IDENTIFICATION OF DIFFERENTIAL PREDICTORS OF ADHD SUBTYPES USING LABORATORY PROCEDURES

By

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To the Faculty of Washington State University:

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IDENTIFICATION OF DIFFERENTIAL PREDICTORS OF ADHD SUBTYPES USING LABORATORY PROCEDURES

ABSTRACT

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A laboratory task shown to differentiate the behavior of hyperactive (Spontaneously Hypertensive rats) and non-hyperactive rats (Wistar-Kyoto rats) was adapted for use with humans. The task allowed for exploring a Dynamic Developmental Theory (DDT) hypothesis (Sagvolden, Johansen, Aase, & Russell, 2005) that responding to fixed interval (FI) reinforcement schedules should differ as a function of ADHD symptomatology (Johansen, Killeen, & Sagvolden, 2007). The translation of the paradigm to humans utilized a computer task that generated reinforcement conditions specified by the DDT and similar to those used with rats. The participants were 152 undergraduates above the age of 18 who completed the computer task, a screening questionnaire, and a widely used ADHD self-report form. Hypotheses were formulated from the DDT concerning differential responding on the computer task as a function of ADHD symptomatology and were tested with respect to mean comparisons between a Hyperactive Impulsive (HI) group, an Inattentive (I) group, and a control group characterized by low HI and I scores. In addition, correlational procedures were used to assess how the two primary symptom dimensions of ADHD (hyperactive-impulsive and inattentive) related to computer task performance. The dependent variables, derived from behavior on the computer
task, included button sampling (i.e., frequency of response and total clicks per trial), response variability (i.e., entropy), and rate of learning (i.e., beta). The results of correlational analyses showed a significant association between ADHD-I symptomatology and button sampling behavior, and no other significant findings. The results of a chi-square analysis revealed a significant difference for button sampling for both ADHD-I and for ADHD-HI compared to control participants. ANOVA and t-test results revealed no differences across groups for any of the dependent variables. The results are discussed with respect to (a) the usefulness of the computer task as a diagnostic tool, and (b) the fact that the FI schedule performance of humans may relate more strongly to Inattentive symptoms than it does to Hyperactive/Impulsive symptoms.
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CHAPTER ONE

INTRODUCTION

ADHD is a diagnostic label used to describe behaviors characterized by inattention, hyperactivity, and impulsivity leading to impairment in at least two of one’s social, academic, and occupational domains (American Psychiatric Association, 2000). ADHD is divided into three general categories, two of which are subtypes characterized by distinct symptom profiles, and a third that represents a combination of the symptoms (American Psychiatric Association, 2000). The three general categories are ADHD-Hyperactive-Impulsive type, ADHD-Inattentive type, and a third combined type (American Psychiatric Association, 2000). Demographically, ADHD shows a 3 – 7% prevalence rate for school-age children (American Psychiatric Association, 2000) with estimates indicating that for every afflicted female, there are between three to nine afflicted males (Bruchmuller, Margraf, & Schneider, 2011; Chhabildas, Pennington, & Willcutt, 2001). These statistics are, however, highly controversial and, while it is generally accepted that there is at least a 3:1 male-to-female gender ratio, some contend that higher ratios may instead reflect therapist biases leading to overdiagnosis of males (Bruchmuller et al., 2011).

The first subtype, ADHD-Hyperactive-Impulsive (ADHD-HI) type is characterized by inappropriately high activity levels, sometimes described as “driven by a motor” (American Psychiatric Association, 2000; Barkley & Murphy, 2005). Males account for the majority of ADHD-HI diagnoses and are more frequently diagnosed with comorbid externalizing disorders characterized by oppositional, defiant, and aggressive behavior (Milich, Balentine, & Lynam,
In turn, externalizing disorders contribute to increased risk of developing Oppositional Defiant Disorder (Burns & Walsh, 2002).

The second subtype, ADHD-Inattentive (ADHD-I) type is characterized by marked difficulty maintaining focus on a primary task or thought process that often results in repeated attention shifts and consequent failure to complete tasks (American Psychiatric Association, 2000). Less is known about this disorder, and this will be a topic of discussion later in this paper. What is known, however, is that girls are more likely to be diagnosed with this disorder than are boys (Gross-Tsur et al., 2006). Also, the disorder is associated with self-consciousness, passivity, and somatization, and may be more highly correlated with internalizing disorders such as anxiety and depressive disorders (Diamond, 2005; Quay, 1997; Gross-Tsur et al., 2006; Milich et al., 2001). The disorder is significantly less likely than ADHD-HI to be associated with aggression or social impairment (Milich et al., 2001; Quay, 1997).

ADHD-Combined (ADHD-C) type is characterized by an elevation in both inattention and hyperactivity where the individual demonstrates clinically significant elevation of six or more symptoms for each of ADHD-HI and ADHD-I (American Psychiatric Association, 2000).

The developmental course of ADHD and its subtypes is interesting and controversial (Chhabildas et al., 2001). For instance, research illustrates that the symptom profiles of individuals with ADHD differ over time (Biederman, Mick, & Faraone, 2000; Chhabildas et al., 2001). This is especially true for those diagnosed with ADHD-HI. Review of the literature on the developmental course of ADHD illustrates that hyperactive and impulsive symptoms decrease over time such that by age twenty there has occurred a significant remission in symptoms (Biederman et al., 2000; Malloy-Diniz, Fuentes, Borges Leite, Correa, & Bechara, 2007). As a result, the distinctions between the two subtypes become less pronounced as
childhood gives way to adolescence and adolescence gives way to adulthood (Malloy-Diniz et al., 2007). In fact, it appears that while the ADHD-HI symptoms become more moderate over time, I symptoms become increasingly impaired, such that as teens these individuals may qualify for combined type, then in adulthood only as inattentive type.

Despite this developmental trajectory, most theorists believe that the HI – I distinction is valid and reflects different underlying neuropsychological deficits. For example, Diamond (2005) has argued that ADHD-HI and ADHD-I are profoundly different disorders. According to Diamond (2005) and others (see Milich et al., 2001), our traditional method for categorizing the two disorders is mistaken. Traditionally, categorization is based on weighing the extent to which HI or I symptoms dominate the clinical picture. Diamond (2005) has suggested an alternative view of this distinction in which I symptoms naturally manifest in individuals with the HI type, but the converse is not true. That is, ADHD-HI symptoms do not manifest in individuals with the ADHD-I type (Diamond, 2005). Therefore, she proposed that making an accurate diagnosis of ADHD-I involves classifying as separate those individuals who manifest high or even moderate I symptoms and very low rates of HI symptoms (Diamond, 2005). According to her, individuals with moderate or high HI symptoms, regardless of the number of I symptoms, have a different disorder than those currently classified as ADHD-I (Diamond, 2005). Theories suggesting a developmental trajectory toward increasingly inattentive symptoms and theories suggesting an independent and truly inattentive disorder are therefore not necessarily mutually incompatible, and according to Diamond (2005), ADHD-HI and ADHD-C symptom profiles reflect the same underlying disorder, and should be distinguished from individuals who display only I symptoms.
Diamond’s (2005) conceptualization of ADHD-HI and ADHD-I as distinct disorders is consistent with field trials prior to release of DSM-IV. Preceding release of DSM-IV, trials to determine optimal psychometrics for ADHD-I revealed several items that predicted inattention associated with hyperactivity, but which failed to isolate inattention unassociated with hyperactivity (Hinshaw, 2001). DSM-IV effectively ignored potential qualitative differences between inattention that occurs in isolation and inattention that occurs together with hyperactive-impulsive symptoms (Hinshaw, 2001). For example, Mitchell (2010) described a motivational dysfunction pathway particularly relevant in the etiology of hyperactive-impulsive symptoms. Specifically, Mitchell (2010) presented research that describes high behavioral approach and dysfunctional reinforcement processes as predictive of hyperactive-impulsive ADHD symptoms. This effect remained after controlling for comorbid conduct disorder and psychopathy symptoms (Mitchell, 2010). Inattention may result as a byproduct of the low inhibition and hyperactivity characteristic of a high behavioral approach, yet inattention occurring in the absence of hyperactivity would not fit within an ADHD classification (Diamond, 2005). A truly inattentive subtype lacking hyperactive symptoms would therefore be considered the result of working memory deficits and an entirely independent diagnosis from that of ADHD-HI or ADHD-C (Diamond, 2005).

In addition to potentially distinct biological and cognitive origins of ADHD-I (Diamond, 2005; see Neurobiological Foundations of ADHD), Milich et al. (2001) commented on behavioral discrepancies and identified distinct symptoms that are both characteristic of the inattentive subtype and discordant with response inhibition and other traits inherent to the ADHD-HI and ADHD-C subtypes (Barkley, 2001). Barkley (2001) explained that while problems with response inhibition typify ADHD-HI and ADHD-C, it is relatively absent in
ADHD-I, and due to maintaining inhibitory control, the inattentive group does not display the marked tendency to develop comorbidities such as conduct disorder and oppositional defiant disorder. In other words, not only does the hyperactive-impulsive subtype have a higher behavioral approach that is independent of comorbidities, but they also show a markedly increased development of these comorbidities (Barkley, 2001; Diamond, 2005). In addition to the poor fit of the inattentive subtype to prevailing functional impairments present in ADHD-HI and ADHD-C (e.g., response disinhibition), characteristic symptoms of the inattentive domain are similarly exclusive. Catania (2005) describes inattention as associated with “profound” learning difficulties that include attention deficit and difficulty learning adaptive behavioral patterns. Given neuropsychological evidence that attention is multifaceted, Barkley (2001) argued that it should be possible to identify “qualitatively distinct disorders of attention.” It follows that any inattention that develops due to response disinhibition may indeed characterize and be consistent with the hyperactive-impulsive symptoms of ADHD, yet be qualitatively different from the potentially multitudinous inattentive symptoms evidenced in what the DSM-IV-TR (American Psychiatric Association, 2000) currently describes as ADHD-I.

Theories that argue that predominantly inattentive type is an entirely distinct disorder from ADHD-HI and ADHD-C (Barkley, 2001; Diamond, 2005; Milich et al., 2001; Mitchell, 2010) are disputed due to indications that the inattentive group may be split into a subthreshold combined group and a cognitively sluggish group where only the cognitively sluggish group qualify as non-ADHD (Hinshaw, 2001). While arguing that neuropsychological evidence does not yet definitively distinguish between the subtypes, Hinshaw (2001) added to Barkley’s (2001) chorus regarding the need for improved phenotype differentiation, explaining that until that time, the ADHD-I group will continue to be split, thus confounding both the ADHD-C and ADHD-I
subgroups. In an effort to reduce this confound, Diamond (2005) offered an alternative mechanism by which to subdivide the groups, focusing on the hyperactive symptom set rather than the inattentive domain. As explained, if inattention develops in the presence of high levels of hyperactive symptoms, then that inattention is likely a side effect of faulty response inhibition processes rather than a consequence of a conversely underactive, overly inhibited, sluggish cognitive tempo. By this argument, individuals with high levels of hyperactivity with or without comorbid inattention would qualify as ADHD-HI or ADHD-C. Another way of understanding this is through Catania’s (2005) argument that behavioral patterns characterized by hyperactivity-impulsivity are so because of discrepant reinforcement; the fact that these maladaptive patterns can be reinforced at all indicates a less severe pathology than one characterized entirely by inattention wherein behavioral patterns are neither strengthened nor extinguished, thus indicating that the presence of hyperactivity, not of inattention, is the arbiter of whether an individual is categorized as ADHD-HI/C. By contrast, for individuals with low levels of hyperactivity, inattention would not be expected to develop as a result of their hyperactivity, and thus high inattention in these individuals would be considered non-ADHD in origin (Diamond, 2005). Hinshaw (2001) explained that this latter group might constitute a sluggish cognitive tempo group while the former group would capture individuals for whom inattention is believed to derive from hyperactive-impulsive association. This subdivision according to relative differences in hyperactive-impulsive and inattentive symptoms would allow for nuanced differentiation based on presumably differing precipitating factors rather than on neuropsychologically similar displays of inattention. While certainly experimental, this approach circumvents current limitations in neuropsychological understanding of inattentiveness and provides a scientifically founded mechanism by which to divide participants with features of
inattention into two subsets differentiated by either very low or significantly elevated hyperactive-impulsive scores. This approach thus addresses the problem of having subthreshold ADHD-C individuals classified instead as ADHD-I. Given that no other alterations to current diagnostic criteria are indicated, research based on these sorting principles ought to either clearly delimit groups or refute this approach by increasing homogeneity across subtypes.
Animal Models

Thus far I have discussed what we know about the manifestation of ADHD in humans and I have reviewed studies that include humans as research participants. There exists also a literature that seeks to understand the basic mechanisms of ADHD using animals. A major milestone in this research was reached when researchers identified the spontaneously hypertensive rat (SHR), a behaviorally hyperactive rodent which many came to accept as an animal analog for ADHD (Hunziker, Saldana, & Neuringer, 1996; McCarty, Chiuheh, & Kopin, 1978; Myers, Musty, & Hendley, 1982; Sagvolden et al., 1992; Wultz, Sagvolden, Moser, & Moser, 1990). Originally inbred from Wistar-Kyoto rats to facilitate blood pressure research (Okamoto & Aoki, 1963), the SHR rodent was adopted by some researchers as a mechanism for studying ADHD due to multiple studies demonstrating SHR behavioral hyperactivity (McCarty & Kopin, 1979; Myers, Musty, & Hendley, 1982; Sagvolden et al., 1992; Wultz et al., 1990).

This dissertation has focused on one body of research which accepted as accurate the neurobiological assertions of Sagvolden, Johansen, Aase, and Russell (2005) and aimed to use a fixed interval (FI) reinforcement schedule to differentiate behavioral responses of SHR, or ADHD-HI, rodents and controls (Johansen, Killeen, & Sagvolden, 2007).

Founded on the premise that WKY and SHR rodents represent physically and behaviorally distinct phenotypes, eight rodents of each type were selected to respectively represent controls and ADHD rodents. The rats were not medicated, but were deprived of water such that they were intrinsically motivated to attain water that could thus function as a reinforcer. Rats were placed individually into an operant chamber which contained a light, an empty water dish, and a wall of twenty holes into which the rats could poke their noses. The following figure
(Figure 1) reproduced from Johansen et al. (2007) displays the placement of stimuli and reinforcement.

Figure 1. Rodent study operant chamber wall

As evident through the above graphical representation, nose-pokes into 19 of the 20 holes elicited sound, light flickering, or no stimulus while a nose-poke into the target hole resulted in water delivery to the previously empty water dish according to an FI reinforcement schedule. FI schedules are believed to be a good method for capturing ADHD-HI behavioral tendencies because reinforcement relies on the subject’s ability to identify behavior that will lead to gratification and then to wait through a fixed period in order to receive a reward (Johansen et al., 2007).

