THE ROLE OF EXPECTATIONS ON ATTENTION PERFORMANCE

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AD/HD medications are shown to be significantly more successful at enhancing attention/concentration performance in individuals with AD/HD than placebo treatments. Few studies, however, have investigated the possibility of a placebo reaction in both medication and placebo groups by comparing placebo treatments to no treatment at all. Using an undergraduate population, I evaluated the effect of expectations about a treatment's efficacy on performance in an attention/concentration task. In addition to cognitive performance outcome measures, I included several physiological measures, such as heart rate variability (HRV) through respiratory sinus arrhythmia (RSA). Contrary to expectations, no differences were observed in performance on attention tasks or physiological measurements as a result of the believed efficacy of an orally administered placebo treatment.
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CHAPTER 1

THE ROLE OF EXPECTATIONS ON ATTENTION PERFORMANCE

Deficits in attention and concentration, such as attention deficit/hyperactivity disorder (AD/HD), affect nearly 5% of the adult population worldwide (Polanczyk, Silva de Lima Lessa Horta, Biederman, & Rohde, 2007). Impairments in this area include poor attention span, difficulty focusing, and impulsivity (Diagnostic and Statistical Manual of Mental Disorders, 4th ed. text rev. [DSM-IV-TR]). Individuals with deficits in attention and concentration tend to overlook details and make careless mistakes. As a result, their work is often messy and haphazard (Wilens, 2009). Additionally, people with these deficits have difficulty finishing tasks they have begun, often times engaging in new endeavors before prior ones are completed (Wilens, 2009; DSM-IV-TR). Such tendencies can impede academic and occupational performance, and hinder social interactions (Teeter, Rumsey, Natoli, Naylor, & Smith, 2000; DSM-IV-TR).

Despite being categorized in the DSM-IV-TR as a mental disorder, attention problems such as AD/HD are more often diagnosed by primary care providers (PCP) than by psychologists or psychiatrists (Bussing, Zima, & Belin, 1998). Moreover, many of these PCP’s rely on patients’ self-reports to diagnose AD/HD, rather than using psychometrics designed for assessing attention and concentration (Wasserman et al., 1999). In fact, of 3,900 physicians surveyed, only 38% used DSM-IV-TR criteria in their diagnosis of AD/HD. The other 62% surveyed used “clinical intuition,” instead of proper AD/HD assessment (Wasserman et al., 1999). This lack of thorough testing can sometimes lead to incorrect diagnoses of AD/HD. Manic episodes associated with bipolar disorder, for example, are sometimes mistaken for the hyperactive component.
seen in AD/HD (Spencer, Biederman, Wozniak, Faraone, Stephen, Wilens, & Mick, 2001; Biederman, 1998). Hence, bipolar disorder is sometimes misdiagnosed as AD/HD (Spencer et al., 2001; Biederman, 1998). Misdiagnoses such as these can lead to ineffective treatments, which may prolong or even exacerbate the expression of symptoms (Perlis, 2005; Biederman, 1998).

Treatment and AD/HD

According to the National Institute of Neurological Disorders and Stroke (NINDS), there is no "cure" for AD/HD. However, pharmacological treatments such as Ritalin (Methylphenidate), and Adderall (Amphetamine) are two of many medications used to treat AD/HD symptoms (Shillington, Reed, Lange, Clapp, & Henry, 2006). Though they are successful at mitigating inattention and impulsivity, many stimulant medications include undesirable side effects such as decreased appetite, headache, nausea, insomnia, and irritability (Faraone, Glatt, Bukstein, Lopez, Arnold, Findling, 2008; Stein, 1999; Cantwell, D. P., 1996). Despite these unwanted reactions, nearly 60% of people with AD/HD who take these drugs report substantial improvement in areas of attention and concentration (Polanczyk et al., 2007; Shillington, Reed, Lange, Clapp, & Henry, 2006). The components of AD/HD medications have a similar effect on the body as cocaine, often resulting in chemical addiction (Prudhimme White, Becker-Blease, & Grace-Bishop, 2006). In fact, the cocaine-like properties and performance enhancing effects of these medications have led to widespread abuse, particularly among college students (Shillington, Reed, Lange, Clapp, Henry, 2006). A 2002 study indicted that, of
the 150 undergraduate college students surveyed, 35% reported using prescription amphetamines illicitly (Low, Gendaszek, 2002).

Though effects of amphetamine medications on attention and concentration performance are evident, the efficacy of their chemical makeup alone is unclear. Many physicians believe that the success of these drugs is due solely to a chemical reaction that increases the concentration of dopamine and norepinephrine: two neurotransmitters associated with attention (Biederman, 2005). Others, however, contend that the expectations of individuals taking these medications may play an important role as well (Barrilleaux & Advokat, 2008).

