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Predicting reward sensitivity in a non-clinical population

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PREDICTING REWARD SENSITIVITY IN A NON-CLINICAL POPULATION

By

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Predicting Reward Sensitivity in a Non-Clinical Population

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ABSTRACT

Reward sensitivity (RS) has been implicated in a range of suboptimal psychological

outcomes, including ADHD, antisocial personality disorder, borderline personality

disorder, and callous-unemotional personality traits. Less known, however, is the

relation between these constructs and RS in the non-clinical population. The current

study investigated the utility of these traits in predicting RS in an undergraduate sample

($N = 225$). Hierarchical multiple regression analyses suggested that impulsive ADHD

symptoms and relational aggression predicted RS, as measured by two distinct

questionnaires ($R^2_{adj.} = .15$ for SPSRQ Sensitivity to Reward [Torrubia, Ávila, Moltó, &

Caseras, 2001]; $R^2_{adj.} = .07$ for BIS/BAS Scales' BAS total score [Carver & White,

1994]). Overall measures of callous-unemotional traits were not significantly related to

RS (ps = .54 - .95), although subscale-level associations suggested a small, inverse

relation between these constructs. These findings highlight the role of RS across the

spectrum of impulse control abilities.

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LIST OF ABBREVIATIONS

ADHD   Attention Deficit/Hyperactivity Disorder

ASPD   Antisocial Personality Disorder

BAARS-IV   Barkley Adult ADHD Rating Scale

BAS   Behavioural Approach System

BAS-T   BIS/BAS Scales BAS Total Score

BIS   Behavioural Inhibition System

BPD   Borderline Personality Disorder

CU   Callous-Unemotional or Callousness-Unemotionality

DSM-5   Diagnostic and Statistical Manual for Mental Disorders, 5th Edition

FFFS   Fight/Flight/Freeze System

ICD   Impulse Control Disorder

ICU   Inventory of Callous-Unemotional Traits

MCAR   Missing Completely at Random

MNAR   Missing Not at Random

RST   Reinforcement Sensitivity Theory

SPSRQ   Sensitivity to Punishment/Sensitivity to Reward Questionnaire

SP   SPSRQ Sensitivity to Punishment subscale

SR   SPSRQ Sensitivity to Reward subscale

RAQ   Relational Aggression Questionnaire

TBI   Traumatic Brain Injury

WISPI-IV   Wisconsin Personality Disorders Inventory

I. LITERATURE REVIEW

Introduction

In our daily experiences, there are many factors that influence human behaviour. Reward and punishment have long been understood to contribute to the development of socially-desirable conduct. With roots in Skinner's theory of operant conditioning, the current understanding of reward and punishment also includes the concept of individual sensitivity. Specifically, those personal factors, such as differences in neurophysiology or temperament, are understood to predispose individuals to respond differently to reward and punishment (Boksem, Tops, Wester, Meijman, & Lorist, 2006; Corr, 2008).

Implicated in a number of suboptimal psychological outcomes, individual responsivity to reward has been identified as an important personality construct. Its role in impulsivity and impulse control disorders (Fowles, 1988), including neurodevelopmental disorders such as attention-deficit/hyperactivity disorder, and some personality disorders, including antisocial personality disorder and borderline personality disorder, suggests both nosological and clinical importance for the reward sensitivity construct. The goal of the present study is to evaluate the relations among reward sensitivity, impulse control disorder (ICD) symptoms, and callous-unemotional personality traits in a non-clinical sample.

This chapter describes the extant findings regarding reward sensitivity as they relate to impulsivity-related constructs. The aim of this chapter is threefold: first, to review the literature on reward sensitivity; second, to discuss impulsivity-related constructs associated with heightened reward sensitivity; and third, to present support for

callous-unemotional personality traits as an additional predictor of reward sensitivity in nonclinical young adults.

Reinforcement Sensitivity: An Overview

Gray's Reinforcement Sensitivity Theory (RST; Gray, 1970, 1981, 1987; Gray & McNaughton, 2000) serves as a theoretical foundation for understanding individual sensitivity to reward and punishment. This theory proposes that behaviour, affect, and motivation itself are regulated by activity in three orthogonal, autonomic neural networks. These networks are the Behavioural Inhibition System (BIS; Gray, 1987), the Fight/Flight/Freeze System (FFFS; Gray & McNaughton, 2000; McNaughton & Corr, 2004), and the Behavioural Approach System (BAS, Gray, 1987; also known as the Behavioural Activation System, Fowles, 1980). The sensitivities of these three independent neural networks—that is, the intensity of a stimulus required for network activation—are posited to govern individual sensitivity to punishment and reward.

According to the most recent version of Gray's theory, the FFFS and BIS underlie the construct of punishment sensitivity (Gray & McNaughton, 2000). Gray proposed that the FFFS governs behaviour in the response to of aversive stimuli, as well as novelty and frustrative non-reward. In contrast, the BIS modulates behaviour in the presence of conflicting motivational signals (e.g. approach-avoidance conflict; Gray & McNaughton, 2000; Corr, 2008).

Gray and McNaughton (2000) proposed that these systems modulate response to aversive stimuli via neural circuits connecting a number of regions, including the frontal lobes (i.e., anterior cingulate cortex, posterior cingulate cortex, dorsal prefrontal cortex) and more posterior subcortical regions (i.e., periaqueductal gray, medial hypothalamic,

septohippocampal, and amygdalar regions). Recent functional neuroimaging research has also suggested that decreased functional connectivity between these areas underlies punishment sensitivity (reviewed in Kennis, Rademaker, & Geuze, 2013).

Revised RST theory describes a third neural network, the sensitivity of which is principally responsible for determining an individual's reward sensitivity (Gray & McNaughton, 2000). Orthogonal to the BIS and the FFFS, the Behavioural Approach System (BAS; Gray, 1970, 1987; Gray & McNaughton, 2000), or the Behavioural Activation System (Fowles, 1980), is purported to underlie motivation to obtain positive stimuli.

Like BIS and FFFS, the revised RST's BAS sensitivity construct is thought to emerge from circuits connecting specific brain regions. According to the revised RST, emotional motivation driving reward pursuit involves four primary neural regions: the prefrontal cortex, the ventral tegmentum, and the ventral pallidum and ventral striatum (Gray & McNaughton, 2000). Accordingly, neuroimaging and electrophysiological findings have supported the existence of reward-based neural networks; the sensitivity of these four regions has been repeatedly identified as foundational to reward sensitivity (Kennis et al., 2013).

In addition to the neuroimaging and electrophysiological methods used to identify areas important for reward processing, two classes of methods are typically employed to measure individual reward sensitivity. First, researchers have used performance-based tasks which measure, for example, reaction time (e.g. Tripp & Alsop, 1999) or the accuracy with which a task is performed (e.g. Carlson et al., 2000). Performance-based

reward sensitivity is then quantified by comparing task performance in a condition in which a reward is offered to performance in a no-reward condition.

Reward sensitivity is also frequently measured via self-report questionnaires. Of note, Carver and White's (1994) BIS/BAS scales are a popular measure of BIS/BAS activity which include three subscales purported to measure various aspects of BAS-related functioning. In addition, a more recent measure of the construct, the Sensitivity to Punishment/Sensitivity to Reward Questionnaire (Torrubia, Avila, Moltó, & Caseras, 2001) is increasingly used to assess BIS and BAS sensitivity. Both measures have allowed researchers to investigate the occurrence and correlates of self-reported reward sensitivity in a range of populations.

Using these methods, researchers have suggested that heightened reward sensitivity relates to a range of personal and nonclinical psychological features. For example, reward sensitivity has been found to be lower in men than in women (Carver & White, 1994). It has also been associated with a number of personality-based constructs; for example, individuals who are more sensitive to reward have been found to be more extraverted (Gray, 1970, 1987; Caseras, Avila, & Torrubia, 2003; Boksem, Tops, Wester, Meijman, & Lorist, 2006) and proactive (Boksem et al., 2006). Greater reward sensitivity also appears to correlate with positive affectivity (Franken & Muris, 2006; Carver & White, 1994) and a propensity for novelty-seeking (Boksem et al., 2006).

Impulsivity and Reward Sensitivity

Germane to the present study, recent research has linked reward sensitivity to normative impulsive behaviour. Although trait-like impulsivity and reward hypersensitivity are distinct constructs (Franken & Muris, 2006; Quilty & Oakman,

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2004), the two appear to be related (Gray & McNaughton, 2000). The terms have been used interchangeably in early versions of Gray's theory as well as by more contemporary researchers (e.g. Ávila & Parcet, 2002). Scores on measures of reward sensitivity correlate positively with self-reported impulsivity (Caseras, Avila, & Torrubia, 2003). Moreover, individuals who are more sensitive to reward also demonstrate higher levels of impulsivity-related behaviours, including more frequent risk-taking behaviours (Zuckerman & Kuhlman, 2000). Greater reward sensitivity has also been related to sensation-seeking (Torrubia, Ávila, Moltó, & Caseras, 2000) and related behaviours such as reckless driving (Scott-Parker, Watson, King, & Hyde, 2011, 2013).

Reward hypersensitivity has also been implicated as a mechanism in disorders for which poor impulse control is a central feature. For example, some research has suggested altered reward processing as a correlate of substance abuse (e.g. Knyazev, Slobodskaya, Kharchenko, & Wilson, 2004) and traumatic brain injury (e.g. Larson et al., 2007). Of particular interest for the present study, a robust body of evidence has also suggested a role for reward hypersensitivity in three disorders of impulse control: attention-deficit hyperactivity disorder (ADHD), antisocial personality disorder (ASPD), and borderline personality disorder (BPD).

Attention-Deficit/Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) is a common disorder of impulse dyscontrol characterized by pervasive inattention and/or hyperactivity (American Psychiatric Association, 2013). Although pediatric ADHD has long been a focus of research, its manifestation in adulthood has been relatively recently described in the

Diagnostic and Statistical Manual for Mental Disorders, Fifth Edition (*DSM-5*;

American Psychiatric Association, 2013).

ADHD is frequently accompanied by significant impairments in social

functioning (Maedgen & Carlson, 2000) and academic abilities (Barbaresi, Katusic,

Colligan, Weaver, & Jacobsen, 2007). Individuals with a history of ADHD in childhood

are also at risk for a number of comorbid disorders associated with poor impulse control,

such as conduct disorder (Mannuzza, Klein, Abikoff, & Moulton, 2004), Cluster B

personality disorders (Halperin, Rucklidge, Powers, Miller, & Newcorn, 2011), substance

abuse (Biederman et al., 1995), and intermittent explosive disorder (Kessler et al., 2006).

While much of these findings relate to clinical ADHD samples, individuals possessing

non-clinical levels of ADHD symptoms may nevertheless experience functional

impairments (Barkley et al., 2006). Given that ADHD symptoms appear to be normally

distributed in the general population (Cornish et al., 2005), the correlates of ADHD

symptomatology are relevant to the population as a whole.

Reward sensitivity and ADHD. It has been suggested that ADHD and similar

disorders result from atypical BAS regulation (Quay, 1988, 1997). In keeping with this

hypothesis, considerable research has implicated reward hypersensitivity as an etiological

mechanism of ADHD. Compared to typically-developing children, those with ADHD

typically demonstrate greater performance gains in response to reward (Luman, Meel,

Oosterlaan, & Guerts, 2012), with some evidence of a "speed-accuracy" tradeoff for this

improved performance in ADHD (reviewed in Luman, Oosterlaan, & Sergeant, 2005).

Heightened reward sensitivity in children with ADHD is also supported by physiological

findings, such as heart rate elevation in reward conditions (Crone, Jennings, & van der

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Molen, 2003; Luman et al., 2005). Individuals with ADHD also demonstrate neurophysiological anomalies in regions which contribute to reward processing, including thinning of the prefrontal cortex (Shaw et al., 2006) and reduced activation in the dorsal anterior mid-cingulate cortex (Bush, 2011). Research has further suggested that individuals with ADHD differ in terms of their dopaminergic response to reward (Tripp & Wiekens, 2008), although the research investigating this theory remains somewhat equivocal (Luman, Tripp, & Scheres, 2010).