Over the course of four trials, rats were habituated to the chamber; during these four trials, all holes were covered and water was disbursed on a random time (RT) schedule. The goal of these trials was for the rats to learn to collect reinforcement when it was made available. Following these trials, the rats’ behavior was hand-shaped by experimenters who aided the rats in finding the target hole. The rats were subsequently subject to 54 FI trials. The FI time intervals increased across trials from 1-second intervals for trials 1 – 6 to 300-second intervals for trials 17 – 54.
Data analysis centered on calculation of total nose-pokes per hole and the time at which each poke occurred. These values provided an overview of total activity and the response variability (i.e., entropy) of the rats’ poking behavior. Analyses compared mean nose-pokes and variability (i.e., entropy) by group across all FI trials as well as the learning curves and delay gradients for each group across the first six trials. Johansen et al. (2007) reported differences in the SHR and WKY behavior, with the SHR rats demonstrating more nose-pokes and higher variability (i.e., entropy) across all FI trials compared to the WKY rats. Rates of learning also differed by group. Total nose-pokes, variability of response (i.e., entropy), and rates of learning calculations were based on mathematical formulas from which basic numerical differences were identified. Relative statistical analysis was not conducted except for a chi-square comparison of the total number of nose-pokes per group across the first six FI trials. This paper also did not report all numerical results and it was therefore not possible to statistically evaluate the original findings. As such, for translational purposes, the primary limitation of the Johansen et al. (2007) study is the exclusion of statistical analyses to statistically validate the reported numerical differences. Another important limitation is that the study did not differentiate an inattentive subgroup; rather, the SHR group is considered to represent the global features of the current DSM-IV diagnostic features of ADHD, namely hyperactivity, impulsivity, and inattention (Johansen et al., 2007). This provides for comparison of global ADHD symptoms to controls, but does not provide data to aid differentiation of the ADHD-subtypes.
Measurable Deficits and Key Experimental Variables in ADHD

The goal of this section is to expand upon terms commonly used in human and animal ADHD research, and which are critical to the primary theories of ADHD. Such a focus reflects a belief that progress in ADHD research is most likely to come from the interface between animals and humans, as is the case with biomedical research.

Deficient Learning Processes and Windows of Reinforcement

Reinforcement-based theories of ADHD explain that following the onset of a stimulus, a window of time exists during which an introduced consequence can become associated with the stimulus (Johansen et al., 2009). This window is represented by a delay-of-reinforcement gradient that represents a function of events in time (Johansen et al., 2009). Small window size is causally and reciprocally related to delay gradients and delay aversion that impede higher-order operant conditioning and lead to incomplete learning of task chains. Some theories identify this as a defining behavioral feature of ADHD.

Catania (2005) described delay gradients as patterns of multiple actions summing to a more significant reinforcement value than that which would have been produced through reinforcement of any one action; earlier actions receive less reinforcement than later actions. Sagvolden et al. (2005) proposed a steepened delay gradient in ADHD, wherein behaviors early in a chain do not become associated with reinforcement, while later behaviors that occur in rapid succession are disproportionately reinforced. It follows that this reinforcement of rapid succession behaviors leads to development of hyperactivity (Catania 2005).

Delay aversion refers to actively seeking to shorten windows of reinforcement by choosing immediate rewards over greater, delayed rewards (Johansen et al., 2009; Antrop et al., 2006). In addition to greater difficulty delaying gratification, poor response to reinforcement...
often leads to failure to respond appropriately to discipline and behavioral expectations (Tripp & Wickens, 2007). Active avoidance of delay may thereby manifest socially as impulsivity and apparent lack of motivation. Antrop et al. (2006) described the stimulation-seeking behavior of ADHD children, particularly in situations of delayed reinforcement. The authors (Antrop et al., 2006) reported that this delay aversive behavior appeared to normalize when ADHD children were provided with a choice of either waiting for a higher reward while self-administering visual cartoon stimulation, or of taking a short delay award, but at the expense of no extra stimulation. Delay aversion and response disinhibition may be conceptualized as distinct, independent characteristics of ADHD (Solanto et al., 2001), or conversely, as effects of the interaction of neurobiological deficits and individual learning styles (Johansen et al., 2005).

**Response Inhibition and Response Disinhibition: Response Rate and Variability**

Response disinhibition, the negation of response inhibition, is defined by failing to inhibit responses (Sagvolden et al., 2005). Quay (1997) described response disinhibition as a biologically mediated, primary deficit in ADHD. Though failure to inhibit responses and response disinhibition may qualitatively appear similar, the former refers to a multidimensional sequence leading to failure of inhibition while the latter refers to a direct cognitive process leading to inability to inhibit. For this reason, Sagvolden et al. (2005) argued that “disinhibition” is misleading because the actual problem is one of excessive motor behavior and increased motor variability rather than one of problematic cognitive processes. This increased motoric behavior and limited inhibition may nonetheless masquerade as inattention of cognitive origin, thus blurring differentiation between sluggish cognitive tempo and ADHD-mediated inattentiveness. Quay (1997) specified that disobedience or lack of response to punishment is not a core deficit of ADHD, but rather that the core deficit is the disruption of chaining between conditioned stimuli
and resulting reinforcement (whether punishment or reward). Apparent lack of responsiveness to punishment may lead to a misinterpretation of the correlation between intentional disobedience and ADHD. Though this literature review supports perspectives describing multiple deficits rather than a core deficit, Quay’s (1997) research on response disinhibition provided important clarification about the stimulus-punishment response system in ADHD, supporting the core deficit hypothesis.

Response disinhibition is commonly tested using the stop-signal task that is administered by teaching subjects a stimulus-response pattern, and then introducing a sound that indicates that on a given trial, the learned response should not occur (Quay, 1997; Chhabildas et al., 2001). Normal subjects show ability to inhibit, and this is therefore a good method for testing response disinhibition. Though Quay’s (1997) support for the connection between an “underresponsive” behavioral inhibition system (BIS) and core deficits in ADHD inherently dismissed a multidimensional perspective, his explanation does offer a method of interpreting why ADHD-I is associated with better controlled inhibition than is ADHD-HI or ADHD-C. That is, Quay (1997) explained that comorbid anxiety, more common in ADHD-I than in either of the other subtypes, is associated with increased BIS activity, and therefore with better performance on tasks requiring inhibition. It therefore follows that ADHD-I children will demonstrate better response inhibition than ADHD-HI or ADHD-C children, and this suggests a different stimulus-response mechanism across subtypes. Moreover, Quay’s (1997) focus on the BIS emphasized poor reinforcement sensitivity, and it is this overactive behavioral approach coupled with low sensitivity to reinforcement that forms the foundation for multidimensional, reinforcement-based models (Mitchell, 2010).
One effect of disrupted stimulus-response task chains is the development of response variability, or inconsistent responding. Specifically, failure to reinforce complete task chains responsible for a given reinforcement leads to a conundrum wherein reinforcement may be inappropriately or disproportionately associated with certain stimuli at the expense of other contributing factors, thus leading to response variability wherein a given stimulus elicits inconsistent responses.

The consequence of inconsistent response patterns may be the development of ineffective responses to punishment. Catania (2005) described response variability following punishment. While undesired behavior may be discontinued immediately following punishment, this subsequent avoidance of punishable behavior does not lead to praise. Because the preferred behavior is none at all, the result is that the improved behavior is not acknowledged or reinforced. This means that the stimulus that previously elicited punishable behavior is not paired with lack of behavior, and consequently, the prior association is not extinguished, and the punishable action may reemerge (Catania, 2005), thus creating a situation of apparent response variability manifest as response disinhibition. To explain further, whereas the stop-signal task alters stimulus-response patterns through use of a new response (sound), in the case just outlined, the new response is actually a lack of response and is therefore not associated with anything concrete (like sound) which can be paired with the original stimulus. As a result, the subject in Catania’s (2005) punishment scenario does not learn to inhibit one behavior for a preferential other behavior (or lack thereof) and may therefore initially stop the behavior only to restart it, thus displaying response variability.
**Extinction**

Extinction refers to elimination of a response due to lack of reinforcement. Pruning refers to the successful elimination of responses as a result of exposure to extinction. Lack of pruning occurs to the extent that exposure to an extinction schedule fails to eliminate responses (Sagvolden et al., 2005). According to the DDT (Sagvolden et al., 2005), many of the symptoms that define hyperactivity stem from a failure of behaviors to extinguish properly for individuals with this disorder. As a result, individuals with ADHD develop, over time, an excess of behaviors. These are sometimes described as giving the sense of being “driven by a motor” (American Psychiatric Association, 2000) or as excessively busy. The DDT (Sagvolden et al., 2005) attributes these extended sequences of “extra” behavior to learning. The behavior of ADHD individuals is differentially affected by organism-environment interactions compared to non-ADHD individuals. Importantly, however, these learning differences are thought to derive from neurological differences. This is in contrast to some behavioral theories of ADHD that attribute the symptoms to a different learning environment.

Frequent reinforcement may compensate for situations in which an individual with a steepened delay gradient would otherwise fail to appropriately associate stimulus-response patterns. Controlling the frequency with which reinforcement is delivered may therefore aid appropriate pruning and behavioral selection in individuals with steepened delay gradients. Dopamine hypotheses (Sagvolden et al., 2005; Tripp & Wickens, 2007; Waldman et al., 1998) suggest neurobiological differences in extinction and reinforcement processes. The following section explores this relationship and, within this context, explains the apparent neurobiological causative relationship between reinforcement and extinction processes.
Neurobiological Foundations of ADHD

Neurological Impairment

Neuroimaging, electrophysiological, and behavioral evidence in both human and rat models provide evidence for the role of frontostriatal impairment in ADHD development, but the mechanism of impairment remains debated (Doyle et al., 2005). Neuropsychological risk factors are proposed to include decreased processing speed and inhibition and visual attention deficits, and family studies reveal a higher proportion of processing speed and inhibitory deficits among non-ADHD family members of persons with ADHD (Doyle et al., 2005). In addition to research that indicates that ADHD is highly heritable and genetically linked (McGough & McCracken, 2006), an integral part of the debate regarding ADHD development is whether impairments in attention may be due to sluggish cognitive tempo (SCT) rather than ADHD (Chhabildas et al., 2001; Diamond, 2005; Milich et al., 2001).

Potential differences in cognitive ability and IQ across subtypes lead to discussion of whether and how to control for and assess intelligence and neuropsychological status. For example, while it is theoretically possible that failure to control for IQ could obscure neuropsychological differences across subtypes, it is equally plausible that controlling for IQ may inadvertently alter performance variability specific to ADHD (Shanahan et al., 2006). Arguments abound both in favor of and opposing controlling for IQ, reading disorder, and other executive dysfunctions commonly associated with ADHD, yet no conclusive agreement has been reached among ADHD researchers (Shanahan et al., 2006).

Diamond (2005) argued that ADHD-I is equivalent to the dysexecutive syndrome described in adults with executive functioning problems, and further stated that ADHD-I without hyperactivity is inclusive of many of the traits of SCT. Diamond (2005) offered behavioral
support for these assertions through examples such as the common trajectory of ADHD-I and dysexecutive syndrome patients in which both tend to begin tasks well, but falter over time as the task becomes tedious or forgotten. As neurobiological support for distinct, separate pathways in ADHD and ADHD-I without hyperactivity, Diamond (2005) explained that hyperactivity is associated with damage to the striatum while damage to the prefrontal cortex is primarily associated with attentional problems. To further emphasize the relationship between brain region and symptoms associated with the ADHD subtypes, Sagvolden et al. (2005) specified that it is hypofunction of the mesolimbic dopamine branch which leads to altered reinforcement and extinction processes, hypofunction of the nigrostriatal dopamine branch which leads to impaired motor functions, and hypofunction of the mesocortical dopamine branch which leads to attentional problems. Though the exact mechanism of genetic influence remains uncertain, much evidence supports explanations wherein genetic influence leads to neurobiological dysfunction of decreased dopaminergic and noradrenergic system activity (Malloy-Diniz et al., 2007).

**Dopamine Hypotheses**

Recognition that dopamine system dysfunction is a significant factor in ADHD developed due to the response of ADHD symptoms to pharmacological treatments such as methylphenidate and dextroamphetamine which primarily affect dopamine activity (Moeller et al., 2001; Sagvolden et al., 2005; Waldman et al., 1998). Waldman et al. (1998) further explained that genes effecting dopaminergic function are highly implicated in ADHD because of known association between dopamine function and effects on motor activity and reward-seeking. Dopamine, the main catecholamine neuromodulator (Missale, Nash, Robinson, Jaber, & Garon, 1998) is usually carefully regulated, but in individuals with an underdeveloped prefrontal cortex, as occurs with ADHD, dopamine levels are not well regulated, and this leads to faulty signal
transmission (Sagvolden et al., 2005). While an underdeveloped prefrontal cortex and frontostriatal impairment are associated with all ADHD subtypes (Sagvolden et al., 2005; Doyle et al., 2005), Diamond (2005) explained that the DAT1 gene is believed to be more highly associated with ADHD while the DRD4 gene is associated more with ADHD-I without hyperactivity, and DAT1 is particularly active in the striatum while DRD4 is particularly active in the prefrontal cortex. According to Waldman et al. (1998), the association of DAT1 to hyperactive-impulsive symptoms gained credibility following human studies by Cook et al. (1995) and by Gill et al. (1997) as well as a mice study by Giros et al. (1996). Waldman et al. (1998) presented results from a study in which they sought to replicate and extend these earlier findings and, as hypothesized, found that DAT1 showed higher correlation to hyperactive-impulsive than to inattentive symptoms (Waldman et al., 1998). Importantly, this effect occurred regardless of ethnic background thus countering arguments that it is not dopamine but rather cultural variations in discipline that lead to development of ADHD symptoms (Aase, Meyer, & Sagvolden, 2006; Waldman et al., 1998).

**Dynamic Developmental Theory.** According to Sagvolden et al. (2005) and the Dynamic Developmental Theory (DDT), in a normal brain, following reinforcement for a novel behavior or situation, a burst of dopamine activity occurs which is associated with the stimulus perceived to be predictive of reinforcement. By contrast, stable-state behavior results in no change to dopamine activity, and the lack of a predicted reinforcement actually leads to a decrease in dopamine activity (Sagvolden et al., 2005). Hypofunctioning dopaminergic systems presumably result in neither high nor low dopamine levels, such that a floor effect is created (Sagvolden et al., 2005). The floor effect likely leads to a biological reduction in the ability of ADHD individuals to extinguish behavior due to relatively stable dopaminergic response to all stimuli,
regardless of whether dopamine activity would otherwise increase or decrease in a non-ADHD individual (Sagvolden et al., 2005). In other words, because of biologically unique stimulus response systems, ADHD individuals are proposed to have difficulty learning whether a behavior is adaptive or maladaptive. Because reinforcing and extinguishing behaviors require highs and lows in dopamine levels, and because these highs and lows do not occur in individuals with ADHD, one can conclude that ADHD individuals have increased difficulty learning which behaviors should be maintained and which should be avoided. Further, both DAT1 and DRD4 are involved in the dopaminergic system, and therefore a neurobiological model of poorly modulated dopamine activity could result in similar reinforcement and extinction patterns in all three ADHD subtypes despite the potential that ADHD-I may be a distinct disorder.

