# AN EXAMINATION OF THE ROLE OF WORKING MEMORY DEMANDS ON OBJECTIVELY MEASURED MOTOR ACTIVITY IN ADULT ADHD, GAD, AND HEALTHY CONTROL GROUPS

By

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AN EXAMINATION OF THE ROLE OF WORKING MEMORY DEMANDS ON OBJECTIVELY MEASURED MOTOR ACTIVITY IN ADULT ADHD, GAD, AND HEALTHY CONTROL GROUPS

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Title of Study: AN EXAMINATION OF THE ROLE OF WORKING MEMORY DEMANDS ON OBJECTIVELY MEASURED MOTOR ACTIVITY IN ADULT ADHD, GAD, AND HEALTHY CONTROL GROUPS

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Abstract: Attention-deficit/hyperactivity disorder (ADHD) is a complex neurocognitive disorder characterized by problems with attention, hyperactivity, and impulsivity. While previously considered a childhood disorder, recent research indicates that currently 4 to 5% of the adult population meets diagnostic criteria for ADHD. Furthermore, the presence of ADHD in adulthood has been associated with a number of negative outcomes such as a lower socioeconomic status, increased risk for substance abuse, traffic violations, and workplace difficulties. Hyperactivity is the primary reason for clinical referrals and is an important symptom in distinguishing ADHD subtype classifications. Additionally, hyperactive behaviors are associated with the most severe lifelong impairment due to their disruptive nature. Most notably, excessive hyperactive behavior is predictive of criminal activity in adulthood. The functional WM model of ADHD suggests that biological factors such as genetics influence the neurobiological system of WM and result in impaired CE functioning and a limited storage capacity in the PH and VS systems. In turn, these impairments lead to deficits in basic learning and attention abilities. Moreover, excessive levels of motor activity serve as a compensatory strategy to increase cortical arousal need to improve WM performance and to meet the environmental demands on central executive (CE) functioning. Notably, excessive motor activity such as restlessness and fidgeting are not pathognomonic symptoms of ADHD, and are often associated with other problems of psychopathology. For example, the diagnostic criteria of Generalized Anxiety Disorder (GAD) includes restlessness as a distinguishing symptom of the disorder. Furthermore, previous research indicates WM deficits in storage and rehearsal components directly affect ruminations or anticipatory processing associated with anxiety disorders. The topographical similarity of excessive motor activity seen in both ADHD and anxiety disorders, as well as similar WM deficits, may indicate a common relationship between WM deficits and increased motor activity across psychopathology. However, to date, no studies have examined the possible relationship between WM deficits and objectively measured motor activity associated with anxiety. Consequently, the current study examined objectively measured motor activity associated with the WM system in adults with ADHD, adults with GAD, and healthy control (HC) adults.

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| | HC (N=20) | ADHD (N=21) | GAD (N=21) | | |
|---|---|---|---|---|---|
| | M (SD) | M (SD) | M (SD) | $\chi^2$ | *F* |
| Gender (% male) | 30.0 | 46.6 | 23.8 | 2.86 | |
| Racial composition (% of group) | | | | 6.99 | |
| Caucasian | 70.0 | 85.7 | 90.5 | | |
| African American | 15.0 | 4.8 | 4.8 | | |
| Native American | 5.0 | 9.5 | 4.8 | | |
| Asian | 5.0 | 0.0 | 0.0 | | |
| Hispanic | 5.0 | 0.0 | 0.0 | | |
| Age in years | 18.80 (1.01) | 19.57 (1.91) | 19.14 (0.72) | | 1.76 |
| IQ Composite (KBIT-2) | 101.45 (8.50) | 102.76 (9.33) | 101.09 (11.82) | | .161 |
| Socioeconomic status[a] | 54.70 (7.95) | 52.09 (10.67) | 49.47 (7.54) | | 1.79 |
| Barkley-Current-Self | 0.10 (0.44) | 14.28 (9.18) | 3.76 (4.74) | | 30.88*** |
| Barkley-Child- Self | 0.60 (1.27) | 10.57 (4.74) | 3.71 (4.27) | | 37.34*** |
| Barkley-Current-Other | 0.30 (0.80) | 12.61 (3.58) | 1.76 (3.72) | | 101.45*** |
| Barkley-Child-Other | 0.20 (0.69) | 8.04 (5.27) | 1.52 (3.07) | | 28.58*** |
| PSWQ | 42.25 (13.09) | 53.66 (13.33) | 69.00(6.56) | | 28.46*** |

*Note.* HC = healthy control; KBIT-2 = Kaufman Brief Intelligence Test-2; PSWQ = Penn State Worry Questionnaire

[a] Scores are based on the Four Factor Index of Social Status (Hollingshead, 1975).

***p < .001

Table 1. Sample and Demographic Variables.

| | HC (N=20) M (SD) | ADHD (N=21) M (SD) | GAD (N=21) M (SD) | LSD Post Hoc |
|---|---|---|---|---|
| Phonological | | | | |
| Composite | 10,726 (4,946) | 18,072 (10,805) | 12,925 (6,399) | ADHD>GAD=HC |
| Set Size 4 | 10,953 (4,644) | 14,712(7,072) | 13,293 (6,807) | ADHD>GAD=HC |
| Set Size 5 | 10,570 (5,538) | 17,125 (8,049) | 11,926 (6,414) | ADHD>GAD=HC |
| Set Size 6 | 10,043 (5,524) | 17,333 (10,185) | 12,567 (6,143) | ADHD>HC; ADHD=GAD |
| Set Size 7 | 10,170 (4,546) | 19,463 (11,053) | 12,728 (6,260 | ADHD=GAD=HC |
| Visuospatial | | | | |
| Composite | 11,268 (7,133) | 16,585 (10,093) | 10,674 (4,160) | ADHD>GAD=HC |
| Set Size 4 | 11,805 (7,504) | 16,143 (10,005) | 10,676 (4,591) | ADHD=GAD=HC |
| Set Size 5 | 10,696 (8,856) | 17,495 (11,824) | 10,149 (5,076) | ADHD>GAD=HC |
| Set Size 6 | 11,305 (7,458) | 16,866 (10,711) | 11,774 (8,022) | ADHD=GAD=HC |
| Set Size 7 | 11,265 (5,900) | 15,836 (9,280) | 10,099 (4,287) | ADHD>GAD=HC |
| Control Conditions | | | | |
| Control 1 | 6,702 (3,508) | 8,755 (7,878) | 6,083 (3,410) | ADHD=GAD=HC |
| Control 2 | 8,830 (3,790) | 8,011 (5,297) | 8,726 (4,244) | ADHD=GAD=HC |

*Note.* HC = healthy control.

[a]Total activity level scores reported in Proportional Integrating Measure (loPIM) units.

Table 2. Composite and Set Size Comparison of Total Extremity Scores[a]

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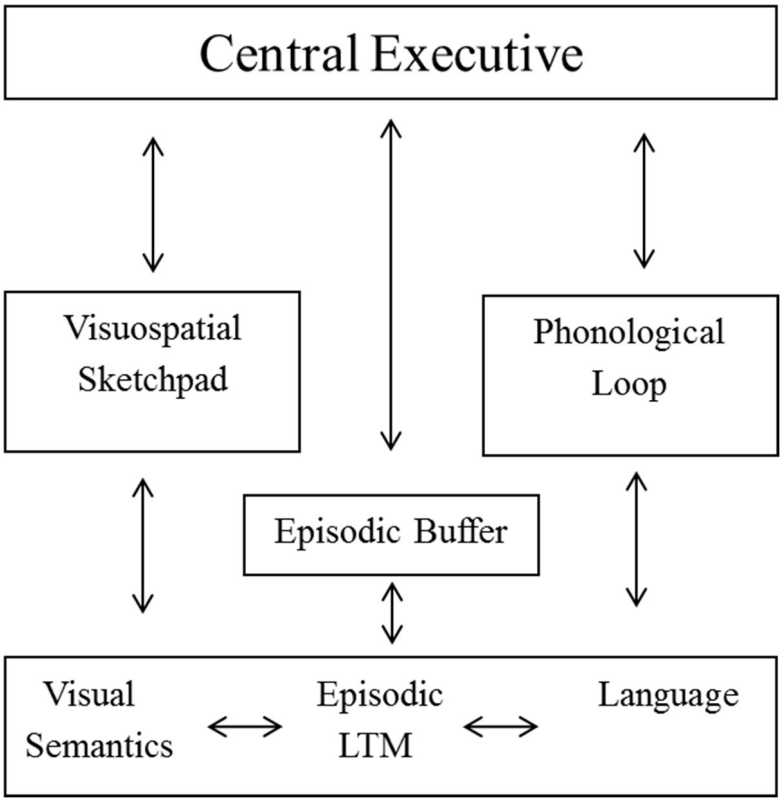



*Note*. LTM= Long term memory.

Figure 1. Visual schematic of Baddeley's (2007) multi-component model of working memory.

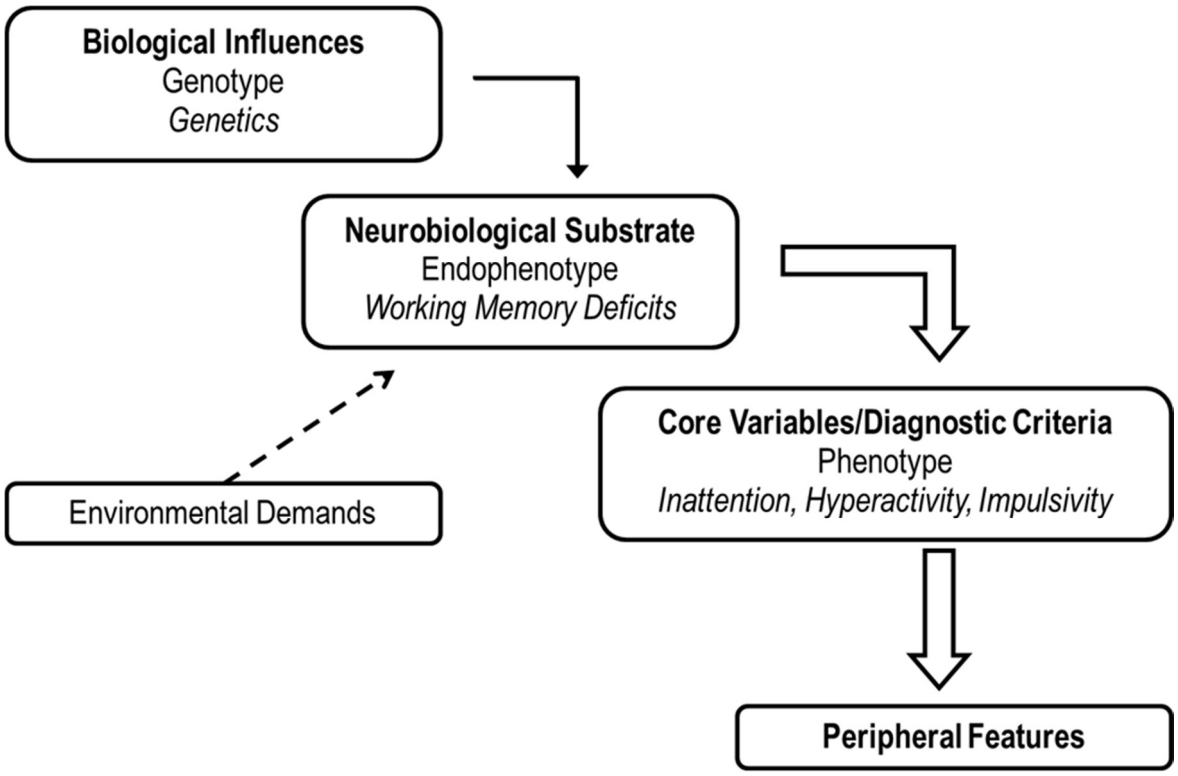
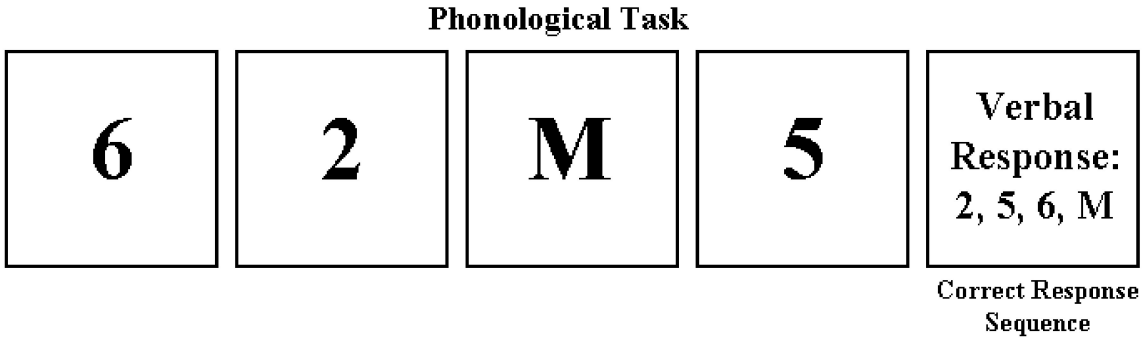



Figure 2. Visual schematic of Rapport and colleagues' (2008) Functional working memory model of ADHD

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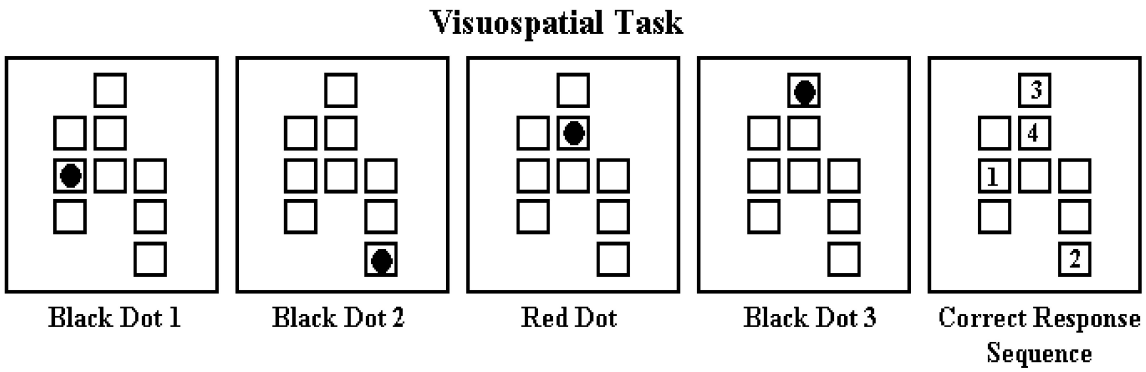


