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Recent reviews indicate that Hyperactive-Impulsive symptoms associated with Attention-Deficit/Hyperactivity Disorder (AD/HD), which are considered the core symptoms of AD/HD, are caused by relatively distinct etiological pathways. Overactive behavioral approach motivation tendencies are among these proposed causal pathways and are addressed within Reinforcement Sensitivity Theory (RST). RST proposes that overactive behavioral approach tendencies are associated with over responsiveness to immediately reinforcing stimuli and result from an overactive appetitive motivational subsystem of the brain—the Behavioral Approach System (BAS). The BAS is dopaminergically-based and is associated with impulsivity-sensation seeking. However, other externalizing disorder symptoms, such as Psychopathy, are also associated with overactive BAS functioning and frequently co-occur with AD/HD symptoms. Given this co-occurrence, any relationship between Hyperactive-Impulsive AD/HD symptoms and the BAS may be spurious. The current study tested the hypothesis that BAS functioning is differentially associated with Hyperactive-Impulsive AD/HD and Psychopathy symptoms (Primary and Secondary forms) among a sample of college undergraduates (n = 232). In addition, a second motivational subsystem of the brain, the Behavioral Inhibition System (BIS), which is associated with sensitivity to punishment and trait anxiety, was also assessed. Correlational analyses indicated that performance on an experimental behavior task of BAS functioning was unrelated or was modestly related to

self-report measures of psychopathology and BAS functioning. However, correlations between self-report measures of BIS and BAS functioning with measures of psychopathology were significantly associated. Therefore, structural equation modeling of self-report measures of BIS and BAS functioning was conducted. This analysis assessed the relationship between each psychopathology variable with a BIS and BAS latent variable while also taking the other psychopathology symptoms into account. This analysis indicated that Hyperactive-Impulsive AD/HD symptoms are associated with high BAS functioning, and Primary Psychopathy symptoms are associated with high BAS and low BIS functioning. Secondary Psychopathy symptoms were positively correlated with BAS functioning, but this relationship failed to reach significance in the structural equation analysis. Instead, Secondary Psychopathy symptoms were associated high BIS functioning. Implications of these findings, particularly theoretical and treatment recommendations for AD/HD and its persistence into adulthood, and future research directions are discussed.

REINFORCEMENT SENSITIVITY THEORY, ADULT AD/HD SYMPTOMS,

AND COMORBIDITY: AN EXAMINATION OF PATHWAYS BASED

ON BEHAVIORAL APPROACH AND INHIBITION

by

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CHAPTER I

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (AD/HD) is a developmental disorder characterized by symptoms of inattention, hyperactivity, and impulsivity according to the *Diagnostic and Statistical Manual of Mental Disorders-IV-TR* (*DSM-IV-TR*; American Psychiatric Association [APA], 2000). Those diagnosed with AD/HD are categorized into Predominantly Inattentive, Predominantly Hyperactive-Impulsive, or Combined subtypes. Based on previous work (Barkley, 2001; Johansen, Aase, Meyer, & Sagvolden, 2002; Milich, Balentine, & Lynam, 2001; Nigg, 2001), the current paper takes the position that: (a) Hyperactive-Impulsive behaviors are the core symptoms of AD/HD, (b) poor sustained attention is a secondary result of such behaviors, and (c) that poor sustained attention is not specific to AD/HD. Therefore, Hyperactive-Impulsive AD/HD symptoms, not Inattentive symptoms, are of interest in the current study. (See Table 1 in Appendix A for a summary of AD/HD symptoms and Appendix A for all remaining tables.) Recently, overactive behavioral approach motivation tendencies have been offered as a likely causal pathway to Hyperactive-Impulsive behaviors characteristic of AD/HD (Nigg, Goldsmith, & Sachek, 2004; Nigg, 2006a). These approach tendencies are addressed within a neuropsychological account of personality called Reinforcement Sensitivity Theory (RST; Gray, 1970, 1982, 1991, Gray & McNaughton, 2000, Pickering & Gray, 1999). The purpose of the current study was to examine the relationship between

Hyperactive-Impulsive AD/HD behaviors among adults and RST variables, while also taking comorbid Psychopathy symptoms into account.

Attention-Deficit/Hyperactivity Disorder

Although historically considered a childhood disorder, follow-up studies of children diagnosed with AD/HD report persistence of the disorder into adulthood (see Barkley, 2006a; Barkley, Murphy, & Fischer, 2008; and Faraone et al., 2000, for reviews). This persistence into adulthood has a number of negative functional consequences in economic, occupational, social, and academic domains (Faraone et al., 2000; Mannuzza & Klein, 1999). Also, AD/HD is implied to be a categorical diagnosis according to the *DSM-IV-TR*, but empirical findings support that Hyperactive-Impulsive AD/HD symptoms are part of the extreme end of a continuum and that AD/HD can be considered a dimensional disorder (Levy, Hay, McStephen, Wood, & Waldman, 1997; see Barkley, 2006b, pp. 95-96, for a summary). Thus, the view that psychopathology represents the extremes of normal personality variation (Clark, 2005; Eysenck, 1987; Pickering, Corr, Powell, Kumari, Thornton, & Gray, 1997) is consistent with the dimensional view of AD/HD as a disorder based on a continuum.

Theoretical causal models of AD/HD emphasize poor inhibitory control (i.e., failure to inhibit goal irrelevant behavior) as the primary cause of Hyperactive-Impulsive behaviors (Barkley, 1997a, 1997b; Quay, 1988, 1997). Nigg (2001) reviewed these poor inhibition, or disinhibition, accounts and proposed a distinction between two different disinhibition types: executive and motivational. Executive disinhibition refers to difficulty withholding a motor or cognitive response so that internally represented goals

in working memory can be reached at a later time; motivational disinhibition refers to difficulty withholding cognitive or behavioral responses to environmental cues (either reward or punishment) that redirect attention to an unanticipated stimulus. Both disinhibition types are proposed to be independent causes of AD/HD (see Nigg, 2006a, for a review). Nigg (2001) categorized RST-based accounts as motivational given its emphasis on reward and punishment, which is discussed in greater detail below.[1] Although motivational disinhibitory accounts based on RST have a history of involvement with inhibitory models of AD/HD (Quay, 1988, 1997), RST has not been properly assessed within the AD/HD literature. For instance, recent findings suggest other aspects of RST should be examined as an alternative to Quay's motivational model of poor inhibitory control (e.g., Mitchell & Nelson-Gray, 2006). In particular, motivationally-based overactive behavioral approach tendencies have been proposed to result in Hyperactive-Impulsive AD/HD symptoms (Nigg, 2006a; Nigg et al., 2004).

Motivational accounts are important to defining disinhibition in AD/HD. First, some executive tasks, such as the Stroop Task, fail to identify differences between AD/HD and control groups (van Mourik, Oosterlaan, & Sergeant, 2005). This is consistent with a recent review of executive disinhibition accounts that concluded they yield modest effect sizes (Halperin & Schulz, 2006), implicating the study of additional causal pathways to AD/HD (i.e., motivational). Second, although motivational and executive accounts are proposed to be distinct causal pathways to AD/HD (reviewed in Nigg, 2006a), some executive tasks (i.e., the Stop Signal Task) are mediated by

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[1] In this context, motivation refers to responsiveness to immediate incentives, as opposed laymen use of this term (i.e., not putting forth effort, or lacking long-term values or goals).

overactive behavioral approach tendencies that are based on an RST motivational account (Avila & Parcet, 2001). Thus, it is important to identify motivational pathways to AD/HD since motivational disinhibition accounts may also be interrelated with executive disinhibition. Indeed, this is consistent with the temperamental literature in which similar regulatory "approach" (analogous to a motivational account based on RST) and "effortful control" tendencies (analogous to executive functioning) are proposed to be interrelated as the former may constrain the latter in early development (Rothbart, Ellis, & Posner, 2004).[2] Overall, these findings suggest assessing a motivational pathway to AD/HD, regardless of how executive and motivational accounts are related.

Third, in addition to Nigg's (2001) distinction between different types of disinhibition, an RST-based motivational account seems relevant to AD/HD given the effectiveness of behavioral modification programs on AD/HD samples and the emphasized role of reinforcement (Luman, Oosterlaan, & Sergeant, 2005; Pelham & Waschbusch, 1999). The emphasis of these programs on positive reinforcement to shape behavior is consistent with aspects of RST that emphasize personality traits associated with reward responsiveness. Finally, biological substrates affected by medications for AD/HD, such as methylphenidate, are also specified in RST (Pickering & Gray, 1999; Volkow, Wang, Fowler, & Ding, 2005; Volkow et al., 2002). Following a discussion of RST below, the application of this theory to AD/HD and hypotheses regarding the relationships between RST and AD/HD are provided.

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[2] It is most likely that these processes are interrelated in infancy and early childhood as suggested by Rothbart et al. (2004), but are separable in later development. However, assessing how executive and motivational accounts are related is outside of the scope of the current study. This paper takes the position consistent with Nigg (2006a) that these accounts are separate. This position is consistent with other AD/HD theories (e.g., Sonuga-Barke 2002, 2003) and empirical findings (Toplak, Jain, & Tannock, 2005).

Reinforcement Sensitivity Theory

Appetitive and aversive motivational systems that are proposed to underlie behavioral response tendencies, affective response tendencies, and stable personality traits have received substantial empirical interest (e.g., Cloninger, 1988; Depue & Collins, 1999; Fowles, 2001). However, one such model, RST, has nearly a 40 year history (Gray, 1970) and has a unique history of involvement with AD/HD in particular (i.e., Quay, 1988, 1997). RST (Gray, 1970, 1982, 1991; Gray & McNaughton, 2000; Pickering et al., 1997; Pickering & Gray, 1999) is an adaptation of Eysenck's (1967) personality model. Gray (1970, 1982, 1991) proposed the rotation of Eysenck's extraversion (E) and neuroticism (N), which he labeled the Behavioral Approach System (BAS) and the Behavioral Inhibition System (BIS). When superimposed on Eysenck's personality dimension, the BIS dimension can be thought of as a 30-degree rotated angle with its ends in the high N-low E quadrant (high BIS) and low N-high E quadrant (low BIS) (Gray, 1972). The BAS personality dimension is a rotation of E and N by 30-degrees so that high BAS is in the high N-high E quadrant and low BAS activity is in the low N-low E quadrant. These rotations place the BAS closer to E than N, whereas the BIS is closer to N than low E. Gray proposed that these rotated dimensions are biologically-based neural systems that reflect fundamental individual differences in sensitivity to cues of punishment and reward that are relatively stable. Normal personality is assumed to be situated on a continuum with psychopathological behavior towards the extreme ends. People toward the ends of the BIS and BAS dimensions are considered

more vulnerable to psychopathology (Pickering & Gray, 1999). Corr (2004) provides a thorough summary of RST's development and its current state.

Behavioral Approach System. The BAS—also referred to as the reward system, the behavioral activation system (Fowles, 1988), or the behavioral facilitation system (Depue & Collins, 1999)—responds to appetitive stimuli for reward (i.e., positive reinforcement) or relief from punishment (i.e., negative reinforcement) (Gray, 1991; Pickering & Gray, 1999, 2001). The BAS activates the organism in response to cues of reward and is characterized as the trait of impulsivity-sensation seeking[3] (see Pickering & Gray, 1999, for a review). Those high on the BAS trait are predicted to experience more motivational consequences (i.e., the person's arousal increases, which energizes any ongoing approach behavior) and reinforcing consequences (i.e., the consequence from learning a response when that response has the effect of eliciting BAS stimuli) of BAS activity than those with lower BAS trait levels (Pickering, 2004a; Pickering & Gray, 2001).

In a highly reactive BAS person, BAS-mediated approach will tend to predominate. These people will exhibit risky, sensation-seeking behaviors (Pickering & Gray, 1999). Problems with self-regulation can occur with overactive BAS behavior or with difficulty interrupting overactive BAS behavior and will appear as impulsive behavior. For example, BAS-mediated behavior is associated with a preference for immediate reward rather than a larger delayed reward (Avila & Parcet, 2000).

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[3] To avoid confusion with use of the term *impulsivity* in the RST and AD/HD literature, *BAS-mediated behavior* or other terminology that invokes the BAS will be used instead of the RST trait label that includes the term *impulsivity*.

Pickering and Gray (1999) provide a detailed account of the BAS neural network and indicate that dopaminergic functioning is involved in appetitive motivation. This account includes a network of ascending dopaminergic projections. Particularly, the mesolimbic dopaminergic pathway is indicated. It is comprised by projections from the ventral tegmental to the nucleus accumbens of the striatum (within the basal ganglia), amygdala, and the prefrontal cortex. These projections appear to play a role in facilitating selection of motor programs. The ultimate effect will be the facilitation of ongoing behavior approach responses. Interestingly, Gray cites the effectiveness of methylphenidate for AD/HD as indirect evidence for the dopaminergic basis of the BAS (Pickering & Gray, 1999) and implies that Hyperactive-Impulsive AD/HD symptoms are related to BAS functioning. Molecular genetic (Reuter, Schmitz, Corr, & Hennig, 2006) and brain imaging findings (Borrós-Loscertales et al., 2006) also support predictions for the BAS and its dopaminergic basis.

Behavioral Inhibition System. The BIS responds to conditioned stimuli for punishment and frustrative nonreward, as well as novelty and innate fear stimuli, to increase passive avoidance and extinction (Gray, 1982, 1991). For example, arousal of the BIS may cause an organism to stop any ongoing behavior and focus attention on environmental cues and lead to an orienting response. Those high on BIS are most sensitive to cues to punishment relative to those low on BIS. Heightened sensitivity to these cues reflects trait anxiety (Gray, 1991). Recent cognitively-based explanations of the BIS emphasize how it inhibits prepotent conflicting behaviors (i.e., behaviors that are dominant) when aroused, assesses risk, and scans the memory to resolve goal conflict

(see Gray & McNaughton, 2000, for a thorough review). These recent updates to RST

also emphasize that the BIS is responsible for the resolution of goal conflict in general,

such as approach and avoidance conflict (see Corr, 2004, for a summary; Gray &

McNaughton, 2000). The BIS is located in the septo-hippocampal system and is

connected to the amygdala and the prefrontal cortex. The septo-hippocampal system has

noradrenergic inputs from the locus coeruleus and serotonergic inputs from the raphe

nucleus (Gray, 1991; Gray & McNaughton, 2000).

Fight-Flight-Freeze System. The third RST system, the Fight Flight Freeze

System (FFFS, Gray & McNaughton, 2000), has traditionally been associated with

responsiveness to unconditioned aversive stimuli (Gray, 1991). However, Gray and

McNaughton's recent revisions also include responsiveness to conditioned aversive

stimuli for the FFFS as well. Although distinct at the neural level, the BIS and FFFS

overlap at the behavioral level. That is, presumably due to evolutionary pressures, the

BIS has a bias for potentially threatening information so that avoidant responses are

favored. Thus, although the BIS resolves conflict between the FFFS and BAS and is

responsive to both, the BIS is biased towards the FFFS (Corr & Perkins, 2006). See

Figure 1 (Appendix B), for an illustration of the interaction between these three systems

(see Appendix B for all remaining figures). The relationship of the FFFS to personality

has historically been more difficult to determine; however, in line with Gray and

McNaughton's (2000) most recent revisions, Corr (2004) has argued that the personality

dimension of high Neuroticism and low Extraversion (i.e., the BIS dimension within

Eysenckian space) actually reflects combined BIS and FFFS functioning. The current

paper takes this position as well and refers to this output as simply BIS output.

Applying RST to AD/HD: Underactive BIS, Overactive BAS, and Response Modulation

Quay (1988, 1997) was the first researcher to explicitly apply RST to AD/HD.

Quay's account proposed that Hyperactive-Impulsive AD/HD behaviors result from an

underactive BIS. According to an underactive BIS account, passive avoidance is the

learned inhibition of behavior following a threat of punishment or nonreward and is

under control of the BIS. In AD/HD, the BIS was proposed to provide little output

following the cue to a threat, which results in Hyperactive-Impulsive AD/HD behaviors.

However, comorbid internalizing disorders resulting from BIS overactivation are

common in children and adults diagnosed with AD/HD (August, Realmuto, MacDonald,

Nugent, & Crosby, 1996; Barkley, 2006c; Biederman et al., 1993). These comorbid

conditions, particularly anxiety, provide conflicting accounts of BIS activity and limit the

underactive BIS proposal. Additionally, questionnaire and experimental behavioral task

studies, discussed below, do not provide support for an underactive BIS.

There are two additional impulsivity accounts based on RST and both rely on

BAS overactivity: they are the overactivation of the BAS and poor response modulation

accounts. First, the overactive BAS account proposes that response inhibition is difficult

in the presence of cues to reward and results in overactive approach behavior (Newman

& Wallace, 1993). Consistent with findings that BAS-mediated behavior is associated

with preference for smaller immediate rewards than larger delayed rewards (Avila &

Parcet, 2000), children diagnosed with AD/HD have a shorter delay-of-reinforcement

gradient than normal controls (Johansen et al., 2002). Therefore, overactivation of the BAS would not be predicted for AD/HD samples when a reinforcing stimulus is presented with a long temporal delay. However, overactive reward responsiveness would be predicted for shorter delays.

Second, the response modulation hypothesis (Newman & Wallace, 1993; Patterson & Newman, 1993) relies on an overactive BAS as well. Impulsive behavior emerges as a result of deficient response modulation between the BAS and BIS. Specifically, poor response modulation is characterized by difficulty suspending a dominant approach set resulting from an overactive BAS and failure to automatically shift attention to more relevant environmental cues or feedback for controlled behavior. When cues to reward are present and activate the BAS in a person with deficient response modulation, the person may be more resistant to cues that activate the BIS. In other words, BAS activity is dominant and the BIS cannot interrupt despite the presence of cues to punishment. Instead, the person is overly-focused on goal-directed behavior associated with immediate reinforcement and has difficulty shifting attention to the nondominant response set. As a result, a person with poor response modulation may appear to have poorer reflective cognition to punishment cues (Patterson & Newman, 1993).

Newman and Wallace (1993) propose that those diagnosed with AD/HD have poor response modulation and thus an overactive BAS. Overall, any account that relies on BAS overactivation conflicts with Quay's (1988, 1997) differentiation between AD/HD and Conduct Disorder (CD). That is, Quay's account claimed that AD/HD is

associated with underactive BIS functioning, whereas CD is associated with overactive

BAS functioning. Thus, in accordance with Quay's account, over-responsiveness to

reward cues (i.e., high BAS) in AD/HD would be an artifact of comorbid CD symptoms.

Conversely, Newman and colleagues (Newman & Wallace, 1993; Patterson & Newman,

1993) state that this responsiveness is a core, generalized predisposition to impulsivity in

both AD/HD and CD.

RST-Based Studies of AD/HD: Questionnaire and Experimental Behavior Tasks

These three impulsivity accounts within RST (i.e., underactive BIS, overactive

BAS, and poor response modulation) have been assessed primarily by questionnaire and

experimental behavioral task methods. Overall, these findings suggest an involvement of

overactive BAS functioning in AD/HD, which implicates both overactive BAS and

response modulation accounts. However, methodological concerns in these studies limit

some findings. For instance, the comorbidity between AD/HD and CD can be

problematic as 25% to 60% of AD/HD children also qualify for a CD diagnosis (Barkley,

Fischer, Edelbrock, & Smallish, 1990; Dulcan & Benson, 1997; Klein & Mannuzza,

1991; Satterfield & Schell, 1997). This comorbidity is particularly pronounced among

children and adolescents who endorse higher Hyperactive-Impulsive AD/HD symptoms

(Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999). CD, typically

assessed among children and adolescents, is an antecedent to Psychopathy and

Psychopathy is largely considered the adult outcome of CD (Abramowitz, Kosson, &

Seidenberg, 2004). Indeed, Psychopathy is considered a core feature of CD (Frick &

Ellis, 1999).[4] Not surprisingly, AD/HD and Psychopathy symptoms frequently co-occur as well (Colledge & Blair, 2001; Mathias, Furr, Daniel, Marsh, Shannon, & Daugherty, in press). Since CD and Psychopathy result from overactive BAS functioning (Kimbrel, Nelson-Gray, & Mitchell, 2007; Knyazev & Wilson, 2004; Newman & Wallace, 1993; Patterson & Newman, 1993; Quay, 1988; Slobodskaya, Safronova, Knyazev, & Wilson, 2001), a spurious relationship between AD/HD and the BAS may appear.[5] Thus, this Psychopathy externalizing dimension should be taken into account when addressing this relationship. Additional limitations of previous research are noted below.