**Dopamine Transfer Deficit Model.** The dopamine transfer deficit model proposed by Tripp and Wickens (2007) offered further support for the relationship between dopamine and altered response to reinforcement in children with ADHD. Specifically, based on rat and non-human primate research, Tripp and Wickens (2007) explained that in normal children, dopaminergic function increases prior to reinforcement due to cues that precede reinforcement, and this leads initially neutral cues to become predictive of reinforcement. Once these cues become associated with impending reinforcement, normal children experience anticipatory dopamine firing and reinforcement (Tripp & Wickens, 2007). By contrast, the dopamine transfer deficit (DTD) model contends that in children with ADHD there is reduced anticipatory firing of dopaminergic cells which results in decreased dopamine and subsequent reduction in association between anticipatory cues and dopamine, thus leading to deficient reinforcement processes (Tripp & Wickens, 2007). As a result, altered reinforcement contingencies trump the globally learned reinforcement contingencies of non-ADHD children, and the child with ADHD thus
appears “off-task,” impulsive, or to be misbehaving even where their behavior may be consistent with their independent reinforcement framework (Tripp & Wickens, 2007). In comparison to the DDT and other dopamine-based theories, the DTD similarly sites disruption of the dopamine signal as causal in development of variable behavior, but the DTD argues that this disrupted dopaminergic function will lead to increased rates of extinction whereas the DDT argues that the result will be decreased and deficient extinction (Tripp & Wickens, 2007). Of particular relevance for the research presented herein, the DTD predicts that continuous reinforcement schedules will lead to normal behavior whereas partial reinforcement schedules should exacerbate ADHD behavioral tendencies thus leading to poorer performance (Tripp & Wickens, 2007). This is also consistent with the fixed interval reinforcement contingency utilized by the most prominent rodent model of the DDT (Johansen et al., 2007). The present experiment does not incorporate neurological imaging or other biological testing and can therefore not assess for the role of dopamine, but this biological overview nonetheless offers a framework for understanding how typical ADHD behavioral patterns may develop, and thus provides a platform for analyzing the effect of different reinforcement contingencies on variability associated with ADHD.
The Dynamic Developmental Hypothesis of ADHD-HI and ADHD-C

Sagvolden et al.’s (2005) dynamic developmental theory (DDT) described the etiology of ADHD-HI and ADHD-C as resulting from environmental effects that stem from neurological dysfunction. This is consistent with other psychiatric and brain-based accounts of ADHD as a disorder of biological origin from which multiple pathways and interactions of genes and environment result in symptomatic ADHD (McGough & McCracken, 2006). Neurologically, the DDT states that hypo-function of dopaminergic systems results in atypical signal transmission that leads to improper control of behavior by reinforcing events that include extinction (Sagvolden et al., 2005). The behavioral, emotional, and cognitive features that define ADHD arise across time from these disordered reinforcement processes (Sagvolden et al., 2005).

In this way the DDT is developmental. Faulty reinforcement processes stemming from neurological deficits give rise, over time and within specific environments, to the diagnostic features of ADHD (Sagvolden et al., 2005; Tripp & Wickens, 2007). Individual behavioral deficits that are evident at very brief time scales (i.e., milliseconds) are the basis for behavioral, cognitive, and emotional symptoms that are evident at larger time scales (e.g., “as if he were driven by a motor”; “he does fine at first, but then quickly gets distracted and bored”) (American Psychiatric Association, 2000).

Therefore, according to the DDT (Sagvolden et al., 2005), an understanding of ADHD requires an evaluation of behavior at multiple time-scales beginning with how environmental events “capture” or fail to capture behavioral responses, and how those responses are pruned. Differences emerge between the behavior of ADHD and non-ADHD individuals with respect to behavior over time and depending on the nature of the schedule of reinforcement active within a specific environmental context. It is this time-based and situation-specific learning, according to
the DDT, that has been overlooked by other theories of ADHD. These behavioral differences manifest only through interactions with the environment. In that way, ADHD is determined by environment-behavior interactions. Neurology gives rise to ADHD via its influence on how the environment affects learning. What symptoms manifest, and when, is determined across environmental contexts in an independent manner. That is to say, in each new environment, ADHD behaviors become manifest as a result of the organism’s interactions within that environment. The DDT hypothesizes that positive parenting and societal styles interact with within-child features in order to produce both short- and long-term behavioral, emotional, and cognitive change (Sagvolden et al., 2005).

The short- and long-term outcomes described by this DDT model define extremely divergent trajectories in which a child becomes more adaptive or maladaptive based on environmental influences (Sagvolden et al., 2005). These divergent paths further clarify how ADHD may be conceptualized based on disruptive behavioral characteristics that lie along a continuum from adaptive-to-maladaptive and from positive-to-negative. Initial, short-term behavioral effects progress into longer-term positive and negative outcomes. The principle goal of the DDT is therefore to describe and understand this complex multidimensional and multidirectional set of relationships in order to determine how to consistently direct development toward more adaptive, positive outcomes (Sagvolden et al., 2005). While conceptualized differently, it is interesting that the within-child and environmental factors integral to the DDT have also been conceptualized by Barkely (1997) as the primary determinants in ADHD. Clearly, despite different formulas for conceptually linking these pieces, models of ADHD consistently identify medication, parental, and societal factors as significant factors in ADHD outcomes.
In summary, the DDT proposes a neurobiological and behavioral analysis of ADHD where two principle behavioral processes related to reinforcement and extinction of behavior lead to development of ADHD (Sagvolden et al., 2005). Sagvolden et al. (2005) explained that apparent executive dysfunctions characteristic of cognitive models may be reformulated as disturbances of behavior caused by problems with reinforcement, extinction, and impaired motor control. In this sense, Sagvolden et al. (2005) approached the development of ADHD and problems with inhibition as originating in disruption to behavioral chains rather than due to failures of cognitive processing. Inattention, however, is identified as a weakly defined descriptor which may be due to incorrect interpretations regarding behavioral changes, as well as to decreased motivation, psychiatric disorder, or cognitive impairment, and due to the abstract and poorly defined nature of inattention, Sagvolden et al. (2005) cautioned that non-ADHD disorders of inattention may appear to mimic ADHD. Additional studies of specific behavioral and neurobiological evidence for inattention in ADHD may either support current diagnostic organization of the inattentive type as an ADHD subtype or lead to identification of a differential diagnosis for ADHD-I which recognizes the presence of inattention without hyperactivity, and thus identifies a new disorder of inattention that is separate from ADHD-I. If a truly inattentive, non-hyperactive disorder is identified, then it would follow that the inattention present in ADHD is different, and ADHD-I may be subsumed under ADHD-C as a diagnosis that reflects both hyperactivity and inattention. Indeed, Sagvolden et al. (2005) and others argue for the presence of two etiologically separate disorders, and it is the role of future behavioral research to further differentiate inattention as seen in conjunction with hyperactivity from that seen without hyperactivity.
Testing DDT Predictions of Altered Reinforcement and Extinction Processes in ADHD

At present, the DDT has support arising from studies utilizing animal analogues of ADHD as well as from a couple of child studies (Aase et al., 2006; Johansen et al., 2007; Johnson, Wiersema, & Kuntsi, 2009). Specifically, Johansen et al. (2007) tested the dynamic developmental theory’s predictions of response variability and extinction by comparing the behavior of hyperactive rats to non-hyperactive rats. The predictions that drove the experiment derived from the DDT’s idea of steepened delay gradients as a primary aspect of ADHD (Sagvolden et al., 2005). Results were based on calculation of delay gradients, and ADHD animals were shown to have greater variability and slower extinction of inefficient responses, both of which are characteristic of steepened delay gradients. These findings are consistent with the predictions of the DDT and suggest the promise of the theory.

In addition to laboratory animal research, Aase et al. (2006) attempted to find support for the DDT as a neurobehavioral disorder by testing whether Western findings proved true in non-Western cultures within a human population. This study compared Norwegian and South African children by using a computer task in which children clicked on one of two squares; 1.5 second cartoons functioned as reinforcement for timely and correctly predicting square location (Aase et al., 2006). The results relevant for this discussion showed that Norwegian and South African children were more alike if they had ADHD than if they did not (Aase et al., 2006). In a similar study of only Norwegian children, Aase and Sagvolden (2006) additionally showed that frequent reinforcement is most effective and reduces the behavioral variability of children with ADHD (Tripp & Wickens, 2007). Neuringer (2009) commented on these findings, projecting that individuals with ADHD are sensitive to reinforcement provided it is offered at a certain frequency, but that infrequent reinforcement is associated with increased variability among those
with ADHD. The cross-cultural similarity of children with ADHD indicates that while non-ADHD characteristics may vastly differ, ADHD symptoms are consistent across cultural barriers, thus further supporting the DDT notion of ADHD as a neurobehavioral disorder, and dispelling the notion that ADHD prevalence in Western nations is a result of cultural issues (Aase et al., 2006). However, while ADHD prevalence is therefore not culturally determined, adaptation may be better in stricter societies, thus leading to lower levels of dysfunction among individuals with ADHD.

Different approaches have been utilized to test the applicability of DDT predictions to humans, and various methodologies center on the same principles employed in animal models, but until the study presented herein, none has employed the identical paradigm of Johansen et al. (2007) with human subjects. Considering that Johansen et al. (2007) presented an apparently reliable animal model of ADHD based on the DDT, we deemed it prudent to map that model to a human population and thereby test the effect of fixed interval reinforcement contingencies with humans. Though Sagvolden (2008) recently proposed a rodent model of ADHD-I, the wealth of research to date has limited animal model applicability to human research of ADHD-HI and ADHD-C, and there is therefore a need for additional research focused on better delimiting ADHD-I and ADHD-HI based on established models.

**DDT Competitors and Collaborators**

The four major and long-standing psychological theories of ADHD correspond to the measureable deficits as outlined in the section titled Measurable Deficits and Key Experimental Variables in ADHD, and include the DDT, Executive Dysfunction theory, State Regulation theory, and Delay Aversion theory (Johnson, Wiersema, & Kuntsi, 2009). The following
sections attend to key components of Executive Dysfunction and Delay Aversion theories, and specifically address similarities and differences between these theories and the DDT.
Executive Dysfunction Theories: Neuropsychological and Cognitive Impairments

Executive dysfunction theories of ADHD describe symptoms as emerging due entirely to neurobiological impairment and altered dopamine sensitivity (Johnson et al., 2009). The Dynamic Developmental Theory (Sagvolden et al., 2005), by contrast, views neurobiological impairment with similar importance, but only as one necessary component in a multi-dimensional framework leading to ADHD symptom expression. Findings of executive dysfunction in ADHD are debated (DuPaul, Weyandt, O’Dell, & Varejao, 2009) with some citing profound deficits in processing speed as well as differences by subtype while others report inconsistent subtype differentiation by executive dysfunction and even state that as many as 20% of those with ADHD display no executive deficiency (Shanahan et al., 2006). Despite these inconsistencies, evidence of frontal-striatal impairment in ADHD-HI and frontal-parietal impairment in ADHD-I provide a functional explanation for executive deficits (Johnson et al., 2009) that may lead to some differences as well as to some similarities that are similar in dysfunction but not causative agent. For this reason, neither similarities nor differences should be taken to preclude or support existence of a truly inattentive, distinct disorder (Diamond, 2005). Despite inconsistent findings, it is nonetheless informative to more generally consider which executive dysfunctions are most implicated in which subtypes.

Whether discussing DSM-III classification of ADD with or without hyperactivity or discussing DSM-IV classification of ADHD by subtypes which vary according to hyperactivity as well as inattention, general conclusions indicate that children with ADHD-HI demonstrate impulsivity and attentional deficits while children with ADHD-I demonstrate slowed perceptual processing and deficient vigilance (Chhabildas et al., 2001). This is consistent with likenesses between ADHD-I and adult dysexecutive syndrome. Additionally, though both combined and
inattentive groups show processing speed deficits, ADHD-C is associated with poorer planning skills than is ADHD-I (Chhabildas et al., 2001). Based on these differences, Chhabildas et al. (2001) hypothesized that ADHD-HI would show inhibition deficits, that ADHD-I would show processing speed and vigilance deficits, and that due to interaction effects, ADHD-C children would show more severe impairment on each of processing speed, vigilance, and inhibition. Chhabildas et al. (2001) used a twin study selected via parent and teacher questionnaires which screened for behavioral problems consistent with ADHD subtypes in order to determine neuropsychological differences of ADHD subtypes. After assessing for diagnostic criteria, inhibition and vigilance were studied using a Continuous Performance Task. Inhibition was additionally tested via the Stop Task, and processing speed was assessed using the Trailmaking tests A and B as well as parts of the WISC-R (Chhabildas et al., 2001). Though performance differences were found across groups, Chhabildas et al. (2001) did not find distinct neuropsychological profiles differentiating ADHD-I or ADHD-C children. More specifically, impairments demonstrated by ADHD-I and ADHD-C children were similar across neuropsychological measures. By contrast, when the effect of inattention was controlled, ADHD-HI children were shown to be non-significantly impaired on vigilance, inhibition, and processing speed tasks, and this suggests that it is inattention rather than hyperactivity/impulsivity that is associated with neurological deficits (Chhabildas et al., 2001). The authors therefore concluded that ADHD-I individuals suffer significant neuropsychological deficits while ADHD-HI individuals do not. In summary, despite distinct neuropsychological profiles, Chhabildas et al. (2001) described inhibitory failures in both ADHD-I and ADHD-HI, and argue that these two subtypes may, respectively, involve more severe cognitive or behavioral disinhibition.
Willcutt, Doyle, Nigg, Faraone, and Pennington (2005), however, indicated that specific neuropsychological executive function deficits may be used to differentiate ADHD-I and ADHD-C, noting especially differences in response inhibition. The conclusion is therefore that ADHD-HI does not show significant neuropsychological impairment, but ADHD-I and ADHD-C are associated with neuropsychological impairment that is considered a result of the symptoms of inattentiveness inherent in both the ADHD-I and ADHD-C groups (Chhabildas et al., 2001).

Malloy-Diniz et al. (2007) researched adults with ADHD-I and ADHD-C subtypes and found that in their sample, general intelligence scores were like those of controls, yet the ADHD adults demonstrated motor, cognitive, and attentional impulsivities which led to impairment on neuropsychological measures. This finding implies that impulsivity cannot be blamed on cognitive deficits. Specifically, on the Iowa Gambling Task (IGT) which is considered to reflect real-life decision making processes, ADHD adults consistently showed poorer decision making skills than controls, yet due to normal cognitive abilities, the decision making deficits evidenced by ADHD individuals on the IGT are clearly not the result of lower intelligence. Rather, the IGT performance of adults with ADHD indicates cognitive impulsivity. Malloy-Diniz et al.’s (2007) research thus supports the idea that impaired inhibition leading to impulsivity is one of multiple factors underlying ADHD behavioral impairment. This is consistent with other research (e.g., DDT of Sagvolden et al., 2005) stating that hyperactivity develops due to a time-dependent process in which neural predisposition is mediated by environmentally-based learning that reinforces impulsive behavioral responses. The earlier meta-analysis by Willcutt et al. (2005) supports the separation of intelligence from executive dysfunctions like cognitive impulsivity by explaining that executive dysfunction remains a central feature of ADHD even after controlling for correlated variables like IQ, reading competency, and comorbidities. Accordingly,
impulsivity can be described as action without forethought despite normal intelligence (Moeller et al., 2001).