In a 2008 study investigating whether adults and children with AD/HD differ in the accuracy of their performance on attention tasks, Barrilleaux and Advokat found that adults contributed their success on the tasks to their medication as well as their effort, whereas children attributed their success to effort alone. This led the authors to suspect that adults with AD/HD may have an inordinate reliance on their medication for improved task performance. To investigate this suspicion Barrilleaux and Advokat suggest that future research include a placebo condition, which may reveal the degree to which expectation in the effects of the medication influenced performance.

Physiology and Attention

Studies indicate that lower heart rate variability (HRV: variation in length of intervals between heart beats) is correlated with increased attention and concentration on a given task (Duscheck, Muckenthaler, Werner, & Reyes del Paso, 2009; Boger, Meer, Ronner, Alberts, Geuze, & Botge, 1998). Duscheck et al., (2009) investigated
several facets of autonomic reactivity in relation to cognitive performance. Among other parameters, HRV decreased markedly during tests requiring attention. Boger et al., (1998) conducted a similar study and found that HRV also indexes effort allocation on continuous performance tests (CPT’s); i.e., the more effort an individual allocates in an attention task, the lower their recorded HRV. The current study used respiratory sinus arrhythmia (an indicator of HRV) as an objective marker of attention and effort allocation on an attention task. In addition, other physiological measures, such as blood pressure (BP), and cardiovascular output (CO) were explored.

Expectations and Outcomes

Prior research suggests that individuals perform in accordance with their expectations (Bandura, 1996; Reinhard & Dickhäuser, 2009; Carver et al., 1979). Rand (2009) looked at the relationship between hope and academic performance among 345 undergraduate students, and concluded that hope directly raises expectations, which leads to better performance. Reinhard and Dickhäuser (2009) supported this finding, but argued that expectations predict performance only when executing a challenging task. Similarly, a 1998 meta-analysis examining the relationship between self-efficacy and performance found an average of 28% increase in performance due to self-efficacy on a given task (Stajkovic & Luthans, 1998).

Self-efficacy is not the only factor that affects the way expectations influence outcomes. Shiv, Carmon, and Ariely (2005) found that pricing can alter the perceived and actual efficacy of a product. In their study, Shiv et al., offered the same energy drink at a "reduced price" and a "regular price." According to the authors, customers
who bought the drink at the reduced price benefited less from the product (i.e., felt less alert, were able to solve fewer puzzles) than those who bought the drink at its regular price. This variance in product efficacy is thought to be due to the consumer’s expectations of the product’s effect, which were higher or lower depending upon the monetary value given to the product.

Such effects of commercial features apply to expectations of the efficacy of medications as well. A study conducted in 2006 recruited volunteers to “test” the effectiveness of an experimental “pain medication,” which was actually a placebo pill: a treatment thought to have no inert powers, such as a sugar pill or saline drip. Of the 82 volunteers who received the placebo, half were told that the regular price of the drug was $2.50 per pill. The other participants were told that the drug was currently discounted to $0.10 per pill (no explanation for the discount was given). Though all participants received identical placebo pills, those who took the “discounted medication” reported significantly less pain reduction than those who received the “medication” at its supposed “regular cost” (Waber, Shiv, Carmon, et al., 2006).

Reported reactions that occur in response to placebo treatments (as seen in the aforementioned studies) are thought to be due to a person’s expectations of the efficacy of that treatment (Stewart-Williams, 2004). For this reason, placebo reactions are often dismissed as faulty assessments of a subjective experience. However, placebo treatments have been shown to influence physiological reactivity, as well as subjective relief. For example, a 2004 study using functional magnetic resonance imaging (fMRI) to examine the variability in pain response regions of the brain in response to heat pain and a topical placebo cream, found that expectations of pain relief were associated with
reductions of neurological activity in areas of the brain involved in experiencing pain (Wager, Rilling, Smith, Sokolik, Casey, Davidson, et al., 2004). Furthermore, a 2001 study investigating the placebo effect in patients with Parkinson’s disease found that participants who received the placebo treatment showed a similar physiological reaction to those who receive the standard Parkinson's medication: A significant increase of dopamine in an area of the brain that contributes to motor function; resulting in fewer tremors and more motor control (Fuente-Fernández et al., 2001; de la Fuente-Fernández et al., 2004). In accordance with prior studies (Bandura et al., 1996; Carver et al., 1979; Reinhard, M., & Dickhauser, 2009), these authors reported that the degree to which dopamine was increased within a participant was highly correlated with his or her expectations of clinical improvement. Findings such as these indicate that the placebo effect may do more than simply influence subjective outcomes. It may, in itself, have the power to be physiologically beneficial.

