of antisocial behavior. Although not significant, relationships between heart rate and the other antisocial behavior measures were in the expected negative direction.

### **Mediation Effects**

**Combined parent and child ratings.** Mediation models were tested in which heart rate during stress served as a mediator of the social adversity-antisocial behavior relationship (Table 1.2, Figure 1.1). Bootstrapping analysis revealed that the indirect effect of social adversity via heart rate on combined parent-reported and child-reported general antisocial behavior was significant (indirect effect = .02,  $p < .01$ ). More

specifically, controlling for low heart rate reduced the percentage of variance in general antisocial behavior explained by social adversity by 9.27%. Testing other mediation models revealed that low heart rate mediated the social adversity-delinquency (indirect effect = .10,  $p < .01$ ) and adversity-child psychopathy (indirect effect = .04,  $p < .05$ ) relationships. Low heart rate marginally mediated the adversity-conduct disorder relationship (indirect effect = .01,  $p < .10$ ). Of the three forms of antisocial behavior, adding heart rate to the mediation model reduced the percentage of variance explained by social adversity most for the outcome of delinquency (19%).

Heart rate also served as a mediator of the relationship between social adversity and the aggressive factor of conduct disorder (indirect effect = .01,  $p < .05$ ) as well as the callous-unemotional (indirect effect = .01,  $p < .01$ ) and impulsivity subscales of the APSD (indirect effect = .01,  $p < .05$ ). The mediated effect of heart rate explained 7.14%, 12.40%, and 14.52% of the direct effect of adversity on aggression in conduct disorder, callous-unemotional traits, and impulsivity respectively. Resting heart rate was also tested as a possible mediator in the mediation models. However, no significant mediation effects of low resting heart rate were found.

**Parent ratings.** Similar mediation effects of low heart rate at stress were found for parent-reported data (Table 1.2, Figure 1.2); 20.35% of the effect of social adversity on delinquency was explained by the mediation effect of heart rate (indirect effect = .07,  $p < .01$ ). Including heart rate in the mediation model also reduced the percentage of variance in general antisocial behavior (indirect effect = .02,  $p < .01$ ), conduct disorder

(indirect effect = .01,  $p < .05$ ), and child psychopathy (indirect effect = .05,  $p < .01$ ) explained by social adversity by 13.38%, 6.03%, and 15.57%, respectively.

Similarly, there was a 10.68%, 8.33%, 10.39%, and 38.46% reduction in the percentage of variance in the aggressive form of conduct disorder (indirect effect = .01,  $p < .05$ ), callous-unemotional traits (indirect effect = .01,  $p < .05$ ), narcissism (indirect effect = .01,  $p < .05$ ), and impulsivity (indirect effect = .02,  $p < .01$ ) explained by social adversity after controlling for heart rate, respectively. No significant mediation effects of low resting heart rate were observed.

**Child ratings.** Regarding the child-reported antisocial behavior measures, bootstrapping analyses found that the mediation effect of low heart rate did not approach significance. Furthermore, no significant mediation effects of low resting heart rate were observed.

### **Potential Confounds**

After controlling for age and gender in the mediation models, results remained substantively unchanged. Low heart rate significantly mediated the relationship between social adversity and general antisocial behavior measured using parent ratings (indirect effect = .01,  $p < .05$ ) and combined parent and child ratings (indirect effect = .01,  $p < .05$ ).

## **Discussion**

The objective of this study was to test whether a biological mechanism may help explain why social adversity relates to antisocial behavior. Results supported the hypothesis that low heart rate partly mediates this relationship. Heart rate on average explained 10.79% (range: 3.60 - 19.00%) and 15.40% (range: 6.03 - 38.46%) of the effect of social adversity on antisocial behavior for combined parent and child ratings and parent-reported ratings, respectively. The strongest mediation effects for parent-reported delinquency (20.35%) and impulsivity in child psychopathy (38.46%) are notable. Low heart rate was also associated with higher child psychopathy scores, extending the existing literature on the heart rate-antisocial behavior link. To the authors' knowledge, this study is the first to use mediation models to document a psychophysiological risk factor as a mediator of the adversity-antisocial behavior relationship in a child or adult sample. This initial empirical finding gives rise to a social neurocriminological theoretical account of crime and delinquency that directs attention to social influences that sculpt biological functions in a way to shape antisocial and criminal behavior.

### **Mediation Mechanism**

Although controlling for heart rate during stress did not by any means completely reduce the variance in antisocial behavior explained by social adversity, findings suggest that social adversity experienced early in life influences the biological risk factor of heart rate at least to a certain extent, which in turn is a robust (albeit modest) correlate of general antisocial behavior, delinquency, conduct disorder, and child psychopathy. However, the precise pathway linking the macro-level variable of social adversity to



heart rate remains unclear. A possible explanation for this finding may be the downregulation of heart rate due to exposure to stress. Social disadvantage is associated with higher levels of stress (Goodman, McEwen, Dolan, Schafer-Kalkhoff, & Adler, 2005). While children who have experienced less adversity may find the social stress test particularly stressful and in response have higher heart rates, children who have experienced more chronic stress from adversity at a young age may have habituated to life stress, such that the laboratory stress test does not evoke the same level of autonomic reactivity. Effectively, sustained exposure to socially adverse conditions may “inoculate” some children from stress by downregulating their autonomic stress reactivity functioning, predisposing them to fearlessness and a proclivity to antisocial behavior. This is consistent with findings of blunted cortisol responses (another biological measure of stress reactivity) only among individuals who had experienced adverse childhood events compared to individuals who had not (Elzinga et al., 2008). Regardless of the mechanism, this study documents an association between two salient and well-replicated social and biological risk factors for antisocial behavior – increased social adversity and low heart rate. Future tests of this fearlessness inoculation hypothesis could further delineate the mechanism of action.

Null results for the mediation effect of resting heart rate may also shed some light on the mechanism underlying another relationship in the mediation model, that between heart rate and antisocial behavior. As significant mediation effects were only observed for heart rate during the stress task, which unlike the rest condition invokes stress, there is some support for the fearlessness theory in relating low heart rate to antisocial behavior. This finding is broadly consistent with that of other researchers documenting support for

the fearlessness theory (Armstrong & Boutwell, 2012), although given findings discussed subsequently, other theoretical interpretations are possible.

### **Low Heart Rate in Relation to Impulsivity and Child Psychopathy**

In contrast to the above-mentioned support for fearlessness theory, the finding that low heart rate is particularly associated with the impulsivity dimension of child psychopathy from the parent-reported scales ( $d = -.37, p < .01$  for heart rate at stress;  $d = -.28, p < .01$  for heart rate at rest) provides support for the alternative impulsive sensation-seeking theory in the criminological literature. As a close link exists between impulsivity and sensation seeking (Zuckerman, 1993), the results reinforce prior empirical findings that impulsive sensation seeking rather than fearlessness mediates the relationship between heart rate and antisocial behavior (e.g., Portnoy et al., 2014b). We caution, however, that we did not employ any measure of “fearless antisocial behavior” alongside our measure of impulsive antisocial behavior to test between these competing theories and consequently the current findings can only provide equivocal support for each theoretical perspective that in turn require further empirical test.

The finding that heart rate is most strongly associated with impulsivity also has implications for other criminological theories. Documenting that low heart rate was associated with impulsivity, which in turn is associated with lack of self-control, supports Gottfredson and Hirschi’s (1990) self-control theory of delinquency that proposes that low self-control constitutes the most important causal factor in explaining antisocial behavior. Furthermore, findings relate to DeLisi and Vaughn’s (2014) temperament-based theory that suggests that temperament, namely, reduced effortful control and

negative emotionality, produces antisocial conduct throughout life, and explains failure to interact with criminal justice practitioners and complete correctional sentences. Effortful control is related to impulsivity and self-control because it partly represents the ability to suppress a dominant response to perform a subdominant response (Rothbart, Ellis, Rueda, & Posner, 2003). Thus, findings on the heart rate-impulsivity relationship provide empirical support for temperament-based criminological theories.

Importantly, this study also advances prior findings on the heart rate-child psychopathy relationship. Results are consistent with notions that psychopathic traits are related to low autonomic system arousal and demonstrate that the negative association between heart rate and child psychopathy replicates across cultures (East Asian and Western) and with the use of different measures of psychopathy (Baker et al., 2009; Raine et al., 2014). This is significant, as child psychopathy has downstream effects in predisposing to adult offending.

### **Informant Differences**

As in many previous studies, only low-level correlations were found between parent and child-reported behavioral constructs (e.g., Baker et al., 2007). Having parent-reported data in addition to self-reported data has utility since behavior symptoms such as conduct disorder symptoms tend to be more commonly reported by parents than their children (Angold et al., 1987; Edelbrock, Costello, Dulcan, Conover, & Kala, 1986) and different etiologies may exist for scales derived from different informants (Baker et al., 2007). In this study, significant heart rate-antisocial behavior associations and mediation effects failed to be observed in child-reported data compared to the parent-reported data.

A possible explanation for the discrepancy in findings from the parent-reported and child-reported measures is that the data from the self-reported antisocial measures have lower construct validity. This is supported by the fact that compared to parent-reported ratings, the child-reported data were less strongly correlated with other variables that are theoretically expected to be associated with antisocial behavior, such as social adversity and heart rate. Indeed, historically, youth self-reports have been considered the least useful source of behavior ratings (Youngstrom, Loeber, & Stouthamer-Loeber, 2000).

Alternatively, consistent with findings of the lowest levels of agreement among caregivers and children for impulsivity scores, it is suggested that children are very likely more impulsive than their caregivers (Steinberg et al., 2008). Impulsive children may consequently make more errors in their self-report antisocial behavior questionnaires, rendering the child-reported data less valid. In this study, the use of multiple informants can be viewed as a methodological strength in comparison to other studies using single informants, especially in light of the significant findings produced when combining parent and child data to measure antisocial behavior.

### **Theoretical Implications for Mainstream Criminology and Future Directions**

Although the current empirical findings are novel, they contribute to a social neurocriminology perspective that can potentially help move the discipline of criminology forward at a theoretical level. First, the current findings and approach can help sharpen existing criminological theories by explaining *why* environmental variables that are identified in many sociological theories of crime are associated with offending. Consistent with the integrative criminology approach (Henry & Einstadter, 2006), this

study provides a first step to advancing future theories by demonstrating empirical proof of concept, that social environmental factors can affect biological variables which in turn lead to antisocial behavior. Such proof of concept has not been empirically shown in criminology or other disciplines such as psychology, sociology, and pediatrics for antisocial and criminal behavior in particular.

Second, social neurocriminology places neurobiological influences into a social context to explain antisocial behavior and promotes greater consideration of the role that social environmental variables play in affecting biological processes in future biosocial research and theory. One example of how a social neurocriminological perspective can complement and contribute to traditional criminological theory is through Gottfredson and Hirschi's (1990) self-control theory of delinquency that argues that social processes predispose to impulsivity and low social control which in turn predisposes to crime. Social neurocriminology invokes into this social theoretical framework a biological variable – hypothesizing that ineffective socialization and lack of nurturance predisposes to low heart rate which in turn predisposes to the lack of self-control that leads to delinquency and crime. The social environment nevertheless remains, at least in theory, at the starting point of this cascade toward crime.

Three important caveats are required to provide an appropriate, scientific balance to the introduction of social neurocriminology theory. First, social neurocriminology places social influences at the forefront of etiological processes that shape crime – a flow from social influences to biological influences to criminal behavior. It aims to provide social scientists added traction in their work and to encourage them to incorporate biological constructs into future studies, thus potentiating the explanatory power of their

revised theories. This is especially possible, as heart rate is a noninvasive, cheap, and relatively quick measure of autonomic nervous system-regulatory activity with replicable relationships to child antisocial behavior and adult offending. Nonetheless, research on other potential biological mediators of the social adversity-antisocial behavior relationship, including genetic, hormonal, and brain imaging factors need to be examined in the future. Studies have indicated that disadvantaged neighborhoods affect cognitive ability which is in turn associated with antisocial behavior (McGloin & Pratt, 2003; Sampson, Sharkey, & Raudenbush, 2008; Sharkey, 2010). Therefore, it would be valuable to examine possible neurocognitive factors that may mediate the link between negative social environments and antisocial outcomes. Second, social neurocriminology does not negate the main premise of neurocriminology that genetic and biological influences can more directly predispose to social, behavioral, and affective risk factors for crime, although it does offer a different conceptual framework that makes competing predictions. Third, as a mediational model, it differs conceptually from moderation models that instead focus on interactions between social and biological variables (Barnes et al., 2014; Beaver et al., 2009; Burt & Simons, 2014) and thus provides a different perspective. As such, it can provide a framework for future theories to integrate concepts that are central to both biological theories of offending, as well as classical, sociological theories of crime that involve environmental variables (Agnew, 1992; Shaw & McKay, 1942).

## **Limitations**

There are several limitations in the current study. First, antisocial behavior was measured concurrently with heart rate at age 11. From a longitudinal standpoint, it remains to be seen whether social adversity early in life predisposes to low heart rate later in childhood which in turn is a risk factor for even later delinquency and adult crime.

Second, genetic influences on social adversity, heart rate, or antisocial behavior are not examined in this study. Research suggests that social environments can influence biological systems through gene expression (Burt & Simons, 2014; Champagne & Curley, 2005). Thus, genetic factors could contribute to the understanding of the how social adversity influences autonomic system functioning.

Third, it should be reemphasized that the mediation effect did not completely explain the effect of social adversity on antisocial behavior. The mediation effect of low heart rate on the social adversity-antisocial behavior relationship, although statistically significant, is small. Clearly, many other social and biological mechanisms likely play important mechanistic roles in forming this relationship.

Fourth, this study adopted a broad approach in examining social adversity as a general construct in relation to antisocial behavior. The use of a more comprehensive measure of social adversity that includes a number of variables regarding parents' health, poverty, as well as elements of breaks in family structure allowed a broad test of the relationship between the environmental risk factor of early adversity and antisocial behavior. The limitation is that this study does not delineate the precise social processes that give rise to antisocial behavior and low heart rate. It has been suggested that heart rate could serve a mediating function in the relationship between the specific environmental factor of supervision/control in education and antisocial behavior (Lösel &

Bender, 1997) and future studies could advance current knowledge by focusing on more specific aspects of social adversity. Additionally, the items in the social adversity index were not weighted. Nevertheless, the current study provides a starting point upon which other more detailed studies may build.

### **Conclusion**

In conclusion, this study documents that low heart rate reduced the percentage in variance of antisocial behavior explained by social adversity by up to 38.46%, with an average of 10.79% for combined parent and child ratings and 15.40% for parent-reported antisocial behavior measures. The effects are small but quite consistent with (a) the modest relationship between heart rate and antisocial behavior and (b) the conceptual reality that multiple processes likely help explain the relationship, including other biological risk factors for offending (Glenn & Raine, 2014). Demonstrating for the first time that low heart rate partly mediates the social adversity-antisocial behavior relationship in children gives rise to a social neurocriminological theoretical perspective that can potentially help stimulate future conceptual and theoretical developments in criminology.



**Paper 1 Tables and Figures**

Table 1.1. Bivariate Correlations for Heart Rate, Social Adversity, and Combined Parent-Reported and Child-Reported Antisocial Behavior Measures

	1	2	3	4	5	6	7	8	9	10	11	12
<i>Heart Rate</i>												
1. Stress	1.00	.83**	-.17**	-.17**	-.12*	-.13*	-.08	-.16**	-.17**	-.09 <sup>†</sup>	-.13*	-.13*
2. Rest		1.00	-.11*	-.11*	-.08	-.10 <sup>†</sup>	-.05	-.10 <sup>†</sup>	-.08	-.06	-.09 <sup>†</sup>	-.05
3. <i>General Antisocial</i>			1.00	.91**	.90**	.82**	.81**	.90**	.52**	.80**	.75**	.37**
4. <i>Delinquency</i>				1.00	.74**	.68**	.66**	.75**	.40**	.65**	.66**	.29**
5. <i>Conduct Disorder (Total)</i>					1.00	.92**	.90**	.69**	.35**	.64**	.59**	.37**
6. <i>Aggressive</i>						1.00	.66**	.62**	.29**	.59**	.53**	.32**
7. <i>Non-Aggressive</i>							1.00	.64**	.36**	.58**	.54**	.35**
8. <i>Child Psychopathy (Total)</i>								1.00	.65**	.88**	.77**	.35**
9. <i>Callous-Unemotional</i>									1.00	.35**	.23**	.33**
10. <i>Narcissism</i>										1.00	.60**	.28**
11. <i>Impulsivity</i>											1.00	.23**
12. <i>Social Adversity</i>												1.00
<i>Mean</i>	83.20	81.52		20.29	3.51	1.78	1.73	17.33	5.72	5.51	5.30	3.92
<i>Standard Deviation</i>	10.81	9.87		13.89	3.70	2.17	1.89	5.73	2.11	2.77	1.89	2.34

*Note.* Stress = Heart rate measured during the social stressor task; Rest = Heart rate measured at rest, General Antisocial = General measure of antisocial behavior; Aggressive = Aggressive subfactor of conduct disorder; Non-Aggressive = Non-aggressive subfactor of conduct disorder; Callous-Unemotional = Callous-unemotional dimension of child psychopathy; Narcissism = Narcissism dimension of child psychopathy; Impulsivity = Impulsivity dimension of child psychopathy.  
<sup>†</sup> $p < .10$ ; \* $p < .05$ ; \*\* $p < .01$ , two-tailed.

Table 1.2. Total, Direct, and Indirect Mediation Effects of Social Adversity on Antisocial Behavior Measures through Heart Rate

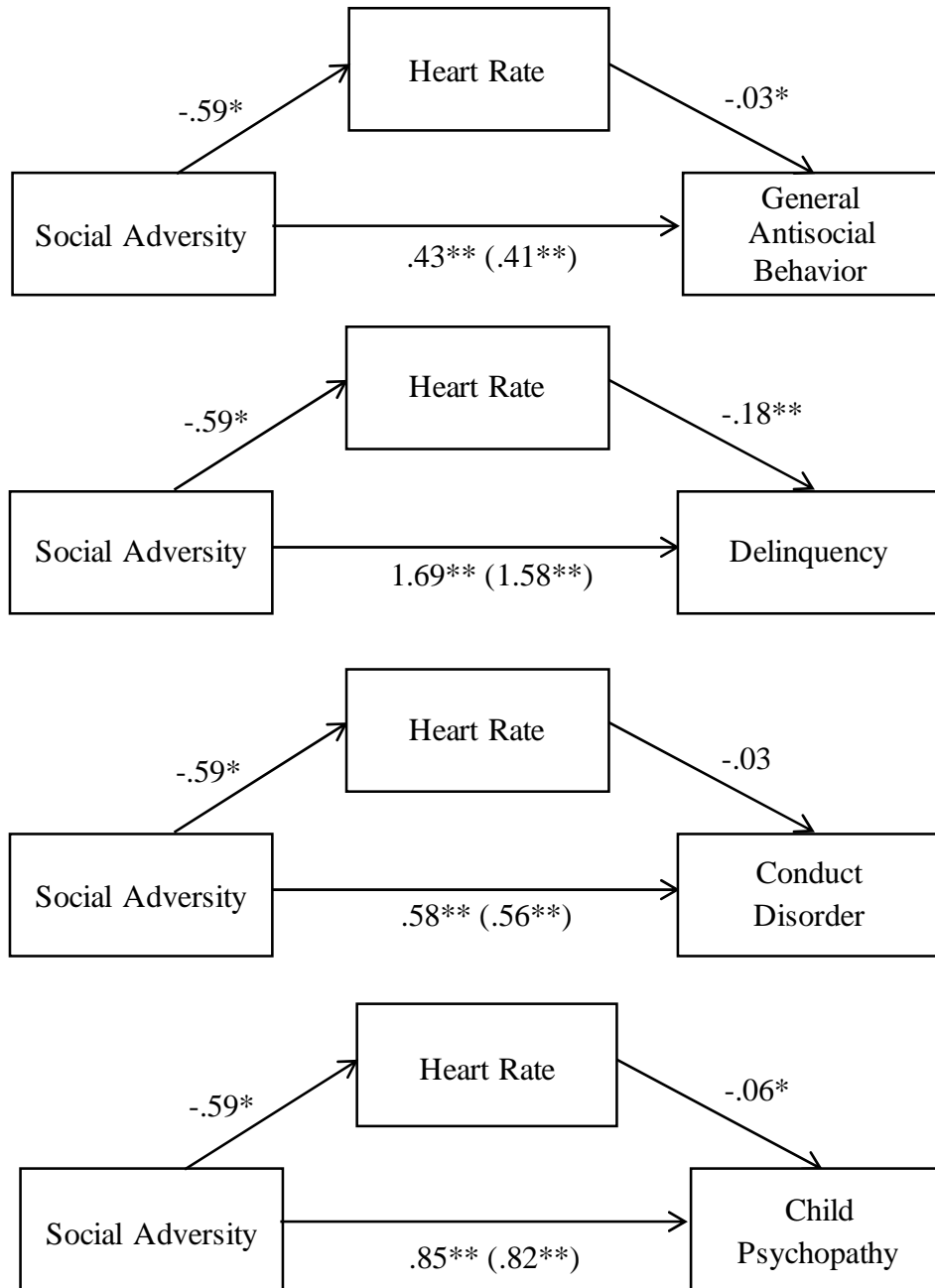
Outcomes	Total Effect: Social Adversity on Outcome		Direct Effect: Social Adversity on Outcome		Mediation Effect: Social Adversity on Outcome		
	B	SE	B	SE	B	SE	95% CI
<b>Parent and Child-Report</b>							
<i>General Antisocial</i>	.43**	.05	.41**	.05	.02**	.01	.004 - .044
<i>Delinquency</i>	1.69**	.29	1.58**	.29	.10**	.05	.025 - .234
<i>Conduct Disorder</i>	.58**	.07	.56**	.08	.01†	.01	-.001 - .048
Aggressive	.30**	.04	.29**	.05	.01*	.01	.001 - .030
Non-Aggressive	.28**	.04	.28**	.04	.004	.01	-.004 - .021
<i>Child Psychopathy</i>	.85**	.12	.82**	.12	.04*	.02	.006 - .090
Callous-Unemotional	.29**	.04	.28**	.04	.01**	.01	.003 - .036
Narcissism	.33**	.06	.32**	.06	.01	.01	-.004 - .030
Impulsivity	.19**	.04	.18**	.04	.01*	.01	.001 - .027
<b>Parent-Report</b>							
<i>General Antisocial</i>	.40**	.05	.38**	.05	.02**	.01	.005 - .046
<i>Delinquency</i>	1.14**	.18	1.07**	.18	.07**	.04	.019 - .167
<i>Conduct Disorder</i>	.40**	.06	.38**	.06	.01*	.01	.001 - .038
Aggressive	.22**	.04	.21**	.04	.01*	.01	.002 - .025
Non-Aggressive	.17**	.03	.17**	.03	.003	.004	-.003 - .014
<i>Child Psychopathy</i>	.85**	.13	.80**	.13	.05**	.02	.011 - .106
Callous-Unemotional	.29**	.05	.28**	.05	.01*	.01	.001 - .031

Narcissism	.33**	.06	.31**	.06	.01*	.01	.001 - .040
Impulsivity	.17**	.04	.16**	.04	.02**	.01	.004 - .040

*Note.* Heart Rate = Heart Rate measured during the social stressor task; General Antisocial = General measure of antisocial behavior; Callous-Unemotional = Callous-unemotional dimension of child psychopathy; Narcissism = Narcissism dimension of child psychopathy; Impulsivity = Impulsivity dimension of child psychopathy; B = unstandardized coefficient, SE = standard error; CI = confidence interval.

† $p < .10$ ; \* $p < .05$ ; \*\* $p < .01$ , two-tailed.