Questionnaire studies. Questionnaire-based studies suggest Hyperactive-Impulsive AD/HD symptoms are associated with overactive BAS functioning. For instance, Kepley (2002) found that a Combined AD/HD subtype group reported higher BAS scores than a predominantly Inattentive AD/HD group of adults. This finding is meaningful because the former group is characterized by elevated Hyperactive-Impulsive symptoms relative to the latter group. Similarly, Mitchell and Nelson-Gray (2006) reported that Hyperactive-Impulsive symptoms in a college sample predicted BAS scores based on two different methods of measurement (i.e., placement based on a composite measure of BAS and placement within Eysenck's dimensions). Johnson, Turner, and Iwata (2003) reported similar results, but did not differentiate between Inattentive and Hyperactive-Impulsive AD/HD symptoms.

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[4] Thus, while CD and Psychopathy may be discussed separately, they are considered different developmental points of the same construct.

[5] Although both AD/HD and CD/Psychopathy may share a common temperamentally-based overactive BAS predisposition and they should both therefore have an overactive BAS, both should have a unique relationship with the BAS while partialing out the symptoms of the other disorder.

This trend is also consistent among child and adolescent samples. For instance, Hyperactivity was significantly correlated with self-report BAS scores on a modified RST measure (Muris, Meesters, de Kanter, & Timmerman, 2005) and externalizing behaviors were significantly correlated with parent-reported BAS scores on another modified RST measure (Colder & O'Connor, 2004). Although the majority of these studies did not control for comorbid CD symptoms that could account for higher BAS scores (Colder & O'Connor, 2004; Johnson et al., 2003; Kepley, 2002; Muris et al., 2005), the same trend emerged when CD symptoms were considered (Mitchell & Nelson-Gray, 2006).

Experimental behavior task performance studies also suggest BAS functioning is related to Hyperactive-Impulsive AD/HD behaviors. In experimental behavioral tasks, performance is assessed following a manipulation of BIS and BAS stimuli (i.e., punishments and rewards, respectively). Depending on which stimuli are manipulated, responsiveness to these cues is considered an index of BIS and BAS functioning. Two tasks that are frequently administered in the AD/HD literature to assess BIS and BAS functioning, the motivated go/no-go task and card playing task, are discussed below.

Motivated go/no-go task studies (Newman, Widom, & Nathan, 1985; see Appendix C for a more thorough description of this task). Gomez (2003) assessed impulsive responding of a Combined AD/HD subtype group of male children and controls in conditions of the motivated go/no-go task that assess BIS functioning (punishment-only condition), BAS functioning (reward-only condition), and response modulation (reward-punishment condition) while controlling for anxiety, depression,

aggression, and intelligence. Although the AD/HD group displayed greater impulsivity in all three conditions, a within group comparison of the AD/HD participants indicated significantly poorer performance in the reward-punishment condition, suggesting AD/HD is associated with poor response modulation. However, there are methodological concerns about the go/no-go task that limit these findings. These concerns include (a) the proposal that the reward-punishment condition assesses response modulation, (b) carryover effects when different conditions of this task are administered to the same participants, and (c) whether both the reward-only and punishment-only conditions inadvertently activate both systems.

First, in the reward-punishment condition of the go/no-go task, this condition does not meet the standards outlined by Patterson and Newman (1993) to test response modulation. That is, this condition does not manipulate rewards to establish approach behavior before introducing cues to punishment. Rather, approach behavior is only assumed to become established, even though behavior can be either punished or rewarded from the onset of the task.

Second, an additional criticism of the go/no-go task is carryover effects between different conditions (Gomez, 2003; Nigg, 2001). For instance, in some studies, two or three of the conditions of the go/no-go task are administered to the same participant. Thus, a participant is administered a highly similar task at least twice with different reinforcement conditions. However, reinforced responding learned in one condition may affect performance in a subsequent condition.

Third and finally, each of the go/no-go task conditions may actually serve as BIS *and* BAS stimuli in all conditions, including those proposed to assess BIS-only activity and BAS-only activity (Gomez, 2003). For instance, in the reward-only condition, participants are (a) rewarded for responding to stimuli they are instructed to respond to or (b) rewarded for refraining from responding to stimuli in which they are instructed to withhold their response. However, it is possible that participants in this condition are expecting a reward for responding to a stimulus in which they are instructed to withhold their response. For instance, if responding to a stimulus does not result in an expected reward, then responding may lead to frustrative nonreward linked to BIS activity (Corr, 2002a). Alternatively, in the punishment-only condition, participants must refrain from responding to punishing stimuli or respond to stimuli to avoid punishment (i.e., passive and active avoidance, respectively). According to RST, active avoidance is attributed to BAS activity (Corr, 2002a). Thus, different conditions of the go/no-go task may inadvertently activate systems they do not intend to activate.

Additional studies that assessed responsiveness to differing conditions of the motivated go/no-go task are listed in Table 2. The limitations of these and other studies include the inclusion of a heterogenous AD/HD group[6], comorbidity (e.g., as already noted, CD and Psychopathy symptoms can affect performance), male-only or

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[6] AD/HD criteria has varied from *DSM-III*, *DSM-III-R*, and *DSM-IV* (*DSM-IV-TR* did not include any content revisions of *DSM-IV*). *DSM III* (1980) taxonomy recognized Attention Deficit Disorder (ADD) with Hyperactivity (ADDH) or without Hyperactivity (ADD). The *DSM-III-R* (1987) relabeled the disorder as Attention Deficit Hyperactivity Disorder. There were no subtypes because the disorder was viewed as unidimensional (i.e., diagnosis required endorsement of eight out of 14 undifferentiated Inattentive, Hyperactive, or Impulsive behaviors). It was not until *DSM-IV* (1994) that symptoms were divided into two dimensions (Inattentive and Hyperactive-Impulsive) and subtypes were introduced: Predominantly Inattentive, Predominantly Hyperactive-Impulsive, and Combined types.

predominantly male samples, small sample size, various reporting sources (i.e., parent, teacher, and self-report), and control for medication effects. Two additional concerns include reinforcement allocation policy (Luman et al., 2005) and reinforcement expectancy (Corr, 2002a; Gray, 1991) that can affect experimental behavioral task performance, which are discussed in more detail below.

Reinforcement allocation policy "concerns whether reinforcement allocation is based on *task performance* or whether it is based on *task participation* (irrespective of response accuracy)" (Luman et al., 2005, p. 186, emphasis added). This policy may confound studies that reward participants regardless of task performance. If there is an expectancy of reward for participating, this may influence the rate of reinforcement learning and task performance (Schultz, 2000).

Recent criticisms about experimental tasks assessing dimensions of RST emphasize the need to assess subjective reinforcement expectancy (Corr, 2002a). For instance, rewarding and punishing stimuli in an experimental task can elicit either BIS-mediated or BAS-mediated behavior. That is, BAS-mediated behavior is elicited by rewarding stimuli or removal of punishing stimuli; BIS-mediated behavior is elicited by punishing stimuli or removal of rewarding stimuli (i.e., frustrative nonreward) (Gray, 1991). Conditions that claim to assess approach behavior with the presentation of rewarding stimuli, for example, may actually elicit BIS-mediated behavior when the stimuli are removed or when approach behavior is elicited but failure to attain the reward results in frustration. Corr (2002a) suggests assessing levels of subjective reward and punishment to ensure reward and punishment manipulations are actually rewarding and

punishing to the participant. Thus, the perceived reinforcement value of the stimulus following its administration must be equal to or greater than the expected reinforcement prior to administration to be considered effective. Corr's (2002a) hypothesis about assessing reinforcement expectancy to ensure manipulations intended to be punishing or rewarding are actually punishing or rewarding has received empirical support (Kambouropoulos & Staiger, 2004). Among the studies reviewed in this dissertation, subjective reward and punishment levels were not assessed and may account for inconsistent findings.

Card playing task (Siegel, 1978; see Appendix D for a more thorough description of this task). The card playing task is proposed to assess response modulation as it assesses the perseveration of an appetitive response despite the introduction of punishment which makes responding maladaptive (Newman & Wallace, 1993; Patterson & Newman, 1993). Participants are financially rewarded for responding at the onset of the task to establish a dominant response set and are gradually punished for responding; the dependent measure is the number of cards that a participant will play before quitting and the amount of money earned.

Only two studies have administered the card playing task to assess AD/HD and yield conflicting results. Milich, Hartung, Martin, and Haigler (1994) administered the card playing task to ninety adolescent participants (60 males, 30 females). AD/HD and CD symptoms were assessed in accordance with *DSM III-R* (1987) criteria. AD/HD symptoms shared a significant relationship with error on the card playing task among both genders independent of CD symptoms, indicative of a response modulation deficit.

Fischer, Barkley, Smallish, and Fletcher (2005) assessed the performance of

young AD/HD adults who were identified as Hyperactive ($n = 147$) in childhood with a

community control group ($n = 70$). The Hyperactive group was divided into those with ($n = 68$) and without ($n = 53$) an adult AD/HD diagnosis (i.e., Hyperactive+AD/HD and

Hyperactive without AD/HD) and compared on a variety of behavioral tasks, including

the card playing task. The Hyperactive+AD/HD group did not perform significantly

worse than the other two groups. However, group differences emerged when the

Hyperactive group was divided into those with ($n = 46$) and without ($n = 100$) a lifetime

CD diagnosis. The Hyperactive+CD group played significantly more cards than the

Hyperactive group without CD. Thus, Fischer et al. suggested that poor response

modulation or overactive approach behavior is specific to CD—not AD/HD.

Some methodological concerns, however, may limit Fischer et al.'s (2005)

conclusions. First, the subtypes of the adult AD/HD group were not reported. Recall that

the Inattentive subtype endorses a lower frequency of Hyperactive-Impulsive symptoms,

the latter of which are the core symptoms of interest. Thus, a group of non-subtyped

AD/HD adults may be a heterogeneous one that includes the Inattentive subtype—this

subtype is not predicted to perform worse since they do not endorse many Hyperactive-

Impulsive symptoms. Additionally, a person diagnosed as Hyperactive in childhood does

not imply that as adults, if diagnosed with AD/HD in adulthood, he or she will meet

criteria for the Combined or Hyperactive-Impulsive subtype instead of the Inattentive

subtype. Lahey, Pelham, Loney, Lee, and Willcut (2005), for example, report that

AD/HD subtypes were likely to shift among a sample of preschoolers into elementary

school. Thus, the instability of subtypes over development suggests it is important to report AD/HD subtypes in longitudinal analyses. Another concern is that anxiety could improve performance on the card playing task since, based on RST, people that are more anxious are more responsive to punishing stimuli (Corr, 2002b). Although not reported, comorbid anxiety could have been more prevalent in the AD/HD group than the CD group and therefore account for the finding that the CD group performed significantly worse on this task. Also, reinforcement allocation policy (Luman et al., 2005) suggests that the $100 paid to participants for participating in the study could have affected performance and reduced the effectiveness of the financial reinforcer manipulated in the card playing task. Finally, the sample was primarily male (89%).

An adapted version of the card playing task for children, the door opening task, has been administered to AD/HD samples (Daugherty & Quay, 1991; Daugherty, Quay, & Ramos, 1993; Matthys, van Goozen, de Vries, Cohen-Kettenos, & van Engeland, 1998). In this computerized task, doors are used instead of cards. Once a door is opened, the child is provided feedback that the door was either a winning or losing door. These studies are summarized in Table 3 and provide some support for the response modulation deficit account. Although not reviewed in detail here, other tasks besides from the card playing task and motivated go/no-go task also manipulate reinforcers and punishers have also found a relationship between reward responsiveness and AD/HD (Toplak, Jain, & Tannock, 2005; Tripp & Alsop, 1999). However, since they are less commonly administered than the card playing task and motivated go/no-go task, they are not

discussed. Overall, however, they are consistent with the overactive BAS functioning

hypothesis and its association with Hyperactive-Impulsive AD/HD symptoms.

Card arranging reward responsiveness objective test (CARROT; Powell, Al-

Adawi, Morgan, & Greenwood, 1996). Finally, the CARROT is an experimental measure

of reward motivation (i.e., BAS activity) that has ecological validity and is linked to

dopaminergic functioning associated with the BAS (Pickering & Gray, 1999). This

promising RST task is a repeated measures card task in which participants are required to

sort a stack of cards as quickly as possible on four consecutive trials. During the third

trial, participants are offered a financial incentive for sorting faster. Their sorting rates are

compared between rewarded and nonrewarded trials to yield a reward responsiveness

index. Powell et al. (1996) demonstrated that performance on this task is mediated by

dopaminergic activity, which is commonly implicated with BAS functioning as its

biological foundation. To the authors knowledge, the CARROT has never been

administered to assess AD/HD symptoms.

Assessing Behavioral Inhibition and Approach

Overall, these questionnaire and behavioral task findings suggest that the most

likely impulsivity account within an RST-based framework would be either overactive

BAS functioning or poor response modulation. Both accounts are based on overactive

BAS functioning. While questionnaire studies are consistent in their findings, this

questionnaire method of measurement of BIS and BAS-related behaviors has proven to

be a difficult task (Pickering & Gray, 2001; Smillie & Jackson, 2005). Currently, the

BIS/BAS Scale (Carver & White, 1994) and the Sensitivity to Punishment and Sensitivity

to Reward Questionnaire (SPSRQ; Torrubia, Avila, Molto, & Caseras, 2001) are the most widely administered and most promising RST self-report scales. However, the BIS/BAS Scales yield a theoretically inconsistent number of factors and include inappropriate item content (Torrubia et al., 2001). While the SPSRQ yields two factors for the BIS and BAS, O'Connor, Colder, and Hawk (2004) reported a better two factor fit with a revision in which some items were omitted. O'Connor et al. (2004) also suggested adding some items and rewording current items for future versions of the SPSRQ. However, in its original version, the SPSRQ does yield two factors (O'Connor et al., 2001) and is associated with over responsiveness to immediately reinforcing and punishing stimuli as predicted (Avila & Parcet, 1997, 2000, 2002). Thus, self-report measures, particularly the SPSRQ, provide important information about RST constructs.

Behavioral tasks that provide converging evidence with self-report measures may strengthen findings. However, tasks such as the motivated go/no-go task have limitations (discussed above). The card playing task, conversely, has only one condition that assesses response modulation. It does not assess BIS- or BAS-only activity. This is problematic as the card playing task cannot test the basic assumptions of the overactive BAS and response modulation accounts as BAS-only tasks can. Although the CARROT has never been administered to assess AD/HD symptoms, it can test the basic assumptions of the overactive BAS and response modulation deficit accounts which the card playing task and motivated go/no-go task have been unable to address. Additionally, it could assess whether both CD/Psychopathy and AD/HD symptoms are uniquely associated with an overactive BAS (Newman & Wallace, 1993) or if CD/Psychopathy symptoms solely

account for this relationship between AD/HD symptoms and the BAS (Quay, 1988). Such an analysis would help to clarify the relationship between CD/Psychopathy, AD/HD, and the BAS.

For the current study, since the sample is composed of adults, Psychopathy symptoms were assessed. Recall that these symptoms are considered the core feature of CD (Frick & Ellis, 1999) and are the adult outcome of childhood CD (Abramowitz et al., 2004). According to the Fowles-Gray-Lykken hypothesis (Fowles, 2001; Gray, 1991; Lykken, 1995), Psychopathy is subdivided into primary and secondary forms. Primary Psychopathy is characterized by undersocialization, aggression, low anxiety, and impulsivity (Blackburn, 1975) and is proposed to result from low BIS functioning and normal BAS functioning. Secondary Psychopathy is similar to Primary Psychopathy, with the exception of relatively higher levels of anxiety and depression (Blackburn, 1975) and thus is proposed to result from normal BIS and high BAS. This differentiation of Psychopathy types in adulthood is consistent with the factor structure of these behaviors in childhood and the identification of two different types of Psychopathic traits. That is, "Type I Psychopathy" in childhood is associated with callous, impulsive, unempathic traits, whereas as "Type II Psychopathy" in childhood is associated with negative affect, impulsive, and antisocial traits (see Nigg, 2006b, p. 409, for a review). Type I Psychopathy in childhood descriptively overlaps with Primary Psychopathy in adulthood, whereas Type II Psychopathy in childhood descriptively overlaps with Secondary Psychopathy in adulthood.

Recently, Newman, MacGoon, Vaughn, and Sadeh (2005) reported that Primary Psychopathy is associated with low BIS and normal BAS, and Secondary Psychopathy is associated with high BAS and normal to high BIS. However, Kimbrel et al. (2007) reported that high BAS is associated with both forms of Psychopathy and that Primary Psychopathy is differentiated in that it is associated with low BIS. Studies that have assessed the relationship between Psychopathy in general and AD/HD indicate that impulsivity associated with both disorders may be an underlying mechanism for the development of AD/HD and Psychopathy, and their comorbidity (Colledge & Blair, 2001; Mathias et al., in press). In accordance with this proposal, BAS functioning may be the proposed underlying mechanism that accounts for both disorders.

Goals and Hypotheses

The goal of the present study was to investigate the relationship between AD/HD and RST, specifically Hyperactive-Impulsive AD/HD symptoms and the BAS. Indeed others have identified that overactive BAS responsiveness and the resultant approach tendencies is a pathway to AD/HD that "remains heuristic and in need of further study" (Nigg et al., 2004, p. 51). Also, the current study aimed to assess whether this relationship is better accounted for by comorbid Psychopathy/CD symptoms as proposed by Quay (1988, 1997) or if both disorders share a similar overactive BAS predisposition as proposed by Newman and colleagues (Newman & Wallace, 1993; Patterson & Newman, 1993). The following relationships were hypothesized:

1. Primary Psychopathy, Secondary Psychopathy, and Hyperactive-Impulsive AD/HD symptoms will correlate positively with performance on a behavior task of BAS

functioning (i.e., the CARROT) and a self-report of BAS functioning (i.e., the Sensitivity to Reward scale of the SPSRQ).

2. Measures of BAS functioning at the self-report (i.e., the Sensitivity to Reward scale of the SPSRQ) and behavioral performance (i.e., the CARROT) levels will have a statistically significant positive relationship. This relationship will be particularly strong for participants who report the reward in the CARROT more reinforcing than their expectations.

3. BIS functioning was assessed as well and was measured by the Sensitivity to Punishment scale of the SPSRQ. While Hyperactive-Impulsive AD/HD symptoms are not predicted to be related to the BIS, Primary Psychopathy will be negatively correlated with the BIS and Secondary Psychopathy will be positively correlated with the BIS.

Structural equation modeling was conducted for the remaining hypotheses. A latent variable of BAS functioning was hypothesized to be created from the SR scale of the SPSRQ and CARROT score. The following predictions for this model are summarized below.

4. The SR scale of the SPSRQ and the CARROT total score will load onto a common latent variable.

5. Hyperactive-Impulsive AD/HD symptoms will be significantly associated with the BAS latent variable while taking the influence of Primary and Secondary Psychopathy symptoms into account.

6. Primary Psychopathy symptoms will be significantly associated with the BAS latent variable while taking the influence of Hyperactive-Impulsive AD/HD symptoms and Secondary Psychopathy symptoms into account.

7. Secondary Psychopathy symptoms will be significantly associated with the BAS latent variable while taking the influence of Hyperactive-Impulsive AD/HD symptoms and Primary Psychopathy symptoms into account.

8. The relationship between the BIS (as indexed by the Sensitivity to Punishment scale of the SPSRQ) and Hyperactive-Impulsive AD/HD, Primary Psychopathy, and Secondary Psychopathy symptoms will be assessed. While Hyperactive-Impulsive AD/HD symptoms were not predicted to be related to the BIS, Primary Psychopathy were predicted to be related to low BIS and Secondary Psychopathy to high BIS.

CHAPTER II

METHOD

Participants

A sample of 252 undergraduate students enrolled in Introduction to Psychology

courses at the University of North Carolina at Greensboro (UNCG) participated. Of these

participants, 20 were excluded from analyses due to invalid item responses as indicated

by Infrequency Scale for Personality Measures scores that exceeded a score of 2, which is

the suggested threshold indicating random response patterns (Chapman & Chapman,

1986). There were no exclusionary criteria based on gender, age, or ethnicity. The

demographics of the remaining 232 participants are summarized in Table 4. The mean

age of the sample was 19.30 (*SD* = 3.51). The composition of the sample was primarily

female (62%) and Caucasian (63%). The gender, age, and ethnicity distribution of the

sample is typical of undergraduate Introduction to Psychology courses at UNCG.

Materials

General Information Questionnaire. This questionnaire (Appendix E) consists of

items addressing general demographic information such as age, gender, race or ethnic

origin, college major, and grade point average.

