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Effects of Extrinsic Rewards and Social Comparison on Inhibitory Control  
in Adults with ADHD Symptoms

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EFFECTS OF EXTRINSIC REWARDS AND SOCIAL COMPARISON ON INHIBITORY  
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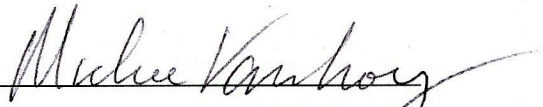
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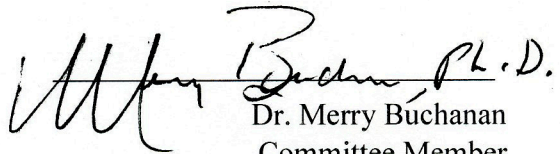
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## Abstract

The current study was an attempt to understand the links between reward processes, upward social comparisons, and behavioral inhibition in adults with ADHD and ADHD symptoms. Studies have shown that motivation can improve inhibitory control in children with ADHD, but little has been done to show the same effect in adults with ADHD. Additionally, social rewards such as praise and positive feedback have been shown to improve inhibitory control in children with ADHD, though not as strongly as tangible rewards. The current study used monetary rewards as well as false information regarding the performance of other participants to elicit an upwards social comparison. Monetary rewards had the greatest effect on the speed of inhibitory control in the ADHD group. Social comparison did not significantly improve the speed of response inhibition in the ADHD group, and in fact seemed to hurt accuracy. On the other hand, it did improve the speed of response inhibition for the non-ADHD control group. Neither monetary rewards nor the social comparison manipulation significantly affected the accuracy of the participants. Overall, the ADHD and control groups performed similarly. Future research needs to examine any differences that may exist in how individuals with ADHD symptoms use social comparison information when compared to their non-ADHD counterparts.

## Acknowledgments

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## TABLE OF CONTENTS

List of Figures.....	ii
List of Tables.....	iii
Chapter 1: Attention Deficit/Hyperactivity Disorder.....	1
Chapter 2: Social Comparison.....	9
Chapter 3: The Current Study.....	13
Chapter 4: Method.....	16
Chapter 5: Results.....	20
Chapter 6: Discussion.....	26
References.....	31
Appendix A: Adult ADHD Self-Report Scale v.1.1.....	42
Appendix B: Behavioral Health Screening Measure.....	46

## LIST OF FIGURES

Figure 1: The Presentation of the Stop-Signal Task.....	18
Figure 2: The Sequence of the Experimental Procedure.....	19
Figure 3: Interaction Effect of Reward and Information on SSRT during Session-1.....	22
Figure 4: Interaction of Reward and Information on SSRT during Session-2.....	23
Figure 5: Main Effect of Monetary Rewards in the ADHD Group.....	24
Figure 6: Main Effect of Information in the Non-ADHD Control Group.....	25

## LIST OF TABLES

Table 1: Mean PRS and SSRT Scores of the ADHD Group .....	20
Table 2: Mean PRS and SSRT Scores of the Non-ADHD (Control) Group .....	21

## CHAPTER 1

### Attention Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder (ADHD) is an increasingly diagnosed neurobehavioral disorder with a prevalence rate of approximately 6-7% in children (Molina et al., 2012) and a prevalence rate of 4.4% in adults (Kessler et al., 2006). The latter is only a recent topic of research, as the bulk of the research for the past couple of decades focuses on the disorder in children. The deficits that occur in individuals with ADHD have a wide-reaching negative impact on their lives. Of the millions of children diagnosed with ADHD, 50-70% of these children will experience some difficulty with social adjustment, functioning, and psychiatric problems in adolescence and adulthood, which can negatively impact relationships with peers (Cantwell, 1996; Sagvolden, Johansen, Aase, & Russell, 2005). ADHD also persists into adulthood in 75% of individuals who were diagnosed in childhood (Biederman et al., 1994).

Hyperactivity and impulsivity are the deficits most often associated with the disorder, in addition to difficulty sustaining attention and poor emotional regulation (Roberts, Fillmore, & Milich, 2011). Impulsive behaviors are commonly characterized by deficient regulation of inappropriate responses, encompassing over-rapid responsiveness, premature responsiveness, carelessness and recklessness, and a general inability to sufficiently plan and execute an appropriate response to an event or stimulus (Barkely, 1997). These types of inhibitory control issues are seen in oppositional-defiant disorder (ODD) and conduct disorder (Oosterlaan, Logan, & Sergeant, 1998), as well as in those with high-functioning autism (Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2004).

Working memory and the central executive functions have been a visible theme in ADHD research. Findings from various studies show that observable neuropsychological



markers of working memory dysfunction, especially short-term verbal memory, persist into adolescence or adulthood (Alderson, Kasper, Hudec, & Patros, 2013). In addition to this type of itemized memory that has been studied extensively, it has also been found that source memory or the context of episodic memory is deficient in adults with ADHD (Fuermaier et al., 2013). This study was one of the first to display a deficiency in source discrimination in adults with ADHD compared to healthy controls, an aspect of the disorder that has been overlooked in past research.

Up to 83% of adults with ADHD report sleep deficits (Philipsen, Hornyak, & Reimann, 2006). Individuals with ADHD show delayed sleep onset and difficulty waking up, increased nocturnal hyperactivity, deficient sleep efficiency, and reduced amount of time in REM (Rapid Eye Movement) sleep (Rybak, McNeely, Mackenzie, Jain, & Levitan, 2007; Sobanski, Schredl, Kettler, & Alm, 2008; Gruber et al., 2009; Boonstra et al., 2007). Significant impairments in the secretion of endocrine factors were found to affect the regulation of the circadian rhythms in humans as well as in the functioning of “clock” genes that generate circadian rhythms (Baird, Coogan, Siddiqui, Donev, & Thome, 2012).

## **Models of ADHD**

### **1. Barkley’s Behavioral Inhibition Model**

This model centers on behavioral inhibition as the core deficit of ADHD. Behavioral inhibition involves three interrelated processes: the inhibition of an initial prepotent response to an event, the stopping of an ongoing response, and interference control whereby individuals can avoid disruption from competing events and responses while attempting to make a self-directed response (Barkley, 1997.) Poor behavioral inhibition in this model would be in the category of executive functions (EF), and would produce the other deficits that are often seen in ADHD.

Such deficits include poor emotional regulation, deficits of working memory, poor regulation of motivation, and trouble internalizing speech. Behavioral inhibition can be further described in terms of intentional motor inhibition, which is known as “executive inhibition” (Nigg, 2001). According to Nigg, executive inhibition involves the ability to cancel a planned action in order to achieve a later, predetermined goal. This would involve suppressing uninvolved internal and external stimuli, and is very close conceptually to the third aspect of behavioral inhibition in Barkley's model.

## **2. The Dynamic Developmental Model**

Deficient reward and reinforcement processes are among the various deficits of ADHD. According to the dynamic developmental behavioral theory, three hypodopaminergic pathways fail to modulate nondopaminergic transmission (Saagvolden et al., 2005). The failure of the dopaminergic processes to modulate GABA and glutamate lead to the exaggerated delay aversion that is seen in individuals with ADHD. Saagvolden et al. (2005) posited that hypofunctioning of the mesolimbic dopamine branch leads to hyperactivity in novel situations, impulsivity, inattention, and poor inhibitory control. Hypoactivity of the mesocortical dopamine branch leads to attention response deficiencies (behavioral and motor responses) and poor behavioral planning. A hypofunctioning nigrostriatal dopamine branch leads to deficient modulation of motor functions, and impairments of learning and memory. These tend to manifest in clumsiness, failures of inhibition when rapid response is required, and an apparent developmental delay. These three pathways are thought to interact with dysregulated frontostriatal circuits to produce the behavioral deficits that are observable in ADHD.