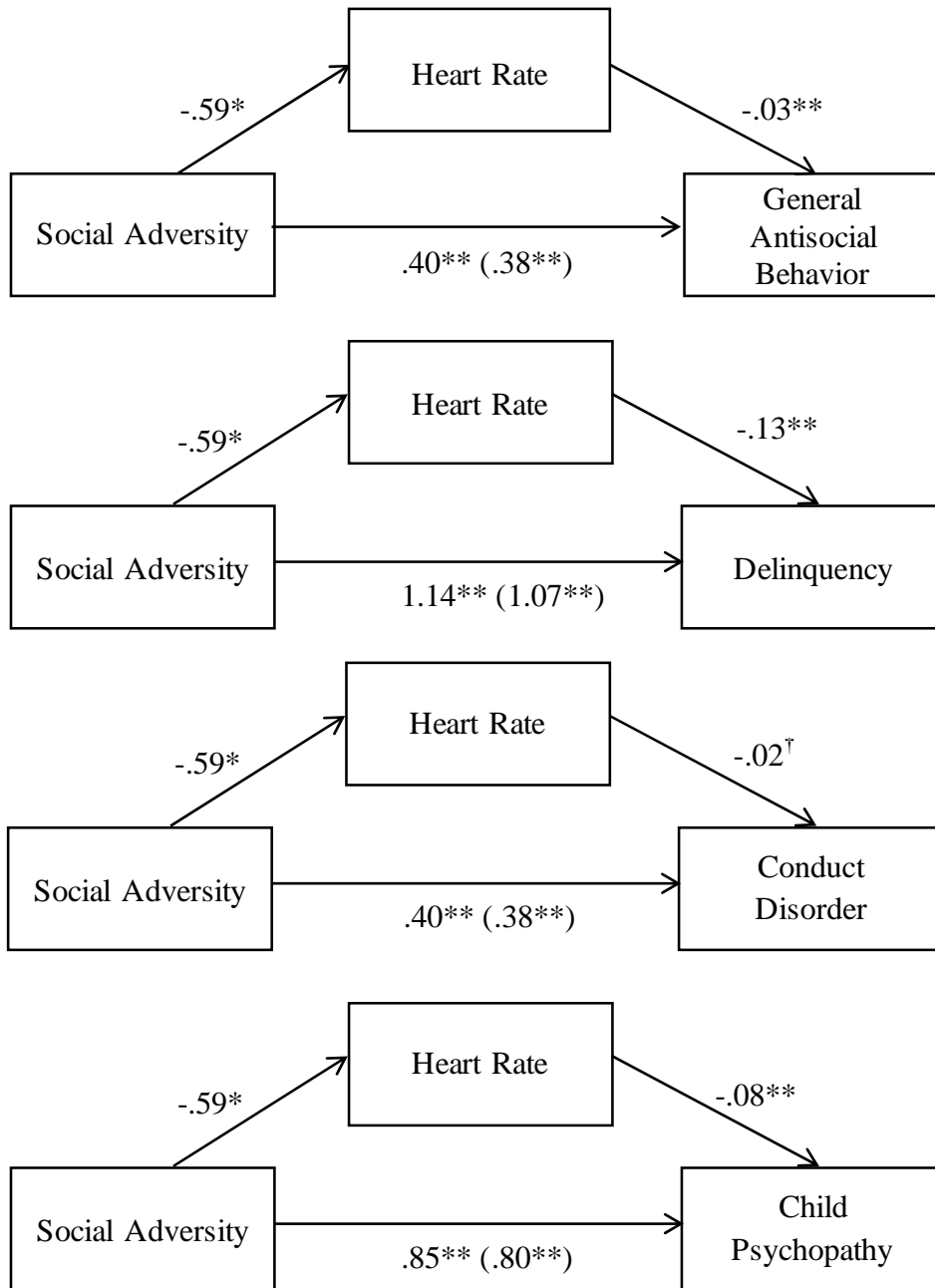
Figure 1.1. Mediation Models for Parent-Child Combined Measures of General Antisocial Behavior, Delinquency, Conduct Disorder, and Child Psychopathy



*Note.* The path coefficients are unstandardized. Values in parentheses indicate the direct effect of the independent variable, social adversity, on the antisocial behavior measures. Heart Rate = Heart rate during stress.

\* $p < .05$ ; \*\* $p < .01$

Figure 1.2. Mediation Models for Parent-Reported Antisocial Behavior of General Antisocial Behavior, Delinquency, Conduct Disorder, and Child Psychopathy



*Note.* The path coefficients are unstandardized. Values in parentheses indicate the direct effect of the independent variable, social adversity, on the antisocial behavior measures. Heart Rate = Heart rate during stress.

$^\dagger p < .10$ ;  $*p < .05$ ;  $**p < .01$ .

## **PAPER 2. THE NEED TO INCORPORATE AUTONOMIC AROUSAL IN DEVELOPMENTAL AND LIFE-COURSE RESEARCH AND THEORIES**

### **Abstract**

Although there is an extensive body of research on the relationship between autonomic measures of physiological arousal and crime and delinquency, little effort has been made to incorporate autonomic arousal into criminological theories. This article examines the empirical literature on the associations between autonomic arousal and key constructs that are included in developmental and life-course theories, including temperament, cognition, life events, and family factors. For an illustrative approach, this article specifically focuses on two measures of physiological arousal, heart rate and skin conductance. Salient features of the literature include empirical findings that suggest that social factors can affect autonomic functioning and that autonomic arousal levels can contribute to individual differences in psychological risk factors for offending. A biopsychosocial model is proposed to show how autonomic arousal can be incorporated into extant criminological theories. If autonomic measures of arousal are included in future developmental and life-course research, the explanation and prediction of offending will be increased.

## **Background**

Since the revival of biological explanations in criminology in the late twentieth century, criminology has developed from a social science-focused discipline to one that is increasingly aware of the contributions of biological sciences. For example, a substantial body of empirical research exists documenting biological factors as risk and protective factors for crime and delinquency (Boutwell et al., 2014; Gao et al., 2011; Grisso, 2007; Miller, 2006; Portnoy, Chen, & Raine, 2013; van Goozen, Fairchild, Snoek, & Harold, 2007; Yang et al., 2011). In light of the relationship between biology and crime, some researchers have also begun to consider how biological factors might be incorporated in criminological theories such as Agnew's general strain theory (Walsh, 2000), rational choice theory (Armstrong & Boutwell, 2012), Moffitt's developmental taxonomy (Barnes, 2013; Barnes, Beaver, & Boutwell, 2011; Boutwell, Barnes, Deaton, & Beaver, 2013; Boutwell & Beaver, 2008; Moffitt & Caspi, 2001), and Lahey and Waldman's (2005) developmental model of the propensity to offend in children and adolescents.

Despite evidence that criminology is becoming more biologically informed, the extent to which biological variables are included differs across research and theories (Wright & Boisvert, 2009). Biological studies tend to be viewed as having empirical merit in documenting a putative risk factor for antisocial behavior, but many theories still fail to incorporate biological influences or explain how such factors relate to antisocial behavior (Armstrong & Boutwell, 2012; Walsh, 2000; Wilson & Scarpa, 2012). This failure to place biological factors in a theoretical context can limit the explanatory power of current criminological theories.

This article aims to address the conceptual gap between biology and criminology by examining the relationship between autonomic arousal and four social and psychological constructs that underlie developmental and life-course (DLC) theories. As DLC theories have been claimed to be more wide-ranging and encompassing than classic criminological theories, the usefulness of autonomic factors to DLC theories is the focus of this review (Farrington, 2005a).

First, we describe the role of autonomic measures of physiological arousal as possible causes of delinquent and criminal behavior, including a broad description of heart rate and skin conductance responses. The second section introduces a model through which autonomic arousal can be incorporated into DLC theories. Third, the empirical literature on the relationships between autonomic arousal and psychological and social constructs underlying DLC theories is reviewed and examples of how autonomic arousal can be incorporated into DLC theories are given. Fourth and last, we address future directions and challenges for the inclusion of autonomic measures in criminology.

### **Autonomic Arousal**

The concept of an optimal level of arousal has contributed significantly to the contemporary understanding of biology in criminology (Eysenck, 1967; Hebb, 1955; Rafter, 2006; Zuckerman, 1991). Eysenck's arousal-based personality theory and Trasler's learning theory proposed that arousal rates are related to crime because conscience consists of a set of classically conditioned emotional responses to adverse environments (Eysenck, 1977; Trasler, 1962). Lower levels of arousal cause poorer



conditioning to environmental stimuli. It is assumed that the strength of the conscience depends on passive avoidance conditioning by parents. Impaired conditioning may result in the absence of or decrease in anticipatory anxiety regarding the consequences of a criminal or antisocial act, which causes a weak conscience. This in turn makes individuals less restrained and predisposes them to crime and antisocial behavior. Individuals with lower levels of arousal may engage in crime to raise their arousal to an optimal level. Aspects of Eysenck's theory have influenced research on biological factors in offending. Empirical studies have documented that autonomic underarousal is associated with crime and delinquency (Cornet, de Kogel, Nijman, Raine, & van der Laan, 2013; Farrington, 1997; Jennings, Piquero, & Farrington, 2013; Lorber, 2004; Ortiz & Raine, 2004).

One index of arousal is autonomic nervous system activity. The autonomic nervous system is a subdivision of the peripheral nervous system that regulates involuntary functions (Brodal, 2004). It comprises of the sympathetic nervous system that is associated with the "fight or flight" response and the parasympathetic nervous system, which is associated with regulatory activity (Beauchaine, 2001; Gilissen, Koolstra, van Ijzendoorn, Bakermans-Kranenburg, & van der Veer, 2007). Specifically, psychophysiological measures are emphasized in this review. They constitute a valid method to determine arousal as physiological responses are universal, sensitive, and objective (Lorber, 2004). They have been used in prospective studies on criminal and antisocial outcomes (Baker et al., 2009; Jennings et al., 2013; Lösel & Bender, 1997; Raine, 2002; Wilson & Scarpa, 2012). Psychophysiology is particularly useful in studying biological influences as specific measures have been identified. In contrast, in

disciplines such as behavioral genetics, with the exception of the monoamine oxidase A (MAOA) gene, no single gene has been robustly identified as associated with crime and delinquency (Beaver, DeLisi, Vaughn, & Barnes, 2010; Beaver, DeLisi, Vaughn, & Wright, 2010; Brunner, Nelen, Breakefield, Ropers, & Van Oost, 1993; Caspi et al., 2002; Fergusson, Boden, Horwood, Miller, & Kennedy, 2011; Goldman & Ducci, 2007; Taylor & Kim-Cohen, 2007). As cardiovascular functioning and electrodermal activity serve as the most popular measures of physiological response, this review is limited to these factors.

### **Heart Rate**

Heart rate is influenced by the sympathetic and parasympathetic branches of the autonomic nervous system (Boucsein, 1992). It is a particularly apt biological variable to assess in relation to crime, as low heart rate is a robust, well-replicated correlate of constructs relevant to criminal behavior (Ellis, Beaver, & Wright, 2009; Scarpa, Fikretoglu, & Luscher, 2000). Studies have revealed significant associations between low resting heart rate and other undesirable outcomes related to crime and delinquency such as antisocial behavior in children and adolescents and conduct problems (e.g., Lorber, 2004; Ortiz & Raine, 2004; Portnoy & Farrington, 2015). A meta-analysis has also documented resting heart rate to be associated with both reactive and proactive aggression (Portnoy & Farrington, 2015), although a recent study found that the association between resting heart rate and aggression was specific to proactive aggression after controlling for reactive aggression (Raine, Fung, Portnoy, Choy, & Spring, 2014). Moreover, there is some evidence that low heart rate may be associated particularly with

non-psychopathic forms of antisocial behavior (e.g., Baker et al., 2009; Portnoy et al., 2014b; Raine et al., 2014).

Similar relationships were observed when heart rate was assessed during a stressor (Ortiz & Raine, 2004). The association between low heart rate and criminal and antisocial behavior has been found across different species of animals, cultures, and genders (Cherkovich & Tatoyan, 1973; Eisermann, 1992; Lorber, 2004; Moffitt, Caspi, Rutter, & Silva, 2001; Ortiz & Raine, 2004; Raine, 2002) and independently of potential confounds such as height, weight, smoking, and physical exercise (Armstrong, Keller, Franklin, & MacMillan, 2009; Cauffman, Steinberg, & Piquero, 2005; Jennings et al., 2013). Furthermore, heart rate is diagnostically specific, as low resting heart rate is associated with conduct disorder, but not with other psychiatric conditions (Raine, 2002).

There are two major theoretical interpretations of underarousal in delinquent and criminal individuals. The stimulation-seeking theory posits that underarousal prompts individuals to commit more antisocial acts as they seek stimulation to raise their arousal levels to an optimal level, while the fearlessness theory proposes that low levels of arousal index a low level of fear in individuals who may then be more likely to engage in antisocial behaviors that require a degree of fearlessness to execute (Armstrong et al., 2009; Quay, 1965; Raine, 2002; Venables, 1987). In an empirical study, Armstrong and Boutwell (2012) concluded that fearlessness, measured by how individuals perceive the costs and benefits of offending, is a mechanism linking low heart rate to antisocial behavior. However, studies by Sijtsema et al. (2010) and Portnoy et al. (2014b) found that impulsive sensation-seeking, rather than fearlessness, partially mediates the heart rate-antisocial behavior association. Thus, the underlying mechanism is unclear.

## **Skin Conductance**

Unlike heart rate, electrodermal activity is regulated exclusively by the sympathetic nervous system (Boucsein, 1992). The skin conductance level and number of nonspecific skin conductance responses measure electrical conductivity at the surface of the skin (Fowles, Kochanska, & Murray, 2000; Kroeber-Riel, 1979). Empirical studies have documented a negative relationship between skin conductance and criminal behavior and delinquency (Ellis et al., 2009; Gatzke-Kopp, Raine, Loeber, Stouthamer-Loeber, & Steinhauer, 2002; Kruesi et al., 1992; Raine, Venables, & Williams, 1990). A similar pattern of results has been observed for other types of antisocial behavior. Low resting electrodermal activity is associated with higher levels of adult psychopathy and aggressive behavior in children, specifically reactive aggression, while lower skin conductance measured during a task is associated with increased conduct problems and psychopathy in adolescents and adults (Lorber, 2004; Posthumus, Böcker, Raaijmakers, Van Engeland, & Matthys, 2009; Scarpa & Haden, 2006).

## **Integrating Measures of Autonomic Arousal into DLC Theories**

### **Biopsychosocial Model of Crime and Delinquency**

A biopsychosocial mediation model is proposed to conceptualize how biological characteristics fit into theories of delinquent and criminal behavior (Figure 2.1). Variations of this mediation model have been suggested by other researchers. For example, the social neurocriminology perspective proposes that the social environment influences biological functions, in part, to predispose to antisocial and criminal behavior

(Choy et al., 2015). In another model, van Goozen et al. (2007) proposed that family factors influence antisocial behavior through biological, cognitive, and emotional mechanisms, while DeLisi and Vaughn (2014) presented a model in which genotypes and neural substrates influence temperament to lead to problem behavior outcomes, all of which can be affected by contextual factors. This article builds on prior literature by (a) presenting a more encompassing framework for crime and delinquency that includes social environmental, biological, and psychological variables; (b) applying a biopsychosocial model to DLC research and theories; and (c) examining the biological factor of autonomic arousal.

In this model, social factors are hypothesized to lead to changes in biological functioning to some extent. Biological factors in turn lead to psychological risk factors that influence criminal and delinquent behavior. Such an approach is consistent with the notion that social causes of crime must act through psychological pathways (Eysenck, 1996), that psychological processes originate from physiological ones (Kroeber-Riel, 1979), and that models of criminal behavior should involve the integration of biological, psychological, and environmental characteristics (Armstrong & Boutwell, 2012; DeLisi, Beaver, Wright, & Vaughn, 2008; Schug & Fradella, 2014; van Goozen et al., 2007).

### **Psychological Risk Factors**

**Temperament.** Temperament is the stable tendency by which an individual experiences and emotionally responds to the environment (DeLisi & Vaughn, 2014; Walsh, 2000). While many categories of temperament exist (Walters, 2011), four

dimensions of temperament are highlighted in the criminological literature, namely effortful control, daring, empathy, and fear.

*Effortful control.* Effortful control is a key feature of self-regulation. It is closely related to impulsivity and is often referred to as inhibitory control or self-control in developmental contexts (DeLisi & Vaughn, 2014; Fowles et al., 2000). It is the ability to suppress a dominant response in order to perform a subdominant one, as well as to modulate and maintain emotions, behaviors, and physiological reactions to achieve a goal (Cole, Martin, & Dennis, 2004; Rothbart & Bates, 2006; Rothbart, Ellis, Rueda, & Posner, 2003; Wilson, Lengua, Tininenko, Taylor, & Trancik, 2009; Wolfe & Bell, 2004).

Studies have shown that lower arousal is associated with lower levels of effortful control. Lower resting heart rates were associated with faster responses in a risk game, indicating that autonomic underarousal is linked to impulsivity (Schmidt, Mussel, & Hewig, 2013). Similarly, Mathias and Stanford (2003) found that underarousal was associated with individuals' ratings of impulsivity. Highly impulsive adults exhibited lower resting heart rates compared to those with normal scores on the impulsivity scale. This is further supported by a recent finding that low resting heart rate was significantly associated with higher impulsivity scores in children, controlling for age, sex, and other traits associated with child psychopathy (Raine et al., 2014).

Similarly, arousal during task periods has been assessed in relation to effortful control. Higher skin conductance levels measured during tasks were associated with higher effortful control ratings in children at age 4 years (Fowles et al., 2000). In a study

involving an arousal challenge of different levels of intensity, more impulsive individuals had significantly lower heart rates than those with a normal range of impulsivity scores during the most challenging three of the four arousal tasks (Mathias & Stanford, 2003). In addition, research on psychopathy, of which impulsivity is a component, has documented that psychopaths show lower electrodermal activity in the face of a stimulus (Lykken, 1995; Zahn, 1986), providing support for the positive relationship between autonomic arousal and effortful control.

***Daring.*** The daring dimension of temperament refers to a style of reacting in the face of novel events (Fox, Henderson, Marshall, Nichols, & Ghera, 2005; Richards & Cameron, 1989). It is characterized by a tendency towards sensation-seeking and novelty-seeking and has been regarded as the inverse of behavioral inhibition (Kagan, Snidman, & Arcus, 1998; Lahey & Waldman, 2003; Lahey & Waldman, 2005).

Low resting heart rate has been linked to lower inhibition or high risk-taking behavior in children and adolescents (Portnoy et al., 2014b; Reznick et al., 1986; Sijtsema et al., 2010) as well as adults (De Pascalis, Valerio, Santoro, & Cacace, 2007; Mawson, 2009; Schmidt et al., 2013), even after controlling for a number of variables. A similar pattern of results has been found for arousal measured during a task. Greater heart rate levels were observed in inhibited compared to uninhibited children during a cognitive task (Reznick et al., 1986). In another study, participants' behaviors were analyzed during a control condition and after experiencing an arousal condition comprising of acute physical exercise that elevated their heart rate levels (Schmidt et al., 2013). Participants were more likely to choose the safer option in a gambling task in the

arousal condition compared to the control condition. The positive association between heart rate and behavioral inhibition has also been observed longitudinally (Kagan, Reznick, Clarke, Snidman, & Garcia-Coll, 1984). Although evidence of a low arousal-high sensation seeking relationship is less consistent in females (Wilson & Scarpa, 2013; Wilson & Scarpa, 2014) and for the measure of skin conductance (De Pascalis et al., 2007), empirical studies have found substantial support for the relationship between low autonomic arousal and a more daring temperament.

*Empathy.* A third dimension of temperament, empathy, refers to concern for the feelings of others (Lahey & Waldman, 2005). Zahn-Waxler, Cole, Welsh, and Fox (1995) found that higher heart rates, measured both during a sadness mood induction period and a non-mood induction period, were associated with greater empathic concern, emotional arousal, and prosocial behavior. On the other hand, lower heart rates were consistently associated with avoidance and joy during another's distress. Findings from more recent studies support these conclusions, as lower heart rate was strongly and consistently associated with active disregard reflected by negative and aggressive responses to others' distress (Van Hulle et al., 2013) and higher heart rate was associated with greater empathy and likelihood of intervening to help (Barhight, Hubbard, & Hyde, 2013; Liew et al., 2003). These results are consistent with the notion that individuals who are highly aroused are more likely to be disregulated and distressed when confronted with another's distress, motivating them to be prosocial in order to alleviate their own distress (Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994).



*Fear.* Within the limited research on the association between autonomic arousal and fear, evidence has been found for an association between a higher resting heart rate and more fear in children (Calkins, Dedmon, Gill, Lomax, & Johnson, 2002; Kagan et al., 1998), as well as distress responses (Fabes et al., 1994; Fox, 1989; Wilson et al., 2009).

### **Integration of autonomic arousal into DLC theories involving temperament.**

Taken together, the results from various studies on temperament provide some evidence that individual differences in autonomic arousal can affect temperament in child and adult populations. It may be beneficial then, to incorporate arousal into temperament-based theories. For example, DeLisi and Vaughn's (2014) temperament-based theory suggests that reduced effortful control and negative emotionality produces antisocial conduct throughout life and explains people's failure to interact with criminal justice practitioners and complete correctional sentences. Individuals with such temperament may have lower social competence, low levels of self-constraint, and difficulty disengaging with negative or threatening stimuli, resulting in blaming others, becoming easily annoyed, and frequently losing their temper. Effortful control also predicts outcomes such as greater depression and higher likelihood of drug dependence, which are associated with criminal behavior (Moffitt et al., 2011). Based on the empirical findings, higher autonomic arousal may lead to better effortful control, which results in better ability to regulate attention, emotion, and behavior to function according to societal rules. In a similar way, autonomic measures can be incorporated into Lahey and Waldman's (2005) DLC theory of the propensity to offend during childhood and adolescence, which proposes that the daring, prosociality, and negative emotionality dimensions contribute to the antisocial propensity

of individuals. Thus, low levels of autonomic arousal can help to explain differences in temperament that have been shown to predict later offending.

**Cognition.** Cognitive functioning, in terms of verbal and spatial ability, decision-making, and memory processes, is influenced by states of physiological arousal. Although it has been suggested that among males, change in noncognitive skills has a larger effect on criminal behavior, measured by probability of incarceration, compared to a corresponding change in cognitive skills (Heckman, Stixrud, & Urzua, 2006), cognition has been shown to be linked to delinquency later in life and in persistently antisocial individuals (Moffitt, Lynam, & Silva, 1994; Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). It is a construct that is central to a number of DLC theories.

***Verbal and spatial ability.*** Greater autonomic activity is positively associated with verbal ability, measured by memory for words (Buchanan, Etzel, Adolphs, & Tranel, 2006). Through three verbal memory tasks, differences in skin conductance responding to arousing word groups, namely taboo and unpleasant words, predicted subsequent recall. Words that elicited greater skin conductance responses were better remembered, suggesting that greater autonomic responses to individual words influence better subsequent memory. Additionally, studies have found that higher blood pressure, which is positively correlated with heart rate (Morcet, Safar, Thomas, Guize, & Benetos, 1999; Zhang & Kesteloot, 1999), is linked to better visuospatial performance (Mathewson, Dywan, Snyder, Tays, & Segalowitz, 2011; Wharton et al., 2006).

***Decision-making.*** Physiological factors are also associated with decision-making (Bechara & Damasio, 2005). In a study by Armstrong and Boutwell (2012), individuals with a low resting heart rate perceived a lower likelihood of arrest for theft and drunk driving and were less likely to anticipate a sense of guilt or shame if they committed an assault during a confrontation. In this way, low resting heart rate was associated with consideration of the costs and benefits of offending. This is consistent with a prior study documenting that higher arousal, measured by heart rate during a complex mental activity, was associated with better performance in solving complex logical problems (Blatt, 1961). The relationship between arousal and decision-making, however, is more consistently observed among males (Blascovich, Nash, & Ginsburg, 1978).

***Memory processes.*** Autonomic measures of arousal at baseline and during recall and recognition tasks are also related to memory processes, with greater autonomic activity associated with better memory. Individuals with higher heart rates at baseline and during cognitive tasks, such as Digit Symbol and Symbology tasks, performed better than those with lower heart rates (Burgess & Hokanson, 1964; Capuana, Dywan, Tays, & Segalowitz, 2012). This finding is replicated with electrodermal activity. Higher skin conductance responses were linked to improved memory (Bradley, Greenwald, Petry, & Lang, 1992). However, there is evidence to suggest that the arousal-memory relationship depends on the level of cognitive demand of the task. High levels of arousal have been associated with better performance on more cognitively demanding tasks such as long-term memory tests compared to immediate tests (Burbridge, Larsen, & Barch, 2005; Craik & Blankstein, 1975) and in a more rigorous *n*-back working memory task

compared to an easier letter memory task (Cellini, Zambotti, Covassin, Sarlo, & Stegagno, 2014).

**Integration of autonomic arousal into DLC theories involving cognition.** In

DLC theories, it can be proposed that autonomic arousal causes individual differences in cognitive abilities. In this way, incorporating autonomic arousal can help to better explain extant DLC theories. For example, lower resting heart rates predicted life-course persistent offenders, but not adolescence-limited offenders in Moffitt's (1993) developmental taxonomy (Moffitt & Caspi, 2001). As cognitive deficits are linked to life-course persistent, but not adolescence-limited offenders, autonomic arousal may account for the neuropsychological variation that increases risk for life-course persistent offending (Moffitt, 1993). In addition, autonomic arousal can influence verbal ability, which constitutes one of the most important factors contributing to antisocial propensity in Lahey and Waldman's (2005) model. It can also influence cognitive processes such as thinking and decision-making that contribute to whether antisocial potential translates into crime and antisocial behavior as in Farrington's (2005b) integrated cognitive antisocial potential (ICAP) theory. It has been proposed that autonomic arousal facilitates learning through its effect on the brain, by establishing a level of cortical excitation without which learning cannot occur (Hebb, 1955). Thus, low levels of autonomic arousal may result in poorer cognitive abilities that affect individuals later in life such as in the form of fewer options to pursue legitimate employment (Moffitt, 1993) and poor decision-making when weighing the perceived costs and benefits of offending (Armstrong & Boutwell, 2012), that then lead to crime and delinquency.

## **Social Risk Factors**

**Life events.** An increasing body of research shows that social variables can influence autonomic system functioning (Ellis & Boyce, 2008; Hackman & Farah, 2009; Luecken, 1998; Wadsworth, 1976). More specifically, social factors in the form of life events such as neighborhood changes and marriage have been studied in relation to autonomic measures. For example, there is evidence of an effect of migration on higher blood pressure, which is positively correlated with heart rate (Morcet et al., 1999; Zhang & Kesteloot, 1999), that is significant even controlling for factors such as weight change and selective migration, particularly among men. These findings are consistent across different countries including New Zealand (Beaglehole, Eyles, & Prior, 1979; Salmond, Prior, & Wessen, 1989), Italy (Modesti et al., 1994), Africa (Poulter et al., 1990), and China (He et al., 1991). In addition, marital status is associated with cardiovascular functioning, as married individuals had higher ambulatory blood pressures than single individuals (Holt-Lunstad, Birmingham, & Jones, 2008).