Placebo responses are generally experienced immediately after treatment is administered (Stewart-Williams, 2004; Wager et al., 2004; Zubieta et al., 2006), but the long-term effects of these treatments are less well known. In a 2004 study, McRae et al. conducted a double-blind-follow-up to investigate quality of life (QOL) in patients with advanced Parkinson’s disease who had undergone either a real or "sham" surgery one-year prior. Both surgeries were part of a trial designed to measure the efficacy of embryonic dopamine neuron transplantation on patients with advanced Parkinson’s disease (Freed, Greene, Breeze, Tsai, DuMouchel, & Kao et al., 2001). McRae et al. found that participants who believed they had received the transplant reported a higher QOL, regardless of which treatment they actually received. Furthermore, medical staff
reported congruent ratings of QOL upon examining the participants. As such, McRae et al. concluded that changes in QOL after 12 months were more dependent upon the perceived treatment rather than the actual treatment a patient received (McRae, Cherin, Yamazaki, Diem, & Vo et al., 2004). The influence that expectations have on performance and symptom relief has been documented in academic environments (Bandura et al., 1996; Reinhard et al., 2009), athletic performance (Lee, 1982), and numerous areas of medicine (Fuente-Fernández et al., 2001; Mcrae et al., 2000; Mayberg, Silva, Brannan, Tekell, et al., 2002; Zubieta et al., 2006). Few studies, however, have investigated the effects of performance expectations on attention tasks. Waschbusch et al. (2009) reviewed some such studies in which children with ADHD were administered placebo treatments in place of a stimulant medication. According to the authors, these studies indicated an average of 20% to 30% improvement in ADHD symptoms after placebo treatments were administered (Waschbusch, Pelham, Waxmonsky, & Johnston, 2009; Swanson et al., 1995; Ottenbacher & Cooper, 1983). The authors intimate, however, that these improvements were based on caregiver reports rather than self-reports or objective measures, and that observed positive effects may be the result of caregiver expectations rather than actual symptom correction (Waschbusch et al., 2009).

When compared to placebo treatments, medications such as Ritalin and Adderall, are shown to be significantly more successful at enhancing attention/concentration performance. Few studies, however, have investigated the possibility of a placebo reaction in both medication and placebo groups by comparing placebo treatments to no treatment at all (Suchman & Adler, 1992). Using an
undergraduate population, I evaluated the effect of expectations about a treatment's efficacy on performance in an attention/concentration task. In addition to cognitive performance outcome measures, I included several physiological measures, such as blood pressure (BP) heart rate (HR) and RSA. The aim of this study was to observe differences in performance on concentration and attention tasks as a result of the believed efficacy of an orally administered placebo treatment. Additionally, this study examined whether individuals who believe they received a medication showed a significant reduction in HRV, as is consistent with literature on physiological components of attention (Duscheck et al., 2009; Boger et al., 1998).
CHAPTER 2

METHODS

Participants

Eighty participants (calculated by G-Power for a statistical power of .95), ages 18 and over were drawn from a large pool of students at the University of North Texas. Eligibility criteria included (a) no prior diagnosis of attention deficit disorder and (b) no current medications taken for problems in the area of attention. This last eligibility criterion was included to assure that no observed enhancements in attention performance were due to AD/HD medications. Participants were randomly assigned to one of four experimental conditions, and received course credit for their participation.

Measures

Conner’s Adult Attention Rating Scale – Self-Report, Screening Version (CAARS-S:SV)

The CAARS-S:SV is used to quantitatively measure AD/HD. This measure uses a self-report assessment that focuses on DSM-IV-TR criteria for quick AD/HD identification. This measure contains a 12-item AD/HD index, as well as the DSM-IV AD/HD Symptom Subscales. Normative data for the CAARS-S:SV were based on 1,026 nonclinical adults.

Number Span Forward (NSF)

This subtest was adapted from the Wechsler Adult Intelligence-Fourth Edition (WAIS-IV) Digit Span subtest, which is a commonly used measure of attention that requires participants to attend to, and recall a series of numbers read aloud by the
examiner. In this assessment, the examiner says a series of numbers in one-second intervals, in a monotone voice. The examinee must repeat the numbers back in the same order they were presented.

**Number Span Backward (NSB)**

This subtest, also adapted from the WAIS-V Digit Span subtest, is similar to NSF in administration, but requires examinees to repeat the numbers to the examiner in backward sequence. In both NSF and NSB, the string of numbers increases until the examinee incorrectly responds to two consecutive numerical sequences. The WAIS-IV Digit Span subtest is often used to assess for AD/HD and other attention processes, and is a good indicator of rout memory as well as working memory.

**Self-Report Performance Questionnaire (SRPQ)**

Participants were asked to indicate how well they felt they performed on the attention tasks. Choices included: worse than usual; same as usual; and better than usual. The survey also asked participants to indicate to what degree the treatment aided in their performance on the task (not at all; a little; somewhat; a great deal).