This relationship between clinical ADHD and reward hypersensitivity is well-supported. However, although some studies have investigated the relationship between ADHD symptoms and reward sensitivity in non-clinical samples, findings from this body of work have not been wholly consistent. Although some have found reward hypersensitivity to be related to the hyperactive/impulsive symptoms of ADHD (Gomez & Corr, 2010; Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008; Mitchell, 2010; Mitchell & Nelson-Gray, 2006), the relation between RST constructs and the inattentive symptoms of ADHD is more equivocal. For example, reward hypersensitivity has been associated with inefficient attention on a performance-based measure (Avila & Parcet, 2002); however, it is unclear whether performance on this computerized, laboratory-based task generalizes to attentional abilities and difficulties observed in the general population. Indeed, others have found that self-report inattention is associated with punishment sensitivity rather than reward sensitivity (Gomez & Corr, 2011), yet still others have found inattention to be linked to heightened reward sensitivity in females (Mitchell & Nelson-Gray, 2006). Given these inconsistent findings, further work is

necessary in order to gain a true understanding of the motivational mechanisms underlying ADHD symptoms in a non-clinical population.

Impulsive Personality Disorders

Personality disorders consist of maladaptive personality traits as well as deficiencies in one's view of self and interpersonal patterns (American Psychiatric Association, 2013). Two such disorders described by the *DSM-5* are antisocial personality disorder (ASPD) and borderline personality disorder (BPD; American Psychiatric Association, 2013). In addition to their momentous personal cost to individuals who suffer from these disorders, ASPD and BPD account for a substantial proportion of the medical and incarceration costs incurred by society (Teplin, 1994; Beauchaine, Klein, Crowell, Derbidge & Gatzke-Kopp, 2012; Bender et al., 2001).

In terms of their *DSM-5* diagnostic criteria, ASPD and BPD represent distinct-but-related constructs. Antisocial personality disorder is typified by a manipulative interpersonal style and pervasive disregard for social norms. Additionally, individuals with ASPD demonstrate a marked disregard for others' rights and feelings, instead motivated to fulfill their own desires and needs (American Psychiatric Association, 2013). Borderline personality disorder, in contrast, represents a constellation of symptoms which result in the appearance of emotional instability and relational volatility (American Psychiatric Association, 2013). As conceptualized by the *DSM-5,* individuals with BPD achieve a sense of fulfillment only through their relationships with others. Individuals with BPD typically idealize their relationships at the onset; however, when slighted, these individuals typically feel abandoned and become aggressive, emotionally reactive, and often suicidal.

While diagnostically discrete entities, ASPD and BPD share a high degree of overlap in their central features. In fact, it has been suggested that ASPD and BPD represent the gender-specific phenotypes of the same disorder (Paris, 1997). Aggression, for example, is a trait often observed in both ASPD and BPD (APA, 2013). In particular, relational aggression—a form of aggression characterized by expressing anger by damaging peers' social status—is a correlate of symptoms of both disorders (Ostrov & Houston, 2008; Werner & Crick, 1999). Although relational aggression is often conceptualized as a predominantly female form of aggression (Crick & Grotpeter, 1995; Werner & Crick, 1999), the above-cited studies have identified this link in mixed-gender samples, suggesting that relational aggression may also apply to males with these disorders.

While relational aggression represents an important feature of these disorders' symptomatic presentation, perhaps the most salient point of overlap between ASPD and BPD is their underlying impulsivity (Fossati et al., 2007; Looper & Paris, 2000; Steel & Blaszczynski, 1998). ASPD and BPD are also frequently comorbid with impulsivity-related issues such as substance abuse (Tragesser, Sher, Trull, & Park, 2007; Trull, Sher, Minks-Brown, Durbin, & Burr, 2000) and pathological gambling (Barry, Stefanovics, Desai, & Potenza, 2011; Slutske et al., 2001; Steel & Blaszczynski, 1998). Reckless driving is also associated with both ASPD and BPD (Malta, Blanchard, & Freidenberg, 2005). In BPD, this characteristic impulsivity may also lead to self-harm (Brodsky, Malone, Ellis, Dulit, & Mann, 1997) and risky sexual behaviours (Rickards & Laaser, 1999).

The impulsivity noted in ASPD and BPD appears to be related to neuroanatomical anomalies in these groups. While controls activate primarily prefrontal regions while inhibiting prepotent responses, ASPD and BPD groups activate diffuse neural networks throughout the frontal and temporal lobes; this pattern suggests that more neural resources are required for successful impulse control in ASPD and BPD (Völlm et al., 2004). Similarly, atypical cortical and subcortical serotonin synthesis differentiates individuals with BPD from normal controls (Leyton et al., 2001). Diminished serotonin synthesis in in frontal areas (particularly the medial frontal gyrus), the striatum, and cortical areas in the temporal lobe relates to the heightened impulsivity demonstrated by BPD subjects (Leyton et al., 2001).

There is some evidence that the patterns of impulsivity observed in ASPD and BPD may be related to an etiological overlap with ADHD. Research has suggested that both ASPD and BPD often proceed from a childhood history of ADHD (Fossati, Novella, Donati, Donini, & Maffei, 2002; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998). Evidence likewise exists for elevated co-occurrence of adult ADHD with BPD (Torok, Darke, & Kaye, 2012; Speranza et al., 2011) and ASPD (Semiz et al., 2008).

The behavioural impulsivity associated with ASPD and BPD has been most frequently studied in clinical samples. However, it must be noted that the relationship between these personality disorder symptoms and impulsivity has also been identified in nonclinical populations. James and Taylor (2007) found a self-report measure of overall impulsivity to be related to ASPD (but not BPD) symptoms in nonclinical subjects. Individuals who demonstrate BPD and ASPD features have also been found to demonstrate more self-reported "motor impulsivity" (i.e., acting without regard for

consequences; Fossati et al., 2004). ASPD and BPD symptoms in nonclinical samples

have also been linked to self-reported impulsive behaviours such as substance abuse

(Taylor, 2005) and self-harm (Casilas & Clark, 2002).

Reward sensitivity in ASPD and BPD. Heightened reward sensitivity appears

to be a possible mechanism for the impulsivity characteristic of these disorders.   Both

ASPD and BPD have been shown to relate to heightened self-reported BAS

hypersensitivity in clinical samples (i.e., personality-disordered; Ross, Keiser, Strong, &

Webb, 2013). Moreover, self-reported ASPD and BPD symptoms are also associated

with heightened reward sensitivity in non-clinical groups (Ross, Keiser, Strong, & Webb,

2013; Pastor et al., 2007).

Additional support for reward hypersensitivity as a feature of ASPD may be

inferred from research investigating a similar construct, psychopathy. Although

psychopathy and ASPD do not appear to be wholly interchangeable (for a review, see

Ogloff, 2006), they share a conceptual core defined by egocentricity, antisocial

behaviour, and impulsivity.   Thus, research identifying reward hypersensitivity in

individuals with psychopathy may have utility in supporting the link between reward

sensitivity and ASPD.

While it has been suggested that varying levels of punishment sensitivity may

distinguish distinct "primary" and "secondary" psychopathy subgroups, it is broadly

accepted that elevated reward sensitivity underlies most forms of psychopathy (e.g.

Blackburn, 2006; Lykken, 1995). Accordingly, empirical findings have found reward

hypersensitivity to underlie both primary and secondary psychopathy subgroups (Ross et

al., 2007; Newman, MacCoon, Vaughn, & Sadeh, 2005), suggesting that heightened

reward sensitivity is an important facet across psychopathy subtypes. In a study which used a passive avoidance learning task in juvenile offenders, psychopathic adolescents differed from non-psychopathic adolescent offenders in their response to reward, but not to aversive stimuli (Scerbo et al., 1990). Individuals with clinically-elevated psychopathy also display poor impulse control in the presence of reward (Masui & Nomura, 2011). While the link between reward sensitivity and psychopathic traits is relatively well-established in clinical groups, it has also been supported in work with a non-clinical population (Masui & Nomura, 2011).

Callous-Unemotional Traits

As outlined above, symptoms of ICDs are thought to be strong predictors of reward hypersensitivity. However, a growing body of evidence has also supported a relation between reward sensitivity and a cluster of personality features known as callous-unemotional (CU) traits. These personality traits appear to be stable throughout adolescence and adulthood (Loney, Taylor, Butler, & Iacono, 2007) and encompass a range of atypical affective features and patterns of social processing. Central to these appear to be a lack of empathy and theory of mind (Pardini, Lochman, & Frick, 2003; Muñoz & Frick, 2012; Stellwagen & Kerig, 2013) and an absence of guilty feelings after wrongdoing (Frick & White, 2008; Muñoz & Frick, 2012).

Additionally, callous-unemotional traits appear to be related to atypical neuroanatomical features. Research has particularly focused on the amygdala, which is implicated in emotion processing (Dawel, O'Kearney, McKone, & Palermo, 2012); accordingly, amygdalar malformation and dysfunction has repeatedly been identified as a neurobiological correlate of CU traits (reviewed in DeLisi, Umphress, & Vaughn, 2009).

Taken together, these biological correlates suggest that CU traits are, at least in part, the result of a neurobiological mechanism.

There is some evidence that CU traits approximate the normal distribution in the general population (e.g. Loney, Butler, Lima, Counts, & Eckel, 2006). It has further been suggested that some degree of CU traits may be normative, particularly in adolescence and early adulthood (reviewed in Moffit, 1993). However, *elevated* callousness-unemotionality has been associated with a range of behavioural and psychiatric disorders. While CU traits have been most frequently investigated as a core feature of psychopathy (Cooke, Michie, & Hart, 2005) emerging evidence suggests that CU traits comprise a cross-disorders construct (Herpers, Rommelse, Bons, Buitelaar, & Scheepers, 2012). It has been suggested, for example, that CU traits may serve as a marker for a more severe subtype of conduct disorder (Kahn, Frick, Youngstrom, Findling, & Youngstrom, 2012). Callous-unemotional traits have further been observed in a subset of individuals with ADHD (Brammer & Lee, 2011) and a subgroup of non-clinical adolescents displaying symptoms of BPD (Chabrol, Valls, van Leeuwen, & Bui, 2012), suggesting that they may be a construct independent of these disorders themselves.

This construct has been identified as a risk factor for an array of maladaptive behaviours, including substance abuse (Wymbs et al., 2012). They have also been shown to incrementally predict future criminal behaviour (Kahn, Byrd, & Pardini, 2012; McMahon, Witkiewitz, Kotler, & the Conduct Problems Prevention Research Group, 2010); similarly, CU traits have been consistently linked with higher displays of instrumental aggression (that is, aggression which does not result from provocation but is rather used as a means to achieve some personal gain; Muñoz & Fick, 2012). Callous-

unemotional traits have also been shown to differentiate psychopathy from pure antisocial behaviour (Christian et al., 1997) and predict antisocial personality disorder diagnosis (McMahon et al., 2010). In addition to the ramifications of their own behaviour experienced by individuals with CU traits, their associated interpersonal style may encourage their peers to delinquency, although individuals who are CU are unlikely to be influenced by their peers themselves (Kerr, van Zalk, & Stattin, 2012).

Reward Sensitivity and Callous-Unemotional Traits

Although the CU construct has come to the fore in only relatively recent years, a few studies have investigated the relation between CU traits and reward sensitivity. This growing body of literature has provided support for altered reward sensitivity in individuals who display CU traits. For example, a number of studies have suggested that CU traits accompany reward hypersensitivity. Children with CU traits appear to respond more to positive reinforcement and reward than more punishment-based (e.g. time-out) behavioural management strategies (Hawes & Dadds, 2005). Individuals with CU traits also appear to overvalue the positive outcomes of aggression while undervaluing the punitive consequences (Lorber, Hughes, Miller, Crothers, & Martin, 2011; Pardini, Lochman, & Frick, 2003).