*Integration of autonomic arousal into DLC theories involving life events.* DLC theories such as Sampson and Laub's (2005) age-graded theory notes that life events in adulthood inhibit offending and foster desistance from crime, while the ICAP theory suggests that life events affect individuals' long-term antisocial potential, which in turn affects criminal behavior (Farrington, 2005b). Although research on the relationship between life events and autonomic arousal is limited and the mechanisms underlying the association are unclear, the findings suggest that the understanding of desistance in DLC

theories can be improved by noting that life events can affect criminal behavior, in part, through their effects on autonomic functioning.

**Family factors.** Family factors are included in most DLC theories, and variables such as bad parenting have long been acknowledged as causes of crime and delinquency. Findings have supported a negative relationship between family factors and autonomic arousal as children who had experienced separation from the home before age 4 years had lower pulse rates at age 11 compared to children from intact homes (Wadsworth, 1976). The association between poor social bonds and low arousal is supported by the finding that low arousal is associated with low empathy (Lorber, 2004; Van Hulle et al., 2013; Zahn-Waxler et al., 1995), which is required for bonding and attachment (Bowlby, 1982; Britton & Fuendeling, 2005; Joireman, Needham, & Cummings, 2002; Laible, Carlo, & Roesch, 2004). However, the relationship between family factors and autonomic arousal is not clear cut as some conflicting results have been documented. One study noted no significant difference in resting heart rate and skin conductance levels between abused children and matched controls (Carrey, Butter, Persinger, & Bialik, 1995), while others have found that poor quality of caretaking is associated with higher heart rates in offspring (Bell & Belsky, 2008; Luecken, 1998; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). These inconsistencies may be attributable to family context and future research should recognize family context as a critical factor to consider in assessing the long-term physiological impact of family factors (Roubinov & Luecken, 2010).

*Integration of autonomic arousal into DLC theories involving family factors.*

The association between family factors and autonomic measures of physiological arousal suggests that autonomic arousal can be incorporated into DLC theories in which family factors play a role, such as Sampson and Laub's (2005) general age-graded theory and Thornberry and Krohn's (2005) interactional theory. Sampson and Laub (2005) propose that family factors such as parental discipline and supervision contribute to the strength of bonding between individuals and parents, which in turn tends to inhibit offending. Thornberry and Krohn (2005) propose that parenting deficits, characterized by poor monitoring, low affective ties, and physical punishment contributes to the early manifestation of antisocial behavior in children from birth to 6 years of age, compared to late starters who begin to engage in antisocial behavior from ages 18 to 25 years. The explanations of offending in these theories can be improved by considering that family factors may lead to crime and delinquency partly through their influence on individuals' levels of autonomic arousal. For example, negative parenting experiences early in a child's life can result in long-term chronic exposure to stress which imposes a biological burden on individuals' physiological systems (McEwen, 1998). This can lead to downregulation of physiological responses, which may help to explain in part how family factors are associated with offending throughout the life course.

**Addressing Key DLC Issues Using Autonomic Arousal**

Autonomic measures of physiological arousal can also contribute to DLC theories by shedding light on key empirical and theoretical issues, such as desistance, the

explanation of within-individual differences in offending, the contribution of learning and decision-making processes in offending, and findings that may challenge DLC theory.

**Protective factors.** One important issue that can be addressed with the incorporation of autonomic arousal lies in the understanding of factors that inhibit offending and the explanation of factors influencing desistance (Farrington, 2005a). Psychological and social factors such as attachment and socialization processes (Farrington, 2005b) and life events (Wikström, 2005) have been proposed in DLC theories as variables that inhibit offending. However, such theories do not consider the role of biological factors as possible protective factors in the development of crime and delinquency. High autonomic functioning, measured by resting heart rate and skin conductance levels, has been documented to protect against criminal and antisocial behavior (Brennan et al., 1997; Portnoy, Chen, & Raine, 2013; Raine, Venables, & Williams, 1995). Similar results have been obtained for orienting and fear conditioning (Raine, Venables, & Williams, 1995; Raine, Venables, & Williams, 1996). To address this issue, it would be desirable to incorporate autonomic measures of physiological arousal in DLC theories.

**Within-individual change.** Autonomic arousal in DLC research can also aid in explaining within-individual differences in offending. The heart rate-antisocial behavior relationship has been confirmed in both cross-sectional and prospective longitudinal research, suggesting that heart rate predicts offending in spite of change in age. However, studies have not examined whether change in an individual's heart rate and skin



conductance level over time explains offending over time. Analyses of within-individual changes in autonomic arousal, psychological risk factors, and later offending can help to establish causal relations as such analyses allow for the controlling of possible between-group factors that can influence offending, as well as the determination of causal order (Farrington, 1988; Murray, Farrington, & Eisner, 2009). Obtaining a more accurate picture of how changes in these constructs affect changes in behavior can enable us to gain a better understanding of the development and causes of offending.

**Learning and decision-making processes.** Another key issue raised in relation to DLC theories is whether there is a learning or decision-making process for offending (Farrington, 2005a). As Figure 2.1 suggests that autonomic arousal influences these cognitive processes to lead to offending, autonomic arousal can play an important role in addressing this key theoretical issue.

**Challenges to the theory.** It is also important to address what findings might challenge the theory shown in Figure 2.1. Although some empirical support for the proposed model has been documented (Choy et al., 2015), one study found that the relationship between resting heart rate and antisocial behavior in children was almost entirely explained by genetic influences (Baker et al., 2009). With the dearth of replicated studies, additional research on autonomic arousal can reveal whether findings that challenge the model exist and thus, shed light on this important issue raised in DLC criminology.

## Conclusion

Research on autonomic measures of physiological arousal supports the incorporation of such constructs in DLC theories. While similar models to Figure 2.1 have been suggested to explain antisocial behavior, by proposing an integrative biopsychosocial mediation framework that builds on existing models, this article demonstrates how biological measures such as autonomic arousal can complement and add coherence to existing DLC theories. The proposed model suggests a biological factor, autonomic arousal, as an explanatory construct that is missing in extant DLC theories. For example, as depicted in Figure 2.1, autonomic arousal may serve as a mechanism linking social factors, such as life events and family factors that are described in Sampson and Laub's (2005) theory, to crime and delinquency. Examining changes in autonomic arousal after experiencing turning points in life and including this missing construct in the theory can thus provide a more complete understanding of the processes leading to offending. This can result in the development of a more comprehensive theory regarding the etiology of criminal and delinquent behavior, adding coherence.

Although this review refers specifically to DLC theories, autonomic arousal can also be incorporated into classical criminological theories. For instance, autonomic arousal is associated with self-control (Fowles et al., 2000; Schmidt et al., 2013), which forms the foundation of Gottfredson and Hirschi's (1990) general theory of crime. Additionally, autonomic arousal is associated with poor parenting, an underlying construct in differential association theory (Sutherland, 1939).

This review is limited to autonomic arousal. However, other biological constructs, including genetic factors, brain deficits, and hormones, can also be incorporated into

DLC theories. For instance, brain functioning in the prefrontal executive attention system and parahippocampal gyri has been linked to temperamental and cognitive dimensions (Brewer, Zhao, Desmond, Glover, & Gabrieli, 1998; Posner, Rothbart, Sheese, & Voelker, 2012; Wagner et al., 1998), while increased striatum volume is linked to reward-seeking in the form of anticipation of monetary awards, which is in turn associated with motives for committing crime (Glenn, Raine, Yaralian, & Yang, 2010). As another example, life events such as marriage (Gray, Kahlenberg, Barrett, Lipson, & Ellison, 2002), involvement in a committed, romantic relationship (Burnham et al., 2003), and fatherhood (Gettler, McDade, Feranil, & Kuzawa, 2011) are associated with lower levels of testosterone in males. Future research could involve using the biopsychosocial model as a conceptual framework to empirically test how social factors may influence biological factors and psychological variables to lead to crime and delinquency later in life. Including other biological factors and adverse social stimuli can result in the development of more complex models. Additionally, possible moderating effects can be taken into account. For example, social factors have been found to moderate the relationship between risky decision-making and antisocial behavior (Gao, Baker, Raine, Wu, & Bezdjian, 2009). Thus, examining possible moderating effects on the proposed mediation model would be revealing. Relatedly, it would be valuable to expand on the model longitudinally to reflect additional relationships, such as the possible influence of offending on life events and family factors.

In incorporating autonomic measures of physiological arousal in DLC research and theories, there are some practical benefits and constraints to consider. One primary challenge lies in the need for expertise and experience in conducting psychophysiological

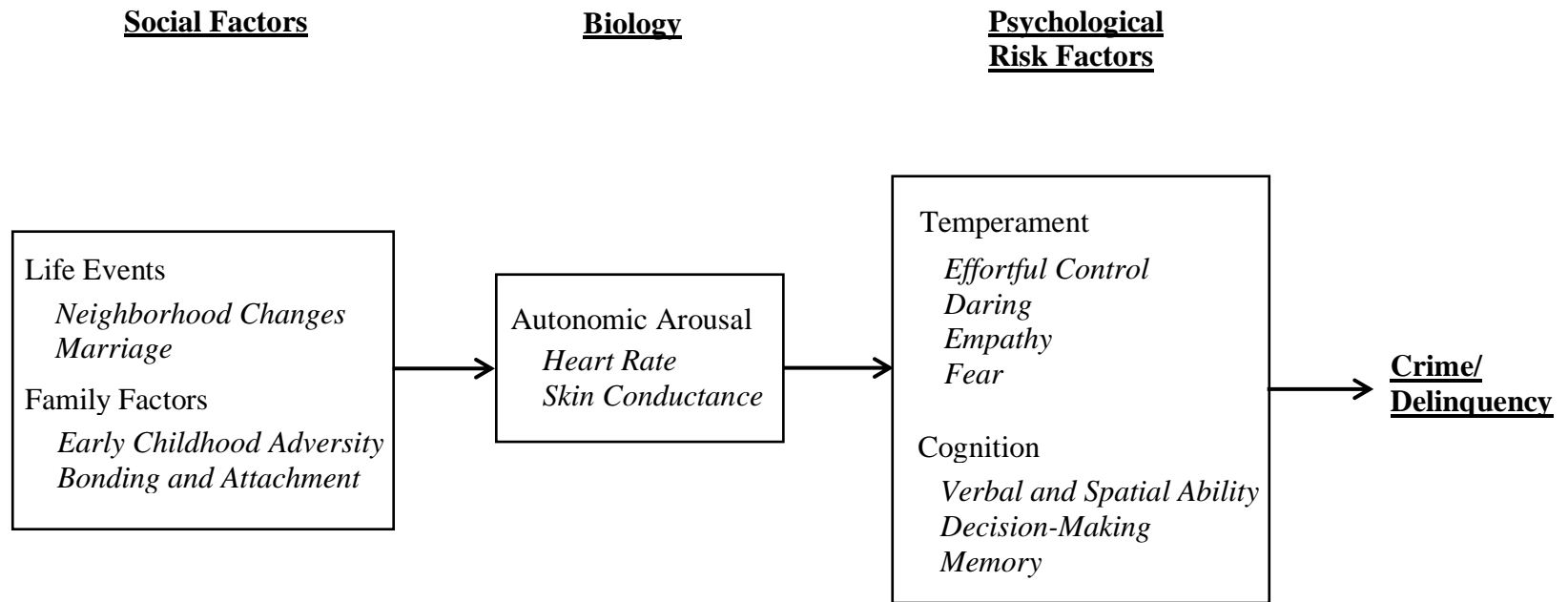
tests. Other practical issues involve the need for equipment for data collection, the choice of task during the measurement of psychophysiological arousal as results may be influenced by speech or the level of cognitive demand of the task (Wilson, 1992), the temperature of the environment, and the placement of electrodes to reduce motion artifacts. Despite the need for training in collecting psychophysiological data, psychophysiological measures are advantageous as they are easier to record and less invasive than many other biological measures. This is particularly true for heart rate. They do not require laboratory conditions and can be measured in community surveys. These features are especially beneficial for longitudinal studies that may take place in multiple environments. Autonomic measures can also be recorded continuously without severely limiting activity and movement, and several variables can be obtained at once. For example, besides resting heart rate or heart rate measured during a task, vagal tone and heart rate variability can be obtained from the data. These benefits render psychophysiological measures apt to include in DLC research.

Several major longitudinal studies in criminology have employed such autonomic measures. For example, cardiovascular functioning has been measured in the Cambridge Study in Delinquent Development (Farrington, Piquero, & Jennings, 2013), the Individual Development and Adaptation Research Program (Bergman, 2000; Magnusson, 1988), the Mater University Study of Pregnancy (Lawlor et al., 2004), and the Dunedin Multi-Disciplinary Health and Development Study (Moffitt et al., 2001), while the Mauritius Child Health Project (Raine, Venables, & Mednick, 1997) and the Pittsburgh Youth Study (Loeber, Farrington, & Stallings, 2011) have included measures of electrodermal activity in addition to heart rate. In light of the ease of measurement,

autonomic measures of arousal should be included in future prospective longitudinal studies. Increasingly, researchers are acknowledging that criminological theories that do not include biological influences of criminal behavior might be limited in scope and incomplete in their potential for contribution (DeLisi, 2012; Wilson & Scarpa, 2012). This article makes progress towards this goal by showing how autonomic arousal can be incorporated in DLC theories.

## Paper 2 Figure

Figure 2.1. The Proposed Biopsychosocial Model



*Note.* This model shows the pathways through which social factors (life events and family factors) lead to changes in biological functioning (autonomic arousal, measured by heart rate and skin conductance), which is in turn associated with psychological risk factors (temperament and cognition) that predispose to crime and delinquency. Subdimensions of each construct are listed in italics.

### **PAPER 3. EXPLAINING THE GENDER GAP IN CRIME: THE ROLE OF HEART RATE**

#### **Abstract**

Although it is well established that males engage in more crime compared with females, little is known about what accounts for the gender gap. Few studies have been aimed at empirically examining mediators of the gender-crime relationship in a longitudinal context. In this study, we test the hypothesis that a low resting heart rate partly mediates the relationship between gender and crime. In a sample of 894 participants, the resting heart rate at 11 years of age was examined alongside self-reported and official conviction records for overall criminal offending, violence, serious violence, and drug-related crime at 23 years of age. A low resting heart rate partially mediated the relationship between gender and all types of adult criminal offending, including violent and nonviolent crime. The mediation effects were significant after controlling for body mass index, race, social adversity, and activity level. Resting heart rate accounted for 5.4% to 17.1% of the gender difference in crime. This study is the first to produce results documenting that lower heart rates in males partly explain their higher levels of offending. Our findings complement traditional theoretical accounts of the gender gap and have implications for the advancement of integrative criminological theory.

## **Background**

The higher rate of offending among males compared with females is a well-documented finding in criminology. The gender gap has been found across time, cultures, and data sources (Archer, 2004; Heimer, Lauritsen, & Lynch, 2009; Rohner, 1976; Schwartz, Steffensmeier, Zhong, & Ackerman, 2009; Steffensmeier, Schwartz, Zhong, & Ackerman, 2005). Evidence also shows that gender differences in antisocial behavior are observed as early as 17 months of age (Baillargeon et al., 2007; Tremblay et al., 1999). Thus, any encompassing theory of crime and antisocial behavior should help to explain the gender difference in offending that is in place from early in life to adulthood.

The reason for the gender gap has become the subject of considerable theoretical debate (Foster, 2014). The limitations of socially grounded criminological paradigms, such as social control, differential association, strain, and reintegrative shaming theories, in fully explaining the gender gap have prompted researchers to move beyond the standard concepts of traditional theories (De Coster, Heimer, & Cumley, 2012). In this article, we address this gap in the literature regarding the incomplete understanding of why males are more criminal than females, while answering recent calls for research on sex differences in biological functioning and behavior (National Institute of Mental Health, 2011; National Institutes of Health Office, 2015).

### **Socially Oriented Theories of Crime and the Gender Gap**

Dominant explanations for the gender gap have stemmed from a socially oriented perspective according to at least four criminological theories — social control theory, differential association theory, strain theory, and reintegrative shaming theory. Social



control theory proposes that differences in parenting and socialization for males and females arise from gender expectations (Gottfredson & Hirschi, 1990). These differences then encourage the development of traits such as empathy and emotion regulation in females, which contribute to a better ability to control impulses. Meanwhile, the lower levels of self-control among males, characterized by impulsivity and risk-taking inclinations, are argued to predispose them toward criminal behavior. Differential association theory in contrast suggests that a higher rate of male offending results from greater association with delinquent peers as girls may be more closely supervised and have fewer opportunities to interact with nonconventional others (Akers, 2009; Burgess & Akers, 1966; Sutherland, 1947). The theory also suggests that as a result of social learning, males are more likely to be taught identities emphasizing traits such as risk-taking and to hold beliefs that encourage offending. General strain theory in turn introduces the argument that the gender gap can be attributed to males' heightened exposure to strain and emotional responses to strain that are more conducive to crime (Agnew, 1992; Broidy & Agnew, 1997). Other conditioning factors such as limited legitimate coping resources, lower levels of social control and emotional support, and greater association with criminal peers increase males' likelihood of responding to strains with crime (Agnew, 2009). Reintegrative shaming theory gives rise to the notion that females experience greater self-conscious emotion than do males, rendering them less prone to engage in crime or delinquency (Braithwaite, 1989; Van Gelder, Elffers, Nagin, & Reynald, 2014).

Some support has been found for these theories. For example, in line with social control theory, empirical findings have suggested that attachment to conventional values

(Liu & Kaplan, 1999), parental involvement and monitoring (Bell, 2009; Svensson, 2003), disruptions of early attachment (Hayslett-McCall & Bernard, 2002), and self-control (e.g., Kim & Kim, 2015; LaGrange & Silverman, 1999; Tittle, Ward, & Grasmick, 2003) can account for the gender gap in offending. Consistent with the differential association theory explanation, higher levels of exposure to delinquent peers have been found among males, which may account, at least in part, for the gender gap (Bell, 2009; Liu & Kaplan, 1999; Jensen, 2003; Mears, Ploeger, & Warr, 1998; Simons, Miller, & Aigner, 1980; Svensson, 2003). Evidence also shows that males are more likely than females to endorse attitudes favorable to violent behavior (Heimer & De Coster, 1999). Partial empirical support for the strain theory explanation is derived from the results of research on stress. Males have been found to experience more strains that are conducive to serious crimes (Broidy & Agnew, 1997). In line with reintegrative shaming theory, anticipated shame has been documented as an intervening mechanism linking gender to crime and delinquency (Rebellon, Wiesen-Martin, Piquero, Piquero, & Tibbetts, 2015; Tibbetts, 1999).

Despite these findings, there are limitations in the ability of these theoretical perspectives to explain the gender gap in full, suggesting that other mechanisms may be in play. First, mixed conclusions have been found in empirical assessments of these theories. For instance, reports that there are no consistent differences in the parenting of boys and girls (Shekarkhar & Gibson, 2011), that over-involvement in activities may promote delinquency (Booth, Farrell, & Varano, 2008), and that differences in self-control levels contribute little to the understanding of the gender gap (Botchkovar & Broidy, 2012) contradict the social control theory explanation. Moreover, in conflict with

strain theory, there is evidence that males and females do not experience different amounts of strain (Broidy & Agnew, 1997) and respond to strain with similar emotions (e.g., Jennings, Piquero, Gover, & Perez, 2009; Piquero, Fox, Piquero, Capowich, & Mazerolle, 2010). For example, some researchers have found that anger and achievement frustration, which are constructs of general strain theory, failed to mediate the relationship between gender and offending (Liu & Kaplan, 1999; Rebellon et al., 2015). Limited empirical support has also been found by researchers for the effect of conditioning variables on the strain-crime link (e.g., Aseltine Jr., Gore, & Gordon, 2000; Botchkovar, Tittle, & Antonaccio, 2009; Jang & Rhodes, 2012).

Second, these theories are not capable of fully explaining the gender gap even when examined simultaneously (Bell, 2009; Booth et al., 2008; Rebellon et al., 2015; Svensson, 2003). Third, the ability to explain the gender gap for different types of offending is lacking. Traditional theories have been proposed to be better at explaining minor delinquency than serious offending (Steffensmeier & Allan, 1996). Fourth, with the exception of Liu and Kaplan (1999), constructs from these theories have not been tested in a formal mediation model with longitudinal data. Fifth, traditional theoretical explanations are seriously challenged by not accounting for why the gender difference in antisocial behavior is observed as early as 17 months of age and persists into adulthood. The early occurrence of the gender difference in physical aggression contradicts explanations involving differential observational learning of patterns of aggressive behavior, and is too early to be the result of gendered socialization processes (Archer, 2004; Archer & Côté, 2005). Moreover, gender differences in crime persist in adulthood,

where differences in supervision by agents of social control such as parents are minimal (Gottfredson & Hirschi, 1990).

### **Biopsychosocial Explanations for the Gender Gap**

Given these limitations, sociologists have appropriately acknowledged that other processes, including biological factors, also may help to explain gender differences in offending (Jensem & Eve, 1976; LaGrange & Silverman, 1999; Steffensmeier & Allan, 1996). This acknowledgment is supported by the fact that even within similar environments, female rates of offending remain lower than that of males (Lauritsen, Heimer, & Lynch, 2009; Sutherland & Cressey, 1970). Socially oriented theories have shed light on psychological mediators for the gender gap. Social control theory and differential association theory are consistent with the social role perspective derived from social psychology, that the female gender role's emphasis on caring and concern for others strengthens their beliefs about the negative consequences of offending. Females' increased guilt and anxiety about causing others to suffer, and their greater fear about the danger that their aggressive behaviors would pose to themselves from retaliation, would inhibit antisocial behavior (Eagly & Steffen, 1986). Social control theory also suggests that externally exerted social control becomes internalized as self-control. Self-control, which has been operationalized as a combination of impulsivity and risk seeking, is a proposed psychological explanation for the gender gap (Campbell, 2006). In general strain theory, personality variables such as negative emotionality condition the effect of social variables on criminal behavior (Agnew, 2009).

Besides psychological processes, biological characteristics have been empirically shown to improve and extend existing models of criminal behavior (Barnes et al., 2014; Beaver & Walsh, 2010; Boisvert, Vaske, Wright, & Knopik, 2012; Cauffman, Steinberg, & Piquero, 2005; DeLisi, Wright, Vaughn, & Beaver, 2010; Vaske, Wright, Boisvert, & Beaver, 2011). They are also consistent with the suggestion that theoretical explanations for the gender gap should involve variables that are influential in both genders but have distinguishable distributions in males and females (Rowe, Vazsonyi, & Flannery, 1995). Despite evidence supporting the role of biopsychosocial explanations, few studies have been aimed at formally testing whether biological factors may underlie the gender gap. One exception is a study with a finding that controlling for characteristics enhanced by male sex hormones (e.g., testosterone) reduced the gender difference specifically in violent offending (Ellis, Das, & Buker, 2008). In another study, researchers documented that after controlling for gray matter volumes in three regions of the prefrontal cortex, the gender difference in antisocial behavior was more than cut in half (Raine, Yang, Narr, & Toga, 2011).

Resting heart rate has been suggested to be a viable candidate to account for the gender difference in offending (Portnoy et al., 2014a). A low resting heart rate is widely regarded as the best-replicated biological correlate of antisocial and criminal behavior. Through a recent meta-analysis on resting heart rate and antisocial behavior among children and adults, researchers documented an effect size of  $d = -.20$  (Portnoy & Farrington, 2015). This relationship is observed for both males and females and for multiple types of antisocial behavior, including violent and nonviolent criminality

(Latvala, Kuja-Halkola, Almqvist, Larsson, & Lichtenstein, 2015; Murray et al., 2016; Portnoy & Farrington, 2015).

Long-standing evidence also shows that resting heart rates differ by gender. In a study on 35,302 individuals, females had significantly higher heart rates when compared with males at every age category from 1 year to 79 years of age (Ostchega, Porter, Hughes, Dillon, & Nwankwo, 2011). This finding has been extended to infants. A study on newborns 0 to 7 days old found that females had significantly higher heart rates versus males, adjusting for a host of variables (Nagy, Orvos, Bárdos, & Molnár, 2000). Thus, the gender differences in heart rate that appear throughout life may be present at birth.

One suggested mechanism linking low heart rate to antisocial behavior is fearlessness. Reduced autonomic arousal may index a lower level of fear in individuals, who are then more likely to engage in antisocial behavior (Armstrong & Boutwell, 2012; Venables, 1987). Nevertheless, the results from recent studies have revealed empirical support for the theory that the personality trait, sensation seeking, mediates the relationship between heart rate and antisocial behavior (Portnoy et al., 2014b; Sijtsema et al., 2010). Sensation-seeking theory posits that underarousal inclines individuals to seek stimulation through antisocial behavior (Quay, 1965). The relationship between low heart rate and sensation seeking suggests that the gender difference observed in sensation seeking may exist because of gender differences in biology (Cross, Cyrenne, & Brown, 2013; Vaske, Boisvert, & Wright, 2015). Thus, a biological factor such as low heart rate may underlie a portion of gender differences in psychological variables such as self-control, anticipated shaming, and emotions as proposed in the social control, differential association, and strain theories.