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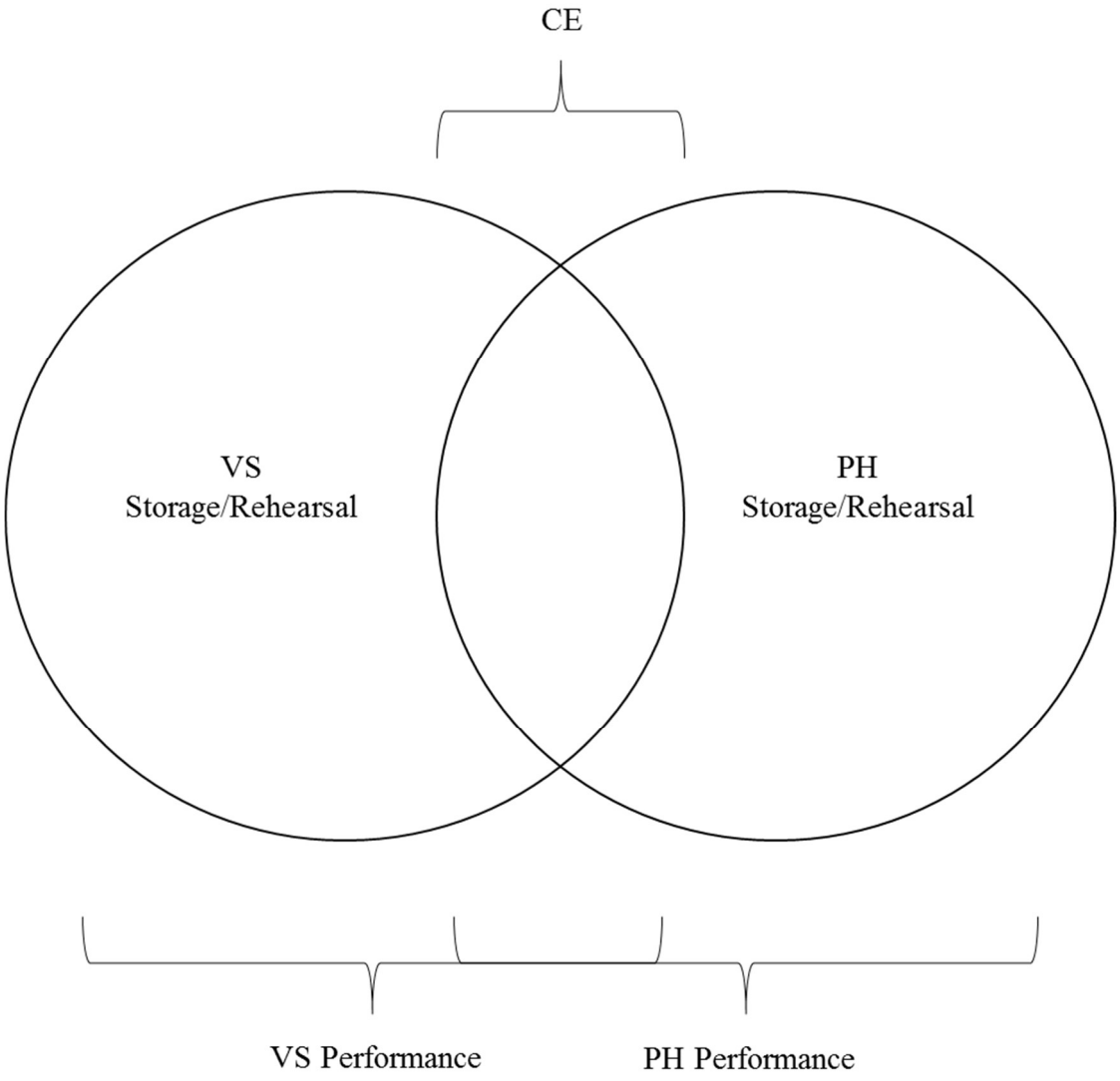


*Note*. CE= Central Executive, PH=Phonological, VS= Visuospatial

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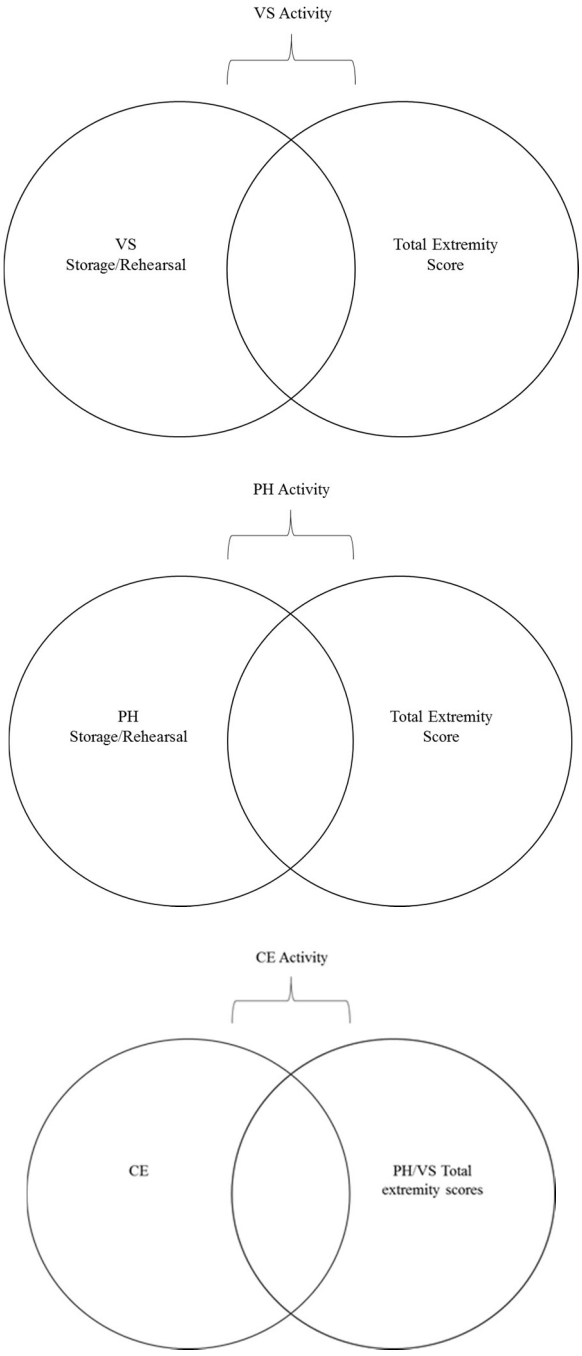


*Note*. CE= Central Executive, PH=Phonological, VS= Visuospatial

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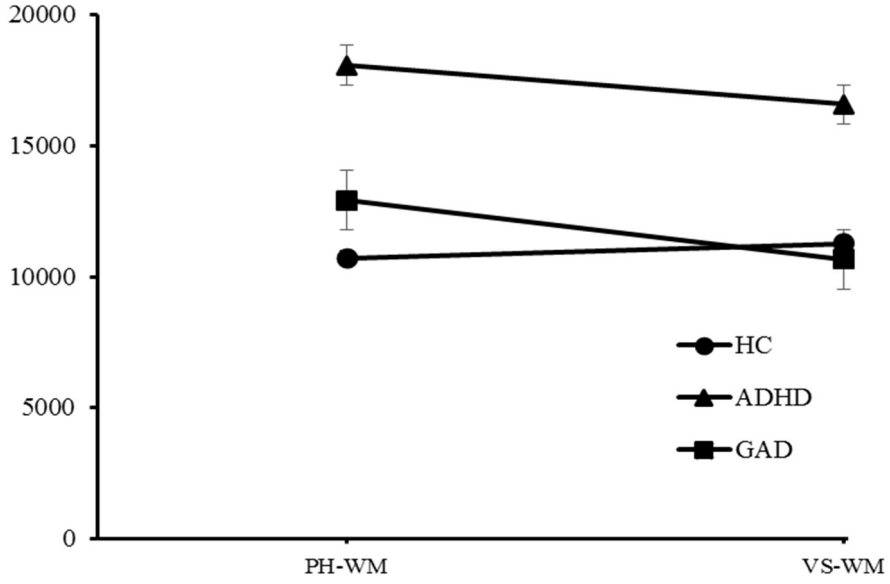
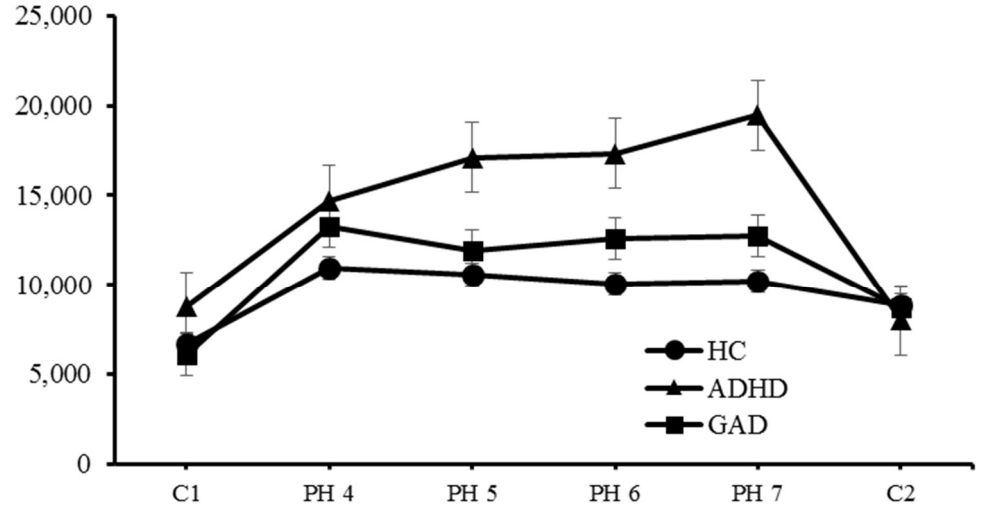



*Note.* Error bars represent standard errors. HC = healthy control; PH = phonological; VS = visuospatial.

Figure 7. Composite total extremity scores for working memory modalities.

(a)

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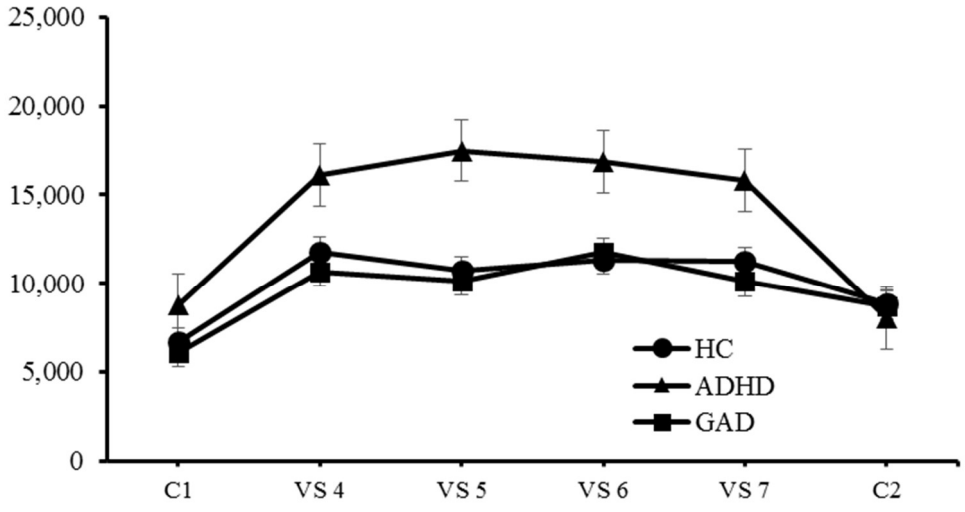


*Note.* Error bars represent standard errors. C= control condition; HC = healthy control; PH = phonological; VS = visuospatial.

Figure 8. Total extremity scores of phonological (a) and visuospatial (b) working memory and control condition

# CHAPTER I

## INTRODUCTION

Attention-deficit/hyperactivity disorder (ADHD) is a complex neurocognitive disorder characterized by problems with attention, hyperactivity, and impulsivity. While previously considered a childhood disorder, recent research indicates that 4 to 5% of the adult population meets diagnostic criteria for ADHD (Barbaresi et al., 2013). The presence of ADHD in adulthood has been associated with a number of negative outcomes such as a lower socioeconomic status, increased risk for substance abuse, traffic violations, and workplace difficulties (Barkley, Fischer, Smallish, & Fletcher, 2006; Biederman et al., 2008). Hyperactivity, the primary reason for ADHD referrals in outpatient clinics (Sayal, Taylor, Beecham, & Byrne, 2002), is associated with the most severe lifelong impairments due to its disruptive nature (Gaub & Carlson, 1997a; Faraone, Bierderman, Weber, & Russell, 1998; Hinshaw, 2002). Most notably, excessive hyperactive behavior is predictive of criminal activity in adulthood (Babinski, Hartsough, & Lambert, 1999), and hyperactive adults are at a higher risk for teen pregnancy (Harpin, 2005), are fired from more jobs, have fewer close friends, and are treated more often for sexually transmitted diseases, relative to non-hyperactive peers (Barkley et al., 2006; see Appendix A for a full review of ADHD).

Although hyperactivity serves as a core feature of ADHD in the DSM-5 (American Psychiatric Association, 2013), a growing body of research (Alderson, Kasper, Hudec, & Patros, 2013; Hudec, Alderson, Patros, & Kasper, 2014; Hudec at al., 2015; Sarver et al., 2016; Rapport et al., 2009) and models (Barkley, 1997; Rapport, Chung, Shore, & Isaacs, 2001; Songue-Barke, 2002) suggest that neurocognitive deficits/endophenotypes underlie ADHD-related hyperactivity that is viewed as a secondary outcome/phenotypic feature of the disorder. In particular, the working memory (WM) model of ADHD (Rapport, et al., 2001) suggests that there is a functional relationship between ADHD-related hyperactivity and demands on working memory (see Appendix B for a full review of theoretical models of ADHD). Specifically, the model suggests that biological factors such as genetics influence the neurobiological system of working memory and result in impaired CE functioning and a limited storage capacity in the PH and VS systems. These impairments lead to deficits in basic learning and attention abilities. Moreover, the model suggests that excessive motor activity serves as a compensatory strategy to increase cortical arousal needed to improve working memory performance and to meet the environmental demands on CE functioning (Rapport, Bolden, Sarver, Raiker, & Alderson 2009). A visual schematic of Rapport and colleagues' model is presented in figure 2.

The functional working memory model is based on Baddeley's (2007) multi-component model of working memory, which suggests working memory involves temporary storage and manipulation of mental information. Baddeley's model divides working memory into four subcomponents; the visuospatial (VS) sketchpad, which is responsible for temporary storage, rehearsal, and processing of visual and spatial

information; the phonological (PH) loop, which is responsible for temporary storage, rehearsal, and processing of auditory information; the domain- general central executive (CE), which is responsible for the division, switching, and maintenance of attention, as well as the manipulation of information in the VS and PH storage/rehearsal slave systems; and the episodic buffer, which is responsible for temporary storage of information presented via multiple modalities and provides a link between short term and long-term memory (Baddeley, 2007; Appendix C provides a review of alternative working memory models). A visual schematic of Baddeley's multi-component model of working memory is presented in figure 1.

Although a number of extant studies have demonstrated a functional relationship between working memory demands and ADHD-related hyperactivity in children with ADHD (Alderson et al., 2012; Hudec et al., 2015; Sarver et al., 2016; Rapport et al., 2009), relatively few studies have examined the relationship between motor activity and working memory demands in samples of adults with the disorder. The first of these studies examined the motor activity of 20 healthy controls and 20 adults with ADHD while they completed a VS 1-back working memory task (Lis et al., 2010). Motor activity was measured by calculating head movements greater than 1mm. Time active, distance of movement, and spatial area travelled were examined to gain a detailed picture of activity exhibited by both groups. Overall, it was determined that adults with ADHD were approximately 3.5 times more active than their healthy control counterparts. However, several factors limit conclusions that can be drawn from this study. For example, the exclusive use of a VS working memory tasks, in lieu of a PH working memory task, precludes inferences about the relationship between motor activity and PH

working memory processes. Moreover, the use of only a 1-back working memory task negates the ability to examine changes in motor activity as working memory load increases or decreases. This study also failed to include control conditions which limits conclusions that can be made about the presence of hyperactivity during both cognitively demanding and non-demanding tasks. That is, use of control (low WM demand) conditions allow for inferences about whether or not hyperactivity is present during all tasks or is dependent on situational demands related to working memory. Finally, the study's measurement of head movement failed to account for more finite movement in the extremities, potentially resulting in deflated estimates of gross motor activity (Hudec et al., 2014; Rapport et al., 2009).