It is argued in the dynamic developmental behavioral theory that there are two main behavioral processes involved in the expression of ADHD: altered or deficient reinforcement of

novel behavior and deficient extinction of previously reinforced behavior. Insufficient glutamate input from the prefrontal cortex to dopamine neurons and the faulty regulation of dopamine release results in the hypodopaminergic pathways that contribute to these behavioral processes. Dopamine neuron activity is associated with the increased potentiation of reinforced behaviors and the weakening potentiation of non-reinforced behaviors. The model predicts that deficient dopamine regulation will result in an individual having more difficulty attaching consequence to behavior in the time window allotted for such an appraisal. They will also display marked deficits in the extinction of previously reinforced behaviors. In a Go/No-Go paradigm in which a participant must respond to several “Go” prompts and then inhibit a response to a “No-Go” prompt, an individual with ADHD would be unable to extinguish the reinforced “Go” response.

### **3. The Dual Pathway Model**

Other models have emerged that challenge the executive dysfunction stance, instead opting for a more motivationally based explanation. Lack of sustained attention when completing a task and an inability to delay gratification have both been shown as consistent deficits in ADHD (Sonuga-Barke, Williams, Hall, & Saxton, 1996; Schweitzer & Sulzer-Azaroff, 1995; Kuntsi, Oosterlaan & Stevenson, 2001). The dual-pathway model postulates a biologically based shortened delay gradient that minimizes the importance of future rewards while leading to a preference for reward immediacy. This explains why children with ADHD seem to be hypervigilant in their search of the environment for cues that will allow them to escape from delay (Sonuga-Barke, De Houwer, De Ruiter, Ajzentstzen, & Holland, 2004).

Sonuga-Barke (2003) illustrates a dual pathway hinging on the interaction between an executive circuit and a reward circuit. The interplay between the circuits is very complex, involving cortical and sub-cortical regions, as well as feedback loops. The executive circuit

features a glutamatergic excitatory feedback loop between the pre-frontal cortex and the caudate nucleus, with reciprocal excitatory feedback back to the pre-frontal cortex and other cortical regions. This circuit is mediated through GABA-based inhibitory mechanisms of the thalamus and other sub-cortical feedback loops. The reward circuit's activity is centered in the nucleus accumbens, with additional excitatory inputs from the thalamus and other frontal regions. It also receives input from the amygdala, which provides support for reinforcement processes.

The model also emphasizes the role of dopamine pathways in the executive circuit and reward circuit. Sonuga-Barke (2003) postulates that the executive circuit is modulated by the mesocortical and nigrostriatal dopamine pathways, while the reward circuit is modulated by the mesolimbic dopamine pathway. Deficiencies in production or distribution of dopamine by these pathways are seen as discrete biological bases for the psychological processes that give rise to executive dysfunction and delay aversion.

#### **4. The Working Memory Model**

The working memory model of ADHD asserts that the working memory construct is a core component in the expression of the disorder (Rapport et al., 2008). Working memory as a construct entails a multi-component storage system composed of a central executive oversight mechanism and two subsystems – the phonological loop (PH) and the visuospatial sketchpad (VS) – that serve as a modality specific temporary storage system and rehearsal mechanism (Baddeley, 2003). The central executive acts as a coordinator for the subsystems, allocates attentional resources, and provides a link between short term storage and long term memory. Children with ADHD displayed impairment in central executive functioning when using a dual task paradigm requiring them to divide their attention between two tasks (Rapport et al., 2008; Karatekin, 2004). However, they were not differentially impaired compared to controls in either

the VS or PH functioning alone (Karatekin, 2004). In contrast, Rapport et al. (2008) found children with ADHD showed more VS impairment relative to PH impairment, and all systems (central executive and sub-systems) were impaired relative to typically developing children.

## **5. The Cognitive-Energetic Model**

The cognitive-energetic model (CEM) posits that certain aspects of inhibition may be deficit in ADHD in a manner dependent upon the energetic state of the child (Sergeant, 2000). This model attempts to provide a more comprehensive account of the deficits that occur in individuals with ADHD, and allows for various factors to influence the behavior of these individuals. The (CEM) is a three-level model that attributes deficits of information processing to both computational factors (which include encoding, search, decision, and motor organization) and state factors (which include effort, arousal, and activation) in the first level. The second level of the model includes three energetic pools: effort, arousal, and activation. Effort is defined as the energy which is necessary to complete a task, and is affected by factors such as cognitive load. Effort is required when the current energetic state of the organism is not sufficient to complete the task at hand. Arousal is the measurable phasic differences in physiological or behavioral markers occurring in response to some input change. Berlyne (1969) described these as “collative variables”, which included changes in intensity of input that was unexpected by an organism, changes in the timing of an input, and a change in “ground” of input, particularly scarce or novel input. Activation was described as the tonic change of an organism to an input as opposed to the more phasic shifts of the arousal mechanisms. The third level of the model is active in planning and inhibition of responses, and is associated with executive functioning (Sergeant, 2000). In essence, the deficits seen in ADHD are due to the interplay between these computational mechanisms, the three energy pools, and executive functioning (Sergeant, 2005).

## 6. Chaotic Intermittency Model of Inattention

The chaotic intermittency model, a non-linear model of inattention that was conceptualized at the neuronal level and effective at the systems level, is an attempt to explain undesired fluctuations of attention while also providing a more thorough look at the root causes of the deficits of sustained attention seen in ADHD a non-linear model of inattention that was conceptualized at the neuronal level and effective at the systems level (Baghdadi, Jafari, Sprott, Towhidkhah, & Golpayegani, 2013). Intermittency in non-linear systems refers to the property of a chaotic system in which dynamics switch between two qualitatively different behaviors even though all control parameters of the system do not fluctuate. Like a chaotically intermittent system, the deficits of sustained attention can be seen even in the absence of external noise. Individuals with ADHD would thus have trouble maintaining focus on a game or any other activity without their attention shifting. The use of non-linear methods is a novel approach to the study of ADHD.

Using non-linear mathematics, the chaotic intermittency model assigns values to both the excitatory and inhibitory mechanisms of the brain as control parameters. In a dynamical, non-linear system such as this a minute change in parameter can result in extremely aberrant behavior in a system. The study showed that intermittency occurs in the system by taking the value of A (in this model A is equivalent to the output of dopamine in the system) from 12.473 (a value that keeps the system from experiencing intermittency) to 12.472. Essentially, one 1/1000<sup>th</sup> of a unit of value A is all it takes to cause the system to go from constant attending to attention switching. The model further elaborates that the system must have a parameter value for the level of dopamine that causes other neurotransmitter levels to change in order to keep the system from reaching a state of intermittency. Indeed, the model shows that a small increase in the value of B

(excitatory neurotransmitters) does indeed stabilize the system. This means that in the ADHD system there must exist some mechanism that keeps the system in intermittency.

## **7. The Default Mode Network (DMN) and ADHD**

The DMN is a network of brain regions active during periods of task-negative processing and suppressed during periods of task-positive processing (Raichle et al., 2001; Greicius & Menon, 2004). The DMN encompasses the posterior cingulate cortex (PCC), the medial prefrontal cortex (mPFC), and the bilateral inferior parietal cortex (IPC) (Greicius & Menon, 2004; Raichle et al., 2001). This network is inversely correlated with a task-positive network, which includes the dorsolateral prefrontal cortex (dPFC), IPC, and supplementary motor area (SMA) (Fransson, 2005, 2006). There is evidence that deactivation of the DMN is attenuated in individuals with ADHD; this attenuation leads to increases in reaction times, and led to a “default-mode interference” hypothesis. (Castellanos et al., 2008; Sonuga-Barke & Castellanos, 2007). This hypothesis states that the deficits of attention in ADHD are due to dysfunctional connectivity between the task-positive and task-negative networks. Abnormal connectivity within the DMN is linked to attentional and working memory deficits, as well as the overall variability in task performance that is characteristic of ADHD (Broyd et al., 2009).

