

Copyright

by

Laura Ranee Marusich

2011

**The Dissertation Committee for Laura Rane Marusich Certifies that this is the approved version of the following dissertation:**

**Contracted Spans of Temporal Integration in Adults with Attention Deficit Hyperactivity Disorder**

**Committee:**

---

David Gildea, Supervisor

---

Caryn Carlson

---

Lawrence Cormack

---

Arthur Markman

---

Roberta Tsukahara

**Contracted Spans of Temporal Integration in Adults with Attention  
Deficit Hyperactivity Disorder**

**by**

**Laura Raneë Marusich, B.A.**

**Dissertation**

Presented to the Faculty of the Graduate School of

The University of Texas at Austin

in Partial Fulfillment

of the Requirements

for the Degree of

**Doctor of Philosophy**

**The University of Texas at Austin**

**December 2011**

## **Acknowledgements**

I would like to thank my advisor David Gilden for inviting me to work in his lab, and for everything I've learned from him in the six years since. I also want to express my gratitude to the other members of the Gilden lab who have offered many contributions to this work – Maryam Ezell, Grant Baldwin, and especially Llewyn Paine, who was a great officemate and who continues to be a great friend.

Thank you to my parents, Kyle and Cheryl Marusich, for your example and for your love and unwavering support through the years.

Finally, I'm so appreciative of all the encouragement from my friends during my time in graduate school – Ashley Brock, Allison Cassady, Curt Cooper, Emily Newhouse, Tracy Harper, Rachel Long, Jenni Pacheco, Jessica Pierson, and Josh Sexton – thank you all!

# **Contracted Spans of Temporal Integration in Adults with Attention Deficit Hyperactivity Disorder**

Laura Raneë Marusich, Ph.D.

The University of Texas at Austin, 2011

Supervisor: David Gilden

ADHD is a highly prevalent disorder in both children and adults that involves significant impairment throughout the lifespan, and yet the core cognitive deficits of the disorder are not well understood. Accumulating evidence of dysfunctioning dopamine systems motivated the theory that delay-of-reinforcement gradients are altered in ADHD in such a way that reinforcers must arrive earlier in time following a response for an association between the two to be learned. The current work is motivated by the conjecture that dopamine dysfunction has consequences for the maximum timescales over which connections can be formed, not just in reinforcement learning, but also in the processes of temporal integration and scene formation that allow humans to understand and navigate their world. There is a maximum window of temporal separation over which discrete events can be integrated into a unified experience, and the current experiments indicate that this maximum window of integration is contracted in ADHD.

The experiments included multiple tasks designed so that the participant response required implicit integration over temporal intervals, and the length of those intervals was varied as an independent variable. Adults with and without ADHD completed these tasks, and the strength of temporal integration was measured with respect to interval length and compared between the two groups. This methodology was applied in five types of tasks: rhythmic tapping, spatial cuing, irrelevant feature priming, and two apparent motion tasks. On the whole, this suite of studies was successful in demonstrating a contraction in the maximum interval over which temporal integration can occur in ADHD relative to controls. Two of the tasks, rhythmic tapping and spatial cuing, generated unexpected and interesting results, and several follow-up tasks were designed to further explore these findings. As a result, a somewhat improved tapping task was discovered. This tapping task, as well as the irrelevant feature priming task and one of the apparent motion tasks, demonstrated potential utility for the diagnosis of adults with ADHD.

## Table of Contents

List of Tables .....	x
List of Figures .....	xi
Introduction.....	1
ADHD in Adults .....	1
Overview.....	1
Diagnostic criteria.....	2
Scaled Delay Gradients in ADHD.....	4
Impaired dopamine systems in ADHD.....	5
Altered reinforcement mechanisms in ADHD: The delay-of-reinforcement gradient .....	7
Salience vs. reward .....	10
Contracted Spans of Temporal Integration in ADHD.....	11
Timing in the ADHD literature.....	13
General Methods.....	16
Participants .....	16
Descriptive measures.....	17
Inclusion/exclusion criteria.....	21
Procedure .....	24
Rhythmic Tapping Experiments .....	25
Experiment 1: Continuation tapping.....	28
Method .....	28
Preliminary Data .....	30
Results and Discussion.....	32
Experiment 1a: Continuation tapping follow-up.....	37
Method .....	37
Results and Discussion.....	38
Experiment 1b: Synchronization tapping .....	42

Method .....	42
Results and Discussion.....	43
Diagnostic utility: Rhythmic tapping experiments .....	44
Cuing and Priming Experiments.....	46
Experiment 2: Posner cuing.....	46
Method .....	48
Preliminary Data .....	51
Results and Discussion.....	52
Experiment 2a: Posner cuing follow-up .....	54
Method .....	54
Results and Discussion.....	55
Experiment 3: Irrelevant feature priming .....	57
Method .....	60
Results and Discussion.....	62
Diagnostic utility: Cuing and priming experiments.....	66
Apparent Motion Experiments.....	67
Experiment 4: Group vs. element motion in the Ternus display .....	69
Method .....	69
Results and Discussion.....	73
Experiment 5: Perceived trajectory of apparent motion .....	76
Method .....	78
Results and Discussion.....	82
Diagnostic utility: Apparent motion experiments.....	86
Combined Diagnostic Utility .....	86
Explicit Timing Assessment .....	87
General Discussion and Conclusions.....	89
Summary of Results.....	89
General observations.....	90
Conclusions.....	92

Appendix: EF Questionnaire.....	95
References.....	96

## **List of Tables**

<i>Table 1.</i> Descriptive characteristics of the primary ADHD and control group. ....	22
<i>Table 2.</i> Descriptive characteristics of participants in Experiments 1a and 1b. ....	37
<i>Table 3.</i> Descriptive characteristics of participants in Experiment 2a.....	55

## List of Figures

<i>Figure 1.</i> Illustration of normal and scaled delay-of-reinforcement gradients.....	10
<i>Figure 2.</i> Example of rhythmic tapping sequences by a single participant at five target tempi. ....	26
<i>Figure 3.</i> Coefficient of variation and autocorrelation data in preliminary version of Experiment 1.....	31
<i>Figure 4.</i> Coefficient of variation and autocorrelation data in Experiment 1.....	32
<i>Figure 5.</i> Regression slope coefficients in Experiment 1.....	34
<i>Figure 6.</i> Coefficient of variation and autocorrelation of regression residuals in Experiment 1.....	35
<i>Figure 7.</i> Lag-1 autocorrelation coefficients of drumming records in Experiment 1a..	39
<i>Figure 8.</i> Regression slope coefficients in Experiment 1a. ....	40
<i>Figure 9.</i> Lag-1 autocorrelation coefficients of regression residuals in Experiment 1a.	41
<i>Figure 10.</i> Average deviation from metronome and average absolute deviation from metronome in Experiment 1b.....	43
<i>Figure 11.</i> Differences in autocorrelation from 90 bpm to 60 bpm versus ADHD index and combined DSM symptoms.....	45
<i>Figure 12.</i> Absolute deviations from metronome at 90 bpm versus ADHD index. ....	46
<i>Figure 13.</i> Trial sequence used in Experiment 2. ....	49
<i>Figure 14.</i> Mean RT for valid and invalid trials in preliminary version of Experiment 2.....	52
<i>Figure 15.</i> Mean RT for valid and invalid trials in Experiment 2. ....	53
<i>Figure 16.</i> Mean RT for valid and invalid trials in Experiment 2a. ....	56
<i>Figure 17.</i> Illustration of irrelevant feature priming.....	59

<i>Figure 18.</i> Trial sequence in Experiment 3. ....	61
<i>Figure 19.</i> Color-position interaction as a function of RSI in Experiment 3. ....	64
<i>Figure 20.</i> Decay slopes of cross strength and response priming strength as functions of RSI in Experiment 3. ....	65
<i>Figure 21.</i> Ternus stimuli used in Experiment 4. ....	70
<i>Figure 22.</i> Data and logistic fit from a single participant in Experiment 4. ....	73
<i>Figure 23.</i> Distributions of resampled average alpha values in Experiment 4. ....	75
<i>Figure 24.</i> Possible trajectories of apparent motion. ....	77
<i>Figure 25.</i> Stimuli used in Experiment 5. ....	79
<i>Figure 26.</i> Motion quartet shown as an example of apparent motion in Experiment 5. ....	81
<i>Figure 27.</i> Means and standard deviations of displacement in Experiment 5. ....	84
<i>Figure 28.</i> Relative errors in explicit timing task. ....	89

# **Introduction**

## **ADHD IN ADULTS**

### **Overview**

Attention deficit hyperactivity disorder (ADHD) is a developmental disorder associated with symptoms of hyperactivity, impulsivity, and inattention (American Psychiatric Association (APA), 2000). It has a strong genetic component; twin studies estimate the heritability to be approximately 80% (Faraone et al., 2005). The prevalence of ADHD in the U.S. and worldwide has been estimated between 5 and 10% in children (APA, 2000; Polanczyk et al., 2007; Scahill & Schwab-Stone, 2000). In as many as 60% of childhood cases, symptoms persist into adulthood (Barkley, Fischer, Smallish, & Fletcher, 2002; Biederman, Mick, & Faraone, 2000), and the prevalence of adult ADHD is estimated to be approximately 4% (Kessler et al., 2006).

ADHD is associated with significant educational and social impairment in childhood, extending to difficulties in employment, driving, and relationships in adolescence and adulthood. For example, adults with ADHD are less likely to enter college, more likely to be fired from a job, and more likely to be involved in automobile accidents than adults without ADHD (Barkley, 2002). ADHD was considered for years to be a disorder that only affected children, but it is now clear that a large number of children with ADHD continue to experience significant problems with the disorder as adults.

## **Diagnostic criteria**

Currently, the diagnosis of ADHD in children and adults is made based on the following DSM-IV criteria (APA, 2000):

A) either (1) or (2)

1) at least 6 out of 9 symptoms of inattention have been present for at least 6 months

2) at least 6 out of 9 symptoms of hyperactivity/impulsivity have been present for at least 6 months

B) the onset of symptoms occurred before the age of 7

C) the symptoms cause impairment in at least 2 different settings

D) clinically significant impairment in social, academic, or occupational functioning is present

E) the symptoms of ADHD are not better explained by another disorder.

An individual who meets all these criteria is diagnosed with ADHD and classified into one of three subtypes. Individuals meeting criteria A1 and A2 are classified as Combined Type, those who meet A1 (but not A2) are classified as Predominantly Inattentive type, and those who meet A2 (but not A1) are classified as Predominantly Hyperactive-Impulsive Type.

Applying the same criteria to children and adults can prove problematic for individuals who seek diagnosis for the first time as adults. For example, the number of reported symptoms often decreases with age, and while an adult may continue to

experience significant impairment due to ADHD symptoms, he or she may not experience the criterion cutoff of 6 symptoms. In addition, it can be difficult to recall and accurately report behaviors from early childhood, which is required in order to demonstrate an age of onset before 7 years.

There is some degree of debate about these aspects of the diagnostic criteria for adults. The symptom cutoff criteria were developed for and tested on children, and there is little evidence that they have the same discriminatory power in adults. For example, it has been argued that a four-symptom cutoff would be more appropriate for adults, as four symptoms represents the same percentile in an adult population that a six-symptom cutoff represents in a childhood population (Barkley, Murphy & Fischer, 2007; Kooij et al., 2005). The age-of-onset criterion of 7 years has also been studied, and there appear to be no negative consequences for diagnostic validity if the age-of-onset is relaxed to age 12 (Faraone et al., 2006; Polanczyk et al., 2010). In fact, one study showed that validity was improved if the age-of-onset criterion was relaxed or removed (Applegate et al., 1997).

Another issue in the diagnosis of adults is the assignment of subtype. As described above, the DSM-IV guidelines for diagnosis allow individuals to be diagnosed with one of three subtypes of ADHD: predominantly inattentive, predominantly hyperactive/impulsive, or combined type. In adults, however, categorization into subtypes is not as straightforward as it is in children. Because symptoms of hyperactivity/impulsivity are more attenuated with age than symptoms of inattention (Biederman, Mick, & Faraone, 2000; Hart, Lahey, Brooks, Applegate, & Frick, 1995), many individuals who met the criteria for combined type ADHD as children only meet

the criteria for predominantly inattentive type as adults. It is not clear whether such individuals change subtypes with age, or if the definitions of the subtypes should be different for adults and children (Barkley et al., 2007).

### **SCALED DELAY GRADIENTS IN ADHD**

Despite the high prevalence of ADHD in both children and adults, and the major life impairments associated with the disorder, many aspects of the etiology and core deficits of ADHD are still not well understood. Many constructs have been proposed to account for the behaviors associated with ADHD, including impaired behavioral inhibition and executive function (Barkley, 1997), delay aversion (Sonuga-Barke, 2002), impairment in the regulation of arousal/activation (Sergeant, 2000), temporal processing deficits (Smith, Taylor, Rogers, Newman, & Rubia, 2002; Sonuga-Barke, Bitsakou, & Thompson, 2010), and, of primary interest to the current work, altered reinforcement mechanisms (Johansen, Aase, Meyer, & Sagvolden, 2002; Sagvolden, Johansen, Aase, & Russell, 2005; Scheres, Tontsch, Thoeny, & Kaczurkin, 2010). These and other theories have enjoyed mixed levels of acceptance and success at accounting for the symptoms of ADHD as well as the behavioral and neurobiological findings associated with the disorder.

Recent work in ADHD emphasizes the bridging of neurobiological accounts with symptom-level descriptions of the disorder through intermediate constructs, known as endophenotypes. Endophenotypes are “heritable quantitative traits that index an individual’s liability to develop or manifest a given disease...thought to be more directly

related than dichotomous diagnostic categories to aetiological factors” (Castellanos & Tannock, 2002, p. 617). One promising candidate endophenotype, which is anchored in the neurobiological evidence of impaired dopamine systems in ADHD, is the idea of altered delay gradients (Sagvolden et al., 2005). Briefly, altered delay gradients are proposed to cause a shortening in the timing for effective reinforcement mechanisms. This perspective informs the theoretical motivation of the present experiments - that ADHD might be associated with a more general contraction in the timing of effective causal association and integration of temporal events.

### **Impaired dopamine systems in ADHD**

Sagvolden et al.’s (2005) theory of altered delay gradients is based upon the finding of dysfunctioning dopamine systems in ADHD. There is an abundance of evidence of altered dopamine systems in ADHD, which is briefly reviewed in this section.

Structural imaging studies have reported volumetric reductions in regions of the brain that are rich in dopamine receptors, including the caudate nucleus (Castellanos et al., 1996; Hynd et al., 1993; Montes et al., 2010; Qiu et al., 2009), the globus pallidus (Aylward et al., 1996; Castellanos et al., 1996; Qiu et al., 2009), and the ventral striatum (Carmona et al., 2009). In addition, PET and SPECT imaging studies have reported increased levels of dopamine transporter (DAT) in the striatum in ADHD, although these results have not uniformly replicated (Spencer et al., 2005; 2007). Dopamine transporter

is the primary mechanism for removing extracellular dopamine from the synapse, so increased DAT should entail lower levels of extracellular dopamine.

The implication of the dopamine system in neuroimaging studies is consistent with what is known about the mechanisms of action of stimulant medications, which are commonly prescribed for the treatment of ADHD. Stimulants appear to exert their primary effect on the dopamine and norepinephrine systems (Solanto, 1998). In a PET study that compared occupancies of DAT after differing doses of methylphenidate, Volkow et al. (1998) demonstrated that methylphenidate functions as a DAT blocker. A later PET study examining dopamine receptor availability showed that the blockade of DAT induced by methylphenidate is effective at increasing extracellular dopamine (Volkow et al., 2001).

In addition to differences in DAT levels, Volkow et al. (2007) found evidence that ADHD is also associated with lower levels of spontaneous striatal dopamine release. This finding was assessed indirectly by comparing the change in dopamine levels induced by methylphenidate in an ADHD and control group. Because methylphenidate increases extracellular dopamine by blocking DAT and not by increasing the release of dopamine, the amount of change in extracellular dopamine caused by methylphenidate is a reflection of the amount of dopamine spontaneously released. Methylphenidate produced a smaller change in extracellular dopamine levels in the ADHD group than in the control group, indicating that there was less dopamine released in the first place in ADHD.

The dopamine system has also been implicated in genetic studies, which have demonstrated the most consistent associations for the dopamine receptor type D4 gene

and the DAT gene in ADHD (see Sharp, McQuillin & Gurling, 2010, and Swanson et al., 2007, for reviews).

There is general agreement that ADHD involves a dysfunctioning dopamine system, but less agreement exists about what the specific dysfunction is (affected levels of dopamine release, dopamine transporter, dopamine receptors, or a combination of these), much less what the implications of the dysfunction are for ADHD cognition. However, the model formulated by Sagvolden et al. (2005) involves a compelling integration of the evidence for dopamine dysfunction. ADHD is explained in terms of altered reinforcement mechanisms, grounded in the well-studied connection between dopamine and reinforcement learning.

### **Altered reinforcement mechanisms in ADHD: The delay-of-reinforcement gradient**

The evidence for dysfunctioning dopamine systems in ADHD, especially findings in the striatum, suggests reinforcement mechanisms as a fruitful area of research (Johansen et al., 2010; Sagvolden et al., 2005; Tripp & Wickens, 2008). The dopamine system, particularly the mesolimbic and nigrostriatal pathways, is known to be involved in reinforcement learning (Beninger & Freedman, 1982; Beninger & Miller, 1998; Robbins & Everitt, 1996). Specifically, dopamine is released in the striatum in response to an unpredicted reinforcer, which allows for long-term potentiation (LTP), or the strengthening of synaptic connections.

Studies show that with repeated experience with reinforcement, the phasic release of dopamine transfers from the onset of reinforcement itself to the response or to earlier

cues that predict the delivery of reinforcement. This process of transfer is the biological evidence of a learned association. Lowered levels of extracellular dopamine are hypothesized to cause this transfer process to occur more slowly in ADHD (Sagvolden et al., 2005; Tripp & Wickens, 2008). In other words, more exposures to the response-reinforcement contingency are required for the transfer to take place. Another way of conceptualizing this idea is that each exposure to the response-reinforcement contingency is somewhat less effective at inducing transfer in ADHD. This attenuation of the transfer process has consequences for the time scales over which reinforcement learning can occur.

The ability to form an association between a reinforcer and the behavior that produced it (the operant response) is subject to the decaying memory trace of the behavior. That is, if the reinforcer is delivered immediately after the operant response, the memory trace of the response will still be quite strong and thus available to be associated with the reinforcement. However, if the reinforcer is delayed substantially, the memory trace of the response will have decayed to a point where it is no longer available to be associated with the reinforcement. In this case, any intervening behaviors or cues that occur between the operant response and the reinforcement are more likely to be reinforced. This concept is illustrated by the delay-of-reinforcement gradient (see Figure 1), which relates the amount of time separating response and reinforcement to the probability of learning an association between the two. This inverse relationship between reinforcement delay and probability of reinforcement has long been recognized in the animal learning literature, and the upper limit on delay for successful reinforcement is

known to be on the order of a few seconds (see, for example, Perin, 1943). Anecdotal experience with the training of pets can confirm the fact that immediate reward stimulates faster and more correct learning than delayed reward, and that after a delay of several seconds, reward is virtually ineffective.

Here, we consider the probability of learning in terms of a simple product:

*Probability = memory trace strength ( $\Delta t$ ) \* magnitude of dopamine signal,*

where  $\Delta t$  is the interval between response and reinforcement. The trace strength, as described above, decays with increasing  $\Delta t$ . An impaired dopamine response, as hypothesized in ADHD, would effectively scale the delay-of-reinforcement curve, as shown in Figure 1. In a scaled gradient, compared to a normal gradient, the reinforcer must be delivered more proximally in time for an equivalent probability of association. Sagvolden et al. (2005) proposed that this shortening in the timescale of reinforcement learning could lead to many of the observed symptoms of ADHD.

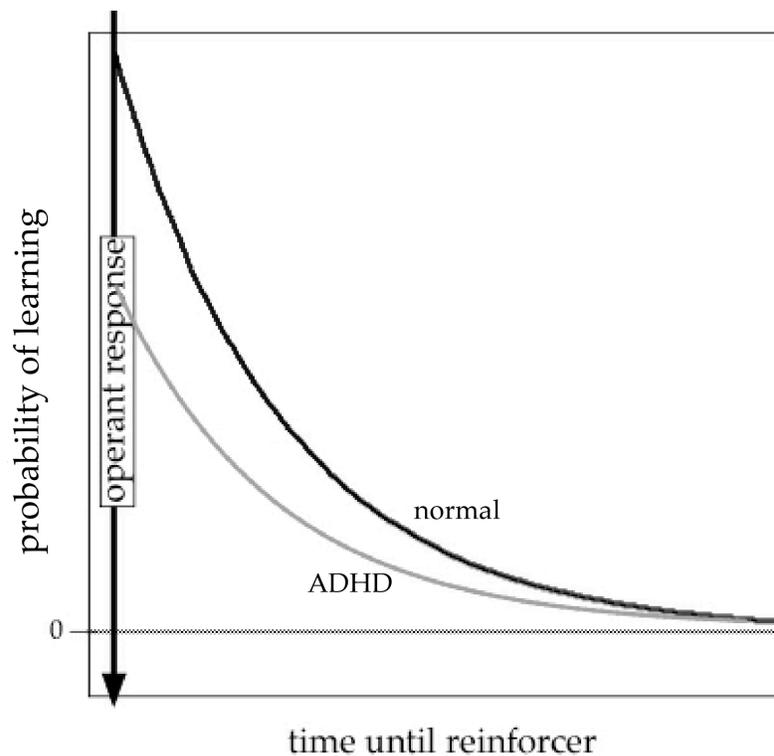


Figure 1. Illustration of normal and scaled delay-of-reinforcement gradients.

### **Salience vs. reward**

Sagvolden et al.'s account of shortened timescales of reinforcement learning in ADHD is compelling. However, dopamine release is known to occur in response not just to rewarding stimuli, but also more generally in response to salient events. This has led to an understanding of dopamine as a signal of salience, which includes, but is not limited to, reinforcing stimuli (Horvitz, 2000; Jensen et al., 2007; Schulz, Dayan, & Montague, 1997). If dopamine function is not limited to reinforcement, then it is obvious that it can affect not only the timing of reinforcement learning, but also the timing of more general types of association formation. This idea is expanded in the following section.