To support heart rate as a partial explanation for the gender gap, heart rate is associated with testosterone and the prefrontal cortex, variables that partly explain the gender difference in antisocial behavior (Ellis et al., 2008; Raine et al., 2011). Consistent findings show that receptors for steroid sex hormones (e.g., testosterone) are present in the heart, suggesting that sex hormones contribute to the gender-related difference in heart rate (Nio, Stöhr, & Shave, 2015; Wittnich, Tan, Wallen, & Belanger, 2013). Additionally, although limited, upregulating prefrontal cortical activity can result in an increased heart rate (Knotkova et al., 2012). These findings, together with the robust relationship between heart rate and offending, and the presence of gender differences in heart rate even in infancy, render a low heart rate as a putative biological mechanism that partly explains the relationship between gender and crime.

### **Current Study**

The purpose of this study is to test whether a biological factor, low heart rate, serves as a mediating variable in partly accounting for differential levels of male and female crime. While not negating the importance of social influences, this study aims to contribute to a more complete explanation of the gender gap. We hypothesize that a low resting heart rate at 11 years of age partly mediates the relationship between gender and criminal offending at 23 years of age. We build on the limited number of studies that have been conducted to test theories of the gender gap empirically by examining the mediation effects on different types of crime in addition to overall criminal offending. In line with the two prior 1-year follow-up studies that were aimed at examining

explanations for the gender gap (Liu & Kaplan, 1999; Smith & Paternoster, 1987), the longitudinal design adopted here allows for the establishment of causal order.

## **Methods**

### **Subjects**

Data were obtained from a subsample of participants derived from a prospective longitudinal study, the Mauritius Child Health Project. The original sample consisted of 1,795 children recruited from the island of Mauritius at 3 years of age. Children born in 1969 to 1970 in two major towns, Quatre Bornes and Vacoas, were recruited into the study such that the original cohort had a racial distribution representative of the country as a whole. At 11 years of age, a major psychophysiological test was conducted, and at 23 years of age, assessments on adult criminal offending were made. Participants with complete data on autonomic arousal and crime at 11 and 23 years, respectively, were included in the analyses. Based on the distribution of heart rate values, four outliers were identified and excluded, resulting in a total sample of 894 participants (497 males, 397 females). Descriptive statistics are reported in Table 3.1.

Of these 894 participants, 69.8% ( $n = 624$ ) were Indian, 24.2% ( $n = 216$ ) were Creole (African origin), and 6.0% ( $n = 54$ ) were of other ethnicities (Chinese, English, and French). No significant differences were observed in the number of self-reported crimes and official court convictions, race, or social adversity between individuals who were included in the analyses and those who were not ( $p > .05$ ). Informed consent was obtained from the mothers of participants at 3 and 11 years of age. Written informed consent was obtained from participants at 23 years of age. Additional information about



participant recruitment and background characteristics of the initial sample are given in Raine, Liu, Venables, Mednick, & Dalais (2010).

## **Measures**

**Resting heart rate.** All participants were tested at 11 years of age under the same controlled conditions in a sound-insulated, temperature-controlled cubicle. Testing was conducted in a research-dedicated building in a quiet, residential area of Quatre Borne. A dehumidifier was used to minimize fluctuations in humidity. During the 5-minute rest period, participants were instructed to relax with their eyes closed and to minimize movement to standardize conditions across participants and to reduce movement artifacts. Heart rate was recorded with Beckman silver/silver chloride electrodes and Cambridge electrode gel. A Standard Lead I recording configuration was used, with the electrocardiogram amplified by using a Grass type 79 polygraph and a 7P5 preamplifier. Data were recorded from the polygraph onto a Racal Store 4 FM tape recorder for off-line analysis. The analog data were digitized with ASYST software and manually cleaned for movement artifacts. Heart rate, in beats per minute, was calculated from the average of artifact-free interbeat intervals over the 5 minutes of the rest condition.

**Crime.** Criminal offending at 23 years of age was assessed from structured interviews about participants' histories of criminal offending in the past 5 years. The adult extension of the self-report delinquency measure from the National Youth Survey was used (Elliott, Ageton, Huizinga, Knowles, & Canter, 1983), asking participants about the perpetration of 41 criminal offenses covering a range of theft, violence, and drug

crimes. In addition, official crime records were obtained from district courts. The registration of offenses, including property, drug, violence, and serious driving offenses (e.g., driving while drunk and dangerous driving), was collected. As a result of the highly skewed nature of the crime variables, criminal behavior was dichotomized and an all-source measure of offending was used, whereby participants who either reported a criminal offense or had a court record for any offense were categorized as criminal (De Coster, Iselin, & Gallucci, 2009; Loeber, Farrington, & Jolliffe, 2008; Murray et al., 2016). Self-report and official records were combined as it has been proposed that they reflect different sources of error and a combination of these records is the most inclusive indicator of criminal activity in adulthood (Babinski, Hartsough, & Lambert, 2001). In addition to overall criminal offending (i.e., having committed any crime), the perpetration of violence, serious violence, and drug-related offenses was analyzed to assess subtypes of offending specifically. For completeness, self-reported data and official court records were later analyzed separately.

To test whether the results were sensitive to our methodology of dichotomizing offending, we conducted additional analyses with the variety of offenses as the dependent variable. As the prevalence of female offending according to official court records was insufficiently common to afford statistical analysis (Table 3.1), only self-reported variety scores were used. The total number of types of offending was calculated. Variety scales are a good reflection of criminality as a result of their lower sensitivity to higher frequency items, lower skew compared with frequency scales, and higher concurrent validity and equal predictive validity compared with other offending scales (Osgood, McMorris, & Potenza, 2002; Sweeten, 2012).

## **Covariates**

Social adversity, body mass index (BMI), race, and activity level were considered as confounders of the relationship between gender (male = 1) and crime. The social adversity index was based on 14 variables collected by social workers who visited the homes of the children at 11 years of age (Raine, Yaralian, Reynolds, Venables, & Mednick, 2002). A total adversity score was calculated by adding 1 point for each of the following 14 variables: living in rented accommodation, house without electricity or water, child has neither good toys nor good books, no television, living in poor housing, father uneducated, mother uneducated, parent psychiatrically ill, parent physically ill, teenage mother (mother 19 years of age or younger when child was born), single-parent family, separation from both parents, five or more siblings, and overcrowded home with five or more family members per house room. As physical size has been argued to be central to understanding gender, sex, and violence (Felson, 2014), BMI was derived from height and weight measured at 11 years of age and was calculated as kilograms/m<sup>2</sup>. Race was categorized as Indian, Creole, or Other. Activity level at 11 years of age was assessed according to the hyperactivity score from the Child Behavior Checklist, controlling for aggression and delinquency (Achenbach & Edelbrock, 1983). Higher scores reflect greater hyperactivity. Data on smoking were not collected as cigarette consumption in Mauritius is low (ITC Project, 2010) and cigarette use at 11 years old was almost unheard of in this sample.

## **Statistical Analyses**

Bivariate relationships between the key study variables were examined as part of a preliminary analysis. Linear regressions were conducted to investigate gender differences in resting heart rate. Gender differences in adult offending and the relationship between heart rate and crime were assessed by using logistic and negative binomial regression when the dependent variables were dichotomous measures of offending and variety scores, respectively. Effect sizes were computed with Cohen's *d*.

The mediation effect of gender on crime at 23 years of age through heart rate at 11 years of age was examined by using two approaches. In the first approach, the PROCESS macro on SPSS version 22.0 (IBM Corporation, Armonk, NY, 2013) was adopted to test the significance of indirect effects of gender on dichotomous measures of offending. A total of 10,000 bootstrapped samples were drawn from the original data (Hayes, 2013). The path from the independent variable (gender) to the mediator (heart rate) was estimated with ordinary least-squares regression, whereas the path from the mediator (heart rate) to the dependent variable (crime) was estimated with logistic regression. The indirect effect of gender on offending was calculated as the product of the ordinary least-squares coefficient for the relationship between gender and heart rate and the logistic regression coefficient for the relationship between heart rate and crime. The total, direct, and indirect effects were obtained as regression coefficients in unstandardized form. The bootstrapping procedure was adopted as it is more rigorous and powerful compared with the Sobel test (Zhao, Lynch, & Chen, 2010). Bootstrapping provides better estimates of standard errors to identify any mediation effect, makes fewer assumptions about the shape of the sampling distribution of the indirect effect, and has

higher power while maintaining reasonable control over the type I error rate (Hayes, 2013; MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2008).

As the path coefficients in the mediation models were estimated with different regression models (logistic and linear), the indirect and total effects were scaled differently. Therefore, to assess the extent of mediation, the coefficients were first standardized to render them comparable across equations. The magnitude of the mediation effect was then expressed as the ratio of the indirect to total effect of gender on criminal offending (Hayes, 2013).

In the second approach, the indirect effect of gender on variety of offending was assessed with the PARAMED command in Stata version 14.0 (Emsley & Liu, 2013; Stata Corp, College Station, TX, 2015). First, the effect of gender on heart rate was estimated with a linear regression model. Second, the effects of gender as well as heart rate on variety scores were estimated with negative binomial regression. The indirect effect was estimated with a counterfactual framework (Valeri & VanderWeele, 2013). The counterfactual approach to mediation was used, rather than the product of coefficients method, as it allows for count outcomes (Valeri & VanderWeele, 2013). Bias-corrected bootstrap confidence intervals for the total, controlled direct, and natural indirect effects were computed from 10,000 bootstrapped samples.

Social adversity, BMI, race, and activity level were included as covariates in all mediation models. As a result of missing data on three covariates, namely social adversity, BMI, and activity level, mediation analyses were conducted on a subsample of 663 participants for whom we had complete data. For the purpose of sensitivity testing

and completeness, we reran the mediation analyses after imputing missing data on the covariates with the maximum likelihood expectation-maximization procedure on SPSS.

## Results

### Bivariate Relationships

**Gender differences in crime and heart rate.** Gender differences were observed in resting heart rate at 11 years of age and in crime at 23 years of age. Females had higher resting heart rates compared with males [ $b = -10.12$ , standard error (SE) = .95,  $p < .001$ ]. Gender accounted for 11% of the variance in resting heart rate levels. Significant gender differences were also observed in the prevalence of overall criminal offending [ $b = 2.26$ , SE = .18,  $p < .001$ , odds ratio (OR) = 9.62], violent offending ( $b = 2.02$ , SE = .24,  $p < .001$ , OR = 7.58), serious violence ( $b = 2.80$ , SE = .35,  $p < .001$ , OR = 16.41), and drug-related crimes ( $b = 3.55$ , SE = .51,  $p < .001$ , OR = 34.80), with males having a higher prevalence rate of offending than that of females. According to the negative binomial regression model, males also had higher variety scores of offending compared with those of females ( $b = 2.18$ , SE = .19,  $p < .001$ ).

**Heart rate and crime.** Bivariate regressions were undertaken to assess the relationship between low resting heart rate and crime. A low resting heart rate at 11 years of age was associated with overall criminal offending at 23 years of age ( $b = -.03$ , SE = .01,  $p < .001$ , OR = .97). More specifically, a low resting heart rate at 11 years of age was associated with violent crime ( $b = -.04$ , SE = .01,  $p < .001$ , OR = .96), serious violence ( $b = -.04$ , SE = .01,  $p < .001$ , OR = .96), and drug offenses ( $b = -.04$ , SE = .01,  $p < .001$ ,

OR = .96) at 23 years of age. The magnitudes (Cohen's  $d$ ) of differences in heart rate levels between offenders and non-offenders range from .46 to .62 (Table 3.2). In addition, a low resting heart rate was associated with a greater variety of offending ( $b = -.04$ ,  $SE = .01$ ,  $p < .001$ ,  $d = -.41$ ).

### **Mediation Effects**

These analyses document that there is a significant total effect of gender on crime. They also establish that a low heart rate is related to being male as well as to increased offending. As such, a low heart rate is a candidate as a process explaining the effect of gender on crime. Consequently, mediation analyses were conducted to test the indirect effect – the path from gender to heart rate to crime.

A low resting heart rate partly mediated the relationship between gender and overall criminal offending (indirect effect = .14,  $SE = .07$ ,  $p < .05$ ), violent crime (indirect effect = .28,  $SE = .09$ ,  $p < .001$ ), serious violent crime (indirect effect = .31,  $SE = .10$ ,  $p < .001$ ), and drug-related crimes (indirect effect = .19,  $SE = .10$ ,  $p < .05$ ). As shown in Figure 3.1, the total and direct effects in all four mediation models indicate that the magnitude of the association between gender and criminal offending, assessed from a combination of self-reports and official court records, decreased after controlling for resting heart rate levels. After rescaling the coefficients, calculating the ratio of the indirect to total effect of gender on crime revealed that 6.0% of the total effect of gender on overall criminal offending was accounted for by low resting heart rate. A low heart rate also accounted for 14.0%, 10.1%, and 5.4% of the total effect of gender on violent offending, serious violence, and drug offenses, respectively.

These findings were replicated with variety scores. The total effect and controlled direct effect of gender on self-reported variety of offending were 2.33 (SE = .24,  $p < .001$ ) and 2.13 (SE = .25,  $p < .001$ ), respectively (Table 3.3). A low resting heart rate partly mediated the association between gender and self-reported variety of offending (natural indirect effect = .20, SE = .08,  $p < .001$ ). The total, direct, and indirect effects indicate that the gender gap significantly decreased after adding heart rate to the model. As the controlled direct effect of gender on variety score was statistically significant, heart rate partly accounted for the gender gap. This reinforces the mediation findings based on the dichotomous measures of offending.

Additional analysis was conducted on self-reported outcome measures specifically. The total, direct, and indirect effects of gender on self-reported dichotomous measures of offending are reported in Table 3.3. The bias-corrected bootstrap confidence intervals for the indirect effects were above zero. Therefore, the indirect effects were statistically significant ( $p < .05$  for overall offending,  $p < .001$  for violence,  $p < .001$  for serious violence,  $p < .05$  for drug offenses). These results suggest that gender is indirectly related to self-reported criminal offending through its association with resting heart rate. A low resting heart rate accounted for 7.1%, 17.1%, 12.5%, and 5.5% of the total effect of gender on self-reported overall offending, violent offending, serious violence, and drug offenses, respectively. As further support for the robustness of these findings, mediation results from analyses on the imputed data remained substantively unchanged (Table 3.4).



## **Discussion**

The main objective of this study was to test whether a low resting heart rate partly explains the gender gap in offending. Males had lower resting heart rates at 11 years of age and committed more crime at 23 years of age. Support for a causal developmental model was documented whereby gender, determined before birth, is associated with a lower resting heart rate at 11 years of age, which is in turn associated with increased crime at 23 years of age. Evidence for mediation was documented for all forms of adult criminal offending, including violent crimes, serious violence, and drug-related offenses, as well as variety scores. Although findings do not document causality and do not suggest that a low heart rate completely accounts for the gender gap, they are to our knowledge, the first to show that lower heart rates in males partly explain their higher levels of offending. This study is one of few to probe statistically any type of mediation model (involving any social or nonsocial mediator) of gender differences in crime in a longitudinal context.

### **Consistency with Socially Oriented Theory Explanations**

The mediation findings documented here have the potential to complement dominant sociological and psychological explanations for the gender gap. Empirical evidence has shown that heart rate is associated with constructs in traditional criminological theories, which suggests that even though socializing influences can affect variables such as self-control, anticipated shaming, and emotion regulation, a low heart rate may also serve as a basis for the processes in socially oriented theoretical explanations for the gender gap. For instance, in empirical studies, researchers have

documented that a low heart rate is associated with higher impulsivity (Mathias & Stanford, 2003; Schmidt, Mussel, & Hewig, 2013) and increased risk-taking and sensation-seeking behavior (e.g., Portnoy et al., 2014b; Schmidt et al., 2013), which are components of low self-control (Schreck, 1999). The notion that a low heart rate may contribute to individual differences in self-control is further supported by findings that a low resting heart rate predicts victimization and involvement in accidents as vulnerability to crime and accident-proneness are increased by lower self-control (Latvala et al., 2015; Schreck, 1999). These findings complement the social control theory explanation for the gender gap as the gender-related difference in heart rate may help explain the gender differences in self-control that in turn lead to differential rates of offending.

Our findings are also in line with reintegrative shaming theory. A low resting heart rate has been documented to be associated with emotions such as a lower likelihood of anticipating guilt or shame for committing an assault (Armstrong & Boutwell, 2012). Additionally, a low heart rate is associated with reduced empathy (Barhight, Hubbard, & Hyde, 2013; Liew et al., 2003; Zahn-Waxler, Cole, Welsh, & Fox, 1995). This association supports the notion from reintegrative shaming theory that self-conscious emotions may mediate the relationship between gender and crime (Rebellon et al., 2015). Higher resting heart rates among females may suggest higher levels of empathy that may inhibit crime.

Our findings can also complement strain theory explanations (Broidy & Agnew, 1997). Coping style in response to stressors has been suggested to be related to autonomic functioning. The finding that greater expression of negative emotions is related to lower heart rates (Ramaekers, Ector, Demyttenaere, Rubens, & Van De Werf,

1998) is consistent with the strain theory notions that males' externalization of negative emotions and poorer coping skills in response to strain account for their higher levels of crime (Broidy & Agnew, 1997). The compatibility between the current study's findings and extant criminological theory explanations provides further support that gender differences in heart rate can enhance our understanding of the reasons for the gender gap.

### **Social Influences on Low Heart Rate**

Although we find that resting heart rate differs across gender, we do not rule out the possibility that social environmental factors can also influence heart rate. In contrast to a study in which researchers reported that the heart rate-antisocial behavior relationship in children was entirely explained by genetic influences (Baker et al., 2009), empirical evidence shows that social adversity is associated with a lower heart rate later in life (Choy et al., 2015; Hagedaars, Stins, & Roelofs, 2012; Wadsworth, 1976).

The relationship between social influences and heart rate may be explained by the fact that chronic stress associated with social disadvantage can disrupt brain architecture, which in turn dampens autonomic functioning (McEwen, 1998; Susman, 2006). For example, chronic stress can result in atrophy of neurons in the prefrontal cortex, which can affect cardiovascular functioning (McEwen, 2007; Tavares, Antunes-Rodrigues, & Corrêa, 2004). Therefore, despite findings of gender-related differences in heart rate, according to social neurocriminology, the social environment can also influence autonomic functioning to in turn shape criminal behavior (Choy et al., 2015). The findings here do not undermine the role of sociocultural factors from criminological

perspectives in the overall explanation of the gender gap. Rather, they integrate a biological perspective with the existing literature to provide a more complete explanation.

## **Contributions**

**Broader implications for criminology.** A unique contribution of this study is the finding, for the first time, that a biological factor, low heart rate, mediates in part the relationship between gender and crime in a longitudinal context. It is acknowledged that classic criminological theories were developed to explain male deviance, with little concern for gender differences in offending and that these theories have been applied to understand gender differences in crime retrospectively (De Coster et al., 2012; Smith & Paternoster, 1987). Nevertheless, the lack of formal empirical tests of constructs from major criminological paradigms as mediators of the gender-crime relationship is surprising.

Consistent with the goals concerning increased research on sex differences recently proposed by the National Institutes of Health (2015), beyond heart rate, the broader implication of this study is to encourage more rigorous tests of constructs from criminological theories in longitudinal, mediation models. Strengthening theoretical testing regarding the gender gap can enable us to arrive at more robust empirical evidence for what accounts for the higher levels of male crime, particularly in relation to serious offending.

**Empirical literature on heart rate and crime.** Our finding that a low heart rate in childhood predicts greater involvement in adult criminal behavior contributes to the

extant literature on low heart rate as a risk factor for crime. Longitudinal studies aimed at examining the heart rate-antisocial behavior relationship have an average follow-up period of only 5.93 years (compared with 12.0 years in the current study), and few longitudinal studies have been conducted to assess offending in adulthood (Portnoy & Farrington, 2015). As mentioned, the sensation-seeking and fearlessness theories provide a psychological account for why a low heart rate may act as a risk factor for crime.

Our longitudinal findings on the heart rate-crime relationship that generalize to many forms of criminal offending are bolstered by recent studies from Brazil (Murray et al., 2016) and Sweden (Latvala et al., 2015). Nevertheless, Latvala et al.'s (2015) study was only conducted on males and could not address the current study's primary question on the gender gap. Furthermore, we show here that heart rate at 11 years of age is associated with adult crime, 7 years earlier than in Latvala et al.'s (2015) study, in which heart rate was assessed at 18 years of age. Our study in Mauritius also adds to the evidence for the cross-cultural generalizability of the heart rate-crime relationship. We did, however, find larger effect sizes ( $d = .46$  to  $.62$ ) than that of  $.20$  reported in Portnoy and Farrington's (2015) meta-analysis. This could be, in part, because African populations have not been represented in prior studies, which have focused on Western cultures. In addition, the recent studies of Latvala et al. (2015) and Murray et al. (2016), which had large sample sizes with significant results in the predicted direction, were not included in Portnoy and Farrington's (2015) meta-analysis. Clearly, there is scope for replication and extension of our current findings from Mauritius to Western countries.

## **Limitations**

Despite the contributions of this study, there are several limitations. First, this study only examined resting heart rate and did not assess heart rate during stress. A meta-analysis on low heart rate and antisocial behavior that specifically assessed child and adolescent samples documented a greater effect size for low heart rate measured during a stress task ( $d = -.76$ ) than heart rate at rest ( $d = -.44$ ) (Ortiz & Raine, 2004). Hence, it may be that our results underestimate the mediating role of low heart rate on the gender-crime relationship.

Second, as this study is limited to heart rate, it does not examine the possible contributions of other factors to the gender gap in crime. One example is cortisol as differential reactivity to stress may influence male and female rates of crime (Loney, Butler, Lima, Counts, & Eckel, 2006). Additionally, given evidence that sex hormones such as estrogen influence the gender difference in heart rate (Johnson et al., 1997; Luczak & Leinwand, 2009; Wittnich et al., 2013), sex hormones may affect heart rate levels, which are in turn linked to offending. We are also unable to control for potential confounding psychological variables about which we lack data, such as levels of inhibition. Nevertheless, whether these variables should be controlled for or considered part of the causal chain remain to be elucidated. Future tests of other biological variables as mediators, such as in a serial mediation model, in conjunction with social and psychological variables, may result in greater predictive and explanatory power.

Third, the mediation models presented in this study suggest causality as they reveal that gender determined before birth influences resting heart rate levels at 11 years of age to in turn predispose to criminal behavior in adulthood, yet they do not establish causality. Future studies may be aimed at extending these findings by manipulating a

process linked to heart rate such as empathy or sensation-seeking, which are constructs in social control theory, in an intervention procedure to elucidate better the causal relationship suggested here.

Fourth, the role of sociocultural definitions and expectations about masculinity and femininity (Gartner, 2011; McCarthy & Gartner, 2014) on variation in crime is not assessed.

Lastly, although the heart rate-antisocial behavior relationship documented here has been replicated in more than 12 countries (Raine, 2015), providing support for our results, the generalizability of our mediation results from Mauritius to other contexts remains to be examined.

### **Conclusion**

In conclusion, in this study, we document that lower heart rates in males *partly* explain their higher rates of crime. Estimates of the percentage of the total effect of gender on crime mediated by resting heart rate ranged from 5.4% to 17.1%. It should be reemphasized that heart rate did not completely explain the gender difference in offending. Other social and biological mechanisms likely play important mechanistic roles. Nevertheless, to our knowledge, these findings demonstrate for the first time in a formal mediation test that a biological characteristic can help to account in part for the well-established gender gap in offending. From a social neurocriminology perspective, this raises the conceptual question of whether by incorporating biological variables into explanatory frameworks for the social and demographic causes of crime, extant

criminological theories can provide richer and ultimately more powerful explications of the mechanisms underlying criminal behavior than hitherto.



Paper 3 Tables and Figures

Table 3.1. Descriptive Statistics (N = 894)

	Males		Females		Total		Range
	Mean/ Proportion	SD	Mean/ Proportion	SD	Mean/ Proportion	SD	
<i>Crime variables</i>							
<i>All-source measures</i>							
Overall offending	.55	.50	.11	.31	.35	.48	0 - 1
Violence	.31	.46	.06	.23	.20	.40	0 - 1
Serious violence	.28	.45	.02	.15	.16	.37	0 - 1
Drug offenses	.26	.44	.01	.10	.15	.36	0 - 1
<i>Self-reported offending</i>							
Overall offending	.47	.50	.11	.31	.31	.46	0 - 1
Violence	.28	.45	.06	.23	.18	.38	0 - 1
Serious violence	.25	.43	.02	.15	.15	.35	0 - 1
Drug offenses	.26	.44	.01	.10	.15	.35	0 - 1
Variety score	1.09	2.07	.12	.44	.66	1.64	0 - 15
<i>Official records</i>							
Overall offending	.16	.36	.01	.07	.09	.29	0 - 1
Violence	.05	.21	0	0	.03	.16	0 - 1
Serious violence	.04	.20	0	0	.02	.15	0 - 1
Drug offenses	.01	.10	0	0	.01	.07	0 - 1
Variety score	.22	.69	.01	.08	.13	.52	0 - 8
Heart rate	88.00	13.21	98.12	15.24	92.49	15.01	54.58 - 144.13
<i>Potential confounds</i>							

Social adversity	2.28	1.62	2.65	1.80	2.44	1.71	0 - 8
BMI	14.51	1.82	14.57	2.11	14.54	1.95	10.97 - 26.67
Hyperactivity	-.11	.94	.12	1.02	-.02	.98	-2.54 - 5.11
Race							
Indian	.70		.70		.70		0 - 1
Creole	.24		.24		.24		0 - 1
Other	.06		.06		.06		0 - 1
<hr/> <i>N</i>	497		397		894		

*Note.* *N* for social adversity = 795; *N* for BMI = 792; *N* for hyperactivity = 812; SD = standard deviation; BMI = body mass index.