Support for the reward hypersensitivity of individuals with CU traits may also be inferred from research investigating similar constructs. For example, high levels of risk-taking behaviours, which have been argued to represent reward hypersensitivity particularly in adolescence, have been observed in individuals with callous-unemotional traits (Centifanti & Modecki, 2013; Essau, Sasagawa, & Frick, 2006; Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Pardini et al., 2004; Sadeh, Verona, Javdani, & Olson,

2009). Of note, however, it is also possible that this increased risk-taking in individuals

with CU traits may instead be driven by low punishment sensitivity.

However, the literature investigating reward sensitivity in individuals with CU

traits is equivocal, as not all studies have found *increased* reward sensitivity to be

associated with CU. In fact, several studies have found diminished reward sensitivity as

a correlate of CU traits (e.g. Centifani & Modecki, 2013; Marini & Stickle, 2010; Roose,

Bijttebier, Claes, & Lilienfeld, 2011; Roose, Bijttebier, Decoene, Claes, & Frick, 2010;

Verona, Patrick, Curtin, Bradley, & Lang, 2004). As such, additional research is

warranted to truly understand the motivational role reward plays in this population.

II. THE PRESENT STUDY

Rationale

As reviewed above, the literature surrounding reward sensitivity includes relatively strong evidence for the association between disorders of impulse control, callous-unemotional traits, and reward sensitivity. However, several limitations of the current literature exist which the present study is designed to address. Firstly, to this author's knowledge, a concurrent analysis of the utility of ICD symptoms and CU traits in predicting reward sensitivity has yet to be conducted. Given that CU traits may co-occur with ICD symptoms (e.g. McMahon et al., 2010; Brammer & Lee, 2011; Chabrol, Valls, van Leeuwen, & Bui, 2012), simultaneous investigation of these features may provide a more ecologically valid understanding of these traits.

Additionally, research investigating the impulsivity-reward sensitivity relation has typically focused on clinical groups and incarcerated individuals. While research in these groups is vital to an understanding of the clinical manifestations of these disorders, it assumes that categorical diagnoses adequately capture the nature of disorder. However, given evidence that impulsivity and callousness-unemotionality are personality traits which approximate a normal distribution (e.g. Cornish et al., 2005; Loney et al., 2006), using an arbitrary cutoff for treatment and research may disregard individuals who nonetheless experience personally significant impairment. Although less is known about the association between reward sensitivity and ICD and CU features in non-clinical groups, this topic is nonetheless of great import.

Using self-report measures of ICD symptoms, CU traits, and reward sensitivity, the present study attempts to address these gaps in the current literature. Based on

previous findings, it is hypothesized that greater self-reported ICD and CU features will predict heightened reward sensitivity in a non-clinical sample. Support for an association between these features may have important implications for motivating individuals who display impulsive and callous-unemotional traits.

Methods

Participants

Two hundred twenty-five participants (186 females, 37 males; 1 indicated "other", 1 did not respond) were recruited through University of Windsor's participant pool. This electronically-based system allows full- and part-time undergraduate students currently enrolled in psychology courses (and select business courses; 21 students reported a business-related major) to receive course credit in exchange for participation in a research study. Upon registration, participants complete a screening questionnaire, allowing researchers to make their study invisible to participants who do not meet inclusion criteria. For the present study, participants were unable to view the online study advertisement and subsequently enroll if they indicated that they were unable to read, write, and/or speak English.

Participant demographic information is presented in Table 1.

Procedure

The procedures used in this study were approved by the University of Windsor's Research Ethics Board (see Appendix A). Students who met the criteria for inclusion were scheduled for participation via the University's Department of Psychology Participant Pool. All testing appointments were conducted by the author in on-campus research lab space. Upon arrival for his or her testing appointment, each student gave

informed consent prior to participation in the study. Through this process, the

participants were apprised of the nature and duration of the study. Further, the minimal

risks of participation were explained, as were the benefits to participants (i.e., course

credit). In all, testing appointments lasted no longer than 90 minutes. Each participant

was awarded 1.5 credits for participation, in accordance with published participant pool

policy.

After consent had been obtained, participants completed a demographic

questionnaire and a packet containing several additional questionnaires, as described

below.   Questionnaires were ordered randomly for each participant. Multiple participants

completed questionnaire packets during the same testing session. To ensure

confidentiality of responses, participants were not seated directly next to or across from

another participant. Further, participants were provided with a cover sheet to conceal his

or her responses, if desired.

Measures

Demographic information. A series of demographic questions was constructed

for the present study (see Appendix B). This questionnaire collected information

regarding participants' age, birth month and year, ethnicity, marital status, and academic

standing. The demographic questionnaire also screened for a history of traumatic brain

injury. Information regarding parents' academic and vocational attainment was also

gathered for the purposes of coding socio-economic status.

Reward sensitivity. The measurement of reward sensitivity has been deemed

"historically elusive" and limited by imperfect tools (Cogswell, Alloy, van Dulmen, &

Fresco, 2006, p. 1657). Nonetheless, two self-report measures are widely used for the

measurement of this construct and are therefore used in the present investigation: the Sensitivity to Reward/Sensitivity to Punishment Questionnaire (Torrubia et al., 2001) and Carver and White's (1994) BIS/BAS scales.

The Sensitivity to Punishment/Sensitivity to Reward Questionnaire (SPSRQ). The SPSRQ (Torrubia et al., 2001) consists of forty-eight items to which individuals may respond either "yes" or "no". The SPSRQ is comprised of two subscales: Sensitivity to Reward (SR) and Sensitivity to Punishment (SP). A total subscale score is derived for both SR and SP by totaling the number of "yes" responses for each subscale. The SR subscale is comprised of the measure's even-numbered items. Questions such as "do you generally give preference to those activities that imply an immediate gain?" and "are you interested in money to the point of being able to do risky jobs?" measure approach behaviours, corresponding with reward sensitivity. The odd-numbered SP items measure avoidance behaviours with questions such as "whenever you can, do you avoid going to unknown places?" and "do you often refrain from doing something you like in order not to be rejected or disapproved of by others"?

The SPSRQ was constructed as a measure of specific reward- and punishment-sensitive behaviours; consistent with Gray's theory, the SPSRQ measures situational manifestations of reward sensitivity rather than one's general tendencies across different types of situations (Torrubia et al., 2001). As such, the SPSRQ items are designed to maximally discriminate individuals who possess a construct (i.e. reward sensitivity or punishment sensitivity) to a high degree from those who are low on the construct. Unlike many other measures of reward and punishment sensitivity, the SPSRQ produces orthogonal scores corresponding to the BIS and the BAS (Torrubia et al., 2001).

While it has been shown to be imperfect (Cogswell et al., 2006), the SPSRQ represents a relative improvement over previously conventional measures of reward sensitivity such as the BIS/BAS scales (Carver & White, 1994), described below. The authors of the SPSRQ and others report "good" internal consistency (α_{males} = .78, $\alpha_{females}$ = .75, Torrubia et al., 2001; α = .75, Cogswell et al., 2006) for the SR subscale in large non-clinical samples. The authors of the SPSRQ also claim "adequate" test-retest reliability for the SR subscale (Torrubia et al., 2001).

The BIS/BAS Scales. As a supplement to the SPSRQ, Carver and White's (1994) BIS/BAS Scales were also administered. The BIS/BAS scales consist of twenty-four self-statements encompassing a range of approach and avoidance behaviours. In comparison to the SPSRQ, the BIS/BAS scale items measure general patterns of BAS-relevant behaviours in response to rewarding stimuli (Torrubia et al., 2001). Participants rate the degree to which they agree with each statement using a four-point Likert scale ([1] "very false" to [4] "very true"). The scale items reflect four content areas related to BIS and BAS sensitivity: BIS (e.g. "I worry about making mistakes"), measuring avoidance of aversive stimuli; BAS Drive (e.g. "When I want something, I usually go all-out to get it"), measuring one's work ethic or drive to obtain desired outcomes; BAS Fun-Seeking (e.g. "I crave excitement and new sensations"), measuring spontaneity and the pursuit of novel positive stimuli; and Reward Responsiveness (e.g. "When I get something I want, I feel excited and energized"), measuring one's affective responses to possible reward.   Utilizing a non-clinical sample, the authors report internal consistency values ranging from "acceptable" (Fun Seeking, α = .66) to "good" (BIS, α = .74; Reward

Responsiveness, α = .73; Drive, α = .76) and also provide some support for its convergent validity (Carver & White, 1994).

Despite their frequent use, the three BAS subscales have garnered recurrent criticism from RST researchers. Of paramount concern, the subdivision of BAS into these three component domains is not rooted in RST theory, problematizing interpretation of any relations between the scales and other constructs (Torrubia et al, 2001; Torrubia, Ávila, & Casearas, 2008). From a psychometric standpoint, the factor structure of the scale items is also unclear. Some argue that Carver and White's four-factor model (BIS and BAS Drive, Fun Seeking, Reward Responsiveness) does fit the scale items (Heubeck, Wilkinson, & Cologon, 1998; Jorm et al., 1999; Leone, Perugini, Bagozzi, Pierro, & Mannetti, 2001; Ross, Millis, Bonebright, & Bailley, 2002), although fit statistics reported in these studies are typically only moderate and do not provide convincing support for a four-factor model (Poythress, Skeem, Lilienfeld, Douglas, & Edens, 2009).

The validity of the individual BAS scales has also been subject to criticism. For example, it has been suggested that the Fun Seeking scale measures BAS correlates, such as impulsivity and novelty-seeking, rather than BAS itself (Caseras et al., 2003; Knyazev, Slobodskaya, & Wilson, 2004; Zelenski & Larsen, 1999); as a result, the Fun Seeking scale may be inappropriate for use as a standalone measure of reward sensitivity. The psychometric properties of the individual BAS scales have also been criticized; for example, internal consistency (as measured by Cronbach's α) for the Reward Responsiveness subscale has been as low as .59 in one sample (Cooper, Smillie, & Jackson, 2008). Further, this subscale has been found to load on both BIS and BAS scales (Carver & White, 1994; Heubeck et al., 1998; Knyazev et al., 2004).

In order to circumvent potential problems associated with using the less robust subscales yet maintain continuity with the majority of previous studies of RST, some researchers working with nonclinical samples have utilized a BAS total score (BAS-T) comprising the combined scores of the three BAS subscales (e.g. Gomez & Gomez, 2002; Jorm et al., 1999; Murphy, Murphy, & Garavan, 2013; Stange et al., 2012). Indeed, the scale authors initially reported that the BAS scales loaded onto a higher-order BAS factor (Carver & White, 1994); others have found that scree values suggest a two-factor (BIS, BAS-T) solution (Jorm et al., 1999). Using this approach, the BAS-T has been demonstrated to have improved internal consistency compared to the subscales individually ($\alpha = .81$ in a large nonclinical sample; Smillie, Jackson, & Dalgleish, 2006). In concordance with this approach (and in recognition that the BIS/BAS scales, in all forms, imperfectly measure BAS sensitivity yet are the cornerstone of self-report RST literature), the BAS-T was used in the present study.

ADHD symptoms. Current symptoms of ADHD were assessed using the self-report Barkley Adult ADHD Rating Scale (BAARS-IV, Barkley, 2011). This questionnaire was designed to assess the degree to which self-reported *DSM* symptoms of ADHD deviate from a normative adult sample. The BAARS-IV consists of a list of 27 symptoms. Using a four-point Likert scale (from [1] "never or rarely" to [4] "very often"), individuals rate the frequency with which each item has applied to them in the past six months.

The BAARS-IV items measure ADHD symptoms across four domains: Inattention (e.g. "difficulty sustaining my attention in tasks or fun activities"), Hyperactivity (e.g. "leave my seat in classrooms or in other situations in which remaining

seated is expected"), Impulsivity (e.g. "blurt out answers before questions have been completed, complete others' sentences, or jump the gun"), and Sluggish Cognitive Tempo (e.g. "I don't seem to process information as quickly or as accurately as others"). While examiners may compute a symptom count for these domains and for overall ADHD symptomology (i.e., how many items were rated as "often" or higher), scale and overall total scores (sum of all item ratings for each domain) were deemed more appropriate for the present non-clinical sample. The BAARS-IV also assesses the pervasiveness of ADHD symptoms (i.e., "In which settings did these symptoms impair your functioning?") and the approximate age of symptom onset.