## **CONTRACTED SPANS OF TEMPORAL INTEGRATION IN ADHD**

Every moment of waking life involves the organization of the moment-to-moment flux in action and thought into intelligible behavior, intelligible streams of thought, and a coherent and stable world in which to live. Animals must have a window of time over which discrete episodes can be integrated into unified experiences. This allows for the experience of events in time. However, these windows of integration must have an upper limit; otherwise there would be no end to an event.

A simple example of the kind of temporal integration process described above is the perception of melody. A melody is composed of individual notes that are separated by short temporal intervals. If these intervals were too long, the perception would be not of music, but rather of the occasional arrival of isolated tones. To perceive melodic contour, the length of the intervals between notes must be relatively small. In fact, this limitation is reflected in the construction of metronomes; the slowest tempo built into most metronomes is 40 beats per minute, or one quarter note every 1.5 seconds. The key point here is that all of experience is filled with temporal Gestalts; we do not hear individual notes in music - we hear melody and harmony. We do not see movement sequences - we see gestures and meaningful activity. The whole is always greater than the sum of the parts, and these wholes are formed by the processes of temporal integration creating bridges across time. It is suggested here that scaled gradients in ADHD have consequences not just for reinforcement learning, but for these processes of temporal integration and scene formation, essentially in how time is bridged in making sense of the world.

The notion of temporal integration implies the activity of an implicit memory system - implicit in the sense that the processes that allow us to connect the sequential notes of a melody operate automatically and outside of conscious awareness. In other words, the perception of a melody does not involve the explicit encoding, storage, and retrieval of pitches. As the temporal span of these memory systems is rather short, on the order of a few seconds, they can be regarded as examples of implicit working memory.

This concept of implicit working memory must be distinguished from the concept of working memory used within the ADHD and larger cognitive literatures, which depends exclusively on explicit methods of memory assessment. These methods involve the learning of unrelated nonsense material (unrelated words, numbers, syllables, etc.) followed by immediate or delayed testing. The use of explicit tests has led to a conception of working memory as a short-term buffer served by a central executive in the execution of normal cognitive functions such as reasoning, language comprehension, and categorization (Baddeley, 1986). None of the experiments proposed here would be viewed as measuring working memory in the sense meant by Baddeley. To avoid confusion, temporal integration or temporal bridging will be used throughout this proposal to refer to the use of implicit working memory systems to connect discrete episodes into meaningful events.

The conjectures at the heart of the current work are 1) that there is a maximum span over which temporal integration can occur, governed by delay gradients and the dopamine system, and 2) that dopamine dysfunction in ADHD entails a contraction of the maximum span of temporal integration.

These conjectures were tested with tasks that required integration across temporal intervals. The length of the intervals was varied, and the strength of integration was compared. It was expected that integration strength would decrease with increased temporal interval, and that the rate of decrease would be steeper in ADHD.

### **Timing in the ADHD literature**

This proposal that ADHD might be fundamentally an impairment in time has a surface similarity to a large body of work in the ADHD literature involving temporal processing. However, these two lines of work have different motivations and make different predictions. The following section describes the difference between the assessments of time used in this work and those typically used in the ADHD literature.

For the most part in the literature on ADHD, timing differences are viewed as a secondary consequence of another primary deficit in ADHD. For example, Barkley's (1997) theory of deficient behavioral inhibition predicts impaired working memory, which in turn predicts difficulties with tasks involving time. One aspect of Sonuga-Barke's (2002) dual-pathway model is delay aversion, the notion that people with ADHD make choices that decrease their experience of delay. He argues that behavior that can look like timing impairment could alternatively be interpreted as a preference for shorter intervals or for ending trials and experimental sessions early. In addition, there has been recent speculation that ADHD may involve a pure timing deficit, separate from working memory or other impairments (Smith et al, 2002; Sonuga-Barke et al., 2010). All of these theories predict ADHD impairment at all temporal intervals, and that the degree of

impairment should increase with increasing interval length. In this proposal, however, it is predicted that ADHD and control behavior will be similar at both short and very long intervals, and that differences will only emerge at interval lengths that are too long for integration in ADHD but still within the maximum window of integration for controls.

The theories described above have led to a large number of studies on timing and ADHD. The findings of these studies are summarized in a recent review by Toplak, Dockstader, and Tannock (2006). While many studies found group differences in timing process, the findings as a whole are quite inconsistent. Toplak et al. speculate that the inconsistencies across studies may be explained by the wide array of tasks, modalities, and time intervals assessed. While these differences are certainly likely to contribute to the lack of consistency, a better explanation may be the explicit conception of time that is universal in these studies.

Examples of the timing tasks typically used are duration discrimination, verbal estimation of time intervals, and the reproduction of single time intervals. These tasks require not just the perception of time, but also the explicit judgment of time. Explicit judgments can be made about any property – length, color, sweetness, pitch, etc. A task that uses explicit judgments may provide some information about the processing of the property to be judged, but it will be conflated with the processing involved in performing comparisons and making decisions based on these comparisons. It is known that on any task where individuals with ADHD make judgments about stimuli, they will generate data with increased variability relative to controls. In light of this fact, it is unsurprising that previous studies of temporal processing in ADHD that use explicit judgments do not

show consistent results, and that when they do find a group difference, it is often one of increased response variability in groups with ADHD (Toplak et al., 2006).

This project is concerned with how time is used, not how it is judged. To summarize, the idea of altered delay gradients predicts ADHD impairment relative to normal controls only at a narrow range of temporal interval. The method by which this impairment can be assessed is through tasks that include time as a medium over which processes of integration occur, not as an explicit property to be judged and/or compared. This is a novel approach in the ADHD literature.

The primary goal of the current work was to measure the maximum span of temporal bridges in normal adults and in adults with ADHD. To accomplish this goal, five experiments were designed to measure the strength of temporal bridges across intervals of varying length. For example, one study employed a spatial cuing paradigm to assess the strength of the effect of a cue upon a target, and the temporal separation between cues and targets was manipulated. It was expected that integration strength would decay with increasing temporal interval in both groups, and that the adults with ADHD would demonstrate faster rates of decay than the control groups. The variable to be analyzed depended upon the nature of the study, but in all cases it was a measure of the connection strength between subsequent events, analyzed as a function of temporal interval size. It is noted again that the participants were at no point required to explicitly monitor or judge time. The methods and findings from these studies are described in detail in subsequent chapters.

## **General Methods**

### **PARTICIPANTS**

One large set of participants was recruited to participate in the original versions of all five experiments reported here, which required them to return for multiple experimental sessions. Participants were recruited initially in the various ways described below. After their first experimental session, they were then given the option of providing contact information so that they could return for additional sessions, for which they all received monetary compensation. Additional follow-up versions of two of the experiments required additional participants, who were recruited as described below but only completed a single experimental session.

The participants without ADHD (controls) were recruited from the PSY 301 participant pool at the University of Texas at Austin and received course credit for their initial session.

Participants with ADHD were recruited primarily through the Services for Students with Disabilities (SSD) office at the University of Texas at Austin. To register with the SSD, students must provide documentation of a DSM-IV or ICD diagnosis of ADHD from a mental or medical health care professional. If this diagnosis was not made within the past three years, students must provide documentation of neuropsychological or psychoeducational evaluation in the past three years demonstrating that their assessment of ADHD is current. The SSD forwarded a recruitment email to all students registered with ADHD at the university. This email included the contact information of

the laboratory, which allowed interested students to contact the lab directly in order to schedule an appointment for an experimental session.

A small number of additional participants with ADHD were recruited from ADD Austin, a local psychological practice that specializes in the diagnosis and treatment of ADHD and related disorders. Fliers were posted in the waiting room, allowing interested patients to contact the lab to schedule appointments for participation. All participants recruited from this location carried a current diagnosis of ADHD.

Participants recruited from the SSD and from ADD Austin received monetary compensation for their initial experimental session.

## **DESCRIPTIVE MEASURES**

Participants in the large primary study group completed a series of questionnaires, as well as an assessment of estimated IQ. Participants who were part of the additional study group completed only one of these questionnaires (the CAARS – S:L). All descriptive measures used are described below.

### *Conners Adult ADHD Rating Scale: Self-Report: Long Version*

The Conners Adult ADHD Rating Scale (CAARS; Conners, Erhardt, & Sparrow, 1999) is designed to assess current symptoms of ADHD in adults. This scale has demonstrated good reliability and validity (Conners et al., 1999; Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999). The long version of the self-report form (CAARS – S:L) includes 66 questions and yields scores on nine subscales: inattention/memory problems, hyperactivity/restlessness, impulsivity/emotional lability, problems with self-

concept, DSM-IV inattentive symptoms, DSM-IV hyperactive-impulsive symptoms, DSM-IV total symptoms, ADHD index, and an inconsistency index. Respondents rate their recent experience of each of the 66 items on a 0 (“not at all, never”) to 3 (“very much, very frequently”) scale.

The scores on each subscale can be compared to appropriate population norms for age and gender of the respondent. Scores that fall above the 94<sup>th</sup> percentile are considered clinically relevant elevations, and can indicate that the respondent experiences symptoms of ADHD that meet the DSM-IV criteria for diagnosis.

This scale was used to confirm that participants in the ADHD group were in fact experiencing current symptoms of ADHD, and that participants in the control group did not report clinically elevated symptoms of ADHD. In addition, the CAARS – S:L was used to initially classify participants with ADHD as experiencing primarily inattentive, primarily hyperactive/impulsive, or both types of symptoms. Finally, scores on the CAARS were correlated with several experimental measures in an assessment of diagnostic utility of these measures.

The CAARS - S:L was administered to all participants in the primary study group during their first experimental session, along with two additional questions: 1) “Have you ever been diagnosed with ADHD/ADD?” and 2) “Do you regularly take any psychiatric medications (e.g. antidepressants, stimulants, etc)?” Participants initially recruited for the control group who answered yes to either of these questions were not asked to return for additional experimental sessions, and the data from their first session were excluded from analysis.

### *Depression Anxiety Stress Scales 21 (DASS-21)*

The Depression Anxiety Stress Scales 21 (DASS-21) is a short version of the longer, 42-item DASS (Lovibond & Lovibond, 1995), which assesses depression, anxiety, and stress. Studies have shown the DASS-21 to have good reliability and construct validity (Antony, Bieling, Cox, Enns, & Swinson, 1998; Henry & Crawford, 2003). Respondents rate their recent experience of each of 21 items on a 0 (“did not apply to me at all”) to 3 (“applied to me very much, or most of the time”) scale. The responses that apply to each of the three subscales of depression, anxiety, and stress are summed to generate three severity scores.

Adult ADHD is often comorbid with depression and anxiety (Barkley et al., 2007; Kessler et al., 2006), which can cause a confound in interpreting results of group differences, especially in cognitive measures. Previous work drawing from a similar participant pool to that of the current studies found that the sample with ADHD did not score significantly higher than the control group on measures of depression or anxiety (Shin, 2006). Nevertheless, the DASS-21 was used to collect information on symptoms of depression and anxiety in both groups, in order to assess potential comorbidity confounds.

### *WAIS-III – Vocabulary and Matrix Reasoning Subtests*

Recent meta-analyses of studies have found evidence that ADHD is associated with lower scores on measures of IQ relative to normal controls (Bridgett & Walker, 2006; Frazier, Demaree, & Youngstrom, 2004), although there are many individuals with high IQ that carry a valid diagnosis of ADHD (Antshel et al., 2009). It is the subject of

much debate whether lowered IQ should be considered a confound in studies of ADHD or as a part of the syndrome. In the current studies, estimated IQ information was collected using two subtests from the Wechsler Adult Intelligence Scale 3<sup>rd</sup> edition (WAIS-III; Wechsler, 1993): vocabulary and matrix reasoning. These two subtests were chosen because they have been shown to provide a good estimate of full-scale IQ, and because of all the WAIS-III subtests, they have been shown to produce the smallest gains due to practice (Kaufman & Lichtenberger, 2006). It is likely that many of the participants with ADHD may have completed an IQ assessment within the previous 1-3 years, as a result of the requirements for registering with the SSD, so it was necessary to use subtests that are relatively less susceptible to practice effects. Previous work using a similar participant pool found no significant between-group differences in IQ (Shin, 2006), but the vocabulary and matrix reasoning subtests were used to generate estimated IQ, in order to assess IQ as a potential confound.

#### *Executive Function Questionnaire*

Barkley's model of ADHD supposes a primary deficit in behavioral inhibition, which causes a disruption in executive function. This has led to the idea that a better symptom list might be developed for diagnosing ADHD that addresses executive function impairment, and recent studies have found that self-reported EF symptoms do seem to successfully distinguish ADHD from non-ADHD adults (Barkley et al., 2007; Kessler et al., 2010). Barkley et al. (2007) assessed a large number of potential symptoms in adults with ADHD and in controls and found that seven of these symptoms best discriminated the two groups, and that this discrimination performance was actually

better than the discrimination performance of the current DSM-IV symptom list. To assess the utility of these seven items in the current sample and to compare it with the results of the experimental measures, these seven items were administered in a form referred to from here forward as the EF Questionnaire (Appendix A). Respondents rated their experience of each symptom in the last 6 months on a 0-3 scale, with 0 being “never or rarely” and 3 being “very often.” The total score on this questionnaire has a possible range of 0 to 21.

#### **INCLUSION/EXCLUSION CRITERIA**

##### *ADHD Group*

Inclusion criteria for participants with ADHD were 1) age 18 to 30 years; 2) previous diagnosis of ADHD from a mental or medical health professional; and 3) consent from participants taking stimulant medication to undergo a 24-hour washout period prior to each experimental session to permit assessment in an unmedicated state. The exclusion criterion was 1) uncorrected vision impairment.

##### *Control Group*

Inclusion criteria for control participants were 1) age 18 to 30 years; 2) no history of diagnosis of ADHD; and 3) a *T*-score below 65 (the 94<sup>th</sup> percentile) on the ADHD Index subscale of the CAARS – S:L. Exclusion criteria were 1) the use of psychotropic medications at the time of study; and 2) uncorrected vision impairment.

It is noted that participants were not excluded from the ADHD group on the basis of subthreshold self-report ratings of ADHD behavior. This is because of the findings

that current symptom thresholds for diagnosing ADHD in adults are too conservative (Barkley et al., 2007). In addition, a range of reported symptom severity in the ADHD group would allow for a better correlational analysis between experimental measures and symptom severity.

	Control		ADHD		<i>t</i>
	<i>M</i> ( <i>SD</i> )	<i>n</i>	<i>M</i> ( <i>SD</i> )	<i>n</i>	
Age	18.9 (1.5)	73	21.0 (2.1)	40	6.7***
CAARS					
Inattention	51.3 (9.2)	73	64.9 (10.2)	40	7.2***
Hyperactivity	48.8 (8.0)	73	62.0 (9.8)	40	7.7***
Impulsivity	47.0 (10.0)	73	57.0 (12.4)	40	4.7***
Self-concept	47.9 (10.2)	73	54.2 (11.5)	40	3.1**
DSM inattentive	54.5 (10.8)	73	75.2 (10.3)	40	9.9***
DSM hyperactive	47.8 (10.2)	73	65.3 (11.7)	40	8.2***
Total symptoms	52.0 (10.5)	73	74.5 (10.1)	40	11.0***
ADHD index	49.0 (7.9)	73	61.9 (10.1)	40	7.5***
DASS-21					
Stress	10.5 (7.9)	44	16.8 (8.4)	39	3.5***
Anxiety	6.4 (6.9)	44	9.2 (6.4)	39	2.0
Depression	6.9 (8.8)	44	9.9 (7.9)	39	1.6
WAIS-III Estimated IQ	110.6 (9.9)	39	109.3 (11.8)	35	0.5
Executive Function	7.2 (3.8)	42	12.3 (4.0)	39	5.9***
					$\chi^2$
Sex	20 M, 53 F		16 M, 24 F		1.9

Table 1. Descriptive characteristics of the primary ADHD and control group.  
*\*p* < .05, *\*\*p* < .01. *\*\*\*p* < .001

Table 1 shows the demographic information, as well as the scores on the descriptive measures for all participants in the primary participant group. On average, the

participants with ADHD were about two years older than the control participants, but the relative numbers of males and females in each group did not differ significantly.

Three control participants scored above 65 on the ADHD Index subscale from the CAARS – S:L and were consequently removed from all subsequent analyses. As would be expected, the two groups differed substantially on the CAARS, with the ADHD group scoring higher on all subscales. In addition, the total scores on the EF questionnaire were significantly different in the two groups, with the participants with ADHD scoring about 5 points higher on average than the control participants.

In the analysis of potential group IQ differences, the scores of one participant with ADHD were excluded from analysis due to a refusal to engage seriously with the assessment, and the scores from three control participants were excluded due to a suspected language barrier. Notably, the groups did not differ significantly on the estimated IQ as measured by the vocabulary and matrix reasoning subtests of the WAIS-III. Neither did they differ on the depression or anxiety scales of the DASS-21. The group with ADHD did score significantly higher than the control group on the stress scale, but the scores for both groups on all three scales of the DASS-21 were in the normal to mild range of severity. Because the measures obtained for IQ, depression and anxiety, which had been anticipated as potential confounds, did not differ between the groups, these variables were not entered as covariates in the analysis of experimental data.

Among the 40 participants in the ADHD group, 16 primarily endorsed inattentive symptoms on the CAARS: S-L, 2 primarily endorsed hyperactive/impulsive symptoms,

and 19 endorsed both types of symptoms. Three participants reported subthreshold levels of both symptoms. The small number of participants classified as predominantly hyperactive/impulsive is not surprising, based on previous findings that the symptoms of hyperactivity/impulsivity are more likely to decline with age than the symptoms of inattention (Biederman et al., 2000; Hart et al., 1995).

The hyperactive/impulsive and subthreshold subgroups were too small to be entered into analyses separately. The inattentive and combined subgroups did not show any substantial differences in their performances on the experimental tasks; as a result, all analyses reported here compare the participants with ADHD as a single group to the control participants.

## **PROCEDURE**

Participants in the primary study group attended three separate hour-long experimental sessions to complete all descriptive measures and experiments. The structure of the three sessions was as follows. In the first session, participants completed Experiment 3 (irrelevant feature priming), followed by the CAARS – S:L, and then Experiment 5 (perceived trajectory of apparent motion). In the second session, participants completed Experiment 2 (Posner cuing), followed by Experiment 1 (rhythmic tapping), and then the DASS-21. In the third session, participants completed Experiment 4 (Ternus display), followed by the vocabulary and matrix reasoning subtests of the WAIS-III, the explicit estimation of a time interval, and then the EF questionnaire.

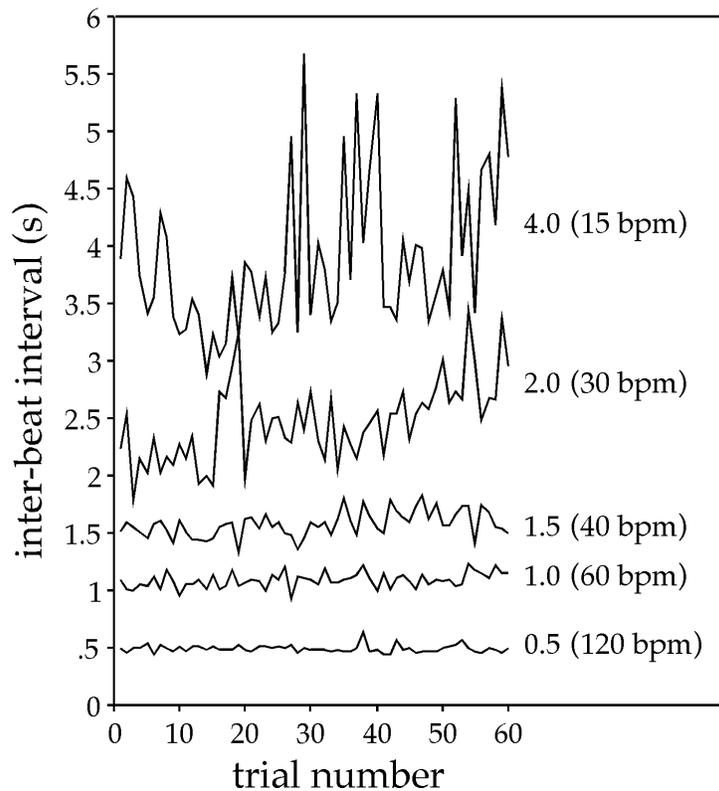
Some of the data from the large study group provoked additional questions, and modified versions of two of the experiments (rhythmic tapping and Posner cuing) were designed to answer these questions. In these follow-up experiments, different participants were recruited to participate. These participants all completed the CAARS – S:L. Different control participants completed the two experiments, while the same participants with ADHD completed both.

### **Rhythmic Tapping Experiments**

One of the most natural ways to study the formation of implicit temporal bridges is through the use of a rhythmic tapping task. The basic idea is to ask participants to produce a stream of drum taps at various tempi and to assess the point where their feeling of rhythm begins to break down. In this task, the “feeling of rhythm” is the evidence of the temporal integration of successive drum taps. At fast tempi, the brief interval between each drum tap falls well within the range of the maximum window for temporal integration, and rhythm is easily felt. At slow tempi, the amount of time between taps exceeds this window, and subsequent taps are not connected; thus, rhythm is not felt.

Early pilot work was performed with normal undergraduates at a wide range of tempi. The purpose of this pilot work was to provide information about the most efficient range of tempi to use in order to span both competent and incompetent drumming in control and ADHD groups. Participants heard 16 counts of a metronome at a given tempo, and they were instructed to begin tapping with the metronome as soon as they were able (the synchronization phase). After 16 counts, the metronome stopped, and the

participants continued on for 60 additional taps (the continuation phase). A characteristic example of the data generated from this task is shown in Figure 2, where the transition from stable to wandering records can be clearly seen. Most participants in this pilot work generated similarly stable performances at 40 bpm and faster. At 30 bpm and slower, the performances began to wander more and more extremely.



*Figure 2.* Example of rhythmic tapping sequences by a single participant at five target tempi.

This general finding of a change of performance regime between 40 and 30 bpm in pilot work marked this tempo range as the region of interest for capturing both stable and wandering drumming in control groups. Contracted spans of temporal integration predict that ADHD performance should become unstable at faster tempi than control

performance, so the tempo range for this experiment was extended to include a faster tempo - 60 bpm.