A more recent study aimed at addressing the limitations of Lis and colleagues' 2010 study. Motor activity was measured via actigraphs located on the participants' non-dominant hand and two ankles. Actigraphs are wristwatch devices in appearance, and record the frequency, intensity, and duration of motor activity 16 times per second. The ability to measure finite motor activity via multiple methods (i.e., frequency, intensity, duration of movement) in the extremities is viewed as a more accurate metric relative to gross trunk or head movement (Hudec et al., 2014; Rapport et al., 2009, Tryon, 2005). Notably, both adults with ADHD and healthy controls exhibited greater activity during working memory conditions in relation to control conditions. Further, the CE and PH subsystems accounted for the largest between-group differences in activity while the VS subsystem did not account for motor activity after controlling for the CE (Hudec et al., 2014; see Appendix D for a review of studies examining working memory, ADHD, and hyperactivity).

Collectively, findings from carefully-controlled studies (Hudec et al., 2014; Lis et al., 2010) suggest that ADHD-related motor activity is functionally related to variability in working memory demands, consistent with predictions from Rapport and colleagues' (2008) functional working memory model of ADHD. Excessive motor activities such as restlessness and fidgeting are not pathognomonic symptoms of ADHD, however, and are often associated with other psychiatric disorders such as mania (Minassian et al., 2010), depression (Finazzi et al., 2010), and anxiety (Tryon, 2009). In particular, restlessness is the first of six symptoms listed as diagnostic criteria for Generalized Anxiety Disorder (GAD) in the DSM-5 (American Psychiatric Association, 2013). It is therefore not clear whether the relationship between working memory demands and motor activity is unique to ADHD, or a more general characteristic of psychopathology (see Appendix E for a review of anxiety disorders and working memory).

Similar to ADHD, anxiety disorders have been associated with a variety of domain-specific and domain-general working memory deficits indicating that these deficits may also be functionally associated with underlying causes of the disorder. For example, a recent meta-analytic review of anxiety and working memory found that high self-reported anxiety is associated with deficits in both storage/rehearsal and CE components of working memory (Moran, 2016; Owens, Stevenson, Hadwin, & Norgate, 2012). Experimental examinations have also found that highly anxious individuals, compared to their non-anxious counterparts, exhibit overall slower response times (MacLeod, & Donnellan, 1993; Miyake et al., 2000) and report experiencing greater feelings of worry and cognitive self-concern during the completion of working memory tasks (Ikeda, Iwanaga, & Seiwa, 1996). More recently, an examination of WM in

individuals with social anxiety found that WM load moderates attentional bias such that socially anxious individuals have problems disengaging or avoiding negative stimuli when WM load increases (Judah, Grant, Lechner, & Mills, 2012). Together, these findings suggest that working memory influences cognitive control and may limit executive resources associated with attentional control in anxiety disorders. Similar to predictions provided by the functional working memory model of ADHD, it follows that excessive motor activity in anxiety disorders may also serve as a compensatory strategy to increase cortical arousal needed to improve attentional control associated with tasks demands.

Although restlessness serves as diagnostic feature of GAD (American Psychiatric Association 2013), remarkably few studies have examined objectively measured motor activity in GAD, and no studies to date have examined objectively measured motor activity outside of the context of sleep (Tryon, 2009; Wicklow & Espie, 2000). Moreover, while the attentional control theory (Eysenck et al., 2007) suggests that worry manifests as verbal based inference that limits the PH storage/ rehearsal system, and implies the possibility of compensatory strategies used to maintain cognitive effectiveness, it does not address the possible relationship between working memory deficits and GAD-related restlessness/increased motor activity.

Collectively, the phenotypic similarities of increased motor activity and working memory impairments that are present in both ADHD and anxiety disorders suggest that the functional relationship between working memory demands and motor activity observed in studies of ADHD, might also generalize to anxiety disorders. This study is the first to examine the potential relationship between objectively measured motor

activity and working memory demands in adults with ADHD, adults with GAD, and healthy control (HC) adults to determine if the relationship between working memory and activity is unique to ADHD, or a more general phenomenon associated with psychopathology. Notably, this is also the first study to utilize actigraphy to objectively examine GAD-related motor activity outside of sleep. Lastly, this study examined the unique contribution of CE, PH storage/rehearsal, and VS storage/rehearsal processes on motor activity in adults with ADHD, GAD, and healthy controls. A priori, it was expected that adults with ADHD would be the most active on both PH and VS tasks followed by adults with GAD, and then healthy control adults. Adults with GAD, relative to the healthy control adults, were expected to be disproportionately more active during the PH condition. Additionally, all adults were expected to exhibit more motor activity during working memory conditions compared to non-working memory conditions. Finally, it was expected that when examining the unique contributions of CE, PH, and VS subsystems of working memory, adults with ADHD would exhibit the highest level of activity as it relates to all subsystems followed by adults with GAD and healthy controls, respectively.

# CHAPTER II

## HYPOTHESES

**Hypothesis I (Examination of Working Memory Modality on the Groups' Motor Activity)**:

A significant interaction between group (ADHD, GAD, healthy controls) and working memory modality (PH, VS) was expected. Adults with GAD, relative to the healthy control adults, were expected to be disproportionately more active during the PH condition. This was expected due to previous literature that suggests ruminations associated with GAD specifically interfere with PH storage rehearsal processes (Rapee, 1993). Adults with ADHD were expected to be the most active followed by adults with GAD, and then healthy control adults. This between-groups difference was expected based on previous literature that highlights working memory deficits and high rates of motor activity in both ADHD and anxious groups (Hudec et al., 2014; Tryon, 2009)

**Hypothesis II (Examination of Motor Activity Across PH Set Sizes):**

A significant interaction between group (ADHD, GAD, healthy controls) and PH working memory demand (C1, set sizes 4, 5, 6, 7, & C2) on activity was predicted. Based on models highlighting task effectiveness, efficiency deficits (Eysenck et al., 2007), and

PH working memory deficits (Rapee, 1993) in anxious adults, this interaction effect was predicted as it associates with disproportionate increase in activity in adults with GAD relative to healthy control adults during conditions of high working memory demand. Adults with ADHD were also expected to exhibit the highest levels of motor activity relative to GAD and healthy control adults.

**Hypothesis III (Examination of Motor Activity Across VS Set Sizes):**

Based on previous literature (Hudec et al., 2014), adults with ADHD were expected to exhibit the highest levels of motor activity relative to GAD and healthy control adults across VS set size. However, due to the limited research on VS working memory processes in adults with GAD, additional predictions were not made at this time.

**Hypothesis IV Examination of Working Memory Components' Contribution to Motor Activity:**

A. Based on previous literature identifying activity associated with PH storage/rehearsal processes in ADHD (Hudec et al., 2014), adults with ADHD were predicted to exhibit the highest levels of activity associated with PH storage/rehearsal, followed by adults with GAD, and then healthy control adults. This prediction was based on previous literature that indicates adults with GAD experience PH storage/rehearsal deficits associated with self-reported anxiety (Eysenck et al., 2007; Eysenck & Calvo, 1992; Rapee et al., 1993).
B. Based on previous literature (Hudec et al., 2014), adults with ADHD were predicted to exhibit the highest levels of activity associated with VS

storage/rehearsal, relative to both adults with GAD and healthy controls. However, additional predictions were not made at this time, due to the limited research on VS working memory processes in adults with GAD.

C. Adults with ADHD were predicated to exhibit the highest levels of activity associated with the CE, followed by adults with GAD than healthy control adults. This prediction was based on previous literature that indicates adults with ADHD experience exceptionally large CE deficits (Hudec et al., 2014), particularly relative to CE deficits associated with anxiety (Eysenck & Calvo, 1992).

**Hypothesis 5: Control Conditions**

Based on previous literature (Hudec et al., 2014), a small magnitude difference in activity between the ADHD group and the other two groups were expected after controlling for working memory. However, due to limited research available on working memory and activity in adults with GAD, additional hypotheses were not made at this time.

# CHAPTER III

## METHODOLOGY

### Participants

All participants were undergraduate students at a midwestern university, between the ages of 18 and 24 years, that completed the study to fulfill a research requirement for a university course. Nine thousand three hundred and thirty-two total participants were available for recruitment via an online participant pool over the course of data collection. Of the participant pool, 908 individuals completed a multi-laboratory online screening questionnaire that included the Barkley's Current Symptom Scale- Self-Report and the Penn State Worry questionnaire, provided contact information, and were sent a recruitment email based on meeting clinical cut-off scores of $\geq 4$ on the Barkley's ADHD Current Symptom Scale- Self-Report or clinical cut-off scores of $\geq 61$ on the Penn State Worry Questionnaire. Participants who reported no psychiatric history, less than four ADHD symptoms, and no clinically significant symptoms of worry were also invited to participate as potential members of the HC group. A total of 144 participants replied to the recruitment email and completed the study. Of the 144 participants, 82 were not included in the final analyses due to their failure to meet inclusion criteria of any of the three groups based on their clinical interview and rating scales. The excluded participants

were comprised of individuals with psychiatric symptoms or disorders not related to ADHD or GAD (e.g., major depressive disorder, *n* = 19; social anxiety disorder, *n* = 15; adjustment disorder, *n* = 43; and post-traumatic stress disorder, *n* = 5). The final sample of 62 participants was comprised 82.3% Caucasian, 8.1% African American, 6.5% Native American, 1.6% Asian, and 1.6% Hispanic participants. All participants gave their informed consent prior to their participation, and the university's Institutional Review Board approved the study procedures prior to the onset of data collection.

**Group Assignment.** Participants were included in the ADHD group if they met the following criteria: (a) a diagnosis of ADHD by a clinical psychologist using DSM-5 (American Psychiatric Association, 2013) criteria for ADHD, based on a semi-structured clinical interview and developmental history; (b) a symptom count ≥ 4 on the Barkley ADHD Current Symptoms Scale- Self-Report, (c) a symptom count of ≥ 6 on the Barkley Childhood Symptoms Scale-Other; and (d) no indication of current comorbid conditions based on supplemental rating scales, mental health history, or clinical interview. A symptom cutoff of 4 on the Barkley Current Symptoms Scale- Self-Report was used based on suggestions that the cutoff of 6 is too restrictive for identifying ADHD symptom presentations in adults (Barkley & Murphy, 2006; Simon, Czobor, Balint, Meszaros, & Bitter, 2009). Twenty-one participants (10 male) comprised the ADHD group and had an average age of 19.57 (*SD* = 1.91) years.

Participants were included in the GAD group (GAD) if they met the following criteria: (a) diagnosis of Generalized Anxiety Disorder by the directing clinical psychologist using DSM-5 (American Psychiatric Association, 2013) criteria based on a semi-structured clinical interview and developmental history; (b) a score of ≥ 61 on the

Penn State Worry Questionnaire (Mayer, Miller, Metzger, & Borkovec, 1990); (c) normal range ratings on the Barkley ADHD Current Symptoms Scale-Self Report, the Barkley Childhood Symptoms Scale-Other, mental health history, and clinical interview. Twenty-one participants (5 male) comprised the GAD group and had an average age of 19.14 (*SD* = .727) years.

Participants were included in the healthy control (HC) group if they met the following criteria: (a) a normal developmental history and no evidence of any clinical disorder based on mental health history and clinical interview; (b) a symptom count of < 61 on the Penn State Worry Questionnaire, (c) a symptom count of < 4 on the Barkley ADHD Current Symptoms Scale- Self-Report (Barkley &Murphy, 2006), and (d) symptom count of < 6 on the Barkley Childhood Symptoms Scale-Other (Barkley &Murphy, 2006). Twenty participants (6 male) comprised the HC group and had an average age of 18.80 (*SD* = 1.01) years.

Participants that presented with a history of seizure disorders, psychosis, gross neurological, sensory, or motor impairments, or a Full Scale IQ score of less than 85 were excluded. Participants with GAD that were taking prescribed anxiety medication were excluded from the study to eliminate any potential confounds associated with medication effects. Participants with ADHD that were taking prescribed psychostimulant medications were asked to discontinue the use of their medications for 24 hours prior to the laboratory session.

## Measures

### Clinical Interview

All participants completed the Kiddie Schedule for Affective Disorders and Schizophrenia-Present and lifetime Version (K-SADS-PL; Kaufam et al., 1997). Consistent with procedures employed in previous studies (Alderson et al., 2013; Belendiuk, Clarke, Chronis, & Raggi, 2007; Hudec et al., 2014; Magnússon et al., 2006), KSADS-PL questions were adapted for an adolescent population by using age-appropriate examples and by probing to measure past and present symptoms of psychopathology. The KSADS-PL has robust criterion and construct validity with adults samples and has a reliability range from 0.70 to 0.90 (Ambrosini, 2000; Belendiuk, et al., 2007; Magnússon et al., 2006). The KSADS-PL was used in lieu of other adult clinical interviews that lack questions about ADHD symptomology.

### ADHD Rating Scales

The Barkley report forms (Current Symptoms Scale-Self-Report and Childhood Symptoms Scale-Other Report, Barkley& Murphy, 2006) are widely administered to determine ADHD symptomology. Participants rated current behavioral and attention problems using Barkley's 18-item questionnaire (Current Symptoms Scale-Self-Report) designed to address DSM-5 diagnostic criteria of ADHD. Each item utilizes a 4-point Likert scale ranging from "never/rarely" to "very often." A parent of each participant retrospectively rated the participants' behavior and attention at ages 5 to 12 years using Barkley's 18-item questionnaire (Childhood Symptoms Scale-Other Report), designed to address DSM-5 diagnostic criteria of ADHD. Each item utilizes a 4-point Likert scale ranging from "never/rarely" to "very often." The Barkley report forms have internal

reliability ranging from .84 to .95 (Katz, Petscher, & Welles, 2009; Zucker, Morris, Ingram, Morris, & Bakeman, 2002) and have strong discriminant validity (Barkley, Murphy, DuPaul, & Bush, 2002).

**Anxiety Rating Scale**

The Penn State Worry Questionnaire (PSWQ, Mayer, Miller, Metzger, & Borkovec, 1990) is a 16-item self-report measure that is widely used to assess the trait of worry. Each item utilizes a 5-point Likert scale ranging from "not at all typical of me" to "very typical of me." The PSWQ has internal reliability ranging from .85 to 95 and has strong test-retest reliability (Mayer et al., 1990).

**Intellectual Functioning**

All participants completed the Kaufman Brief Intelligence Test-Second Edition (K-BIT2; Kaufman& Kaufman, 2004). The KBIT-2 consists of three subtests that comprise two subtest scores (Nonverbal, Verbal) and an overall IQ composite score. K-BIT2 scores have a mean of 100 and a standard deviation of 15. The overall IQ composite score has strong content and predicative validity, and has high internal–consistency reliability that ranges from .89 to .96 and test-retest reliability that ranges from .76 to .93 (Kaufman & Kaufman, 2004).