**Physiological Measures**

An impedance cardiograph (Mindwave Technologies, Ltd.) was used to assess the electrocardiogram (ECG), basal thoracic impedance ($Z_0$) and first derivative of the impedance signal (dZ/dt). Three electrodes were placed on each participant (right collar bone, left lower rib, and right lower rib) for ECG collection. To measure impedance,
additional electrodes were placed on the suprasternal notch, ziphosternal junction and corresponding locations on the back. A 4mA AC current passed through these electrodes allowed for the collection of impedance and the derivation of $Z_0$ and $dZ/dt$. Stroke volume (SV) was derived through the Kubicek equation (Sherwood, Allen, Fahrenberg, et al., 1990), and cardiac output (CO) was estimated using the equation $HR \times (SV/1000)$. Total peripheral resistance (TPR) was calculated in resistance units (dynes-s X cm-5) using mean arterial pressure (MAP) and applying the equation $MAP/CO \times 80$. Respiratory sinus arrhythmia (RSA), which reflects parasympathetic control of the heart and is derived through digitized interbeat intervals (IBI), was analyzed and corrected for artifacts as per Bernstron, Quigly, Jang, et al. (1990). A “weighted” best algorithm of the IBI series was used to create a heart period time series. Sharp transitions in the heart period time series were detected using the Bernstrom et al. (1990) algorithm and removed by smoothing. Very large ultralow frequency trends were removed from the input signal via a high pass filter by fitting and subtracting a first order linear polynomial from the heart period time series (Litvack, Oberlander, Carney, et al., 1995). Fast Fourier transform was used to calculate the power spectrum of the heart period time series scaled to msec²/Hz. RSA was averaged for each minute and calculated as the natural log of the area under the heart period power spectrum within the corner frequencies of the band-pass filter (Litvack et al., 1995).

Procedure

Participants met the examiner at the university’s Psychophysiology Laboratory, where they read and endorsed an informed consent document. Next, I administered the
baseline Number Span (forward and backward) tests. The CAARS-S:SV was then administered. Each participant read a specific script, determined by one of four conditions to which the participant was randomly assigned: 1) medication, 2) placebo, 3) medication or placebo, and 4) control. Participants in Conditions 1, 2, and 3 were given a placebo capsule made of cornstarch (Brondeur, D. W., 1965).

Condition 1 was read a script meant to induce high expectations in the efficacy of the placebo capsule: “I am going to ask you to ingest a trial medication being tested for its performance enhancing properties in areas of attention and concentration.” Condition 2 was told that they would receive a placebo capsule, which would have no impact upon their performance: “I am going to ask you to ingest a placebo capsule. This capsule is made of cornstarch and has no attention/concentration enhancing properties.” Condition 3 was informed that they would either receive a medication or a placebo treatment: “I am going to ask you to ingest a capsule. This capsule will either be a medication being tested for its performance enhancing properties in areas of attention and concentration, or a placebo pill, made of cornstarch, that has no attention/concentration enhancing properties.” Condition 4 was informed that they would not be given the cornstarch capsule: “You have been assigned to the control condition of this study, and will not receive the medication.” After the capsule was administered to Conditions 1-3, participants were hooked up to the impedance cardiograph equipment, and a ten-minute physiological baseline was conducted. Following the physiological baseline, participants once again were administered two Number Span tests. After both administrations were completed, participants were given the SRPQ. In addition,
participants in Condition 3 were asked to indicate whether they believe they received a medication or placebo capsule.

**Physiological Baseline**

Participants were hooked up to the impedance cardiograph equipment in one of two experimental rooms, and were separated from physiological monitors and observed via audio and visual recording equipment. A ten-minute “vanilla” baseline period (Jennings, Kamarck, Stewart, et al., 1992) was conducted during which the participants engaged in a minimally demanding task. Cardiac impedance measures were collected continuously during this period.

**Debriefing**

After the tasks were completed, participants were given a debriefing form informing them of the study's aim, as well as notifying them that the capsule they took was a cornstarch placebo.

**Analytic Strategy**

Consistent with prior recommendations, change scores (i.e., task minus average baseline values) were computed for each physiological measure during the number span forward, number span backward, and recovery periods (Llabre et al., 1991). Change scores within periods were averaged to increase reliability. As baseline levels can affect degree of change (Benjamin, 1967) they were included in analyses of covariance (ANCOVAs). Two-way ANCOVAs, 2 (Condition; Medication, Placebo,
Med/Placebo, Control) X 2 (Sex; Male, Female), were calculated for each physiological variable (i.e., SBP, DBP, MAP, HR, RSA). Significant interaction effects were explicated with individual comparisons (Bernhardson, 1975). Because sex is a well-known moderator of cardiovascular responses it was included as a between-subjects factor in all analyses.