One issue in the study of rhythmic feeling is how best to objectively measure its presence from a time series of inter-tap intervals (ITIs). Madison (2001), in a study of isochronous finger tapping in healthy adults at a range of tempi (0.4 s to 2.2 s ITIs), examined the lag-1 correlation coefficient, as well as the autocorrelation function out to lag 15 in each of the tapping sequences. To estimate the amount of drift in each series, he calculated the slope of the best-fitting regression line through the series, as well as the difference in the mean interval produced between the first half of the series and the second. Drift is an important measure of tapping performance, in that it indicates how well the performer can feel the target tempo. Most studies of tapping assume independent timing errors distributed around a stationary mean target interval. This assumption is often at least approximately met when the target intervals are short (e.g., less than one second). Few studies examine longer intervals as Madison does, because this requires that drift be taken into account or somehow removed from the data prior to analysis. However, this drift is an important indicator of the loss of rhythmic feeling and is therefore a focus of assessment in the current work.

In Experiment 1, two measures of the data were initially assessed. The coefficient of variation (calculated as the standard deviation divided by the mean) of the ITIs produced provides information about the relative magnitude of errors generated at the various tempi, and the autocorrelation coefficient at lag-1 provides information about the degree of wandering in the performances. As drumming performance begins to wander

away from a target tempo, the successive inter-tap intervals (ITIs) become more correlated with each other, as in a random walk. Truly independent errors around a stationary mean are perfectly uncorrelated and unpredictable.

The measures of drift employed by Madison (regression slope and the difference between the first and second half of the series) were not initially used here. This was because the series length used by Madison was somewhat short (35 taps). When drift occurs in such a short series, it can typically be estimated as a linear drift. In longer series such as those used in the current study (60 taps at a minimum), the series can wander away from the target tempo and back again, which would not be captured by a measure of linear drift.

## **EXPERIMENT 1: CONTINUATION TAPPING**

### **Method**

#### *Participants*

Thirty-nine participants with ADHD and forty-four control participants from the primary participant group completed this study. None were excluded from analysis.

#### *Materials*

A Roland Handsonic MIDI drum pad was used for the collection of data. This device provided participants with good tactile and auditory feedback. It had the look and feel of a drum pad, and it was set to play a simulated conga drum sound through the participant's headphones with every tap. The Handsonic was connected to a MIDI sequencer, which played the synchronizing metronome beats and collected the drum-

tapping records. A computer screen positioned directly in front of participants displayed large, non-technical text, which the participants read while tapping. The purpose of this text was to prevent participants from subdividing the longer intervals by counting.

### *Procedure*

The experimenter brought participants into a small experiment room and explained the nature of the task. Participants were given the opportunity to practice tapping on the drum, and they received feedback as to whether their tapping was strong enough to produce a MIDI signal in the sequencer. The experimenter then played an example of the metronome click and explained that in each of the four conditions, the metronome would play for 16 counts, after which the participant should continue tapping to the same beat. The participants were also instructed to read aloud from the text in front of them while tapping.

As a guideline, the experimenter recommended that participants listen to the metronome for several counts, begin to tap along with the metronome for several more counts, and then begin reading as well. This procedure was implemented in the preliminary version of the study (described below), and it allowed the participants to acquire a good feel for the synchronization rhythm and to smoothly transition from the synchronization phase to the continuation phase with minimal confusion about the concurrent reading task.

After the 16-beat synchronizing metronome, the continuation phase lasted for 3 minutes. This procedure was repeated for each of four tempo conditions: 60, 40, 30, and 20 bpm.

A preliminary version of this task was performed with a smaller sample and with only three of the four tempo conditions (60, 40, and 30 bpm). The results from this preliminary study are described below, in order to provide a point of comparison for the results obtained in Experiment 1.

### **Preliminary Data**

Twenty-two participants (11 with ADHD, 11 control) between the ages of 18 and 30 completed a preliminary version of this experiment (Gilden & Marusich, 2009) that included only three tempo conditions: 60, 40, and 30 bpm. All participants with ADHD were University of Texas students and received monetary compensation for their participation. Approximately half of the control participants were University of Texas students who received course credit for their participation, and the remainder were individuals from the Austin community who received monetary compensation for their participation.

The coefficients of variation (CV) and lag-1 autocorrelation coefficients at each tempo condition are displayed in Figure 3. The CV reflects the overall accuracy of the tapping performance, and the autocorrelation provides a measure of the amount of wandering that occurs in the performance.

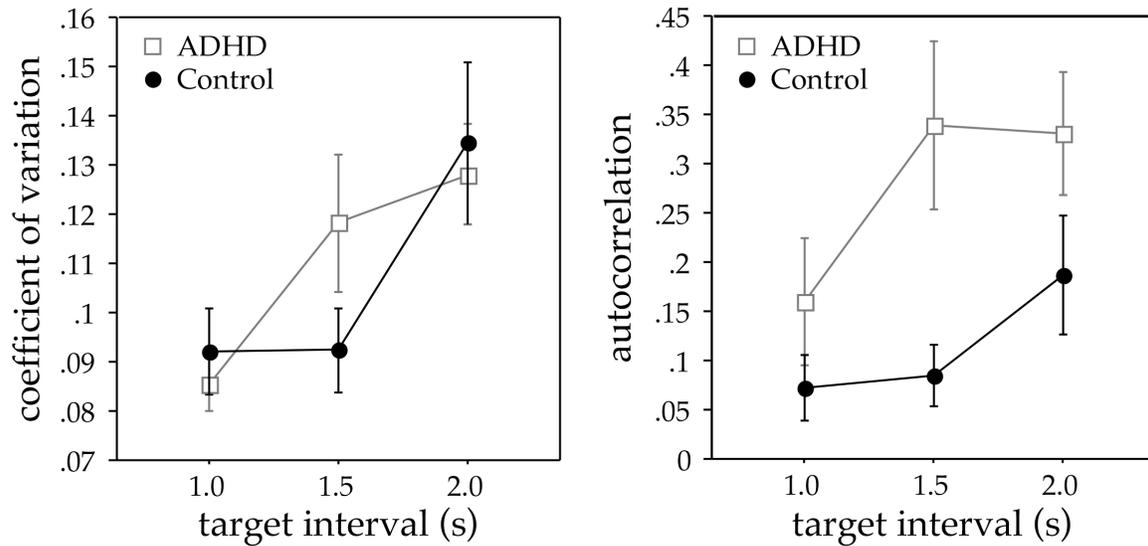


Figure 3. Coefficient of variation and autocorrelation data in preliminary version of Experiment 1.

In both groups, there is clearly a change in drumming stability as the tempo decreases from 60 bpm to 30 bpm. Both the CV and the lag-1 autocorrelation increase with slower tempo. At 60 bpm, both groups are fairly accurate and exhibit a small amount of wandering. At 30 bpm, both groups are similarly inaccurate and demonstrating higher amounts of wandering. At 40 bpm, however, the groups differ. The control group appears to be equally accurate and stable at 40 bpm as at 60 bpm, while the group with ADHD generates data that mimics the 30 bpm condition. These preliminary results suggested that the maximum span of temporal integration is between 1.5 and 2 seconds in typical adults, and that it is contracted to between 1 and 1.5 seconds in adults with ADHD.

## Results and Discussion

The coefficient of variation and autocorrelation at lag 1 at each of the four tempo conditions in Experiment 1 are shown in Figure 4. The coefficient of variation results only show a main effect of group, where the group with ADHD is more variable than the control group at all tempo conditions, and a main effect of tempo, where performance is more variable at slower tempo in both groups. There does not appear to be any interaction between the two variables.

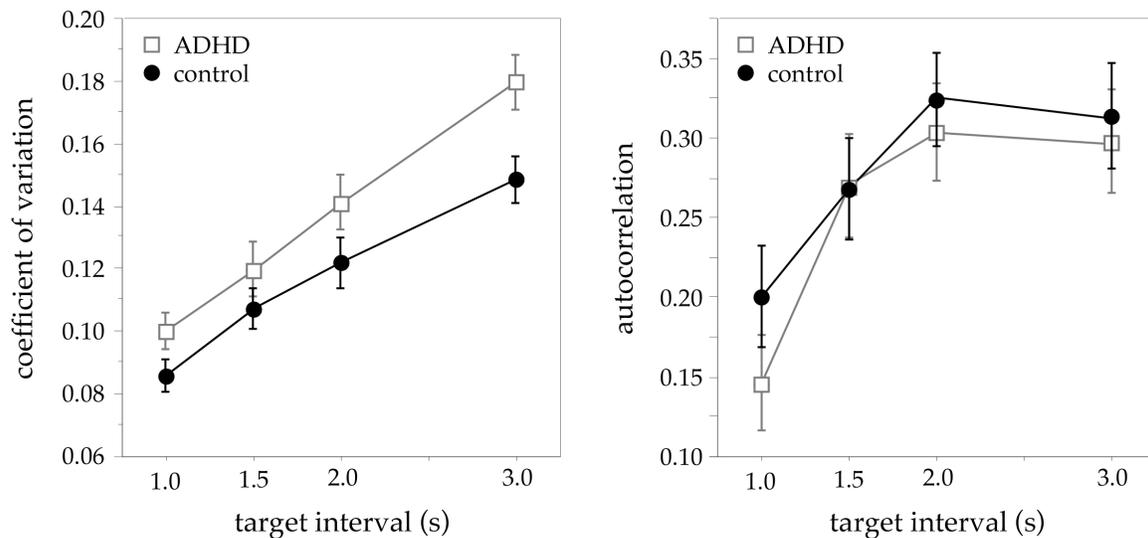


Figure 4. Coefficient of variation and autocorrelation data in Experiment 1.

Additionally, the CV results do not show two regimes of performance, as was seen in the preliminary data. There is no clear regime of competent drumming versus incompetent drumming in either group. In the autocorrelation data, there may be more evidence for this idea of two regimes, as the autocorrelation coefficients do not monotonically increase with interval size, but instead seem to asymptote around 0.3 in

both groups. Both groups start out with low autocorrelations at 60 bpm, and both groups demonstrate high autocorrelations at the slowest tempi, but the control group seems to stabilize after 30 bpm, while the ADHD group may be stabilizing after 40 bpm. However, these differences are clearly not significant.

Another aspect of this data that differs from the preliminary findings is that the autocorrelation coefficients are much higher at the fastest tempo in this study than they were in the preliminary version. Upon examination of individual drum records, it became clear that there were some participants who, at the fastest tempo where rhythm should be easily felt, did not generate stable performances centered around the target tempo. Yet their records did not look like the wandering, large-error records seen at long intervals in most participants. Instead, their records contained a steady, linear drift, with low variability apart from this linear trend. It is not clear that a steady linear drift should be considered as an indication of loss of rhythm the same way that a wandering record is, but the coefficient of variation and the autocorrelation coefficient will be similarly high in both these cases. To attempt to assess wandering separately from linear drift, a linear regression was performed to assess the slope of the best-fitting line through each record, as done in Madison (2001). Then the variability and the lag-1 autocorrelation of the residuals were computed. A truly wandering performance would have not just a linear drift away from the target tempo, but hills and valleys in the record, which will be preserved in the residuals from a linear regression. The residuals from a wandering record should thus still generate high CV and  $r$  when the linear trend is removed. Similarly, in cases where the performance was stable and centered at the target tempo, the

regression will have no effect on the measures of rhythmic feeling, as there is no linear trend to remove. It should simply lower the CV and  $r$  in performances where these measures are inflated due the participant having an overall tendency to either steadily speed up or steadily slow down through the length of the performance.

The results of this analysis are shown in Figures 5 and 6. Figure 5 shows the absolute value of the slope coefficients from the regression, averaged across participants in each tempo condition. In both groups, the linear drift increases with increasing target interval, but there are no significant differences in this trend between the two groups.

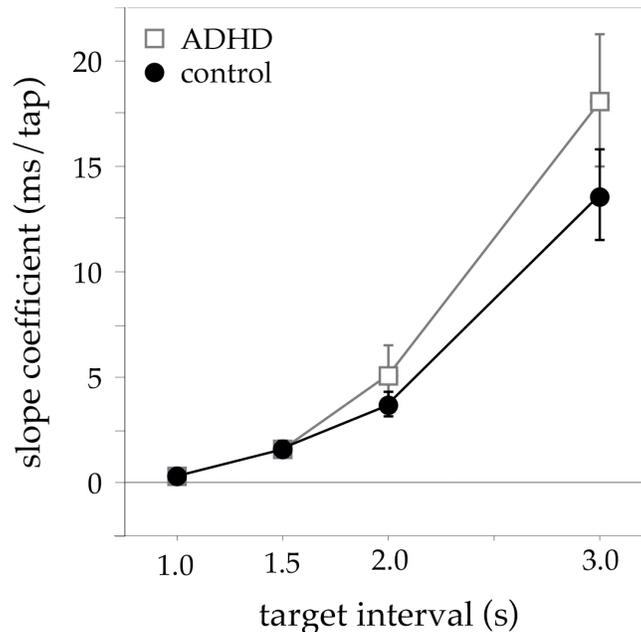


Figure 5. Regression slope coefficients in Experiment 1.

Figure 6 shows the variability in the regression residuals. To make this measure similar to the coefficient of variation, the standard deviation of the residuals is divided by the mean of the original performance. Figure 6 also shows the lag-1 autocorrelation of

the regression residuals. These figures show that removing the linear drift from the performances put the range of values in the same range as in the preliminary study, but the pattern of results doesn't change extensively. Again, the CV increases monotonically with increasing interval, and the group with ADHD has a higher CV at every tempo. The autocorrelation results are more in line with the pattern of the preliminary results, but with much more overlap between the groups.

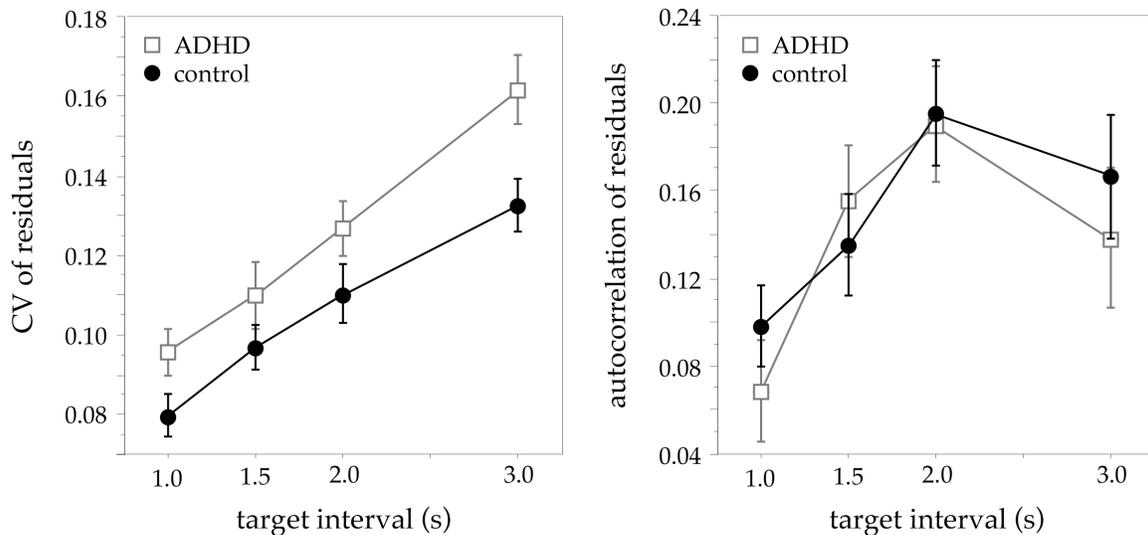


Figure 6. Coefficient of variation and autocorrelation of regression residuals in Experiment 1.

The results were not expected, in that the two groups did not differ, but they were also different from the preliminary data in other unexpected ways. In particular, the control group in this study does not show the same performance as the control group in the preliminary study. Their statistics at the fastest tempo are much higher in this study. In fact, many people generated coefficients of variation of above 15% at 60 bpm, which

is quite a large amount of error. This suggests a possible reason that two distinct regimes of drumming performance are not demonstrated in this data; perhaps a large number of participants in both groups are already in an incompetent drumming regime even at the fastest tempo. It is speculated that the secondary reading task was more disruptive in Experiment 1 than it was in the preliminary version of the study. The text was different in the two versions of the study, and perhaps it was less difficult in the preliminary version in ways that were not immediately apparent. If both groups were primarily in the regime of incompetent drumming at all tempos, then large group differences would not be expected. Differences are only expected to occur in the transition between competent and incompetent drumming.

To assess this conjecture, a follow-up study was designed. In this study, the secondary reading task was removed from the procedure. This increased the risk that participants would subdivide long intervals by counting, but they were explicitly asked to avoid doing this, and their data were analyzed after each performance to look for evidence of counting, which would be demonstrated by a CV that never increased, even at long intervals. This was never the case. In addition, the 20 bpm condition was removed, as it had been extremely difficult for the participants to complete, and a 90 bpm condition was added in order to ensure that the transition point between stable and wandering drumming performance was captured in the design.

## EXPERIMENT 1A: CONTINUATION TAPPING FOLLOW-UP

### Method

#### *Participants*

Twenty-three control participants and fourteen participants with ADHD completed this study (see Table 2). The control participants were recruited through the PSY 301 subject pool and received course credit for their participation. The participants with ADHD were recruited through the SSD and received \$20 for their participation.

	Control		ADHD		<i>t</i>
	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	
Age	19.8 (1.6)	15	21.0 (1.9)	14	2.1*
CAARS					
Inattention	51.3 (8.1)	15	68.1 (10.0)	14	5.6***
Hyperactivity	51.6 (8.6)	15	63.4 (6.2)	14	4.5***
Impulsivity	46.4 (6.6)	15	58.1 (9.6)	14	4.4***
Self-concept	47.9 (8.6)	15	57.1 (10.3)	14	3.0**
DSM inattentive	55.7 (11.3)	15	78.4 (10.8)	14	6.0***
DSM hyperactive	53.3 (9.0)	15	70.1 (11.3)	14	5.0***
Total symptoms	56.0 (10.9)	15	78.0 (10.6)	14	6.0***
ADHD index	50.8 (7.6)	15	64.1 (6.9)	14	5.4***
					$\chi^2$
Sex	11 M, 12 F		7 M, 7 F		0.02

Table 2. Descriptive characteristics of participants in Experiments 1a and 1b.

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

#### *Procedure*

In this follow-up study, the tempo conditions were changed to 90, 60, 40, and 30 bpm, and the number of synchronizing metronome beats was lowered from 16 to 8. In

addition, participants were not given a secondary reading task; instead, they were simply asked not to count, either aloud or mentally. In all other ways, the procedure was identical to that of Experiment 1.

## **Results and Discussion**

The results of Experiment 1a are shown in Figure 7. The coefficient of variation was not included, as it displayed the same pattern of results as in Experiment 1, in that it rose steadily with tempo and was again somewhat higher in the group with ADHD. It seems to be the case that the CV is not a reliably useful measure for depicting the transition between stable and wandering drumming performance, which is not entirely surprising, as the quantities of mean and standard deviation are not meaningful when the record they describe is non-stationary. In the preliminary version of the study, the CV showed two clear regimes of performance in the same way that the autocorrelation did. In subsequent versions, it instead shows a continuous increase with increasing ITIs. The lag-1 autocorrelation, however, does still appear to have two regimes. This suggests that the autocorrelation may indeed be a more reliable tool to measure the stability of a drumming performance.

It is clear from the figure that there is a difference between the two groups in terms of where performance is stable and where it is wandering. In the group with ADHD, a dramatic change in performance occurs between 90 bpm and 60 bpm ( $t(13) = 3.05, p = .005$ ). In the control group, however, a dramatic change occurs between 60 bpm and 40 bpm ( $t(22) = 3.65, p < .001$ ).

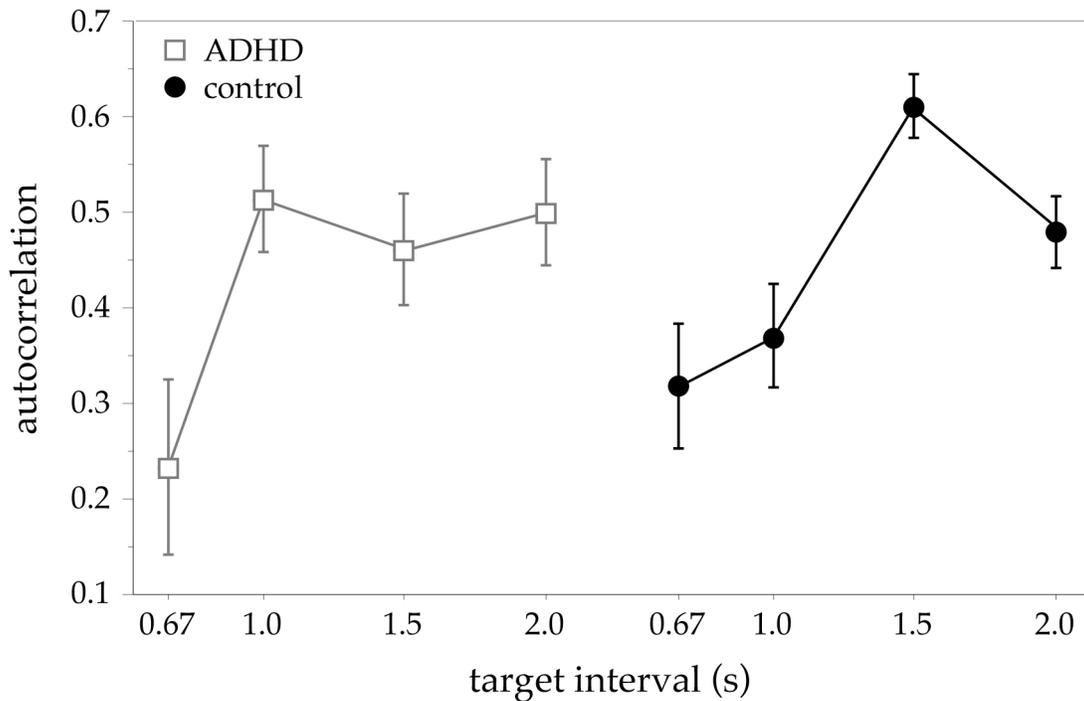


Figure 7. Lag-1 autocorrelation coefficients of drumming records in Experiment 1a

Although the data from this follow-up study were closer in shape to the expected results, the actual values of the autocorrelation coefficients were still somewhat elevated in relation to the preliminary study. Inspection of individual records revealed that there were again instances of purely linear drift, even at the fast tempi. For this reason, a linear regression analysis was again applied to each record. The slope coefficients from this analysis are plotted in Figure 8. The pattern of results here is somewhat different from that of Experiment 1. The slopes of the two groups are only significantly different at 60 bpm ( $t(35) = 2.08, p = .02$ ), which is the tempo at which their autocorrelation coefficients are also different. This raises the possibility that all of the difference in the autocorrelation between the two groups is due to a difference in the amount of linear

drift. This possibility was assessed by analysis of the lag-1 autocorrelation of the residuals.