**Phonological Working Memory Task**

The PH working memory task used in the current study has been utilized in multiple examinations of PH working memory in child (Rapport et al., 2008: Rapport et al., 2009) and adult (Alderson et al., 2013; Hudec, et al., 2014) samples of ADHD. The PH working memory tasks were programmed using SuperLab 4.0 (SuperLab Pro, 2008) and are stylistically similar to the Letter-Numbering Sequencing subtest in Wechsler

intelligence tests (Wechsler, 2008). Participants heard a computer say a series of single digit numbers and one letter taken from a prerecorded stimuli sequence. The position of the single letter in the sequence of stimuli was counterbalanced across all trials to occur equally in all positions, except first or last to reduce primacy and recency effects. Further, no number was repeated in the sequence. Each stimulus was followed by a 200-ms interstimulus interval, and each trial was followed by an auditory click and the appearance of a green light on the computer screen to signal that participants should give their verbal response. Participants were instructed by the experimenter to recall the numbers and letters and say the numbers aloud in order from the smallest to largest, followed by the letter last. Indicating the position of the letter last, rather than its position during the presentation of the stimuli is expected to place high demands on the CE component of working memory as it requires attentional shifts, rehearsal/maintenance of information temporarily held in the buffer and storage components, and mental manipulation of information into the correct response. After verbally responding, participants touched the computer screen to advance to the next trial. Trials also advanced if participants failed to touch the screen after a delay corresponding with 10 seconds for each stimuli presented in the trial (i.e. 60 seconds during trials of six stimuli). Each verbal response was followed by an inter-trial interval of 1,000-ms and an auditory click to indicate the beginning of a new trial. Trials consisted of four to seven stimuli and each set-size block consisted of 24 trials for a total of 96 trials. The presentation order of set size blocks was counterbalanced across participants to avoid possible carry over and practice effects. Five practice trials were administered and participants were required to provide a correct verbal response to 80% (4 trials) of the practice trials before proceeding

to experimental trials. Participants did not receive feedback about their performance during practice or experimental trials. All verbal responses were coded individually by two research assistants in a nearby room out of the participants' view. The coded verbal responses were then compared by a third research assistant to ensure reliable and valid coding procedures. Any discrepancies in codes were addressed by reviewing video recordings of the experimental session and recoding until all discrepancies were eliminated. A visual schematic of the PH working memory task is provided in Figure 3.

**Visuospatial Working Memory Task**

The VS working memory task used for this study has also been used in multiple examinations of VS working memory based on Baddeley's (2007) model (Alderson et al., 2013; Hudec et al.,2014) and was programmed using SuperLab 4.0 (SuperLab Pro, 2008). The VS working memory task was presented to participants on a 17 x 14 inch touch-screen monitor. A series of 2.5 centimeter in diameter dots were presented to participants for 800 ms each. Each dot was presented in one of nine 3.2-centimeter squares arranged in three offset columns. These columns were offset to reduce the likelihood of PH coding of the stimuli (e.g., assigning numbers to each square location). One dot in each trial was red and all other dots were black. No two dots appeared in the same location during a single trial and the location of the red dot was counterbalanced across trials to appear in each square an equal number of times. Similar to the letter in the PH working memory task, the red dot never appeared first or last in a sequence to avoid primacy or recency effects. After the presentation of each dot, there was a 200-ms interstimulus interval followed by an auditory click and the appearance of a blank grid of boxes. Participants were instructed to respond by first touching the boxes on the screen in

the same order and location in which the black dots appeared, and then where the red dot appeared. The reordering of stimuli and the placement of the red dot last is intended to serve the same purpose as the letter in the PH task. Participants were allowed to respond for a maximum of 10,000 ms per stimulus (i.e 60,000 ms for set size 6). Each response was followed by an inter-trial interval of 1,000 ms and an auditory click that indicated the beginning of a new trial. Trials consisted of four to seven stimuli and each set size bloc consisted of 24 trials for a total of 96 trials. The presentation order of set sizes was counterbalanced across participants to avoid possible carryover or practice effects. Five practice trials were administered to each participant before experimental trials and participants were required to provide a correct verbal response to 80% (4 trials) of the practice trials before proceeding. Participants did not receive feedback about their performance during practice or experimental trials. A visual schematic of the VS working memory task is provided in Figure 4.

**Control Conditions**

The control conditions were based on previous protocols established in examinations of working memory in children and adults with ADHD (Alderson et al., 2013; Hudec et al., 2014; Rapport et al., 2008; Rapport et al. 2009). Participants were asked to draw or paint anything of their choice with the Microsoft Paint program. This computer-based task was expected to place minimal working memory demands on participants because they are not required to temporarily store, recall, or rehearse information (Hudec et al., 2014). Participants used the program for five consecutive minutes before (C1) and after (C2) completing the working memory tasks. The use of

two control conditions allowed for the examination of possible fatigue effects on overall motor activity.

**Activity Measurement**

MicroMini Motionlogger ® Actigraphs (Ambulatory Monitoring INC., 2010) are devices similar to wristwatches in appearance that measure frequency, intensity, and duration of motor activity 16 times per second. Actigraphs have been previously used as objective measures of motor activity in studies examining children and adults with ADHD (Hudec et al., 2014; Rapport et al., 2009), as well as studies examining sleep disturbances in anxious children and adults (Kain and Cadlwell-Andrews, 2003; Wicklow and Espie, 2000). Actigraphs have been found to be reliable and valid (Tryon, 2005; Tryon & Williams, 1996), and have a re-test reliability ranging from 0.90 to 0.99 (Tryon, 1985). Actigraphs were attached with a Velcro band to each participants' non-dominant wrist and above each ankle. Actigraphs were not placed on participants' dominant hand to eliminate any activity measurements associated with task response. Participants were informed of the actigraphs' purpose to record physiological data, but no other information was provided to reduce potential changes in natural motor activity that may occur due to demand characteristics. All actigraphs were set on the proportional integrating measure (loPIM) mode to measure participants' gross motor activity intensity, rather than frequency. The Observer XT (Noldus Information Technology, 2008) live observation software was used to record time stamps for the start and stop of each task. Actigraph data was uploaded into the Action4 (Ambulatory Monitoring Inc., 2010) computer software program and was then matched to corresponding time stamps for analyses.

### Dependent Variables

A Total Extremity Score (TES) (Rapport et al., 2009) was calculated by adding activity scores from the non-dominant hand, left ankle, and right ankle. The TES was used as the dependent variable representing overall activity during each condition. This procedure was used instead of averaging activity scores across the three locations to provide a broader examination of individual variability in activity associated with any one extremity. This procedure also eliminates any potential deflation effects resulting from motion variability across extremities.

## Procedure

Participants first independently completed a multi-laboratory online screening questionnaire required for course credit in their Introduction to Psychology undergraduate course. This screening questionnaire included the PSWQ, the Current Symptoms Scales-Self-Report, and a question that asked if they had been previously diagnosed with ADHD. Participants who self-reported a PSWQ score of $\geq 61$ were invited via email to participate in the study as a likely member of the GAD group. Participants with no psychiatric history, a score of $< 61$ on the PSWQ, and fewer than 4 symptoms on the Current Symptoms Scales Self-Report, were invited via email to participate as a likely member of the HC group. Finally, participants with a previous diagnosis of ADHD and/or $\geq 5$ symptoms on the Current Symptoms Scales Self-Report were invited via email to participate as a likely member of the ADHD group. Individuals who did not qualify for any group were eliminated during this initial screening procedure. Prior to the laboratory session, participants were asked to complete an additional online questionnaire that

assessed health history, employment history, and social development. This online questionnaire required 20-30 minutes to complete.

Each participate completed one laboratory session, and upon the arrival a session administrator reviewed an informed consent form and obtained the participants' consent to participate. The K-SADS-PL was administered followed by a three to five minute break and the administration of the KBIT-2. Each interview was video recorded to allow another advanced doctoral student coder to review the clinical interview for interrater agreement of group assignment and diagnosis. The interrater agreement for group assignment and diagnosis was 100%. Participants subsequently completed the PH, VS, and control conditions alone, seated on a swivel chair approximately .70 m from a computer monitor in the testing room. Set size and working memory modality were counterbalanced to control for possible carryover or order effects. The two control conditions always occurred first and last. All participants were offered 2 to 3 minute breaks as needed and between tasks. The entire laboratory session lasted approximately 2.5 hours.

# CHAPTER IV

# RESULTS

## Preliminary Analyses

**A priori power analyses.** A prior power analyses were conducted using G* Power software (v 3.12; Faul, Erdfelder, Lang, & Buchner, 2007) to determine the number of participants required for this study to reliably detect within-subject, between-subject, and interaction effects in a repeated measure ANOVA. A Cohen's *d* effect size of 1.40 was chosen based on recent studies investigating motor activity and executive functions in adults with ADHD (Lis et al., 2010; Hudec et al., 2014). The effect size associated with adult ADHD research was chosen due to the lack of studies examining motor activity and executive functioning in GAD samples. Based on Cohen's (1992) conventions, power was set at 0.80 and an alpha level of 0.05 was chosen. Based on these values and the inclusion of 3 groups and 4 conditions (set sizes 4, 5, 6, and 7), 18 total participants (6 per group) are needed to detect within-subject, between-subject, and interaction effects. The current study's sample included 62 participants (21 ADHD, 21 GAD, 20 HC), suggesting it is adequately powered Sample and demographic variable are presented in table 1.

**Outliers**

Data from each laboratory task was screened for values ≥ 3.29 standard deviations above or below the group mean (i.e., $p < .05$) that could potentially influence analyses (Tabachnick & Fidell, 2001). One HC participant's TES on VS4, VS5, and VS6 were identified as outliers. One GAD participant's TES on VS6 was identified as an outlier. Each TES outlier score was replaced with the activity value equal to 3.29 *SD*s for the group mean. A Chi-squared test revealed no significant between-group differences in race/ethnicity, $\chi^2$ (2) = 6.99, $p = .537$ or gender, $\chi^2$ (2) = 2.86, $p = .240$. Further, a one-way ANOVA revealed no significant between-group differences in SES, $F$ (2, 59) = 1.79, $p = .177$, age, $F$ (2, 59) = 1.76, $p = .181$, or IQ, $F$ (2, 59) = .161, $p = .852$. A one- way ANOVA revealed significant between-group differences in Self-report Current, $F$ (2, 59) = 30.88, $p < .001$, Self-report Child, $F$ (2, 59) = 37.34, $p < .001$, Other-report Current, $F$ (2, 59) = 101.45, $p < .001$, and Other report-Child, $F$ (2, 59) = 28.58, $p < .001$, ADHD rating scales. LSD post hoc analyses revealed that the ADHD group had significantly higher scores compared to the GAD group and the HC control group on Self-report Current, Self-report Child, Other-report Current, and Other-report Child (all $p <.001$). The GAD group had significantly higher scores than the HC group only on the Self-report Child ADHD scale ($p = .011$). This result is not surprising due to the symptom overlap (e.g., attention and concentration difficulties) between the GAD and ADHD diagnoses. A one- way ANOVA revealed significant between-group differences in the PSWQ, $F$ (2, 59) = 28.46, $p < .001$. LSD post hoc test revealed the GAD had significantly higher rates of reported anxiety than both the ADHD ($p < .001$) and HC

groups ($p < .001$), while the ADHD group had significantly higher self-reports of anxiety than the HC group ($p = .002$) .

**Tier 1: Examination of Working Memory Modality on the Groups' Motor Activity**

A composite TES for each modality (PH and VS) was computed by averaging the TES from each modality's set sizes (4, 5, 6, and 7), and a 3x2 mixed-model ANOVA was used to examine differences in activity across working memory modalities (PH, VS) and groups (ADHD, GAD, & HC). The main effect for working memory modality, $F$ (1, 59) = 3.25, $p$ =.077, and the interaction effect, $F$ (1, 59) = 1.71, $p$ = .190, were not significant. However, there was a significant main effect for group, $F$ (2, 59) = 4.78, $p$ = .012. LSD post hoc comparisons indicated that the ADHD group exhibited significantly greater activity relative to the GAD ($p$ = .016, $d$ = .760) and the HC ($p$ = .006, $d$ = .871) groups. The activity exhibited by the GAD group was not significantly different compared to activity exhibited by the HC group ($p$ = .714, $d$ = .110).

**Tier 2: Examination of Motor Activity Across PH Set Sizes**

A 3x7 mixed-model ANOVA was used to examine differences in activity across PH-working memory conditions (C1, set sizes 4, 5, 6, 7, & C2) and group (ADHD, GAD, & HC). The interaction between condition and group was significant, $F$ (10, 295) = 3.79, $p < .001$. Six post-hoc ANOVAs were completed to probe between-group main effects at each condition and revealed no significant between groups differences on C1 ($F$ (2, 59) = 1.42, $p$ = .251), C2 ($F$ (2, 59) = .208, $p$ = .813), or PH7 ($F$ (2, 59) = 2.83, $p$ = .067), and significant between group effects for PH4 ($F$ (2, 59) = 4.85, $p$ = .011), PH5 ($F$ (2, 59) = 4.85, $p$ = .011), and PH6 ($F$ (2, 59) = 3.84, $p$ = .027).

Three post-hoc repeated measures ANOVAs, one for each group, were used to probe for activity differences across conditions. Main effects for condition were significant for the HC group, $F$ (5, 95) = 4.55, $p$ =.001, the ADHD group, $F$ (5, 100) = 16.30, $p$ <.001, and the GAD group, $F$ (5, 100) = 5.00, $p$ <.001. Post hoc pairwise comparisons found that HC adults and adults with GAD exhibited significantly less activity on C1 compared to C2, PH4, PH5, PH6, and PH7 (all $p$ < .05, $d$ = .582-1.30). Further, there were no significant differences in activity on PH4, PH5, PH6, and PH7. Additionally, adults with GAD exhibited significantly less activity on C2 compared to PH4 ($p$ = .013, $d$ = .805), PH6 ($p$ = .022, $d$ = .727), and PH7 ($p$ = .010, $d$ = .748), while HC adults did not. Post hoc pairwise comparison revealed no significant activity differences between C1 and C2 ($p$ = .589, $d$ = .110) in the ADHD group. Moreover, adults with ADHD exhibited significantly less activity on both C1 and C2 compared to PH4 ($d$ = 1.11), PH5 ($d$ = 1.05), PH6 ($d$ = .942), and PH7 ($d$ = .795; all $p$ < .001). Additionally, the ADHD group exhibited higher activity on PH7 compared to PH4 ($p$ = .029, $d$ = .512). There were no significant differences in activity across PH4, PH5, PH6, or PH7.

**Tier 3: Examination of Motor Activity Across VS Set Sizes**

A 3x7 mixed model ANOVA was used to examine differences in activity across VS-working memory condition (C1, set sizes 4, 5, 6, 7, & C2) and group (ADHD, GAD, & HC). The interaction between condition and group was significant, $F$ (10, 295) = 3.28, $p$ < .001. Six post-hoc ANOVAs were completed to probe between-group main effects at each condition and revealed no significant between group differences on C1 ($F$ (2, 59) = 1.42, $p$ = .251), C2 ($F$ (2, 59) = .208, $p$ = .813), VS4 ($F$ (2, 59) = 2.99, $p$ = .058), or VS6

($F$ (2, 59) = 2.82, $p$ = .068), and significant between group effects for VS5 ($F$ (2, 59) = 4.76, $p$ = .012) and VS7 ($F$ (2, 59) = 4.13, $p$ = .021).

Three post- hoc repeated measures ANOVAs, one for each group, were used to probe for activity differences across conditions. Main effects for condition were significant within the HC group, $F$ (5, 95) = 5.00, $p$ <.001, ADHD group, $F$ (5, 100) = 17.74, $p$ <.001, and GAD group, $F$ (5, 100) = 5.00, $p$ <.001. Post hoc pairwise comparisons found that both HC adults and adults with GAD exhibited significantly less activity on C1 compared to C2, VS4, VS5, VS6, and VS7 (all p < .05, $d$ = .582-1.13). Further, there were no significant differences in activity on VS4, VS5, VS7, or C2. However, adults with GAD had significantly higher activity on VS6 compared to C2 ($p$ = .044, $d$ = .714). Post hoc pairwise comparison revealed the ADHD exhibited no significant activity differences across C1 and C2 ($p$ = .589 $d$ = .110), but exhibited significantly less activity on both C1 and C2 compared to VS4 ($d$ = .820), VS5 ($d$ = .869), VS6 ($d$ = .862), and VS7 ($d$ = .822; all $p$ <.001).