AD/HD Symptoms. The Conners' Adult ADHD Rating Scale (CAARS; Conners, Erhardt, & Sparrow, 2000; Appendix F) is a 66-item self-report measure of AD/HD behaviors in adults. Response options are on a four-point Likert scale and range from "not at all true" to "very much true." Among its scales, the CAARS yields symptom scales based on the *DSM-IV* (APA, 1994) criteria for AD/HD (i.e., Inattention and Hyperactivity-Impulsivity) with modified wording for adults (e.g., "I am restless or overactive," as opposed to "often runs about or climbs excessively in situations in which remaining seated is expected") and provides *DSM-IV* cut-off scores to identify those experiencing elevated AD/HD symptoms. The CAARS has demonstrated adequate reliability (internal consistency ranging from 0.86 to 0.92, test-retest reliability ranging from 0.80 to 0.91) and validity (Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999). The Hyperactivity-Impulsivity symptom severity score from the CAARS was used for the current study.

Psychopathy Symptoms. Levenson's Self-Report Psychopathy Scale (LSRP; Levenson, Kiehl, & Fitzpatrick, 1995; Appendix G) is a self-report measure designed to assess Primary and Secondary Psychopathy. The 16-item Primary Psychopathy scale assesses callous, selfish, and manipulative attitudes, whereas the 10-item Secondary Psychopathy scale assesses impulsive and self-defeating behaviors. Participants respond to items on a 4-point Likert scale that ranges from "disagree strongly" to "agree strongly." The LSRP has shown acceptable internal consistency and good test-retest reliability over an 8-week period (Lynam, Whiteside, & Jones, 1999). In addition, the

LSRP has shown good convergent validity with Psychopathy and anti-social behavior measures (Levenson et al., 1995; Lynam et al., 1999).

BIS and BAS self-report. The Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ; Torrubia et al., 2001; Appendix H) is a 48-item binary response self-report measure designed to assess respondent's levels of impulsivity and anxiety along Gray's personality dimensions. Several studies provide support for this measure's ability to reliably and validly reflect individual differences along Gray's dimensions. Additional construct and discriminant validity findings are provided by Avila (2001). The Sensitivity to Punishment (SP) subscale indexed BIS functioning and the Sensitivity to Reward (SR) subscale indexed BAS functioning.

Infrequency Scale for Personality Measures (Chapman & Chapman, 1986). This 13-item infrequency scale (Appendix I) was intermixed in the questionnaire packets for validation assessment. These infrequency items were presented in a "yes" or "no" format and pertained to events that are unlikely to occur and indicate a random response style. Participants that endorsed greater than two items were discarded.

Experimental BAS Behavioral Task. The Card Arranging Reward Responsiveness Objective Test (CARROT; Powell et al., 1996) measures the increase in speed on a simple card-sorting task in response to a financial reward. Participants sort a pack of cards, each card with five digits, one of the digits being a one, two, or three, into three trays labeled one, two, and three. There are four trials (T1-T4). T1 involves sorting 60 cards into trays, with the researcher recording the number of seconds it takes to sort the cards. This trial also controls for any sensory, motor, or cognitive deficits (Powell,

Pickering, Dawkins, West, & Powell, 2004). T2 involves sorting 100 cards and sorting

them again into trays until the researcher tells the participant to stop. The time limit for

this trial is equal to the number of seconds it took the participant to sort the cards in T1.

T3 is the same as T2, except that a monetary reward ($.20) is provided for every five

cards sorted correctly. Money is placed in front of the participant as the fifth card is

sorted into the correct tray. T4 is the same as T2. T4 controls for fatigue or practice

effects that may arise during the task, both of which may influence response speed. A

final score is computed by subtracting T3 from the average of T2 and T4 (final score =

T3-[(T2+T4)/2)]). The CARROT score assessed BAS functioning in the current study.

See Appendix J for more administration details and examples of stimulus materials.

Reinforcement expectancy. Reinforcement expectancy was assessed just prior to

and following CARROT administration. Reinforcement expectancy was estimated by

subtracting perceived reward following stimulus presentation (i.e., "how rewarding did

you find this task to be?") from expected reward prior to stimulus presentation (i.e., "how

rewarding do you expect this task to be?") in accordance with Kambouropoulos and

Staiger's (2004) guidelines. Participants rated their expected and perceived reward on a

scale from 1 (not at all) to 100 (extremely). The reinforcer is considered effective if the

difference score is positive.

Procedure

Undergraduate students enrolled in Introduction to Psychology classes at UNCG

were eligible to participate—there were no required qualifying criteria. Posted times

allowed students to sign up to participate and earn experimental course credit for

participation. This study received Institutional Review Board approval from the University of North Carolina at Greensboro.

When participants arrived to complete the study, they were informed of the confidentiality of their responses, guided through consent procedures, and signed a standard consent form (Appendix K). Following, participants were asked to complete the general information questionnaire, CAARS, SPSRQ, LSRP, and additional measures not reported in this study. The order of these questionnaires was randomized. While completing the self-report measures, participants were randomly selected at different times during this session to complete the CARROT administered by a research assistant in another room. Just prior to and following the CARROT, reinforcement expectancy was assessed. Five research assistants who received training in CARROT administration from the primary investigator administered the CARROT for this study. Of the five research assistants, two were female research assistants and administered the CARROT to 79 participants, whereas three were male research assistants and administered the CARROT to 153 participants. While the number of participants varied to whom each research assistant administered the CARROT (range of $n = 16$ to 111), there were no statistically significant differences in CARROT scores between research assistants. That is, a univariate ANOVA was performed to assess if there were any differences in scores on the CARROT between different research assistants, which were then followed by post hoc planned comparisons using Tukey's HSD procedure. Although the ANOVA indicated a between group difference, $F(4, 227) = 2.81$, $p = .03$, post hoc comparisons indicated that there were no statistically significant group differences (p's ranged from .11 to .94). This

indicates uniformity in CARROT administration in the current study across research assistants who administered the CARROT. In addition, whether the gender of the research assistant administering the CARROT affected participant performance on the CARROT was also assessed. A univariate ANOVA indicated that CARROT scores were not statistically different based on the gender of the research assistant administering the CARROT, F (1, 230) = .43, p = .51. Also, male and female participant CARROT scores did not statistically differ based on the gender of the research assistant. That is, among the male sample (n = 89), 30 were administered the CARROT by a female research assistant, whereas 59 were administered the CARROT by a male research assistant. A univariate ANOVA indicated that CARROT scores were not statistically different for the male sample based on the gender of the research assistant, F (1, 87) = .63, p = .43. Among the female sample (n = 143), 49 were administered the CARROT by a female research assistant, whereas 94 were administered the CARROT by a male research assistant. A univariate ANOVA indicated that CARROT scores were not statistically different for the female sample based on the gender of the research assistant, F (1, 141) = .03, p = .87. Following completion of all questionnaires and the CARROT, participants were debriefed about the nature of the study.

CHAPTER III

RESULTS

Preliminary Data Analyses

Alpha was set at .05 for all analyses. A check of variable distributions indicated

that all personality and psychopathology variables were normally distributed. For

instance, histograms appeared to conform to a normal distribution. Skewness and kurtosis

were also examined to assess normality. The absolute values of skewness and kurtosis

were all less than 1.0, with the exception of the kurtosis index for total CARROT scores

(kurtosis = 1.30). Following Kline's (2005) guidelines for skewness and kurtosis, all of

these values appeared normally distributed. Table 5 lists Cronbach coefficient alpha

values. Internal consistency values ranged from adequate to very good, indicating that

responses were consistent across items within each respective scale. Mean scores for the

psychopathology and RST variables are listed in Table 6.

Correlational Analyses

Hypotheses 1 through 3 regarding the relationships among Hyperactive-Impulsive

AD/HD symptoms, Primary Psychopathy symptoms, Secondary Psychopathy symptoms,

the CARROT score, the Sensitivity to Reward scale score from the SPSRQ, and the

Sensitivity to Punishment scale score from the SPSRQ were assessed by analyzing a

bivariate, zero-order correlation matrix. Pearson product-moment correlations for the

Sensitivity to Reward, Sensitivity to Punishment, CARROT, Hyperactivity-Impulsivity, Primary Psychopathy, and Secondary Psychopathy scale scores are listed in Table 6. These correlations indicated that the Sensitivity to Punishment scale was significantly correlated only with the Secondary Psychopathy scale—it was not significantly related to any other variables.

Table 6 also indicates that the multi-method assessment of the BAS with the Sensitivity to Reward scale and the CARROT total score yielded a positive, yet modest relationship ($r = .12$, $p = .06$). This relationship could be attenuated by including participants that did not find the manipulation effects of the CARROT reinforcing (i.e., reinforcement expectancy ratings exceed reinforcement experience ratings). However, among the participants that reported their reinforcement experience was greater than their expectancies ($n = 188$), the correlation between CARROT total score and Sensitivity to Reward scale score did not increase and was not statistically significant ($r = .09$, $p = .22$). Among psychopathology variables for this portion of the sample, CARROT total score correlations did not increase either. That is, the correlations between the CARROT total score and Hyperactive-Impulsive symptoms ($r = .08$, $p = .30$), Primary Psychopathy symptoms ($r = .16$, $p = .03$), and Secondary Psychopathy symptoms ($r = .10$, $p = .18$) were positive, yet modest for this subset of the sample.

Conversely, among participants who indicated that the manipulation was not effective (i.e., reinforcement expectancy rating exceeded reinforcement experience rating) ($n = 43$), the relationship between CARROT scores and other variables such as the Sensitivity to Reward scale should decrease. Although the relationship is not statistically

significant, the direction of the relationship between the CARROT total score and Sensitivity to Reward did not decrease as predicted ($r = .26$, $p = .09$). Also, the relationship between the CARROT total score and Hyperactivity-Impulsivity, Primary Psychopathy, and Secondary Psychopathy symptom scores did not decrease either. That is, the correlation between CARROT total scores and these psychopathology symptoms were positive, but not statistically significant (r's ranged from .14 to .18).

Overall, these findings indicate that participant expectancy and experience of reinforcement is not related to performance in the current sample and does not account for the modest correlations between the CARROT and the Sensitivity to Reward, Hyperactivity-Impulsivity, Primary Psychopathy, and Secondary Psychopathy scale scores. One possible explanation for this muted relationship between CARROT scores with the Sensitivity to Reward scale and psychopathology symptom variables is a limited range of CARROT scores. Indeed, Figure 3 is a scatterplot of CARROT scores with Sensitivity to Reward scale scores and indicates there is little variance among CARROT scores. CARROT scores, therefore, are not considered in the remaining analyses given that they have a consistent restricted relationship with all of the psychopathology symptom and RST variables. Additional details, limitations, and future directions for the CARROT are addressed in the Discussion section.

Consistent with the hypotheses, Table 6 indicates that the Sensitivity to Reward scale is significantly correlated with Hyperactive-Impulsive, Primary Psychopathy, and Secondary Psychopathy scale scores. In addition, the significant correlations among Hyperactive-Impulsive, Primary Psychopathy, and Secondary Psychopathy scores

indicate a high degree of symptom co-occurrence. Because of this overlap, any

significant correlations between either of these symptoms with the Sensitivity to Reward

scale score can be a spurious relationship. Pearson product-moment correlations were

also calculated by gender as well (Table 7). This table indicates that the relationships

between psychopathology and RST variables are generally consistent between males ($n =$

89) and females ($n = 143$).

Structural Equation Modeling Analyses

    Structural equation modeling overview. To test the remaining hypotheses,

structural equation modeling based on maximum likelihood estimation was conducted

using version 7.0 of AMOS (Analysis of Moment Structures; Arbuckle, 2006). Structural

equation modeling involves assessing how well a proposed model accounts for the

covariance among measures of interest. In other words, structural equation modeling

assesses the fit of a proposed model with data. Models that lack fit are indicative of other

relationships among variables or error that was not specified and is thus not accounted for

in the model.

    Structural equation modeling can be viewed as a combination of path analysis and

confirmatory factor analysis (Kline, 2005). A path analysis will assess the independent

relationships of different variables to assess hypothesized causal effects (i.e., the

structural portion of the model), whereas factor analysis will allow a latent variable based

on SR and CARROT scores to be created (i.e., the measurement portion of the model).

Additionally, structural equation modeling will control for the overlap among predictor

variables (i.e., Psychopathy and Hyperactive-Impulsive AD/HD symptoms) to determine

unique effects on the latent criterion variable (i.e., BAS) and allow for the assessment of alternative models. For example, the predicted model that included the CARROT (see Appendix B, Figure 2[7]) may be tested against a null model and an alternative model. As an example of an alternative model, Hyperactive-Impulsive symptoms may be artificially related to the BAS because of overlap with Psychopathy symptoms as proposed by Fischer et al. (2005). Thus, a model in which Psychopathy symptoms mediate Hyperactive-Impulsive symptoms and the BAS can be assessed.

The current sample size ($n = 232$) is considered large and acceptable for structural equation modeling (Kline, 2005; Thompson, 2000). Structural equation modeling was conducted because it has a number of advantages over other techniques (e.g., regression or path analysis). For instance, traditional multivariate procedures either do not assess for or correct measurement error (e.g., regression and general linear models assumes error in the independent variable is negligible and not present), whereas structural equation modeling provides estimates of error variance parameters (Bryne, 2001). Also, structural equation modeling allows for assessing both observed and latent variables, whereas traditional data analytic methods are based on observed variables only (Bryne, 2001). In addition, structural equation modeling tests an entire model in one analysis while simultaneously testing individual parameter estimates, whereas alternative methods such as regression analysis would require running multiple regressions. Performing the latter would increase the likelihood of committing Type I errors.

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[7] Note that Figure 2 is a depiction of the original model prior to assessing the data that yielded restricted relationships with the CARROT and the removal of this variable from the remaining analyses.

According to structural equation modeling terminology, the predictor variables are referred to as exogenous variables, whereas the dependent variables are referred to as endogenous variables. The use of circles or ellipses in Figure 2 represents a latent variable; the use of squares or rectangles in Figure 2 represents observed variables. Although omitted for simplicity in Figure 2, structural equation modeling also yields two types of error: (a) measurement error associated with observed variables that are thought to underlie a latent variable (e.g., error associated with the CARROT and SR scores in Figure 2) and (b) residual error terms, or disturbances, that represent error in the prediction of endogenous variables from exogenous variables (e.g., error associated with BAS and BIS variables in Figure 2) (Bryne, 2001; Kline, 2005). The first type of error derives from random measurement error (e.g., resulting from a measure that has poor internal consistency) and nonrandom measurement error (Bryne, 2001). Nonrandom error refers to some characteristic that is specific to a particular indicator variable, such as effects due to a particular measurement method or particular stimuli that make up some task (Kline, 2005). The second type of error (i.e., disturbance terms), "represent all causes of an endogenous variable that are omitted from the structural model" (Kline, 2005, p. 69). Thus, disturbances can be considered unmeasured exogenous variables or omitted causes in the model. All endogenous variables in structural equation modeling have a disturbance term. Overall, both measurement and residual error terms are representative of unobserved variables, which is why it is standard in structural equation modeling to represent these sources of error in circles (Bryne, 2001).

The model in Figure 2 and all other models assessed in this study are recursive (i.e., it includes arrows that indicate one direction, as opposed to bi-directional arrows) and overidentified. An overidentified model refers to a property of the model, not the data, and indicates that a unique estimate of each parameter is possible (i.e., the model is testable) and is therefore preferable for a model (Kline, 2005). Identification is calculated by identifying the number of observations in a model and the number of parameters in a model (Kline, 2005). The total number of observations is calculated with the following formula: $v(v+1)/2$, where v is the number of observed variables. The total number of parameters equals "the total number of variances and covariances (i.e., unanalyzed associations) of exogenous variables that are either observed or unobserved (i.e., disturbances) and direct effects on endogenous variables from other observed variables" (Kline, 2005, p. 101). In an overidentified model, the number of parameters is less than the number of observations, which results in positive degrees of freedom (Bryne, 2001). For example, Figure 2 yields 21 observations (v = 6) and 17 distinct parameters to be estimated. Therefore, $21 - 17 = 4$ degrees of freedom in Figure 2.

Two other types of model identification properties are just-identified models and underidentified models. Models that are just-identified indicate that the number of parameters is the same as the number of observations in a model. Therefore, it has no degrees of freedom and can never be rejected (Bryne, 2001). Models that are underidentified indicate that the number of parameters is greater than the number of observations in a model. Therefore, it has negative degrees of freedom and an infinite number of solutions are possible (Bryne, 2001). Thus, overidentified models are

preferable in structural equation analyses. Models that are not overidentified should be respecified prior to data collection (Kline, 2005).

Measurement portion of the model. Given that the CARROT is not included in any additional analyses, the measurement portion of the proposed model (Figure 2) was modified. The inclusion of the CARROT total score as an indicator in Figure 2 allowed for creating a latent variable of the BAS. Accordingly, omitting the CARROT total score makes the Sensitivity to Reward scale of the SPSRQ the only indicator of the BAS latent variable and therefore makes the BAS an observed variable. As an alternative to using single indicators of the BAS variable (i.e., the Sensitivity to Reward scale of the SPSRQ as the only observed variable of BAS) and BIS variable (i.e., the Sensitivity to Punishment scale of the SPSRQ), item parcels were used to create a latent variable of BAS and BIS (see Coffman & MacCullum, 2005, for a review and demonstration of the advantages of using parcels instead of total scale scores). Item parcels refer to a total score of a set of homogenous items from a larger scale (i.e., a miniscale) (Kline, 2005). For example, the Sensitivity to Reward scale was split into four subscales composed of six items each (i.e., parcel one was composed of items 2, 4, 6, 8, 10, and 12 from the SPSRQ, parcel two was composed of items 14, 16, 18, 20, 22, and 24 from the SPSRQ, parcel three was composed of items 26, 28, 30, 32, 34, and 36 from the SPSRQ, and parcel four was composed of items 38, 40, 42, 44, 46, and 48). These subscales or parcels are now considered indicators of a latent BAS variable. Similarly, the Sensitivity to Punishment scale of the SPSRQ was also split into four subscales or parcels composed of six items each. Both Sensitivity to Punishment and Reward scale parcels were determined

by grouping the first six items together, then the second set of six items, and so on for each respective scale. To assess whether the results are dependent on the specific grouping of items for each parcel, different groupings of the items into parcels were considered and led to similar conclusions. The revised model in Figure 4 summarizes the measurement portion of the model. The entire revised model that includes the measurement and structural portions are illustrated in Figure 5.

Structural equation models with item parcels as indicators are called partial disaggregation models, whereas total disaggregation models refer to structural models in which all individual items of a scale load onto a factor (Bagozzi & Heatherton, 1994; Leone, Perugini, Bagozzi, Pierro, & Mannetti, 2001). Partial disaggregated models are more advantageous because total disaggregated models (a) require more parameters to be estimated and thus require larger samples and (b) include only one item as an indicator and are thus more vulnerable to measurement error and sample specificity (Leone et al., 2001; Little, Cunningham, Shahar, & Widaman, 2002). Since partial disaggregation models reduce the number of observed variables and parameters, smaller sample sizes are permitted, and computational problems are less likely. Also, creating parcels to create a latent variable is advantageous because the score reliability of parcels tends to be greater than that for individual items (Kline, 2005).

When a latent variable is created in structural equation modeling, a scale for the latent variable has to be determined since it has no definite scale. The standard approach to assigning this scale to a latent variable is to constrain a single factor loading for a latent variable to 1.0, which is typically called a unit loading identification constraint. In

reference to Figure 4, one of the four pathways from the BAS and BIS latent factors is

designated as a fixed value (Bryne, 2001). This common scaling procedure is also

conducted for disturbance and error terms as well, which are illustrated in Figure 5 as

well (Kline, 2005).

   Structural portion of the model. Prior to assessing individual pathways, assessing

the overall fit of a structural equation model requires assessing fit statistics for the entire

model. In structural equation modeling, however, there is no one standard fit index.

Instead, a number of fit indices are typically reported as different fit indices are affected

by different factors. For example, it is standard to report a chi-square statistic, although

reporting this statistic only can be problematic since it is sensitive to sample size (Kline,

2005). It is standard, therefore, to report and interpret a minimal set of fit indices, the

most typical of which are the chi-square statistic, the root mean square error of

approximation (RMSEA), the comparative fit index (CFI), the goodness-of-fit index

(GFI), the adjusted goodness-of-fit index (AGFI), and the normed fit index (NFI).

Acceptable model fit indices (listed above) and fit criteria proposed by Kline (2005) were

followed to assess the fit of the current model and alternative models. A good fit can be

concluded if (a) the *p*-value of a model chi-square statistic is greater than .05 since this

statistic assesses the magnitude of unexplained variance, so a statistic that is not

statistically significant (i.e., $p > .05$) suggests good fit; (b) RMSEA is less than .08; (c)

CFI is greater than .90; (d) GFI is greater than .90; (e) AGFI is greater than .90; and (f)

NFI is greater than .90 (Kline, 2005). Bryne (2001) suggests reporting the 90%

confidence interval around the RMSEA value as well. In addition, Kline (2005)

advocates that a hypothesized model not be rejected if the lower bounds of a 90%

confidence interval for an RMSEA value is less than or equal to .05 and the upper bound

is less than or equal to .10. In addition, AMOS yields a *p* test for closeness of fit for

RMSEA values in which higher *p* values indicate better fit (Loehlin, 1998).