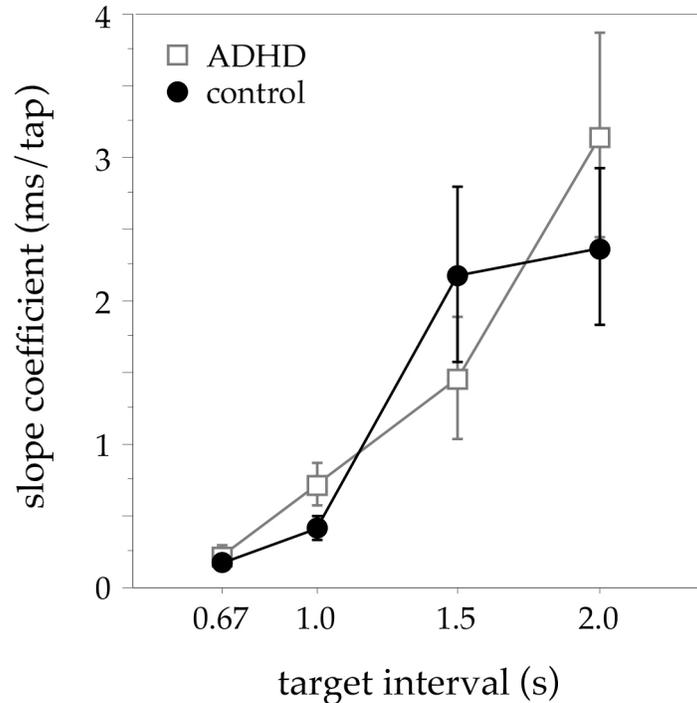


Figure 8. Regression slope coefficients in Experiment 1a.

The results of this analysis of the residuals are shown in Figure 9. It is clear that the data turn over at different intervals in the two groups. Again, the change in regime occurs between 90 and 60 bpm in the group with ADHD ( $t(13) = 2.29, p = .02$ ), and between 60 and 40 bpm in the control group ( $t(22) = 3.31, p = .003$ ). This would indicate that perhaps there are two components to the change in regime from stable to unstable drumming performance. The first is an increase in a linear drift away from the target tempo. The second is the additional predictability that comes from the hills and valleys

of a wandering performance. In this follow-up study, it is clear that both components transition at smaller intervals in the ADHD group than they do in the control group.

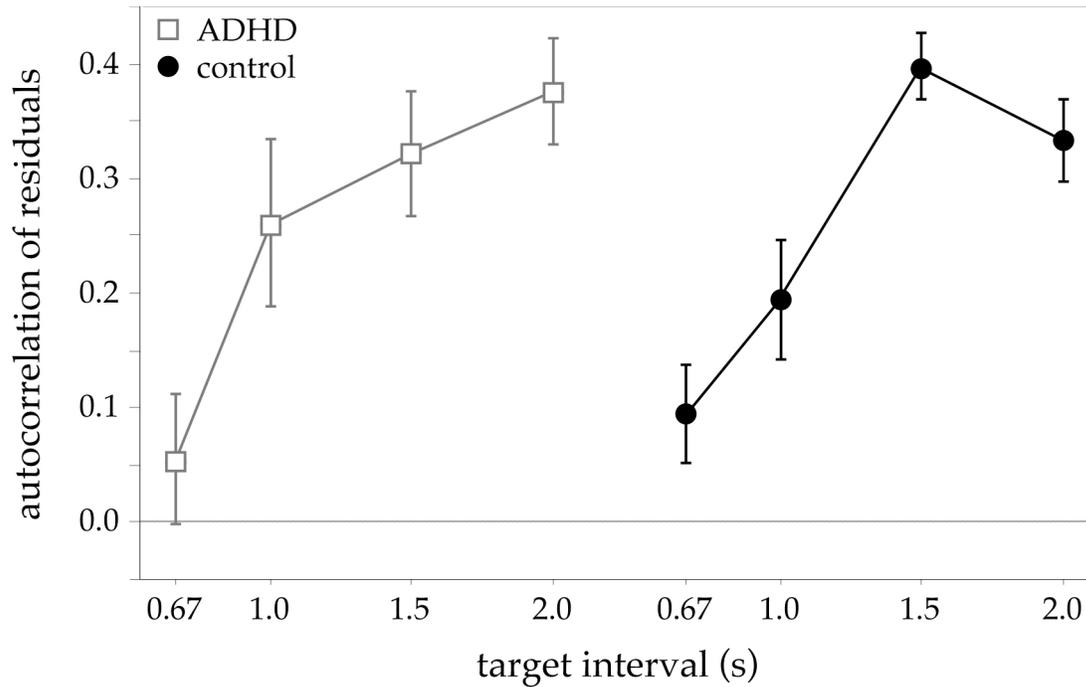


Figure 9. Lag-1 autocorrelation coefficients of regression residuals in Experiment 1a.

The findings of Experiment 1a indicate that rhythmic drumming is in fact a useful task for discriminating between the ADHD and control limits of temporal integration. It does seem to be the case, however, that a secondary reading task is not only unnecessary, but also may push the performances into an unstable zone even at fast tempo, depending on the text being read.

Another version of a tapping task was devised that was conducted at the same time as Experiment 1a. Instead of a synchronization phase followed by a continuation phase, this task would only include synchronization to a metronome. The measure of

interest in this case would not be how far away the performance drifted from the target tempo, but instead the average magnitude of the deviations from the metronome, as well as the direction of those errors (i.e., whether the taps were early or late, in respect to the metronome). This study is described below.

## **EXPERIMENT 1B: SYNCHRONIZATION TAPPING**

### **Method**

#### *Participants*

The same participants who completed Experiment 1a also completed this study. Three of the control participants were excluded from analysis in the synchronization study, however, because they did not consistently play with the metronome; at various points in the recording, these participants would tap midway between metronome beats instead.

#### *Procedure*

The metronome was played for 68 beats at each tempo. Participants began tapping at some point within the first 8 beats, and recording began on the 9<sup>th</sup> beat, so that every performance record was 60 beats long.

#### *Analysis*

The average deviation from the metronome was computed for each participant at each tempo condition. The autocorrelation was not used in this version of the study, as the metronome provides a continuous correcting signal that prevents drift from occurring in the record and which would produce negative lag-1 autocorrelations.

## Results and Discussion

The average deviations from the metronome are shown in the left panel of Figure 10. Both groups tend to have negative deviations, which indicates being early, or ahead of the beat, but this was not universal, so the absolute value of the deviations was taken. This measure is shown in the right panel of Figure 10. It is clear that both groups generate their largest deviation at the longest target interval of 2 s per beat. In the control group, the smallest deviations occur at 60 bpm, whereas in the group with ADHD, the smallest deviations occur at 90 bpm. The difference between the two groups at 90 bpm is quite large ( $t(32) = 2.2, p = 0.03$ ). The shape of these curves suggests that there may be an optimal tempo for tapping with a metronome, and that this optimal tempo may be somewhat faster in ADHD.

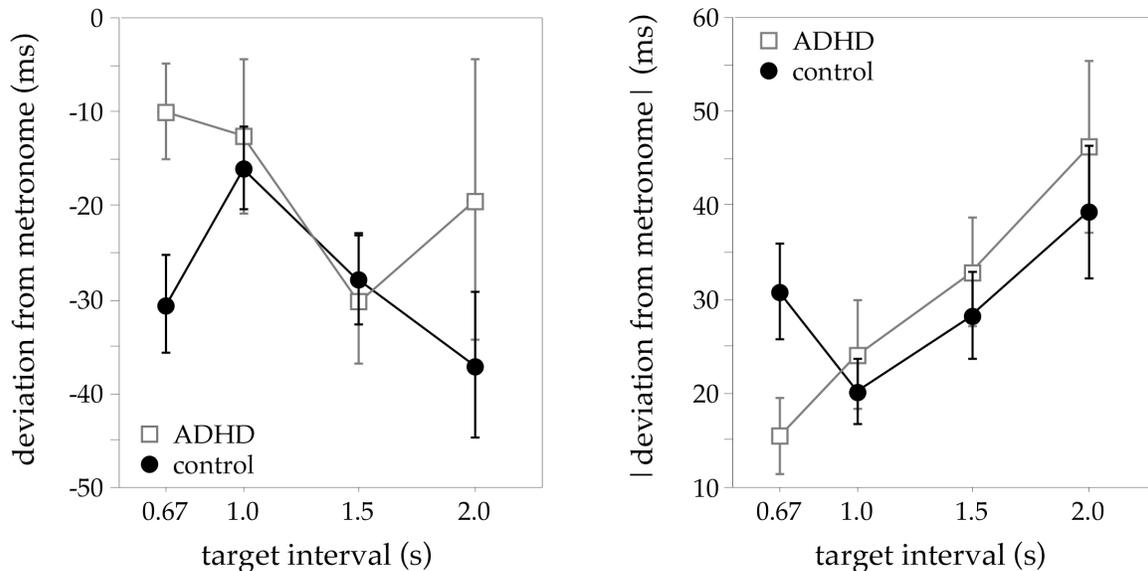


Figure 10. Average deviation from metronome and average absolute deviation from metronome in Experiment 1b.

## DIAGNOSTIC UTILITY: RHYTHMIC TAPPING EXPERIMENTS

The large group differences seen in the results of Experiments 1a and 1b suggest that these measures might be useful to examine as predictors of symptom severity and of group membership. The participants who completed these studies also completed the CAARS – S:L questionnaire, so the experimental measures could be explored in relation to whether the participant was in the control or ADHD group, and in relation to his or her reported ADHD symptoms.

As shown in Table 2, the members of the two groups differed greatly in their reported symptoms. The ADHD index subscale of the CAARS – S:L has been found to be most useful for distinguishing adults with ADHD from normal controls (Conners, Erhardt, & Sparrow, 1999), and in this sample, the ADHD index is a significant predictor of group membership in a logistic regression analysis ( $\chi^2 = 21.6$ ). Including additional CAARS subscale scores as predictors does not improve the model, in terms of significance and in terms of the Akaike information criterion (AIC).

In Experiment 1a, a single measure that can represent the difference between the two groups is the change in the autocorrelation coefficient from 90 bpm to 60 bpm. The value of this measure is 0.05 in the control group and 0.28 in the group with ADHD ( $t(35) = 2.2, p = 0.02$ ). As shown in Figure 11, this measure is somewhat correlated with the ADHD index subscale ( $r = 0.26$ ) and the total DSM-IV symptoms subscale ( $r = 0.23$ ). When entered in a logistic regression, it is a significant predictor of group membership ( $\chi^2 = 4.6$ ). However, the ADHD index is a better predictor of group membership ( $\chi^2 = 21.6, AIC = 29.5$ ), and this model is not improved by adding the drumming measure ( $\chi^2 =$

22.5, AIC = 30.6). Nonetheless, the fact that this measure is able to predict group membership with over 70% accuracy and is correlated with the two CAARS measures suggests that it may be tapping into the core cognitive deficits of ADHD.

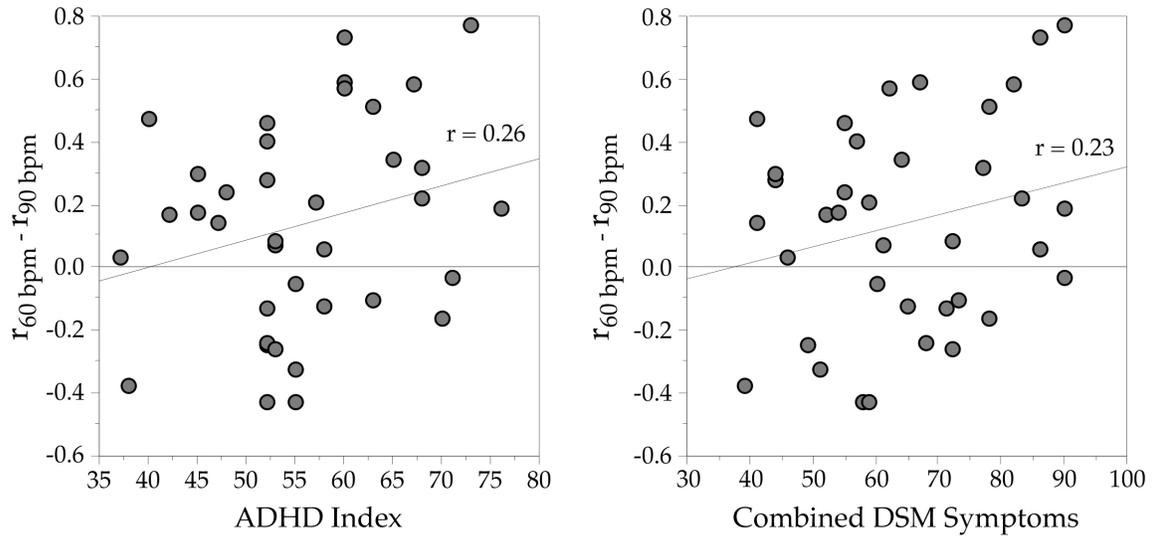


Figure 11. Differences in autocorrelation from 90 bpm to 60 bpm versus ADHD index and combined DSM symptoms

In Experiment 1b, a single measure that can represent the difference in group performance is the absolute average deviation from the metronome signal at 90 bpm. The value of this measure is 31 ms in the control group and 16 ms in the group with ADHD ( $t(32) = 2.2, p = 0.03$ ). This measure also correlates (negatively) with the ADHD index subscale ( $r = -0.34$ ), as shown in Figure 12. When entered in a logistic regression, it is a significant predictor of group membership ( $\chi^2 = 5.1$ ). This measure is substantially correlated with the measure from Experiment 1a ( $r = -0.38$ ), and they do not predict group membership better combined than alone. This suggests that the two measures may be tapping into the same construct, and that one or the other may be used productively on

its own. If this is the case, the synchronization task might be the most efficient to implement in diagnostic scenarios, as it requires records of shorter length than the continuation task.

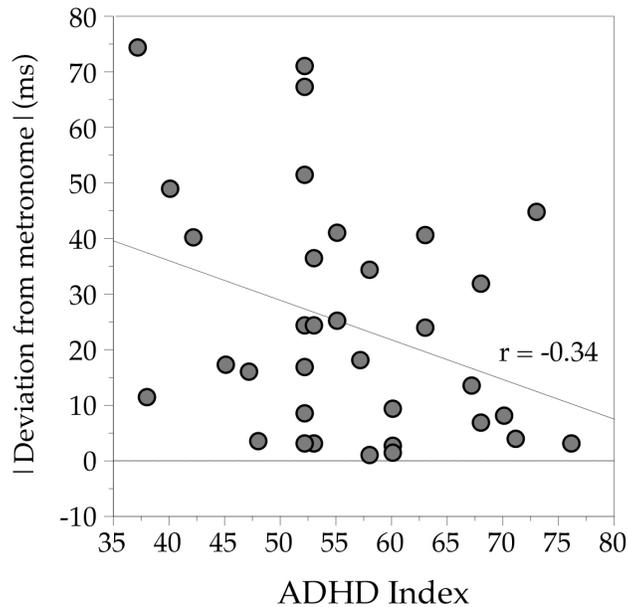


Figure 12. Absolute deviations from metronome at 90 bpm versus ADHD index.

## Cuing and Priming Experiments

### EXPERIMENT 2: POSNER CUING

The experience of rhythm is only one demonstration of temporal integration. Many well-studied cognitive paradigms also involve the bridging across time of discrete events. In Experiment 2, a variation on the Posner spatial cuing paradigm (Posner, 1980) was used to assess the maximum window of temporal integration in adults with and without ADHD.

In the standard version of the Posner paradigm, participants respond to the location of a target, preceded on every trial by a cuing stimulus. This cue is either valid, meaning that it indicates the same location in which the subsequent target appears, or invalid, meaning that it indicates a different location. Typically, the number of valid trials is greater than the number of invalid trials, allowing observers to learn that the cue is informative. This manifests in faster reaction times on valid trials than on invalid trials. This reaction time (RT) difference between valid and invalid trials is referred to as the validity effect.

The Posner paradigm has previously been adopted for the assessment of children and adults with ADHD, primarily to assess hemispheric deficits in visuo-spatial attention (Carter, Krener, Chaderjian, Northcutt, & Wolfe, 1995; Epstein, Conners, Erhardt, March, & Swanson, 1997; McDonald, Bennett, Chambers, & Castiello, 1999; Swanson et al., 1991; Tomporowski, Tinsley, & Hager, 1994). In these studies, the prediction was that the RT difference between valid and invalid trials would diminish or disappear for one half of the visual field. Huang-Pollock and Nigg (2003) conducted a meta-analysis of this body of work, looking not just for visual field effects, but also for differences in more general attentional processes. The authors found that the results from these studies were generally small and heterogeneous, and they concluded that the Posner paradigm may not be useful for measuring attentional differences between ADHD and control populations.

The motivation for the use of the Posner paradigm in the current work is unrelated to the issues that have guided previous investigations in ADHD populations. The interest

here is in the element of temporal bridging that connects the cue and target, forming a unified cue-target event. The validity effect in RT would be the evidence of this integration, and it should decay with increased temporal separation between cue and target. At short cue-target separations, say less than a second (Posner cuing studies are typically run in this regime), it is the case that observers use the cue, responding quickly when it is valid and more slowly when not. At longer separations, say more than several seconds, it is predicted that RTs of valid and invalid trials will approach a common value that is simply the time to mark that a target has appeared.

Because the connection between cue and target is an example of temporal bridging in the same way as is the connection between successive beats in the feeling of rhythm, it was predicted that intervals of 2-3 seconds would be sufficient to annihilate all validity effects in normal adults. The hypothesis to be tested was whether the adults with ADHD would produce validity effects that are extinguished on slightly shorter time scales than controls. This hypothesis has not been previously tested; earlier studies of Posner cuing in ADHD populations used short delays precisely because they promote strong validity effects.

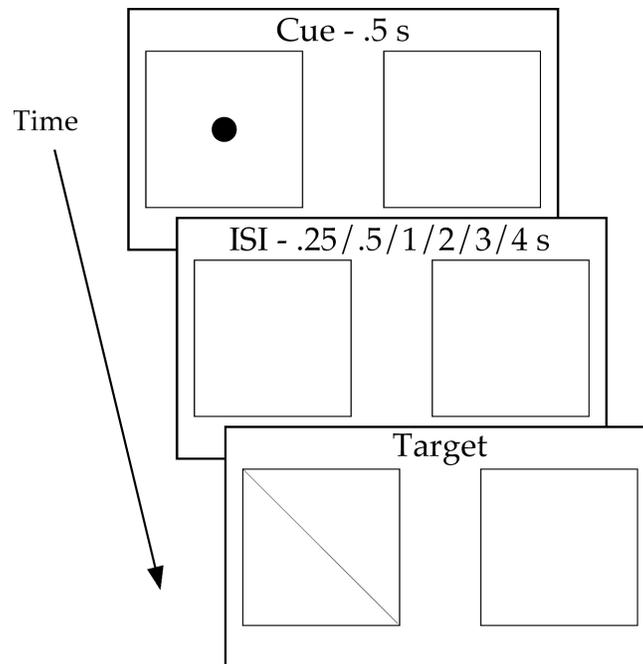
## **Method**

### *Participants*

Thirty-nine participants with ADHD and fifty-one control participants from the primary participant group completed this study. None were excluded from the analysis.

### *Stimuli*

On every trial, two boxes appeared, just to the left and right of the center of the screen, respectively (see Figure 13). In one of the boxes, a cue consisting of a small black dot appeared for 500 ms. After an interstimulus interval (ISI) of variable length (250, 500, 1000, 2000, 3000, or 4000 ms), the target appeared. The target consisted of a diagonal line extending from the upper left corner to the lower right corner of one of the two boxes. The target remained visible until participant response. The location of the cue was randomized from trial to trial, and the cue was valid (correctly predicted the target location) on 80% of trials.



*Figure 13.* Trial sequence used in Experiment 2.

### *Procedure*

Participants were instructed to respond to the appearance of the target stimulus with a keypress of “1” when the target appeared in the left box and “2” when the target appeared in the right box. Participants were asked to respond as quickly as possible, and they were given feedback after practice and test blocks about their overall speed and accuracy. This feedback was designed to ensure that participants completed test blocks quickly enough to maintain rates of accuracy between approximately 90 and 95%.

All participants completed one practice block of 60 trials and four test blocks of 120 trials each. In total, participants responded to 64 valid and 16 invalid trials at each of the six different ISI conditions, which were randomly interleaved within each block.

### *Analysis*

The variable of interest in this experiment was the validity effect (the difference in RTs between valid and invalid trials) and the rate at which this validity effect decays with increased ISIs. For each participant, the mean RT for valid and invalid trials was computed at each ISI, and the validity effect was then calculated at each ISI by subtracting the mean for valid trials from the mean for invalid trials.

A preliminary version of this task was performed with a smaller sample and with only five of the six ISI conditions (no 4 s condition). The results from this preliminary study are described below, in order to provide a point of comparison for the results obtained in Experiment 2.

## **Preliminary Data**

Twenty-nine students at the University of Texas (15 ADHD, 14 control) participated in a preliminary version of this experiment. All participants with ADHD were recruited through the SSD office and received monetary compensation for their participation. All of the control participants were recruited from the PSY 301 subject pool and received course credit for their participation.

In this preliminary version of the experiment, there were five ISI conditions: .25, .5, 1, 2, and 3 seconds. Participants completed one practice block of 50 trials and three test blocks of 150 trials each.

Data from the two groups are illustrated in Figure 14. It is clear that for both groups, there is a relatively large validity effect at small ISIs, and that the magnitude of the effect diminishes at larger ISIs. The validity effect first reaches zero at 2-second intervals in the group with ADHD, while it does not vanish until 3-second intervals in the control group. This is reflected in the large group difference in the validity effect at the 2-second ISI condition ( $t(27)=2.25, p=0.02$ ).