Internal consistency values for the BAARS-IV, as reported by the scale's author, range from "good" (α = .78, Hyperactivity; α = .81, Impulsivity) to "excellent" (α = .90, Inattention; α = .91, Current ADHD Total Score; Barkley, 2011). Satisfactory test-retest reliability is also reported when participants were retested after an interval of two to three weeks (*r*s ranging from .66 to .88). As evidence for the validity of this measure, the author cites small to medium correlations between BAARS-IV Current scale scores and a continuous performance test, and medium to large correlations with a self-report measure of executive functioning (Barkley, 2011). Although developed for clinical use, the BAARS-IV has been applied in some studies using non-clinical samples (e.g. Flannery, Becker, & Luebbe, 2014; Langberg, Becker, Dvorsky, & Luebbe, 2014).

ASPD and BPD symptoms. Features of ASPD and BPD were assessed using the Wisconsin Personality Disorders Inventory (WISPI-IV; Klein & Benjamin, 1996). Derived from the *DSM* personality disorder diagnostic criteria, the WISPI-IV items consist of a series of statements typifying the behaviour, affective functioning, and

cognition associated with each of the personality disorders. The full-length WISPI-IV consists of 214 items. For the current study, however, only the items corresponding to each *DSM-5* Cluster B diagnosis were administered in an effort to minimize participant fatigue.

For each of the items, participants are asked to rate how much each statement has applied to his or her "usual self" over the past five or more years. Responses fall on a ten-point scale, ranging from "never/not at all" (1) to "always/extremely" (10). A mean score for each personality diagnosis may be derived. Additionally, participants' mean scores for each diagnosis may be compared to a normative sample using scoring software. Scores for each of the WISPI-IV diagnostic dimensions have demonstrated "good" to "excellent" internal consistency in a partially non-clinical population (Klein et al., 1993). The WISPI-IV also boasts reasonable convergent and discriminant validity compared to other personality disorder measures (i.e. the Structured Clinical Interview-II; Smith, Klein, & Benjamin, 2003). This measure has been previously applied in studies investigating personality functioning in the non-clinical population, including RST correlates of personality disorder symptomology (e.g. Kimbrel, Mitchell, Hundt, Robertson, & Nelson-Gray, 2012).

Relational Aggression Questionnaire. Aggression is an important feature of the *DSM-5* diagnostic criteria for both ASPD and BPD. However, these criteria (and tools based on them, such as the WISPI-IV) are largely biased toward acts of physical aggression. It has been suggested that the manifestation of aggression varies by gender; while males may enact aggression physically, it has been suggested that females' aggressive acts are frequently directed relationally (e.g. damaging relationships through

gossip, excluding others, etc.; Crick & Grotpeter, 1995; Werner & Crick, 1999). Given the overrepresentation of females in the university participant pool, inclusion of a measure of relational aggression may better capture the aggressive ASPD and BPD features of the current university sample.

For the current study, a seven-item Relational Aggression Questionnaire (RAQ) was included as a brief and general measure of this construct. The items were originally developed as a peer nomination tool (Werner & Crick, 1999); in this original form, participants provided the name of a peer who typifies each item. Individuals who received more frequent peer nominations were deemed to exhibit more relational aggression. In order to feasibly use this measure to assess relational aggression in a university sample, the seven items were adapted into a self-report questionnaire. Participants were asked to rate the frequency at which each of the original items applies to him- or herself on a five-point Likert scale (ranging from [1] "never true" to [5] "always true"). A total score was then calculated, with higher scores reflecting a more pervasive pattern of relational aggression. In its original form, this measure boasts good reliability (α = .87; Werner & Crick, 1999). Although applied for the first time as a self-report tool in the present sample, its reliability in the current study was adequate (α = .60).

Callous-unemotional traits. Finally, callous-unemotional traits were assessed using the Inventory of Callous/Unemotional Traits (ICU; Frick, 2004). The ICU consists of twenty-four items designed to measure the behavioural and affective features of callousness-unemotionality. The ICU items comprise three orthogonal factors (Essau, Sasagawa, & Frick, 2006; Kimonis et al., 2008): Uncaring (e.g. "I always try my best"),

Callous (e.g. "I do not care who I hurt to get what I want"), and Unemotional (e.g. "I am very expressive and emotional"). Participants were asked to rate their agreement with each statement on a four-point Likert scale ([1] "not at all true" to [5] "definitely true").

An ICU total score as well as scores for each factor were calculated by summing the response values for corresponding items. The internal consistency and test-retest reliabilities of the ICU total score and the Uncaring and Callous factor scores have been noted to be "good" (α = .83, ICU Total Score; α = .79, Callousness; α = .77, Uncaring; α = .73, Unemotional; Roose et al., 2010). Because it has only five items, the Unemotional subscale has demonstrated "poor" internal consistency in an incarcerated juvenile sample (α = .57; Kimonis et al., 2008). However, the reliability of the Unemotional subscale was found to be adequate in a non-clinical population (Roose et al., 2010). Moreover, evidence exists for the convergent validity of the self-report ICU in non-clinical populations. The ICU scores have been found to correlate significantly with measures of psychopathy, personality, and reward and punishment sensitivity in a non-clinical sample (Roose et al., 2010).

Data Analysis

The aforementioned measures yield several variables of interest in the current analyses. Potential predictors of reward sensitivity (as measured by the SPSRQ and BIS/BAS BAS-T) included ADHD symptoms (BAARS-IV total score), antisocial and borderline personality disorder symptoms (ASPD and BPD scores from the WISPI-IV, as well as a relational aggression total score from the RAQ), and callous-unemotional traits (ICU total score and scale scores).

Missing data analysis. The extent of missing data and the patterns by which data are missing are important factors for consideration when addressing missing data (Tabachnik & Fidell, 2001). For example, the generalizability of results may suffer for datasets which are missing more than five percent of data points, or in which the data are systematically missing (i.e., Missing Not at Random [MNAR]). In contrast, sparse, random missing data are seen as less problematic.

Given importance of data missingness for informing subsequent analysis decisions (Tabachnik & Fidell, 2001), the dataset was examined for missing data points on all variables to be included in the analysis. Data were missing on 17.2% of the variables of interest (i.e., questionnaire items, and as a result, total scores). Of the 225 participants, 33 (14.7%) had one or more missing data points. Overall, 0.1% of the total dataset was missing. Little (1988)'s MCAR test was not statistically significant (χ^2 [5919] = 5763.780; p = .924), indicating that the data were likely missing completely at random. Taken together, these findings suggest that the small amount of missing data is unlikely to influence analysis results or their generalizability. Given the negligible quality of the missing data, missing data points were imputed using expectation maximization, a standard strategy for missing data imputation. This imputation and all subsequent analyses were conducted in SPSS, v. 19.

Multiple regression analyses. A hierarchical multiple regression analysis was designed for each of the two reward sensitivity variables: 1) SPSRQ Sensitivity to Reward and 2) the BAS total score from the BIS/BAS Scales. Predictors for each model were the aforementioned variables representing ICD symptoms and callous-unemotional

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traits. A theoretical entry order for potential predictor variables was predetermined as follows:

1. In the first block of the analysis, potential confounding variables correlated with the outcome variable were entered. For example, research has shown gender differences in reward processing (e.g. Torrubia et al., 2001); for this reason, gender was entered as a dummy-coded variable in Block 1. Because both participant age and parental education (as estimated by the highest level of parental education attained) have been shown to be related to functioning in reward-related neural networks (Christakou, Brammer, & Rubia, 2011; Gianaros et al., 2011), these variables were also entered in Block 1. This allowed a less biased assessment of the incremental ability of impulsivity-based measures to predict reward sensitivity.

2. Given that they have garnered the greatest degree of support as potential correlates of reward sensitivity, variables representing symptoms of ADHD (BAARS-IV ADHD total score; Inattention, Hyperactivity, Impulsivity, and/or Sluggish Cognitive Tempo domain scores) were entered in the second block.

3. Cluster B personality symptoms (WISPI-IV ASPD and BPD scores; Relational Aggression Questionnaire total scores) were entered in Block 3.

4. Finally, to assess their incremental utility in predicting reward sensitivity, callous-unemotional personality traits were entered in Block 4.

While this model was constructed to systematically guide the regression analyses, any variables which were not significantly correlated with the outcome variables were not

included in the regression analyses. Likewise, variables which did not incrementally

predict reward sensitivity were removed before construction of a final model.

III. RESULTS

Descriptive statistics for the aforementioned variables of interest in predicting reward sensitivity are displayed in Table 2. Skew and kurtosis values were examined in order to assess the degree to which measured traits approximated the normal distribution (as indicated by a skew statistic $< |2|$ and kurtosis statistic $< |3|$). With the exception of age, ASPD symptoms, and ICU Callousness, all variables of interest adequately approximated the normal distribution in the current sample (Table 3; Field, 2009).

Predicting SPSRQ Sensitivity to Reward

A series of hierarchical multiple regression models was conducted to identify those variables which best predict reward sensitivity, as measured by the SPSRQ Sensitivity to Reward (SR) scale.

Participants Included

Sixteen participants (7.1%) endorsed a history of traumatic brain injury (TBI) resulting in a loss of consciousness. Because self-reported history of TBI was associated with significantly higher SPSRQ Sensitivity to Reward scores ($t(221) = -2.63$; $p = .009$), these participants were excluded from the analysis predicting SPSRQ scores, along with the two participants who did not respond to this question. Gender was not significantly related to history of TBI ($\chi^2(3) = 4.18$, $p = .24$.).

Model Construction

Correlations between each potential predictor and SPSRQ SR (in addition to all relevant variable intercorrelations) are reported in Table 4. Contrary to preliminary hypotheses, SR was not significantly correlated with variables measuring callous-unemotional personality traits (ICU total and scale scores; $r = .00$ to $.10$, $p = .17$ to $.99$).

Several variables were likewise correlated with Sensitivity to Reward but failed to account for unique variance in the outcome variable when entered into the hierarchical regression model. In particular, although a marginally significant relation emerged between SR and the two dummy-coded gender variables (each isolating either participants who identified themselves as males or females; point-biserial r_{males} = -.12, p = .08; $r_{females}$ = .14, p = .05), the dummy-coded gender variables did not concurrently predict SR scores when added with age in the first entry step of the model. They were therefore eliminated as a possible predictor of SR scores.

Similarly, the BAARS-IV Impulsivity Total Score emerged as the only robust ADHD-related variable in predicting SPSRQ Sensitivity to Reward; when entered concurrently with this Impulsivity variable, neither the other BAARS-IV scale scores (Inattention, Hyperactivity, and Sluggish Cognitive Tempo) nor the BAARS-IV Total Score accounted for significant variance in SPSRQ Sensitivity to Reward. Thus, of the BAARS-IV variables, only Impulsivity was retained for the final regression model. Though significantly correlated with SR, WISPI-IV ASPD and BPD scores also failed to account for unique variance in Sensitivity to Reward when entered in Step 3 and were removed prior to construction of the final model.

Final Model

A final hierarchical regression model was constructed which accounted for 16.4% of the variance in SPSRQ Sensitivity to Reward scores ($R^2_{adj.}$ = .15; Table 5). In the first block, age significantly predicted Sensitivity to Reward ($R^2_{adj.}$ = .04; p = .002). The addition of BAARS-IV Impulsivity in the second block significantly increased the amount of variance in Sensitivity to Reward accounted for by the model (ΔR^2 = .06;

F_{change} (1, 204) = 13.88, p < .001). The final addition of Relational Aggression

Questionnaire total scores likewise improved the model (ΔR^2 = .06; F_{change}(1, 203) = 14.