Hypothesis

(1) I hypothesized that participants assigned to the medication condition would perform with significantly fewer errors on the post-manipulation number span tasks than those in other conditions. (2) I also hypothesized that participants in the experimental condition, who believed they received a medication, would perform on the post-manipulation number span tasks with significantly fewer errors than those who believed they received a placebo. (3) I hypothesized that individuals who rated higher on the CAARS-S:SV (as indicated by a greater number of endorsed items on the measure) would perform better in the medication condition on the post-manipulation number span tasks than those in other conditions. (4) I also hypothesized that those who believed they received the medication, as defined by group condition, would exhibit lower RSA, indicating enhanced attention and increased effort on the task, than those who believed they received a placebo or who received no treatment at all.
CHAPTER 3
RESULTS

Baseline Equivalence and Task Effects on Physiological Reactivity

Condition Effects on Pre-Manipulation Task Performance

Associations between performance on the number span tasks as well as perceptions of performance are displayed in Table 1. Performance on the number span forward and number span backwards were moderately associated, $r = .349$. Condition differences on task performance are displayed in Table 2. There were no condition differences in performance on the pre-manipulation number span forward task, all $Fs(3,69) < 2.632, p = ns$. It should be noted that there was a marginally significant condition difference in performance of the pre-manipulation number span backwards task. Specifically, participants in the control condition performed with fewer errors than the medication condition (7.97 vs. 10.0, respectively), $F(1,35) = 5.67, p < .05$. With respect to sex differences, men and women performed equally at baseline on both tasks, all $Fs(3,69) < 1.356, p = ns$. Finally, a condition by sex interaction was observed on performance of the pre-manipulation number span forward task, $F(3,69) = 3.88, p < .05$. This effect appears to be driven by sex differences within the control condition where men performed with fewer errors relative to women (2.88 vs. 5.46), $F(1,17) = 11.01, p < .01$.

Condition Differences in Baseline Physiological Parameters

Condition differences on key physiological variables during baseline are displayed in Table 3 with a comparison of sex in Table 4. No differences were found
between condition or sex for MAP, HR, PEP, RSA, or CO, all Fs(3,69) < 1.65, p = ns. A group difference was observed in DBP, with the medication condition presenting significantly lower DBP than the placebo condition (60.7 vs. 68.9), $F(3,69) = 3.481, p < .05$. Additionally, an expected sex difference was observed in SBP, which was driven by men (115.5 vs. 105.5), $F(1,69) = 17.131, p < .01$.

**Self-Reported CAARS-S:SV Scores and Pre-Manipulation Task Performance**

Self-endorsed AD/HD symptoms on the CAARS-S:SV was not associated with pre-manipulation performance on the number span forward or backward tasks, all $r$'s < .17, $p = ns$. Likewise, when the pre-manipulation number span tasks were examined as possible predictors of the CAARS-S:SV score, no relationship was found; multiple $R = .17$, $R^2 = .029$, $F(2,75) = 1.12, p = ns$.

**Task Performance and Physiological Response**

Repeated-measures ANCOVA was used to examine whether the task was associated with increases in CVR. As shown in Figure 1, cardiovascular responses (SBP, DBP, MAP, and HR) increased from baseline to the number span tasks and then decreased during recovery, all $F$'s(3,76) > 4.43, $p < .05$. No increase, however, was found in CO or SV during the tasks, all $F$’s(3,76) < .45, $p = ns$. Contrary to expectations, RSA did not decrease significantly from baseline to the task and then increased during recovery, $F(3,74) = .868, p = ns$.

Next, task performance was examined as a determinant of physiological reactivity using regression where the baseline physiological variable was entered into
the first block with the task performance entered into the second block via the enter method. Although baselines were generally associated with task levels, no associations were found between performance on number span forward and change in SBP, DBP, MAP, HR, PEP, or RSA during the task. However, performance on number span forward was associated with change in CO during the task, \( R^2D = .06, F(1, 73) = 5.69, p < .03. \) Similarly, no associations were found between performance on number span backward and change in SBP, DBP, MAP, HR, PEP, RSA, or CO during the task.

**Post-Manipulation Results**

*Condition Effects on Task Performance*

As shown in Table 1, a strong positive association was observed between the post-manipulation number span forward and backward tasks \( r = .53. \) Contrary to expectations, there was no effect of condition, sex, or their interaction on the performance of post-manipulation number span forward, all \( F_{s}(3, 69) < 1.073, p = ns. \) Similarly, there were no observed main effects of condition or sex on post-manipulation number span backwards, \( F_{s}(3, 69) < .357, p = ns. \) However, a significant interaction between condition and sex was observed, \( F_{s}(3, 69) = 3.72, p < .05. \) As shown in Figure 2, follow-up contrasts revealed that men performed with significantly fewer errors than women in the placebo group (7.2 vs. 9.6), \( F_{s}(1,18) < 6.48, p < .05, \) but with significantly more errors than women in the med/placebo group (9.5 vs. 6.3), \( F_{s}(1,16) < 7.57, p < .05. \)
**Condition Effects on Physiological Responses**

No condition differences were found for SBP, DBP, HR, PEP, RSA, or CO during task performance following the manipulation, all $F$s(3,67) = .717 $p = ns$. However, a condition by sex interaction was observed in MAP during number span forward $F(3,69)$ = 2.932 $p < .05$. As shown in Figure 3, follow-up contrasts revealed that this interaction was present only in the control and placebo conditions, with women exhibiting higher MAP in the control condition (5.25 and -1.21), and men exhibiting lower MAP placebo condition(1.89 and -1.39).