**Tier 4: Examination of Working Memory Components' Contribution to Motor Activity**

Three dependent variables that reflect estimates of CE, PH, and VS processes were created using a regression approach as outlined in previous literature (Alderson, et al., 2013; Hudec, et al., 2014). Briefly, PH performance scores (i.e., average stimuli recalled correctly) for each set size were regressed on to corresponding VS scores to create four predicted scores representing shared variance between the variables. Similarly, VS performance scores were regressed onto the corresponding PH score at each set size to create four additional predicted scores. The resulting eight predicated

scores were averaged to create a variable that reflect estimated CE processes. Residual scores created from these regressions provided estimates of VS and PH storage/rehearsal processes. A visual schematic of the regression approach is depicted in Figure 5.

VS storage/rehearsal performance scores at each set size were regressed onto the corresponding set-size TES to create four new variables that represent the shared variance between performance and activity (i.e., activity associated with VS processes). The four variables were then averaged to create a single variable that represents the overall activity that is associated with VS storage/rehearsal processes. The identical procedure was completed for the PH tasks to create a single variable that represents the overall activity that is associated with PH storage/rehearsal processes. Finally, the CE performance variables for each set size were regressed onto the related PH and VS TES. This created eight variables that represent the shared variance between CE performance and activity (i.e., activity associated with CE processes). The eight variables were then averaged to create a single variable that represents the activity that is associated with CE processes. A visual schematic of the regression approach is depicted in Figure 6.

One-way ANOVAs revealed a significant between-group main effects on activity associated with the VS storage/rehearsal processes, $F$ (2, 61) = 3.31, $p$ = .043, PH storage/rehearsal processes, $F$ (2, 61) = 5.98, $p$ = .004, and CE processes, $F$ (2, 61) = 4.58, $p$ = .014. LSD post hoc analyses revealed that the ADHD group exhibited significantly greater activity associated with VS storage/rehearsal processes than both the GAD group ($p$ = .023, $d$ = .709) and the HC group ($p$ = .040, d = .569). Post hoc analyses also indicated that the ADHD group had significantly greater activity associated with PH storage/rehearsal processes than both the GAD group ($p$ = .020, $d$ = .657) and the HC

group ($p$ = .001, $d$ = 1.00). Finally, the ADHD group had significantly greater activity associated with CE processes than both the GAD group ($p$ = .020, $d$ = .696) and the HC group ($p$ = .007, $d$ = .773). There were no differences, however, between the GAD and HC groups with regard to activity associated with VS ($p$ = .839, $d$ = .080), PH ($p$ = .319, $d$ = .393) or CE processes ($p$ = .670, $d$ = .174).

**Tier 5: Control Conditions**

Performance variables were regressed onto activity at each control condition to examine between group differences in activity during the control condition after controlling for activity associated with CE, PH, and VS processes. That is, residual scores from each regression represented activity during the control conditions not associated with working memory. A 3 (ADHD, GAD, & HC) x 2 (C1, C2) mixed model ANOVA revealed that the main effects for condition, $F$ (1, 59) = .000, $p$ =.995, and group, $F$ (2, 59) = 1.55, p = .221, were not significant. Additionally, the interaction between group and condition was not significant, $F$ (2, 59) = 1.16, $p$ = .319

# CHAPTER V

## DISCUSSION

The current study examined the role of working memory demands on objectively measured motor activity across adults with ADHD, GAD, and healthy controls. While previous research has supported a functional relationship between working memory demands and motor activity in both children (Alderson et al., 2012; Porrino et al., 1983; Rapport et al., 2009) and adults with ADHD (Lis et al., 2010; Hudec et al., 2014), few studies have examined motor activity in GAD (Tryon, 2009; Wicklow & Espie, 2000), and no studies to date have examined objectively-measured, GAD-related motor activity outside of the context of sleep difficulties.

Overall, the groups exhibited similar activity across PH and VS working memory modalities. These findings are not surprising with respect to the ADHD group, as they are consistent Hudec et al.'s (2014) previous findings, as well as recent meta-analytic findings that revealed similar magnitude PH and VS performance deficits in adults with the disorder (Alderson et al., 2013). However, these results are somewhat surprising as they relate to the GAD group, given previous literature that suggests ruminations associated with GAD specifically interfere with PH storage rehearsal processes (Rapee,1993). However, more recent examinations have indicated that anxiety may not affect working memory in a domain specific fashion (Moran, 2016), but rather more globally. Consistent with this study's a priori hypotheses and findings from a previous study of ADHD-related motor activity in adults, examinations of between-group effects

across analyses in the current study indicated that adults with ADHD regularly exhibited greater motor activity compared to adults with GAD and healthy controls across both PH and VS working memory modalities. A closer examination of between group differences at each control and set-size condition revealed a few exceptions, however. First, as expected, the groups' activity was not significantly different during the control conditions, suggesting motor activity was not ubiquitous. Next, the groups were significantly different at PH set sizes 4, 5, and 6. Less clear is the finding that VS between-group differences were only significant at set sizes 5 and 7. This unexpected pattern of findings might reflect insufficient power, such that a larger sample size would likely yield significant between-group effects at set sizes 4 and 6 as well (Button et al., 2013). Lastly, adults with GAD, did not differ significantly in motor activity relative to healthy controls. These results are surprising given the role of restlessness as a diagnostic feature of GAD. However, theoretical conceptualizations of GAD do not specify a relationship between GAD-related working memory deficits and increased motor activity (Behar et al., 2009). Therefore, it is possible that manipulation of alternative unexamined variables may elicit between group differences. A closer examination of activity changes across increasing PH and VS working memory demands (i.e., C1, C2, and set sizes 4, 5, 6, and 7) revealed a number of notable findings. First, there was a significant interaction between group and working memory demands in both the PH and VS analyses. As expected, all groups exhibited increases in motor activity from C1 to all PH and VS set sizes. In addition, with just one exception (i.e., ADHD activity was greater during PH7 relative to PH4), the groups' activity remained relatively stable across the PH and VS set sizes. This finding is particularly notable for the GAD group, given previous research that

has highlighted task effectiveness/efficiency deficits (Eysenck et al., 2007), and consequently suggest task efficiency would decrease as cognitive load increases. It would be expected that as adults with GAD become more taxed at higher PH working memory loads, lack of efficiency would lead to disproportion increases in activity as activity would serves as a compensatory strategy needed to increase task effectiveness. Similar findings were revealed in studies of children (Rapport et al., 2009) and adults (Hudec at al., 2014) with ADHD, and likely suggest that changes in motor activity are likely due to increased CE demands, rather than changes in storage/rehearsal processes. That is, CE demands are expected to remain relatively constant across set sizes that place varying demands on buffer capacity (Baddeley, 2012) and rehearsal processes (Baddeley,2007).

Surprisingly follow-up within-group analyses also found an increase in C2 as it relates to C1 in both adults with GAD and healthy controls, but not ADHD. This unexpected finding might be explained by a fatigue effect and/or a cumulative depletion of cognitive resources. That is, the adults with GAD and healthy controls may have successfully allocating available resources to complete the working memory task, which in turn taxed and depleted cognitive resources and result in activity increases during non-working memory tasks (Baddeley, 2003; Muraven & Baumeister, 2000). This hypothesis appears consistent with the attentional control theory of anxiety, which suggests compensatory strategies can be used to maintain cognitive effectiveness during difficult tasks, but overall efficiency decreases following depletion of resources (Eysenck & Calvo, 1992). Collectively, findings from post-hoc between- and within-group probes appear to suggest that, while all groups exhibit increased motor activity during high working memory demand conditions (PH and VS set sizes 4-7), the significant

interactions resulted from disproportionate increases in activity by the ADHD group, while the GAD and HC groups appeared to covary.

Lastly, similar to previous research (Hudec et al., 2014; Rapport et al., 2009), the current study examined the contribution of CE and storage/rehearsal processes to motor activity. Collectively, greater activity exhibited by the ADHD group, relative to the GAD and HC groups, was associated with PH and VS storage/rehearsal processes, as well as the CE. Again, this finding is generally consistent with previous research, albeit Hudec and colleagues' (2014) study did not yield a significant effect associated with the VS storage rehearsal. Again, the lack of significant differences in activity associated with CE and PH storage/rehearsal process in adults with GAD and healthy controls is particularly surprising given previous literature noting deficits in these systems in adults with GAD (Eysenck et al., 2007; Eysenck & Calvo, 1992; Rapee et al., 1993).

Overall, findings from this study provide important information about the relationship between motor activity and working memory adults in GAD and ADHD. Nevertheless, this study is not without limitations. First, the sample examined in the current study is relatively small. Small samples are associated with increased risk of Type II errors and limited generalizability to larger groups of individuals with ADHD or GAD, and consequently, findings should be interpreted with caution. Our a priori power analyses, however, indicated that the current sample size was considerably greater then what was needed to detect interaction effects, as well as between- and within-group main effects. Further, significant interaction effects were found in nearly all of the primary analyses of interest indicating sufficient power. Another potential limitation is that participants were recruited online via a participant pool that received course credit at a

public university. That is, since participants were non-treatment seeking individuals, it is possible that symptomology experienced by the GAD sample is less severe than what might be found in a clinical sample. However, all participants included in this study were carefully assessed and diagnosed using multidimensional assessment procedures consistent with typical clinical diagnostic practices. Lastly, the current study's inclusion of individuals with the predominately inattentive presentation of ADHD may have served as a limitation, considering the primary purpose of this study was to examine motor activity which is often not as prominent in the inattentive presentation. Regardless, significant results were found in the primary analyses of interest and future studies may aim to examine the relationship between the various ADHD presentations and motor activity.

Collectively, findings from the current study suggest that the relationship between working memory and objectively measured motor activity may be uniquely associated with underlying features associated with ADHD. Furthermore, current findings ultimately suggest that adults with GAD are no more active, restless, or fidgety than healthy control adults. This is particularly surprising due to the GAD DSM-5 diagnostic criteria of "restlessness" (American Psychiatric Association, 2013). Moreover, it seems apparent that the diagnostic symptom relies more on a subjective clinical evaluation of an individual rather than core and objective symptoms of the disorder. Notably, current theoretical models of GAD (Borkovec, 1994; Dugas & Keroner, 2005; Mennin, et al., 2004; Roemer & Orsillo, 2005; Wells, 2005) also do not attempt to explain or conceptualize the role of the DSM-5 diagnostic symptom of restlessness (American Psychiatric Association, 2013). Based on these findings, a more in depth examination of

the diagnostic symptoms of GAD and a reevaluation or the core symptoms of the disorder are likely warranted. As the current criteria stand, little empirical evidence is available to support increased motor activity as a diagnostic feature of GAD.

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APPENDICES

## Appendix A

### Overview of Attention Deficit/Hyperactivity Disorder

Attention-deficit/ hyperactivity disorder (ADHD) is a complex neurocognitive disorder characterized by difficulties with inattention, impulsivity, and hyperactivity that interfere with daily functioning and development in childhood (Barkley, 2006). Examples of inattention include frequent off task behavior and concentration difficulties (Bauermeister et al., 2005), while impulsivity is typically defined as acting without foresight (Winstanley, Eagle, & Robbins, 2006) and an inability to delay gratification (Williams & Dayan, 2005). ADHD-related hyperactivity refers to developmentally inappropriate excessive motor activity, restlessness, and fidgeting (Barkley, 1998). Factor analytic studies have indicated that the predominant symptoms associated with ADHD fall within three main clusters, hyperactivity and impulsivity, inattention, and a combination of hyperactivity/impulsivity and attention deficits (ADHD-C; Gaub & Carlson, 1997a). These clusters are reflected in the DSM-5 (American Psychiatric Association, 2013) as predominantly hyperactive/impulsive presentation (ADHD-H), predominantly inattentive presentation (ADHD-I), and combined presentation. However, some factor analytic reviews indicate that two symptom clusters – hyperactive/impulsive and inattentive - more accurately represents clinician reported variation in symptoms of ADHD (Lahey et al. 1998). Finally, previous reviews indicate that ADHD-C and ADHD-

I subtypes may be distinct disorders due to the differences in impairment, prevalence, and gender (Carlson, Shin & Booth, 1999; Milich, Balentine, & Lynam, 2001). ADHD is a pervasive disorder that affects 3 to 7% of the current childhood population (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). Meta-analytic reviews suggest that ADHD-I is the most prevalent of the three subtypes in the general population, representing 44% (Wolraich., Hannah, Pinnock, Baumgaertal, & Brown, 1996) to 51% (Baumgartel, Wolraich, & Dietrich, 1995) of cases, while ADHD-C represents 27 to 33%, and ADHD-H presents 20 to 23% (Carlson, et al., 1999). Prevalence rates of ADHD-C in clinical samples, however, equal or exceed those of the ADHD-I subtype (Faraone, Biederman, Weber, & Russell, 1998) indicating that children with the ADHD-C subtype are more likely to be referred for treatment (Carlson, Shin, Booth, 1999). Early conceptualizations of the disorder suggested that ADHD symptoms, particularly hyperactive-impulsive behaviors, attenuate during adolescence. Lower prevalence rates reported in adult studies relative to child studies contributed to this conceptual framework (Fayyd et al., 2007). More recent examinations of the disorder, however, have found that symptoms continue to manifest and cause significant impairment in multiple adult related settings (e.g., workplace and social settings; Barkley, Murphy, Fischer, 2010). Further, epidemiological data suggests that ADHD continues to affect 36.3 to 70% of individuals after childhood (Harpin, 2005; Kessler, et al., 2006). Consequently, ADHD is a prevalent adult condition that affects 4 to 5% of the adult population (Barbaresi et al., 2013).

In addition to its high prevalence rate, ADHD occurs frequently with other clinical disorders. Available research suggests that approximately 42.7 to 93% of children with ADHD exhibit symptoms and associated impairment consistent with a comorbid

diagnosis of oppositional defiant disorder (ODD) and/or conduct disorder (CD; Biederman et al., 2005; Kuhnes, Schachar, & Tannock, 1997; Spencer, Biederman, & Wilens, 1999). Research also suggests that 15 to 25% of adults with substance use disorders (SUD) of drugs or alcohol would meet criteria for ADHD (Wilens, 2004). Finally, between 13.0 and 50.8% of children with ADHD meet criteria for a comorbid diagnosis of an internalizing disorders (e.g. generalized anxiety, separation anxiety, and major depressive disorder; Jensen, Martin, & Cantwell, 1997; Gau et al., 2010).

**Gender differences in ADHD**. ADHD is most often diagnosed in males compared to females, with a clinical diagnostic ratio of 2:1 in children and 1.6:1 in adults (Gershon, 2002; Lee, Oakland, Jackson, & Glutting, 2008). Females diagnosed with ADHD are more likely to be diagnosed with the ADHD-I subtype relative to males, who are more often diagnosed with the ADHD-H and ADHD-C subtypes (Biederman et al., 2002). However, a previous meta-analytic review indicates that there are no gender differences in impulsivity, academic performance, social functioning, and fine motor skills in individuals diagnosed with the disorder (Gaub & Carlson, 1997b). Furthermore, findings from the meta-analytic review suggest that differences in symptom presentations and referral biases may influence the clinical diagnostic prevalence rates identified in previous studies. For example, hyperactive behavior in females often manifests as hyper-talkativeness and is often seen as less disruptive than excessive motor activity exhibited by males with the disorder. Collectively, differences in perceived disruptiveness indicates a possible gender bias in the observation and reporting of the outward hyperactive symptoms associated with ADHD (Quinn, 2005).