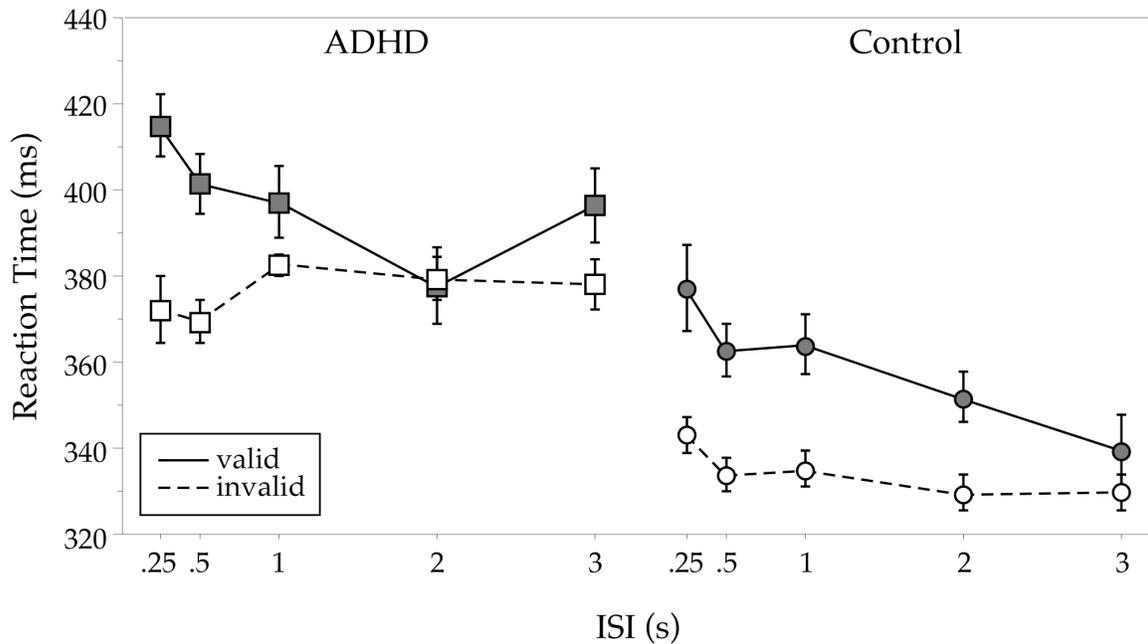


Figure 14. Mean RT for valid and invalid trials in preliminary version of Experiment 2.

## Results and Discussion

The results of Experiment 2 are shown in Figure 15. These data are similar to the results of the preliminary version of the study, in that the group with ADHD has slower reaction times overall, and both groups show the largest validity effects at the shortest ISIs. However, the results are unexpectedly different from the preliminary study in that neither group demonstrates real decay. They both maintain substantial validity effects out to 2- and 3-second delays between the cue and target. The fact that there is no clear data signature that indicates that the maximum interval for cue-target integration has been surpassed presents a major problem for this task as an assessment of temporal integration limits.

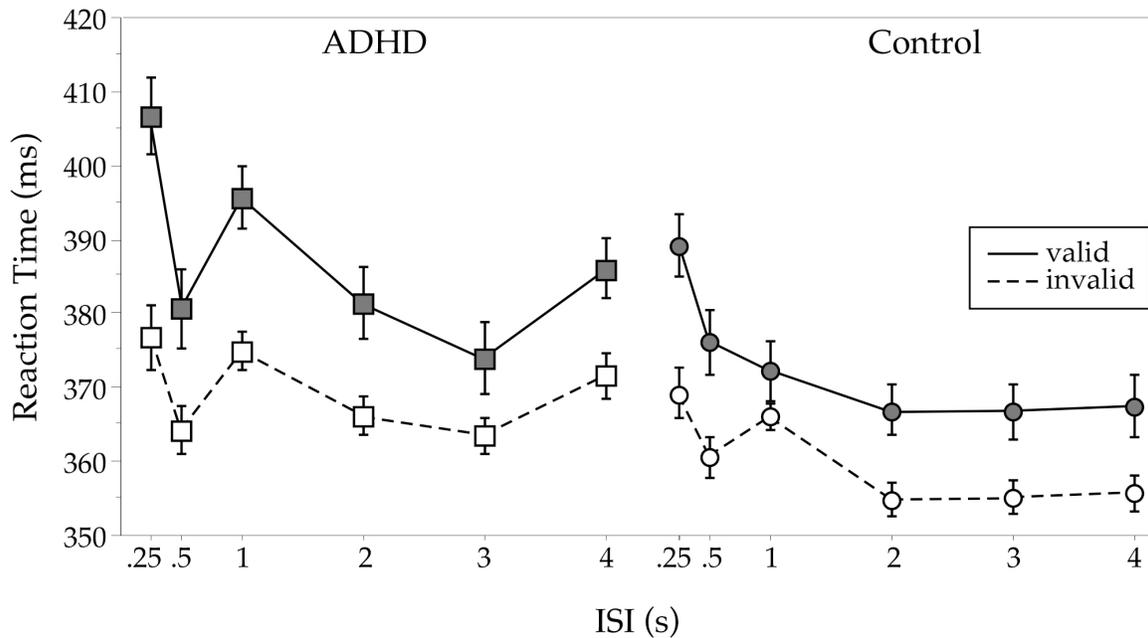


Figure 15. Mean RT for valid and invalid trials in Experiment 2.

The lack of decay that occurred in Experiment 2 was quite surprising, as all previous pilot work using this paradigm in normal undergraduates had shown a clear diminishing of the validity effect to zero at 2-3 second intervals. The only unique feature of the design of this version of the paradigm was the inclusion of a 4-second condition. It is speculated that adding this 4-second condition changed the nature of the task, because half of the trials involve wait times of over a second, and the average wait time is nearly 2 seconds. With such a large proportion of the trials involving long wait times, it is speculated that implicit temporal integration of the cue and target is traded for a more explicit strategy of noting the position of the cue and consciously orienting towards that location to prepare for the arrival of a target. If this strategy were indeed adopted, there

is no reason to think that the validity effect would ever disappear, no matter the size of the ISI.

To determine whether decaying validity effects could be replicated when the 4-second condition was removed from the design, a follow-up version of Experiment 2 was implemented. This follow-up study is described below.

## **EXPERIMENT 2A: POSNER CUING FOLLOW-UP**

### **Method**

#### *Participants*

Fifteen control participants and fourteen participants with ADHD completed this study (see Table 3). The control participants were recruited through the PSY301 subject pool and received course credit for their participation. The participants with ADHD were recruited through the SSD and received \$20 for their participation.

	Control		ADHD		<i>t</i>
	<i>M (SD)</i>	<i>n</i>	<i>M (SD)</i>	<i>n</i>	
Age	19.3 (3.0)	15	21.0 (1.9)	14	2.5*
CAARS					
Inattention	48.3 (9.5)	15	68.1 (10.0)	14	5.5***
Hyperactivity	46.7 (4.7)	15	63.4 (6.2)	14	8.2***
Impulsivity	42.3 (4.8)	15	58.1 (9.6)	14	5.6***
Self-concept	45.8 (9.3)	15	57.1 (10.3)	14	3.1**
DSM inattentive	54.3 (11.6)	15	78.4 (10.8)	14	5.8***
DSM hyperactive	47.1 (6.6)	15	70.1 (11.3)	14	6.8***
Total symptoms	51.4 (8.3)	15	78.0 (10.6)	14	7.6***
ADHD index	46.5 (6.3)	15	64.1 (6.9)	14	7.2***
					$\chi^2$
Sex	9 M, 6 F		7 M, 7 F		0.3

Table 3. Descriptive characteristics of participants in Experiment 2a.  
 \* $p < .05$ , \*\* $p < .01$ . \*\*\* $p < .001$

## Results and Discussion

The results of Experiment 2a are shown in Figure 16. The first thing to note is that both groups demonstrate decay of the validity effect in this follow-up design that only includes ISIs up to 3 seconds. The validity effect is greatest at the .25-second interval condition, and it decreases fairly smoothly to zero in the 3-second condition. However, the slopes of this decay in the validity effect are not significantly different in the two groups. If anything, the group with ADHD demonstrates slightly slower decay.

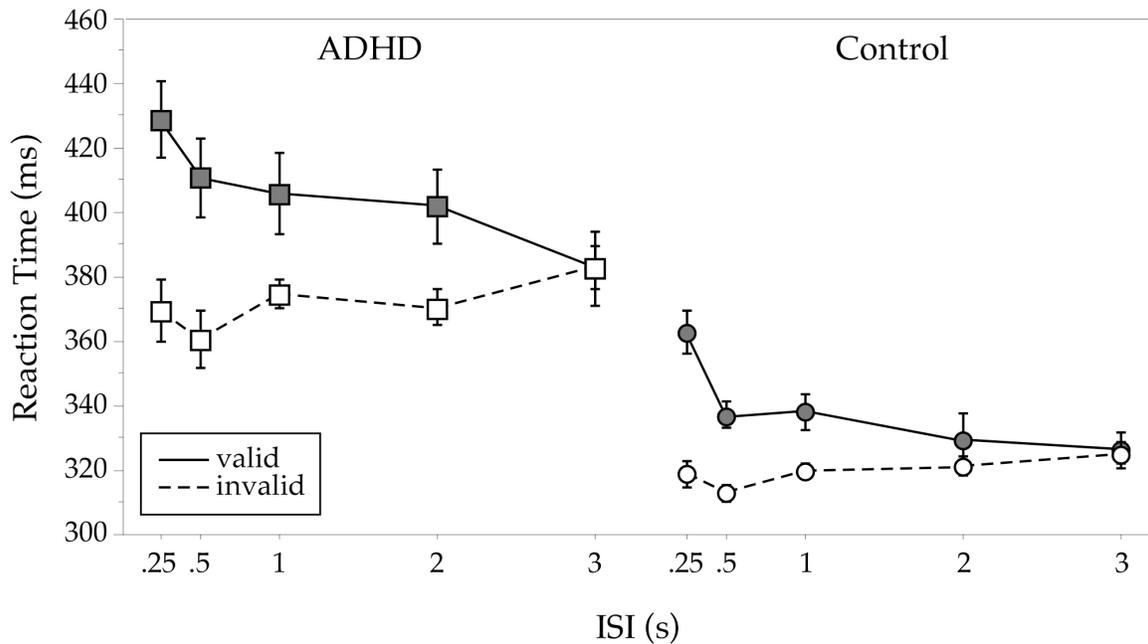


Figure 16. Mean RT for valid and invalid trials in Experiment 2a.

The results of this improved follow-up study suggest that perhaps the Posner cuing paradigm is not in fact the best methodology for assessing the limits of temporal integration. This could be because of large variation in the strategy used to complete the task. The cue could be used implicitly, in which case the expected decay would occur, or explicitly, by consciously attending to the cued location, in which case decay might not be expected to occur. In addition, observers could be switching strategies during the task. This was hypothesized as a possible reason for the increase in the validity effect in the ADHD group in the pilot study. At the longest interval, it was suggested that this group switched from an implicit to an explicit strategy. In all three replications of the task, there is a large amount of variability between subjects in terms of the magnitude of the validity effect and the nature of its decay. It is only on the average that the shape above appears.

In the follow-up study, there is a great deal of variability in whether or not decay occurs in both groups, even though on the average, both groups decay. If there is strategy switching, it is difficult to determine where and when it occurs in a single participant. It seems preferable to use a task where an explicit strategy is either prevented, as in the drumming study, or not helpful to the task, as in the priming and apparent motion experiments.

### **EXPERIMENT 3: IRRELEVANT FEATURE PRIMING**

Sequential priming is a nearly universal outcome in studies of speeded response. Reaction time latencies appear to be extraordinarily sensitive to repetitions in response selections and response executions made on the previous few trials. In typical RT studies, these correlations between sequential trials are rarely of interest. In fact, these priming effects are one of the reasons that multiple observations in every treatment cell are required in RT experiments; multiple observations allow priming effects to be averaged over when estimating the effect of the independent variable of interest. However, in the current study, the RT effects that occur as a result of sequential priming are the object of inquiry. The fact that RTs are influenced by prior responses and prior stimuli is evidence that some type of temporal bridging is occurring across trials. Experiment 3 was designed to assess the conjecture that moment-to-moment bridging, manifested by patterns of sequential priming, is attenuated at somewhat shorter temporal intervals between trials in ADHD than in normal controls.

The specific priming paradigm used here was selected because it has been used in other contexts and rarely fails to produce consistent and replicable priming patterns (Feinstein, 2007; Paine, 2010). The paradigm is outlined in Figure 17. On every trial, a red or blue dot appears in one of two possible positions. Although there are four distinct trial types, participants respond only to color, so there are only two possible responses. Extensive work (Feinstein, 2007; Hommel, 1998; Huang, Holcombe, & Pashler, 2004; Paine, 2010) with normal undergraduates has demonstrated an effect known as irrelevant feature priming. Reaction times are influenced by the color of the stimulus from the previous trial, and this influence is modulated by the position of the previous stimulus, even though position is irrelevant to participant response.

The crossed interaction in Figure 17 indicates an implicit coupling of the two dimensions (one relevant to response, one irrelevant), such that fast responding only occurs when there is change consistency, i.e. both dimensions change or neither dimension changes. That is, if trial  $n+1$  is identical in both color and position to trial  $n$  (case 1 in Figure 17), RTs on trial  $n+1$  will be fast. RT will also be fast if both color and position change (case 3). RTs are slow when only one dimension changes while the other is repeated (cases 2 and 4).

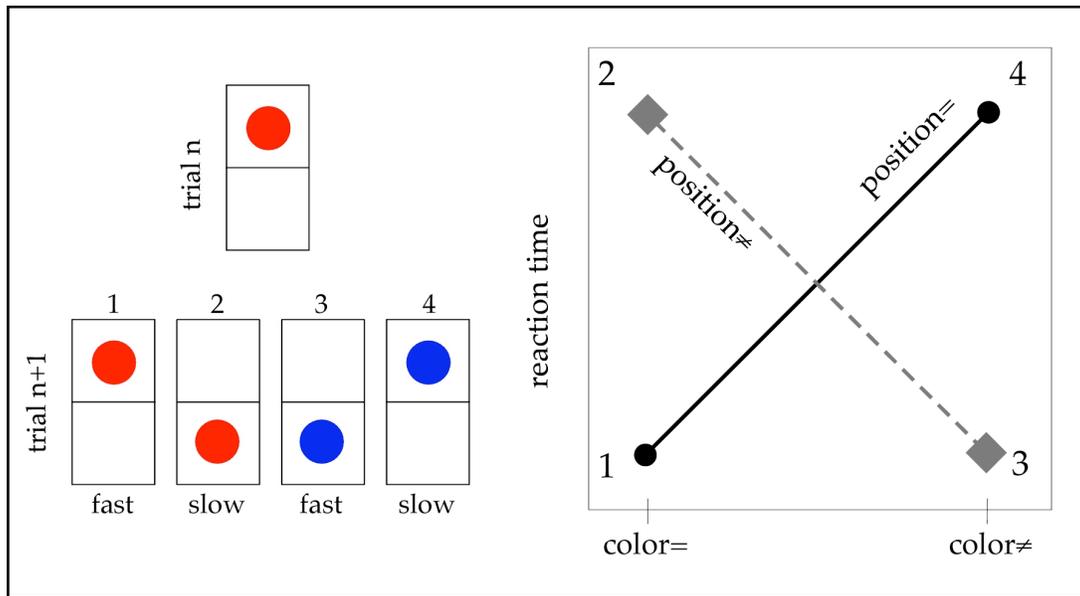


Figure 17. Illustration of irrelevant feature priming.

The crossed interaction was expected to behave as a measure of the strength of temporal integration between successive trials. At short intervals between trials, observers would integrate the two dimensions; as interval length increases, the amplitude of the interaction would monotonically diminish. At sufficiently long times, previous trials would exert no influence on reaction time. The rate of decay of the amplitude of the crossed interaction was compared between the ADHD and control group. Normal response priming, which is just the effect of the relevant feature upon the RTs of subsequent trials, was also examined. Again, delays up to a few seconds comprise the critical range of temporal intervals for the measurement of the span of temporal integration.

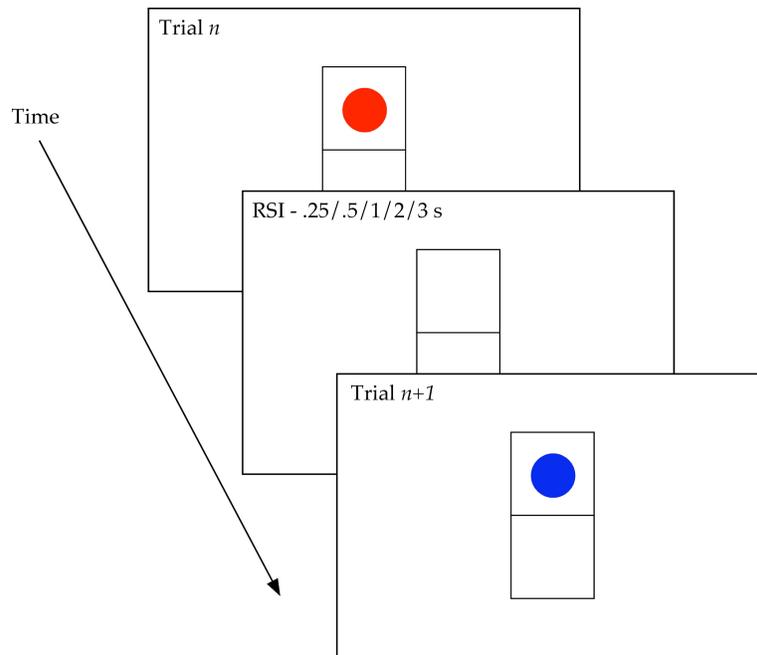
## **Method**

### *Participants*

Forty participants with ADHD and sixty-six control participants from the primary participant group completed this experiment. None of the control participants were excluded from analysis; however, the data from one participant with ADHD was removed because only 3 of the 5 blocks were completed.

### *Stimuli*

Throughout the experiment, a figure consisting of two adjacent squares, one above the other, remained on the screen. On each trial, a red or blue dot appeared in one of the two squares (see Figure 18). The color and position of the dot varied randomly from trial to trial. Response-to-stimulus intervals (RSIs) varied by block and could be 0.25, 0.5, 1, 2, or 3 seconds.



*Figure 18.* Trial sequence in Experiment 3.

### *Procedure*

Participants were instructed to respond with a key press of “1” when a red dot appeared on the screen and “2” when a blue dot appeared. Participants were asked to respond as quickly as possible and were given feedback after practice and test blocks about their overall speed and accuracy. This feedback was designed so that participants completed test blocks quickly enough to maintain rates of accuracy between approximately 90 and 95%.

All participants completed one practice block of 60 trials at an RSI of 0.5 s, after which they completed one test block of 120 trials at each RSI condition. The order of the 5 test blocks was randomized for each participant, and the order of stimuli presented was randomized within each block.

### *Analysis*

Prior to the analysis of sequential dependencies, all main effects due to color and position were removed on a block-by-block basis for each participant. Then the mean RT was computed for each of the cells illustrated in Figure 17: color (same or different) x position (same or different). The amplitude of the crossed interaction in each block of data was computed as

$$\text{cross strength} = \frac{1}{2}[(\text{RT}(2)-\text{RT}(1)) + (\text{RT}(4)-\text{RT}(3))]$$

where RT(n) refers to the mean RT for one of the four cells notated in Figure 17. This expression is equivalent to subtracting the slope of the position-equal line from the slope of the position-not-equal line. Large values indicate a strong cross, while a value of 0 indicates no interaction.

Normal response priming was calculated by averaging over position (equal and not equal) as

$$\text{response priming strength} = -\frac{1}{2}[(\text{RT}(2)+\text{RT}(1)) - (\text{RT}(4)+\text{RT}(3))].$$

For each subject, the best fitting regression line was calculated for both cross strength and priming strength as a function of ISI. This represents a measure of rate of decay. The slopes of these two regression lines were then compared between groups.

### **Results and Discussion**

The results of Experiment 3 are shown in Figure 19. As is commonly seen in reaction time experiments with ADHD populations, the overall reaction time is somewhat slower in the ADHD group than in the control group. In addition, both groups show a

main effect of RSI on reaction time, where RT is fastest at the .5-second interval. Both groups demonstrate crossed interactions between color and position, and these crosses are generally tilted, indicating an overall response priming effect as well.