The DMN interference has ramifications for the allocation of attentional resources, which is a function of the task-positive network (Broyd, Helps, & Sonuga-Barke, 2011). Children with ADHD showed an increase in reaction time variability associated with the failure to sufficiently deactivate mPFC during a working memory task; a failed suppression such as this is mitigated with methylphenidate in children with ADHD (Fassbender et al., 2009; Liddle et al., 2011).

## CHAPTER 2

### Social Comparison

The act of relying on the behavioral cues of others in order to make decisions in the absence of objective information is known as social comparison (Festinger, 1954). Social comparison affects the way people make decisions, as well as their general well-being (Fliessbach et al., 2007). It also can affect performance both in typically developed individuals and those with psychopathology through perceived and direct competition (Geurts, Luman, & Van Meel, 2008; Winickoff, Coltin, Morgan, Buxbaum, & Barnett, 1984). Compliance and energy conservation behaviors increase when others in a neighborhood display these energy saving behaviors (Nolan, Schultz, Caldini, Goldstein, & Giskevicius, 2008). In studies that involve social comparison based on observation, watching or comparing oneself to someone who is proficient in a task yields important information on how to improve one's own performance (Buunk & Ybema, 1997). These comparisons also seem to increase the motivation to perform better on a task. Performance appears to be improved in a number of task-relevant factors (Levine, 1983), including attention to task (Santrock & Ross, 1975), self-monitoring of performance (Hake, Vukelish, & Kaplin, 1973), time spent on a task (Nicholls, 1975), task vigilance under threat of failure (Brown & Inouye, 1978), and level of performance (Halisch & Heckausen, 1977; McClintock & Van Avermaet, 1975; Rijnsman, 1974). Humans tend to trust the actions of others when objective cues for behavior are absent. Social comparison theory would state that individuals should be more willing to engage in a behavior if it is consistent with the actions of others (Festinger, 1954). Much of the work on social comparison stems from Festinger's work.



## **Festinger's Social Comparison Theory**

Social comparison theory states that individuals have a drive to evaluate their own abilities, thoughts, and beliefs relative to other individuals (Festinger, 1954). The evaluation of an individual's ability depends on how others evaluate their ability in that area when that ability is of an ambiguous nature. Festinger uses the ability to write poetry as an example of an ambiguous ability appraisal, in which there is no objective criteria in which to order the performance. An instance of an unambiguous ability appraisal would be a runner striving to beat a predetermined time. Appraisals of ability and "levels of aspiration" become unstable across time when social comparison is not available. The level of aspiration for a given task is the objective score that one deems to be a good performance. When no social comparison is available, it is found that the "level of aspiration" becomes unstable over time. When one exceeds a previous level of aspiration, then the original aspiration is no longer a good score. Likewise, if one scores less than the original aspiration, then the aspiration level drops. Festinger points out that performance appraisal continues to fluctuate even when an individual has ample experience with a task.

Social comparison behaviors tend to decrease as the difference between one's own ability and the person being compared increases. Individuals prefer comparing themselves to people whose abilities are relatively close to their own, and in fact become less accurate with their appraisals when their comparisons are done to people whose abilities are far greater than theirs (or far less than theirs). In this case, individuals typically do not make comparisons, instead relying on their own subjective judgments regarding ability.

## **Social Comparison and the Classroom**

Social comparison used in classroom settings seems to facilitate greater learning and performance (Huguet, Dumas, Monteil, & Genestoux, 2001). Children tended to compare upwards with close friends whose performance was slightly above their own, identifying them as a model for self-improvement. This identification came when children described having a strong locus of control over their standing and performance relative to the comparison-target. Children who nominated a social –comparison target in their class performed better compared to controls with no offered social comparison, even on a task that required a powerful inhibitory response, such as the Stroop task (Blanton, Buunk, Gibbons, & Kuyper, 1999). The peer that was chosen was one who slightly outperformed the child. This stemmed from Festinger’s idea that social comparison fails when the target’s ability is highly different (in either direction) than one’s own ability level. Blanton et al. (1999) studied a group of Dutch ninth graders and found that those who compared their own exam scores with those who performed slightly better were more likely to improve across a variety of academic domains. Those with a higher level of comparative evaluation (how one evaluates their own performance in comparison to a group or individual’s performance) showed a higher level of academic achievement. Another study found that forced social comparison facilitates better performance when the target performs slightly better than the individual, though there were no findings for how performance is affected when the social comparison occurs in a downward fashion (Seta, 1982). The drive to perform better is thought to stem from the way in which schools are structured. Schools are often the first introduction for children to a center of authority, where performance and following prescribed rules are rewarded. This instills the sense of unidirectional (upward) evaluation and comparison to those

who perform and behave slightly better than oneself. Additionally, observing others do well seems to inspire a sense of potential in an individual (Wheeler, Martin, & Suls, 1997).

## CHAPTER 3

### The Current Study

The purpose of the current study was to observe the effects that upwards social comparisons had on behavioral inhibition deficits in adults with ADHD, with and without a paired monetary reward. Research supports the efficacy of monetary reinforcement in improving performance but little has been done to show that performance feedback can positively affect inhibitory control in individuals with ADHD. Performance feedback in this study was operationalized as feedback about the participant's performance, as well as information about other participants' performance on the same task (the task involved producing correct inhibitory responses to a stop-signal trial). Thus, performance feedback consisted of false information in the form of an "average score" that other participants had made in the first session. This "average score" was set at five responses higher than the participant themselves had scored in a previous session. This number was set arbitrarily as there was no precedent in the literature to guide this portion of the study. Instead, given how participants were expected to perform, five responses was selected as a number that would be high enough to improve performance without being high enough to hurt performance.

The reward and consequence processing deficiencies in ADHD seem to speak to some kind of motivational deficit, though the literature show conflicting results. The perceived motivational deficit has been attributed to dysfunction of the dopamine reward pathway (Volkow, Wang, & Baler, 2011). Individuals with ADHD had significant correlations between low scores on Achievement measures on the Multidimensional Personality Questionnaire (MPQ) and lower than normal activity in the mesoaccumbens dopamine pathway, which has been hypothesized to play a role in reward and motivational deficits in ADHD. In this view, ADHD is

not just a disorder characterized by poor inhibitory control and inattentiveness, but also by pervasive deficits of motivation. These motivational deficits seem to be physiologically, if not genetically, based. In a group of stop-signal task studies, several found that motivational incentives normalized performance in ADHD children (Slusarek, Velling, Bunk, & Eggers, 2001; Konrad, Gauggel, Manz, & Scholl, 2000; Michel, Kerns, & Mateer, 2005), while others found conflicting results (Oosterlaan & Sergeant, 1998; Scheres, Oosterlaan, & Sergeant, 2001; Stevens, Quittner, Zuckerman, & Moore, 2002). Shanahan, Pennington, and Willcutt (2008) found that children with ADHD performed worse than their non-ADHD peers on a stop task irrespective of incentives, which was interpreted as support for the behavioral inhibition theory of ADHD over both the motivational and dual pathway models. A more recent study (Herrera, Speranza, Hampshire, & Bekinschtein, 2014) manipulated magnitudes and schedules of monetary rewards in healthy individuals. Participants were given one of four types of reward feedback during a stop signal task: no reward, 1, 10, and 20-cent tokens. The monetary rewards were given in one of three random schedules: increasing reward, decreasing reward, and random presentation. The findings showed that the greatest impact came from the decreasing schedule with the highest magnitude, in which the highest reward was given at the beginning of the trial.