The modified hypothesized model (Figure 5), labeled Model 1a (*df* = 38), yielded

model fit indices that were excellent (see Table 8) and were comparatively stronger than

the null model of independence that assesses a zero correlation relationship among

variables in the model. Rejection of the null model of independence indicates that a more

elaborate model, such as Model 1a, is required to account for the data. Thus, whereas the

null independent model was rejected by all indices, Model 1a fit almost all of the indices.

The only statistic in which the model did not demonstrate adequate fit was the chi-square

statistic, although this statistic is sensitive to sample size (i.e., chi-square statistics are

likely to reject a model as sample size increases, even though differences between the

observed and predicted covariances are slight) and thus it is important to report other fit

indices that are less dependent on sample size (Kline, 2005). Given that chi-square was

the only fit index indicating a poor fit, this finding is likely a result of sample size. For

example, Model 1a meets Kline's (2005) guidelines about the range of RMSEA values

based on a 90% confidence interval and all other guidelines for a good model fit (see

Table 8).

The structural portion of Model 1a (Figure 6) indicates that all of the parcels load

strongly onto their respective latent variables. In other words, all SP parcels load

significantly onto the BIS latent variable and all SR parcels load significantly onto the

BAS latent variable. In structural equation modeling, these factor loading values are interpreted as regression coefficients (see Table 9 for these values as well). An examination of the individual pathways from a structural portion of the model in Figure 6, however, indicated some relationships that were not significant (i.e., the pathway between Hyperactive-Impulsive symptoms and the BIS and the pathway between Secondary Psychopathy symptoms and the BAS) and may improve the model fit if they are excluded.[8] Thus, the model was revised so that the pathway from Hyperactive-Impulsive AD/HD symptoms and the BIS latent variable was omitted, as well as the pathway from Secondary Psychopathy symptoms to the BAS latent variable. This revised model, labeled Model 1b ($df = 40$), demonstrated an excellent and improved fit (see Figure 7 and Table 8[9]). An alternative model in which the relationship between Hyperactive-Impulsive AD/HD symptoms and the BAS are mediated by Primary Psychopathy symptoms was assessed as well. Secondary Psychopathy symptoms were taken into account as well, however, their covariance with Hyperactive-Impulsive AD/HD symptoms was considered instead of considering its mediational role since it was not related to the BAS latent variable in the previous models. This model (Figure 8), labeled Model 2 ($df = 42$), did not demonstrate a better fit than Model 1b (see Table 8).

Overall, Models 1a and 1b provide a better fit to the data than the null independent model and Model 2. Since Models 1a and 1b are nested, a chi-square difference test can assess if the two models are statistically different. Referring to both

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[8] Additional details regarding the interpretation of pathway coefficients are discussed below.
[9] Due to rounding fit index values, Models 1a and 1b appear identical. However, Model 1b actually yields improved estimates than 1a without rounding off these values.

models as nested indicates that they are hierarchically arranged such that particular

parameters are freely estimated in one model, but fixed to zero in another model (such as

the pathway from Hyperactive-Impulsive symptoms to the BIS latent variable in Models

1a and 1b) (Bentler & Chou, 1987; Bollen, 1989). This statistic is calculated by

subtracting the chi-square statistic (χ^2) from one model against another model. The final

result is the chi-square difference statistic (χ^2_D). Thus, for Model 1a and Model 1b, χ^2_D

$(2) = 61.77 - 60.12 = 1.65$, $p = .44$.[10] This test indicates that both models do not

statistically differ. For the remaining analysis, Model 1b will be considered because it

was the most consistent with the original hypotheses, whereas Model 1a was more

exploratory. That is, the structural portion of Model 1a tested all pathways between

psychopathology symptom and RST variables, including those that were not predicted. In

addition, Model 1b is more parsimonious than Model 1a, which also supports considering

Model 1b over Model 1a. The final pathway values for each model, however, are listed in

Tables 9 (Model 1a) and 10 (Model 1b).

The next step in structural equation modeling involves assessing individual

pathways within the model that displays the best fit. Before assessing the structural

portion of the model, we will first review the findings at the measurement level of the

model (i.e., the SP and SR parcels and the BAS and BIS latent variables). Recall that in

structural equation modeling, factor loadings are interpreted as regression coefficients. In

Figure 7, these standardized regression coefficients indicate that the observed variables

for the BAS and BIS latent variables (i.e., the parcels) load strongly onto their respective

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[10] The 2 in this equation indicates differences in degrees of freedom for both models (i.e., 40 degrees of freedom for Model 1b and 38 degrees of freedom for Model 1a).

latent variables and are considered "large" standardized beta coefficients (Kline, 2005).

Figure 7 also indicates that these loadings are statistically significant (also see Table 10).

The disturbance and error terms (Table 10) are interpreted as standardized

estimates that are proportions of unexplained variance, which is a unique characteristic of

structural equation modeling in comparison to other techniques that ignore error, such as

multiple regression (Kline, 2005). These values are indicated, but not explicitly listed, in

Figure 7. That is, in Figure 7, the .31 over the BAS latent variable denotes that amount of

variance in the BAS latent variable that its predictors (i.e., Hyperactive-Impulsive

AD/HD and Primary Psychopathy symptoms) explain. The remaining variance (1 − .31 =

.69) indicates the proportion of unexplained variance accounted for by the disturbance

term (see Table 10). Additional disturbance and error terms are interpreted in the same

way. Thus, in Figure 7, the BAS latent variable as a predictor of Sensitivity to Reward

scale parcel 1 explains 46% of its variance. Thus, the error variance is 54% (1 − .46 =

.54). This latter percentage is listed in Table 10 as measurement error variance for parcel

1 of the Sensitivity to Reward scale. The disturbance and error term values associated

with this model are within the typical range for structural equation modeling.

As Figure 7 indicates, Hypotheses 5 and 6 were supported. That is, (a)

Hyperactive-Impulsive AD/HD symptoms were significantly associated with the BAS

latent variable while taking the influence of Primary and Secondary Psychopathy

symptoms into account and (b) Primary Psychopathy symptoms were significantly

associated with the BAS latent variable while taking the influence of Hyperactive-

Impulsive AD/HD and Secondary Psychopathy symptoms into account. Figure 7

indicates that the estimated standardized path coefficients (β's) for the direct effects of Hyperactive-Impulsive AD/HD symptoms and Primary Psychopathy symptoms on the BAS (while taking each other and Secondary Psychopathy symptoms into account) are 0.38 ($B = .07$, $SE = .01$, $p < .001$) and 0.33 ($B = .03$, $SE = .01$, $p < .001$), respectively. This means that a level of Hyperactive-Impulsive AD/HD symptoms one full standard deviation above the mean predicts BAS level almost 0.38 standard deviations above the mean. This would also apply to Primary Psychopathy symptoms and that one full standard deviation above the mean predicts BAS level almost 0.33 standard deviations above the mean. The magnitude of these standardized path coefficients is considered "typical" or "medium" (Kline, 2005, p. 122). In addition, the curved arrows linking the psychopathology variables indicates high correlations between symptoms that would be expected among disorders that are highly comorbid and are interpreted as correlation values (all p's $< .001$).

Hypothesis 7 that Secondary Psychopathy symptoms will be significantly associated with the BAS latent variable while taking the influence of Hyperactive-Impulsive AD/HD and Primary Psychopathy symptoms into account was not supported. Based on the correlational analysis that indicated these symptoms were positively associated with the SR scale, it appears that the relationship between the BAS latent variable and Secondary Psychopathy is an indirect result of their relationships with Primary Psychopathy and Hyperactive-Impulsive AD/HD symptoms. Instead, consistent with Hypothesis 8, Secondary Psychopathy symptoms were statistically significantly associated with high BIS functioning ($B = .07$, $SE = .02$, $\beta = .33$, $p < .001$). Conversely,

Primary Psychopathy symptoms were negatively associated with BIS functioning (B = -

.03, SE = .01, β = -.22, p = .007). Counter to Quay's (1988, 1997) hypothesis,

Hyperactive-Impulsive AD/HD symptoms were not associated with BIS functioning.[11]

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[11] Recently, Hundt, Kimbrel, Mitchell, and Nelson-Gray (in press) demonstrated that high BAS and low BIS scores predicted Hyperactive-Impulsive symptoms among a college sample when Inattentive AD/HD symptoms were partialled out in a regression analysis. The current study did not include the latter AD/HD symptoms due to a lack of theoretical rationale and since the current study was concerned with externalizing behaviors only. However, as an exploratory analysis, these symptoms were incorporated into the same structural equation model as Model 1a with Inattentive symptoms from the CAARS included as a predictor. Including these Inattentive symptoms did not change the relationship between Hyperactive-Impulsive symptoms and the BIS latent variable (B = -.03, SE = .02, β = -.12, p = .19) or any of the relationships between both sets of Psychopathy symptoms and RST variables. One likely explanation for the findings between Hyperactive-Impulsive symptoms and BIS scores in Hundt et al. is that comorbid Primary and Secondary Psychopathy symptoms were not taken into account as they were in the current study.

CHAPTER IV

DISCUSSION

The purpose of the current study was to assess the relationship between core symptoms of AD/HD (i.e., Hyperactive-Impulsive symptoms), Primary and Secondary forms of Psychopathy symptoms, and their relationship with RST. Structural equation modeling supported the hypothesis that Hyperactive-Impulsive AD/HD symptoms are uniquely associated with BAS functioning, while taking Primary and Secondary Psychopathy symptoms into account. This finding supports Nigg's (2006a) proposal that overactive approach behavior resulting from overactive BAS functioning is one pathway to AD/HD. The predicted positive relationship between Primary Psychopathy symptoms and the BAS was also supported while controlling for Hyperactive-Impulsive AD/HD symptoms and Secondary Psychopathy symptoms. Also, Primary Psychopathy symptoms were also inversely associated with underactive BIS functioning. Finally, the prediction that Secondary Psychopathy symptoms will share a significant positive relationship with the BAS was supported at the bivariate correlational level. However, when the high correlations with Hyperactive-Impulsive AD/HD symptoms and Primary Psychopathy symptoms were considered, this relationship, although positive, did not reach statistical significance. Instead, Secondary Psychopathy symptoms were significantly positively

associated with BIS functioning while taking Hyperactive-Impulsive AD/HD symptoms and Primary Psychopathy symptoms into account.

Overall, these findings indicate that, while considering the high correlation among Hyperactive-Impulsive AD/HD, Primary Psychopathy, and Secondary Psychopathy symptoms, distinct profiles based on BIS and BAS functioning emerge for each symptom set. That is, Hyperactive-Impulsive AD/HD symptoms are associated with overactive BAS functioning, Primary Psychopathy symptoms are associated with overactive BAS functioning and underactive BIS functioning, and Secondary Psychopathy symptoms are primarily associated with overactive BIS functioning. The current study also adds to current multiple pathway theories of AD/HD since it supports one proposed pathway (e.g., Nigg, 2006a; Sonuga-Barke, 2002, 2003; discussed in greater detail below) and is the first to consider highly comorbid Psychopathy symptoms to demonstrate how symptoms from both forms of psychopathology are related to RST in the same model. These findings address issues related to comorbidity and a potential underlying mechanism that accounts for the co-occurrence among these disorders within an RST-based theoretical framework. This study also overcame limitations associated with previous studies (e.g., this study included a large sample composed of males and females). Another notable strength of this study is the *a priori* model approach to testing and directly testing this model against alternative accounts. Implications for these findings, particularly for AD/HD and how these findings fit with AD/HD etiological theories, are discussed below. In addition, the current study failed to demonstrate a multi-method approach to assessing BAS functioning. This finding is also discussed below,

along with implications for future studies to generalize these findings to alternative methodologies beyond self-report.

Implications for AD/HD Theory, Development, and Biological Underpinnings

Theoretical and developmental factors. The finding that Hyperactive-Impulsive AD/HD symptoms are associated with BAS functioning and not the BIS has a number of implications for etiological theories of AD/HD. These findings fail to support Quay's (1988, 1997) underactive BIS hypothesis and other similar accounts (i.e., Beauchaine, 2001).[12] Instead, these findings support claims that overactive BAS functioning underlies these core AD/HD symptoms (Newman & Wallace, 1993; Patterson & Newman, 1993). Also, these findings support more recent proposals that AD/HD develops via separable and independent multiple pathways (i.e., etiological heterogeneity), one of which includes an overactive BAS motivational account (Nigg, 2006a; additional pathways are discussed below). That is, the current study confirmed this one pathway that "remains heuristic and in need of further study" (Nigg et al., 2004, p. 51). In addition, the current study addresses previous concerns that symptoms of other disorders which are comorbid with AD/HD and are associated with overactive BAS functioning (i.e., CD symptoms in childhood and Psychopathy symptoms in adulthood) mediate the relationship between AD/HD symptoms and overactive BAS functioning (Nigg et al., 2004). Based on the current findings, overactive BAS functioning may be an underlying predisposition for AD/HD and Psychopathy (mainly in its Primary form). This shared predisposition could

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[12] However, Beauchaine's (2001) account is different from Quay's (1988, 1997). Whereas Quay proposed an underactive BIS account, Beauchaine proposed that AD/HD results from an underactive BIS *and* an overactive BAS.

also account for the high degree of co-occurrence among symptoms of both disorders as well. Given such high rates of co-occurrence, a proposed mechanism to account for this link is crucial. In accordance with Mathias et al.'s (in press) proposal that impulsivity may be this underlying mechanism for the development of AD/HD and Psychopathy and their co-occurrence, the current findings support that impulsivity resulting from overactive behavioral approach tendencies (i.e., over responsiveness to immediately reinforcing stimuli in comparison to delayed reinforcement) as that mechanism.

The current findings supporting Nigg's (2006a) multiple pathways proposal are also noteworthy because of the potential utility of this account as it relates to temperamentally-based developmental accounts.[13] That is, Nigg (2006a) proposed three basic temperamental traits labeled approach (which is related to extraversion, positive affect, and BAS output), withdrawal (which is related to neuroticism, negative affect, and BIS output), and conscientiousness or constraint (which is related to effortful control and executive functioning). According to Nigg, the former two accounts are motivational, whereas the latter account is executive. These pathways converge with other temperamental accounts that attempt to link psychopathology and personality. For example, Clark (2005) labeled these dimensions as positive affect, negative affect, and disinhibition, respectively (see Mitchell, Kimbrel, Hundt, Cobb, Nelson-Gray, & Lootens, in press, for empirical support of these dimensions). In addition, these

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[13] Regarding the relationship between temperament and personality, temperamental traits are considered as changing over development and combine with experience to give rise to personality traits in adulthood (Rothbart & Bates, 1998). Given its biological emphasis on reactive brain-behavior systems, RST can be considered as overlapping with temperamental constructs (e.g., Nigg's (2006a) inclusion of RST variables within a temperamental view). Also see Blair (2003), Blair, Peters, and Granger (2004), and Farmer (2005) for additional examples of applying RST to a temperamental level.

temperamental traits converge with Rothbart's developmental theory of temperament as well (Rothbart, 2007; Rothbart, Ahadi, & Evans, 2000; Rothbart, Ahadi, Hershey, & Fisher, 2001; Rothbart & Bates, 1998).

This convergence with temperamentally-based developmental accounts is important because it identifies potential precursors to the development of AD/HD. According to Nigg's (2006a) account, AD/HD precursors are most likely to be high behavioral approach tendencies associated with overactive BAS functioning (a motivational process) and poor cognitive control (an executive process). Indeed, Goldsmith, Lemery, and Essex (2004) demonstrated in a prospective study of children from birth until the first grade that low effortful control and high approach tendencies are associated with Hyperactive-Impulsive AD/HD symptoms. This type of research informs heterogeneous etiological links to AD/HD, provides an empirical and theoretical basis for "telling a developmental story" of AD/HD, and is consistent with the current findings. In terms of the latter, this consistency with the current findings regarding overactive behavioral approach tendencies (a) provides empirical evidence that the current findings can be linked with temperamentally-based developmental accounts and (b) further supports an etiological account partially based on RST. Given that these temperamental traits are precursors to other disorders as well (e.g., Clark, 2005), addressing issues related to comorbidity is possible, which is relevant to the AD/HD literature given that the majority of AD/HD diagnoses are also accompanied by another disorder (see Barkley, 2006c, for a review). Since BIS and BAS output are included in two of these three dimensions and these two dimensions draw heavily from RST (e.g., see Rothbart et al.,

2001, for a review linking RST with their temperamental model), the utility of applying RST is also noteworthy.

The current findings, based largely on Nigg's (2001, 2006a) motivational and executive heuristic, can also be incorporated with other AD/HD theories as well. For example, Barkley's (1997a, 1997b) proposal that poor response inhibition leads to poor executive functioning, which Nigg (2006a) categorizes as an executive account, is a separate alternative pathway to AD/HD. Thus, rather than a competing theory, the overactive BAS account in the current study would be a more complimentary account of AD/HD.

Other recent AD/HD theories are more consistent with Nigg's (2006a) etiological heterogeneity account in that they propose multiple pathways to AD/HD. These accounts can also be incorporated with the current findings. For example, Sagvolden, Johansen, Aase, and Russell (2005) have proposed that altered functioning of dopaminergic branches lead to different AD/HD symptoms, particularly in the form of executive functions and abnormal reward responsiveness. These deficits associated with AD/HD resemble Nigg's (2001) executive and motivational accounts, respectively. Similarly, Sonuga-Barke (2002, 2003) proposed a dual pathway model that deficient executive and reward processes independently predict AD/HD. Regarding the latter process, Sonuga-Barke characterizes this as a "motivational style," similar to Nigg's (2006a) motivational process, in which there is an aversion to delayed reinforcement. This deficient reward process account is also similar to Sagvolden et al.'s (2005) account as well. For example, Sonuga-Barke (2002) acknowledged Sagvolden, Aase, Zeiner, and Berger's (1998)

evidence of abnormal reward responsiveness as evidence for his delay aversion account. According to Sonuga-Barke's account, as a result of this delay aversion, people diagnosed with AD/HD rate immediate rewards as more reinforcing than delayed rewards in comparison to controls. In terms of associating this account with overactive BAS functioning and the current findings, people who exhibit high BAS activity are associated with over responsiveness to immediately reinforcing stimuli and, as a result, appear averse to delayed reinforcement (e.g., Avila & Parcet, 2000).

The advantage of combining the reward dysfunction accounts from Sonuga-Barke (2002, 2003) and Sagvolden et al. (2005) within an RST framework is that it allows for a comprehensive theoretical framework that not only incorporates different theoretical accounts, but also accounts for comorbidity associated with AD/HD (Nigg et al., 2004). That is, RST can account for not just behavioral characteristics associated with AD/HD, but also how these disorders are comorbid (e.g., via shared predispositions associated with BIS or BAS). Thus, AD/HD accounts need to account not only for AD/HD, but also disorders that co-occur with AD/HD as well.

Although the current findings for Hyperactive-Impulsive AD/HD symptoms are interpreted within Nigg's (2001, 2006a) independent motivational and executive processes (see Sonuga-Burke, 2003, for another supporting argument for this separable processes position), how these two processes interact across development deserves attention in future research. For instance, although separate at later points in life (Toplak et al., 2005), these processes may be more intertwined in early development. For example, Rothbart et al. (2004) propose that temperamental regulatory "approach"

(analogous to a motivational account based on overactive BAS functioning) and "effortful control" tendencies (analogous to executive functioning) are interrelated as the former may constrain the latter in early development. Although not the focus of the current study, future research should clarify these processes and how they interact over development. This interaction could have implications for how AD/HD symptoms develop.

Biological factors. In terms of biological factors, the current findings have implications for AD/HD as well. For instance, Borrós-Loscertales et al. (2006) assessed the gray matter volume in areas associated with reward responsiveness and scores on the Sensitivity to Reward scale of the SPSRQ in a non-clinical male sample. A significant negative relationship between Sensitivity to Reward scores and gray matter volume in the dorsal striatum and the prefrontal cortex emerged, indicating that reduced gray matter volume in these areas (i.e., fronto-striatal circuits) is associated with greater reward sensitivity/BAS responsiveness. These findings are consistent with imaging studies that implicate the basal ganglia with AD/HD. For example, Seidman, Valera, and Makris (2005) report that 69% of AD/HD studies have shown significantly smaller volumes of the caudate nucleus. Similar findings are reported in affected monozygotic twins with AD/HD (Castellanos, Sharp, Gottesman, Greenstein, Giedd, & Rapoport, 2003). Seidman et al. (2005) also report that a smaller pallidum is implicated with AD/HD. In support of the proposed relationships with biological findings from the AD/HD literature, the findings from the current study demonstrated that the same scale is associated with

Hyperactive-Impulsive AD/HD symptoms while taking comorbidity concerns into account as well.