22, p < .001). This final model significantly fit the data (F(3, 203) = 13.24, p < .001).

The predictor with the greatest weight was relational aggression (β = .25), though the

weight of impulsivity scores in predicting SR was nearly equivalent (β = .21). Age was

given the least weight in predicting SR (β = -.16).

Because β-weights may be influenced by the variables included in or excluded

from the model (Cohen, Cohen, West, & Aiken, 2003), additional statistics are reported

in Table 6 to provide a more global estimate of each variable's importance in predicting

SR. These include squared partial correlations (indicating the proportion of variance in

the outcome variable that is both attributable to the given predictor and not accounted for

by other predictors; Cohen et al., 2003), squared semi-partial correlations (indicating the

proportion of overall variance in the outcome variable that is attributable to the given

predictor; Cohen et al., 2003), and squared structure coefficients (indicating the

proportion of the variance accounted for by the model that is attributable to the given

predictor, calculated as a correlation between a predictor and predicted scores;

Thompson, 2006).

Of note, the pattern of squared structure coefficients supported the relative

importance of each variable in predicting SR. By this metric, Relational Aggression had

the greatest weight in predicting SR (squared structure coefficient = .53), followed by

Impulsivity (squared structure coefficient = .46). Age had the least weight in predicting

SR (squared structure coefficient = .27).

Cross-Validation of Findings

While the adjusted R^2 values reported above provide some estimation of the

shrinkage in predicted power expected when generalizing this model to the population as

a whole (derived via Wherry's equation for R^2; Stevens, 2002), the variance accounted

for by the model predicting SPSRQ was further cross-validated by using Stein's formula

for a cross-validated R^2 (Field, 2009; Stevens, 2002). This equation suggests that the

final SPSRQ model would account for slightly less variance were the model built upon

population-wide data, $R^2_{cv} = .13$.

Assumptions of Multiple Regression

In order to assess the validity and generalizability of these findings, variables

included in the final model were checked for violations of the assumptions of multiple

regression analysis. Multiple regression assumes an absence of outliers and influential

observations (Cohen et al., 2003; Tabachnik & Fidell, 2001). In the model predicting

SPSRQ Sensitivity to Reward, no cases had values which represented outliers on Y (i.e.

all standardized residuals < 3.27; Tabachnik & Fidell, 2001). However, while eight cases

had extreme values for one or more of the predictor variables (with leverage values >

$[3\{k + 1\}/N]$; Cohen et al., 2003), the data did not contain influential observations (all

Cook's d values < 1.0; Stevens, 2002). Thus, cases which represented outliers on the

predictor variables were retained to preserve the integrity of the regression parameters

(Stevens, 2002).

In addition to an absence of outliers and influential observations, multiple

regression assumes an adequate sample size (Cohen et al., 2003). Conventionally, fifteen

observations per predictor variable are considered sufficient to produce a model which

will generalize to the population (Stevens, 2002); as such, the sample of 207 participants would be adequate for the current three-predictor model. To verify, a *post hoc* power analysis was run using G*Power (Faul, Erdfelder, Lang, & Buchner, 2007) using the sample size of 207 and three predictors. The statistical power for the SPSRQ model exceeded .99, indicating a low likelihood that the null hypothesis was incorrectly rejected in the detection of a significant effect for the model.

Further analyses explored possible violations of additional assumptions of regression. Intercorrelations between predictors were small (*r* values ranging from -.09 to .16). Taken together with in-range collinearity diagnostic statistics (i.e., all tolerance values > .1 [range: .96 - .98], Field, 2009; all Variance Inflation Factor [VIF] values < 10 [range: 1.02 - 1.05], Stevens, 2002), the variables appear to demonstrate an acceptable absence of multicollinearity. The residuals adequately approximated the normal distribution, as indicated by histograms and a non-significant test of normality (Shapiro-Wilk statistic (207) = .992; *p* = .32).

Multiple regression also assumes homoscadasticity, or that the variance for residuals is equal across all values of the predictor variables, and that the predictors are linearly related to the residuals (Field, 2009). Visual inspection of bivariate scatter plots did not indicate violations of these assumptions. The model likewise demonstrated independence of errors (Durbin-Watson statistic = 2.01). Additionally, multiple regression assumes that variables are measured without error. Cronbach's alpha values are reported in Table 2. Internal consistency of the variables in the equation ranged from "adequate" (α = .60, Relational Aggression Questionnaire Total Score) to "good" (α = .72, SPSRQ Sensitivity to Reward scale score; α = .72, BAARS-IV Impulsivity).

Finally, an assumption critical to the validity of a regression model is that of independent observations (Stevens, 2002). The design of the present study makes both final regression models constructed minimally susceptible to violations of this assumption. Although it is likely that some of the participants in the present study interact in courses or in their respective programs of study, the variables included in the final model measure somewhat static personality features rather than behaviours that are readily influenced by peer involvement. Second, the non-experimental design of the present study meant that participants provided information for only one time point and were not selected into groups, which would invite further peer influence. Finally, participants were discouraged from speaking during questionnaire completion, and efforts were made to conceal participants' answers from fellow participants. In sum, while a minor violation of this assumption is inevitable--particularly in a university sample--its effects on the validity of the study's findings are likely minimal.

Predicting BAS Total Score

A second series of hierarchical multiple regression models was constructed to identify those variables that best predict reward sensitivity as measured by the BAS-T.

Participants Included

The 16 participants reporting a history of traumatic brain injury with loss of consciousness did not differ significantly in terms of BIS/BAS BAS total scores ($t(221) = -1.03$; $p = .31$) and were therefore retained for this analysis. Individual scores on the three BAS subscales were likewise unrelated to history of TBI ($ps = .12 - .76$).

Model Construction

Correlations between each potential predictor and the outcome variables are reported in Table 4. Contrary to preliminary hypotheses, BAS-T was not significantly related to age (r = -.05, p = .45), dummy-coded gender variables (r_{male} = -.09, p = .20; r_{female} = .08, p = .23), or parent education (r = -.10, p = .14). BAS-T scores were likewise uncorrelated with the symptoms of ASPD (r = .09, p = .20) or BPD (r = .01, p = .87).

Additionally, although both the ICU Uncaring and Callousness scales were marginally related to the outcome variable ($r_{uncaring}$ = -.13, p = .06; $r_{callousness}$ = .11, p = .09), only the Uncaring scale score accounted for unique variance in BAS-T scores when entered concurrently with Callousness. Thus, Callousness scores were removed prior to construction of the final model. Finally, analysis of structure coefficients after removal of Callousness suggested that ICU Uncaring scores were acting as a suppressor variable, as indicated by a structure coefficient of practically zero (r = -.03, p = .65). This suggests that Uncaring scores were improving the R^2 of the model via their shared variance with the other predictors, rather than the outcome variable (Stevens, 2002). For this reason, Uncaring scores were also removed from the final model.

Final Model

A final hierarchical regression model was constructed which accounted for 8.0% of the variance in BAS-T scores ($R^2_{adj.}$ = .07; Table 7). Because no demographic variables significantly predicted BAS-T scores, the first block consisted of BAARS-IV Impulsivity ($R^2_{adj.}$ = .04; p = .003). The addition of RAQ scores in the second block significantly increased the amount of variance in BAS-T accounted for by the model (ΔR^2 = .04; F_{change}(1, 222) = 9.76, p = .002). This final model significantly fit the data

($F_{(3,222)} = 9.68$, $p < .001$). The strongest predictor of BAS-T scores was relational

aggression ($\beta = .20$), followed by Impulsivity ($\beta = .17$).

Additional statistics are reported in Table 8 to aid in interpretation of the

importance of these effects. The pattern of squared structure coefficients supported the

finding that relational aggression was the most important predictor in predicting BAS-T

(squared structure coefficient = .67), followed by Impulsivity (squared structure

coefficient = .49).

Cross-Validation of Findings

To supplement the adjusted R^2 values (calculated using Wherry's equation)

reported above, Stein's formula for cross-validated R^2 was also applied.   It is estimated

that the current model would account for less variance were the model constructed from

the population of data, $R^2_{cv} = .06$.

Assumptions of Multiple Regression

In assessing the adherence of these data to the assumptions of multiple regression,

procedures and cut-off values applied were identical to those used in the first series of

analyses predicting SR. For the final two-variable model predicting BAS-T Scores, no

variables had values which represented outliers on Y. Additionally, while nine cases had

extreme values for one or more of the predictor variables, these cases (and all others) did

not contain influential observations and were therefore not removed.

Although the sample size of 225 exceeds the conventional rule of fifteen

observations per predictor needed for adequate sample size, a *post hoc* analysis was

conducted using G*Power (Faul et al., 2007) using the sample size of 225 and two

predictors. The statistical power for the BAS-T model was .98. It is therefore unlikely that the finding of statistical significance for this model represents a Type I error.

The small intercorrelation between the two predictors ($r = .17$) and acceptable collinearity diagnostic statistics (tolerance = .97, VIF = 1.03) suggest low multicollinearity between the two predictors. Statistical analysis and visual inspection of histograms suggested that the residuals were adequately normally distributed (Shapiro-Wilk statistic (225) = .995; $p = .73$). Bivariate scatterplots likewise suggested adequate homoscedasticity. The assumption of independence of errors was not violated (Durbin-Watson statistic = 2.17). The reliability of included variables ranged from "acceptable" ($\alpha = .60$, Relational Aggression Questionnaire Total Score) to "good" ($\alpha = .72$, BAARS-IV Impulsivity; $\alpha = .79$, BAS Total Score). For a discussion of the assumption of independence of observations inherent in the study design and applying to both final models, see above.

IV. DISCUSSION

The present study sought to predict reward sensitivity (RS) using self-reported symptoms of impulse control disorders (ICDs) and callous-unemotional personality traits in a sample of undergraduate university students. It was hypothesized that the symptoms of ICDs−such as attention-deficit/hyperactivity disorder (ADHD), antisocial personality disorder (ASPD), and borderline personality disorder (BPD)−would be associated with heightened reward sensitivity, as measured by two different self-report measures. It was further anticipated that callous-unemotional personality traits would predict reward sensitivity beyond the effects of ICD symptoms, although this aspect of the study was largely exploratory.

Hierarchical multiple regression analyses, in part, supported these hypotheses. As measured by the Sensitivity to Reward/Sensitivity to Punishment Questionnaire (SPSRQ; Torrubia et al., 2001) Sensitivity to Reward (SR) subscale, reward sensitivity was most strongly predicted by relational aggression, followed by the impulsive symptoms of ADHD and age. A similar model emerged for predicting reward sensitivity as measured by the BAS total score (BAS-T) of the BIS/BAS scales (Carver & White, 1994). Here, relational aggression was again the most robust predictor of reward sensitivity, followed by the impulsive symptoms of ADHD.

Impulsive Symptoms and Reward Sensitivity

In the present study, a measure of impulsivity (the Impulsivity subscale of the BAARS-IV) was a significant predictor of greater scores on both measures of reward sensitivity. This finding is in line with prior work; RST theory suggests that reward sensitivity contributes to trait impulsivity (Corr, 2008). Accordingly, a link between

reward sensitivity and impulsivity has been identified in previous studies using both the

BIS/BAS scales (e.g. Quilty & Oakman, 2004; Smillie, Jackson, & Dalgleish, 2006) and

the SPSRQ (e.g. Quilty & Oakman, 2004; Torrubia et al., 2001).

Important to note, however, is that impulsivity and reward sensitivity have been

shown to be conceptually distinct constructs, often via factor analytic studies (Franken &

Muris, 2006; Quilty & Oakman, 2004). Impulsivity is accepted to be a multi-faceted

construct rather than a unitary trait (e.g. Dawe & Loxton, 2004; Quilty & Oakman, 2004;

Reynolds, Ortengren, Richards, & de Wit, 2006), and it has been suggested that variants

of impulsive behaviour are only one manifestation of high Behavioral Activation System

(BAS) activity, which produces greater reward sensitivity (Quilty & Oakman, 2004).