In summary, this section describes the neuropsychological differences between ADHD subtypes as reliant upon inattention, and the shared problem of disinhibition present across studies, and introduces key components of the executive dysfunction theory of ADHD according to which ADHD symptoms arise from frontoparietal and frontostriatal abnormalities that lead to reduced executive control (Johnson et al., 2009). Recall that Chhabildas et al. (2001) demonstrated that ADHD-I and ADHD-C children, both of whom share inattention as a primary feature, are neuropsychologically similarly impaired while ADHD-HI children, for whom inattention is not a primary feature, only show significant neuropsychological impairment prior to controlling for inattention. Willcutt et al. (2005) offered support for the primary conclusion of Chhabildas et al. (2001) that ADHD-HI is not significantly neuropsychologically impaired while ADHD-C and ADHD-I are due to the shared factor of inattention, though Willcutt et al. (2005) additionally argued that ADHD-I and ADHD-C may in fact be differentiated by executive function deficits, specifically in response inhibition. Malloy-Diniz et al. (2007) demonstrated cognitive impulsivities in both ADHD-I and ADHD-C, and like Willcutt et al. (2005), executive function deficits are described as a central feature of ADHD. Taken together, these studies demonstrate neuropsychological differences across subtypes. Moreover, in addition to the previously stated behavioral evidence for multi-dimensional genetic, neurobiological, and environmental origins of ADHD, treatment response differences between the subtypes have led some to question whether they may in fact be separate disorders with unique neurobiological etiologies (Sagvolden et al., 2005). Consideration of the relationship between executive function and ADHD further differentiates between symptomology and either primary- or multiple-deficit
causality, and also provides a framework for bridging the DDT and cognitive theories of executive function.
Delay Aversion Theory, Motivation, and Memory: Revisiting Cognitive Theories

The delay aversion theory of ADHD was originally proposed by Sonuga-Barke, Wiersema, van der Meere, and Roeyers (2010), and provided a motivation-based explanation for why children with ADHD will opt for immediate rather than delayed reward (Johnson et al., 2009). The argument proposed that these children were not cognitively deficient or unable to wait, but rather that they felt an aversion to waiting and thus chose not to do so (Johnson et al., 2009). By this argument, inattention and hyperactivity develop due to situations where the child is unable to avoid delay (Johnson et al., 2009). The delay aversion hypothesis argues for a motivational underpinning for development of ADHD behaviors, and in so doing contests cognitive accounts of an executive dysfunction hypothesis (Antrop et al., 2006). According to Tripp and Wickens (2007), what Sonuga-Barke et al. (2010) cited as delay aversion may alternatively be viewed as preferential attendance to immediate reinforcement due to faulty dopamine processes. This account is also consistent with the DDT that describes a confluence of faulty dopamine and environmental processes as leading to the emergence of ADHD behaviors (Sagvolden et al., 2005). Delay aversion and preferential attendance to specific stimuli are therefore not mutually exclusive, but rather preferential attendance due to faulty reinforcement might precede delay aversive behavior (Tripp & Wickens, 2007). Interpretation and adoption of the DDT as a behavioral account of dysfunctional motivational impulses led Mitchell (2010) to combine DDT principles with motivational testing to review subsequent inhibition and hyperactive-impulsive behavioral symptoms. This perspective of motivational or delay aversive behaviors as complementary to DDT principles provides a dual explanation of behavioral outcomes as shaped by cognitive as well as environmental and motivational factors (Mitchell, 2010). ADHD research has been hampered by exclusive theoretical alliances that undermine
potentially valuable merging across disciplines. There have been some attempts toward integration across theories (Mitchell, 2010; Sonuga-Barke et al., 2009) coupled with criticism that none of the predominant theories focus on homogeneity and overlap thus opening rather than further sequestering discussion of ADHD causality and symptomology (Johnson et al., 2009). Excessive emphasis on theoretical perspectives at the expense of a priori testable hypotheses is a further criticism of the current ADHD models (Johnson et al., 2009). The following discussion therefore attempts to elucidate overlapping concepts and to thereby offer a broader understanding of the measurable deficits and key experimental variables of ADHD as they were presented in the section titled Measurable Deficits and Key Experimental Variables in ADHD.

Killeen (2005) provided a reinterpretation of the DDT in terms of cognitive psychology. He argues that the behavioral concept of reinforcement gradient is synonymous with, or at least compatible with, the cognitive notion of memory. This represents an important breakthrough for the DDT because it allows it to be linked with the copious work on cognitive deficits in ADHD. Also, Killeen’s cognitive reinterpretation should allow for better communication across the spectrum of disciplines that seek to explain ADHD. Johansen et al. (2009) also helped to bridge the cognitive-behavioral divide by describing key elements of the DDT as compatible with the term attention. Specifically, they note how neuropsychological processes previously interpreted as constituting the neurological basis for differential attention and memory decay can operate in terms of the delay-of-reinforcement gradients described by the DDT. Specifically, the delay-of-reinforcement gradient is a behavioral function which leads to characteristic ADHD symptomology, and which itself is shaped by multiple factors, including the neurological deficits that are manifest as attention and memory (Johansen et al., 2009).
Sagvolden et al. (2005) describe the effect on ADHD of shortened delay gradients, and the potential for behavioral modification via frequent reinforcement. They explain (Sagvolden et al., 2005) that in a normal delay gradient with infrequent reinforcers, as the window of time between behavior and reinforcers decreases, the effect of reinforcement increases. Extending this, the longer the window of time between behavior and reinforcement, the more diminished is the effect of reinforcement. By contrast, for ADHD, in order to obtain a comparatively sized reinforcement to that for a non-ADHD person, behavior must occur much closer in time to reinforcement than with a normal delay gradient (Sagvolden et al., 2005). This is because there is a shortened delay gradient in ADHD. In turn, to provide consistent reinforcement in ADHD, it is necessary to compress each behavior-reinforcer sequence into a shortened delay framework (Sagvolden et al., 2005). Through such a framework, ADHD behavior may more reliably be associated with reinforcement (or lack thereof), and consequently, behavior may be modified. Similarly, Catania (2005) explained that a shortened delay gradient differentially strengthens responses that occur in rapid succession. This rapid response rate coupled with decreased reinforcement occurring for early responses leads to a combination of hyperactive behavior with deficient attention, or ADHD-HI (Catania, 2005).

Both Catania’s (2005) and Sagvolden et al.’s (2005) explanations demonstrate the relationship between reinforcement and time as a function of varying delays of reinforcement gradients. By contrast, Killeen’s (2005) reinterpretation of the DDT in terms of cognitive psychology involved understanding that the time until reinforcement gradient may effectively be flipped such that both reinforcement and time are viewed as a function of memory trace strength rather than a function of the delay of reinforcement gradient. Killeen (2005) explained that as the reinforcement gradient decreases as time decreases, the memory trace is also decreasing,
leading to what may be considered either a congruent or perhaps even a combined effect of decreased memory trace occurring as the time until reinforcement (or reinforcement delay) increases. In simplified form, Killeen (2005) explained that the greater time that a person must wait for behavioral reinforcement results in weaker reinforcement, and this may also be interpreted as the effect of reduced memory trace.

The attempt to explain delay-of-reinforcement and memory trace functions as similar is further explained by Johansen et al. (2007) that delay-of-reinforcement presumably occurs as a result of the decay of the response trace over time. Having explained this relationship between the cognitive notion of memory and the behaviorist and DDT notion of delay-of-reinforcement gradients, it is worthwhile to briefly consider how either or both of memory decay and interference during learning may be causally related to ADHD symptoms of response disinhibition and poor association of behavior with delayed reinforcement.

Hervey, Epstein, and Curry (2004) described the interaction of response disinhibition with working memory, concluding that lack of inhibition leads to processing of extraneous stimuli that interfere with successful verbal working memory. Specifically, response disinhibition leads to distracted attention and subsequent inability to focus processing to that which is most relevant. The Stroop Color-Word Interference Test offers a good example as ADHD individuals show more difficulty inhibiting undesirable response than do controls (Hervey et al., 2004), thus exhibiting how interference may disrupt learning. Decay, by contrast, occurs as a result of deficient reinforcements across time associated with specific behaviors such that weak associations are formed between behaviors and consequences, leading to decayed memory of those behaviors for which there has been no recent reinforcement.
Computer Task Assessment of ADHD

The Continuous Performance Test (CPT) is one of several established, though frequently criticized computerized assessment tools for ADHD. The CPT is believed to assess for inattention, impulsivity, and vigilance (Brocki, Tillman, & Bohlin, 2010), and an important agenda for computerized ADHD assessment tools is to identify behavioral variability associated with ADHD. Neuringer (2009) regarded variability as a continuum where reinforcement contingencies control levels of predictability and repetition at one end and “stochastic unpredictability” at the other. ADHD, in turn, is associated with high levels of variability that impacts reaction time, as well as with disinhibition, and together these attributes lead to unusually large response classes for those with ADHD as well as what was initially construed as “non-contingent influences,” or failure to be normally influenced by reinforcement (Neuringer, 2009). The theory of non-contingent influences has met with criticism from evidence arguing for a more nuanced understanding of reinforcement sensitivity in ADHD. For example, in one study of these factors in children with ADHD, Saldana and Neuringer (1998) found higher rates of off-task responding, but similar behavioral variability to controls. This might be explained by previously discussed findings (Aase & Sagvolden, 2006) that show that frequent reinforcement leads to similar behavior across samples, but that infrequent reinforcement predicts a rise in variability for those with ADHD. In other words, contingent on frequency of reinforcement, ADHD does show sensitivity to reinforcement, and both novel situations and highly rewarding activities improve ADHD behavior such that it mirrors that of controls (Aase & Sagvolden, 2006; Sagvolden, Metzger, & Sagvolden, 1993; Saldana & Neuringer, 1998).

Spawned from these findings and computerized assessment tools, recently developed treatment paradigms for ADHD include computerized learning and classroom aids (Shalev, Tsal,
& Mevorach, 2007). Computerized assessment and treatment offers potential for quickly and accurately evaluating the effect of reinforcement contingencies on behavior as well as the extent to which variability shifts with environmental cues. Given flexibility to alter reinforcement contingencies, reduction in experimenter error, and ability to collect greater amounts of data thus allowing for review of variability and the effect of reinforcement on that variability, the present study utilized a fixed interval reinforcement computer task.

In preparation for presentation of my dissertation research which utilizes computerized assessment to compare ADHD symptom categories to each other and to controls, this literature review described fundamentally accepted differences between ADHD subtypes as defined by the DSM-IV-TR (American Psychiatric Association, 2000), introduced the laboratory terminology and conceptual framework for animal-to-human translational research, explained neurobiological and behavioral interaction in the development of ADHD, and discussed computerized assessment tools.
Purpose of the Study

This dissertation aims to translate the Johansen et al. (2007) rodent study to a human sample. The translational application involved two parts, referred to as Study 1 and Study 2.

Study 1 sought to examine a simple symptom-severity model of ADHD in which we explored how the severity of three symptom types relates to computer task performance. The symptom clusters correspond to the three symptom categories of ADHD specified in the DSM-IV-TR (American Psychiatric Association, 2000), namely: ADHD-HI and ADHD-I, and ADHD-C. For the purposes of evaluating these relationships, ADHD-HI is defined by a total HI symptom count, ADHD-I is defined by total I symptom count, and ADHD-C is defined by an aggregation of both HI and I symptoms. In this model, no effort is made to account for how the symptom categories relate to one another. The main hypothesis driving this investigation derives from Sagvolden et al.’s (1992; 2005) suggestion that it is the severity of HI symptoms that determines poorer task performance on fixed interval reinforcement schedules such as those appearing in the hole poking apparatus developed by Johansen and colleagues (2007) and the computer program utilized in the present study.

Study 2 seeks to explore a more complex model of ADHD in which subgroups are determined by the specific clustering of ADHD-I and -HI symptoms. This model was proposed by Diamond (2005) and defines ADHD-I not only by the presence of I symptoms, but also by the absence of HI symptoms. According to this conceptualization, participants with such a symptom profile have a qualitatively different disorder than individuals for whom I symptoms co-occur with even moderately high levels of HI symptoms. An interesting aspect of this conceptualization of ADHD is that it identifies ADHD-HI and ADHD-C as the same disorder known here and elsewhere as ADHD-HI/C. Therefore, in Study 2 an effort is made to utilize
self-report data from the larger sample of participants to generate the following three groups: ADHD-I, ADHD-HI/C, and a control group characterized by low I and HI scores.

**Predictions**

The dependent variables are the same for Study 1 and Study 2 and concern aspects of computer task performance. For both studies, comparisons are made within and between diagnostic groups with respect to the following variables: button sampling (e.g., how frequently the participant clicks computer buttons), variability of responding (e.g., entropy), and rate of learning (i.e., beta values) for the first six trials of a 15 trial computer task. An operational definition of each of these variables is provided in the Methods section of this paper. What follows here are the hypotheses regarding how ADHD symptomatology will relate to each of these variables. Study 1 predictions concern how computer task performance will relate to symptom severity for each of the three symptom categories. Study 2 predictions concern how computer task performance will differ across the three diagnostic groups that derive from Diamond’s (1995) conceptualization of ADHD subtypes.

**Study 1 hypotheses.** Study 1 hypotheses derive primarily from the DDT (Sagvolden et al., 2005) and the results of the Johansson et al. (2007) study, which provided support for that theory. According to the DDT principles tested by Johansen et al. (2007), hyperactivity symptoms should result in task performance characterized by higher total button sampling (i.e., clicks), greater variability of responding (i.e., entropy), and a faltering rate of learning (i.e., beta) characterized by relatively high initial learning that tapers off as the task novelty wears off. These hypotheses were to be tested by examining the zero order correlation coefficient between ADHD-HI and each of these measures.
The DDT and the Johansson et al. (2007) study were concerned with the impact of hyperactive-impulsive symptoms on fixed interval schedule responding. Therefore, neither source makes predictions about how inattention relates to the dependent variables of the present study. Other sources (Diamond, 2005; Barkley 2011), however, suggest that inattention might impact performance in ways that differ from hyperactivity. ADHD-I symptoms are expected to correlate positively with decreased total button sampling (i.e., clicks) and variability of response (i.e., entropy) that mirrors that seen when hyperactivity is present. Higher endorsement of ADHD-I symptoms was expected to correlate positively to an initially lower rate of learning (i.e., beta). Across the first several trials, these correlations were expected to reverse such that ADHD-I symptom endorsement would indicate higher rates of learning. We tested this by exploring both the zero order correlation between ADHD-I symptoms and all computer variables, and also with a partial correlation in which ADHD-HI symptoms were partialled. In that way, we could explore the extent to which I symptoms might operate independently from HI symptoms with respect to their impact on computer task performance.

**Study 2 hypotheses.** Study 2 seeks to evaluate how computer task performance differs across the diagnostic groups, ADHD-HI/C and ADHD-I, and a selected control group. According to Neuringer (2009) and Aase and Sagvolden (2006), one characteristic of ADHD is variability that is elevated in comparison to controls, but which may be reduced when individuals with ADHD are placed in a situation that involves frequent reinforcement. Thus, higher variability of responding (i.e., higher entropy) and greater button sampling (i.e., clicks) indicate more difficulty focusing and sustaining focus than do lower variability (i.e., lower entropy) and button sampling (i.e., fewer total clicks). Therefore, it was predicted that the ADHD-HI/C group would have higher mean button sampling, or clicking, scores than either the ADHD-I group or
the control group. And, given that deficits in cognitive processing speed and working memory are indicated in slowed reaction times (Diamond, 2005), the ADHD-I group was predicted to have lower mean button sampling scores than the control group. The ADHD-HI/C and ADHD-I groups were predicted to have higher mean variability (i.e., entropy) scores than the control group. The mean rate of learning (i.e., beta) scores across trials 1 – 6 were predicted to be initially similar for the ADHD-HI/C and control groups and both groups were expected to display higher mean initial rates of learning (i.e., higher betas or faster learning) than the ADHD-I group. Across trials 4 – 6 it was predicted that the control group would have the highest mean rates of learning (i.e., betas) except for a brief dip in trial 5 and that the ADHD-HI/C group would show the lowest rate of learning (i.e., lower betas or slower learning). The following figure (Figure 2) displays these predictions.