**Condition Differences in Recovery Physiological Parameters**

Condition differences on key physiological variables during recovery are displayed in Table 5. No condition differences were found for DBP, HR, PEP, RSA, or CO during recovery, all $F$'s(3,65) < 2.03, $p = ns$. However, an expected sex difference was found for SBP, with men exhibiting higher SBP (114.23 and 102.64), $F(3,69) = 22.9, p < .05$.

**Self-Reported CAARS-S:SV Scores and Main Task Performance**

As seen in the baseline comparisons, self-endorsed ADHD symptoms on the CAARS-S:SV was not associated with performance on the post-manipulation number span forward or backward tasks, all $r$'s < -.13. Additionally, when the post-manipulation number span tasks were examined as possible predictors of the CAARS-S:SV score, no relationship was found $R^2 = .023, p = ns$. 
Hypothesis Testing

Hypothesis 1

It was hypothesized that participants assigned to the medication condition would perform with significantly fewer errors on the second number span tasks than those in other conditions.

As described above, there was no effect of condition on performance of the post-manipulation trials of number span forward or number span backwards, all $F$'s(3,69) < 1.073, $p = ns$.

Hypothesis 2

It was hypothesized that participants in the experimental condition, who believed they received a medication, would perform on the post-manipulation number span tasks with significantly fewer errors than those who believed they received a placebo.

Unfortunately, only one (1) participant endorsed the belief that they received medication despite condition instructions. Thus, no analyses were performed.

Hypothesis 3

It was hypothesized that individuals who rated higher on the CAARS-S:SV (as indicated by a greater number of endorsed items on the measure) would perform better in the medication condition on the post-manipulation number span tasks, than those in other conditions.

CAARS-S:SV AD/HD subscale scores were coded into two groups: those who scored 60 and above (indicating a belief in significant attention difficulties), and those
whose scores fell below 60 (indicating no significant belief of attention deficits). An ANOVA was then run using the coded groups as categorical independent variables against number span performances as dependent variables. No significant effects were found for either number span forwards or backwards, all $F_s(1,76) < 1.72, p = ns$.

**Hypothesis 4**

It was hypothesized that those in the medication condition would exhibit lower RSA, indicating enhanced attention and increased effort on the task, than those assigned to the placebo or control conditions.

As reported above, there was no condition differences in any of the physiological indices including RSA during task performance following the manipulation, all $F_s(3,67) = .717 p = ns$. 
CHAPTER 4

DISCUSSION

The primary aim of this study was to observe differences in performance on concentration and attention tasks following an orally administered placebo treatment. Using an undergraduate sample of men and women I compared task performance before and after administration of a faux medication. There were four conditions which differed by description of the faux medication such that a quarter of the sample were told either that the administration was indeed a medication to improve attention and concentration, told that it was a placebo which would have no effect, told they would receive either a medication or placebo, and a control condition where participants did not receive the faux medication. Prior to the manipulation, there were no observed condition differences in baseline physiological characteristics or performance on the first trial of the tasks. Contrary to expectations, there were no differences in task performance or in physiological responses to the tasks by condition, following the manipulation. Moreover, where I expected individual differences in self-reported AD/HD symptoms to moderate performance, no such effects were found. These results suggest that within the context of this study, there was not an association between instructions regarding condition and either performance or physiological responses during performance.

One potential explanatory factor for these results is the degree to which participants believed or bought into the manipulation. Although participants were randomized to specific conditions, the efficacy of the study depended upon congruence between their belief in and expectations associated with that condition. As noted above,
only one participant in the ‘medication or placebo’ condition endorsed believing they received a medication. It is possible that this lack of belief in the administered medication was present in the ‘medication’ condition as well. This may be partially responsible for the lack of differences observed in task performance between conditions.

Regarding the physiological findings, a potential explanatory factor may be the method by which the attention task was executed. Prior studies observing RSA in response to attention have used visually based tasks that require sustained visual attention (Duscheck et al., 2009; Boger et al., 1998). The current study differed from prior studies in that the task required a working memory component with an auditory stimulus (i.e., remembering and manipulating numbers read allowed). The lack of findings in the current study suggests that RSA reactivity may be less correlated with attention to auditory, than to visual stimuli. This possibility is corroborated by a 2003 study in which HRV was correlated with a visually based working memory task, but not with a task requiring auditory tone discrimination (Tripathi, Mukundan, & Mathew, 2003).

Additionally, the lack of relationship observed between self-reported AD/HD symptoms and quality of performance on the pre or post-manipulation number span task, in any condition, may indicate that individuals have difficulty accurately gauging their ability to attend and respond to a given task.