Molecular genetic AD/HD studies, although still an emerging field, have identified a number of catecholamine-regulating genes, which primarily includes dopamine receptor genes, as important to AD/HD. These emerging genetic markers in the AD/HD literature overlap considerably with genetics associated with the BAS. For example, the roles of dopamine transporter and dopamine D4 receptor genes in particular have received substantial support for their association with AD/HD. Other receptor genes, such as dopamine D2, D3, and D5 receptor genes, have received less attention, but are also promising potential candidates associated with AD/HD that have received some support (see Nigg, 2006a, pp. 201-207, for a review). In a detailed biological account of the BAS, Pickering and Gray (1999) identify the effectiveness of methylphenidate on the dopamine transporter gene as indirect evidence for the BAS. Other studies, such as Reuter et al. (2006) have reported that the dopamine D2 receptor gene is associated with the BAS. Reuter et al. also identify other genes, such as the dopamine D4 receptor gene, as worthy of future study but were not assessed in their study. Overall, addressing the overlap between AD/HD and RST biological factors is beneficial given the theoretical foundation of RST.

AD/HD Treatment Implications

Pharmacological treatment. The current findings also have implications for pharmacological and psychosocial treatments. At the pharmacological level, Pickering and Gray (1999) indicate that impulsive behavior resulting from overactive BAS

functioning can be improved by stimulant treatment, particularly methylphenidate. Consistent with this, stimulant treatment, particularly methylphenidate, is a first-line pharmacotherapy and is the most effective for children and adults with AD/HD (Connor, 2006; Prince, Wilens, Spencer, & Biederman, 2006). The finding that Hyperactive-Impulsive AD/HD symptoms are associated with BAS functioning provides a theoretical rationale for why this pharmacological treatment should be effective. Indeed, targeting biological substrates and neural pathways implicated with the BAS should enhance the value of delayed rewards through dopamine release (Nigg, 2006a). This is consistent with Taylor and Jentsch's (2001) finding that stimulant medication affects dopaminergic functioning associated with reward responsiveness.

Psychosocial treatment. Psychosocial treatment options are also implicated. That is, Farmer and Nelson-Gray (2005) review behavioral treatment strategies that are implicated with overactive BAS functioning. Based on the appetitive nature of BAS output (i.e., the function of the impulsive behavior is to attain immediate positive reinforcement), self-control strategies can increase the saliency of other reinforcement contingencies that are incompatible with the impulsive behavior and thereby decrease the frequency of impulsive behavior. In other words, self-control strategies could be administered to bring behavior under the influence of reinforcement contingencies that are incompatible with impulsive forms of behavior. Applying these types of techniques based on an RST framework may be beneficial. Overall, however, there is little empirical literature for well-controlled studies of psychosocial interventions for adults with AD/HD, although there are exceptions. For instance, one recent manualized cognitive-

behavioral treatment for adult AD/HD (Safren, Perlman, Sprich, & Otto, 2005) has demonstrated beneficial affects beyond pharmacotherapy treatment only (Safren, Otto, Sprich, Winett, Wilens, & Biederman, 2005).

Among the modules in this cognitive-behavioral treatment is a module on stimulus control techniques that are consistent with recommendations from Farmer and Nelson-Gray (2005). Stimulus control techniques refer to techniques that involve identifying and modifying a discriminative stimulus (i.e., antecedent conditions that influence behavior via their previous association with reinforcement contingencies) to change behavior (Farmer & Nelson-Gray, 2005). Thus, behavior is under stimulus control when its performance is contingent on the presence of certain stimuli. For example, if a client's goal were to decrease the amount of time spent on the computer, he or she could place a colored adhesive dot on his monitor that serves as a discriminative stimulus that cues a statement, such as "Am I doing what I'm supposed to be doing" or "Am I wasting time?" In this example, the past behavior may have been reinforcing in the short-term (i.e., time spent on the computer), but the dot serves to influence this previous association by also prompting the person to consider other contingencies, such as long-term ones that are negative (e.g., missing appointments or being late). Similarly, this cognitive-behavioral treatment also includes other self-control strategies, such as self-evaluation of behavior in relation to short-term and long-term goals. Implicit within this technique is consideration of temporal properties of reinforcing stimuli. Consistent with an RST explanation, someone who exhibits impulsive behavior associated with overactive BAS functioning is more responsive to immediate reinforcers than delayed reinforcers. Based

on the current findings, a component analysis of this cognitive-behavioral treatment should yield the strongest effects on improving Hyperactive-Impulsive AD/HD symptoms following administration of the stimulus control techniques module (and any other portions of the treatment involve forms of self-control training) above the other modules.

Primary and Secondary Psychopathy

The current findings also have implications for Psychopathy and the Fowles-Gray-Lykken hypothesis (Fowles, 2001; Gray, 1991; Lykken, 1995). This hypothesis posits that Primary Psychopathy is associated with low BIS functioning and normal BAS functioning, whereas Secondary Psychopathy is associated with normal BIS functioning and high BAS functioning. Previous studies have reported that Primary Psychopathy is associated with low BIS, whereas Secondary Psychopathy is associated with high BAS (Newman et al., 2005). Others have reported that low BIS is associated with Primary Psychopathy, whereas high BAS is associated with both Primary and Secondary Psychopathy (Kimbrel et al., 2007). The current findings, however, support that high BAS and low BIS are associated with Primary Psychopathy symptoms, whereas high BIS is associated with Secondary Psychopathy symptoms. Sample composition may partially account for discrepant findings from the current study and from Newman et al. (2005). That is, in the latter study, the Primary Psychopathy and control groups were both composed of male inmates. This comparison may result in higher BAS scores among both groups in comparison to a normal, non-incarcerated control group since some forms of criminal activity can be characterized as impulsive, reward-seeking behavior. As a

result of artificially higher BAS scores among inmates in general, non-significant group differences in BAS scores may have emerged.

In addition, the high correlation between Primary and Secondary Psychopathy symptoms in the current study may account for findings that associate Secondary Psychopathy symptoms and the BAS in other studies (Hundt, Kimbrel, Mitchell, & Nelson-Gray, in press; Kimbrel et al., 2007; Newman et al., 2005). That is, findings from previous studies indicating a significant relationship between Secondary Psychopathy and the BAS may be spurious. This relationship may emerge from the high correlation both variables share with Primary Psychopathy symptoms. In support of this explanation, although a correlational analysis indicated that Secondary Psychopathy symptoms and BAS scores were significantly correlated (Table 6), this relationship is not statistically significant when Primary Psychopathy symptoms are considered (Figures 6 and 7). Given that (a) the main distinction between Primary Psychopathy and Secondary Psychopathy is the presence of internalizing features (i.e., anxiety and depression) in the latter (Blackburn, 1975) and (b) the current findings indicate that Secondary Psychopathy symptoms are higher on BIS functioning and Primary Psychopathy symptoms are lower on BIS functioning, the current findings are consistent with this main distinction between both forms of Psychopathy.

The relationship between Primary Psychopathy symptoms and RST is consistent with Corr's (2002b) joint subsystem hypothesis, which suggests that the simultaneous activity of both BIS and BAS may provide a more accurate account of behavior than examination of BIS or BAS alone. In general, this study was the first to assess the

interdependent relationship between AD/HD and Psychopathy symptom variables and how they independently relate to RST variables.

The relationship between the BIS and Secondary Psychopathy symptom replicates recent finings that support a positive association between these variables (Hundt et al., in press). However, one issue that is not entirely clear from the current study is the role of impulsivity in Secondary Psychopathy symptoms. That is, Secondary Psychopathy symptoms share impulsive behavioral components with Primary Psychopathy symptoms (Blackburn, 1975), but the current findings fail to confirm a relationship between Secondary Psychopathy symptoms and the BAS. Although the current model did not support that Secondary Psychopathy symptoms are uniquely associated with high BAS functioning, these symptoms were significantly positively correlated with externalizing symptoms that were correlated with the BAS latent variable (i.e., Hyperactive-Impulsive AD/HD symptoms and Primary Psychopathy symptoms). Thus, based on the current findings, it is likely that impulsivity associated with Secondary Psychopathy is the result of externalizing symptom comorbidity with AD/HD and Primary Psychopathy either at the clinical or subclinical levels. In addition to this comorbidity hypothesis, "anxious impulsivity" may also account for impulsive behaviors associated with Secondary Psychopathy symptoms. According to Wallace, Newman, and Bachorowski (1991), increased sensitivity to punishment cues could lead to behavior that appears impulsive. In situations in which an anxious state is established and requires a response, people high on Secondary Psychopathy symptoms may be more likely to behave in an unplanned, spontaneous way that appears impulsive. In contrast to impulsivity associated with the

BAS characterized by responsiveness to immediate reinforcing stimuli, impulsivity

associated with the BIS differs in function since it is more concerned with responsiveness

to punishing stimuli instead. Future research should test these hypotheses to establish

whether either is empirically supported.

Limitations and Future Directions

The findings from the current study are novel, although additional work is needed

to replicate and generalize these results. These current findings should be interpreted in

light of several limitations. First, the current study was cross-sectional and therefore

limits causal inferences. In structural equation modeling, inferring causality requires

more than a correspondence between a model and data. Instead, the following conditions

must be met: (a) evidence of the causal variable preceding the outcome variable, (b) the

direction of the causal relationship is correctly specified (e.g., X causes Y, instead of Y

causes X), and (c) the relationship between the causal and outcome variable is maintained

when external variables are considered (Kline, 2005). Thus, prospective studies could

address these constraints to demonstrate causality. However, given the cost of such

studies, cross-sectional studies such as the current study provide a basis for pursuing a

more intricate longitudinal design. Also, regarding the second point above, the direction

of the relationship between both variables needs to be addressed in causal modeling.

Although the model in the current study illustrates that Hyperactive-Impulsive AD/HD

symptoms lead to the BAS latent variable, the opposite direction would actually be

predicted in a prospective study (i.e., BAS $\rightarrow$ Hyperactive-Impulsive AD/HD

symptoms). This directional portion of the model in the current study (i.e., the direction

between psychopathology symptom variables and personality variables in general) is for

practical purposes only since it allowed for controlling for the correlation between

AD/HD and Psychopathy symptoms. Instead, the personality variables in the current

study should predict psychopathology symptom variables. Therefore, in a prospective

study, overactive BAS functioning should be observed prior to Hyperactive-Impulsive

AD/HD symptoms. Indeed, emerging findings provide support for this relationship

(Goldsmith et al., 2004). This association between the BAS and temperamental constructs

(e.g., Rothbart et al., 2000) also demonstrates the utility of an RST approach to address

this issue related to AD/HD since RST can be applied in adulthood and childhood. (Also

see Blair, 2003, Blair et al., 2004, Coplan, Wilson, Frohlick, & Zelenski, 2006, and

Farmer, 2005, as examples of studying RST as a temperamental model with children.)

Second, the sample was composed primarily of female college students. Thus, this

limitation concerns both gender of the sample and the type of sample (i.e.,

undergraduate). Regarding gender, the current sample was predominantly female, while

the gender proportion in self-referred clinical samples is more balanced among adults

(Biederman, Faraone, Monuteaux, Bober, & Cadogen, 2004). Evidence from the current

study, however, indicates support for the findings across gender. That is, Table 7

demonstrates that the correlational findings between genders were consistent overall.

Regardless, given that structural equation modeling is a large sample technique, future

studies that include larger samples of both males and females should be conducted to

address any gender-specific effects. Regarding the non-clinical sample, this type of

approach reduces confounds associated with current psychopathology (e.g., medication

side effects). However, it also limits the generalizability of the results. Therefore, the direct clinical relevance of these findings are limited. Future studies should consider including clinical participants along with RST-specific measures to address this limitation.

Third, although attempts at multi-method assessment were made, the current study relied on self-report measures. This methodology leaves open the possibility of error due to shared method variance. However, the current findings were generally consistent with non-RST behavior task findings (see Tables 2 and 3). Despite this consistency, future studies should address this limitation. Also relevant to the current study is the limited variance associated with CARROT scores (see Figure 3) and the consistent null or restricted findings associated with this behavioral measure (see Table 6). Given that the CARROT appears to be a promising measure of reward responsiveness (i.e., BAS functioning), future studies should address ways to improve this measure.

One possible explanation for the muted CARROT findings in the current study is a ceiling effect in sorting rate. Specifically, the reinforcement trial in the CARROT is during the third sorting trial. Prior to this trial, participants may have already reached their capacity for sorting quickly for a number of reasons (e.g., covert cues such as gaining social reinforcement from the experimenter). Pickering and Gray (1999) recommend that an alternative administration approach that may overcome this limitation is introducing the reinforcement trial earlier, such as trial two. However, this suggestion has limitations itself. For example, an increase in sorting during the second trial may emerge because of practice effects. These practice effects could emerge simply because

of changes in sorting strategies, which the CARROT does not control for. For instance, the directions for the CARROT do not specify if participants should use his or her dominant hand, or if he or she should use one hand or two hands while sorting. This lack of control could allow for more error to enter into CARROT scores. Although research assistants in the current study were all trained by the principal investigator who obtained explicit CARROT administration directions from its author (see Appendix J), this method of task administration may have introduced error to CARROT scores. This limitation may be overcome by creating a computerized version of the CARROT to improve the standardization of CARROT administration. Other issues, such as reinforcement magnitude and frequency should be considered as well. For instance, perhaps a larger amount of money or a different type of reward may be more likely to elicit BAS output. Future studies should address these limitations of the CARROT to improve multi-method assessment of RST variables.

A fourth issue that deserves mention and is a limitation in terms of methodology is the complicated nature of self-report measurement of RST variables (see Smillie, Pickering, & Jackson, 2006, for a review of RST updates and issues related to self-report measurement) and how updates to RST by Gray and McNaughton (2000) have only exacerbated these concerns (Corr, 2004). Smillie et al. (2006) recently reviewed RST updates by Gray and McNaughton (2000) and how these updates affect RST personality measurement. RST updates have two major implications for future research and are summarized below. Although beyond of the scope of the current study, these implications are reviewed given their potential impact on AD/HD theory in the future.

First is the differentiation between the BIS (a conflict detection and resolution system associated with anxiety) and the FFFS (a system associated with responsiveness to punishing stimuli and fear) in personality measurement. Evolutionarily, BIS and FFFS outputs are similar and highly correlated (e.g., Corr, 2004; Corr & Perkins, 2006); however, animal ethoexperimental paradigms are able to differentiate these two systems. In RST research in humans, this distinction is yet to be established and is currently an area under development (see Perkins & Corr, 2006; Perkins, Kemp, & Corr, 2007, for measurement issues applicable to human participants). Thus, as mentioned above, this is beyond the scope of the current study. However, in terms of implications for AD/HD research once this issue related to measurement is caught up with theory, differentiating the FFFS from the BIS will require updates to AD/HD theories. That is, Quay's underactive BIS theory was based on Gray's (1982) "old RST," which asserted that BIS functioning was associated with conditioned punishing stimuli. However, updates to RST now associate the FFFS with punishing stimuli (both conditioned and unconditioned). Thus, studies that can differentiate these two systems in accordance with the "new RST" have implications for AD/HD theory. For example, it could be that difficulty detecting approach avoidance conflicts associated with the BIS (but not punishment sensitivity now associated with the FFFS) is associated with AD/HD in addition to overactive BAS functioning. The current study considered BIS and FFFS output as combined based on Corr's (2004) argument and we conclude, based on the current data, that Hyperactive-Impulsive AD/HD symptoms are not associated with combined BIS and FFFS output.

However, the empirical differentiation of the output of both systems may require future revision of the relationship between RST variables and AD/HD.

A second implication for updates to RST reviewed by Smillie et al. (2006) is the relationship between the BAS (or reward reactivity) and trait impulsivity. Although a "controversial" topic (Smillie et al., 2006, p. 328), Smillie et al. propose that BAS functioning is more aligned with Extraversion, but not with trait impulsivity as proposed by Gray (e.g., see Gray, 1991). Instead, they hypothesize that trait impulsivity is more strongly associated with Psychoticism. Accordingly, BAS output does not necessarily result in impulsive behavior. Although this differentiation between trait impulsivity (associated with Psychoticism) and reward responsiveness (associated with the BAS and Extraversion) is important for future research (see Ashton, Lee, & Paunonen, 2002; Franken & Muris, 2006 for opposing evidence; or Smillie, Jackson, & Dalgleish, 2006, for alternative theories, such as that trait impulsivity and reward responsiveness share a biological substrate that is the BAS), the interpretation of the current findings is that BAS functioning is associated with impulsive behavior, and that BAS functioning is associated with Hyperactive-Impulsive AD/HD symptoms. Indeed, the measurement of BAS functioning in the current study has been associated with impulsive behavior in past behavioral studies (e.g., Avila & Parcet, 2000, 2001). As another example of how the Sensitivity to Reward scale of the SPSRQ as an index of BAS output is associated with trait impulsivity, Mitchell et al. (in press) reported that the Sensitivity to Reward scale loads onto an impulsivity factor commonly found in hierarchical analyses of the five factor model of personality (e.g., DeYoung, 2006; Digman, 1997). This impulsivity

factor from the five factor model is similar to trait impulsivity as discussed by Smillie et al. (2006).

Despite the complicated nature of RST-based self-report measures, the current findings (a) are largely consistent with previous analyses of RST (or non-RST measures that have implications for RST measurement) and AD/HD, and (b) are generally consistent with the hypotheses in the current study. Regarding the former comment, however, the current findings are the first to demonstrate that Hyperactive-Impulsive AD/HD symptoms are associated with BAS functioning with RST-specific measures and within a theoretical framework that includes not just AD/HD, but other disorders as well that may mediate this relationship and make the relationship between AD/HD and the BAS spurious. Relatedly, although BIS and BAS are brain-behavior systems that regulate responsiveness to reinforcing stimuli and the scales from the SPSRQ assess inter-individual variation in the functioning of the BIS and BAS (i.e., Sensitivity to Punishment and Sensitivity to Reward scales of the SPSRQ are scales, not the actual brain-behavior systems they are proposed to underlie), recent studies that support an association between scales from the SPSRQ and biological substrates hypothesized to be related to them are encouraging and provide a basis for linking the current findings to RST brain-behavior systems (e.g., Borrós-Loscertales et al., 2006). Overall, to address any concerns related to self-report measurement, studies that incorporate alternative methods of measurement, as discussed above, should be incorporated.

In terms of future directions, the current study focused on a similar predisposing variable to psychopathology (i.e., BAS and its association with AD/HD and Psychopathy

in general) and supported claims by others that these disorders share a common predisposition via the BAS (Fowles, 2001; Newman & Wallace, 1993; Nigg et al., 2004; Patterson & Newman, 1993). However, how these distinct disorders differentiate above and beyond personality variables would address additional issues not addressed in the current study. That is, although Hyperactive-Impulsive AD/HD, Primary Psychopathy, and Secondary Psychopathy symptoms all exhibited differential profiles with RST variables, other differentiating factors account for etiology as well (e.g., socioeconomic status, ineffective parenting, or parental modeling of violence). Also, additional motivational and executive pathways (Nigg, 2006a) may differentiate these disorders. For example, whereas poor response modulation associated with overactive BAS functioning and underactive BIS functioning may be associated with Primary Psychopathy, overactive BAS functioning and poor executive functioning may be associated with AD/HD. Now that the current study has demonstrated that a central tenet to overactive BAS and response modulation accounts (i.e., overactive BAS functioning) is associated with both AD/HD and Psychopathy symptoms, future studies can address additional aspects of how these processes are associated with different forms of psychopathology. For example, a study could address whether there are differences between Hyperactive-Impulsive AD/HD and Primary Psychopathy symptoms on response modulation tasks or executive functioning tasks.

Conclusion

The current study assessed the relationship between Hyperactive-Impulsive AD/HD symptoms and the BAS, while taking symptoms of comorbid disorders also

associated with overactive BAS functioning (i.e., Primary and Secondary forms of Psychopathy symptoms) into account. Structural equation modeling allowed for this overlap to be considered. Overall, these findings indicate that, while considering the high correlation among Hyperactive-Impulsive AD/HD, Primary Psychopathy, and Secondary Psychopathy symptoms, distinct profiles based on BIS and BAS functioning emerge for each symptom set. That is, Hyperactive-Impulsive AD/HD symptoms are associated with overactive BAS functioning, Primary Psychopathy symptoms are associated with overactive BAS functioning and underactive BIS functioning, and Secondary Psychopathy symptoms are primarily associated with overactive BIS functioning.