During the forced social comparison condition, the target of an individual is not freely chosen. This eliminates the concept of “comparison-level choice”, which is the level of performance that an individual would most often compare their own performance to (Blanton et al., 1999). The body of research has established the effects of upwards social comparisons on performance, yet there is very little to show the same effect in a clinical ADHD population. Testing how forced social comparison interacts with monetary rewards to improve inhibitory control in adults with ADHD could help improve future therapeutic interventions, as well as

potentially improving performance in other areas of life, such as employment or school. The current study may also demonstrate differences in how individuals with ADHD and those without ADHD utilize social comparison information. There are very few studies that show how social comparison is used to increase performance in adults with ADHD. Forced social comparison which facilitates better performance in a normal population may possibly have a negative effect on an individual with ADHD.

There were two measures of behavioral inhibition: Stop-Signal Reaction Time (SSRT) and Probability to Respond to Stimulus (PRS). The SSRT is the indirect measure of how long a participant takes to inhibit a response, while the PRS is the percentage of “STOP” signals that a participant responded to (the lower the PRS, the higher the accuracy of inhibition).

It was hypothesized that upwards social comparisons elicited by performance feedback would improve inhibitory control in adults with ADHD and ADHD symptoms. SSRT and PRS scores would be lower in the performance information condition compared to the no-information condition.

It was predicted that monetary rewards would increase performance. This would be reflected in a decrease in SSRT and PRS scores as amount of rewards increased.

Finally, it was expected that the combination of large monetary rewards and performance information would result in the greatest inhibitory control. Participants receiving the largest monetary reward and performance feedback would have the lowest SSRT and PRS scores.

## CHAPTER 4

### Method

#### Participants

During the course of the current study, 883 individuals completed the screening questionnaire. Of those, ninety-three students at a Midwestern metropolitan university were recruited to participate voluntarily or in partial fulfillment of course requirements. Participants were recruited from *General Psychology* via SONA Systems, and through flyers posted at the university's Disability Support Services office and across campus. Participants completed the Adult ADHD Self-Report Scale (ASRS v.1.1) and the Behavioral Health Screening Measure (BHSM) online as screening measures prior to participation in the study. Those who exhibited clinically significant symptoms on the screening measures including those who disclosed an ADHD diagnosis were assigned to the experimental group (n=46). All others were assigned to the non-ADHD control group (n=47). Participants were asked to report if they were taking any medication for ADHD before beginning the study. Those who reported taking medication were asked to take the same dose when completing the second session.

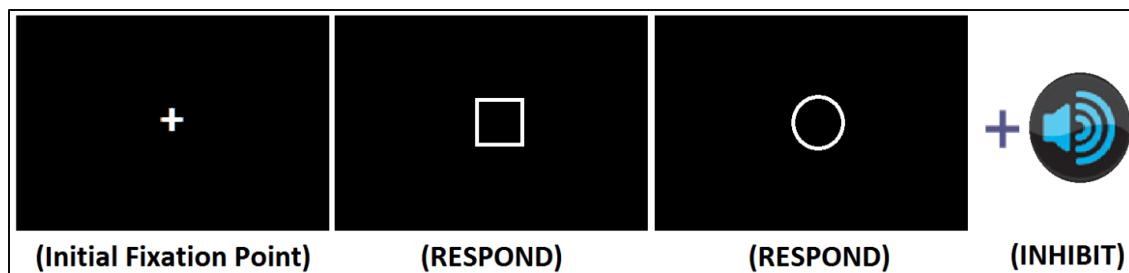
#### Materials

The screening measures (ASRS and BHSM) used to identify participants with ADHD symptoms are presented in Appendices A and B. All visual stimuli were presented using a Hewlett Packard Envy M6 touchscreen laptop with an AMD A10-5750 2.5 GHz APU.

**The stop-signal task.** A modified version of the computerized stop-signal task (STOP-IT) created by Verbruggen, Logan, & Stevens (2008) was used. Modifications included a counter in the upper right hand corner that displayed the amount of money that a participant in the low-reward or high-reward conditions had earned, as well as a static counter that displayed the false

feedback during the second session. Visual stimuli were either a white circle or square on a black background (Figure 1). Each experimental session consisted of one practice block of 32 trials and four experimental blocks of 64 trials. The no-signal trials (shape only) were presented 75% of the time (48 NS trials per block, 192 NS trials per session, 384 NS trials total per participants). Stop-signal trials in which the auditory stimulus (750Hz tone for 75ms) were presented 25% of the time (16 SS trials per block, 64 SS trials per session, 128 SS trials total per participant) following the visual stimulus presentation. The visual stimulus remained on the screen for a maximum of 1,250 ms, at which point the next trial would begin. The inter-trial interval was set at 2,000ms. The latency between the presentation of the visual stimulus and the stop signal was the Stop-Signal Delay (SSD). The SSD was initially set at 250ms in each block, but was adjusted by +/- 50ms for each successful or failed inhibitory response. When inhibition was successful, the SSD increased by 50ms and decreased by 50ms for each failed inhibition. Participants responded to the visual stimuli using a GameStop Xbox 360 game controller. Participants responded to circle stimuli by pressing the right trigger button and the square stimuli by pressing the left trigger button. The program calculated an estimate of latency of the stop process i.e. the Stop Signal Reaction Time (SSRT) = mean reaction time – mean SSD. Additionally, the program recorded how often a participant failed to inhibit a response, i.e. the probability to respond to the stimulus (PRS). Computations of the PRS and SSRT were done with the program ANALYZE-IT, an accompanying program for the STOP-IT program.





*Figure 1.* The Presentation of the Stop-Signal Task

Initial fixation point was replaced by the visual stimuli (either square or circle) after 250 ms. The visual stimulus remained until participants responded or 1,250 ms had passed.

On 25% of trials, an auditory stimuli was presented with the visual stimuli to indicate that a participant was to withhold their response.

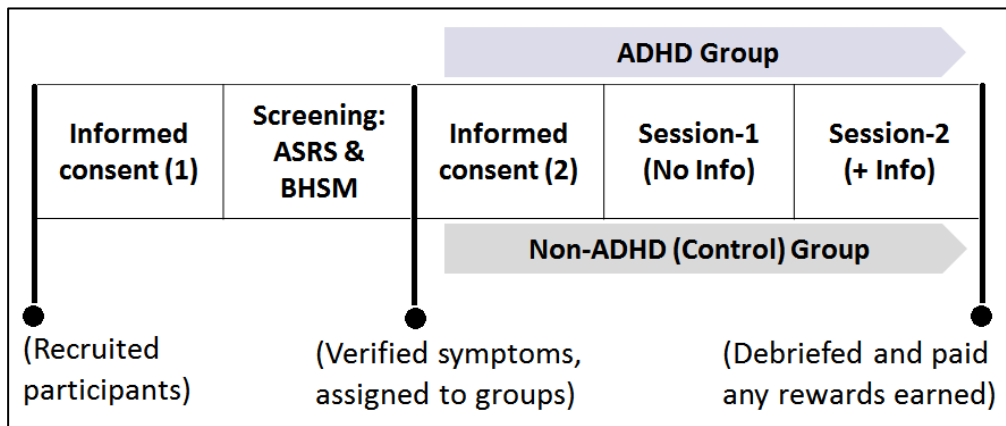
### **Procedure**

The experimental portion of the study consisted of two 20 minute sessions held on two separate days.

**Session – 1.** Participants were seated in a secluded, well-lit room approximately 24” from the laptop. The task instructions were presented on the screen, and the experimenter explained the task instructions to the participant. The participant was instructed to respond to the visual stimuli as quickly and accurately as possible. When the stop-signal (auditory tone) was presented, the participant was instructed to withhold their response. There was no reward for this in the no-reward condition. For those in the low-reward and high-reward conditions, correct response inhibition on the stop-signal trials earned one cent and ten cents respectively. A counter in the upper right hand corner of the screen kept track of how much money the participant had earned. The participant was made aware that any money earned during the course of the experiment would be paid to them at the conclusion of the second session. At the conclusion of the first session, participants scheduled a second session with the experimenter.