Further evidence for a distinction between impulsivity and reward sensitivity

comes from research investigating other personality constructs. Specifically, measures of

impulsivity and reward sensitivity are differentially related to Big Five personality traits,

which are seen by many as the current paradigm for understanding personality from both

a psychometric and neuroanatomic perspective. Measures of reward sensitivity such as

the BAS Reward Responsiveness and Drive subscales and SPSRQ SR subscale are

correlated with Extraversion, while measures of trait impulsivity generally correlate with

Psychoticism (Caseras et al., 2003; Smillie et al., 2006). Neuropsychological and

genetic studies further support this distinction; although the same neural networks appear

to underlie reward sensitivity and extraversion (Boksem et al., 2006; Cohen, Young,

Baek, Kessler, & Ranganath, 2005), activation of these networks does not appear to

correlate with facets of impulsivity which lack an affective component, such as constraint

(reviewed in Depue & Collins, 1999).

The findings of the present study, then, align with the extant research demonstrating an overlap between the two conceptually different constructs of reward sensitivity and impulsivity. Given this demonstrated distinction, then, it is perhaps unsurprising that impulsive symptoms (in particular, those associated with the impulsive variant of ADHD) only accounted for a modest amount of variance in reward sensitivity scores in the present investigation.

ADHD Symptoms

An important finding related to this impulsivity-reward sensitivity relation was that only those ADHD symptoms defined by impulsivity accounted for unique variance in predicting reward sensitivity. Although some have suggested that deficits in reward processing exist across ADHD subtypes (reviewed in Luman et al., 2005), the dissociation between hyperactive/impulsive and inattentive symptoms in the present study supports theories suggesting a "dual pathway" etiology of ADHD. Specifically, it has been suggested that ADHD--Predominantly Inattentive subtype may be attributable to difficulties in executive functioning (Sonuga-Barke, 2003) and cognitive control (Martel & Nigg, 2006), whereas Predominantly Hyperactive/Impulsive ADHD and Combined Type ADHD may be attributable to differences in motivational networks (Gomez & Corr, 2010; Martel & Nigg, 2006).

This theory of a motivational mechanism for hyperactive/impulsive ADHD was initially constructed for application in clinical contexts. However, the findings of the present study add to extensive work extending this dissociation to the non-clinical population. Numerous studies have found an association between hyperactive/impulsive

symptoms and reward sensitivity in non-clinical groups (Gomez & Corr, 2010; Hundt et al., 2008; Mitchell, 2010; Mitchell & Nelson-Gray, 2006).

Impulsive Personality Disorder Symptoms

In contrast to the impulsive symptoms of ADHD and contrary to hypotheses, traditional measures of ASPD and BPD symptoms (i.e., the Wisconsin Personality Disorders Inventory--Fourth Edition; Klein & Benjamin, 1996) were not significantly associated with BAS-T. Moreover, though they were significantly correlated with SPSRQ SR scores, ASPD and BPD scores did not contribute unique variance to the prediction of SPSRQ SR after impulsive ADHD symptoms were added to the model.

The relative insignificance of these variables in predicting reward sensitivity is surprising, given previous associations between ASPD and BPD symptoms and reward sensitivity. In particular, Ross and colleagues (2013) found that symptoms of Cluster B personality disorders, including ASPD and BPD, were associated with greater BAS-T scores in both clinical and non-clinical samples. A similar finding was produced in a Spanish-speaking non-clinical sample (Pastor et al., 2007).

A few possibilities exist for explaining the lack of a significant contribution of ASPD and BPD symptoms to the prediction of reward sensitivity scores. First, although ASPD and BPD were significantly correlated with SPSRQ SR, they did not contribute unique variance to the model. In other words, it appears that the variance ASPD and BPD symptoms shared with SR was already accounted for in the model by the impulsive symptoms of ADHD.

Accordingly, several studies have identified commonalities between impulsive personality disorders and ADHD. As mentioned previously, many children with ADHD

demonstrate ASPD and BPD features in adulthood (Fossati et al., 2002; Halperin et al., 2011; Storebø & Simonsen, 2013a; Storebø & Simonsen, 2013b), suggesting an etiological link between these disorders. In adult clinical samples, individuals with ASPD or BPD have been found to be at increased risk for comorbid ADHD (Philipsen, 2006; Semiz et al., 2008; Speranza et al., 2011; Torok et al., 2010), to the extent that some have suggested that these disorders are etiologically related (Philipsen, 2006). However, few studies have identified an association between ASPD/BPD symptoms and ADHD symptoms in the non-clinical population, as was found in this study (Table 4).

While this shared variance may be a function of meaningful overlap between these ICD constructs, other possible explanations for this unexpected result must be noted.   Indeed, in non-clinical samples, the BAS-T has been shown to correlate with other measures of Cluster B personality disorder symptoms, such as the Minnesota Multiphasic Personality Inventory-Second Edition (MMPI-2; e.g. Pastor et al., 2007) and the Schedule for Nonadaptive and Adaptive Personality (SNAP; e.g. Ross et al., 2013). Thus, the lack of any significant correlation between the BAS-T and personality disorder symptoms hints at a potential limitation inherent in using the WISPI-IV in this sample.

In the present study, the WISPI-IV was selected purposefully to allow for identification of specific clinical features of ASPD and BPD in the non-clinical population. However, although the WISPI-IV was validated, in part, in a non-clinical sample (Klein et al., 1993) and has received some use in non-clinical investigations (e.g. Kuhlken, Robertson, Benson, & Nelson-Gray, 2014; Samuel & Widiger, 2010), it is possible that the WISPI-IV items are not sensitive to the subclinical manifestations of antisocial and borderline traits. Accordingly, a few of the items' wordings may be too

extreme to warrant endorsement in this sample. Items such as "before I was 15 years old, I forced someone to have sex and I didn't give a damn about their feelings" and "anybody who tries to push me around could end up dead" may be too specific for use outside of a clinical or incarcerated sample. Participants who possess subclinical levels of the corresponding traits may fail to endorse these items at all because they have never committed rape or murder, though they may have engaged in less severe, related behaviours.

Relational aggression. Novel to this study is the identification of an association between relational aggression and reward sensitivity. Individuals who more frequently expressed aggression by damaging or threatening harm to others through relationships (e.g. spreading rumours, isolating targets socially) reported greater reward sensitivity in the present study.

Relational aggression, as measured by an adapted version of a questionnaire developed by Werner and Crick (1999), was included in the current investigation with the intent of capturing ASPD- and BPD-like traits in the non-clinical population. Relational aggression is a trait particularly central to the diagnostic presentation of both ASPD and BPD (Ostrov & Houston, 2008; Werner & Crick, 1999), and is likewise more typical of female aggressive behaviour than physical acts of aggression (Crick & Grotpeter, 1995; Werner & Crick, 1999). Thus, though WISPI-IV ASPD and BPD scores did not significantly predict reward sensitivity, the import of relational aggression in predicting both measures of reward sensitivity may nonetheless suggest a role for impulsive personality disorder symptomology in this predominantly (82.7%) female, non-clinical sample.

The significance of relational aggression in predicting reward sensitivity may also reflect this construct's overlap with other disorders of impulse control, as demonstrated in the current study. For example, in the present study, small to medium correlations existed between the symptoms of ADHD and relational aggression. This finding is echoed in previous work in both clinical and non-clinical groups. School-age and adolescent girls with hyperactive/impulsive ADHD have been shown to exhibit more relational aggression than their predominantly inattentive (Zalecki & Hinshaw, 2004) and neurotypically-developing peers (Mikami, Hinshaw, Lee, & Mullin, 2008; Ohan & Johnston, 2007). Additionally, a study identifying risk factors for future offending in a low-socio-economic status group of 10- to 11-year-olds found that ADHD symptoms were greater in a group which endorsed high levels of aggression, including relational aggression (McLoughlin, Rucklidge, Grace, & McLean, 2010). This association has also been found in mixed-gender samples (Ostrov & Godleski, 2009).

Support for a link between relational aggression and ADHD symptoms in adulthood may be extrapolated from the numerous studies identifying increases in other forms of aggression (e.g. Kern, Rasmussen, Byrd, & Wittschen, 1999; Richards, Deffenbacher, & Rosén, 2002; Theriault & Holmbeg, 2001) and social problems (e.g. Canu & Carlson, 2003, 2007; Paulson, Buermeyer, & Nelson-Gray, 2005; Ramirez et al., 1997; Shaw-Zirt, Popali-Lehane, Chaplin, & Bergman, 2005) in adults with symptoms of ADHD; however, excepting the present study, little work has *overtly* investigated this association beyond childhood and adolescence.

In addition to its well-supported co-occurrence with ADHD symptomology, relational aggression has also been identified as a correlate of Cluster B personality

pathology across the lifespan. In a sample of elementary school-aged girls, relational aggression predicted borderline personality disorder outcomes over a one-year span (Crick, Murray-Close, & Woods, 2005). Peer-nominated relational aggression in undergraduate women (but not men) was significantly associated with a number of BPD features, including affective instability, negative relationships, identity disturbances, and self-harm (Werner & Crick, 1999).

An association has likewise been found between relational aggression and ASPD symptoms across the lifespan. University students reporting a stronger tendency to relational aggression also possess a greater degree of ASPD symptoms (Werner & Crick, 1999; Ostrov & Houston, 2008) and symptoms of the ASPD-related trait cluster of psychopathy (Miller & Lynam, 2003). Of note, gender effects have been found for the degree of ASPD symptoms endorsed (with males typically endorsing more ASPD traits; e.g. Miller & Lynam, 2003; Ostrov & Houston, 2008) and the specific ASPD symptoms associated with relational aggression (e.g. Werner & Crick, 1999).

These studies, in conjunction with the present results, suggest an important tie between relational aggression and clusters of impulsive symptoms. However, given that relational aggression contributed unique variance to the prediction of reward sensitivity *beyond* the effect of ICD symptoms (i.e. impulsive symptoms of ADHD), the association between relational aggression and reward sensitivity merits consideration beyond the constructs' overlap with impulsivity.

Though no prior studies have overtly identified a link between relational aggression and reward sensitivity, further evidence for this relation may be gleaned from studies of human neuroendocrine functioning. It has been suggested that the BAS is

analogous to the human hypothalamic-pituitary-gonadal (HPG) axis, responsible for the release of testosterone and consequent inhibition of cortisol release (Terburg, Morgan, & van Honk, 2009). This body of research has also demonstrated that increased testosterone (and therefore decreased cortisol) increases the likelihood of aggression by focusing attention on the perceived threat; increased HPG activity also inhibits the cognitive control processes which would normally serve as a "conscience", modulating reward-sensitive impulses (reviewed in Terburg et al., 2009).

Importantly, low salivary cortisol, which would indicate greater activity in the BAS-analogous HPG axis, has been identified as a correlate of relational aggression in a non-clinical group of children (Murray-Close, Han, Cicchetti, Crick, & Rogosch, 2008). Contrary to their hypotheses, Murray-Close and colleagues found this cortisol effect on relational aggression to be consistent across gender groups. Similarly, the present study found a relation between these variables in a mixed-gender group. However, given that the vast majority of participants in the present sample were female, any analyses designed to rule out a moderating effect of gender on the relational aggression-reward sensitivity relation would be statistically underpowered. Further studies which equally sample across genders are needed to support the existence of this effect in both males and females. Nonetheless, the present study suggests that relational aggression stands as an important variable for consideration as a correlate of reward-sensitive personality.

Callous-Unemotional Personality Traits and Reward Sensitivity

Contrary to hypotheses, callous-unemotional (CU) personality traits (operationalized *a priori* as the total score from the Inventory of Callous-Unemotional

Personality Traits; Frick, 2004) were significantly related to neither the BAS total score nor the SPSRQ Sensitivity to Reward.