**Negative outcomes associated with ADHD**. A diagnosis of ADHD is associated with an increased risk for a variety of negative outcomes among both child and adult populations. Academic problems are the most common impairment seen in individuals with ADHD and can include underachievement in reading, writing, and mathematics, (Berry, Lyman, & Klinger, 2002; Mannuzza, Klein, Bessler, & LaPadula, 1993; Marshall, Hynd, Handwerk, & Hall, 1997), as well as disruptive classroom behaviors resulting from hyperactivity (Pelham, Foster, & Robb, 2007). Compared to their typically developing peers, children with ADHD have been found to exhibit higher levels of motor activity at all times of the day and most significantly during academic activities related to math and reading (Porrino et al.,1983). Furthermore, increased hyperactive behavior in academic settings directly influences the amount of academic failure in children with ADHD (Cunningham & Barkley, 1978). Problems in academic settings can lead to rejection from typically developing peers and overall low self-esteem (Harpin, 2005). Children with ADHD also experience higher rates of impairment in social functioning with peers (Hoza, 2007), siblings (Mikami, & Pfiffner, 2008), and adults (Wehmeir, Schacht, & Barkley, 2010). These impairments are particularly associated with pervasive hyperactivity and can range from disobedience and noncompliance toward authority, as well as annoying, overbearing and obtrusive behavior towards peers (Pelham, et al., 2007; Keown & Woodard, 2006).

Many of the ADHD-related impairments experienced in childhood continue to follow individuals into adulthood. For example, late adolescents and young adults with ADHD are at an increased risk for dropping out of high school or college (Harpin, 2005). Adults with ADHD also experience difficulties concentrating and remaining vigilant

during driving, and have an increased risk for speeding violations and traffic accidents (Murphy, & Barkley, 1996), as well as negative outcomes in occupational settings (e.g. excessive errors, lateness, interpersonal problems, and problems balancing workload) leading to high rates of suspensions and dismissals (Barkley, Murphy, & Fischer, 2010). Additional research suggests that adults with ADHD are at a higher risk, relative to their healthy peers, for developing a substance use disorder. The lifetime prevalence rates of substance use disorders in adults with ADHD is approximately 52%, compared to only 27% in adults without the disorder (Biederman et al., 1995).

Excessive hyperactivity is one of the most predictive ADHD symptoms of lifelong negative outcomes (Gaub & Carlson, 1997; Faraone, et al., 1998; Hinshaw, 2002). For example, recent research indicates that hyperactivity and impulsivity in childhood are predictive of future criminal behavior in adulthood (Babinski et al., 1999). Furthermore, teacher rated hyperactivity in adolescents is a significant predictor of violent criminal behaviors and risky substance use in adulthood (Klinteberg, Andersson, Magnusson, & Stattin, 1993). Hyperactive adults are also at a higher risk for teen pregnancy (Harpin, 2005), are fired from mores jobs, have fewer close friends, and are treated more often for sexually transmitted diseases relative to non-hyperactive peers (Barkley et al., 2006).

In addition to being associated with the most severe impairment (Hinshaw, 2002), hyperactivity is often considered a hallmark feature of ADHD and is currently the most prominent symptom associated with clinical referrals due to its disruptive nature in school, workplace, social, and home settings (Sayal et al., 2002). The overall importance of hyperactivity in the diagnostic understanding and subtype classification of ADHD has

resulted in extensive and continued examinations of the symptom's underlying core features and role in the disorder.

**Diagnostic history of ADHD related hyperactivity**. Emphasis on hyperactivity as a core symptom of ADHD has varied throughout the history of the disorder. The first recognized mentioning of ADHD-related symptoms occurred in a medical textbook that detailed attention disorders (Weikard, 1790). In this textbook, conceptualizations related to attention disorders identified distractibility and inattention as the core symptom of these disorders, excluded the inclusion of hyperactive symptoms, noted the problems occurred more often in youth, and defined the cause of the disorder as poor child rearing (Barkley & Peters, 2012). Hyperactivity then became a featured symptom of focus in the early twentieth century medical and clinical literature due to research identifying "deficits in moral control of behavior" as the prominent symptoms of the disorder (Still, 1902). For some time, hyperactivity remained the symptom of focus with the creation of several monikers focusing on hyperactivity as the underlying core difficulty. These included "hyperkinetic disease of infancy" (Kramer & Pollnow, 1932), "hyperkinetic disorder" (Laufer, Denhoff, & Solomons, 1957), "hyperactive child syndrome" (Chess, 1960), and the DSM-II diagnosis "hyperkinetic reaction to childhood" (American Psychiatric Association, 1968). The underlying causes of hyperactivity associated with these monikers varied from social factors such as lack of purposefulness and high distractibility (Kramer & Pollnow 1932), to biological factors such as minimal brain dysfunction and prenatal "cerebral hypoxic lesions" (Towbin, 1971).

Views of the disorder shifted in the early 1970's to focus on inattention and impulsivity as the core symptoms (Douglas 1972; Campbell, Douglas, & Morgensterr,

1971; Lange et al. 2010). Ultimately, this shift led to the creation of the DSM-III diagnosis of attention deficit disorder (ADD), which excluded excessive hyperactivity as a diagnostic criterion (American Psychiatric Association, 1980). The change stemmed from research comparing attentional difficulties in ADHD to attentional difficulties in learning disorders and to typically developing children (Douglas, 1972; Campbell et al., 1971). The removal of hyperactivity as a core symptom sparked notable controversy and critique claiming a lack of empirical evidence to justify the change (Barkley, 2006). The diagnostic criteria for ADHD was subsequently revised in the DSM-IV to include three subtypes --Predominately hyperactive/impulsive type (ADHD-H), Predominately inattentive type (ADHD-I), and Combined type (ADHD-C). (American Psychiatric Association, 1994). The creation of these subtypes emerged from the use of rating scales and clinical field trials larger than previous DSM field trials, and were based on findings from cluster and factor analytic examinations of structured diagnostic clinical interviews. Further, these research findings indicated different symptom presentations but similar levels of impairment across subtypes (Lange, Reichl, Lange, Tucha, & Tucha, 2010; Lahey et al, 1994).

The diagnostic description and criteria for ADHD, including the inclusion of hyperactivity as core symptom, remained unchanged in the text revision of the fourth edition of the Diagnostic and Statistical Manual of Mental disorders (2000). Most recently, the diagnostic criteria for ADHD was updated in the release of the DSM-5 to generalize hyperactive symptoms to affected adults (American Psychiatric Association, 2013). These changes have included recognizing impairment in work situations and indicating that symptoms of the disorder must be present before the age of 12 years old,

rather than 7 years old. In addition, adults and late adolescents are only required to experience five symptoms rather than the six required for a diagnosis in childhood (American Psychiatric Association, 2013).

Appendix B

## Theoretical Models of ADHD

Due to the pervasive nature and high prevalence rates of ADHD, a growing body of literature on the core deficits/endophenotypes of the disorder has developed. Endophenotypes are features measurable on the cognitive or neurobiological level, are less genetically complex, and are considered to be closer to biological etiologies of clinical disorder than behavior phenotypes (Gau & Shang, 2010). Various conceptualizations of the underlying core deficits of ADHD have further developed into well-researched models of the disorder. Many existing models of ADHD feature similar core deficits but vary on which processes play central versus secondary roles in the disorder (Rapport et al., 2008).

**Behavioral inhibition model**. One of the most researched models of ADHD is Barkley's Behavioral Inhibition model (1997). Barkley's model holds that deficits in behavioral inhibition are the primary underlying cause for deficiencies in the executive functions such as working memory, reconstitution, internalization of speech, and self-regulation, which in turn leads to deficient fine and gross motor control (Barkley, 1997). Behavioral inhibition is the ability to inhibit prepotent responses, the ability to stop an ongoing response, and the ability to control interference (Barkley, 1997; Bronowksi, 1977; Fuster, 1989; Logan, Cowan, & Davis, 1984). Individuals with deficits in interference control are unable to prevent irrelevant stimuli and information from gaining access to executive functions' resources, such as working memory (Brocki et al., 2008).

Barkley's (2007) model implies that hyperactivity is a ubiquitous symptom of the ADHD resulting from inability of the executive function systems to control off task

activity (Alderson, Rapport, & Kofler, 2007). However, recent research examining behavioral inhibition and executive processes indicates that behavioral inhibition fails to account for deficits in working memory, suggesting that behavioral inhibition is downstream of other executive functions (Alderson, Rapport, Hudec, Sarver, & Kofler, 2010). Furthermore, research focusing on excessive motor activity associated with ADHD suggests that increased activity is functionally related to non-inhibitory working memory functions (Rapport et al., 2009) and that motor activity does not significantly differ during inhibitory and non-inhibitory conditions (Alderson, Rapport, Kasper, Sarver, & Kofler, 2012).

**Cognitive energetic model**. Sergeant's (2000) cognitive energetic model of ADHD hypotheses three levels of deficits that result in ADHD symptoms (Sergeant, 2000). The first level of the cognitive energetic model identifies a "lower" set of information processes that include encoding information, central processing for memory searching and decision making, and response/motor organization (Sergeant & van der Meere, 1990). At this level, deficits in the motor organization subsystem influence ADHD-related motor activity (Sergeant, 2000; Sergeant & van der Meere, 1990). The second level of Sergeant's model describes energetic pools that are further broken into the subsystems of arousal, activation, and effort (Sergeant, 2000). At the second level, the cognitive energetic model holds that ADHD deficits relate to activation and effort pools. The activation pool represents physiological activity and effort pools are described as the necessary energy to complete and meet the demands of a task (Pribram & McGuinness, 1975; Sergeant, 2000). Finally, the last level of the model includes management and evaluation mechanisms (i.e. planning, and detecting; Sergeant, 2005) that are associated

with executive functioning concepts outlined in Barkley's (1997) behavioral inhibition model (Sergeant, 2000; Sergeant, Oosterlaan, & van der Meere, 1999). Collectively, the cognitive energetic model of ADHD suggests that problems in the activation and effort pools account for the primary deficits associated with ADHD, while secondary ADHD deficits are associated with impairments occurring at other levels (Sergeant, Oosterlaan, van der Meere, 1999). The CEM is difficult to evaluate empirically, however, due to a lack of adequate measures available that can assess the arousal, activation, and effort concepts. Without these measures available, many conclusions made by the model are hypothetical; most notably, no testable prediction related to the purposed role of excessive motor activity in ADHD has been proposed based on this model (Sergeant, 2005, Rapport et al., 2009).

**Dual pathways model**. Sonuga-Barke's (2003) dual pathways model of ADHD describes two psychophysiological pathways based in separate, yet related, brain circuits (Songua-Barke, 2003). Distinctly different psychological processes mediate both pathways but have a common neurobiological framework. Furthermore, while the dual pathway model of ADHD indicates the possibility of other social or environmental pathways involved in ADHD deficits, the executive and reward circuits are thought to underlie the primary endophentypes of the disorder (Songue-Barke, 2005). The first pathway identified in this model, the executive circuit, is modulated by mesocortical dopamine. Deficits in this area can lead to executive and inhibitory problems associated with ADHD. The second identified pathway is the reward circuit, which is modulated by mesolimbic dopamine. Deficits in this area are hypothesized to lead to problems with delay aversion (Songue-Barke, 2005). Within this model, children with ADHD are

hypothesized to have a greater emotional and motivational sensitivity to situations that contain a delay, and therefore attempt to escape or avoid delays. According to the dual-pathway model, impulsiveness occurs when individuals choose relatively immediate-small rewards to reduce or eliminate delays, while excessive motor activity serves as a distraction in situations where individuals are forced to wait (Songue-Barke, 2002). These predications, however, focus primarily on deficits experienced in childhood ADHD and fail to account for continued deficits and excessive motor activity into adulthood.

**Neurodevelopmental Model**. Halperin & Schulz's (2006) neurodevelopmental model of ADHD was formulated in response to other neurological models of ADHD that suggest the primary deficits of the disorder are related to the development of the prefrontal cortex and its connections with the striatum (Himelstein, Schulz, Newcorn, & Halperin, 2000; Schultz, Himelstein, Halperin, & Newcorn, 2000; Swanson & Castellanos, 2002). The need for this revised model arose from emerging evidence indicating that the developmental course and symptom manifestation of ADHD is inconsistent with the development of the prefrontal cortex (Benes, 1989; Hynd at al., 1993) and executive functions (McKay, Halpperin, Schwartz, & Sharma, 1994; Welsh, Pennington, & Groisser, 1991). Furthermore, research examining early prefrontal cortex damage in young children has indicated that children who experience this damage do not always exhibit or develop symptoms of ADHD (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Tripp & Aslop, 1999). This suggests that prefrontal damage or underdevelopment is not a sufficient explanation for ADHD symptomology in and of itself. Additionally, some evidence suggests that the development of the prefrontal cortex

over time and its overall plasticity might be responsible for the remediation of ADHD related symptoms. The revised neurodevelopmental model of ADHD instead suggests that a wide variety of neurological deficits, lesions, or underdeveloped regions could contribute to the manifestation of ADHD symptoms. Moreover, the development of the prefrontal area of the brain is responsible for the overall improvement and reduction of ADHD related symptoms over time (Halperin & Schultz, 2006). However, the lack of specificity and large number of suggested neurological deficits that could be associated with ADHD requires significant and extensive future research to narrow down the possible neurological manifestations of ADHD. Furthermore, the neurodevelopmental model fails to provide testable hypotheses regarding the relationship between executive deficits and increased motor activity, or provide an explanation for the general increased activity levels of children and adults with ADHD.

**Functional Working Memory Model.** Rapport and colleagues' (2008) model of ADHD suggests that biological factors, such as genetics, influence the neurobiological system of WM and result in impaired CE functioning and a limited storage capacity in the PH and VS systems. These impairments are hypothesized to be the underlying core deficits of the disorder, which in turn result in problems with basic learning and attention abilities (see Figure 2). Notably, Rapport's model of ADHD is the only model of ADHD that provides specific and testable hypotheses that explicitly explain hyperactive behaviors in children and adults with the disorder.

Experimental examinations of the functional WM model have found deficits in all three WM subsystems in children with ADHD, with the largest deficits appearing in the CE component (Alderson et al., 2010; Kofler, Rapport, Bolden, Sarver, & Raiker, 2010;

Rapport et al., 2008; Rapport et al., 2009). Meta analytic reviews of WM and ADHD have yielded moderate to large effect sizes (ES) associated with the unique components of WM (Kasper, Alderson, & Hudec, 2012; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) and identified a variety of potential moderating variables (Kasper et al., 20112). Specifically, Kasper and colleagues found that studies with a relatively low percentage of females in the sample, greater numbers of experimental trials, high demands on the CE, and required recall rather than recognition processes, were associated with larger magnitude between-group PH and VS effect sizes. Further, findings from the review, via the "best case estimate" procedure, indicated that studies that include fewer females, younger children, recall tasks, large number of trials, high CE demands, and stimuli correct as the dependent variable, are expected to yield VS and PH effect sizes of 2.15 and 2.01, respectively (Kasper, Alderson, & Hudec, 2012).

Previous studies have also demonstrated that WM impairments underlie DSM-5 defined core and secondary features of ADHD. For example, in an examination or WM deficits and social skills, WM was found to have an indirect relationship with parent and teacher reported social skills deficits, such that WM deficits in switching and dividing attention result in inattentive, impulsive, and hyperactive behavioral outcomes that subsequently affect social interactions (Kofler et al., 2011). Academic performance has also been linked to WM deficits, suggesting that WM deficits are risk factors for poor academic achievement in children with ADHD (Alloway, Gathercole, & Elliot, 2010; Rogers, Hwang, Toplak, Weiss, & Tannock, 2011). Specifically, PH WM deficits appear to be associated with lower achievement in reading and mathematics, while VS WM

deficits are associated with lower achievement in mathematics only (Rogers et al., 2011). More recent experimental examinations of the functional WM model in relation to other models of ADHD suggest that WM deficits underlie behavioral inhibition deficits, which were previously considered the core deficits in children with ADHD (Alderson et al., 2010). Specifically, results revealed that the CE and VS WM mediated the relationship between task performance and group membership, while behavioral inhibition mediated the relationship between the CE and group, but could not account for VS or PH deficits in children with the disorder. Additionally, an examination of the relationship between WM, behavioral inhibition, and objectively measured impulsivity suggests that accounting for CE WM attenuates group differences in impulsivity in children with ADHD and healthy controls (Raiker, Rapport, Kofler, & Sarver, 2012). Collectively, these findings provide empirical support for behavioral inhibition deficits serving as secondary symptoms of ADHD accounted for by limited WM resources.