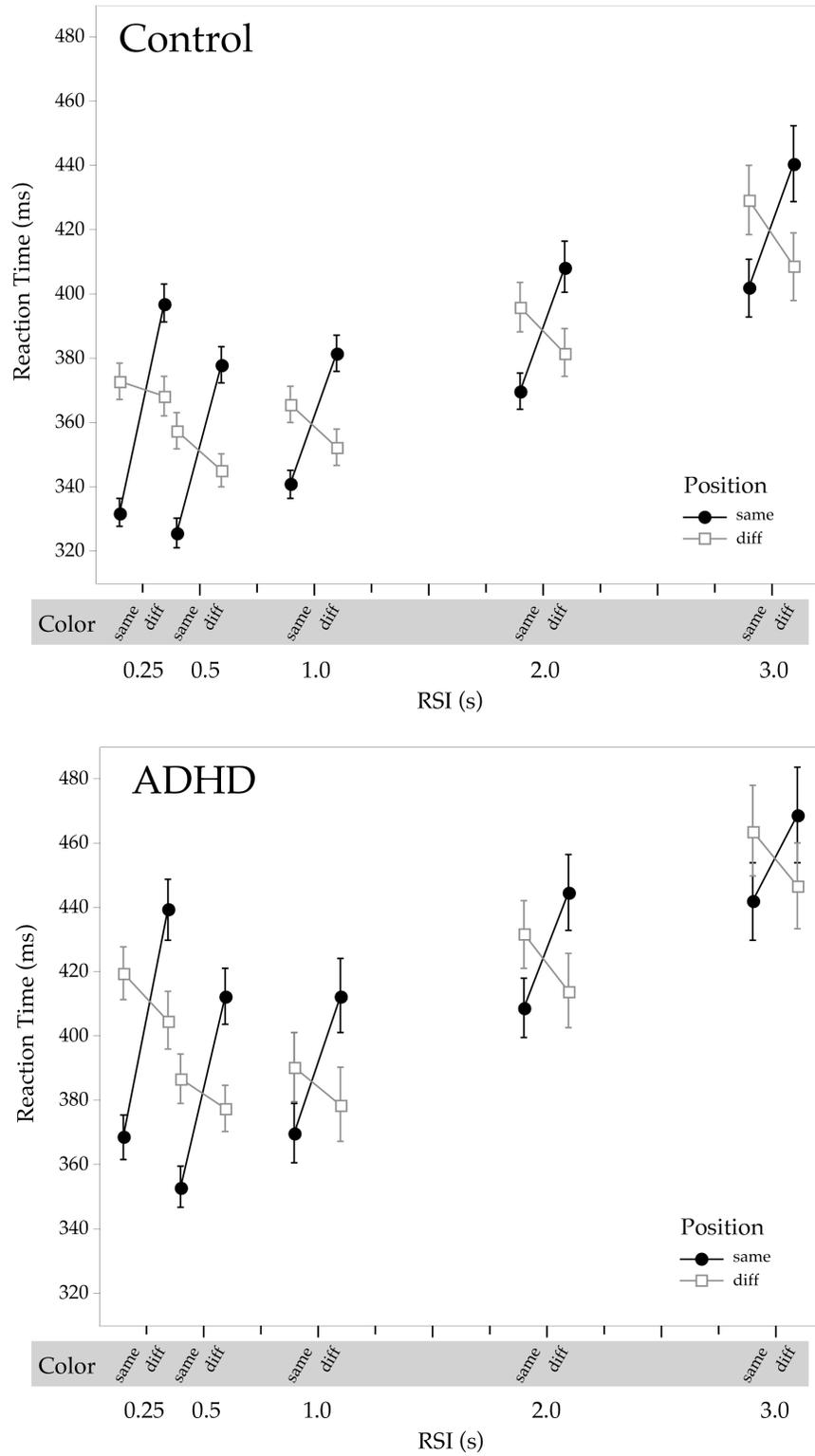


Figure 19. Color-position interaction as a function of RSI in Experiment 3.

The question of interest is whether these priming effects decay, and if they decay faster in the ADHD group than in the control group. Figure 19 shows that the crosses are of larger amplitude in both groups at the smallest RSIs, and that the cross is more collapsed at 3 seconds in the group with ADHD than it is in the control group. Figure 20 directly compares the slopes of the cross strength function and the response priming function between the two groups. For cross strength, the group with ADHD produced much steeper slopes than the control group ( $t(103) = 2.41, p = 0.009$ ). For response priming strength, ADHD slopes were also steeper, but not as substantially ( $t(103) = 1.13, p = 0.13$ ).

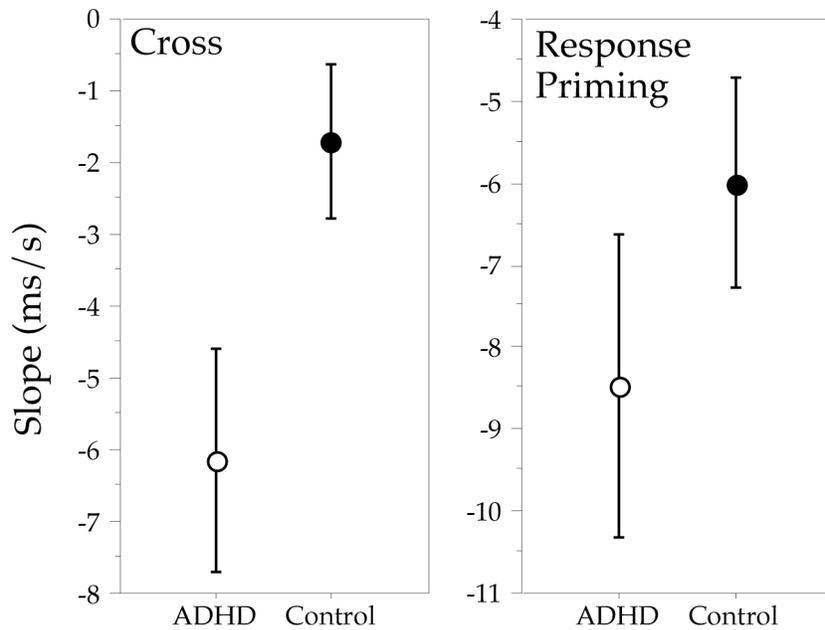


Figure 20. Decay slopes of cross strength and response priming strength as functions of RSI in Experiment 3.

## DIAGNOSTIC UTILITY: CUING AND PRIMING EXPERIMENTS

Apart from the preliminary experiment, neither version of Experiment 2 produced stable group differences in terms of differences in temporal integration. For this reason, Experiment 2 was not assessed as a predictor of ADHD symptom severity or of group membership. Experiment 3, however, did appear to produce stable group differences. The slope of the decay of the crossed interaction with increasing RSI was assessed in relation to symptoms and group membership.

The slope of the decay of the crossed interaction was not correlated with the CAARS subscale scores, but it was a significant predictor of group membership in a logistic regression ( $\chi^2 = 5.9$ ). Alone, the ADHD index subscale ( $\chi^2 = 44.1$ ) and the total DSM-IV symptoms subscale ( $\chi^2 = 73.1$ ) from the CAARS – S:L are each better predictors than the slope of the interaction decay. However, adding the slope measure as a predictor does significantly improve the fit of each model. Overall the best model, as determined by the AIC, combines both the total DSM-IV symptom subscale with the slope measure ( $\chi^2 = 78.0$ ). This model correctly classifies 89% of the 105 cases entered in this analysis. These findings indicate that the rate of decay of the crossed interaction produced by irrelevant feature priming might be useful as a diagnostic aid when combined with more standard self-report measures of symptom severity.

## **Apparent Motion Experiments**

Apparent motion, first systematically studied by Wertheimer (1912), is a phenomenon in which discrete flashes of identical stimuli in different locations can elicit the perception of motion. Wertheimer demonstrated that when the temporal separation between the flashes of the two stimuli is very short, observers perceive simultaneity of the two stimuli. When the temporal separation is very long, observers perceive succession: the appearance of first one stimulus, then the appearance of the second stimulus. However, at intermediate separations, the perception is of one object moving between two locations.

The influence of the length of the temporal separation on the apparent motion effect recommended this paradigm as another domain in which the span of temporal integration could be productively studied. Many studies of apparent motion since Wertheimer's have explored the effect of temporal separation on the perception of motion (Burt & Sperling, 1981; Korte, 1915; Farrell, 1983; Gepshtein & Kubovy, 2007). However, these studies have primarily focused on the minimum temporal separations required to achieve the perception of apparent motion. The focus here, rather, was on the maximum temporal separation that allows for motion perception. It should be noted that the range of intervals typically assessed in this field is on the order of tens of milliseconds, which is much shorter than the range of intervals assessed in the studies described above. It was not obvious if altered delay gradients would predict a shorter span of integration in the perception of apparent motion, but there were reasons to expect that this might be so, described in more detail below.

Apparent motion is thought to be divided into two classes: the long-range process and the short-range process (Anstis, 1980; Braddick, 1974; Braddick, Ruddock, Morgan, & Marr, 1980; Pantle & Picciano, 1976; Petersik, 1989). Short-range apparent motion generally occurs over small separations in space and temporal interval, and is attributed to low-level processes of motion detection in the visual system. Long-range apparent motion, on the other hand, often occurs over longer separations in space and time, and is thought to involve higher-level interpretive processes in the visual system. The conjecture that was tested in these experiments was whether these processes that generate long-range apparent motion are governed by the time scale of delay-of-reinforcement gradients.

Two apparent motion studies were conducted. One was a classic display originally developed by Ternus (1926) and illustrated in Figure 21. When the ISIs separating the two frames of this display are short, only one dot appears to move, bouncing from the leftmost position in frame 1 to the rightmost position in frame 2. This perception is referred to as element motion. When ISIs are longer, all three dots appear to shift back and forth together; this is referred to as group motion. The two regimes of element and group motion are thought to represent the short and long range apparent motion, respectively (Braddick et al., 1980; Pantle & Picciano, 1976; Petersik & Pantle, 1979). This study assessed the ISI that corresponds to the indifference point between element and group motion in adults with ADHD and normal controls.

The second apparent motion experiment was a study of perceived trajectory in long-range apparent motion. The display used here was borrowed from Proffitt, Gilden,

Kaiser, and Whelan (1987), who assessed effects of orientation upon the perceived trajectory of apparent motion. In this version, the effect of varying ISI was investigated. It was expected that longer ISIs would reduce the perception of a stable path, and this effect was compared in the two groups.

#### **EXPERIMENT 4: GROUP VS. ELEMENT MOTION IN THE TERNUS DISPLAY**

Previous work (Pantle & Picciano, 1976; Petersik & Pantle, 1979) using the Ternus display, as well as pilot data, indicate that the indifference point (the ISI where responses of “group motion” or “element motion” are made with equal probability) of typical (non-ADHD) observers is approximately 50-60 ms. A range of ISIs was used that bracketed this interval length; this was done to allow all participants to experience multiple trials of both element and group motion perception.

#### **Method**

##### *Participants*

Thirty-nine participants with ADHD and forty-four control participants from the primary participant group completed this study. The data from several participants in each group were excluded from analysis, as described below.

##### *Design*

The Ternus display used in this study consists of three grey dots appearing on a white background and surrounded by a black rectangle. At a viewing distance of about 60 cm, the rectangle subtends approximately 14 degrees of visual angle, and the distance

between the location of the leftmost appearing dot from the location of the rightmost appearing dot is approximately 10 degrees of visual angle.

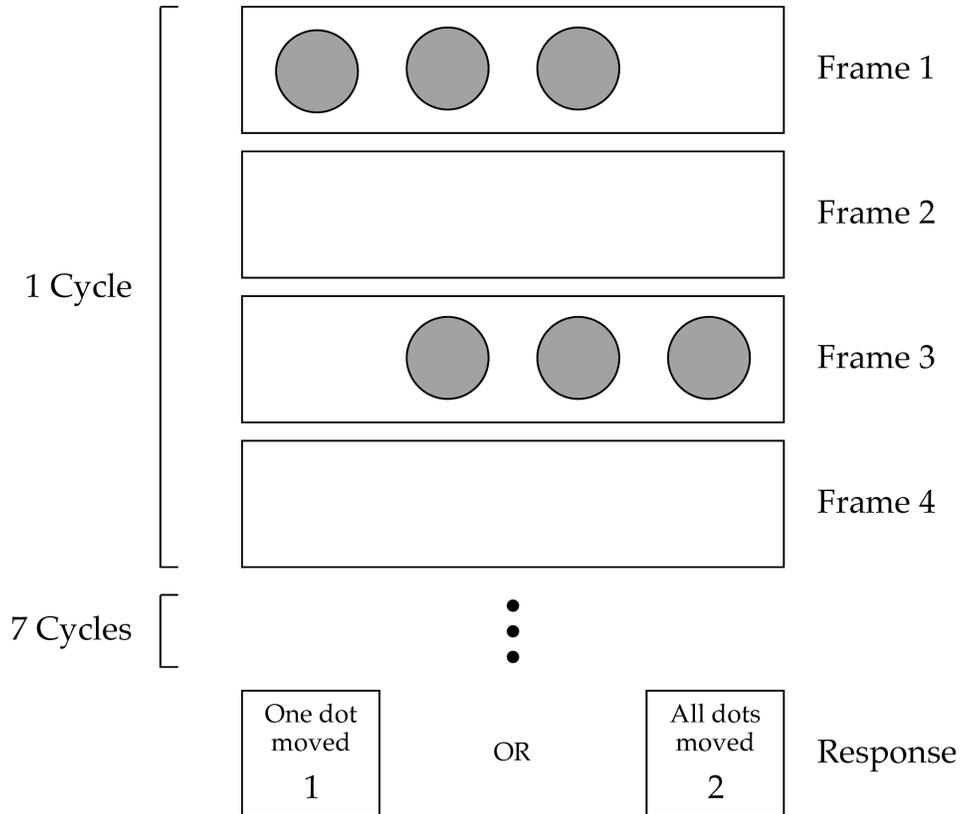


Figure 21. Ternus stimuli used in Experiment 4.

*Procedure*

Participants viewed an instructional screen, which included 12 cycles each of two displays that were designed to provide clear examples of the two types of motion that could be perceived. The first example used an ISI of 240 ms, a long enough interval to elicit a strong perception of group motion in virtually all observers. The second example used an ISI of 0 ms, which almost universally elicits the perception of element motion.

The experimenter described the two different types of apparent motion and confirmed that the participant experienced group and element motion, respectively, in the two examples. Participants were instructed that they would be asked to make repeated judgments of the type of motion perceived, and that some of the judgments might be more difficult than those in the instructional display. For these more difficult cases, participants were told to choose the type of motion that they perceived more strongly throughout the trial.

Participants completed four blocks of 60 trials each. In every block, each ISI condition occurred five times. The order of presentation was randomized for each participant in each block. Each trial consisted of 8 cycles of the display, after which participants were asked to choose if they experienced element or group motion (see Figure 21).

### *Analysis*

Each participant completed four test blocks of 60 trials each, which totals to 20 decisions about each of the 12 ISI conditions. The proportion of “group motion” responses were plotted as a function of ISI for each participant, and a logistic was fit to each participant’s data to estimate the ISI indifference point, where the participant is predicted to make “group motion” and “element motion” responses with equal probability (see Figure 22). The average indifference points were compared between groups.

A resampling procedure was conducted to determine if any disparity in indifference points between the two groups was statistically reliable. An example of one

participant's data and logistic distribution fit are shown in Figure 22. The alpha parameter of the logistic distribution represents the indifference point, and this value is represented in the figure as a vertical dashed line. This participant's data was resampled and fit to a logistic distribution, and the range of alpha parameter values generated from this resampling procedure is plotted with a 95% confidence interval on the same figure. This confidence interval, which spans approximately 12 ms, illustrates how stable the estimated indifferent point is for this observer. If the experiment were replicated with the same observer 1000 times, the estimated indifference point from the logistic fit would be within about 6 ms of the original estimate in 95% of the replications.

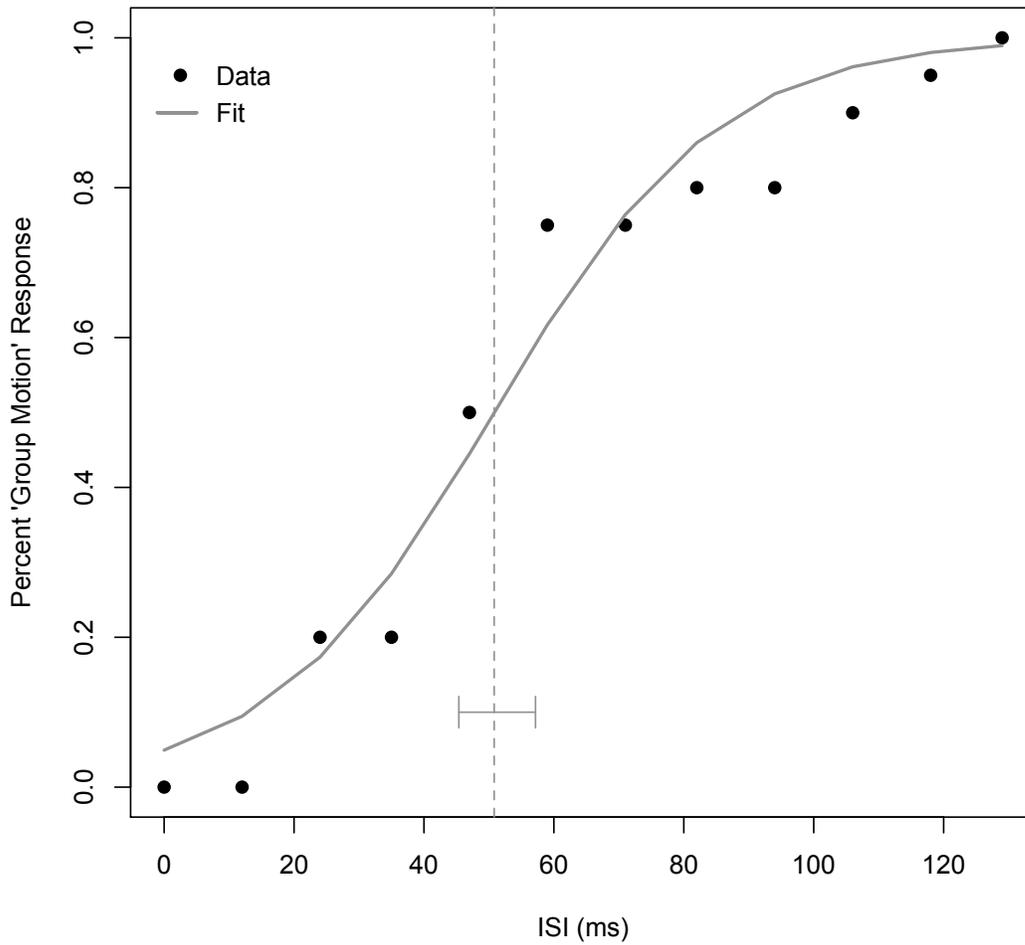


Figure 22. Data and logistic fit from a single participant in Experiment 4.

## Results and Discussion

Several participants from each group (7 control, 11 ADHD) were excluded from analysis because their data indicated that they did not have a full understanding of the task. These exclusions were made when a participant did not indicate a perception of group motion at least 75% of the time in any of the ISI conditions.

The estimated alphas were averaged across participants. This average alpha value (across both groups) was approximately 55 ms. This value is consistent with previous work and demonstrates that the methodology used here was sound. The average for the control group was 54.1 ms, and the average for the group with ADHD was 56.0 ms, a difference of less than two milliseconds. The data from the participants were resampled and fitted as described above, and the average of the alpha parameters was recorded for each group. This produced an estimate of how stable the two group means are, and how substantial the 2 ms difference is relative to the variability in the experiment. The results of this resampling procedure are plotted in Figure 23. The distribution of ADHD average alphas is much wider than that of the control group, as a result of larger between-subject variability in the ADHD group. Unsurprisingly, the mean of the resampled ADHD distribution is approximately 2 ms larger than the mean of the control distribution, but it is clear that the distributions overlap extensively. In fact, in more than one third of the simulations, the control average was actually larger than the ADHD average.

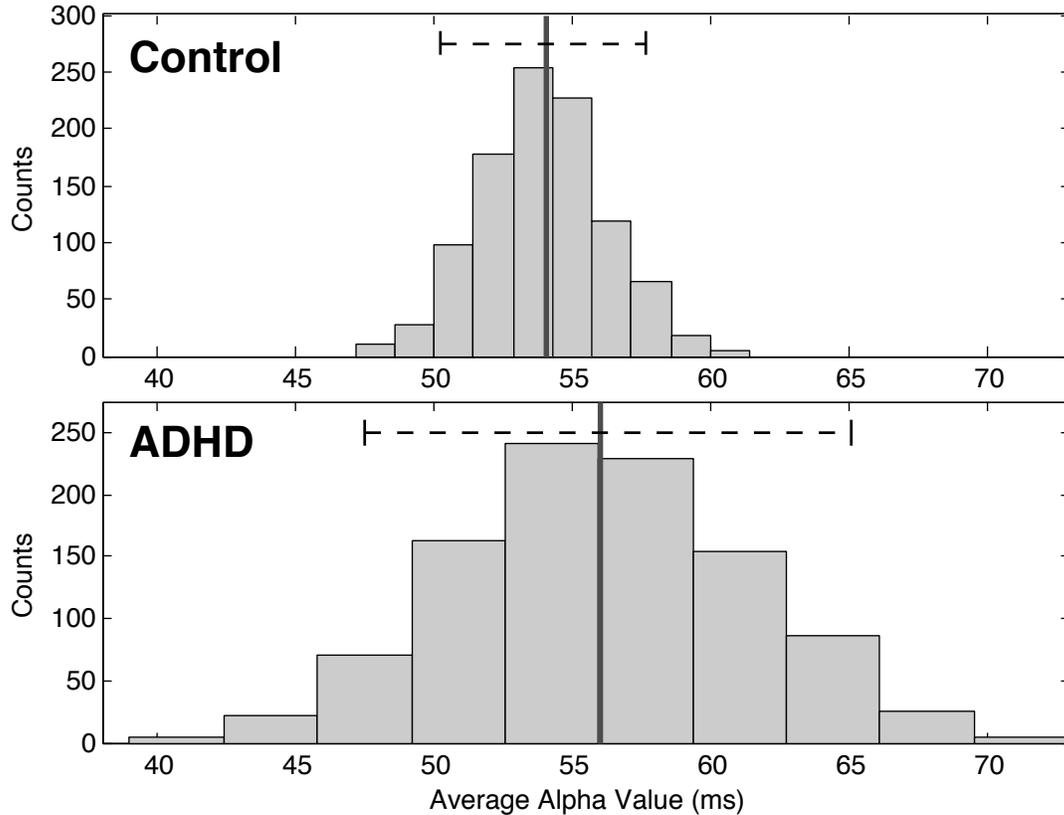


Figure 23. Distributions of resampled average alpha values in Experiment 4. Vertical lines indicate group means from the original data. Dashed lines indicate 95% confidence intervals.

The results of this study do not indicate any replicable group differences in indifference points between ADHD and control. Both groups generated indifference points that were exactly in the middle of the range of values seen in previous studies of the Ternus display (Pantle & Picciano, 1976; Petersik & Pantle, 1979). The lack of group differences was not unexpected, given that the temporal intervals involved in the transition from group to element motion are on the order of tens of milliseconds, as opposed to the 2-3 second intervals of interest in temporal integration. Nonetheless, this

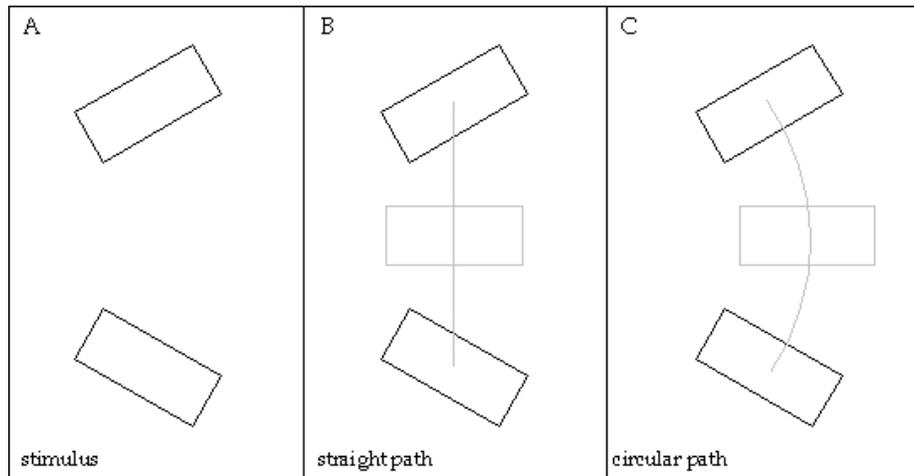
is an interesting result in that it provides evidence that ADHD does not involve a general timing impairment at all time intervals – rather a contraction of maximum intervals for integrating events into meaningful scenes.