The current study also adds to current multiple pathway theories of AD/HD since it supports one proposed pathway (e.g., Nigg, 2006a) and is the first to consider highly comorbid Psychopathy symptoms to demonstrate how symptoms from both forms of psychopathology are related to RST in the same model. These analyses indicated that Hyperactive-Impulsive AD/HD and Primary Psychopathy symptoms are uniquely associated with overactive BAS functioning, which supports previous claims that these disorders share a similar predisposition (e.g., Newman & Wallace, 1993; Patterson & Newman, 1993). The model that illustrated this relationship exhibited an improved fit with the data in comparison to alternative models, such as Psychopathy symptoms mediating the relationship between Hyperactive-Impulsive AD/HD symptoms and the BAS. This finding with Hyperactive-Impulsive AD/HD symptoms also provides empirical evidence for an etiological pathway (i.e., high BAS functioning) proposed to be of heuristic value to AD/HD in need of future study (Nigg et al., 2004).

Although Hyperactive-Impulsive AD/HD symptoms were not associated with BIS functioning, which Quay (1988, 1997) proposed as a central deficit that yields Hyperactive-Impulsive AD/HD symptoms, differential findings based on BIS functioning were supported for Primary and Secondary Psychopathy symptoms. That is, Primary Psychopathy symptoms were associated with low BIS output, whereas Secondary Psychopathy symptoms were associated with high BIS output.

The current study addresses issues related to multi-method assessment of RST variables, overcomes limitations associated with previous shortcomings in the AD/HD literature (e.g., gender distribution), concurrently demonstrates not just how AD/HD is associated with RST but also how Psychopathy symptoms are associated with RST, and reviews these findings in light of other AD/HD theories in an effort to make interpretation of these differing accounts parsimonious.

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Appendix A

Tables

Table 1

Summary of DSM-IV-TR Inattentive and Hyperactive-Impulsive AD/HD Symptoms (APA, 2000)

Inattentive symptoms

    a) Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities

    b) Often has difficulty sustaining attention in tasks or play activities

    c) Often does not seem to listen when spoken to directly

    d) Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)

    e) Often has difficulty organizing tasks and activities

    f) Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)

    g) Often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)

    h) Is often easily distracted by extraneous stimuli

    i) Is often forgetful in daily activities

Hyperactive-Impulsive symptoms

    a) Often fidgets with hands or feet or squirms in seat

    b) Often leaves seat in classroom or in situations in which remaining seated is expected

c) Often runs about or climbs excessively in situations in which it is inappropriate

d) Often has difficulty playing or engaging in leisure activities quietly

e) Is often "on the go" or often acts as is "driven by a motor"

f) Often talks excessively

g) Often blurts out answers before questions have been completed

h) Often has difficulty awaiting turn

i) Often interrupts or intrudes on others (e.g., butts into conversations or games)

Table 2

Summary of Motivated Go/No-Go Behavior Task Studies

Study	Overall Sample Age	Condition	Limitations	AD/HD Subtype	Findings
Milich, Hartung, Martin, & Haigler (1994)	16.30 ($SD = 2.10$)	Reward-punish, reward-only	Medication effects Comorbid anxiety Reinforcement allocation (not addressed) Reinforcement expectancy	n/a[1]	Response Modulation Deficit for males Overactive BAS for females
Iaboni, Douglas, & Baker (1995)	10.68 ($SD = 1.45$)	Reward-punish, punish-only, reward-only	Comorbidity Small N Male-only sample Reinforcement expectancy	n/a[1]	Generalized Deficit
Hartung, Milich, Lynam, & Martin (2002)	15.21 ($SD = 1.37$)	Reward-punish, punish-only	Comorbid anxiety Reinforcement allocation (not addressed) Reinforcement expectancy	Not Specified[2]	Response Modulation Deficit for both males and females No support for low BIS
Gomez (2003)	10.66 ($SD = 1.14$)	Reward-punish, punish-only, reward-only	Male-only sample Reinforcement allocation (not addressed) Reinforcement expectancy	C	Response Modulation Deficit Generalized Deficit
Farmer & Rucklidge (2006)	14.98 ($SD = 1.51$)	Reward-punish	Small N Reinforcement allocation (not addressed) Reinforcement expectancy	C and IA	Response Modulation Deficit

Note. In the AD/HD Subtype column, 1 = diagnosis based on *DSM-III-R* criteria, 2 = diagnosis based on *DSM-IV* criteria, n/a = subtype is not applicable based on *DSM* diagnostic criteria, C = Combined, IA = Inattentive; Small *n* is defined as < 20 participants in at least one group.

Table 3

Summary of Card Playing Behavior Task Studies

Study	Overall Sample Age	Limitations	AD/HD Subtype	Findings
Daugherty & Quay (1991)*, [A]	11.0 ($SD = 0.92$)	Comorbidity with CD Medication effects Small N Reinforcement allocation (not addressed) Reinforcement expectancy	ADDH/CD[1]	Response Modulation Deficit
Daugherty, Quay, & Ramos (1993)*, [A]	11.20 ($SD = 1.10$)	Comorbidity Medication effects Small N Reinforcement allocation (not addressed) Reinforcement expectancy	ADDH/CD[1]	Null results
Matthys, van Goozen, de Vries, Cohen-Kettenos, & van Engeland (1998)*	10.0 ($SD = 1.10$)	Comorbidity Small N Male-only sample Reinforcement allocation (not addressed) Reinforcement expectancy	Not Specified[3]	Response Modulation Deficit
Milich, Hartung, Martin, & Haigler (1994)	16.30 ($SD = 2.10$)	Medication effects Comorbid anxiety Reinforcement allocation (not addressed) Reinforcement expectancy	n/a[2]	Response Modulation Deficit

Fischer, Barkley, Smallish, & Fletcher (2005)	20.90 (*SD* = 1.0)	Comorbid anxiety in CD group Reinforcement allocation Reinforcement expectancy Primarily male sample	Not Specified[3]	Poor response modulation among adults with a lifetime CD diagnosis, but not for AD/HD without a lifetime CD diagnosis

Note. In the AD/HD Subtype column, 1 = diagnosis based on *DSM-III* criteria, 2 = diagnosis based on *DSM-III-R* criteria, 3 = diagnosis based on *DSM-IV* criteria, n/a = subtype is not applicable based on diagnostic criteria; ADDH/CD indicates Attention Deficit Disorder with Hyperactivity and comorbid Conduct Disorder; Small *n* is defined as < 20 participants in at least one group; * = modified card playing task administered (door opening task); [A] = This study also included an Attention Deficit Disorder without Hyperactivity group, but they would not be expected to be associated with high BAS functioning and are therefore not included in the table.

Table 4

Summary of Sample Demographics

Variable	
Mean Age	19.30 (3.51)
Gender	
Male	89
Female	143
Ethnicity	
Caucasian	145
African American	57
Asian	7
Hispanic	4
Native American	2
Other	12
Missing	5
Mean Grade Point Average	3.14 (0.65)

Note. Standard deviation listed in parentheses.

Table 5

Cronbach Coefficient Alpha Values

Scale	α
Sensitivity to Punishment	.82
Sensitivity to Reward	.78
Hyperactivity-Impulsivity	.73
Primary Psychopathy	.86
Secondary Psychopathy	.74

Table 6

Means, Standard Deviations, and Pearson Product-Moment Correlations

	Mean	SD	1	2	3	4	5	6
1. Sensitivity to Punishment	11.26	5.16	---	.11	.02	.12	-.06	.20**
2. Sensitivity to Reward	12.70	4.31		---	.12	.40**	.37**	.35**
3. CARROT total	1.38	4.10			---	.09	.16*	.11
4. Hyperactivity-Impulsivity	8.39	4.33				---	.25**	.48**
5. Primary Psychopathy	30.67	7.80					---	.49**
6. Secondary Psychopathy	21.02	4.59						---

Note. SD = Standard Deviation, ** Correlation is significant at the 0.01 level (2-tailed), * Correlation is significant at the 0.05 level (2-tailed).

Table 7

Pearson Product-Moment Correlations by Gender

	1	2	3	4	5	6
Males (*n* = 89)						
1. Sensitivity to Punishment	---	.21*	.12	.15	-.04	.19
2. Sensitivity to Reward		---	.15	.44**	.29**	.38**
3. CARROT total			---	.14	.21*	.16
4. Hyperactivity-Impulsivity				---	.26**	.42**
5. Primary Psychopathy					---	.43**
6. Secondary Psychopathy						---
Females (*n* = 143)						
1. Sensitivity to Punishment	---	.09	-.02	.11	-.04	.21**
2. Sensitivity to Reward		---	.09	.39**	.40**	.34**
3. CARROT total			---	.06	.11	.08
4. Hyperactivity-Impulsivity				---	.24**	.51**
5. Primary Psychopathy					---	.52**
6. Secondary Psychopathy						---

Note. ** Correlation is significant at the 0.01 level (2-tailed), * Correlation is significant at the 0.05 level (2-tailed).

Table 8

Model Fit Index Summary

	Chi-Square statistic ($p > .05$)	GFI (>.90)	AGFI (>.90)	NFI (>.90)	CFI (>.90)	RMSEA (<.08)
Model 1a	60.12 ($p = .01$)	.96	.93	.92	.97	.05 (.02 - .07)
Null Independent Model	794.82 ($p < .001$)	.53	.44	.00	.00	.24 (.23 - .26)
Model 1b	61.77 ($p = .02$)	.96	.93	.92	.97	.05 (.02 - .07)
Model 2	140.32 ($p < .001$)	.90	.85	.82	.87	.10 (.08 - .12)

Note. Reference for a good fit for fix indices are listed in parentheses next to their respective index (based on Kline, 2005); 90% confidence intervals are listed in parentheses in the RMSEA column; RMSEA closeness of fit p values = 0.47 for Model 1a, $p < .001$ for the Null Independent Model, $p = 0.52$ for Model 1b, and $p < .001$ for Model 2, which suggests Models 1a and 1b have the most optimal fit.

Table 9

Maximum Likelihood Standardized Parameter Estimates for Model 1a

Parameter	Estimate	SE	Parameter	Estimate	SE
Factor loadings			Measurement error variances		
BAS→SRp1	.68	---	E_SRp1	.54	.10
BAS→SRp2	.64	.16	E_SRp2	.59	.17
BAS→SRp3	.67	.12	E_SRp3	.55	.09
BAS→SRp4	.73	.16	E_SRp4	.46	.15
BIS→SPp1	.66	---	E_SPp1	.57	.15
BIS→SPp2	.78	.13	E_SPp2	.39	.14
BIS→SPp3	.69	.13	E_SPp3	.52	.14
BIS→SPp4	.76	.15	E_SPp4	.43	.19
Direct effects			Disturbance variances		
HI→BAS	.34	.02	BIS	.91	.18
HI→BIS	.03*	.02	BAS	.69	.09
Prim→BAS	.29	.01			
Prim→BIS	-.23	.01			
Sec→BAS	.10*	.02			
Sec→BIS	.31	.02			

Correlations

Prim – Sec	.49	2.62
Sec – HI	.48	1.44
Prim – HI	.25	2.28

Note. Estimates for measurement errors and disturbances are proportions of unexplained variance; $p < .001$ for all estimates except Prim→BIS pathway ($p = .006$) and * (indicates $p > .05$); Standard errors are not available for paths used for scaling of latent variables; Standard error values are calculated for unstandardized solutions; SE = Standard Error; BAS = Behavioral Approach System; BIS = Behavioral Inhibition System; SR = Sensitivity to Reward scale; SP = Sensitivity to Punishment scale; p1 = parcel 1; p2 = parcel 2; p3 = parcel 3; p4 = parcel 4; HI = Hyperactive-Impulsive AD/HD symptoms; Prim = Primary Psychopathy symptoms; Sec = Secondary Psychopathy symptoms; E = Error.

Table 10

Maximum Likelihood Standardized Parameter Estimates for Model 1b

Parameter	Estimate	SE	Parameter	Estimate	SE
Factor loadings			Measurement error variances		
BAS→SRp1	.67	---	E_SRp1	.54	.10
BAS→SRp2	.64	.16	E_SRp2	.59	.17
BAS→SRp3	.67	.12	E_SRp3	.56	.10
BAS→SRp4	.73	.16	E_SRp4	.46	.15
BIS→SPp1	.66	---	E_SPp1	.57	.15
BIS→SPp2	.78	.13	E_SPp2	.39	.14
BIS→SPp3	.69	.13	E_SPp3	.52	.14
BIS→SPp4	.76	.15	E_SPp4	.43	.19
Direct effects			Disturbance variances		
HI→BAS	.38	.01	BIS	.91	.18
Prim→BAS	.33	.01	BAS	.69	.09
Prim→BIS	-.22	.01			
Sec→BIS	.33	.02			
Correlations					
Prim – Sec	.49	2.62			
Sec – HI	.48	1.44			

Prim – HI	.25	2.28

Note. Estimates for measurement errors and disturbances are proportions of unexplained variance; *p* < .001 for all estimates except Prim→BIS pathway (*p* = .007); Standard errors are not available for paths used for scaling of latent variables; Standard error values are calculated for unstandardized solutions; SE = Standard Error; BAS = Behavioral Approach System; BIS = Behavioral Inhibition System; SR = Sensitivity to Reward scale; SP = Sensitivity to Punishment scale; p1 = parcel 1; p2 = parcel 2; p3 = parcel 3; p4 = parcel 4; HI = Hyperactive-Impulsive AD/HD symptoms; Prim = Primary Psychopathy symptoms; Sec = Secondary Psychopathy symptoms; E = Error.

Appendix B

Figures

Figure 1. An interacting network model of the BIS (Behavioral Inhibition System), BAS (Behavioral Approach System), and FFFS (Fight Flight Freeze System; adapted from Pickering, 2004b). S_R and S_P are system inputs (i.e., reinforcing or punishing stimulus strength, both conditional and unconditional), whereas W_R, W_P, and W_C are system sensitivities. Thus, excitatory activation of the BAS, for example, is driven by both current reward stimulus strength (S_R) and BAS sensitivity (W_R) that reflect temperament and learning history influences. The AND indicates interaction of the FFFS and BAS (both need to be engaged) that activate the BIS. In this model, the output on the left is a combination of FFFS and BIS output based on Corr and Perkins (2006) argument that the BIS favors FFFS output (also see Corr, 2004), which is based on Gray and McNaughton's (2000) revisions to Reinforcement Sensitivity Theory.
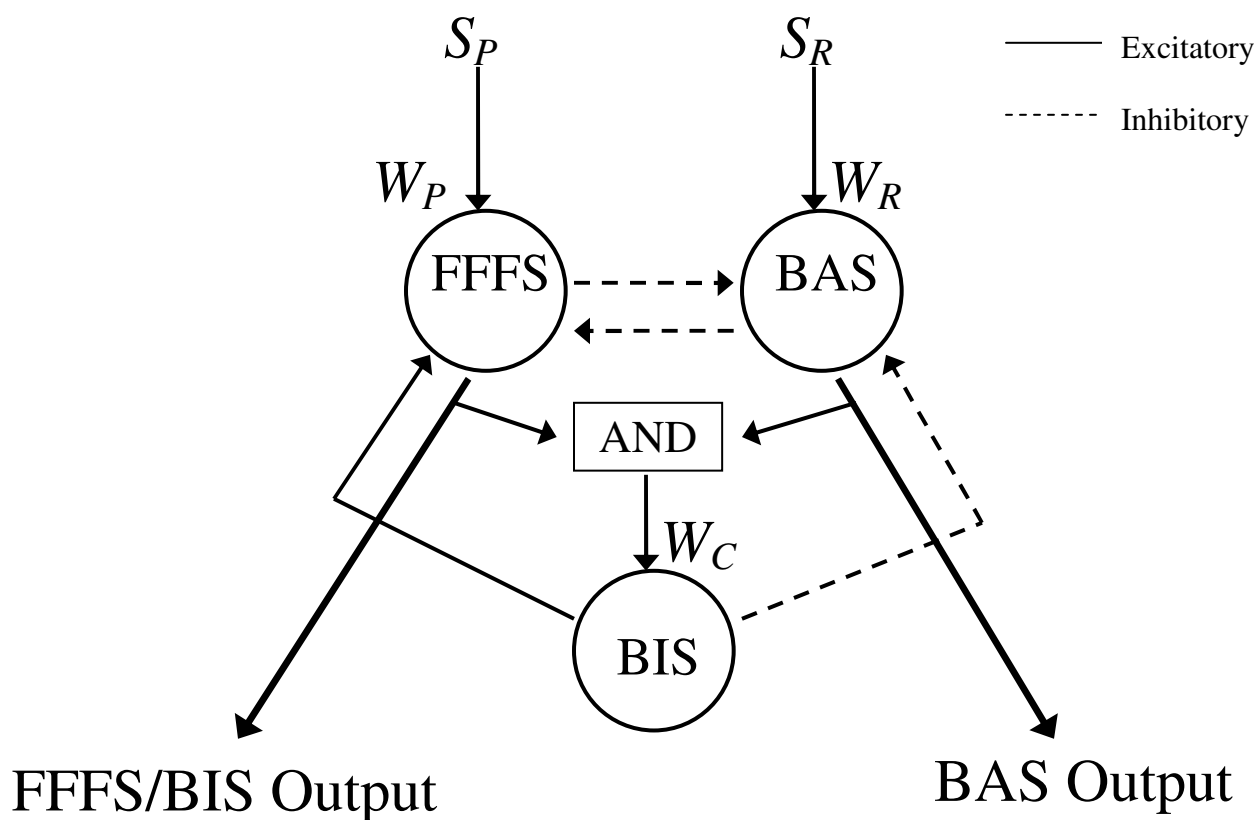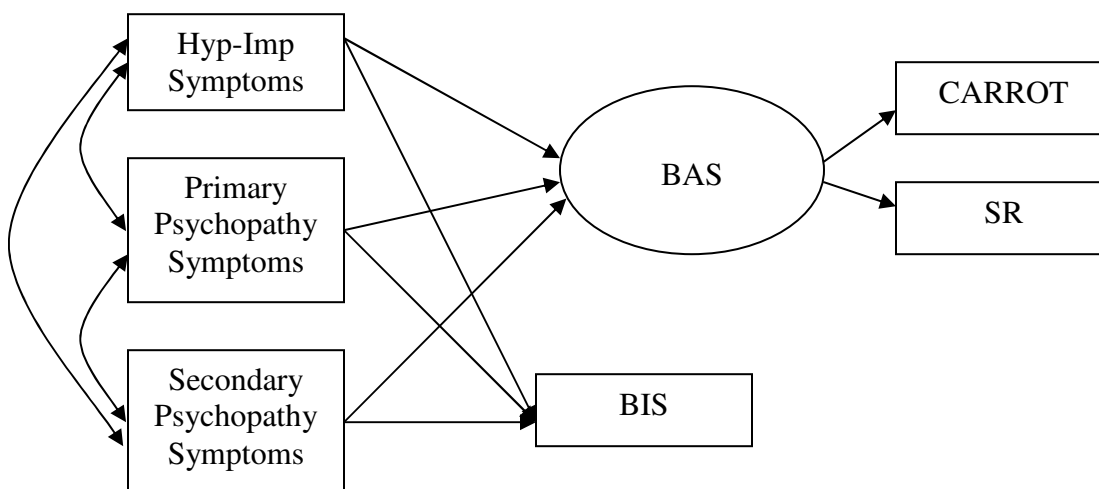
Figure 2. A simplified structural equation model assessing the relationship between AD/HD, Psychopathy, and BAS.

Note. In this recursive, overidentified model (error and disturbance terms are omitted for simplicity), the relationship between Hyperactive-Impulsive AD/HD symptoms, Primary Psychopathy, and Secondary Psychopathy symptoms with the BIS and BAS are examined. The curved arrow linking AD/HD and Psychopathy symptoms (a) indicates that they are correlated (i.e., in structural equation modeling, double-headed curved arrow represents covariance between a pair of variables) and (b) controls for this overlap when assessing how they are associated individually with the BAS and BIS variables. Although conceptually the BAS or BIS should predict Hyperactive-Impulsive AD/HD symptoms and Psychopathy symptoms, the direction of this model allows for controlling for the correlation between AD/HD and Psychopathy symptoms and is for practical purposes only. The structural portion of this model includes Hyperactive-Impulsive AD/HD symptoms, Psychopathy symptoms, BIS, and the BAS latent variable; the measurement portion of this model includes the BAS latent variable and the self-report (i.e., SR) and experimental behavior task (i.e., CARROT) measures. The BAS is a latent variable with loadings from the CARROT and SR scores. Hyp-Imp = Hyperactive-Impulsive AD/HD; SR = Sensitivity to Reward subscale of SPSRQ; CARROT = Card Arranging Reward Responsiveness Objective Test; BAS = Behavioral Approach System; BIS = Behavioral Inhibition System as indexed by the Sensitivity to Punishment scale from the SPSRQ.