A variety of neurological studies also provide strong support for WM deficits in ADHD. For example, a meta-analytic review examining potential neural correlates associated with ADHD has found notable hypoactivity in the anterior cingulate, dorsolateral prefrontal, inferior prefrontal, and orbitofrontal cortices in the frontal lobe (Dickstein, Bannon, Castellanos, & Milham, 2006). More general electroencephalography (EEG) examinations have found increased slow wave activity in the frontal lobe regions of children (Chabot & Serfontein, 1996) and adults with ADHD (Loo & Barkley, 2005). Furthermore, magnetic resonance imaging (MRI) studies have found that cerebellar volumes as well as deceased frontal grey matter significantly correlate with parent and clinician rated measures of ADHD symptomology, and

individuals with ADHD have significantly reduced volume in in all brain regions (Castellanos et. al., 2002). Finally, an examination of neural activity and WM performance in healthy controls and adults with ADHD found that adults with ADHD exhibited decreased activity in both the cerebellar and occipital lobes while completing a 2-back WM task (Valera, Faraone, Bierderman, Poldrack, & Seidman, 2005), consistent with predictions from Rapport and colleagues' WM model of ADHD.

Findings from studies examining neuropsychological and WM deficits in adults with ADHD have concluded that overall WM deficits persist into adulthood (Alderson, et al., 2013; Alderson, Kasper, Hudec, & Patros, 2013; Hervey, Epstein, & Curry, 2004; Hudec, Alderson, Patros, & Kasper, 2014). While earlier meta-analyses (Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005; Hervey et al., 2004; Schoechlin & Engel, 2005) focused primarily on gross neuropsychological performance deficits, the most recent meta-analysis focused primarily on ADHD-related WM deficits in adulthood. Overall, adults with ADHD exhibited moderately decreased performance on both PH and VS WM tasks when compared to healthy controls. Furthermore, best-case estimate procedures outlined by this meta-analysis generated large effect estimates (ES= 1.44 for PH-WM, ES=1.22 for VS-WM) when utilizing best practice moderators (Alderson et al., 2013). More recent experimental research used methodology consistent with the best estimate procedures outlined by Kasper and colleagues (2012) and found that adults with ADHD experience large-magnitude PH deficits and moderate-magnitude VS deficits, relative to health-control adults (Alderson et al., 2013). Moreover, examination of WM components revealed that adults with ADHD exhibit the greatest deficits associated with CE and PH storage/rehearsal processes.

## Appendix C

### Theoretical Model of Working Memory

**Cowan's embedded-processes model.** Cowan's embedded- processes model of WM (1988) defines WM as a cognitive process that maintains easily accessible mental information (Cowan, 2005). The embedded- processes model holds the WM processes are not a part of a separate system, but instead are activations of specific areas of the long-term memory. Additionally, these activation areas decay over time unless they are maintained by continued attention or verbal rehearsal (Cowan, 1988). Cowan's model emphasizes controlled-focus of attention which includes both voluntary and involuntary processes (Cowan, 1999), and has the capacity to hold and manipulate four items or chunks of items (Cowan, 2005). While Cowan's model is fundamentally different from Baddeley's working memory model, Cowan's recognition of the importance of verbal rehearsal can be seen as similar to the phonological loop (Baddeley, 2010).

**Individual differences models.** Individual differences theories combine experimental and correlation methods to address why some individuals are able to maintain higher levels of cognitive performance across WM-span tasks. These models stemmed from research on the capability of WM-span tasks to predict individual abilities (Daneman & Carpenter, 1980), and focus on a variety of explanations for individual variability in cognitive performance. One such explanation focuses on an individual's ability to use gaps between stimuli presentation in span tasks to rehearse information and avoid a time-related decay in the storage components of memory (Barrouillet, Bernardin, & Camos, 2004). Another similar explanation focuses on an individual's ability to resist time decay and become efficient in switching between tasks that require WM (Towse,

Hitch, & Hutton, 2000). Other models emphasize an individual's ability to ignore interference (Saito & Miyake, 2004), or an individual's inhibitory ability which can remove potential disruption from the WM system (Engle & Kane, 2004).

**Computational models.** Computational models of WM attempt to provide a more detailed account of WM with the use of computer simulations (Baddeley, 2012). Originally, these complex models attempted to account specifically for language processes, but eventually developed to include other aspects such as motor control, emotion, and awareness (Bernard, 1987). In addition, some computational models distinguish procedural and declarative WM. In these representations, procedural WM is associated with processing and manipulating information, while declarative WM is responsible for maintaining and providing information to procedural WM. Furthermore, both subsystems are comprised of an activated portion of long-term memory, direct access or binding regions, and a specific focus of attention (Oberauer, 2010). While these models provide changes or reconceptualization to concepts established in Baddeley's (2007) multi-component model of WM, they have not yet gained strong empirical support (Baddeley, 2012).

**Baddeley's multi-component model.** Baddeley's multi- component model defines working memory as an executive function responsible for the temporary storage, maintenance, and manipulation of visual and auditory information (Baddeley, 2007). WM is divided into four distinct subcomponents including the visuospatial (VS) sketchpad, the phonological (PH) loop, the domain-general central executive (CE) system, and the episodic buffer. The PH loop is responsible for the temporary storage, rehearsal, maintenance, and processing of auditory information, while the VS sketchpad

is responsible for the temporary storage, rehearsal, maintenance, and processing of visual and spatial information. The CE is responsible for allocating resources to the VS and PH systems, as well as dividing, switching, and maintaining attention, and manipulating information that is temporarily stored in the VS and PH rehearsal systems (Baddeley, 2007). The newer episodic buffer component is responsible for the temporary storage of information presented via multiple modalities, and provides a link between short term and long-term memory. Findings from neuroimaging studies suggest the PH loop is associated with the left temporoparietal region, the VS sketchpad is associated with similar areas in the right hemisphere, and the CE component is most recognizably associated with the frontal lobes (Baddeley, 2007; Henson, 2001; Jonides et al., 1993; Paulesu, Frith, & Frckowiak, 1993; Smith & Jonides, 1997). A visual schematic of Baddeley's multi-component model of working memory is provided in Figure 1.

## Appendix D

### Working Memory and ADHD Related Hyperactivity

Correlational studies that have examined the relationship between behavioral measures of hyperactivity and WM performance have produced somewhat mixed conclusions. For example, WM deficits correlate highly with attention span and distractibility, but not hyperactive behaviors on rating scales (Alloway et al., 2009; Gathercole et al., 2008; Martinussen & Tannock, 2006; Willcutt et al., 2005). However, important confounds in these studies may have resulted in a failure to identify a relationship between motor activity and WM. Specifically, these studies utilized rating scales that asked parents and teachers to approximate children's activity level in multiple settings throughout the day rather than the specific time the children were completing WM tasks. Retrospective approximation of activity levels may have resulted in deflated activity scores due to lower activity levels exhibited in tasks that require little WM processing. Additionally, observer bias related to perceived hyperactivity could have artificially deflated overall activity scores. Finally, previous research has indicated notably low agreement ($r=0.32$ to $r=0.58$) between subjective and objective measures of motor activity (Rapport, Kofler, & Himmerich, 2006), suggesting subjective measures of motor activity could misrepresent or under represent actual motor activity levels.

The first experimental study of the relationship between activity and situational/cognitive demands investigated the activity of boys with and without ADHD across multiple home and academic settings (Porrino et al., 1983). Motor activity in both groups was measured via an acceleration sensitive device placed around the waist for a 24 hour, seven-day period. Results indicated that boys with ADHD displayed higher

activity levels compared to their typically developing peers during all times of the day including sleep and weekend activities. Additionally, boys with ADHD displayed the most significantly elevated levels of activity during school-based activities, specifically those including reading or mathematics. This finding was the first to suggest variations in cognitive demands were functionally related to increased motor activity. Several limitations of the study, however, precludes strong conclusions. For example, only waist-based motor activity levels were examined, which may fail to account for excessive activity in the extremities resulting in artificially deflated activity levels. Furthermore, this study did not report on-task versus off-task behavior during school-based activities, which limits the study's ability to examine relationship between activity and tasks that require executive functions. This information is necessary to determine the relationship between motor activity and attentional demands.

A more recent experimental study examined the functional relationship between motor activity and demands on the WM system in children with and without ADHD (Rapport et al., 2009). Activity was objectively measured with actigraphs placed on the non-dominant wrist and both ankles of participants, while children completed control, VS-WM, and PH-WM tasks. Results indicated that all children exhibited significantly more activity during WM conditions, relative to control conditions, and that children with ADHD exhibited disproportionately higher levels of motor activity during WM conditions, compared to their typically developing peers. Further analyses revealed that increased activity was related to CE processing but not PH or VS storage/rehearsal processes. These findings supported Rapport et al.'s (2008) hypothesis that ADHD-related hyperactivity is functionally related to environmental demands on CE functioning.

A subsequent study examined motor activity in children with ADHD and their typically developing counterparts during a control tasks, a stop signal task, a choice-reaction time task where they were asked to ignore an auditory tone, and a choice-reaction time task without an auditory tone (Alderson et al., 2012). Results indicated that motor activity in children with ADHD disproportionately increased in relation to typically developing children during tasks that placed the greatest demand on controlled-focused attention (CE), rather than behavioral inhibition or control conditions (Alderson et al., 2012).

Although findings from a recent meta-analytic review suggest that adults with ADHD experience large-magnitude PH and VS WM performance deficits (Alderson et al., 2013; Hervey et al., 2004; Schoechlin & Engel, 2005), relatively few studies have examined the relationship between motor activity and WM demands in samples of adults with the disorder. The first of these studies examined the motor activity of 20 healthy controls and 20 adults with ADHD while they completed a VS *n*-back WM task (Lis et al., 2010). Motor activity was measured by calculating head movements greater than 1mm. Additionally, time active, distance of movement, and spatial area travelled were examined to gain a more detailed picture of activity levels in both groups. Overall, it was determined that adults with ADHD were approximately 3.5 times more active than their healthy control counterparts. However, several limitations influence the possible conclusions that can be drawn for this study. For example, the exclusive use of a VS WM tasks, in lieu of a PH WM task, precludes inferences about the relationship between motor activity and PH working memory processes. Additionally, the use of only a 1-back WM task lacks the ability to examine changes in motor activity as WM load increases or

decreases. Even more, the study's measurement of head movement fails to account for more finite movement in the extremities.

A more recent study found that adults with ADHD exhibit significantly higher levels of objectively measured motor activity while completing both PH and VS WM tasks compared to healthy controls (Hudec et al.,2014). In this study, motor activity was measured via actigraphs located on the participants' non-dominant hand and two ankles. These actigraphs were able to measure finite motor activity in the extremities resulting in a more accurate measure of overall motor activity. Notably, both groups exhibited greater activity during WM conditions in relation to control conditions. Further, the CE and PH subsystems accounted for the largest between-group differences in activity.

Collectively, findings from carefully-controlled studies of children (Alderson et al., 2012; Porrino et al., 1983; Rapport et al., 2009) and adults (Hudec et al., 2014; Lis et al., 2010) with ADHD suggest that ADHD-related motor activity is functionally related to variability in working memory demands, consistent with Rapport and colleagues' (2008) functional working memory model of ADHD. Previous studies, however, have not included other clinical groups (e.g., anxious). As a result, it is not clear whether the relationship between WM demands and motor activity is specific to ADHD, or psychopathology more generally.

Notably, excessive motor activity such as restlessness and fidgeting are not pathognomonic symptoms of ADHD, and are often associated with other psychiatric disorders such as mania (Weiss, Foster, Reynolds, & Kupfer, 1974), depression (Avery, & Silverman, 1984), and anxiety (Tryon, 2009). In particular, restlessness is one of six symptoms used to diagnose Generalized Anxiety Disorder (GAD) in the DSM-5

(American Psychiatric Association, 2013). Further, emerging research suggests that PH WM storage/rehearsal processes are adversely affected by articulatory suppression related to ruminations or anticipatory processing associated with anxiety disorders (Derakshan & Eysenck, 1998). The phenotypic similarities of increased motor activity and WM impairments that are present in both ADHD and anxiety disorders suggests that the established relationship between WM demands and motor activity in ADHD might also be generalizable to anxiety disorders. However, to date, no studies have examined the possible relationship between WM deficits and variability in anxiety-related motor activity.

## Appendix E

### Anxiety Disorders Brief Review

*Anxiety disorders* is a blanket term used to categorize a variety of disorders where pathological anxiety is a core feature (Emillien, Durlach, Lepola, & Dinan, 2002). Feelings of anxiety are often divided into two subcategories, "subjective" (cognitive) feelings such as fear, worry, and terror (Stone, 2010), and "objective" (somatic) feelings such as nausea, abdominal pain, and restlessness (Berrios, Link, & Clark, 1995; Emillian et al., 2002). These disorders are often chronic lifelong conditions (Anthony & Setin, 2009) associated with impairment in daily functioning and frequent use of primary psychological care (Kessler & Greenberg, 2002; Baxter, Scot, Vos, Whiteford, 2013).

Contemporary research and clinical conceptualizations of anxiety disorders have varied with regard to which disorders fall under the anxiety disorder classification (Andrews, 1996; Tryer, 1989). Many of these concerns relate to the overlapping features some anxiety disorders share with other diagnoses and to the possibility of compromised discriminate validity that comes with expanded nosologies (Brown & Leyfer, 2009). The current DSM-5 identifies 12 separate disorders that fall under the category of anxiety disorder: separation anxiety disorder, selective mutism, specific phobia, social anxiety disorder, panic disorder, panic attack, agoraphobia, generalized anxiety disorder, substance/medication induced anxiety disorder, anxiety disorder due to other medical condition, other specified anxiety disorder, and unspecified anxiety disorder (American Psychiatric Association, 2013).

**Gender differences in anxiety disorders.** Anxiety disorders are one of the most prevalent mental conditions affecting around 1 in 5 adults in the population (Kessler et

al., 2005). Substantial sex differences in prevalence rates have been observed in research on all anxiety disorders (Yonkers & Kidner, 2002), and generally, approximately 31% of females and only 19% of males will experience an anxiety disorder in their lifetime (Kessler et al., 1994). Furthermore, females, compared to males, are twice as likely to have experienced anxiety by the age of 6 years (Lewinsohn, Gotib, Lewinsohn, Seeley, &Allen, 1998). Gender differences might be explained by the higher frequency rates of anxiety vulnerable alleles in females over males (Altemus & Epstein, 2007). Alternatively, women's more frequent hormonal changes across their lifespan could account for possible dysregulations in the complex neurochemical subsystems of the brain (Altemus & Epstein, 2007; Altemus et al., 2004). Further, females diagnosed with an anxiety disorder are more likely to develop additional anxiety disorders and depressive disorders than males, although males diagnosed with anxiety disorder are more likely to be diagnosed with comorbid ADHD and substance use disorders then females (McLean, Asnaani, Litz, & Hofmann, 2011). These differences in comorbidities are likely related to the gender differences in the prevalence rates of other psychological disorders.

**Brief diagnostic history of anxiety disorders.** Recognition of anxiety as an impairing disorder dates back to Greek classical age (Stone, 2010). In early medical literature, conditions describing anxious states were often identified as having possible connections with physical symptoms (Berrios & Link, 1995, Burton, 1621). However, these early writings did not specifically use the term "anxiety" (Stone, 2010). The first mentioning of anxiety as an abnormal condition identified a person as in an "anxious state" if they "[trouble] themselves with everything" (Flecknoe, 1658). As the medical and mental health fields progressed, a variety of terms such as "panophobia", "nervous

disorders", and "neurosis" were used to describe psychological anxiety (Bienvenu, Wuyek, & Stein, 2010; Stone 2010).