**Session – 2.** The participants completed the same task (with similar blocks of trials) for the second session, with the exception of a static counter in the upper right hand corner that displayed a number that represented the average number of correct response inhibitions that other participants had scored in the first session. This number was always set at five more correct response inhibitions than the participant themselves had scored in the first session. The money counter for the low-reward and high-reward condition reset to zero for the second session.

Participants in the ADHD group and the non-ADHD control group went through both experimental sessions in a similar manner (Figure 2). At the completion of the study, all participants were debriefed regarding the nature of the deception used in the study, and any rewards earned were distributed.



*Figure 2.* The Sequence of the Experimental Procedure

Participants were recruited and screened using the ASRS. Based on the screening results, they were assigned to the ADHD or non-ADHD control group and then randomly assigned to a reward condition. Each experimental session lasted approximately 20 minutes. Participants were debriefed and paid any rewards earned at the end of the second session.

## CHAPTER 5

### Results

#### Experimental Design

A mixed factorial design was used within each group of participants: monetary reward (no, low, and high) was the between-subjects factor and performance information (without and with) was the within-groups factor.

#### Data Analysis

Tables 1 and 2 show the descriptive statistics for each condition for the ADHD group and non-ADHD control group respectively.

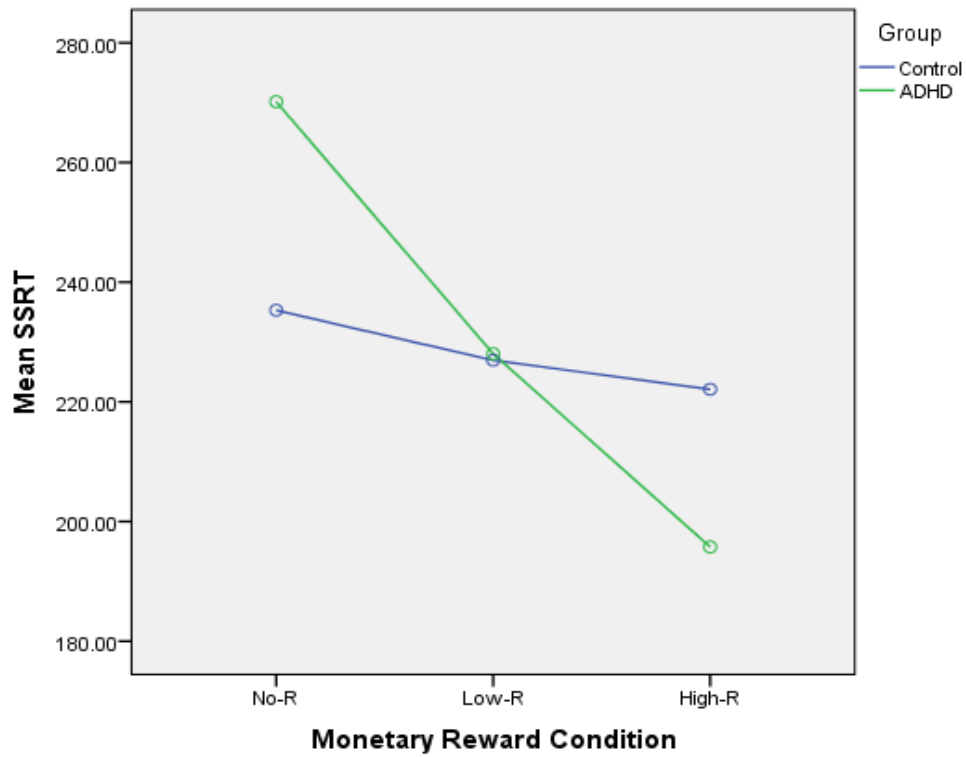
Table 1. Mean PRS and SSRT Scores of the ADHD Group

ADHD Group		Monetary Reward Condition					
		No-Reward ( <i>n</i> =15)		Low-Reward ( <i>n</i> =15)		High-Reward ( <i>n</i> =16)	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
PRS	No-Info	.47	.031	.47	.047	.45	.032
	Info	.48	.044	.45	.030	.46	.038
SSRT (ms)	No-Info	268.66	40.8	255.20	61.3	233.50	74.25
	Info	270.15	58.1	228.02	39.3	195.74	80.31

Table 2. Mean PRS and SSRT Scores of the Non-ADHD (Control) Group

Non-ADHD (Control) Group		Monetary Reward Condition					
		No-Reward (n=15)		Low-Reward (n=16)		High-Reward (n=16)	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<b>PRS</b>	<b>No-Info</b>	.45	.035	.45	.037	.46	.036
	<b>Info</b>	.47	.026	.46	.029	.45	.033
<b>SSRT</b> (ms)	<b>No-Info</b>	257.03	40.2	256.98	63.1	239.26	66.7
	<b>Info</b>	235.30	34.4	226.95	35.1	222.09	21.5

Data were analyzed using a 3x2x2 mixed MANOVA. There was an interaction effect of monetary reward condition and information for the SSRT variable ( $F(2,87)=6.34, p<.01, Wilk's \Lambda = 0.87, \text{partial } \eta^2 = .13, \text{power} = .89$ ). There was a main effect of information ( $F(1,87)=6.68, p<.01, \text{partial } \eta^2 = .071, \text{power} = .72$ ) and a main effect of condition ( $F(2,87)=6.89, p<.01, \text{partial } \eta^2 = .137, \text{power} = .914$ ). Figures 3 and 4 illustrate the interaction effect of information and reward condition for the No-Information and Information conditions, respectively.



*Figure 3.* Interaction Effect of Reward and Information on SSRT during Session-1.

There was a significant interaction of monetary reward condition and information condition for the SSRT measure.