Limitations

Variability in observations and effect size between placebo studies have been linked to several distinct moderators including examiner/participant rapport (Kaptchuk, 2002), duration of treatment (Wedge & Keck, 2003), context in which treatment is
received, and the rout by which it is administered (Craen, Tijssen, Gans & Kleigen, 2000). Several of these moderators may have influenced the findings of the current study, including limited interaction between examiner and participant and the use of an orally administered, “single-dose” treatment. Further, the current study was conducted in a psychophysiology laboratory, rather than in a medical setting, such as the student health center. As such, the context in which the placebo was given may have weakened any existing placebo effects.

Several other limitations of the current study may also have influenced the findings. The participants in this study were all drawn from a population of undergraduate psychology students. As such, many of the participants are familiar with placebo research. Given that this study was conducted in the Psychology Department, participants may have anticipated that the “medication” received was indeed an inert substance. Moreover, several participants indicated they had prior knowledge that the treatment given in this study was “fake” suggesting that they had discussed the study with prior participants and learned of the deception. Such an exchange of information between participants may have mitigated the anticipated placebo effect. Prior experience with AD/HD medications may have influenced findings as well. While participants were screened for current AD/HD medication use, they were not screened for prior experience with such medications. It is possible that previous experience with these medications may have interfered with the subjective effects of the placebo treatment (Looby & Earleywine, 2011).
Conclusions

Future studies should control for the aforementioned limitations of the current study. Though the delicate nature of influencing and capturing expectations make the placebo effect difficult to study, the prevalence of medication use (particularly the use of AD/HD medication among college students) make it an important subject for further investigation.

Table 1

*Correlations between Performance on the Number Span Forward (NSF) and Number Span Backward (NSB), as well as Self-Reported Performance (SRPQ)*

<table>
<thead>
<tr>
<th></th>
<th>NSF1</th>
<th>NSB1</th>
<th>NSF2</th>
<th>NSB2</th>
<th>SRPQ1</th>
<th>SRPQ2</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSF1</td>
<td>1</td>
<td>.349</td>
<td>.587</td>
<td>.466</td>
<td>-.137</td>
<td>.039</td>
</tr>
<tr>
<td>NSB1</td>
<td>.349</td>
<td>1</td>
<td>.398</td>
<td>.586</td>
<td>-.151</td>
<td>-.045</td>
</tr>
<tr>
<td>NSF2</td>
<td>.587</td>
<td>.398</td>
<td>1</td>
<td>.525</td>
<td>-.133</td>
<td>-.169</td>
</tr>
<tr>
<td>NSB2</td>
<td>.466</td>
<td>.586</td>
<td>.525</td>
<td>1</td>
<td>.001</td>
<td>-.258</td>
</tr>
<tr>
<td>SRPQ1</td>
<td>-.137</td>
<td>-.151</td>
<td>-.133</td>
<td>.001</td>
<td>1</td>
<td>-.023</td>
</tr>
<tr>
<td>SRPQ2</td>
<td>.039</td>
<td>-.045</td>
<td>-.169</td>
<td>-.258</td>
<td>-.023</td>
<td>1</td>
</tr>
</tbody>
</table>

*Note.* SRPQ1: self-report performance questionnaire, item 1: how well participants believed they performed on the number span tasks before receiving the “medication”; SRPQ2: self-report performance questionnaire, item 2: how well participants believed they performed on the number span tasks after receiving the “medication.”
Table 2

*Condition Differences on Number Span Forward (NSF) and Number Span Backward (NSB) Task Performance*

<table>
<thead>
<tr>
<th></th>
<th>Medication</th>
<th>Placebo</th>
<th>Med/Placebo</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSF1</td>
<td>4.45 (1.76)</td>
<td>4.00 (1.89)</td>
<td>3.74 (1.82)</td>
<td>4.37 (2.09)</td>
</tr>
<tr>
<td>NSB1</td>
<td>4.42 (.41)</td>
<td>4.02 (.41)</td>
<td>3.85 (.43)</td>
<td>4.17 (.42)</td>
</tr>
<tr>
<td>NSF2</td>
<td>3.7 (.37)</td>
<td>3.60 (.36)</td>
<td>3.92 (.38)</td>
<td>3.28 (.37)</td>
</tr>
<tr>
<td>NSB2</td>
<td>8.44 (.60)</td>
<td>8.43 (.59)</td>
<td>7.90 (.63)</td>
<td>7.73 (.61)</td>
</tr>
</tbody>
</table>

*Note. NSF1: Number span forward, trial 1; NSB1: Number span backward, trial 1; NSF2: Number span forward, trial 2; NSB2: Number span backward, trial 2.*