Table 3.2. Mean Resting Heart Rates at Age 11 Years for Adult Offenders and Non-Offenders, Together with Effect Sizes for Group Differences (Cohen's *d*) (N = 894)

	Offenders		Non-offenders		Cohen's <i>d</i>
	Mean	SD	Mean	SD	
Overall offending	88.20	13.31	94.83	15.37	.46
Violence	86.53	13.30	93.95	15.05	.52
Serious violence	85.33	12.56	93.89	15.05	.62
Drug offenses	86.02	12.84	93.64	15.08	.55

*Note.* SD = standard deviation.

Table 3.3. Total, Direct, and Indirect Mediation Effects of Gender on Self-Reported Measures of Offending through Heart Rate (N = 663)

	<b>Total Effect: Gender on Offending</b>		<b>Direct Effect: Gender on Offending</b>		<b>Mediation Effect: Gender on Offending</b>		
	<b>B</b>	<b>SE</b>	<b>B</b>	<b>SE</b>	<b>B</b>	<b>SE</b>	<b>95% CI</b>
Overall offending	2.12*	.23	1.98*	.24	.15	.07	.02 - .31
Violence	1.80*	.28	1.53*	.29	.32	.09	.16 - .51
Serious violence	2.81*	.47	2.51*	.47	.36	.10	.18 - .58
Drug offenses	3.46*	.59	3.30*	.60	.19	.10	.01 - .39
Variety score	2.33*	.24	2.13*	.25	.20	.08	.07 - .35

*Note.* Models were adjusted for race, social adversity, BMI, and activity level. The PROCESS macro uses listwise deletion based on all variables in the model. Gender was coded as 0 for female and 1 for male. Coefficients for the variety score are in unexponentiated form. As the prevalence of female offending according to official court records was insufficiently common to afford statistical analysis, official records were excluded from mediation analyses. B = unstandardized coefficient; SE = standard error; CI = bias-corrected bootstrap confidence interval.

\* $p < .001$ , two-tailed.

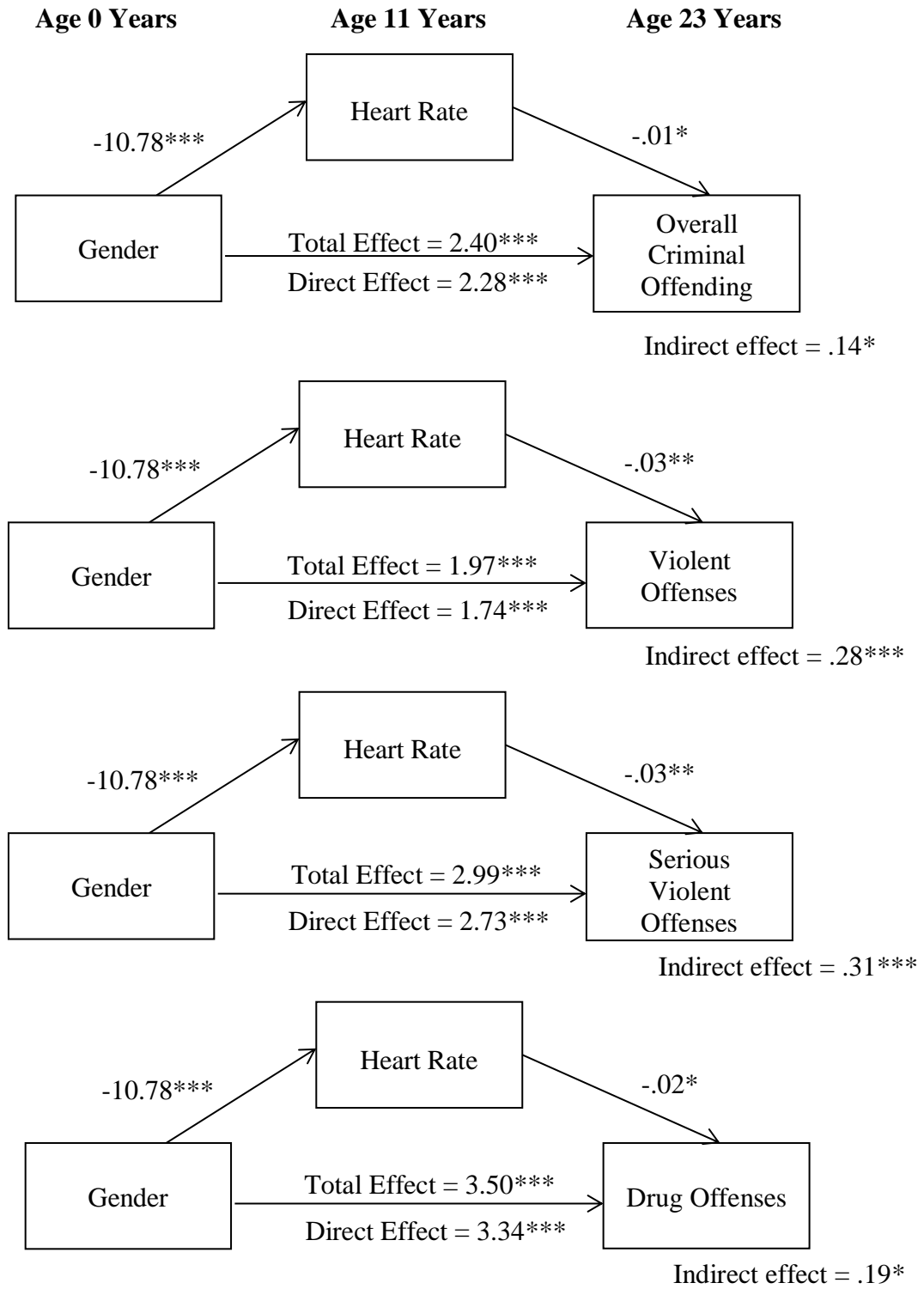
Table 3.4. Total, Direct, and Indirect Mediation Effects of Gender on All-Source Measures of Offending and the Self-Reported Variety Score through Heart Rate After Expectation-Maximization Estimation (N = 894)

	<b>Total Effect: Gender on Offending</b>		<b>Direct Effect: Gender on Offending</b>		<b>Mediation Effect: Gender on Offending</b>		
	<b>B</b>	<b>SE</b>	<b>B</b>	<b>SE</b>	<b>B</b>	<b>SE</b>	<b>95% CI</b>
Overall offending	2.25*	.19	2.14*	.19	.12	.06	.01 - .25
Violence	2.02*	.24	1.83*	.25	.22	.07	.08 - .37
Serious violence	2.78*	.35	2.56*	.36	.26	.08	.12 - .44
Drug offenses	3.57*	.51	3.41*	.52	.18	.08	.04 - .36
Variety score	2.18*	.20	2.00*	.20	.17	.06	.07 - .29

*Note.* Models were adjusted for race, social adversity, BMI, and activity level. Gender was coded as 0 for female and 1 for male. Coefficients for the variety score are in unexponentiated form. B = unstandardized coefficient; SE = standard error; CI = bias-corrected bootstrap confidence interval; Variety score = self-reported variety score.

\* $p < .001$ , two-tailed.

Figure 3.1. Mediation Models for the Effect of Gender on Crime in Adulthood through Heart Rate (N = 663)



*Note.* Models were adjusted for race, social adversity, BMI, and activity level. The PROCESS macro uses listwise deletion based on all variables in the model. Gender was coded as 0 for female and 1 for male.

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

## **PAPER 4. VITAMIN D AS A PROTECTIVE FACTOR FOR CHILD ANTISOCIAL BEHAVIOR**

### **Abstract**

Vitamin D insufficiency and child antisocial behavior are public health concerns that are associated with mental and physical health problems. It is unknown whether vitamin D plays a role in antisocial outcomes and there is a need to investigate protective factors that may reduce antisocial behavior in children. This study examines whether higher levels of vitamin D can protect children who are exposed to early social adversity from antisocial behavior. The Healthy Brains and Behavior Study recruited children from the community in Philadelphia between 2009 and 2012. In a sample of 300 children aged 11-12 years (151 females, 149 males), serum concentrations of 25-hydroxyvitamin D [25(OH)D] were assessed alongside social adversity determined from interviews with caregivers, and parent and child-reported antisocial behavior. Vitamin D moderated the association between early social adversity and both parent and child-reported antisocial behavior. Higher social adversity was associated with increased antisocial behavior among vitamin D-insufficient [25(OH)D < 30 ng/mL], but not vitamin D-sufficient children [25(OH)D ≥ 30 ng/mL], after adjusting for other variables. At serum vitamin D concentrations above 27.16 - 30.69 ng/mL, the effect of social adversity on child antisocial behavior outcomes was nullified. To our knowledge, this study is the first to document a protective effect of vitamin D on antisocial behavior. Our findings in a pediatric population suggest a possible role of vitamin D supplementation in interventions to reduce antisocial behavior. They are consistent with the broader literature



on the clinical and overall health benefits of vitamin D, as well as evidence that the most advantageous vitamin D concentrations begin at 30ng/mL.

## Background

Childhood antisocial behavior is a public health concern. Antisocial children are at greater risk for mental health problems including internalizing psychopathology (Odgers et al., 2007; van der Molen et al., 2015) and substance abuse (Bardone et al., 1998; van der Molen et al., 2015), physical health problems such as respiratory problems (Odgers et al., 2007; Paradis, Koenen, Fitzmaurice, & Buka, 2016) and sexually transmitted diseases (Bardone et al., 1998), poorer well-being (von Stumm et al., 2011), and premature mortality (Jokela, Ferrie, & Kivimäki, 2009). Individuals who experience social adversity early in life are at an elevated risk for antisocial behavior (Duke, Pettingell, McMorris, & Borowsky, 2010; Piotrowska, Stride, Croft, & Rowe, 2015). This may be due to the chronic strain from early social adversity that can exert a biological burden on the body (McEwen, 1998). For example, the literature on allostatic load documents that the most consistent neurobiological findings in children with adverse psychosocial experiences are linked to impaired prefrontal cortical functioning (Danese & McEwen, 2012), which is a risk factor for antisocial behavior (Yang & Raine, 2009).

However, some children may develop resilience to the effects of adversity. For instance, good child-rearing (Farrington & Ttofi, 2011), high non-verbal intelligence, (Ttofi et al., 2016) positive peer relationships (Criss, Pettit, Bates, Dodge, & Lapp, 2002; Lansford, Criss, Pettit, Dodge, & Bates, 2003), higher heart rate (Farrington, 1997), and a high activity monoamine oxidase-A genotype (Kim-Cohen et al., 2006) constitute buffering protective factors that moderate the association between psychosocial risk factors and antisocial behavior. Despite the need to identify protective factors that may support resilience, scientific foundation for the notion that youth in high-risk

environments can be protected from engaging in antisocial behavior remains limited (Hall et al., 2012). This is especially salient as the estimated cost of saving a high-risk youth at age 10 years is \$3.2-\$5.5 million (Cohen & Piquero, 2009). Moreover, prevention and intervention efforts that seek to reduce antisocial behavior are least costly and most effective if they are implemented in childhood (Conduct Problems Prevention Research Group, 2015; Frick, 2016).

One candidate protective factor is vitamin D. Vitamin D serves as a vitamin and prohormone as it can be ingested orally and produced endogenously in the skin by ultraviolet B conversion. In the pediatric population, insufficient vitamin D is associated with increased risk of illnesses including tuberculosis (Williams, Williams, & Anderson, 2008), type 1 diabetes (Hyppönen, Läärä, Reunanen, Järvelin, & Virtanen, 2001), cystic fibrosis (Green et al., 2008), and respiratory tract infections (Esposito & Lelii, 2015), as well as neurodevelopmental disorders such as autism (Mazahery et al., 2016), schizophrenia (McGrath, Burne, Féron, Mackay-Sim, & Eyles, 2010), and attention deficit hyperactivity disorder (ADHD; Kamal, Bener, & Ehlayel, 2014). Low vitamin D status is thus acknowledged as a worldwide health problem (Hosseini-nezhad & Holick, 2013; Palacios & Gonzalez, 2014). A possible protective effect of vitamin D on antisocial behavior is supported by evidence that vitamin D is a regulator of brain development and function through its role in calcium signaling, neurotrophic actions, and neuronal differentiation, maturation, and growth (Kalueff & Tuohimaa, 2007; Mackay-Sim, Féron, Eyles, Burne, & McGrath, 2004; Groves, McGrath, & Burne, 2014). Vitamin D has also been suggested to have a neuroprotective effect and may protect against the damaging neurobiological effects of stress (Groves et al., 2014). Although multivitamin and mineral

supplementations have been found to reduce antisocial behavior among children (Benton, 2007) and low vitamin D intake levels have been observed among prisoners (Eves & Gesch, 2003), it is unknown whether vitamin D has protective effects on antisocial behavior in children. This study sought to contribute to the literature on resilience to child antisocial behavior and more specifically, on outcomes related to vitamin D insufficiency.

## **Methods**

### **Participants**

The Healthy Brains and Behavior Study recruited 454 children aged 11 and 12 years from the city of Philadelphia and contiguous suburbs between 2009 and 2012. Recruitment involved targeted mailings, flyers in the community, advertisements, personal referrals, and enrollment through health care providers. Children were excluded if they had a psychotic disorder, mental retardation, or pervasive developmental disorder diagnosis, a seafood allergy, or medication that may modify lipid metabolism. Eighteen participants were found to be ineligible or withdrew from the study. Additional information about participant recruitment and exclusionary criteria have been reported. (Liu et al., 2013; Richmond, Cheney, Soyfer, Kimmel, & Raine, 2013)

A total of 300 children (151 females, 149 males) who had complete data on the key study variables, namely vitamin D, social adversity, and antisocial behavior were included in the analysis. The sample was predominantly African American (80.0%). 11.3% were Caucasian and 8.3% were of other ethnicities (Asian American, Native American, Hispanic, or multiracial). One participant declined to respond. The mean age

of the sample was 11.44 (SD = .50) years. No significant differences in demographic characteristics, antisocial behavior, social adversity, or vitamin D levels were observed between individuals who were included in the analysis and those who were not ( $p > .05$ ). The caregiver of each child also participated in the study as an informant for the child's behavior. Caregiver participation primarily involved the biological mothers (90.7%). Written informed consent and assent were obtained from parents and children, and the study protocols were approved by the Institutional Review Board of the University of Pennsylvania and the Philadelphia Department of Health.

## **Measures**

**Social adversity.** Following recommendations for the assessment of adverse childhood experiences, a social adversity index that encompassed a range of psychosocial factors was used to accommodate the breadth of adversity experienced by children (Wade, Shea, Rubin, & Wood, 2014). The scale reflected early childhood adversity that is associated with long-term stress. Based on responses to demographic items obtained from an interview with caregivers, a total social adversity score was calculated by adding 1 point for each of the following indicators: mother's low education, father's low education, parent unemployment, teenage pregnancy (mother aged 20 years or younger at child's birth), problems with living accommodation, five or more siblings, not living with both biological parents, early maternal separation between age 6 months and 2.5 years, absence of child supervision at home, living in government housing, more than one person per room, moved 4 or more times in the past 12 years, mother's physical illness, father's physical illness, mother's mental illness, father's mental illness, father had been

detained or imprisoned, mother had been arrested (Choy et al., 2015). Higher scores reflected greater social adversity. For this scale, a simple imputation procedure was used to predict social adversity scores for cases with missing values. Full details of the procedure have been reported (Choy et al., 2015).

**Vitamin D.** 25-hydroxyvitamin D [25(OH)D], in nanograms per milliliter (ng/mL), was measured from blood samples collected from participants. Serum 25(OH)D concentration is widely-acknowledged to be the best indicator of vitamin D status (Holick et al., 2011; Thatcher & Clarke, 2011). Based on the distribution of vitamin D levels, 3 outliers greater than 3 standard deviations above the mean were identified and rescaled by assigning them to the nearest value lying within the valid, non-outlying distribution. This Winsorization approach has been recommended as effective in maximizing power and the accuracy of standard errors (Erceg-Hurn & Mirosevich, 2008).

Vitamin D sufficiency was defined as 25(OH)D  $\geq$  30 ng/mL (75nmol/L). Children with 25(OH)D levels below 30 ng/mL were considered vitamin D-insufficient. This definition of an optimal level of vitamin D is consistent with recommendations provided for pediatric populations (Cole et al., 2010; Esposito & Lelii, 2015; Godel et al., 2007; Lautenbacher, Jariwala, Markowitz, & Rastogi, 2016; Thorisdottir et al., 2016; Walker & Modlin, 2009; Williams et al., 2008).

**Child antisocial behavior.** Antisocial behavior was reported by participants and their caregivers. Children completed 5 questionnaires that yielded 14 subscores. The Buss-Perry Aggression Questionnaire produced scores for physical aggression, verbal

aggression, anger, hostility, and indirect aggression (Bryant & Smith, 2001; Buss & Perry, 1992). The Reactive-Proactive Aggression Questionnaire yielded scores for reactive and proactive aggression (Raine et al., 2006). The Youth Self-Report (YSR) was used to assess the rule-breaking and aggressive behavior syndromes (Achenbach & Rescorla, 2001). The Antisocial Personality Screening Device (APSD) evaluated three dimensions of child psychopathy: callous-unemotional traits, narcissism, and impulsivity (Frick & Hare, 2001). The Conduct and Oppositional Defiant Disorder Scale (CODDS), modeled on the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) symptoms, assessed conduct disorder and oppositional defiant disorder (Raine, 2008).

Caregivers completed 4 assessments about their child's behavior, comprising 9 subscales. Parents responded to the supplement of the YSR, the Child Behavior Checklist (Achenbach & Rescorla, 2001), as well as the APSD and CODDS (Frick & Hare, 2001; Raine, 2008). In addition to the questionnaires, parents were interviewed by trained research assistants using the National Institute of Mental Health Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV), to assess symptoms of conduct disorder and oppositional defiant disorder (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000).

Full details for the factor analyses that were conducted separately for child and parent reports have been documented elsewhere (Raine et al., 2016). The overall measures of parent-reported and child-reported antisocial behavior, and two subfactors obtained from the child reports (aggressive-reactive and callous-proactive), were examined.

**Covariates.** Demographic characteristics, sex (1 = male, 0 = female) and race (1 = African American, 0 = non-African American) were included in all analyses. Body mass index (BMI), intelligence, ADHD, and calcium levels were examined as possible confounders. Despite being determinants of serum vitamin D concentration, season of serum collection and skin pigmentation were also considered as sources of potential confounding as they may affect antisocial behavior through pathways independent from vitamin D (Ascherio, Munger, & Simon, 2010; Brehm et al., 2010; Ross et al., 2011).

BMI in kilograms/m<sup>2</sup> was calculated from the average of two height and weight measurements conducted at a hospital and by a research assistant at the testing site. Intelligence was assessed based on raw scores for the digit span, coding, vocabulary, and matrix reasoning tasks in the Wechsler Intelligence Scale for Children (Wechsler, 2003). Diagnoses of ADHD in the past year were made using the NIMH DISC-IV (Shaffer et al., 2000). As low calcium intake may be associated with vitamin D insufficiency (Steingrimsdottir, Gunnarsson, Indridason, Franzson, & Sigurdsson, 2005), the average daily consumption of calcium, in milligrams, was assessed via three interviews using the dietary recall procedure of the National Health and Nutrition Examination Survey. Consistent with other studies, season of serum collection was defined as winter (December to February), spring (March to May), summer (June to August), and fall (September to November) (Bodnar et al., 2014; Dong et al., 2010). Skin color was rated by separate researchers based on an 11-point scale (Massey & Martin, 2003). Ratings were highly correlated ( $r = .92$ ). Higher ratings denoted darker skin color.

## **Statistical Analysis**



Bivariate associations were assessed using Pearson correlations, independent *t*-tests, chi-square tests, and one-way analysis of variance as appropriate to the nature and distribution of the variables. Variables were included in subsequent analyses as covariates if they were significantly associated with an antisocial outcome and one of the main predictors (social adversity or vitamin D insufficiency).

To assess whether vitamin D moderated the social adversity-antisocial behavior relationship, a regression-based model including the interaction term, social adversity x vitamin D insufficiency, was tested using the PROCESS macro on SPSS (Hayes, 2013). In sensitivity analysis, interaction effects were assessed using vitamin D as a continuous variable. The continuous variables were centered prior to all moderation analyses. The Johnson-Neyman technique was used to identify the level of vitamin D at which social adversity no longer significantly affects antisocial behavior (Hayes, 2013; Johnson & Fay, 1950). Furthermore, due to the large proportion of African Americans in the sample, a subanalysis was performed to examine moderation effects among African Americans specifically. Statistical analyses were conducted using SPSS, version 24.0 (IBM Corp).

## **Results**

The mean serum vitamin D concentration was 24.22 ng/mL (Table 4.1). 74.7% of the sample had vitamin D levels lower than 30 ng/mL. The descriptive statistics for all of the study variables are presented in Table 4.1. As intelligence, ADHD diagnosis, and skin tone were the only hypothesized confounders to be significantly associated with antisocial behavior and one of the main predictors (Table 4.2), they were included as covariates in the moderation analyses.

### **Moderating Effect of Vitamin D on Antisocial Behavior**

A significant interaction effect of social adversity x vitamin D insufficiency ( $b = .14$ ,  $SE = .04$ ,  $t = 3.17$ ,  $p = .002$ ) was observed for parent-reported overall antisocial behavior (Table 4.3). Among children with vitamin D insufficiency, greater social adversity was associated with higher levels of parent-reported antisocial behavior ( $b = .07$ ,  $SE = .02$ ,  $t = 4.16$ ,  $p < .001$ ; Figure 4.1). However, social adversity was not associated with antisocial behavior for children who met the threshold for vitamin D sufficiency ( $b = -.03$ ,  $SE = .04$ ,  $t = -.75$ ,  $p = .46$ ). The results suggest that having sufficient vitamin D is associated with nullification of the effect of social adversity on antisocial behavior.

Significant interactions between social adversity and vitamin D insufficiency were also detected in predicting self-reported overall antisocial behavior ( $b = .11$ ,  $SE = .04$ ,  $t = 2.63$ ,  $p = .01$ ) and the aggressive-reactive ( $b = .12$ ,  $SE = .05$ ,  $t = 2.60$ ,  $p = .01$ ) and callous-proactive factors ( $b = .10$ ,  $SE = .05$ ,  $t = 2.13$ ,  $p = .03$ ). Children with insufficient vitamin D exhibited increases in self-reported antisocial behavior when they experienced social adversity early in life, while vitamin D-sufficient children displayed non-significant changes in antisocial behavior as a function of social adversity (Figure 4.1).

When vitamin D was assessed as a continuous variable, results did not substantively change. The interaction effects were significant in predicting parent-reported antisocial behavior ( $b = -.01$ ,  $SE = .002$ ,  $t = -2.99$ ,  $p < .01$ ), child-reported overall antisocial behavior ( $b = -.01$ ,  $SE = .002$ ,  $t = -2.78$ ,  $p < .01$ ), aggressive-reactive

scores ( $b = -.005$ ,  $SE = .002$ ,  $t = -2.46$ ,  $p = .01$ ), and callous proactive scores ( $b = -.01$ ,  $SE = .002$ ,  $t = -2.89$ ,  $p < .01$ ). The Johnson-Neyman technique showed that when children have vitamin D concentrations above 28.94 ng/mL, social adversity is no longer significantly related to parent-reported antisocial behavior (Figure 4.2). As vitamin D levels decrease, the relationship between social adversity and antisocial behavior becomes more positive. The effect of social adversity on child-reported overall antisocial behavior, aggressive-reactive behavior, and the callous-proactive factor transitions to non-significance at vitamin D levels of 28.88 ng/mL, 27.16 ng/mL, and 30.69 ng/mL respectively.

In analyses restricted to African Americans, the pattern of findings from the main analyses persisted (Figure 4.3). The influence of early social adversity on antisocial behavior was observed among vitamin D-insufficient children, but not among those meeting vitamin D sufficiency. However, while the interaction effect of social adversity x vitamin D insufficiency remained significant for parent-reported antisocial behavior ( $b = .16$ ,  $SE = .06$ ,  $t = 2.78$ ,  $p < .01$ ), child-reported overall antisocial behavior ( $b = .14$ ,  $SE = .06$ ,  $t = 2.35$ ,  $p = .02$ ), and the aggressive-reactive factor ( $b = .15$ ,  $SE = .06$ ,  $t = 2.35$ ,  $p = .02$ ), it was attenuated for the callous-proactive factor in the African American subsample ( $b = .12$ ,  $SE = .06$ ,  $t = 1.87$ ,  $p = .06$ ; Table 4.4).

## Discussion

This study tested the hypothesis that vitamin D sufficiency confers resilience to child antisocial behavior. Our results show a protective-stabilizing effect of vitamin D (Luthar, Cicchetti, & Becker, 2000) as having sufficient vitamin D helped to neutralize

the association between early social adversity and antisocial behavior. Children with high adversity and vitamin D sufficiency had similar levels of antisocial behavior as individuals without social risk. Specifically, the effect of social adversity on child antisocial behavior outcomes became non-significant when vitamin D concentrations were above 27.16 - 30.69 ng/mL. The examination of the vitamin D levels required for optimum protective effects can have public health implications. Although the threshold reflecting vitamin D sufficiency is highly debated, our findings are consistent with the evidence that the most advantageous serum vitamin D concentrations begin at 30ng/mL and support guidelines for vitamin D sufficiency to be defined as 25(OH)D  $\geq$  30 ng/mL (Bischoff-Ferrari, Giovannucci, Willett, Dietrich, & Dawson-Hughes, 2006; Holick et al., 2011).