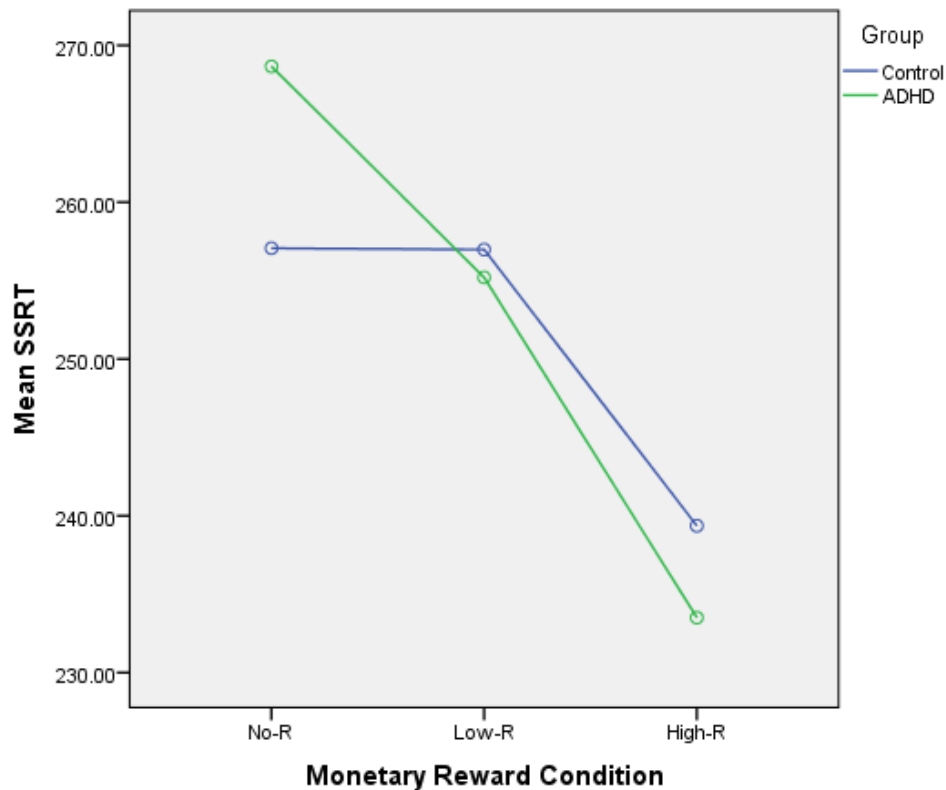


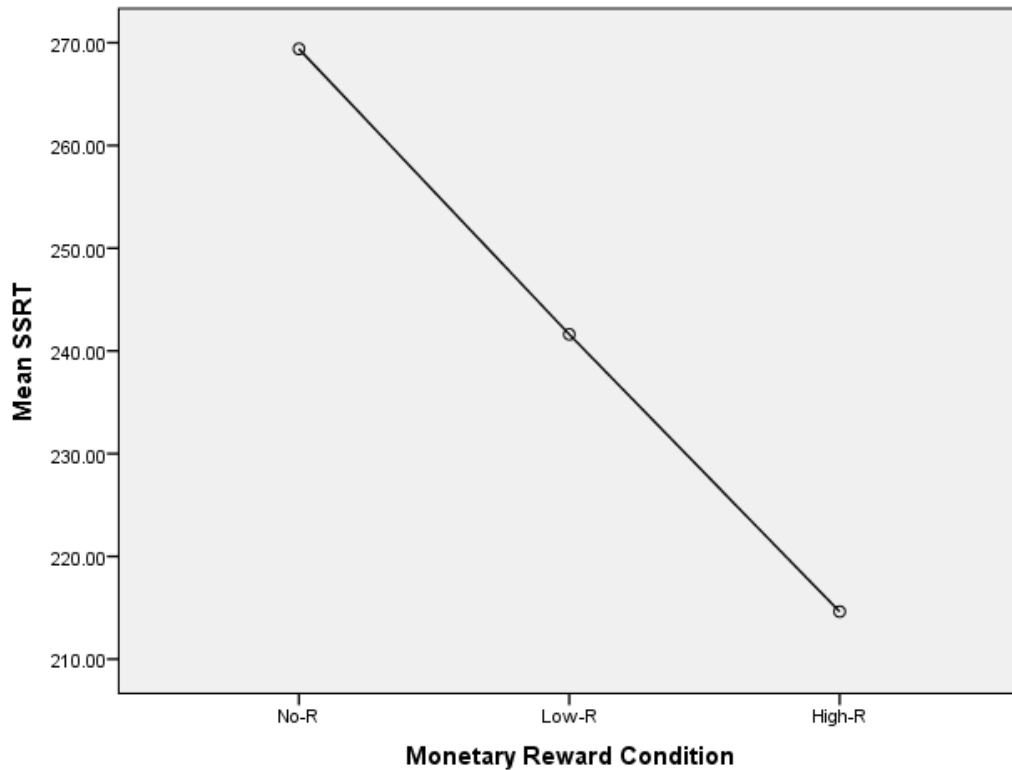
Figure 4. Interaction of Reward and Information on SSRT during Session-2.

There was a significant interaction between the monetary reward condition and the information condition.

To further investigate the MANVOA findings, data were analyzed using two 3x2x2 mixed ANOVAs, one for each dependent variable - PRS and SSRT. A comparison of the ADHD group and the non-ADHD control group yielded no interaction effects, in either dependent measure (PRS or SSRT). No main effects were found when investigating PRS scores. With regards to SSRT, main effects of information ( $F(1, 87) = 7.47, p < .01, \eta p^2 = .08, \text{power} = .77$ ) and reward condition ( $F(2, 87) = 6.64, p < .01, \text{partial } \eta p^2 = .13, \text{power} = .90$ ) were found. Post hoc analysis (Tukey's HSD) found significant differences between the No-reward and High-reward

condition ( $p < .01$ ). Group (ADHD or non-ADHD) did not have a main effect.

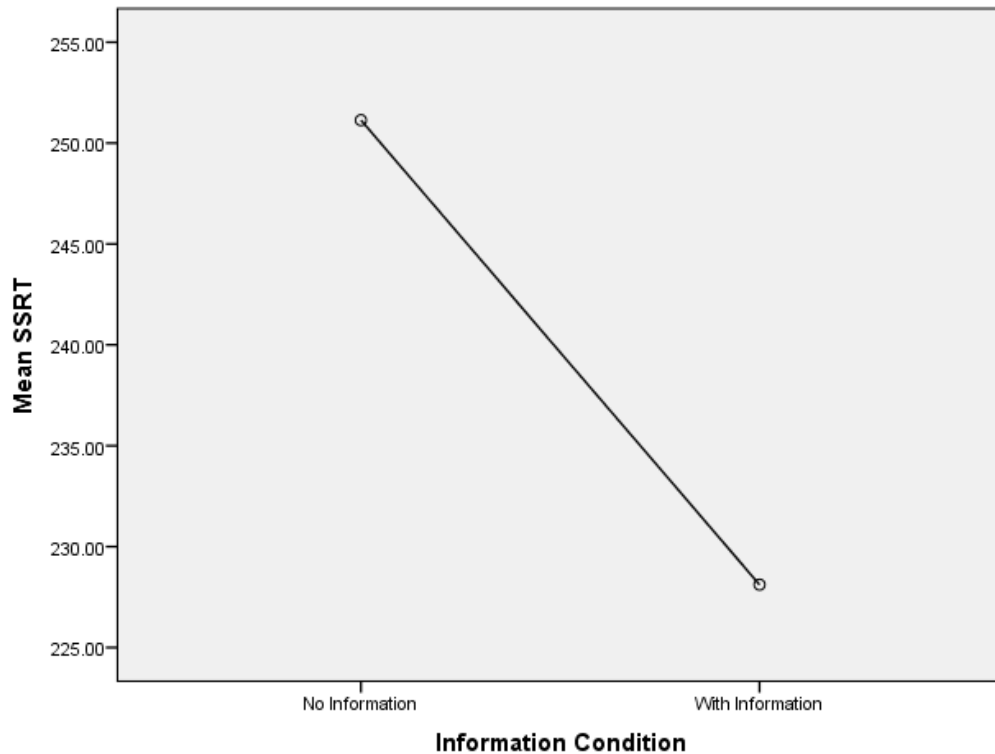
Four separate 3x2 ANOVAs were conducted to investigate how the experimental manipulations affected each group of participants. The data from the ADHD group (Table 1), show that monetary rewards affected SSRT scores,  $F(2, 43) = 5.89, p < .01$ , partial  $\eta^2 = .22$ , power = .85, but not PRS scores. The presence of performance information did not affect either dependent measure. Post hoc analysis (Tukey's HSD) found significant differences between the Low-reward and High-reward conditions within the ADHD group ( $p < .01$ ). Figure 5 illustrates this main effect.



*Figure 5.* Main Effect of Monetary Rewards in the ADHD Group

There was a main effect of monetary reward for the ADHD group, such that higher rewards resulted in lower SSRT scores.

The data from the non-ADHD/control group (Table 2) show that performance information did not affect PRS scores but did affect SSRT,  $F(1,44)= 5.00$ ,  $p= .03$ , partial  $\eta^2 = .10$ , power= .59. Monetary rewards did not affect either dependent measure. Figure 6 illustrates the main effect of information.



*Figure 6.* Main Effect of Information in the Non-ADHD Control Group

There was a main effect of information in the control group, such that the presence of social comparison information improved SSRT scores.

Overall, there were no interactions (money x information) within either the ADHD group or within the non-ADHD control group for PRS or SSRT.