Figure 3. A scatterplot of CARROT total scores with the Sensitivity to Reward Scale.
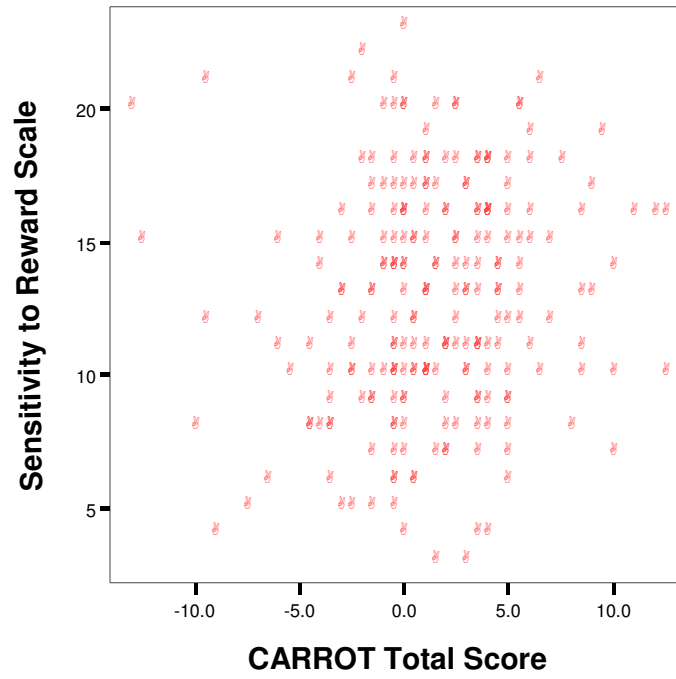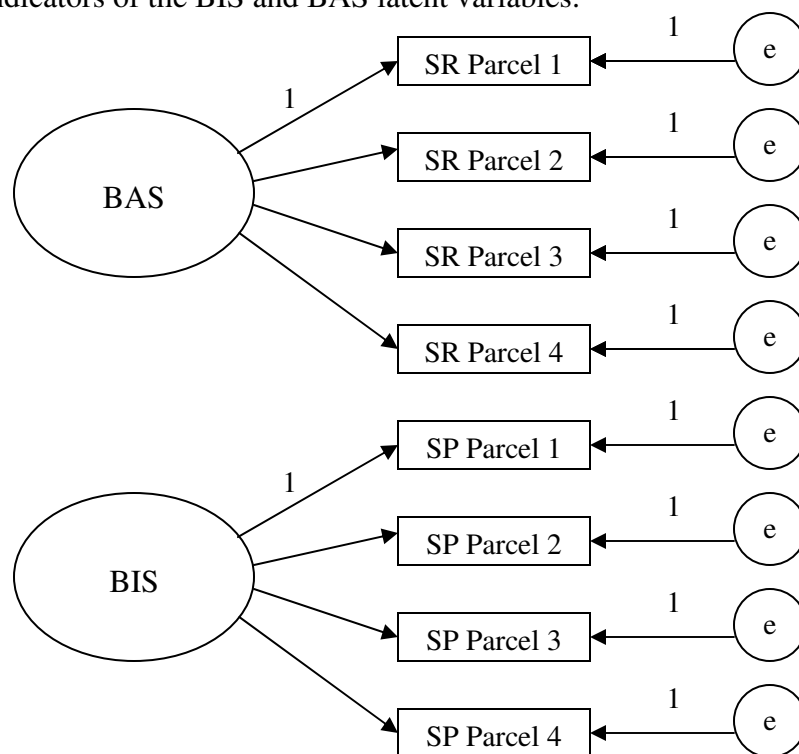
Figure 4. The revised measurement model of the structural equation model with item parcels as indicators of the BIS and BAS latent variables.
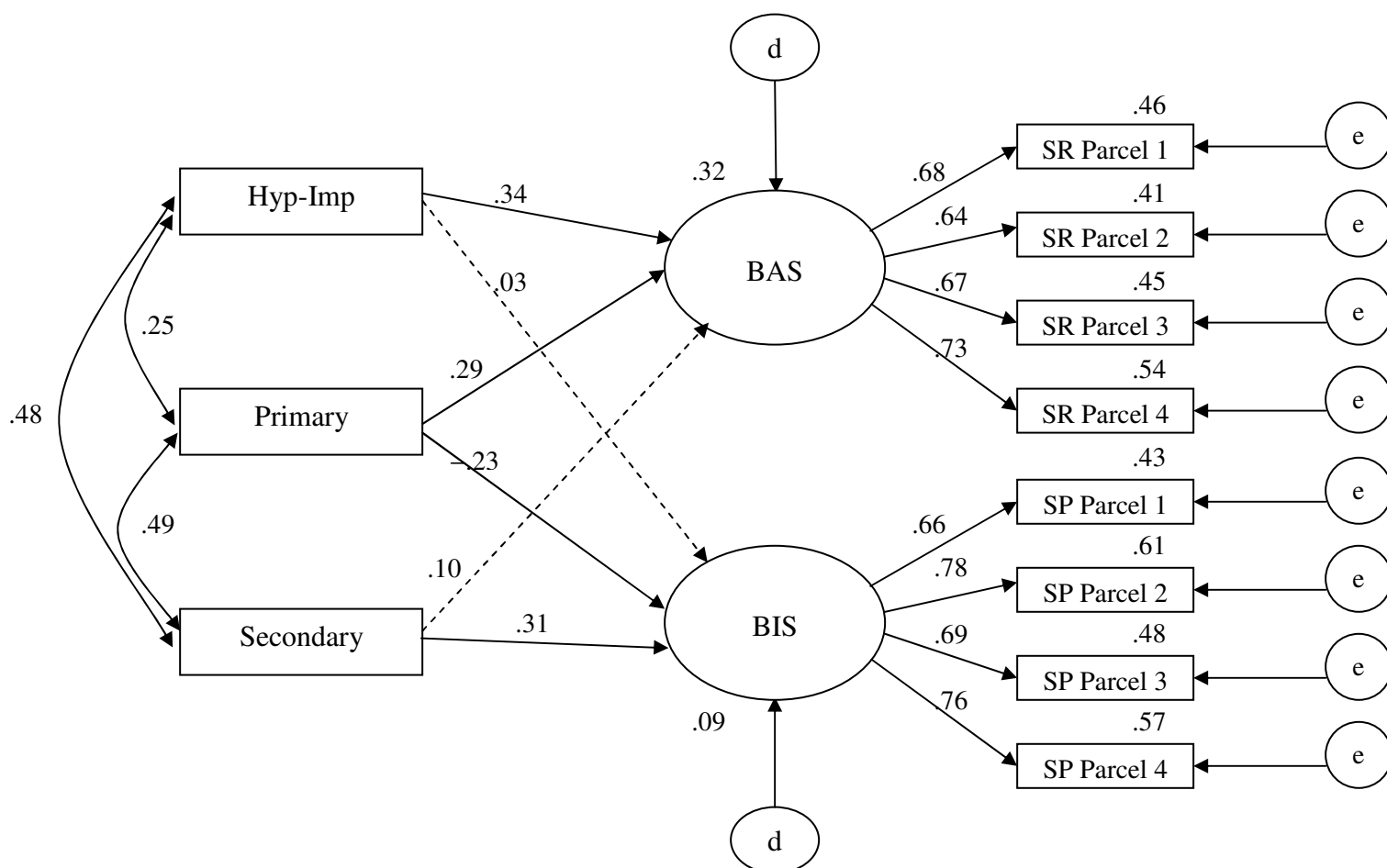

Note. SP = Sensitivity to Punishment Scale; SR = Sensitivity to Reward Scale; BAS = Behavioral Approach System; BIS = Behavioral Inhibition System; e = error term.

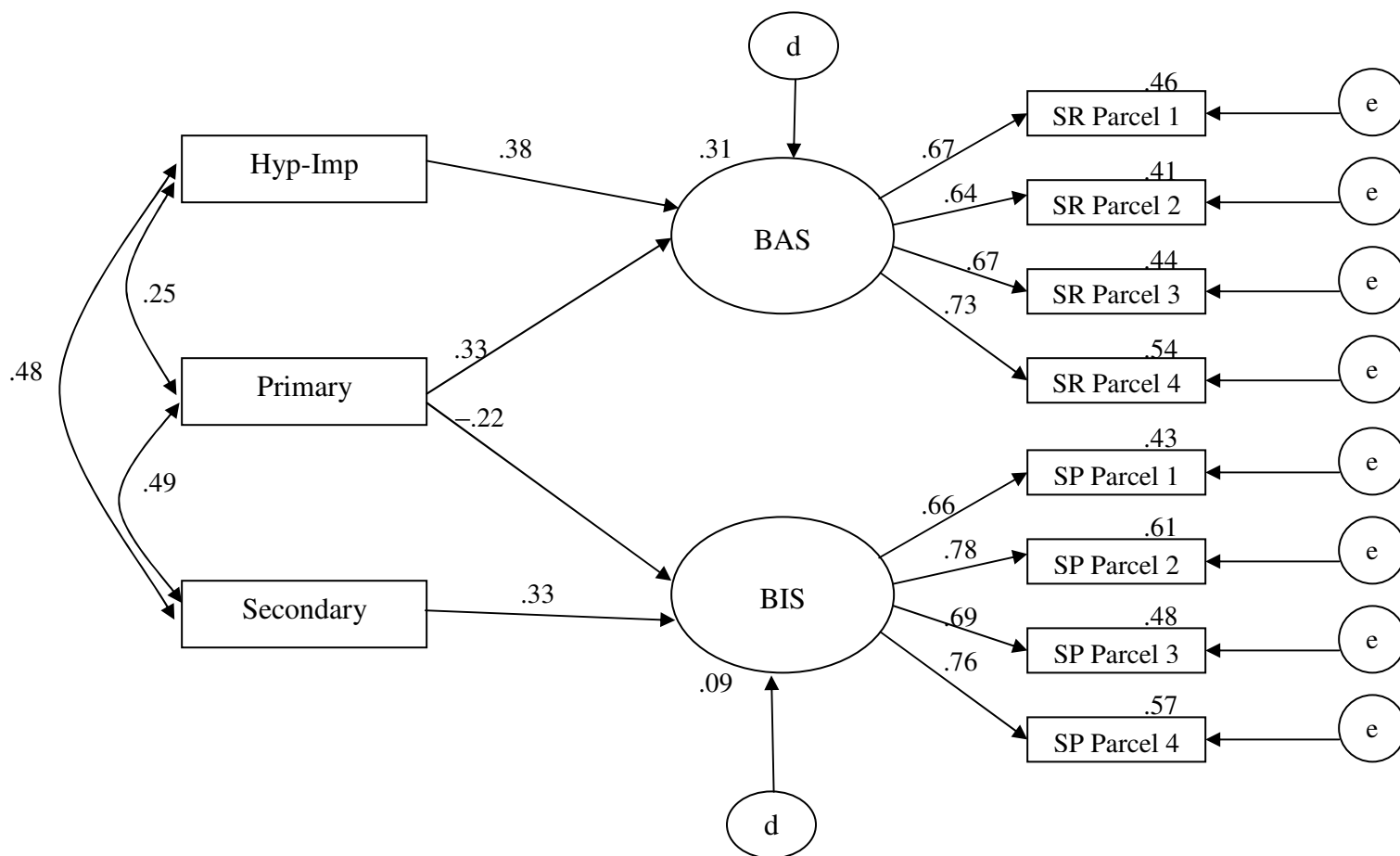*Figure 5*. The revised model, including the measurement and structural portions.
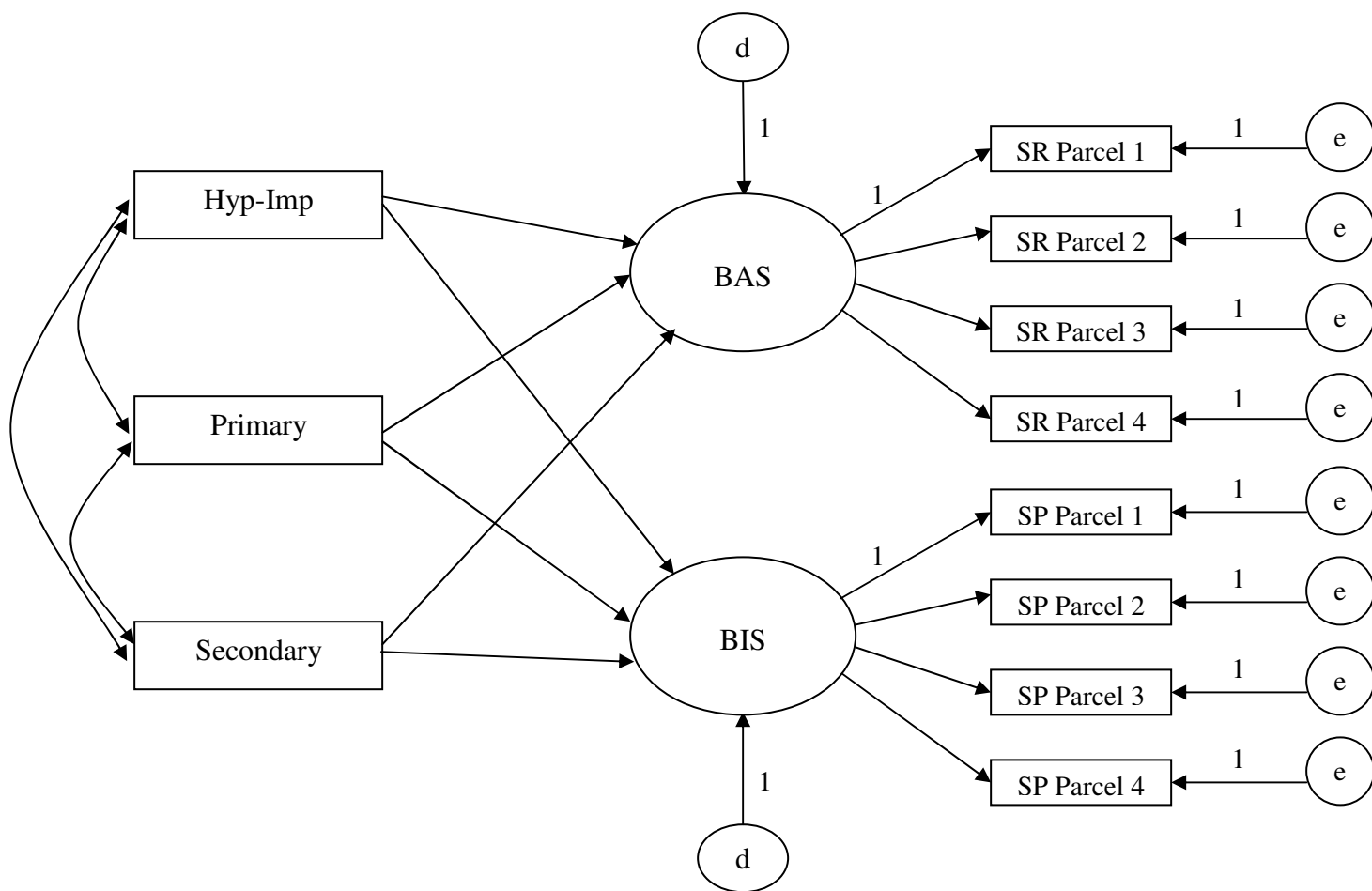
Note. Hyp-Imp = Hyperactive-Impulsive AD/HD symptoms; Primary = Primary Psychopathy symptoms; Secondary = Secondary Psychopathy symptoms; d = disturbance term; e = error term; BIS = Behavioral Inhibition System; BAS = Behavioral Approach System; SP = Sensitivity to Punishment scale; SR = Sensitivity to Reward scale.
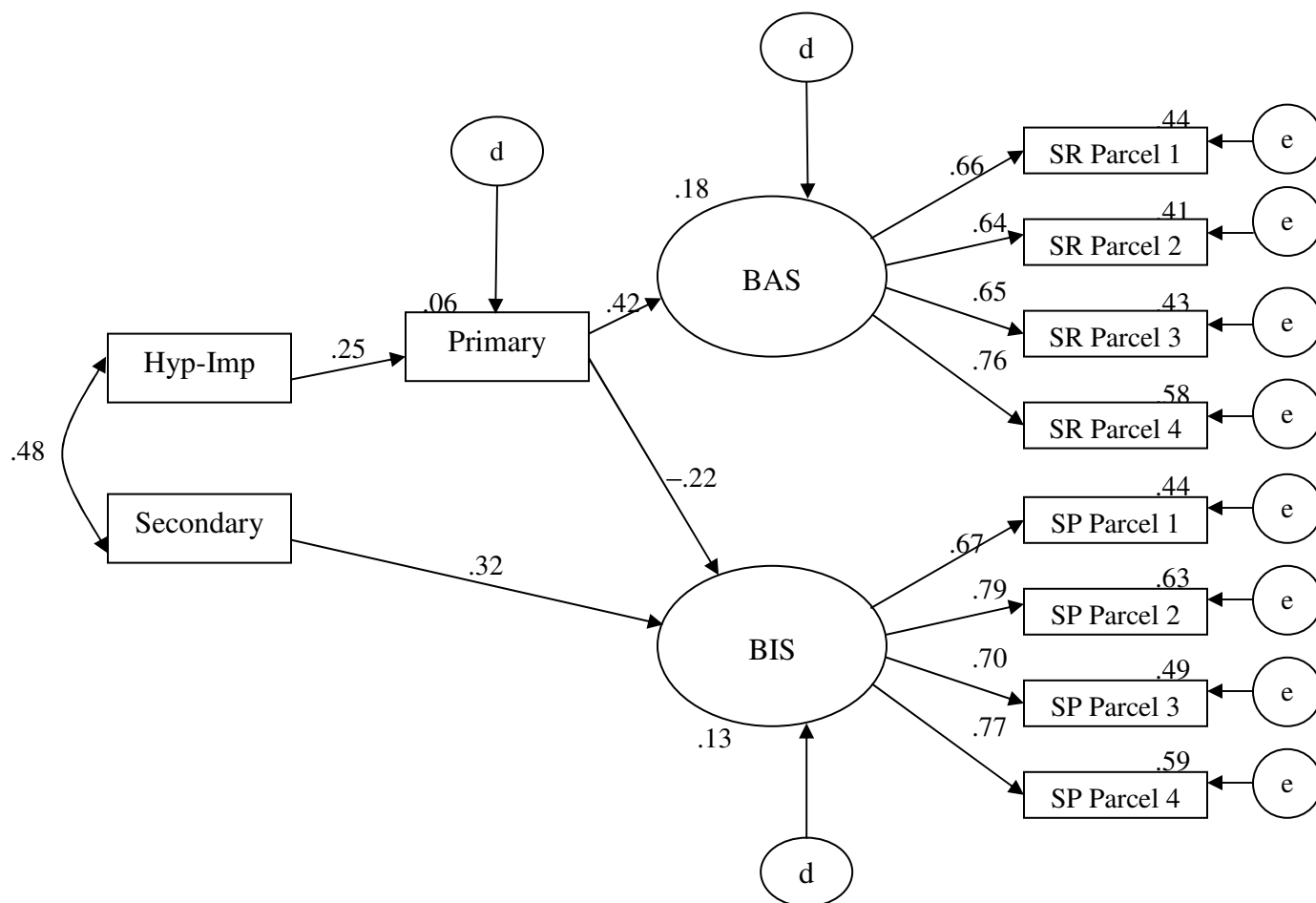
Figure 6. Model 1a.

Note. Dashed lines indicate non-statistically significant paths, all other pathways are statistically significant at the *p* < .001 level (with the exception of Primary Psychopathy to BIS pathway with *p* = .006); Hyp-Imp = Hyperactive-Impulsive AD/HD symptoms; Primary = Primary Psychopathy symptoms; Secondary = Secondary Psychopathy symptoms; d = disturbance term; e = error term; BIS = Behavioral Inhibition System; BAS = Behavioral Approach System; SP = Sensitivity to Punishment scale; SR = Sensitivity to Reward scale.

Figure 7. Model 1b.

Note. All pathways are statistically significant at the *p* < .001 level, with the exception of Primary Psychopathy to BIS pathway with *p* = .007; Hyp-Imp = Hyperactive-Impulsive AD/HD symptoms; Primary = Primary Psychopathy symptoms; Secondary = Secondary Psychopathy symptoms; d = disturbance term; e = error term; BIS = Behavioral Inhibition System; BAS = Behavioral Approach System; SP = Sensitivity to Punishment scale; SR = Sensitivity to Reward scale.