![Figure 2. Predicted rates of learning by group](image-url)
CHAPTER TWO
PILOT STUDIES

Prior to conducting Studies 1 and 2, five pilot studies were conducted to create a computer program that allowed for generating performance variables for humans that mapped onto those generated for rodents in the hole-poking apparatus. A total of 70 undergraduates were recruited in a manner similar to the Study 1 and Study 2 participants, in order that they complete the computer task in order to generate a program that best reproduced the animal study paradigm used by Johansen et al. (2007). Table 1 summarizes the pilot studies, includes the number of participants and what was accomplished, and indicates the changes made following each study. The program was considered complete once it mapped onto the Johansen et al. (2007) study in terms of having distinct FI blocks (e.g., trials 1 – 6, trials 7 – 12, and trials 13 – 15) that participants could reliably complete within the allotted time. These individual studies utilized computer performance data from samples that ranged in size from 9 to 22 participants, and were conducted with the purpose of testing game functionality. In addition, a post-participation questionnaire was used to see if participants could describe the operative contingencies. The results of the final pilot study indicated that performance variables fell within an expected normal range where participants were generally able to identify the intended behaviors. Earlier pilot studies varied between too easy and too difficult such that there was little participant variability. Results from the last pilot study indicated that participants generally understood the rules yet performance variability was maintained. The only problem in the final pilot study 5 related to alternating the number of reinforcers (Rs) that participants were required to collect. Requiring an inconsistent number of Rs per trial introduced a variable that was difficult to account for and also made it difficult to compare responses across trials. For example, if trial 1
had 10 required Rs while trial 2 had 20, and trial 3 had 10 again, the learning measured across
trials 1 – 3 would involve 40 Rs rather than 30 Rs and would therefore introduce nonlinear
changes in the data.

Table 1

*Overview of Pilot Studies 1-5*

<table>
<thead>
<tr>
<th>Pilot</th>
<th>Participants</th>
<th>Trials</th>
<th>Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22</td>
<td>1</td>
<td><strong>Noted programming error in which game functionality was inverted such that reinforcement occurred for fast clicking; game was also time-limited rather than based on trials</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Added trials with 1, 2, 3, 5, and 15 second FI reinforcement schedules and with 10 Rs per trial; fixed programming error; no longer gave 1 point for clicking lights/sound nor 2 points per R, but instead just 1 point per R; changed instructions</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>5</td>
<td>Reduced trial#5 FI schedule from 15 to 10 seconds</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>5</td>
<td>Added 7 trials with variable FI schedules and 10 Rs each</td>
</tr>
<tr>
<td>4</td>
<td>13</td>
<td>12</td>
<td>Added 3 trials; altered required Rs to be variable across trials, ranging from 10-20 per trial</td>
</tr>
</tbody>
</table>
Methods

Study 1 Participants

The participants in this study included 152 college students age 18 and older recruited through a WSU Psychology Department subject pool of introductory psychology students. Participants were recruited using two methods. All participants completed a prescreening questionnaire. Some experimental trials were limited to individuals who had endorsed multiple ADHD symptoms on the prescreen measure while other trials were open to all individuals enrolled as an introductory psychology student. By using a prescreen, it was possible to request participation from individuals who might fit the requirements for Study 2; given the relatively low percentage of ADHD in a normal sample, this approach made it possible to generate groups of at least 10 for Study 2. Similarly, the trials that were open to any participants were used to maximize the total number of individuals presenting for the sake of the correlational Study 1. Two individuals were turned away due to presenting intoxicated. Participants earned course credit for their participation. A total of 152 participants completed Study 1 of whom 97 were female and 55 were male. Their mean age was 20.5 years ($SD = 2.97$) and their mean grade point average (GPA) was 3.02 ($SD = 0.44$).

Study 2 Participants

As stated in the Introduction and in the section titled Neurobiological Foundations of ADHD, Diamond (2005) proposed that an ADHD-I designation be defined by moderate-to-high ADHD-I symptoms and few if any ADHD-HI symptoms. Furthermore, Diamond (2005) proposed that individuals who score high on ADHD-HI symptoms and ADHD-C symptoms be categorized together (see also Barkley, 2011). Consistent with this logic, an ADHD-I group was generated that included individuals who illustrated moderate-to-high elevation on the CSS-SR.
(see Measures; Barkley & Murphy, 2005) inattention scale and minimal elevation on hyperactive-impulsive symptom endorsement. Specifically, to be included in the extreme ADHD-I group, an individual’s summary score had to be a minimum of 11 for the inattentive domain and a maximum of 8 on the hyperactive-impulsive domain. According to the CSS-SR criteria, an I symptom scale score of 11 lies one standard deviation above the mean for a normal sample. Similarly, according to normative standards for the CSS-SR, the mean HI score in a normal sample equals 8.5. The inclusion criteria for the ADHD-I group in this study are therefore based on allowing for a maximum HI score equal to the mean HI score in a normal sample combined with an I score equal to at least one standard deviation above the mean I score in a normal sample. These criteria are thus derived from a combination of the CSS-SR normative data (Barkley & Murphy, 2005) and theories that argue that it is elevation on HI that accounts for differences in behavior between ADHD-I and ADHD-HI groups (Diamond, 2005).

Similarly, an ADHD-HI/C group was generated that included participants who illustrated moderate to high elevations on the CSS-SR hyperactive-impulsive scale, regardless of inattention symptomatology. To be included in the ADHD-HI/C group, the individual had to have a CSS-SR hyperactivity-impulsive summary score greater than or equal to 15. This is consistent with CSS-SR scoring instructions that state that hyperactivity-impulsivity is likely if scores exceed 15.6 (Barkley & Murphy, 2005).

Finally, a control group was generated that included participants who illustrated very limited elevations of the CSS-SR hyperactive-impulsive and inattention scales. Specifically, control participants had to have CSS-SR summary scores on both scales that were less than four. For participants ages 17 – 29, adult norms from a sample of 720 adults indicated a mean HI summary score of 8.5 with a 1 standard deviation range of 3.8 to 13; for participants ages 17 – 29, adult
norms indicate a mean I summary score of 6.3 with a 1 standard deviation range of 1.6 to 11 (Barkley & Murphy, 2005). A summary score of 0 – 3 on each scale falls within 1 to 2 standard deviations below the mean for HI and I symptom endorsement. As such, the control group requirements were chosen in order to generate an extreme comparison group.

Based on the above criteria, 10 participants qualified as ADHD-I, 11 qualified as ADHD-HI, and 12 qualified as controls. Demographic data that includes gender, mean age, mean GPA, and mean HI and I summary scores is presented in the Results section’s (Table 5) description of the sample characteristics for Study 2. The mean reported GPA was 2.90 ($SD = 0.60$) and the mean ranged from 2.76 to 3.06 with the ADHD-I group reporting the lowest GPA and the control group reporting the highest GPA. Mean ages ranged from 19.8 for both the control ($SD = 1.96$) and ADHD-HI/C ($SD = 2.00$) groups to 25.1 ($SD = 8.05$) for the ADHD-I group. The ADHD-I group’s higher mean age is due to inclusion of two participants ages 37 and 43; without these participants, the mean age of the ADHD-I group falls to 21.38 ($SD = 1.19$). The ADHD-I and control groups were split evenly with respect to gender while the ADHD-HI/C group was primarily female with only one male participant. These findings are consistent with those that state that the male-to-female ratio of ADHD becomes insignificant in adulthood (Malloy-Diniz et al., 2007); these findings are inconsistent with childhood statistics that show higher prevalence of ADHD-HI among males (Chhabildas et al., 2001) and higher prevalence of ADHD-I among females (Gross-Tsur et al., 2006).

**Procedures**

The present studies were run in a WSU Psychology Department research lab which is equipped with one large classroom and multiple private offices each of which contains one computer and speakers. The doors to each office were closed thus preventing participants from
observing one another, yet a window in each door made it possible for the experimenter to observe individual participants. All trials were scheduled for one hour, though the average duration of each computer trial for control participants only required 20 – 30 minutes. Participants were initially gathered in the classroom and provided with information regarding the experiment and their right to consent to participate (see Appendix C. Research Study Consent Form). After participants indicated agreement through signature, they were placed randomly into different individual offices.

After being seated, participants were told to follow the on-screen instructions: “Complete each of multiple game rounds using as few clicks as possible.” No further instructions were given. If a participant requested further instruction from the experimenter, the participant was told to do their best, to just keep trying to figure it out, or some other encouraging, but non-instructive response. Even if the participant asked directly, she was not told how many trials were necessary to complete the task.

All participants completed 15 trials of the computer task prior to completing computer administered versions of the CSS-SR (see Barkley & Murphy, 2005) and the Demographic and Performance Questionnaire (see Appendix B. Demographic and Performance Questionnaire). In the event that a participant did not finish the computer task within 50 minutes, the experimenter intervened to stop the task and to administer a paper version of the questionnaires. Following the experiment, individual debriefing included reminding participants of the confidentiality of the experiment, thanking them for their participation, and providing each an opportunity to provide more information that they thought relevant about their performance.
Measures

**Barkley Current Symptoms Scale—Self-Report Form.** The Barkley Current Symptoms Scale (CSS-SR) is an 18-item self-report measure of characteristics of ADHD and Oppositional Defiant Disorder (Barkley & Murphy, 2005). Items are rated according to a 4-point Likert scale where a score of 0 corresponds to “never or rarely,” 1 corresponds to “sometimes,” 2 corresponds to “often,” and 3 corresponds to “very often.” The 18 items are divided into nine items that assess for inattentive symptoms and nine that assess for hyperactive/impulsive symptoms where 8 of these 18 items additionally assess for ODD traits. Likert ratings for the nine hyperactive-impulsive questions and for the nine inattentive questions on the Barkley CSS-SR are summed to provide summary scores for both dimensions. The CSS-SR is a particularly useful adult assessment tool because it offers insight into how ADHD-traits have interfered over the preceding 6 months with functionality in various domains including home, work, and school (Murphy & Adler, 2004). The CSS-SR is a widely used screening measure for ADHD that derives its questions directly from the currently accepted 18 items identified in the DSM-IV-TR (American Psychiatric Association, 2000) diagnostic criteria. Diamond (2005) reports that previous studies have demonstrated validity, and others report an internal reliability coefficient of 0.93 (Katz, Petscher, & Welles, 2009). This instrument can be found in Barkley and Murphy (2005) and is not reproduced here due to copyright restrictions.

**Demographic and performance questionnaire.** This questionnaire includes 11 items that assess demographic status and experiential data regarding participation in the experiment. Specifically, information was obtained about the participant’s age, gender, ethnicity, primary language, major and GPA. In addition, three questions taken from the Padua Inventory-WSUR (Burns, 1995) assess obsessive-compulsive type behaviors, although these questions are not the
subject of the present study. With respect to their participation in the study, participants were asked to describe how they approached the computer task. This question seeks to determine if a general strategy was followed that involved matching, pattern and sequence-based responding, time-based responding, random responding, or none of the above. The last question encouraged the participants to share anything additional believed to be relevant. The data generated from this instrument was used primarily during the Pilot Study phase of this project and appears as Appendix B. Demographic and Performance Questionnaire.

**Computer Task Apparatus**

The computer task was developed to provide a human analog situation to the hole-poking apparatus developed by Johannson and colleagues. As such, it sought to generate a display that appears in Figure 1. As with the hole poke apparatus, the computer program exposed participants to a fixed interval (FI) schedule of reinforcement that provides reinforcement for behavior that occurs after a set time following the last reinforcer. The computer display observed by participants is presented in Figure 3. In this computer task, the dollar sign is the reinforced operant, and clicking on that button according to the task’s FI schedule results in awarding of game points which are displayed in a bar under the panel of buttons. Completion of the computer task requires participants to complete fifteen trials in which ten reinforcers (e.g., ten game points) must be attained per trial in order to advance to the next trial. Participants were not informed of the number of trials required. Table 2 indicates the number of trials, reinforcers, and FI schedule used in this computer task.
**Table 2**

*Computer Task’s Fixed Interval Reinforcement Schedule*

<table>
<thead>
<tr>
<th>Trials</th>
<th>FI reinforcement schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-6</td>
<td>1 second</td>
</tr>
<tr>
<td>7-12</td>
<td>3 seconds</td>
</tr>
<tr>
<td>13-15</td>
<td>5 seconds</td>
</tr>
</tbody>
</table>

Note. 10 reinforcers were required per trial

The computer task involves responding to a display (see Figure 3) that is composed of a panel of four rows of five identical buttons per row that appear on the computer screen. This computer-based apparatus is adapted from the experimental apparatus developed by Johansen et al. (2007) for use with rats. Consistent with the Johansen et al. (2007) study, the computer is programmed such that responding to 5 of the 20 buttons produces no effect, responding to 7 of the 20 buttons produces a sound, responding to seven other buttons produces flickering of a light on the face of the button, and responding to one of the buttons results in flashing of a dollar sign on the face of the button and reinforcement according to the FI schedule. The button functions remain the same throughout the experiment. Therefore, despite the fact that a number of outcomes can result from clicks on the display, reinforcement is obtained only by clicking one of the buttons.

Figure 3 illustrates the functions of each button as follows: blanks indicate no response, bugles indicate buttons for which a sound results, light bulbs indicate buttons that result in the light display, and the dollar sign results in reinforcement in the form of points that are displayed at the bottom of the screen.
Figure 3. Computer task state space

As stated, the computer task events mapped directly onto the original study’s operant chamber nose-poke holes (see Figure 1). A description of variables captured and the strategies used to analyze them follows.

**Computer Task (Dependent) Variables**

The computer task allows for calculating three dependent variables (e.g., button sampling, variability of responding, and rate of learning) relevant for testing variability and responsiveness to reinforcement. The dependent variables for the present study are as follows.

**Button sampling: Frequency of response per button and total clicks per trial.** For each participant, the number of total button clicks and the specific number of clicks per button (1 – 20) per trial was calculated. These calculations provide an overview of the individual’s button sampling per trial, and when averaged across trials, provide an overview of the participant’s button sampling across all three FI reinforcement blocks.

**Variability of responding (entropy).** A second measure of variability can be calculated as entropy, which involves multiplying the sum of the probability of clicking each button by the logarithms of those probabilities. This formula is: \( U = -\sum p \log_2(p) \). Entropy ranges from 0 to
4.32 with a score of zero indicating that only one button was clicked while a score of 4.32 indicates even distribution of clicking across all buttons. Higher entropy scores thereby indicate greater variability while scores closer to zero indicate better focusing on the target stimulus (the dollar sign). Mean entropy is calculated as a function of the number of reinforcers received during the 15 computer task trials averaged across all similarly categorized participants.

**Rate of learning (β).** A rate of learning parameter based on the Euclidean distance of button clicks from the reinforcer was calculated for each participant group for the reinforcers collected in trials 1 – 6, 7 – 12, and 13 – 15. The model of the learning process is a typical learning curve power function \( d_n = d_1 n^{-\beta} \), which can be interpreted that \( d_n \) (the mean Euclidean distance preceding each reinforcer n) equals \( d_1 \) (the mean Euclidean distance preceding the first reinforcer) multiplied by \( n \) to the power of the negative learning rate \( \beta \). \( d_1 \) and \( \beta \) are parameters of the learning model that will be fitted by taking the logarithm of each side of the equation (yielding \( \log d_n = \log d_1 - \beta \log n \)), and estimating \( \log d_1 \) and \( \beta \) by linear regression. Rate of learning scores across time (e.g., for each reinforcer per trial) indicate the relative speed of learning based on the distance away from the reinforcer of the buttons that the participant clicks preceding each reinforcer. A high rate of learning is observed when a participant hones in on the reinforcement button by either clicking only in the immediate proximity or on the button itself. A low rate of learning is observed when a participant clicks buttons further away from the reinforcement button.

**Analytical Strategy**

**Study 1: Correlational analysis of HI and I symptoms.** Recall that the Study 1 hypotheses were concerned with how hyperactive-impulsive symptoms, inattentive symptoms, and combined symptoms related to the three computer performance dependent variables: button
sampling (i.e., clicks), variability of responding (i.e., entropy), and rate of learning (i.e., beta) for each of the three FI blocks. Therefore, analyses involve exploring the zero order Pearson correlations between these variables. A partial correlation coefficient was also calculated that involved exploring the relationship between I symptoms and computer functioning, having partialled HI symptoms. Paired samples correlations were also conducted to evaluate differences in the rate of learning from one trial to the next.