Like Experiment 4, Experiment 5 also used an apparent motion task, but instead of assessing the transition point between two types of motion, it probed the transition from perceived motion to succession.

### **EXPERIMENT 5: PERCEIVED TRAJECTORY OF APPARENT MOTION**

There are an infinite number of paths that could be inferred from the flashing of two stimuli in different locations, but the paths that are actually perceived by observers in long-range apparent motion tend to be those that minimize motion (Wertheimer, 1912).

One well-studied example is illustrated in Figure 24. The stimuli in this example include both a translation and an orientation change. Previous work has examined whether observers tend to perceive a circular path, which represents a single rotation, or a straight path, which represents translational motion plus a rotation (Farrell, 1983; Foster, 1975). Proffitt et al. (1988) showed that the type of motion seen depended on the nature of the stimuli. Observers tended to indicate paths that were closer to circular path than straight, although this was mitigated by orientation changes in the stimuli. A constant, short ISI was used in all their manipulations. Experiment 5 tested the effect of varying ISI length upon the type of motion perceived, as well as the stability of that perception.



*Figure 24.* Possible trajectories of apparent motion. A) visible stimulus, B) illustration of the perception of a straight path (translation plus rotation), C) illustration of the perception of a circular path (single rotation).

This study used the same task as the Proffitt et al. (1998) experiments: participants were asked to indicate the location where the rectangle should appear in the middle of the perceived path. This procedure provided a more objective measure of path perception than could be achieved by asking participants to describe the nature of apparent motion perceived on each trial. The location of the test stimulus was compared across different ISI conditions to see if the type of paths perceived changed as a function of ISI. In addition, the variability of estimations within a given ISI condition was compared to determine if the variability in path estimation increased with increasing ISI.

## **Method**

### *Participants*

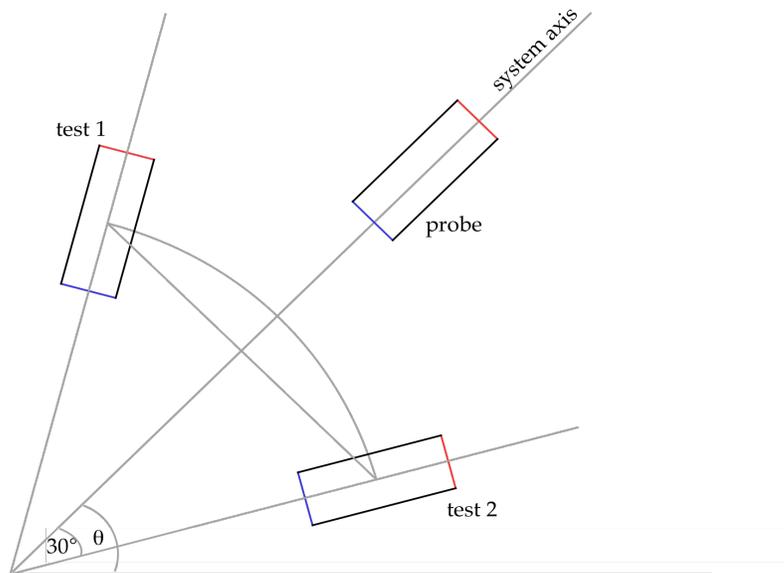
Thirty-nine participants with ADHD and seventy-two control participants from the primary participant group completed this study. The data from several participants in each group were excluded from analysis, as described below.

### *Design*

The stimuli in this experiment consisted of two test rectangles flashing in turn in fixed positions on the screen. After a specified number of cycles of this display, a flashing probe rectangle appeared that could be moved by the observer. One side of each rectangle was colored blue, while the other was colored red; this was done to make clear the orientation change between the two test rectangles. The dimensions of each rectangle were 4.5 by 1.5 cm, and the distance between the centers of the two test rectangles was 20.5 cm.

An illustration of the system appears in Figure 25. The system had two parameters that varied from trial to trial: 1) the angle ( $\theta$ ) of the system axis (a line equidistant at every point to the centers of two test rectangles) relative to the horizontal, and 2) the ISI that elapsed after the disappearance of one test rectangle before the appearance of the other. The possible values for  $\theta$  were 45, 135, 225, and 315 degrees, and the possible ISI values were 200, 300, 500, 1100, 1700, and 2300 ms. This ISI between the test rectangles remained constant before and after the onset of the flashing probe rectangle. The duration of all three stimuli was 100 ms.

The probe rectangle was constrained to move along the system axis (which was not visible to observers), and it flashed in sequence with the test rectangles. The location of the probe rectangle could be manipulated with the mouse, and a mouse click recorded the current position of the probe and ended the trial. On every trial, the flashing probe rectangle first appeared in a randomized location along the system axis, so that the observer could not learn a stereotyped motor pattern for placing the rectangle.



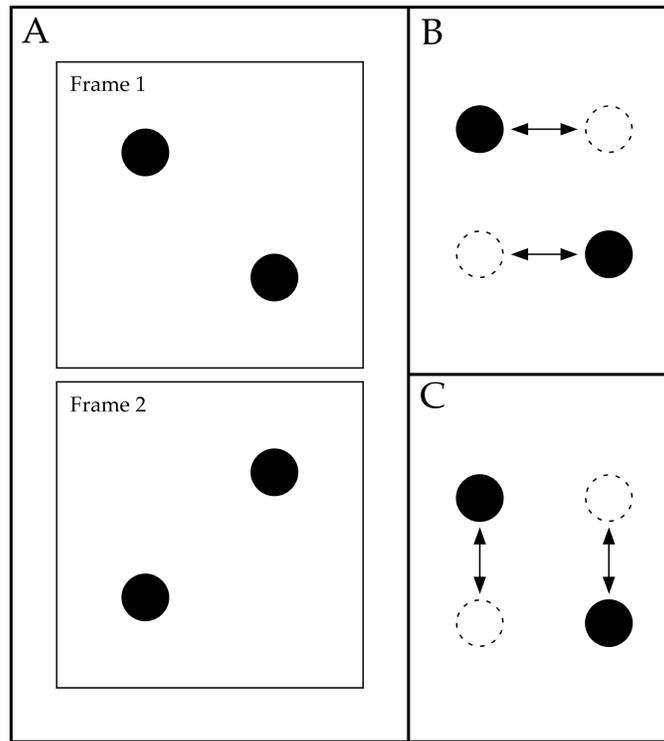
*Figure 25.* Stimuli used in Experiment 5.

### *Procedure*

Participants viewed an instructional screen, which began with a demonstration of the concept of apparent motion. This demonstration consisted of a motion quartet (see Figure 26). Participants viewed the motion quartet for several cycles, after which two points were added to the display to constrain the perception to horizontal motion. These

points disappeared, and then reappeared in a location that constrained the perception to vertical motion. During this demonstration, the experimenter explained that the display could be seen in more than one way, including either horizontal or vertical motion. The experimenter asked if the participant could see that there were at least two ways to interpret the motion, and if there was a particular one that he or she had initially seen.

If the participant understood this demonstration, the display was advanced from the motion quartet to the flashing rectangle display. In this demonstration, the test rectangles flashed at a relatively short ISI (200 ms), allowing for strong perception of apparent motion along a path. After 10 cycles, the probe rectangle began to flash in sequence in the display. The experimenter explained the purpose of the test rectangle and demonstrated how its position on the screen could be manipulated with the mouse. The participant was then given control of the mouse and had the opportunity to practice placing the probe rectangle. If the participant placed the probe rectangle in the general vicinity that would indicate either a circular or straight path, the experimenter began the test block. Otherwise, the participant was asked to explain the type of motion he or she was seeing. This did not occur in many cases, and when it did, there was an underlying misunderstanding of the task that was easily resolved.



*Figure 26.* Motion quartet shown as an example of apparent motion in Experiment 5. A) Stimulus shown to the observer. B) Horizontal interpretation of motion. C) Vertical interpretation of motion.

The participants then completed a test block of 24 trials. There were four possible angles of rotation of the system, and six possible ISIs. In total, observers executed one trial at each angle/ISI condition. In analysis, the data were collapsed across the four angle conditions in analysis, yielding 4 observations at every ISI condition. On every trial, the coordinates of the placement of the center of the probe rectangle were recorded.

*Analysis*

The displacement of the placed test rectangle from the straight path was calculated for each trial. Positive displacement values indicated displacement in the direction of the circular path, while negative values indicated displacement in the

opposite direction of the circular path. It was speculated that displacements might decrease with ISI and that the variability of the displacements would increase with ISI. A steeper rate of change in either or both of these measures in the ADHD group would provide further evidence of a contraction of the timescale for temporal integration.

## **Results and Discussion**

A small amount of pre-processing and exclusions had to take place before the two main measures could be calculated and averaged. First of all, several participants reported that they clicked the mouse before moving the test rectangle on one or more trials. These trials were typically easy to locate, as the test rectangle first appeared in locations that were quite far from the average participant placement. If an observer only had one error of this nature, only that trial was removed. If, however, the observer made more than one such error, all of that participant's data was removed from analysis. In addition, some data sets indicated that the observers did not understand the task in the same way that the majority did. This was typically indicated by displacements that were highly dependent on the angle of the system axis ( $\theta$ ) in such a way that the participant seemed to be attempting to mark the location of the pivot point of the system, rather than the path the rectangle would travel through. The data from these participants were also removed from analysis. This resulted in the exclusion of 14 participants (3 ADHD, 11 control).

The results of Experiment 5 are shown in Figure 27. The top panel shows the average displacements (in pixels) from the straight path for both groups. Displacement

values of 0 would indicate that the observers placed the test rectangle at the exact location of the straight path, while displacement values of 107 would indicate that the observers placed the test rectangle at the exact location of the circular path. In all ISI conditions, both groups tended to place their rectangles at locations that were close to, but not quite at, the circular paths. There is not a great deal of movement across the different ISIs in terms of the type of path perceived. There is a small trend, especially in the control group, for the estimates to become closer to the circular path as the intervals decrease, but this effect is not significant. Another effect worth noting from this figure is that the between-subject variability in placements, indicated by the size of the error bars, is very large at the long ISIs and becomes much smaller at the short ISIs.

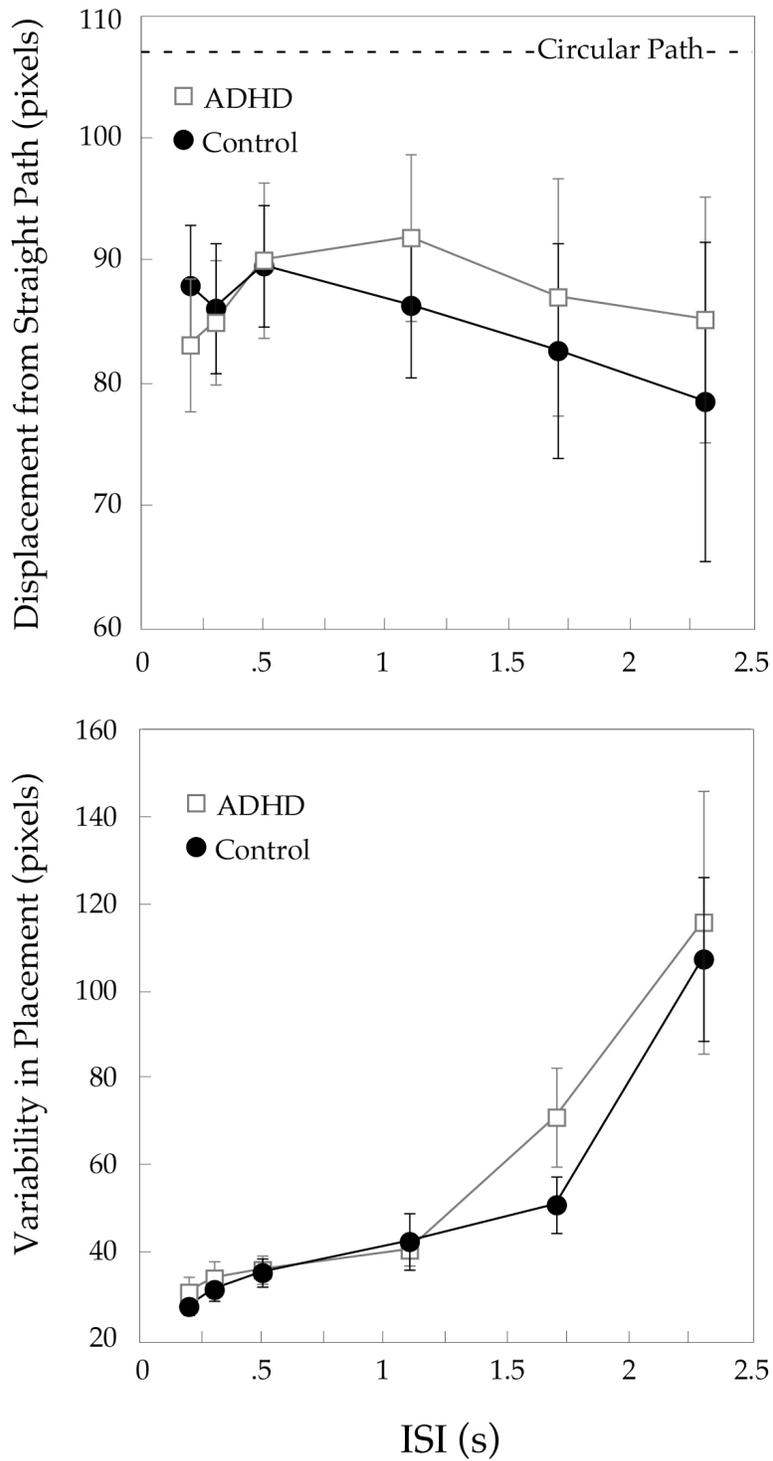


Figure 27. Means and standard deviations of displacement in Experiment 5.

The bottom panel of Figure 27 shows the average variability in placement of the test rectangles. This measure represents how variable each observer was in his or her placement at a given interval, and unlike the mean displacements, it shows a large effect of ISI. Observers in both groups are extremely variable at the longest interval of 2.3 seconds – the average standard deviation for both groups is approximately 110 pixels. Such high variability suggests that observers were likely unable to see a path at all and are placing the test rectangle at wildly different locations on the screen within a given delay interval condition. This variability rapidly decreases as the intervals decrease, with both groups demonstrating stable variability of about 30-40 pixels when the intervals were 1.2 seconds or smaller. There appears to be a difference in the rate at which the variability decreases in the two groups. The control group shows a large decrease between 2.3 and 1.7 seconds, and only small decreases as the intervals continue to become shorter. The ADHD group, however, shows fairly large decreases between both 2.3- to 1.7-s ISIs, and 1.7- to 1.2-s ISIs. As a result, at 1.7-s intervals, the group with ADHD has higher variability than the control group ( $t(95) = 1.65, p = 0.05$ ).

The results of Experiment 5 indicate a group difference in the rate of variability change across intervals. This indicates that the perception of stable paths as a result of apparent motion may be an example of temporal integration, and therefore that it is subject to the same limits on the maximum intervals over which integration can occur. It is worth noting that the interval of difference between the two groups in this experiment (1.7 seconds) is in the same range as the intervals in the previous experiments that showed group differences.

## **DIAGNOSTIC UTILITY: APPARENT MOTION EXPERIMENTS**

Experiment 4 was designed with the expectation that the two groups would not differ in their transition point between element motion and group motion, and in fact, they did not. For this reason, the results of Experiment 4 were not assessed as predictors of ADHD symptom severity or group membership. Experiment 5, however, did seem to produce stable group differences. The variability in estimates at the second longest ISI was assessed in relation to symptoms and group membership.

The variability measure in Experiment 5 did not correlate with the CAARS subscale scores. Alone, the ADHD index subscale and the total DSM-IV symptoms subscale from the CAARS – S:L are each better predictors of group membership than the variability measure. However, adding the variability measure from Experiment 5 to each of these models does significantly improve the fit of the model. Overall the best model, as determined by the AIC, combines both the total DSM-IV symptom subscale with the Experiment 5 variability measure ( $\chi^2 = 70.8$ ). This model correctly classifies 87% of the 95 cases entered in this analysis.

### **Combined Diagnostic Utility**

The measures of interest from Experiment 3 and Experiment 5 both proved to be significant predictors of group membership when combined with subscales of the CAARS – S:L. Because the same primary participant group completed both of these studies, it was possible to assess their predictive power in combination as well. The best

model, as determined by the minimum AIC, combines the total DSM-IV symptoms subscale and the two experimental measures from Experiments 3 and 5 as predictors ( $\chi^2 = 72.4$ ). This model correctly classifies 87% of the 91 cases entered in this analysis.

The total scores on the Executive Function (EF) Questionnaire were highly correlated with all of the CAARS subscale scores. However, the EF scores were not significantly correlated with the slope measure from Experiment 3 or with the variability measure from Experiment 5, suggesting that the group differences seen in these tasks are not related to executive function impairments in ADHD. Logistic regression revealed that the EF scores were significant predictors of group membership ( $\chi^2 = 28.9$ ). A smaller number of participants completed the EF questionnaire than the CAARS – S:L, so the models that include EF produce somewhat different results from those described in earlier sections. However, when the two experimental measures, EF scores, and CAARS scores are all analyzed in a multiple logistic regression, the best model (as determined by the minimum AIC) includes EF, total DSM-IV symptoms subscale, and the variability measure from Experiment 5 as predictors. This model correctly classifies 85% of the 75 participants who completed all of these measures.

### **Explicit Timing Assessment**

After completing the IQ assessment in their third and final experimental session, participants were asked to estimate the amount of time that had elapsed during that assessment. This was done to provide a measure of explicit timing ability, as a point of comparison to the implicit use of time studied in Experiments 1 through 5. On average,

participants tended to under- rather than over-estimate the amount of time that had elapsed, with the group with ADHD making larger underestimations. On average, the control group underestimated the elapsed time by 1.4 minutes, and the group with ADHD underestimated by about 3 minutes, although this group difference is not significant ( $t(73) = 1.1, p = 0.3$ ). However, the actual amount of time that elapsed was several minutes longer on average in the ADHD group (19.1 minutes) than in the control group (15.1 minutes). When the relative errors, (estimate – actual / actual), were compared, the groups were quite similar (see Figure 28).

The lack of a significant group difference in explicit timing ability suggests that the ability to explicitly time is not related to the processes that govern the maximum intervals of temporal integration. Furthermore, most accounts of ADHD as an explicit timing disorder would predict that delay aversion and reward discounting would lead to time intervals being perceived as longer than they are in ADHD. This is not what the finding here; instead, the participants with ADHD estimated the elapsed time as being shorter than it actually was, even more so than the control participants. Regardless, the magnitude of the errors was not significantly different in the two groups, providing evidence against theories of general timing impairment in ADHD.

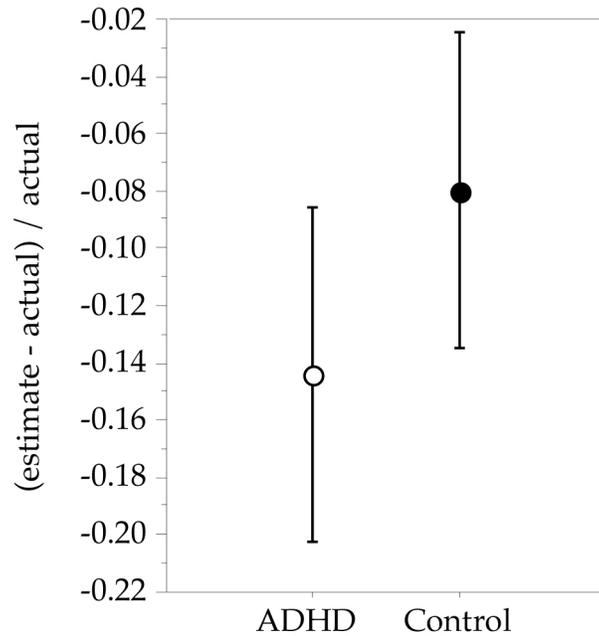


Figure 28. Relative errors in explicit timing task.

## General Discussion and Conclusions

### SUMMARY OF RESULTS

Five experiments were originally conducted to assess the maximum intervals of temporal integration in both normal adults and in adults with attention deficit hyperactivity disorder. Experiment 1, which studied rhythmic tapping at varied tempos, and Experiment 2, which studied spatial cuing across varying delays, both produced surprising and interesting results in relation to previous work using both tasks. These results led to a refinement in the methodologies of both tasks in subsequent follow-up studies.

On the whole, the revised rhythmic tapping tasks, the priming task of Experiment 3, and the apparent motion task of Experiment 5 all produced significant group differences in the rate of decay of temporal integration with increasing intervals over which integration was required. In addition, these three tasks generated measures that were found to be potentially useful as diagnostic aids for adult ADHD.

It was concluded that the spatial cuing task of Experiment 2 was not a reliable measure of temporal integration, as implicit temporal integration was only one possible strategy that could be productively used to complete the task. The transition from element to group motion in the Ternus display was, predictably, concluded to not be an example of the maximum limit of temporal integration.

The two groups only demonstrated differences in the maximum spans of temporal bridging; that is, in their implicit use of time. When participants were required to explicitly judge elapsed time intervals, the group with ADHD did not show a significantly greater impairment than the control group.