Figure 8. Model 2.

Note. All pathways are statistically significant at the *p* < .001 level, with the exception of Primary Psychopathy to BIS pathway with *p* = .002; Hyp-Imp = Hyperactive-Impulsive AD/HD symptoms; Primary = Primary Psychopathy symptoms; Secondary = Secondary Psychopathy symptoms; d = disturbance term; e = error term; BIS = Behavioral Inhibition System; BAS = Behavioral Approach System; SP = Sensitivity to Punishment scale; SR = Sensitivity to Reward scale.

Appendix C
Motivated Go/No-Go Task Description

The motivated go/no-go task (Newman, Widom, & Nathan, 1985) is proposed to assess BIS activity, BAS activity, and response modulation with its three conditions (punishment-only, reward-only, and mixed incentive, respectively). In all conditions, participants start with 64 cards that contain eight different two-digit numbers per card (e.g., 17, 98, 24, 32, 49, 67, 71, 88, 13). These eight numbers are repeated eight times in random order for a total of 64 individual trials. Participants are instructed to respond to four of the numbers (i.e., go) and not to respond to the other four numbers (i.e., no-go).

In the punishment-only condition, participants are financially punished for (a) responding to a number they are instructed not to respond to (inappropriately responding to no-go stimuli) or (b) not responding to a number they are instructed to respond to (inappropriately not responding to go stimuli). In this condition, someone with an underactive BIS would be predicted to perform poorly because they are less responsive to punishment.

In the reward-only condition, participants are financially rewarded for (a) responding to a number they are instructed to respond to (appropriately responding to go stimuli) or (b) not responding to a number they are instructed not to respond to (appropriately not responding to no-go stimuli). In this condition, someone with an overactive BAS would be predicted to over respond because they are more responsive to reward cues.

In the mixed incentive condition, the reward and punishment conditions are combined. Participants are punished for inappropriate responses (lose money for responding to no-go stimuli) and rewarded for appropriate responses (win money for responding to go stimuli). In this condition, assuming this is an index of response moduation, someone with a response modulation deficit would be predicted to perform poorly.

Appendix D
Card Playing Task Description

        The card playing task (Siegel, 1978) assesses the perseveration of an appetitive response despite the introduction of punishment which makes responding maladaptive (i.e., response modulation; Newman & Wallace, 1993). It has only one condition and does not assess BAS-only or BIS-only activity. People hypothesized to have a response modulation deficit are likely to perform worse on this task. Participants start with 100 playing cards and are instructed that they will be financially rewarded when the next card in the deck is a face card. However, they will also be financially punished with a number card. They are not allowed to skip any cards, but they may stop at any time and keep the money they earned up to that point. One card is presented at a time. At the onset of the task, approach behavior is elicited. The cards are prearranged so that 9 out of the first 10 cards are face cards, then 8 out of 10, then 7 out of 10, and so on. A dominant response set is established as a result of a high amount of reinforcement for responding (i.e., high BAS activity). However, as the task continues, responding (i.e., approach behavior) becomes maladaptive. Adaptive performance requires participants to shift their attention to the changing probabilities of rewards and punishments, which is gradually becoming more punishing. Quitting requires the person to suspend goal-directed behavior (i.e., continue and seek rewards) and to respond to the change in environmental cues (i.e., increase in the occurrence of punishment). The dependent measure is the number of cards that a subject will play before quitting and amount of money earned. An adapted child version of this task is called the door opening task. In this computerized task, doors are used instead of cards. Once a door is opened, the child is provided feedback that the door was either a winning or losing door.

Appendix E

Demographics Form

Please respond to each question below by circling the appropriate response or filling in the blank space.

1. What is your age?

    _____

2. What is your race or ethnic origin?
    a. White/European American
    b. African American
    c. Hispanic
    d. Asian
    e. Other: _____

3. What is your family's annual household income?
    a. less than $10,000
    b. $10,000 to $20,000
    c. $20,000 to $30,000
    d. $30,000 to $50,000
    e. above $50,000

4. How many years have you been in college?

    _____

5. What is your gender?
    a. male
    b. female

6. What is your College Major?

    _____

7. What is your Grade Point Average (GPA)? (high school GPA if you do not have a college GPA)

    _____

8. What was your total score (verbal plus quantitative) on the SAT?

    _____

Appendix F
CAARS

The entirety of this measure cannot be displayed due to copyright laws. Please refer to Table 1 (Appendix A) for a listing of *DSM-IV-TR* (APA, 2000) Hyperactive-Impulsive AD/HD symptoms. These symptoms were assessed by the CAARS in the current study.

Additional information regarding the CAARS is listed in the following citation:
Conners, C.K., Erhardt, D., & Sparrow, E. (2000). *Conners Adult ADHD Rating Scales*. North Tonawanda, NY: Multi-Health Systems.

Appendix G

LSRP

Participant #: ☐☐☐☐

Using the following scale, indicate the degree to which you agree with each statement by filling in the corresponding bubble.

1 = Disagree Strongly	2 = Disagree Somewhat	3 = Agree Somewhat	4 = Agree Strongly

1. Success is based on survival of the fittest; I am not concerned about the losers.. ① ② ③ ④

2. For me, what's right is whatever I can get away with .... ① ② ③ ④

3. In today's world, I feel justified in doing anything I can to succeed .... ① ② ③ ④

4. My main purpose in life is getting as many goodies as I can .... ① ② ③ ④

5. Making a lot of money is my most important goal.. ① ② ③ ④

6. I let others worry about higher values; my main concern is with the bottom line ① ② ③ ④

7. People who are stupid enough to get ripped off usually deserve it .... ① ② ③ ④

8. Looking out for myself is my top priority .... ① ② ③ ④

9. I tell other people what they want to hear so that they will do what I want them to do .... ① ② ③ ④

10. I would be upset if my success came at someone else's expense .... ① ② ③ ④

11. I often admire a really clever scam .... ① ② ③ ④

12. I make a point of trying not to hurt others in the pursuit of my goals .... ① ② ③ ④

13. I enjoy manipulating other people's feelings .... ① ② ③ ④

14. I feel bad if my words or actions cause someone else to feel emotional pain .... ① ② ③ ④

15. Even if I were trying very hard to sell someone something, I wouldn't lie about it... ① ② ③ ④

16. Cheating is not justified because it is unfair to others .... ① ② ③ ④

17. I find myself in the same kinds of trouble, time after time .... ① ② ③ ④

18. I am often bored .... ① ② ③ ④

19. I find that I am able to pursue one goal for a long time .... ① ② ③ ④

20. I don't plan anything very far in advance .... ① ② ③ ④

21. I quickly lose interest in tasks I start .... ① ② ③ ④

22. Most of my problems are due to the fact that other people just don't understand me .... ① ② ③ ④

23. Before I do anything, I carefully consider the possible consequences .... ① ② ③ ④

24. I have been in a lot of shouting matches with other people .... ① ② ③ ④

25. When I get frustrate, I often "let off steam" by blowing my top .... ① ② ③ ④

26. Love is overrated .... ① ② ③ ④

Appendix H

SPSRQ

ID number

○ Yes ○ No 1. Do you often refrain from doing something because you are afraid of it being illegal?

○ Yes ○ No 2. Does the good prospect of obtaining money motivate you strongly to do some things?

○ Yes ○ No 3. Do you prefer not to ask for something when you are not sure you will obtain it?

○ Yes ○ No 4. Are you frequently encouraged to act by the possibility of being valued in your work, in your studies, with your friends or with your family?

○ Yes ○ No 5. Are you often afraid of new or unexpected situations?

○ Yes ○ No 6. Do you often meet people that you find physically attractive?

○ Yes ○ No 7. Is it difficult for you to telephone someone you do not know?

○ Yes ○ No 8. Do you like to take some drugs because of the pleasure you get from them?

○ Yes ○ No 9. Do you often renounce your rights when you know you can avoid a quarrel with a person or an organization?

○ Yes ○ No 10. Do you often do things to be praised?

○ Yes ○ No 11. As a child, were you troubled by punishments at home or in school?

○ Yes ○ No 12. Do you like being the center of attention at a party or a social meeting?

○ Yes ○ No 13. In tasks that you are not prepared for, do you attach great importance to the possibility of failure?

○ Yes ○ No 14. Do you spend a lot of your time on obtaining a good image?

○ Yes ○ No 15. Are you easily discouraged in difficult situations?

○ Yes ○ No 16. Do you need people to show their affection for you all the time?

○ Yes ○ No 17. Are you a shy person?

○ Yes ○ No 18. When you are in a group, do you try to make your opinions the most intelligent or the funniest?

○ Yes ○ No 19. Whenever possible, do you avoid demonstrating your skills for fear of being embarrassed?

○ Yes ○ No 20. Do you often take the opportunity to pick up people you find attractive?

○ Yes ○ No 21. When you are with a group, do you find you have difficulties selecting a good topic to talk about?

○ Yes ○ No 22. As a child, did you do a lot of things to get people's approval?

○ Yes ○ No 23. Is it often difficult for you to fall asleep when you think about things you have done or must do?

○ Yes ○ No 24. Does the possibility of social advancement move you to action, even if this involves not playing fair?

○ Yes ○ No 25. Do you think a lot before complaining in a restaurant if your meal is not well prepared?

○ Yes ○ No 26. Do you generally give preference to those activities that imply an immediate gain?

○ Yes ○ No 27. Would you be bothered if you had to return to a store when you noticed you were given the wrong change?

○ Yes ○ No 28. Do you often have trouble resisting the temptation of doing forbidden things?

○ Yes ○ No 29. Whenever you can, do you avoid going to unknown places?

○ Yes ○ No 30. Do you like to compete and do everything you can to win?

○ Yes ○ No 31. Are you often worried by things that you said or did?

○ Yes ○ No 32. Is it easy for you to associate tastes and smells to very pleasant events?

○ Yes ○ No 33. Would it be difficult for you to ask your boss for a raise (salary increase)?

○ Yes ○ No 34. Are there a large number of objects or sensations that remind you of pleasant events?

○ Yes ○ No 35. Do you generally try to avoid speaking in public?

○ Yes ○ No 36. When you start to play with a slot machine, it is often difficult for you to stop?

○ Yes ○ No 37. Do you, on a regular basis, think that you could do more things if it was not for your insecurity or fear?

○ Yes ○ No 38. Do you sometimes do things for quick gains?

○ Yes ○ No 39. Comparing yourself to people you know, are you afraid of many things?

○ Yes ○ No 40. Does your attention easily stray from your work in the presence of an attractive stranger?

○ Yes ○ No 41. Do you often find yourself worrying about things to the extent that performance in intellectual abilities is impaired?

○ Yes ○ No 42. Are you interested in money to the point of being able to do risky jobs?

○ Yes ○ No 43. Do you often refrain from doing something you like in order not to be rejected or disapproved of by others?

○ Yes ○ No 44. Do you like to put competitive ingredients in all of your activities?

○ Yes ○ No 45. Generally, do you pay more attention to threats than pleasant events?

○ Yes ○ No 46. Would you like to be a socially powerful person?

○ Yes ○ No 47. Do you often refrain from doing something because of your fear of being embarrassed?

○ Yes ○ No 48. Do you like displaying your physical abilities even though this may involve danger?

Appendix I

Infrequency Scale

True False 1. On some mornings, I didn't get out of bed immediately when I first woke up.

True False 2. There have been a number of occasions when people I have known said hello to me.

True False 3. There have been times when I have dialed a telephone number only to find that the line was busy.

True False 4. At times when I was ill or tired, I have felt like going to bed early.

True False 5. On some occasions I have noticed that some other people are better dressed than myself.

True False 6. Driving from New York to San Francisco is generally faster than flying between these cities.

True False 7. I believe that most light bulbs are powered by electricity.

True False 8. I go at least once every two years to visit either northern Scotland or some part of Scandinavia.

True False 9. I cannot remember a time when I talked with someone who wore glasses.

True False 10. Sometimes when walking down the sidewalk, I have seen children playing.

True False 11. I have never combed my hair before going out in the morning.

True False 12. I find that I often walk with limp, which is the result of a skydiving accident.

True False 13. I cannot remember a single occasion when I have ridden on a bus.

Appendix J
CARROT Administration Directions

The Card-Arranging Reward Responsivity Objective Test (CARROT; Powell et al., 1996)
The CARROT is a simple psychomotor task designed to assess responsiveness to reward and involves four trials of card sorting (see Table below for a summary of trials).

The cards, which are slightly larger than playing cards, each display five digits listed vertically ranging from 1 to 9 (see example below). One of the numbers on each card (and only one) is either a '1' or a '2' or a '3' and participants are asked to sort the cards into corresponding piles according to these critical numbers whilst ignoring the others. A practice trial of twenty cards is firstly conducted to ensure that participants understand the task. After successful completion of this practice trial, participants are informed that the main task will now begin and they will be given more cards to sort in the same way. At the beginning of each trial, the following instructions are given: *"Please sort through the pile of cards as quickly as you can without making any mistakes. If you do make a mistake however, don't worry about it and just carry on sorting but try not to make any more."*

On trial one (baseline), the participant is presented with a pile of 60 cards and the time taken to sort these cards is recorded. This individually determined time then becomes the time that participant is given for card sorting on subsequent trials. For trials two, three and four, the participant is given a larger pile of 100 cards and informed that he or she will be told when to stop sorting. The procedure for trials two (NR1) and four (NR2) are identical and these comprise the non-rewarded trials. On the third trial (R) however, participants are offered a financial incentive for speed of sorting. Specifically, he or she is informed that: *"This time there is a reward for fast sorting .For every five cards you sort, you will be given twenty cents which I will place on the table in front of you. There are no 'catches' to this and at the end of the trial you will be given the money and it will not be taken away from you later."*

CARROT Trial Summary Table

Trial	Trial Type	Description
1	Baseline (B)	Time (in x seconds) to sort 60 cards
2	Non-Reward (NR1)	No. of cards (out of 100) in 'x secs.'
3	Reward (R)	No. of cards (out of 100) in 'x secs.'
4	Non-Reward (NR2)	No. of cards (out of 100) in 'x secs.'

To maintain consistency in the unit of measurement across all trials and from time 1 to time 2, scores were converted to rates (i.e. number of cards sorted per second: B^{RATE}, $NR1^{RATE}$, R^{RATE}, $NR2^{RATE}$).

The CARROT yields a 'reward responsivity index' which can be computed by subtracting the average of the two non-rewarded trials (NR^{RATE} = [$NR1^{RATE}$ + $NR2^{RATE}$ /2]) from the rewarded trial (R^{RATE}). Two non-rewarded trials, one either side of the rewarded trial, are included to account for practice and fatigue effects, and the mean of these two trials is used. A positive reward responsivity index therefore reflects an increase in the speed of card sorting with reward relative to non-reward while a negative score reflects a decreased speed of card sorting with reward relative to non-reward. In a pilot study of the CARROT in a sample of 71 healthy males, Al-Adawi (unpublished doctoral dissertation) reported that speed of card sorting was significantly faster in the rewarded than the non-rewarded condition.

Examples of CARROT cards

<table>
<tr><td>

4
6
2
9
5

</td><td>

7
5
9
4
1

</td></tr>
<tr><td>

4
6
9
3
7

</td><td>

1
9
5
7
8

</td></tr>
</table>
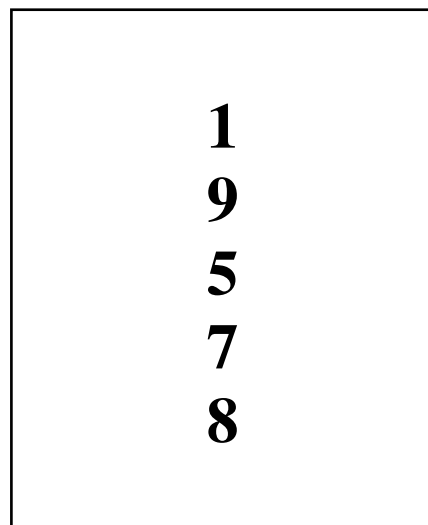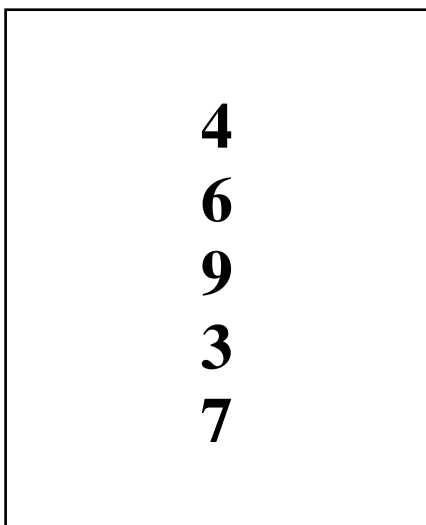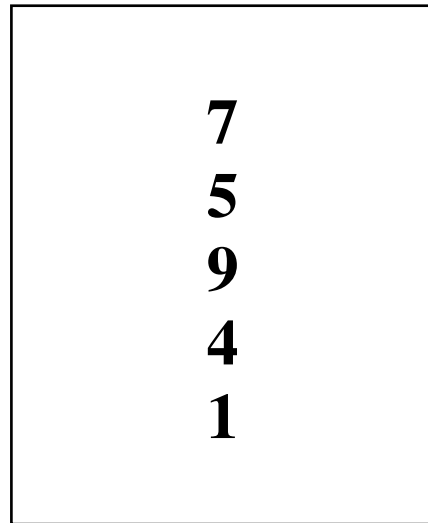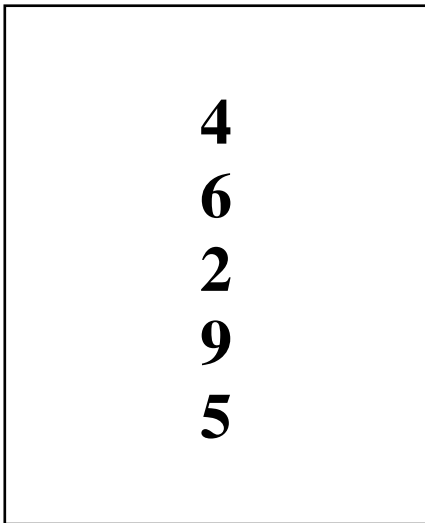
Appendix K

Consent Form

Project Title: Personality and Young Adult Functioning

Project Directors: John Mitchell, M.A. and Rosemery Nelson-Gray, Ph.D.

Participant's Name: _____

Date of Consent: _____

DESCRIPTION AND EXPLANATION OF PROCEDURES: This project, in which you will be participating, is designed to examine how different traits are related to peoples' overall functioning. Participation involves the completion of questionnaires and a behavioral task and will take between 90 and 120 minutes. For your participation as an introductory psychology student, you will receive experimental credits as appropriate for the time you spend completing questionnaires and participating in this research project. A copy of this consent form may be obtained upon request.

RISKS AND DISCOMFORTS: You may become mildly uncomfortable during your participation in this project because of the questions to which you will be asked to respond. Any distress you may feel, however, is not anticipated to be any greater than that experienced in daily living. Your participation in this project is entirely voluntary and, should you become uncomfortable or distressed, you are free to refrain from answering any questions or to withdraw from the study altogether at any point without penalty or prejudice.

All information that you give and questions you answer during the course of this project will be kept in confidentiality. No information you furnish will identify you personally in publications or presentations. Data will be kept in a secured site and destroyed after 5 years.

POTENTIAL BENEFITS: Participants will benefit in an increased understanding of issues related to psychological research and will have an opportunity to learn more about themselves through responses to questionnaires. Broader benefits will enable researchers and clinicians to better understand the overall functioning of young adults and lead to better treatment programs for adults having difficulty in their overall functioning.

COMPENSATION/TREATMENT FOR INJURY: Not Applicable.

CONSENT: The research and this consent form have been approved by the University of North Carolina at Greensboro Institutional Review Board which insures that research involving people follows federal regulations. Questions regarding your rights as a participant in this project can be answered by calling Dr. Beverly B. Maddox-Britt at (336) 334-5878. Questions regarding the research itself will be answered by calling John Mitchell at (336) 256-0050. Any new information that develops during the project will be provided to you if the information might affect your willingness to continue participation in the project.

By signing this consent form, you agree that you understand the procedures and any risks and benefits involved in this research. You are free to refuse to participate or to withdraw your consent to participate in this research at any time without penalty or prejudice; your participation is entirely voluntary. Your privacy will be protected because you will not be identified by name as a participant in this project.

By signing this form, you are agreeing to participate in the project described to you by either John Mitchell or the research assistant running this project.

_____   _____

Participant's Signature   Witness to Signature