## CHAPTER 6

### Discussion

There were several unexpected findings in the current study. There was no significant effect of group, indicating that ADHD and control participants performed similarly on average. Participants in the ADHD group did show slightly slower SSRT's overall relative to controls, but their accuracy as measured by PRS was not significantly different. Several decades of research has found that individuals with ADHD perform worse relative to controls on the SST (for a review, see Alderson, Rapport, & Kofler, 2007). Specifically, individuals with ADHD show slower SSRT, indicating a longer latency between a "STOP" signal and successful response inhibition. This was not the case in the current study. Neither the social comparison manipulation nor the monetary condition significantly affected the inhibitory accuracy (PRS) for either group. It was found, however, that higher monetary rewards generally improved performance in the ADHD, which was expected. Additionally, social comparison information improved performance in the control group. There was also a significant interaction effect of monetary reward condition and information for the SSRT measure when analyzed by MANOVA, but the effect disappeared when investigated with smaller analyses (ANOVA both between and within groups). The lack of improvement in inhibitory accuracy (PRS) for both groups, as well as the lack of significant differences between the ADHD and control groups, could have been influenced by certain limiting factors:

#### **Ceiling Effects**

There was some evidence that a ceiling effect could have impacted the within-subjects (social comparison information) outcome. According to Logan & Cowan (1984) and Verbruggen et al., (2008), the dynamic adjustment of the SSD generally results in a successful inhibition rate

of 50%. More successful inhibition trials results in slower SSD latency. It theoretically should be more difficult to successfully inhibit a response if the participant successfully inhibits previous responses. Both the ADHD and control group experienced successful inhibition on average 44-48% of trials, which is significantly higher than the theorized percentage. Given the dynamic nature of the SST, it was improbable that participants who performed this well on the first session would be able to improve their performance regardless of manipulations. This could explain the lack of significant differences between the ADHD and control groups, which one would expect to see.

### **The ADHD Sample**

The characteristics of the individuals in the ADHD group could have contributed to the similarities in the data between the ADHD and control groups. It was difficult to recruit a sufficient number of individuals who had been previously diagnosed with ADHD. This necessitated that individuals with ADHD symptoms also be included to ensure statistical power. The lack of statistical differentiation between the ADHD and control group could be due to the mixed ADHD sample. However, the SST has rarely been investigated using adults with ADHD, as the bulk of the literature typically uses children. It is possible that adults with ADHD symptoms do not differ much from other typical adults, unlike children with ADHD who do significantly differ from their typical counterparts.

Finally, there were some minor limitations to the study that could have influenced the outcome. There was variance in how long participants waited between the first and second session (typical time was between 24-72 hours, though some had a week or more in between). Additionally, some participants were not able to use the same room for both sessions due to laboratory circumstances. It is doubtful that these minor limitations had a significant impact on

the study, though in the future all care should be taken to correct these missteps.

## **Implications**

As predicted, tangible extrinsic rewards (such as money) improve performance in individuals with ADHD. Specifically, higher rewards resulted in faster response inhibition for the participants in the ADHD group. While the rewards did not necessarily increase the participants' inhibitory accuracy (PRS), they did decrease the amount of time it took to inhibit their responses (SSRT). This finding relates back to the race model of inhibition from Logan and Cowan (1984). Response inhibition can be thought to "race" with response execution. If the response times for the response execution are faster than the response times for the response inhibition, then the resulting action will be a response execution. Thus, lower SSRT scores indicate that the response inhibition process was generally faster than the response execution process. Surprisingly, the control group did not show the same improvement based on condition as the ADHD group. One possible reason for this could stem from confusion about the participant actually receiving the monetary reward. Although it was made clear in the instructions before completion of the study that the participant would be paid any rewards at the conclusion of the study, some expressed surprise when the monetary reward was actually presented to them. If the majority of the individuals in the control condition did not actually expect a reward, then the reward may not have influenced their performance.

The social comparison manipulation improved performance in the non-ADHD (control) sample. Specifically, receiving information about other participants' performances seemed to improve their speed of response inhibition. This shows that social comparison information can improve inhibitory control, although it did not seem to help the participants in the ADHD group. In some cases, performance actually declined in the social comparison (i.e. information) session.

This could reflect a fundamental difference in the way that social comparison information is used by individuals with ADHD compared to those without any symptoms of ADHD. Very little has been done to show how social comparison affects adults with ADHD, which makes this a very important field of study. Social comparison is a ubiquitous part of human behavior and observing the process in populations with psychopathologies can be both interesting and impactful. In the current study, the upwards comparisons typically ranged from an 8-12% score increase, with the comparison always set at five points above the participant's score in the first session. This may have been insufficient as a motivating factor. It could have also negatively impacted the participant by increasing cognitive load. In fact, during debriefing several participants in the ADHD group reported being negatively affected by knowing how others had performed on the task.

In the future, it would be beneficial to manipulate the level of upwards comparison, perhaps in the same way that reward conditions were manipulated in this study. For example, participants could be assigned to one of several groups that differ based on the gap between the participant's score and the comparison score such as No-difference (no comparison), 2-more (low-comparison), 5-more (high-comparison), etc. Investigating performance improvement in each condition in both an ADHD and a non-ADHD (control) population could yield insight into any existing differences in how social comparison is used, as well as how it affects performance.

Finally, it would be interesting to use non-linear methods such as those in the chaotic intermittency model to examine these phenomena. In particular, time series analysis would allow for the use of a smaller sample, as well as investigate the data trends that would not be apparent when relying on standard inferential statistics. This type of method would allow a researcher to investigate individual differences within the sample, as well as look at the trends as a whole.

Future investigators could also utilize different extrinsic rewards to improve inhibitory control, such as social rewards, like praise or other types of affirmation. Finding ways to improve inhibitory control could improve performance on behavioral tasks, particularly in classroom settings, so the literature should be expanded to include non-monetary rewards.

## **Conclusion**

In summary, monetary rewards had the greatest effect on inhibitory control in the ADHD group, specifically improving the speed of response inhibition (SSRT). Social comparison did not significantly improve the speed of response inhibition in the ADHD group, and in fact seemed to hurt accuracy (PRS). Accuracy involves the number of inhibitory responses that an individual was able to successfully complete. In a naturalistic setting, this may include the number of times that an individual inhibits the urge to interrupt a coworker or is able to ignore interference when attempting to complete a task. However, the social comparison manipulation did improve the speed of response inhibition for the control group. Overall, the ADHD and control groups performed similarly. Even with these findings, the literature is fairly established when it comes to inhibitory control deficits in individuals with ADHD. They are prominent in both children and adults and finding ways to improve these deficits is of paramount importance when it comes to improving their quality of life. It could be that traditional linear statistics do not tell the whole story. By using non-linear statistics, such as a time series analysis, a researcher could begin to pick apart the individual nuances of performance and ADHD symptoms. Understanding the ways that individuals with ADHD utilize social comparison information is also of importance moving forward. As a very important social process, social comparison affects the ways that these individuals interact with the world around them. Finally, developing non-pharmacological interventions could provide a valuable alternative towards helping

individuals who may be unable to use psychotropic medications. There is much more to explore in regards to this disorder. It will be important for future researchers to recognize our gaps in knowledge, and to have the insight and desire to fill them.

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## Appendix A

### Adult ADHD Self-Report Scale v1.1

#### **Adult ADHD Self-Report Scale (ASRS-v1.1) Symptom Checklist - Instructions**

*The questions on the back page are designed to stimulate dialogue between you and your patients and to help confirm if they may be suffering from the symptoms of attention-deficit/hyperactivity disorder (ADHD).*

Description: The Symptom Checklist is an instrument consisting of the eighteen DSM-IV-TR criteria. Six of the eighteen questions were found to be the most predictive of symptoms consistent with ADHD. These six questions are the basis for the ASRS v1.1 Screener and are also Part A of the Symptom Checklist. Part B of the Symptom Checklist contains the remaining twelve questions.