**Study 2: Analysis of ADHD-HI/C and ADHD-I participant groupings.** Study 2 sought to explore whether computer performance differences existed across diagnostic groups. Analysis of Variance and t-test procedures were used to test the Study 2 hypotheses. First, ANOVAs were performed to evaluate whether the groups differed with respect to means for button sampling (i.e., clicks), variability of responding (i.e., entropy), and rate of learning (i.e., beta) across each of the three FI blocks. Post hoc analyses consisting of t-tests were performed in order to evaluate hypotheses related to differences across two groups (i.e., comparing the entropy of ADHD-I versus ADHD-HI/C). Bonferroni corrections were implemented to control for type I error arising from multiple tests. Paired samples t-tests were also conducted to evaluate group differences in the rate of learning from one trial to the next.

**Graphical analysis of participant groupings.** In addition to traditional correlational and mean differences statistical tests, we also utilized observational analytic procedures similar to those of Johansen et al. (2007). Primarily based on observational graphical data, Johansen et al. (2007) justify their analyses as consistent with predictions made by the DDT. In other words, the DDT predicts that fewer stimuli preceding a reinforcer will be captured and thus repeated at a later stage as a result of the initial reinforcer. According to the DDT and Johansen et al. (2007), various aspects of this phenomenon can be captured through the graphical analyses utilized by
Johansen et al. (2007). Regarding sample size, Johansen et al. (2007) state that use of SHR and WKY rats as ADHD and control analogues is validated, and SHR rats have been shown to graphically display specific behavioral patterns relative to WKY rats on measures of distribution of response to reinforcement. Like Johansen et al. (2007), we present graphical results of the mean button sampling (i.e., clicks) and a chi-square analysis comparing these graphs as well as graphical results of the mean variabilities (i.e., entropies) and rates of learning (i.e., betas) for the Study 2 ADHD-HI/C, ADHD-I, and control groups.
CHAPTER THREE

RESULTS

Study 1: Correlational Analysis of HI and I Symptoms

Sample characteristics. The mean CSS-SR total score for the entire sample was 15 ($SD = 8.24$), and scores ranged from 0 to 39 out of a total possible score of 54. This is consistent with the sample on which this measure was normed (Barkley & Murphy, 2005) where the mean CSS-SR total ADHD score for 17 – 29 year olds was 14.7 ($SD = 8.7$). For 17 – 29 years olds in the sample on which the CSS-SR was normed (Barkley & Murphy, 2005), the mean ADHD-HI summary score was 8.5 ($SD = 4.7$) and the mean ADHD-I summary score was 6.3 ($SD = 4.7$). The summary scores of participants in Study 1 were consistent with the normative data. Specifically, the mean CSS-SR hyperactive-impulsive summary score for Study 1 participants was 7.8 ($SD = 4.3$) with a range of 0 – 21 symptoms endorsed and the mean inattentive summary score was 7.6 ($SD = 4.7$) with a range of 0 – 23 symptoms endorsed. Of 152 participants, adherence to the Barkley CSS criteria indicated seven ADHD-C and 12 ADHD-I participants that accounts for 19 individuals or 12.5% of the total sample.

Correlational results. Testing the Study 1 hypotheses involved evaluating how each symptom dimension correlates with each of the computer performance variables (e.g., button sampling/clicks, variability/entropy, and rate of learning/beta) for each of the three sets of trials (see Computer Task Apparatus for an overview of trials 1 – 15). Before reviewing these analyses, Table 3 presents the means and standard deviations for the three dependent variables for each FI block. The button sampling (i.e., clicks) variable indicates the mean number of clicks made by all participants across each FI block, mean entropy values indicate the variability of
clicking shown by all participants, and the rate of learning (i.e., beta) values indicate the mean speed of learning for all participants across each FI block.

Table 3

Means and Standard Deviations of Clicks, Entropy, and Beta for Entire Sample

<table>
<thead>
<tr>
<th>DVs</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trials 1-6</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>184.86</td>
<td>89.46</td>
</tr>
<tr>
<td>Entropy</td>
<td>1.66</td>
<td>0.70</td>
</tr>
<tr>
<td>Beta</td>
<td>0.12</td>
<td>0.11</td>
</tr>
<tr>
<td><strong>Trials 7-12</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>375.81</td>
<td>341.91</td>
</tr>
<tr>
<td>Entropy</td>
<td>1.90</td>
<td>1.09</td>
</tr>
<tr>
<td>Beta</td>
<td>0.11</td>
<td>0.50</td>
</tr>
<tr>
<td><strong>Trials 13-15</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>204.72</td>
<td>212.42</td>
</tr>
<tr>
<td>Entropy</td>
<td>1.92</td>
<td>1.08</td>
</tr>
<tr>
<td>Beta</td>
<td>0.40</td>
<td>0.45</td>
</tr>
</tbody>
</table>

Note. DVs = Dependent Variables

The results of the correlational analyses are presented in Table 4. Note that it was necessary to control for skewness and this was accomplished by transforming the mean click variables for trials 1 – 6 and trials 7 – 12. As can be seen in the table, all correlations between computer performance scores and symptom dimension scores were insignificant with the exception of the correlations of clicking rate to reported total ADHD symptoms and to reported inattentive symptoms for trials 1 – 6. These results are surprising because it was predicted that computer task performance would correlate with the HI dimension, not the I dimension.
Table 4

Correlations between HI, I, and Combined ADHD Scores and Computer Performance Variable Scores for the Three Blocks of Trials

<table>
<thead>
<tr>
<th>DVs</th>
<th>ADHD (Combined)</th>
<th>HI</th>
<th>I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trials 1-6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>.164*</td>
<td>.098</td>
<td>.197*</td>
</tr>
<tr>
<td>Entropy</td>
<td>.050</td>
<td>.002</td>
<td>.088</td>
</tr>
<tr>
<td>Beta</td>
<td>-.013</td>
<td>.049</td>
<td>-.067</td>
</tr>
<tr>
<td>Trials 7-12</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>.112</td>
<td>.068</td>
<td>.134</td>
</tr>
<tr>
<td>Entropy</td>
<td>.104</td>
<td>.055</td>
<td>.131</td>
</tr>
<tr>
<td>Beta</td>
<td>-.071</td>
<td>-.068</td>
<td>-.062</td>
</tr>
<tr>
<td>Trials 13-15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>.066</td>
<td>-.001</td>
<td>.116</td>
</tr>
<tr>
<td>Entropy</td>
<td>.087</td>
<td>.024</td>
<td>.130</td>
</tr>
<tr>
<td>Beta</td>
<td>.055</td>
<td>.074</td>
<td>.028</td>
</tr>
</tbody>
</table>

Note. DVs = Dependent Variables  
*p < .05

Study 2: Comparison of ADHD-HI/C, ADHD-I, and Control Groupings

Sample characteristics. Of the 152 participants, 21 individuals were classified according to our criteria for extreme groupings (see Study 2 Participants). These groupings consisted of 10 classified as ADHD-I and 11 classified as ADHD-HI/C. These 21 individuals represent 13.8% of the total sample. Twelve control subjects were also chosen based on stated criteria and these 12 controls represent 7.9% of the total sample. Table 5 provides an overview of the participants’ demographics and CSS-SR scores by group assignment. Regarding differences between groups, ANOVA comparing mean GPA did not reveal a significant difference between groups. ANOVA comparing mean age did reveal significant variance in reported ages and a significant difference between groups (p<.02) which is due to the two outliers in the ADHD-I group; this is not considered a problem since all participants are adult college students. Regarding gender, it is
important to note that the majority of participants in Study 1 were female (64%) and Study 2 revealed identical gender ratios (64% female). Neither ANOVA nor chi-square comparing gender across groups was significant, $X^2 (2, N=33) = 5.30$, ns, however it is relevant to interpretation of the results to note the higher female ratio in the ADHD-HI/C group relative to the other groups. This is consistent with the WSU Psychology Department subject pool's gender balance of 59% female participants during the time of data collection.

In addition to reviewing differences between groups for each of the variables of GPA, age, and gender, independent samples t-tests were conducted to evaluate group differences in symptom endorsement on the CSS-SR. Comparison of the HI/C group mean CSS-SR HI score to that for the I group revealed significant differences, $t(19)=13.03$, $p<.05$. The HI/C and I groups did not, however, differ significantly with respect to mean CSS-SR I scores. The HI/C and control groups differed significantly with respect to both CSS-SR HI scores, $t(13)=21.28$, $p<.05$, and with respect to CSS-SR I scores, $t(12)=12.51$, $p<.05$. Similarly, the I and control groups differed significantly with respect to both CSS-SR HI scores, $t(20)=6.79$, $p<.05$, and with respect to CSS-SR I scores, $t(10)=9.53$, $p<.05$. These results reveal that the three groups differ in significant ways with respect to the two dimensions of ADHD.

Table 5

*Demographic and CSS-SR (Barkley & Murphy, 2005) Data by Participant Group*

<table>
<thead>
<tr>
<th>Demographics</th>
<th>HI/C</th>
<th>I</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size</td>
<td>11</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Males</td>
<td>1</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Females</td>
<td>10</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>Age</td>
<td>19.82</td>
<td>2.00</td>
<td>25.10</td>
</tr>
<tr>
<td>GPA</td>
<td>2.81</td>
<td>0.79</td>
<td>2.76</td>
</tr>
<tr>
<td>HI score</td>
<td>17.45</td>
<td>2.21</td>
<td>6.10</td>
</tr>
<tr>
<td>I score</td>
<td>15.64</td>
<td>3.56</td>
<td>13.60</td>
</tr>
</tbody>
</table>
**ANOVA results.** Study 2 sought to compare the computer performance of three groups of participants according to the criteria based on arguments presented in Diamond (2005) and Barkley (2011). Table 6 presents means and standard deviations for the three dependent variables by participant group and for each FI block.

Table 6

**Means and Standard Deviations of Clicks, Entropy, and Beta by Group**

<table>
<thead>
<tr>
<th>DVs</th>
<th>HI/C</th>
<th>I</th>
<th>Controls</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Trials 1-6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>193.36</td>
<td>76.24</td>
<td>206.10</td>
<td>118.60</td>
</tr>
<tr>
<td>Entropy</td>
<td>1.76</td>
<td>0.78</td>
<td>1.76</td>
<td>0.57</td>
</tr>
<tr>
<td>Beta</td>
<td>0.13</td>
<td>0.07</td>
<td>0.07</td>
<td>0.08</td>
</tr>
<tr>
<td>Trials 7-12</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>423.82</td>
<td>238.25</td>
<td>395.00</td>
<td>290.00</td>
</tr>
<tr>
<td>Entropy</td>
<td>2.31</td>
<td>0.99</td>
<td>2.15</td>
<td>1.33</td>
</tr>
<tr>
<td>Beta</td>
<td>0.02</td>
<td>0.59</td>
<td>0.08</td>
<td>0.51</td>
</tr>
<tr>
<td>Trials 13-15</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>162.55</td>
<td>156.37</td>
<td>237.00</td>
<td>175.00</td>
</tr>
<tr>
<td>Entropy</td>
<td>1.74</td>
<td>0.96</td>
<td>2.28</td>
<td>0.81</td>
</tr>
<tr>
<td>Beta</td>
<td>0.43</td>
<td>1.07</td>
<td>-0.17</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Note. DVs = Dependent Variables

Table 7 presents the results of ANOVAs that were conducted to determine if the means of the three participant groups differed with respect to the three computer performance variables button sampling (i.e., frequency of response and total clicks per trial), response variability (i.e., entropy), and rate of learning (i.e., beta). As can be seen, all ANOVAs were insignificant, which is inconsistent with predictions (see Predictions). Specifically, the groups did not differ with respect to performance on any of the three dependent variables across any of the three FI blocks. Moreover, visual inspection of the means did not reveal directional effects consistent with
predictions. For instance, Figures 5 and 6 show differences in response variabilities and rates of learning between the Johansen et al. (2007) study and this Study 2.

Table 7

**ANOVA of Participant Groups for Computer Performance Variables across Three Blocks of Trials**

<table>
<thead>
<tr>
<th>DVs</th>
<th>F(2, 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trials 1-6</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>0.30</td>
</tr>
<tr>
<td>Entropy</td>
<td>0.02</td>
</tr>
<tr>
<td>Beta</td>
<td>1.42</td>
</tr>
<tr>
<td>Trials 7-12</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>0.11</td>
</tr>
<tr>
<td>Entropy</td>
<td>0.06</td>
</tr>
<tr>
<td>Beta</td>
<td>0.06</td>
</tr>
<tr>
<td>Trials 13-15</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>0.96</td>
</tr>
<tr>
<td>Entropy</td>
<td>1.06</td>
</tr>
<tr>
<td>Beta</td>
<td>0.84</td>
</tr>
</tbody>
</table>

Note. DVs = Dependent Variables  
*p < .05

**T-test results.** Due to the exploratory nature of the study, we also conducted t-tests to evaluate if differences emerged when comparing two rather than all three groups. Once again, results were insignificant for all comparisons with respect to mean button sampling (i.e., clicks), variability of responding (i.e., entropy), and rate of learning (i.e., beta) values across three FI blocks. Table 8 displays the results.
**Table 8**

_T-values for Comparisons across the Three Groups of Participants for All Computer Performance Variables across Three Blocks of Trials_

<table>
<thead>
<tr>
<th>DVs</th>
<th>Trials 1-6</th>
<th>Trials 7-12</th>
<th>Trials 13-15</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HI/C – I</td>
<td>HI/C – controls</td>
<td>I – controls</td>
</tr>
<tr>
<td>Clicks</td>
<td>-0.11</td>
<td>0.64</td>
<td>0.67</td>
</tr>
<tr>
<td>Entropy</td>
<td>-0.01</td>
<td>-0.17</td>
<td>-0.18</td>
</tr>
<tr>
<td>Beta</td>
<td>1.66</td>
<td>0.86</td>
<td>-0.89</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>0.46</td>
<td>0.159</td>
<td>-0.32</td>
</tr>
<tr>
<td>Entropy</td>
<td>0.32</td>
<td>0.150</td>
<td>-0.20</td>
</tr>
<tr>
<td>Beta</td>
<td>-0.27</td>
<td>-0.314</td>
<td>-0.02</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clicks</td>
<td>-1.03</td>
<td>-1.33</td>
<td>-0.36</td>
</tr>
<tr>
<td>Entropy</td>
<td>-1.39</td>
<td>-1.08</td>
<td>0.31</td>
</tr>
<tr>
<td>Beta</td>
<td>1.33</td>
<td>0.49</td>
<td>-0.82</td>
</tr>
</tbody>
</table>

*Note. DVs = Dependent Variables
*p < .05

**Visual and Nonparametric Comparisons of Graphical Results**

Similar to the analytic procedures used by Johansen et al. (2007), button sampling frequency across trials 1 – 6 was graphed for the ADHD-HI/C and control groups, and the distribution of responses across trials 1 – 6 were graphed for each of the ADHD-HI/C, ADHD-I, and control groups.

*Button sampling: Frequency of response per button and total clicks per trial.* Figure 4a from Johansen et al. (2007) shows sampling by hole for trials 1 – 6 for each of the SHR and WKY groups. Prior to reproducing the Johansen et al. (2007) graphs, it was necessary to balance the number of Study 2 participants per group. This was approached in two different ways. First the control participant with the greatest total clicks was excluded. However, given the sample size and potential impact of removing even one participant, the second approach instead calculated
mean clicks per hole for each group and then multiplied those means by 8 to aid comparison with Johansen et al. (2007). Results from the second approach are shown in Figure 4a.