The failure of the ICU total score to predict reward sensitivity is somewhat surprising given a modest body of work suggesting altered reward processing in individuals possessing CU traits. Importantly, however, the majority of research in this domain has sampled individuals who display CU traits in the context of clinical concerns, particularly conduct disorder (Hawes & Dadds, 2005), incarceration (Marini & Stickle, 2010; Pardini et al., 2003; Verona et al., 2004), or a sample of mixed behaviour disorders (Lorber et al., 2011). Importantly, these studies' results have diverged, with some identifying increased reward sensitivity in these groups (e.g. Hawes & Dadds, 2005; Lorber et al., 2011; Pardini et al., 2003) and others finding *decreased* reward sensitivity in individuals with CU traits (e.g. Marini & Stickle, 2010; Verona et al., 2004).

The limited studies investigating altered reward sensitivity as a correlate of CU traits in non-clinical groups has provided more consistent evidence for an inverse relation between CU traits and reward sensitivity. Adolescents with CU traits appear to be less driven by rewards on a gambling task (Centifani & Modecki, 2013). Another study found the BIS/BAS Scales' Reward Responsiveness subscale to be negatively associated with a measure of CU traits (Roose, Bijttebier, Claes, & Lilienfeld, 2011). This finding echoes an earlier study (Roose et al., 2010), which found the ICU's Unemotional subscale to be inversely correlated with the BIS/BAS Scales' Reward Responsiveness subscale, while BAS Drive and Fun Seeking correlated positively with the ICU's Uncaring and Callousness scales. Moreover, ICU Unemotional was negatively related to scores on the Drive and Fun Seeking subscales in that study.

While these non-clinical studies are inconsistent with the failure of CU traits (i.e. the ICU Total Score) to correlate with reward sensitivity total scores in the present study, they are consistent with correlations which emerged from these data on a subscale level (see Table 4). As was found by Roose and colleagues (2010), scores on the ICU Callousness scale were associated with greater BAS Drive and Fun Seeking scale scores. However, the "unacceptable" ($\alpha = .44$) internal consistency for the Callousness subscale of the ICU suggests that this subscale is not measuring a unitary construct in this sample, and that any conclusion drawn from its significant correlations with these BIS/BAS scales are likely invalid.

Although the remaining subscale-level correlations between ICU and reward sensitivity measures are small in magnitude, aspects of this pattern of associations are of theoretical interest and will be briefly discussed. Specifically, correlations were statistically significant between BIS/BAS Reward Responsiveness and the ICU's Uncaring scale (reverse-scored) and total score. Of interest is the directionality of these relations: individuals endorsing more Reward Responsiveness items endorsed fewer Uncaring items and, perhaps as a result, had lower ICU total scores. Inspection of the corresponding BIS/BAS Reward Responsiveness and reverse-scored Uncaring items provides some insight into this finding; conceptually, both scales contain items which appear to tap constructs such as drive and positive affectivity. As such (and contrary to the hypothesized effect), there actually appears to be a theoretically-based, inverse relationship between some aspects of reward sensitivity and callous-unemotional personality traits. To this author's knowledge, this finding has not been produced in other studies; however, the negative correlation between ICU Unemotional and BAS

Reward Responsiveness found by Roose and colleagues (2011) may similarly tap an affectivity-related construct.

In sum, although the CU total score was neither significantly related to nor predictive of reward sensitivity summary variables, significant correlations at the subscale level highlight the conceptual overlap between these two constructs. These findings are congruent with previous findings suggesting diminished reward sensitivity in non-clinical individuals (e.g. Centifani & Modecki, 2013; Roose et al., 2010, 2011).

Limitations

While the present study yielded significant results, several limitations inherent to this study warrant attention. First, all relevant variables were measured via self-report. Using exclusively self-report instruments may introduce common method variance (Podsakoff, MacKenzie, Jeong-Yeon, & Podsakoff, 2003). Future studies may be made more robust by utilizing a multi-method approach in the measurement of key variables, perhaps including a combination of the behavioural and physiological measures previously described as well as self-report and rating scales completed by a reliable informant.

In a similar respect, this study's findings are limited by the lack of reliable measures of both reward sensitivity and the included predictor variables. As reviewed above, research exploring the reward sensitivity construct has been limited by the nonexistence of highly reliable and valid measures. Similarly, the present study employed clinical measures such as the WISPI-IV and the BAARS-IV to best capture ICD symptoms. While these questionnaires have been shown to be valid and reliable measures of clinical symptoms, scales specifically designed to measure the full spectrum

of these impulsivity-related constructs may more adequately capture ICD symptoms in non-clinical groups.

Additionally, the cross-sectional design of the present study limits conclusions drawn from this work. While this study has produced insight regarding the non-clinical, impulsivity-related correlates of reward sensitivity, future studies using longitudinal methods are needed to better understand the relation between these variables, and possible long-term outcomes associated with reward hypersensitivity and impulsive behaviour.

Finally, this study focused expressly on ICD symptoms as predictors of reward sensitivity in order to explore the reward sensitivity-impulsivity relation. However, future studies may benefit from the inclusion of other relevant variables, including Big Five personality traits. Elucidating the role such personality factors play (concurrently with impulsivity variables) in predicting reward sensitivity−as well as any interactions between these constructs−may provide further insight into mechanisms driving human motivation.

Conclusions

Despite these limitations, the present study has produced important findings regarding the motivational correlates of ICD symptomology in a non-clinical sample. While supporting previous work suggesting a link between reward sensitivity and ADHD symptoms, this study is the first to highlight the role of reward sensitivity in relational aggression. These findings are also critical for understanding impulse control from a theoretical standpoint, suggesting that personality and motivational factors such as reward sensitivity may play a role across a spectrum of impulsive behaviours.

These findings have broad implications for guiding intervention planning. In individuals with impulsive and socially alienating behaviour, those interventions that are likely to improve functioning could be those which employ tactics to capitalize on this reward-sensitive style. Future studies exploring specific implementation of this theoretical finding are necessary. However, by garnering greater understanding of the motivational mechanisms underlying a spectrum of impulsive symptoms and relational aggression, this study provides a theoretical grounding allowing the design of more effective interventions.

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Table 1
Participant Demographics

Categorical variables			
		N	%
Gender	female	186	82.7
	male	37	16.4
	other	1	0.4
	no response	1	0.4
Ethnic background	Aboriginal/First Nations	4	1.8
	Black/African descent	12	5.3
	Asian descent	25	11.1
	Hispanic/Latino	1	0.4
	Caucasian or non-Hispanic White/European descent	149	66.2
	Arab/Middle Eastern descent	17	7.6
	Other	14	6.2
	Prefer not to answer	3	1.3
Year of study	1	36	16.0
	2	41	18.2
	3	76	33.8
	4	54	24.0
	5	16	7.1
	6+	2	0.9
Employment status	Currently employed outside home	154	68.4
	Not currently employed	69	30.7
	Prefer not to answer	2	0.9
Relationship status	Single	121	53.8
	In a relationship	90	40.0
	Married / in a civil union	6	2.7
	Cohabitating	54	24.0
	Divorced	1	0.4
	Widowed	0	0.0
	Prefer not to answer	0	0.0
Continuous variables			
		M (*SD*)	Range
Age		21.09 (3.43)	17 - 43
Parental education (higher of parents' years of education)		15.49 (2.96)	8 - 26

Table 2
Descriptive Statistics

	Mean (SD)	Range	α	N_{items}
BAARS-IV Inattention	15.52 (4.68)	9 - 31	.85	9
BAARS-IV Hyperactivity	9.40 (2.83)	5 - 20	.62	5
BAARS-IV Impulsivity	6.65 (2.42)	4 - 14	.72	4
BAARS-IV Sluggish Cognitive Tempo (SCT)	18.58 (6.04)	9 - 36	.90	9
BAARS-IV ADHD Total	31.57 (8.03)	18 - 58	.91	27
WISPI-IV ASPD	1.36 (0.51)	1 - 4.33	.84	27
WISPI-IV BPD	2.60 (1.30)	1 - 6.61	.88	18
Relational Aggression	11.04 (2.84)	7 - 23	.60	7
ICU Uncaring	13.85 (3.30)	8 - 23	.68	8
ICU Callousness	13.74 (2.27)	11 - 24	.44	9
ICU Unemotional	12.38 (3.39)	5 - 20	.79	5
ICU Total	39.96 (6.50)	24 - 63	.76	24
SPSRQ Sensitivity to Reward	35.12 (3.89)	27 - 46	.72	24
BAS Reward Responsiveness	17.37 (1.99)	11 - 20	.66	5
BAS Drive	10.36 (2.54)	4 - 16	.78	4
BAS Fun Seeking	11.31 (2.18)	4 - 16	.60	4
BAS Total	39.04 (5.18)	25 - 52	.79	13

Note. Abbreviations: *SD* = standard deviation. α = Cronbach's alpha. BAARS-IV = Barkley Adult ADHD Rating Scale, Fourth Edition. RAQ = Relational Aggression Questionnaire. WISPI-IV = Wisconsin Personality Disorders Inventory. ICU = Inventory of Callous-Unemotional Traits.

Table 3

Normality Diagnostics

	Skew	Kurtosis
Age	3.08	12.81
Parental education (higher of parents' years of education)	0.56	1.25
BAARS-IV Inattention	0.76	0.16
BAARS-IV Hyperactivity	0.66	0.65
BAARS-IV Impulsivity	1.07	0.79
BAARS-IV Sluggish Cognitive Tempo (SCT)	0.54	-0.25
BAARS-IV ADHD Total	0.62	0.28
WISPI-IV ASPD	2.79	10.34
WISPI-IV BPD	0.80	-0.18
RAQ Total Score	1.14	1.98
ICU Uncaring	0.63	-0.09
ICU Callousness	1.77	3.89
ICU Unemotional	0.08	0.16
ICU Total	0.74	0.85
SPSRQ Sensitivity to Reward	0.07	-0.46
BAS Total	0.02	-0.38
BAS Drive	-0.06	-0.25
BAS Fun Seeking	-0.30	0.13
BAS Reward Responsiveness	-0.50	-0.33

Note. Abbreviations: BAARS-IV = Barkley Adult ADHD Rating Scale, Fourth Edition. RAQ = Relational Aggression Questionnaire. WISPI-IV = Wisconsin Personality Disorders Inventory. ICU = Inventory of Callous-Unemotional Traits.

Table 4
Variable Intercorrelations

	1	2	3	4	5	6	7	8	9	10	11
1. Age	1.00										
2. [2]Gender (dummy code isolating females)[2]	.05	1.00									
3. Gender (dummy code isolating males)	-.05	-.97‡	1.00								
4. Parental education (higher of parents' years of education)	-.15*	-.10	.10	1.00							
5. Inattention[3]	-.06	-.07	.03	-.08	1.00						
6. Hyperactivity[3]	-.14*	-.13	.13	-.07	.53‡	1.00					
7. Impulsivity[3]	-.13	.06	-.10	-.03	.42‡	.43‡	1.00				
8. Sluggish Cognitive Tempo (SCT)[3]	-.06	-.01	-.01	-.07	.72‡	.49‡	.30‡	1.00			
9. ADHD Total[3]	-.12	-.07	.03	-.08	.89‡	.79‡	.69‡	.68‡	1.00		
10. ASPD[4]	-.09	-.13	.15*	.04	.29‡	.24‡	.12	.17†	.29‡	1.00	
11. BPD[4]	-.11	.03	-.01	-.00	.56‡	.45‡	.31‡	.57‡	.57‡	.53‡	1.00
12. Relational aggression[5]											
13. Uncaring[6]											
14. Callousness[6]											
15. Unemotional[6]											
16. ICU Total[6]											
17. Sensitivity to Reward[1,7]											
18. Reward Responsiveness[8]											
19. Drive[8]											
20. Fun Seeking[8]											
21. BAS Total[8]											

Note. [1]Participants with history of traumatic brain injury excluded ($N_{included}$ = 207). [2]Point-biserial correlation. [3]Barkley Adult ADHD Rating Scale IV. [4]Wisconsin Personality Disorders Inventory IV. [5]Relational Aggression Questionniare. [6]Inventory of Callous-Unemotional Traits.. [7]Sensitivity to Punishment/Sensitivity to Reward Quesitonnaire. [8]BIS/BAS Scales. *$p < .05$ †$p < .01$ ‡$p < .001$