The consistent pattern of results found across multiple informants and for different antisocial outcomes add credence to our findings. Prior studies have found low to moderate levels of agreement between caregiver and children reports (Achenbach, McConaughy, & Howell, 1987; Shakoor et al., 2011). Despite the variations between reports by different informants, vitamin D served a protective function against overall antisocial behavior. Additionally, there has been little research on whether protective effects operate in the same way across races (Jolliffe, Farrington, Loeber, & Pardini, 2016). Sensitivity analyses provided evidence that the buffering protective effect of vitamin D sufficiency persisted in a subsample of African Americans.

Several mechanisms might be responsible for a protective role of vitamin D on antisocial behavior. One process is through brain development and functioning (Groves et al., 2014). Early deprivation may disrupt brain development and neuronal functioning

that are relevant for the regulation of behavior, and vitamin D may be able to counter these effects. Another mechanism may be the regulation of serotonin levels. This may occur through the role of vitamin D in activating the transcription of tryptophan hydroxylase 2 (Patrick & Ames, 2015). Vitamin D may contribute to the adaptation of stress in this way as higher serotonin levels are associated with lower antisocial behavior (Moore, Reise, Gur, Hakonarson, & Gur, 2015; Moul, Dobson-Stone, Brennan, Hawes, & Dadds, 2013). Future studies including neuroimaging data may provide greater insight into the vitamin D-antisocial behavior relationship.

There are several limitations to this study. The cross-sectional analysis limited our ability to determine the temporal relationship between vitamin D and antisocial behavior and to investigate longer-lasting protective effects of vitamin D on behavior. Conclusive recommendations based on these findings await the outcome of randomized controlled trials (RCTs) among individuals predisposed to antisocial behavior as well as in antisocial populations. Additionally, although we attempted to include many covariates related to social adversity, vitamin D, and antisocial behavior, our effect estimates may have been influenced by other unmeasured or unknown confounders. We were unable to adjust for variables for which we lacked data, such as clothing practices or use of sunscreen, which could affect serum vitamin D levels. Nevertheless, as a first investigation of the protective function of vitamin D on antisocial behavior, the findings here are hypothesis-generating and can be used to develop protocols for RCTs. They suggest that there is a public health case to be made for undertaking exploratory trials involving an easily modifiable, safe, and publicly accessible resource.

## **Conclusion**

Vitamin D insufficiency is surprisingly common. The high prevalence of vitamin D insufficiency observed in this study is in line with prevalence rates from nationally representative samples of US children, which have documented 73% of children aged 6 to 11 years (Mansbach, Ginde, & Camargo Jr, 2009) and 70% of individuals aged 1 to 21 years (Kumar, Muntner, Kaskel, Hailpern, & Melamed, 2009) to have vitamin D levels below 30 ng/mL. Consistent findings of a buffering protective effect of vitamin D across multiple antisocial outcomes suggests a potential role for vitamin D supplementation in efforts to reduce antisocial behavior, particularly in children with social risk. Besides being essential for overall health and well-being (Holick, 2004), serum 25(OH)D levels above 30 ng/mL may have a beneficial influence on behavior.

Paper 4 Tables and Figures

Table 4.1. Descriptive Statistics and Differences Between Vitamin D Sufficient and Insufficient Groups

	<b>Total sample</b>	<b>Vitamin D insufficient</b>	<b>Vitamin D sufficient</b>		
	Mean/Proportion (SD)	Mean/Proportion (SD)	Mean/Proportion (SD)	Range	<i>p</i>
Parent report	.17 (.82)	.17 (.84)	.18 (.78)	-1.03 – 3.09	.91
Overall antisocial behavior					
Child report					
Overall antisocial behavior	.01 (.72)	.06 (.75)	-.15 (.57)	-1.29 – 2.88	.01
Aggressive-reactive	.01 (.76)	.06 (.78)	-.15 (.68)	-1.53 – 2.16	.03
Callous-proactive	.01 (.78)	.06 (.85)	-.13 (.53)	-.99 – 4.49	.02
Social adversity	3.91 (2.36)	4.08 (2.36)	3.42 (2.32)	0 – 10.13	.04
Vitamin D	24.22 (10.71)	19.32 (6.38)	38.65 (7.34)	6.23 – 53.90	.00
Covariates					
BMI	21.87 (5.79)	22.66 (5.99)	19.57 (4.46)	13.24 – 48.76	.00
Intelligence	36.25 (7.53)	35.65 (7.26)	37.97 (8.05)	16.00 – 56.00	.02
ADHD	.17 (.37)	.14 (.35)	.25 (.44)	0 – 1	.02
Calcium	837.56 (337.42)	813.83 (323.75)	907.14 (368.20)	166.22 – 2148.31	.04
Skin tone	5.29 (2.20)	5.66 (1.81)	4.24 (2.81)	1 – 9	.00
Season					
Winter	.17	.20	.09	0 – 1	.00
Spring	.29	.34	.15	0 – 1	
Summer	.33	.25	.55	0 – 1	
Fall	.21	.21	.21	0 – 1	
Sex	.50	.56	.34	0 – 1	.00

Race	.80	.88	.55	0 – 1	.00
N	300	224	76		

*Note.* Vitamin D = serum vitamin D; BMI = body mass index; Season = season of serum collection; Sex = 1 if male; Race = 1 if African American. N for BMI = 296; N for intelligence = 294; N for ADHD = 295; N for calcium = 291; N for skin tone = 267.



Table 4.2. Bivariate Correlations Between Key Study Variables and Covariates

<b>Variable</b>	<b>BMI</b>	<b>Intelligence</b>	<b>ADHD</b>	<b>Calcium</b>	<b>Skin tone</b>	<b>Season</b>	<b>Sex</b>	<b>Race</b>
Parent report								
Overall antisocial behavior	.002	-.25**	.40**	.09	.16**	.05	.17**	.07
Child report								
Overall antisocial behavior	.10	-.10	.05	.06	.08	.06	.07	.03
Aggressive-reactive	.10	-.02	.02	.07	.05	.04	.02	.01
Callous-proactive	.07	-.27**	.11	.02	.13*	.10	.15*	.08
Social adversity	.06	-.30**	.14*	-.09	.21**	.07	-.03	.04
Vitamin D insufficiency	.23**	-.14*	.13*	-.12*	.29**	-.17**	-.19**	.35**

*Note.* N = 267 - 300. BMI = body mass index; ADHD = 1 if ADHD is present; Season = season of serum collection; Sex = 1 if male; Race = 1 if African American; Vitamin D insufficiency = 1 if 25(OH)D < 30ng/mL.

\* $p < .05$ ; \*\* $p < .01$ .

Table 4.3. Regression Coefficients for the Effects of Social Adversity, Vitamin D Insufficiency, and their Interaction on Antisocial Behavior Outcomes

	<b>Parent report</b>		<b>Child report</b>	
	Overall antisocial behavior	Overall antisocial behavior	Aggressive-reactive	Callous-proactive
	b (SE)	b (SE)	b (SE)	b (SE)
Social adversity	-.03 (.04)	-.02 (.04)	-.03 (.04)	.01 (.04)
Vitamin D	-.02 (.11)	.26* (.11)	.30* (.12)	.18 (.12)
Social adversity x Vitamin D	.14** (.04)	.11** (.04)	.12* (.05)	.10* (.05)

*Note.* N = 257 as the PROCESS macro uses listwise deletion based on all variables in the model. Models were adjusted for sex, race, intelligence, ADHD, and skin tone. Sex was coded as 0 for female and 1 for male. Race was coded as 0 for non-African Americans and 1 for African Americans. b = unstandardized coefficient; SE = standard error. Vitamin D = 1 if 25(OH)D < 30ng/mL.

\* $p < .05$ ; \*\* $p < .01$ .

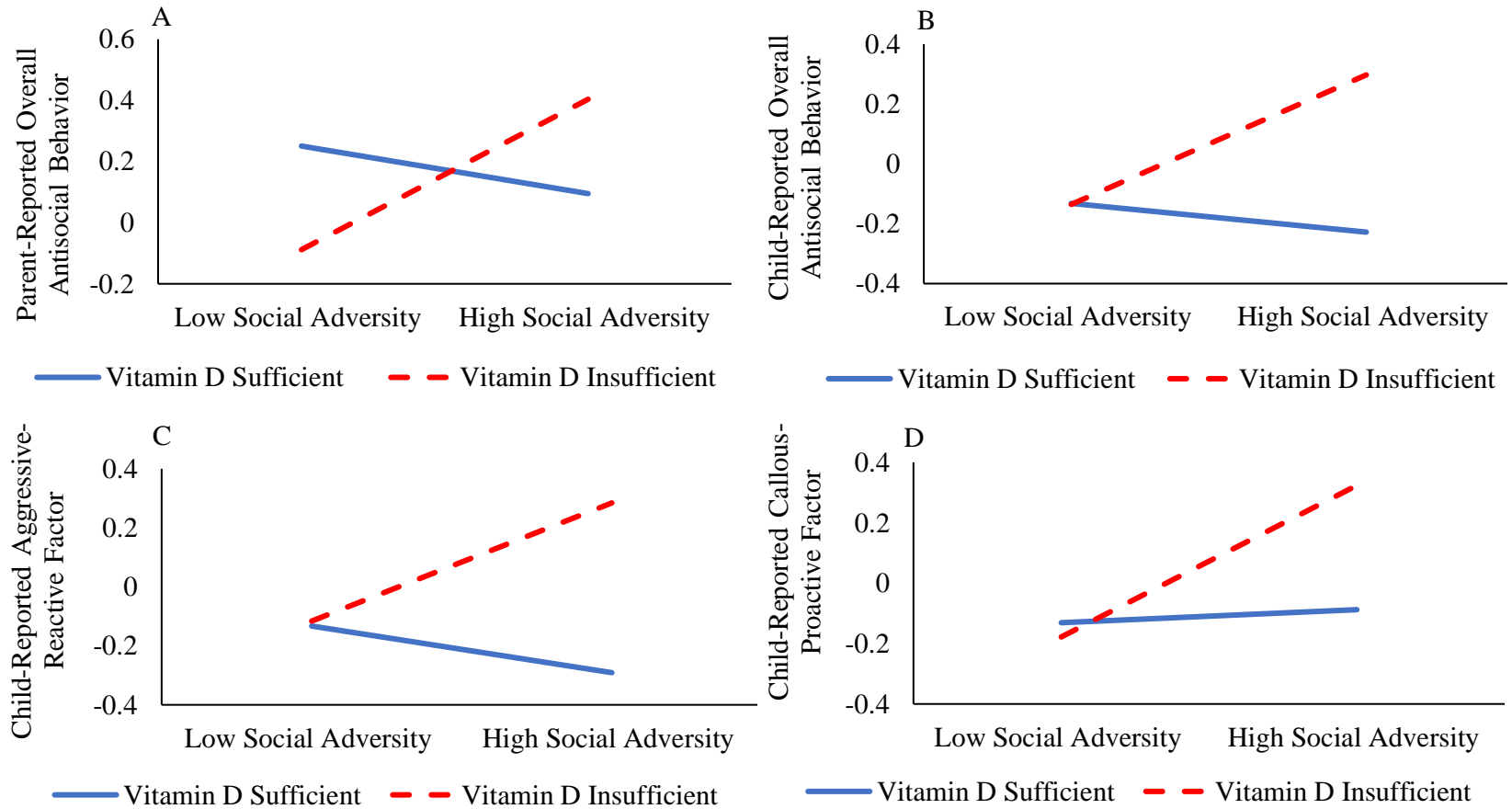
Table 4.4. Regression Coefficients for the Effects of Social Adversity, Vitamin D Insufficiency, and their Interaction on Antisocial Behavior Outcomes in a Subsample of African American Children

	<b>Parent report</b>		<b>Child report</b>	
	Overall antisocial behavior	Overall antisocial behavior	Aggressive- reactive	Callous- proactive
	b (SE)	b (SE)	b (SE)	b (SE)
Social adversity	-.04 (.05)	-.04 (.05)	-.06 (.06)	-.01 (.06)
Vitamin D	.03 (.13)	.36** (.14)	.41** (.14)	.26 <sup>†</sup> (.15)
Social adversity x Vitamin D	.16** (.06)	.14* (.06)	.15* (.06)	.12 <sup>†</sup> (.06)

*Note.* N = 204 as the PROCESS macro uses listwise deletion based on all variables in the model. Models were adjusted for sex, intelligence, ADHD, and skin tone. Sex was coded as 0 for female and 1 for male. b = unstandardized coefficient; SE = standard error. Vitamin D = 1 if 25(OH)D < 30ng/mL.

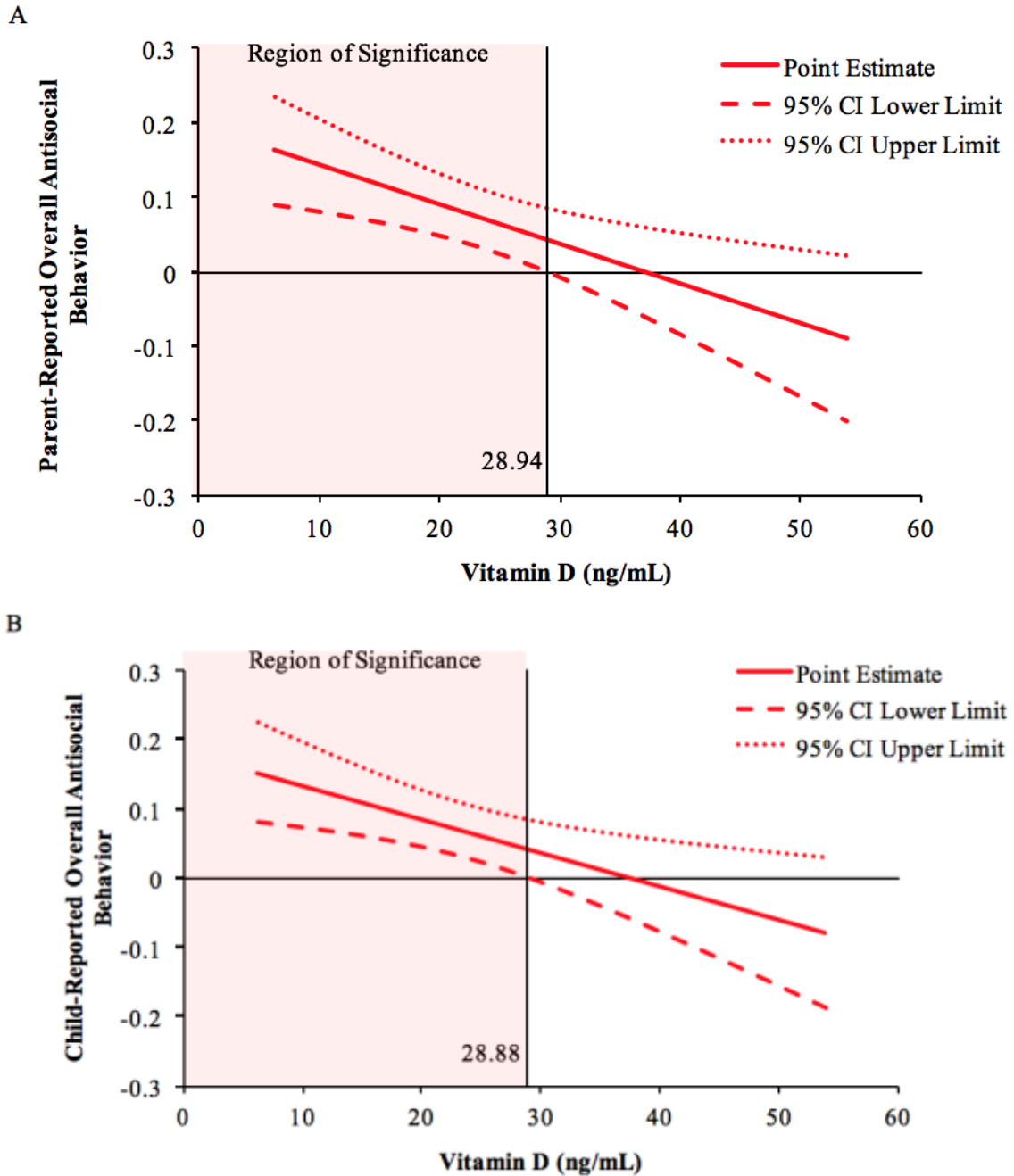
<sup>†</sup> $p < .10$ ; \* $p < .05$ ; \*\* $p < .01$ .

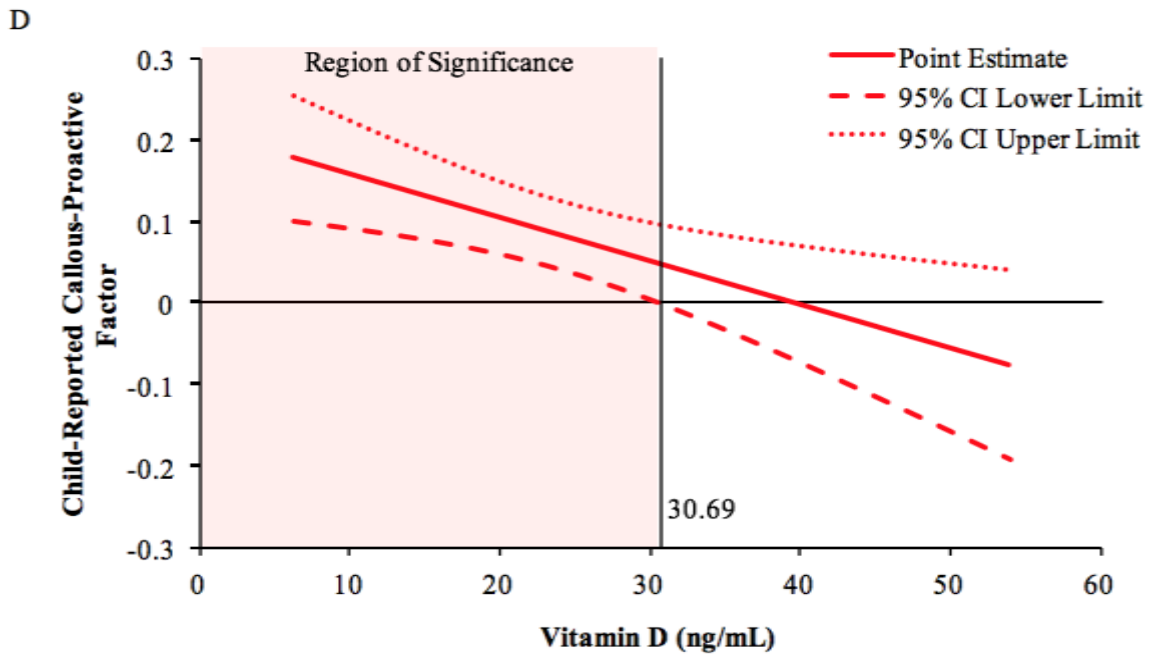
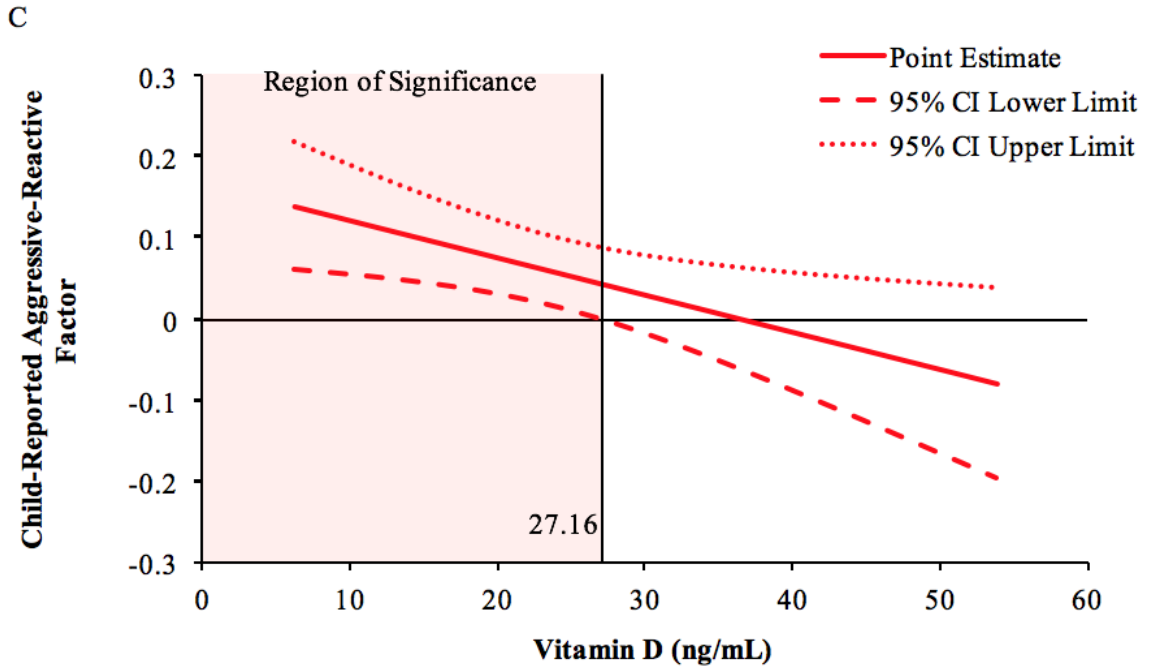
Figure 4.1. Effect of Social Adversity and Serum Vitamin D Level (Sufficient/Insufficient) on (A) Parent-Reported Antisocial Behavior and Child-Reported (B) Overall Antisocial Behavior, (C) Aggressive-Reactive Behavior, and (D) Callous-Proactive Behavior Scores



*Note.* Social adversity groups are 1 SD above and below the mean. Models were adjusted for sex, race, intelligence, ADHD, and skin tone.

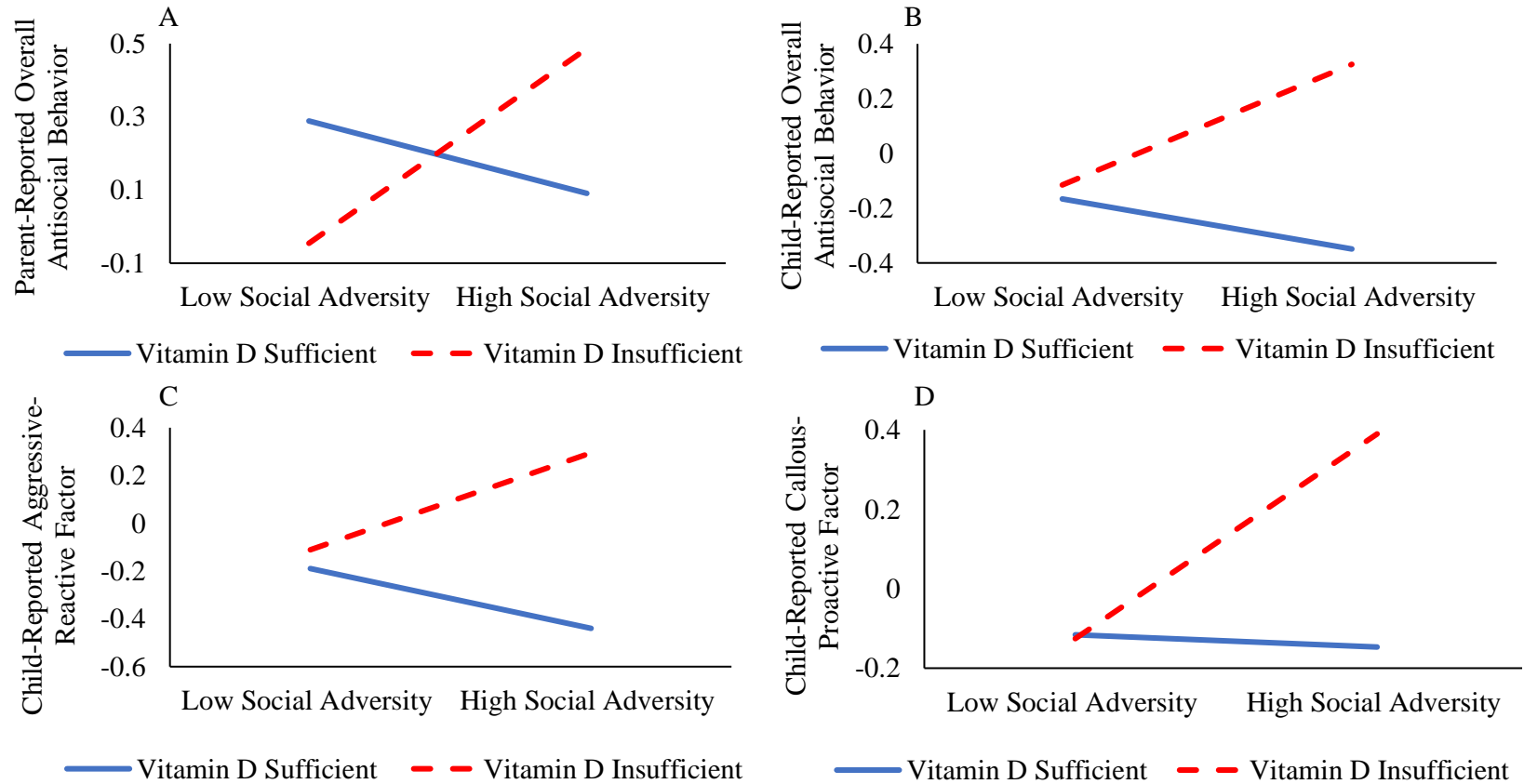
Figure 4.2. Johnson-Neyman Plot of the Region of Significance for the Conditional Effect of Social Adversity on (A) Parent-Reported Antisocial Behavior and Child-Reported (B) Overall Antisocial Behavior, (C) Aggressive-Reactive Behavior, and (D) Callous-Proactive Behavior Scores across the Range of Vitamin D Levels





*Note.* Dashed lines represent the upper and lower limits of the 95% confidence interval. The horizontal line denotes a conditional effect of 0. The vertical line represents the boundary of the region of significance ( $p < .05$ ), which is delineated by the shaded area.