Figure 4a. Hole and button choices across trials 1 – 6 (truncated at 400) for the rodent study and the Study 2 control and ADHD-HI/C groups

Also consistent with Johansen et al. (2007), a chi-square test of independence was performed to study the relationship between participant group and level of button clicking in trials 1 – 6. Johansen et al. (2007) studied the relationship between ADHD-HI and controls and found the two graphs to be significantly different from one another, \( \chi^2 (19, N=16) = 218, p<.01 \). Like the Johansen et al. (2007) study, chi-square analysis of the balanced ADHD-HI and control groups with one excluded control participant was significantly different, \( \chi^2 (19, N=20) = 65.37, p<.01 \). Chi-square analysis of these groups balanced by using mean values rather than total sums
was also significantly different, $X^2 (19, N=21) = 57.25, p<.01$. Consistent with Johansen et al.’s (2007) findings, both methods of computing chi-square indicate that ADHD-HI/C participants are more likely to click all holes and to thus demonstrate a flatter spatial generalization than controls. Johansen et al. (2007) explain that a flatter generalization represents increased behavioral variability among the SHR group; the origin for this increased variability may be due to a spatial discrimination problem among the SHR group (Johansen et al., 2007).

The Johansen et al. (2007) study did not include an ADHD-I analog rat and therefore offered no results relevant to distinguishing an ADHD-I subgroup from a control group. Given that Study 1 found a significantly positive correlation between the ADHD-I dimension and button clicking across trials 1 – 6, a chi-square analysis comparison of the ADHD-I and control groups was conducted and found a significant difference between these two groups, $X^2 (19, N=22) = 36.45, p<.01$. An additional chi-square analysis comparison of the control group and the ADHD-I group minus the two age outliers was conducted and similarly found a significant, though somewhat less powerful, difference, $X^2 (19, N=20) = 31.09, p<.04$. Figure 4b shows sampling by hole for trials 1 – 6 for the full ADHD-I group. Compared to the Study 2 ANOVAs and t-test calculations, this data analysis represents an alternative method of comparing total clicks.

Figure 4b. Hole and button choices across trials 1 – 6 (truncated at 400) for the ADHD-I group.
Variability of responding (entropy and Euclidean distances). Johansen et al. (2007) explored the initial distribution of responses across trials 1 – 6. This involved calculation of Euclidean distances and mean variability of response (i.e., entropy). Consistent with Johansen et al. (2007), Study 2 participant data was plotted for mean entropies across trials 1 – 6. These plots appear in Figures 5 and 6 together with reproductions of the original rodent study plots (Johansen et al., 2007). All plots show the SHR and ADHD-HI/C groups in red ink, the WKY and controls in blue ink, and the ADHD-I group in green ink. The Johansen et al. (2007) graphs demonstrated greater Euclidean distances preceding each reinforcer as well as higher entropy across trials 1 – 6 for the SHR group, thus indicating that the SHR group displayed a more variable rate of response than the WKY group. The graphs from Study 2 do not present the clear pattern of results observable in the Johansen et al. (2007) graphs.

Figure 5. Entropy as a function of the number of reinforcers received during trials 1 – 6
Initial learning

Upon initial exposure to all holes, all rats probed most of the holes. Over the course of the first 6 sessions of FI 1 s with all holes available, the distribution of responses narrowed, becoming both more focused on the target hole, and becoming less variable overall. This is visible in Figure 3, where the average distance of hole-pokes from the target hole is plotted as a function of number of reinforcers ($d_n$). The curves are simple power functions, which are often used to describe learning curves:

$$d_n = d_1 \times n^{-\beta}$$

where $d_n$ is distance in cm around the time of the $n$th reinforcer, the parameter $d_1$ is the average distance projected to the time of the first reinforcer, and $\beta$ is the rate of learning. Both strains start from an average distance of $d_1 = 7.1$ cm, but the rate of learning is faster for WKY ($\beta = 0.32$) than for SHR ($\beta = 0.22$).

In Figure 4, response variability, expressed as entropy, is plotted as a function of reinforcers during acquisition. For both strains, the decreasing variability is described by Equation 1. The SHR start slightly more variable ($U = 3.7$) and may focus more slowly ($\beta = 0.13$) than WKY ($U = 3.0$,$\beta = 0.18$). Given the width of the error bars, however, all that can be said with confidence is that the entropy curve for the SHR lies above that for the WKY. The reduction in variability of responding was largely due to the convergence of behavior onto the operant target hole. The holes around the periphery provided additional stimulation which seemed more attractive than the neutral holes. Figure 2 shows, however, that any additional attractiveness of the stimulus holes may be attributed to their spatial layout, not their sensory consequences.

Delay-of-reinforcement gradients

To what extent can a reinforcer increase the probability of not only the response that immediately preceded it, but also the probability of other, earlier responses? Figure 5 shows real delay-of-reinforcement gradients calculated from all sessions testing FI < 300s and the last 21 sessions testing FI 300 in the manner detailed in the procedure section. They are shown on a logarithmic $x$-axis to highlight the time intervals closest to reinforcement. The data are pooled across all animals within a strain. The curves through the data are exponential processes, such as those represented in Equation 2, where the parameter $c$ gives the height of the gradient above its asymptotic level, $b$, at the time of reinforcement ($t = 0$). The parameter lambda gives the rate of decrease in the gradient as a function of the time between a response and the ensuing reinforcer. The additive constant $b$ measures the asymptotic probability of emitting the same response on succeeding trials.

$$dd_n = -E(1)$$

Figure 6. Mean distance of holes/buttons visited prior to each reinforcer for trials 1 – 6
CHAPTER FOUR

DISCUSSION

The purpose of this dissertation was to translate research from a rodent study of ADHD (Johansen et al., 2007) to a human sample with the goal of evaluating if the theoretical model — the DDT (Sagvolden et al., 2005) — is supported in humans. The DDT predicts that the performance of individuals — rodents or humans — with ADHD-HI symptoms will differ from those without ADHD-HI symptoms on tasks that utilize fixed interval reinforcement schedules to maintain behavior. According to the theory, which received support from the Johansen et al. (2007) rodent study, ADHD-HI individuals should show clear patterns of behavior with respect to the following three variables: button sampling (i.e., total clicks), variability (i.e., entropy), and rate of learning (i.e., beta). Specifically, it was predicted that individuals endorsing ADHD-HI/C symptoms would show higher rates of button sampling and greater variability than those endorsing ADHD-I symptoms or individuals endorsing few ADHD symptoms. Rate of learning was expected to decrease with increased symptoms of ADHD; among those endorsing ADHD symptoms, it was expected that those endorsing mostly I symptoms would start more slowly than those endorsing HI/C symptoms, but that they would show greater learning across time. Each of these differences was reported in the Johansen et al. (2007) study.

This dissertation was comprised of two studies as well as graphical and nonparametric comparison to the original rodent study (Johansen et al., 2007). Studies 1 and 2 showed that HI/C and I scores are generally unrelated to the three computer-task generated performance variables of button sampling (i.e., total clicks), variability (i.e., entropy), and rate of learning (i.e., beta). Study 1 did, however, find a significant correlation between button sampling (i.e., total clicks) and ADHD, and specifically ADHD-I, symptom endorsement for trials 1 – 6.
Consistent with this finding and that of Johansen et al. (2007), graphical and nonparametric analysis of the Study 2 data demonstrated a significant chi-square difference in spatial generalization (i.e., higher or more variable button sampling) of the ADHD-HI/C group compared to the control group as well as of the ADHD-I group compared to the control group. Interestingly, ANOVAs conducted in Study 2 did not find evidence of a significant difference in clicking rate by group. While the button sampling findings were consistent with those found by Johansen et al. (2007), graphical comparisons of variability (i.e., entropy) and rate of learning (i.e., beta) did not display consistent results. Overall, findings from Studies 1 and 2 and from the graphical comparison to Johansen et al. (2007) reveal an inconsistent pattern with some significant, but mostly insignificant results. The pattern of significant correlational and chi-square results across trials 1–6 for the button sampling variable for Study 1 and the graphical comparison to Johansen et al. (2007) despite the lack of significant ANOVA differences in Study 2 indicates that differences are directional, but not substantial and robust enough to be defended. This suggests that with clinical groupings, differences might be more apparent and that it might also be possible to modify the computer tool to differentiate controls from individuals with ADHD-HI/C.

**Study 1: Correlational Analysis of HI and I Symptoms**

Study 1 organized participants based on self-reported ADHD symptoms categorized according to the CSS-SR (Barkley & Murphy, 2005), and the goal of Study 1 was to determine whether reported symptom severity correlates to any of the three measures of computer task performance.

Increased button sampling (i.e., total clicks) was expected to correlate positively with the number of reported HI symptoms and negatively with the number of I symptoms. Study 1 did
not support these hypotheses. In fact, there was a significant positive correlation between I symptoms and button sampling but not between HI symptoms and button sampling. ADHD symptoms as a whole did, however, correlate positively to increased button sampling. The ADHD-HI button sampling results are contrary to the Johansen et al. (2007) rodent study findings and to indications that ADHD leads to higher rates of off-task responding (Saldana & Neuringer, 1998). They are not, however, inconsistent with arguments that ADHD-HI does not manifest in novel situations (Aase & Sagvolden, 2006; Sagvolden et al., 1993; Saldana & Neuringer, 1998) or with the argument that ADHD-I and ADHD-C have more pronounced difficulties with response inhibition and neuropsychological impairment than either ADHD-HI or controls (Chhabildas et al., 2001; Willcutt et al., 2005). Recall specifically that frequent reinforcement and novel situations can lead to an improvement in focus for individuals with ADHD such that their behavior mirrors that of controls (Aase & Sagvolden, 2006; Neuringer, 1999; Sagvolden et al., 1993; Saldana & Neuringer, 1998). Given that most of the individuals in this study that reported I symptoms also reported a high number of HI symptoms, interaction effects typical of the ADHD-C subtype are expected (Chhabildas et al., 2001). The significant positive correlation between total ADHD symptoms and increased button sampling is therefore consistent with the notion that the ADHD-C subtype demonstrates the most severe impairment on measures of processing speed, vigilance, and inhibition (Chhabildas et al., 2001).

Variability (i.e., entropy) was expected to correlate positively with HI and I symptoms, but this was also not an accurate hypothesis. The findings were, however, consistent with those of Saldana and Neuringer (1998) who found behavioral variability to be similar for ADHD and control participants. Rate of learning (i.e., beta) was expected to change across sessions and in particular, the ADHD-I group was expected to show an overall higher rate of learning. This was
also not an accurate hypothesis, and in fact the inverse was shown; the ADHD-HI group showed a trend toward a positive correlation with rate of learning while the ADHD-I group did not. Due to greater emphasis on cognitive difficulties, ADHD-I is believed by some to indicate worse prognosis than ADHD-HI (Catania, 2005; Chhabildas et al., 2001), a perspective that would be consistent with the findings. By contrast, the predictions herein were based on the school of thought that hyperactivity is of primary importance in the evaluation of ADHD (Diamond, 2005). Furthermore, both variability and rate of learning are impacted by the amount of clicking that occurs on buttons other than the reinforcement button. It is therefore possible that ADHD symptoms could be related to non-target clicking of a distracted or hyperactive origin while controls might also click frequently as a result of attempting to construct a pattern. This is one potential explanation for these unexpected results.

**Study 2: Comparison of ADHD-HI/C, ADHD-I, and Control Groupings**

Study 2 organized participants into two symptom groups, one comprising individuals with elevated hyperactivity and the other comprising individuals with elevated scores on inattention but minimal elevation in reported hyperactivity. Based on the organizational logic of Diamond (2005), this second study sought to identify differences in the computer task performance of participants endorsing elevated levels of inattention with and without comorbidly elevated levels of hyperactivity as well as of both groups relative to controls. It was predicted that the ADHD-HI/C group would demonstrate higher mean button sampling than the control group who was in turn predicted to demonstrate higher mean button sampling than the ADHD-I group. The ADHD groups were predicted to show higher mean variability (i.e., entropy) than the control group, and rate of learning (i.e., beta) was predicted to be initially highest for the ADHD-HI/C and control groups, but across the first six trials, it was expected that the ADHD-HI/C group would show a
decrease in rate of learning. Predictions were inconsistent with results. ANOVAs showed no significant results for differences across groups and t-test comparisons similarly revealed no significant results. Perhaps the greatest reason for the lack of significant differences is the fact that the particular FI schedule utilized in this study may have eliminated differences due to offering frequent reinforcement and a novel environment (Aase & Sagvolden, 2006; Sagvolden et al., 1993; Saldana & Neuringer, 1998), and also due to the potential that control participants may have engaged in pattern-based responding. The lack of significant findings in Study 2 was inconsistent with expectations and initially raised questions of a critical flaw in the program design as opposed to a problem with group assignment. This perspective seems inaccurate, however, when considered in context of the Study 1 findings.

While Study 1 was inconsistent with predictions, the significant correlation between total ADHD, and specifically between ADHD-I, and rate of clicking across trials 1 – 6 demonstrated that the computer program captured some differences between ADHD and control behavior. Several of the identified differences were consistent with literature opposing the literature on which the predictions were made, and as such, while inconsistent with our predictions, the differences were consistent with other presented literature and established descriptions of ADHD (Aase & Sagvolden, 2006; Chhabildas et al., 2001; Neuringer, 1999; Sagvolden et al., 1993; Saldana & Neuringer, 1998; Willcutt et al., 2005). Given that correlational differences with respect to symptom endorsement were identified in Study 1, then those differences ought to have been magnified in Study 2. The fact that Study 2 demonstrated no significant comparisons is thus remarkable because it implies that the group assignments made in Study 2 were not optimally configured. This implication gains further strength when considered in context of the chi-square comparisons made as part of the graphical analysis of the Study 2 data.
Graphical Analysis of Participant Groupings

Graphical comparison of the Johansen et al. (2007) data and the Study 2 data (Figures 5 and 6) primarily revealed inconsistencies between the behaviors observed in rats and humans. Chi-square analysis indicated graphical similarities (see Figure 4a) with respect to button clicking. Like in the Johansen et al. (2007) study, the flatter spatial generalization of the ADHD-HI/C group indicated less focused clicking on the reinforcement button and surrounding buttons. In this respect, the control groups in both studies may have behaved similarly to one another by focusing on the holes/buttons surrounding the target. In addition, chi-square analysis indicated that ADHD-I also displayed a significantly flatter spatial generalization than the control group. This is of particular relevance when viewed in context of the Study 1 correlational results because, in contrast to the Study 2 findings, these findings together indicate that I symptoms might be of greater importance than HI symptoms in determining behavior on FI tasks.

Graphical comparison of entropy scores indicated that the Johansen et al. (2007) study observed divergent behavioral tendencies clustered by participant group whereas this dissertation observed behaviors clustered by group, but with frequent cross-over from one behavioral pattern to another. Interestingly HI and I groups followed a pattern inverse to one another with respect to the trials in which each group demonstrated the greatest or least variability. By contrast, the control participants seemed to demonstrate a more stable pattern than either the HI or I groups, yet that pattern was stable at an elevated entropy score thus indicating consistently elevated variability of response. As mentioned in the discussion of both Studies 1 and 2, one explanation for the control participants showing an elevated entropy score may be that control participants may have used pattern based responding. In other words, due to 1-second reinforcement contingencies, it is possible that control participants were inadvertently reinforced for a sequence
of clicks rather than simply the one click on the reinforcer button. In this case, it would be logical that the ADHD-HI participants might display similar behaviors to – due to different underlying causes than – the control group. The current program design does not provide a means to identify differences in underlying cause for different outcomes.