Table 4 (continued)
Variable Intercorrelations

	12	13	14	15	16	17[1]	18	19	20	21
1. Age	-.08	-.06	-.01	-.04	-.05	-.21†	-.03	-.01	-.08	-.05
2. Gender (dummy code isolating females)[2]	-.01	-.03	-.13*	-.24‡	-.19†	-.12	.13*	-.01	.08	.08
3. [2]Gender (dummy code isolating males)[2]	-.01	.03	.15*	.23†	.19†	.14*	-.13*	.01	-.10	-.09
4. Parental education (higher of parents' years of education)	.03	-.06	.00	.05	.06	.09	-.05	-.07	-.10	-.10
5. Inattention[3]	.28‡	.35‡	.42‡	.15*	.40‡	.08	-.08	-.08	.21†	.02
6. Hyperactivity[3]	.12	.08	.28‡	.18†	.23†	.14*	.00	.02	.18†	.09
7. Impulsivity[3]	.17*	.08	.19†	-.01	.10	.28‡	.06	.12	.28‡	.20†
8. Sluggish Cognitive Tempo (SCT)[3]	.26‡	.18†	.31‡	.13*	.27‡	.13	.01	-.12	.15*	.01
9. ADHD Total[3]	.26‡	.25‡	.40‡	.15*	.35‡	.18	-.03	-.00	.27‡	.10
10. ASPD[4]	.34‡	.46‡	.57‡	.22†	.55†	.15*	-.10	.16*	.10	.09
11. BPD[4]	.33‡	.27‡	.36‡	.12	.33‡	.18*	-.06	-.03	.11	.01
12. Relational aggression[5]	1.00	.21†	.34‡	.16*	.31‡	.29‡	.17†	.24‡	.11	.23‡
13. Uncaring[6]		1.00	.43‡	.21†	.77‡	.00	-.25‡	.06	-.01	-.13
14. Callousness[6]			1.00	.23‡	.67‡	.10	-.10	.15*	.19†	.11
15. Unemotional[6]				1.00	.71‡	-.06	-.09	.03	-.04	-.03
16. ICU Total[6]					1.00	.01	-.21†	.04	.04	-.04
17. Sensitivity to Reward[1,7]						1.00	.35‡	.29‡	.40‡	.45‡
18. Reward Responsiveness[8]							1.00	.40‡	.37‡	.73‡
19. Drive[8]								1.00	.40‡	.81‡
20. Fun Seeking[8]									1.00	.76‡
21. BAS Total[8]										1.00

Note. [1]Participants with history of traumatic brain injury excluded ($N_{included} = 207$). [2]Point-biserial correlation. [3]Barkley Adult ADHD Rating Scale IV. [4]Wisconsin Personality Disorders Inventory IV. [5]Relational Aggression Questionnaire. [6]Inventory of Callous-Unemotional Traits.. [7]Sensitivity to Punishment/Sensitivity to Reward Questionnaire. [8]BIS/BAS Scales. *$p < .05$ †$p < .01$ ‡$p < .001$

Table 5

Summary of Hierarchical Regression for Variables Predicting SPSRQ Reward Sensitivity (N = 207)

	B	*SE B*	β
Step 1			
Constant	39.92	1.64	
Age	-0.24	0.07	-.21***
Step 2			
Constant	36.46	1.39	
Age	-0.19	0.08	-.17**
BAARS-IV Impulsivity	0.39	0.11	.25***
Step 3			
Constant	32.88	2.02	
Age	-0.18	0.07	-.16*
BAARS-IV Impulsivity	0.33	0.10	.21*
RAQ Total	0.33	0.09	.25***

Note. $R^2 = .04$ for Step 1, $\Delta R^2 = .06$ for Step 2 ($p < .001$), $\Delta R^2 = .06$ for Step 3 ($p < .001$). Model: $R^2 = .16$, $R^2_{adj.} = .15$. *p < .05; **p < .01; ***p < .001. Abbreviations: BAARS-IV = Barkley Adult ADHD Rating Scale, Fourth Edition. RAQ = Relational Aggression Questionnaire.

Table 6
SPSRQ Final Model: Correlations of Predictors with SR

	Correlations with SPSRQ Reward Sensitivity					Structure coefficient	Squared structure coefficient
	Zero-order	sr	sr^2	pr	pr^2		
Age	-.21**	-.16	.03	-.17	.03	-.52***	.27
BAARS-IV Impulsivity	.28***	.21	.04	.22	.05	.68***	.46
RAQ Total	.29***	.24	.06	.26	.07	.73***	.53

Note. sr = semipartial correlation, pr = partial correlation. *p < .05 **p < .01
***p < .001. Abbreviations: SPSRQ = Sensitivity to Reward/Sensitivity to Punishment Questionnaire, SR = Sensitivity to Reward, BAARS-IV = Barkley Adult ADHD Rating Scale, Fourth Edition, RAQ = Relational Aggression Questionnaire, sr = semipartial correlation, pr = partial correlation.

Table 7

Summary of Hierarchical Regression for Variables Predicting BIS/BAS BAS Total (N = 225)

	B	*SE B*	β
Step 1			
Constant	36.19	1.00	
BAARS-IV Impulsivity	0.43	0.14	.20**
Step 2			
Constant	32.57	1.52	
BAARS-IV Impulsivity	0.35	0.14	.17*
RAQ Total	0.37	0.12	.20**

Note. $R^2 = .04$ for Step 1, $\Delta R^2 = .04$ for Step 2 ($p = .002$). Model: $R^2 = .08$, $R^2_{adj.} = .07$. $*p < .05$; $**p < .01$; $***p < .001$. Abbreviations: BAARS-IV = Barkley Adult ADHD Rating Scale, Fourth Edition. RAQ = Relational Aggression Questionnaire.

Table 8

BAS Total Score Final Model: Correlations of Predictors with BAS-T

	Correlations with BAS-T					Structure coefficient	Squared structure coefficient
	Zero-order	*sr*	*sr*^{2}	*pr*	*pr*^{2}		
BAARS-IV Impulsivity	.20***	.16	.03	.20	.04	.70***	.49
RAQ Total	.23***	.20	.04	.21	.04	.82***	.67

Note. sr = semipartial correlation, *pr* = partial correlation. *$p < .05$ **$p < .01$ ***$p <$ .001. Abbreviations: BAS-T = BIS/BAS BAS Total Score, BAARS-IV = Barkley Adult ADHD Rating Scale, Fourth Edition, RAQ = Relational Aggression Questionnaire, ICU = Inventory of Callous-Unemotional Traits. *sr* = semipartial correlation, *pr* = partial correlation.

APPENDICES

Appendix A

Today's Date: September 03, 2013
Principal Investigator: Miss Brianne Brooker
REB Number: 31041
Research Project Title: REB# 13-146 Predicting Reward Sensitivity in a Non-Clinical Population"
Clearance Date: September 3, 2013
Project End Date: November 28, 2014
Milestones:
REB Clearance-2013/09/03(Completed)
Renewal Due-2014/11/28(Pending)

_____

This is to inform you that the University of Windsor Research Ethics Board (REB), which is organized and operated according to the Tri-Council Policy Statement and the University of Windsor Guidelines for Research Involving Human Subjects, has granted approval to your research project on the date noted above. This approval is valid only until the Project End Date.

A Progress Report or Final Report is due by the date noted above. The REB may ask for monitoring information at some time during the project's approval period.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the REB. Minor change(s) in ongoing studies will be considered when submitted on the Request to Revise form.

Investigators must also report promptly to the REB:
a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
b) all adverse and unexpected experiences or events that are both serious and unexpected;
c) new information that may adversely affect the safety of the subjects or the conduct of the study.

Forms for submissions, notifications, or changes are available on the REB website: www.uwindsor.ca/reb. If your data is going to be used for another project, it is necessary to submit another application to the REB.

We wish you every success in your research.

Pierre Boulos, Ph.D.
Chair, Research Ethics Board
301 Assumption University
University of Windsor
519-253-3000 ext. 3948
Email: ethics@uwindsor.ca

Appendix B

Demographic Questionnaire

Personal information:

1. Month and year of birth (e.g. January 1992): _____

2. Today's date (e.g. October 20, 2013): _____

3. Your current age: _____ years old

4. Gender (<u>please circle one</u>): male   female   other   prefer not to answer

5. How do you describe your ethnicity (<u>circle all that apply</u>)?
    a. Aboriginal/First Nations
    b. Black/African descent
    c. Asian descent
    d. Hispanic/Latino
    e. Caucasian or non-Hispanic White/European descent
    f. Arab/Middle Eastern descent
    g. Other (please describe): _____
    h. Prefer not to answer

6. Your native (first) language: _____

7. Other languages spoken (if applicable): _____

8. Relationship status (circle one):
    a. Single
    b. In a relationship
    c. Married / in a civil union
    d. Cohabitating
    e. Divorced
    f. Widowed
    g. Prefer not to answer

9. Are you employed outside of the home?
    a. yes (please describe position): _____
    b. no
    c. prefer not to answer

Academic information:

1. Current year of study (please circle one):   1   2   3   4   5   6+

2. Current major: _____

3. Current GPA (if unsure, estimate): _____

4. Have you ever been diagnosed with a learning disability (circle one):
   a. yes (describe) _____
   b. no

5. Have you **ever** received educational accommodations (e.g. Individual Education Plan [IEP], extra time to take tests, etc.)? Please include time before you entered the University of Windsor.
   a. yes (describe) _____
   b. no

6. Are you **currently** receiving educational accommodations or services through the Student Disabilities Office (e.g. Individual Education Plan [IEP], extra time to take tests, etc.)?
   a. yes (describe) _____
   b. no

For the following three questions, please circle the response that best applies to you:

1	2	3	4
very false for me	somewhat false for me	somewhat true for me	very true for me

	very false for me	somewhat false for me	somewhat true for me	very true for me
1. I enjoy school.................................	1	2	3	4
2. I am excited by my program of study.	1	2	3	4
3. School is difficult.	1	2	3	4

Explain (optional): _____

Medical information:

1.   Have you ever had a concussion / head injury / traumatic brain injury?

a.   Yes (describe): _____

     _____ *(if yes, please answer questions 3-5)*

b.   No *(if no, proceed to Family History section on following page)*

If you answered yes to #2, please answer:

2.   Were you hospitalized?

a.   Yes (describe how long): _____

b.   No

If you answered yes to #2:

3.   Did you lose consciousness (i.e., did you pass out)?

a.   Yes (describe how long): _____

b.   No

If you answered yes to #2:

4.   Did you have trouble remembering anything as a result of your head injury (<u>circle all that apply</u>):

     a. Yes, I couldn't remember what happened for _____(minutes/hours) <u>before</u> my injury

     b. Yes, I couldn't remember what happened for _____(minutes/hours) <u>after</u> my injury

     c. No, I did not have trouble remembering as a result of my head injury

Family history:

Biological mother (if applicable):

    a. Years of education completed: _____

    b. Occupation, if currently employed: _____

    c. Number of biological children:_____

Biological father (if applicable):

    a. Years of education completed: _____

    b. Occupation, if currently employed: _____

    c. Number of biological children:_____

Other parent (if applicable, describe): _____

    a. Years of education completed: _____

    b. Occupation, if currently employed: _____

    c. Number of biological children:

Other parent (if applicable, describe): _____

    a. Years of education completed: _____

    b. Occupation, if currently employed: _____

    c. Number of biological children:_____

<u>Thank you for completing this survey!</u>

VITA AUCTORIS

Brianne Brooker was born in St. Louis, Missouri in the United States in 1990. She graduated from Calvin College in Grand Rapids, Michigan, in 2012, where she earned Bachelor's degrees in Psychology and German. She is currently a graduate student in Clinical Neuropsychology at the University of Windsor.   After earning her M.A. in 2014, she will continue studying in pursuit of a Ph.D. in Clinical Neuropsychology.