Figure 4.3. Effect of Social Adversity and Serum Vitamin D Level (Sufficient/Insufficient) on (A) Parent-Reported Antisocial Behavior and Child-Reported (B) Overall Antisocial Behavior, (C) Aggressive-Reactive Behavior, and (D) Callous-Proactive Behavior Scores in a Subsample of African American Children



*Note.* Social adversity groups are 1 SD above and below the mean. Models were adjusted for sex, intelligence, ADHD, and skin tone.

## **PAPER 5. STIMULATION OF THE PREFRONTAL CORTEX REDUCES INTENTIONS TO COMMIT AGGRESSION: A RANDOMIZED CLINICAL TRIAL**

### **Abstract**

Although prefrontal brain impairments are one of the best-replicated brain imaging findings in relation to aggression, little is known about the causal role of this brain region. This study tests whether stimulating the dorsolateral prefrontal cortex (DLPFC) using transcranial direct current stimulation (tDCS) reduces the likelihood of engaging in aggressive acts, and the mechanism underlying this relationship. In a double-blind, stratified, placebo-controlled, randomized trial, 81 adults (36 males, 45 females) were randomly assigned to an active ( $N = 39$ ) or placebo ( $N = 42$ ) condition, and followed up one day after the experiment session. Intentions to commit aggressive acts and behavioral aggression were assessed using hypothetical vignettes and a behavioral task, respectively. The secondary outcome was the perception of the moral wrongfulness of the aggressive acts. Participants who received anodal stimulation reported being less likely to commit physical and sexual assault ( $p < .01$ ), and judged aggressive acts as more morally wrongful ( $p < .05$ ) compared to the sham controls. 31% of the total effect of tDCS on intentions to commit aggression was accounted for by perceptions of greater moral wrongfulness regarding the aggressive acts. Results provide the first experimental evidence that increasing activity in the prefrontal cortex can reduce intentions to commit aggression and enhance perceptions of moral judgment. Findings shed light on the biological underpinnings of aggression and theoretically have the potential to inform future interventions for aggression and violence in clinical populations.



## **Background**

Aggression, fighting, and anger are diagnostic features of some mental disorders, including antisocial personality disorder, intermittent explosive disorder, conduct disorder, and borderline personality disorder (American Psychiatric Association, 2013). Aggressive and violent behavior, including sexual assault, have also been documented to be significantly increased among individuals with substance use disorders, bipolar disorder, schizophrenia, major depressive disorder, panic disorder with agoraphobia, and paranoid, schizoid, histrionic, and obsessive-compulsive personality disorders (Pulay et al., 2008; Volavka, 2013). Recent proposals have been made for a symptom-based research approach in psychiatry, which emphasizes the assessment of groups according to predominant psychopathological symptoms and the examination of etiological processes for these distinct symptoms (Fleeson, Furr, & Arnold, 2010; Schmidt, 2015). This perspective is supported by evidence that individual symptoms of mental disorders can have clinical significance on their own (Ellison, Rosenstein, Chelminski, Dalrymple, & Zimmerman, 2016). In line with this psychiatric research approach, there is a pressing need for a better understanding of the determinants of aggression and the investigation of cost-effective interventions that may reduce this symptom.

Prefrontal brain impairment is one of the best-replicated risk factors for aggressive behavior. Evidence from neurological research shows that patients with damage to the frontal cortex exhibit more aggressive and violent behavior (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Grafman et al., 1996; Heinrichs, 1989). In addition to head injury and lesion studies, the imaging and neuropsychological literature has documented structural and functional prefrontal deficits in antisocial individuals

(Brower & Price, 2001; Yang & Raine, 2009). Findings on the role of the frontal cortex in modulating aggression and violence also extend to sexual offending. Rapists show decreased fractional anisotropy (connectivity) in white matter near the medial frontal pole compared to matched controls (Chen et al., 2016). Within the prefrontal cortex, a meta-analysis of 43 imaging studies found that impairments of the dorsolateral prefrontal cortex (DLPFC) are particularly implicated in antisocial behavior (Yang & Raine, 2009). This may be due to the DLPFC's broad connection to functions related to aggression, including moral judgment (Jeurissen, Sack, Roebroek, Russ, & Pascual-Leone, 2014; Mendez, 2009; Tassy et al., 2012) that can in turn influence the risk of engaging in aggression, a deduction consistent with the neural moral model of antisocial behavior (Raine & Yang, 2006). Additionally, the DLPFC has widespread connections with other brain regions which are involved in these functions, including the ventromedial prefrontal cortex (Hare, Hakimi, & Rangel, 2014) and anterior cingulate cortex (Kolling et al., 2016).

Despite these findings, little is known about the *causal* role of the prefrontal cortex on aggressive behavior. Conclusions drawn from extant research on the neural foundations of aggression have largely been correlational. Three known studies have tested the effect of prefrontal cortex upregulation on aggression using the Taylor Aggression Paradigm and transcranial direct current stimulation (tDCS), a non-invasive technique that influences neural excitability by delivering a direct, continuous, low-intensity electrical current to cortical areas between anodal and cathodal electrodes (Brunoni et al., 2012). However, findings have been mixed as one study documented that upregulating the right DLPFC reduced proactive aggression in males (Dambacher et al.,

2015a) while another revealed that increasing left DLPFC activity resulted in more aggressive behavior when participants were angry (Hortensius, Schutter, & Harmon-Jones, 2011). In contrast, upregulation of the inferior frontal cortex did not have a significant effect on aggression (Dambacher et al., 2015b). Whether stimulation targeting the DLPFC can reduce intentions to engage in aggressive acts or behavioral aggression using other measures have not been examined and to our knowledge, no studies have experimentally investigated the intermediary mechanisms linking prefrontal deficits to aggressive behavior.

Given the association between prefrontal impairments and aggression, this study tests the hypothesis that upregulating the prefrontal cortex using a non-invasive form of brain stimulation, tDCS, will reduce a symptom of some mental disorders, namely intention to commit an aggressive act. This study additionally extends the limited literature on tDCS and aggression by employing a larger sample. Furthermore, as similarities have been found between the neural mechanisms underlying moral cognition in normal individuals and brain mechanisms that are impaired in antisocial populations, (Raine & Yang, 2006) we also assess whether prefrontal upregulation improves judgments of moral wrongfulness, which may in turn partly account for any effect of prefrontal enhancement on reducing intent to commit aggressive acts.

## **Methods**

### **Trial Design**

The study consisted of a double-blind, placebo-controlled, stratified, randomized trial comparing an anodal tDCS intervention with a sham control group. Baseline

assessments and one session of tDCS or sham intervention were conducted during the experimental session, while outcome measures were assessed the following day. Tasks and questionnaires were administered in a fixed order. The study was approved by the Institutional Review Board of the University of Pennsylvania and the trial protocol was registered (ClinicalTrials.gov NCT02427672).

### **Participants**

Eighty-six healthy adults ( $\geq 18$  years of age) were recruited in Philadelphia between April 2015 and April 2016. The experiment took place during the course of one visit to the study site. In addition to assessments conducted at baseline, participants were followed up one day after the experimental session using a web-based questionnaire. Exclusion criteria included contraindications to brain stimulation, including metallic implants near the electrode sites, unstable medical conditions, neurological, cardiovascular, or psychiatric illness, participation in another non-invasive brain stimulation study on the same day, history of adverse reactions to tDCS, and lack of email access. Written informed consent was obtained from all participants.

### **tDCS Intervention**

tDCS was administered by trained study personnel using a battery-driven, constant-current stimulator (TCT Research). Two anodal electrodes were placed over the DLPFC bilaterally (F3 and F4) according to the International 10-20 EEG system. A constant current of 2mA (1mA to each DLPFC site) was applied for 20 minutes through saline-soaked sponge electrodes (5x5cm). A single extracephalic cathodal electrode

(5x7cm) was placed at the posterior base of the neck in order to minimize unintentional effects of inhibitory stimulation on brain activity.

Following standard tDCS protocol, stimulation commenced after a 30-second ramp-up period. The current was ramped down over the last 2 seconds. The tasks performed during tDCS are understood to influence the behavioral after-effects of stimulation (Gill, Shah-Basak, & Hamilton, 2015). Thus, during the stimulation session, all participants performed the Psychology Experiment Building Language (Mueller & Piper, 2014) version of two cognitive tasks that are known to engage the DLPFC, the Psychomotor Vigilance Task (Cui et al., 2015; Dinges & Powell, 1985), followed by the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994; Ernst et al., 2002). Although participants in both intervention arms received the same electrode placement and ramp-up/down times, stimulation for the sham control group was discontinued after 30 seconds. This has proven to be effective for blinding as participants habituate to the sensation of stimulation within seconds of current initiation (Gandiga, Hummel, & Cohen, 2006).

**Intentions to commit aggression.** Behavioral intentions to commit aggressive acts were assessed using two hypothetical vignettes (Hannon, Hall, Nash, Formati, & Hopson, 2000; Mazerolle, Piquero, & Capowich, 2003). Brief scenarios describing two types of aggression, physical assault and sexual assault, were presented to participants who responded to the anticipated likelihood that they would commit the aggressive act. Responses were measured on a scale ranging from zero (no chance at all) to ten (100 percent chance). Details of the vignettes, which have been studied in samples with similar

characteristics to ours (Hannon et al., 2000; Mazerolle et al., 2003) are presented in Appendix D.

**Perceptions of moral wrongfulness.** To assess moral perceptions of the aggressive acts, participants were asked to rate how morally wrong it would be to act as the protagonist in the scenario on a scale from 0 (not at all) to 10 (very). Aggregate measures of aggressive intent and perception of moral wrongfulness were created by combining responses from the physical and sexual assault scenarios (Armstrong & Boutwell, 2012).

**Aggression.** The voodoo doll task is a reliable and validated behavioral analog measure of aggression (DeWall et al., 2013). In this task, participants were shown a computer-based image of a doll that represented a partner or a close friend. They were told that they were given the opportunity to release their negative energy to that individual by inserting as many pins (0-51) in the doll as they wished. Instructions did not use the word “voodoo”. Stabbing the doll with more pins indicated higher levels of aggression.

### **Randomization and Stratification**

At the initial visit, participants were randomized into an active stimulation or sham/placebo condition using a computerized urn randomization procedure (Stout, Wirtz, Carbonari, & Del Boca, 1994). The stratification factors were age (18 years/19 years/20

years and above), sex (male/female), and ethnicity (Caucasian/non-Caucasian). This stratification was used to balance groups on key demographic variables.

### **Blinding**

Participants and experimenters were blind to the tDCS condition assignment. The trial adhered to established procedures to maintain separation between staff that conducted the stimulation and staff that engaged with the participant. In each experimental session, only one experimenter who set up the tDCS procedure had knowledge of the participant's allocation. To further ensure blinding, all participants were kept blind to the objective of the study and outcome measures were not taken in the presence of research staff as they could lead to biased results.

In the 3 cases where double blinding was compromised due to the inability of having more than one experimenter at a session, the cases were excluded from analyses. To assess adherence to blinding procedures, James' and Bangs' blinding indices were calculated using the participants' and blinded experimenters' guesses about group assignment at the end of the experimental session (Bang, Ni, & Davis, 2004; James, Bloch, Lee, Kraemer, & Fuller, 1996).

### **Statistical Analyses**

One-way ANCOVA was used to test group differences in intentions to commit aggression and the behavioral measure of aggression. Baseline measures were examined as possible covariates: variety of self-report crime throughout the lifetime, aggression (Raine et al., 2006), GPA, trait anxiety (Spielberger, 1983), social adversity, psychopathy

(Paulhus, Neumann, & Hare, 2009), the lack of premeditation and sensation-seeking dimensions of impulsivity (Lynam, 2013), and self-control (Tangney, Baumeister, & Boone, 2004). Following recommendations, stratification variables and baseline measures that were associated with the outcomes were adjusted for, while variables with baseline imbalances were not (Committee for Proprietary Medicinal Products, 2003; Kahan, Jairath, Doré, & Morris, 2014). Effect sizes were calculated using partial eta squared.

To provide information on a mechanism of action accounting for any effect of tDCS on aggressive intent, change in perceptions of moral wrongfulness was examined using ANCOVA. We tested whether enhanced moral judgment mediated group differences in intent to commit aggressive acts through a bootstrapping approach using the PROCESS macro on SPSS (Hayes, 2013; IBM Corp, 2016). 10,000 bootstrapped samples were drawn from the original data. The indirect effect of tDCS on intent to commit aggression was calculated as the product of the ordinary least squares regression coefficients for the relationship between tDCS and moral judgment and the association between moral judgment and aggressive intent. The percent mediated,  $P_M$ , is expressed as the ratio of the indirect to total effect of treatment group on intention to commit aggression (Ditlevsen, Christensen, Lynch, Damsgaard, & Keiding, 2005; Hayes, 2013). Hypothesis tests were two-tailed. Blinding indices were obtained using STATA version 14.0 (Stata Corp, 2015). All other statistical analyses were conducted using SPSS version 24.0 (IBM Corp, 2016).



## Results

### Participant Flow and Recruitment

Data were analyzed on a total sample of 81 (see Figure 5.1 for details or reasons for loss). No participants were lost to follow-up. There was no evidence of selection bias as no significant differences were observed between participants who were included in the analyses and those who were not ( $p > .05$ ; Table 5.1).

### Demographics and Adherence to Protocol

Baseline distributions of the hypothesized covariates were generally well balanced between the treatment groups. With the exception of social adversity, demographic variables and baseline characteristics did not differ across groups (Table 5.2). As the James' blinding indices were greater than .5 and Bang's blinding indices did not approach 1 or -1, participants were considered to have been blinded successfully on average (Table 5.3; Bang et al., 2004; James et al., 1996).

### Aggression Outcomes

Prognostic covariates were determined based on bivariate associations between the hypothesized covariates and outcome measures (Table 5.4). A one-way ANCOVA controlling for stratification variables, self-report crime, and baseline aggression levels revealed a main effect of treatment group on aggressive intent, with the active tDCS group reporting a significantly lower likelihood of engaging in aggression compared to the sham control group (Table 5.5). There were no significant interaction effects between treatment group and sex,  $F(1, 70) = .57, p = .45, \eta_p^2 = .01$ , and between treatment group

and ethnicity,  $F(1, 70) = .01, p = .92, \eta_p^2 < .001$ . Further analyses revealed that intent to commit both physical assault ( $p = .02$ ) and sexual assault ( $p = .02$ ) were lower in the active tDCS group (Table 5.5). However, there was no significant group difference in behavioral aggression assessed using the voodoo doll task ( $p = .26$ ; Table 5.5).

Additional sensitivity analysis conducted on log-transformed and square root-transformed data for the aggression measures yielded substantively similar findings (see Table 5.6).

### **Mechanisms Accounting for the Reduction in Intent to Commit Aggression**

ANCOVA also revealed that compared to controls, the active tDCS group perceived aggressive acts as more morally wrong (Table 5.5). Higher ratings of moral wrongfulness partly mediated the reduction in intention to commit aggressive acts (indirect effect:  $b = -.51, 95\% \text{ CI } -1.14 \text{ to } -.10, p < .05$ ). After controlling for perceptions of moral wrongfulness, treatment group was not a significant predictor of aggressive intent (Figure 5.2). 31% of the total effect of treatment group on overall aggressive intent was accounted for by moral perception.

Further analysis revealed that moral wrongfulness partly mediated the reduction in likelihood of committing sexual assault (indirect effect:  $b = -.34, 95\% \text{ CI } -1.11 \text{ to } -.03, p < .05$ ), but not physical assault (indirect effect:  $b = -.32, 95\% \text{ CI } -.89 \text{ to } .10, p > .05$ ). Perceptions of moral wrongfulness accounted for approximately half ( $P_M = .56$ ) of the total effect of treatment group on intent to commit sexual assault. For completeness, sensitivity analyses that included the demographic variables and social adversity as covariates did not substantively change the mediation results (Table 5.7).

## **Adverse Events**

tDCS was associated with minimal side effects. No major adverse events were reported over the duration of the study. According to Fertonani, Rosini, Cotelli, Rossini, and Miniussi's (2010) scale and consistent with other tDCS studies (Brunoni et al., 2012; Fertonani et al., 2010), reported side effects included itchiness (85.2%), lightheadedness (40.7%), pain (46.9%), burning (49.4%), warmth (51.2%), pinching (45.7%), iron taste (7.4%), and fatigue of light to moderate intensity (35.0%). No participants withdrew due to these minor events.

## **Discussion**

This study tested a new approach to reducing a symptom of some mental disorders, including antisocial personality disorder, intermittent explosive disorder, conduct disorder, and borderline personality disorder. Individuals who underwent bilateral anodal stimulation of the DLPFC using tDCS reported a lower likelihood of committing an aggressive physical and sexual assault one day after stimulation compared to a sham control group. The treatment-aggressive intent relationship was partly accounted for by enhanced perception that the aggressive acts were more morally wrongful, resulting from prefrontal upregulation. Findings help to strengthen conclusions from neurological, neuroimaging, and neuropsychological research (Damasio, 2000; Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994; Liljegren et al., 2015; Rogers & De Brito, 2016; Yang & Raine, 2009) by documenting experimentally the role of the prefrontal cortex on the likelihood of engaging in aggression and the perception of such

acts as morally wrong. This symptom-oriented approach could help to shed light on the biological underpinnings of clinical disorders comorbid with aggression.

Beyond examining experimentally the role of the prefrontal cortex on a behavioral symptom, the finding that moral judgment partly mediates the effect of tDCS on the likelihood of sexual assault contributes to our mechanistic understanding of the etiology of sexual violence. It also provides partial support for the neuro-moral theory of violent behavior that violence is due in part to impairments in brain regions subserving moral cognition and emotion (Raine & Yang, 2006). The null mediation effect observed for physical assault suggests that moral judgment plays a greater role on intentions to commit sexual assault, which is consistent with empirical evidence that sexual offenses such as rape are rated as more morally wrongful than physical violence (Akman, Normandeau, Sellin, & Wolfgang, 1968; Hsu, 1973). This indicates that moral judgment is likely only one of several processes underlying the prefrontal-aggression relationship.

Given empirical evidence that changes in intentions precede behavioral change (Webb & Sheeran, 2006), our results indicating lower intent to engage in aggressive acts following anodal prefrontal stimulation suggest that tDCS may be an initial step towards the reduction of aggression. While the treatment and control groups did not differ on the behavioral measure of aggression, this finding is consistent with the concept that a single session of tDCS may have a limited effect on behavioral change. The longer-lasting therapeutic effects of tDCS are suggested to be associated with repeated, rather than single sessions of stimulation (Nitsche et al., 2008). Therefore, beyond intent to engage in aggression, future studies need to evaluate whether behavioral changes may be observed with increased intervention sessions.

Several caveats are in order. First, the trial findings are limited to an ostensibly healthy population. As the first study to test the effect of prefrontal cortical upregulation on aggressive intentions, the generalizability of the findings to other samples remains to be seen. A second limitation is that moral judgment and aggressive intent were measured concurrently. Thus, we were unable to confirm the temporal order of the mediator and outcome variable. However, empirical evidence that moral judgments shape behavior (Reynolds & Ceranic, 2007) provide support that the mediation model presented reflects the expected temporal effects. Third, this study measured aggressive inclinations one day after the intervention. Further research is needed to determine if tDCS can produce longer-term reductions in aggressive intent as well as aggressive behavior. Fourth, although the findings demonstrate that anodal tDCS resulting in a current flow through the DLPFC influences intentions to commit aggression, they do not negate the involvement of other brain areas. Future studies using complimentary non-invasive neurostimulation approaches such as transcranial magnetic stimulation and high definition-tDCS may elucidate the anatomical specificity of this effect.

There has been increasing discussion of biological interventions on antisocial and aggressive behavior in both children and adults (Gesch, Hammond, Hampson, Eves, & Crowder, 2002; Hübner & White, 2016; Raine, Portnoy, Liu, Mahoomed, & Hibbeln, 2015). Our initial findings that are limited to intentions to commit aggression and moral judgment require extensive replication. Nevertheless, among other etiological mechanisms, the role of biological factors on the development of antisocial behavior, including aggression, has been increasingly acknowledged (Glenn & Raine, 2014; Latvala, Kuja-Halkola, Almqvist, Larsson, & Lichtenstein, 2015). It has been suggested

that treatment programs will be improved by considering biological mechanisms that potentially regulate aggression (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Thus, it can be argued that further investigation of basic science trials on tDCS may offer a potentially promising new biological approach for a significant feature of some clinical disorders.

### **Conclusion**

The link between aggression and mental disorders renders it of relevance to psychiatry (Pulay et al., 2008). Indeed, asking patients whether they have had thoughts about harming others has long been a standard part of mental status examinations (Appelbaum & Gutheil, 1991; Borum & Reddy, 2001). Understanding of the etiology of aggression and the development of new interventions are paramount to a public health approach to violence reduction (Butchart, Phinney, Check, & Villaveces, 2004; Slutkin, 2017; World Health Organization, 2014). This first known application of prefrontal tDCS to intentions to commit aggression takes a modest step towards translating prior brain imaging research on offenders into practice by testing a novel, evidence-based method of reducing aggression perpetration. Findings provide experimental evidence for the role of the prefrontal cortex on both physical and sexual assault, and suggest how the brain may be therapeutically amenable to change using a non-invasive tool with transient and relatively minor adverse effects (Fertonani, Ferrari, & Miniussi, 2015; Poreisz, Boros, Antal, & Paulus, 2007).

## Paper 5 Tables and Figures

Table 5.1. Comparison of Participants who were Included and Excluded in Statistical Analyses

<b>Characteristic</b>	<b>Included (n = 81)</b>	<b>Excluded (n = 3)</b>	<b>Statistic</b>	<b>p value</b>
<i>Demographic variables</i>				
Sex				
Female	45	1	Chi <sup>2</sup> = .58	.45
Male	36	2		
Age, y	20.21 (3.31)	20.00 (1.73)	t = .11	.91
Race				
Caucasian	36	1	Chi <sup>2</sup> = .15	.70
Non-Caucasian	45	2		
<i>Baseline measures</i>				
GPA	3.59 (.77)	3.66 (.29)	t = -.17	.87
Social adversity	1.10 (1.48)	1.00 (1.00)	t = 2.32	
Variety of offending	16.85 (6.21)	15.00 (5.00)	t = .51	.61
Baseline aggression	9.37 (4.72)	10.33 (2.08)	t = -.35	.73
Psychopathy	23.17 (12.20)	23.00 (13.75)	t = .02	.98
Lack of premeditation	1.61 (.49)	1.92 (.14)	t = -1.08	.28
Sensation-seeking	2.89 (.64)	2.58 (.52)	t = .80	.42
Anxiety	38.60 (8.86)	45.67 (13.05)	t = -1.34	.19
Self-control	36.26 (6.95)	37.00 (1.73)	t = -.18	.86
<i>Outcome variables</i>				
Aggressive intent	2.26 (3.56)	1.33 (.58)	t = .45	.66
Aggression (voodoo doll task)	3.91 (10.29)	3.33 (5.77)	t = .10	.92
Moral wrongfulness	15.20 (3.48)	16.33 (3.51)	t = -.56	.58

*Note.* Data for continuous variables are presented as mean (SD), with comparisons conducted using independent samples *t*-tests or chi-square tests as appropriate. For 8 individuals missing data on GPA scores, mean imputation was conducted. Missing values were replaced with the mean of the observed data as suggested in Kahan et al. (2014).

Table 5.2. Baseline Characteristics by Treatment Arm

<b>Characteristic</b>	<b>tDCS Group (n = 39)</b>	<b>Sham Group (n = 42)</b>	<b>Statistic<sup>b</sup></b>	<b>p value</b>
Sex				
Female	24	21	Chi <sup>2</sup> = 1.09	.30
Male	15	21		
Age, y	20.26 (4.13)	20.17 (2.36)	t = -.12	.90
Race				
Caucasian	17	19	Chi <sup>2</sup> = .02	.88
Non-Caucasian	22	23		
GPA	3.55 (.27)	3.47 (.33)	t = -1.18	.24
Social adversity	.72 (1.15)	1.45 (1.67)	t = 2.32	.02
Variety of offending	17.36 (6.25)	16.38 (6.22)	t = -.71	.48
Aggression	9.92 (4.97)	8.86 (4.48)	t = -1.02	.31
Psychopathy	23.33 (11.85)	23.02 (12.65)	t = -.11	.91
Lack of premeditation	1.59 (.49)	1.63 (.49)	t = .38	.71
Sensation-seeking	2.89 (.65)	2.88 (.65)	t = -.07	.94
Anxiety	38.79 (8.53)	38.43 (9.25)	t = -.19	.85
Self-control	37.05 (6.69)	35.52 (7.18)	t = -.99	.33

*Note.* Data for continuous variables are presented as mean (SD). Differences in baseline scores were compared using two-tailed independent *t*-tests and chi-square tests. For 8 individuals missing data on GPA scores, mean imputation was conducted. Missing values were replaced with the mean of the observed data as suggested in Kahan et al. (2014).