The Johansen et al. (2007) study provided graphical results of the Euclidean distance from the poked hole to the target hole by group as a measure of distance and thus also of rate of learning (i.e., beta). Visual comparison of Euclidean distance graphs showed marked differences between the animal and human studies where the human control participants demonstrated higher mean distances relative to the ADHD-HI group whereas the animal controls demonstrated lower mean distances. It is proposed that the contradictory entropy findings and the unexpected distance findings may share an explanation. Indeed, if control participants were inadvertently reinforced for a sequence of clicks rather than for one click on the reinforcer button, then it would follow that the control group’s mean distances prior to each reinforcer would be higher than was expected.

**Limitations**

It is important to identify limitations of the presented studies, and to discuss how they may be overcome in the future. The limitations discussed are related to sample size, group assignment, gender differences across groups, and functional difficulties of computer program assessment in humans as opposed to operant chamber rodent experimentation. Limitations related to group assignment and gender are specific to Study 2, but otherwise all limitations discussed were universal across both studies.

**Sample size.** One limitation with respect to conducting the ANOVAs is that of the sample size. Due to preferential selection of prescreened individuals, the total sample size of 152
participants contained 13.2% of the target population, a percentage that represents a higher number of target participants than that found in the general adult population or in other comparably sized ADHD studies. Mean percentages of adult, and specifically college adult, ADHD have been estimated somewhere between 1 – 8% (DuPaul et al., 2009; Kessler et al., 2005). As the research indicates, it is unrealistic to expect high ratios of ADHD individuals in the adult population. The most significant problem regarding sample size was to sample enough total participants to obtain a higher number of ADHD-I and ADHD-HI/C participants. Specifically, the samples in Study 2 consisted of too few extremes to constitute high statistical power when comparing the ADHD-I and ADHD-HI/C groupings.

**Group assignment.** The difficulties connected to increasing our target populations, and particularly ADHD-I, are consistent with those found by Barkley (2011), DuPaul et al. (2009), Davidson (2008), and Diamond (2005). Difficulty obtaining a larger human sample size of participants expressing ADHD symptoms highlights a conundrum wherein animal models are used precisely because rodent groups are specifically bred to exhibit behavior currently believed to be representative of human ADHD. Optimal translational research therefore relies on larger human sample sizes than are typically obtained even where resources are immense (Barkley, 2011).

A failure to screen participants beyond use of the CSS-SR further interfered with group selection and assignment. Participants in the current study were not explicitly assessed for medication use or diagnosed developmental or learning disabilities, including ADHD. Participants had the option of offering additional information that they self-identified as important and, in fact, multiple participants did offer information relevant to an ADHD study with two specifically revealing an “ADD” diagnosis as well as use of Ritalin. Conversely, others
self-identified as “ADD,” but given that there was no standardized format for assessment of prior clinical diagnosis or medication use, none of this information could be used to sort groups. Given that medication use is intended to alter the very behaviors studied by the utilized computer task, and given that the groups were already suboptimally sized, addressing this problem might result in significantly different outcomes.

**Gender.** As indicated in the Methods and Results sections, the ADHD-I and Control groups were split evenly with respect to gender, but the ADHD-HI/C group was primarily female with only one male participant. This is inconsistent with childhood and adolescent data that indicate higher prevalence of ADHD-HI/C among males (Chhabildas et al., 2001), but is consistent with suggestions that gender differences become insignificant with age (Malloy-Diniz et al., 2007). Given that Study 1 was 64% female and that the selected participants for Study 2 also were 64% female, it is believed that one reason for the gender differences displayed is that the population was disproportionately female; indeed, 59% of the WSU Psychology Department subject pool participants were female during the period in which Studies 1 and 2 were conducted. The approach used to classify individuals for Study 2 may have selected a disproportionate number of females for the ADHD-HI/C group because of the relatively high rates of inattention among those selected. This is another indication that the Study 2 participant selection methods may be flawed. To remove this confound, it would be advisable to conduct these studies with gender-homogenous and clinically diagnosed groups.

**Computer task.** As indicated in Chapter Two, the computer task was thoroughly tested and modified across five pilot studies. The program effectively recreated a computerized version of the original Johansen et al. (2007) rodent study. Despite these pilot studies and success in replicating most aspects of the rodent experiment with humans, the translation from animal-to-
human research resulted in a couple of unique problems in the human research that developed due both to the expectation that basic human behavior would be far more advanced than rodent behavior as well, conversely, to human conceptual capabilities that resulted in creating a more multidimensional experience than that experienced by the rodents. Specifically, the rodent study (Johansen et al., 2007) included an initial training paradigm that was determined to be unnecessary with a human sample. Most participants during the pilot studies gave no indication that a training paradigm was necessary and in fact, several correctly assessed the game and performed well. The lack of a training paradigm may, however, have increased imaginative learning and thus increased the likelihood of reinforcing pattern-based or other rule-governed behavior. This eventuality led to the addition of question 1 in the Appendix B. Demographic and Performance Questionnaire that asked participants to state their method of responding. Correlational analysis revealed, however, that the indicated method of responding (pattern, matching, random, time, or none) was not significantly correlated to ADHD-HI symptom endorsement or to ADHD-I symptom endorsement. Despite this lack of correlation between reported method of responding and symptom endorsement, the data reveals trends wherein some participants were quickly reinforced for clicking at least one button prior to the reinforcer, and this behavior among control participants could be misleading with respect to measures of total buttons sampled (i.e., clicked), variability or entropy, and rates of learning (i.e., betas).

Another possible flaw in the computer task may be that frequent rewards have been shown to reduce or even eliminate differences in behavior when comparing WKY and SHR rats (Sagvolden et al., 1993). Recall also the findings of Aase and Sagvolden (2006) who showed that frequent reinforcement resulted in similar findings for ADHD and control participants. As indicated in Table 2, the FI reinforcement schedule utilized by the computer assessment began
with 1-second FI reinforcement and only increased to a maximum wait time of 5-second FI reinforcements. These rewards may have been too frequent to allow for divergence of ADHD-HI/C, ADHD-I, and control behavioral patterns. It is believed that a better approach would be to have a FI schedule that began with frequent reward as part of a training paradigm followed by infrequent reward.

As reviewed here, limitations of this study include sample size, limited collection of information of potential importance to group assignment, and problems related to optimal design of the computer task. Due possibly to these limitations and despite numerical trends suggesting some differences between groups, these studies indicate that the computer task is of no clinical utility for differentiation of participants as HI/C or I. Possible future directions and alterations to this research are therefore provided based upon the limitations believed to have had the greatest detrimental impact on hypothesized outcomes.

**Future Directions**

Recommendations to improve this experiment logically follow from the limitations discussed relating to each of sample size, screening for group assignment, and problems with the computerized assessment tool.

**Sample size and group assignment.** To address the problems of sample size and screening, it is recommended that a clinically diagnosed human sample be used to assess the utility of the computer assessment tool when replicating the Johansen et al. (2007) study exactly. Positive findings would thus indicate support for the rodent model of ADHD as well as support for the translated assessment device. Following from these findings and given that clinical diagnosis according to current DSM-IV-TR (American Psychiatric Association, 2000) criteria would eliminate the ability to sort data according to alternative techniques (Barkley, 2011;
Diamond, 2005), it would then be recommended that extreme groupings be created via
diagnostic interview and categorization according to the criteria presented in this dissertation.
Using a clinical sample would eliminate issues related to sample size as well as ambiguities
surrounding medication use and potentially relevant comorbid conditions such as learning
disabilities.

**Computer task.** Finally, the computer tool ought to be modified by providing more
explicit directions and by adding a shaping procedure. The focus should be on whether
participants with ADHD-symptoms are capable of behaving according to structured rules rather
than on whether or not they are capable of self-identifying those rules and then following them.
Explicit directions and a shaping procedure ought to also reduce problems related to self-directed
rule-governed behavior.

It is believed that problems related to insufficient motivation and rule-governed behavior
are the primary reasons that the methodology did not transfer. Redesign that addresses the
problems identified herein might result in this methodology being diagnostically useful.

**Conclusion**

Although the computer task methodology tested in the present study does not appear to
discriminate groups to the extent necessary for use as a diagnostic instrument, the results of the
study are interesting from a theoretical perspective. Specifically, Study 1 correlational analyses
revealed a significant association between inattentive symptoms, but not hyperactive-impulsive
symptoms, and variability of responding on the FI computer task. In addition, chi-square
analyses revealed spatial generalization differences such that both ADHD-I and ADHD-HI
participants illustrated a greater tendency to click all holes than did the control participants.
While these results are consistent with the notion that ADHD may influence responding on FI
schedules, they are contrary to the notion that effects are driven by HI symptoms. Instead, the present results point to the possibility that, at least with college students, differences in FI schedule performance may be driven more so by I symptoms rather than by HI symptoms.

Although these findings are contrary to the one aspect of the DDT which postulates that hyperactivity-impulsivity drives the relationship between ADHD and FI behavior, these findings are consistent with Catania’s (2005) notion that extreme I symptoms may dictate variability of responding and may even represent more severe disorder than one characterized by HI symptoms. Similarly, Diamond (2005) and others (see also Milich et al., 2001) have speculated that a disorder exists that is characterized by inattention without hyperactivity or impulsivity, although little is known about the underlying behavioral features of that disorder. Therefore, it may be worthwhile for future studies to investigate how FI responding relates to the two symptom dimensions of ADHD. Such investigations must take into account the fact that the symptom profiles of individuals change over the course of development (Biederman et al., 2000; Chhabildas et al., 2001), and that much remains unknown about how ADHD manifests in young adults.
REFERENCES


Diamond, A. (2005). Attention-Deficit Disorder (Attention-Deficit/Hyperactivity Disorder without hyperactivity): A neurobiologically and behaviorally distinct disorder from
Attention-Deficit/Hyperactivity Disorder (with hyperactivity). *Developmental Psychopathology*, 17, 803-825.


APPENDIX


**ADHD-I Symptoms**
1. Often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
2. Often has difficulty sustaining attention in tasks or play activities
3. Often does not seem to listen when spoken to directly
4. 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)
5. Often has difficulty organizing tasks and activities
6. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
7. Often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)
8. Is often easily distracted by extraneous stimuli
9. Is often forgetful in daily activities

**ADHD-HI Symptoms**
1. Often fidgets with hands or feet or squirms in seat
2. Often leaves seat in classroom or in other situations in which remaining seated is expected
3. Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
4. Often has difficulty playing or engaging in leisure activities quietly
5. Is often “on the go” or often acts as if “driven by a motor”
6. Often talks excessively
7. Often blurts out answers before questions have been completed
8. Often has difficulty awaiting turn
9. Often interrupts or intrudes on others (e.g., butts into conversations or games)
B. Demographic and Performance Questionnaire

Questionnaire: indicate answers by circling choice or filling in the blank

1. Which technique most closely describes your approach to this game?
   • Matching game
   • Patterns and sequences
   • Time-based
   • Just clicked randomly
   • None of the above

2. I have to do things several times before I think they are properly done.*
   • Not at all
   • A little
   • Quite a lot
   • A lot
   • Very much
   • Prefer not to answer

3. When I handle money, I count and recount it several times.*
   • Not at all
   • A little
   • Quite a lot
   • A lot
   • Very much
   • Prefer not to answer

4. When I read, I have the impression I have missed something.*
   • Not at all
   • A little
   • Quite a lot
   • A lot
   • Very much
   • Prefer not to answer

5. Age: ______

6. Gender: ______

7. Year: ______

8. Is English your first (or one of your first) language(s)? ______

9. How would you describe your ethnic identity? ______

10. Is there anything you would like to share? ______

11. Major: ______

12. GPA: ______

C. Research Study Consent Form

WASHINGTON STATE UNIVERSITY
Psychology Department

Research Study Consent Form

Study Title: Predicting Concentration, Attention, and Problem Solving Using a Computer Game

Researchers:

PI: Paul Strand, Ph.D., Psychology Department – WSU Tri-Cities, 372-7177
Co-investigator: Dana Grip, Psychology Department – WSU Pullman, danagrip@wsu.edu

You are being asked to take part in a research study carried out by Dr. Paul Strand and Dana Grip. This form explains the research study and your part in it if you decide to join the study. Please read the form carefully, taking as much time as you need. Ask the researcher to explain anything you don’t understand. You can decide not to join the study. If you join the study, you can change your mind later or quit at any time. There will be no penalty or loss of services or benefits if you decide to not take part in the study or quit later.

What is this study about?
This research study is being done to investigate differences in how people process certain types of information, and how those differences relate to concentration, attention, and problem solving. You are being asked to take part because your answers on the human subject pool questionnaire identified you as having or not having difficulties related to concentration, attention, and problem solving. Taking part in the study will require about 60 minutes of your time. You cannot take part in this study if you are under 18.

What will I be asked to do if I am in this study?
If you take part in the study, you will be asked to:
• Play a computer game (10-30 minutes)
• Answer a brief questionnaire (10-20 minutes)
• The questionnaire will ask you questions about your age, your GPA, and a couple of general questions about academic performance.
• You may refuse to answer any question or withdraw from the study.
• As this is a comparative study in which individual results are held and information is confidential, there will be no individual feedback about performance.

Are there any benefits to me if I am in this study?
The potential benefits to you for taking part in this study are: There is no direct benefit to you from being in this study.

Ultimately, results from this research may help us better understand concentration, attention, and problem-solving.

Are there any risks to me if I am in this study?
The potential risks from taking part in this study are:
• Psychological stress and questions about your own functioning: Some subjects may wish to obtain more information about their psychological functioning as a result of completing the self report forms. Such individuals will be provided with contact information for the University Counseling Center.
Will my information be kept private?
The data for this study will be kept confidential to the extent allowed by federal and state law. No published results will identify you, and your name will not be associated with the findings. Under certain circumstances, information that identifies you may be released for internal and external reviews of this project.

- After data are obtained, names will be replaced with participant numbers and the cover page (Consent Form) containing each participant’s name will be removed from the data sheets and stored separately.
- Electronic media will contain only subject numbers.
- All surveys will be maintained in a locked file cabinet.
- Only the PI and co-investigator will have access to the data.

The results of this study may be published or presented at professional meetings, but the identities of all research participants will remain anonymous.

The data for this study will be kept for three years.

Are there any costs or payments for being in this study?
There will be no costs to you for taking part in this study.

You will receive credits toward fulfillment for research participation for taking part in this study.

Who can I talk to if I have questions?
If you have questions about this study or the information in this form, please contact the researcher [Dr. Strand: Psychology Department – WSU Tri-Cities, 372-7177] or the Psychology Department [509-335-2632 in JT 233].

What are my rights as a research study volunteer?
Your participation in this research study is completely voluntary. You may choose not to be a part of this study. There will be no penalty to you if you choose not to take part. You may choose not to answer specific questions or to stop participating at any time.

What does my signature on this consent form mean?
Your signature on this form means that:

- You understand the information given to you in this form
- You have been able to ask the researcher questions and state any concerns
- The researcher has responded to your questions and concerns
- You believe you understand the research study and the potential benefits and risks that are involved.
Statement of Consent

I give my voluntary consent to take part in this study. I will be given a copy of this consent document for my records.

__________________________________  ______________________
Signature of Participant               Date

__________________________________  ______________________
Printed Name of Participant

Statement of Person Obtaining Informed Consent

I have carefully explained to the person taking part in the study what he or she can expect. I certify that when this person signs this form, to the best of my knowledge, he or she understands the purpose, procedures, potential benefits, and potential risks of participation. I also certify that he or she:

- Speaks the language used to explain this research
- Reads well enough to understand this form or, if not, this person is able to hear and understand when the form is read to him or her
- Does not have any problems that could make it hard to understand what it means to take part in this research.

__________________________________  ______________________
Signature of Person Obtaining Consent  Date

__________________________________  ______________________
Printed Name of Person Obtaining Consent  Role in the Research Study