#### **Instructions:**

##### **Symptoms**

1. Ask the patient to complete both Part A and Part B of the Symptom Checklist by marking an X in the box that most closely represents the frequency of occurrence of each of the symptoms.
2. Score Part A. If four or more marks appear in the darkly shaded boxes within Part A then the patient has symptoms highly consistent with ADHD in adults and further investigation is warranted.
3. The frequency scores on Part B provide additional cues and can serve as further probes into the patient's symptoms. Pay particular attention to marks appearing in the dark shaded boxes. The frequency-based response is more sensitive with certain questions. No total score or diagnostic likelihood is utilized for the twelve questions. It has been found that the six questions in Part A are the most predictive of the disorder and are best for use as a screening instrument.

##### **Impairments**

1. Review the entire Symptom Checklist with your patients and evaluate the level of impairment associated with the symptom.
2. Consider work/school, social and family settings.

- Symptom frequency is often associated with symptom severity, therefore the Symptom Checklist may also aid in the assessment of impairments. If your patients have frequent symptoms, you may want to ask them to describe how these problems have affected the ability to work, take care of things at home, or get along with other people such as their spouse/significant other.

**History**

Assess the presence of these symptoms or similar symptoms in childhood. Adults who have ADHD need not have been formally diagnosed in childhood. In evaluating a patient’s history, look for evidence of early-appearing and long-standing problems with attention or self-control. Some significant symptoms should have been present in childhood, but full symptomology is not necessary.

**Adult ADHD Self-Report Scale (ASRS-v1.1) Symptom Checklist**

Patient Name: \_\_\_\_\_ Today’s Date: \_\_\_\_\_

Please answer the questions below, rating yourself on each of the criteria shown using the scale on the right side of the page. As you answer each question, place an X in the box that best describes how you have felt and conducted yourself over the past 6 months. Please give this completed checklist to your healthcare professional to discuss during today’s appointment.	Never	Rarely	Sometimes	Often	Very Often
1. How often do you have trouble wrapping up the final details of a project, once the challenging parts have been done?					
2. How often do you have difficulty getting things in order when you have to do a task that requires organization?					
3. How often do you have problems remembering appointments or obligations?					
4. When you have a task that requires a lot of thought, how often do you avoid or delay getting started?					
5. How often do you fidget or squirm with your hands or feet when you have to sit down for a long time?					
6. How often do you feel overly active and compelled to do things, like you were driven by a motor?					
7. How often do you make careless mistakes when you have to work on a boring or difficult project?					

8. How often do you have difficulty keeping your attention when you are doing boring or repetitive work?					
9. How often do you have difficulty concentrating on what people say to you, even when they are speaking to you directly?					
10. How often do you misplace or have difficulty finding things at home or at work?					
11. How often are you distracted by activity or noise around you?					
12. How often do you leave your seat in meetings or other situations in which you are expected to remain seated?					
13. How often do you feel restless or fidgety?					
14. How often do you have difficulty unwinding and relaxing when you have time to yourself?					
15. How often do you find yourself talking too much when you are in social situations?					
16. When you're in a conversation, how often do you find yourself finishing the sentences of the people you are talking to, before they can finish them themselves?					
17. How often do you have difficulty waiting your turn in situations when turn taking is required?					
18. How often do you interrupt others when they are busy?					

### **The Value of Screening for Adults With ADHD**

Research suggests that the symptoms of ADHD can persist into adulthood, having a significant impact on the relationships, careers, and even the personal safety of your patients who may suffer from it.<sup>1-4</sup> Because this disorder is often misunderstood, many people who have it do not receive appropriate treatment and, as a result, may never reach their full potential. Part of the problem is that it can be difficult to diagnose, particularly in adults.

The Adult ADHD Self-Report Scale (ASRS-v1.1) Symptom Checklist was developed in conjunction with the World Health Organization (WHO), and the Workgroup on Adult ADHD that included the following team of psychiatrists and researchers:

- **Lenard Adler, MD**  
Associate Professor of Psychiatry and Neurology  
New York University Medical School

- **Ronald C. Kessler, PhD**  
Professor, Department of Health Care Policy  
Harvard Medical School
- **Thomas Spencer, MD** Associate  
Professor of Psychiatry Harvard  
Medical School

As a healthcare professional, you can use the ASRS v1.1 as a tool to help screen for ADHD in adult patients. Insights gained through this screening may suggest the need for a more in-depth clinician interview. The questions in the ASRS v1.1 are consistent with DSM-IV criteria and address the manifestations of ADHD symptoms in adults. Content of the questionnaire also reflects the importance that DSM-IV places on symptoms, impairments, and history for a correct diagnosis.<sup>4</sup>

The checklist takes about 5 minutes to complete and can provide information that is critical to supplement the diagnostic process.

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1. Schweitzer JB, et al. *Med Clin North Am.* 2001;85(3):10-11, 757-777.
2. Barkley RA. *Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment.* 2nd ed. 1998.
3. Biederman J, et al. *Am J Psychiatry.* 1993;150:1792-1798.
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Appendix B  
Behavioral Health Screening Measure

**BHSM**

**NAME** \_\_\_\_\_ **DATE** \_\_\_\_\_

The following statements refer to experiences that many people have in their everyday lives. Circle the number that best describes **HOW MUCH** that experience has **DISTRESSED or BOTHERED** you during the **PAST TWO WEEKS**. The numbers refer to the following verbal labels:

0	1	2	3	4
None of the time	A little of the time	Some of the time	Most of the time	All of the time

1.	Emotionally, I am not doing very well.	0	1	2	3	4
2.	I am <i>not</i> satisfied with my life.	0	1	2	3	4
3.	I feel unhappy, sad, or depressed.	0	1	2	3	4
4.	I am less interested in things I used to enjoy.	0	1	2	3	4
5.	I have thoughts of ending my life.	0	1	2	3	4
6.	I feel fearful, nervous, or anxious without knowing why.	0	1	2	3	4
7.	I cannot relax.	0	1	2	3	4
8.	I am afraid that something bad is going to happen.	0	1	2	3	4
9.	I have tried to cut down on my drinking or drug use.	0	1	2	3	4
10.	I feel unhappy or guilty about my drinking or drug use.	0	1	2	3	4
11.	I have been criticized for my drinking or drug use.	0	1	2	3	4
12.	I feel afraid of someone else.	0	1	2	3	4
13.	I am easily irritated or annoyed.	0	1	2	3	4
14.	I feel out of control of my anger.	0	1	2	3	4

15.	I have rapid or strong mood swings.	0	1	2	3	4
16.	I have racing thoughts.	0	1	2	3	4
17.	I have thoughts or ideas that others might find unusual or odd.	0	1	2	3	4
18.	I am worried that there is something wrong with my mind.	0	1	2	3	4
19.	I go to extreme measures to avoid gaining weight.	0	1	2	3	4
20.	I cannot control how much I eat.	0	1	2	3	4
21.	Emotional problems or difficulties interfere with my relationships with friends.	0	1	2	3	4
22.	Emotional problems or difficulties interfere with my ability to accomplish as much as usual at work or school.	0	1	2	3	4

### **Behavioral Health Screening Measure (BHSM)**

#### Administration & Scoring

The BHSM is a screening instrument for detecting possible emotional problems in adults. It is a 22-item measure that covers many symptoms of mental illness. Respondents rate how much the statements fit them on a 5-point Likert scale.

Scores are generated by adding the item scores. The possible range of scores is 0-88. Mean score for patients is 26.31 ( $SD = 15.31$ ). Recommended cutoff score is 16, with scores equal or above that indicating possible mental health difficulty.

Reference:

Zygowicz, K.M., & Saunders, S.M. (2003). A behavioral health screening measure for use with young adults in primary care settings. *Journal of Clinical Psychology in Medical Settings*, 10 (2), 71-77.