Table 5.3. Participant and Experimenter Conjectures about Group Assignment and Blinding Indices

<b>Participant's guess, n (%)</b>							
<b>Intervention</b>	<b>tDCS</b>	<b>Sham</b>	<b>Do not know</b>	<b>Total</b>	<b>James' BI</b>	<b>Bangs' BI</b>	<b>95% CI</b>
tDCS	26 (32.1)	3 (3.7)	10 (12.3)	39 (48.1)		.59	.42, .76
Sham	18 (22.2)	9 (11.1)	15 (18.5)	42 (51.9)		-.21	-.41, -.02
Total	44 (54.3)	12 (14.8)	25 (30.9)	81 (100)	.57		.49, .65

<b>Experimenter's guess, n (%)</b>							
<b>Intervention</b>	<b>tDCS</b>	<b>Sham</b>	<b>Do not know</b>	<b>Total</b>	<b>James' BI</b>	<b>Bangs' BI</b>	<b>95% CI</b>
tDCS	12 (15.4)	0 (0)	25 (32.1)	37 (47.4)		.32	.20, .45
Sham	2 (2.6)	2 (2.6)	37 (47.4)	41 (52.6)		0	-.08, .08
Total	14 (17.9)	2 (2.6)	62 (79.5)	78 (100)	.84		.76, .91

*Note.* Due to missing data, 3 cases were omitted from calculations of the blinding indices from experimenters' guesses; CI = confidence interval.

Table 5.4. Relationships between Outcome Variables (Aggressive Intent, Moral Wrongfulness, Behavioral Aggression) and Baseline Characteristics of the Sample, Assessed Using *t*-Tests for Dichotomous Demographic Variables (Upper Section) and Pearson Correlations for Continuous Baseline Variables (Lower Section)

<b>Characteristic</b>	<b>Aggressive intent</b>	<b>Moral wrongfulness</b>	<b>Behavioral aggression</b>
Sex	-2.10*	4.21***	.11
Race	-.08	.06	-.52
Age	-.07	-.01	-.02
GPA	.13	.02	.15
Social adversity	-.08	.09	-.05
Variety of offending	.36**	-.21	.001
Aggression	.42***	-.07	.08
Psychopathy	.17	-.30**	.20
Lack of premeditation	-.07	.11	.28*
Sensation-seeking	.17	-.06	.19
Anxiety	-.02	-.07	.22
Self-control	.01	-.07	.22

*Note.* Sex was coded as 0 for female and 1 for male. Race was coded as 0 for Caucasian and 1 for non-Caucasian.

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

Table 5.5. Group Means and SDs for Antisocial Behavior Outcomes at Follow-Up

Outcome	tDCS group (n = 39)	Sham group (n = 42)	Main group effect, <i>F</i> (df = 1)	Partial eta squared
Aggressive intent	1.41 (2.07)	3.05 (4.41)	8.40**	.11
Physical assault	1.15 (1.98)	2.19 (2.60)	5.61*	.07
Sexual assault	.26 (.55)	.86 (2.38)	5.64*	.08
Aggression	5.31 (11.84)	2.62 (8.56)	1.31	.02
Perception of moral wrongfulness	16.03 (3.06)	14.43 (3.70)	4.64*	.06
Physical assault	6.56 (2.79)	5.74 (2.54)	.96	.01
Sexual assault	9.46 (.94)	8.69 (2.21)	6.81*	.09

*Note.* Group effects on aggressive intent were determined from ANCOVA, controlling for stratification variables (sex, age, ethnicity), self-report offending, and baseline aggression. Group effects on the behavioral measure of aggression were determined after controlling for stratification variables and lack of premeditation scores. Group effects on perceptions of moral wrongfulness were determined after controlling for stratification variables and baseline psychopathy levels.

\* $p < .05$ ; \*\* $p < .01$ .

Table 5.6. Group Means and SDs for Log-Transformed and Square-Root Transformed Antisocial Behavior Outcomes at Follow-Up

Outcome	tDCS group (n = 39)	Sham group (n = 42)	Main group effect, <i>F</i> (df = 1)	Partial eta squared
<b>Log-transformed variables</b>				
Aggressive intent	.27 (.30)	.42 (.39)	7.44**	.10
Physical assault	.21 (.30)	.37 (.34)	6.43*	.08
Sexual assault	.07 (.15)	.13 (.28)	4.68*	.06
Aggression	.34 (.55)	.18 (.42)	1.43	.02
<b>Square-root transformed variables</b>				
Aggressive intent	.81 (.88)	1.27 (1.21)	7.61**	.10
Physical assault	.64 (.87)	1.11 (.99)	6.53*	.09
Sexual assault	.23 (.46)	.39 (.85)	4.50*	.06
Aggression	1.16 (2.02)	.62 (1.51)	1.44	.02

*Note.* Group effects on aggressive intent were determined from ANCOVA, controlling for stratification variables (sex, age, ethnicity), self-report offending, and baseline aggression. Group effects on the behavioral measure of aggression were determined after controlling for stratification variables and lack of premeditation scores.

\* $p < .05$ ; \*\* $p < .01$ .

Table 5.7. Total, Direct, and Indirect Effects of tDCS on Aggressive Intent, Controlling for Demographic Variables and Social Adversity

	Total effect: tDCS on aggressive intent		Direct effect: tDCS on aggressive intent		Mediation effect: tDCS on aggressive intent		
	B	SE	B	SE	B	SE	95% CI
Overall aggressive intent	-1.68*	.81	-1.30	.81	-.38	.23	-1.01, -.05
Physical assault	-.97 <sup>†</sup>	.53	-.74	.50	-.23	.23	-.81, .12
Sexual assault	-.71 <sup>†</sup>	.42	-.36	.39	-.35	.25	-1.06, -.03

*Note.* B = unstandardized coefficient; SE = bootstrap standard error; CI = bias-corrected bootstrap confidence interval.

<sup>†</sup> $p < .10$ ; \* $p < .05$ .

Figure 5.1. CONSORT Flowchart of the Screening and Enrollment of Study Participants who were Randomly Assigned to Anodal Prefrontal Stimulation or a Sham Control Group

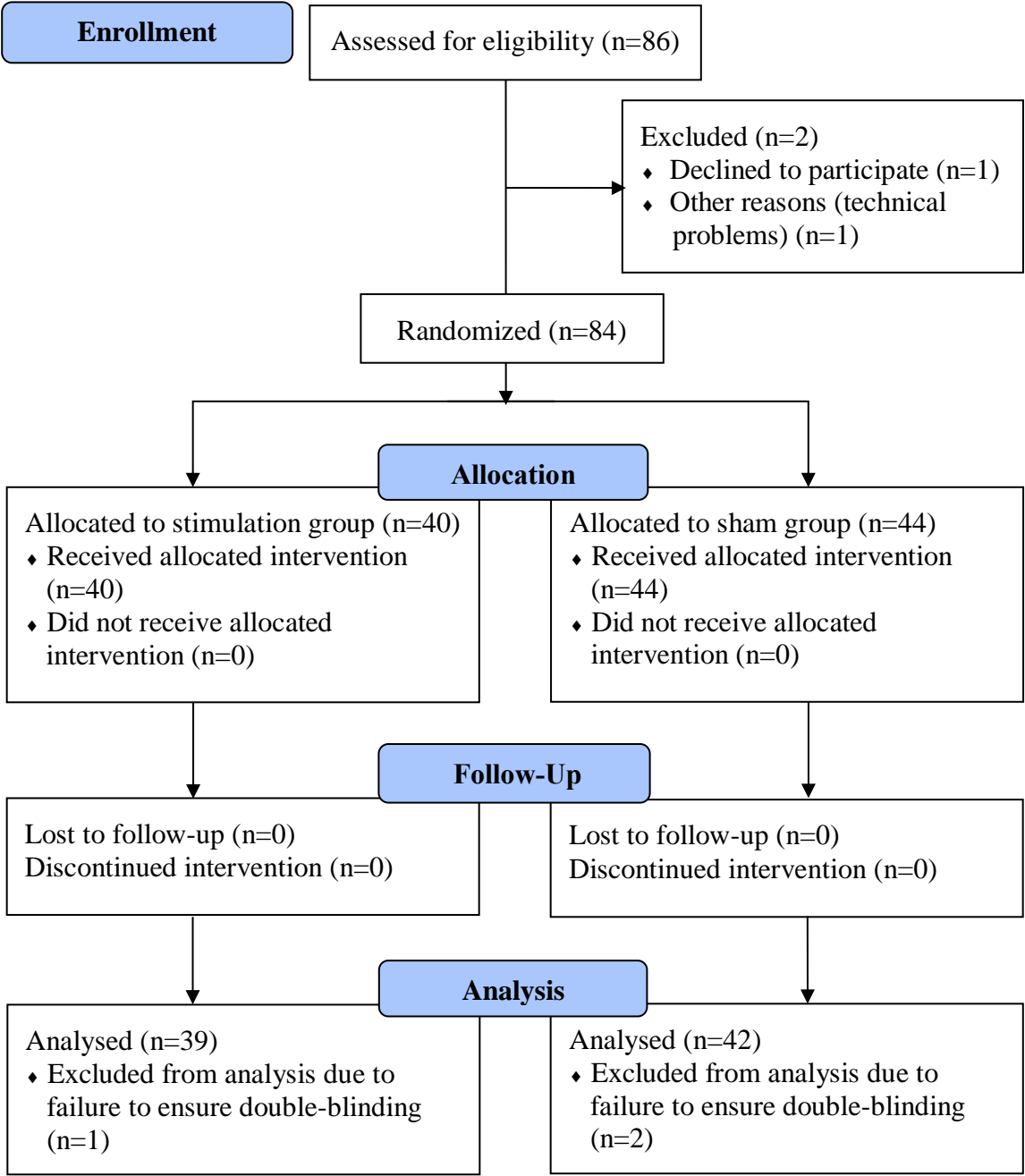
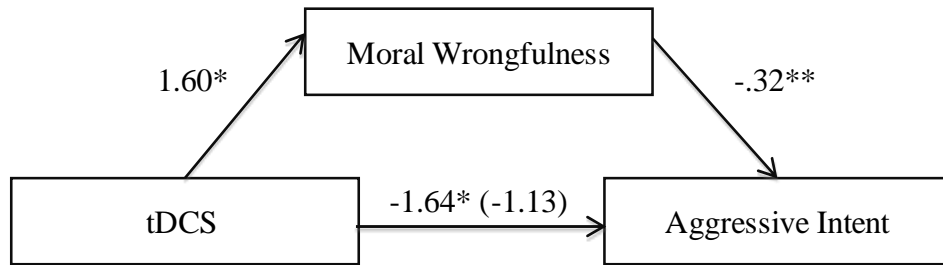


Figure 5.2. Bootstrapped Mediation Model Documenting that Perceptions of Greater Moral Wrongfulness Mediated the Effect of Anodal tDCS on Reducing Intentions to Commit Aggression



*Note.* The path coefficients are unstandardized. Value in parentheses indicates the direct effect of tDCS on intention to engage in aggression.

\* $p < .05$ ; \*\* $p < .01$ .

## GENERAL DISCUSSION

This dissertation aimed to examine whether biological factors can advance traditional theoretical accounts of crime, add to the literature on protective factors, and have a causal role on antisocial behavior. Paper 1 found that greater social adversity early in life was associated with a lower heart rate during stress at age 11 and 12 years, which was in turn associated with delinquency, conduct disorder, and child psychopathy. This finding placed neurobiological influences in a social context to explain antisocial behavior. Paper 2 built on these results to propose an integrative biopsychosocial mediation framework in which social factors lead to changes in biological functioning, which are in turn associated with psychological risk factors that predispose to crime and delinquency. Paper 3 documented that lower heart rates in males at age 11 years partly explain their higher levels of crime at age 23 years. Paper 4 found that serum vitamin D levels moderated the association between early social adversity and both parent and child-reported antisocial behavior at age 11 and 12 years, such that greater social adversity was associated with increased antisocial behavior among vitamin D-insufficient children, but not for individuals who met the threshold for vitamin D sufficiency. Evidence was found for high vitamin D as a protective factor for antisocial behavior. Paper 5 provided the first experimental evidence that increasing activity in the prefrontal cortex can reduce intentions to commit aggression, which was partly accounted for by enhanced perceptions of the moral wrongfulness of the aggressive acts.

These findings can have relevance to the broader discipline of criminology. One contribution of these studies concerns theoretical refinement and development. Overall,



the studies suggest that biological research can help to support and strengthen extant theories of crime, such as strain theory (Agnew, 1992), social control theory (Gottfredson & Hirschi, 1969), reintegrative shaming theory (Braithwaite, 1989), and developmental and life-course theories (e.g., Farrington, 2005b; Lahey & Waldman, 2005; Sampson & Laub, 2005; Thornberry & Krohn, 2005). Importantly, these perspectives do not minimize the importance of social environmental or psychosocial processes. Instead, the papers in this dissertation detail how sociological theory can be buttressed by biological research. The division between “social” and “biological” is increasingly being recognized as a false dichotomy. For instance, the empirical proof of concept for the development of a new “social neurocriminology” perspective draws our attention to how social environmental factors influence biological processes to in turn give rise to crime.

Beyond theoretical development, the findings may have implications for prevention and intervention efforts. Researchers have highlighted that the relevance of biosocial research to practical applications such as interventions is better elucidated using experimental methods to help establish causal relations between biological variables and antisocial outcomes (Cornet, 2015; Wright & Boisvert, 2009). In the first known application of prefrontal tDCS to intentions to commit aggression, results suggest that the brain may be therapeutically amenable to change in order to reduce the likelihood of engaging in aggression. In addition, from a resilience perspective, evidence that higher levels of vitamin D protected individuals with social risk against antisocial behavior underscores the potential role of vitamin D supplementation in efforts to reduce such behavior. There is clearly scope for replication and extension of our current findings.

Nevertheless, they provide initial support for further exploratory trials that may potentially offer new approaches to reducing antisocial behavior.

In conclusion, the papers in this dissertation demonstrate how the biological factors, autonomic arousal, vitamin D, and prefrontal cortical functioning may contribute to a richer and more complete understanding of the mechanisms underlying antisocial behavior. They highlight the importance of considering an interplay between sociological, psychological, and biological variables in research to inform theory and advance prevention and treatment strategies to reduce antisocial behavior.

## APPENDICES

### Appendix A

#### Antisocial Behavior Measures

##### Delinquency

The *Rule-Breaking Behavior Syndrome* was measured using 15 items: Drinks alcohol without parents' approval; doesn't feel guilty about misbehaving; breaks rules at home, school, or elsewhere; hangs around with others who get in trouble; lies or cheats; prefers being with older kids; runs away from home; sets fires; steals at home; steals outside the home; swears or uses obscene language; thinks about sex too much; smokes, chews, or sniffs tobacco; engages in truancy/skips school; uses drugs for nonmedical purposes, excluding alcohol or tobacco.

The *Aggressive Behavior Syndrome* included the following 17 items: Argues a lot; exhibits cruelty, bullying, or meanness to others; demands a lot of attention; destroys his/her own things; destroys things belonging to his/her family or others; disobedient at home; disobedient at school; gets in many fights; physically attacks people; screams a lot; is stubborn, sullen, and irritable; exhibits sudden changes in mood or feelings; is suspicious; teases others a lot; has temper tantrums or a hot temper; threatens to hurt people; is unusually loud.

The internal reliability (Cronbach's alpha) of the delinquency scale was .80.

##### Conduct Disorder

Conduct disorder was measured by assessing how many times subjects had done the following in the past year: (1) bullied or threatened someone, (2) started a physical

fight, (3) used a weapon to harm someone, (4) been physically cruel to someone, (5) been physically cruel to an animal, (6) stolen or grabbed things from someone, (7) forced someone into a sexual activity, (8) started a fire to damage things, (9) destroyed people's things, (10) broken into a house, building, or car, (11) lied to get things or favors, or to avoid doing something, (12) stolen things or shoplifted, (13) stayed out at night without permission, (14) ran away from home for a while, (15) stayed off school without permission. The aggressive and non-aggressive subscales were assessed using items 1-7 and 8-15 respectively. The internal reliability (Cronbach's alpha) of the scale was .83.

### **Child Psychopathy**

The *Callous-Unemotional* subscale of child psychopathy was assessed according to how well the following 6 statements characterize the subject: Not concerned about how well he/she does at school/work; not good at keeping promises; does not feel bad or guilty when he/she does something wrong; not concerned about the feelings of others; does not show feelings or emotions; does not keep the same friends. The *Narcissism* subscale was measured with 7 items: Emotions seem shallow and not genuine; brags excessively about his/her abilities, accomplishments, or possessions; uses or "cons" other people to get what he/she wants; teases or makes fun of other people; acts charming at times to get things he/she wants; becomes angry when corrected or punished; thinks that he/she is better or more important than other people, while the *Impulsivity* subscale was measured using 5 items: Blames others for his/her mistakes; acts without thinking of the consequences; gets bored easily; engages in risky or dangerous activities; does not plan

ahead, or leaves things until the “last minute”. The internal reliability (Cronbach’s alpha) of the scale was .81.

## **Appendix B**

### **Social Adversity**

#### **Items**

The social adversity index consists of 18 items that were recoded as either 1 (adverse) or 0 (not adverse). The items included mother's education (1 = less than 12 years, 0 = graduated high school), father's education (1 = less than 12 years, 0 = graduated high school), parents' employment status (1 = neither parent was employed at time of initial visit, 0 = at least one parent was employed), mother's age at child's birth (1 = 20 or younger, 0 = above 20), problems with living accommodation (1 = yes, 0 = no), number of siblings (1 = 5 or more, 0 = less than 5), living with both biological parents (1 = no, 0 = yes), separation from mother between age 6 months and 2.5 years (1 = yes, 0 = no), absence of child supervision at home (1 = yes, 0 = no), living in government housing (1 = yes, 0 = no), ratio of people per room, including bedrooms, living room, dining room, and kitchen (1 = 1.0 and above, 0 = less than 1.0), number of times moved in the past 12 years (1 = 4 or more times, 0 = less than 4 times), presence of physical illness, such as heart or lung problems, in mother (1 = yes, 0 = no), presence of physical illness in father (1 = yes, 0 = no), presence of mental illness, such as alcoholism, major depression, schizophrenia, and anxiety in mother (1 = yes, 0 = no), presence of mental illness in father (1 = yes, 0 = no), father has been detained or imprisoned (1 = yes, 0 = no), mother has been arrested (1 = yes, 0 = no). We dichotomized the demographic variables based on face validity. We examined the distributions for the responses to each item and determined cut-points or jumps in the frequencies of responses.

## Statistical Analyses

As missing values were primarily found for the social adversity index, listwise deletion excluded up to 156 subjects from the analyses. In the simple imputation procedure, a mean social adversity score for each subject was calculated based on the available responses, by dividing each subject's total adversity score by the number of items that they responded to. We then multiplied this score by the total number of items (18) to obtain an imputed score for each subject. Subjects who required imputed values on the social adversity questionnaire did not differ from those with complete data on heart rate and most antisocial behavior measures. Significant differences between the two groups were found for parent-reported general antisocial behavior and parent-reported child psychopathy. It is apt to note that heart rate during stress mediated the social adversity-antisocial behavior relationship even with the use of listwise deletion.

## Results

General antisocial behavior was significantly associated with mother's education ( $r = -.22, p < .001$ ), father's education ( $r = -.13, p = .02$ ), mother's age at child's birth ( $r = -.11, p < .03$ ), number of siblings ( $r = .22, p < .001$ ), ratio of people per room ( $r = .15, p < .01$ ), and number of moves in the last 12 years ( $r = .19, p < .001$ ). Significant differences in general antisocial behavior scores were observed in terms of parental unemployment ( $t = -2.44, df = 143.04, p = .02$ ), problems with living accommodation ( $t = -2.05, df = 67.71, p = .04$ ), living circumstances ( $t = -5.52, df = 281.85, p < .001$ ), separation from mother early in life ( $t = -2.24, df = 58.09, p = .03$ ), living in government housing ( $t = -2.78, df = 97.74, p = .01$ ), mother's mental illness ( $t = -2.08, df = 383, p$

= .04), father's imprisonment ( $t = -5.14, df = 289.61, p < .001$ ), and mother's arrest ( $t = -2.84, df = 383, p = .01$ ). No significant differences were found in combined parent and child-reported general antisocial behavior based on child's supervision ( $t = .30, df = 384, p = .76$ ), mother's physical illness ( $t = -.90, df = 379, p = .37$ ), father's physical illness ( $t = -.90, df = 327, p = .37$ ), and father's mental illness ( $t = -.91, df = 322, p = .36$ ).

**Prevalence of Adversity for Each Item in the Social Adversity Scale**

<b>Item</b>	<b>Prevalence (%)</b>
(1) Mother's low education	10.6
(2) Father's low education	12.6
(3) Parent unemployment	25.3
(4) Teenage pregnancy	23.7
(5) Problems with living accommodation	15.1 ( $n = 383$ )
(6) Five or more siblings	19.0 ( $n = 384$ )
(7) Not living with both biological parents	71.9
(8) Early maternal separation between age 6 months and 2.5 years	12.6
(9) Absence of child supervision at home	4.7 ( $n = 386$ )
(10) Living in government housing	20.3 ( $n = 385$ )
(11) More than one person per room	18.2 ( $n = 384$ )
(12) Moved 4 or more times in the past 12 years	25.9 ( $n = 386$ )
(13) Mother's physical illness	22.6 ( $n = 381$ )
(14) Father's physical illness	14.9 ( $n = 329$ )
(15) Mother's mental illness	14.8 ( $n = 385$ )
(16) Father's mental illness	8.3 ( $n = 324$ )
(17) Father has been detained or imprisoned	46.8 ( $n = 331$ )
(18) Mother has been arrested	20.3 ( $n = 385$ )
<b>Mean</b>	3.92
<b>Standard Deviation</b>	2.34
<b>Range</b>	0 to 10.13



## Appendix C

### Bivariate Correlations and Concordance between Parent-Reported and Child-Reported Antisocial Behavior Measures

	Pearson Correlations						R <sub>c</sub>
	<i>Child-Report</i>			<i>Parent-Report</i>			
	Heart Rate – Stress	Heart Rate – Rest	Social Adversity	Heart Rate – Stress	Heart Rate – Rest	Social Adversity	
<i>General Antisocial</i>	-.09 <sup>†</sup>	-.05	.22**	-.18**	-.14**	.35**	.38
<i>Delinquency</i>	-.09 <sup>†</sup>	-.05	.16**	-.19**	-.13**	.30**	.36
<i>Conduct Disorder</i>	-.07	-.05	.24**	-.13*	-.10*	.33**	.34
Aggressive	-.06	-.04	.21**	-.14**	-.13*	.30**	.35
Non-Aggressive	-.06	-.04	.24**	-.08	-.05	.28**	.23
<i>Child Psychopathy</i>	-.07	-.03	.17**	-.18**	-.14**	.32**	.27
Callous- Unemotional	-.08	-.03	.15**	-.13*	-.08	.30**	.25
Narcissism	-.03	.007	.15**	-.12*	-.12*	.26**	.25
Impulsivity	-.04	-.04	.07	-.18**	-.14**	.20**	.17

*Note.* Heart Rate – Stress = Heart rate measured during the social stressor task; Heart Rate – Rest = Heart rate at rest; General Antisocial = General measure of antisocial behavior; Aggressive = Aggressive subfactor of conduct disorder; Non-Aggressive = Non-aggressive subfactor of conduct disorder; Callous-Unemotional = Callous-unemotional dimension of child psychopathy; Narcissism = Narcissism dimension of child psychopathy; Impulsivity = Impulsivity dimension of child psychopathy; R<sub>c</sub> = Concordance Correlation Coefficient.

<sup>†</sup>*p* < .10; \**p* < .05; \*\**p* < .01, two-tailed.

## Appendix D

### Physical and Sexual Assault Scenarios

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<b>Physical Assault</b>	<p>It's Friday night. Chris and Lisa, who have been dating for two years, go for a few beers and dinner. While drinking their beers, Chris excuses himself and goes to the bathroom. While he is away, another guy, Joe, who is with his friends, starts talking to Lisa and sits down at her table. Chris returns just as Joe is asking Lisa for her phone number and asks the guy if he has a problem, because he is coming on to his girlfriend. Joe stands up and tells Chris that Lisa does not have a ring and is therefore allowed to talk to whomever she wants. Chris does not like this very much, so he motions to Lisa for her hand so they can leave. Meanwhile, Joe's friends stare Chris down. Then Joe pushes Chris' hand down. Chris grabs a beer bottle off the table and hits Joe in the head with the bottle.</p>
<b>Sexual Assault</b>	<p>Josh and Patricia know each other from a class they are taking. One day after class, they went to a local cafe to discuss an upcoming assignment. After a few hours of enjoyable conversation during which each admitted to being attracted to the other, they decided to meet again over the weekend to watch a movie at Patricia's apartment. During the movie, they began flirting and kissing. After a while, both Patricia and Josh were sexually aroused, and both became more involved in flirting, kissing and intimately touching each other. Patricia was uncomfortable with what was happening, and stopped and pulled away. Josh and Patricia talked for a little while, Josh encouraging Patricia to be more intimate. Patricia again said, "I'm uncomfortable with this. I want to stop," and moved away from Josh. Josh then continued and they had sexual intercourse despite Patricia's protests to stop.</p>

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