#### **GENERAL OBSERVATIONS**

One interesting finding from these studies is that the delay interval that marks the maximum interval for integration is not exactly the same in all types of experiments. In fact, it is not exactly the same in different versions of the same experiment. For example, in one version of the rhythmic drumming studies the maximum interval for stable performance was 1.5 seconds in controls, and in another version it was 1 second in controls. It is possible that task difficulty could explain such differences; a more difficult

task might not allow for bridging to occur across as long of a delay between events as a simpler task. However, the maximum intervals are consistently in the same range of a few seconds. And critically, no matter what the maximum time interval is in controls, we almost universally see that that maximum interval is shifted to be somewhat smaller in ADHD. When this did not occur in Experiment 4, the intervals of interest were less than 100 ms, and the process being tested was thought to be more related to low-level visual processes than the Gestalt processes of perceptual organization. Experiment 4, then, could be considered the exception that proves the rule.

Another finding of interest arising from this ensemble of experiments is that there is some evidence that not only is the breakdown of temporal integration shifted to shorter intervals in ADHD, but that the peak performance of integration may also be shifted to shorter intervals. The most evident indication of this was in Experiment 1b, the synchronization follow-up study, where the group with ADHD was able to tap much closer to the metronome than the control group at the fastest tempo. In addition, the group with ADHD demonstrated larger validity effects at the fastest tempos than the control group did in the Posner cuing studies. Finally, in Experiment 3, the priming study, the group with ADHD showed larger priming effects at the fastest tempos than the control group. These observations may be evidence that ADHD is not detrimental in all contexts, and that there may be instances where it could instead be a benefit. For example, in the studies of rhythmic tapping, the intervals studied ranged from the slow extreme of music to tempos slower than what is even found on a metronome. At faster tempos that are typical in popular music, it is possible, based on the above evidence, that

musicians with ADHD might “feel the rhythm” better than their non-ADHD counterparts. Many people with ADHD are musicians – this could be attributed to the appeal of the less-structured work environment, but it could also be seen as an opportunity for a benefit of the disorder to be expressed. This is obviously speculative, but may be worth further study.

## CONCLUSIONS

The experiments presented here provide convergent evidence that the span of temporal integration is foreshortened in ADHD cognition. In this specific sense, timing function is found to be compromised in ADHD. The key to these studies is the focus on the timescale over which past experience exerts influences on present behavior, rather than on the explicit awareness of time as something that might be judged – as the explicit awareness of any sensory quality might be judged. These findings are quite specific to the maximum delays over which temporal bridges are built.

Earlier studies of ADHD timing have mostly involved a search for a sense in which ADHD timing performance is impaired relative to normal controls, but their findings have so far lacked consistency across studies. The conclusion that has been drawn from the variety of results is that there is some type of timing impairment in ADHD, but the exact nature of that impairment and how it manifests is still unclear. This inconsistency of results can be better explained by problematic theoretical motivation and problematic methodology. The formulation of ADHD as a disorder of executive function is vague and itself lacking in solid empirical support (Castellanos, Sonuga-Barke,

Milham, & Tannock, 2006). Further, the connection between executive function and timing has not been adequately developed. In the absence of specific mechanisms connecting timing to the central executive it has generally been supposed that timing deficits in ADHD would be diffuse and not localized to specific systems or abilities (Barkley, 1997). This lack of specificity provides little guidance for selecting a methodology by which to study timing function. Secondly, and possibly more importantly, timing assessments have mostly involved explicit comparison processes. There is no question that the explicit comparison of time intervals might offer productive insights on timing process - quite a bit has been learned about scalar timing in humans and other animals using these techniques - but it is now clear after a number of thorough studies (Toplak et al., 2006) that such assessments in the context of ADHD provide information about the variability of comparison process in ADHD and have not been as productive in clarifying the nature of timing impairments.

The current work, in contrast, benefits from two literatures: one on the consequences of delay of reinforcement upon learning, the other on the effects of dopamine upon learning. These two literatures lead to specific predictions about how dopamine dysfunction shrinks the maximum window for temporal integration across discrete events. By focusing the experimental work precisely on these windows, it was possible to design experiments that spotlight the critical durations where ADHD and control behavior are distinguished. The findings suggest that ADHD might be more productively studied not as a disorder of attention or behavioral inhibition, but rather as a

disorder that expresses itself by shortening the intervals over which temporal bridges may be built.

## Appendix: EF Questionnaire

### Questionnaire

---

Participant ID: \_\_\_\_\_ Sex: M F Age: \_\_\_\_\_ Date: \_\_\_\_\_

Instructions: Please circle the number next to each item that best describes your behavior *during the past 6 months*.

Items:	Never or rarely	Sometimes	Often	Very often
1. Easily distracted by irrelevant thoughts	0	1	2	3
2. Make decisions impulsively	0	1	2	3
3. Have difficulty stopping my activities or behavior when I should do so	0	1	2	3
4. Start a project or task without reading or listening to directions carefully	0	1	2	3
5. Show poor follow-through on promises or commitments I may make to others	0	1	2	3
6. Have trouble doing things in their proper order or sequence	0	1	2	3
7. More likely to drive a motor vehicle much faster than others (excessive speeding)	0	1	2	3

## References

- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders: Text revision (DSM-IV-TR)* (4th ed.). Washington, DC: American Psychiatric Association.
- Anstis, S.M. (1980). The perception of apparent movement. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 290, 153-168.
- Antony, M.M., Bieling, P.J., Cox, B.J., Enns, M.W., & Swinson, R.P. (1998). Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales in clinical groups and a community sample. *Psychological Assessment*, 10, 176-181.
- Antshel, K.M., Faraone, S.V., Maglione, K., Doyle, A., Fried, R., Seidman, L., & Biederman, J. (2009). Is adult attention deficit hyperactivity disorder a valid diagnosis in the presence of high IQ? *Psychological Medicine*, 39, 1325-1335.
- Applegate, B., Lahey, B.B., Hart, E.L., Biederman, J., Hynd, G.W., Barkley, R.A., et al. (1997). Validity of the age-of-onset criterion for ADHD: A report from the DSM-IV field trials. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 1211-1221.
- Aylward, E.H., Reiss, A.L., Reader, M.J., Singer, H.S., Brown, J.E., & Denckla, M.B. (1996). Basal ganglia volumes in children with attention-deficit hyperactivity disorder. *Journal of Child Neurology*, 11, 112-115.
- Baddeley, A. (1986). *Working Memory*. New York, NY: Oxford University Press.

- Barkley, R.A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*, 65-94.
- Barkley, R.A. (2002). Major life activity and health outcomes associated with attention-deficit/hyperactivity disorder. *Journal of Clinical Psychiatry*, *63* (suppl 12), 10-15.
- Barkley, R.A., Fischer, M., Smallish, L., & Fletcher, K. (2002). The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *Journal of Abnormal Psychology*, *111*, 279-289.
- Barkley, R.A., & Murphy, K.R. (2006). *Attention-Deficit Hyperactivity Disorder: A Clinical Workbook*. (3<sup>rd</sup> ed.). New York, Guilford.
- Barkley, R.A., Murphy, K.R., & Fischer, M. (2007). *ADHD in Adults: What the Science Says*. Guilford Press: New York.
- Beninger, R.J., & Freedman, N.L. (1982). The use of two operants to examine the nature of pimozide-induced decreases in responding for brain stimulation. *Physiological Psychology*, *10*, 409-412.
- Beninger, R.J., & Miller, R. (1998). Dopamine D1-like receptors and reward-related incentive learning. *Neuroscience & Biobehavioral Reviews*, *22*, 335-345.
- Biederman, J., Mick, E., & Faraone, S.V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. *American Journal of Psychiatry*, *157*, 816-818.

- Braddick, O. (1974). A short-range process in apparent motion. *Vision Research*, *14*, 519-527.
- Braddick, O.J., Ruddock, K.H., Morgan, M.J., & Marr, D. (1980). Low-level and high-level processes in apparent motion. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *290*, 137-151.
- Bridgett, D.J., & Walker, M.E. (2006). Intellectual functioning in adults with ADHD: A meta-analytic examination of full scale IQ differences between adults with and without ADHD. *Psychological Assessment*, *18*, 1-14.
- Burt, P., & Sperling, G. (1981). Time, distance, and feature trade-offs in visual apparent motion. *Psychological Review*, *88*, 171-195.
- Carmona, S., Proal, E., Hoekzema, A., Gispert, J.-D., Picado, M., Moreno, I., et al. (2009). Ventro-striatal reductions underpin symptoms of hyperactivity and impulsivity in attention-deficit/hyperactivity disorder. *Biological Psychiatry*, *66*, 972-977.
- Carter, C.S., Krener, P., Chaderjian, M., Northcutt, C., & Wolfe, V. (1995). Asymmetrical visual-spatial attentional performance in ADHD: Evidence for a right hemispheric deficit. *Biological Psychiatry*, *37*, 789-797.
- Castellanos, F.X., Giedd, J.N., Marsh, W.L., Hamburger, S.D., Vaituzis, A.C., Dickstein, D.P., et al. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry*, *53*, 607-616.

- Castellanos, F.X., Sonuga-Barke, E.J.S., Milham, M.P., & Tannock, R. (2006). Characterizing cognition in ADHD: Beyond executive dysfunction. *Trends in Cognitive Sciences, 10*, 117-123.
- Castellanos, F.X., & Tannock, R. (2002). Neuroscience of attention-deficit/hyperactivity disorder: The search for endophenotypes. *Nature Reviews Neuroscience, 3*, 617-628.
- Conners, C.K., Erhardt, D., & Sparrow, E. (1999). *Conners' Adult ADHD Rating Scales (CAARS)*. New York: Multi-Health Systems.
- Epstein, J. N., Conners, C. K., Erhardt, D., March, J. S., & Swanson, J. M. (1997). Asymmetrical hemispheric control of visual-spatial attention in adults with attention deficit hyperactivity disorder. *Neuropsychology, 11*, 467-473.
- Erhardt, D., Epstein, J.N., Conners, C.K., Parker, J.D.A., & Sitarenios, G. (1999). Self-ratings of ADHD symptoms in adults: II. Reliability, validity, and diagnostic sensitivity. *Journal of Attention Disorders, 3*, 153-158.
- Faraone, S.V., Perlis, R.H., Doyle, A.E., Smoller, J.W., Goralnick, J.J., Holmgren, M.A., & Sklar, P. (2005). Molecular genetics of attention-deficit/hyperactivity disorder. *Biological Psychiatry, 57*, 1313-1323.
- Faraone, S.V., Biederman, J., Spencer, T., Mick, E., Murray, K., Petty, C., et al. (2006). Diagnosing adult attention deficit hyperactivity disorder: Are late onset and subthreshold diagnoses valid? *American Journal of Psychiatry, 163*, 1720-1729.
- Farrell, J.E. (1983). Visual transformations underlying apparent movement. *Perception & Psychophysics, 33*, 85-92.

- Feinstein, T. (2007). Interactions in short-term implicit memory and inhibition of return (Doctoral dissertation, University of Texas at Austin, 2007). *Dissertation Abstracts International*, 67, 4126.
- Frazier, T.W., Demaree, H.A., & Youngstrom, E.A. (2004). Meta-analysis of intellectual and neuropsychological test performance in attention-deficit/hyperactivity disorder. *Neuropsychology*, 18, 543-555.
- Gepshtein, S., & Kubovy, M. (2007). The lawful perception of apparent motion. *Journal of Vision*, 7, 1-15.
- Gilden, D.L., & Marusich, L.R. (2009). Contraction of time in attention deficit hyperactivity disorder. *Neuropsychology*, 23, 265-269.
- Hart, E.L., Lahey, B.B., Loeber, R., Applegate, B., & Frick, P.J. (1995). Developmental change in attention-deficit hyperactivity disorder in boys; A four-year longitudinal study. *Journal of Abnormal Child Psychology*, 23, 729-750.
- Henry, J.D. & Crawford, J.R. (2003). The short-form version of the Depression Anxiety Stress Scales (DASS-21): Construct validity and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, 44, 227-239.
- Hommel, B. (1998). Event files: Evidence for automatic integration of stimulus-response episodes. *Visual Cognition*, 5(1/2), 183-216.
- Horvitz, J.C (2000). Mesolimbocortical and nigrostriatal dopamine responses to salient non-reward events. *Neuroscience*, 96, 651-656.
- Huang, L., Holcombe, A. O., & Pashler, H. (2004). Repetition priming in visual search: Episodic retrieval, not feature priming. *Memory and Cognition*, 32(1), 12-20.

- Huang-Pollock, C. L., & Nigg, J. T. (2003). Searching for the attention deficit in attention deficit hyperactivity disorder: The case of visuospatial orienting. *Clinical Psychology Review, 23*, 801-830.
- Hynd, G.W., Hern, K.L., Novey, E.S., Eliopoulos, D., Marshall, R., Gonzales, J.J., & Voeller, K.K. (1993). Attention deficit-hyperactivity disorder and asymmetry of the caudate nucleus. *Journal of Child Neurology, 8*, 339-347.
- Jensen, J., Smith, A.J., Willeit, M., Crawley, A.P., Mikulis, D.J., Vitcu, I., & Kapur, S. (2007). Separate brain regions code for salience vs. valence during reward prediction in humans. *Human Brain Mapping, 28*, 294-302.
- Johansen, E.B., Killeen, P.R., Russell, V.A., Tripp, G., Wickens, J.R., Tannock, R., et al. (2009). Origins of altered reinforcement effects in ADHD. *Behavioral and Brain Functions, 5*, 7.
- Kaufman, A.S., & Lichtenberger, E.O. (2006). *Assessing Adolescent and Adult Intelligence*. Hoboken, NJ: John Wiley & Sons.
- Kessler, R.C., Adler, L., Barkley, R., Biederman, J., Conners, C.K., Demler, O., et al. (2006). The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. *American Journal of Psychiatry, 163*, 716-723.
- Kessler, R.C., Green, J.G., Adler, L.A., Barkley, R.A., Chatterji, S., Faraone, S.V., et al. (2010). Structure and diagnosis of adult attention-deficit/hyperactivity disorder: Analysis of expanded symptom criteria from the adult ADHD clinical diagnostic scale. *Archives of General Psychiatry, 67*, 1168-1178.

- Kooij, J.J.S., Buitelaar, J.K., van den Oord, E.J., Furer, J.W., Rijnders, C.A.T., & Hodiament, P.P.G. (2005). Internal and external validity of attention-deficit hyperactivity disorder in a population-based sample of adults. *Psychological Medicine*, 35, 817-827.
- Korte, A. (1915). Kinematoskopische Untersuchungen. *Zeitschrift für Psychologie*, 72, 194-296.
- Lovibond, S.H. & Lovibond, P.F. (1995). *Manual for the Depression Anxiety Stress Scales* (2<sup>nd</sup> Ed.) Sydney: Psychology Foundation.
- Madison, G. (2001). Variability in isochronous tapping: Higher order dependencies as a function of intertap interval. *Journal of Experimental Psychology: Human Perception and Performance*, 27, 411– 422.
- McDonald, S., Bennett, K.M.B., Chambers, H., & Castiello, U. (1999). Covert orientation and focusing of attention in children with attention deficit hyperactivity disorder. *Neuropsychologia*, 37, 345-356.
- Montes, L.G.A., Ricardo-Garcell, J., De La Torre, L.B., Alcántara, H.P., García, R.B.M, Fernández-Bouzas, A. (2010). Clinical correlations of grey matter reductions in the caudate nucleus of adults with attention deficit hyperactivity disorder. *Journal of Psychiatry & Neuroscience*, 35, 238-246.
- Nigg, J.T., Swanson, J.M., & Hinshaw, S.P. (1997). Covert visual spatial attention in boys with attention deficit hyperactivity disorder: Lateral effects, methylphenidate response and results for parents. *Neuropsychologia*, 35, 165-176.

- Paine, L.E. (2010). *Modulation of implicit working memory in temporal grouping* (Doctoral dissertation, University of Texas at Austin, 2010).
- Pantle, A., & Picciano, L. (1976). A multistable movement display: Evidence for two separate motion systems in human vision. *Science, 193*, 500-502.
- Perin, C.T. (1943). A quantitative investigation of the delay-of-reinforcement gradient. *Experimental Psychology, 32*, 37-51.
- Petersik, J.T. (1989). The two-process distinction in apparent motion. *Psychological Bulletin, 106*, 107-127.
- Petersik, J.T., & Pantle, A. (1979). Factors controlling the competing sensations produced by a bistable stroboscopic motion display. *Vision Research, 19*, 143-154.
- Polanczyk, G., Caspi, A., Houts, R., Kollins, S.H., Rohde, L.A., & Moffitt, T.E. (2010). Implications of extending the ADHD age-of-onset criterion to age 12: Results from a prospectively studied birth cohort. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*, 210-216.
- Polanczyk, G., de Lima, M.S., Horta, B.L., Biederman, J., & Rohde, L.A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry, 164*, 942-948.
- Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology, 32*, 3-25.

- Proffitt, D.R., Gilden, D.L., Kaiser, M.K., & Whelan, S.M. (1987). The effect of configural orientation on perceived trajectory in apparent motion. *Perception & Psychophysics*, *43*, 465-474.
- Qiu, A., Crocetti, D., Adler, M., Mahone, E.M., Denckla, M.B., Miller, M.I., & Mostofsky, S.H. (2009). Basal ganglia volume and shape in children with attention deficit hyperactivity disorder. *American Journal of Psychiatry*, *166*, 74-82.
- Robbins, T.W., & Everitt, B.J. (1996). Neurobehavioural mechanisms of reward and motivation. *Current Opinion in Neurobiology*, *6*, 228-236.
- Sagvolden, T., Johansen, E.B., Aase, H., & Russell, V.A. (2005). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Sciences*, *28*, 397-468.
- Scahill, L., & Schwab-Stone, M. (2000). Epidemiology of ADHD in school-age children. *Child and Adolescent Psychiatric Clinics of North America*, *9*, 541-555.
- Scheres, A., Tontsch, C., Thoeny, A.L., & Kaczurkin, A. (2010). Temporal reward discounting in attention-deficit/hyperactivity disorder: The contribution of symptom domains, reward magnitude, and session length. *Biological Psychiatry*, *67*, 641-648.
- Schultz, W., Dayan, P., & Montague, P.R. (1997). A neural substrate of prediction and reward. *Science*, *275*, 1593-1599.

- Sharp, S.I., McQuillin, A., & Gurling, H.M.D. (2010). Genetics of attention-deficit hyperactivity disorder (ADHD). *Neuropharmacology*, *57*, 590-600.
- Shin, M. (2006). Different time course of negative priming in the subtypes of ADHD (Doctoral dissertation, University of Texas at Austin, 2006). *Dissertation Abstracts International*, *67*, 2244.
- Smith, A., Taylor, E., Rogers, J.W., Newman, S., & Rubia, K. (2002). Evidence for a pure time perception deficit in children with ADHD. *Journal of Child Psychology and Psychiatry*, *43*, 529-542.
- Solanto, M.V. (1998). Neuropsychopharmacological mechanisms of stimulant drug action in attention-deficit hyperactivity disorder: A review and integration. *Behavioural Brain Research*, *94*, 127-152.
- Sonuga-Barke, E.J.S. (2002). Psychological heterogeneity in AD/HD – a dual pathway model of behaviour and cognition. *Behavioural Brain Research*, *130*, 29-36.
- Sonuga-Barke, E.J.S., Bitsakou, P., & Thompson, M. (2010). Beyond the dual pathway model: Evidence for the dissociation of timing, inhibitory, and delay-related impairments in attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child Psychiatry*, *49*, 345-355.
- Spencer, T.J., Biederman, J., Madras, B.K., Faraone, S.V., Dougherty, D.D., Bonab, A.A., & Fischman, A.J. (2005). In vivo neuroreceptor imaging in attention-deficit/hyperactivity disorder: A focus on the dopamine transporter. *Biological Psychiatry*, *57*, 1293-1300.

- Spencer, T.J., Biederman, J., Madras, B.K., Faraone, S.V., Dougherty, D.D., Bonab, A.A., Livni, E. et al. (2007). Further evidence of dopamine transporter dysregulation in ADHD: A controlled PET imaging study using Altropane. *Biological Psychiatry*, 62, 1059-1061.
- Swanson, J.M., Kinsbourne, M., Nigg, J.T., Lanphear, B., Stefanatos, G.A, Volkow, N, et al. (2007). Etiologic subtypes of attention-deficit/hyperactivity disorder: Brain imaging, molecular genetic and environmental factors and the dopamine hypothesis. *Neuropsychology Review*, 17, 39-59.
- Ternus, J. (1926). Experimentelle untersuchungen über phänomenale identität. *Psychological Research*, 7, 81-136.
- Tomporowski, P.D., Tinsley, V., & Hager, L.D. (1994) Visuospatial attentional shifts and choice responses of adults and ADHD and non-ADHD children. *Perceptual and Motor Skills*, 79, 1479-1490.
- Toplak, M.E., Dostkader, C., & Tannock, R. (2006). Temporal information processing in ADHD: Findings to date and new methods. *Journal of Neuroscience Methods*, 151, 15-29.
- Tripp, G., & Wickens, J.R. (2008). Research review: Dopamine transfer deficit: A neurobiological theory of altered reinforcement mechanisms in ADHD. *Journal of Child Psychology and Psychiatry*, 49, 691-704.
- Volkow, N.D., Wang, G.-J., Fowler, J.S., Gatley, S.J., Logan, J., Ding, Y.-S., et al. (1998). Dopamine transporter occupancies in the human brain induced by

- therapeutic doses of oral methylphenidate. *American Journal of Psychiatry*, 155, 1325-1331.
- Volkow, N.D., Wang, G.-J., Fowler, J.S., Logan, J., Gerasimov, M., Maynard, L., et al. (2001). Therapeutic doses of oral methylphenidate significantly increase extracellular dopamine in the human brain. *The Journal of Neuroscience*, 21, RC121.
- Volkow, N.D., Wang, G.-J., Newcorn, J., Telang, F., Solanto, M.V., Fowler, J.S. et al. (2007). Depressed dopamine activity in caudate and preliminary evidence of limbic involvement in adults with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, 64, 932-940.
- Wechsler, D. (1993). *Wechsler Adult Intelligence Scale*, 3<sup>rd</sup> edn. Psychological Corporation: San Antonio, TX.
- Wertheimer, M. (1912). Experimentelle studien uber das sehen von bewegung. *Zeitschrift fur Psychologie*, 61, 161-265.
- Williams, J., & Dayan, P. (2005). Dopamine, learning, and impulsivity: A biological account of attention-deficit/hyperactivity disorder. *Journal of Child and Adolescent Psychopharmacology*, 15, 160-179.