Table 3

*Condition Differences on Key Physiological Variables during Baseline*

<table>
<thead>
<tr>
<th></th>
<th>Medication</th>
<th>Placebo</th>
<th>Med/Placebo</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td>109.47 (2.39)</td>
<td>108.87 (2.35)</td>
<td>111.36 (2.48)</td>
<td>112.48 (2.43)</td>
</tr>
<tr>
<td>DBP</td>
<td>60.90 (1.73)</td>
<td>68.67 (1.73)</td>
<td>65.51 (1.80)</td>
<td>65.85 (1.76)</td>
</tr>
<tr>
<td>MAP</td>
<td>79.15 (1.94)</td>
<td>82.75 (1.91)</td>
<td>80.71 (2.01)</td>
<td>82.19 (1.97)</td>
</tr>
<tr>
<td>HR</td>
<td>73.80 (2.27)</td>
<td>82.81 (2.24)</td>
<td>73.65 (2.36)</td>
<td>74.25 (2.31)</td>
</tr>
<tr>
<td>PEP</td>
<td>111.76 (4.64)</td>
<td>112.43 (4.72)</td>
<td>112.51 (4.82)</td>
<td>113.72 (4.92)</td>
</tr>
<tr>
<td>RSA</td>
<td>10.6 (5.03)</td>
<td>15.70 (5.12)</td>
<td>6.49 (5.23)</td>
<td>6.17 (5.33)</td>
</tr>
<tr>
<td>CO</td>
<td>19.88 (8.82)</td>
<td>34.31 (8.98)</td>
<td>27.40 (9.17)</td>
<td>27.37 (9.34)</td>
</tr>
</tbody>
</table>

*Note. SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure; HR: Heart rate; PEP: Pre-ejection period; RSA: Respiratory sinus arrhythmia; CO: Cardiovascular output.*
Table 4

Sex Differences in Baseline Systolic Blood Pressure (SBP) between Conditions

<table>
<thead>
<tr>
<th></th>
<th>Medication</th>
<th>Placebo</th>
<th>Med/Placebo</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>114.92</td>
<td>112.22</td>
<td>115.63</td>
<td>119.41</td>
</tr>
<tr>
<td></td>
<td>(12.17)</td>
<td>(11.44)</td>
<td>(4.14)</td>
<td>(6.91)</td>
</tr>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP</td>
<td>104.03</td>
<td>105.52</td>
<td>107.10</td>
<td>105.55</td>
</tr>
<tr>
<td></td>
<td>(10.76)</td>
<td>(15.25)</td>
<td>(10.79)</td>
<td>(6.19)</td>
</tr>
</tbody>
</table>

Table 5

Condition Differences on Key Physiological Variables during Recovery

<table>
<thead>
<tr>
<th></th>
<th>Medication</th>
<th>Placebo</th>
<th>Med/Placebo</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SBP</strong></td>
<td>109.64</td>
<td>107.64</td>
<td>109.58</td>
<td>112.48</td>
</tr>
<tr>
<td></td>
<td>(2.39)</td>
<td>(2.36)</td>
<td>(2.49)</td>
<td>(2.43)</td>
</tr>
<tr>
<td><strong>DBP</strong></td>
<td>62.46</td>
<td>68.51</td>
<td>64.04</td>
<td>66.49</td>
</tr>
<tr>
<td></td>
<td>(2.36)</td>
<td>(2.32)</td>
<td>(2.45)</td>
<td>(2.40)</td>
</tr>
<tr>
<td><strong>MAP</strong></td>
<td>79.83</td>
<td>83.12</td>
<td>76.03</td>
<td>80.03</td>
</tr>
<tr>
<td></td>
<td>(2.75)</td>
<td>(2.70)</td>
<td>(2.85)</td>
<td>(2.79)</td>
</tr>
<tr>
<td><strong>HR</strong></td>
<td>58.58</td>
<td>57.51</td>
<td>54.63</td>
<td>58.17</td>
</tr>
<tr>
<td></td>
<td>(1.23)</td>
<td>(1.26)</td>
<td>(1.28)</td>
<td>(1.25)</td>
</tr>
<tr>
<td><strong>PEP</strong></td>
<td>116.46</td>
<td>111.34</td>
<td>118.89</td>
<td>119.61</td>
</tr>
<tr>
<td></td>
<td>(3.49)</td>
<td>(3.55)</td>
<td>(3.71)</td>
<td>(4.92)</td>
</tr>
<tr>
<td><strong>RSA</strong></td>
<td>5.91</td>
<td>13.72</td>
<td>9.46</td>
<td>9.03</td>
</tr>
<tr>
<td></td>
<td>(3.03)</td>
<td>(3.11)</td>
<td>(3.23)</td>
<td>(3.22)</td>
</tr>
<tr>
<td><strong>CO</strong></td>
<td>29.37</td>
<td>47.14</td>
<td>24.42</td>
<td>25.39</td>
</tr>
<tr>
<td></td>
<td>(8.17)</td>
<td>(8.30)</td>
<td>(8.65)</td>
<td>(8.61)</td>
</tr>
</tbody>
</table>

*Note.* SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure; HR: Heart rate; PEP: Pre-ejection period; RSA: Respiratory sinus arrhythmia; CO: Cardiovascular output.
Figure 1. Cardiovascular responses during baseline, number span forward, number span backward, and recovery.

Figure 2. Condition and sex differences on number span backward.
Figure 3. Condition and sex differences in mean arterial pressure (MAP) during number span forward.
REFERENCES