The first edition of the *Diagnostic and Statistical Manual of Mental Disorders'* (American Psychiatric Association, 1952) "psychoneurotic disorders" category was used to classify "anxiety reaction", "depressive reaction", "phobic reaction", "conversion reaction", "obsessive compulsive reaction", and "dissociative reaction"(American Psychiatric Association, 1952). This broad category reflected the belief that these types of symptom clusters were a result of a condition of the nerves or neurosis (Bienvenu et al., 2010). The predominance of the term neurosis continued into the second edition of the DSM with the creation of the neuroses disorders category (American Psychiatric Association, 1968), which continued to include depressive and dissociative conditions. Due to the psychoanalytic implications of the "neurosis" moniker, the term was retired for the publication of the DSM-III, and anxiety disorders received their own category and became separate from depressive and dissociative disorders based on research suggesting core differences between the disorders (Fava, Rafanelli, & Tossani, 2008; Bienvenuet al., 2010). In later editions of the DSM, the anxiety disorders category remained largely unchanged until the publication of the DSM-5, which further divided anxiety disorder into anxiety disorders, obsessive-compulsive disorders, and trauma and stressor disorders (American Psychiatric Association, 2013). This division was based on an increased focus on the distinctiveness of these disorders but the interest in keeping the disorders interconnected (Kupfer, Kuhl, & Regier, 2013).

**Negative outcomes associated with anxiety.** A variety of negative outcomes have been associated with the presence of anxiety disorders, such as increased risk of

long term mental health problems (Berg et a., 1989; Keller et al., 1992; Pine et al., 1998). Adolescents and young adults with anxiety disorders are at an increased risk for developing depressive disorders, nicotine dependence, alcohol dependence, narcotics dependence, suicidal behavior, educational underachievement, and early parenthood (Woodward & Ferguson, 2001). Additionally, adults with anxiety disorders experience significantly higher levels of work impairment (Mendlowicz & Stein, 2000), and substantially lower enjoyment, satisfaction, and quality of life compared to healthy adults (Rapaport, Clary, Fayyad, & Ednicott, 2005). Anxiety disorders also account for a large economic burden for individuals as research indicates the cost associated with anxiety disorders in the United States is over 46.6 billion dollars annually, 31.5% of total cost of all mental disorders (DuPont et al., 1996; Rice & Miller, 1998).

**Working Memory and Anxiety**

Specific examinations of the WM system in individuals with anxiety have identified multiple deficits. For example, in a study involving traditional digit span and sequencing tasks, highly anxious individuals experienced a significant decline in performance associated with both storage/rehearsal and central executive (manipulation) processes of WM (Darke, 1988). Further, individuals with high trait anxiety experience a decrease in reading-span task performance in stressful environmental conditions. In non-stressful conditions, however, individuals with high trait anxiety performed better on span tasks than individuals with low trait anxiety indicating that situational stressors effect WM capacity in anxious individuals (Sorg, & Whitney, 1992). Additionally, highly anxious individuals, compared to their non-anxious counterparts, exhibit slower response times (MacLeod, & Donnellan, 1993; Miyake et al., 2000) and report experiencing

greater feelings of worry and cognitive self-concern, during the completion of WM tasks (Ikeda, Iwanaga, & Seiwa, 1996). More recently, an examination of WM in individuals with social anxiety found that WM load moderates attentional bias such that socially anxious individuals have problems disengaging or avoiding negative stimuli when WM load increases (Judah, Grant, Lechner, & Mills, 2012). These findings suggest that WM influences cognitive control and may limit executive resources associated with attentional control in anxiety disorders. Finally, a recent meta analytic review of anxiety, working memory, and academic performance found that high levels of anxiety, and specifically test anxiety, creates deficits in the CE component of WM and leads to a decrease in academic performance (Owens, Stevenson, Hadwin, & Norgate, 2012). While there are few studies examining WM deficits and generalized anxiety disorder (GAD) specifically, GAD is sometimes considered the clinical manifestation of high trait anxiety (Rapee, 1991) indicating that findings associated with individuals with high trait anxiety could be applied to the disorder.

**Working Memory and Theoretical Models of Anxiety**

Recently, theoretical models of anxiety have begun to focus on the relationship between anxiety and executive functions. These more general models of anxiety focus primarily on states of anxiety experienced within the normal population (Eysenck, Derakshan, Santos, & Calvo, 2007). Although these models provide theoretical conceptualizations of anxiety, no models to date provide possible explanations for restless motor behavior or its relationship with WM.

**Processing efficiency theory.** The processing efficiency theory (Eysenck & Calvo, 1992) of state anxiety has contributed theoretical explanations of state anxiety and

its relationship with cognitive performance. This model emphasizes the difference between task effectiveness and efficiency. *Effectiveness* is described as the quality, accuracy, or correctness of task performance while *efficiency* is described as the effort or resources utilized to complete a task. Moreover, the processing efficiency model of anxiety suggests that anxiety more directly and negatively effects task efficiency over task performance or effectiveness. In relation to WM, this model identifies worry and worrisome thoughts as a primary component of state anxiety, which directly interferes with the limited-resource CE component of WM, which in turn decreases its availability for task completion. The PH-WM system is also affected by articulatory suppression occurring from internal verbal worries (Rapee, 1993).

Although the processing efficiency theory of anxiety provides a hypothesis for the relationship between state anxiety, WM, and cognitive performance, several limitations warrant consideration. For example, the model fails to provide specific explanations related to how and to what extent state anxiety decreases the efficiency of the CE. Furthermore, the model fails to account for the performance of anxious individuals when presented with distracting and/or threating stimuli. This limitation is notable due to the evidence suggesting that cognitive performance of anxious individuals is more affected by distracting (Calvo & Eysenck, 1996; Hopko, Ashcraft, Gute, Ruggiero, & Lewis, 1998) and threatening (Egloff & Hock, 2001; Judah et al., 2012) stimuli then their non-anxious peers. In addition, contrary to predictions from the processing efficiency theory of anxiety, existing studies have found that anxious individuals often out-perform their non-anxious counterparts (Byrne & Eysenck, 1995; Sorg & Whitney, 1992; Standish &

Champion, 1960). Finally, the processing efficiency theory of anxiety does not explain why anxious individuals exhibit restless/increased motor activity.

**Attentional control theory.** The attentional control theory of anxiety (Eysenck et al.,2007) was developed to address the limitations of the processing efficiency theory and to primarily focus on the relationship between attentional control, cognitive performance, and anxiety. Generally, the attentional control theory of anxiety suggests that anxiety decreases the CE's attentional control and focus. More specifically, the attentional control theory identifies two distinct attentional systems (Corbetta & Shulman, 2002): a goal directed system focused on current goals, and a stimulus directed system focused on directed attention to relevant stimuli. The model hypothesizes that increased anxiety increases focus on the stimulus driven system, rather than the goals driven system, resulting in deficits in CE processes such as inhibition (i.e., ability to inhibit prepotent responses and inhibit attention away from task irrelevant stimuli), shifting (i.e., using attentional control to switch between multiple tasks), and updating (i.e., updating and modifying stimuli within the WM system; Miyake et al., 2000). Moreover, impairments in inhibition increase when CE demands increase (Graydon & Eysenck, 1989), and result from anxious individuals' inability to control prepotent responses to task irrelevant stimuli. Impairments and slowing in shifting between tasks occur when anxiety interferes with the overall efficiency of attentional control (Eysenck et al., 2007). Finally, impairments in the updating function of the CE are hypothesized to only occur during stressful situations since updating does not directly require attentional control (Miyake et al., 2000). However, anxious individuals experience increased demands on the CE system

during stressful situations, which can result in lowered efficiency and in turn, can lead to impaired updating abilities.

Unlike the processing efficiency theory, the attentional control theory of anxiety explains the seemingly paradoxical finding that anxious individuals often outperform non-anxious individuals on WM tasks. That is, the attentional control theory of anxiety suggests that anxiety impairs efficiency over effectiveness, suggesting that anxious individuals must allocate more effort/resources to regulation of attentional focus (Eysenck et al., 2007). It is noted that this emphasis on efficiency over effectiveness implies that anxious individuals might engage in some type of compensatory strategy to maintain task performance.

While the attentional control theory of state anxiety provides a more detailed explanation of the relationship between anxiety, WM, and cognitive performance, a number of limitations reduce its usefulness. Specifically, the model fails to generalize its theoretical implications to clinical manifestations of anxiety and does not consider other possible cognitive deficits, beyond attentional control, that may influence anxiety's relationship with executive functioning. Furthermore, while this model implies the possibility of compensatory strategies used to maintain cognitive effectiveness, it fails to address the possible relationship between excessive motor activity and WM.

**Working Memory and Theoretical Models of GAD**

While there are several theories aimed at identifying the relationship between broad anxiety and cognitive functioning, there are several more specific and prevalent models aimed at developing theoretical conceptualizations on GAD.

**Avoidance worry model**. The avoidance worry model (Borkovec, 1994) suggests that worry manifests as verbal thoughts. This worry interferes with emotional processing and is negatively reinforced by creating avoidance of negative or aversive stimuli. Furthermore, individuals with GAD participate in cognitive avoidance that involves using strategies such as distraction to avoid threating or aversive stimuli. Although this model does not specifically mention WM in relation to GAD, the linguistic based worry and cognitive avoidance identified in the model implies the possibility of internal articulatory suppression that can interfere with PH-WM storage rehearsal processes. However, the model does not mention or account for the diagnostic criteria of restlessness or excessive motor activity commonly associated with GAD.

**Intolerance of uncertainty model.** The intolerance of uncertainty model (Dugas & Keroner, 2005) also highlights the importance of cognitive avoidance, but suggests that individuals with GAD experience chronic worry in situations that could be considered ambiguous or uncertain. The worry related to uncertainty in turn leads to orientations towards negative problems and cognitive avoidance. Individuals who are oriented to negative problems often experience lack of confidence, frustration, and pessimism which can influence cognitive or task performance. The intolerance of uncertainty model does not explicitly address WM deficits in relation to GAD; however, explanation of chronic worry suggests a relationship to overall cognitive performance. Further, similar to the avoidance worry model, this model fails to mention possible explanations for restlessness in GAD.

**Metacognitive model of GAD**. The metacognitive model of GAD (Wells, 2005) focuses on two distinct types of worry experienced by individuals with GAD. The first

type of worry is related to external causes or events, and while the second type of worry is frequently referred to as "worry about worry" (Wells, 2005), or worry centered on the uncontrollable nature of worry. The latter type of worry often leads to poor coping strategies that center on reducing emotional reactions and controlling worrisome thoughts. These ineffective coping strategies often reinforce worry when they fail and can influence general functioning. Like other models of GAD, however, the metacognitive model does not specifically address WM performance deficits, and does not mention or explain GAD-related restlessness/hyperactivity.

**Emotion dysregulation model.** The emotion dysregulation model suggests that there are four key components to the theoretical understanding of GAD (Mennin, Turk, Heimberg, & Carmin, 2004). These components have a basic foundation in conceptualization of emotional dysregulation in borderline personality disorder (Linehan, 1993) The components in this model state that individuals with GAD experience hyper arousal or intense emotions, have a poor understanding of their own emotions, have negative beliefs about emotions in general, and have maladaptive emotion regulation skills. This model primarily focuses on emotions and negative affect with little emphasis placed on cognitive processes. Additionally, no explanation is provided for the function of restless motor activity in the disorder.

**Acceptance-based model.** The acceptance-based model (Roemer & Orsillo, 2005) of GAD involves the recognition of internal experiences, a problematic relationship with these experiences, experiential avoidance, and behavioral restriction (Behar, DiMarco, Hekler, Mohlman, & Staples, 2009). This model hypothesizes that individuals with GAD recognize their internal experiences, have a negative reaction to

these experiences, and subsequently try to avoid these experiences both cognitively and behaviorally (Roemer & Orsillo, 2005). The avoidance is reinforced by the reduction of anxious feelings and behavior, and consequently increases over time. Similar to the emotion dysregulation model, the acceptance based model primarily explains internal experiences and emotions, and fails to provide clear explanations for cognitive functioning or the function of restlessness in GAD.

Collectively theoretical conceptualizations of GAD do not specifically explicate WM deficits that have been reliably observed in individuals with the disorder in previous studies (Behar et al., 2009). Moreover, no extant model/theory of GAD provides an explanation or conceptualization for the role of restlessness and fidgeting behaviors commonly associated with the disorder. Since restlessness or fidgeting currently serves as a key diagnostic feature of GAD, failure to include the symptom in the theoretical conceptualizations of the disorder results in less than complete understanding of the underlying features or causes of GAD

**Excessive Motor Activity and Anxiety**

While restlessness serves as diagnostic feature of GAD (American Psychiatric Association 2013), remarkably few studies have examined objectively measured motor activity in GAD, and no studies to date have examined objectively measured motor activity outside of the context of sleep. That is, examinations of sleep disturbances in anxious individuals have indicated that persistent worry and trait anxiety predict nighttime restlessness and sleep latency as measured by actigraphs (Tryon, 2009; Wicklow & Espie, 2000). However, due to the lack of research examining daytime motor activity in anxious individuals, outside of anecdotal inferences, the extent to which

anxiety disorders are associated with elevated motor activity (i.e., restlessness and fidgeting) is currently unknown. Needless to say, the relationship between anxiety-related WM deficits and activity has also not been investigated in previous research.

# Appendix F

## Oklahoma State University Institutional Review Board

| | | | |
|---|---|---|---|
| Date | Monday, May 06, 2013 | **Protocol Expires:** | **5/5/2014** |
| IRB Application No: | AS0948 | | |
| Proposal Title: | Attention-Deficit/Hyperactivity Disorder (ADHD) in Late Adolescence: An Examination of Potential Core Deficits | | |
| Reviewed and Processed as: | Expedited<br>**Continuation** | | |
| Status Recommended by Reviewer(s) | **Approved** | | |

Principal Investigator(s)

R. Matt Alderson
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Approvals are valid for one calendar year, after which time a request for continuation must be submitted. Any modifications to the research project approved by the IRB must be submitted for approval with the advisor's signature. The IRB office MUST be notified in writing when a project is complete. Approved projects are subject to monitoring by the IRB. Expedited and exempt projects may be reviewed by the full Institutional Review Board.

☒ The final versions of any printed recruitment, consent and assent documents bearing the IRB approval stamp are attached to this letter. These are the versions that must be used during the study.

The reviewer(s) had these comments:

New subject enrollment still in progress; addition of 40 participants to study is approved.

Signature : Shelia R. Kennison

Shelia Kennison, Chair, Institutional Review Board

Monday, May 06, 2013
Date

VITA

Sarah Elizabeth Lea

Candidate for the Degree of

Master of Science

Thesis: AN EXAMINATION OF THE ROLE OF WORKING MEMORY DEMANDS ON OBJECTIVELY MEASURED MOTOR ACTIVITY IN ADULT ADHD, GAD, AND HEALTHY CONTROL GROUPS

Major Field: Psychology

Biographical:

Education:
Completed the requirements for the Master of Science in Psychology at Oklahoma State University, Stillwater, Oklahoma in May, 2016.

Completed the requirements for the Bachelor of Arts in Psychology at University of Alabama, Tuscaloosa, Alabama in 2013.

Experience:

| | |
|---|---|
| Oklahoma State University Center for Research of Attention and Behavior | 2013- Present |
| University of Alabama ADHD and Anxiety Program | 2011-2013 |
| Institute for Social Sciences Research | 2010-2011 |

Professional Memberships:

| | |
|---|---|
| Association of Psychological Sciences Student Member | 2013- Present |
| Psychology Graduate Student Association | 2013-Present |